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Monitoring of Intracranial Pressure (ICP) and Tissue Oxygen Pressure (Ptio2) in Acute Brain Injury: Diagnostic and Therapeutic Approach

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ABSTRACT
Acute Brain Injury, stemming from various causes such as trauma, hemorrhages, and diseases, leads to intracranial hypertension and cerebral oxygen deprivation. The lack of prevention and treatment can be fatal, necessitating Multimodal Neuromonitoring for these patients in Intensive Care Units. Despite the historical significance of Intracranial Pressure (ICP), cerebral oxygenation monitoring, measured by Ptio2, revealed that normal ICP values do not ensure optimal brain oxygenation. The integration of Cerebral Tissue Oxygen Pressure (Ptio2) monitoring has improved outcomes, reducing social, economic, and health-related costs. This work aims to serve as a theoretical-practical tool for professionals managing neurocritical patients, examining the monitoring and management of ICP and Ptio2 in acute brain injury. The findings emphasize the importance of Ptio2 monitoring associated with ICP, enabling more precise therapeutic decisions for patients with acute brain injury.

Keywords: Acute brain injury, Intracranial pressure, Cerebral tissue oxygen pressure, Cerebral hypoxia

INTRODUCTION

Acute brain injury (AHI), arising from various etiologies such as traumatic brain injury (TBI), intracerebral hemorrhages (ICH), tumors with mass effect, vasospasm secondary to subarachnoid hemorrhage (SAH), ischemic cerebrovascular disease (CVD) and infectious neurological pathology, converge in a common final stage: intracranial hypertension and hypoxia of brain tissue. Ischemic hypoxia is its most common modality, which leads to the extinction of energy production and, if not prevented, identified, and treated properly, can result in death, according to Domínguez-Roldán, J. M (2020).

Intracranial pressure (ICP) monitoring is considered integral in the clinical care of a variety of life-threatening brain injuries, including severe brain trauma, subarachnoid hemorrhage, and malignant cerebrovascular infarction, among others. This technique informs about the expansion of brain injuries, prevents or treats hernias, and helps manage pressure that affects the supply of nutrients to the brain (such as oxygen and glucose). It facilitates the calculation of cerebral perfusion pressure (CPP) and the estimation of cerebrovascular autoregulation status. In some cases, additional monitoring, such as partial pressure of oxygen in brain tissue (PbtO2), can be used to interpret ICP monitoring information and tailor treatment in a personalized way for each patient (Hawryluk, 2022).

Therefore, in neurocritical patients it is crucial to optimize hemodynamic, metabolic and brain oxygenation variables to improve results. Multimodal monitoring plays a crucial role in guiding the physician toward the best treatment for each patient. It has been observed that the correlation between intracranial pressure, tissue oxygen pressure and cerebral perfusion pressure helps to optimize management. Including the measurement of tissue oxygen pressure in multimodal monitoring in patients with acute brain injuries helps to make more accurate decisions, allowing treatments to follow a logical order, according to Toro López, S (2016). Continuous monitoring of cerebral oxygenation in neurologically severe patients is a current challenge in critical care medicine. Although there are several techniques to monitor brain oxygenation, brain tissue oxygen monitoring offers relevant information about oxygen levels in brain tissue. Its development not only addresses technical aspects, but also the importance of alterations in brain oxygenation in neurocritical patients, according to Domínguez-Roldán, J.M (2020).

Until two decades ago, intracranial pressure (ICP) monitoring was the only way to detect cerebral hypoxia through cerebral perfusion pressure. Subsequently, jugular oxygen saturation (SjO2) began to be measured and

it was shown that there could be normal ICP and CPP regardless of the degree of cerebral oxygenation, later corroborated with the measurement of Cerebral Tissue Oxygen Pressure (PtiO2) as part of Multimodal Neuromonitoring, according to Murillo-Cabezas, F (2014). In the clinical management of patients with traumatic brain injury and other acute neurological pathologies, the parameters used for monitoring are intracranial pressure and cerebral oxygenation, according to Murillo-Cabezas F (2016) (currently). The understanding of brain metabolic variables expands the information for the management of neurocritical patients with acute brain injury, including the measurement of ICP as a basic parameter and the direct measurement of cerebral tissue oxygen pressure (PtiO2) as part of advanced monitoring for cerebral oxygenation, considered the gold standard in the monitoring of cerebral oxygenation at the patient's bedside. being the second parameter measured after ICP.

The present review aims to review current knowledge related to the monitoring of intracranial pressure (ICP) and tissue oxygen pressure (Ptio2) in acute brain injury; and to become a theoretical-practical tool for health personnel working in hospitals that manage neurocritical patients with acute brain injury.

MATERIAL AND METHODS

This study is based on the compilation of updated information available in databases such as Scopus, SciELO, Web of Science, EMBASE, Ovid-Medline, Google Scholar and PubMed to date. The following keywords were used for the search: "Intracranial pressure", "Cerebral tissue pressure of oxygen", "Cerebral hypoxia" and "acute brain injury". We identified studies related to monitoring brain tissue oxygen pressure together with intracranial pressure, which showed greater effectiveness compared to monitoring intracranial pressure alone in improving outcomes in patients with acute brain injury. The author carried out a thorough analysis of the articles before the final writing of this document.

RESULTS

Physiology And Pathophysiology: Intracranial Pressure

Intracranial Pressure (ICP) throughout history

Intracranial Pressure (ICP) is defined as the pressure inside the cranial vault and has been studied over time. Cerebral edema and the benefits of skull opening were understood by Galen, Hippocrates, Egyptian physicians, and Incas. However, the modern understanding of intracranial pressure (ICP) and its determinants can be traced back to the work of Alexander Monro and George Kellie in the late eighteenth century. His model, the Monro-Kellie doctrine, was later refined by Harvey Cushing, detailing the basic principles governing the PIC. This pressure is determined by the interrelationship between the skull and its intracranial volume, which is constant and is made up of the brain (1300 cc), blood (110 cc) and cerebrospinal fluid (65 cc).

Monro-Kellie Doctrine: Basic Principles of the CIP

The Monro-Kellie Doctrine refers to the interrelationship between a continent and a content. The continent, represented by the skull, is a non-expandable bony structure that generates a constant volume within the cranial cavity. This cavity will be occupied by a constant volume under normal conditions, composed of the brain parenchyma (80%), cerebrospinal fluid (CSF) (10%), and blood (10%) (arterial 3-4% and venous 6-7%). When one of the intracranial components increases, it is compensated by the displacement of the other two. This mechanism allows the intracranial volume and, therefore, the intracranial pressure to be kept constant, according to Harary, M., Dolmans, R., & Gormley, W. B. (2018).

However, when this compensation is exhausted, a linear increase in volume leads to an exponential increase in intracranial pressure (ICP), as we can see in the pressure-volume curve (Figure 1A). This curve has the following zones: (1) High complacency with a good compensatory reserve; (2) Diminished complacency with gradual depletion of the compensatory reserve; (3) Minimal complacency with poor compensatory reserve and increased risk of ischemia and brain hernia; (4) Critically high ICP leading to collapse of the cerebral microvasculature and impaired cerebrovascular reactivity.

Normal and Pathological Intracranial Pressure

Intracranial pressure is defined as the pressure within the cranial vault and has physiological limits of 7 to 15 mm Hg in adults in the supine position, 3 to 7 mm Hg in children, and 1.5 to 6 mm Hg in infants. However, mean intracranial pressure in pediatric populations can vary by age and is not as well established. At all ages, frequent elevations with physiological increases in intrathoracic pressure can occur during Valsalva maneuvers, according to (Kukreti V, 2014). Alterations in intracranial pressure become clinically important and require treatment when increases exceed 22 mm Hg and remain elevated for more than 5 minutes, according to the BTF (Carney, 2017).

ICP Wave Amplitude (PICA)

The normal amplitude of the intracranial pressure wave (PICA) has an absolute value ranging from 1 to 5 mmHg, calculated by finding the difference between the maximum and minimum value of the intracranial pressure wave (Figure 1B).

Intracranial Hypertension and its Variants

Intracranial hypertension is an elevation of intracranial pressure above normal values due to various causes, which can become a medical-surgical emergency. For its management, criteria for intracranial hypertension are established when the ICP is greater than 22 mmHg, considering patients with Decompressive Craniectomy (DC) or lesions in the posterior fossa, temporal and deep frontal, where an ICP greater than 15 mmHg is considered, which directly affects the brainstem with a high risk of interlocking.

On the other hand, Refractory Intracranial Hypertension is considered when the ICP is greater than 22 mmHg or greater than 15 mmHg, depending on the case, without response to First Level measurements during a period of 30 minutes.

ICP Wave Studies

By obtaining the PIC, in addition to the absolute value, we can observe its morphology to understand how changes in it can provide information about self-regulation. The ICP wave is composed of two components: a cardiac and a respiratory component, related to the heartbeat and the respiratory cycle, respectively.

The ICP wave, due to its relationship with the heartbeat, is made up of three components or peaks:

- P1 Percussion wave: reflects the systolic blood pressure in the skull.
- P2 Tidal wave: represents 80% of P1 and is related to brain compliance.
- P3 Dirot wave: reflects venous flow in the skull, according to Hall, A., & O'Kane, R. (2016).

The graphical characteristics of continuous ICP monitoring were described by Lundberg. Under physiological conditions, the recording is pulsatile and is associated with cardiac and respiratory cycles. However, when there are pathological conditions that elevate ICP, continuous recording may present three different morphologies known as pathological Lundberg waves:

- A-waves, or plateaus, indicate a decrease in intracranial compliance, observed in patients with expansive lesions or hydrocephalus. These waves result from a hemodynamic phenomenon associated with vasodilation and the consequent decrease in cerebral perfusion pressure (CPP), reaching ICP of 50 to 100 mmHg. They are related to alterations in vasomotor tone and can cause global hypoxic-ischemic alterations.
- B waves, are observed in patients with periodic breathing and are directly related to changes in PaCO2. The ICP could reach values of 20 to 50 mmHg.
- C waves, less relevant, manifest themselves with changes in blood pressure and can reach 20 mmHg.

Lundberg type A and B waves appear in pathological conditions and have clinical relevance (Figure 2B), according to Hall, A., & O'Kane, R. (2016).

Cerebral Hemodynamics and Autoregulation

In a normal brain, cerebral blood flow (CBF) remains constant at a level of 50 ml per 100 grams per minute, with a mean arterial pressure (MAP) of 60 to 150 mm Hg; this process is known as self-regulation, according to Bruzzone, P. (1998). Brain autoregulation operates to maintain adequate cerebral perfusion pressure (CPP), defined as the pressure needed to perfuse nerve tissue and ensure optimal metabolic functioning. This CPP is determined by the difference between MAP and intracranial pressure (ICP), and is adjusted by altering cerebral arteriolar resistance, according to Kinoshita K. (2016). However, self-regulation is only effective within a PPC range of 50 to 150 mmHg; outside this range, hypoperfusion or cerebral edema may occur, respectively (Figure 1C). In addition, the ability to self-regulate is also influenced by the blood pressure of carbon dioxide (PaCO2). Hypercapnia causes the dilation of the cerebral vessels, increasing the CBF and increasing the risk of hypoperfusion. Conversely, hypocapnia leads to vasoconstriction, increasing the risk of ischemia, according to Meng, L., & Gelb, A. W. (2015).

Vasodilation and Vasoconstriction Cascades

Vasodilation and vasoconstriction cascades represent extensive processes of self-regulation that can influence the diameter of cerebral blood vessels, thereby affecting cerebral blood volume and intracranial pressure, even in the presence of brain injury. These cascades were described by Rosner, based on clinical observations with significant relevance, by demonstrating that ICP can be reduced by increasing the supply of nutrients to the brain, according to Hawryluk, 2022.

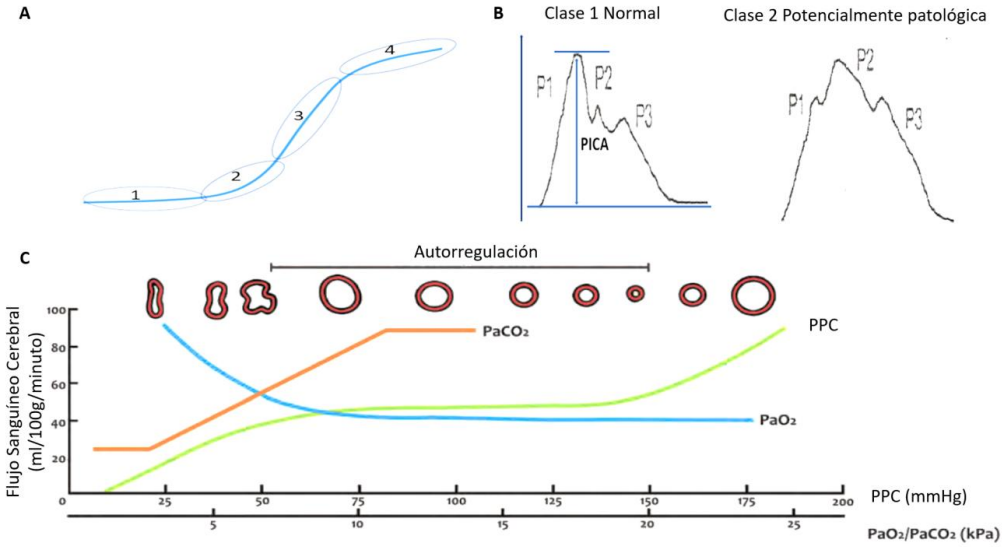

Figure 1. Brain physiology. A. Pressure-volume curve for intracranial pressure represented in 4 zones. B. Illustrative examples of the normal and potentially pathological class characteristic of intracranial pressure pulse waveforms. Class 1: P1 dominating over P2 and P3, Class 2: Increased prominence of P2 with P1 equaling or surpassing P3. C. Effect of changes in mean arterial pressure, oxygen and carbon dioxide blood pressures, and cerebral vascular resistance on cerebral blood flow. In original idopma English

Cerebral Oxygenation

Brain Metabolism

Between 15 and 25% of cardiac output is directed to the brain, with a cerebral blood flow (CBB) of 40-50 ml per 100 grams of brain tissue per minute. CSF is regulated by cerebral metabolic oxygen consumption (CMRO2), controlled through autoregulation by cerebral vascular resistance (CVR), and by cerebral perfusion pressure (CPP). CMRO2 is divided into approximately 40% basal energy expenditure (mainly to maintain membrane potential, unmodifiable by drugs but sensitive to temperature) and 60% functional energy expenditure (not sensitive to temperature but modifiable by drugs). Of this CMRO2, 90% is related to neuronal tissue and only 10% to supporting tissue or glia (despite the fact that the latter constitutes more than 50% of the brain volume). CMRO2 is in the range of 4-6 ml per 100 grams of brain tissue per minute. Therefore, pathological conditions such as anemia or hypoxia could reduce arterial oxygen content, which can result in inadequate brain oxygen delivery, according to G. Rodríguez (2015).

Brain Tissue Oxygen Pressure (Ptio2)

It is assumed that the value obtained from PtiO2 corresponds to the partial pressure of oxygen at the end of the capillary circuit, this being an average value of the vascular, intracellular and extracellular compartment. PtiO2 represents the partial pressure of oxygen in the cerebral extracellular space, a parameter of cerebral oxygenation that reflects the balance between cerebral perfusion, oxygen diffusion into brain tissue, and cellular oxygen consumption. It may present low or pathological values (cerebral hypoxia) in the presence of normal ICP and CPP levels. The pathophysiological basis for this fact lies in the description of the different types of tissue hypoxia (specifically, cerebral tissue hypoxia) provided by the Sigaard-Andersen classification, according to Domínguez-Roldán (2020). (Table 1)

Brain Oxygen Tissue Pressure Monitoring (Ptio2)

Tissue oxygen pressure is an important prognostic marker. The value of Ptio2 as a prognostic marker is widely recognized. Normal values of PTiO2 are considered to be greater than 25-30 mmHg. It is classified as mild hypoxia when it is below 20 mmHg, moderate between 15 and 20 mmHg, severe between 10 and 15 mmHg, and critical below 5 mmHg. Therefore, the therapeutic goal is to achieve values above 20 mmHg. A significant increase in the chances of death or severe disability has been observed in sustained periods with PTiO2 < 15 mmHg. A mortality of 50% is associated with values below 15 mmHg maintained for more than 4 hours. If PTiO2 is less than 10 mmHg for more than 30 minutes, mortality rises to 56%. The contribution of a low PTiO2 to a poor prognosis is independent of the PIC or PPC figures. Episodes of moderate and severe tissue hypoxia have been documented that have resulted in the appearance of areas of cerebral infarction, even with normal values of CPP, according to A.M. Domínguez-Berrot (2014). (Table 1)

Table 1. International consensus on the monitoring of cerebral tissue oxygen pressure in neurocritical patients.

Types Of Cerebral Hypoxia Based On The Sigaard-Andersen Classification	Types Of Hypoxia Detectable By Ptio2 Monitoring
1. Ischemic hypoxia 2. Hypoxia due to low extraction 3. Hypoxia due to dysperfusion 4. Hypermetabolic hypoxia 5. Hypoxia by shunt 6. Histotoxic hypoxia 7. Decoupling hypoxia	1. Ischemic hypoxia 2. Hypoxia due to low extraction 3. Hypoxia due to dysperfusion 4. Hypermetabolic hypoxia 5. Hypoxia by shunt

Technological Considerations

Ptio2 Measurement and Monitoring Systems

For the measurement and monitoring of cerebral tissue partial pressure (PtiO2), it is achieved from the introduction into the brain parenchyma of a small caliber oxygen-sensitive catheter, based on the Clark electrode described in the fifties (55), and applied in humans in the early nineties with Meixensberger and his team suggesting that it could prevent hypoxic events in neurocritical patients and improve their prognosis. according to Domínguez-Roldán (2020) There are currently two monitoring systems for Ptio2. The Licox system, Integra Lifesciences, Plainsboro, NJ is the most widely used and is considered the "Gold Standard"; Also present are the Neurovent-P Temp, Raumedic AG, Münchberg, Germany.

The licox system is based on oxygen reduction with Clark-electrode (electro-chemical measurement method), which allows the quantification of oxygen pressure in a medium by the polygraphic method. This electrode has an electrolyte aqueous substance inside it and two metal electrodes (anode and cathode) covered by a membrane semipermeable to oxygen that on its entry is oxide-reduced in the tasting generating an electric current proportional to the pO2 in the medium and can be quantified. (according to A.J. MARÍN-CABALLOS 2008). The Neurovent-P Temp system, Raumedic is based on the oxygen quenching method of fluorescent light (oxygen quenching with fluorescent dye), so it is an optical measurement method, for which it has an oxygen microsensor called optode. The Licox system measures an area of 14-18 mm2 and has great long-term stability, even after one week of implantation. The Neurovent-p is more recent, comes with ICP included in the same catheter and measures a surface area of 24 mm2, according to Lubillo, S (2014).

Indications for PIC Monitoring- Cerebral Tissue Oxygen Pressure (PtiO2)

Apart from TBI, PtiO2 monitoring has many other uses such as in neurosurgery of aneurysms, arteriovenous malformations and resection of brain tumors, in the study of the effect of therapeutic maneuvers such as increased CPP, hyperventilation, normobaric hyperoxia, hypothermia, decompressive craniotomy, hyperosmolar treatment with mannitol, the use of anesthetic agents, etc., according to Matthew A (2016). (Table 2)

Table 2. Main indications for oxygen pressure monitoring in brain tissue and near-infrared spectroscopy in perioperative and intensive care settings, based on clinical data.

Modality	**Directions**	**Evidence**	**Higher Quality Evidence**
PIC-PtiO2	**INTENSIVE CARE UNIT**		
	Severe head trauma	A low PtiO2 is associated with higher mortality, a lower Glasgow Coma Scale result, and an increase in neuropsychological deficits.	Prospective observational
		Treatment of low PtiO2 may improve outcomes.	Randomized Clinical Trial
		PtiO2 can help define individual CSP thresholds.	Prospective observational
		Response to PtiO2-guided therapy is associated with a reduction in mortality.	Retrospective analysis of prospective observational data
	Low-grade subarachnoid hemorrhage	Low PtiO2 values are associated with higher mortality, but the relationship with morbidity is less clear.	Prospective observational
		Assessment of the self-regulating oxygen reactivity index derived from PtiO2 can predict the risk of late cerebral ischemia and unfavorable outcomes.	Prospective observational
		Response to PtiO2-guided therapy is associated	Retrospective analysis

		with improved long-term functional outcomes.	of prospective observational data
	Intracerebral hemorrhage	PtiO2 monitoring can help identify optimal targets of cerebral perfusion pressure.	Retrospective
		Reduced perihematomamal PtiO2 values are associated with a poor outcome.	Retrospective
	Acute cerebrovascular event	The cereal perfusion pressure of the oxygen reactivity index; PtiO2-derived can predict the development of malignant middle cerebral artery infarction	Prospective observational
	PERIOPERATIVE		
	Cerebral angiography	Low PtiO2 values are correlated with a severe reduction in intracranial angiographic arterial caliber in patients with low-grade subarachnoid hemorrhage.	Retrospective
	Aneurysm surgery	A PtiO2 threshold of 15 mm Hg is found as a sensitive indicator of the likelihood of developing procedure-related ischemia	Prospective observational
	Arteriovenous malformation surgery	A correlation is observed between reduced PtiO2 values and the development of cerebral ischemic infarction.	Case Report

Protocol Method Of Placement: Insertion Of The Sensor

Catheter Placement and Functional Verification

The catheter is placed by a trained neurosurgeon or neurointensivist, usually after perforation, using a double-lumen catheter in the Licox system: one for ICP and one for Ptio2. In contrast, in the Neurovent-p system, both measurements are performed on a single catheter that includes PIC, Ptio2, and Temperature. It is crucial that the catheter tip is located in the white matter because of its greater metabolic stability compared to the gray matter. Once placed, its operation is verified by increasing the FiO2 to 100%, which should cause a notable increase in the PtiO2. A brain CT scan is then performed to confirm its proper placement. It is important to consider that even the slightest injury caused by the insertion of the sensor into the brain parenchyma can affect the reliability of initial PtiO2 readings over a period of 30 to 120 minutes, according to Lubillo, S (2014). (Figure 2)

Risks and Complications

There is a risk of infection that manifests itself approximately from the fifth day of implantation. Sometimes, sensors may be placed in other areas of the brain. In addition, possible complications include bleeding, according to Li L, Timofeev I, Czosnyka M (2010).

Figure 2. Sequence of Steps for the Placement of the PIC-Cerebral Oxygen Catheter. In original language English

Protocols For The Treatment Of Intracranial Hypertension-Cerebral Hypoxia Acting in a Patient with Low Ptio2

A simple scheme is proposed that can facilitate decision-making in cases of cerebral hypoxia, evidenced by a value below 15-20 mmHg. The most common cause of cerebral hypoxia is ischemic hypoxia, which occurs in cases of elevated ICP or insufficient CPP. A study published by Oddo et al. details the causes of cerebral hypoxia in 103 patients: 50% had a combination of high ICP and low CPP; in the remaining 25%, there was normal ICP with low CPP, while in the remaining 25% the causes were other, such as hypothermia, relative anemia or hypoxemia, according to Domínguez-Roldán (2020).

Protocols for the Treatment of Intracranial Hypertension-Cerebral Hypoxia

The first treatment algorithms for severe traumatic brain injury, published by the BTF, widely recommended measuring ICP and CPP, and in the Level III recommendation, monitoring oxygen in brain tissue (PtiO2). This recommendation was removed due to conflicting evidence obtained from the third to fourth editions of the BTF in 2016, which left a gap in neuromonitoring. To address this problem, the Consensus Conferences In SPAIN 2020 emerged. The continuous monitoring of cerebral oxygenation and its application in severe neurological patients are current challenges in critical medicine. Although there are several techniques described for monitoring cerebral oxygenation, cerebral tissue oxygen monitoring provides relevant information about oxygen levels in brain tissue. Its development has sought not only technical aspects but also the meaning of the alteration of brain oxygenation values in neurocritical patients.

Management algorithms have been developed, such as that of the Seattle International Consensus Conference on Severe Brain Injury (SIBICC), developed by an international and multidisciplinary group of more than forty experts in severe TBI. This algorithm is available in different languages in https://globalneuro.org/EN/resources/sibicc-stbi-algorithm. html "SIBICC Severe TBI Algorithm for patients with ICP monitoring". (Table 3)

Table 3. SIBICC algorithm commented by expert in Neurointensivism.

SIBICC II ALGORITHM	
TYPE A: PtiO2 > 20 mmHg - PIC < 22 mmHg **BASIC CARE:** Applicable to all Patients with Severe TBI	
EXPECTED INTERVENTIONS:	
• Admission to the ICU • Endotracheal intubation and mechanical ventilation • Serial assessments of neurological status and pupillary reactivity • Raise the head of the bed (HOB) 30-45° • Analgesia to manage signs of pain (not directed at the ICP) • Sedation to prevent agitation, ventilator asynchrony, etc. (not directed to the PIC) • Temperature management to prevent fever: measure the core temperature and treat it if it is above 38°C • Consider anti-seizure medications only for 1 week (in the absence of indication to continue)	• Maintain Cerebral Perfusion Pressure (PPC) initially ≥ 60mmHg • Maintain Hb > 7g/dL • Avoid hyponatremia • Optimize venous return from the head (e.g., keep the head in midline, make sure the cervical necks are not too tight) • Arterial line for continuous blood pressure monitoring Maintain SpO2 ≥ 94%
RECOMMENDED INTERVENTIONS:	
• Insertion of a center line	• ETCO2 monitoring
TYPE B: PtiO2 > 20 mmHg - ICP > 22 mmHg (ELEVATED ICP – NORMAL CEREBRAL OXYGENATION)	
LEVEL 1	
• Maintain CPP 60-70 mmHg • Increase analgesia to reduce ICP • Increase sedation to reduce ICP • Maintain maximum PaCO2 at the low end of normal (35-38 mmHg/4.7-5.1 kPa) • CSF drainage if an external ventricular drainage device is in place	• Administer mannitol in an intermittent bolus (0.25-1.0 g/kg) • Intermittent bolus hypertonic saline • Consider anticonvulsant prophylaxis for one week only (unless indicated to continue) • Consider EEG monitoring
LEVEL 2	
• Mild hypocapnia (range 32-35 mmHg/4.3-4.6 kPa) • Neuromuscular paralysis in adequately sedated	• Initiate or adjust a vasopressor or inotropic to increase MAP by 10 mmHg for no more than 20

patients if effective in reducing ICP • Perform mean arterial pressure (MAP) challenge to assess brain autoregulation and guide MAP and CPP targets in individual patients. • Must be performed under the direct supervision of a physician who can assess response and ensure safety • No other therapeutic adjustments (i.e., sedation) should be made during the MAP challenge	minutes • Monitor and record key parameters (MAP, CPP, ICP and PbtO2) before, during and after the challenge • Adjust vasopressor/inotropic dose based on study findings Increase CPP with boluses of fluids, vasopressors, and/or inotropics to reduce ICP when autoregulation is intact
LEVEL 3	
• Eat with pentobarbital or thiopental titrated to control ICP if effective • Secondary decompressive craniotomy • Mild hypothermia (35-36°C) by active cooling measures	• Hyperventilation up to PaCO2 of 30-32 mmHg/4.0-4.3 kPa
TYPE C: PtiO2 < 20 mmHg - PIC < 22 mmHg (NORMAL ICP – CEREBRAL HYPOXIA)	
LEVEL 1	
• Maintain maximum CPP of 60 to 70 mmHg • Increase CPP to a maximum of 70 mmHg with fluids, vasopressors, and/or inotropics • Keep PaCO2 > (35 mmHg/4.7 kPa)	• If PaO2 is already in the desired range, further increase PaO2 by increasing FiO2 to 60% • Consider EEG monitoring
LEVEL 2	
• Ventilatory management to increase PaO2 up to 150 mmHg/20 kPa • Reduce ICP to a threshold < 22 mmHg • Consider CSF drainage • Increasing sedation to improve mechanical ventilation and PbtO2 • Neuromuscular paralysis in adequately sedated patients if effective in increasing PbtO2 • Perform mean arterial pressure (MAP) challenge to assess brain autoregulation and guide MAP and CPP goals in individual patients - Must be performed under the direct supervision of a physician who can assess response and ensure safety	- No other therapeutic adjustments (e.g., sedation) should be made during the MAP challenge - Initiate or adjust a vasopressor or inotropic to increase MAP by 10 mmHg for no more than 20 minutes - Monitor and record key parameters (MAP, CPP, ICP, and PbtO2) before, during, and after the challenge - Adjust vasopressor/inotropic dose based on study findings • Increase CPP to increase PbtO2 when supported by the MAP Challenge • Increase CPP above 70 mmHg with boluses of liquids, vasopressors, and/or inotropics
LEVEL 3	
• Increase PaCO2 to 45-50 mmHg/6.0-6.7 kPa (but avoid intracranial hypertension) • Consider normobaric hyperoxia up to a PaO2 above 150 mmHg/20 kPa	• If PbtO2 remains < 20 mmHg despite PaO2 and CPP/MAP optimization, consider transfusion of 1 unit of red blood cells if Hb <9g/L
TYPE D: PtiO2 < 20 mmHg - ICP > 22 mmHg (ELEVATED ICP – CEREBRAL HYPOXIA)	
LEVEL 1	
• Maintain CPP of 60 to 70 mmHg • Increase CPP to a maximum of 70 mmHg with fluids, vasopressors, and/or inotropics • Increase analgesia to reduce ICP/improve ventilation and PbtO2 • Increase sedation to reduce ICP/improve ventilation and PbtO2 • Keep PaCO2 > (35 mmHg/4.7 kPa) • Administer mannitol in an intermittent bolus (0.25-1.0 g/kg) • Intermittent bolus hypertonic saline	• CSF drainage if an external ventricular drainage device (EVD) is in place • Consider placement of EVD to drain CSF if a parenchymal tube was initially used • If PaO2 is already in the desired range, further increase PaO2 by increasing FiO2 to 60% • Consider anticonvulsant prophylaxis for one week only (unless indicated to continue) • Consider EEG monitoring
LEVEL 2	
• Ventilatory management to increase PaO2 up to 150 mmHg/20 kPa • Increase sedation to improve ICP and PbtO2	- Initiate or adjust a vasopressor or inotropic to increase MAP by 10 mmHg for no more than 20 minutes

• Neuromuscular paralysis in adequately sedated patients if effective in decreasing ICP or increasing PbtO2 • Perform mean arterial pressure challenge to assess brain autoregulation and guide MAP and CPP targets in individual patients - Must be performed under the direct supervision of a physician who can assess response and ensure safety - No other therapeutic adjustments (e.g., sedation) should be made during the MAP challenge	- Monitor and record key parameters (MAP, CPP, ICP, and PbtO2) before, during, and after the challenge - Adjust vasopressor/inotropic dose based on study findings • Increase CPP to decrease PIC and/or increase PbtO2 when supported by the MAP Challenge • Increase CPP above 70 mmHg with boluses of liquids, vasopressors, and/or inotropics
LEVEL 3	
• Eat with pentobarbital or thiopental titrated to control ICP if effective • Secondary decompressive craniotomy	• Consider normobaric hyperoxia up to a PaO2 above 150 mmHg/20 kPa • If PbtO2 remains < 20 mmHg despite PaO2 and CPP/MAP optimization, consider transfusion of 1 unit of red blood cells if Hb <9g/L

CONCLUSIONS

This work can become an instrument of help from the theoretical-practical point of view for health personnel who work in hospitals that care for neurocritical patients with acute brain injuries.

In the systematic review, two studies were identified that addressed the research problem posed, which proposed a different prediction model for the admission of patients diagnosed to an intensive care unit. These studies were critically assessed in the present review, identifying design problems and a high risk of bias.

The intracranial pressure monitor has shown practical utility in severe cases where a significant recovery in results has been observed. Although improvement has been observed even when the monitor has not been used, a particular case is mentioned at the beginning, a mild head trauma without the use of a monitor, which led to death. This raises the possibility of using the monitor also in mild and moderate cases, although this case was isolated and not statistically significant.

No statistically significant difference has been observed between the state at baseline (GOES) and at the end (GOS), maintaining an association from the beginning of the study: those with greater severity were those who were placed on the monitor.

The author suggests that future field research should be carried out to evaluate the usefulness of the monitor only in mild and moderate cases to identify possible differences, since the use of the monitor is currently established only in severe cases; and to evaluate the possible side effects of the use of the monitor and to establish evidence-based criteria for its use in health services, especially in moderate and mild cases.

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26. SIBICC Severe TBI Algorithm for patients with ICP monitoring https://globalneuro.org/EN/resources/sibicc-stbi-algorithm.html Video link placement of PIC-Ptio2. https://youtu.be/QaA--ExdmFI

Annexes

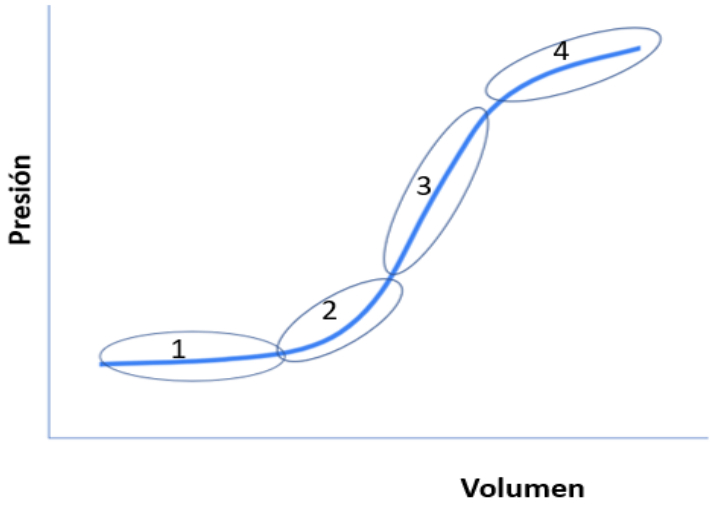

Figure 1. Pressure-volume curve. In original language English

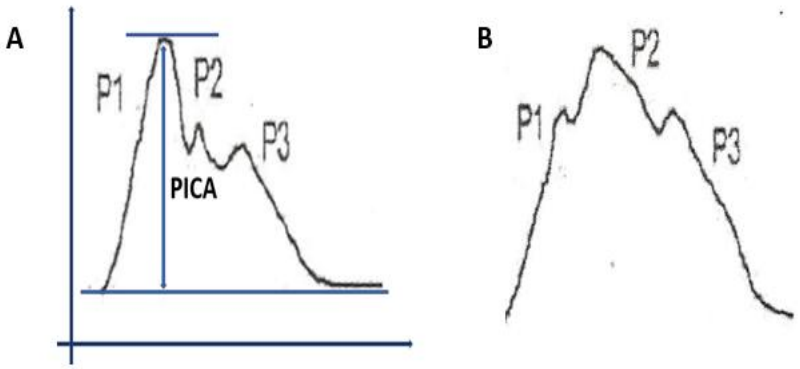

Figure 2. ICP pressure waves: A. Normal wave, B. Pathological wave. In original language English

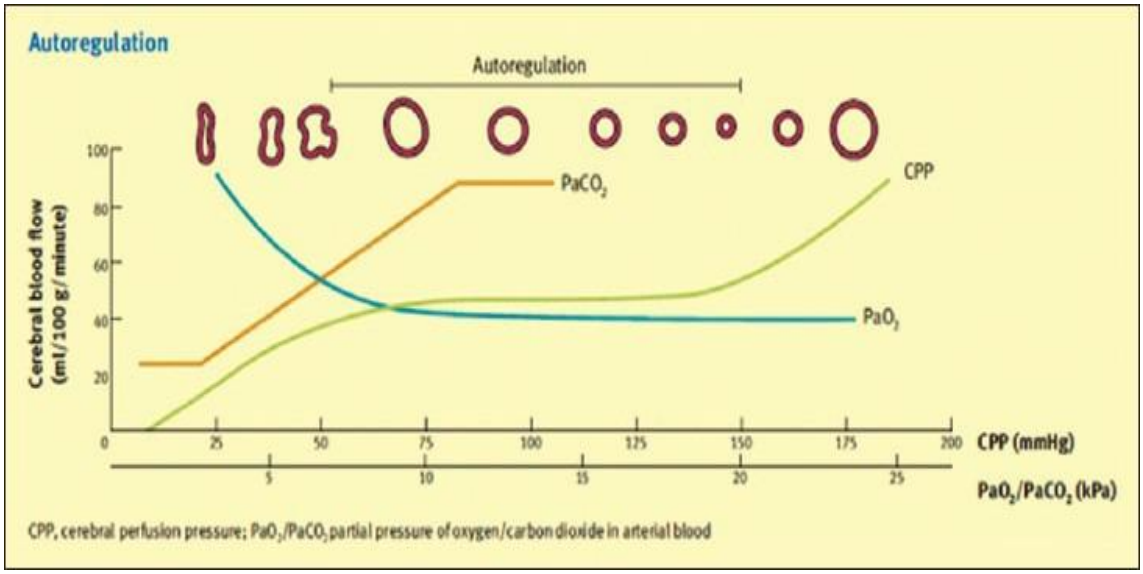

Figure 3. Brain autoregulation and the effect of blood gases, according to Tameem, A. and Krovvidi, H. (2013). In original language English

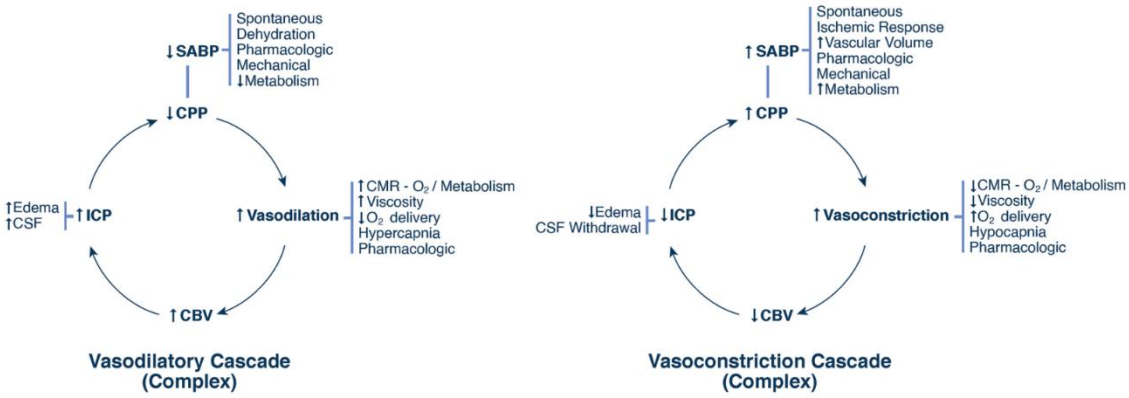

Figure 4. Rosner's vasodilation and vasoconstriction cascades. In original language English

Tabla 1 – Tipos de hipoxia cerebral basada en la clasificación de Sigaard-Andersen

1.- Hipoxia isquémica
2.- Hipoxia por baja extracción
3.- Hipoxia por disperfusión
4.- Hipoxia hipermetabólica
5.- Hipoxia por shunt
6.- Hipoxia histotóxica
7.- Hipoxia por desacoplamiento

(Consensus 2020, Spain)

Tabla 2 – Tipos de hipoxia detectables por la monitorización de la PtiO2

1.- Hipoxia isquémica
2.- Hipoxia por baja extracción
3.- Hipoxia por disperfusión
4.- Hipoxia hipermetabólica
5.- Hipoxia por shunt

(Consensus 2020, Spain)

Table 3. The main indicators of brain.

The main indications for brain tissue oxygen tension monitoring and near-infrared spectroscopy in the perioperative and intensive care settings, based on clinical data

Modality	Indications	Evidence	Highest Quality Evidence
$Ptio_2$	*ICU*		
	Severe TBI	Low $Ptio_2$ is associated with worse mortality,[47] lower GOS,[6,9] and increased neuropsychological deficits.[48]	Prospective observational
		Treatment of low $Ptio_2$ may improve outcomes.[10,20]	Randomized controlled trial
		$Ptio_2$ can help define individual CPP thresholds.[49]	Prospective observational
		Response to $Ptio_2$-guided therapy is associated with reduced mortality.[50]	Retrospective analysis of prospective observational data
	Poor-grade SAH	Low $Ptio_2$ values are associated with increased mortality, but the relationship with morbidity is less clear.[51]	Prospective observational
		$Ptio_2$-derived ORx autoregulation assessment can predict the risk of DCI[13] and unfavorable outcome.[52]	Prospective observational
		Response to $Ptio_2$-guided therapy is associated with improved long-term functional outcomes.[19]	Retrospective analysis of prospective observational data
	ICH	$Ptio_2$ monitoring may help identify optimal CPP targets.[53]	Retrospective
		Reduced perihematomal $Ptio_2$ values are associated with poor outcome.[53]	Retrospective
	AIS	$Ptio_2$-derived CPP-ORx may predict the development of malignant MCA infarction.[54]	Prospective observational
	Perioperative		
	Cerebral angiography	Low $Ptio_2$ values are correlated with severe intracranial angiographic arterial caliber reduction in patients with poor-grade SAH.[55]	Retrospective
	Aneurysm surgery	$Ptio_2$ threshold of 15 mm Hg is found a sensitive indicator of the likelihood of developing procedure-related ischemia.[56]	Prospective observational
	AVM surgery	Correlation is observed between reduced $Ptio_2$ values and development of a periprobe ischemic infarction.[57]	Case report

(Matthew A.2016)