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Review

# Anesthetic management of the traumatic brain injury patients undergoing non-neurosurgery

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This article describes the anesthetic management of patients with traumatic brain injury (TBI) undergoing non-neurosurgery, primarily targeting intraoperative management for multiple-trauma surgery. The aim of this review is to promote the best clinical practice for patients with TBI in order to prevent secondary brain injury. Based on the current clinical guidelines and evidence, anesthetic selection and administration; maintenance of optimal cerebral perfusion pressure, oxygenation and ventilation; coagulation monitoring; glucose control; and temperature management are addressed. Neurological recovery, which is critical for improving the patient's quality of life, is most important; therefore, future research needs to be focused on this aspect.

**Keywords:** Anesthesia; Blood coagulation; Cerebral perfusion pressure; General surgery; Intracranial pressure; Temperature; Traumatic brain injury.

## INTRODUCTION

Traumatic brain injury (TBI) is defined as an alteration in brain function or any other evidence of brain pathology as a result of an external force. Alteration in brain function here implies loss of consciousness or a decreased level of consciousness, loss of memory, neurologic deficits, or alteration in mental state at the time of the injury [1]. TBI has traditionally been classified using clinical severity scores, and the Glasgow Coma Scale [2] is a universally-accepted tool for TBI classification; however, severity will not be described separately in this article. Only the management of anesthesia in patients with TBI who undergo surgery other than neurosurgery is discussed here. The most common situation is when multiple-trauma patients require surgical treatment for body parts other than the head; therefore, the main focus of this article is the intraoperative management for trauma surgery. Considerations may differ depending on whether the non-neurosurgery is performed before or after primary treatment for brain injury. In addition, neurosurgical consultation may be necessary for intracranial pressure (ICP) monitoring and management.

When a patient with TBI needs to undergo non-neurosurgery, proper attention should be given to the prevention of secondary brain injury while performing the surgery and administering anesthesia. Understanding the systemic physiological changes caused by trauma and preparing for the resulting cerebral hemodynamic changes are the main starting points for safe anesthesia management, with the ultimate goal being an improved outcome wherein the patient's brain function is restored and preserved. Clinical choices and decisions based on current clinical guidelines and evidence will be reviewed in this article.

## PRIMARY AND SECONDARY BRAIN INJURY

Primary brain injury refers to damage that occurs directly

at the time of the initial trauma and includes the mechanical impact to the head and resulting epidural/subdural/intracranial hematomas, skull fractures, and diffuse axonal injuries [3]. Secondary brain injury is a consequence of these physiological insults and develops over time after the onset of the initial injury, causing further damage to the cerebral physiology and worsening outcomes in TBI patients [4]. The major initiating factors of secondary injury are hypotension and hypoxemia; other factors include hyperthermia, hypoglycemia/ hyperglycemia, and hyponatremia/hypernatremia [4,5]. Prevention of secondary brain injury should be included among the goals of anesthesia for TBI patients undergoing non-neurosurgery [4].

## GUIDELINES

The Brain Trauma Foundation has published evidence-based clinical guidelines describing the management of severe TBI (Guidelines for the Management of Severe Traumatic Brain Injury, 4th edition, 2016) [6]. The contents related to anesthesia management are summarized in Table 1. Details regarding the topics listed in the table are described, as and when required, in the following sections.

## INTRAOPERATIVE MANAGEMENT

### Systemic blood pressure and ICP management: threshold

In TBI, the increased intracranial volume caused by bleeding or tissue edema is initially compensated for by a decrease in cerebrospinal fluid (CSF) volume. However, there is a limit to this compensation, with further increases in space-occupying lesions increasing the ICP excessively, which restricts blood flow to the skull, resulting in tissue ischemia [7]. Elevated ICP is also transmitted to the brain parenchyma and can lead to uncal herniation [7].

Cerebral perfusion pressure (CPP) = Mean arterial pressure (MAP) – ICP

In order to maintain adequate CPP, monitoring and management of both systemic blood pressure and ICP is required. Both a decrease in MAP or an elevation in ICP will deleteriously alter the effective perfusion pressure.

Management of TBI includes maintaining adequate cerebral blood flow and oxygenation to avoid secondary brain injury. Previously, this therapy was directed at managing ICP, but there has recently been a shift towards strategies aimed at maintaining adequate CPP. Maintaining systolic blood pressure at ≥ 100 mm Hg for patients 50–69 years old and at ≥ 110 mm Hg or above for patients 15–49 or over 70 years old may be considered to decrease mortality and improve outcomes (level III) [6]. ICP monitoring remains a level IIB recommendation in the latest Brain Trauma Foundation guidelines [6]. Using information obtained by ICP monitoring for the management of patients with severe TBI is recommended to reduce in-hospital and 2-week post-injury mortality [8].

According to The Brain Trauma Foundation, ICP > 22 mmHg is associated with increased mortality (level IIB). There are no Class I study data that indicate an optimal CPP threshold or any specific optimal CPP in patients with TBI. It has been found that maintaining the CPP below 50 mmHg produces signs of ischemia and raising it above 60 mmHg avoids cerebral oxygen desaturation in the injured brain [9,10]; that is, it is suggested that the critical threshold for CPP lies between 50 and 60 mmHg and that CPP below 50 mmHg should be avoided. One study that proposed a critical CPP threshold of 60 mmHg suggested that the treatment goal should be to maintain the CPP at 70 mmHg to remain above the threshold [11]. Current guidelines recommend maintaining CPP at 60–70 mmHg and acknowledge that optimal CPP may vary depending on cerebral blood flow autoregulation [6].

### How to estimate ICP

Both invasive and non-invasive methods can be used to evaluate ICP [12,13]. Invasive methods generally involve the use of either external ventricular drains (EVDs) or intraparenchymal monitors. The EVD method serves the dual purposes of CSF diversion and continuous ICP measurement and is considered the gold standard [12]. Non-invasive methods for monitoring ICP include pupillometry, optic nerve sheath diameter measurement, and transcranial Doppler; however, these have various limitations with regard to intraoperative use. Although ICP monitoring in patients with TBI is a level IIB recommendation according to the Brain Trauma Foundation guidelines [6], ICP monitoring during extracranial surgery requires the patient to be equipped with a device, such as an EVD, that can be connected to pressure monitors used in the operating room. If there is no attached device, cooperation with a neurosurgeon is required, which

**Table 1.** Summary of the Recommendations Related to Anesthetic Management from the Brain Trauma Foundation Guidelines for the Management of Severe TBI, 4th Edition

| | | |
|---|---|---|
| Prophylactic hypothermia | • Early (within 2.5 h) or short-term (48 h post-injury) prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse injury. | Level IIB |
| Hyperosmolar therapy | • Insufficient evidence. | |
| CSF drainage | • An EVD system centered at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent drainage. | Level III |
| | • Use of CSF drainage to lower ICP in patients with an initial Glasgow Coma Scale score < 6 during the first 12 h after injury may be considered. | |
| Ventilation therapies | • Prolonged prophylactic hyperventilation with $PaCO_2$ of 25 mmHg or less is not recommended. | Level IIB |
| Anesthetics, analgesics, and sedatives | • Administration of barbiturates to induce burst suppression measured by EEG as prophylaxis against the development of intracranial hypertension is not recommended. | Level IIB |
| | • High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy. | |
| | • Although propofol is recommended for the control of ICP, it is not recommended for improvement in mortality or 6-month outcomes. Caution is required, as high-dose propofol administration can result in significant morbidity. | |
| Steroids | • The use of steroids is not recommended for improving outcomes or reducing ICP. In patients with severe TBI, high-dose methylprednisolone is associated with increased mortality and is contraindicated. | Level I |
| Tracheostomy | • Early tracheostomy is recommended to reduce mechanical ventilation days when the overall benefit is assessed to outweigh the risk of complications associated with such a procedure. However, there is no evidence that early tracheostomy reduces mortality or the rate of nosocomial pneumonia. | Level IIA |
| ICP monitoring | • Management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality. | Level IIB |
| CPP monitoring | • Management of severe TBI patients using guidelines-based recommendations for CPP monitoring is recommended to reduce 2-week mortality. | Level IIB |
| Advanced cerebral Monitoring | • Jugular bulb monitoring of arteriovenous oxygen content difference may be considered to reduce mortality and improve outcomes at 3 and 6 months post-injury. | Level III |
| Blood pressure thresholds | • Maintaining systolic blood pressure at ≥ 100 mmHg for patients 50 to 69 years old and at ≥ 110 mmHg for patients 15 to 49 or over 70 years old may be considered to reduce mortality and improve outcomes. | Level III |
| ICP thresholds | • Treatment for ICP above 22 mmHg is recommended, because values above this level are associated with increased mortality. | Level II B |
| CPP thresholds | • The recommended target CPP value for survival and favorable outcomes is between 60 and 70 mmHg. Whether the minimum optimal CPP threshold is 60 or 70 mmHg is unclear and may depend upon the patient's autoregulatory status. | Level II B |
| | • Avoiding aggressive attempts to maintain CPP above 70 mmHg by using fluids and vasopressors may be considered because of the risk of adult respiratory failure. | Level III |
| Advanced cerebral monitoring thresholds | • Jugular venous saturation < 50% may be a threshold to avoid in order to reduce mortality and improve outcomes. | Level III |

CSF: cerebrospinal fluid, ICP: intracranial pressure, CPP: cerebral perfusion pressure, EVD: external ventricular drain, EEG: electroencephalography, TBI: traumatic brain injury.

will depend on the clinical environment at each institution.

## Methods to maintain CPP: reducing ICP

The fastest way to decrease ICP is to allow CSF drainage, if possible. Another simple alternative to promote cerebral venous drainage is head-of-bed elevation to 30° (the reverse Trendelenburg position) while maintaining the patient's neck in a neutral position. Hyperosmolar medications can lower ICP by allowing osmotic mobilization of water across the intact blood-brain barrier (BBB) into the intravascular space, thus reducing cerebral water content; commonly used agents include mannitol and hypertonic saline. If the patient is stable, 0.25–1 gm/kg of mannitol can be carefully administered slowly over 15 min to avoid intravascular volume depletion and hypotension due to urinary losses. Currently, there is not sufficient data to reach a definitive conclusion—although hypertonic saline showed a trend towards lower mortality and a more beneficial effect on CPP compared to mannitol, there were no statistically significant

differences in mortality and neurological outcomes between hypertonic saline and mannitol administration [14].

Although the ideal serum sodium concentration is not well established, close monitoring of blood sodium levels is imperative to prevent hypernatremia, as rapid changes in serum sodium levels may be a causative factor for central pontine myelinolysis. A serum sodium concentration greater than 155 mmol/L has been suggested to be an independent predictor of acute kidney injury [15]. It should also be considered that the effects of administering a hyperosmolar agent will be altered in case of BBB rupture, which is common in patients with TBI [7].

## Methods to maintain CPP: increasing systemic blood pressure

Although decreasing ICP is the first-line therapy, increasing MAP to maintain CPP should also be considered. Patients with TBI who need to undergo non-neurosurgery may be in an acute state, in which systemic hemodynamics are unstable due to trauma, or a subacute state, in which patients are recovering from the trauma. Accordingly, blood pressure must be appropriately managed to maintain CPP even before the induction of anesthesia, and special attention is required to avoid exacerbating hypotension due to anesthetics or bleeding during surgery. If there is a modifiable cause of hypotension, its management should be prioritized, and vasopressor administration must always be considered. Selecting which vasopressor to use is less straightforward. A retrospective analysis showed that phenylephrine was the preferred vasopressor, with a greater increase in MAP and CPP after the start of infusion compared to norepinephrine or dopamine [16]. At equal MAP, using ephedrine resulted in better brain microcirculation and oxygen delivery than with the use of phenylephrine [17]. However, clear clinical evidence regarding the different effects of vasopressors in conditions with BBB disruption is still lacking and needs further investigation [18]. Given the ample evidence that autoregulation of cerebral blood flow in response to changes in CPP is impaired in both severe and mild TBI [19-21], careful attention to blood pressure management is necessary, because low blood pressure can be directly linked to reduced cerebral blood flow.

## Airway management

The incidence of cervical spine injury in trauma patients is reported to range from 3.5 to 6.2% [22,23]. Notably, cervical spine injury must always be suspected in patients with TBI, and caution should be exercised during endotracheal intubation and while repositioning patients. Video-guided laryngoscope with cervical immobilization was reported to reduce the upper cervical spine motion than Macintosh laryngoscope [24,25] and facilitated a more rapid tracheal intubation compared with the flexible bronchoscope [26]. Awake tracheal intubation using a flexible bronchoscope can minimize cervical spine movement [27], but requires sufficient procedural experience and may be difficult to perform depending on the level of consciousness of patients with brain trauma. In anesthetized patients without cervical immobilization, tracheal intubation using a video-guided laryngoscope resulted in greater cervical spine movement than a flexible bronchoscope, and jaw thrust during flexible bronchoscopy also causes movement of the cervical spine [28]. Therefore, an awake bronchoscopic approach is expected to be more effective for experienced clinicians when the situation, including the patient's condition, allows, whereas using a flexible bronchoscope, lightwand, or video-guided laryngoscope with cervical immobilization may be a better option for inexperienced clinicians. Nasotracheal intubation is better to be avoided in case of patients with skull base fractures; in such cases, there is a risk that the tube may pass through the cribriform plate and enter the frontal brain region as it advances [29].

Tracheostomy is commonly performed on patients with TBI in the intensive care unit—in approximately 32% of cases—and is most frequently performed after the first week in the intensive care unit [30]. A recent systematic meta-analysis reported that early tracheostomy in patients with severe TBI contributes to reducing mechanical ventilation duration, intensive care unit and hospital stay duration, as well as the incidence of ventilator-associated pneumonia [31]. The Brain Trauma Foundation guidelines also recommend early tracheostomy when the overall benefits appear to outweigh the risk of complications [6].

## Choice of induction agents for anesthesia

Intravenous anesthetics, which are mainly used for the induction of general anesthesia, have various effects on cerebral blood flow and the cerebral metabolic rate of oxygen ($CMRO_2$). Propofol, thiopental, midazolam, and etomidate reduce $CMRO_2$, resulting in decreased cerebral blood flow and ICP. However, thiopental and propofol can cause hypo-

tension and reduce CPP; therefore, caution must be exercised with regard to dose titration. Etomidate has a limited effect on MAP and is advantageous for managing CPP. Unfortunately, a single dose of etomidate is sufficient to cause adrenal insufficiency, and there are concerns that its use may be associated with mortality in patients with sepsis [32]. Even though a recent meta-analysis supports its use [33], the evidence is insufficient to guarantee the safety of etomidate use under septic conditions [34]. Ketamine has historically not been indicated for patients with brain trauma, based on the belief that it has detrimental effects on ICP. However, recent clinical data support the neuroprotective effects of ketamine via reducing glutamate levels and inhibiting cortical spreading depolarization [35,36]. A systematic review concluded that there was no evidence of harm due to ketamine use in patients with acute brain injury [37]. When inducing anesthesia in a patient with TBI, etomidate can be considered preferentially in patients with unstable hemodynamics, whereas ketamine can be an alternative option if septic shock is the cause of unstable hemodynamics.

## Anesthetics for maintenance

There is no evidence to indicate that TBI outcomes can be improved based on the type of anesthetic agent used. Maintenance of anesthesia may be accomplished using inhaled or intravenous anesthetics, with careful consideration of the hemodynamic management goals. Total intravenous anesthesia using propofol and opioids is advantageous for cerebral hemodynamic management and neurophysiological monitoring and is frequently used in neurosurgery. As a highly selective alpha-2-adrenergic agonist, dexmedetomidine, which is expected to reduce ICP by reducing cerebral blood flow, has been reported to reduce anesthetic and opioid requirement and postoperative nausea and vomiting when used as an adjunct to general anesthetics [38], but evidence regarding its optimal dose as an adjuvant and its comparative effects versus other anesthetics is lacking [39]. Low concentrations of isoflurane and sevoflurane suppress brain metabolism and constrict cerebral blood vessels [40]. At higher concentrations, the direct vasodilatory effects of volatile anesthetics dominate, increasing both cerebral blood flow and ICP. Nitrous oxide is another cerebral vasodilator that increases cerebral blood flow and ICP; therefore, it should ideally be avoided [41]. When a volatile anesthetic is selected for general anesthesia, a minimum alveolar concentration of < 1 seems to be appropriate for patients with TBI [40].

## Oxygenation and ventilation

Hypoxemia (generally defined as $PaO_2$ < 60 mmHg) is a major factor in the development of secondary brain injury and should be avoided [4,42]. Despite the evidence being insufficient to conclude whether it affects clinical outcomes, hyperoxia after TBI has been suggested to be associated with higher mortality [43]. Therefore, a balanced approach that avoids the higher and lower extremes is suggested for oxygenation management.

$PaCO_2$ is a potent mediator of cerebrovascular reactivity. Hypercapnia causes cerebral vasodilation via CSF acidosis; therefore, even a small increase in $PaCO_2$ may have deleterious effects on ICP in TBI patients with low intracranial compliance [44,45]. Hypocapnia causes cerebral vasoconstriction via an increase in CSF pH, and sustained reduction in $PaCO_2$ can lead to cerebral ischemia [44]. The Brain Trauma Foundation recommends avoiding hypocapnia [6] and hyperventilation in patients at risk of herniation, for short-term control of ICP, should be cautious. According to a recent meta-analysis, approximately 20% of TBI patients have acute respiratory distress syndrome (ARDS), which leads to worse neurological outcomes and higher mortality [46]; this indicates the need to use lung-protective ventilation during general anesthesia, especially in TBI patients with multiple-trauma, although concerns regarding resulting hypercapnia remain. Given the risks and benefits of lung-protective ventilation, the consensus when applying mechanical ventilation to patients with brain injury strongly recommends lung-protective ventilation in the presence of ARDS and no elevated ICP, and weakly recommends lung-protective ventilation in the absence of ARDS or elevated ICP [47]. Positive end-expiratory pressure (PEEP) can promote improved oxygenation and reduced intrapulmonary shunting; however, increased PEEP can lead to hypotension and alveolar overdistension with increased dead space, resulting in a higher $PaCO_2$ [48]. Furthermore, the increased intrathoracic pressure following elevated PEEP may reduce the pressure gradient of cerebral venous outflow and lead to an increase in ICP [49]. Therefore, as a practical strategy for safe PEEP titration in patients with ARDS and elevated ICP, it was suggested that the level of PEEP should be adjusted by monitoring the response of blood pressure and ICP such that increasing the PEEP does not increase ICP [50].

## Coagulation monitoring

At the time of admission to the emergency department, coagulopathy is present in up to 25–35% of trauma patients [51]. During the initial hours of trauma-induced coagulopathy development, hypocoagulability is typically present, resulting in bleeding; later, coagulopathy is characterized by a hypercoagulable state associated with venous thromboembolism and multiple organ failure [52]. Coagulopathy is an integral component of a vicious cycle when combined with acidosis and hypothermia [53].

Ongoing intracranial bleeding can lead to increased ICP, brain herniation, and death. Tranexamic acid reduces bleeding by inhibiting the enzymatic breakdown of fibrin blood clots [54]. The role of tranexamic acid in the management of TBI remains unclear—a recent randomized controlled study showed that tranexamic acid administration within 3 h of injury reduced all-cause 24-h mortality in cases of mild to moderate injury, but had no effect on 28-day all-cause mortality [55], while a recent meta-analysis showing that tranexamic acid has no effect on mortality or neurological recovery, although its use probably does not increase the risk of adverse events [56].

Current approaches to trauma resuscitation focus on controlling bleeding and managing trauma-induced coagulopathy through the timely administration of hemostatic therapy [57]. Conventional coagulation tests include platelet counting and fibrinogen level and PT and aPTT measurement; however, they are relatively slow and do not provide a measure of platelet function or fibrinolysis [58,59]. Due to these shortcomings, anesthesiologists and trauma critical-care surgeons often prefer viscoelastic hemostatic assay-guided blood component therapy for evaluating bleeding patients in whom coagulopathy is common [58]. The two most commonly used viscoelastic hemostatic assays are thromboelastography and rotational thromboelastometry. Viscoelastic hemostatic assays provide clinicians with real-time data and a complete view of the coagulation process, from clot initiation and formation to clot stability and fibrinolysis measurements [57]. Although recent studies and reviews have described no differences in clinical outcomes between resuscitation guided by viscoelastic hemostatic assays and those guided by conventional coagulation tests [59,60], clinicians can use both methods to monitor hemostasis across heterogeneous groups of patients.

## Glycemic control

Trauma triggers an increase in stress hormone and cytokine levels, resulting in enhanced glucose production, reduced insulin production, and insulin resistance in peripheral tissues [61]. In the context of acute illness or injury, this has been termed stress-induced hyperglycemia and is defined as a transient plasma glucose level > 200 mg/dl in patients who are normally euglycemic [62]. There is no consensus on the best approach for glycemic control in patients with TBI. Studies on the association between stress-induced hyperglycemia and patient outcomes have consistently reported higher morbidity and mortality rates [63,64]. However, it remains unclear whether stress-induced hyperglycemia has a direct causative effect on worsening outcomes or is simply a marker of more severe disease [62]. The prospective, multicenter Normoglycemia in Intensive Care Evaluation-Survival Using Glucose Algorithm Regulation study reported a higher 90-day mortality with strict glucose control than with standard care, without any effect on organ failure or length of stay, suggesting that the target of glycemic control can be abandoned [65]. In a study that evaluated outcomes before and after strict glucose control therapy with a target glucose level of < 140 mg/dl in critically ill trauma patients, mortality, length of hospital stay and intensive care unit stay, and number of ventilator days did not differ between comparison periods [66]. These trial results do not allow for conclusive conclusions to be drawn regarding the exact glucose level that should be maintained. Tight glycemic control to maintain blood glucose levels below 110 mg/dl is likely not required and may even be detrimental to patient outcomes [62], including causing hypoglycemia [67]. A moderate level of glycemic control aimed at stabilizing glucose levels while reducing hyperglycemic and hypoglycemic events appears to be safe [68].

## Temperature management

A systematic review published in 2003 showed that clinically induced hypothermia could reduce the risks of mortality and poor neurological outcomes in adults with TBI [69]. It has been hypothesized that hypothermia has a neuroprotective effect in TBI by disrupting post-injury biochemical and inflammatory cascades [70]. However, a multicenter randomized trial demonstrated that early prophylactic hypothermia did not improve neurological outcomes at 6 months compared to normothermia in patients with severe

TBI [71]. Similarly, the results of another recent meta-analysis do not support the use of early prophylactic hypothermia (within 6 h after injury) as a neuroprotective strategy in adult patients with TBI, although they indicate that hypothermia could effectively reduce refractory high ICP [72]. Meanwhile, the incidence of central or neurogenic fever is considerably high in patients with severe TBI, especially in those with diffuse axonal or frontal lobe injury [73]; fever due to infection is also common in patients with severe trauma [74]. The detrimental effects of fever on the neurological recovery of patients with acute brain injury have been increasingly recognized [75-77]. Evidence supports the maintenance of normothermia in patients with TBI [78], and the Brain Trauma Foundation guidelines do not recommend prophylactic hypothermia [6]. Additionally, core body temperature is not a reliable indicator of brain temperature [79], and direct measurement remains the best way to monitor brain temperature in patients with brain injury, although brain temperature monitoring is currently not routinely applicable.

## TIMING OF SURGERY

There is no evidence to specify the exact and safe time following TBI for elective surgery. Some studies recommend elective non-neurosurgery 6 months after recovery from TBI [80], based on the fact that patient symptoms generally do not improve or worsen 6 months after severe TBI [81]. However, in a meta-analysis comparing the effects of early and late fracture fixation on the prognosis of patients with limb fractures and concomitant TBI, late fixation (performed > 14 days after trauma) was associated with nonunion or malunion, and early fixation (within 24 h) did not affect the incidence of mortality, pneumonia, ARDS, or neurologic adverse events [82]. A retrospective cohort study reported that early orthopedic and facial fracture fixation (≤ 24 h after injury) under general anesthesia was not associated with worse neuropsychological or functional outcome than late surgery in multisystem trauma patients with TBI [83]. Currently, there are no contraindications to anesthesia in TBI, and once the decision to proceed with surgery has been made, steps should be taken to reduce the risks associated with surgery based on an understanding of the pathophysiology of TBI and the interactions between surgery and application of anesthesia [84].

## FUTURE RESEARCH AND CHALLENGES

Due to the clinical characteristics of trauma patients, it is difficult to conduct randomized controlled studies on trauma cases. In TBI, the most important aspect is to recover the patient's cognitive function. To improve this outcome, it is necessary to pursue the best medical practices by accumulating and analyzing a large amount of clinical evidence. Further research is expected to focus on the recovery of patient neurological function and improving their quality of life.

## CONCLUSION

The overall strategy for anesthesia management in non-neurosurgery for patients with TBI is similar to that in neurosurgery for TBI. However, especially when ICP is elevated following trauma and decompressive craniectomy is not performed, there is a risk of secondary brain injury during non-neurosurgery, because the brain still has impaired autoregulation. Thus, related indicators, including blood pressure, oxygen saturation, ICP, temperature, and coagulation status, should be properly monitored and controlled to prevent secondary brain injury while administering anesthesia for non-neurosurgery.

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## CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

## DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article, as no datasets were generated or analyzed during the current study.

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