Chapter 8
Jugular Bulb Oximetry

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8.1 Introduction

Patients with acute spontaneous or traumatic neurological injuries are susceptible to secondary brain damage. Episodes of hypotension, hypoxia, increased intracranial pressure, and local inflammatory cascades are the main events involved. Minimizing secondary insults is the main objective of the medical team [25]. However, to do so, such insults must first be detected. Older technologies allowed continuous monitoring of systemic variables, including blood pressure and oxygen saturation, which were then applied to brain physiology. Technological developments have allowed the brain to be directly monitored with catheter implantation that permits, in addition to continuous monitoring of intracranial pressure, monitoring of brain temperature, tissue pressure, and, more recently, brain microdialysis [22, 25]. Cerebral venous flow oxygen saturation (SjO2) has been investigated as a neuromonitoring parameter for over 50 years. The first works on jugular bulb oximetry are from 1930 to 1940 [17, 18]. SjO2 currently provides an indirect assessment of brain oxygen use and is used to guide physiological management decisions in a variety of clinical settings [33]. This chapter is an overview of SjO2 monitoring and an update on its clinical applications.

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8.2 Anatomy

The internal jugular veins drain practically all the blood from the brain. The drainage veins of the telencephalic and diencephalic structures direct their contents to the venous sinuses. Superior sagittal sinus, inferior sagittal sinus, straight sinus, and occipital sinus unite at the confluence of the sinuses (Herophili torcular) [15]. From this point, the venous blood is directed to the right and left transverse sinuses and sigmoid. These two sinuses pass through their jugular foramina at the base of the skull and dilate to form the jugular bulb—the dilated cephalic part of the internal jugular vein. A small proportion of effluent brain blood can drain through the vertebral venous plexus, which is most pronounced when in the upright position [35].

The jugular bulb receives drainage from both intracranial and extracranial compartments, and it is located posterolaterally within the pars vascularis of jugular foramen [39, 40]. The tributaries vein include: the middle thyroid vein, superior thyroid vein, lingual vein, facial vein, pharyngeal vein, and inferior petrosal sinus, apart from the vertebral venous plexus, venous plexus of the hypoglossal canal, posterior condylar emissary vein, and veins along the petroclival fissure [41, 42].

Anteriorly, the jugular bulb is limited by the internal carotid artery, cochlear aqueduct, inferior petrosal sinus, meningeal branch of the ascending pharyngeal artery, lower cranial nerves, and posterior meningeal artery. The posterior limits of the jugular bulb include the sigmoid sinus, occipital bone, and facial nerve, while the superior limits of the jugular bulb include the external auditory canal, middle ear, posterior semicircular canal (SCC), vestibule, and internal auditory canal [45, 46].

The upper limit of the jugular bulb is commonly found under the hypotympanum within the middle-ear cavity, and an atypical presentation of the jugular bulb may be visualized as an upward extension of the bulb that invades into the hypotympanum. Sasindran et al. define this extension of jugular bulb presenting in the middle-ear space with a thin or nonexistent bony septum as a high-riding jugular bulb (HRJB), which has been previously subclassified as “with dehiscence” or “without dehiscence.” An alternate definition of an HRJB has been proposed when it is observed above the tympanic annulus or no greater than 2 mm from the IAC. Singla et al. postulated the jugular bulb as high riding when the distance of the summit of the jugular fossa from the round window or IAC was less than or equal to 2 mm or if there is no distance between the jugular fossa and the slit on which the endolymphatic sac opens [47].

8.2.1 Classifications of Jugular Bulb

Shao et al. [52] classified jugular bulbs as (1) grade 1, jugular bulb located less than 1 mm above the lower border of IAC; (2) grade 2, jugular bulb between 1.5 and 3 mm above the lower border of the IAC; and (3) grade 3, jugular bulb greater than

3 mm above the lower border of the IAC. We believe that these anatomical boundaries of the jugular bulb and extent of the bulb itself are not evolving forms, and hence the term "classification" seems to be more appropriate than "grading."

Park et al. subclassified HRJB into two types based on axial CT images: type 1, in which the bulb dome reaches above the inferior part of the round window; and type 2, when the dome is higher than the inferior edge of the IAC. It is apparent that there is no consensus on the exact definition of HRJB, and multiplanar structures that define the critical microsurgical boundaries (SCC, IAC, round window, and endolymphatic sac) of the skull base cannot often be analyzed based only on limited standard axial CT sections of temporal bone, without reconstruction [39].

The current proposal of the Manjila and Semaan classification accounts for the relationship of the IAC, posterior SCC, and presence or absence of dehiscence into the middle ear or IAC. This straightforward and practical classification system with easy subcategorization based on local skull base landmarks would be extremely useful in preoperative planning of surgical corridors and thus achieve satisfactory clinical outcomes.

8.2.2 *Manjila and Semaan Classification of Jugular Bulb Location*

Type 1 No jugular bulb
Type 2 Below inferior margin of posterior SCC
Type 2a Without dehiscence into the middle ear
Type 2b With dehiscence into the middle ear
Type 3 Between inferior margin of posterior SCC and inferior margin of IAC
Type 3a Without dehiscence into the middle ear
Type 3b With dehiscence into the middle ear
Type 4 Above inferior margin of IAC
Type 4a Without dehiscence into the IAC
Type 4b With dehiscence into the IAC
Type 5 Combination of dehiscence

8.2.3 *Development of the Jugular Bulb*

The jugular bulb develops during childhood, particularly when the child has gained the ability to stay upright around 2 years of age. The jugular bulb continues to develop through childhood and becomes stable in adulthood. Once an erect posture is attained in life, the ascending negative pulse waves originating from the right atrium are postulated to be transmitted rostrally into the jugular sinus leading to the dilation or formation of the jugular bulb. Consequently, an HRJB is also considered

a risk factor for jugular bulb dehiscence. Because there is growth and plateauing of jugular bulb development with age, there is debate on the role of temporal bone pneumatization and the orientation of SCCs in determining the location of the jugular bulb.

8.3 Normal and Physiopathology

The concept of obtaining information on brain oxygen status from measuring oxygen saturation in mixed cerebral venous blood is based on the following assumptions derived from the Fick principle. Fick's principle states that the amount of oxygen transported to a tissue compartment minus the amount of oxygen consumed is equal to the amount of oxygen exiting this tissue compartment through the venous circulation. Therefore, jugular bulb blood saturation is related to cerebral blood flow (CBF), arterial saturation, brain oxygen extraction, and cerebral oxygen metabolism (CMRO2) (Spiss 1998; [48]). In other words, the cerebral oxygen supply (DO2) is described by the following equation (CaO2 arterial oxygen content):

$$\mathrm{DO2} = \mathrm{CBF} \times \mathrm{CaO2}$$

Where CaO2 = (% saturation × 1.34 × Hb) + (arterial or venous O2 tension × 0.003).

While brain oxygen consumption (CMRO2) is described by the equation (CjvO2 jugular venous blood oxygen content):

$$\mathrm{CMRO2} = \mathrm{CBF} \times \left(\mathrm{CaO2} - \mathrm{CjvO2}\right)$$

The difference in oxygen content between arterial and jugular venous blood is expressed by the term (CaO – CjvO2) or AjvDO2. Rearranging the above equation, it is evident that:

$$\mathrm{AjvDO2} = \mathrm{CMRO2} / \mathrm{CBF}$$

Usually, AjvDO2 is stable in 4–8 mL O2/100 mL blood [44, 55]. If CMRO2 remains constant, changes in AjvDO2 should reflect changes in CBF. If AjvDO2 is ±4 mL O2/100 mL blood, the oxygen supply is assumed to be greater than the demand (i.e., exuberant). An AjvDO2 ~ 8 mL of O2/100 mL of blood suggests that demand exceeds supply (i.e., ischemia) [48]. As the hemoglobin (Hb) level is practically constant between arterial and venous circulation and dissolved oxygen is negligible, it can be observed that CaO2 varies with saturation. Thus, SjO2 is a function of arterial saturation, CMRO2, and CBF. There is a debate about what the normal range of SjO2 is, but most studies assume that 50–54% is the lower limit of normal and 75% is the upper limit. Different methods for defining normality have caused controversy over the exact lower limit of normal SjO2, with some authors assuming 50% [21, 53] and 54% [7, 8, 31]. None of them are wrong, as shown in a recent

article comparing SjO2 monitoring with brain tissue oxygenation (PtiO2) [27], where PtiO2 values of 8.5 ± 11.0 mmHg (although with large variability) corresponded to 50 ± 54% SjO2 values.

It is noteworthy that in certain pathological situations, such as cerebral infarction, Sjv02, or AjvDO2 itself may not accurately reflect the presence of cerebral ischemia or hypoxia. With the onset of cerebral infarction, regional CMR02 may be decreased, leading to normal Sjv02 or AjvDO2, despite substantial impairment of cerebral perfusion. In such cases, the determination of the lactate oxygen index is a valuable complement to jugular venous bulb oximetry. It is a measure of the ratio between the amount of anaerobically metabolized glucose and the amount that is aerobically metabolized.

The lactate oxygen index is calculated as follows: lactate oxygen index (LOI): –(arterial–jugular bulb lactate concentration)/AjvDO2. If the LOI is less than 0.08, a normal AjvDO2 indicates that the brain is flow coupled with metabolism. If this number increases above 0.08, ischemia is likely to be present [49]. The extent of the impact of a given brain region on Sjv02 depends on the size of that particular region and its regional CMR02. Both factors determine the absolute amount of oxygen extracted from the arterial blood.

Therefore, small brain regions with high CMR02 can contribute as much as large brain regions with a lower CMR02 to the total amount of oxygen that is consumed during aerobic metabolism. These considerations are of great importance when interpreting changes in Sjv02 and their relationship to clinical events. In general, monitoring of Sjv02 is useful whenever we expect a mismatch between oxygen supply and consumption, which may be short-lived or even avoided if recognized in advance. Some situations where there is a mismatch between supply and consumption are:

- Oxygen supply drop: Cerebral ischemia: systemic hypotension, increased intracranial pressure, vasospasm, hyperventilation, internal carotid artery occlusion (for intraoperative monitoring situations)
- Drop-in arterial oxygen content: hypoxemia (ARDS), anemia, carbon monoxide poisoning, and hemoglobinopathies
- Increased consumption (metabolism): Seizure and hyperthermia

8.4 Which Side to Choose?

The choice of which cannular jugular bulb can potentially influence outcomes. The jugular bulb cannot be assumed to contain exclusively cerebral venous blood, as it may be contaminated by extracranial drainage. According to Shenkin et al. [54], in eight patients without cerebrovascular lesions in which dye was injected into the external carotid artery demonstrated that up to 6.6% (with an average of 2.7%) of blood in the jugular bulb was derived from extracranial sources. This is because the frontal veins and emissary veins drain into the sagittal sinus beyond the connection

of the cavernous sinus to the sigmoid sinus and the jugular bulb through the petrous sinuses [43]. The primary source of potential contamination is the facial vein that joins the internal jugular vein a few inches below the jugular bulb. Experiments show that the rate of blood withdrawal from the jugular bulb affects the composition of the sample. A rate of 1 ± 2 mL per minute is ideal so that venous blood is not aspirated from the facial vein [43].

It is observed that the composition of the jugular bulbs is not equal. Cortical tissue drains into the superior sagittal sinus, while subcortical tissue drains into the rectum sinus. These join together to form the confluence of the breasts (Torcular Herophili) that divides into the two transverse sinuses that eventually drain into the jugular bulb through the sigmoid sinuses. The mixing of blood from the cortex and deeper brain regions is incomplete; the lateral sinus is more significant to the right in 62%, left in 26%, and equal in 12% of individuals [23]. In an autopsy study, Gibbs et al. [17] found that blood from subcortical areas draining from the right sinus tended to flow to the left lateral sinus, while blood from cortical tissue draining from the sagittal sinus flowed mainly to the right lateral sinus. Shenkin et al. [54] suggested that two-thirds of the contents of the internal jugular vein are from the ipsilateral hemisphere and one-third from the contralateral hemisphere. Simultaneous sampling of right and left jugular bulbs in healthy subjects showed that oxygen saturation at the level of the internal jugular vein is equal on both sides [18].

On the other hand, according to Stoccheti et al., in a study with 32 patients with head trauma, in which both jugular bulbs were cannulated, and 171 blood samples were collected. Differences in the saturation of parallel samples were approximately 5%. Probably, in healthy individuals, there is no difference in saturation between the right and left sides, but there are differences in patients with head trauma [57]. In clinical practice to test the dominant side, compression of the right and left jugular vein can be performed and to evaluate that it generates a more significant increase of intracranial pressure [1, 11]. The side with the highest increase in ICP drains most of the blood and thus allows SjO2 monitoring of the largest vascular territory of the brain. If there is no difference between the two sides, the right side is commonly used in diffuse lesions, as it is more likely to be the dominant anatomical side [29]. In diffuse lesions, other authors recommend measuring the jugular foramen dimensions to choose which side to cannulate [34, 38].

8.5 Cannulation Technique

Retrograde cannulation of the internal jugular vein is a simple and safe procedure in experienced hands [20]. The jugular vein may be punctured more distally between the two heads of the sternocleidomastoid muscle [37] or more proximal at the cricoid ring level [1]. The patient is positioned horizontally, always aware of the ICP values so that they do not exceed 20 mmHg, if possible. The head should be laterally contralateral to the side of the puncture and slightly deflected. Routine antiseptic measures, as with all other invasive catheterization techniques, are required. The

carotid artery is palpated medially to the sternocleidomastoid muscle at the lower edge level of the thyroid cartilage. Lateral to the carotid artery, a 21 G needle with a coupled syringe containing normal saline is advanced at a 30° angle toward the ipsilateral external acoustic meatus. Blood aspiration should occur at a depth of approximately 4 cm from the skin. A 5 fr (french) introducer is placed using the Seldinger technique and fixed in position. After a preinsertion calibration, the fiber optic catheter is inserted through the introducer into the internal jugular vein and advanced to the base of the skull (approximately 15 cm). After feeling the resistance of reaching the bulb, the catheter is pulled for about 1 cm. To prevent increases in intracranial pressure that occur when the head is down (to facilitate puncture), there is a description of cannulation in the raised head position with the aid of a needle with an in situ Doppler probe [50].

The catheter tip should be above the C1/C2 disc to minimize facial vein contamination; therefore, the position of the catheter tip should be checked by X-ray to ensure accurate measurement and reduce complications. An over penetrated lateral radiograph is the simplest and most reliable X-ray type [24]; alternatively, anteroposterior radiographs may be preferred [2].

8.6 Clinical Indications

8.6.1 General Principles

Jugular bulb oximetry is the first continuous and bedside brain monitoring method estimating cerebral perfusion adequacy. The fact that most of the regulatory mechanisms of cerebral perfusion are failing in patients suffering from severe neurologic lesions explains the growing interest in monitoring cerebral perfusion adequacy in these critically ill patients. Moreover, brain ischemia has been observed in not less than 80% of all patients dying from severe head injury, illustrating the frequent occurrence of brain ischemia after severe head injury.

An important mainstay of the intensive management is accordingly to avoid the occurrence of cerebral ischemia. A distinction is often made between primary and secondary ischemia, with primary ischemia considered as resulting from the ischemic insults that occurred at the scene of the accident and secondary ischemia as the ischemic insults occurring during the further intensive care management. To avoid secondary ischemia during the intensive care management, monitoring of the cerebral perfusion state seems obligatory [36].

8.6.2 Outline in Detecting Physiological Insult

In acute brain injuries, multimodal monitoring is critical. These include pulse oximetry, electroencephalogram, intracranial pressure, blood pressure, tissue oxygen pressure, cerebral perfusion pressure, and not least oxygen saturation of the jugular

bulb. This is an integral component with significant contributions to clinical management, driving effective therapeutic strategies. SjO2 monitoring has applications in neurosurgery, neurointensive, cardiac surgery with cardiopulmonary bypass, and hypothermia [56]. Jugular venous oxygen is an indirect assessment of brain oxygen use. In a simplified way, when demand exceeds supply, the brain extracts more oxygen, resulting in lower oxygen saturation of the jugular bulb. If cerebral blood flow (CBF) decreases, a point is finally reached at which the brain can no longer fully compensate for decreased CBF by an additional increase in oxygen extraction. At this point, oxygen consumption decreases, and anaerobic metabolism with lactate production occurs when brain oxygen supply exceeds demand, jugular bulb oxygen saturation increases [33, 48]. If CMRO2 increases without an increase in CBF, the brain draws more oxygen from the blood, and there is a decrease in oxygen content of venous blood saturation of the brain. Jugular venous oxygen saturation is usually approximately 55–75% [19], which is lower than mixed venous systemic oxygen saturation.

If hemoglobin concentration is stable, arterial oxygen saturation is approximately 100%, and the amount of dissolved oxygen in plasma is physiological, SjVO2 is a direct correlation with AjvDO2. As SjVO2 is a global measure, SjVO2 monitoring has high specificity but low sensitivity to ischemia, that is, normal saturation may not reflect focal areas of ischemia, but low saturation is indicative of low flow. If SjVO2 is less than 50%, therapies directed at increasing brain oxygen supply and decreasing demand should be initiated. The decrease in hemoglobin (Hb) to deficient levels resulted in a decrease in CMR02 with an unchanged Sjv02 [9].

This observation indicates that cerebral oxygen extraction may be limited in these situations, that is, decreased Hb causes a decreased oxygen supply to the brain that cannot be compensated for by extracting more oxygen from arterial blood. Briefly, the main objective is to detect and treat cerebral hypoperfusion to minimize secondary insults. In acute brain injury, low values are indicative of increased cerebral oxygen extraction as a result of systemic arterial hypoxia, low cerebral blood flow from systemic hypotension, vasospasm, or increased intracranial pressure with low cerebral perfusion pressure (CPP). Chan et al. [5] demonstrated a reduction in SjO2 when PPC falls below 70 mmHg. Fever and seizures (which may not be correctly diagnosed in sedated patients) also result in low SjO2 due to increased brain metabolic needs. On the other hand, an increase in SjO2 may be the result of hyperemia [4] or failure of oxygen extraction. Another possibility exists in patients with high SjO2 who may have a very low CPP due to a high ICP as a preterminal event with arterial blood deviation [12] Fig. 8.1.

The fact that SjvO2 provides only information about global and nonregional disorders of brain oxygen consumption may lead to misinterpretation of typical SjvO2 values. In cases where posterior circulation is compromised, the brainstem oxygen supply is threatened. Posterior circulation contributes a small amount of drainage to the jugular bulb, and impaired brainstem oxygenation may go unnoticed with normal SjvO2 levels.

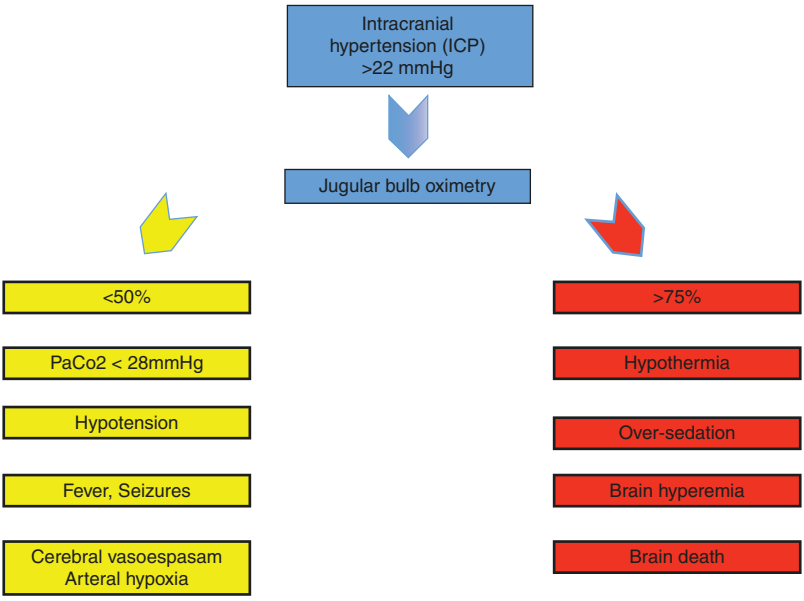

Fig. 8.1 Flowchart for evaluating jugular bulb oximetry results

8.6.3 Head Trauma

Traumatic brain injury (TBI)—the "silent epidemic"—contributes to death and disability worldwide more than any other traumatic insult. Sixty-nine million (95% CI: 64–74 million) individuals are estimated to be victims of head injury annually. One of the leading causes is auto-accidents. The proportion of TBIs resulting from traffic crashes is highest in Africa and Southeast Asia (both 56%) and the lowest in North America (25%). The overall incidence of TBI per 100,000 people is highest in North America (1300: 100,000) and Europe (1012: 100,000) [14]. According to Lewis et al. [31] in patients with acute blunt head injury, it is possible to safely detect episodes of cerebral venous desaturation mainly caused by hypocapnia in 45% of all observations, hypoperfusion in 22%, increased ICP in 9%, or a combination of any of these events by 24% [31]. These results demonstrate that these patients are at risk of a mismatch between oxygen supply and consumption and that most of the triggering factors can probably be avoided. Early monitoring after the initial injury is essential because many subsequent events occur in this early period. The most unstable period is usually in the early days when many episodes of jugular bulb desaturation were demonstrated. Eighty-three percent of these events occurred within 48 hours of injury [12, 13]. CBF is at its lowest level during the first 12 h after injury [3]. Many studies consider that CPP is more critical than ICP and that CPP should be maintained above 70 mmHg to prevent cerebral ischemia, evidenced by a low SjO2 [5].

Besides, as acute events noted, the brain's self-regulating mechanisms do not function properly, and SjO2 monitoring becomes an even more critical tool for guiding therapy. An example is a fact that mannitol may initially reduce brain oxygenation. This can be detected early by SjO2 monitoring and treated when PaCO2 is increased [28]. In addition to confirming the deleterious effects of low CPP or systemic desaturation, SjO2 can be used to guide interventional therapies. The initial active treatment for a high ICP is to "hyperventilate" the patient to lower PaCO2 levels and thus decrease ICP via cerebral vasoconstriction. It is impossible to know the PaCO2 threshold for each patient before cerebral ischemia occurs unless SjO2 is monitored [49]. This is because an individual's PaCO2 threshold depends on their CPP, steady-state PaCO2, and comorbid factors such as atherosclerosis. SjO2 monitoring allows therapeutic hypocapnia until SjO2 is near the lower limit of normal.

Extreme hyperventilation is harmful in severe intracranial lesions. Cruz et al. showed a reduction in SjO2 when hypocapnia was induced for ICP control. Therefore, hyperventilation as a therapeutic maneuver is a strong indication for SjO2 monitoring. In its absence, hyperventilation with a minimum PaCO2 is restricted to 30 mmHg [32]. We point out that in recently published guidelines, prophylactic hyperventilation is not recommended, and there is no evidence to justify that jugular bulb oxygen saturation as an auxiliary method for how hyperventilation can be performed. Although there is no evidence for measuring SjVO2, there is no contrary evidence either. Another author describes hyperoxia as another therapeutic maneuver to allow the lowest possible PaCO2, preserving global oxygenation as monitored by SjO2. Other studies do not corroborate such measures, and recent studies are demonstrating that hyperoxia increases mortality in head injury patients [10].

8.6.4 Subarachnoid Hemorrhage

One of the leading causes of morbidity and mortality in subarachnoid hemorrhage (SAH) is vasospasm. Patients suffering from intracerebral or subarachnoid hemorrhage had a 90% chance of developing episodes of cerebral venous desaturation during their disease, as opposed to just 50% in head trauma patients [49]. Despite this, jugular venous oximetry is occasionally used in aneurysmal subarachnoid hemorrhage as a tool to identify changes in cerebral oxygen metabolism. The use of transcranial Doppler (DTC) to diagnose vasospasm has become quite common. One of the difficulties with TCD is distinguishing hyperemia from vasospasm, and the management of these two conditions is markedly different. SjVO2 may be used in this setting because patients with hyperemia would demonstrate marked venous oxygen saturation, while in severe vasospasm, saturation would be decreased [59]. Von Helden et al. demonstrated that SjvO2 could be valuable for monitoring cerebral ischemia in SAH. They described a patient who had a 60% SjvO2 initially with a fall to 55% when TCD demonstrated an increase in flow velocity consistent with

vasospasm. Later, the flow velocity increased. Further, it was noted that SjvO2 fell below 50%.

The patient suffered a heart attack and died [58]. Fandino et al. also confirmed the ability of the jugular bulb oximetry to detect cerebral oxygenation patterns in vasospasm and differentiate them from nonvasospastic conditions that may also occur in SAH. They evaluated SjvO2 before, during, and after intra-arterial papaverine infusion, with or without balloon angioplasty, in patients with symptomatic vasospasm. Twenty-three vascular territories in 10 patients were treated. A significant improvement in SjvO2 was observed in all cases, with an improvement in brain oxygenation after endovascular vasospasm treatment ($P = 0.005$).

In their study, there was no attempt to use jugular venous oximetry as an aid to detect or predict vasospasm. Few studies have been performed demonstrating the AVDO2 baseline observed correctly in SAH before vasospasm, and the results were neither consistent nor clear. Gibbs et al. studied 50 normal young men and found a SvjO2 average of 61.8%, with a range of 55–71%. Kawamura et al. [26] studied nine patients with SAH and demonstrated that in the prevasospasm stage, the brain was relatively hyperemic, with CBF close to regular and smaller than usual CMRO2, compared to the vasospasm stage when CMRO2 decreased in parallel with CBF. The study by Heran et al. confirmed the jugular oximetry ability to predict the onset of clinical vasospasm in a small group of patients. In the four patients who developed vasospasm, a significant increase ($P < 0.001$) of cerebral AVDO2 was demonstrated on average, 26 hours before clinical changes were noted. This was not observed in patients who did not develop clinical vasospasm.

8.6.5 Ischemic Stroke and Children

In patients with an ischemic stroke, there are few studies with inconclusive results, and routine monitoring of venous saturation of the jugular bulb is not indicated. At present, there is no evidence to support that monitoring SjvO2 in children would have any additional prognostic benefit over other established prognostic predictors such as cerebral perfusion pressure, and GCS. Given the potential risk in children (thrombosis, infection, etc.), the benefit of using SjvO2 monitoring is undetermined. Additional studies on the overall value of monitoring jugular venous saturation to guide therapy in children are needed before widespread use is recommended.

8.6.6 Jugular Bulb Oximetry and Clinical Outcomes

SjO2 abnormalities were associated with a poor outcome compared with patients who did not demonstrate this physiological disorder. In a study of more than 100 patients admitted to intensive care after traumatic brain injury, Gopinath et al. reported a correlation between SjvO2 desaturation and final neurological prognosis

in case of brain injury. A poor final neurological prognosis was obtained in 90% of patients with recurrent SjvO2 desaturation events. In patients who had no SjvO2 desaturation events, the poor neurological prognosis was found in only 55% of these patients [21]. Cormio et al. reported that in 450 patients with severe head trauma treated at the intensive care unit, 25.6% of patients with increased SjvO2 had a better outcome (functional recovery, moderate disability), 25.6% recovered with severe disability, and 48.8% died or remained a vegetative state. In other prognostic models, as shown by Senapathi et al. [51]. Desaturation episodes measured in the jugular bulb and FOUR scores were considered significant predictors of mortality. Sharf and El-Gebali also reported that GCS ($P = 0.008$) and SjvO2 ($P< 0.001$) were significant predictors of mortality.

In a series of 50 patients with severe head trauma followed in 42% by multiple injuries, who were promptly treated were transferred to hospital, some aspects such as age, tomographic findings and clinical severity (as judged by the Glasgow Coma Scale, pupillary reactivity, and APACHE score), showed no statistically significant correlations with the outcome. Only the occurrence of two or more episodes of jugular venous desaturation correlated with clinical outcome. The nature of the population studied, and the relatively small number of patients may, however, have influenced the results. On the other hand, high values of SjO2 were also associated with poor results [16].

In general, even though studies in adults show the importance of measuring jugular oximetry mainly in trauma and subarachnoid hemorrhage, there are methodological limitations of the studies that do not allow absolute conclusions to use this monitoring method as a prognostic tool. It is observed that the more extensive discussion related to the diagnostic methods and prognostic estimation is influenced by the actions taken from the obtained results.

8.7 Complications

As with any invasive procedure, iatrogenic lesions may occur, the incidence of which is reduced if standard cannulation techniques are followed. The main complications relate to puncture and length of stay of the catheter. Some studies reported increased intracranial hypertension, but such findings were not corroborated. In a study with pediatric patients, Goetting measured intracranial pressure in 28 patients with jugular bulb catheters and found that neither cannulation nor the presence of in situ catheters further increased pressure [20]. In an observational study of 44 patients with jugular bulb catheters, Coplin et al. [6] concluded that complications related to catheter insertion were rare and clinically insignificant and that the risk of catheter-related bacteremia was negligible. However, on ultrasound, the incidence of subclinical thrombosis of the internal jugular vein after monitoring the jugular bulb catheter was up to 40% with in situ catheters for up to 6 days. Patients with proven thrombus had no symptoms.

Regarding infection in a study by Latronico et al., 73 catheters used in the study population and 11 (15%) cultures revealed colonization (Staphylococcus epidermidis, nine patients, Staphylococcus aureus, two patients). In two patients, *S. aureus* bacteremia was documented; however, microorganisms were isolated in the jugular and subclavian vein catheters [30]. In a risk balance, considering insertion complications and invasive catheter-related infections, monitoring the jugular bulb oximetry is a safe procedure with a low infection rate.

8.8 Conclusions

There is abundant evidence to support the fact that physiological insults secondary to the injured brain result in further brain damage. Monitoring jugular bulb saturation provides early warning of cerebral ischemia due to systemic disorders such as hypotension and hypoxia, and allows therapeutic maneuvers to be performed safely without inducing cerebral ischemia. The benefits far outweigh the risks associated with the technique. After years of enthusiasm, interest in jugular saturation has waned, and more modern methods such as tissue oxygen monitoring are now available. Jugular saturation monitoring has low sensitivity, with the risk of losing low saturation but high specificity. In addition, it is inexpensive when used with intermittent sampling. SjO2 monitoring should be considered, mainly if associated with other diagnostic methods such as ICP monitoring, transcranial Doppler, PtiO2 monitoring, brain microdialysis as well as imaging methods (tomography and resonance). In this context, the final neurological damage represented by cerebral ischemia may have minor clinical consequences.

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