Review Article

Anesthetic considerations for oral maxillofacial surgery in neurologically injured patients: a comprehensive narrative review

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Background and Objective: The co-occurrence of oral-maxillofacial and brain injuries is common and present unique perioperative challenges. This narrative review aims to provide a comprehensive summary of the considerations and best practices related to balancing cerebral oxygen supply with demand, regulating ventilation, upholding hemodynamic stability, sustaining optimal fluid and electrolyte balance, and employing strategies for optimal patient outcomes.

Methods: A literature review using the PubMed and Google Scholar databases was employed to gather data from controlled trials, cohort studies, and meta-analyses on neurological injury and oral and maxillofacial surgery (OMFS). Relevant keywords included traumatic brain injury, facial trauma, airway management, pain management, prophylactic anticoagulation, and intracranial pressure. Titles and abstracts were screened for their relevance to the research question and included articles were assessed in full. Articles written in English were prioritized, and the data was categorized into pertinent themes. A narrative synthesis was conducted for each category to ensure a balanced and comprehensive overview of the current literature and guidelines from professional organizations.

Key Content and Findings: We identified key elements in secondary brain injury pathophysiology and discussed approaches to minimize delayed neurological injury. Trauma patients at risk for blunt cerebrovascular injuries are discussed and the diagnostic approaches and management options outlined. We have also focused on airway concerns, hemodynamic considerations, and optimization of coagulation and cellular homeostasis in this cohort. Perioperative goals and anesthetic options for neurological injury patients requiring OMFS are highlighted, emphasizing hemodynamic stability and cerebral perfusion. Comprehensive perioperative and postoperative care to improve cerebral perfusion and cellular homeostasis are crucial for optimal outcomes.

Conclusions: Patients with neurologic injuries requiring OMFS necessitate specialized perioperative care. Understanding the pathophysiology, potential complications, and best practices for management can optimize patient outcomes. This narrative review serves as a thorough guide for clinicians involved in the care of these patients.

Keywords: Neurological injuries; oral maxillofacial surgery; perioperative management; ischemic brain injury; traumatic brain injury (TBI)

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Introduction

The co-occurrence of oral-maxillofacial (OMF) and brain injuries is relatively common, typically stemming from high-impact incidents such as motor vehicle collisions (MVCs) and blunt traumas. Additionally, individuals who experience a stroke may sustain OMF injuries for a variety of reasons, including complications arising from falls or mishaps due to impaired mobility or balance. Perioperative management of these patients poses a risk of exacerbating the neurological injuries and introduces a complex array of physiological challenges (1). Accordingly, these cases call for highly specialized anesthesia considerations to optimize patient outcomes.

A comprehensive grasp of the pathophysiology of neurological disorders and a careful approach to the physiological ramifications of anesthetic decisions can help optimize cellular homeostasis in the injured brain and minimize the progression of neurological injury. It is crucial to adeptly balance cerebral oxygen supply with demand, diligently regulate ventilation, uphold hemodynamic stability, sustain optimal fluid and electrolyte balance to curtail the risk of cerebral edema, and employ strategies for optimal pain management (2). The intricacy of these considerations amplifies when injuries prompt uncertainty in prioritization of management, as seen in trauma patients who endure concurrent brain and significant OMF injuries. This narrative review aims to provide a comprehensive understanding of the co-occurrence of OMF and brain injuries, emphasizing the specialized anesthesia considerations required for optimal patient outcomes. We present this article in accordance with the Narrative Review reporting checklist (available at https://joma.amegroups.com/article/view/10.21037/joma-23-31/rc).

Methods

A comprehensive literature review was conducted in July 18, 2023 using PubMed and Google Scholar to locate relevant articles (*Table 1*). The search strategy involved the use of several pertinent keywords and their combinations including: traumatic brain injury (TBI), venous thromboembolism (VTE), oral and maxillofacial surgery (OMFS), low molecular weight heparin (LMWH), inferior vena cava (IVC) filters, nonsteroidal anti-inflammatory drug (NSAID), tranexamic acid (TXA), and intracranial pressure (ICP). Titles and abstracts were screened followed by full-text appraisal. The selection favored high-quality studies such as randomized controlled trials, prospective cohort studies, and meta-analyses, alongside key review articles. The inclusion criteria were articles written in English. Exclusion criteria included non-English publications that were not relevant to the research topic. Key information from each selected source was documented systematically. The information derived from the included sources was sorted into relevant themes corresponding to the research question. A narrative synthesis of these findings was

Table 1 The search strategy summary

Items	Specification
Date of search	7/18/2023
Databases and other sources searched	PubMed, Google Scholar
Search terms used	'Pathophysiology', 'Secondary Brain Injury', 'Penetrating and Blunt Traumatic Brain Injury', 'Ischemic and Hemorrhagic Strokes', 'Hemodynamic monitoring', 'Fluid and Sodium Management', 'Hemostasis and Coagulation Management', 'Airway Management', 'Perioperative Anesthesia Considerations', and 'Postoperative Management'
Timeframe	1990–2023
Inclusion criteria	Narrative review; article in English
Selection process	Authors involved in writing the narrative review conducted the selection

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Figure 1 General considerations of patients with neurologic injury requiring OMFS. SBP, systolic blood pressure; CPP, cerebral perfusion pressure; TBI, traumatic brain injury; GCS, Glasgow coma scale; SIADH, syndrome of inappropriate secretion of antidiuretic hormone; CSWS, cerebral salt wasting syndrome; LMWH, low molecular weight heparin; VTE, venous thromboembolism; IVC, inferior vena cava; TXA, tranexamic acid; ENT, ear, nose, and throat; ICP, intracranial pressure; RSI, rapid sequence induction; OMFS, oral and maxillofacial surgery.

performed to provide a comprehensive overview of each theme. A systematic approach to gathering and synthesizing information was adopted, with the goal of providing a balanced and comprehensive review of anesthetic considerations for OMFS in patients with neurological injuries.

Pathophysiological considerations in patients with brain injuries

TBI

A comprehensive understanding of the primary and secondary mechanisms TBIs is paramount for devising a safe and effective perioperative care plan (*Figure 1*). Primary injuries result from direct, shearing, or rotational forces, either penetrating or blunt, and can be categorized as focal or diffuse. Focal injuries, commonly associated with severe and moderate TBIs, tend to predominantly affect the frontal and temporal lobes. These injuries can result in notable impairments in executive functioning, memory, attention, and emotional regulation (3). These injuries can also result in reperfusion injury, focal ischemia, and hematomas causing brain compression (4). On the other hand, diffuse injuries involving widespread neuronal damage are often marked by microvascular changes and axonal disconnection (4). Notably, axons in white matter are particularly vulnerable to rotational forces, a fact that can be visualized using diffusion tensor imaging with magnetic resonance imaging (MRI) (5).

Secondary brain injury refers to the cascade of cellular, molecular, and biochemical events that occur in the hours, days, or weeks following the initial injury. The mechanisms involved in secondary brain injury include delayed ischemia, impaired blood-brain-barrier and cerebral edema, oxidative stress, excitotoxicity, inflammation, and apoptosis (*Figure 2*) (6). The resulting ATP depletion disrupts the

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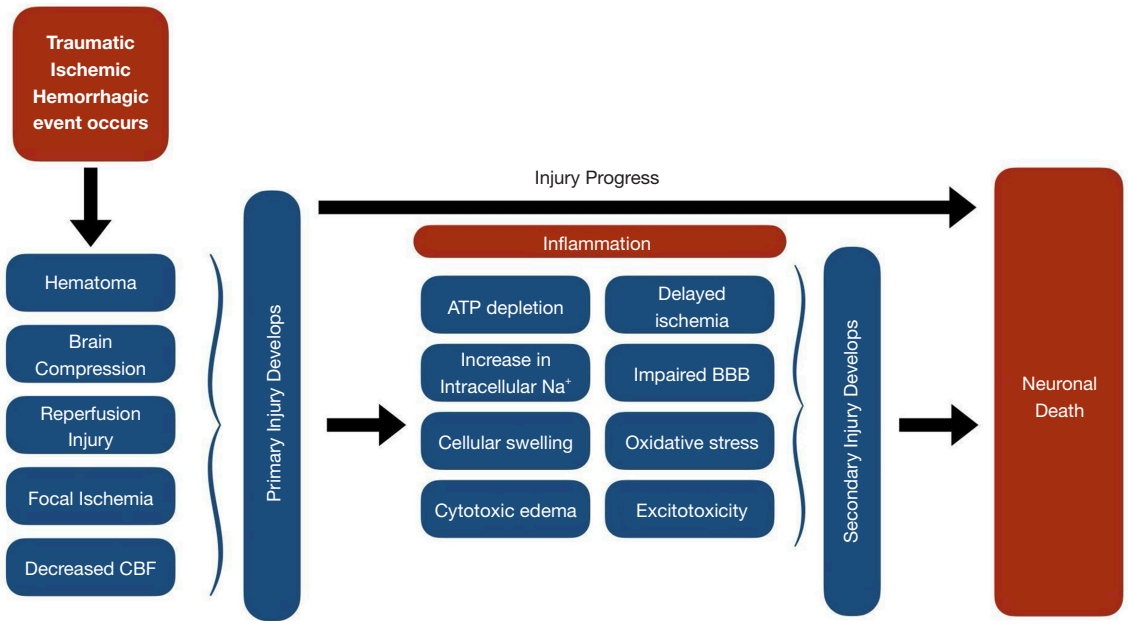

Figure 2 Timeline of primary and secondary injury events. CBF, cerebral blood flow; BBB, blood-brain barrier.

sodium-potassium pump, leading to intracellular sodium accumulation, water influx, cellular swelling, and cytotoxic edema. These processes can lead to neuronal cell death and impair the blood-brain barrier (BBB) (7). Vasogenic edema secondary to BBB breakdown typically peaks within 6–24 hours and can lead to fluid accumulation within the interstitial space, increasing oncotic pressure and obstructing smaller vessels (8). This local hypoperfusion can further exacerbate ionic gradient disturbances worsening cytotoxic edema. Increased ICP can contribute to herniation, along with reductions in cerebral perfusion pressure (CPP) and cerebral blood flow (CBF), leading to hypoxia.

Ischemic and hemorrhagic brain injury

Ischemic injuries associated with TBI can stem from various causes, including hypoxemia, impaired blood flow from large vessel occlusion, or atherosclerotic disease (9). In patients who have experienced cardiopulmonary arrest, accurately assessing the duration of circulatory and respiratory deficits is crucial in gauging the degree of neurological injury, with recovery prospect and length of perfusion deficit directly proportional to each other (9). Neuronal damage occurs within minutes of ischemia as ATP depletion disrupts the Na^+/K^+/ATPase pump (9,10). Concurrently, anoxic depolarization activates several metabolic and inflammatory cascades and enzymes that destroy neurons (9-11). In the acute phase, macrophage and neutrophil infiltration recruit inflammatory cells and directly damage neurons (11). This inflammation is further amplified by necrosed cells releasing damage-associated molecular patterns (DAMPs) into the extracellular space. Additionally, ongoing research has identified perfusion disturbances after restoration of spontaneous circulation, referred to as the no-reflow phenomenon, that may be associated with coagulation and intravascular fibrin formation (12).

Ischemic brain injuries, such as acute ischemic strokes (AIS), can lead to a myriad of perioperative neurological complications. Patients with recent AIS require a carefully designed surgical plan, with emphasis on the timing of procedures with respect to neurologic injury (*Figure 3*). The American Heart Association (AHA) recommends an interval of 6–9 months before scheduling an elective surgery in these patients (13). Those undergoing noncardiac procedures within 3 months after their initial stroke face a 68% increase in the risk of recurrent stroke (13). The AHA and American Stroke Association (ASA) also state that the risk of perioperative stroke plateaus at approximately 9 months after AIS (13,14).

Hemorrhagic strokes are also associated with significant perioperative morbidity and mortality. The pathophysiology

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Category	Pathophysiology	Important Considerations
Traumatic	• Result of direct, shearing, or rotational forces • Either penetrating or blunt	• Additional concern of secondary injury • Expanding hematomas • Penetrating trauma warrants immediate assessment - Heightened infection risk
Ischemic	• Oxygen and/or nutrient deprivation in neuronal tissue halting ATPase function, driving Na^+ influx • Na^+ influx leads to anoxic depolarization, edema, Ca^{2+} influx, driving glutamate release and activating inflammatory pathways	• Wait 6-9 months for surgery if possible following stroke • Maintain BP <185/110 mmHg prior to fibrinolytic treatment for stroke patients
Hemorrhagic	• Increased blood volume in intracranial cavity • Blood brain barrier (BBB) description • Heightened oxidative stress and inflammation	• 30% of all hemorrhagic brain injury patients face hematoma expansion within 6 hours of ICH, furthering risk of secondary injury • Vasoconstriction furthers risk of secondary injury

Figure 3 Comparison of pathophysiology and consideration of neurologic injuries. BBB, blood-brain barrier; BP, blood pressure; ICH, intracerebral hemorrhage.

of hemorrhagic brain injuries is complex, involving damage from the bleeding itself, as well as the mass effect, BBB disruption, oxidative stress, and inflammation (15). Approximately 30% of patients with intracerebral hemorrhage (ICH) will face hematoma expansion (HE) within 6 hours of their bleeding (*Figure 3*) (15). Additionally, vasospasm can increase the incidence or severity of secondary brain injury (16), as does cerebrospinal fluid (CSF) flow disruption and hydrocephalus. Similar to ischemic injury, there is also a release of proinflammatory cytokines and DAMPs, which can further exacerbate inflammatory pathways and compromise the BBB.

Trauma: penetrating versus blunt

Patients suffering from blunt and penetrating injuries share common therapeutic objectives. Although only 0.1% of trauma patients experience blunt cerebrovascular injuries (BCVIs), clinicians must exercise heightened vigilance when treating these individuals because of the risk for severe associated complications (17). Indicators of BCVI include epistaxis, significant facial bleeding, a detectable bruit, or thrill, expanding hematomas, and/or focal or lateralizing neurological deficits. The presence of these indicators should prompt clinicians to swiftly pursue imaging studies with computed tomography angiography (CTA) or magnetic resonance angiography (MRA) (*Figure 4*). Diagnostic imaging should also be considered in patients with severe TBI and a Glasgow Coma Scale (GCS) <6, mandible fracture, displaced Le Fort II or III injuries, skull fractures, or cervical spine fractures or dislocation (18). Patients who experience arterial hemorrhage from midface fractures (Le Fort II or III) have also an elevated risk for BCVI (19). Due to the abundant vascular supply from the carotid artery branches, maxillofacial fractures can be associated with significant bleeding and hematoma development, potentially compromising the airway or impacting CBF (20).

Anesthesiologists play a vital role in the management of patients with facial trauma who may also suffer vision-threatening injuries (21). Complications such as swelling, blood loss, and certain treatments can increase the risk of vision loss in these patients. By applying the principles of Advanced Trauma Life Support (ATLS) to assess, stabilize, and effectively manage these complex cases, anesthesiologists can mitigate the risk without impeding ongoing resuscitation and treatment efforts (21). The primary goal of prompt initial evaluations, especially in less responsive patients, is to rule out immediate sight-threatening injuries. Facial trauma can affect the orbit, leading to direct injury to the optic nerve or retinal detachment (22). Various treatment approaches, such as corticosteroid therapy and surgical decompression, are common, but there is a lack of consensus on the most effective treatment. The expertise and vigilance of anesthesiologists are therefore pivotal in managing facial trauma patients at risk of vision-threatening injuries.

Providing care for patients with penetrating brain injuries (PBIs) necessitates an immediate focused evaluation of factors such as trajectory, depth, and the extent of tissue damage. Patients who experience PBI face an increased risk of infection (23). Foreign bodies, bone fragments, and

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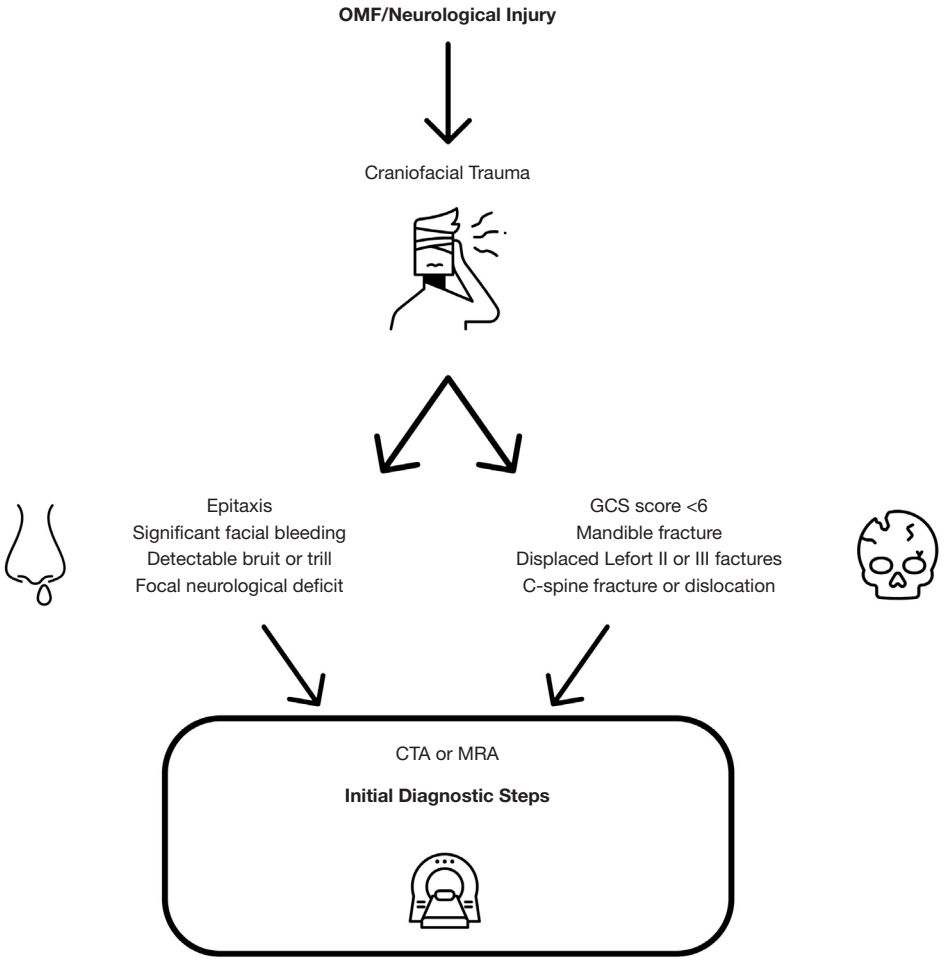

Figure 4 Initial diagnostic management of OMF trauma patients with intracranial injury. OMF, oral-maxillofacial; GCS, Glasgow Coma Scale; CTA, computed tomography angiography; MRA, magnetic resonance angiography.

debris in the wound can lead to increased inflammation and tissue swelling, furthering the risk of elevated ICP. While *Staphylococcus aureus* is the most frequent causative organism, gram-negative bacteria can also lead to intracranial infections. The clinical benefits of prophylactic antibiotics have not been established in this population, yet clinicians are commonly advised to initiate a short course of broad-spectrum antibiotics, with an extended duration in those with gross contamination of the wound (24). Cephalosporins are an appropriate choice in most cases, with the addition of metronidazole if organic debris are present (24). Certain prognostic factors, including advanced age, close-range injuries, perforating injuries, hypotension, and elevated ICP are predictive of high mortality (25). The Surviving Penetrating Injury to the Brain (SPIN) score can be used to estimate survival following a PBI (26).

High-velocity PBI is a severe form of TBI, resulting from high-energy events such as firearm or blast injuries, transmitted to the brain and surrounding tissues (27). The cavitation to a projectile can collapse and re-expand cyclically, leading to extensive neuronal injury, hematomas, and parenchymal contusions (27). When a projectile travels through brain tissue, it induces an asynchronous motion in the skull and the brain, which can cause diffuse axonal injury (28). Secondary injuries due to bleeding, increased edema, and ICP elevation can also occur.

Airway management

Patients with TBI and facial trauma present unique challenges for anesthesiologists as facial trauma can distort the normal anatomy, posing potential intubation

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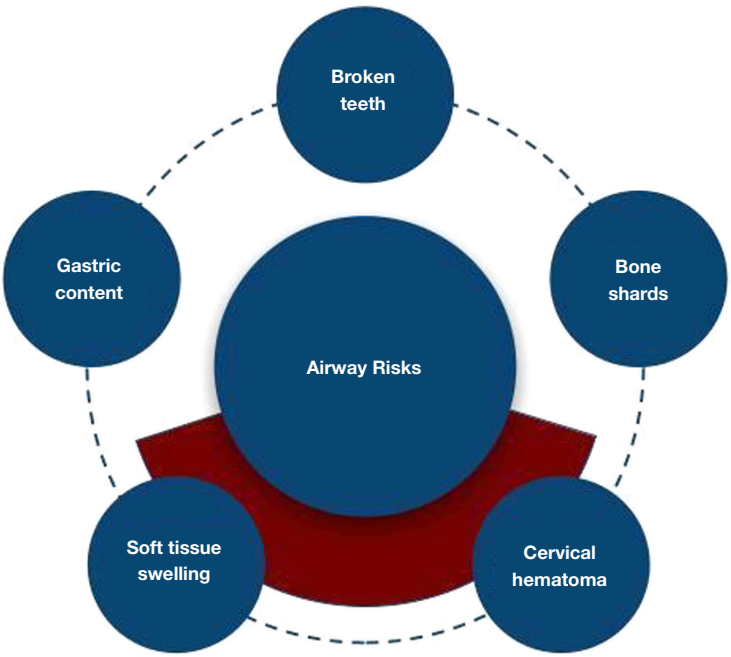

Figure 5 Airway obstruction risks associated with head and neck trauma patients.

challenges. Head injuries that cause loss of consciousness may also lead to functional airway obstruction (29). In patients with craniofacial trauma, GCS scores of 8 or lower indicate a critical condition that may require immediate intervention to secure the airway (30). Hutchison *et al.* [1990] identified six specific scenarios linked to maxillofacial trauma that can be predictive for difficult airway management, listed in *Figure 5* (31). Aspiration of blood or gastric content can complicate airway management (32). Every TBI patient in need of immediate intubation should be treated as full stomach, and their airway management should also take into consideration the potential for cervical spine injuries (33).

The presence of facial fractures in trauma patients can act as a clinical indicator of possible CNS injury, heightening awareness and the need for timely intervention (34-36). Continuous monitoring of neurologic function may be warranted, and cerebrovascular and spine imaging considered. In TBI patients with concurrent cervical spine injuries, maintaining cervical spine precautions during airway management is important to prevent any additional injury. The ATLS guidelines emphasize the importance of immobilizing the cervical spine during airway management in patients who have endured severe injuries (37). Cautious mask ventilation may be considered to avoid progressive hypoxemia (30) but pre-oxygenating and positive pressure mask ventilation may present unique challenges, as these patients may have experienced anatomical alterations of the face, oral cavity, and/or oropharynx (32). Pneumocephalus has also been reported, both after mask ventilation and ventilation using a laryngeal mask airway (38).

As laryngoscopy and intubation can temporarily elevate ICP due to heightened sympathetic adrenergic activity, premedication with lidocaine, fentanyl, or esmolol should be considered (39). Rapid sequence induction (RSI) may be used to reduce the risk of aspiration. Etomidate and propofol are shown to decrease the cerebral metabolic rate for oxygen ($CMRO_2$) and CBF, mitigating the rise in ICP during intubation. The previously reported association between ketamine and elevated ICP has been questioned in multiple recent reviews, which found no detrimental increase in CBF or ICP (40). For muscle relaxants, succinylcholine has been suggested to cause elevations in ICP, but studies have failed to prove clear detrimental outcomes (41). Rocuronium should be considered as the primary neuromuscular blocking agent to eliminate any potential risk for adverse ICP effects. Additional considerations to minimize ICP elevation include employing ventilation strategies to minimize hypoxia and hypercarbia, as well as careful titration of positive end-

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expiratory pressure (PEEP). The effects of PEEP on ICP remain controversial, but recent evidence suggests that in the absence of severe lung injury, PEEP can be applied safely in patients with TBI (42).

Severe facial trauma may lead to respiratory failure or acute airway obstruction, making airway management particularly difficult (43). Strategies for airway management should not only include endotracheal intubation but also surgical airway via tracheostomy or cricothyrotomy. Factors such as the patient's anatomy, the extent of facial and/or airway trauma, and the anesthesiologist or surgeon's expertise should all be considered in managing the airways of these patients. In facial fracture patients who require early airway management, the presence of TBI does not affect the site of airway management (44). In the absence of contraindications, endotracheal intubation is the preferred method of airway access in patients with a suppressed state of arousal (43). Options may include direct laryngoscopy, video laryngoscopy, or fiberoptic intubation. In patients with cervical spine injuries, video laryngoscopy, and fiberoptic intubation are thought to minimize neck movement and the associated risk for worsening spinal cord injury (45,46). In a critical emergent scenario, the surgical airway becomes the preferred course of action when all other attempts to establish a patent airway prove unsuccessful. In such instances, surgical cricothyroidotomy emerges as a more expedient and straightforward option. Tracheostomy can be considered once the patient's condition stabilizes, thereby reducing the risk of subglottic stenosis (20).

Recognition of bilateral fractures of the anterior mandible is important because of the potential for the fractured symphysis and the tongue to displace posteriorly, resulting in oropharyngeal obstruction when the patient is in a supine position (32). It is also crucial to emphasize that in cases of limited mouth opening due to a temporomandibular joint injury, sedation will not ameliorate mouth opening and may exacerbate the situation (47). For patients with comminuted midface or nasal fractures where nasal intubation is contraindicated, submental orotracheal intubation can provide an alternative airway securing method. For this approach, an incision is made midway between the chin and the angle of the mandible. Following orotracheal intubation, a blunt dissection is performed through the oral floor, including the superficial fascia, platysma, and deep fascia (32). It is important to note that this type of intubation should not be employed in patients with comminuted mandibular fractures. Extubation of patients with TBI and facial trauma must also be carefully planned to avoid airway compromise due to laryngospasm, aspiration, or loss of airway patency (48).

Hemodynamic considerations

Despite the rich vascular supply, transfusion of blood products is infrequently required in maxillofacial trauma patients in the absence of coagulopathy, systemic disease, or concomitant injuries (18). However, given that up to 5% of these patients can face severe life-threatening bleeding (49,50), it is imperative to reduce the likelihood of intraoperative blood loss (IOB). Strategic patient positioning, controlled hemodynamics, and the use of antifibrinolytic agents and topical vasoconstrictors can help reduce transfusion requirements (51).

Any decision to employ controlled hypotension and establish a target blood pressure (BP) to control blood loss should be rigorously deliberated and jointly agreed upon in preoperative discussions. This approach is usually not recommended for patients with prior or current neurological injuries, including those with TBI, as they are at risk for significant neurological deterioration if their cerebral perfusion is compromised through induced hypotension. Avoiding fluctuations in BP should be a key focus in every patient with TBI, as both hypotensive and hypertensive episodes have been associated with unfavorable outcomes (52-55). Although the exact parameters for safe BP are not set, current guidelines recommend maintaining a systolic blood pressure (SBP) of ≥100 mmHg for TBI patients 50 to 69 years of age, and ≥110 mmHg for individuals 15 to 49 or over 70 years old (55). A CPP of 60 to 70 mmHg is also recommended to improve survival and favorable neurological outcomes.

The underlying pathophysiological mechanisms of secondary brain injury include risk factors such as hypotension and decreased CPP. However, hypertension with a loss of autoregulatory response can also lead to worse outcomes (56). Catecholamine-induced hypertension can exacerbate vasogenic edema in brain regions with compromised blood-brain-barrier integrity (57,58). Cerebral edema is a leading cause of mortality after TBI and is associated with unfavorable neurological prognosis (8). Furthermore, systemic hypertension stands as a well-recognized risk factor for HE, prominently observed in patients experiencing acute ICH (59). Accordingly, a target SBP range of <140–160 mmHg is currently recommended in these patients (60).

Similarly, in AIS patients with large vessel occlusion

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undergoing mechanical thrombectomy, the exact BP recommended for the best neurologic outcome remains undetermined. Lower BP after infarction has been associated with hypoperfusion of brain tissue and exacerbation of concomitant cardiac disease (61-63). Patients additionally have a higher risk for early recurrent stroke and fatal brain edema (63,64). Following an AIS, cerebral autoregulation can be impaired, increasing the effects of systemic pressures on cerebral perfusion. Current guidelines based on the INTERACT-2 and ATACH-2 trials note that a goal SBP <140 (or 130–150) mmHg is safe and reasonable in ischemic stroke patients (65,66). It is important in this setting to remember that more intensive BP reduction can lead to an increased incidence of acute kidney injury (67). Current AHA/ASA guidelines also recommend a BP <185/110 mmHg prior to fibrinolytic treatment, with continued BP maintenance in the following 24 hours after tissue plasminogen activator (tPA) or mechanical thrombectomy (68). Rapid BP reduction can worsen cerebral ischemia, so an initial reduction of BP by 15% is recommended for patients with cardiac comorbidities such as acute coronary syndrome or acute heart failure (69). For patients with acute hemorrhagic stroke, early BP lowering to 140 mmHg systolic is safe, although it does not demonstrate a significant reduction in the rate of death or disability (69). When bundled with strict blood glucose, temperature, and coagulation management, a SBP <140 mmHg was, nevertheless, associated with better neurological outcome (70).

Fluid and sodium management

In patients with severe TBI, fluid and electrolyte management is important because of its significant impact on cellular homeostasis and ICP. This aspect of care becomes even more critical considering the high incidence of serum sodium dysregulation, which can affect up to 68% of patients (71). These alterations may manifest as hypernatremia through diabetes insipidus (DI), or hyponatremia via syndrome of inappropriate secretion of antidiuretic hormone (SIADH) or cerebral salt wasting syndrome (CSWS) (72). These conditions can be induced by the primary brain injury or the subsequent events and management and can increase morbidity and mortality after TBI (73,74).

Central DI-induced hypernatremia can arise from an insult to the hypothalamic-pituitary axis, resulting in decreased antidiuretic hormone (ADH) production, or from impairment of osmoreceptors leading to hypotonic polyuria. Management should prioritize fluid replenishment (75). Regular observation and correction of electrolytes and fluid balance, as well as plasma and urine osmolalities, is prudent (76). Patients with DI and a urine output greater than 250 mL/h may be considered for hormone replacement therapy with desmopressin (77). Hypernatremia should be corrected gradually to prevent rapid shifts in serum osmolality, which can lead to cerebral edema, seizures, or cardiac arrhythmias (78). The recommended rate of correction is no more than 0.5 to 1 mEq/L per hour and 10 to 12 mEq/L over 24 hours. Frequent monitoring of serum sodium levels is crucial, and patients' neurological status, fluid balance, urine output, and electrolyte levels should be closely monitored. As plasma sodium concentration increases, the extracellular shift of water can cause a rapid decrease in the brain volume with an associated risk of hemorrhage and neurological damage (79). Mortality of TBI patients with severe hypernatremia has been recorded as high as 76–86% (74,80). Hyperchloremia is also a predictor of mortality in these patients (81).

Common causes of hyponatremia in patients with TBI include disruption of the BBB, SIADH, and CSWS (82). Although SIADH and CSWS can have overlapping signs, symptoms, and laboratory results, differentiation is important as each condition requires distinct treatment approaches for effective management. In SIADH, patients can be normovolemic or hypervolemic. Interventions should center on fluid balance and management of ADH secretion. Mild cases can be treated with fluid restriction as a first-line approach, or with hypertonic saline (HTS) or enteral salt tablets. In rare cases of prolonged SIADH, demeclocycline is effective and well-tolerated (83). In CSWS, patients may be hypovolemic, and management will focus on replacement with sodium-containing solutions. For mild to moderate cases, patients can be started on isotonic saline. More aggressive therapy with HTS or salt tablets and free water restriction may be required for severe hyponatremia. If sodium levels are resistant to correction, rescue with mineralocorticoids such as low-dose fludrocortisone may be considered (84,85). Caution should be taken with fludrocortisone, as it can contribute to hypokalemia and high BP (86). Hyponatremia may also occur due to reduced oral intake. Additionally, some medications, including antiepileptic drugs, can affect sodium and fluid balance and contribute to hyponatremia (87). When correcting hyponatremia, patients

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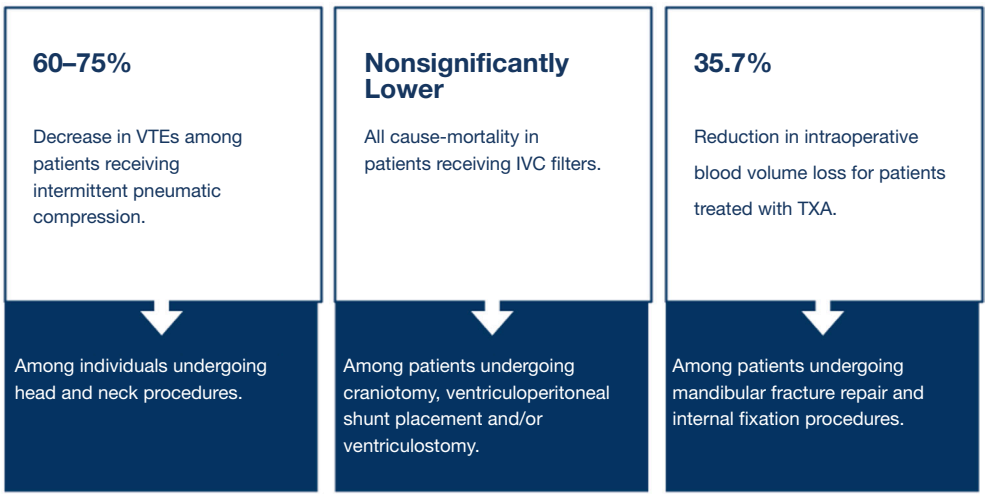

Figure 6 Reduction in VTE and IOB through various management strategies. VTE, venous thromboembolism; IVC, inferior vena cava; TXA, tranexamic acid; IOB, intraoperative blood loss.

should be closely monitored to prevent rapid shifts in serum osmolality (88). The recommended rate of correction is no more than 0.5 to 1 mEq/L per hour to 10 to 12 mEq/L over 24 hours.

There is a critical phase of cerebral edema when osmotic therapy is employed to control increased ICP in TBI patients. In these cases, patients present with neurological deterioration due to compromised brain perfusion. Acute hyponatremia can worsen brain swelling and metabolic encephalopathy with overall mortality up to 34% (89). Two primary osmotic agents utilized for serum sodium correction are HTS and mannitol, with mannitol found to be slightly more effective in reducing ICP (90). However, HTS may have a more sustained effect than mannitol on the ICP (90). Both treatment options should be carefully titrated to minimize the risk of rapid cerebral edema reversal. Current guidelines suggest that there is insufficient evidence to support the use of a particular hyperosmolar agent in patients with severe TBI (55).

Hemostasis, coagulation management, and VTE prophylaxis

In the setting of trauma, a combination of factors, including hemorrhagic shock, consumption coagulopathy, hypothermia, or hypoperfusion-induced metabolic acidosis can lead to disruptions in hemostasis (91,92). In TBI patients, acute coagulopathy has been associated with worse outcomes (93,94). Perioperative management is multifactorial, focusing on bleeding control and timely administration of hemostatic therapy. Conventional coagulation testing includes platelet number, fibrinogen level, prothrombin time (PT), and partial thromboplastin time (PTT), but a viscoelastic hemostatic assay can yield quicker results (95). In cases of major hemorrhage, physicians may be reluctant to initiate VTE prophylaxis. However, patients may transition from a hemorrhagic state to a prothrombotic state due to the hypercoagulability associated with resuscitation and the acute phase response (96). While VTEs are high mortality events, prophylactic anticoagulation is a double-edged sword that carries a heightened risk-benefit ratio for patients receiving head and neck surgeries (*Figure 6*).

In patients who experience no complications during the first 48 hours after TBI, consideration may be given to initiating thrombosis prophylaxis using LMWH (97). However, patients with compromised renal function and those facing significant risk for bleeding may be better candidates for unfractionated heparin (97). In patients with moderate risk of bleeding complications, a stability head CT should be obtained prior to the initiation of prophylactic anticoagulation; high-risk TBI patients may be better candidates for mechanical devices (98). Patients with active brain hemorrhage, recent intracranial surgery, and coagulation disorders may not be candidates for chemoprophylaxis. Internal mechanical devices, such as IVC filters, may offer protection in reducing pulmonary embolisms in some trauma patients (99). External devices

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such as intermittent pneumatic compression (IPC) have also been shown to decrease deep vein thrombosis (DVT) by 60–75% with minimal complications in patients receiving head and neck procedures (99-102). The American College of Chest Physicians and American Society of Clinical Oncology both maintain that LMWH administered along with mechanical measures is the gold standard in reducing VTE risk (102,103).

The CRASH-3 and ULTRA trials found that TXA, an antifibrinolytic agent, had no significant effect on survival for patients with severe TBI or subarachnoid hemorrhage (104,105). However, a significant reduction in morbidity and complications was noted among CRASH-3 patients with mild TBI (GCS 3–8) when TXA was given within 3 hours of initial injury. The PATCH-Trauma trial demonstrated that severe TBI patients given prophylactic prehospital TXA had lower early hospital mortality but similar functional outcomes 6 months after injury compared to a placebo (106). Thus, the most important factors to consider when using TXA in TBI patients are severity of injury and time from injury (107). In OMFS, TXA may significantly reduce IOB, help maintain a higher hemoglobin level, and potentially decrease operating time by an average of 15 minutes (108,109). Patients taking continuous vitamin K antagonists (VKAs) undergoing minor oral surgery experienced significant reductions in postoperative bleeding after receiving TXA (110). Perioperative management of patients on long-term oral anticoagulation or antiplatelet therapy may include "bridging" their home treatment with short-acting anticoagulants such as heparin for 24–72 hours postoperatively before resuming systemic anticoagulation (111,112).

Perioperative considerations

When managing OMF trauma patients with intracranial injury, it is important to consider the entire continuum of care from preoperative to intraoperative to postoperative stages (*Figure* 7). Intraoperative hypotension is common in patients with isolated TBI undergoing craniotomy. Factors contributing to an increased risk of hypotension include the presence of a subdural hematoma, higher heart rate, and longer duration of anesthesia (113,114). Similarly, up to 60% of patients undergoing orthopedic surgeries are reported to experience intraoperative hypotension (115). The exact mechanism of this perioperative instability varies between cases, but in patients with severe brain injuries the associated sympathetic surge may cause myocardial or pulmonary dysfunction, which can be exacerbated by anesthetic agents that impact cerebral and cardiovascular physiology (116-118). If the patient has suffered a facial fracture or traumatic injury to the skull, a TBI must be assumed to be present until proven otherwise and special precautions must be taken (*Figure 8*). The timing of operative care is influenced by factors including the severity of the TBI, the extent of additional injuries, and the patient's comorbidities. Anesthesiologists should perform a thorough preoperative assessment prior to induction of anesthesia, though the acuity of the patient's condition and altered neurological status secondary to TBI may limit examination.

Given the increased risk for secondary brain injury, consider invasive BP monitoring via arterial catheterization. Monitoring of ICP can also be helpful as subtle changes in hemodynamics, fluid status, patient position, and other factors can lead to rapid shifts in ICP. Since noninvasive methods of ICP monitoring has not been validated, patients with severe TBI should be managed under the presumption of elevated ICP until evidence suggests otherwise (119). Intraoperative neuromonitoring with EEG can assess the presence and location of seizure or other abnormal electrical activity secondary to neuronal injury (120). Other methods of intraoperative neuromonitoring, such as invasive brain tissue oxygenation measurement, jugular venous oximetry, and cerebral microdialysis are available but less commonly utilized (120,121). Furthermore, it is essential to establish venous access with at least two large-bore intravenous lines, ensuring readiness for fluid management and potential emergencies. A central venous line is valuable and allows for rapid, safe infusion of fluids, blood products, and anesthetics. However, the utility of these tools must be balanced with the need to correct emergent intracranial processes.

Key factors that must be considered regardless of the agent selected for TBI patients include maintenance of hemodynamic stability and cerebral perfusion, and prevention of ICP increase. Curtailing the progression and formation of additional injuries that will either damage the central nervous system or worsen the patient's prognosis is paramount (122). Agents with anticonvulsant and anxiolytic properties, such as benzodiazepines and barbiturates, may reduce cerebral metabolic demand and mitigate the adverse physiological effects of intubation and mechanical ventilation. However, these medications may also reduce the ability to accurately assess neurological status, as well as increase the risk of respiratory depression

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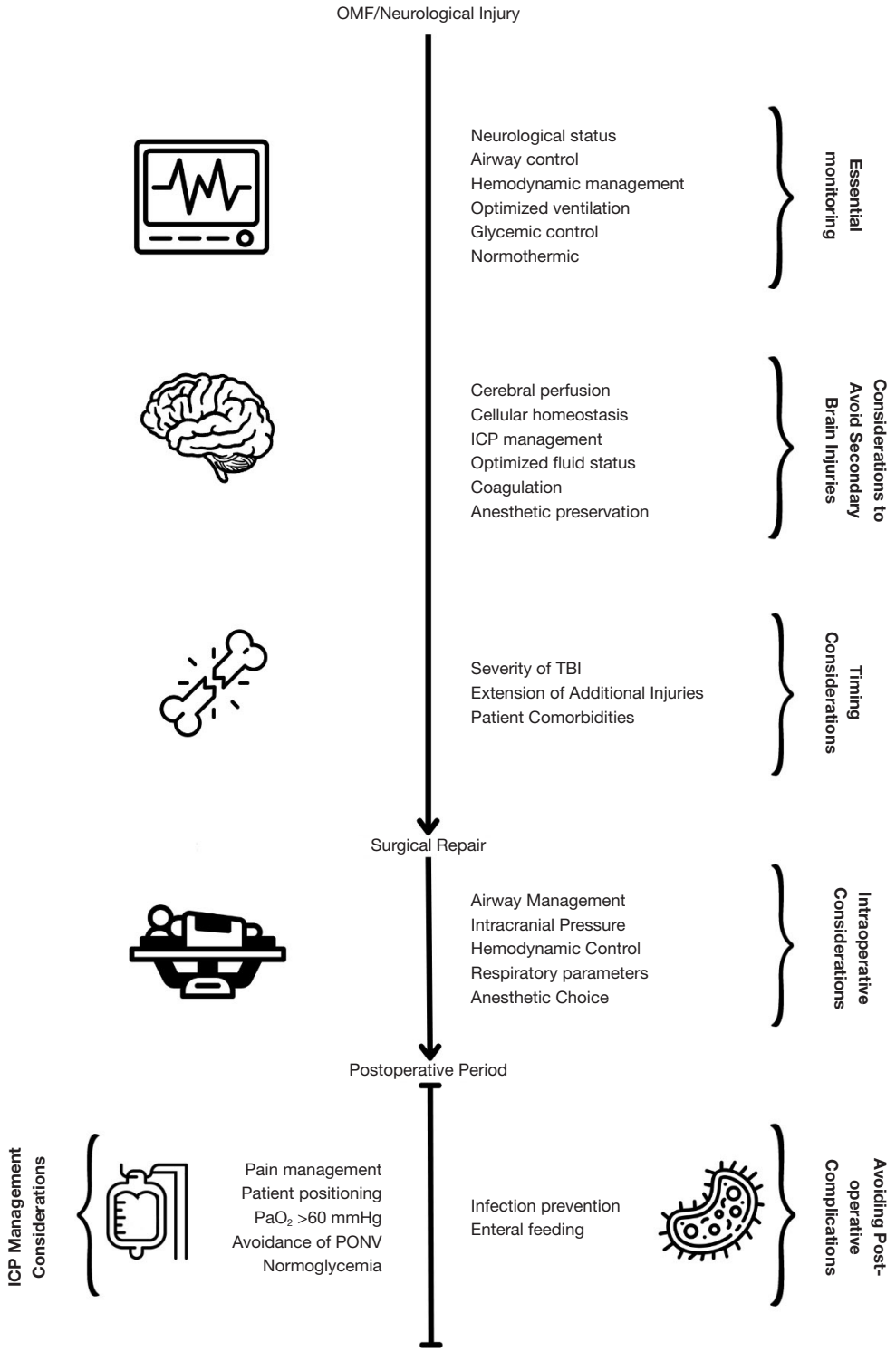

Figure 7 Management and considerations of OMF trauma patients with intracranial injury from preoperative to intraoperative to postoperative care. OMF, oral-maxillofacial; ICP, intracranial pressure; TBI, traumatic brain injury; PONV, postoperative nausea and vomiting.

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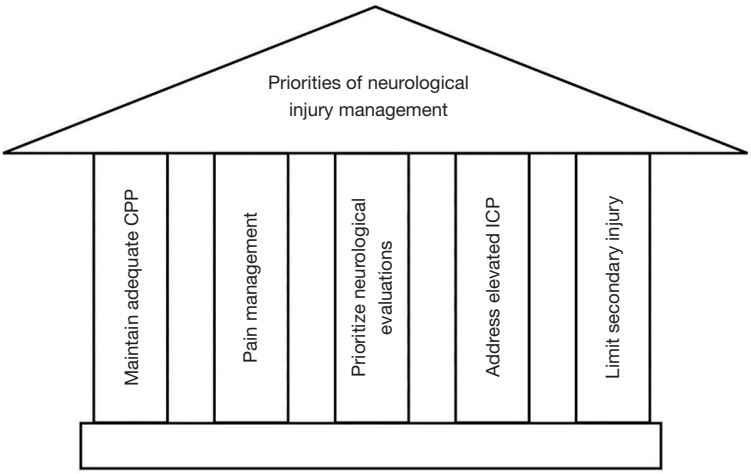

Figure 8 Five priorities of neurological injury management. CPP, cerebral perfusion pressure; ICP, intracranial pressure.

and aspiration in TBI patients (123). When emergent operative intervention is required, patients with suspected TBI should be assumed to have a full stomach, and standard RSI practices should be followed keeping in mind the patient's facial injuries. Studies have been inconsistent regarding succinylcholine's effect on transient ICP elevation (124,125). Commonly used agents appropriate for use in TBI include fentanyl, propofol, and etomidate. The use of ketamine as an induction agent has gained considerable interest in recent years due to its possible neuroprotective effects. When administered with propofol, ketamine was found to result in lower rates of hypotension and vasopressor requirements (126).

The ideal protocol for anesthesia maintenance in the setting of TBI and facial trauma is also debated. Inhaled anesthetics, such as sevoflurane, may be used as long as its effects on ICP, systemic pressures, and CPP are considered (127). Some studies suggest that total intravenous anesthesia (TIVA) results in better outcomes with reduced CBF and $CMRO_2$ (128). Other research has failed to show a significant difference between volatile anesthetics and propofol (127). A large recent study, on the other hand, found reduced rates of postoperative ischemic stroke with increasing doses of volatile anesthetics, suggesting superior perioperative neuroprotection with these agents (129). Dexmedetomidine may be a reasonable adjunct as this agent can reduce ICP and the need for sedatives and analgesics, and improve survival (130-132). Corticosteroids were once widely used to reduce cerebral edema and ICP. However, further studies have demonstrated significantly higher rates of mortality and worse outcomes in patients given high-dose corticosteroids (133).

Postoperative management

In the post-surgical care of TBI patients with accompanying facial injuries, clinicians must ensure that monitoring equipment is accurately calibrated and fully operational (*Figure 7*). It is important to identify any unusual increases in the ICP to minimize the risk of hypoperfusion, herniation, and delayed neurological injury. After major intracranial surgery, especially skull base and spine procedures, many patients typically experience pain for the initial 48 hours, which can elevate ICP (134). Proper pain management with a multimodal regimen may aid in achieving better neurological evaluations, prevent significant variations in BP, and promote faster recovery.

In patients with refractory intracranial hypertension, neuromuscular blocking agents may be considered to prevent coughing and ventilator dyssynchrony (115). Patient positioning is also important to ensure no hindrance to cerebral venous drainage and to avoid excessive neck flexion or rotation. Whereas short periods of hyperventilation may be considered for acute ICP control, its prolonged application should be avoided given concern for cerebral ischemia. Ventilation should also be optimized to maintain a PaO_2 above 60 mmHg (135). Both mannitol and HTS can be used to treat intracranial hypertension, with some guidelines recommending the use of HTS as the initial agent with symptom-based dosing instead of a target serum concentration (136). An external ventricular drain (EVD) facilitates continuous ICP monitoring while

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allowing cerebrospinal fluid drainage to manage ICP fluctuations. While cooling the brain might theoretically mitigate secondary brain injuries by reducing inflammation and intracranial hypertension, the Eurotherm3235 Trial demonstrated that patients with an ICP over 20 mmHg post-TBI did not benefit more from therapeutic hypothermia combined with standard care (137).

About 50% of craniotomy patients experience postoperative nausea and vomiting (PONV) after TBI (134). Vomiting can increase the risk of gastric content aspiration as well as elevate ICP due to systemic hypertension and increased abdominal and chest pressures (137). To counteract PONV, medications such as ondansetron, dexamethasone, antihistamines, phenothiazines, and even scopolamine patches can be considered (134). Hyperglycemia is associated with aggravated secondary brain injury and worse outcomes following TBI (138). Given the current evidence, a target glucose range of 80–140 mg/dL in the perioperative period appears reasonable (115).

Proper care of facial wounds to prevent infection and promote healing is essential in the postoperative management of facial trauma patients with TBI. Goh *et al.* [2021] found infections to be the most reported postoperative complication in this cohort (139). Fracture stabilization should be prioritized as immobilization can reduce the risk of infection (140). Typically, in patients with penetrating TBI and those with foreign materials, such as intracranial electrodes for deep brain stimulation or epilepsy management, prophylactic antibiotics are recommended. They may also be considered during invasive ICP monitoring, in situations with contaminated wounds, or when there is a heightened risk of infection, as seen with a CSF leak. Due to the potential for bacterial contamination in maxillofacial fractures, antibiotic prophylaxis is frequently employed (140). It is worth mentioning that prolonged antibiotic prophylaxis has not been shown to be beneficial for surgically treated facial fractures (141). Oral hygiene is also important as bacterial invasion from the fracture, loose or absent teeth, and disruptions in natural cleaning mechanisms heighten infection risks. Saline, peroxide, or chlorhexidine gluconate may be used for oral care. While the effectiveness of chlorhexidine in preventing ventilator-associated pneumonia has been a topic of debate, a recent meta-analysis has reaffirmed its efficacy in promoting wound healing after oral surgery (142).

After TBI and surgery, enteral feeding should begin orally, through a nasogastric tube, or via a transgastric tube as soon as possible. It is important to note that extensive facial trauma with associated skull base and sphenoid sinus fractures or bone defects may allow inadvertent intracranial penetration of a nasogastric tube (143). Recommendations suggest reaching nutritional targets within 5 to 7 days post-injury (144). Post-extubation management can be challenging in the presence of facial injuries and altered anatomy. Important factors to consider include the extent of the brain injury, the patient's state of arousal and ability to protect their airway, the application of maxillomandibular fixation, and other concurrent health conditions and injuries. Careful planning for extubation over an airway exchange catheter through the oro-/nasotracheal route may be considered (145).

Limitations

This narrative review has several limitations that warrant consideration. We predominantly relied on articles written in English, potentially omitting valuable insights from non-English publications. Our selection criteria also favored high-quality studies, such as randomized controlled trials and meta-analyses, which might have led to the exclusion of smaller scale or less rigorous studies that could offer additional insights into the management of neurologically injured patients undergoing OMFS. Additionally, the review's scope was confined to the thematic areas identified through our search strategy, possibly overlooking other pertinent aspects not encapsulated by the selected keywords. Moreover, the narrative synthesis approach, aiming to provide a balanced overview of the current literature and guidelines, might have introduced subjective interpretations by the authors. This could affect the objectivity of the findings presented.

Conclusions

Traumatic, ischemic, and hemorrhagic brain injuries in OMFS patients present significant challenges due to their intricate management and long-term implications. Recognizing both primary and secondary neurological injuries is crucial, emphasizing the role of anesthesiologists in preventing further damage during surgeries. Managing facial and brain injuries demands attention to hemodynamics, fluid balance, hemostasis, and coagulation. Anesthesiologists can encounter significant challenges in managing the airway, and during perioperative and postoperative care, particularly in cases with altered

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anatomy and potential for cervical spine instability.

For achieving optimal patient outcomes, continuous neurological monitoring is essential, along with a judicious selection of anesthetic agents and techniques. Furthermore, comprehensive intra- and postoperative care, which includes effective pain management, glycemic control, and maintaining cellular homeostasis, are critical components of the overall treatment strategy.

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