Mediterranean Journal of Emergency Medicine
https://mjemonline.com/

MJEM
Mediterranean Journal of Emergency Medicine

Severe traumatic brain injury

Citation: Ismail N. Severe traumatic brain injury. Med Emergency, MJEM 2020; 28:33-42.
DOI: 10.26738/MJEM.2017/MJEM28.2020/NI.RAE.100719
Key words: brain trauma, Glasgow Coma Scale, TBI, traumatic brain injury

ABSTRACT

Traumatic brain injury (TBI) is a worldwide major cause of morbidity and mortality particularly in the vulnerable population young males, low-income individuals and members of ethnic minority groups.

Severe traumatic brain injury, defined as head trauma associated with a Glasgow Coma Scale (GCS) score of 3 to 8 with loss of consciousness duration and altered mental status greater than 24 hours and post traumatic amnesia more than seven days.

In this resume of protocols article, a helpful review of the current status of management of severe TBI according to the recent up-dated brain trauma foundation 2016 and The National Institute for Health and Care Excellence (NICE) 2014 guidelines is present. A concise overview of the optimal medical management, and both the non-invasive and invasive monitoring strategies, as well as the indications of surgical interventions necessary in particular instances. It is important not only for trauma team but for all healthcare personnel to be aware of the management and prevention of complications of severe traumatic brain injury.

Authors' affiliation:

Corresponding author: Nora Ismail, MD
Critical Care department, Faculty of medicine, Cairo University,
Kasr el Ainy hospital Al-Saray Street, El Manial Cairo, Egypt
E-mail: Narnar_77@yahoo.com

Article history / info:

Category: Review article
Received: May 8, 2019
Revised: June 19, 2019
Accepted: July 10, 2019

Conflict of interest statement:

There is no conflict of interest to declare

INTRODUCTION

Traumatic brain injuries (TBIs) can affect people of all ages and are a serious explanation for death and disability, with an incidence of around ten million people worldwide [1]. TBI is that the major and most significant explanation for one-third to one-half of trauma-related deaths, and also the leading reason behind disability under forty years-old (15-20/100,000 populations per year) [2-4]. Men are twice more likely to sustain TBI than women [5]. In this resume of protocols, a helpful summary of the present status of management of severe TBI consistent with the recent updated guidelines is presented. A concise overview of the optimal medical management, both the non-invasive and invasive monitoring strategies, also because the indications of surgical interventions necessary particularly instances is provided it's important not just for trauma team except for all healthcare professionals to bear in mind of the management and prevention of complications of severe traumatic brain injury.

METHODS

A review of literature was conducted using internet search engines on Pubmed® mainly additionally to Google Scholars® involving all systematic reviews and guidelines for severe traumatic brain injury management till 2017. A selected highlight was given to reviews that symbolize powerful information sources for practitioners searching for state-of-the art evidence to guide their decision-making and work practices. All TBI clinical practice guidelines published within the last decade were selected if their scope included management of severe TBI, systematic methods for evidence search and clear defined recommendations.

HIGH-RISK POPULATIONS

High risk populations at increased risk of traumatic brain injury include young males, low-income individuals, unmarried individuals, members of ethnos groups, residents of inner cities, individuals with a history of drug abuse, individuals who have suffered a previous TBI [6;7]. TBI defined as head trauma related to a Glasgow Coma Scale (GCS) score of three to eight with loss of consciousness duration and altered mental status greater than 24 hours and post traumatic amnesia quite seven days [8]. Severe TBI is a very important major challenge in medical specialty and demanding care medicine. The economic, social and psychological impact is important, thanks to the direct and indirect expenses of acute treatment, rehabilitation and permanent sequelae during which patients are victims. The sequels are after all much devastating in middle and low income countries [1]. Severe TBI patients form 10% of all civilian TBI. Severe TBI patients have the best incidence of intracranial mass lesions, and need intense medical and sometimes surgical intervention. Approximately, 25% of severe TBI patients will have another organ injury. Permanent disability is assumed to occur in 10% of mild injuries, 66% of moderate injuries, and 100% of severe injuries [9;10]. Severe-TBI fatality rates after admission to hospital are 25-40% [11;12]. Updated management Guidelines, Scores and reported biomarkers are also mentioned.

MANAGEMENT STEP I

Advanced Trauma Life Support (ATLS) guidelines and International Trauma Life Support (ITLS) guidelines [13].

AIRWAY

Examine for agitation and cyanosis, which suggest hypoxia. Auscultate breath sounds bi-laterally. Gurgling and snoring suggest occlusion of the pharynx. Hoarseness could also be induced by laryngeal obstruction. Securing the airway could be a necessity dictated by conscious level and not only pulmonary function disorder.

A. Chin-lift: Place fingers of 1 hand under the mandible and perform a delicate anterior chin-lift. Depress the lower lip to open the patient's mouth together with your thumb. Avoid hyperextension of the neck.

B. Jaw-thrust: Grasp angles of the mandibular bone, one hand on either side, and displace the mandible forward [14].

C. Suction: Use rigid suction catheter to clear airway.

D. Oropharyngeal airway: Insert into the mouth behind the tongue.

E. Endotracheal intubation: Contraindicated if mid-facial injuries are present. Pre-oxygenate with 100% oxygen. Endotracheal intubation is that the route of choice as nasotracheal intubation may lead to further neurological injury within the face of unknown fracture of the skull base injury.

F. Cricothyroidotomy: A skin incision is made through the cricothyroid membrane. Dilate with a hemostat and insert a small endotracheal tube or tracheostomy tube. Cricothyroidotomy should be avoided in children younger than 12 years.

G. Jet insufflation: Needle cricothyroidotomy involves insertion of a large-caliber plastic cannula, 12 or 14 gauge, into the trachea below the level of the obstruction [14].

PRE-INTUBATION PRECAUTIONS

BREATHING

Administer supplemental oxygen to keep up SpO_2 > 92% and maintain PaO_2 80-120 mmHg. Maintain $PaCO_2$ 35-40 mmHg. Hyperventilation should be avoided during the primary 24 hours after injury when CBF often is reduced critically [16]. Prolonged prophylactic hyperventilation with $PaCO_2$ of < 25 mmHg isn't recommended [16]. Provide judicious analgesia and sedation to manage pain and agitation: a) Fentanyl 25-500 $\mu g.hr^{-1}$ IV infusion. Bolus with dose increases for elevated ICP; Preferentially increase fentanyl (and/or oral opioid therapy) over propofol. Treat significant pain with small doses of intravenous opioids titrated against clinical response and baseline cardio-respiratory measurements [17]. b) Propofol 10-50 $\mu g.kg^{-1}.min^{-1}$ IV infusion. High dose propofol infusions (> 83 $\mu g.kg.min^{-1}$ for greater than 24 hours) are related to fatal acidosis, rhabdomyolysis, and refractory arrhythmias (known as "Propofol infusion syndrome") [18]. In general, doses 50 $\mu g.kg^{-1}.min^{-1}$ are both effective and safe. Short-term infusions of high dose propofol (> 50 $\mu g.kg^{-1}.min^{-1}$) is also deemed necessary to manage ICP after neurosurgical discussion [19]. Despite the utility of propofol for the acute control of elevated ICP, its use didn't show improvement in mortality or six months outcomes [16].

CIRCULATION

Maintaining SBP > 100 mmHg for patients 50 to 69 years old or > 110 mmHg or above for patients 15 to 49 or > 70 years old could also be considered to decrease mortality and improve outcomes [16]. One episode of hypotension with a systolic vital sign not up to 90 mmHg was related to increased morbidity and doubled mortality [16].

Consider central blood pressure monitoring (CVP line or arterial blood vessel catheter); consider insertion of arterial line. Maintain target mean blood pressure (MAP) ≥ 80 mmHg if no ICP monitor is in situ. Ensure adequate volume resuscitation. Aggressively resuscitate shock, and look for underlying causes (head injuries don't usually cause shock except in terminal stages). Ensure hemoglobin > 9 $g.dL^{-1}$ during the patient's acute resuscitation phase. Consider advanced hemodynamic monitoring. Consider adding norepinephrine 0.05 $\mu g.kg^{-1}.min^{-1}$, titrate to stay MAP ≥ 80 mmHg or central perfusion pressure (CPP) ≥ 60 mmHg. Maintain euvolemia (fluid balance positive by 500-1000 mL in first 24 hours). After establishing IV access: complete blood count, complete metabolic profile, cross-match for blood, fresh frozen plasma, platelets and coagulation studies, blood gas, and toxicology [14;20]. Six units of red cells should be cross-matched urgently. Insert a urinary catheter for adequate urine output monitoring unless a ruptured urethra is suspected (blood at the urinary meatus, severe fractured pelvis) during which case a supra-pubic catheter is indicated.

DISABILITY

The most accurate assessment is full clinical neurological assessment within the absence of medication or sedatives. Lateralizing which include extremity weakness, change in sensation or pupillary changes. Pupillary function is vital for higher cognitive process and prognosticating. It's important to notice the dimensions, shape, reactivity, and symmetry. Pupillary asymmetry of but one mm is normal and has no pathological significance. Hypoxemia, hypotension, hypothermia and medicines can even produce dilated pupils reinforcing the actual fact that it's necessary to resuscitate the patient before assessing papillary function. Examine skull for clinically detectable depressed skull fracture, battle's sign (ecchymosis over mastoid process), raccoons eye (periorbital ecchymosis), and body fluid rhinorrhea or otorrhea [14]. For unconscious patients whole spine X-ray or X-raying (CT-scan) should be made. Severe TBI is related to cervical spine fracture in 22% of cases [21]. NB: a standard neurologic examination doesn't rule out a C-spine injury [14].

EXPOSURE AND ENVIRONMENT CONTROL

The patient should be completely exposed in preparation for the detailed secondary survey. Following complete removal of the patient's clothes, both the front and back of the patient should be assessed after log rolling. confirm that the patient is roofed adequately after exposure. Check for temperature.

STEP II SECONDARY ASSESSMENT

Injuries are easily missed in emergencies especially if one injury is apparent. A secondary and even tertiary survey should be performed.

STEP III DIFFERENTIAL DIAGNOSIS

If GCS is strictly under height, with or without unequal pupils, with lateralizing deficits, or with open head injuries, the foremost likely cause may be a large intracerebral mass or a diffuse axonal injury [14].

ACUTE EXTRADURAL HEMATOMA (EDH)

The patient presents with loss of consciousness, and as many as 56% present in coma on admission, 47% of surgical EDH regained consciousness, becoming lucid, only to surrender to the rapidly expanding hematoma. Untreated patients typically have deterioration in conscious level frequently with an ipsilateral dilated or dilating pupil, observed in 18-44%, and contralateral hemiparesis or hemiplegia [22].

ACUTE SUBDURAL HEMATOMA (SDH)

Between 37 and 80% of patients with acute SDH present with initial GCS of eight or less. Pupillary abnormalities are observed in up to 50% of patients on admission. An association with other intracranial and extra-cranial injuries is common [23].

TRAUMATIC SUBARACHNOID HEMORHAGE

Traumatic subarachnoid hemorrhage is that the most frequent finding in TBI and occurs in over half severe TBI patients. Mortality is increased twofold within the presence of traumatic subarachnoid hemorrhage. The presence of subarachnoid hemorrhage within the basal cisterns implies an unfavorable outcome of roughly 70% [24;25].

DIFFUSE AXONAL INJIRY (DAI)

The CT-scan findings in isolated DAI are strikingly disproportionate to the neurological condition of the patient, as is additionally frequently seen in hypoxic or anoxic injury. Extension of CT-scan cuts to the cervical spine with sagittal and coronal reconstruction adds little time and may help further guide intervention [25].

INTRACEREBRAL HEMATOMA

Traumatic parenchymal mass lesion occurs in up to 82% of all TBI and 80% of severe TBI [26]. The bulk of small parenchymal lesion doesn't require surgical intervention. Parenchymal lesion tends to evolve within the presence of coagulopathy. Timing of surgery from neurological deterioration greatly affects the result [25].

STEP IV SPECIFIC MANAGEMENT

Maintain head in neutral position to avoid jugular vein constriction. Head elevation of the bed to 30 degrees has rapid effects to cut back cerebral edema and ICP unless contraindicated. Although the mean carotid pressure is reduced during head of bed elevation, ICP is reduced and cerebral blood flow (CBF) is unaffected [27]. In patients with suspected or documented spine injury, this can be best achieved by placing the patient's bed within the reverse Trendelenburg position. Elevation of the top of bed greater than 30 degrees has not been demonstrated to be beneficial [17]. Maintain serum sodium $\geq$ 140 m Eq.L^{-1} with isotonic IV fluids (no dextrose).

Traumatic brain injury patients on antiplatelet

Discontinue antiplatelet agents when traumatic intracranial hemorrhage is suspected. Aspirin therapy testing or reversal isn't necessary. Don't transfuse platelets for patients with antiplatelet-associated intracranial hemorrhage who won't undergo a neurosurgical procedure, irrespective of kind of platelet inhibitor, platelet function assay (PFA) results, hemorrhage volume, or neurologic exam [28]. In TBI patients and known history of ADP-inhibitor antiplatelet therapy (e.g. clopidogrel, prasugrel, ticagrelor, ticlopidine) and who have a planned neurosurgical procedure: check baseline platelet function assay (PFA-clopidogrel) also called P2Y12 assay and if positive: administer one unit of apheresis platelets (one unit = 6-10 pack) IV and transfuse platelets at the time of maximal desired benefit [29-31]. Consider the addition of one of the subsequent options in patients with renal dysfunction (i.e. blood urea nitrogen > 20 and/or serum creatinine > 2) and active bleeding: desmopressin injection

(dDAVP) 0.3 $\mu g.kg^{-1}$ IV in 50 mL normal saline or cryoprecipitate one unit IV STAT [31-33].

Correction of Coagulopathy

Correct coagulopathy with the acceptable reversal agent in life – threatening bleeding – Patient on warfarin and INR > 2: FEIBA (anti-inhibitor coagulant complex) NF 1000 units IV-syringe infusion over 20 minutes [17]. If not available fresh frozen plasma and fat-soluble vitamin 1 to 3 mg slow IV. Regarding new oral anticoagulants (NOACs): patient on factor Xa inhibitors (rivaroxaban): FEIBA 2000 units IV-syringe infusion over 20 minutes. Tranexamic acid 1 g IV over ten minutes - Patient on dabigatran: idarucizumab 2.5 g IV ten minutes, double doses [17]. A recent study concluded that elderly traumatic brain injury patients on direct oral anticoagulants have significantly lower rate and fewer have to reversal agents compared with warfarin [34]. Maintain normothermia (temperature 36-37°C) within six hours. Acetaminophen 500 mg per os (PO) ou per gastric tube (PT) in four hours scheduled if temperature > 37°C [17]. Consider Ibuprofen 800 mg PO/PT in 6 hours (if unable to regulate fever with acetaminophen) taking into consideration increased risk of bleeding. Although a recent study concluded that pre-injury ibuprofen use wasn't related to progression of initial intracranial hemorrhage and therefore the need for neurosurgical intervention. Pre-injury use of Ibuprofen as an variable quantity mustn't warrant the requirement for a routine high resolution CT-scan [35]. Acetaminophen is truly the foremost frequently used antipyretic agent, with ibuprofen getting used much less frequently. Judicious use of those agents within brain trauma patients may have occurred because of potential for paramol toxicity in uncontrolled fever and also the increased bleeding risk of the ibuprofen and other non-steroidal drugs [36;37]. Maintain serum glucose ≥ 70 $mg.dL^{-1}$ and ≥ 180 $mg.dL^{-1}$. Prevent deep phlebothrombosis (DVT). Unless contraindicated, initiate low mass heparin or low dose un-fractionated subcutaneous heparin within 24 hours of injury together with mechanical compression stockings [16]. TBI is that the second highest risk factor for the event of venous thromboembolism (VTE), second only to acute medulla spinalis injury and also the incidence of DVT seven to ten days after TBI is as high as 31.6% even with mechanical prophylaxis. Some protocols recommend weekly routine screening for DVT in severe TBI patients [38]. Initiate gastrointestinal stress ulcer prophylaxis ranitidine or sucralfate. Prevent skin breakdown / ulcer formation through appropriate pressure reduction bed surface.

CEREBRAL PERFUSION THRESHOLD

Cerebral blood flow (CBF) is directly associated with the CPP, which is defined as MAP – ICP and is inversely associated with the cerebral vascular resistance (CVR): CBF = CPP/CVR [39]. Measure CPP at the extent of the tragus. Low CPP, generally defined as but 50 millimeter of mercury, should be prevented (Grade B). Aim is to take care of CPP between 60-70 mmHg. Aggressive maintenance of CPP > 70 mmHg should be avoided because of an increased risk of over-resuscitation and acute respiratory distress syndrome (ARDS) [16]. If CPP < 60 mmHg: Ensure adequate volume resuscitation and consider advanced hemodynamic monitoring. Consider adding norepinephrine 0.05 $\mu g.kg^{-1}.min^{-1}$ and titrate to stay CPP > 60 mmHg.

Intracranial cerebral pressure

Management of severe TBI patients using information from ICP monitoring is usually recommended to scale back in-hospital and period post-injury mortality. ICP should be monitored altogether salvageable patients with a TBI (GCS 3 to 8 after resuscitation) and an abnormal CT-scan. An abnormal CT-scan of the top is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns. ICP monitoring is indicated in patients with severe TBI with a traditional CT-scan if three or above of the subsequent features are noted at admission: age > 40 years, unilateral or bilateral motor posturing (decorticate/decerebrate), or SBP < 90 mmHg [16]. Patients with TBI who won't be examinable for a protracted period of your time. Use of an ICP monitor with external ventricular drainage (EVD) is preferred over an ICP monitor alone. ICP above 22 mmHg is related to increased mortality and warrants treatment [16].

Management of sustained ICP > 22 mmHg for ten minutes

Verify correct ICP waveform on EVD and notify neurosurgery if ICP waveform is wrong or there's no drainage level EVD at the external meatus [17]. Close EVD and level at zero mmHg upon insertion to observe ICP. If ICP > 22 mmHg for ten minutes and EVD clamped – open EVD at zero mmHg for quarter-hour [17]. If EVD is opened quite thrice within 90 minutes, leave EVD open at zero mmHg continuously and consult neurosurgery [17].

Osmolar Therapy

First line therapy for ICP > 22 mmHg for ≥ ten minutes; 7.5% binary compound 250 mL IV bolus over quarter-hour, only once if serum sodium < 160 $mEq.L^{-1}$ [17]. Hypotensive patients with severe TBI may like either 250 mL of seven; 5% as an acute resuscitative intervention to lift MAP, reduce ICP, and avoid crystalloid over-resuscitation. Hypotensive TBI patients resuscitated with hypertonic saline were twice as likely to survive compared with normal saline resuscitation. Hypertonic sodium lactate ends up in improved cognitive function post-TBI in comparison to the employment of hypertonic binary compound solution. Lactate solution is also administered peripherally and it's not related to hyperchloremia [17].

Alternate therapy

Mannitol 0.25 to 1.0 $g.kg^{-1}$ IV-push, only once if serum sodium < 160 $mEq.L^{-1}$ and/or serum osmolality < 320 $mOsm.L^{-1}$ [17]. IC P-lowering effect is dose dependent and it appears to be maximal with a 1 $g.kg^{-1}$ dose infused over half-hour. For continuous use, it's tapered to a maintenance dose of 0.25 to 0.50 $g.kg^{-1}$ IV bolus every four to six hours. Mannitol changes blood rheology reducing blood viscosity and thus increasing cerebral blood flow and is osmotic diuretic [41]. Mannitol shouldn't be administered within the absence of ICP monitoring unless the patient is showing signs of trans-tentorial herniation [16;42]. Measure serum sodium every four hours (and serum osmolality every four hours if using

Table 1: Causes of elevated intracranial cerebral pressure (ICP) in traumatic brain injury (TBI) patients [40]

EXTRACRANIAL	INTRACRANIAL
Mass lesion (i.e. epidural or subdural hematomas, hemorrhagic contusions) Hydrocephalus Diffuse brain edema Depressed skull fracture Brain edema – Cytotoxic (intracellular) or vasogenic (extracellular) Disturbed in cetebro spinal fluid dynamics with or without ventricular enlargement Hyperemia – Vasomotor paralysis or loss of autoregulation Venous sinus thrombosis	Airway obstruction Hypoventilation Hypoxia Hypercarbia Head position or posture Cranial venous outflow obstruction Hyperpyrexia Hyponatremia Agitation, pain Diabetic ketoacidosis Convulsive or non-convulsive seizure Increased intrathoracic or intra-abdominal pressure (i.e., Valsalva maneuvers, mechanical ventilation) Drugs (tetracycline, doxycycline): Increased intra-abdominal pressure (including compartment syndrome) Liver failure Hypo-osmolarity

mannitol). Hold hypertonic saline therapy for serum sodium ≥ 160 m Eq.L^{-1}. Hold mannitol therapy for serum sodium ≥ 160 mEq.L^{-1} and/or serum osmolality ≥ 320 mOsm.L^{-1}.

NB: In patients who have severe congestive cardiomyopathy and intolerance to mannitol, administration of hypertonic saline can serve as alternative agent.

The following interventions should be considered if ICP is persistently > 22 mmHg for more than 60 minutes after discussion with neurosurgeons: Repeat osmolar therapy as long as serum sodium < 160 mEq.L^{-1} – recommend: use 7.5% sodium chloride 250 mL IV-bolus, one time when volume resuscitation also needed with serum sodium checks every four hours [17]. Consider continuous EEG monitoring to rule out non-convulsive status epilepticus. Consider bolusing and then increasing sedative and analgesic therapy.

Paralysis

Ensure RASS-5 Richmond agitation-sedation scale before initiation of paralytic. Start rocuronium (50 mg IVP loading dose, then 8 µg.kg^{-1}.hr^{-1}); adjust dose according to train of four 1/4 [17]. Consider short-term hyperventilation ($PaCO_2$ 30-34 mmHg) to acutely reduce ICP. Hyperventilation should be avoided in the first 24 hours after injury [16].

Mild hyperventilation

Begin mild hyperventilation with goal $PaCO_2$ 30-34 mmHg. The CBF decreases by 1 mL per 100 g.min^{-1} for each 1 mmHg decrease in $PaCO_2$ [43]. If hyperventilation is used, jugular bulb oxygen saturation (SjO_2) or brain tissue oxygen tension ($BtpO_2$) measurements are recommended to monitor oxygen delivery. Jugular bulb monitoring of arterio-jugular differences of Oxygen $AVDO_2$, as a source of information for management decisions, may be considered to reduce mortality and improve outcomes at three and six month post-injury. Jugular venous saturation of < 50% may be a threshold to avoid in order to reduce mortality [16].

Refractory intracranial hypertension

If all above measures fail and ICP remains elevated > 22 mmHg. Ensure that medical therapy with hypertonic saline is maximized (e.g. serum sodium 155-160 mEq.L^{-1}) [17]. Consider revised ICP threshold of 25 mmHg with strict adherence to CPP > 60 mmHg. Initiate continuous EEG (if not already present) [17]. Surgical decompression [17]. Craniotomy solely for management of ICP does not improve long-term neurological outcome. Consider decompressive craniotomy or craniotomy in patients with a surgical lesion.

Barbiturate coma

If not a surgical candidate, and refractory to all above interventions, consider pentobarbital coma. Pentobarbital 10 mg.kg^{-1} IV over 30 minutes, then 5 mg.kg^{-1}.h^{-1} over three hours, then 1 mg.kg^{-1}.h^{-1} IV infusion [44]. As alternative, sodium thiopental might be used as follows: 2.5-10 mg.kg^{-1} IV, slow bolus, then 0.5-2 mg.kg^{-1}.h^{-1}. Titrate pentobarbital to the minimal dose required to achieve EEG burst suppression, as – three to five bursts per minute (one or two bursts per screen) [17]. Discontinue all other sedative agents and paralytics after pentobarbital loading doses complete (four hours). Consider invasive hemodynamic monitoring (such as pulmonary artery catheter) due to the negative inotropic effects of pentobarbital [45]. Once ICP ≤ 22 mmHg for 48 hours, wean pentobarbital dose over the next 48-72 hours [17]. Prophylactic barbiturates administration to induce burst suppression measured by EEG is not recommended [16].

Hemodynamic stability is essential before and during barbiturate therapy. Although propofol was recommended for the control of ICP, it is not recommended for improvement in mortality or six months outcomes. Caution is required as high-dose propofol can produce significant morbidity [16].

Cerebrospinal fluid drainage

An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use [16]. Use of CSF drainage to lower ICP in patients with an initial GCS of five or under during the first 12 hours after injury may be considered [16]. If present, these catheters should be placed 5-10 cm above the head and opened for drainage every one to four hours. ICP monitoring should be continued until the patient can be assessed clinically, ICP stabilized (< 20-25 mmHg) and cerebral edema resolved on CT-scan (usually takes seven days) [46].

HYPOTHERMIA

Early (within 2.5 hours), short-term (48 hours post-injury), prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse injury. Induced hypothermia (temperatures 30-33 C°) is associated with prolonged ventilation and predisposes to nosocomial infection and may be associated with increased morbidity and mortality in head trauma patients. Currently it cannot be recommended on current basis [46].

SEIZURES MANAGEMENT

Prophylactic use of phenytoin or valproate is not recommended for preventing late post-traumatic seizures (PTS) occurrence after seven days of injury. Phenytoin is recommended to decrease the incidence of early PTS (within seven days of injury) although early PTS have not been associated with worse outcomes [16]. Levetiracetam (cheap, safe alternative to phenytoin with fewer drug interactions and does not require level monitoring) 500 mg IV/PO/PT every 12 hours during seven days (discontinue after seven days if no seizure activity). If seizures occur, treat with lorazepam, 4 to 8 mg IV, repeated until seizures are controlled, followed by phenytoin, 17 $mg.kg^{-1}$ IV loading at a rate of 50 $mg.min^{-1}$ [14].

NUTRITION

Early appropriate nutritional support is highly important (within 24 hours; post-pyloric feeding preferred). Feeding patients to attain basal caloric replacement at least by the fifth day and at most by the seventh day post-injury is recommended to decrease mortality [16]. Transgastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.

INFECTION PROPHYLAXIS

Early extubation in qualified patients. Early tracheostomy is recommended to reduce mechanical ventilation days although, there is no evidence that early tracheostomy reduces mortality or the rate of nosocomial pneumonia [16]. The use of poviodine iodine (PI) oral care is not recommended to reduce ventilator-associated pneumonia and may cause an increased risk of acute respiratory distress syndrome. Antimicrobial-coated catheters may be considered to prevent catheter-related infections during external ventricular drainage [16]. Routine ventricular catheter change or prophylactic antibiotic use for ventricular catheter placement is not recommended to reduce infection [42]. Give flucloxacillin 1 g IV or cefuroxime 1.5 g IV if a penetrating or compound skull fracture or intracranial air is found, and tetanus prophylaxis [20].

SURGICAL MANAGEMENT

Epidural hematoma (EDH)

EDH > 30 cm^3 should be evacuated regardless of the GCS. Hematomas < 30 cm^3 with GCS of 8 or under, or thickness > 15 mm or midline shift > 5 mm or a focal deficit should undergo immediate evacuation after vital stabilization [22].

Subdural hematoma (SDH)

SDH > 10 mm thick or with midline shift > 5 mm should be evacuated regardless of the GCS. SDH < 10 mm thickness and midline shift < 5 mm should also be evacuated if the patient has GCS decrease by 2 points, asymmetric pupils or fixed pupils, or ICP > 20 mmHg [23]. Closed depressed fracture is indicated for operative management

1. Bone depressed more than skull thickness
2. Gross cosmetic deformity.

Compound break [34] is indicated for operative management:

A- Bone depressed quite the thickness of calvaria

B- Bone depressed but skull thickness and:

1. Evident wound infection (redness, pus, etc.)
2. Gross wound contamination (hair, dirt, etc.)
3. Significant underlying intracranial hematoma
4. Evident dural laceration (clinically or radiologically)
5. Sinus involvement
6. Gross cosmetic deformity
7. Pneumocephalus

Intracranial parenchymal lesions > 50 cm^2 should be evacuated operatively. Lesions 20-50 mL should be evacuated operatively in medically refractory ICP elevations or patients who show neurological deterioration as a result of the lesion, patients with GCS six to eight with > 5 mm midline shift or cistern compression [26].

Bifrontal decompressive craniectomy (DC) isn't recommended to enhance outcomes as measured by the extended Glasgow outcome score (GOS-E score) at six-months post-injury in severe TBI patients with diffuse injury (without mass lesions), and with ICP elevation to values > 20 mmHg for over 15 min within a 1 hour period that are refractory to first-tier therapies. However, this procedure has been demonstrated to scale back ICP and to reduce days within the intensive care unit (ICU). An outsized fronto-temporoparietal DC (not but 12 x 15 cm or 15 cm diameter) is suggested over a tiny low fronto-temporoparietal DC for reduced mortality and improved neurologic outcomes in patients with severe TBI [48]. Severe TBI patients undergoing prolonged emergency surgery should ideally have intracranial pressure monitoring placed as soon as possible.

Table 2: Side effects of commonly used treatment modalities for intracranial cerebral pressure (ICP) [15]

ICP monitoring	Intracranial hemorrhage; infection; pain at insertion site.
Hyperventilation	Auto-regulatory dysfunction; cerebral ischemia (regional or global).
Mannitol	Congestive heart failure; circulatory overload; hypo- or hypertension; chills, convulsions, dizziness, headache; volume depletion; pulmonary and peripheral edema; electrolyte abnormalities (pseudohyponatremia); osmotic nephropathy (especially when volume depleted); metabolic acidosis, water intoxication; acute tubular necrosis (> 200 $g.day^{-1}$; serum osmolality > 320 $mOsm.L^{-1}$); subdural hematomas that result from shearing of bridging veins due to hyperosmolar contracture of brain.
Hypertonic saline	Central nervous system changes (encephalopathy, lethargy, seizures, coma); central pontinemyelinolysis (often seen in alcoholic and malnutrition patients); congestive heart failure; transient hypotension (during bolus); electrolyte derangements; cardiac arrhythmias; pulmonary and peripheral edema; hyperchloremic metabolic acidosis; subdural hematomas that result from shearing of bridging veins due to hyperosmolar brain shrinkage; hemolysis with rapid infusions, resulting in sudden osmotic gradients in serum; phlebitis with infusion via peripheral route; coagulopathy; rebound hyponatremia leading to cerebral edema with rapid withdrawal.
Barbiturates (thiopental/pentobarbital)	Respiratory depression and hypercarbia; nausea; vomiting; hypotension and cardiac suppression; infection; confusion, paradoxical reactions, constipation, diarrhea, phlebitis.
Propofol	Hypotension; hypopnea; arrhythmia; decreased cardiac output. Propofol infusion syndrome (acute refractory bradycardia leading to asystole, with one or more of the following: metabolic acidosis, rhabdomyolysis, hyperlipidemia, enlarged or fatty liver).
Therapeutic hypothermia	Electrolyte abnormalities (hypokalemia, hypocalcemia); cardiac suppression, arrhythmias (including asymptomatic electrocardiographic changes); infection due to immune suppression; reduced creatinine clearance (during the active phase of hypothermia); pancreatitis.
Decompressive craniectomy	Subdural hygroma formation, contralateral development of subdural or epidural hematoma, hydrocephalus, excessive herniation through the skull defect, and intracranial infections. The syndrome of the trephined includes headaches, memory disturbance, mood alteration, dizziness and contralateral upper extremity weakness not due to the initial injury and It is reversed by cranioplasty.

SUPPLEMENTS

Chest X-Ray. All head trauma patients receive the routine "trauma series" of x-rays chest, pelvis, cervical spine (AP, lateral and Peg view also called open mouth view) [46], 85-90% of cervical spine injuries are evident in lateral view radiographs.

CT-scan. All severe traumatic brain injury patients should undergo CT-scan brain furthermore as of the cervical spine within one hour of initial assessment within the ED. A provisional written radiology report should be made available within one hour of the scan being performed. CT-scan Imaging should never delays lifesaving treatment. CT-scan findings with predictive value with reference to outcome: presence or absence of the basal cisterns, midline shift, traumatic subarachnoid hemorrhage, and hemorrhage into the basal cisterns [13]. Traumatic brain injury patients with a hemorrhagic but non-surgical lesion found on initial brain CT-scan are recommended to be scanned again at four hours post-initial CT-scan scan unless there's rapid deterioration. Approximately 25% of posttraumatic hemorrhages expand during this time. For safety, logistic and resource reasons, resonance imaging (MRI) scanning isn't recommended because the primary investigation for clinically important brain injury in patients who have sustained a head injury, although sometimes it can detect additional important prognostic information is recognized. MRI imaging is indicated if there are neurological signs and symptoms referable to the cervical spine [13].

PROGNOSTIC SCORES

CLINICAL SCORES

GCS is the most generally used system to predict outcome after TBI [49]. The motor component of the GCS score is most predictive of the severity of the brain injury and correlates most strongly with overall outcome. The GCS should be measured after airway, breathing, and circulation are assessed, and after necessary ventilatory or circulatory resuscitation has been performed preferably before administering sedative or paralytic drugs.

GOS-E score with five ordered categories in its original version: death, vegetative state, severe disability, moderate disability, and good recovery. The GOS-E scale splits each of severe disability, moderate disability, and good recovery into lower and upper categories to permit for greater differentiation between the degree of recovery which will be achieved [50;51].

Radiological scores: Both Marshal and Rotterdam CT-scan grading systems are good in predicting early mortality after moderate and severe TBI. Because the Rotterdam system also includes additional variables like subarachnoid hemorrhage, EDH and basal cistern compression it's going to be preferable, particularly in patients with diffuse injury [52].

Serum biomarkers: serum levels of Thrombomodulin (TM) and doc factor (vWF) are valuable in evaluating endothelial damage in severe TBI moreover as prediction of Delayed Traumatic intracerebral hematoma [53]. S-100β correlated with TBI severity and prognosis [54].

KEY NOTES TO REMEMBER

1. Transport patients who have sustained TBI on to a hospital that has the resources to further resuscitate them and to research and initially manage multiple injuries, appropriate to the patients' age [13].

2. Prevent and emergency treatment of secondary brain injury causes hypoxia, hypo (hyper) glycemia, hyperpyrexia, hypo (hyper) tension, prolonged hypo (hyper) capnea.

3. All primary brain injury causes are irreversible except surgically evacuable mass.

4. The foremost effective methods of reducing intracranial pressure are mechanical interventions as removal of mass lesions, drainage of spinal fluid or decompressive craniotomy.

5. Cerebral angiography should be considered accordance with the rules set forth by the Eastern Association for the Surgery of Trauma (EAST): when carotid dissection is suspected by; (a) Large iso-dense lesion in CT-scan after TBI, (b) Patient clinical condition not in step with CT-scan (dense hemiparesis within the absence of mass lesion), (c) High-impact trauma, a history of cervical hyperextension, flexion, or rotation, (d) trauma patients presenting with epistaxis from a suspected arterial source after trauma, (e) Asymptomatic patients with significant blunt head trauma as defined below are at significantly increased risk for BCVI and screening should be considered. Risk factors are as follows: *GCS score of 8 or under; *Petrous bone fracture; *Diffuse axonal injury; *Cervical spine fracture particularly those with (i) fracture of C1 to C3 and (ii) fracture through the foramen transversarium; *Cervical spine fracture with subluxation or rotational component; and *Lefort II or III facial fractures.

6. The utilization of steroids isn't recommended for improving outcome or reducing ICP. In patients with severe TBI, high dose methylprednisolone was related to increased mortality and is contraindicated [16].

7. While fever could also be seen in up to 70% of TBI patients. In these patients, an infectious etiology is present but 50% of the time, with the rest being classified as "central fever." Early fever following TBI has been related to lower GCS, presence of diffuse axonal injury, cerebral edema, hypotension, hypoglycemia, leukocytosis, neurologic impairment, and prolonged ICU stay [55].

8. Clinical signs of herniation include dilated and unreactive pupils, asymmetric pupils, or a motor exam that identifies extensor posturing. Cushing reflex (also said because the vasopressor response) could be a physiological systema nervosum response to increased (ICP) that ends up in Cushing's triad of increased force per unit area, irregular breathing, and bradycardia [56].

9. Consider or suspect abuse as a contributory factor to or reason for head injury in children. Abuse may coexist with a head injury. Ascribe depressed conscious level to intoxication only after a major brain injury has been excluded.

10. It's not monitoring as such that affects outcomes; rather, it's using the data from monitoring to direct treatment.

REFERENCES

1. Hyder A, Wunderlich C, Puvanachandra P, Gururaj G, Kobusingye O. The impact of traumatic brain injuries: a global perspective. NeuroRehabilitation 2007; 22:341-53.
2. Kamal VK, Agrawal D, Pandey RM. Epidemiology, clinical characteristics and outcomes of traumatic brain injury: evidences from integrated level 1 trauma center in India. J Neurosci Rural Pract 2016; 7:515-25.
3. Fleminger S, Ponsford J. Long term outcome after traumatic brain injury. BMJ 2005; 331:1419-20.
4. De ramirez SS, Hyder AA, Herbert HK, Stevens K. Unintentional injuries: magnitude, prevention, and control. Annu Rev Public Health 2012; 33:175-91.
5. Kraus JF, Black MA, Hessol N, Ley P, Rokaw W, Sullivan C, et al. The incidence of acute brain injury and serious impairment in a defined population. Am J Epidemiol 1984; 119:186-201.
6. Tieves K, Yang H, Layde P. The epidemiology of traumatic brain injury in Wisconsin, 2001. WMJ 2005; 104:22-5.
7. Dawodu, ST. 2017. Traumatic brain injury: Definition, epidemiology, pathophysiology, Available from: http://www.emedicine.com/pmr/topic212.htm (Last access 03/04/2019).
8. Management of Concussion/mTBI Working Group. VA/DoD clinical practice guideline for management of concussion/mild traumatic brain injury. J Rehabil Res Dev 2009; 46:CP1-68.
9. Frey LC. Epidemiology of posttraumatic epilepsy: A critical review. Epilepsia 2003; 44(s10):11-7.
10. Jallo JI, Narayan RK. Craniocerebral trauma. In: WG Bradley, RB Daroff, GM Fenichel, et al. Eds. Neurology in clinical practice. Boston: Butterworth-Heinemann 2000; 1055-87.

11. Maegele M, Engel D, Bouillon B, Lefering R, Fach H, Raum M, et al. Incidence and outcome of traumatic brain injury in an urban area in Western Europe over 10 years. Eur Surg Res 2017; 39:372-9.
12. Jennett B. Epidemiology of head injury. J Neurol Neurosurg Psychiatry 1996; 60:362-9.
13. Hodgkinson S, Pollit V, Sharpin C, Lecky F. Early management of head injury: summary of updated NICE guidance. BMJ 2014; 348:g104.
14. Gossman W, Plantz S. Pearls of Wisdom Emergency Medicine. 5th ed. New York: McGraw-Hill; 2008.
15. Hall JB, Shmidt GA, Kress JP. Intracranial pressure: monitoring and management. In Principles of critical care. 4th ed. New York: McGraw-Hill 2015; 786-820.
16. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery 2017; 80:6-15.
17. Department of surgical education. Antiplatelet agent reversal in adults with traumatic intracranial hemorrhage. Available from: http://www.surgicalcriticalcare.net/Guidelines/Antiplatelet%20Reversal%20Guideline.pdf (Last access 03/04/2019).
18. Cremer OL, Moons KG, Bouman EA, Kruijswijk JE, de Smet AM, Kalkman CJ. Long-term propofol infusion and cardiac failure in adult head-injured patients. Lancet 2001; 357:117-8.
19. Stevens R, Shoykhet M, Cadena R. Emergency Neurological Life Support: Intracranial hypertension and herniation. Neurocrit Care 2015; 23 (s2):S76-82.
20. Brown A, Codigan M. Emergency medicine diagnosis and management 7th ed. CRC Press; 2016.
21. Nazir M, Khan S, Raja R, Bhatti S, Ahmed E. Cervical spinal injuries in moderate to severe head injuries. J Ayub Med Coll Abbottabad 2012; 24:100-2.
22. Bullock M, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell D, et al. Surgical management of acute epidural hematomas. Neurosurgery 2006; 58:S7-15.
23. Bullock M, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell D, et al. Surgical management of acute subdural hematomas. Neurosurgery 2006; 58:S16-24.
24. Youmans J. Neurological surgery: A comprehensive reference guide to the diagnosis and management of neurosurgical problems. 4th ed. Philadelphia Saunders; 1996.
25. Rabinovici R, Frankel HL, Kirton O. Trauma, critical care and surgical emergencies. 1st ed. Informa UK; 2010.
26. Bullock M, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell D, et al. Surgical management of traumatic parenchymal lesions. Neurosurgery 2006; 58:S23-46.
27. Feldman Z, Kanter M, Robertson C, Contant C, Hayes C, Sheinberg M, et al. Effect of head elevation on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow in head injured patients. J Neurosurg 1992; 76:207-11.
28. Frontera J, Lewin J, Rabinstein A, Aisiku I, Alexandrov A, Cook A, et al. Guideline for reversal of antithrombotics in intracranial hemorrhage. Neurocrit Care 2016; 24:6-46.
29. Ranucci M, Nano G, Pazzaglia A, Bianchi P, Casana R, Tealdi DG. Platelet mapping and desmopressin reversal of platelet inhibition during emergency carotid endarterectomy. J CardothoracVasc Anesth 2007; 21:851-4.
30. Floradal PA, Sahlin S. Use of desmopressin to prevent bleeding complications in patients treated with aspirin. Br J Surg 1993; 80:723-4.
31. Department of surgical education. Antiplatelet agent reversal in adults with traumatic intracranial hemorrhage. Available from: http://www.surgicalcriticalcare.net/Guidelines/Antiplatelet%20Reversal%20Guideline.pdf (Last access 17/04/2019).
32. Hedges SJ, Dehoney SB, Hooper JS, Amanzadeh J, Busti AJ. Evidence-based treatment recommendations for uremic bleeding. Nature Clin Prac 2007; 3:138-53.
33. Voils S. Pharmacologic interventions for the management of critical bleeding. Pharmacotherapy 2007; 27:69S-84S.
34. Prexl O, Bruckbauer M, Voelckel W, Grottke O, Ponschab M, Maegele M, et al. The impact of direct oral anticoagulants in traumatic brain injury patients greater than 60-years-old. Scand J Trauma Resusc Emerg Med 2018; 26:20.
35. Zangbar B, Pandit V, Rhee P, Khalil M, Kulvatunyou N, O'Keeffe T, Tang A, et al. Clinical outcomes in patients on preinjury ibuprofen with traumatic brain injury. Am J Surg 2014; 209:921-6.
36. Thompson H, Kirkness C, Mitchell P. Intensive care unit management of fever following traumatic brain injury. Intensive Crit Care Nurs 2007; 23:91-6.
37. Holtzclaw BJ. Use of thermoregulatory principles in patient care: Fever management. The Online Journal of Clinical Innovations 2002; 5:1-64.
38. Ekeh AP, Dominguez K, Markert R, McCarthy M. Incidence and risk factors for deep venous thrombosis after moderate and severe brain injury. J Trauma 2010; 68:912-5.
39. Prabhakar H, Sandhu K, Bhagat H, Durga P,Chawla R. Current concepts of optimal cerebral perfusion pressure in traumatic brain injury. J Anaesthesiol Clin Pharmacol 2014; 30:318-27.
40. Laskowitz D, Grant G. Translational research in traumatic brain injury. boca raton (FL): CRC Press/Taylor and Francis Group; 2016.
41. LIorente G, Mejia MC. Mannitol versus hypertonic saline solution in neuroanaesthesia. Rev Colomb Anestesiol 2015; 43:29-39.
42. Brain Trauma Foundation; American association of neurological surgeons; Congress of neurological surgeons; Joint section on neurotrauma and critical care, AANS/CNS, Carney NA, Ghajar J. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007; 24:S1-106.
43. Giardino N, Friedman S, Dager S. Anxiety, respiration, and cerebral blood flow: implications for functional brain imaging. Compr Psychiatry 2007; 48:103-12.
44. Eisenberg HM, Frankowski RF, Contant CF, Marshall LF, Walker MD. High dose barbiturates control of elevated intracranial pressure in patients with severe TBI. J Neurosurgery 1988; 69;15-23.
45. Haddad S, Arabi Y. Critical care management of severe traumatic brain injury in adults. Scand J Trauma Resusc Emerg Med 2012; 20:12.
46. Bersten A. Oh's intensive care manual. 7th ed. Butterworth Heinmann Elsevier; 2014.
47. Bullock MR, Chesnut R, Gghagar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of depressed cranial fractures. Neurosurgery 2006; 58:S56-60.
48. Moon JW, Hyun DK. Decompressive craniectomy in traumatic brain injury: A review article. Korean J Neurotrauma 2017; 13:1-8.
49. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974; 2:81-4.
50. Teasdale GM, Pettigrew LE, Wilson JT, Murray G, Jennett B. Analyzing outcome of treatment of severe head injury: A review and update on advancing the use of the Glasgow Outcome Scale. J Neurotrauma 1998; 15:587-97.

51. Wilson JTL, Pettigrew LE, Teasdale GM. Structured interviews for the Glasgow outcome scale and the extended glasgow outcome Scale: Guidelines for their use. J Neurotrauma 1997; 15:573-85.
52. Deepika A, Prabhuraj AR, Saikia A, Shukla D. Comparison of predictability of Marshall and Rotterdam CT-scan scoring system in determining early mortality after traumatic brain injury. Acta Neurochir (Wien) 2015; 157:2033-8.
53. Yokota H, Naoe Y, Nakabayashi M, Unimito K, Kashimoto S. Cerebral endothelial injury in severe head injury: the significance of measurement of serum thrombomodulin and von vellibrand factor. J Neurotrauma 2002; 19;1007-15.
54. Mercier E, Boutin A, Lauzier F, Fergusson D, Simard JF, Zarychanski R, et al. Predictive value of S-100β protein for prognosis in patients with moderate and severe traumatic brain injury: systematic review and meta-analysis. BMJ 2013; 346:f1757.
55. Cairns CJ, Andrews PJ. Management of hyperthermia in traumatic brain injury. Curr Opin Crit Care 2002; 8:106-10.
56. Ayling, J. Managing head injuries. Emerg Med Serv 2002; 31:42.