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Traumatic Brain Injury: A View from Anesthesiology

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ABSTRACT

Traumatic brain injury (TBI) is one of the main causes of death and disability in children and young adults, with an increasing incidence in developing countries, secondary to motor vehicle accidents. Many survivors, are left with significant sequelae, which translates into a significant socioeconomic burden. TBI frequently occurs in the context of polytrauma; therefore, anesthetic management of these patients is complex and requires a multidisciplinary approach. In particular, surgery and anesthesia can lead to secondary injury to the brain, thereby worsening prognosis. This review addresses the epidemiology, pathophysiology, and anesthetic management of adult patients with severe TBI.

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Definition

According to the Center for Disease Control and Prevention (CDC), head trauma (TBI) is an injury that affects brain function. It can be caused by a blow, jolt to the head, or penetrating head injury [1,2]. According to severity, it is classified according to the Glasgow coma scale as mild (GCS 13-15 points), moderate (9-12 points), and severe (<9 points).

Epidemiology

Estimating the global burden of TBI is a complex task. Many patients with TBI, particularly those with minor injuries, do not seek medical attention. Furthermore, most of the high-quality epidemiological data come from high-income countries, while the majority of the world's population resides in low- and middle-income countries. Moreover, existing data suggest that the incidence of TBI varies substantially among countries and regions.

The Global Burden of Disease (GBD) study published the following estimates of TBI in 2016 [3].

- The global annual incidence was estimated at 27.08 million, with an age-standardized incidence rate of 369 per 100,000 inhabitants.
- The worldwide prevalence of TBI is estimated at 55.5 million, with an age-standardized prevalence rate of 759 per 100,000 people.
- In 2016, traumatic brain injuries resulted in 8.1 million years of disabled life worldwide (111 per 100,000, age-standardized).
- Stratified by the sociodemographic index (ISD), the age-standardized incidence rate was higher in the upper-middle category (468 per 100,000 inhabitants). While the global incidence increased by 3.6% compared to 1990, it fell by 9.4% in countries with the highest DSI, increased by 21.8% in countries in the medium DSI category, and fell by 9.3% in the lowest ISD category.
- Falls were the most common cause of TBI across all ISD categories, followed by motor vehicle accidents. It is thought that the reduction in TBI in countries with high DSI is the result of road safety regulations.

In Chile, the most affected group is in the 20-39 age range, and is Is responsible for 40% of deaths due to traffic accidents [4]. In the Public Assistance Emergency Hospital, of a total of 66,692 discharges (years 2011-2018), the diagnosis of TBI corresponded to 3.84%, with an average age of 46 years, which is slightly higher than that described in the literature; 79.4% of them, men. A total of 50.91% were admitted to the Critical Patient Unit (UPC), constituting 32.13% of the total discharges from this unit, with a consequent impact on both the biography of the victims and their families, as well as on hospital expenses [5].

Classification

TBI has traditionally been classified using injury severity scores; the most widely used is the Glasgow Coma Scale (GCS) (Table 1) [6]. A GCS score of 13-15 is considered a mild injury, 9-12 is considered a moderate injury, and 8 or less is considered a severe traumatic brain injury. The GCS is universally accepted as a tool for the classification of TBI because of its simplicity, reproducibility, and predictive value for the overall prognosis. However, it is limited by confounding factors, such as intoxication, sedation, paralysis, and endotracheal intubation. These confounding problems are particularly important in patients with a low GCS score [7,8].

Table 1

Glasgow Coma Scale Score
Eye opening
Spontaneous 4
Response to verbal command 3
Response to pain 2
No answer 1
Best verbal response
Oriented 5
Confused 4
Inappropriate words 3
Incomprehensible sounds 2
No answer 1
Best motor response
Obey verbal command 6
Locate the pain 5
Retreat to pain 4
Flexion to pain 3
Extension to pain 2
No answer 1

In addition, it is advisable to complement the diagnosis with classification based on morphological findings of computed tomography (CT) of the brain [5]. Among the lesions that can be identified by neuroimaging are [9].

- Skull fracture.
- Epidural hematoma (EDH).
- Subdural hematoma (HSD).
- Subarachnoid hemorrhage (SAH).
- Intraparenchymal hemorrhage.
- Brain contusion.
- Intraventricular hemorrhage.
- Focal and diffuse patterns of diffuse axonal injury with cerebral edema.

The Marshall and Rotterdam scales are two rating scales based on CT findings:

The Marshall score uses CT findings to classify injuries into six different categories (Table 2) [10]. It is widely used in neurotrauma centers and has been shown to accurately predict the risk of increased intracranial pressure (ICP) and outcomes in adults, although it lacks reproducibility in patients with multiple types of brain injuries [10]. The Rotterdam score is a more recent CT-based classification that was developed to overcome the limitations of the Marshall scale. It has shown promising initial results and an adequate correlation between the initial score and the prediction of early mortality [11].

Table 2: Marshall Score

Classification	Definition
Diffuse Injury I	No Visible Intracranial Pathology
Diffuse Injury II	Midline shift 0 to 5 mm, basal cisterns remain visible
Diffuse Injury III	Midline shift 0 to 5 mm Basal Cisterns Compresxed or Completely Effaced
Diffuse injury IV	Midline shift > 5 mm
Evacuated Mass Lesión V	Any Lesión Evacuated Surgically
Non-Evacuated Mass Lesión VI	High or Mixed Density Lesions > 25 cm3

Pathophysiology

The main mechanisms of TBI are classified as (a) focal brain damage due to contact type injuries resulting in contusion, laceration, and intracranial hemorrhage, or (b) diffuse brain damage due to acceleration/deceleration type injuries resulting in diffuse axonal or cerebral edema [12].

The outcome of a brain injury is determined by two substantially different mechanisms/stages:

(a) Primary injury that occurs at the moment of impact. In terms of treatment, this type of injury is exclusively sensitive to preventive measures; but not therapeutic.(b) Secondary lesions representing consecutive pathological processes initiated at the time of trauma with delayed clinical presentation that are amenable to therapeutic interventions [13]. Common mechanisms of primary injury include direct impact, acceleration/deceleration, penetrating injuries, and blast waves. Although these mechanisms are heterogeneous, they all come from external mechanical forces transferred to the intracranial contents that can result in skull fracture, brain contusion, and vascular and/or parenchymal injury, causing bleeding and increased intracranial pressure (ICP) [14]. An inflammatory process, edema formation, and excitotoxicity follow, leading to a further increase in ICP and a reduction in cerebral perfusion pressure (CPP) [14,15].

Although the severity of the primary lesion is the main factor that determines the outcome of patients with TBI, secondary damage caused by physiological disturbances contributes to worsening outcomes. It is characterized by increased excitotoxicity and formation of free radicals in cell membranes, electrolyte imbalance, mitochondrial dysfunction, nflammation, apoptosis, secondary ischemia due to vasospasm, focal microvascular occlusion, and vascular injury [14-17]. Among the most important secondary injuries are hypotension (systolic blood pressure [SBP] <90 mmHg in adult patients) and hypoxemia (PaO2 <60 mmHg), both independently associated with increased morbidity and mortality in severe TBI and with hypoperfusion and cerebral ischemia [16]. Other common secondary injuries include hypoglycemia, hyperglycemia, hypercapnia, hypocapnia, fever, seizures, and increased ICP [12]. All of these can manifest sooner or later during the course of TBI. Furthermore, the consequences of this may be evident in other organ systems besides the brain and may require immediate attention.

Systemic manifestations

The consequences of TBI may be evident in organ systems other than the brain and may require immediate attention (Table 3). Approximately one-third of patients with severe TBI develop coagulopathy, which is associated with an increased risk of bleeding progression, poor neurological outcomes, and death [17,18]. Although coagulopathy may be due to the patient's pre-existing medications, acute traumatic brain injury (TBI) is also thought to cause coagulopathy through the systemic release of tissue factors and brain phospholipids into circulation, leading to coagulation. intravascular and consumptive coagulopathy [19].

Cardiovascular dysfunction is common in patients with severe brain injury and occurs in 80%-90% of patients admitted to intensive care units [20]. Cardiovascular complications such

as electrocardiogram (ECG) changes, especially repolarization abnormalities, ischemia, and acute heart failure, mediated by pro-inflammatory cytokines and elevated catecholamines, can delay recovery or cause death [21,22]. However, some studies have the absence of cardiac dysfunction 24 h after TBI [23,24]. Between 20% and 25% of patients with TBI develop respiratory failure, which is associated with an increase in oxygen demand and/or a partial pressure of arterial oxygen/FrO ratio (Pa/Fi < 300). [25,26]. Acute pulmonary distress syndrome (ARDS) is characterized by increased vascular permeability, resulting in the deposition of protein-rich fluid in the alveolar space observed in TBI [27]. In addition, there is evidence that ARDS is due, at least in part, to lung cell damage and death, likely secondary to increased expression of various apoptosis-regulating proteins [28,29].

Table 3: Systemic Manifestations of TBI

• Cardiorespiratory
Abnormal Breathing/Hypoventilation/Apnea
Neurogenic Pulmonary Edema
Pulmonary Emboli
ARDS
Myocardial Ischemia or Stunned Myocardium.
• Metabolic
Hyperglycemia and Insuline Resistance
Increased Catecholamine Release
Catabolic State
• Autonomic Dysfunction
• Endocrinologic
Anterior Pituitary Gland Dysfunction (GH deficit, hypothyroidism, ACTH deficit).
Posterior Pituitary Gland Dysfunction (SIADH, DI).
Adrenal insuficciency
Cerebral Salt waste Syndrome
• Hematologic.
Coagulopathy
DIC
• GI
Stress Ulcers
GI dysfunction and Bacterial Translocation

Preoperative Evaluation

The goal of the preoperative evaluation of patients with TBI is to minimize secondary brain injury. Patients with TBI may require surgery for other associated injuries (e.g., orthopedic, abdominal, or thoracic). Even if such procedures are delayed beyond the immediate post-injury period, the goals of anesthesia should include prevention of secondary brain injury. The evaluation should be as complete as possible, although it may be limited by the accuracy of the clinical situation. In most cases, patients present to the operating room after initial evaluation and management. Clinical Assessment: This process should include assessment of the airway, breathing, and circulation; a rapid assessment of neurological status; and associated extracranial injuries, including details of comorbidities and medications. A patient's neurological status may interfere with their ability to provide a clinical history. Associated thoracic, abdominal, spinale, and long bone injuries may be stable or evolve during the perioperative period and should be considered in the differential diagnosis of new-onset hypotension, anemia, hemodynamic instability, or hypoxemia during anesthesia and surgery [13]. A rapid neurologic assessment was performed using the Glasgow Coma Scale (GCS) to stratify TBI severity of TBI (Table 1). The severity of TBI estimates the degree of physiological alteration, determines the urgency of a surgical procedure and postoperative care plan, and predicts the risk of complications and general prognosis [13].

Trauma surgery may be associated with hypotension and reduction in cerebral perfusion pressure, which can lead to secondary brain injury. For non-emergency extracranial surgery, patients with TBI should be resuscitated and stabilized before surgery to minimize the possibility of intraoperative hypotension. However, recovery from brain autoregulation can be delayed for weeks after TBI, particularly in patients with lower GCS scores, diffuse brain injury, and elevated ICP [30]. Patients with late recovery from cerebral autoregulation tend to have poorer outcomes [30].

Laboratory tests: Preoperative laboratory tests should be reviewed, including hemoglobin, platelet count, and coagulation parameters, electrolytes, glucose, and arterial blood gases. A sample should be subjected to crossmatching if significant blood loss is expected. Coagulation parameter abnormalities are common in patients with TBI and require ongoing evaluation and correction [13].

Anesthetic Management

The main goals of anesthetic management of TBI are to facilitate early decompression, provide adequate analgesia and amnesia, treat intracranial hypertension, maintain adequate cerebral perfusion, provide optimal surgical conditions, and avoid secondary insults, such as hypoxemia, hypercapnia, hypocapnia, hypoglycemia, and hyperglycemia.

Monitoring

The main objective of monitoring is to avoid events that may contribute to secondary brain injuries. In addition to the standard ASA monitoring recommendations (ECG, non-invasive blood pressure, pulse oximetry, capnography, temperature), it is usually recommended to place an arterial line for continuous BP measurement and frequent blood work, in addition to bladder catheterization, especially if the use of mannitol is planned [31]. Venous access should be tailored to the planned procedure, expected intraoperative blood loss, and the estimated preoperative blood loss. Even isolated TBI, scalp wounds, scalp dissection, and intracranial hemorrhage can cause large volumes of blood loss [13].

A central venous line (CVL) can be placed in patients with TBI to ensure venous access and, administration of vasopressors and hypertonic fluid. However, CVL placement should not delay evacuation of a rapidly expanding intracranial hematoma, and it is very uncommon to install them prior to cerebral decompression [13,32]. The choice of site for access varies by institution; some centers avoid the internal jugular site for central venous access in TBI patients because of the potential to increase jugular venous pressure and, therefore, increase ICP [12].

An ICP sensor is usually installed and is, useful for monitoring and/or managing changes associated with anesthetic/surgical treatment [13]. Novel techniques have recently been developed for advanced or multimodal neurological monitoring, mainly used in intensive care units, including jugular bulb oxygen saturation (SvyO2) and tissue oxygen pressure (PtiO2) monitoring were used more frequently, which is useful for guidinge ICP management measures [12].

Airway management

Although many patients arrive on the ward already intubated, some, particularly those with extradural hematoma, may be conscious and breathe spontaneously [30]. Airway management in TBI is complicated by several factors, including the urgency of the situation (due to pre-existing or worsening hypoxia), uncertainty of the status of the cervical spine, uncertainty of the airway (caused by the presence of blood, vomiting, debris in the oral cavity, laryngopharyngeal injury, or skull base fracture), full stomach, intracranial hypertension, and uncertain intravascular volume status. All patients with TBI requiring urgent surgery should be assumed to have a full stomach, and airway management should consider possible underlying cervical spine injury, the incidence of which is 4-8% in patients with severe TBI [32-34]. In any case, a backup plan is recommended if faced with a difficult airway.

The choice of technique for tracheal intubation is determined by urgency, individual experience, and available resources and generally incorporates rapid sequence intubation (RSI) with cricoid pressure and manual inline stabilization [35]. Newer airway devices, particularly video laryngoscopes, have recently gained popularity for use in trauma victims and may be useful in difficult airway settings [36].

Selection of drugs

Appropriate pharmacological selection is important for airway management without complications. The patient's hemodynamic status must always be considered to maintain CPP, avoid hypertension, the risk of intracranial hemorrhage, and increase ICP [13].

Propofol (1.5 -2 mg/kg IV): CMO2, CBF, cerebral blood volume (CV), and ICP are reduced by induction doses of propofol preservinge autoregulation and CO2 responsiveness [37-39]. There is a risk of hypotension and, the induction dose should be adjusted based on patient factors.

Thiopental (3-6 mg/kg IV): Barbiturates cause a dose-dependent reduction in CMO2, CBF, and ICP, while autoregulation and CO2 response are maintained [40,41]. Thiopental can produce isoelectric EEG. Therefore, the risk of arterial hypotension should also be considered. The induction dose should be decreased in elderly patients and in those at an increased risk of hypotension.

Etomidate (0.15 to 0.3 mg/kg IV): It is an induction agent that does not lower blood pressure or cardiac output. Administration of an induction dose decreases CBF and; CMO2 and reduces ICP without negatively affecting CPP; while preserving CO2 responsiveness [42,43]. However, even with a single induction dose, etomidate inhibits corticosteroid production in the adrenal gland for up to 24 hours [44,45]. Ketamine (1-2 mg/kg IV): Classically, it has been implicated in an increase in ICP, based on reports from the 1970s in spontaneously ventilating patients [46-49].

Subsequent studies that demonstrated the maintenance or even improvement of BP and CPP; did not report significant increases in ICP, and decreases in it were evident in patients with TBI and controlled ventilation. Furthermore, by blocking the NMDA receptor, ketamine has the potential to modulate the pain response and is neuroprotective by decreasing glutamate release associated with excitotoxicity [50,51].

Opioids: Opioids are usually administered as part of the induction of anesthesia to reduce the required dose of the induction agent, suppress airway reflexes, and attenuate the hemodynamic response to laryngoscopy and intubation. In this context, opioids have minimal effects on cerebral physiological parameters as long as the mean arterial pressure (MAP) is maintained [52,53].

Fentanyl, sufentanil, and alfentanil were used for rapid effects. Remifentanil, an ultrashort-acting opioid, can be used for induction but should be administered by infusion, with or without an initial bolus, to avoid abrupt compensation and resultant hypertension and tachycardia [54].

Lidocaine

Lidocaine (1-1.5 mg/kg IV) can be administered during induction of anesthesia to suppress the cough reflex during laryngoscopy and to attenuate, but not eliminate, the hemodynamic response to intubation Neuromuscular blockers: The choice of neuromuscular blocker (NMBB) for rapid sequence induction is between succinylcholine and rocuronium [55-57]. Non-depolarizing NMBs do not directly affect brain physiology. Some benzylisoquinolines (atracurium and mivacurium) could theoretically cause a reduction in CPP because of a decrease in MAP, cerebral vasodilatation, and an increase in ICP [58].

Succinylcholine, a depolarizing NMB, may contribute to increased ICP secondary to fasciculations and increased PaCO2, the clinical significance of which is questionable [59-62]. More importantly, hypoxia and hypercapnia during airway interventions are more likely to cause clinically significant increases in ICP. Therefore, succinylcholine cannot be avoided in patients with TBI, if a difficult airway is anticipated [62].

Maintenance

It has been experimentally shown that halogenated anesthetics produce an uncoupling between CMRO2 and CBF, decreasing the cerebral metabolic rate of oxygen and increasing CBF, resultings in a net increase in ICP. This occurs only at a minimum alveolar concentration (MAC) of approximately 1.5, although the increase in ICP is negligible if volatile agents are kept below 1 MAC [63]. However, even when kept below 1 MAC, volatile anesthetics have direct vasodilatory effects; that are independent of anesthetic depth and could theoretically affect ICP [64]. In contrast, intravenous anesthetics such as propofol decrease both CBF and CMRO2, resulting in an overall decrease in ICP. In practice, however, there is no clinical evidence to suggest the benefit of intravenous versus volatile anesthetics on patient outcomes [65]. It is important to note that decreased blood flow from hypotension is still very harmful to TBI patients and should be avoided regardless of the method of maintenance anesthesia.Nitrous oxide (N2O) can cause increases in FSC, CMRO2, and PIC. Autoregulation in response to changes in CO2 appears to be preserved when N2O is administered [66-69]. The magnitude of changes in brain physiology with N2O is affected by the administration of other anesthetic drugs and ventilation; as follows:

When used as the sole anesthetic, it can cause substantial increases in ICP and CBF in healthy patients and intracranial pathology. When added to halogenated inhalation anesthesia, N2O can cause a substantial increase in CBF (69).Co-administration of barbiturates opioids, benzodiazepines, and propofol has resulted in minimal or no increase in CBF.The presence of pre-existing pneumocephalus or pneumothorax should be considered, which may aggravate with its administration owing to its exchange with nitrogen [70-75].

The BNM was titrated to one or two responses to a train of four4 stimuli. If rocuronium was used for induction, maintenance was performed using the same agent. If succinylcholine is used,

maintenance can be performed with any nondepolarizing NMB [74].

Oxygenation and Ventilation:
Oxygenation and ventilation goals for patients with TBI should be a blood pressure of O2 (PaO2) >60 mmHg and a blood pressure of carbon dioxide (PaCO2) of 35-45 mmHg, unless hyperventilation is required. therapeutic [75-77]. Hypoxemia is associated with increased mortality and poor neurological outcomes in patients with TBI [78].

Regarding the effect of hyperoxia in these patients, it is not clear; some studies report worse outcomes with extreme hyperoxia, while others report better outcomes [79,80]. The mechanism by which hyperoxia may be detrimental later in TBI is unclear but may involve hyperoxic cerebral vasoconstriction and/or production of reactive O2 species. Given the established risk to the brain from hypoxia and the concern for hyperoxic injury, it seems advisable to maintain a PaO2 of 60-200 mmHg, although more data are lacking to make a recommendation in this regard [81].

Hypercapnia should always be avoided in these patients, as elevations in PaCO2 result in increased CBF and ICP. Ventilation should be guided by blood gases rather than end-expiratory CO2 (ETCO2) [82]. Although ETCO2 generally correlates well with PCO2, several factors can lead to significant discrepancies [82].

Therapeutic hyperventilation, is recommended as a temporary measure to reduce elevated ICP and try to avoid it during the first 24 h after injury, when CBF is often critically reduced. If hyperventilation is used, SvyO2 or PtiO2 should be measured sto monitor the oxygen delivery [77].

When TBI and ARDS occur concomitantly, ventilatory management can be very challenging, as ventilatory goals are often in conflict with these two pathologies.Recruitment maneuvers and the use of high PEEP can improve pulmonary gas exchange and respiratory mechanics by reducing ventilation-perfusion mismatch and opening collapsed alveoli to reduce intrapulmonary shunting. However, they may be associated with the development of intracranial hypertension and decreased CPP by affecting venous flow and preventing cerebral venous return to the right atrium, respectively [83].

Literature on the management of patients with concomitant TBI and ARDS is lacking; therefore, a pragmatic approach is needed for this group of patients.Hemodynamic management in patients with TBI requires the judicious use of fluids, blood products, vasopressors, and inotropes. The goal of intraoperative hemodynamic management in patients with TBI is to maintain adequate CBF to avoid secondary injuries. Through autoregulation, the normal cerebral vasculature maintains adequate CBF over a wide range of MAP [12]. However, after TBI, CBF autoregulation may be impaired or abolished, even in those with mild injuries, which can occur immediately after injury and persist for up to two weeks or more [31,84]. Patients with impaired cerebral autoregulation are described as "pressure dependent" such that a sudden increase in MAP can lead to secondary hemorrhage, edema, and increased ICP due to increased cerebral blood volume and hyperemia. Conversely, falls in MAP may be associated with hypoperfusion and ischemia [85].

However, bedside FSC measurements are difficult to obtain. CPP, which is the difference between MAP and ICP (CPP = MAP – ICP), is a proxy measure. Episodes of hypotension (low MAP), elevated ICP, and/or low CPP are associated with secondary brain injury and poor clinical outcomes [85].

The Brain Trauma Foundation, in its latest 2017 guidelines, recommends keeping ICP <22 mmHg and a CPP goal of 60 mmHg to 70 mmHg, and avoiding aggressive attempts to achieve CPP above 70 mmHg. Efforts to optimize CPP must first address intracranial hypertension (ICH) [77]. Patients with more severely impaired autoregulation may be more susceptible to responding to efforts to reduce ICP than to therapy focused on increasing blood pressure [86].

Avoiding hypotension in patients with severe TBI is a primary perioperative goal, as data suggests that hypotension is associated with poor neurological outcomes in these patients. The BTF recommendations suggest maintaining an SBP ≥100 mmHg in patients aged 50-69 years and ≥110 mmHg in patients aged 15-49 years and >70 years [77].

Patients with TBI associated with multiple traumas are at risk of hypotension related to blood loss and other organ damage. A low GCS score on admission, preoperative tachycardia and hypertension, late surgery, subdural hematoma (SHD), multiple lesions on CT scans, and prolonged duration of anesthesia have been associated with an increased risk of intraoperative hypotension [87]. Sudden hypotension may occur in these patients after bone flap removal and dural incision, which may be due to a sudden decrease in ICP [88].

Hypotension during emergency craniotomy for TBI occurs in up to 65% of adult patients [85]. Even a single hypotensive episode (SBP < 90 mmHg) is associated with a worse outcome, and each subsequent hypotensive episode has a cumulative effect on mortality risk [89].

Fluids
Fluid therapy should be directed at maintaining a euvolemic, isotonic, or mildly hypertonic state, and should ideally be goal-directed based on dynamic indicators of fluid responsiveness [90]. Saline may be preferable to albumin; the latter was associated with increased mortality in a post hoc analysis of TBI patients enrolled in the SAFE clinical trial, which compared saline with albumin for fluid replacement in the intensive care unit (ICU) [91].

Balanced crystalloid solutions (lactated Ringer´s solution´ and plasmalite) are used to decrease the risk of acute kidney injury in critically ill patients. Normal saline is preferred in TBI, as balanced solutions are relatively hypotonic, can worsen cerebral edema, and have not shown benefit in this subgroup of patients [92].

Red blood cells
The optimal intraoperative transfusion strategy for patients with TBI remains unclear. In theory, anemia may cause secondary brain injury by reducing O 2 supply to the brain and is associated with poorer outcomes [93]. However, the available literature on the effects of transfusion on outcomes in patients with TBI does not support the use of a liberal (Hb:10 g/dL) versus restrictive (Hb:7 g/dL) transfusion threshold, since the former did not improve outcomes at six months and was associated with a higher incidence of thromboembolic events [94].

During procedures to control life-threatening bleeding or emergency neurosurgery, red cell transfusion is recommended only when Hb falls < 7 g/dL if the patient is hemodynamically stable [95]. If the patient is hemodynamically unstable or has pre-

existing cardiovascular disease, transfusion management should be tailored to individual needs [95].

Vasoactive drugs

Vasoactive drugs (VADs) are commonly administered during anesthesia to achieve BP goals. The effects of these drugs on brain physiology are complex and reflect baseline BP, the state of autoregulatory mechanisms, the mechanism of drug effects, and the magnitude of BP change. Available evidence does not support a preference for a particular vasopressor over others. Selection of vasopressors should be based on patient factors and clinical situation [13, 75]. Phenylephrine, a direct alpha 1 adrenergic agonist, is the first choice in this setting. However, the effects of phenylephrine on CBF remain controversial. Pure alpha agonists are believed to be systemic but not cerebral vasoconstrictors. Therefore, under most circumstances, when BP is increased by phenylephrine, CBF is increased [96]. However, several studies in anesthetized patients and awake volunteers have suggested that, at least in some circumstances, phenylephrine may be associated with decreased cerebral oxygenation. Norepinephrine has a more predictable and consistent effect in increasing CPP than dopamine [99]. Furthermore, norepinephrine increases oxygen levels in brain tissue and significantly reduces the regional oxygen extraction fraction [97-100].

If the patient is hypertensive, it is recommended to avoid vasodilator drugs since they dilate cerebral circulation, which will increase CBF and, therefore, ICP if adequate MAP is maintained. In anesthetized patients, hypnosis and analgesia should be optimized, taking care not to exceed 1 MAC in the case of inhalation anesthesia. If; hypertension persists, drugs that do not influence cerebral autoregulation, such as labetalol or urapidil, are recommended (75).

Beta-blockers reduce or have no effect on CBF and cerebral metabolic rate (CMRO2) and have been proposed as a neuroprotective treatment after TBI, although evidence of long-term benefits is lacking [101].

Coagulation

Approximately one-third of patients with severe TBI develop coagulopathy, which is associated with an increased risk of bleeding progression, poor neurological outcomes, and death [102]. Coagulopathy may be secondary to drugs used chronically by the patient, such as vitamin K antagonists (VKA), new oral anticoagulants (NOACs), antiplatelet agents, or those associated with the trauma itself [19].

In the case of VKA with an INR >1.4, the use of 4-factor prothrombin complex concentrate (PCC) is recommended at an initial fixed dose of 1,500 to 2,000 U at 100 U/min, associated with 10 mg/IV vitamin K. If CCP is not available, fresh frozen plasma (FFP) can be used at a dose of 10-15 mg/kg associated with vitamin K 10 mg/IV [103]. We rechecked PT/INR approximately 30 min after CCP (or plasma) administration and periodically thereafter every four-six hours for the first 24 h and then daily for a few days [104].

For direct factor Xa inhibitor NOAC users, treatment with andexanet alfa (for patients who received a lower dose of factor Xa inhibitor (e.g., rivaroxaban ≤10 mg, apixaban ≤5 mg, or edoxaban ≤ 30 mg) or if 8 h or more have elapsed since the last dose of factor Xa inhibitor, 400 mg bolus at 30 mg/min over 30 min, followed by a 480 mg infusion given at 4 mg/min for a maximum 120 min;for those who received a higher dose of factor Xa inhibitor (e.g., rivaroxaban >10 mg, apixaban >5 mg, edoxaban >30 mg) or an unknown dose in the previous eight hours bolus of 800 mg at 30 mg/min over 30 min, followed by a 960 mg infusion given at 8 mg/min over a maximum of 120 min) or 4-factor PCC at doses similar to those recommended for VKA [105].

The patient uses direct thrombin inhibitors (dabigatran), idarucizumab (5 g/IV divided into 2 doses of 2.5 g), or activated prothrombin complex concentrate (APCC; FEIBA) should be used at a dose of 50 to 80 units/kg (with low level of evidence) [106,107]. For patients who use low molecular weight heparin (LMWH) at therapeutic doses, it is recommended: in the case of enoxaparin administered within the last 8 h, administer 1 mg protamine per 1 mg of enoxaparin administered (maximum 50 mg). If it was administered within 8-12 h, half of the dose was administered. In the case of dalteparin, it is recommended to administer 1 mg of protamine every 100 anti-Xa units of the drug (maximum 50 mg). If bleeding persists and there is a risk to life, protamine (0.5 mg) should be administered for 100 anti-Xa units of dalteparin or for an additional 1 mg of enoxaparin. In both cases, if the last dose received was beyond 3-5 half-lives, reversal was not recommended [108].

Thrombocytopenia and abnormal platelet function are also associated with increased mortality and progression of intracranial hemorrhage [107]. However, the optimal trigger for platelet transfusion has not yet been determined. Recent guidelines recommend the maintenance of a platelet count > 50,000/mm3 in polytrauma patients and further recommend a more stringent limit (> 100,000/mm3) in cases of continued bleeding and/or TBI [108]. For antiplatelet agents, it is suggested to transfuse a platelet apheresis unit only into candidates for surgery, ideally after a platelet function test, and not to administer it if it is normal [109]. In polytraumatized patients with TBI, tranexamic acid (TXA) is recommended only for mild to moderate TBI. In severe TBI, TXA does not appears to provide benefits [110].

Glycemic control

Intraoperative hyperglycemia occurs in 15%-20% of patients with TBI requiring craniotomy, which could increase secondary injury by producing tissue acidosis as a result of anaerobic metabolism and; generation of radicals. free and increased BBB permeability [111]. Although both hyperglycemia and hypoglycemia are associated with worse outcomes in severe TBI, the optimal target for blood glucose is unclear, and tight glycemic control in these patients is controversial. Serial glucose monitoring is recommended during surgery and the maintenance of blood glucose levels between 80 and 180 mg/dL. Strict glycemic control (80 – 110 mg/dL) is not recommended as it has been associated with increased mortality [112].

Temperature control

Fever is common in patients with severe TBI and is associated with poor outcomes [113].

The use of induced hypothermia to control ICP is not recommended; because it does not improve functional results and has been associated with immunosuppression and a higher rate of pneumonia [114,115].

Eizure Prophylaxis

In patients with severe TBI, the rate of clinical post-traumatic seizures can be as high as 12%, whereas that of subclinical seizures detected on EEG can be as high as 20-25% [116]. Post-TBI seizures are classified as early (< 7 days) or late (> 7 days).

The latest BTF guidelines recommend antiepileptic therapy to decrease the incidence of early seizures when the overall benefit outweighs the complications associated with such treatment and do not recommend the prophylactic use of antiepileptic drugs to prevent late-onset seizures [77]. There is insufficient evidence to recommend the use of levetiracetam over phenytoin to prevent seizures [77].

Osmotic therapy

The most commonly used drugs are mannitol and hypertonic saline (HSS). Osmotic agents reduce brain tissue volume by drawing free water from the brain tissue into the systemic circulation, where it is excreted by the kidneys [117].

Mannitol works by increasing serum osmolality, resulting in an osmotic gradient from the interstitial to the intravascular space, reducing cerebral edema, and consequently, ICP. Its strong osmotic force was due to its high reflection coefficient ($\sigma = 0.9$). Mannitol also acts through other mechanisms such as induction of reflex cerebral arteriolar vasoconstriction, improvement of blood rheology, reduction of CSF formation, and free radical scavenging [118].

The recommended dose to reduce ICP of mannitol 20% is 0.25 to 1 g/kg every 6 h, although doses < 0.5 g/kg are generally considered less effective [119]. Serum osmolality should be maintained at 310-320 mOsm/l. Mannitol is completely excreted in the urine, and there is a risk of acute tubular necrosis if serum osmolality exceeds the recommended levels. Other adverse effects of mannitol include hypotension, electrolyte disturbances (hyperkalemia, hypokalemia, hypomagnesemia, and hypophosphatemia), and rebound cerebral edema after prolonged use. Mannitol is contraindicated in patients with renal failure due to the risk of osmotic nephrosis [120]. possible pulmonary edema, and heart failure. SSH has been used as an alternative to mannitol. Compared to mannitol, it has a higher reflection coefficient (1.0 vs. 0.9, respectively). Therefore, SHH is less able to cross the BBB and may have stronger osmotic action. Thus, it reduces ICP by decreasing cerebral edema while simultaneously increasing CPP by improving the MAP. Other mechanisms of action include the induction of reflex cerebral arteriolar vasoconstriction, improvement of blood rheology, and an anti-inflammatory effect due to reduced adhesion of polymononuclear cells to the cerebral microvasculature [121]. In the literature, the SHH concentrations used to treat intracranial hypertension (ETH) ranged from 3% to 23.4%. Bolus doses are generally administered in response to a measured ICP and can be repeated as needed until the ICP is in an acceptable range or serum sodium concentrations have risen above normal (>145–155 mEq/L) [122].

The possible adverse effects of SHH include rebound cerebral edema, electrolyte abnormalities (hypokalemia), congestive heart failure, renal failure, hyperchloremic metabolic acidosis, phlebitis, transient hypotension, hemolysis, pontine myelinolysis, subarachnoid hemorrhage, seizures, and muscle spasms [123].

Although the BTF does not recommend the use of one osmolar therapy over another [77], a Cochrane review concluded that mannitol for the management of HT may have a beneficial effect on mortality compared with pentobarbital treatment but may have a detrimental effect on mortality compared to SHH [124]. A more recent meta-analysis, including seven RCTs and 191 patients, also highlighted the superiority of SHH over mannitol in the treatment of elevated ICP. With respect to the 6-month mortality, no differences were observed, and limited adverse reactions have been reported [125].

Other Therapies

Glucocorticoids: The use of glucocorticoid therapy after head injury was found to be detrimental in a large trial of patients with moderate-to-severe TBI [126]. In patients treated with methylprednisolone, higher mortality rates were observed at 2 and 6 months [127].

Progesterone: No benefit was found for the administration of intravenous (IV) progesterone in two randomized trials of acute severe TBI [128,129].

Citicoline: Citicoline was not found to be effective in improving outcomes in a randomized trial of 1213 TBI patients [130].

Erythropoietin (EPO): Erythropoietin has been postulated to have neuroprotective effects, and although no such benefit has been demonstrated in TBI, the mortality in the EPO group was significantly lower [131].

HTE management

The goal of medical treatment of severe traumatic brain injury is to ensure that nutrient delivery to the brain is optimized during the period of abnormal physiology and brain inflammation followings injury [132]. The incidence of HTE in patients with severe TBI varies according to the definition, from 91% when considering any episode of ICP > 20 mmHg to 77% when considering ICP > 20 mmHg for more than 5 min [133,134].

In patients with TBI, an ICP of ≥ 20 mmHg triples mortality and the risk of poor neurological prognosis, but if it is greater than 40 mmHg, mortality is mortality is multiplie mortality by seven [135]. In this regard, the latest BTF guidelines recommend treatment guided by neuromonitoring (ICP, CPP measurement, and advanced cerebral monitoring) to reduce in-hospital and 2-week mortality [77].

The BTF first and second edition guidelines include management algorithms; however, because TBI treatments have been studied separately, almost no evidence exists regarding their relative efficacy, sequential order or combination, as would be required to create evidence-based treatment protocols [136,137].

Given the lack of algorithms that would simplify the treatment of ETH in TBI, a group of experts met in 2019 to develop a management algorithm with the condition that the patient is admitted to the ICU with an ICP sensor [138].

This includes 3: levels plus a "zero level" that considers the basic management provided for these patients. They have also added a table with non-recommended treatments.

Zero Level (Does not Require PIC captor):
Expected Interventions:
• ICU admission.
. Endotracheal intubation and+ mechanical ventilation.
• Serial evaluation of neurological status and+ pupillary reactivity.
• Elevation of the head at 30º-45º
. Analgesia for managing pain signs
• Sedation to prevent agitation and asynchrony with a ventilator.
• Temperature management to prevent fever.
o Measure core temperature.

o Treat temperature ≥ 38º.
Recommended interventions:

1. Installation of central venous catheter (CVC).
2. Measurement of expired CO2.
3. Consider antiepileptics (for one week only).
4. Maintain CPP ≥ 60 mmHg.
5. Maintain Hb ≥ 7 g/dL.
6. Avoid hypothermia.
7. Optimize venous return (Keep head in midline, make sure cervical collar is not too tight).
8. Continuous blood pressure monitoring.
9. Maintain SpO2 ≥ 94%.

Level 1 (Requires PIC Catcher):
• Maintain CPP between 60-70 mmHg.
• Improve analgesia to decrease ICP.
• Increase in sedation to decrease ICP.
• Maintain PaCO2 in the lower limit of the normal range (35-38 mmHg).
• Intermittent mannitol boluses (0.25 g/kg – 1 g/kg)*.
SHH-intermittent boluses*
• CSF drainage if EVD is an EVD in situ.
• Consider installing DVE.
• Consider antiepileptic prophylaxis (only one week).
• EEG monitoring was considered.

*Plasma sodium 155 mEq/L and plasma osmolality 320 mOsm are recommended as the upper limits for the administration of SHH and mannitol, respectively.

Therefore, recommend re-examininge the patient and considering a new CT scan, reconsidering surgery for potential surgical injuries, considering other causes of increased ICP, checking that physiological and laboratory parameters are in the normal range (CPP, arterial blood gas), and requesting help from a neurointensivist.

Level 2:
- Induces mild hypocapnia.
- Start neuromuscular block, if effective**
- The "MAP challenge" (139) is recommended for assessing brain autoregulation and guidinge the objectives of CPP and MAP individually.
- Increased CPP with crystalloid boluses, vasopressors, and/or inotropes to decrease ICP when autoregulation is intact.

** Continuous infusion is recommended only if a single dose is shown to be effective.
Level 3:
- Eats with barbiturates (Thiopental or Pentobarbital) ***
- Decompressive Craniectomy.
- Mild hypothermia (35º-36º).

***Continuous infusion is recommended only if proven effective.
Treatments not recommended:
- Continuous infusion of mannitol without a bolus.
- Programmed infusion of hyperosmolar solution (eg every 4-6 fixed hours).
- Lumbar CSF drainage.
- Furosemide.
- Routine Steroide Use.
- Routine use of Therapeutic Hypothermia at Temperatures below 35º.
- Propofol in High Doses to try "Burst Suppression"
- Routine decrease in PaCO2 below 30 mmHg.
- Routinely raise CPP above 90 mm Hg.

Perioperative goals for physiologic and laboratory variables are summarized in Table 4 [77,139].

Table 4: Perioperative Goals of TBI Anesthetic Management

Parameter	Recommended Value
$PaO2/SpO_2$	60 mmHg/>90%
$PaCO_2$	35-40 mmHg
ICP	< 22 mmHg
SBP	≥ 110 mmHg
CPP	60-70 mmHg
$PbtO_2$	≥ 15 mmHg
$SvyO_2$	55-75%
Temperature	36,0 – 38,3
Blood glucose	140 – 180 mg/dL
Platelets	≥ 100.000/mm3
Hemoglobin	≥ 7 g/dL
INR	< 1,5

TBI and Extracranial Surgery

Management of the acute phase after severe TBI with polytrauma is challenging for each member of the trauma team [140].

Polytrauma (significant injuries of three or more Abbreviated Injury Scale (AIS) points in two or more regions along with one or more additional variables of these five physiologic parameters: SBP ≤90 mmHg, GCS ≤8, base excess (BE) ≤−6.0, international normalized ratio (INR) ≥1.4/activated partial thromboplastin time (aPTP) ≥40 s and age ≥70 years) is a complication in up to 70% of traumatic brain injury (TBI)cases, which is one of the leading causes of mortality and disability worldwide [141,142]. Hemorrhage is the most common cause of premature death and traumatic brain injury is the most common cause of late mortality and disability [143].

Uncontrolled bleeding in patients with polytrauma caused by TBI may often require simultaneous multisystem surgery (CMS). The main objective of this study was to control bleeding and to avoid or minimize secondary brain injuries [144].

Therefore, it differs from the treatment of multiple trauma patients without TBI in some respects, but overlaps with others. The most notable distinction relates to hemodynamic management; in polytrauma patients with moderate-to-severe TBI, "permissive arterial hypotension" (SBP >90 mmHg) should be aggressively avoided, while this strategy is highly recommended in polytrauma patients without TBI [145].

In its 2019 consensus guidelines, the World Society for Emergency Surgery recommends maintaining SBP > 100 mmHg or MAP > 80 mmHg during extracranial surgery as well as during emergency neurosurgery [92].

A positive fluid balance was recently shown to be associated with worse outcomes in patients with TBI, suggesting that normovolemia should be the goal in this setting [146]. In this regard, advanced hemodynamic monitoring to assess cardiac output or fluid response (i.e., stroke volume variation, pulse pressure variation, etc.) could be useful in the perioperative period [147].

Variables that appear to be managed similarly in all trauma patients include aggressive prevention of hypoxia Transfusion thresholds based on Hb and coagulation, and transfusion ratios [94]. It should be noted that a higher than usual platelet count is recommended for emergency neurosurgery [94]. The use of TXA is recommended only in selected polytrauma settings when associated with TBI [140].

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