Brain and Spine Trauma

Adam Kelly, MD
University of Rochester
Rochester, New York

Traumatic injury to the brain and spinal cord, either acutely or related to subacute or chronic consequences of trauma, is a common indication for neurologic consultation. The initial manifestations of brain trauma can vary from mild concussion to severe brain injury, which can make prognostication challenging. This section discusses brain and spinal cord trauma as well as sequela of these injuries.

Brain Trauma

Traumatic brain injury (TBI) is a common occurrence in the general population with increased prevalence in certain groups such as military personnel and athletes participating in contact sports. Nearly 2 million new cases of TBI are thought to occur each year, although this is almost certainly an underestimate since many mild cases likely go unreported. The Glasgow Coma Scale is often used to categorize the severity of TBI; specifically, scores of 13 to 15 are categorized as mild, scores of 9 to 12 are categorized as moderate, and scores below 8 indicate severe injury. TBI can occur through a variety of different mechanisms with falls being the most common cause in the general population.
Concussion

The terms *concussion* and *mild TBI* are often used interchangeably, although many organizations consider them to be overlapping but related terms. Mild TBI is the pathologic state of the brain following minor injury to the brain and is classified by a patient’s score on initial Glasgow Coma Scale testing. Concussion is the clinical syndrome of altered brain function that often results from a mild TBI. The following section specifically focuses on the clinical syndrome of concussion.

Roughly 75% of TBIs are mild and therefore may potentially result in concussion. The clinical definition of mild TBI includes a head trauma associated with some combination of the following: loss of consciousness (usually brief) following the injury; amnesia for some period of time immediately before and after the injury; mental status changes (ie, being dazed) at the time of the injury; or some transient focal neurologic symptoms. By definition, the duration of loss of consciousness must not exceed 30 minutes, and the duration of amnesia must not exceed 24 hours. The pathophysiology of mild TBI continues to be an active area of research, although most researchers believe that even mild brain trauma sets off a cascade of biochemical changes, including glutamate release, increased metabolic activity, and lactate accumulation from glycolysis.

Shortly after mild TBI, patients may begin to experience the classic symptoms of concussion, which include headache, nausea, difficulty with attention and concentration, trouble focusing visually, sensitivity to loud noises and bright lights, sleep dysfunction, and mood/personality changes. More objective findings on examination can include slurred speech, prolonged pauses before answering questions, nystagmus, and balance/coordination dysfunction. Although focal neurologic symptoms are rarely seen,
these raise concerns of a potential intracranial hemorrhage, which should be ruled out with imaging before being attributed to a concussion. Without focal findings or other atypical features of the injury or the examination, imaging is not generally needed in the diagnosis of concussion since it is unlikely to be abnormal and does not affect management.

The mainstays of concussion treatment include rest and symptom control. Nonsteroidal anti-inflammatory medications or acetaminophen can be used to control headaches, and antiemetics can be used for nausea. Sleep and quiet rest should be encouraged. Excessive stimulation (eg, exposure to loud noises or bright lights) should be avoided when possible. No other specific disease-modifying therapy has been shown to be beneficial in this setting.

**Return to Work/Play Following Concussion.** Evidence from observational studies shows that patients (especially athletes) who experience a first concussion are at higher risk of developing concussion symptoms with subsequent head injuries, and the recovery process from subsequent concussions can be prolonged. As a result, national organizations (including the American Academy of Neurology) recommend that patients with concussion symptoms not be cleared to return to activities that place them at high risk for recurrent TBI (such as return to contact sports) until their concussion symptoms have completely resolved. It is also recommended that a health care provider also examine such patients to be sure that objective signs have likewise resolved. The rationale for this period of rest is the presumption that the biochemical and metabolic changes described earlier are more likely to be ongoing if signs and symptoms of concussion persist. Once symptoms have resolved at rest or with minimal activity,
patients can begin a graduated process of returning to their prior level of physical and cognitive function to ensure that symptoms do not re-emerge as activity level increases.

**Postconcussive Syndrome.** Most patients with concussion will have improvement and resolution of their symptoms with rest and other conservative measures within the course of several days. A proportion of patients will have persistent symptoms or will have the emergence of symptoms 1 week or longer after their head trauma, with estimates of this proportion ranging from 30% to 80%. The symptoms of this postconcussive syndrome overlap considerably with symptoms of the initial concussion. These symptoms include headaches, irritability, sleep dysfunction, and mood and personality changes. No specific therapeutic intervention exists for postconcussive syndrome that is supported by clinical trial evidence; management is largely directed toward symptomatic relief. Amitriptyline or other tricyclic antidepressants are often used for headache management since the anticholinergic effects of these medications can also help with insomnia. Postconcussive symptoms lasting more than several months are highly unusual and should prompt investigation for alternative diagnoses or psychosocial issues such as pending litigation.

**Diffuse Axonal Injury**

Severe head trauma can result in shear or rotational injuries to nerve fibers, a condition known as diffuse axonal injury (DAI). DAI can result in severe encephalopathy and should be considered as a possibility in patients with TBI with coma and no evidence of elevated intracranial pressure on monitoring. Patients in this setting often have a poor prognosis for meaningful neurologic recovery. Imaging in DAI shows injury and
hemorrhages in subcortical white matter tracts; hemorrhages occur because of shear injuries to penetrating blood vessels as a result of the same shear forces that result in axonal injury.

**Cerebral Contusion**

Penetrating brain injuries (eg, from a gunshot) can result in damage to intracranial blood vessels and intracranial hemorrhage. However, blunt force head injuries can also result in petechial hemorrhages or frank hematomas within brain tissue, also known as a cerebral contusion. Contusions can occur at the site of the head trauma (referred to as a *coup* injury) or at remote locations, often on the contralateral side of the brain (referred to as a *contrecoup* injury). The inferior frontal and inferior temporal lobes are particularly susceptible to contrecoup injuries since the bony floors of the anterior and middle cranial fossa are not entirely smooth in their lining. A common clinical scenario that can represent a diagnostic dilemma is the finding of intracranial hemorrhage in patients who are found unresponsive after they have fallen; in this setting, it can be difficult to know if the hemorrhage occurred first and caused neurologic symptoms including the fall, or whether a fall occurred for other reasons (eg, tripping, syncope) and head trauma resulted in a contusion. One clue to a contusion is other evidence of trauma (either on examination or imaging) in the subcutaneous tissues adjacent to the areas of bleeding. If this is the case, the bleed is more likely to be the result of the fall, not the cause. Neurologists should also be aware that contusions can sometimes present in a delayed fashion (not immediately after a head trauma), especially in patients taking anticoagulant medications.
Other Traumatic Intracranial Hemorrhages

In addition to traumatic hemorrhages within the brain parenchyma (contusion), head trauma can result in bleeding in other intracranial compartments. Epidural hematomas usually result from blunt trauma to the lateral skull, often in association with fracture of the squamous portion of the temporal bone. With or without fracture, blood vessels coursing along the inner aspect of the skull, especially the middle meningeal artery, can rupture and result in bleeding into the epidural space. Patients are often dazed or confused as a result of their head injury, then experience a lucid interval before the hematoma causes mass effect and neurologic symptoms. Epidural hematomas arise from bleeding from arterial sources, and thus hematoma volumes can build up quickly.

Management of these cases is usually surgical.

Subdural hematomas are a common manifestation of head trauma, and in older adults, can occur from rapid acceleration-deceleration events without frank trauma. These mechanisms can cause shearing of bridging veins that course through the subdural space. Older adults or other patients with significant brain atrophy are at higher risk of subdural hematomas since bridging dural veins are often already stretched from the decreased brain volume and are more prone to injury with additional stretching or shear forces. These hemorrhages are almost always under venous, not arterial, pressure and, therefore, can accumulate slowly over time. Management can be surgical in cases where there is significant mass effect and focal neurologic symptoms or cortical irritability resulting in seizures. However, many cases are managed conservatively with reversal or stopping of antithrombotic medications and close observation for signs of hematoma enlargement.
The most common cause of subarachnoid hemorrhage is trauma, not ruptured intracranial aneurysm. Similar to contusion, blunt force head trauma can result in injury to small pial blood vessels in the region adjacent to the trauma, causing small areas of subarachnoid hemorrhage. Management of subarachnoid hemorrhage is conservative with reversal or stopping of antithrombotic medications and other supportive interventions as needed (eg, analgesia). It should be noted that nimodipine, seizure prophylaxis, and other interventions used in the management of aneurysmal subarachnoid hemorrhage are typically not needed in patients with traumatic subarachnoid hemorrhage.

**Brain Death**

Patients with severe TBI or other severe brain injuries are at risk for progression to death by brain criteria, or brain death. Brain death is considered equivalent to death from cessation of cardiopulmonary function, although considerable knowledge gaps continue to exist in the public and the medical community. When the diagnosis of brain death is considered, a proximate cause (eg, trauma, stroke, hypoxic-ischemic injury) should be identified, and all confounding factors should be reversed or addressed. This includes the correction of metabolic factors such as electrolyte or acid-base disorders; allowing for the clearance of any medications with sedating effects; and adjustment of the core body temperature to at least 32°C (89.6°F).

Once this has occurred, examination of the patient who is brain dead will show cessation of all brain function, including no response to stimulation other than reflexive movements and the loss of brainstem reflexes (pupillary light reflex, corneal reflex, oculocephalic reflex including caloric testing, and gag reflex). Apnea testing is then
carried out, and a rise in the PCO₂ of more than 20 mm Hg with no respiratory effort is consistent with a diagnosis of brain death. In patients meeting all of these criteria, confirmatory diagnostic testing is not needed, although it can be carried out in select situations, often when a patient’s cardiopulmonary status is too tenuous to tolerate a full apnea test. Confirmatory testing options include cerebral perfusion scanning, EEG, cerebral angiography, somatosensory evoked potentials, and transcranial Doppler.

**Chronic Traumatic Encephalopathy**

Over time, mounting evidence has supported chronic long-term effects of repeated head trauma. Much of this evidence comes from athletes in collision sports (eg, football, boxing) or in military veterans. The term chronic traumatic encephalopathy (CTE) now refers to a constellation of symptoms including cognitive impairment, neuropsychological changes (including an increased risk of suicide), parkinsonism, and speech changes. The number of head trauma events seems to correlate with the risk of developing CTE; genetic risk factors may be present as well since epidemiologic data suggest an increased risk in apolipoprotein ε4 carriers. Histopathologically, CTE appears to be a tauopathy that is pathologically distinct from other neurodegenerative conditions related to tau deposition. At present, no disease-modifying therapy for CTE exists.

**Spine Trauma**

Acute trauma to the spine can result from a variety of different mechanisms, including penetrating injuries (eg, stabbing, gunshot), mechanical forces (eg, hyperextension, transection), or secondary consequences (eg, damage to blood vessels resulting in an
epidural hematoma). Milder repetitive injuries to the spine can result in degenerative changes to the bones and disks of the spine, especially in the cervical and lumbar segments, causing radiculopathy, myelopathy, and spinal stenosis in some instances. This section focuses on some of these acute and chronic manifestations of spine trauma.

**Acute Spinal Cord Injury**

As noted previously, acute spinal cord injury (SCI) can result from a variety of different injury mechanisms. SCI is relatively common with an estimated 12,000 new cases per year and can be associated with significant morbidity and mortality. Late neurologic consequences of SCI can include quadriplegia or paraplegia, depending on the site of the injury, and many related comorbidities may result from these neurologic sequelae, including deep vein thrombosis, neurogenic bladder, and autonomic dysreflexia.

One of the more common neurologic presentations of SCI is central cord syndrome, which is thought to develop secondary to a hyperextension of the spine (most commonly the cervical spine). This hyperextension results in buckling of the ligamentum flavum and contusion of the spinal cord at the site of the injury and often occurs in areas of spondylosis where a reduction of the diameter of the spinal canal is already present. As its name implies, central cord syndrome impacts structures in the central aspect of the spinal cord; since motor fibers projecting to the arms are located more medially and centrally, patients often have more profound weakness in the arms as compared with the legs. Sensation below the level of the injury and bladder function can also be affected in patients with central cord syndrome.
With penetrating spinal cord injuries, the Brown-Séquard syndrome (hemicord syndrome) can occur. In this situation, patients can experience ipsilateral loss of motor function and light touch/vibration sense with contralateral loss of pinprick and temperature sense (due to crossing of the spinothalamic projections at the level of entry into the spinal cord). Horner syndrome can also be seen with cervical lesions due to involvement of the intermediolateral cell column. Depending on the specific areas of the spinal cord involved in a SCI, anterior or posterior cord syndromes can also occur, although anterior spinal cord syndromes are much more likely to be secondary to ischemic injuries through involvement of the anterior spinal artery.

Early management of the patient with SCI should first center on medical stabilization and the ABCs (airway, breathing, and circulation). Patients with upper cervical injuries are at risk for phrenic nerve dysfunction and may need respiratory support, and spinal shock can result from involvement of descending sympathetic nerve fibers. Depending on the extent and mechanism of injury and the patient’s medical comorbidities, surgical intervention should be carried out as soon as it is felt to be safely possible with the goals of stabilizing the spine, reducing any mass effect, and minimizing secondary cord injury. Although prior clinical trials had shown some potential benefit, more recent studies and consensus guidelines now caution against the use of corticosteroids in the management of acute SCI. No other specific disease-modifying therapies have been shown to be beneficial at this time. Following stabilization, management of the patient with SCI involves respiratory management, treatment of medical complications, and rehabilitation.
Spinal Epidural Hematoma

Spinal epidural hematomas are a specific type of acute SCI due to rupture of a blood vessel in the epidural space of the spinal canal. Spinal epidural hematomas can occur from trauma or as a consequence of a bleeding disorder (eg, anticoagulation, antiplatelet use), but the most common cause is vessel injury during a dural puncture, such as during a lumbar puncture or spinal anesthesia. This vascular injury can either be venous or arterial, and the source of the bleeding (and hence the blood pressure supply it is under) can dictate how quickly blood accumulates and neurologic symptoms develop. Typically, patients present with back pain and radicular symptoms around the site of the hematoma, then develop neurologic symptoms as the hematoma volume and pressure on the spinal cord increase. Neurologic symptoms usually include sensory loss and weakness at levels below the site of bleeding. Patients with pain and neurologic symptoms similar to this following a spinal procedure or trauma should undergo urgent spinal imaging, preferably with MRI. Many patients require surgical evacuation of the hematoma to relieve mass effect on the spinal cord, although some patients with minimal symptoms can be managed conservatively with close observation.

Cervical Spondylosis and Myelopathy

Spondylosis refers to degenerative changes to the vertebral bodies and intervertebral disks of the cervical spine, usually occurring as a result of mild recurrent trauma. These changes include flattening and herniation of the disks, osteophyte formation on bony structures, and hypertrophy of the posterior longitudinal ligament. As these accumulate, narrowing of the intraspinal canal can occur, which can ultimately result in mass effect
on the spinal cord. If mass effect increases to a clinically significant level, cervical myelopathy can result. In addition to mass effect, vascular changes at the site of spinal canal narrowing (both arterial and venous) are thought to potentially play a role in the pathophysiology of myelopathy.

The clinical presentation of cervical myelopathy is dependent on the specific level of the spondylitic changes, with middle to lower cervical segments being most likely affected. In general, lower motor neuron or radicular changes commonly occur at the level of the severe spondylosis, including neck pain radiating into one or either arm, sensory changes in a dermatomal pattern corresponding to the affected level(s), weakness and atrophy in muscle groups innervated by nerve roots at affected level(s), and hyporeflexia in expected segments based on affected roots (eg, depressed triceps reflex with C7 or C8 involvement). In addition, upper motor neuron findings and other evidence of long tract dysfunction typically occur at levels below the areas of significant spondylitic changes. These can include lower extremity weakness; spasticity, hyperreflexia, and Babinski signs; sensory deficits in the lower extremities that more commonly affect dorsal column functions and do not follow a dermatomal or peripheral nerve pattern (a sensory level can also be seen in some occasions); gait dysfunction, often with a spastic quality; and bladder dysfunction (eg, urgency, frequency).

Patients with suspected cervical myelopathy should undergo imaging of the cervical spine, preferably with MRI. In patients who are unable to undergo MRI (eg, because of metal fragments, pacemaker placement), CT or myelography are other options. Electrophysiologic studies can also help pinpoint the areas of significant lower
motor neuron dysfunction, but localization can usually be accomplished through the examination and imaging studies.

Management of cervical spondylosis and myelopathy can range from conservative measures to surgical intervention. Many patients will have some degree of spondylitic changes as they age and so the presence of degenerative disease in the bones and disk of the cervical spine alone is not an indication to proceed with surgery. Conservative management usually includes analgesia and a trial of neck immobilization along with avoidance of activities that place the neck at risk for injury (eg, contact sports). Some patients will have minor symptoms as a result of new herniations of disks or hyperextension of the neck, which can resolve spontaneously. In patients with progressive or severe symptoms, a number of different surgical options can be considered, with the specific choice dependent upon the anatomy of the spine and affected levels.

**Lumbar Spinal Stenosis**

Degenerative changes in the bones and disks of the lumbar spine can result in a number of neurologic symptoms, including radiculopathy and stenosis of the lumbosacral spinal canal. Although spondylitic changes are the most common cause of lumbar stenosis, other causes of nerve root dysfunction in the lumbosacral region (eg, lipomas, metastatic disease) should be considered in the appropriate clinical context.

As with cervical spondylosis, the primary pathophysiology of lumbar stenosis is thought to be mechanical compression of nerve roots in a narrowed spinal canal, although this narrowing can also result in changes in both arterial and venous circulations in this
area. Symptoms of lumbar stenosis are almost always worse when patients are standing erect since this can cause relative hyperextension of this spinal segment and result in buckling of the ligamentum flavum. Both of these processes can further reduce the diameter of the spinal canal. Another common finding in lumbar stenosis is worsening pain and neurologic function with ambulation (so-called neurogenic claudication). It is unclear whether this phenomenon occurs because of repeated mechanical injury with the movements associated with walking, or whether exertion results in ischemia and other vascular changes to the cauda equina where blood flow is already impaired.

The most common presentations of lumbar stenosis are back pain, weakness, and sensory dysfunction in the legs. As noted above, patients almost invariably report worsening of their symptoms with prolonged standing or walking and conversely report improvement with flexed positions or sitting. The weakness and numbness may be bilateral or unilateral and is often asymmetric. It also typically does not follow a distribution of a specific lumbosacral root, probably because the stenosis affects multiple roots. Bowel, bladder, and erectile dysfunction can occur, especially when sacral roots are preferentially affected.

In cases where lumbar stenosis is suspected, imaging of the lumbosacral spine should be performed, usually with MRI if possible. In cases where MRI cannot be performed, CT or CT myelography can be considered. Electrophysiologic studies can also be considered and are often done to rule out alternative explanations for the lower extremity symptoms (eg, peripheral neuropathy). Likewise, other causes of lower extremity claudication, such as peripheral vascular disease, should be evaluated for in the appropriate clinical context.
Management of lumbar stenosis can include nonsurgical and surgical options. Principles of nonsurgical management include physical therapy, analgesia and other symptomatic treatments, and epidural injections. In cases of severe spinal stenosis or when conservative strategies are ineffective, surgical options can be considered with the specific technique dependent on the spinal anatomy and the surgeon’s expertise. In general, younger patients with better preservation of walking function and fewer medical comorbidities tend to have better outcomes following surgery.

**Annotated Bibliography**


This is a systematic review of clinical trials evaluating corticosteroid therapy in the treatment of acute spinal cord injury. In contrast to the guideline statement from the neurosurgery community, this article describes a benefit to steroid therapy without additional adverse effects. The differences between these publications highlight the current controversy behind this treatment.


In this study, patients who were deemed to be good candidates for surgical management of their lumbar spinal stenosis were randomized to surgery or ongoing physical therapy. Physical function scores were no different at 2 years between these two groups.

This systematic review evaluates various clinical characteristics and results of rating scales for their ability to predict abnormal imaging results in patients with minor head trauma. High likelihood of skull fracture, multiple vomiting events, and trauma resulting from a pedestrian being struck by a motor vehicle were some possible predictors of occult intracranial injury.


This paper describes a prospective cohort of patients between the ages of 11 and 22 years whose postconcussive symptoms were assessed in a systematic fashion. Headache was the predominant early symptom with emotional lability developing later in the course and cognitive symptoms present throughout. Although primarily studying a pediatric population, these results may also apply to adult patients with concussion.

This evidence-based review from the American Academy of Neurology covers several topics important in the evaluation and management of concussion, including the initial assessment of the severity of the injury and decisions regarding timing of return to play.


This publication is a guideline from the Congress of Neurological Surgeons and the American Association of Neurological Surgeons that argues against the use of methylprednisolone in acute spinal cord injury. Their consensus opinion is that no high level studies support a benefit of this therapy, yet data strongly suggest increased side effects in patients receiving corticosteroids.


This paper is a thorough overview of the common clinical features and management options, both surgical and nonsurgical, for patients with lumbar spinal stenosis.

This article provides an excellent overview of the epidemiology, presumed pathophysiology, and research priorities for chronic traumatic encephalopathy in athletes who have sustained repeated head trauma.

doi:10.1212/01.CON.0000461091.09736.0c.
This article reviews the major features of an acute spinal cord injury, including common spinal cord syndromes, stabilization, and management of neurologic and secondary consequences of the injury.

Tavee JO, Levin KH. Myelopathy due to degenerative and structural spine diseases. Continuum (Minneap Minn) 2015;21(1 Spinal Cord Disorders):52–66.
doi:10.1212/01.CON.0000461084.71618.35.
This article reviews the clinical characteristics, imaging findings, and management options for patients with cervical spondylitic myelopathy.

doi:10.1212/WNL.0b013e3181e242a8.
This evidence-based review published by the American Academy of Neurology provides a comprehensive summary of the approach to the patient with suspected brain death,
including necessary components of the physical examination and the role for confirmatory testing.