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REVIEW ARTICLE

The Role of Brain Tissue Oxygenation Monitoring in the Management of Subarachnoid Hemorrhage: A Scoping Review

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Abstract

Monitoring of brain tissue oxygenation ($PbtO_2$) is an important component of multimodal monitoring in traumatic brain injury. Over recent years, use of $PbtO_2$ monitoring has also increased in patients with poor-grade subarachnoid hemorrhage (SAH), particularly in those with delayed cerebral ischemia. The aim of this scoping review was to summarize the current state of the art regarding the use of this invasive neuromonitoring tool in patients with SAH. Our results showed that $PbtO_2$ monitoring is a safe and reliable method to assess regional cerebral tissue oxygenation and that $PbtO_2$ represents the oxygen available in the brain interstitial space for aerobic energy production (i.e., the product of cerebral blood flow and the arterio-venous oxygen tension difference). The $PbtO_2$ probe should be placed in the area at risk of ischemia (i.e., in the vascular territory in which cerebral vasospasm is expected to occur). The most widely used $PbtO_2$ threshold to define brain tissue hypoxia and initiate specific treatment is between 15 and 20 mm Hg. $PbtO_2$ values can help identify the need for or the effects of various therapies, such as hyperventilation, hyperoxia, induced hypothermia, induced hypertension, red blood cell transfusion, osmotic therapy, and decompressive craniectomy. Finally, a low $PbtO_2$ value is associated with a worse prognosis, and an increase of the $PbtO_2$ value in response to treatment is a marker of good outcome.

Keywords: Subarachnoid hemorrhage, Cerebral oxygenation, Treatment, Mortality, Neurological outcome

Introduction

Nontraumatic subarachnoid hemorrhage (SAH), mostly secondary to aneurysmal rupture, accounts for 5% of all strokes [1] and is an important cause of morbidity and mortality and of potential years of life lost [1, 2]. About one quarter of patients admitted to the hospital after SAH will eventually die, and among survivors, half will have persistent severe neurological disability [3].

Immediately after blood enters the subarachnoid space, a complex pathophysiological process called early brain injury (EBI) leads to intracranial hypertension, cerebral edema, microcirculatory failure, neuroinflammation, and cerebral ischemia [4–7]. Patients with SAH are also susceptible to late ischemic complications, which can worsen prognosis, identified as delayed cerebral ischemia (DCI) [8–11].

Multimodal neuromonitoring, including brain tissue oxygenation ($PbtO_2$) monitoring, has been recommended to identify patients with EBI and DCI and to optimize treatment [12], especially in those in whom neurological examination is difficult. Indeed, the primary goal of

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neuromonitoring is to enable the detection of secondary brain insults before they cause irreversible damage to the brain [13]. Because the final common pathway in acute brain injury is the failure of oxygen delivery [14], detecting low oxygen cerebral states is vital to reduce secondary brain damage [15], provide a better understanding of complex brain physiology, and help guide management [16]. Preliminary observational studies suggest that monitoring $PbtO_2$ and treating patients with low $PbtO_2$ values may be associated with improved outcomes after SAH [16, 17]. However, there are no randomized control trials (RCTs) specifically assessing the impact of $PbtO_2$ monitoring and $PbtO_2$-guided therapy on the outcome of patients with SAH. The aim of this scoping review was therefore to provide a summary of the role of $PbtO_2$ in the management of adult patients with SAH by assessing the existing and emerging literature on this topic [28].

Methods

The review protocol was preregistered on April 11, 2019, on the Open Science Framework (https://osf.io/zyj7r/) and published in open access [18]; further details regarding the search strategy can be found in the Methods section of the Electronic Supplementary Material. This scoping review followed the five-stage framework proposed by Arksey and O'Malley [19], expanded by Peters et al. [20], and further developed by Levac et al. [21] and the Joanna Briggs Institute [22]. We also followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for scoping reviews guidelines [23].

The aims of the review were to describe in patients with SAH (1) the physiological bases of invasive brain oxygenation monitoring, (2) the technique of invasive brain oxygenation monitoring, (3) the indications and the utility of brain oxygenation monitoring, (4) the role of invasive brain oxygenation monitoring to guide medical and surgical therapy, and (5) the impact of $PbtO_2$ monitoring and $PbtO_2$-guided therapy on the outcome of these patients. We included all available scientific information from fully peer reviewed articles and gray literature that mentioned $PbtO_2$ monitoring in the context of SAH in adult patients. We excluded studies that focused only on a pediatric population (patients < 18 years old) and experimental studies performed exclusively in animals. There were no language limitations or sample size restrictions.

The search was performed on August 1, 2022. Three authors (EGB, MF, and AM) screened different databases for relevant abstracts and studies in a two-phase process (see Methods section of the Electronic Supplementary Material). All disagreements were resolved by consensus. Data were extracted to predefined charts, including the following information: study population; type of probe used; technique and adverse events; the indication for $PbtO_2$ monitoring, including to diagnose neurological complications such as EBI and DCI and to guide therapy; physiological determinants of $PbtO_2$ (oxygen delivery, oxygen extraction, and oxygen consumption); the effect of different treatments and strategies (including but not limited to pharmacological, respiratory, and hemodynamic therapy) on $PbtO_2$ values; outcomes (neurological outcomes and mortality).

Results

The initial search identified 21,550 references, of which 301 met our inclusion criteria (Fig. 1): 1 pilot RCT, 138 non-RCTs (prospective or retrospective), 1 cross-sectional study, 30 published abstracts, 16 case reports/case series, 20 book chapters, 70 reviews (5 of which were systematic reviews), 6 consensus statements, 6 editorials, 3 theses, 2 technical notes, 1 audit, 1 viewpoint, 6 conference statements/lectures, and 7 letters to the editor. A detailed description of all 301 references can be found in the Supplementary Tables S1, S2, and S3.

$PbtO_2$ Monitoring: Physiological Bases

$PbtO_2$ values represent the balance between oxygen delivery $\{DO_2 = \text{cardiac output} \times [1.39 \times \text{hemoglobin} \times \text{oxygen arterial saturation } SaO_2 + (0.003 \times PaO_2)]\}$, determined by cerebral blood flow (CBF), hemoglobin, and arterial oxygenation and oxygen consumption [VO_2 = (cardiac output + arterial content of oxygen) − (cardiac output − venous content of oxygen)], determined by brain metabolism, mitochondrial function, body temperature, and extraction (determined by blood–brain barrier and microcirculation) of brain cells [24, 25]. In this setting, the reasons for low $PbtO_2$ values are often multifactorial (Fig. 2), and several interventions have the potential to correct brain hypoxia [26]. $PbtO_2$ can be reduced because of a decrease in oxygen delivery either as a consequence of reduced CBF, leading to ischemia, or because of changes in the arterio-venous oxygen tension difference [27] caused by systemic hypoxemia secondary to impaired lung function [28, 29] (reduced arterial partial pressure of oxygen [PaO_2]) and anemia [30]. $PbtO_2$ can also be reduced because of increased oxygen consumption as a consequence of hypermetabolic states (e.g., seizures, fever, and shivering), because of mitochondrial dysfunction, and/or because of impaired oxygen extraction due to limited oxygen diffusion caused by brain edema and microvascular dysfunction [15, 31–34].

Normal $PbtO_2$ values are highly variable but are generally defined as values between 23 and 35 mm Hg [35]. Several thresholds have been used to describe brain tissue hypoxia in this setting, ranging from 10 to 19 mm Hg for at least 5 min [36–39]; the most commonly

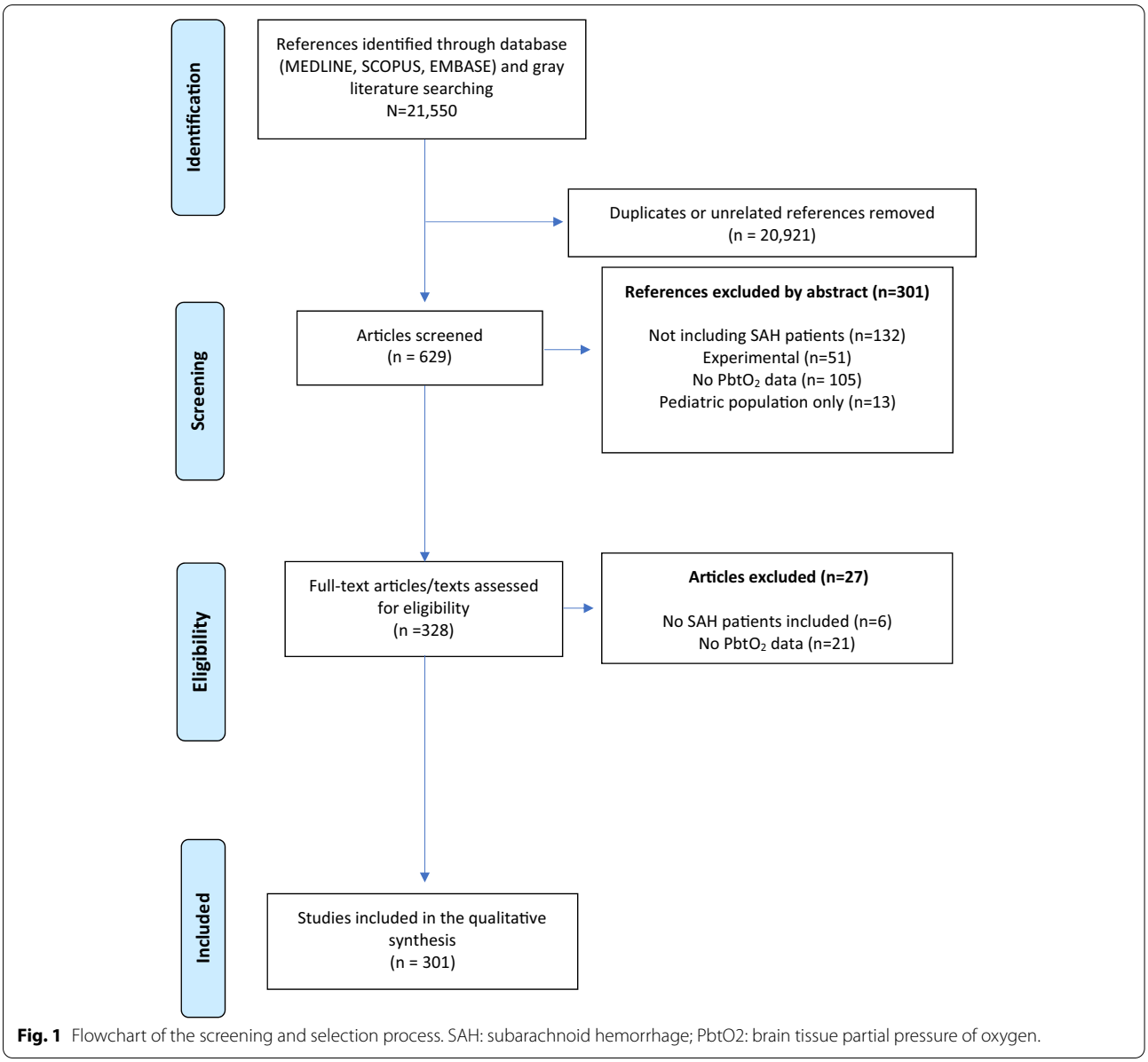

Fig. 1 Flowchart of the screening and selection process. SAH: subarachnoid hemorrhage; PbtO2: brain tissue partial pressure of oxygen.

used threshold is < 20 mm Hg [40, 41]. Tissue necrosis and cell death have been associated with $PbtO_2$ values of < 10 and < 5 mm Hg, respectively [42–45]. A $PbtO_2$ value of 0 mm Hg usually precedes diagnosis of brain death [46].

$PbtO_2$ Monitoring: The Technique

Two methods are used to measure $PbtO_2$ [47]: the luminescence quenching method used by the Neurovent-PTO (Raumedic AG, Munchberg, Germany), the Oxylab pO_2 (Oxford Optronix Ltd, Oxford, United Kingdom) probes [16, 48], and the polarographic method used by the Licox brain oxygen monitor (Integra Neuroscience, Plainsboro, New Jersey) [49, 50]. Importantly, $PbtO_2$ measurements from the two methods are not entirely interchangeable [47, 51–54].

$PbtO_2$ monitoring involves the insertion of a probe into the brain parenchyma, ideally in the subcortical white matter, by using a bolt (single or multiple) or by tunneling, allowing continuous (every 2 s) $PbtO_2$ monitoring [34, 40, 55]. $PbtO_2$ is a local measurement [16, 56], reflecting the tissue oxygenation of an area of 2 mm to 22 mm^2, depending on the device used, the type of probe, and the probe location (i.e., injured vs. noninjured area) [36]. Therefore, a cerebral computed tomography (CT) scan is required following the placement of the $PbtO_2$

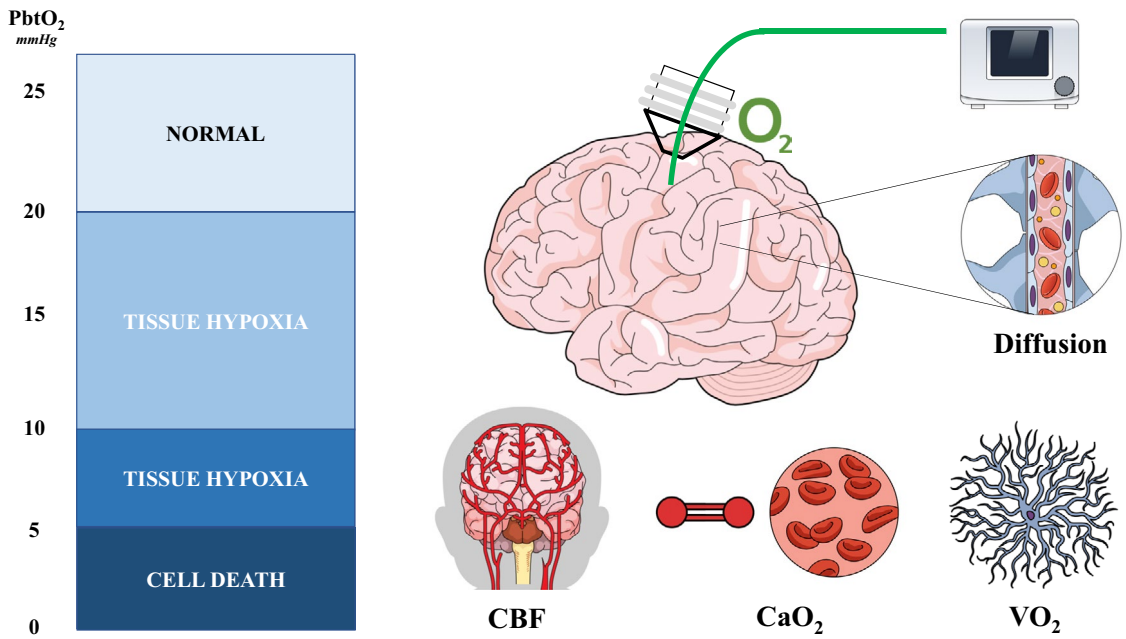

Fig. 2 Physiological determinants of brain tissue oxygenation ($PbtO_2$). $PbtO_2$ represents a balance between oxygen delivery (DO_2), oxygen extraction, and consumption (VO_2) in the brain. Oxygen delivery to the brain is determined by the cerebral blood flow (CBF) and arterial content of O_2. VO_2 depends on brain metabolism, mitochondrial function, body temperature, and extraction (determined by blood–brain barrier and microcirculation) of brain cells. CaO2: arterial content of oxygen.

probe to confirm its adequate location [37, 57], as well as to exclude peri-procedural complications, which can include mispositioning or minor bleeding [33, 58–61].

Placing the $PbtO_2$ catheter in areas where the ruptured aneurysm had been identified can provide reliable monitoring of the secondary ischemic insult (vasospasm and DCI), specially for aneurysms of the anterior circulation [62, 63]. For aneurysms of the posterior circulation, the optimal placement position is not well defined. Probe placement can potentially be guided by CT scan [64], Xenon CT [65], single photon emission CT [66], or transcranial Doppler [67] to increase the likelihood of monitoring the at-risk area or penumbra area of ischemia [68].

Importantly, probe placement causes microtrauma to the subcortical matter, making the first readings unreliable. It is recommended to wait at least 1 h before relying on the monitor [33, 59, 61, 69]. For further verification of the function and responsiveness of the catheter, an oxygen challenge should be performed [33, 59, 61, 69] by increasing inspired fraction of oxygen (FiO_2) to 100% for 2–5 min [28, 33, 70–72]; if the probe is well-positioned and accurate, it will show an increase in the $PbtO_2$ value of around two times baseline values [33, 59].

$PbtO_2$ devices are considered safe and accurate with negligible zero drift [40, 61]. Hemorrhage and hematoma formation rates vary from 0 to 40%, including tract hemorrhages, although hematoma needing surgical intervention is rare [45, 61, 73–76]. Central nervous system infections are also rare [73, 75].

$PbtO_2$ Monitoring: Indications and Uses

In general, patients are selected for $PbtO_2$ monitoring when intracranial pressure (ICP) monitoring is required [40] and neurological evaluation is unreliable [77]. Criteria to initiate multimodal monitoring in patients with SAH include patients with Glasgow Coma Scale (GCS) < 9 who are unlikely to regain consciousness within the next 48 h and have a high probability of surviving for the next 48 h [78]. Rass et al. initiated multimodal monitoring in patients with SAH who required prolonged mechanical ventilation and/or had clinical or radiological signs suggestive of increased ICP [79]. Good-grade patients with delayed deterioration of their neurological status are also candidates for $PbtO_2$ monitoring in the context of cerebral vasospasm and DCI [80].

$PbtO_2$ monitoring has been employed for detecting ischemic events during aneurysm clipping [81, 82] and temporary artery occlusion [39, 83–91]. $PbtO_2$ has also been used to direct therapy in the operating room [92–94]. In the first 72 h of SAH, $PbtO_2$ monitoring is indicated to help detect microvascular injury [95] and ischemic events occurring in the context of EBI [96]. In fact, brain tissue hypoxia is common in the first 48 h after SAH [97]. $PbtO_2$ can be used as a surrogate measure

of regional CBF [98, 99], which can aid in the diagnosis of vasospasm and DCI [100, 101], especially in comatose patients with suboptimal clinical assessments who have angiographic vasospasm as well as in symptomatic patients who have suboptimal imaging [102]. In fact, several studies have focused on the use of $PbtO_2$ to help detect DCI [77, 80, 103–105] because there appears to be a correlation between decreasing $PbtO_2$ values and cerebral vasospasm detected by transcranial Doppler [85, 106, 107] when the $PbtO_2$ probe is adequately placed in the at-risk area for vasospasm [108]. A $PbtO_2$ threshold less than 20 mm Hg has a sensitivity of 71% and a specificity of 89% for prediction of vasospasm, with an area under the receiver operating characteristic curve of 0.90 [100]. Moreover, low $PbtO_2$ values associated with signs of anaerobic metabolism assessed by cerebral microdialysis (CMD) can be present before visible CT scan infarction, enabling an early diagnosis of silent ischemia [78]. In fact, in patients with good-grade and poor-grade SAH, introduction of multimodal monitoring, including of $PbtO_2$, resulted in earlier detection and earlier treatment of DCI, thus reducing DCI-related infarction [80, 109].

$PbtO_2$ also has a role in assessing autoregulation. Impaired cerebral autoregulation is an important pathophysiological pathway of acute brain injury [110, 111] and can represent an independent risk factor for poor outcome in patients with SAH [112]. The oxygen reactivity index (ORx) is expressed as the moving correlation coefficient between cerebral perfusion pressure (CPP) and $PbtO_2$, calculated every 30 s; a high ORx indicates impaired autoregulation [32]. The ORx has been proposed as a better predictor of cerebral hypoperfusion, DCI, and outcome than $PbtO_2$ in patients with SAH [32, 113]. Interestingly, Jaeger et al. found that both ORx and $PbtO_2$ values were lower in a group of patients with poor functional outcome [112]. Other studies have failed to show an association between ORx and cerebral ischemia or neurological outcome [114, 115].

$PbtO_2$ can also be used as an adjunct monitor to assess the impact of ictal discharges and seizures in patients with SAH [116–118] and to monitor changes in brain oxygenation during mobilization of the patients [119, 120] and during transportation [121–123].

The $PbtO_2$ probe should be kept in situ for a maximum of 7 to 10 days [40, 124] and can be removed if the patient is awake (motor GCS of 6 or motor GCS of 5 if patient is aphasic or unable to communicate) or if there is a medical indication for removal of the probe (such as infection or bleeding associated with the catheter) [33]; additionally, if ICP is normal (< 20 mm Hg) for 24 h without specific treatment and $PbtO_2$ values are > 20 mm Hg for 48 h, it is also reasonable to remove multimodal neuromonitoring [33].

$PbtO_2$ Monitoring: Assessing the Efficacy of Different Therapies

Numerous studies have been performed to monitor the efficacy of various therapies, especially focused on vasospasm and DCI. Regarding so-called triple H therapy (i.e., hypervolemia, hemodilution, and hypertension), Muench et al. [125] observed that vasopressor-induced hypertension, but not hypervolemia and hemodilution, could improve $PbtO_2$ values in a population of patients with poor-grade SAH with cerebral vasospasm. Similarly, Raabe et al. noted that an increase in $PbtO_2$ was far more frequent in patients who received induced hypertension compared with those who received hypervolemia [126]. These observations have helped shift treatment strategies from triple H therapy to induced hypertension alone.

Transluminal balloon angioplasty, used as rescue therapy to treat refractory vasospasm, can improve $PbtO_2$ levels and reduce metabolic distress [103, 127]. In many studies, $PbtO_2$ significantly improved during intermittent and continuous chemical spasmolysis with intra-arterial nimodipine (IAN), followed by resolution of vasospasm on angiography [128–133]. A recent observational study compared two strategies for treatment of vasospasm refractory to induced hypertension: one group of patients was treated with induced hypertension targeting a systolic blood pressure (SBP) of 180 mm Hg plus lower doses of continuous IAN, and the other group received higher doses of IAN without induced hypertension (SBP target = 120 mm Hg) [104]. Patients in the latter group had higher $PbtO_2$ levels after IAN without relevant adverse events.

The effects of other vasodilatory agents on $PbtO_2$ have also been studied in the context of refractory vasospasm: intra-arterial papaverine hydrochloride [134], intraventricular sodium nitroprusside [135], and intra-arterial verapamil [136, 137]. Only one studied showed a clear improvement in $PbtO_2$ [136]. Inhaled nitric oxide (iNO) was also used in a pilot study to treat refractory DCI in seven patients; all patients experienced an increase of at least 5 mm Hg in $PbtO_2$ after iNO [138]. Interestingly, the use of erythropoietin in patients with poor-grade SAH and vasospasm tended to increase $PbtO_2$ [139].

$PbtO_2$ can also be used to optimize CPP [126, 140–147] because higher CPP is associated with fewer episodes of brain tissue hypoxia and cerebral infarction [126, 142, 148]. In fact, strategies to increase mean arterial pressure (/CPP, such as fluid resuscitation [149], vasopressors [125], and the use of inotropes to augment cardiac output [150], can also promote an increase in $PbtO_2$ levels, especially when accompanied by an increase in cardiac index [149, 151] in patients with low baseline $PbtO_2$ [147, 151].

Osmotic therapy with hypertonic saline to treat intracranial hypertension has been shown to improve $PbtO_2$

[152, 153]. On the other hand, mannitol may have no impact on $PbtO_2$, especially if the baseline $PbtO_2$ is > 20 mm Hg [154]. Decompressive craniectomy to treat refractory intracranial hypertension can also improve $PbtO_2$ [155, 156], which typically decreases progressively before intervention [157]. The use of barbiturates to treat refractory intracranial hypertension may improve $PbtO_2$ in some but not all patients with SAH [72].

Because brain temperature can influence CBF and $PbtO_2$ measurements [158], some authors have investigated the effects of antipyretic drugs on $PbtO_2$ [159] and have found that the degree of change in $PbtO_2$ correlates with the reduction in core temperature. Mild hypothermia can also improve $PbtO_2$ [160]. Moreover, in a cohort of patients with poor-grade SAH, higher $PbtO_2$ measures were more frequently linked to normothermia (compared with fever) [161].

$PbtO_2$ can also be used to monitor the effects of red blood cell (RBC) transfusion in optimizing oxygen delivery. Interestingly, $PbtO_2$ response to RBC transfusion varies, with most patients showing an increase in $PbtO_2$ (especially those with baseline hypoxia before transfusion and lower hemoglobin levels) and other patients having no change or even a decrease in $PbtO_2$ [162–164].

In patients with acute brain injury and acute respiratory distress syndrome, $PbtO_2$ may assist the clinician in assessing the effects of recruitment maneuvers [165] and the prone position [29, 166] on brain oxygenation. $PbtO_2$ values can also be used to titrate FiO_2 because normobaric hyperoxia usually results in increased $PbtO_2$ [167–169]. During a hyperoxia challenge, smaller increases in $PbtO_2$ are associated with higher CMD lactate and a higher risk of ischemia [170]. Importantly, the impact on outcome of improving $PbtO_2$ by increasing FiO_2 is still uncertain [171–173].

The impacts of different sedative drugs, such as propofol and dexmedetomidine, on $PbtO_2$ were studied, and showed that for a similar Richmond Agitation and Sedation Scale, both drugs had a similar impact on cerebral oxygenation [174], usually leading to a modest elevation in $PbtO_2$ [175]. $PbtO_2$ has also been used to assess the success of a neurological wake-up test, in which reduction in $PbtO_2$ was a criterion of test failure [176].

$PbtO_2$ Monitoring: Impact on Outcome in Patients with SAH

Several studies found that patients with short- and long-term favorable neurological outcomes had higher $PbtO_2$ values for longer periods of time than those with unfavorable outcome [85, 101, 133, 152, 177, 178]. The association between low $PbtO_2$ values and unfavorable outcome is stronger when concomitant metabolic brain dysfunction is present [63]. However, other studies have failed to show an independent association between low $PbtO_2$ levels and unfavorable outcome [63, 179].

Nonsurvivors have consistently lower $PbtO_2$ levels during longer periods of time than survivors [148, 180–182]. In fact, low $PbtO_2$ levels are independently associated with mortality, especially when accompanied with brain energetic dysfunction [28, 148, 183].

The rationale behind $PbtO_2$ monitoring is that improving $PbtO_2$ will translate into a better outcome [33]. However, studies that have investigated the impact of a $PbtO_2$-guided therapy have yielded conflicting results. Bohman et al. analyzed patients with SAH managed with a goal-directed treatment aimed at maintaining a $PbtO_2 \geq 20$ mm Hg. The mean rate of response to the directed treatment was independently associated with a favorable functional outcome (defined as modified Rankin Scale < 4 and Glasgow Outcome Scale-Extended ≥ 3) [178]. Similarly, Al-Rawi et al. also found that a sustained increase in $PbtO_2$ after treatment (hypertonic saline) was associated with a favorable outcome at 12 months in patients with SAH [152]. In a mixed cohort of patients with traumatic brain injury and SAH who underwent treatment when $PbtO_2$ was 15 mm Hg for more than 10 min, patients had a decreased risk of unfavorable outcome and mortality [64]. In another mixed cohort of patients, Monteiro et al. found that multimodal monitoring (including of $PbtO_2$) was associated with better short- and long-term neurological outcomes [184]. Additionally, Veldeman et al. found similar results for long-term outcomes [109].

Conversely, in patients with good-grade SAH who developed DCI, multimodal monitoring did not improve neurological outcome [80]. The lack of association between $PbtO_2$-guided therapy and outcome can be explained by the results of a study conducted by Rass et al. in which, despite a protocolized $PbtO_2$-guided therapy approach, episodes of cerebral hypoxia ($PbtO_2 < 20$ mm Hg) still occurred in 81% of patients [179]. Similarly, Gouvea Bogossian et al. [185] did not find an association between ICP/$PbtO_2$-guided therapy and outcomes compared with ICP-only guided therapy. Importantly, to date, no RCT has investigated the impact of $PbtO_2$-guided therapy on outcome in patients with SAH.

Limitations

This review has some limitations that are inherent to scoping reviews: the findings are often broad, and synthetizing all the results can be challenging. In this review, we did not perform quality analysis, and all available data were charted and summarized. Because of the broad study period, some studies may be outdated and concepts may have changed. Therefore, this

review provides only a summary of the available evidence regarding $PbtO_2$ monitoring in patients with SAH; a systematic review and meta-analysis are needed to answer specific questions with a higher-quality standard. Moreover, RCTs in patients with SAH are needed to specifically assess if the use of $PbtO_2$-guided therapy can improve outcome.

Conclusions

Maintenance of adequate brain oxygenation represents one of the primary objectives in neurocritical care, and the assessment of tissue oxygenation is important to patient management. Integration of $PbtO_2$ into a multimodal neuromonitoring approach may help clinicians in the early detection of physiological derangements that can compromise oxygen supply to the brain, providing both a trigger and a target for interventions.

Supplementary Information
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Author contributions
MF, EGB, and FST conceived the study. AM, MF, and EGB performed the screening and selected the articles for the review. EGB, SF, AM, and GG extracted the data from the articles. SF, AM, GG, and EGB wrote the first draft. DB, MF, CR, and FST revised the text for intellectual content. All authors read and approved the final manuscript.

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Conflict of interest
The authors declare that they have no conflict of interest.

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