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REVIEW ARTICLE

Emergency Neurological Life Support: Traumatic Brain Injury

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Abstract Traumatic brain injury (TBI) was chosen as an Emergency Neurological Life Support topic due to its frequency, the impact of early intervention on outcomes for patients with TBI, and the need for an organized approach to the care of such patients within the emergency setting. This protocol was designed to enumerate the practice steps that should be considered within the first critical hour of neurological injury.

Keywords Secondary brain injury · Hemicraniectomy · ICP monitoring · Protocol

Introduction

In the United States, approximately 1.7 million people sustain a traumatic brain injury (TBI) each year. Of these, 275,000 are hospitalized and 52,000 die. Worldwide, TBI is the leading cause of death and disability for children (>1 year old) and young adults. Falls and motor vehicle collisions account for the largest proportion of TBI cases in civilian populations. Among military personnel, blast injuries are the most common cause [1].

In patients who survive their initial traumatic event, the cascade of secondary insults that begins immediately thereafter influences clinical outcome. Secondary insults occur on both a microscopic cellular level (e.g., as a result of hypoxemia) and a macroscopic level (e.g., a subdural hematoma that requires surgical intervention). The attempt to identify, prevent, and reverse secondary injury defines and guides the first minutes and hours of TBI management. In addition, the prevention and management of secondary injury is fundamental to the subsequent care of hospitalized TBI patients.

The ENLS suggested algorithm for the initial management of traumatic brain injury is shown in Fig. 1. Suggested items to complete within the first hour of evaluating a patient with traumatic brain injury are shown in Table 1.

Traumatic Brain Injury: Diagnosis and Classification

The diagnosis of TBI is based on identifying a mechanism consistent with TBI and/or physical signs of trauma in a patient with neurological signs or symptoms. Severe TBI is defined as TBI in a patient with an identified mechanism who is unconscious and has a Glasgow Coma Scale (GCS) < 9. Consideration for the anatomic status of the patient (e.g., scalp laceration or depressed skull fracture) and the mechanism (e.g., fall > 20 feet or motor vehicle accident exceeding 30 mph) are important historical and physical findings.

The GCS should be determined by examining the patient (seeing if s/he can follow commands or, if not, observing

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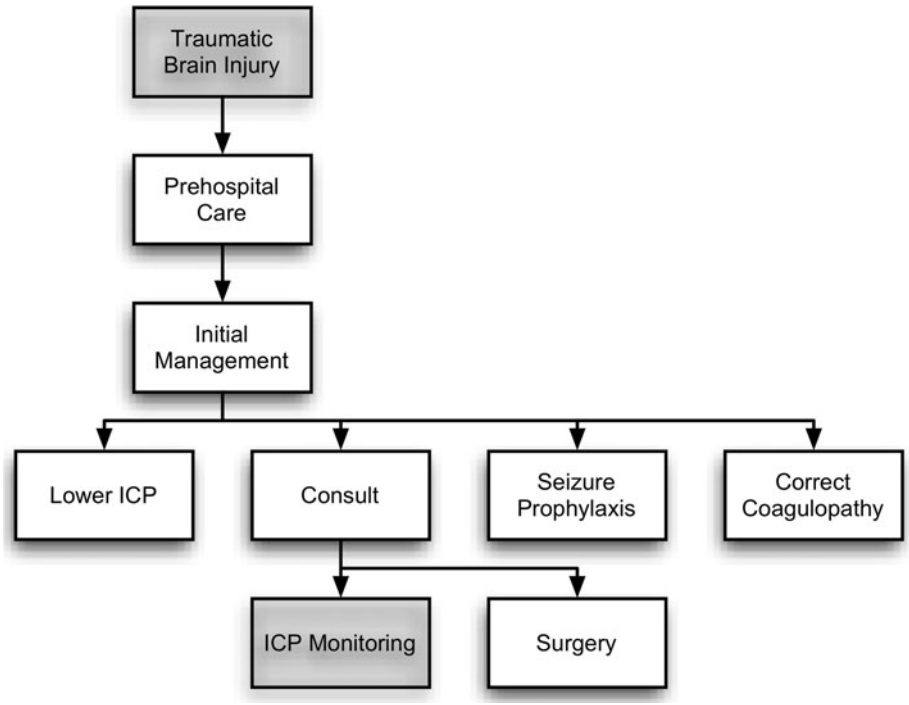

Fig. 1 ENLS traumatic brain injury protocol

Table 1 Traumatic brain injury checklist for the first hour

☐ Airway
☐ Keep SBP >90 mmHg
☐ C-spine precautions
☐ Head CT
☐ Treat herniation

his/her response to pain) and, if possible, should be calculated after any resuscitation but before any sedative or paralytic agents are administered. Every attempt should be made to identify and manage vascular, metabolic, infectious, environmental, toxicological, and other non-traumatic causes, as these causes may co-exist with TBI.

When the mechanism is not clear or history is lacking, the many non-traumatic causes of decreased level of consciousness (LOC) should be considered. With limited patient history, it may be difficult to identify these during the first minutes of care. In such cases, a systematic approach that considers the possibility of non-traumatic conditions which may mimic or co-exist with TBI is essential.

Information gathered at the scene by pre-hospital personnel may be very helpful in diagnosing TBI. Patients involved in single motor vehicle accidents and those without significant signs of external trauma may raise suspicion for other causes of decreased LOC. In addition, a patient may have sustained a TBI after a stroke or intracranial hemorrhage. Causes of deceased LOC that are reversible in the field include airway obstruction, tension pneumothorax, hypoglycemia, and opiate overdose. The influence of drugs and alcohol should also be considered.

Prehospital Care

The prehospital phase of TBI management is critical to outcome. Basic life support maneuvers are directed at protection of the cervical spine (C-spine), maintenance of a patent airway, and assurance of adequate ventilation and oxygenation. If any of these are compromised, secondary injury proceeds at an accelerated pace, and the risk of mortality increases. Moreover, the impact of general cardiopulmonary resuscitation on neurological outcome overshadows most other details of management further downstream in the patient's course.

- Spinal precautions should be maintained until the spine is cleared.
- Assess GCS.
- Pupils should be monitored throughout the resuscitation. Eye trauma may mimic or mask the signs of herniation and should be considered in each case.
- Pupil asymmetry is defined as >1 mm difference in diameter.
- A fixed pupil is defined as <1 mm response to bright light.
- Basic and advanced airway management is indicated to maintain oxygen saturation greater than 90 %.
- In the patient who is breathing spontaneously, the airway should be stabilized while maintaining C-spine

precautions. Supplemental oxygen should be delivered by face mask. If easily tolerated (considering C-spine precautions), insertion of an oral or nasal airway device is helpful to prevent the tongue from occluding the airway.

- The use of paralytics to assist endotracheal intubation in the prehospital setting is not recommended. Exceptions to this recommendation include small closed systems, such as aeromedical transport teams that possess special training in emergency airway management.
- In urban settings and those with short emergency medical system (EMS) transport times, endotracheal intubation is not recommended.
- Normal breathing should be maintained (End Tidal CO_2 ($ETCO_2$) 35–40 mmHg) and hyperventilation avoided ($ETCO_2 < 35$ mmHg) unless there are signs of herniation. If signs of herniation are present, hyperventilation (20 breaths per min in the adult) can be used as temporary measure until signs of herniation resolve.
- Oxygenation (pulse oximetry) and blood pressure (BP) should be continuously monitored.
- Obtain intravenous (IV) access.
- For adults, maintain systolic BP > 90 mmHg.
- Hypotensive patients should be treated with isotonic fluids (500 cc to 1 L of normal saline), or smaller volumes of hypertonic saline solutions can be given in adult trauma victims with systolic BP < 90 mmHg or with signs of hypoperfusion (e.g., poor capillary refill or decreased urine output).
- Hypotonic fluids, such as D5W, should be avoided, as they may exacerbate brain edema.
- GCS may decline with hypotension and improve as BP is restored.
- Diagnose hypoglycemia, and, if hypoglycemic, give D50 (50 ml IV push).

Large studies that have examined the role of pre-hospital care in trauma have concluded that simple first aid and basic life support (BLS) measures, combined with rapid transport to hospital, are preferred over models that utilize more highly trained personnel. The largest prehospital study to compare the effectiveness of advanced life support (ALS) to BLS is the Ontario Prehospital Advanced Life Support (OPALS) study. This study compared a large population of patients before and after the implementation of ALS in pre-hospital care. No improvement was detected in the outcomes of trauma patients subsequent to the addition of ALS transport [2].

The limited studies available suggest that the performance of advanced procedures in the field or during transport, in particular, endotracheal intubation, may worsen outcomes in some patients. Most studies that examine prehospital intubation are retrospective and suffer from selection bias (i.e., the patients who are intubated are likely sicker, making intubation seem deleterious). The best data available is from a pediatric EMS study in which severely ill and injured children were randomized by day of the month to receive either BLS airway management (e.g., bag-valve-mask ventilation) or endotracheal intubation. Patients in the BLS group fared no worse with similar rates of good neurological recovery. The average transport time in the pediatric EMS study was 6 min [3]. This data cannot be directly extrapolated to adults and to EMS systems with longer transport times, such as those in rural or remote areas.

The use by prehospital personnel of paralytic medications in the field to facilitate endotracheal intubation in patients with head injuries has received considerable attention. There are numerous case series of successful advanced airway management by paramedics and nurses in tightly regulated and highly specialized systems. However, the best data comes from a large, controlled trial on field rapid sequence intubation (RSI) that was performed across a variety of ground-based paramedic units. When compared to historical controls, patients who received RSI for intubation in the field had a higher rate of mortality and a decreased likelihood of a favorable neurological outcome than historical controls.

The study involved mostly rapid transport times in urban areas, and the paramedics included received a limited amount of training in RSI [4]. Analysis of field records from this study revealed that RSI doubled scene time from 13 to 26 min. Oximetry and capnography records revealed that prolonged episodes of hypoxemia, hypercapnia, and hypocapnea, often lasting several minutes, were associated with field RSI. These observations may explain the poor outcomes in the RSI cohort.

With continued concerns about the safety of prehospital intubation in trauma, there has been a renewed focus on supraglottic devices, which are easier and faster to place. These have been adopted in many EMS systems as either a primary or rescue airway device when conventional endotracheal intubation has failed. A recent systematic review of the prehospital literature concluded there are no statistically significant differences in patient oriented outcomes between patients managed with alternative airway devices and with field attempts at conventional endotracheal intubation [5]. However, experimental data is lacking.

Permissive hypotension is the practice of withholding fluids in the resuscitation of trauma patients until definitive hemostasis can occur in a controlled setting, such as the operating suite. This practice gained acceptance after a randomized trial that studied victims of penetrating torso

trauma [6]. However, hypotension in the field phase disproportionately worsens outcome in trauma patients with TBI, hence appropriate fluid administration is indicated in TBI patients with shock [7].

Hypertonic saline (HTS) has been studied extensively as a resuscitation fluid in patients with TBI. HTS is theoretically ideal in TBI patients with multiple injuries, as it is both an effective volume expander and capable of decreasing brain edema. Thus, unlike mannitol, HTS can be used in traumatized patients who present in shock. However, one randomized, controlled trial failed to show decreased mortality or improved neurological outcome in patients with severe TBI and hypotension when HTS was used during prehospital care [8].

Initial Hospital Management

Upon arrival to the ED, the major priorities from the field are unchanged: ensuring cerebral perfusion and preventing brain herniation remain the primary goals. In addition to pulse oximetry and electrocardiography (ECG) monitoring, capnography is useful to guide cerebral resuscitation.

- Spinal precautions should be maintained at all times.
- Advanced airway management should be performed to ensure airway protection, oxygen saturation >90 %, and control of ventilation (if inadequate or inappropriate).
- Oxygenation, blood pressure, cardiac rhythm, and pCO_2 should be continuously maintained.
- Obtain parenteral access (IV or interosseus).
- In the adult, systolic BP should be >90 mmHg.
- Reassess GCS: the GCS obtained in the emergency department (ED) may be a more reliable assessment of the severity of TBI than the GCS obtained in the field.
- Diagnose hypoglycemia: if hypoglycemic, give D50 50 ml IV.
- Address immediate, life-threatening, and non-TBI injuries.
- Obtain and review a chest X-ray.
- Obtain head computed tomography (CT) without contrast.

Although a GCS of eight or less during the initial evaluation is an indication for endotracheal intubation, severe extra-cranial injuries or a rapidly declining mental status may also be indications. Patients can be ventilated initially with FiO_2 of 100 % until it can be titrated down to maintain $SaO_2 > 90$ %. In patients with multiple injuries, the next priority is to achieve hemostasis. The initial resuscitation may be followed by more definitive therapy in the operating room or procedural suite and should be directed toward controlling bleeding in the torso or extremities.

Immediate surgical intervention for life-threatening hemorrhage may mean that neuroimaging (e.g., CT) is not performed initially. Emergency neurosurgical procedures, such as ventriculostomy or burrhole, may in some cases be performed concurrently with other life-saving measures, but these generally await the conclusion of the procedure until CT can be performed.

CT remains the emergency neuroimaging modality of choice. Despite advances in magnetic resonance (MR) technology, CT remains much faster and more practical for the critically injured patient. The primary purpose of the initial CT is to identify any hemorrhagic lesions that require surgery. As a general guide, patients with an extra-axial hematoma (extra- or sub-dural) >1 cm in thickness, an intra-parenchymal hematoma >3 cm in diameter, and a >5 mm midline shift associated with a hematoma are considered surgical lesions.

Acute blood is hyperdense, but, in some patients, it may be iso- or hypodense if the patient is coagulopathic or anemic or the CT is obtained very soon after injury. Contusions can be hyperdense, hypodense, or have a "salt and pepper" appearance. Intracranial air suggests an open skull fracture, craniofacial trauma, or injury to an air sinus. Surgical decision making is guided by the amount of mass effect; hence, it should be determined whether the perimesencephalic cisterns are open, compromised, or closed, and the degree of midline shift at the level of the third ventricle should be identified. These findings are useful but not always associated with abnormal intracranial pathophysiology [9]. Therefore, they should be interpreted with clinical findings in mind.

The cranial vault and facial bones should be assessed for fractures, displacement of bone fragments, or penetrating objects. Because C-spine injuries occur in up to 10 % of head-injured patients, radiologic imaging of TBI is not complete without neck imaging including X-rays, CT, and/or magnetic resonance imaging (MRI) where appropriate. Until neck imaging is complete, the C-spine should be continuously protected by a rigid collar or manual in-line stabilization.

In some patients, the cerebrovasculature, including the intracranial or extracranial vessels, requires imaging [10]. This may be accomplished through CT angiography, MR angiography or venography, or conventional angiography in selected patients. These studies should be considered when there is:

- Penetrating injury.
- Fracture over a venous sinus.
- A neurologic deficit that is not explained by head CT scan.

- Selected C-spine injuries (e.g., severe flexion or extension injury or a fracture through the transverse foramen).
- Petrous bone fracture.
- Lefort II or III facial fractures.
- A suspected cause for the injury (e.g., aneurysm rupture).

In addition, patients who are victims of near hanging, have seat-belt abrasions of the neck, or have soft tissue swelling of the anterior neck should undergo vascular imaging to exclude blunt injury to the carotid or vertebral arteries.

Lower ICP

In the first hour of neurotrauma, placement of intracranial pressure (ICP) monitoring devices not always practical. Therefore, whether the patient already has intracranial hypertension at presentation should be considered. Indications of this are examination-based and rely upon identifying signs of brain herniation. Signs include dilated and non-reactive pupil(s) or asymmetric pupils; motor exam that demonstrates extensor posturing or no response, progressive decline in neurologic condition (decrease in GCS $>$ 2 points) that is not associated with non-TBI causes, and Cushing's response (increased BP, decreased pulse, and irregular respirations).

In these circumstances, empirical treatment of presumptive high ICP (a patient can have herniation without elevated ICP) may be helpful. Treatments include:

Mannitol or Hypertonic Saline

- Administer 20 % mannitol 0.25–1 g/kg IV as a rapid (5 min) IV infusion.
- If BP (systolic) $<$90 mmHg in adults, hypertonic saline rather than mannitol should be used; administer 3 % NaCl 150 ml IV over 10 min.

Hyperventilation

- Target a pCO_2 of 28–35 mm HG (20 breaths per min in an adult).

Cerebral Perfusion Pressure (CPP)

- Goal is Approximately 60 mmHg.
- If mean arterial pressure (MAP) $<$ 80 mmHg, consider colloid fresh frozen plasma (FFP) if international normalized ration (INR) $>$ 1.5, additional crystalloid if ICP allows.
- If lactate is not elevated, administer phenylephrine 10–100 mcg/min, other pressors as needed.
- Transfuse red blood cells (RBCs) if active bleeding or hemoglobin (Hgb) $<$ 8 gm/dl.

An understanding of two fundamental aspects of brain physiology—the Monro–Kellie doctrine and cerebral autoregulation (CAR)—provides a framework on which to base ICP management [11]. The Monro–Kellie doctrine states that the total volume of the intracranial contents is constant, provided the cranial vault is intact. This fixed volume of the skull contains brain, blood, cerebrospinal fluid (CSF), and, if present, a mass lesion. The combined volumes of these compartments determine the ICP. The blood and CSF can shift a portion of their volume outside of the skull through natural means (i.e., there can be some degree of compensation for an increase in other compartments).

It follows that if there is an expanding mass lesion (e.g., a post-traumatic hematoma), ICP will remain normal, provided there are reductions in volume of the other compartments. In this compensated state, the volume increase associated with a mass lesion or edema is offset by shifting CSF into the spinal subarachnoid space and venous blood out of the intracranial space. However, as the volume of the mass lesion increases, ICP begins to increase, and—once compensation is exhausted—a rapid increase in ICP occurs. When ICP is $>$20 mmHg, brain tissue begins to herniate from areas of high pressure to areas of low pressure.

An additional concept should be considered in relation to ICP management: compliance. Compliance represents how the intracranial contents compensate for volume changes. Cerebral compliance is defined as the ICP response (ΔP) to a given change in volume (ΔV) described by the pressure volume index. As compliance becomes compromised (i.e., there is a rightward shift of the pressure volume index), the pressure response for a given volume increases. Once the brain becomes non-compliant, a small change in volume can result in a rapid increase in ICP or cause acute herniation. Compensation for a volume change is time dependent; rapid volume changes are less buffered and can have a greater influence on ICP than slow changes. Rapid volume changes may accompany nasotracheal suctioning, patient turning, or hypercarbia (increased cerebral blood volume), all of which may increase ICP when cerebral compliance is reduced.

Changes in ICP influence CPP, defined as (MAP–ICP). Similarly, a decrease in blood pressure can compromise CPP. Adequate CPP is necessary to maintain cerebral blood flow (CBF). Though there are regional differences in CBF, normal CBF is generally considered to be approximately 50 mL/100 g brain/min. When CBF is $<$20 mL/100 g brain/min, cerebral ischemia results, and when CBF is $<$5 mL/100 g brain/min, cell death occurs. Cell death also depends on how long the blood flow is low.

CAR refers to the ability of the brain to maintain a constant CBF. There are two forms of CAR: metabolic and pressure. Metabolic CAR is influenced by cellular requirements. In pressure CAR, vasoconstriction or dilation of the cerebral vasculature helps maintain constant CBF across a broad range of perfusion pressures, believed to be between 50 and 150 mmHg in the normal individual. However, when CAR is compromised or absent, as is frequently observed in TBI, CBF can simply parallel changes in CPP, and inadequate tissue oxygen and glucose delivery may result. When ICP equals CPP, CBF can no longer be maintained, and cerebral circulatory arrest occurs.

In resuscitation and management, the fundamental goal is to ensure normal ICP, based in part on understanding the Monro–Kellie hypothesis and CAR. For example, if a large mass lesion is present, surgery is required to manage ICP. Alternately, when diffuse injury and cerebral edema are present, osmotherapy is indicated, since much of the edema is cytotoxic in origin [12].

To facilitate management of intracranial pathology, the following physiological parameters should be maintained as part of goal-directed TBI care, during both resuscitation and subsequent ICU care.

- Pulse oximetry $\geq$ 90 %.
- $PaO_2 \geq 100$ mmHg.
- $PaCO_2$ 35–45 mmHg.
- SBP $\geq$ 90 mmHg.
- pH 7.35–7.45.
- ICP $<$ 20 mmHg.
- Brain Tissue Oxygen Pressure ($PbtO_2$) $\geq$ 15 mmHg.
- CPP $\geq$ 60 mmHg.
- Temperature 36.0–38.3 °C.
- Glucose 80–180 mg/dL.
- Physiologic Na + 135–145, if using hypertonic saline (HTS) 145–160.
- INR $\leq$ 1.4.
- Platelets $\geq$ 75 $\times$ 103/mm3.
- Hgb $\geq$ 8 gm/dl.

In summary, during the initial and subsequent intensive care unit (ICU) phases of severe TBI management, CPP (i.e., ICP and MAP) is the most important physiological parameter to follow. Other parameters should be appropriately adjusted to optimize CPP. Once resuscitated, care can be guided by placement of intracranial monitors, including ICP and brain oxygen. A MAP of $\geq$80 mmHg is a reasonable goal. However, the optimal blood pressure levels for brain resuscitation remain the subject of debate, and higher target MAPs may increase the risk of pulmonary dysfunction. It is very rare for isolated TBI to cause hypotension. Spinal cord injury should be suspected in patients whose blood pressure does not respond to fluid resuscitation.

Coagulopathy

Coagulopathy may compound secondary brain injury by permitting intracranial bleeding. Identification of a pre-existing or rapidly acquired coagulopathy is important in the first hour and during the remainder of the hospital stay. Patients with known end-stage hepatic or renal disease are at risk, as are patients who have been taking anticoagulants or antiplatelet therapies.

Laboratory measures of prothrombin time (PT)/INR/partial thromboplastin time (PTT) and platelet counts should be routine in neurotrauma. At present, newer anticoagulants (thrombin inhibitors and oral Factor Xa inhibitors) do not have antidotes. However, it is important to know whether a patient has taken such agents, as this may alter surgical options or follow-up.

For patients taking warfarin, vitamin K and FFP should be considered even empirically before the INR has been measured, to expedite reversal of coagulopathy. For clinical hemorrhage, purified factor concentrates have been shown to more rapidly reverse warfarin coagulopathy, as they require no thawing and are administered in smaller fluid volume. Patients who have renal disease or are taking antiplatelet drugs (e.g., clopidogrel, aspirin) can receive platelet transfusion depending on the level of concern for bleeding. In addition, desmopressin can be administered to patients in renal failure to temporarily improve uremic platelet dysfunction, and it may also improve platelet function in patients taking antiplatelet agents. Patients taking Dabagatran (Pradaxa) may require hemodialysis.

The incidence of coagulopathy in TBI patients, including those with isolated TBI, is high, approaching 40–50 % in severe TBI. Coagulopathy associated with trauma has several possible mechanisms, but in TBI and isolated TBI, the principal process involves tissue factor (TF) release. Several factors, such as increased age, hypotension at the scene of injury, a low GCS ($\leq$8), injury severity score (ISS) $\geq$ 16, severity of TBI reflected by the head abbreviated injury score (AIS), intraparenchymal lesions, penetrating injury, and base deficit are associated with TBI coagulopathy [13–15]. In industrialized countries, approximately 1 % of the population receives anticoagulation with warfarin. Therefore, many TBI patients may have a therapeutic coagulopathy. Many more adults take antiplatelet agents, and alcohol abuse can also affect coagulation status.

The presence of coagulopathy is an independent factor associated with the evolution of intracranial hematomas and with poor outcome. In addition, the presence of a coagulopathy will affect the appearance of an acute intracranial hemorrhage on head CT scan, i.e., it may appear hypo- or isodense rather than hyperdense, or a fluid level will be present.

Seizure Prophylaxis

Use of antiepileptic drugs (AEDs) is indicated if seizures accompany the presentation, the patient has a decreased level of consciousness (GCS $\leq$ 10), and/or the patient has an abnormal CT scan.

- Administer phenytoin 18 mg/kg IV at 25 mg/min and not more than 50 mg/min. If available, fosphenytoin can be administered with a dose of 18 mg/kg phenytoin-equivalents IV no faster than 150 mg/min.
- Patients who are still seizing need immediate treatment to stop their seizures. For full treatment regimen, see the *Status Epilepticus* protocol.
- Stop anticonvulsant medications after 7 days if there is no seizure activity.

Seizures that occur after head injury may be classified as early (within 7 days of TBI) or late (occurring $>$ 7 days after head injury). Post-traumatic seizures (PTSs) should be prevented. Particularly during the acute phase, a seizure can cloud the neurologic evaluation, aggravate intracranial pathology, and, in some patients, be the precipitating event that leads to herniation—in part because seizures are accompanied by changes in oxygen delivery and CBF, altered blood pressure, and increases in ICP.

In addition, prolonged seizures ($>$30 min) can cause secondary cerebral injury. PTSs that are clinically manifest occur in approximately 5 % of patients admitted to hospital with closed head injuries and in 15 % of those with severe TBI. In patients who undergo continuous electroencephalography (cEEG), the incidence appears to be higher, i.e., non-convulsive seizures are identified in up to 30 % of patients with severe TBI. There are several risk factors for PTS including a GCS $<$ 10, cortical contusion, subdural hematoma, epidural hematoma, intracerebral hematoma, depressed skull fracture, penetrating head wound, and seizure within 24 h of injury [16].

Phenytoin has proven efficacy in preventing early PTSs in TBI patients [16]. Phenytoin (or fosphenytoin) is therefore recommended as seizure prophylaxis in all patients admitted to hospital with moderate or severe TBI and an abnormal head CT scan. The seizure prophylaxis is stopped after 7 days if no seizure occurs. Late prophylactic therapy (i.e., after 7 days) in patients without evidence of a previous seizure is not effective and may cause harm to the patient. Therefore, prophylactic use of phenytoin or valproate is not recommended to prevent late PTSs [17]. There is a paucity of data on the use of levetiracetam in TBI patients.

ICP Monitoring

Though in many EDs, early placement of ICP monitors is difficult to achieve, if it is possible then these monitors can help to guide care. If an ICP monitor is not placed while the patient is in the ED then one should be inserted when in the ICU. General indications for ICP monitoring include:

- GCS 3–8 and abnormal CT scan.
- GCS 3–8 with normal CT and two or more of the following:
  - Age $>$ 40 years.
  - Motor posturing.
  - Systolic blood pressure (SBP) $<$90 mmHg.
- GCS 9–15 and CT scan:
  - Mass lesion (extra-axial $>$ 1 cm thick temporal contusion, intracranial hemorrhage, or ICH, $>$ 3 cm).
  - Effaced cisterns.
  - Shift $>$ 5 mm.
- Following craniotomy.
- Neurological examination cannot be followed (i.e., requires another surgical procedure, sedation).

Only part of the damage to the brain following TBI occurs at the moment of impact. Much of the poor outcome after TBI is associated with the development of secondary cerebral insults and brain injury that evolve over time. Intracranial hypertension develops in about 50 % of severe TBI patients [18]. An ICP $>$ 20 mmHg, particularly if sustained, is associated with significantly increased mortality [17].

As discussed above, it is difficult to diagnose elevated ICP by clinical means alone; the clinical exam early in TBI is more likely to reveal signs of brain herniation. Moreover, while CT findings indicate mass effect and risk for increased ICP, a reliable relationship does not exist between the admission CT and subsequent development of intracranial hypertension during a patient's ICU course [9]. Late ICP increases, particularly in a sedated patient, can only be detected by pupil dilation or changes in blood pressure and pulse associated with the Cushing response. Therefore, an ICP monitor is essential for timely diagnosis and targeted ICP treatment.

There are several published TBI guidelines that describe monitoring in adults after TBI, including the European Neurointensive Care and Emergency Medicine consensus on neurological monitoring [19], European Brain Injury Consortium [20], Italian Societies of Neurosurgeons and Intensivists [21], and the Brain Trauma Foundation (BTF) Guidelines [17]. The 2007 BTF Guidelines are currently the most widely accepted international guidelines for TBI management.

At present, there are no reliable non-invasive ICP monitors. ICP is best monitored invasively with a ventricular catheter or an intraparenchymal monitor [17, 22].

These devices are typically placed by neurosurgeons, yet, in some institutions, neurointensivists may insert ICP monitors with neurosurgical back-up [23].

Hemorrhage is the most common procedural complication, identified in 1 % of parenchymal monitors and 5 % of ventriculostomies. The majority is identified on imaging and is not of clinical importance. An INR $\leq$ 1.6 is considered safe to place a ventricular catheter after TBI [24]. A platelet count of $>$100,000 appears necessary to safely place an ICP monitor; however there is limited available literature on the topic. The use of FFP or other blood products is generally not required to place a parenchymal monitor [25] unless there is coagulopathy. There are no clinical outcome studies demonstrating that one monitoring technology is superior to others.

A ventriculostomy or external ventricular drain (EVD) to monitor ICP also allows therapeutic drainage of CSF. Control of elevated ICP is observed in approximately 50 % of patients where an EVD is inserted after other initial measures fail [26]. However, a ventricular catheter with an external transducer only allows intermittent ICP measurements when the drain is closed. Simultaneous ICP monitoring and CSF drainage is feasible with commercially available catheters that have a pressure transducer within their lumen. Ventricular catheters can be difficult to insert after TBI due to small ventricular size or shift. In addition, catheter blockage and displacement often occurs, causing ICP to be underestimated during simultaneous ICP monitoring and ventricular CSF drainage [27]. Bacterial ventriculitis and meningitis are also risks of these indwelling devices; antibiotic impregnated catheters reduce ventricular infection rates.

When an EVD is draining, it can miss episodes of increased ICP. Flushing with 1–2 ml of normal saline may be needed to restore catheter patency. This and other catheter manipulations, including CSF sampling, increase the risk of infection that is identified in 5–20 % of patients [28, 29]. Apart from a dose of antibiotics at the time of insertion, there is no role for continued antibiotic prophylaxis [28].

Intraparenchymal devices are easier to place than EVDs and are inserted into the brain parenchyma through a small burr hole. These monitors are secured in the skull by a specially designed, bolt-like cranial access device. Many bolts also permit insertion of other monitors (e.g., brain oxygen, brain temperature, microdialysis, and CBF probes). Parenchymal ICP monitors are typically placed into what appears to be normal white matter on the admission CT, in the non-dominant frontal lobe in diffuse injuries or on the side of maximal pathology in cases of focal abnormality. Different values may be obtained depending on device location and its proximity to a focal abnormality. Therefore, ICP values must be interpreted with the clinical examination and imaging studies.

There are several different intraparenchymal monitor technologies, including fiber-optic, strain gauge, and pneumatic technologies. Drift is very rare in fiber-optic catheters [25]. The risk associated with intraparenchymal ICP monitors is significantly less than that associated with external ventricular catheters; hemorrhage is reported in about 1 % of devices, and infections are reported even less frequently.

Continuous ICP recording is feasible. Technical complications (e.g., catheter breakage or dislodgement) may occur in approximately 4 % of cases. These complications often occur during transport, nursing maneuvers, or patient activities. (See ENLS protocols Elevated ICP and Herniation for further discussion on ICP management).

Surgery

The decision to perform surgery following TBI depends on the patient's neurological status and his/her CT scan findings, and it should be made after neurosurgical consultation. In general, extraxial (extradural or subdural) hemorrhage or mass $>$ 1 cm in thickness, midline shift $>$ 5 mm, ICH $>$ 3 cm in diameter, penetrating injury, depressed skull fracture, or intracranial hypertension (ICH) that fails to respond to medical therapy are all indications for surgery.

Recent evidence-based recommendations provide some direction [30–34]. Patients with a GCS $\leq$ 8 who demonstrate a large mass lesion upon CT should undergo prompt evacuation of the lesion. Effacement of the basal cisterns, particularly the perimesencephalic cisterns and $>$5 mm of midline shift, are regarded as CT findings of significant mass effect. Surgery must be expedited when there is deterioration on repeat evaluation, a focal neurological deficit, or the pupils show anisocoria or fixation/dilation.

In general, all acute extra-axial (i.e., extra-dural and subdural, hematomas $\geq$ 1 cm thick or associated with $\geq$5 mm of midline shift) should be considered surgical lesions regardless of the patient's clinical condition [30–34]. Similarly, intracerebral hematomas $>$ 50 mL in volume or $>$3 cm in diameter, particularly with mass effect, should be considered surgical lesions, though the decision to operate will also depend on hematoma location. A smaller size threshold is recommended for lesions in the posterior fossa, where even minimal enlargement of a small lesion can cause brainstem compression, or in the medial temporal lobe, where herniation may occur suddenly.

Relative contra-indications to this CT-driven approach to surgery include advanced patient age, particularly when there are multiple pre-morbid or associated medical problems, and severe coagulopathy. However, coagulopathy should not slow the procession to the operating room, since it can usually be corrected during surgery itself.

Depressed skull fractures that are displaced greater than the thickness of the skull table, and particularly those that are open or compound, typically require surgical repair. Possible exceptions include a depressed fracture immediately over a major venous sinus. Fractures of air sinuses, penetrating trauma, or craniofacial injuries all require special consideration.

Decompressive craniectomy (DC), either a unilateral hemicraniectomy or bilateral frontal craniectomy, can be used to treat ICH. There is strong evidence that DC in selected patients effectively controls ICP, though the effect on patient outcome is still being elucidated [35, 36]. DC may be considered when there are multiple temporal and frontal contusions.

Several questions exist about the way DC is performed:

- When should DC be performed? Some data suggest that patients do poorly if they undergo DC early in their course [37]. However, other data assert that if a patient arrives in the ED in poor condition and there is diffuse brain swelling or multiple contusions on head CT, DC is a reasonable option—in part because it may help avoid any side-effects associated with medical management of increased ICP.
- Should DC be routine when a large hematoma and, in particular, an acute subdural hematoma (SDH) is evacuated? Some surgeons routinely leave the bone flap off. However, if the brain not is markedly swollen, this may not be necessary in many cases. In addition, it is rarely indicated after evacuation of an acute epidural hematoma (EDH).
- What size of DC is adequate? Experimental data suggest DC may increase edema of the underlying brain if the procedure is inadequately performed or the decompression is too small [38]. The general rule is "the bigger, the better:" a bony diameter of at least 12–15 cm is necessary. This may require significant lateral head turning if the C-spine is cleared or a lateral position if there is a C-spine injury or it has not been excluded. Surgical decompression should extend to the middle fossa floor to achieve optimal decompression of the perimesencephalic cisterns [39, 40]. In addition, the dura must be adequately opened and augmentation duraplasty considered. In a bifrontal DC, this also requires dividing the falx far anteriorly. Finally, when DC is performed, the surgeon must plan for bone flap replacement. This includes use of dural substitutes to prevent adhesions forming between the dura and scalp and careful handling of the temporalis muscle to optimize long-term cosmetic results. The removed bone flap may be stored in a bone bank or subcutaneous pocket on the patient's abdomen. In general, bone flaps should be replaced as early as possible. This may help avoid the syndrome of trephine and alterations in CSF dynamics.

Table 2 Traumatic brain injury communication regarding assessment and referral

☐ Patient age
☐ Pre-injury health, if the information is available
☐ Mechanism of injury
☐ Post resuscitation GCS
☐ Pupil size, reaction, and symmetry
☐ Focal motor findings
☐ Coagulation status
☐ Other injuries
☐ State of C-spine: cleared, not cleared, injury
☐ CT scan results

Consultation

A neurosurgical consultation is appropriate for a patient with TBI if:

- GCS $\leq$ 13.
- The patient has seizures.
- There are lateralizing findings on neurological examination (e.g., unequal pupils or focal weakness).
- Head CT scan is abnormal.
- Head CT is not consistent with the clinical signs.
- Signs of CSF leak (clear fluid that tests positive for B-transferrin).
- Signs of basal skull fracture.
- Penetrating injury.
- Cerebrovascular injury.
- Suspected C-spine injury.

Communication

When communicating to an accepting or referring physician about this patient, consider including the key elements listed in Table 2.

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