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sphincter musculature. Detrusor spasticity is treated with anticholin ergic drugs (oxybutynin, 2.5–5 mg qid) or tricyclic antidepressants with anticholinergic properties (imipramine, 25–200 mg/d). Failure of the sphincter muscle to relax during bladder emptying (urinary dyssynergia) may be managed with the α-adrenergic blocking agent terazosin hydrochloride (1–2 mg tid or qid), with intermittent cath eterization, or, if that is not feasible, by use of a condom catheter in men or a permanent indwelling catheter. Surgical options include the creation of an artificial bladder by isolating a segment of intestine that can be catheterized intermittently (enterocystoplasty) or can drain continuously to an external appliance (urinary conduit). Bladder areflexia due to acute spinal shock or conus lesions is best treated by catheterization. Bowel regimens and disimpaction are necessary in many patients to ensure at least biweekly evacuation and avoid colonic distention or obstruction.

Patients with acute cord injury are at risk for venous thrombosis and pulmonary embolism. Use of calf-compression devices and anticoagu lation with low-molecular-weight heparin are recommended. In cases of persistent paralysis, anticoagulation should probably be continued for 3 months. PART 13 Neurologic Disorders Prophylaxis against decubitus ulcers should involve frequent changes in position in a chair or bed, the use of special mattresses, and cushioning of areas where pressure sores often develop, such as the sacral prominence and heels. Early treatment of ulcers with care ful cleansing, surgical or enzyme debridement of necrotic tissue, and appropriate dressing and drainage may prevent infection of adjacent soft tissue or bone. Spasticity is aided by stretching exercises to maintain mobility of joints. Drug treatment is effective but may result in reduced function, as some patients depend on spasticity as an aid to stand, transfer, or walk. Baclofen (up to 240 mg/d in divided doses) is effective; it acts by facilitating γ-aminobutyric acid–mediated inhibition of motor reflex arcs. Diazepam acts by a similar mechanism and is useful for leg spasms that interrupt sleep (2–4 mg at bedtime). Tizanidine (2–8 mg tid), an α2 adrenergic agonist that increases presynaptic inhibition of motor neu rons, is another option. For nonambulatory patients, the direct muscle inhibitor dantrolene (25–100 mg qid) may be used, but

it is potentially hepatotoxic. In refractory cases, intrathecal baclofen administered via an implanted pump, botulinum toxin injections, or dorsal rhizotomy may be required to control spasticity. Despite the loss of sensory function, many patients with spinal cord injury experience chronic pain sufficient to diminish their quality of life. Randomized controlled studies indicate that gabapentin or prega balin is useful in this setting. Epidural electrical stimulation and intra thecal infusion of pain medications have been tried with some success. Management of chronic pain is discussed in Chap. 14. A paroxysmal autonomic hyperreflexia may occur following lesions above the major splanchnic sympathetic outflow at T6. Headache, flushing, and diaphoresis above the level of the lesion, as well as hyper tension with bradycardia or tachycardia, are the major symptoms. The trigger is typically a noxious stimulus—for example, bladder or bowel distention, a urinary tract infection, or a decubitus ulcer—below the level of the cord lesion. Treatment consists of removal of offending stimuli; ganglionic blocking agents (mecamylamine, 2.5–5 mg) or other short-acting antihypertensive drugs are useful in some patients. Emerging neuro-therapeutic technologies, including rehabilitation robotics and brain-machine interfaces, offer real hope to advance prospects for full and productive lives in patients with disabling myelopathies (Chap. 500). ■ ■FURTHER READING Badhiwala JH et al: Degenerative cervical myelopathy—update and future directions. Nat Rev Neurol 16:108, 2020. Bradshaw MJ et al: Neurosarcoidosis: Pathophysiology, diagnosis, and treatment. Neurol Neuroimmunol Neuroinflamm 8:e1084, 2021. Garg RK et al: Spinal cord involvement in COVID-19: A review. J Spinal Cord Med 46:390, 2023. Gritsch D, Valencia-Sanchez C: Drug-related immune-mediated myelopathies. Front Neurol 13:1003270, 2022.

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Concussion and Other

Traumatic Brain Injuries Traumatic brain injury (TBI) represents a significant global public health problem. In the United States, estimates of the frequency of TBI range between 2.5 and 4.8 million cases per year, depending on the study and methods used to define and include cases. Age-specific rates show a bimodal distribution, with highest risk in younger individuals and older adults. The most common mechanism of injury in the young is motor vehicle accidents and is more common in men, whereas in older adults, falls are the major cause of injury and are more likely to occur in women. Nonfatal TBI-related hospitalization rates are com parable across non-Hispanic white, non-Hispanic black, and Hispanic persons with age adjustment. TBI imposes substantial

demands on health care systems. Worldwide, at least 10 million TBIs are serious enough to result in death or hospi talization, producing a global economic burden of $400 billion annu ally. In the United States, the estimated annual cost is >$76 billion. Due to advances in medical care and other factors, more people are surviving TBI than ever before. Brain injury accounts for more lost productivity at work among Americans than any other form of injury. An estimated 5.3 million Americans are living with significant disabili ties resulting from TBI that complicate their return to a full and pro ductive life. Increased media attention to military and sports-related TBI has highlighted the growing concern that injuries that were previ ously dismissed can have lifelong consequences for some individuals. Head injuries are so common that almost all physicians will be called upon to provide some aspect of immediate care or to see patients who are suffering from various sequelae. Patients and their families initially need education regarding the natural history of TBI along with treatment of acute symptoms such as headache. Continued follow-up is important to ensure that the sequelae experienced by some patients— such as the persistent postconcussion symptoms (PPCS) of headache, disturbances in balance, depression, and sleep disorder—are identified and treated appropriately. Effective management of TBI and its conse quences often requires a coordinated multidisciplinary care team.

■ ■DEFINITION AND CLASSIFICATION TBI is commonly defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force, and character ized by the following: (1) any period of loss or decreased level of con sciousness (LOC), (2) any loss of memory for events immediately before (retrograde) or after (posttraumatic) the injury, (3) any neurologic deficits, and/or (4) any alteration in mental state at the time of injury. Evidence of TBI can include visual, neuroradiologic, or laboratory confirmation of damage to the brain, but TBI is more often diagnosed on the basis of acute clinical criteria. In addition to standard com puted tomography (CT) imaging and conventional clinical magnetic resonance imaging (MRI), advanced MRI imaging (functional MRI, cerebral blood flow, diffusion MRI) techniques show increasing sen sitivity, and it is likely that sensitive blood-based biomarkers will play an increasingly important role in the diagnosis and treatment of these patients (described below). Mechanisms of TBI Common mechanisms of TBI include the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement, a foreign body penetrating the brain, or forces generated from events such as a blast or explosion. Unintentional falls and motor vehicle crashes have his torically been cited as the most common cause of TBI. All forms of transportation, however, are common causes of TBI, including motor cycle crashes, bicycle accidents, skateboarding, and pedestrian injuries. The other leading causes of TBI are falls, assaults, and sports, with var ied frequency across the lifespan. Certainly, there has been an increased focus on the high frequency of mild TBI (mTBI), often referred to as concussion, encountered by athletes participating in contact and colli sion sports at all competitive levels, as well as the potential short-term effects and long-term risks associated with sport-related concussion. Classification of TBI Severity Numerous systems have been developed over the years to define and classify TBI severity along a con tinuum from mild to moderate to severe. These systems are usually most applicable to closed head injuries. In nearly all classification systems, TBI severity is graded based on acute injury characteristics rather than post acute injury status, as other factors can intervene to influence functional outcome. This can be problematic, as some patients with severe TBI will have a full recovery and some with mild TBI will be left with lifetime disability. Historically, the presence and duration of unconsciousness and amnesia have been the main points of distinction along the gra dient of TBI severity. Efforts to develop more advanced classification systems that incorporate

patho-anatomical features reflecting biological response to injury, such as blood-based biomarkers, as well as socioen vironmental factors that may influence ultimate prognosis are currently underway. A recent working group convened by the National Institutes of Health has proposed a new framework for TBI characterization that includes clinical features, blood-based biomarkers, and imaging features, along with modifiers. As this more precise nomenclature is validated, it is anticipated that it will replace the current approach of characterizing patients based solely on their clinical exam and symptoms. The Glasgow Coma Scale (GCS) is the most recognized and widely used method for grading TBI severity. The GCS provides a practical indicator of gross neurologic status by assessing motor function, verbal responses, and the patient's ability to open his or her eyes voluntarily or in response to external commands and stimuli. The grading is applied to the best response that can be elicited from the patient at the time of assessment, preferably before any paralyzing or sedating medication is administered or the patient is intubated, as these interventions con found interpretation of the score. The GCS assessment produces scores ranging from 3 to 15 (Table 454-1). Upon the 40th anniversary of the GCS in 2014, the wording for responses was revised, and recommendations were made to improve its utility. Importantly, individual patients are best described by the three components of the coma scale (eye, verbal, motor, e.g., E3V4M6); the derived total coma score (e.g., 13) is less informative and should only be used to characterize groups of patients. Several injury-classification systems have been developed to go beyond GCS score or acute injury characteristics and incorporate chief

TABLE 454-1 Glasgow Coma Scale EYE OPENING (E) VERBAL RESPONSE (V) Spontaneous

Oriented

To speech

Confused

To pressure

Words

None

Sounds

None

Best Motor Response (M) Obeying commands

Localizing

Normal flexion

Abnormal flexion

Extension

None

CHAPTER 454 Note: Revised GCS (2014). Source: Reproduced with permission from G Teasdale et al: The Glasgow Coma Scale at 40 years: Standing the test of time. Lancet Neurol 13:844, 2014. signs and symptoms in defining mTBI. The use of multiple severity indicators is intended to improve sensitivity in the detection of mTBI (GCS 13–15), while also taking into consideration traditional acute injury characteristics that have been presumed to predict outcome fol lowing mild and moderate brain injury. LOC and posttraumatic amne sia (PTA) remain the most common injury characteristics referenced in these classification systems. In the case of moderate (GCS 9–12) and severe (GCS 3–8) TBI, GCS score and the duration of LOC and PTA can be robust predictors of long-term outcome and morbidity. In cases of mTBI, however, while PTA and LOC are important indicators of acute injury, they are less predictive of eventual recovery time and outcome, particularly within sport-related concussion. Concussion and Other Traumatic Brain Injuries ■ ■TBI TYPES AND PATHOLOGIES Mild TBI (Concussion) It is estimated that 70–90% of all treated TBIs are mild in severity based on traditional case definitions and acute injury characteristics, with most reported estimates in the order of 85%. The published figures likely underrepresent the true incidence of mTBI because of variable case definitions and heterogeneous methods. Moreover, because a subgroup of individuals with milder brain injuries does not seek medical attention, epidemiologic studies that depend on hospital-based data also underestimate the true incidence. In fact, it has been estimated that current data collection sources and methods may only capture one of every nine mTBI/concussions in the United States. The term concussion, while popular, is vague and is not based on widely accepted objective criteria, resulting in multiple definitions from various groups. There has been debate as to whether concussion is part of the TBI spectrum or a separate entity. The Concussion in Sports Group has concluded that “concussion is a traumatic brain injury” as part of the consensus statement definition of the injury. By firmly plac ing concussion in the spectrum of TBI, the underlying pathophysiologic processes common to all TBI presentations can now be considered together. CT imaging is often normal in this population. However, emerg ing evidence indicates that 3-tesla (3T) MRI scans with greater image resolution can identify pathology consistent with acute brain injury such as contusion and microhemorrhage. When patients with mTBI have CT and/or MRI abnormalities, they are often referred to as having complicated mTBI and are more likely to have an unfavorable outcome. SKULL FRACTURE, EXTRA-AXIAL HEMATOMA, CONTUSION, AND AXONAL INJURY ■ ■SKULL FRACTURE A blow to the skull that exceeds the elastic tolerance of the bone causes a fracture. Intracranial lesions accompany roughly two-thirds of skull fractures, and the presence of a fracture increases many-fold

the chances of an underlying subdural or epidural hematoma. Con sequently, fractures are primarily markers of the site and severity of injury. If the underlying arachnoid membrane has been torn, fractures also provide potential pathways for entry of bacteria to the cerebrospi nal fluid (CSF) with a risk of meningitis and for leakage of CSF outward through the dura. If there is leakage of CSF, severe orthostatic headache results from lowered pressure in the spinal fluid compartment.

Most fractures are linear and extend from the point of impact toward the base of the skull. Basilar skull fractures are often exten sions of adjacent linear fractures over the convexity of the skull but may occur independently owing to stresses on the floor of the middle cranial fossa or occiput. Basilar fractures are usually parallel to the petrous bone or along the sphenoid bone and directed toward the sella turcica and ethmoidal groove. Although most basilar fractures are uncomplicated,

they can cause CSF leakage, pneumocephalus, and delayed cavernous-carotid fistulas. Hemotympanum (blood behind the tympanic membrane), ecchymosis over the mastoid process (Battle sign), and periorbital ecchymosis (“raccoon sign”) are clinical signs associated with basilar fractures. PART 13 Neurologic Disorders ■ ■EPIDURAL AND SUBDURAL HEMATOMAS Hemorrhages between the dura and skull (epidural) or beneath the dura (subdural) have characteristic clinical and imaging features. They are sometimes associated with underlying brain contusions and other injuries, often making it difficult to determine the relative contribution of each component to the clinical state. The mass effect of raised intra cranial pressure (ICP) caused by these hematomas can be life threaten ing, making it imperative to identify them rapidly by CT or MRI scan and to surgically remove them when appropriate. Epidural Hematoma (Fig. 454-1) These highly dangerous lesions usually arise from an injury to a meningeal arterial vessel and evolve rapidly. They are often accompanied by a “lucid interval” of several minutes to hours prior to neurologic deterioration. They occur in up to 10% of cases of severe head injury but are less often associated with underlying cortical damage compared to subdural hematomas. Rapid surgical evacuation and ligation or cautery of the damaged ves sel, usually the middle meningeal artery that has been lacerated by an overlying skull fracture, is indicated. If recognized and treated rapidly, patients often have a favorable outcome. Acute Subdural Hematoma (Fig. 454-2) Direct cranial trauma may be minor and is not always required for acute subdural hemor rhage to occur, especially in the elderly and those taking anticoagulant medications. Acceleration forces alone, as from whiplash, are some times sufficient to produce subdural hematoma. Up to one-third of patients have a lucid interval lasting minutes to hours before coma FIGURE 454-1 Acute epidural hematoma. The tightly attached dura is stripped from the inner table of the skull, producing a characteristic lenticular-shaped hemorrhage on noncontrast computed tomography scan. Epidural hematomas are usually caused by tearing of the middle meningeal artery following fracture of the temporal bone.

FIGURE 454-2 Acute subdural hematoma. Noncontrast computed tomography scan reveals a hyperdense clot that has an irregular border with the brain and causes more horizontal displacement (mass effect) than might be expected from its thickness. The disproportionate mass effect is the result of the large rostral-caudal extent of these hematomas. Compare to Fig. 454-1. supervenes, but most are drowsy or comatose from the moment of injury. A unilateral headache and slightly enlarged pupil on the side of the hematoma are frequently, but not invariably, present. Small subdu ral hematomas may be asymptomatic and usually do not require surgi cal evacuation if they do not enlarge. Stupor or coma, hemiparesis, and unilateral pupillary enlargement are signs of larger hematomas. The bleeding that causes larger subdural hematomas is primarily venous in origin, although arterial bleeding sites are sometimes found at opera tion, and a few large hematomas have a purely arterial origin. In an acutely deteriorating patient, an emergency craniotomy is required. In contrast to epidural hematomas, there is significant morbidity and mortality associated with acute subdural hematomas that require surgery. Chronic Subdural Hematoma A subacutely evolving syndrome due to subdural hematoma occurs days or weeks after injury with drowsiness, headache, confusion, or mild hemiparesis, usually in the elderly with age-related atrophy and often after only minor or unno ticed trauma. On imaging studies, chronic subdural hematomas appear as crescentic clots over the convexity of one or both hemispheres, most commonly in the frontotemporal region (Fig. 454-3). A history of trauma may or may not be elicited in relation to chronic subdural hematoma; the injury may have been trivial and forgotten, particularly in the elderly and those with clotting disorders. Headache is common FIGURE 454-3 Computed tomography scan of chronic bilateral subdural hematomas of different ages. The

collections began as acute hematomas and have become hypodense in comparison to the adjacent brain after a period during which they were isodense and difficult to appreciate. Some areas of resolving blood are contained on the more recently formed collection on the left (arrows).

but not invariable. Additional features that may appear weeks later include slowed thinking, vague change in personality, seizure, or a mild hemiparesis. The headache typically fluctuates in severity, some times with changes in head position. Drowsiness, inattentiveness, and incoherence of thought are generally more prominent than focal signs such as hemiparesis. Rarely, chronic hematomas cause brief episodes of hemiparesis or aphasia that are indistinguishable from transient ischemic attacks. CT without contrast initially shows a low-density mass over the convexity of the hemisphere. Between 2 and 6 weeks after the initial bleeding, the clot becomes isodense compared to adjacent brain and may be inapparent. Many subdural hematomas that are several weeks in age contain areas of blood and intermixed serous fluid. Infusion of contrast material demonstrates enhancement of the vascular fibrous capsule surrounding the collection. MRI reliably identifies both sub acute and chronic hematomas. Clinical observation coupled with serial imaging is a reasonable approach to patients with few symptoms and small chronic subdural collections that do not cause mass effect. Treatment with surgical evacuation through burr holes is usually successful, if a cranial drain is used postoperatively. The fibrous membranes that grow from the dura and encapsulate the collection may require removal with a craniotomy to prevent recurrent fluid accumulation. ■ ■TRAUMATIC SUBARACHNOID HEMORRHAGE Subarachnoid hemorrhage (SAH) is common in TBI. Rupture of small cortical arteries or veins can cause bleeding into the subarachnoid space. Traumatic SAH is often seen in the sulci and is frequently the only radiographic finding on CT following mild TBI. SAH occurs dif fusely after severe TBI and confers an increase in mortality. In mild TBI, SAH provides an objective imaging biomarker for TBI and, in some patients, is associated with unfavorable outcomes. ■ ■CONTUSION (FIG. 454-4) A surface bruise of the brain, or contusion, consists of varying degrees of petechial hemorrhage, edema, and tissue destruction. Contusions and deeper hemorrhages result from mechanical forces that displace and compress the hemispheres forcefully and by deceleration of the brain against the inner skull, either under a point of impact (coup lesion) or, as the brain swings back, in the antipolar area (contrecoup lesion). Trauma sufficient to cause prolonged unconsciousness usu ally produces some degree of contusion. Blunt deceleration impact, as occurs against an automobile dashboard or from falling forward onto a hard surface, causes contusions on the orbital surfaces of the frontal lobes and the anterior and basal portions of the temporal lobes. With lateral forces, as from impact on an automobile door frame, contusions are situated on the lateral convexity of the hemisphere. The clinical signs of contusion are determined by the location and size of the lesion; FIGURE 454-4 Traumatic cerebral contusion. Noncontrast computed tomography scan demonstrating a hyperdense hemorrhagic region in the anterior temporal lobe.

often, there are no focal abnormalities with a routine neurologic exam, but these injured regions are later the sites of gliotic scars that may produce seizures. A hemiparesis or gaze preference is fairly typical of moderately sized contusions. Large bilateral contusions produce stupor with extensor posturing, while those limited to the frontal lobes cause a taciturn state. Contusions in the temporal lobe may cause delirium or an aggressive, combative syndrome. Torsional or shearing forces within the brain can cause hemorrhages of the basal ganglia and other deep regions. Large contusions and hemorrhages after minor trauma should raise concerns for coagulopathy due to an underlying disease or more commonly anticoagulant therapy.

Acute contusions are easily visible on CT and MRI scans, appearing as inhomogeneous hyperdensities on CT and as hyperintensities on T2 and fluid-attenuated inversion recovery (FLAIR) MRI sequences; there is usually surrounding localized brain edema and some subarachnoid bleeding. Blood in the CSF due to trauma may provoke a mild inflam matory reaction. Over a few days, contusions acquire a surrounding contrast enhancement and edema that may be mistaken for tumor or abscess. CHAPTER 454 ■ ■AXONAL INJURY (FIG. 454-5) Traumatic axonal injury (TAI) is one of the most common injuries after TBI. There is disruption, or shearing, of axons at the time of impact, and this is associated with microhemorrhages. It occurs follow ing high-speed deceleration injuries, such as motor vehicle collisions (see Johnson et al, 2013, in Further Readings). The presence of four or more areas of TAI is called diffuse axonal injury (DAI) and, when widespread, has been proposed to explain persistent coma and the veg etative state after TBI (Chap. 30). Only severe TAI lesions that contain substantial blood are visualized by CT, usually in the corpus callosum and centrum semiovale. More commonly, the CT will be negative for TAI, but subsequent MRI, particularly gradient-echo or susceptibilityweighted imaging, will show hemosiderin deposits reflective of micro hemorrhages in addition to the axonal damage on diffusion sequences. Traditionally, TAI and DAI have been considered as sequelae much more likely to result from moderate and severe injuries. Accumulating evidence has demonstrated that diffuse white matter abnormalities purportedly reflective of axonal injury, such as changes in microstruc ture and neurite density, are quite common in mild TBI as well. The degree of these changes correlates with metrics of injury severity (e.g., symptom burden) and recovery duration. Concussion and Other Traumatic Brain Injuries ■ ■CRANIAL NERVE INJURIES The cranial nerves most often injured with TBI are the olfactory, optic, oculomotor, and trochlear nerves; the first and second branches of the trigeminal nerve; and the facial and auditory nerves. Anosmia and an apparent loss of taste (actually a loss of perception of aromatic flavors, FIGURE 454-5 Multiple small areas of hemorrhage and tissue disruption in the white matter of the frontal lobes on noncontrast computed tomography scan. These appear to reflect an extreme type of the diffuse axonal shearing lesions that occur with closed head injury.

with retained elementary taste perception) occur in ~10% of persons with serious head injuries, particularly from falls on the back of the head. This is the result of displacement of the brain and shearing of the fine olfactory nerve filaments that course through the cribriform bone. At least partial recovery of olfactory and gustatory function is expected, but if bilateral anosmia persists for several months, the prognosis is poor. Partial optic nerve injuries from closed trauma result in blurring of vision, central or paracentral scotomas, or sector defects. Direct orbital injury may cause short-lived blurred vision for close objects due to reversible iridoplegia. Diplopia limited to downward gaze and cor rected when the head is tilted away from the side of the affected eye indi cates trochlear (fourth nerve) nerve damage. It occurs frequently as an isolated problem after minor head injury or may develop for unknown reasons after a delay of several days. Facial nerve injury caused by a basilar fracture is present immediately in up to 3% of severe injuries; it may also be delayed for 5–7 days. Fractures through the petrous bone, particularly the less common transverse type, are liable to produce facial palsy. Delayed facial palsy occurring up to a week after injury, the mechanism of which is unknown, has a good prognosis. Injury to the eighth cranial nerve from a fracture of the petrous bone causes loss of hearing, vertigo, and nystagmus immediately after injury. Deafness from eighth nerve injury is rare and must be distinguished from blood in the middle ear or disruption of the middle ear ossicles. Dizziness, tinnitus, and high-tone hearing loss occur from cochlear concussion.

PART 13 Neurologic Disorders ■ ■SEIZURES Convulsions are surprisingly uncommon immediately after TBI, but a brief period of tonic extensor posturing or a few clonic movements of the limbs just after the moment of impact can occur. However, the corti cal scars that evolve from contusions are highly epileptogenic and may later manifest as seizures, even after many months or years (Chap. 436). The severity of injury roughly determines the risk of future seizures. It has been estimated that 17% of individuals with brain contusion, sub dural hematoma, or prolonged LOC will develop a seizure disorder and that this risk extends for an indefinite period of time, whereas the risk is ≤2% after mild injury. The majority of convulsions in the latter group occur within 5 years of injury but may be delayed for decades. Penetrat ing injuries have a much higher rate of subsequent epilepsy. CLINICAL SYNDROMES AND TREATMENT OF HEAD INJURY ■ ■CONCUSSION/MILD TBI The patient who has briefly lost consciousness or been stunned after a minor head injury usually becomes fully alert and attentive within minutes but may complain of headache, dizziness, faintness, nausea, a single episode of emesis, difficulty with concentration, a brief amnestic period, or slight blurring of vision. This typical concussion syndrome has a good prognosis with little risk of subsequent deterioration. Chil dren are particularly prone to drowsiness, vomiting, and irritability, symptoms that are sometimes delayed for several hours after apparently minor injuries. Vasovagal syncope that follows injury may cause undue concern. Generalized or frontal headache is common in the following days. It may be migrainous (throbbing and hemicranial) in nature or aching and bilateral. After several hours of observation, patients with minor injury may be accompanied home and observed for a day by a family member or friend, with written instructions to return if symp toms worsen. Persistent severe headache and repeated vomiting in the context of normal alertness and no focal neurologic signs is usually benign, but CT should be obtained and a longer period of observation is appro priate. The decision to perform imaging tests also depends on clinical signs that indicate that the impact was severe (e.g., persistent confu sion, repeated vomiting, palpable skull fracture); the presence of other serious bodily injuries, an underlying coagulopathy, or age >65 years; and on the degree of surveillance that can be anticipated after dis charge. Guidelines have also indicated that older age (>65 years), two or more episodes of vomiting, >30 min of retrograde or persis tent anterograde amnesia, seizure, and concurrent drug or alcohol

intoxication are sensitive (but not specific) indicators of intracranial hemorrhage that justify CT scanning. Though not incorporated into conventional clinical practice guide lines, growing evidence suggests that MRI improves sensitivity for detection of small intracranial hemorrhages and other lesions in mild TBI patients, particular among those with negative findings on CT. Specifically, intracranial abnormalities are fairly common on MRI (27%) in CT-negative patients. Further, acute MRI findings have prog nostic utility in predicting recovery and outcome after mTBI/concus sion (e.g., risk of functional impairment, time to return to activity). Blood-based (serum and plasma) biomarkers of astrocyte damage/ astrogliosis (glial fibrillary acidic protein [GFAP]) and neuronal injury (ubiquitin carboxy-terminal hydrolase L1 [UCHL1]) also hold promise in improving detection and outcome prediction across the full spec trum of TBI. With development and regulatory approval of new rapid assay systems, these biomarkers can now be used for real-time pointof-care assessment; GFAP in particular has high discriminant ability to detect intracranial abnormalities, as well as potential to differentiate CT+, CT–/MRI+, and CT–/MRI– patients. Similar to MRI, emerging biomarkers appear to have not only diagnostic but also prognostic util ity in predicting the trajectory of recovery and functional impairments weeks and months after TBI. ■ ■SPORT-RELATED CONCUSSION Concussion is a frequent injury in contact and collision sports (e.g., football, hockey, wrestling) at all levels of participation, including youth sports. Head injury associated with

sport and recreational activ ity accounts for 45% of TBI-related emergency department visits in children age 17 years and under. Over the last decade, data from the Centers for Disease Control and Prevention indicate a 27% decrease in emergency department visits for sport- and recreation-related TBI in the United States between 2012 and 2018, with a specific decline in contact sport–related visits by 32%. Given that national and state surveillance systems continue to report increased sport-related concus sion rates over the same time period, it could be inferred that diagnosis and management of sport-related concussion outside of the emergency department have increased. The natural history of clinical recovery following sport-related con cussion has been a subject of substantial ongoing research. Recent large prospective studies have reinforced earlier indications that the acute recovery is favorable. For example, a longitudinal study of over 34,000 collegiate athletes (1751 who experienced concussion) from 22 institu tions found that median time to symptom resolution was 6.4 days and median time for return to sport was 12.8 days. By 1 month after injury, 92% of athletes had experienced resolution of symptoms and 85% had been cleared to return to sport. Across several studies, postinjury symptom burden is the most robust predictor of recovery and risk of prolonged symptoms. Other injury/postinjury factors associated with longer return to sport time were continued play following injury and access to health care providers. Several other prospective studies have replicated that the overwhelming majority of athletes across ages (pediatric to adult) achieve a complete recovery in symptoms, cognitive functioning, postural stability, and other functional impairments over a period of 1–4 weeks following concussion. There continues to be a growing focus toward a more rapid return to activity and early rehabilitation following injury. Specifically, while experts agree that initial rest after injury is beneficial for recovery, extended inactivity beyond 5 days can be detrimental and increase risk for protracted recovery. Rather, active rehabilitation involv ing supervised subthreshold exercise has been shown to decrease the duration of symptoms and reduce risk of a protracted recovery. Cervicovestibular rehabilitation has been recently recommended for those who experience neck pain, dizziness, or headaches for >10 days after injury. Preliminary evidence suggests that earlier return to learn/school is associated with faster injury recovery, and there has been an increased focus on facilitating a faster and optimal return to learn/school through accommodations. This could include, but is not limited to, a shortened schedule or built-in breaks for fatigue, opportunity for a quiet work

environment to prevent headaches or attentional difficulties, and setting expectations for course work to reduce anxiety, among other accommodations. Most patients (>90%) experience a full return to learn without accommodations by 10 days. There are a small, select percentage of athletes who remain symp tomatic or impaired on functional testing well beyond the window of recovery commonly reported in group studies. The greatest challenge arguably still facing sport medicine clinicians and public health experts is how to most effectively manage and reduce risk in this subset of athletes who do not follow the “typical” course of recovery. The precise likelihood that an athlete will not follow the typical course of rapid, spontaneous recovery and instead exhibit prolonged postconcussive symptoms or other functional impairments after concussion remains unclear. In addition to the injury-related or postinjury factors identi fied above, a preinjury mental health disorder, migraine, and prior concussion have been consistently associated with the potential for prolonged recovery. Following acute concussion, multimodal advanced neuroimaging has demonstrated a variety of changes, including decreased cerebral blood flow, increased global and local functional connectivity, and alterations in white matter microstructure reflecting axonal organiza tion. In general, these metrics correlate with measures of injury severity, and resolution of

these changes tends to parallel clinical recovery. How ever, a number of studies have shown that slight changes on advanced multimodal imaging can persist even after symptoms have fully resolved, supporting the concept that the “tail” of neurobiologic recov ery may extend beyond the time course of apparent clinical recovery. In the current absence of adequate data, a commonsense approach to athletic concussion has been to remove the individual from play immediately and avoid contact sports for at least several days after a mild injury, and for a longer period if there are more severe injuries or if there are protracted neurologic symptoms such as headache and difficulty concentrating. No individual should return to play unless all concussion-related symptoms have resolved and an assessment has been made by a health care professional who has experience with treatment of concussion. Validated symptom inventories, such as the Rivermead Post-Concussion Symptom Questionnaire (Table 454-2), have been developed to aid clinicians with recording and quantifying the diverse range of physical, cogni tive, and behavioral symptoms that can occur following concussion. In addition to characterizing the constellation of acute symptoms and their severity, symptom inventories can be beneficial to track the course and resolution of symptoms through recovery. Differ entiating concussion-related symptoms from factors that may be also influencing endorsement (e.g., preinjury mood disorders) is an important component of managing recovery from sport-related concussion. Once cleared, the individual can then begin a gradu ated program of increasing activity. These guidelines are designed TABLE 454-2 Review of Concussion Symptoms PHYSICAL COGNITIVE BEHAVIORAL Headaches Forgetfulness or poor memory Being irritable, easily angered Dizziness Poor concentration Feeling depressed or tearful Nausea and/or vomiting Taking longer to think Feeling frustrated or impatient Noise sensitivity Restlessness Sleep disturbance Fatigue Blurred vision Light sensitivity Double vision Note: Items were adapted from the Rivermead Post-Concussion Symptom Questionnaire. Each item is rated on a 5-point Likert scale (0–4), as follows: 0 = Not experienced at all; 1 = No more of a problem now than preinjury; 2 = A mild problem; 3 = A moderate problem; 4 = A severe problem. Total scores can range from 0–64.

in part to avoid a perpetuation of symptoms but also to prevent the rare second-impact syndrome, in which diffuse and fatal cerebral swelling follows a second minor head injury.

■ ■POSTCONCUSSIVE STATES There has been a recent paradigm shift from the term postconcussion syndrome (PCS) to persistent postconcussion symptoms (PPCS) in an effort to avoid classifying prolonged sequelae under a nonspecific diagnostic label and a shifting focus toward identifying and targeting treatment toward direct areas of persistent difficulty. PPCS following mild TBI could include symptoms of fatigue, dizziness, headache, and/ or difficulty in concentration. Management is difficult and generally requires identification and management of the specific problem or problems that are most troubling to the individual. A clear explanation and education around the symptoms that may follow concussion have been shown to reduce subsequent complaints. Care is taken to avoid prolonged use of drugs that produce dependence. Headache may ini tially be treated with acetaminophen and small doses of amitriptyline. Vestibular exercises (Chap. 24) and small doses of vestibular suppres sants such as promethazine (Phenergan) may be helpful when dizziness is the main problem. After mild or moderate injury, patients who have difficulty with memory or with complex cognitive tasks at work may be reassured to know that these problems usually improve over several months, and a reduced workload or other accommodations may be prescribed in the interim. For select cases, speech and language therapy intervention may be appropriate. CHAPTER 454 Concussion and Other Traumatic Brain Injuries For

the vast majority of individuals with mTBI, symptoms of PPCS subside and resolve within a few weeks of injury. For a subset of indi viduals with mTBI, however, complaints of postconcussion symptoms persist beyond the expectation derived from TBI severity markers. Subtypes of PPCS have been proposed to improve characterization of specific symptoms or types of sequelae following mTBI. These include neurologic, cognitive, behavioral, or somatic complaints that continue beyond the acute and subacute periods, becoming chronic and often operationalized as persisting beyond 3 months. Although the overall risk of experiencing PPCS following mTBI is low, the frequency of mTBI patients who present in a clinical setting is believed to be higher. mTBI patients with PPCS frequently present to the outpatient clinics of primary care physicians, physiatrists, or neurologists seeking relief for lingering related symptoms. While some patients will have already received an initial medical workup to rule out a more serious brain injury during the acute phase, many patients will have had no prior contact with health care specialists. A medical workup ordered in the outpatient setting for PPCS-related complaints is typically unremark able for any identifiable neurologic cause to account for the persist ing symptoms reported by the patient. The development of uniform decision trees or “standard of care” treatment regimens for PPCS has been limited by the diversity of symptoms that patients experience, even within mTBI subgroups that have sustained very similar injury patterns. While some patients experience somatic symptoms, others complain of subjective cognitive or behavioral changes. Symptom inventories (Table 454-2) can be helpful in documenting the broad range of these symptoms and serve as a metric for improvement fol lowing symptom-based treatment. Active rehabilitation for the treatment of PPCS involving subthresh old exercise has increased in popularity over recent years and has gained empirical support for its effectiveness as a useful intervention for protracted recovery. PPCS are often influenced by diverse cognitive, emotional, medical, psychosocial, and motivational factors. Because of this complexity, treatments targeting persistent and refractory symptoms should be tailored to the needs and expectations of the individual patient, with referrals to specialists as needed for assistance with management of headache, neck and back pain, dizziness and vertigo, and other persist ing symptoms. A comprehensive review of concussion and persisting symptoms, presented in Table 454-2, allows for development of an individualized approach that leverages currently available treatment for those sequelae that are most bothersome to the patient (e.g., vestibu lar or cervicovestibular rehabilitation therapy for vertigo, melatonin

for sleep disturbance). Patients are frequently referred to behavioral health providers such as neuropsychologists, rehabilitation psycholo gists, health psychologists, and/or psychiatrists for a variety of reasons, but particularly when they are experiencing persistent cognitive, emotional, or behavioral changes. Patients with mood disorders (e.g., depression), anxiety disorders (e.g., posttraumatic stress disorder), or adjustment reactions may benefit from psychiatric consultation for appropriate medication trials or from time-limited psychotherapy such as cognitive behavioral therapy.

Due to the complexity of presentation and varying diagnostic cri teria, there are limited studies regarding overall prognosis of PPCS. However, treatment targeted to the individual’s specific persisting difficulties can improve functional outcomes and patient-rated quality of life. Further, collaborative care has been shown to improve out comes among patients experiencing PPCS. These improved outcomes are likely due to a multidisciplinary team’s ability to simultaneously address the diverse set of difficulties that can occur with PPCS.

OF INTERMEDIATE SEVERITY Patients who are not fully alert or have persistent confusion, behav ioral changes, extreme dizziness, or focal neurologic signs such as hemiparesis should be admitted to the hospital and undergo a cere bral imaging study. A cerebral contusion or hematoma will usually be found. Common syndromes include (1) delirium with a disinclina tion to be examined or moved, expletive speech, and resistance if dis turbed (anterior temporal lobe contusions); (2) a quiet, disinterested, slowed mental state (abulia) alternating with irascibility (inferior frontal and frontopolar contusions); (3) a focal deficit such as aphasia or mild hemiparesis (due to subdural hematoma or convexity contu sion or, less often, carotid artery dissection); (4) confusion and inat tention, poor performance on simple mental tasks, and fluctuating orientation (associated with several types of injuries, including those described above, and with medial frontal contusions and interhemi spheric subdural hematoma); (5) repetitive vomiting, nystagmus, drowsiness, and unsteadiness (labyrinthine concussion, but occasion ally due to a posterior fossa subdural hematoma or vertebral artery dissection); and (6) diabetes insipidus (damage to the median emi nence or pituitary stalk). Injuries of this degree can be complicated by drug or alcohol intoxication, and clinically inapparent cervical spine injury may be present. Blast injuries are often accompanied by rupture of the tympanic membranes. After surgical removal of hematomas, patients in this category improve over weeks to months. During the first week, the state of alertness, memory, and other cognitive functions often fluctuate, and agitation and somnolence are common. Behavioral changes tend to be worse at night, as with many other encephalopathies, and may be treated with small doses of antipsychotic medications. Subtle abnor malities of attention, intellect, spontaneity, and memory return toward normal weeks or months after the injury, sometimes abruptly. How ever, the full extent of recovery may not be realized for several years. Persistent cognitive problems are discussed below. ■ ■SEVERE INJURY Patients who are comatose from the moment of injury require imme diate neurologic attention and resuscitation. After intubation, with care taken to immobilize the cervical spine, the depth of coma, pupil lary size and reactivity, limb movements, and Babinski responses are assessed. As soon as vital functions permit and cervical spine x-rays and a CT scan have been obtained, the patient should be transported to a critical care unit. Hypoxia should be reversed, and normal saline used as the resuscitation fluid in preference to albumin. The finding of an epidural or subdural hematoma or large intracerebral hemorrhage is usually an indication for prompt surgery and intracranial decom pression in an otherwise salvageable patient. Measurement of ICP with a ventricular catheter or fiberoptic device in order to guide treat ment has been favored by many units but has not improved outcome. Similarly, induced hypothermia has shown no benefit. Hyperosmolar intravenous solutions are used in various regimens to limit intracranial pressure. Prophylactic antiepileptic medications are recommended for

7 days and should be discontinued unless there are multiple seizures after injury. Management of raised ICP, a frequent feature of severe head injury, is discussed in Chap. 318. Despite the improvement in mortality for severe TBI over the past few decades, a great deal of therapeutic nihilism persists in TBI. The common use of a 6-month outcome for TBI clinical studies reinforces this misconception. The recovery from severe TBI can take years. Fur thermore, the ability to predict long-term outcome is limited and fre quently incorrect. Best-practice guidelines recommend, in the absence of brain death, that aggressive therapy be instituted for at least 72 h in the acute injury period. ■ ■LONG-TERM OUTCOMES IN TBI Continued follow-up of prospective studies has increased awareness of TBI as a chronic condition with evolving changes and needs over sev eral years after injury. While the majority of individuals remain broadly stable, clinically meaningful improvement and decline across multiple domains (psychiatric, cognitive, and

functional outcomes) have been observed from 2 to 7 years after injury, regardless of initial TBI severity. The direction of the changes beyond 1 year after injury can be variable (may decline and improve later, or vice versa). Collectively, this indicates that functional status remains dynamic beyond 1 year after injury, a conventionally considered plateau of recovery. Future investigation is required to better understand which factors are associated with improvement and decline beyond 1 year after injury. Regardless, there is growing awareness that TBI is an evolving condition that requires ongoing monitoring, rehabilitation, and support to address individual patient needs. Chronic difficulties associated with TBI (characterized above), as opposed to level of risk for Alzheimer's disease and related demen tias (ADRD), is an important distinction in determining long-term outcomes of TBI. TBI (aggregated mild to severe) is associated with a 63–96% increased risk of all-cause dementia. The degree of risk for dementia ranges along the gradient of TBI severity (i.e., greatest risk among severe injuries). There is some evidence that repeated mTBI or sport-related concussions may be associated with elevated risk as well. To date, however, investigations have less reliably established mTBI as a robust risk factor for dementia, likely due to methodologic heterogene ity (e.g., use of different diagnostic criteria, exposure misclassification, self-report vs physician diagnoses of TBI or dementia). Though an identified risk factor for all-cause dementia, pathophysi ologic and epidemiologic factors that underlie the association between TBI and risk of specific neurodegenerative pathologies and dementia subtypes are not well understood. As a result, associations between TBI and clinical syndromes (e.g., Alzheimer's disease, Parkinson's dis ease, amyotrophic lateral sclerosis) or distinct neuropathologies (e.g., beta-amyloid, Lewy bodies, transactive response DNA-binding protein 43) have been inconsistently reported in the literature. In a large study involving clinical and neuropathologic data from three pooled prospec tive studies of community-based cohorts, a significant relationship was found between TBI with LOC >1 h and subsequent Parkinson's disease diagnosis, progression rate of parkinsonism, and Lewy body accumu lation at postmortem examination. Positron emission tomography (PET) studies have allowed for in vivo investigation of neuropathologic deposition and have failed to consistently observe associations between remote TBI (mild through severe) and amyloid or tau deposition. Among former contact and collision sport athletes, exposure to repetitive head impacts (RHI) involving external blows that do not produce signs and symptoms of mTBI/concussion is common. The brains of these patients with substantial RHI exposure may display a characteristic deposition of tau protein in neurons located in the super ficial cortical layers and perivascular regions and particularly in the depths of sulci. This pattern has been defined as the pathognomonic lesion of chronic traumatic encephalopathy (CTE). The degree to which this neuropathologic finding is present in nonathlete populations with potential RHI exposure (e.g., former military service members and veterans, victims of intimate partner violence) is uncertain, although the prevalence was found to be low in a large autopsy study of military service members.

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