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REVIEW

The Association of Cerebral Oxygen Desaturation with Postoperative Cognitive Dysfunction in Older Patients: A Review

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Abstract: Postoperative cognitive dysfunction (POCD) is a neurological complication associated with surgery and anesthesia that is commonly observed in older patients, and it can significantly affect patient prognosis and survival. Therefore, predicting and preventing POCD is important. Regional cerebral oxygen saturation (rSO_2) reflects cerebral perfusion and oxygenation, and decreased intraoperative cerebral oxygen saturation has been reported to increase the risk of POCD. In this review, we elucidated the important relationship between the decline in rSO2 and risk of POCD in older patients. We also emphasized the importance of monitoring rSO2 during surgery to predict and prevent adverse perioperative cognitive outcomes. The findings reveal that incorporating intraoperative rSO2 monitoring into clinical practice has potential benefits, such as protecting cognitive function, reducing perioperative adverse outcomes, and ultimately improving the overall quality of life of older adults.

Keywords: anesthesia, surgery, prognosis, perioperative adverse outcome, cognitive function

Introduction

Postoperative cognitive dysfunction (POCD) is a type of cognitive dysfunction associated with anesthesia and surgery. POCD severely affects the quality of life and prognosis of patients and increases postoperative morbidity and mortality, posing a burden on patients, families, and healthcare systems.[1–5] The incidence of POCD following cardiac surgery is 30–65%.[6] Although POCD can occur in patients of all ages after noncardiac surgery, older patients are at a higher risk.[7,8] With the aging population and advancements in medical technologies, the number of older patients undergoing large-scale surgeries has increased; therefore, early prevention and prediction of POCD in older patients are essential.

Regional cerebral oxygen saturation (rSO_2) reflects the supply and demand of cerebral oxygen and brain metabolism; therefore, monitoring rSO_2 is beneficial for the early diagnosis and treatment of cerebral ischemia and hypoxia. Decreased rSO_2 levels have been reported to be associated with the development of neurological complications.[9] A low intraoperative rSO_2 value in older patients is significantly correlated with and a potential predictor of POCD.[10–12] Intraoperative monitoring of cerebral oxygen combined with interventions to mitigate low rSO_2 may reduce the incidence of POCD and improve perioperative outcomes.[13–15] Since intraoperative monitoring of rSO_2 is effective, current studies are exploring the relationship between rSO_2 and POCD. Therefore, this review summarizes the available data on the effect of decreased rSO_2 on POCD in older patients.

POCD

Definition and Diagnosis of POCD

POCD is characterized by impaired cognitive function, including memory, executive function, attention, language, and visuospatial ability,[16] which persists for weeks to months following surgery.[17] In 2018, a multispecialty working group recommended naming POCD based on the clinical nomenclature of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). The authors recommended 'perioperative neurocognitive disorders' as an overarching

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term for preoperative or postoperative cognitive disorders, which provides POCD new specific definitions and criteria. Perioperative neurocognitive disorders include cognitive decline diagnosed before surgery (described as a neurocognitive disorder), any form of acute cognitive decline event (postoperative delirium), and cognitive decline diagnosed up to 30 days (delayed neurocognitive recovery) and 12 months post-surgery (POCD).[18] Several tests are available to assess cognitive impairment in the perioperative period; however, uniform diagnostic criteria are not available for POCD. Neuropsychological tests can be used to assess cognitive function, including the Montreal Cognitive Assessment, Brief Mental State Examination, and Wechsler Memory Scale. In addition, a portfolio of neuropsychological tests is available to assess cognitive status, including the Visual/Auditory Verbal Learning Test, Stroop Color Interference Test, and Conceptual Switching Test. Neuropsychological tests can also be used to assess patients using computers or even tablets.[19–21] In addition, several methods can be used to determine whether a patient has developed POCD, including neurophysiological examinations,[22] transcranial Doppler ultrasonography,[23] and magnetic resonance imaging.[24]

Risk Factors and Pathogenesis of POCD

The risk factors for POCD fall into three categories, namely patients, surgeries, and anesthesia (Table 1). The patient factors include age (> 65 years), educational level, mental health status, electrolyte abnormalities, alcohol or illicit drug abuse, comorbidities, and preoperative cognitive decline. Surgical factors include major surgery (eg, orthopedic and cardiothoracic surgery), severe intraoperative bleeding (> 1000 mL), poor glycemic control, intraoperative hypotension, and hypocapnia. Anesthesia factors include type of anesthesia, depth of anesthesia, anesthetic drugs, and poor pain control.[25–27] Despite these known risk factors, the pathogenesis of POCD remains unclear.

Neuroinflammation, dysfunction of the cholinergic system, danger-associated molecular patterns, neuronal damage, changes in neurotransmitters and synapses, abnormal β-amyloid function, and abnormalities in the microbial–gut–brain axis are associated with the development of POCD.[28–30] Recent studies have shown that decreased cerebral oxygen saturation is a risk factor for POCD.[11,12] Therefore, the relationship between cerebral oxygen levels and POCD has attracted the attention of researchers.

Cerebral Oxygen and Cognitive Function

The adult brain tissue accounts for 2% of the total body mass but consumes approximately 20% of systemic oxygen. The brain tissue is sensitive to ischemia and hypoxia. Monitoring cerebral oxygen levels can reflect changes in oxygen supply and consumption, which provides insights into patient prognosis. Decreased or excessive cerebral oxygen saturation is associated with an increased risk of neurological complications. Previous studies have confirmed that cognitive impairment and severity of cerebral hypoxia are positively correlated.[31–33] However, the mechanism whereby cerebral hypoxia impairs cognitive function is not fully understood but may involve a combination of the following mechanisms (Figure 1).

1. S100 calcium-binding protein A8 (S100A8) is secreted from neurons under hypoxia, which in turn induces neuronal apoptosis via several pathways. For example, S100A8 activates the secretion of tumor necrosis factor-α(TNF-α) and interleukin-6(IL-6) by phosphorylating microglial extracellular signal-regulated kinase (ERK) and c-Jun N-terminal kinase. Furthermore, S100A8 induces the priming of the nucleottide-binding oligomerization domain-like receptor protein 3(NLRP3)

Table 1 Risk Factors for Postoperative Cognitive Dysfunction

Patient Factors	Surgical Factors	Anesthetic Factors	Reference
Age > 65 years Alcohol or illicit drug abuse Mental health status Educational attainment Preoperative cognitive decline Electrolyte abnormalities Malnutrition	Major surgery (orthopedic, cardiothoracic) Severe intraoperative bleeding (> 1000ml) Intraoperative hypotension and hypocapnia Poor blood glucose control	Use of anesthetic drugs Depth of anesthesia Type of anesthesia Pain control	[25] [26] [27]

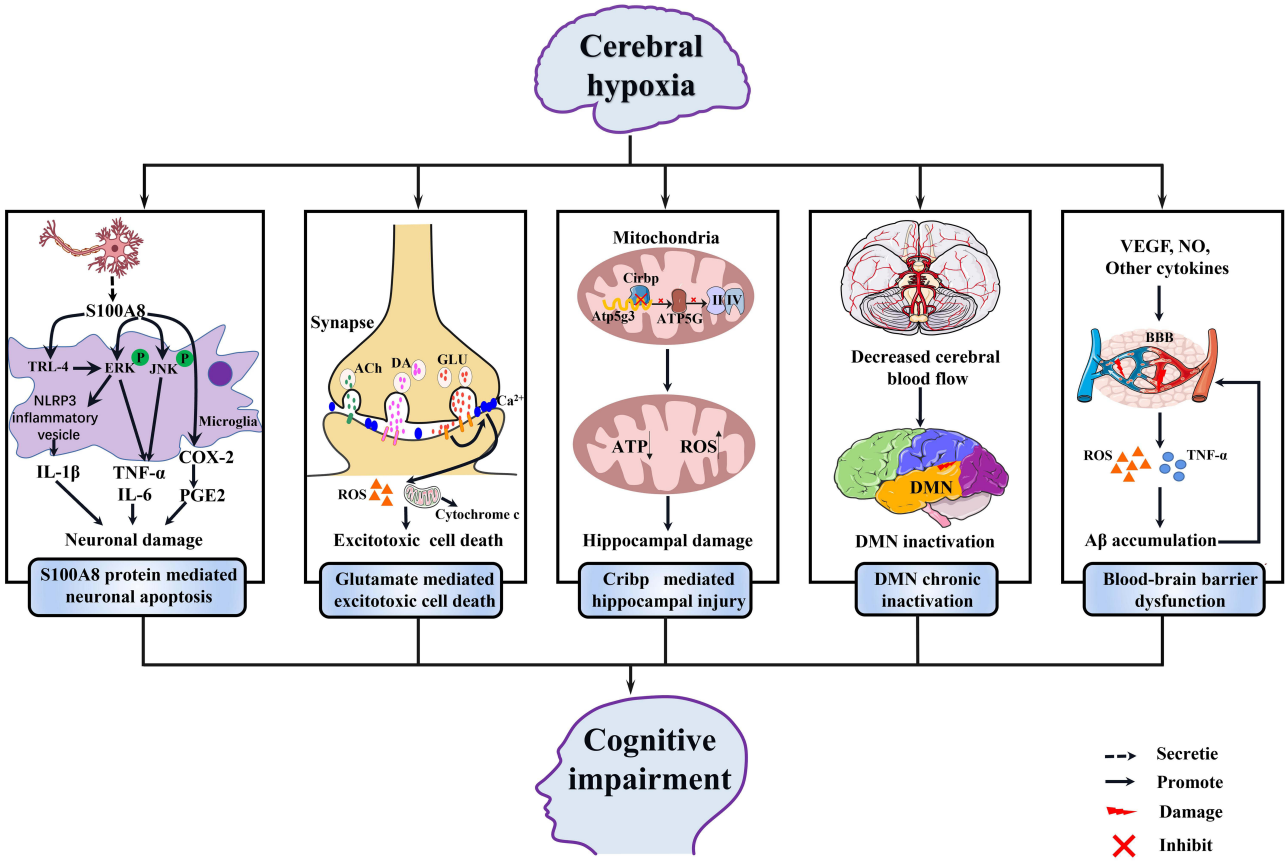

Figure 1 Mechanisms of cerebral hypoxia impairment of cognitive function. Cerebral hypoxai impairs cognitive function via a combination of multiple mechanisms. (1) The S100 calcium-binding protein A8 (S100A8) secreted from neurons under hypoxia induces neuronal apoptosis through several pathways. S100A8 activates ERK, JNK, and the priming signals of the NLRP3 inflammasome through TLR4 receptors in microglial cells. This, in turn, promotes the secretion of TNF-α, IL-6, and IL-1β. In addition, microglial S100A8 expression can activate COX-2 expression and PGE2 secretion. (2) Hypoxia reduces the release of presynaptic membrane acetylcholine and stimulates the release of dopamine and glutamate, ultimately leading to excitotoxic neuronal death and subsequent impairment of cognitive function. (3) Hypoxia reduces Cirbp expression, resulting in reduced ATP production, ROS accumulation, and mitochondrial damage, which leads to damage to the hippocampus and impaired cognitive and memory functions. (4) Cerebral hypoxia deactivates the brain's DMN, causing cognitive impairment. (5) Brain hypoxia damages the blood–brain barrier, which allows for the accumulation of Aβ in the brain and leads to cognitive impairment and dementia.

Abbreviations: S100A8, S100 calcium-binding protein A8; TLR4,toll-like receptor 4; NLRP3, nucleottide-binding oligomerization domain-like receptor protein 3; ROS, reactive oxygen species; ATP, adenosine triphosphate; Ach, acetylcholine; DA, dopamine; GLU, glucose; Cirbp, cold-inducible RNA-binding proteins; ERK, extracellular signal-regulated kinase; JNK, c-Jun n-terminal kinase; TNF-α, tumor necrosis factor-α; IL-1β, interleukin-β; IL-1, interleukin-1; COX-2, cyclooxygenase-2; PGE2, prostaglandin E2; DMN, default mode network; Aβ, amyloid-β; BBB, blood–brain barrier; VEGF,vascular endothelial growth factor; NO, nitrogen monoxide.

inflammasome via toll-like receptor 4(TLR4) -mediated ERK phosphorylation. Under hypoxic conditions, the expression of S100A8 in microglia induces cyclooxygenase-2(COX-2) expression and prostaglandin E2(PGE2) secretion, which in turn induce apoptosis in neurons.[34]

2. Hypoxia reduces the release of presynaptic membrane acetylcholine and stimulates the release of dopamine and glutamate. Increased stimulation of ionotropic receptors by glutamate allows calcium to accumulate postsynaptically, leading to oxidative stress and cytochrome c release from the mitochondria, which trigger excitotoxic neuronal cell death and subsequently impair cognitive function.[35–37]

3. Cold-inducible RNA-binding proteins (Cirbps) promote adenosine triphosphate(ATP) production and eliminate endogenously produced reactive oxygen species. Hypoxia reduces Cirbp expression and impairs the binding of these proteins to Atp5g3 mRNA, thus affecting their expression at the post-transcriptional level and reducing the expression of Cirbp-mediated partial respiratory chain complex subunits. This leads to ATP reduction, reactive oxygen species accumulation, and mitochondrial damage, ultimately causing damage to the hippocampus and impaired cognitive and memory function.[38]

4. The default mode network (DMN) of the brain may be chronically inactivated. The DMN is the most important functional network in the brain associated with cognition and consciousness, and DMN inactivation may lead to cognitive impairment.[39]

5. The release of proinflammatory cytokines, vascular endothelial growth factor, and nitogen monoxide(NO) under hypoxia-ischemia increases the permeability of the blood–brain barrier,[40] which in turn triggers neuroinflammation and oxidative stress, thereby reducing the clearance of amyloid-β (Aβ) and promoting its production in the brain. The accumulation of Aβ in the brain and blood–brain barrier dysfunction can create a feedback loop, causing cognitive impairment and the onset of dementia.[41]

This suggests that the mechanisms underlying cerebral hypoxia-mediated impairment of cognitive function overlap with those involved in the pathogenesis of POCD. Therefore, the correlation between cerebral oxygen and POCD warrants further investigation.

Cerebral Oxygen Saturation Monitoring Technology

rSO_2 monitoring assesses the balance between cerebral oxygen delivery and consumption and has been used in cardiac surgery. rSO_2 monitoring has attracted increasing attention, not only in various major surgeries, such as thoracic, orthopedic, and abdominal surgery but also in the treatment of trauma patients and those in the intensive care unit. There are various methods for monitoring rSO_2 with varying advantages and disadvantages, including near-infrared spectroscopy (NIRS), jugular venous bulb oxygen saturation ($SjvO_2$) monitoring, and brain tissue partial pressure of oxygen ($PbtO_2$) monitoring (Table 2). Other methods have also been developed in recent years, such as electroencephalography, positron emission tomography, functional magnetic resonance imaging, and transcranial Doppler ultrasound.[42–44]

Table 2 Summary of the Advantages, Disadvantages, and Overall Characteristics of Different Cerebral Oxygen Saturation Monitoring Methods in Perioperative Patients

Method	Invasiveness	Advantage	Disadvantage	Reference
NIRS	Non-invasive	1. High security 2. Low cost 3. Real-time monitoring 4. High temporal resolution 5. Significantly reduces the number and severity of perioperative complications during cardiac surgery	1. Signal may be influenced by extracranial tissue 2. Low spatial resolution 3. Low signal-to-noise ratio 4. Baseline variability of rSO_2 between individuals 5. Lack of standardization between different devices 6. Lack of standard threshold and normal range criteria for cerebral hypoxia/ischemia	[44] [45] [46]
$SjvO_2$	Minimally Invasive	1. Simple and convenient 2. Wide range of application 3. Monitoring of whole-brain oxygen saturation 4. Suitable for evaluating capillary hemoglobin concentration	1. Insensitive to local ischemia and hypoxia in brain tissue with prolonged monitoring 3. High $SjvO_2$ values may be associated with pathologic arteriovenous shunting and brain death 4. Extracerebral contamination	[44] [47] [49]
$PbtO_2$	Invasive	1. Simple and convenient 2. High reliability 3. Real-time monitoring 4. With the most reliable evidence base 5. Useful indicators of brain death	1. Longer time is required to obtain valid data 2. Invasive 3. False monitoring results	[40] [50] [51]

Abbreviations: rSO_2, regional cerebral oxygen saturation; $SjvO_2$, jugular venous bulb oxygen saturation; NIRS, near-infrared spectroscopy; $PbtO_2$, brain tissue partial pressure of oxygen.

NIRS-Based Monitoring

NIRS-based monitoring is performed by placing non-invasive electrode pads bilaterally on the forehead while emitting infrared light of different wavelengths through a spectrometer emitter to determine the unique absorption spectra of oxygen, hemoglobin, and deoxygenated hemoglobin in brain tissue. Oxygen saturation is calculated using Cope and Delpy's modified Beer–Lambert law.[45] NIRS monitoring is a simple, non-invasive, and continuous bedside technique for monitoring rSO_2. However, it has some disadvantages, such as low signal-to-noise ratio and spatial resolution. Therefore, the choice of monitoring point impacts the results.[44] Furthermore, potential "contamination" of the signal by extracranial tissue is another challenge, and differences in spectral wavelengths and measurement algorithms used by different devices limit the comparison of the monitoring results between devices.[46]

$SjvO_2$ Monitoring

$SvjO_2$ monitoring was the first bedside monitoring method used for cerebral oxygenation, and this parameter is measured by placing the catheter tip in the jugular venous bulb for intermittent or continuous sampling using a fiber-optic catheter. SjvO2 reflects the dynamic balance between the whole-brain oxygen supply and oxygen consumption, providing a non-quantitative estimate of cerebral perfusion adequacy.[47] The advantage of $SjvO_2$ monitoring is that it can monitor whole-brain oxygen saturation and capture the trend of cerebral oxygen saturation in real time. However, it has some limitations: first, prolonged monitoring may increase the risk of carotid artery puncture, hematoma formation, infection, thrombosis, and intracranial pressure;[48] second, $SjvO_2$ is less sensitive to local cerebral ischemia and hypoxia.[44] In addition, the catheter may compromise $SjvO_2$ measurements, even with a slight deviation from the optimal position, owing to anatomical factors.[49]

$PbtO_2$ Monitoring

$PbtO_2$ monitoring emerged with the development of electronic and fiber-optic technologies, and this parameter is used to monitor rSO2. $PbtO_2$ monitoring is the most reliable method for monitoring cerebral oxygenation.[50] This method allows for the direct measurement of dynamic changes in local $PbtO_2$ values by inserting a polarographic microcatheter into the target brain tissue. $PbtO_2$ reflects the oxygenation, perfusion, and circulatory status of the brain tissue at the cellular level.[51]

$PbtO_2$ monitoring has unique advantages, such as easy operation and high reliability and sensitivity; however, it also has some disadvantages. First, it may lead to erroneous estimation if the microelectrodes are placed in the area of brain injury; second, it is an invasive technique that may cause local damage to brain tissue and increase the risk of intracranial infection; and finally, it is time-consuming.[44]

Association of Cerebral Oxygen Saturation with POCD in Different Types of Surgery

Cardiac Surgery

The incidence of POCD increases following cardiac surgery.[52,53] Cardiopulmonary bypass during cardiac surgery affects oxygen delivery to the brain. Most patients experience one or more episodes of rSO_2 during cardiopulmonary bypass.[54] Patients undergoing cardiac surgery with low rSO_2 are at an increased risk of developing complications, such as respiratory failure, myocardial infarction, and POCD. Therefore, monitoring rSO_2 is a common practice in cardiac surgery.

Although a consensus has not been reached on whether a decrease in rSO_2 during cardiac surgery is correlated with the development of POCD, a number of scholars believe that low rSO_2 is associated with POCD. A randomized controlled trial showed that the incidence of POCD was significantly lower in the intervention groups that maintained rSO_2 > 80% of the baseline values or > 50% of the absolute values compared to that in the control group.[55] In addition, improved cerebral blood oxygenation during cardiac surgery improves neurocognitive outcomes.[56] Qin et al concluded that monitoring the decline in rSO_2 during cardiac surgery could predict the occurrence of POCD.[57] However, scholars have also expressed the opposite view. For example, Semrau et al reported an inconsistent relationship between rSO_2 and neurological complications after cardiac surgery, including stroke, delirium, and POCD;[58] and Zheng et al showed low-level evidence linking low rSO_2 during cardiac surgery with postoperative neurological complications.[59]

Thus, conclusive evidence has not been obtained on the relationship between decreased rSO_2 and POCD following cardiac surgery. However, intraoperative rSO_2 monitoring is important to optimize anesthetic management and improve patient prognosis. The different results of these studies may be attributed to the different definitions and assessment methods of POCD, baseline definitions of rSO_2, and critical thresholds of brain desaturation. Future studies must define the standard baseline rSO_2 and thresholds of cerebral hypoxia and use uniform neurocognitive assessment methods.

Thoracic Surgery

A decrease in rSO_2 during thoracic surgery is correlated with POCD. One-lung ventilation (OLV) is commonly used in thoracic surgery. Patients with OLV develop hypoxemia due to reduced pulmonary ventilation, functional residual air volume, pulmonary arteriovenous shunts, pulmonary ischemia-reperfusion, and systemic inflammatory responses.[60] Hypoxemia underlies the mechanisms that lead to the disruption of cerebral tissue oxygenation. Several studies have confirmed that a decrease in rSO_2 occurs with OLV during thoracic surgery.[28,60,61] Decreased intraoperative $rScO_2$ is associated with an increased incidence of early POCD following thoracic surgery. Tang et al conducted a retrospective study and reported that the timing and extent of $rScO_2$ decline during OLV were associated with early POCD.[62] Li et al found that POCD in older patients undergoing thoracic surgery may be associated with intraoperative rSO_2 decline,[63] whereas Cui et al found that decreased absolute values of cerebral tissue oxygen saturation were associated with cognitive dysfunction.[64]

Given the evidence outlined above, we recommend strengthening OLV management during thoracic surgery. Monitoring rSO_2 changes and addressing rSO_2 may help to avoid brain desaturation and improve postoperative cognitive function and patient prognosis. However, many problems with the use of rSO2 monitoring during thoracic surgery remain to be resolved. For example, different studies have used different methods, with different definitions of brain desaturation and small sample sizes, to measure rSO_2. Therefore, large-scale, standardized, multicenter trials are warranted to define the role of cerebral oxygen saturation monitoring in thoracic surgery.

Orthopedic Surgery

A consensus on the association between decreased rSO_2 and POCD development during orthopedic surgery has not been reached. The beach chair and prone positions are often used in orthopedic surgery, and they can lead to decreased cerebral perfusion and hypoxia and unfavorable neurological complications. rSO_2 decreases when patients are in the beach chair position during shoulder arthroscopy.[65,66]

Larsen et al conducted an observational cohort study and found that POCD in patients undergoing shoulder surgery was associated with low intraoperative rSO_2.[67] Zhu et al found a significant correlation between cognitive dysfunction and rSO_2 in older orthopedic patients 3 months postoperatively.[68] Trafidło et al suggested that the measurement of rSO_2 may help to mitigate postoperative cognitive complications in patients undergoing prone lumbar surgery.[14] Murniece et al found that in patients exhibiting rSO_2 values that decreased by more than 20% from baseline values or values lower than 50% absolute values, intervention may help avoid postoperative cognitive impairment following spinal surgery.[69] Nakao et al conducted a clinical study and observed no significant correlation between cerebral desaturation and POCD during shoulder surgery.[70] Thanaboriboon et al found a high risk of decreased intraoperative saturation during beach chair positioning surgery.[71] However, no association was found between an intraoperative decrease in oxygen saturation and postoperative cognitive decline. Laflam et al suggested that the rSO_2 decline during beach chair surgery did not affect postoperative cognitive function.[72] Therefore, further studies are warranted to confirm whether rSO_2 during orthopedic surgery affects postoperative cognitive function.

Abdominal Surgery

Changes in rSO_2 in older patients undergoing major abdominal surgeries are significantly associated with POCD, and timely interventions can improve neurological outcomes. Li et al found that decreased rSO_2 in hypertensive patients undergoing major abdominal surgery may contribute to early postoperative cognitive decline.[73] Casati et al found that monitoring rSO_2 in older patients undergoing abdominal surgery reduced the occurrence of cerebral hypoxia and may reduce its impact on cognitive function.[74] Yu et al conducted a clinical study and found that serum Aβ levels were

significantly higher and rSO_2 levels were significantly lower in the POCD group than the control groups. Therefore, the combined expression of Aβ and rSO_2 can be used as a diagnostic and predictive indicator of POCD post-subtotal gastrectomy in older patients.[75] These results suggest a correlation between changes in rSO_2 and POCD during abdominal surgery. However, further investigations are needed to confirm this hypothesis.

Given the evidence outlined above, a consensus has not been reached on whether a decrease in rSO_2 during different types of surgery is correlated with the development of POCD (Table 3).

Improving Cerebral Oxygen Saturation May Prevent POCD

An increasing number of studies have confirmed that POCD can be effectively reduced by improving rSO_2 when intraoperative cerebral hypoxia occurs.[69,76,77] However, a consensus has not been reached on the critical threshold for cerebral ischemia and hypoxia associated with rSO_2. Previous studies have often used absolute values of $rScO_2 \leq 50\%$ or reductions from baseline $\geq 20\%$ as the thresholds for improving cerebral oxygenation.[43,69,78,79] Current interventions that are commonly used include the following: changing the head position to exclude mechanical obstruction that may alter the cerebral blood oxygen supply; increasing cerebral oxygen delivery, including increasing intraoperative fraction of inspiration O2, increasing partial pressure of carbon dioxide levels, dilation or raising arterial blood pressure with vasoactive drugs, increasing cardiac output, and administering blood transfusions in cases of significant blood loss; and reducing brain oxygen consumption, such as deepening anesthesia and lowering temperature (Figure 2). These measures

Table 3 Summary of Views–Relationship Between Cerebral Oxygen Saturation and POCD

Type of Surgery	Article View	Reference	Year
Cardiac	1. Prolonged rSO2 desaturation is a predictor of postoperative cognitive decline.	[55]	2015
	2. Improved cerebral blood oxygenation during cardiac surgery improves neurocognitive outcomes.	[56]	2022
	3. POCD can be predicted by monitoring the decline range of rSO_2 intraoperatively.	[57]	2021
	4. Relationship between rSO_2 and neurological complications after cardiac surgery is inconsistent.	[58]	2021
	5. Low-level evidence links low $rScO_2$ to postoperative neurologic complications.	[59]	2015
Thoracic	1. Decline in $SctO_2$ during single-lung ventilation in thoracic surgery is associated with early postoperative cognitive dysfunction.	[62]	2012
	2. Postoperative cognitive dysfunction in patients undergoing thoracic surgery may be associated with intraoperative rSO2 decline.	[63]	2015
	3. Decreased $SctO_2$ values are associated with postoperative cognitive dysfunction.	[64]	2022
Orthopedic	1. Postoperative cognitive dysfunction in patients undergoing shoulder surgery is associated with intraoperative low rSO_2.	[67]	2021
	2. Significant correlation between postoperative cognitive dysfunction and cerebral oxygen saturation in older orthopedic patients.	[68]	2021
	3. Measurement of cerebral oxygen saturation may help to reduce postoperative cognitive complications.	[14]	2021
	4. Intervention for intraoperative rScO2 decline may help to avoid cognitive impairment after spinal surgery.	[69]	2019
	5. Significant relationship was not observed between the occurrence of intraoperative brain desaturation and POCD.	[70]	2019
	6. No association between intraoperative brain saturation decline and postoperative cognitive decline.	[71]	2021
	7. Intraoperative rSO_2 decline does not affect postoperative cognitive function.	[72]	2015
Abdominal	1.Intraoperative rSO2 reduction may contribute to early postoperative cognitive decline.	[73]	2018
	2.Use of rSO2 monitoring may reduce the occurrence of cerebral hypoxia,which may reduce the impact on cognitiv function.	[74]	2005
	3. Combined expression of Aβ and rSO_2 can be used as a diagnostic and predictive indicator of POCD.	[75]	2016

Abbreviations: POCD, postoperative cognitive dysfunction; rSO2, regional cerebral oxygen saturation; rScO2, regional cerebral oxygen saturation; SctO2, cerebral tissue oxygen saturation;

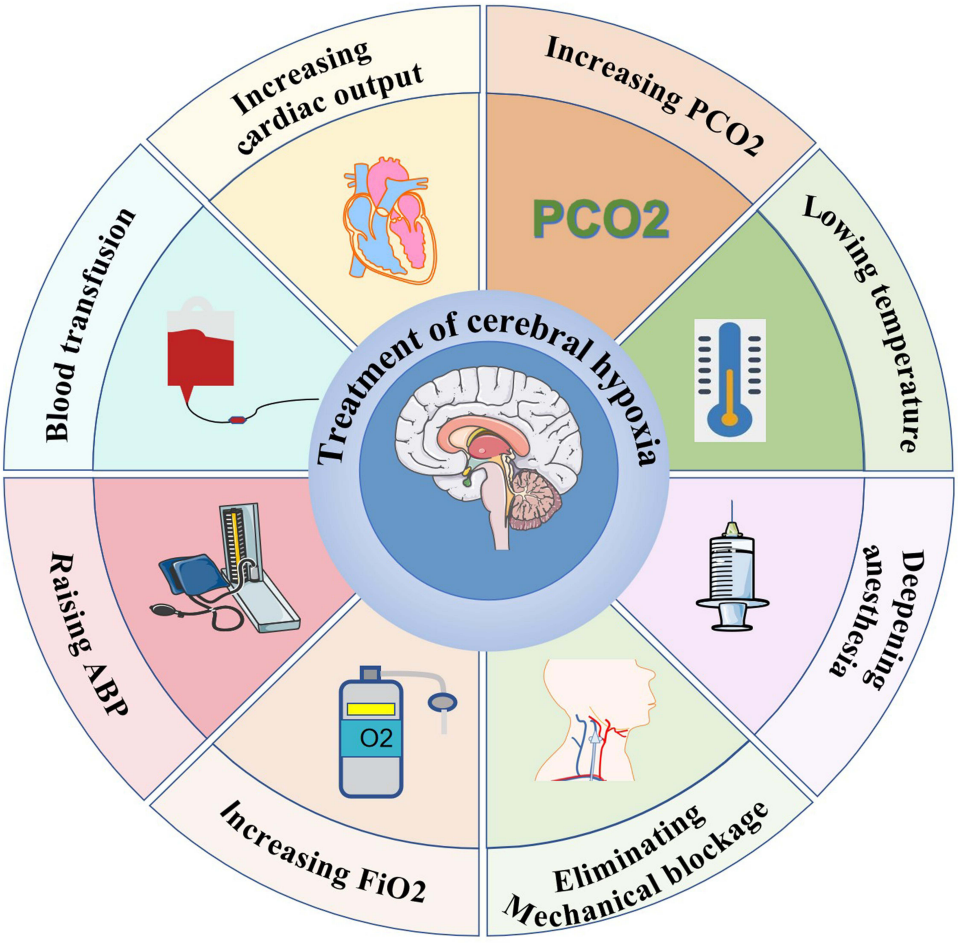

Figure 2 Methods of improving cerebral hypoxia.
Abbreviations: ABP, arterial blood pressure; FiO_2, fraction of inspired O_2; PCO_2, partial pressure of carbon dioxide; O2, oxygen.

can effectively increase rSO_2;[80,81] Evidence suggests that a beneficial early POCD outcome is associated with improved rSO_2 during major surgeries.[82]

Conclusions and Future Directions

In our view and in relation to the literature, rSO_2 monitoring can effectively assess the balance between cerebral oxygen supply and demand and changes in cerebral blood flow, and it can also prevent and predict adverse perioperative reactions in patients. Thus, it represents an important component of perioperative and intensive care unit multimodal neuromonitoring. Decreases or excessive increases in rSO_2 may lead to neurological complications. Data on hyperoxia and POCD are limited; therefore, the specific relationship between hyperoxia and POCD was not discussed in this review. The reliability of decreases in intraoperative rSO_2 in predicting POCD has been controversial due to the different definitions and assessment methods of POCD, baseline definitions of rSO_2, thresholds for clinical rSO_2 desaturation, and clinical intervention criteria. However, previous studies have shown an association between the two. Therefore, future studies should use standardized definitions and assessments of POCD, identify rSO_2 thresholds that affect cognitive function, specify rSO_2 thresholds for cerebral hypoxia, and determine interventions that effectively improve brain desaturation. Furthermore, studies with large sample sizes and argumentative clinical trials are warranted to explore the relationship between intraoperative rSO_2 decrease and POCD to better facilitate the clinical application of rSO_2 monitoring. However, conclusive evidence showing that decreased rSO_2 during surgery predicts adverse neurological outcomes in older patients remains lacking. Early monitoring-based interventions can potentially improve cognitive outcomes. Intraoperative rSO_2 monitoring can be used to protect the brains of older patients,

reduce adverse perioperative clinical outcomes, shorten hospital stays, and improve the quality of life. Therefore, monitoring and maintaining intraoperative rSO_2 are effective for predicting and preventing POCD in older patients and thus have important clinical implications.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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