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Management of Traumatic Brain Injury

Soe Wunna Htay

Abstract

Head trauma or traumatic brain injury (TBI) is one of the most serious, life-threatening conditions in trauma victims. Prompt and appropriate therapy is essential to obtain a favorable outcome. The aim of the acute care of patients with brain injury is to optimize cerebral perfusion and oxygenation and to avoid secondary brain injury. Secondary brain injury develops with times and cause further damage to nervous tissues. The common denominators of secondary injury are cerebral hypoxia and ischemia. A systemic approach such as the Advanced Trauma Life Support (ATLS) algorithm has been recommended for managing head injury patients. Quick initial assessment of the patient's neurologic condition thoroughly is mandatory. There should be attention in evidence of intrathoracic or intraperitoneal hemorrhage in multiple traumatized patients. Optimizing the open airway and adequate ventilation depending on patient's neurologic condition is first step in emergency therapy. Cerebral perfusion pressure should be maintained between 50 and 70 mmHg. Systemic hypotension is one of the major contributors to poor outcome after head trauma. Careful stabilization of the blood pressure with fluid resuscitation and a continuous infusion of an inotrope or vasopressor may be necessary. Standard monitoring with direct arterial blood pressure monitoring and periodical measurement of arterial blood gases, hematocrit, electrolytes, glucose, and serum osmolality are important. Brain monitoring as with an electroencephalogram, evoked potentials, jugular venous bulb oxygen saturation (Sjo₂), flow velocity measured by transcranial Doppler (TCD), brain tissue oxygenation (btPo₂), and ICP monitoring may be used. The reduction of elevated ICP by means of giving barbituates, hyperventilation, diuretics and hyperosmolar fluid therapy, body posture and incremental CSF drainage are critical. Seizure prophylaxis, early enteral feeding, stress ulcer prophylaxis, prevention of hyperglycemic state, fever and prophylaxis against deep venous thrombosis in neurointensive care unit are also important after successful resuscitation of head trauma patients.

Keywords: traumatic brain injury, head trauma

1. Introduction

Head trauma or traumatic brain injury (TBI) is one of the most serious, life-threatening clinical problem related with long-term neurobehavioral and socioeconomic consequences in trauma victim [1].

Prompt and appropriate therapy is necessary to obtain a favorable outcome. The management of patient with head injury focuses aggressively on the

stabilization and resuscitation of the patient from hypoxia, hypoventilation and cardiovascular collapse. These preventable and treatable secondary insults can complicate the course of patients with head injuries and adversely affect outcome.

2. Systemic and intracranial causes of secondary brain injury

Systemic causes	Intracranial causes
Hypotension	Intracranial hypertension
Hypoxia	Edema
Hypoglycemia/hyperglycemia	Vasospasm
Acidosis	Seizures
Sepsis	Infection
Hyperthermia	
Coagulopathy	
Anemia	

The neurosurgical team members especially anesthesiologists manage perioperative course, taking the patients from the emergency room to the neuroradiology suite, the operating room, and the neurointensive care unit.

3. Emergency management

3.1 Initial assessment of the patient’s condition

Glasgow Coma Scale (GCS) assessment can be used for assessing neurologic condition of head trauma patient. Trained health care providers can measure GCS. GCS is based on 15 point scale for estimating severity of brain injury following trauma [2].

- 1. GCS score of 3 to 8 represents severe head injury.
- 2. GCS score of 9 to 12 represents moderate injury.
- 3. GCS score of 13 to 15 represents mild injury [3].

Pupillary responses (size, light reflex) and symmetry of motor function in the extremities should be quickly examined [4].

Head trauma patients are also associated with injury to other parts of body. If the patients presented with shock, thoracic and abdominal injury should be assessed for intrathoracic or intrabdominal bleeding.

3.2 Advanced trauma life support (ATLS) algorithm

There is best accomplished by using a systemic approach, Advanced Trauma Life Support (ATLS) algorithm, which consists of primary and secondary surveys of the patient.

3.2.1 Primary survey

A brief history taking and examination have to be performed within a short period. The history is obtained according to the AMPLE mnemonic (allergies, medications, past medical history, last meal and event). Examination and immediate resuscitation are performed according to the ABCDE mnemonic (airway, breathing, circulation, disability, exposure).

- i. Airway management of the patient: The careful monitoring of changes in mean arterial pressure (MAP), intracranial pressure (ICP), and partial pressures of arterial carbon dioxide (PaCO_2) and oxygen (PaO_2) during airway management of traumatic brain injury patient is essential.

Indications for intubation include

- inability to protect the airway,
- difficulty with either oxygenation or ventilation,
- shock,
- GCS score <9, or
- Rapid neurologic deterioration [5].

If the cervical spine injury has not been precluded, manual in line stabilization of head and neck is important during endotracheal intubation. Rapid sequence induction and intubation have to perform in patient with full stomach, using direct laryngoscopy. Flexible fiberoptic intubation may be valuable in patient who have difficult airway and unstable cervical spine fractures. Laryngeal mask airways (LMAs) including the intubating LMAs and surgical airway techniques such as cricothyroidotomy and tracheotomy are useful back up techniques for ventilation and intubation.

Intravenous uses of lidocaine, 1.5 mg/kg as a pretreatment before endotracheal intubation has been shown to blunt the increase in ICP in response to airway manipulation [6]. If the vital signs of patient are stable, using propofol and thiopental during induction can decrease intracranial pressure and cerebral metabolic rate of oxygen consumption (CMRO_2). While hemodynamic condition of the patient is unstable etomidate 0.3 mg/kg may be a better choice [7].

Use of muscle relaxants facilitate tracheal intubation and decrease the risk of straining. 1 to 1.5 mg/kg of depolarizing muscle relaxant, succinylcholine can be given in emergence condition. Succinylcholine is contraindicated in TBI associated with spinal cord crush, or burn injury owing to the risk of hyperkalemia [8]. Nondepolarizing neuromuscular blocking drugs (NDNMB) including rocuronium, 1 mg/kg, and mivacurium, 0.2 mg/kg, do not increase ICP and can be used in endotracheal intubation in emergence condition. However, nondepolarization muscle relaxants use have a slower onset of action (60 to 90 seconds) and caution with allergy using these agents [9].

- ii. Breathing considerations include the following: Supplemental high-flow oxygen is provided to all patients to prevent hypoxia (PaO_2 <90 mm Hg)

regardless of patient's neurologic condition. Positive pressure ventilation is provided to maintain adequate ventilation and oxygenation [10]. In patients who are hypovolemic, PEEP >10 cm H₂O may reduce CBF. Continuous infusion of sedative and analgesic drugs is beneficial in mechanical ventilated patients for synchronizing of ventilation strategy [11].

- iii. Cardiovascular stabilization: Decreasing mean arterial pressure (MAP) of head trauma patient is strongly associated with poor outcome [12]. Hypovolemia is often masked by a relatively stable blood pressure secondary to either sympathetic hyperactivity or the reflex response to increased ICP. Systemic hypotension due to hypovolemic or cardiogenic shock should be identified and controlled or definitively treated (e.g., by the release of tension pneumothorax). Careful stabilization of the blood pressure (systolic blood pressure should be maintained at or above 90 mm Hg) with fluid resuscitation and a continuous infusion of an inotrope or vasopressor may be necessary [13].
- iv. Disability: If the condition of the patient is stable, neurologic disability should be performed before giving sedative or neuromuscular blocking agents. Pupillary response and the presence of lateralizing signs and spinal motor and sensory levels are carefully noted.
- v. Exposure: Unless the patient is hypothermic, the patient is fully undressed and examined for any other associated injuries.

3.2.2 Secondary survey

Thorough history taking and physical examination, laboratory testing such as metabolic panel, complete blood count, prothrombin time (PT) and partial thromboplastin time (PTT), urinalysis, ethanol level, urine drug screen, and blood type and screen, radiological examination of the whole body should be carried out in secondary survey.

4. Monitoring of patients with traumatic brain injury

4.1 Intracranial pressure (ICP) monitoring

In clinical practice, invasive and non-invasive methods of ICP monitoring are used aiming to determine the optimal cerebral perfusion pressure (CPP). Monitoring of ICP is useful, not only as a guide to therapy, but also for assessing the response to the therapy and determining the prognosis.

Brain Trauma Foundation Guidelines lists the following indications [14]:

- 1. Moderate to severe head injury patient with normal CT scan
- 2. Two or more following features are noted in admission:
 - Age > 40 years,
 - BP < 90 mmHg and
 - Unilateral or bilateral motor posturing

4.1.1 Invasive ICP monitoring

Today, the intraventricular catheter remains the gold standard for ICP monitoring, as it measures global ICP [14]. Moreover, the intraparenchymal catheters used for ICP monitoring have integrated as a CSF drainage catheter and catheters that detect parameters, such as brain tissue O_2 partial pressure ($PbtO_2$) and cerebral blood flow (CBF).

4.1.2 Non-invasive ICP monitoring

A non-invasive ICP monitor should be readily available throughout the hospital, be inexpensive, accurate and convenient to use.

- i. Brain computed tomography (CT): This is the fastest and the most cost-effective method to evaluate raised ICP and associated pathology. A non-contrast CT head can be ruled out the presence of mass lesions, intracranial bleeding or hydrocephalus, as a cause of intracranial hypertension. Findings suggestive of a raised ICP include cerebral edema, midline shift, compression of basal cisterns and changes in gray-white differentiation [15].
- ii. Brain magnetic resonance imaging (MRI): This imaging is costly and time consuming so that it is not first line investigation in acute care setting. MRI can evaluate in detail of soft tissue and cerebral parenchymal lesions, which are not detected on CT scan, e.g. diffuse axonal injury [16].
- iii. Transcranial Doppler (TCD) Ultrasonography: TCD can monitor the velocity of blood flow in cerebral arteries indirectly. It is easy to use and can be measured as a bedside procedure. The most commonly used artery is middle cerebral artery (MCA). The flow velocity of the blood causes a phase shift in the specific sound wave frequency emitted and recorded by the probe, whereas the wave frequency is either increased or decreased in correlation with the speed of the blood. The blood flow volume can be determined if the diameter of the vessel is known. Showing of reduced flow volume indicates impairment to cerebral blood flow and indirectly increased ICP. But the accuracy of the technique depends on the experience of the operator [17].
- iv. Optic Nerve Sheath Diameter (ONSD): The space between the optic nerve and its sheath is filled with CSF and its pressure equals ICP [18]. Optic nerve sheath diameter can be measured by using a transocular ultrasound in brain trauma patients. The studies proved that ONSD $>5-6$ mm corresponds to ICP ≥ 20 mmHg [19]. Limitations of its use are patients with chronic ocular disease and malignant hypertension [19]. The ONSD measurement technique is cheap, efficient and non-time consuming, but operator dependent.
- v. Tympanic membrane displacement (TMD): It measures transmission ICP to perilymphatic space by the use of communication between subarachnoid space and inner ear through the cochlear aqueduct. An increase in ICP is directly transmitted to the footplate of the stapes, displacing the tympanic membrane. Inwards displacement indicates increased ICP, and outwards normal or low ICP [20]. Nevertheless, this practice is lack of accuracy and has to be reconsidered in clinical practice as in quantitative assessment.

4.1.3 Additional tools in ICP monitoring

- i. Brain tissue O₂ partial pressure (PbtO₂): Measurement of PbtO₂ is invasive means of monitoring regional cerebral oxygen tension by inserting a micro-catheter in the white matter [21]. The method can only measure approximately 15 mm² of brain tissue around the tip. Normal baseline PbtO₂ values range from 25 to 35 mmHg. Current guidelines consider PbtO₂ of less than 20 mmHg as threshold to consider intervention [22]. It can be useful in multimodal monitoring in neurocritical care as conjunction with ICP monitor.
- ii. Jugular bulb saturation (SjvO₂): Measurement of SjvO₂ by inserting a catheter placed in the jugular bulb can provide information about cerebral oxygen extraction and adequacy of global cerebral blood flow [17]. SjvO₂ distinguishes deficient oxygen supply due to reduced cerebral perfusion (SjvO₂ < 50%) from hyperemia (SjvO₂ > 80%) because of reduced cerebral oxygen consumption. Increased ICP is mainly associated with reduced SjvO₂. It is more difficult to use and less reliable than PbtO₂ monitoring [22].
- iii. Cerebral microdialysis: Cerebral microdialysis allows bedside monitoring to detect cerebral hypoxemia on a cellular level. The method measures glucose, glutamate, lactate, pyruvate, and glycerol concentrations. An increased in lactate/pyruvate ratio and decreased in brain glucose level is associated with poor outcome after TBI [17]. Microdialysis cannot be used extensively due to its time-consuming maintenance and additional costs [22].
- iv. Near infrared spectroscopy (NIRS): NIRS is a noninvasive tool to measure cerebral oxygenation by detecting oxygenated to deoxygenated hemoglobin concentration [21]. There are difficulty to use if there is a scalp swelling [16]. That why the use of NIRS is limited in clinical practice.
- v. Continuous electroencephalography (cEEG): The use of cEEG is indicated in detection of convulsive and non-convulsive seizures [23]. Focal slowing of underlying rhythms or global EEG suppression or flat EEG patterns provide information of intracranial hypertension. [16]. cEEG can help predict outcome and titrate treatments throughout giving barbiturates [24].

4.2 Others standard monitoring

Baseline monitoring should include electrocardiography, pulse oximetry, capnography and urine output. Invasive hemodynamic monitorings like invasive arterial pressure measurement and central venous pressure is essential in TBI patients [25]. Some of hemodynamic unstable patients need pulmonary artery catheter placement.

Invasive arterial pressure monitoring permits assessment of beat-to-beat variation in blood pressure and regular arterial blood-gas sampling. Central venous pressure monitoring helps optimization of fluid balance and giving vasoactive drugs and parenteral nutrition. Insertion of a pulmonary artery catheter allows the accurate measurement of pulmonary vascular pressure and calculation of cardiac output. Blood glucose, electrolytes, hematocrit, serum osmolality and coagulation should be monitored periodically [25].

Insertion of an indwelling urinary catheter facilitates measurement of urinary volume and composition of urine. It helps diagnosis of conditions of altered urinary output associated with TBI such as diabetes insipidus (DI), the syndrome

of inappropriate antidiuretic hormone (SIADH) secretion, cerebral salt wasting syndrome and the hyperosmolar state [26].

5. General critical measures

Multiple treatment options exist to treat acute intracranial hypertension. The goal of these therapies is to control ICP to less than 20 mmHg [27] and improving parameters. The most recent TBI guidelines from the Brain Trauma Foundation (BTF) suggest that the ICP goal should be less than 22 mmHg [13].

5.1 Intubation and mechanical ventilation

Early and rapid intubation and mechanical ventilation have to be practiced in moderate to severe head trauma patients [5]. During intubation, adequate depth of sedation and elimination of reflexes such as cough and vomiting should be achieved. Mechanical ventilation should aim at avoiding hypoxemia, hypercapnia and hypocapnia [5]. The usual PCO_2 should be kept at values between 35 and 40 mmHg [27]. Generally positive end expiratory pressure (PEEP) can increase intrathoracic pressure and decrease cerebral venous drainage from superior vena cava [28]. PEEP >15 cmH₂O can be applied safely in patients with acute brain injury as it does not have a clinically significant effect on ICP or CPP [10].

5.2 Blood pressure (BP): CPP optimization

Cerebral perfusion pressure (CPP) is key component in management of traumatic brain injury. Cerebral perfusion pressure is defined as mean arterial pressure (MAP) minus intracranial pressure (i.e., $CPP = MAP - ICP$) [29]. The recommended goal of CPP per BTF guideline is 50–70 mm Hg [30]. CPP less than normal limit may result in ischemic brain injury [30]. CPP directed therapy is based on theoretical aids that maintaining optimal cerebral blood flow is necessary to meet the metabolic needs of the injured brain [31]. The “Lund therapy” is a therapeutic approach that focuses on the reduction of ICP by decreasing intracranial volumes [32].

Brain trauma foundation guidelines suggest that SBP ≥ 100 mmHg should be maintained for patients 50 to 69 years old or ≥ 110 mmHg for patients 15 to 49 years or > 70 years old to decrease mortality and improve outcomes [13].

Improving outcome of high-risk surgical patients depend on optimizing cardiac output and oxygen delivery guided by goal-directed fluid therapy (GDT) [33]. Crystalloids, colloids and blood components are used for fluid resuscitation and conserving cardiovascular stability to ensure adequate tissue perfusion. Fluid resuscitation should be guided not only by blood pressure but also by urinary output and central venous pressure (CVP) [34]. Hypotension may worsen neurologic outcome [35].

0.9% normal saline remains widely used as a resuscitation fluid and remains the fluid of choice for patients with brain injury [36]. Lactated Ringer's solution is slightly hypotonic relative to plasma. Osmolarity should be frequently checked if large amount of lactate ringer solution is used [37]. Hypoosmolar solutions like 5% dextrose in water increase brain water content and consequently increase ICP. Glucose containing solutions are avoided because hyperglycemia is associated with worsened neurologic outcomes [38].

Large volumes (>500 mL) of 6% hetastarch should not be used because they may cause coagulopathy [39]. Patients who have hemoglobin (Hb) (7-8 mg/dl) may require blood and blood products transfusion to optimize oxygen delivery [40]. By reducing oxygen delivery, anemia may aggravate secondary insult of traumatic brain injury [41].

If the blood pressure and cardiac output cannot be restored through fluid resuscitation, continuous administration of inotropic and vasopressor drugs is necessary. An infusion of either phenylephrine or dopamine is recommended to maintain cerebral perfusion pressure (CPP) [13, 15].

5.3 Body positioning

Elevation of head position 20–30 degree may be helpful in managing ICP [42]. Preventing excessive flexion or rotation of the neck, avoiding restrictive neck taping, and minimizing stimuli that could induce cough and Valsalva responses and uses of lignocaine during endotracheal suctioning are important in management of intracranial hypertension [43].

5.4 Temperature control

It has been shown that patients who develop a body temperature $> 37.5^{\circ}\text{C}$ within the first 72 hours, have significantly worse outcomes determined as Glasgow outcome scale (GOS) 1 or 2 [5, 16, 44]. These include intravenous and enteral antipyretic medications, control of room temperature, and cooling blankets or pads [16].

Although hypothermia (32 to 34°C) decreases cerebral metabolism and may reduce CBF and ICP [19], therapeutic hypothermia does not improve long-term outcome [13, 45]. Serious adverse effects such as hypokalemia, atrial and ventricular arrhythmias, hypotension and coagulopathy may be associated with hypothermia [16].

5.5 Glycemic control

Hyperglycemia is associated with increased mortality in patients with TBI [46]. Target glycemic control between 4.4 to 6.7 mmol/L have been shown shortened hospital stay and improve outcome [47]. Hyperglycemia (> 11.1 mmol/L) is associated with 3.6 fold increased risk of mortality [48].

5.6 Seizure prophylaxis

Post-traumatic seizure (PTS) is a long-recognized and debilitating complication after traumatic brain injury [49, 50]. PTS are classified into immediate PTS (occurring within 24 hours of injury), early PTS (occurring within 7 days after injury), and late PTS (occurring after 7 days of post injury) [51]. Seizures can exacerbate intracranial hypertension by increasing cerebral blood flow corresponding with the need of brain oxygen and glucose [52, 53]. Continuous video recording of Encephalography (EEG) can be used as a diagnosis tools for PTS after TBI [54].

Seizure prophylaxis is recommended during the first week after TBI, particularly in high-risk patients such as those who have GCS scores <10 ; cortical contusion; depressed skull fracture; subdural, epidural, or intracerebral hematoma; penetrating head trauma; or seizures occurring within the first 24 hours after injury [5, 55]. The Brain Trauma Foundation Guidelines recommended the use of phenytoin in early PTS [16, 56, 57].

5.7 Hyperventilation

Hyperventilation is an effective and rapid method of treating intracranial hypertension. In the setting of intracranial hypertension, the goal of PaCO_2 should

be lower to 30 mmHg or 25–30 mmHg in extreme cases [13]. Reduction of PCO_2 acutely induces vasoconstriction of cerebral arterioles and a decrease in cerebral blood volume, resulting in ICP reduction [19]. The effect supports within 30 minutes after hyperventilation, but generally lasts less than 24 h, due to buffering capacity of CSF compensations [16].

Both global and regional CBF are markedly decreased within 24 to 48 hours after head trauma [58]. Reduction of CBF in early phase of post injury is significantly associated with poor prognosis. Therefore, hyperventilation may have a role as a temporizing measure for the reduction of elevated ICP [59]. Meanwhile, SjvO_2 or PbtO_2 measurements can be used to monitor oxygen delivery [45, 59]. Hyperventilation should not be abruptly discontinued but should be tapered slowly over 4–6 h to avoid vasodilatation of cerebral arterioles and rebound increases in ICP [60].

5.8 Hyperosmolar therapy

Hyperosmolar therapy has critical role of medical treatment in acute intracranial hypertension by reducing brain volume. The most commonly used medications are mannitol and hypertonic saline (HS) [45].

Mannitol increase serum osmolality, resulting in an osmotic gradient from interstitial to intravascular space, reduction of cerebral edema and ICP. Mannitol also acts by other mechanisms, such as induction of reflex cerebral arteriolar vasoconstriction, improvement in blood rheology, reduction of CSF formation, and free radicals scavenging. The recommended ICP lowering dose of 20% mannitol is 0.25 to 1 g/kg every 6 hours [61]. Adverse effects of mannitol include acute renal failure, electrolyte disturbances and rebound of existing cerebral edema [62, 63].

Hypertonic saline (HS) is used alternatively to Mannitol and induce induction of reflex cerebral arteriolar vasoconstriction, improved deformability of erythrocytes with enhanced microcirculation, and an anti-inflammatory effect due to reduced adhesion of polymononuclear cells in the cerebral microvasculature [16]. Bolus and repeated doses are required until serum sodium level have been raised above normal (145–155 mEq/L) [64]. Possible adverse effects of HS include rebound cerebral edema, electrolyte disturbances (hypokalemia), congestive heart failure, renal failure, hyperchloremic metabolic acidosis, phlebitis, transient hypotension, hemolysis, osmotic demyelination, subarachnoid bleeding, seizures and muscle twitching [65].

5.9 Sedation and analgesia

Sedation and analgesia are an integral part of medical treatment. Patient-ventilator dyssynchrony and agitation increase intrathoracic pressure, which increase CBV and consequently increase ICP [27]. Ideal sedative drugs should have rapid onset and recovery for a quick neurological assessment, a predictable clearance independent of end organ failure and reducing cerebral blood flow and cerebral metabolic rate of oxygen consumption [11].

Opioids, benzodiazepines, propofol, barbituates and dexmedetomidine can be used to provide the sedation goal. The preferred regime is combination of an opioids such as fentanyl (1–3 $\mu\text{g/kg/hr}$) or sufentanil (0.1–0.6 $\mu\text{g/kg/hr}$) to provide analgesia and propofol (0.3–3 mg/kg/hr) for sedation [60]. According to the BTF guidelines, the administration of barbiturates is generally reserved for intracranial hypertension, refractory to maximum standard medical and surgical treatment [57].

Combination of nondepolarizing muscle relaxants and sedatives may be used during posturing, coughing, or agitation in head trauma care. When a neuromuscular blockade is used, EEG should be monitored to rule out convulsive states [66].

5.10 Corticosteroids

Recently, the BTF guidelines do not recommend the use of steroids for improving outcome or reducing ICP in TBI patients [59] because steroids are not effective in cytotoxic edema [60]. Steroid use is only indicated for reducing ICP in abscesses or neoplasms associated with vasogenic edema [60].

5.11 Decompressive craniectomy (DC)

Surgical removal of skull bone in effected side followed by evacuation of hematoma is considered if the patient deteriorating or ICP continues to rise [19]. Prompt removal of an acute subdural, epidural, or large solitary intracerebral hematoma is useful measure in traumatic brain injury treatment [67]. Decompressive craniectomy is risky and adverse effects are common. The complications are weighted against the life-threatening circumstances under which surgery is performed [68].

5.12 Cerebrospinal fluid (CSF) drainage

Procedure of CSF removal via external ventricular drainage device, lumbar drain or serial lumbar puncture is simple and tends to reduce intracranial pressure [16, 19]. Use of CSF drainage during the first 12 hours after injury may be considered in patients with an initial GCS <6 [57]. The major risks of EVD placement and CSF drainage include infection, hemorrhage and herniation [69].

5.13 Other measures

Nutritional support is required to facilitate recovery and should be initiated seventh day of post injury is recommended to improve outcome [57]. Recent guidelines suggest that transgastric jejunal feeding is recommended to reduce the incidence of ventilator associated pneumonia [59].

Proton-pump inhibitors, pantoprazole, 40 mg daily is suggested for stress ulcer prophylaxis in critical care settings [70].

Supporting with pneumatic compression devices and use of LMWH or low-dose unfractionated heparin should be initiated as soon as possible for prophylaxis against deep venous thrombosis (DVT) [57]. The benefit of using heparin is considered to outweigh the risk of intracranial hemorrhage.

Author details

Soe Wunna Htay

Department of Anesthesiology and Critical Care, May Myo Military Hospital,
Mandalay, Myanmar

*Address all correspondence to: swunnah@gmail.com

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References

- [1] Stelmasiak, Z., A. Dudkowska-Konopa, and K. Rejdak, Head trauma and neuroprotection. *Medical Science Monitor*, 2000. 6(2): p. RA426-RA432.
- [2] Liew, B., et al., Early management of head injury in adults in primary care. *Malaysian family physician: the official journal of the Academy of Family Physicians of Malaysia*, 2017. 12(1): p. 22.
- [3] Mena, J.H., et al., Effect of the modified Glasgow Coma Scale score criteria for mild traumatic brain injury on mortality prediction: comparing classic and modified Glasgow Coma Scale score model scores of 13. *The Journal of trauma*, 2011. 71(5): p. 1185.
- [4] Aitkenhead, A.R., et al., Smith and Aitkenhead's Textbook of Anaesthesia E-Book: Expert Consult-Online & Print. *Neurosurgical Anesthesia*. Vol. Sixth Edition. 2013: Elsevier Health Sciences. pp 654-656.
- [5] Cottrell, J.E. and W.L. Young, Cottrell and Young's neuroanesthesia. *Care of Acutely Unstable Patient*. Vol. Fifth Edition. 2016: Elsevier Health Sciences. 161-169.
- [6] Lev, R. and P. Rosen, Prophylactic lidocaine use preintubation: a review. *The Journal of emergency medicine*, 1994. 12(4): p. 499-506.
- [7] Turner, B.K., et al., Neuroprotective effects of thiopental, propofol, and etomidate. *AANA journal*, 2005. 73(4): p. 297.
- [8] Muñoz-Martínez, T., et al., Contraindications to succinylcholine in the intensive care unit. A prevalence study. *Medicina intensiva*, 2014. 39(2): p. 90-96.
- [9] Tran, D., et al., Rocuronium vs. succinylcholine for rapid sequence intubation: a Cochrane systematic review. *Anaesthesia*, 2017. 72(6): p. 765-777.
- [10] Videtta, W., et al., Effects of positive end-expiratory pressure on intracranial pressure and cerebral perfusion pressure, in *Intracranial Pressure and Brain Biochemical Monitoring*. 2002, Springer. p. 93-97.
- [11] Peluso, L., B.M. Lopez, and R. Badenes, Sedation in TBI Patients, in *Traumatic Brain Injury-Neurobiology, Diagnosis and Treatment*. 2019, IntechOpen.
- [12] Hutchison, J.S., et al., Impact of hypotension and low cerebral perfusion pressure on outcomes in children treated with hypothermia therapy following severe traumatic brain injury: a post hoc analysis of the Hypothermia Pediatric Head Injury Trial. *Developmental neuroscience*, 2010. 32(5-6): p. 406-412.
- [13] Ragland, J. and K. Lee, Critical care management and monitoring of intracranial pressure. *Journal of Neurocritical Care*, 2016. 9(2): p. 105-112.
- [14] Nag, D.S., et al., Intracranial pressure monitoring: Gold standard and recent innovations. *World journal of clinical cases*, 2019. 7(13): p. 1535.
- [15] Abraham, M. and V. Singhal, Intracranial pressure monitoring. *Journal of Neuroanaesthesiology and Critical Care*, 2015. 2(03): p. 193-203.
- [16] Schizodimos, T., et al., An overview of management of intracranial hypertension in the intensive care unit. *Journal of Anesthesia*, 2020. 34: p. 741-757.

- [17] Ristic, A., R. Sutter, and L.A. Steiner, Current neuromonitoring techniques in critical care. *Journal of Neuroanaesthesiology and Critical Care*, 2015. 2(02): p. 097-103.
- [18] Rajajee, V., et al., Optic nerve ultrasound for the detection of raised intracranial pressure. *Neurocritical care*, 2011. 15(3): p. 506-515.
- [19] Sadoughi, A., I. Rybinnik, and R. Cohen, Measurement and management of increased intracranial pressure. *The Open Critical Care Medicine Journal*, 2013. 6(1).
- [20] Gwer, S., et al., The tympanic membrane displacement analyser for monitoring intracranial pressure in children. *Child's Nervous System*, 2013. 29(6): p. 927-933.
- [21] Tasneem, N., et al., Brain multimodality monitoring: a new tool in neurocritical care of comatose patients. *Critical care research and practice*, 2017. 2017.
- [22] Le Roux, P., et al., Consensus summary statement of the international multidisciplinary consensus conference on multimodality monitoring in neurocritical care. *Neurocritical care*, 2014. 21(2): p. 1-26.
- [23] Kurtz, P., K.A. Hanafy, and J. Claassen, Continuous EEG monitoring: is it ready for prime time? *Current opinion in critical care*, 2009. 15(2): p. 99-109.
- [24] Peacock, S.H. and A.D. Tomlinson, Multimodal neuromonitoring in neurocritical care. *AACN advanced critical care*, 2018. 29(2): p. 183-194.
- [25] Frost, E.A., Essentials of Neuroanesthesia and Neurointensive Care. *Anesthesia and Analgesia*, 2009. 108(2): p. 678-679.
- [26] Capatina, C., et al., Diabetes insipidus after traumatic brain injury. *Journal of clinical medicine*, 2015. 4(7): p. 1448-1462.
- [27] Godoy, D.A., S. Lubillo, and A.A. Rabinstein, Pathophysiology and management of intracranial hypertension and tissular brain hypoxia after severe traumatic brain injury: an integrative approach. *Neurosurgery Clinics*, 2018. 29(2): p. 195-212.
- [28] Frost, E.A., Effects of positive end-expiratory pressure on intracranial pressure and compliance in brain-injured patients. *Journal of neurosurgery*, 1977. 47(2): p. 195-200.
- [29] Robertson, C.S., Management of cerebral perfusion pressure after traumatic brain injury. *The Journal of the American Society of Anesthesiologists*, 2001. 95(6): p. 1513-1517.
- [30] Smith, M., Cerebral perfusion pressure, in *BJA: British Journal of Anaesthesia*. 2015, Oxford University Press: <https://doi.org/10.1093/bja/aev230> London. p. Pages 488-490.
- [31] Prabhakar, H., et al., Current concepts of optimal cerebral perfusion pressure in traumatic brain injury. *Journal of anaesthesiology, clinical pharmacology*, 2014. 30(3): p. 318.
- [32] Nordström, C.-H., Physiological and biochemical principles underlying volume-targeted therapy—the “Lund concept”. *Neurocritical care*, 2005. 2(1): p. 83-95.
- [33] Alvis-Miranda, H.R., S.M. Castellar-Leones, and L.R. Moscote-Salazar, Intravenous fluid therapy in traumatic brain injury and decompressive craniectomy. *Bulletin of Emergency and Trauma*, 2014. 2(1): p. 3.

- [34] De Backer, D. and J.-L. Vincent, Should we measure the central venous pressure to guide fluid management? Ten answers to 10 questions. *Critical Care*, 2018. 22(1): p. 1-6.
- [35] Zornow, M.H. and D.S. Prough, Fluid management in patients with traumatic brain injury. *New Horiz*, 1995. 3(3): p. 488-98.
- [36] Alvis-Miranda, H.R., et al., Fluid therapy in neurotrauma: basic and clinical concepts. *Reviews in Health Care*, 2014. 5(1): p. 7-22.
- [37] Thompson, M., et al., Comparison of crystalloid resuscitation fluids for treatment of acute brain injury: a clinical and pre-clinical systematic review and network meta-analysis protocol. *Systematic reviews*, 2018. 7(1): p. 1-7.
- [38] Zornow, M. and D. Prough, Fluid management in patients with traumatic brain injury. *New horizons (Baltimore, Md.)*, 1995. 3(3): p. 488-498.
- [39] Kozek-Langenecker, S.A., Effects of hydroxyethyl starch solutions on hemostasis. *The Journal of the American Society of Anesthesiologists*, 2005. 103(3): p. 654-660.
- [40] Badenes, R., et al., Hemoglobin concentrations and RBC transfusion thresholds in patients with acute brain injury: an international survey. *Critical care*, 2017. 21(1): p. 1-10.
- [41] Lelubre, C., et al., Anemia management after acute brain injury. *Critical care*, 2016. 20(1): p. 1-11.
- [42] Schulz-Stübner, S. and R. Thiex, Raising the head-of-bed by 30 degrees reduces ICP and improves CPP without compromising cardiac output in euvoletic patients with traumatic brain injury and subarachnoid haemorrhage: a practice audit. *European Journal of Anaesthesiology (EJA)*, 2006. 23(2): p. 177-180.
- [43] Gholamzadeh, S., et al., Examination of the effect of lidocaine on intracranial pressure during endotracheal suctioning in severe head-injured patients in Shiraz-Iran: P 089. *European Journal of Anaesthesiology| EJA*, 2008. 25: p. 31.
- [44] Greer, D.M., et al., Impact of fever on outcome in patients with stroke and neurologic injury: a comprehensive meta-analysis. *Stroke*, 2008. 39(11): p. 3029-3035.
- [45] Stocchetti, N. and A.I. Maas, Traumatic intracranial hypertension. *New England Journal of Medicine*, 2014. 370(22): p. 2121-2130.
- [46] Shi, J., et al., Traumatic brain injury and hyperglycemia, a potentially modifiable risk factor. *Oncotarget*, 2016. 7(43): p. 71052.
- [47] Hermanides, J., et al., Glycaemic control targets after traumatic brain injury: a systematic review and meta-analysis. *Critical Care*, 2018. 22(1): p. 1-11.
- [48] Griesdale, D.E., et al., Glucose control and mortality in patients with severe traumatic brain injury. *Neurocritical care*, 2009. 11(3): p. 311.
- [49] Evans, R.W. and S.C. Schachter, Post-traumatic seizures and epilepsy. *UptoDate*. Available from: <https://www.uptodate.com/contents/post-traumatic-seizures-and-epilepsy>, 2014.
- [50] Pingue, V., C. Mele, and A. Nardone, Post-traumatic seizures and antiepileptic therapy as predictors of the functional outcome in patients with traumatic brain injury. *Scientific reports*, 2021. 11(1): p. 1-12.
- [51] Nichol, H., J. Boyd, and J. Trier, Seizure Prophylaxis Following Moderate

to Severe Traumatic Brain Injury: Retrospective Investigation of Clinical Practice and the Impact of Clinical Guidelines. *Cureus*, 2020. 12(4).

[52] Marguc, K., et al., Measurements of CBF in Patients with Epilepsy, in *Cerebral Blood Flow and Metabolism Measurement*. 1985, Springer. p. 202-204.

[53] Posner, J.B., F. Plum, and A. Van Poznak, Cerebral metabolism during electrically induced seizures in man. *Archives of Neurology*, 1969. 20(4): p. 388-395.

[54] Piccenna, L., G. Shears, and T.J. O'Brien, Management of post-traumatic epilepsy: An evidence review over the last 5 years and future directions. *Epilepsia open*, 2017. 2(2): p. 123-144.

[55] Ding, K., P.K. Gupta, and R. Diaz-Arrastia, Epilepsy after traumatic brain injury. *Translational research in traumatic brain injury*, 2016.

[56] Fordington, S. and M. Manford, A review of seizures and epilepsy following traumatic brain injury. *Journal of neurology*, 2020. 267: p. 3105-3111.

[57] Carney, N., et al., Guidelines for the management of severe traumatic brain injury. *Neurosurgery*, 2017. 80(1): p. 6-15.

[58] Stocchetti, N., et al., Hyperventilation in head injury. *Chest*, 2005. 127(5): p. 1812-1827.

[59] Bullock, M. and J. Povlishock, Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, AANS/CNS Joint Section on Neurotrauma and Critical Care. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma*, 2007. 24(suppl 1): p. S1-106.

[60] Mayer, S.A. and J.Y. Chong, Critical care management of increased intracranial pressure. *Journal of Intensive Care Medicine*, 2002. 17(2): p. 55-67.

[61] Shawkat, H., M.-M. Westwood, and A. Mortimer, Mannitol: a review of its clinical uses. *Continuing education in anaesthesia, critical care and pain*, 2012. 12(2): p. 82-85.

[62] Shi, J., et al., Hypertonic saline and mannitol in patients with traumatic brain injury: A systematic and meta-analysis. *Medicine*, 2020. 99(35).

[63] Schwimmbeck, F., et al., Hypertonic saline versus mannitol for traumatic brain injury: a systematic review and meta-analysis with trial sequential analysis. *Journal of neurosurgical anesthesiology*, 2021. 33(1): p. 10-20.

[64] Ennis, K.M. and G.M. Brophy, Management of intracranial hypertension: Focus on pharmacologic strategies. *AACN advanced critical care*, 2011. 22(3): p. 177-182.

[65] Georgiadis, A.L. and J.I. Suarez, Hypertonic saline for cerebral edema. *Current neurology and neuroscience reports*, 2003. 3(6): p. 524-530.

[66] Shapiro, H.M., Intracranial hypertension: therapeutic and anesthetic considerations. *The Journal of the American Society of Anesthesiologists*, 1975. 43(4): p. 445-471.

[67] Nakagawa, K. and W.S. Smith, Evaluation and management of increased intracranial pressure. *CONTINUUM: Lifelong Learning in Neurology*, 2011. 17(5): p. 1077-1093.

[68] Stiver, S.I., Complications of decompressive craniectomy for traumatic brain injury. *Neurosurgical focus*, 2009. 26(6): p. E7.

[69] Grady, M.S., Lumbar drainage for increased intracranial pressure. *Journal of neurosurgery*, 2009. 110(6): p. 1198-1199.

[70] Ye, Z., et al., Gastrointestinal bleeding prophylaxis for critically ill patients: a clinical practice guideline. *Bmj*, 2020. 368.