

Head and Spinal Cord Injury: Diagnosis and Management

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- Traumatic brain injury • Intracranial pressure • Management
- Cerebrovascular injury • Pediatric • Spinal cord injury
- Vertebral injury

INTRACRANIAL PRESSURE MANAGEMENT

In modern neurotraumatology, intracranial pressure (ICP) management is a central tenet. The incidence of raised ICP or intracranial hypertension (ICHTN) is very high in modern neurotrauma units. In patients with demonstrable mass lesions, up to 63% may have ICHTN. By contrast, up to 13% of patients with a normal initial computed tomogram of head (CT Head) may have ICHTN.¹ Elevated ICP, in turn, is an independent predictor of worse outcomes in patients with severe traumatic brain injury (TBI).²⁻⁴ Increased ICP is also directly related to increased mortality in such patients.² In patients with sustained ICHTN, control of ICP within thresholds leads to improved outcomes.⁵ In addition, inability to control ICP is a predictor of poor outcomes.¹ Since the 1970s, significant reductions in morbidity and mortality have been achieved in patients with severe TBI with intensive management protocols. ICP control is an integral part of management protocols.⁶

Pathophysiology

In TBI, ICHTN can be caused by various factors. Cerebrospinal fluid (CSF) parameters may be responsible for up to one-third of ICP elevation; predominantly due to a decrease in CSF absorption and an increased resistance to outflow. Vascular factors

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may be responsible for the remaining two-thirds. Vascular factors include an increase in cerebral blood volume (CBV) via hyperemia, vasogenic edema due to damaged blood-brain barrier (BBB), cytotoxic edema, and ischemic edema.⁷ Hyperemia and edema may lead to changes in compliance of brain, leading to an abnormal volume-pressure response in the intracranial cavity.⁸ Brain compliance is represented by the pressure-volume index (PVI). In practical terms PVI can be understood as the amount of volume that must be added or removed to change the ICP tenfold (**Fig. 1**).⁹ The normal PVI is 26 ± 4 mL, that is, 26 mL of volume raises ICP from 1 to 10 mm Hg. The same volume will also raise the ICP from 10 to 100 mm Hg.¹⁰ Changes in PVI can occur in TBI independent of edema.¹¹ Changes in CSF outflow resistance may play an important role in altered PVI in addition to hyperemia and edema.

Indications for ICP Monitoring

With ubiquitous availability of imaging methods, most patients with severe TBI undergo imaging with CT Head at the time of presentation. Despite the advances in imaging technology, the presence of ICHTN cannot be reliably predicted by imaging studies alone.¹² The current recommendations regarding indications for ICP monitoring include all salvageable patients with a severe TBI (defined as Glasgow Coma Scale [GCS] score of 3–8 after resuscitation) and an abnormal computed tomography (CT) scan (defined as presence of hematomas, contusions, swelling, herniation, or compressed basal cisterns). Monitoring is also indicated in patients with severe TBI with a normal CT scan if 2 or more of the following are present: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure less than 90 mm Hg.⁶ The effect of untreated ICHTN on outcomes is unknown. However, implementation of protocol-based ICP management leads to improvements in outcomes.¹³ In addition, an increased frequency of monitoring has also been associated with improved outcomes.¹⁴ Prophylactic treatments without an ICP monitor, such as hyperventilation, mannitol, barbiturates, and paralysis, may result in poor outcomes. ICP monitoring is thus recommended in severe TBI because it leads to improved outcomes.⁶

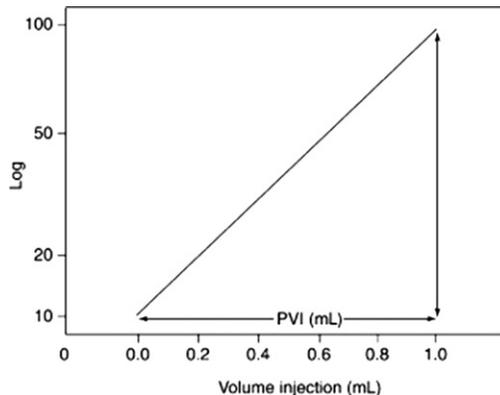


Fig. 1. The pressure-volume index (PVI) is plotted as a straight line on a semilogarithmic scale. This graph shows the relationship between pressure and volume in the cerebrospinal fluid space. (From Marmarou A, Beaumont A. *Physiology of the Cerebrospinal Fluid and Intracranial Pressure*. In: Winn HR, editor. *Youman's Neurological Surgery*. 4th edition. Elsevier Health Sciences; 2004. p.75–194; with permission.)

Goals of ICP Management

The debate regarding the numerical threshold for initiating therapy in monitored patients is ongoing. Based on available data, the current recommendation places the ICP threshold at 20 mm Hg.¹⁵ Both lower and higher thresholds have been proposed.^{2,16} In a large prospective study, logistic regression techniques were used to analyze the effect on ICP outcomes of increments of 5 mm Hg. A threshold at 20 mm Hg had the highest predictive value for outcomes.² Although there is no consensus on a definite threshold for treatment, groups reviewing the literature to suggest guidelines have settled on 20 to 25 mm Hg as a threshold for treatment. In the United States, recommendations by the Brain Trauma Foundation (BTF) suggest a treatment threshold of 20 mm Hg.¹⁵ The European Brain Injury Consortium suggests that "...generally ICP elevations above 20–25 mm Hg should be treated."¹⁷ For the pediatric population, although lower thresholds have been suggested, the current recommendations continue to support a threshold of 20 mm Hg.¹⁸

Approach to ICP Management

Current recommendations

Many well-designed clinical and experimental studies have approached the issue of ICP management in TBI. Consensus groups including a large body of practicing neurosurgeons, traumatologists, and intensivists have attempted to suggest guidelines based on current available evidence. The BTF published the third edition of evidence-based guidelines in 2007.¹⁹ Based on the strength of available evidence, levels of recommendations are classified as Level I, II, and III. Level I recommendations are based on the strongest evidence for effectiveness, and represent principles of patient management that reflect a high degree of clinical certainty. Level II recommendations reflect a moderate degree of clinical certainty. For Level III recommendations, the degree of clinical certainty is not established.²⁰ Guidelines for the management of pediatric TBI were published in 2003 as consensus statements by a varied group of surgical and pediatric societies. The recommendations are classed as Standards, Guidelines, and Options, based on degrees of certainty. Standards are accepted principles of patient management that reflect a high degree of clinical certainty; Guidelines are a particular strategy or range of management strategies that reflect a moderate clinical certainty. Options are the remaining strategies for patient management for which there is unclear clinical certainty.^{21–23}

Table 1 presents an overview of the current recommendations for management of ICP in TBI.

General measures for patients with TBI in neurotrauma units

The general methods include airway control, breathing, circulation (ABC protocol), normovolemia, sedation, analgesia, posture, temperature, and seizure prophylaxis (**Table 2**). These measures ensure adequate control of ICP in addition to ensuring adequate cerebral perfusion pressure (CPP). Adequate ventilation must be maintained to keep the SaO₂ above 90%, and mean arterial pressure should be maintained above 90 mm Hg.⁴⁷ Normal saline or hypertonic saline (3%–7.5% NaCl) may be used to maintain volume status. Sedative and analgesic medications frequently aid maintenance of ICP by blunting the effects of nursing care. Although useful in the short term in the intensive care unit (ICU) for managing patients with TBI, the effects of propofol on long-term prognosis remain unknown.⁴⁸ Posture is maintained to ensure adequate CPP while reducing ICP. The head is kept straight to prevent venous kinking⁴⁹ and the head of bed is elevated to 30°. Keeping the head of bed flat has been

Topic	Consensus Group	Recommendation	Level of Recommendation
Indications for ICP monitoring	BTF ⁶	ICP should be monitored in all salvageable patients with a severe TBI (GCS score of 3–8 after resuscitation) and an abnormal CT scan. An abnormal CT scan of the head is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns	Class II
		ICP monitoring is indicated in patients with severe TBI with a normal CT scan if 2 or more of the following features are noted at admission: age over 40 y, unilateral or bilateral motor posturing, or systolic blood pressure \leq 90 mm Hg	Class III
	Pediatric ²⁴	ICP monitoring is appropriate in infants and children with severe TBI (GCS score \leq 8)	Options
	EBIC ¹⁷	None	N/A
ICP threshold	BTF ¹⁵	Treatment should be initiated with ICP thresholds above 20 mm Hg	Level II
	Pediatric ¹⁸	Treatment for intracranial hypertension, defined as a pathologic elevation in ICP, should begin at an ICP of 20 mm Hg	Options
	EBIC ¹⁷	ICP elevations above 20–25 mm Hg should be treated	N/A
ICP monitoring technology	BTF ⁶	The available technologies are ranked as follows: 1. Intraventricular devices: fluid-coupled catheter with an external strain gauge 2. Intraventricular devices: micro strain gauge or fiber-optic 3. Parenchymal pressure transducer devices 4. Subdural devices 5. Subarachnoid fluid-coupled devices 6. Epidural devices	
	Pediatric ²⁵	In pediatric patients who require ICP monitoring, a ventricular catheter or an external strain gauge transducer or catheter tip pressure transducer device is an accurate and reliable method of monitoring ICP. A ventriculostomy catheter device also enables therapeutic CSF drainage	Options
	EBIC ¹⁷	None	N/A

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Table 1 (continued)			
Topic	Consensus Group	Recommendation	Level of Recommendation
Hyperosmolar therapy	BTF ²⁶	Mannitol is effective for control of raised ICP at doses of 0.25 g/kg to 1 g/kg body weight. Arterial hypotension (systolic blood pressure \leq 90 mm Hg) should be avoided	Level II
		Restrict mannitol use before ICP monitoring to patients with signs of transtentorial herniation or progressive neurologic deterioration not attributable to extracranial causes	Level III
	Pediatric ²⁷	Hypertonic saline is effective for control of increased ICP after severe head injury. Effective doses as a continuous infusion of 3% saline range between 0.1 and 1.0 mL/kg of body weight per hour, administered on a sliding scale. The minimum dose needed to maintain ICP \leq 20 mm Hg should be used. Mannitol is effective for control of increased ICP after severe TBI. Effective bolus doses range from 0.25 g/kg body weight to 1 g/kg body weight	Options
		Euvoemia should be maintained by fluid replacement. A Foley catheter is recommended in these patients to avoid bladder rupture Serum osmolarity should be maintained below 320 mOsm/L with mannitol use, whereas a level of 360 mOsm/L appears to be tolerated with hypertonic saline, even when used in combination with mannitol The choice of mannitol or hypertonic saline as a first-line hyperosmolar agent should be left to the treating physician	
EBIC ¹⁷	Preferably mannitol given repeatedly in bolus infusions, or as indicated by monitoring	N/A	
	Serum osmolarity should be maintained at \leq 315 mOsm/L Other agents, such as glycerol or sorbitol, are not advocated If osmotherapy has an insufficient effect, furosemide can be given additionally		

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Topic	Consensus Group	Recommendation	Level of Recommendation
Hyperventilation	BTF ²⁸	Prophylactic hyperventilation (Paco ₂ of 25 mm Hg or less) is not recommended	Level II
		Hyperventilation is recommended as a temporizing measure for the reduction of elevated ICP	Level III
	Pediatric ²⁹	Hyperventilation should be avoided during the first 24 h after injury when CBF is often critically reduced	Options
		If hyperventilation is used, jugular venous oxygen saturation (SjO ₂) or brain tissue oxygen tension (Pbro ₂) measurements are recommended to monitor oxygen delivery	
	EBIC ¹⁷	Mild or prophylactic hyperventilation (Paco ₂ ≤35 mm Hg) in children should be avoided	N/A
		Mild hyperventilation (Paco ₂ 30–35 mm Hg) may be considered for longer periods for intracranial hypertension refractory to sedation and analgesia, neuromuscular blockade, cerebrospinal fluid drainage, and hyperosmolar therapy	
		Aggressive hyperventilation (Paco ₂ ≤30 mm Hg) may be considered as a second tier option in the setting of refractory hypertension. CBF, jugular venous oxygen saturation, or brain tissue oxygen monitoring is suggested to help identify cerebral ischemia in this setting	
		Aggressive hyperventilation therapy titrated to clinical effect may be necessary for brief periods in cases of cerebral herniation or acute neurologic deterioration	
		If other methods of ICP control fail, more intensive hyperventilation (Paco ₂ ≤30 mm Hg) may be used, preferably with monitoring of cerebral oxygenation to detect cerebral ischemia	

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Topic	Consensus Group	Recommendation	Level of Recommendation
Anesthetics, analgesics, and sedatives	BTF ³⁰	Prophylactic administration of barbiturates to induce burst-suppression EEG is not recommended High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy Propofol is recommended for the control of ICP, but not for improvement in mortality or 6-mo outcome. High-dose propofol can produce significant morbidity	Level II
	Pediatric ³¹⁻⁴⁶	High-dose barbiturate therapy may be considered in hemodynamically stable patients with salvageable severe head injury and refractory intracranial hypertension If high-dose barbiturate therapy is used to treat refractory intracranial hypertension, appropriate hemodynamic monitoring and cardiovascular support are essential	Options
	EBIC	Sedation and analgesia are accepted methods of management of ICP	N/A

Abbreviations: BTF, Brain Trauma Foundation; CBF, cerebral blood flow; CT, computed tomography; EBIC, European Brain Injury Consortium; EEG, electroencephalography; GCS, Glasgow Coma Scale; ICP, intracranial pressure; N/A, not available; TBI, traumatic brain injury.

shown to increase ICP, whereas a higher elevation may lead to a reduced CPP.⁵⁰ Seizures may occur in up to 17% of patients with TBI. The risk seems to increase with the severity of injury.⁵¹ Seizures may increase the cerebral metabolic rate acutely and lead to raised ICP. However, the effect of seizures on long-term outcomes remains unclear.⁵² The current guidelines suggest the use of anticonvulsants to prevent early seizures after TBI. The use of anticonvulsants may not improve either the rate of late seizures or neurologic outcomes.⁵³

Specific measures for management of raised ICP

CSF drainage Ventriculostomies are frequently inserted in patients with TBI.⁵⁴ CSF drainage for ICP management is performed typically by releasing a few milliliters of CSF into the drainage bag. Immediate reduction in ICP is seen with drainage of fluid. Although CSF drainage decreases ICP in the short term, there is no improvement in cerebral circulation or oxygenation. The effect of CSF drainage on long-term outcomes remains unclear.⁵⁰

General Measure	Intervention	Level of Evidence
Airway	Secure early in the following patients: GCS <9, signs of respiratory distress, declining O ₂ saturation (<90%), increasing O ₂ requirement (FiO ₂ >50%), labored breathing Rising Pco ₂ (>45 mm Hg) in patients without COPD Patient unable to clear out secretions due to respiratory/oropharyngeal weakness Patients with severe agitation requiring sedation that may compromise	N/A
Breathing	Maintain Pao ₂ between 80 and 120 mm Hg, Paco ₂ 35–40 mm Hg, Sao ₂ >90%	Level II
Circulation	Maintain euolemia with goal CVP >5 mm Hg 0.9% NaCl at 1–3 mL/kg/h maintenance fluid 0.9% NaCl 0.5–1.0 L IV bolus as needed Maintain MAP >70 mm Hg and/or SBP >90 mm Hg Phenylephrine infusion at 10–1000 µg/min Norepinephrine infusion at 2–100 µg/min Dopamine infusion at 10–1000 µg/min Epinephrine infusion at 1–12 µg/min	N/A
Posture	HOB elevation, keep head at 30° (except in large ischemic stroke) Keep neck straight to prevent venous kinking	N/A
Temperature	Keep temperature below 38°C Acetaminophen 650 mg PO/PR every 4 h Cooling blanket Surface cooling Endovascular cooling	N/A
Sedation and analgesia	Propofol IV drip at 0.1–5 mg/kg/h Fentanyl IV drip at 50–200 µg/h Morphine 2–4 mg IVP every 2–4 h as needed Ativan 1–2 mg IVP every 4–6 h as needed	Level II
Seizure prophylaxis	Phenytoin (or fosphenytoin) 1 g LD IV then 100 mg every 8 h for 7 d Levetiracetam 500 mg PO twice a day for 7 d	Level II

The goals of therapy include maintenance of adequate ICP and CPP. Levels of evidence as determined by the BTF recommendations.

Abbreviations: COPD, chronic obstructive pulmonary disease; CVP, central venous pressure; HOB, head of bed; IV, intravenous; IVP, intravenous perfusion; LD, loading dose; MAP, mean arterial pressure; N/A, not available; PO, by mouth; PR, per rectum; SBP, systolic blood pressure.

Data from Bratton SL, Chestnut RM, Ghajar J, et al. Guidelines for the management of severe traumatic brain injury. XI. Anesthetics, analgesics, and sedatives. *J Neurotrauma* 2007;24(Suppl 1): S71–6.

Pharmacologic therapy Hyperosmolar therapy remains the cornerstone for ICP management in modern neurotrauma units. Hyperosmolar therapy including mannitol and hypertonic saline are used in 73% to 83% of units taking care of patients with severe TBI.⁵⁴ The use of corticosteroids in acute TBI to control ICP is not recommended,⁵⁵ and this practice is falling out of favor.⁵⁴ Opioids, midazolam, and barbiturates have been used to control ICP. Bolus opioid infusions may actually result in raised ICP by raising cerebral blood flow (CBF). Although they are routinely used to maintain

analgesia (fentanyl and remifentanyl), their use to control spikes in ICP is not recommended. Both midazolam and barbiturates have no effect on ICP. In addition, midazolam may decrease mean arterial pressure and CPP.⁵⁶

Mannitol Mannitol is an isomer of sorbitol. Although it is commonly thought that mannitol acts by drawing excess fluid from the cranial cavity, the precise mechanism of action is unknown.⁵⁶ The immediate reduction in ICP probably results from a reduction of blood viscosity and plasma expansion. The effect lasts for 3 to 4 hours. It is typically administered in doses ranging from 0.25 to 1 mg/kg,^{26,57} though there might be some support for using higher doses.⁵⁶ Monitoring of serum osmolarity is recommended when administering mannitol. The effectiveness of mannitol in reducing ICP is related to the osmolarity. In addition, serum osmolarity greater than 320 mOsm may predispose to renal failure.⁵⁸

Hypertonic saline Hypertonic saline (3%–7.5% NaCl solution) is being increasingly used in neurotrauma units for the management of ICP. In addition to a reduction in ICP, hypertonic saline also expands the intravascular volume, increases cardiac output, improves CPP, and improves absorption of CSF.⁵⁹ The mechanism of action for hypertonic saline is likely attributable to a hyperosmolar effect. Hypertonic saline may have advantages over mannitol when used in severe TBI. Unlike mannitol, repeated administration does not diminish the ICP-lowering effect, and does not cause a rebound increase in ICP. It may also be used during intractable ICHTN unresponsive to mannitol and barbiturates.⁶⁰ There is strong evidence that hypertonic saline is superior to mannitol in its ability to lower ICP.⁵⁶ Hypertonic saline may be used as a continuous infusion of 3% solution or in bolus doses of 7.5% to 23.5% solution. Continuous infusions are typically given at a rate of 0.1 to 1.0 mL/kg/h.

Barbiturates Barbiturates induce a suppression of cerebral metabolic rate. In patients with an intact flow-metabolism coupling, a reduction in the metabolic rate leads to a reduction in blood flow and consequently reduced ICP.⁵⁹ In addition, barbiturates offer protection during episodes of cerebral hypoxia.⁶¹ Barbiturates are used less frequently for ICP control than other modalities,⁵⁴ partly because of the conflicting evidence regarding the effect of barbiturates in improving outcomes in TBI.⁵⁶ Systemic hypotension is a constant companion to barbiturate use, and these agents may also cause cardiac depression, sepsis, electrolyte abnormalities, and renal dysfunction.⁵⁹ At present, barbiturate therapy for management of ICP is recommended for refractory ICHTN only. Attention must be given to prevent hemodynamic instability.^{30,56,62} When used in clinical situations, pentobarbital is given as an infusion at the rate of 1 to 8 mg/kg/h with loading doses of 1 to 5 mg every 15 minutes to achieve acute control of ICP. Alternatively, thiopental may be used at the rate of 1 to 6 mg/kg/h with bolus doses of 300 to 400 mg every 30 minutes (total 4 g over 1–5 hours).⁵⁹ There is no difference between these agents when used to control ICP.⁵⁶

Chemical paralysis In ICHTN due to agitation, posturing, or coughing, nondepolarizing muscle paralytics may reduce ICP acutely. Paralytics are used along with sedatives and analgesics in ventilated patients.⁶³ With the use of paralytics neurologic status cannot be easily assessed, and necessitates withholding paralytics daily for assessment. Routine paralytic use in all patients with TBI is not recommended, due to increased incidence of extracranial adverse effects such as pneumonia and sepsis. The use of paralytics has also been associated with an increased length of stay in the ICU and an increased incidence of critical illness myopathy.^{64,65}

Hyperventilation Hyperventilation induces hypocapnia, leading to cerebral vasoconstriction. The subsequent reduction in CBV causes a reduction in ICP. An increased oxygen extraction compensates for a reduction in flow, thereby preserving normal metabolic processes to continue.⁵⁰ Aggressive hyperventilation to reduce P_{aCO_2} to less than 25 mm Hg had been the cornerstone of ICP management for more than 2 decades. However, recent studies have questioned the use of chronic hyperventilation. Evidence for inadequate increase in oxygen extraction⁶⁶ may mean that hyperventilation may predispose to cerebral ischemia.^{28,67} This finding has been confirmed in metabolic imaging of patients undergoing hyperventilation. Despite improvements in ICP and CPP, larger volumes of severely hypoperfused regions have been observed with hyperventilation.⁶⁸ Chronic hyperventilation leads to significantly worse outcomes, although some of this effect may be attenuated with the use of tromethamine (THAM) intravenously (dose: THAM [ml] = body weight [kg] × base deficit).⁶⁹ At present, hyperventilation is only recommended as a temporizing measure for the reduction of ICP with measurements of jugular venous oxygen saturation (S_{jO_2}) or brain tissue oxygen tension (P_{brO_2}) measurements to monitor oxygen delivery. In addition, hyperventilation is avoided during the first 24 hours after injury when CBF is often critically reduced.²⁸

Hypothermia Induced hypothermia reduces ICP by a reduction in the cerebral metabolic rate. In addition to a reduction of CBF, hypothermia may reduce inflammatory responses as well as glutamate and free radicals. Prolonged hypothermia may be associated with adverse effects including arrhythmias, coagulopathies, sepsis, and pneumonia.^{50,59} Methods of hypothermic intervention include systemic hypothermia or selective hypothermia. Systemic hypothermia is achieved by surface cooling,⁷⁰ by endovascular catheters,⁷¹ and occasionally by gastric lavage.⁷² Alternatively, selective hypothermia is achieved by using a cooling cap or band.⁷³ The target temperatures may range from mild (34°–36°C) to moderate (32°–34°C) hypothermia. It is suggested that hypothermia may have higher chances of reducing mortality when cooling is maintained for more than 48 hours.⁷⁴ Hypothermia is maintained for 24 to 72 hours or longer depending on ICP response. Slow, controlled rewarming over 24 hours is critical, because of the development of rebound cerebral edema, hypotension, and electrolyte abnormalities. The current recommendations recognize that prophylactic hypothermia is not significantly associated with decreased mortality. It is noted that preliminary findings suggest a greater decrease in mortality risk is observed when target temperatures are maintained for more than 48 hours. Prophylactic hypothermia has also been associated with significantly higher Glasgow Outcome Scale scores when compared with scores for normothermic controls.⁷⁴

Surgical therapies In cases of intractable ICHTN, removal of a section of skull is a drastic measure undertaken to control ICP.⁷⁵ Decompressive craniectomy involves removing a large portion of skull in the absence of a large mass-occupying lesion such as subdural hematoma, epidural hematoma, or cerebral contusion. Decompression results in an immediate drop in ICP, but is only performed after all other measures have been exhausted.⁵⁰ Recently there has been an increased interest in assessing the outcomes following decompressive craniectomy for intractable ICHTN. When compared with early, prophylactic surgery, improved outcomes were observed when decompression was performed in response to intractable ICHTN.⁷⁶ The timing for performing decompression is critical. The emphasis is on performing the decompression before irreversible ischemic damage occurs.⁷⁷ Although the immediate goal of reducing ICP is met in most patients (85%),⁷⁵ the effect on outcomes remains

to be clarified.⁵⁰ Due to the surgical, drastic nature of the procedure, there is a paucity of randomized controlled trial (RCT) data regarding efficacy and safety. The two published randomized studies report on the importance of timing (early) and size (larger) of decompressive craniectomy in patients with severe TBI.^{78,79} At present, there is strong evidence that decompressive craniectomy reduces elevated ICP immediately after surgery but does not improve long-term outcomes in children. There is also moderate evidence, based on one RCT, that a larger bone flap results in greater reduction of ICP, with limited evidence that patients with a GCS less than 6 show no improvement. There was conflicting evidence as to whether a decompressive craniectomy affects long-term outcomes.^{50,78,79}

CEREBROVASCULAR TRAUMA

Traumatic injury to the intracranial or extracranial vasculature is rare and can be life threatening. Vascular trauma can be caused by either blunt or penetrating injuries. The incidence of cerebrovascular trauma has been reported as between 0.1% and 1.5%.^{75–79} The cervical carotid artery is more frequently involved than the vertebral artery.^{77,79,80} Blunt trauma is more common than penetrating injuries, and motor vehicle accidents remain the most common cause of blunt trauma, accounting for 40% to 70% of cases.⁷⁵ Mortality for blunt carotid trauma has been reported as between 20% and 40%.⁸¹ Cerebrovascular injury can range from a simple intimal tear to a complete transection of the vessel. Both arterial and venous sides of the vasculature can be affected. On the arterial side, dissections, traumatic aneurysms, or arteriovenous fistulae may develop. On the venous side, one may encounter a dural sinus tear or venous thrombosis.⁸²

Traumatic Dissection

An arterial dissection begins with a small intimal tear, allowing blood to enter the wall of the artery, splitting its layers, and resulting in a stenosis, irregularity of the wall or, occasionally, an aneurysmal dilatation.^{82,83} Dissections occur in approximately 1% of all patients with blunt force trauma.⁸⁴ Intracranial dissections are rare; if present, they involve the supraclinoid segment of the internal carotid artery (ICA).⁸² Extracranial dissections are the most common, and carotid involvement is twice as likely to occur than vertebral artery involvement.⁷⁹ Carotid injury is usually associated with TBI and/or basal skull fractures, whereas vertebral dissections are associated with cervical spine fractures.⁸⁵ There are several other risk factors and accompanying physical findings that may be present in traumatic dissections.⁸⁶ Screening protocols have been developed to identify patients at risk (**Box 1**).^{75,80} The use of screening protocols combined with imaging studies has led to an increase in the identification of these lesions. Contrast-enhanced CT and magnetic resonance (MR) imaging might demonstrate an intramural thrombus, narrowing of the arterial lumen, and/or an intimal flap, pointing to the presence of a true and false lumen; there may also be parenchymal cerebral or cerebellar changes.^{78,87} Angiographically, dissections present with an irregular lumen with stenosis or occlusion, intimal flaps, tapering of the lumen of the ICA or slow flow from the ICA to the middle cerebral artery.^{88,89} The degree of luminal compromise can range from a mild irregularity to a complete occlusion. At the carotid level, dissections usually spare the bulb, differentiating this from atherosclerotic disease (**Fig. 2**).⁸⁸ Carotid dissections usually occur at the cervical and petrous segments whereas vertebral dissections occur at the V3 segment, where the vertebral artery enters the foramen magnum and is not protected by bony foramina.^{90,91}

Box 1**Screening protocols for blunt cerebrovascular trauma***Denver Criteria*

- Cervical spine fracture
- Unexplained neurologic deficit
- Skull base fracture into carotid canal
- LeFort II or III fracture
- Expanding cervical hematoma
- Cervical bruit
- Ischemic stroke on secondary CT scan
- Head trauma with GCS <6
- Near hanging with anoxic injury

Memphis Criteria

- Cervical spine fracture
- Unexplained neurologic deficit
- Skull base fracture into carotid canal
- LeFort II or III fracture
- Horner syndrome
- Neck soft-tissue injury



Fig. 2. Cervical carotid dissection. Digital subtraction angiography showing tapering of the proximal third of the right internal carotid artery with complete occlusion above the carotid bulb.

Management is primarily aimed at preventing thromboembolic complications and maintaining patency of the involved artery.^{92,93} Whereas 85% of spontaneous dissections improve or resolve on follow-up studies, only 55% of trauma-related dissections improve and 25% will progress to complete occlusion.⁹⁴ The data on the management for blunt cerebrovascular trauma are limited. Most investigators agree that some form of antithrombotic treatment is appropriate. Intravenous heparin is the most widely reported regimen, with complication rates ranging from 8% to 16%^{77,80,95–97} and 30% to 36% in patients who harbor some kind of contraindication for anticoagulant therapy.^{98,99} Recent recommendations describe a target partial thromboplastin time of 40 to 50 seconds.^{80,100}

Antiplatelet therapy (single or dual with clopidogrel) is an alternative that may avoid the risks associated with systemic heparinization.^{96,99,101,102} Several studies have reported similar or better neurologic outcomes in comparison with heparin.^{98,99,101} At present, endovascular or surgical interventions are reserved for patients who have resilient or recurrent symptoms despite medical management. Endovascular interventions are the nonmedical treatment of choice, and options include stenting, with balloon or self-expandable stents, and/or intra-arterial thrombolysis for acute strokes.

Traumatic Aneurysms

Traumatic aneurysms are rare lesions caused by dissection or rupture of the arterial wall as a result of blunt or penetrating trauma. Wall damage forms a contained hematoma or “pseudoaneurysm.” These lesions account for fewer than 1% of all intracranial aneurysms in adults, but their proportion increases in children to about 20%.^{103,104} Intracranial traumatic aneurysms can be located at the skull base (petrous, cavernous, supraclinoid segments), subcortical (close to falx or tentorium), or be distally associated with skull fractures or hematomas.^{82,97} The most common site involved is the cavernous segment of the ICA, which is associated with base-of-the-skull fractures. Extracranial traumatic aneurysms also occur secondary to blunt, penetrating, or iatrogenic trauma.¹⁰⁵ Blunt trauma is commonly caused by rotatory hyperextension, strangulation, or fracture of the mandible. Penetrating trauma-related aneurysms are more common, and result from stabbing or gunshot wounds.¹⁰⁶

Traumatic intracranial aneurysms may present symptoms acutely or in a delayed fashion, even weeks after the event.^{107,108} These lesions often present with intracranial hemorrhage (intraparenchymal, subdural, intraventricular, or subarachnoid), with some cases causing life-threatening epistaxis due to bleeding of the ICA into the sphenoid sinus.^{107,109–111}

Diagnosis of traumatic aneurysms relies on a high level of clinical suspicion. A trauma patient who presents with delayed or unexplained neurologic deterioration, unusual location of cerebral hemorrhage, and/or large epistaxis warrants further investigation. Although CT angiography is a valid imaging study, further angiographic investigation is still necessary.^{112,113} The presence of a skull-base fracture involving the carotid canal also should raise suspicion for a traumatic pseudoaneurysm.¹¹⁴ Time between trauma and the rupture of the aneurysm can range from days to months; half of the cases present with rupture within 3 weeks.^{115–117} Certain angiographic features can point to the traumatic origin of an aneurysm, delayed filling and emptying of the aneurysm, location (peripheral and away from branching points), irregular contour, absence of a neck, and/or the history of the patient (**Fig. 3**).^{82,118}

Treatment is recommended in all traumatic intracranial aneurysms because mortality rates range between 34% and 54%.^{86,115–117,119–122} Treatment strategy should consider that this “aneurysm” is an extravascular pouch and that the walls are made by thrombus and not arterial wall layers, making it more fragile.^{82,123–125}

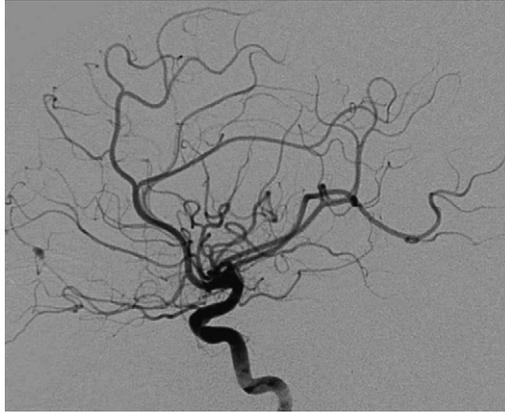


Fig. 3. Traumatic pseudoaneurysm. Lateral digital subtraction angiography showing a distal frontal pseudoaneurysm of the anterior cerebral artery. The aneurysm is distally located, irregular in shape, and away from branching points in a patient with history of head trauma.

The goal of treatment should be immediate proximal occlusion of the parent artery by embolization (coils, balloons, glue) or surgical vessel occlusion.^{82,86,126} Endovascular occlusion of the parent vessel, selective embolization of the aneurysm, and stent placement have shown favorable results in the treatment of traumatic aneurysms.^{104,105,123,127–129}

Traumatic Arteriovenous Fistulae

An arteriovenous fistula (AVF) is an abnormal communication between an artery and a vein, and tends to occur where these two structures are in close proximity. Direct carotid cavernous fistulae (CCF) are the most common traumatic AVF, and are usually secondary to a skull-base fracture; however, iatrogenic AVF after transsphenoid surgery might also be encountered.^{77,82,86} The fistula develops when the ICA and/or its meningeal branches tear within the cavernous sinus, producing a direct communication with the cavernous sinus.¹³⁰

Clinical findings include objective vascular bruit, pulsating exophthalmos, chemosis, and ophthalmoplegia.¹³¹ Loss of vision may occur, secondary to increased intra-ocular pressure related to venous congestion.¹³²

Imaging should include a high-resolution CT scan to assess for fractures. Approximately 75% of all patients have a skull-base fracture associated with CCF, and the incidence of a CCF in a patient with a sphenoid bone fracture is around 3% to 5%.^{133–136} CT angiography or MR angiography usually can confirm the diagnosis; findings include enlargement and early enhancement of the cavernous sinus and superior ophthalmic vein. Angiography is necessary to accurately define the location of the defect and to look for high-risk features such as retrograde cortical venous drainage, which entails a high risk for hemorrhage and warrants early treatment.^{82,104,137} Endovascular management is the treatment of choice; intervention can be performed using coils and/or liquid embolic material, covered stent repair, or ICA occlusion (**Fig. 4**).^{104,105,124,138–141}

Traumatic Venous Lesions

Traumatic venous lesions are often overlooked, and might be more frequent than is reported.⁸ Venous thrombosis may develop following penetrating or blunt trauma

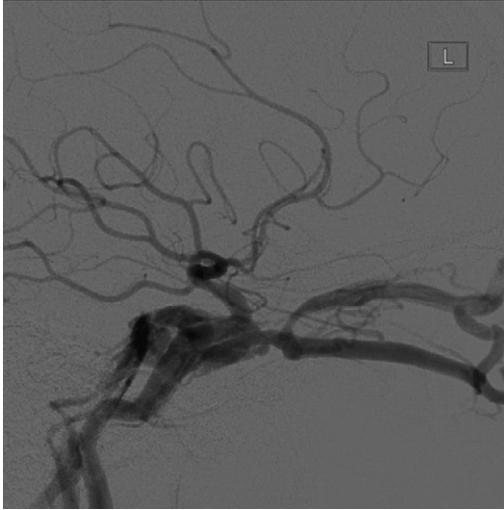


Fig. 4. Direct carotid cavernous fistula. Lateral digital subtraction angiography of the left internal carotid artery showing a direct communication with the cavernous sinus with venous drainage to the superior and inferior ophthalmic veins and inferior petrosal sinus. The patient had a history of base skull fracture after a motor vehicle accident.

due to tear or laceration of dural sinuses, fractures, extrinsic compression, or increased pressure.^{139,142–144} An acute blood clot or thrombus might extend, leading to venous hypertension and increased ICP.¹⁴⁵ The clinical presentation is variable but often includes headaches, seizures, visual disturbances, and/or papilledema, among others.¹⁴⁵ Clinical presentation is closely related to location and extent of the cerebral venous thrombosis (CVT). If CVT is suspected clinically, imaging is needed for confirmation.¹⁴⁶ Acute thrombosis within the dural sinuses can be identified as a high-attenuation lesion on nonenhanced CT. If thrombosis of the superior sagittal sinus occurs, a triangular filling defect (empty delta sign) can be seen on postcontrast images. MR imaging is the modality of choice for CVT; it may be difficult to recognize an acute thrombus on conventional sequences, and early changes, such as edema and gyral swelling, are subtle.¹⁴⁶ Stroke lesions that do not correspond to an arterial territory suggest CVT; hemorrhage often is an accompanying factor.^{82,146,147} On angiography there may be a delayed transit time, filling of collateral veins, or nonvisualization of the affected structure.^{82,137}

Treatment is aimed at avoiding the progression of the thrombosis. Intravenous heparin is considered standard therapy.^{148,149} Patients who present with rapidly progressive thrombosis should be considered for endovascular management. Direct thrombolytic infusion by a percutaneous or retrograde venous approach is feasible, with the potential benefit of avoiding systemic hemorrhagic effects caused by intravenous heparinization.^{150–154}

NEUROSURGICAL TRAUMA IN CHILDREN

Trauma is the leading cause of death in children 18 years and younger. The major determinant of morbidity and mortality in pediatric trauma is TBI. TBI is broadly defined as an alteration of brain function caused by an external force. It can range from mild or concussive to severe or comatose. There are approximately half a million emergency department visits for TBI in children of age 0 to 14 years each year (**Table 3**).

Age Group	Emergency Department Visits	Hospitalizations	Deaths	Total
Children (0–14 years)	473,947	35,136	2174	511,527
Older adults (≥65 years)	141,998	81,499	14,347	237,844

The estimated average annual number of TBIs that occur among children aged 0 to 14 years is 511,257. By contrast, the number of TBIs in adults aged 65 years and older is 237,844. TBI-related emergency department visits accounted for a larger proportion in children (92.7%) than in older adults (59.7%).

Data from Eisenberg HM, Frankowski RF, Contant CF, et al. High-dose barbiturate control of elevated intracranial pressure in patients with severe head injury. J Neurosurg 1988;69(1):15–23.

Falls are the major external force causing TBI in children, especially those aged 0 to 4 years, followed by events in which a child is struck by or against an object, including collision with a moving or stationary object (Fig. 5). TBI is often referred to as a “silent epidemic” because complications, such as memory and learning difficulties, that result from TBI are not always immediately apparent.¹⁵⁵ This section discusses the diagnosis and management of the most common neurosurgical injuries that can occur as a result of the primary impact in TBI.

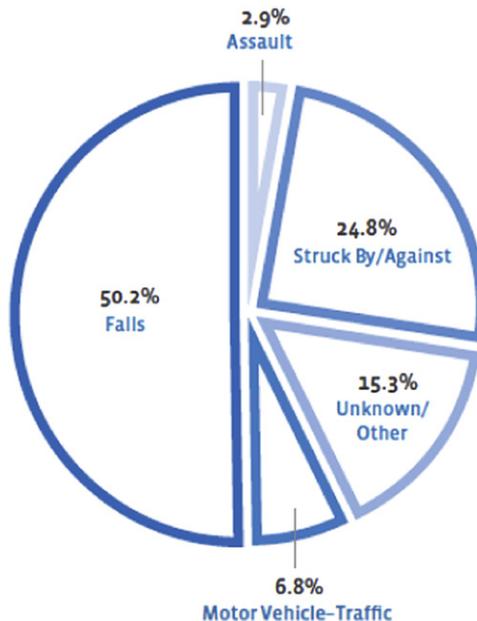


Fig. 5. Estimated average percentage of annual traumatic brain injury-combined emergency department visits, hospitalizations, and deaths among children 0 to 14 years, by external cause, United States, 2002 to 2006.

Skull Fractures

Skull fractures (**Fig. 6**) can occur in up to 20% of children with head injuries, yet the majority will not require any intervention. However, the presence of a fracture on imaging studies should raise concern of an underlying brain injury; therefore a CT scan is the best tool for initial diagnosis and management. Plain skull radiographs, while very good for diagnosing fractures, are unable to show underlying brain injury or hemorrhage that may need immediate attention. Most fractures in children are closed, simple, linear fractures involving the parietal bone that will heal without intervention. Approximately 25% of skull fractures will be depressed because of a focal impact on the skull.^{156,157} These fractures most commonly involve the parietal bone and will be greenstick fractures, whereby the bone is connected but fixed in a new depressed position. Examination will reveal subgaleal swelling and tenderness. In infants and young children a fracture line may be palpable, and in depressed fractures a small dent may be felt. Treatment of depressed greenstick skull fractures in infants remains somewhat controversial. Without intervention, many of these fractures will elevate over time, due to rapid intracranial and skull growth. However, if the depression is large or if it does not improve with observation, surgical elevation of the fracture should be performed. In older children and adolescents, depressed fractures should be elevated surgically if the fragment is depressed 1 cm or more from the skull.^{158,159}

Growing Skull Fractures (Leptomeningeal Cyst)

With complex, comminuted skull fractures, laceration of the dura with or without underlying injury to the cortex can occur. When dural lacerations occur with these complex fractures, a child can be at risk for the development of a growing skull fracture (**Fig. 7**). The pathogenesis of the growing fracture is from CSF pulsations through the lacerated dura causing herniation of the cerebral cortex into the epidural space.¹⁶⁰ This process causes the fracture sites to push apart, often with resorption of the fractured bone. Such a situation most frequently occurs in children younger than 2 years, in whom brain growth is extremely rapid. Children will present with pulsatile masses or fluid collections at the site of the fracture, and CT scan will demonstrate herniation of cerebral tissue with increased separation of the fractured bone. All will require surgical repair of the lacerated dura with plating of the fracture site.

Intracranial Hemorrhage

Intracranial hemorrhage can be classified as extra-axial or intra-axial. Extra-axial hemorrhages are located beneath the skull but outside of the cerebral cortex, whereas

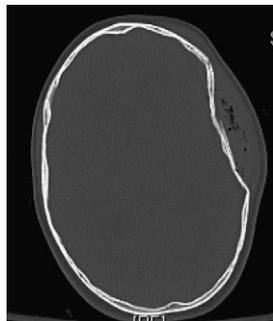


Fig. 6. Noncontrast computed tomography (CT) scan of bone window, showing left fronto-temporal depressed skull fracture.

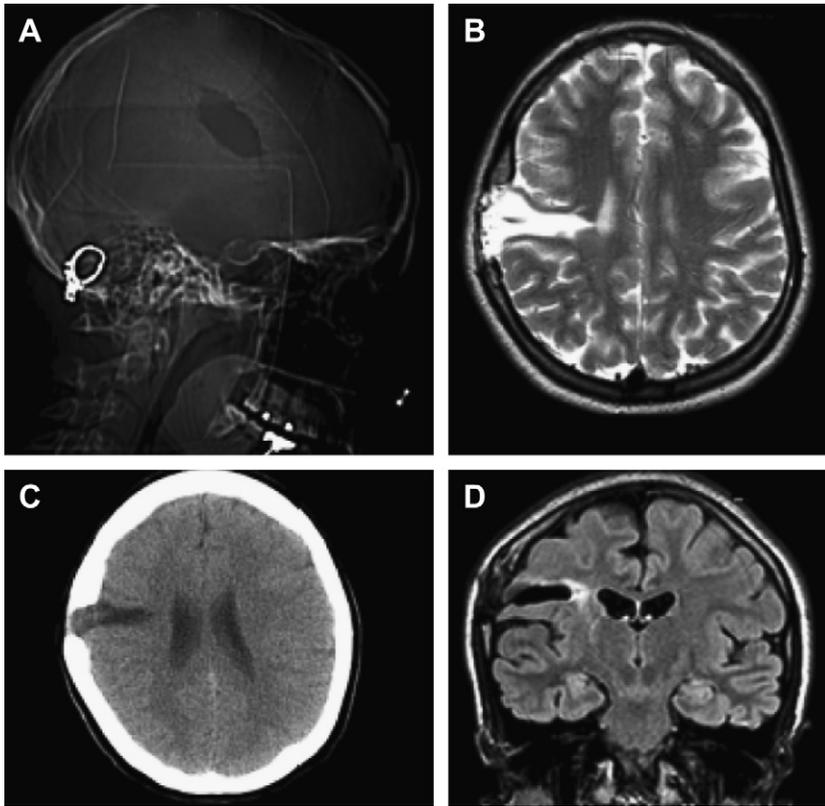


Fig. 7. CT of the brain revealed a skull defect on the localizing sagittal image (A), with axial views demonstrating underlying gliosis and demarcated volume loss within the lateral right sensorimotor cortex (B). The cerebral volume loss and bony defect are striking on the subsequent axial T2-weighted (C) and coronal fluid-attenuated inversion recovery (D) magnetic resonance imaging sequences.

intra-axial hemorrhages are within the cerebral cortex. All present with similar signs and symptoms such as headache, nausea, vomiting, hemiplegia or hemiparesis, and altered mental status, indicating raised ICP. CT scan is the best and quickest imaging study to assess for these injuries in the acute trauma setting.

Epidural Hematoma

Epidural hematomas are hemorrhages located between the skull and the dura (**Fig. 8**). These hematomas are most frequently located in the supratentorial region, usually in the temporal area. Most often these hemorrhages occur from rupture and bleeding of the middle meningeal artery, which runs just under the temporal bone in the area of the pterion. Frequently the patient will have a lucid interval after the initial injury, followed by a quick decline in his or her neurologic examination. Almost all patients will require emergent surgical evacuation because these hematomas can rapidly expand, causing mass effect and possible cerebral herniation.

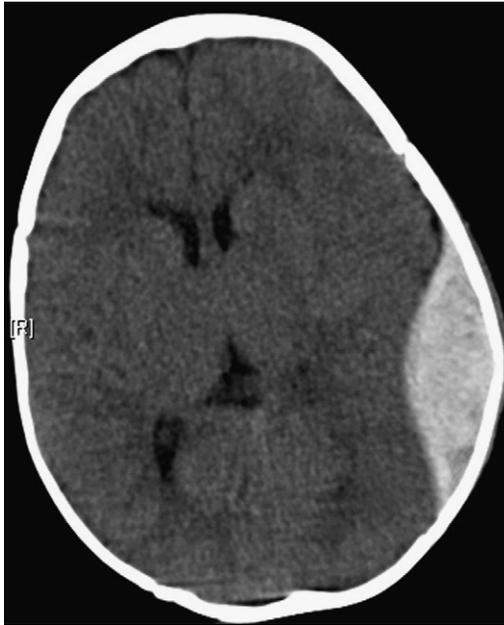


Fig. 8. Non contrast CT scan showing left-sided epidural hematoma.

Subdural Hematoma

A subdural hematoma is a hemorrhage that occurs between the dura and the pial surface of the brain (**Fig. 9**). It is often a result of tearing of bridging veins on the surface of the cortex that drain to a venous sinus. Often these patients have more severe brain



Fig. 9. Noncontrast CT scan showing large left-sided subdural hematoma with mass effect and midline shift.

injury and are comatose shortly after the initial injury. Surgical management with urgent evacuation of the clot is usually required, as these hemorrhages can cause significant mass effect and midline shift, with possible cerebral herniation.

Intracerebral Hemorrhage (Cerebral Contusions)

These contusions are areas of bleeding within the cortex itself (Fig. 10). Such hemorrhages are relatively uncommon in infants after trauma. Older children develop cerebral contusions most often from acceleration/deceleration injuries, most commonly in the frontal and temporal lobes. Cerebral contusions can be observed and conservatively managed if they remain small; this requires serial imaging and neurologic examination to monitor the patient. Some contusions will blossom over the first 24 to 48 hours after the initial injury. If a cerebral contusion becomes large and/or exhibits significant mass effect on the surrounding brain and is easily accessible, surgical evacuation of the intracerebral hemorrhage may be necessary.

Diffuse Axonal Injury

Diffuse axonal injury (DAI) is a shearing injury to the axons located in the deep white matter, arising from severe deceleration injuries. Children with DAI are unconscious on arrival at the hospital, and CT does not show any evidence of significant intracranial pathology. Other symptoms include abnormal motor signs, even decorticate or decerebrate posturing, and papillary dysfunction. CT scans do not readily show DAI, and MR imaging is much more sensitive. MR imaging will reveal multifocal hyperintense foci at the gray-white interfaces, corpus callosum, and brainstem on T2-weighted images.¹⁶¹ Treatment involves ICP monitoring and clinical examination, although most DAI patients will have normal ICP. Recovery from DAI is variable among patients, and can take weeks to months. Factors associated with a poor outcome in DAI are bilateral unreactive pupils, multiple corpus callosum or brainstem lesions, and older age.¹⁶²

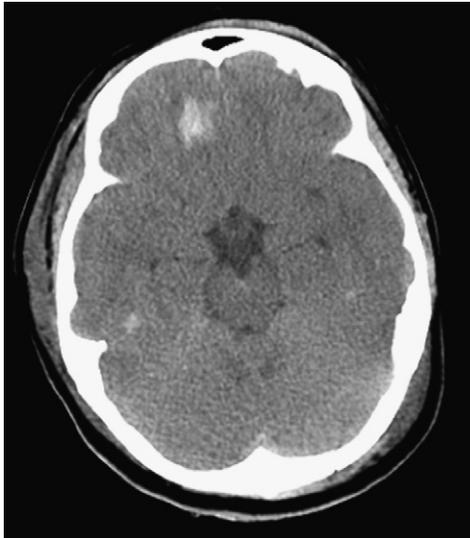


Fig. 10. Noncontrast CT scan showing right frontal intracerebral hemorrhage and punctate bilateral temporal lobe hemorrhages.

Spinal Cord Injury Without Radiographic Abnormality

Spinal cord injury without radiographic abnormality (SCIWORA) is a common and difficult problem attributable to lack of radiographic abnormalities. It has a wide range of incidence, 5% to 70%, most likely due to a varied clinical awareness of the syndrome.¹⁶³ Patients with true SCIWORA have spinal cord injury and have a detailed radiologic evaluation, including plain radiographs, flexion/extension views, CT and MR studies, which do not show any structural cause for the spinal cord injury. SCIWORA accounts for up to two-thirds of severe cervical injuries in children younger than 8 years, and is most common in the upper cervical and thoracic regions of the spinal cord.¹⁶⁴ The elasticity of the pediatric spine allows for a transient subluxation at the time of injury and elastic recoil to a normal alignment at presentation. Children with this injury may have a fracture through the cartilaginous endplates, which cannot be visualized on plain films or an unrecognized interspinous ligamentous injury. Flexion/extension views in an awake patient may demonstrate these underlying injuries. Most patients will present acutely with a complete cord injury, but some patients may present in a delayed fashion up to 4 days after the initial injury.¹⁶⁴ Treatment of SCIWORA includes spinal immobilization, usually a cervical collar, for 3 months.¹⁶⁵ Before immobilization is discontinued, dynamic films, namely flexion/extension radiographs, should be obtained to confirm spinal stability. Neurologic outcome is based on the presence and degree of spinal cord signal changes at the time of injury.^{156,157,164,166} Those patients with no evidence of cord signal changes have an excellent chance for full neurologic recovery. Patients with signal changes consistent with edema or microhemorrhages are associated with significant improvement of function with time. Cord disruption or frank hematomyelia is most often associated with severe neurologic injury and poor chance of recovery of function.^{156,157}

SPINAL CORD INJURY

Acute traumatic spinal cord injury (SCI) remains one of the most devastating events that can occur in an individual. Not only is SCI associated with the obvious harsh physical setbacks of the injury itself, but also permanently affects the individual's social, economic, and psychological realms. According to the National Spinal Cord Injury Statistical Center in Birmingham, Alabama, it is estimated that the annual incidence of SCI, not including those who die at the scene of the accident, is approximately 40 cases per million population in the United States (or approximately 12,000 new cases each year). Since 2005, motor vehicle crashes have accounted for approximately 42% of reported SCI cases (**Fig. 11**).

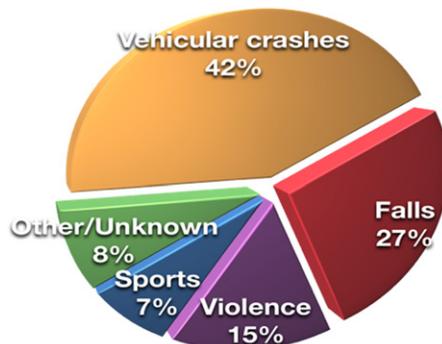


Fig. 11. Causes of spinal cord injury in the United States.

The average age at the time of injury is 39.5 years, and about 77.8% of these injuries occur in males. Incomplete tetraplegia is the most common type of SCI, followed in frequency by complete paraplegia, complete tetraplegia, and incomplete paraplegia. In economic terms the lifetime costs incurred by an individual with SCI range from \$500,000 to \$3 million, depending on the extent of the injury and the age of the injured patient (not including the loss of wages and so forth). In total, about \$4 billion is spent annually on the acute treatment and chronic care of SCI-injured individuals.¹⁶⁷

Diagnosis of SCI

The diagnosis of SCI is usually not established until the patient is transferred to the emergency department and is evaluated by a physician. Secondary to this, all patients who are victims of a significant trauma, trauma patients with loss of consciousness, and minor trauma victims with complaints referable to the spine or spinal cord should be treated as having an SCI by the field team until proved otherwise. Other clues that might point toward SCI are the presence of abdominal breathing, inability to move any one limb, numbness or tingling in any dermatomal distribution, or the occurrence of priapism. Management on the field should include, in addition to the mandatory ABCs, immobilization before extrication to prevent any active or passive movements of the spine, maintenance of blood pressure, maintenance of oxygenation, and caution with intubation with uncleared cervical spines.¹⁶⁸

Once in the emergency department, the initial evaluation and stabilization should take place by the trauma team in the original restraints from the field. When the patient is deemed stable, transfer to the CT scanner for further imaging should take place, still in the original restraints from the field.

The patient should then be examined and the images should be evaluated. The American Spinal Cord Injury Association has defined a grading system for SCI based on neurologic function. The injury, regardless of its etiology, is always classified in terms that describe the retained neurologic function of the injured patient. The classification's nomenclature spans the letters A through E (**Fig. 12**).

Pathophysiology of SCI

There are two known phases of SCI pathophysiology: primary and secondary. The initial traumatic event that physically disrupts the anatomic structures involved in the injury accounts for the primary phase of the insult. The most common form of acute SCI is a compressive-contusive type injury, whereby the displaced elements of the supporting vertebral structures exert force on the neural elements causing the immediate injury, and often sustained compression. The subsequent events that rapidly follow this mechanical disruption include phenomena such as cellular edema, vascular dysfunction, ischemia, excitotoxicity, inflammation, and cell death, and encompass the secondary phase (**Table 4**). As data on these mechanisms continue to amass, our understanding of the processes influencing secondary injury increases, and this has led to the adoption of neuroprotective strategies in an attempt to attenuate these noxious effects. For example, the acknowledgment that ongoing ischemia may worsen secondary damage has led to the implementation of clinical guidelines to promote spinal cord perfusion and aggressively avoid hypotension through an elevated mean arterial pressure. However, few of these research efforts have been able to drive a pharmacologic agent from the preclinical stages into clinically relevant remedies for patients with SCI.¹⁶⁹ All of the drugs currently in use have failed to show substantive benefit, with only methylprednisolone showing modest success in the 1990 landmark study.¹⁷⁰ Nevertheless, the risk for complications related to its use

Patient Name _____
 Examiner Name _____ Date/Time of Exam _____

ASIA INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY **ISCS**

MOTOR
 KEY MUSCLES (scoring on reverse side)

R	L		
C5	<input type="checkbox"/>	<input type="checkbox"/>	Elbow flexors
C6	<input type="checkbox"/>	<input type="checkbox"/>	Wrist extensors
C7	<input type="checkbox"/>	<input type="checkbox"/>	Elbow extensors
C8	<input type="checkbox"/>	<input type="checkbox"/>	Finger flexors (distal phalanx of middle finger)
T1	<input type="checkbox"/>	<input type="checkbox"/>	Finger abductors (palm finger)

UPPER LIMB TOTAL (MAXIMUM) (25) (25) (50)

Comments: _____

KEY MUSCLES (scoring on reverse side)

L2	<input type="checkbox"/>	<input type="checkbox"/>	Hip flexors
L3	<input type="checkbox"/>	<input type="checkbox"/>	Knee extensors
L4	<input type="checkbox"/>	<input type="checkbox"/>	Ankle dorsiflexors
L5	<input type="checkbox"/>	<input type="checkbox"/>	Long toe extensors
S1	<input type="checkbox"/>	<input type="checkbox"/>	Ankle plantar flexors

(VAC) Voluntary anal contraction (Yes/No)

LOWER LIMB TOTAL (MAXIMUM) (25) (25) (50)

SENSORY
 KEY SENSORY POINTS

0 = absent
 1 = altered
 2 = normal
 NT = not testable

Diagram of human body with key sensory points marked: C2-C8, T1-T12, L1-L5, S1-S5. Includes diagrams for hand (Dorsum/Volar) and foot (Dorsum/Volar).

NEUROLOGICAL LEVEL

NEUROLOGICAL LEVEL	R	L
SENSORY	<input type="checkbox"/>	<input type="checkbox"/>
MOTOR	<input type="checkbox"/>	<input type="checkbox"/>

SINGLE NEUROLOGICAL LEVEL

COMPLETE OR INCOMPLETE?
 Incomplete = Any sensory or motor function in S4-S5

ASIA IMPAIRMENT SCALE (AIS)

ZONE OF PARTIAL PRESERVATION
 (in complete injuries only)
 Most caudal level with any preservation

SENSORY R L
 MOTOR R L

TOTALS: LIGHT TOUCH SCORE (max: 112), PIN PRICK SCORE (max: 112), (DAP) Deep anal pressure (yes/No), PIN PRICK SCORE, LIGHT TOUCH SCORE

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Muscle Function Grading

- 0 = total paralysis
- 1 = palpable or visible contraction
- 2 = active movement, full range of motion (ROM) with gravity eliminated
- 3 = active movement, full ROM against gravity
- 4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position.
- 5 = (normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired person.
- 5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present.
- NT = not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

ASIA Impairment (AIS) Scale

- A = Complete.** No sensory or motor function is preserved in the sacral segments S4-S5.
 - B = Sensory Incomplete.** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5 (light touch, pin prick, or deep anal pressure (DAP)), AND no motor function is preserved more than three levels below the motor level on either side of the body.
 - C = Motor Incomplete.** Motor function is preserved below the neurological level** and more than half of key muscle functions below the single neurological level of injury (NLI) have a muscle grade less than 3 (Grades 0-2).
 - D = Motor Incomplete.** Motor function is preserved below the neurological level** and, at least half (half or more) of key muscle functions below the NLI have a muscle grade ≥ 3 .
 - E = Normal.** If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.
- **For an individual to receive a grade of C or D, i.e. motor incomplete status, they must have either (1) voluntary anal sphincter contraction or (2) sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body. The Standards at this time allow even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B versus C).

NOTE: When assessing the extent of motor sparing below the level for distinguishing between AIS B and C, the motor level for that side of the body. The Standards at this time allow even non-key muscle function more than 3 levels below the motor level to be used in determining motor incomplete status (AIS B versus C).

Steps in Classification

- The following order is recommended in determining the classification of individuals with SCI.
- Determine sensory levels for right and left sides.
 - Determine motor levels for right and left sides.
 Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.
 - Determine the single neurological level.
 This is the lowest segment where motor and sensory function is normal on both sides, and is the most cephalad of the sensory and motor levels determined in steps 1 and 2.
 - Determine whether the injury is Complete or Incomplete. (i.e. absence or presence of sacral sparing)
 If voluntary anal contraction = No AND all S4-S5 sensory scores = 0 AND deep anal pressure = No, then injury is COMPLETE. Otherwise, injury is incomplete.
 - Determine ASIA Impairment Scale (AIS) Grade:
 Is injury Complete? NO YES
 If YES, AIS=A and can record ZPP (lowest dermatome or myotome on each side with some preservation)
 Is injury motor Incomplete? NO YES
 If NO, AIS=B (Yes-voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)
 Are at least half of the key muscles below the single neurological level graded 3 or better? NO YES
 If NO, AIS=C YES, AIS=D
- If sensation and motor function is normal in all segments, AIS=E
 Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Fig. 12. American Spinal Cord Injury Association (ASIA) scoring sheet for the patient with spinal cord injury. (Courtesy of American Spinal Injury Association, Atlanta, GA; with permission.)

Time After SCI	Injury Phase	Phases and Key Events	
		Key Processes and Events	Therapeutic Aims
≤2 h	Primary Immediate	Mechanical Injury Traumatic axonal severing Gray matter hemorrhage Hemorrhagic necrosis Microglial activation Release of factors (IL-1 β , TNF- α , IL-6 and others)	Neuroprotection
≤48 h	Early Acute	Vasogenic edema Cytotoxic edema Lipid peroxidation Glutamate mediated excitotoxicity Continued hemorrhage Continued necrosis Neutrophil invasion Peak BBB permeability Early demyelination Neuronal death Axonal swelling Systemic shock Spinal shock Hypotension Hypoxia	Neuroprotection Immune modulation Cell-based remyelination Glial scar degradation
≤2 wk	Secondary subacute	Macrophage infiltration Reactive astrogliosis BBB repair Resolution of edema	
≤6 mo	Intermediate	Continued formation of glial scar Cyst formation Lesion stabilization	Glial scar degradation
≥6 mo	Chronic/late	Prolonged Wallerian degeneration Persistence of spared, demyelinated axons Potential structural and functional plasticity of spared spinal cord tissue	Rehabilitation Neuroprosthesis

Abbreviations: BBB, blood-brain barrier; IL, interleukin; TNF, tumor necrosis factor.

has found its detractors, and its widespread use as the standard of care has been questioned.^{171,172}

Timing of Intervention

There has long been a debate regarding the optimal time to intervene in patients with acute SCI. Preclinical data dating back from the 1970s demonstrated in animal studies that injury to the spinal cord could be worsened by sustained compression. The damage caused by such an insult can be attenuated by decompressive surgery, thereby relieving the pressure being applied to the spinal cord.^{173–180} There is,

however, no clear consensus regarding the optimal timing of decompressive surgery after SCI, but it is evident that there is no increased morbidity in early decompression.^{181,182} In addition, an article by Furlan and colleagues¹⁸³ published the opinion of a panel of 10 experts who recommended that “surgical decompression of the injured spinal cord is performed within 24hrs when medically feasible.” This opinion seems to echo the current standard used in their practice by a large proportion of the international spine community.¹⁸⁴ Thus, although not definitely proved with class I data, the preclinical evidence as well as lower-quality clinical studies seem to favor early decompression. It is imperative to reiterate that the benefit of early decompression in the setting of SCI is contingent on the presence of ongoing spinal cord compression.

Radiologic Evaluation

Having access to imaging modalities such as radiography, CT, and MR imaging to quantify the amount of compression is imperative to be able to both diagnose SCI and determine the patients’ potential for benefit from early surgical intervention. Measurements such as anteroposterior (AP) canal diameter, transverse canal diameter, canal area, and the ratio between AP diameter and transverse diameter have been used in the past, and are described in the literature as useful tools in determining spinal canal compromise.^{185–189} However, the most reliable means to quantify spinal cord compression was set forth in a study by Fehlings and colleagues.¹⁹⁰ This method calculates maximal spinal cord compression by comparing the AP cord diameter at the level of maximum injury with the AP cord diameter at nearest normal levels above and below.

Surgical Decision Making

Important considerations when assessing whether a patient with spinal trauma requires surgery include the presence of spinal cord compression and a neurologic deficit. However, additional concepts to be taken into account that will help decide on how to steer the therapeutic decision making must include the mechanism of injury, the presence or absence of biomechanical instability, osteoligamentous integrity and fracture configuration. Several investigators have attempted to incorporate some or all of these elements to create a comprehensive spinal trauma classification system for use in the clinical realm. However, no single system has gained widespread use. Two novel classification systems for spinal trauma have been recently developed by members of the Spine Trauma Study Group (STSG). The first, known as the Subaxial Cervical Spine Injury Classification (SLIC), addresses injuries to the subaxial cervical spine. The second, contributed by the STSG and known as the Thoracolumbar Injury Classification and Severity Score (TLICS), addresses spinal injuries at the thoracolumbar junction based on an algorithm virtually identical to SLIC. The investigators have recommended that patients with an SLIC or a TLICS score of less than 4 should be treated nonoperatively, those with an SLIC or a TLICS score of greater than 4 should be treated operatively, and those with an SLIC or a TLICS score of exactly 4 can be managed with or without surgery at the discretion of the clinical management team (**Table 5**).^{191,192} The combination of radiographic and clinical information gathered on the individual patient will determine the necessity of operative intervention; however, the surgeon involved will still need to make a decision as to the type of operation to be performed. This decision is largely based on the location of the primary pathology. However, there seems to be a degree of discretion on the surgeon’s behalf to interpret the evidence, his or her personal experience, and his or her proficiency at

Table 5		
Subaxial and thoracolumbar classification systems		
Injury Variable	Weighed Severity Points for SLIC	Weighed Severity Points for TLICS
Morphology		
No abnormality	0	0
Compression	1	1
Distraction	3	3
Rotation/Translation	4	4
Discoligamentous Complex Integrity		
Intact	0	0
Indeterminate	1	2
Disrupted	2	3
Neurologic Status		
Intact	0	0
Root injury	1	2
Complete/conus cord injury	2	3
Incomplete cord injury	3	2
Cord compression with neural deficit/cauda equina	+1	3

Abbreviations: SLIC, Subaxial Cervical Spine Injury Classification; TLICS, Thoracolumbar Injury Classification and Severity Score.

executing a certain approach over another, to formulate what would be the best modality of treatment for a particular patient.

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