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Pediatric athletic head injuries are a significant problem, especially for emergency medicine physicians. The dedication of many young athletes to performance and their avoidance of restrictions makes it imperative that ED physicians recognize and give appropriate recommendations to parents and coaches of young athletes. The cumulative damage that may result from repetitive minor head trauma is not recognized by the majority of athletes, and the ED physician has the burden of conveying these potential risks to the family. This article provides a comprehensive review of sports-related head injuries and recommendations on grading of concussions, imaging, and the safe return of the athlete to competition.

—The Editor

Sports-Related Head Injuries: Learn the Rules of this Serious Game

Authors: **Don W. Penney, MD, MSc, FACEP,** Neurological Surgeon; Professor, Department of Emergency Medicine, Medical College of Georgia, Augusta; and **Ronald M. Perkin, MD, MA, FAAP, FCCM,** Professor and Chairman, Department of Pediatrics, The Brody School of Medicine at East Carolina University, Greenville, NC.

Peer Reviewer: **Norman C. Christopher, MD,** Director, Emergency/Trauma Services, Children's Hospital Medical Center of Akron, Akron, OH.

Introduction

There are approximately 300,000 sports-related head injuries each year in the United States.[1-3] Most of these head injuries are considered mild to moderate in severity, and many go unrecognized and untreated. Increasingly, team trainers, coaches, and parents are utilizing the emergency department to provide care and management recommendations. Optimal care of the head-injured athlete requires an appreciation of the types of head injuries that occur and a firm understanding of the treatment options. It is equally important to understand and recognize the constellation of signs and symptoms that diagnose a post-concussion syndrome. Prevention of the potentially fatal second-impact syndrome (SIS) by accurate assessment of the athlete who has suffered a concussion and ensuring the resolution of his signs and symptoms prior to his return to play is mandatory.[4,5]

Sports-related head injuries commonly are seen in the pediatric population. Children with mild head injuries comprise up to 93% of all head-injured children admitted to the hospital.[6] As many as 20% of all pediatric head injuries have been reported to be sports related.[7] As in the adult population, it is incumbent upon health care professionals, trainers, and coaches to properly identify the severity of a child's head injury and provide the appropriate medical management before clearing him to return to competition.

Despite the high frequency of athletic-related head injuries in both the pediatric and adult populations, research on the topic is

scarce.[8-10] The fact that there is a dearth of Class I evidence (evidence provided by one or more well-designed randomized controlled clinical trials) attests to this. Management recommendations and current guidelines are based on Class II and Class III evidence only. At least 25 different guidelines for the evaluation of concussion have been published.[1,11] Although there is a common thread in each of these guidelines, there is no uniform consensus as to which of the published guidelines is most appropriate.

This manuscript provides a review of athletic head injuries and suggested methods of grading of concussions. A method of sideline neurological assessment, including a mini-mental status exam, also is presented. Discussions of post-concussion syndrome and SIS, as well as current recommendations for return to competition, are included.

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Epidemiology

The incidence of sports-related head injury varies regionally, and is influenced by the types of sports played (contact vs non-contact, organized vs non-organized, officiated vs non-officiated), the age of the population, and the existence of active injury prevention programs.

Sports are categorized by their probability for collision or contact.[12] In "collision" sports (e.g., boxing, ice hockey, football, and rodeo), athletes purposely hit or collide with each other or inanimate objects, including the ground, with great force. In "contact" sports (e.g., basketball and soccer), athletes routinely make contact with each other or inanimate objects, but usually with less force than in collision sports. Efforts usually are not made to separate collision and contact sports, because there is no clear dividing line between them. In "limited-contact" sports (e.g., softball and squash), contact with other athletes or inanimate objects is infrequent or inadvertent. Some limited-contact sports (e.g., downhill skiing and gymnastics) can be as dangerous as contact or collision sports. The categorization, however, gives an idea of the comparative likelihood that participation in a particular sport will result in acute traumatic injuries resulting from blows to the body.[12]

Certain sports, in particular, have a higher incidence of head injuries. These include football, boxing, rugby, ice hockey, soccer, martial arts, equestrian sports, and wrestling.

In the United States, football injuries far exceed other sports injuries. Football, because of the number of participants and the nature of the game, has the highest incidence of head injuries. With approximately 1.5 million participants yearly and up to 20% of the players sustaining a concussion, the issue of appropriate management is not inconsequential.[13,14] There are up to eight football-related head injury deaths each year in the United States.[15]

One group reported that 19% of high school athletes experience at least one minor head injury.[16] Alarmingly, the author stated that 70% of the injured players returned to competition the same day as an injury that resulted in loss of consciousness.

The National Athletic Trainer's Association (NATA) conducted a three-year study of the frequency, type, and severity of injuries in high school football for the 1986-1988 seasons.[17] This study involved 351 teams with more than 21,000 players, 5260 games, and 27,288 practices. There were 12,786 injuries reported; 598 (4.7%) were identified as concussions.

Types of Athletic Head Injuries

Sports should contribute to the physical and mental health of children. Unfortunately, head injuries occur in sports and can cause permanent, irreversible damage to the central nervous system.[18]

Most acute head injuries are caused by a combination of rotational (angular) and translational (linear) acceleration, as well as the force of impact to the head.[18] Rotational acceleration of the brain (caused by rapid turning of the head) can result in diffuse shearing forces deep in the brain, causing axonal injury. Rotational vectors applied to the head often produce injury in the

brain at anatomical sites of fixation of the brain, particularly in the midbrain as it passes through the tentorial opening at the foramen magnum. Translational acceleration will cause tensile (pulling apart) and compressive forces, which usually result in skull fractures and injury to the adjacent structures (i.e., blood vessels, brain parenchyma, dura). Brain acceleration can occur without impact to the head; 35% of concussions in college football involved no impact to the head.[18] Because adolescents are larger, faster, stronger, and more skilled at delivering a forceful impact than their younger counterparts, the incidence of head injury is higher in adolescents.[18,19]

Acute brain injuries can be divided into two categories: 1) focal brain injuries, including epidural hematomas (EDH) and subdural hematomas (SDH) as well as cerebral contusions; and 2) diffuse axonal damage, ranging from concussions to diffuse axonal injuries (DAI). DAI pathophysiologically results from shearing of white matter tracts from the cortex through the brainstem, producing coma in the most severe form. Recognition of focal brain injuries is crucial, because they potentially are the most life-threatening and require emergency transport and neurosurgical evaluation.[20] The most common focal brain injury and etiology of sports-related fatality (87% of all head-related football fatalities) is the SDH.[7,18,19]

The most common sports-related head injury is the concussion, the syndrome characterized by immediate and transient impairment of neural function due to mechanical force.[9,15,21] A concussion can occur even if there is no loss of consciousness (LOC).[18] The most common signs and symptoms include confusion, memory loss (antegrade and retrograde), decreased level of consciousness, headache, slowed information processing, diminished attention span, and decreased coordination immediately or within minutes of the head injury.[15,18]

Repeat head injury is of special concern because the effects of repeated concussion now are being recognized as cumulative in nature.[1,15,22-24] SIS results when a second concussive injury occurs prior to the resolution of symptoms from the first head injury.[1,4] The mechanism of SIS is believed to be loss of vascular autoregulation, which may result in cerebral edema and increased intracranial pressure (ICP).[23] Cognitive impairment and diminished neuropsychological functioning can result from repeated concussions.[23,24] Sports-related mild traumatic brain injuries (MTBI) in still-developing young athletes can have significant long-term consequences involving cognitive, memory, and fine motor functions.[25-27]

Concussion. Derived from the Latin *concussus* ("a shaking"), concussion is the most common type of head injury that occurs in athletes. There is no universal agreement on the standard definition or nature of concussion.[1,2,28] A frequently used definition of a concussion is a trauma-induced alteration in mental status that may or may not involve LOC.[15,29]

Concussions are sometimes described as MTBI.[1,26] The American Congress of Rehabilitation Medicine has proposed this definition of MTBI: A traumatically induced physiologic disruption of brain function with at least one of the following: 1) any period of documented loss of consciousness; 2) memory loss of events just before or just after the injury event; 3) any alteration of mental state at the time of injury event; 4) focal neurologic deficits that may or may not be transient.[30]

There are several common features of concussions that help define the nature of the concussive head injury. The features of concussion as listed by one group include: 1) it is caused by either a direct blow to the head or elsewhere on the body with an "impulsive" force transmitted to the head; 2) it results in an immediate and short-lived impairment of neurologic function; 3) it may result in neuropathologic changes, although the acute clinical symptoms largely reflect a functional disturbance rather than structural injury; and 4) it may result in a graded set of clinical syndromes that may or may not involve LOC.[31] The resolution of the clinical and cognitive symptoms typically follows a sequential course.

The hallmarks of cerebral concussion are confusion and amnesia.[15,29] The athlete with a concussion may demonstrate easy distractibility and poor vigilance. He or she may be unable to maintain a coherent stream of thought or carry out a sequence of goal-directed actions. The confusion and memory disturbance can be of immediate or delayed onset, necessitating close observation of the head-injured athlete.[29,32]

Consciousness is a multifaceted function of the brain dependent on alertness and awareness of self and the environment. These functions likewise involve sensation, perception, memory, attention, and cognition. Consciousness is not localized to a specific brain region, but permeates the entire brain.[33] Alteration of consciousness results when the integrity of the reticular activating system located throughout the brainstem is disrupted.[33] Other anatomical areas involved in consciousness include the lamina terminalis and the anterior frontal lobe. Historically, grading of concussions had been based on the presence or absence of a loss of consciousness. By definition, an athlete does not have to experience a loss of consciousness to have suffered a concussion.[29] Any immediate post-traumatic impairment of neural function, including an altered mental status, qualifies as a concussion.

Different schools of thought exist on grading the severity of concussion. *(See Table 1.)*[1,2,34-37] Some classifications place greater emphasis on the duration of confusion and post-traumatic amnesia, while others place more emphasis on the presence of a loss of consciousness.[28] All classifications of concussion are in agreement that the hallmarks of cerebral concussion are confusion and amnesia. No system of grading is foolproof.[18] Often the history obtained from the athlete is distorted by the very presence of the head injury, which produces confusion of the specific detail of the injury and is compounded by the athlete's willingness to under-report signs and symptoms in hopes of an earlier return to competition.

Recognition of a head injury is easy if the athlete has lost consciousness.[4] It is much more difficult to recognize the far more frequent head injuries in which there is no loss of consciousness, but rather only a transient loss of alertness. More than 90% of all cerebral concussions fall into this mild category, in which there

Table 1. Diagnostic Grading Scales for Sports-Related Concussion

Guideline	Severity of Grade 1	2	3
Cantu	1) No loss of consciousness 2) Post-traumatic amnesia lasts fewer than 30 min	1) Loss of consciousness lasts fewer than 5 min OR 2) Post-traumatic amnesia lasts longer than 30 min	1) Loss of consciousness lasts longer than 5 min OR 2) Post-traumatic amnesia lasts longer than 24 hr
Colorado	1) Confusion without amnesia 2) No loss of consciousness	1) Confusion with amnesia 2) No loss of consciousness	1) Loss of consciousness (of any duration)
Practice Parameter, American Academy of Neurology	1) Transient confusion 2) No loss of consciousness 3) Concussion symptoms or mental status change resolve in fewer than 15 min	1) Transient confusion 2) No loss of consciousness 3) Concussion symptoms or mental status change last longer than 15 min	1) Loss of consciousness (brief or prolonged)

has not been an LOC but only a brief period of post-traumatic amnesia or loss of mental alertness.[4] Because SIS can occur after a mild concussion, just as it can after a more serious head injury, it becomes important to recognize all grades of concussion.[4,15]

Attempts to qualify post-traumatic amnesia to determine the severity of head injury can be arduous. Post-traumatic amnesia (PTA) is the term used to describe the period following head injury in which the athlete is unable to remember. The duration of amnesia is determined by questioning the athlete after he or she has fully recovered. In practice, quantification of the duration of PTA can be unsatisfactory. Problems include the fact that it is determined retrospectively and depends on the athlete's own judgment of the time memory returned and his or her recollections of the associated events which allow it to be timed. Consideration of the duration of post-traumatic disorientation has been considered as an alternative to post-traumatic amnesia; however, research studies have not found the two to be coterminous. Other problems associated with determining PTA include variability from one examiner to another; whether PTA estimates include the time elapsed since the injury or time from clinical assessment; and variability of estimates from the athlete, if he or she is questioned more than once. Head-injured athletes commonly will experience a return of "islands of memory" before the return of continuous memory. Estimates of PTA duration made in the first hours after the injury have been reported to change in more than 25% of cases when athletes were interviewed after complete recovery.[38]

The importance of careful questioning to establish the precise return of continuous memory cannot be over-estimated. One group hypothesizes that there are two phases in the amnestic changes due to concussion; one occurs immediately after impact, and the second phase develops up to an hour later.[39] The return of "memory islands" could represent recovery of the first phase before the second has developed. Explanations of the second phase are speculative; however, localized cortical edema and biochemical changes have been proposed. "Islands of memory," as described by one research team, are considered to occur when a strong stimulus is applied during recovery from the immediate injury.[40]

A mild cerebral concussion does not result in an LOC. Athletes often have confusion and amnesia and can be distracted easily, demonstrating an inability to focus their attention and carry out goal-specific tasks. They often continue to play, as their gross motor skills usually are not impaired. This particular type of head injury is difficult to diagnose unless the athlete complains of symptoms, or a vigilant team of health care providers appreciates the occurrence of a head injury, often through an altered mental status.

A classic cerebral concussion is associated with a transient loss of consciousness. Unconsciousness occurs at the time of impact and recovery occurs within minutes. After regaining consciousness, athletes often will be confused and amnestic and can complain of headache, nausea, dizziness, and visual changes. Neuropsychological signs and symptoms that often are present include poor concentration, reduced attention span, reduced capacity to process information, and difficulty in integrative and abstract thinking. Unless the athlete is observed, questioned, and examined, a team physician or trainer could easily be misled that the athlete has had a complete recovery. Physically, the player may demonstrate nothing more than a vacant stare and some minor motor incoordination. It is imperative that an athlete with this type of injury not be allowed to return to competition because of the increased risk of re-injury and potential for development of SIS.[28]

Returning an athlete to competition following a head injury is determined by the severity of the injury and the history of previous head injuries. This implies the need for some form of medical record for each and every athlete that documents a history of antecedent injury. Studies have demonstrated that a player who

has suffered a concussion has a four times greater likelihood of having a second one, when compared to the athlete who has never had a concussion.[34]

Generally, the decision to return the athlete to active competition will be based on several factors, including:

- Presence of head injury;
- Presence of LOC;
- The length of the LOC;
- The presence of altered mental status (i.e., confusion, agitation, disorientation);
- The presence and length of amnesia;
- A period of observation and re-evaluation;
- The clearance of all concussive symptoms and sensorium; and
- A medical history with no previous head injuries or concussions.

In 1986, Robert Cantu proposed a set of guidelines for the safe return to competition for athletes who have suffered a concussion.[35,41] *(See Table 2.)* His guidelines, which have become widely accepted, are based on a grading of the concussion that is determined by the presence or absence of a loss of consciousness, the length of time of unconsciousness, and by the presence and length of post-traumatic amnesia. Numerous other classifications and guidelines for return to competition have subsequently been published. The Colorado Medical Society and the American Academy of Neurology guidelines are two that have been well accepted. It is important that one system be adopted and utilized by a team and that records are maintained on each individual player suffering a traumatic brain injury. The effect of concussion can be cumulative and if return to competition occurs too early, it can render the athlete vulnerable to repeat injury.[22]

Recognizing and managing Grade 2 and Grade 3 level concussions (per any grading system) require removal from the game or practice and a period of observation and retesting before return to competition. Generally, return to competition after concussion must be deferred until all symptoms have abated and diagnostic studies are normal.[28] Head computed tomography (CT) may be required if neurological symptoms continue. In the pediatric population, those who have suffered a sports-related head injury with an associated LOC; are amnestic at the time of evaluation; or who have continued headache, vomiting, or focal neurological deficit have a prevalence of intracranial injury detectable on CT that ranges from 0-7%.[10]

After any form of concussion, continued headache or associated concussive symptoms would require further evaluation and CT scanning. Any CT-documented post-traumatic pathology (i.e., contusion, hemorrhage, or edema) would conclude the player's season.

Post-Concussion Syndrome. Patients with minor head injuries may continue to have residual complaints stemming from their initial concussion.[8] Symptoms such as continued headache (especially with exertion), nausea, vertigo, dizziness, irritability, impaired memory, and impaired concentration and attention that fail to resolve after a head injury may represent a post-concussion syndrome.[18,28]

Proposed diagnostic criteria for the diagnosis of a post concussion syndrome have been published in the Diagnostic and Statistical Manual (1994) of the American Psychiatric Association (DSM-IV). The DSM-IV proposal criteria requires that two of three major criteria be present for the diagnosis of a post concussion syndrome. However, a significant number of concussions of the mild to moderate type (Cantu/Colorado classification) would not satisfy the DSM-IV criteria and, therefore, would not be eligible for the diagnosis of a post-concussion syndrome. Perhaps the presence of continued signs and symptoms after head injury should support a diagnosis of post-concussion syndrome.

Physicians and trainers should be alerted to continued complaints and symptoms in the head-injured athlete. Referral for further neurological evaluation and, possibly, imaging is recommended. Formal neuropsychological assessment can be beneficial in managing these patients.[22] A large number of patients with a post concussion syndrome have no neurological deficits on clinical examination, yet their symptoms can be disabling and may preclude them from participation in sports. Neuropsychological testing can detect subtle but significant cognitive abnormalities with much greater sensitivity than a standard neurological examination.[24]

Second-Impact Syndrome. SIS refers to a condition in which a fatal brain swelling, at times subclinical, can develop after a minor head injury in an athlete who still is symptomatic from a previous concussion.[4,28,32,42,43] What one group[44] called the "second-impact syndrome of catastrophic head injury" in 1984 was first described in 1973.[4,45] SIS underscores the need for strict adherence to return to competition guidelines, circumventing this potentially catastrophic occurrence.

Typically, the athlete suffers post-concussion symptoms after the first head injury. These may include headache, labyrinthine dysfunction, visual, motor, sensory changes, or mental difficulty, especially thought and memory process.[4] Before these symptoms resolve, which may take days to weeks, the athlete returns to competition and receives a second blow to the head. Affected athletes may appear stunned, but usually remain on their feet. What happens in the next seconds to minutes sets this syndrome apart from a concussion or even a SDH.[4,28] The athlete, conscious yet stunned, quite precipitously collapses to the ground, semicomatose with dilating pupils, loss of eye movement, and evidence of respiratory failure or apnea.[28]

Pathophysiologically, it is believed that subclinical brain edema from a previous head injury renders the brain more susceptive to further injury, even minor in nature. Current understanding of this syndrome is that the initial injury/concussion deranges the brain's autoregulatory and metabolic systems enough to produce vascular engorgement and poor brain compliance, allowing for marked changes in ICP with small changes in intracranial volume.[4,32] Brain compliance refers to the intracranial relationship of volume change to pressure change. Essential to this discussion is the fact that during the minutes to few days after concussion injury, brain cells that are not irreversibly destroyed remain alive but exist in a

Table 2. Examples of Differing Concussion Classification Systems and Return-to-Play Recommendations

CANTU GUIDELINES

Severity	First Concussion	Second Concussion	Third Concussion
Grade 1 (mild): No loss of consciousness; post-traumatic amnesia < 30 min	May return to play if asymptomatic	May return in 2 weeks if asymptomatic at that time for 1 week	Terminate season; may return next year if asymptomatic
Grade 2 (moderate): Loss of consciousness < 5 min or post-traumatic amnesia > 30 min	Return after asymptomatic for 1 week	Wait at least 1 month; may return if asymptomatic for 1 week; consider terminating season	Terminate season; may return next year if asymptomatic
Grade 3 (severe): Loss of consciousness > 5 min or post-traumatic amnesia > 24 hr	Wait at least 1 month; may return then if asymptomatic for one week	Terminate season; may return next year if asymptomatic	

COLORADO MEDICAL SOCIETY GUIDELINES

Severity	First Concussion	Second Concussion	Third Concussion
Grade 1 (mild): Confusion without amnesia; no loss of consciousness	May return to play if asymptomatic for at least 20 min	Terminate contest or practice for the day	Terminate season; may return in 3 months if asymptomatic
Grade 2 (moderate): Confusion with amnesia; no loss of consciousness	Terminate contest/practice; may return if asymptomatic for at least 1 week	Consider termination of season, but may return if asymptomatic for 1 month	Terminate season; may return to play next season if asymptomatic
Grade 3 (severe): Loss of consciousness	May return after 1 month if asymptomatic for 2 weeks at that time; may resume conditioning sooner if asymptomatic for 2 weeks	Terminate season; discourage any return to contact sports	

Cantu guidelines adapted from: Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Phys Sports Med* 1986;14:75-83.
Colorado Medical Society guidelines adapted from: Colorado Medical Society. *Report of the Sports Medicine Committee: Guidelines for the Management of Concussions in Sports* (revised). Denver:Colorado Medical Society;1991.

vulnerable state. This concept of injury-induced vulnerability describes the fact that patients suffering from head injury are extremely vulnerable to the consequences of even minor changes in cerebral blood flow and increases in ICP and apnea.[32] When head injury occurs, major compensatory mechanisms are set into action to accommodate for any increase in intracranial volume. If the limits of these compensatory mechanisms are exceeded, ICP begins to rise; a substantial increase in pressure with any subtle change in volume occurs. This increased ICP can result in herniation, cerebral ischemia, and brain death.[43,46]

The risk factors for SIS currently are not understood. Limited knowledge suggests that children and adolescents are at higher risk; hence, increased clinical vigilance may be necessary after all head injuries in this age group.[43]

Approximately 50 cases of SIS have been reported since 1980. The National Institute for Catastrophic Sports Injury Research reported 35 football-related cases between 1980 and 1993.[4]

The treatment of SIS should be prevention by adherence to return-to-competition guidelines after an initial head injury. Any athlete sustaining a second head injury prior to the resolution of symptoms from the antecedent injury has the potential to develop SIS.[4,32] An athlete who is symptomatic from a head injury must not participate in contact or collision sports until all cerebral symptoms have subsided, and preferably for at least one week after. Whether it takes days, weeks, or months to reach the asymptomatic state, the athlete must never be allowed to practice or compete while still suffering post concussion symptoms.[32]

Recognition of the condition on the field and initiating measures to reduce ICP is indicated. This form of head injury should be managed as a severe head injury, including control of the airway and rapid sequence intubation.[9] If signs of raised ICP are apparent,

or there is clinical evidence of herniation, hyperventilation and intravenous administration of mannitol should be considered until more definitive treatment is available at a medical facility. Prevention of hypotension and hypoxia is crucial. Rapid transport to a hospital with neurosurgical capabilities is mandatory.

Diffuse Axonal Injury. Diffuse axonal injury (DAI) is most common in victims of motor vehicle accidents but occasionally is seen in severe athletic-related head trauma. Severe DAI unaccompanied by a mass lesion occurs in almost half of patients with severe head injury and is responsible for one-third of all head-injury-related deaths.[15] It is the most common cause of persistent vegetative state and significant disability following head injury.

DAI is believed to be caused by angular or rotational forces applied to the head that result in a shearing type injury that stretches and tears axons diffusely.[1,47] When the reticular activating system of the brainstem is disrupted, consciousness is lost. Unconsciousness also can be produced by a diffuse injury to both cerebral hemispheres. The cerebral impairment may be so severe that the respiratory drive is affected, and the athlete hypoventilates or becomes apneic, hypoxic, and hypercapnic.[47,48] The extent of apnea is directly related to the amount of energy transmitted to the brainstem.[48]

The number of axons injured and degree of edema increase with the traumatic force. Less severe axonal injury is reversible; more severe injury can be permanent.[47]

Cerebral concussion is considered a mild form of DAI. Magnetic resonance imaging (MRI) can be helpful in demonstrating characteristic lesions (tissue tear hemorrhages) commonly associated with more severe forms of DAI; however, it does not determine the severity of DAI, which remains a clinical determination.

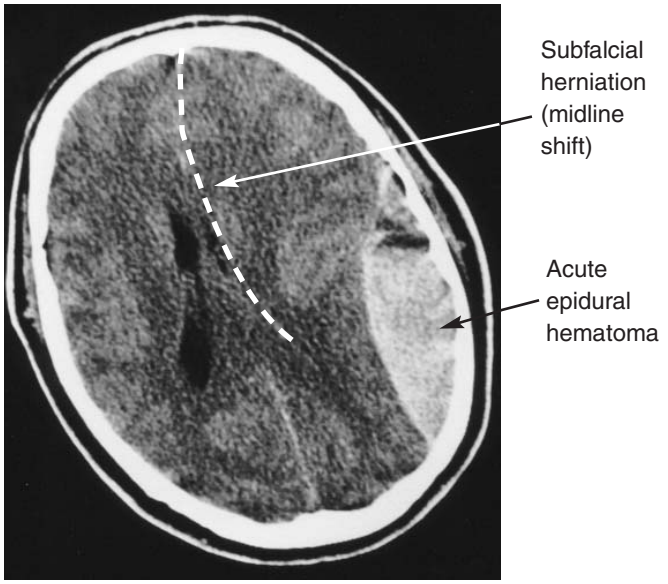
Intracranial Hemorrhage. Intracranial hemorrhage is the most common cause of death in athletes.[28] The four types of primary intracranial hemorrhages are epidural hematoma (EDH), SDH, intraparenchymal hematoma, and subarachnoid hematoma (SAH).

An athlete who has sustained severe head trauma can develop any form of intracranial hemorrhage. SAH is the most common post-traumatic hemorrhage; however, acute SDH is the most lethal and is the most common cause of death in athletes.

Epidural Hematoma. EDH is a hemorrhage that occurs between the periosteal layer of the dura mater and the skull. EDHs often are associated with skull fractures, usually are arterial in origin, and most commonly occur in the middle cranial fossa. The high incidence in the middle cranial fossa arises from the fact that the middle meningeal artery is located close to the squamous temporal bone, which easily fractures with trauma. The bony fragments can tear the vessel, resulting in an EDH. *(See Figure 1.)*[15]

An EDH usually is the most rapidly progressing intracranial hematoma; it may reach a fatal size in 30-60 minutes.[28] The athlete may have a lucid interval, although this does not always occur. Thus, the athlete initially may remain conscious or regain consciousness after the head trauma and then experience an increasing headache and progressive decline in level of consciousness. This occurs as the clot accumulates and the ICP increases. This lesion, if present, almost always will declare itself within 1-2 hours from the time of injury.[20,28] Usually, the brain substance is free from direct injury; thus, if the clot is evacuated promptly, full recovery may be expected.

Figure 1. Epidural Hematoma



Subdural Hematoma. SDHs are located between the dura mater and the arachnoid and usually are crescentic-shaped and associated with a high mortality. Athletes who suffer a severe impact at the time of injury generate brain acceleration within the skull, with over-stretching and rupture of the parasagittal bridging veins. Unlike EDH, with this injury there often is associated injury to the brain tissue.

An acute SDH generally becomes apparent immediately following impact to the head.[20] After contact to the head, or in some instances a blow to another part of the body that shakes the brain sufficiently, the athlete becomes disoriented, nauseated, and loses consciousness. In severe cases, the athlete develops posturing and may have pupillary changes. These patients need immediate treatment, hospitalization, and, often, surgical intervention.

The majority of SDHs are supratentorial in location and most commonly are found in the frontoparietal region. (*See Figure 2.*) The high degree of mortality associated with an acute SDH, even those treated surgically, arises from the significant amount of associated underlying brain injury.[15,28,41] This underscores the need to rapidly transport severely head-injured athletes to hospitals with neurosurgical capabilities to diagnose and surgically evacuate those with significant mass effect. This form of intracranial hemorrhage usually renders an athlete unconscious from the time of impact, and emergent resuscitation may be necessary.[28] Improved outcome has been achieved by resuscitation, rapid transport, and early surgery (within four hours of injury).[18]

Intraparenchymal Hemorrhage. This usually is a well-defined hematoma within the brain parenchyma that occurs post-trau-

Figure 2. Subdural Hemorrhage

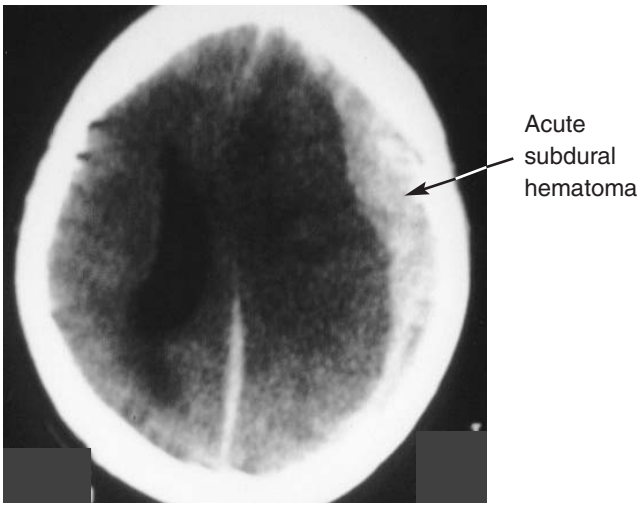
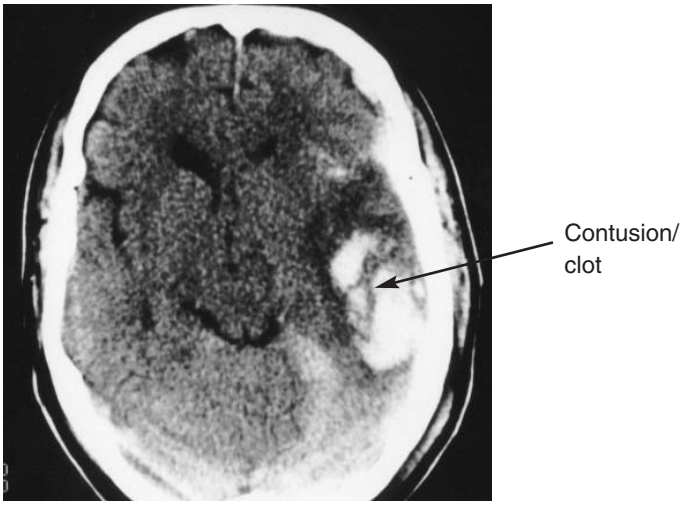


Figure 3. Intraparenchymal Hemorrhage



matically as a result of a disruption of a cerebral blood vessel. Progression in size or severity of the hemorrhage occurs in up to 30% of patients within 24 hours.[28]

Differentiation from a hemorrhagic contusion can be difficult, owing to the fact that contusions can evolve and coalesce into a collection of blood. The mechanism of injury is acceleration of the brain within the calvarium occurring at the time of impact, resulting in disruption of one or more parenchymal vessels. *(See Figure 3.)*

Subarachnoid Hematoma. Extravasation of blood into the subarachnoid space may appear as a high density collection within the cerebrospinal fluid spaces, including sulci, cisterns, and fissures. The irritative properties of the blood may precipitate a seizure. CT demonstration of post-traumatic SAH is limited to a few days as a result of erythrocyte lysis and rapid absorption. SAH in excess of the degree of trauma may signify an underlying brain anomaly (i.e., aneurysm or arteriovenous malformation).[28,41] If clinical suspicion exists, further investigation may be indicated.

Cerebral Contusion. A cerebral contusion, or bruising of the brain, usually is cortical in location.[49] It represents a combination of necrosis, hemorrhage, infarction and edema of the brain involved. Cortical contusions usually are direct coup or contrecoup injuries that occur when the brain impacts against the bony prominences of the skull. Contusions commonly involve the inferior aspect of the frontal lobes and the anterior temporal lobes, where they lie buttressed against the greater wing of the sphenoid bone. Signs and symptoms of contusion are unconsciousness, disturbance in strength or sensation, changes in visual awareness, or focal neurologic signs such as seizures.

Emergency Sports Head Injury Assessment

After an athlete is injured, the examiner must take control of the situation, both on the field during the immediate post-injury period and on the sideline subsequent to transport off the field. A systematic head-to-toe evaluation should be completed. Proper cervical spine precautions should be exercised, and a cervical spine injury must be assumed until proven otherwise, especially in anyone who has had an LOC or has a post-traumatic altered mental status.[18] In dealing with a helmeted unconscious athlete, the helmet should be left in place until adequate assistance is available and insurance that a medical team familiar with a technique for removal of helmets is in place. Those providing medical coverage at athletic events can develop and practice the safe removal of helmets in injured athletes with available protocols. *(See Table 3.)* Sports medicine experts advise against removal of the helmet in the field, to prevent neurological injury. Removal of the helmet is a high-risk maneuver, and when performed by an inadequately trained medical staff can lead to further neurological injury.[50,51] One study estimated that up to 25% of spinal cord injuries occur after the initial traumatic event, during transfer and early management.[52] If cardiopulmonary resuscitation is necessary, removal of the face mask will allow access to the airway.

Common situations that increase morbidity and mortality in the injured athlete include airway obstruction, respiratory failure, cardiac arrest or arrhythmia, severe head injury, cervical spine injury, hyperthermia, and coagulopathy.

Proper assessment and management of head-injured athletes require adherence to guidelines presented in the Advance Trauma Life Support (ATLS) protocols prepared by the American College of Surgeons Committee on Trauma. As recommended, these guidelines include the primary survey, resuscitation, a secondary survey, and re-evaluation and definitive care.

The key to assessment of vital signs is to constantly be vigilant for change from baseline values. Be aware of trends in parameter changes (i.e., How has it changed? What does the change imply?).

The respondent's initial obligation is to determine whether the injured player is breathing spontaneously, has an unobstructed airway, and has a pulse. Second, medical personnel should quickly determine whether further evaluation on the sidelines is appropriate or whether emergent transport to a hospital is need-

ed. If the athlete has an adequate airway, respiration, and pulse, the initial evaluation of the level of consciousness should be performed in the position in which the athlete lies.

In the absence of a pulse and adequate respiration, the neck should be stabilized by an experienced person. With the assistance of two or three trained personnel, the athlete may be log-rolled into a supine position so that cardiopulmonary resuscitation can be initiated effectively.

Athletes with head injuries frequently have a blank expression, may appear confused, exhibit delayed verbal responses, and seem emotionally labile.[32] The standard method of assessing the level of consciousness is by establishing a Glasgow Coma Score (GCS). By observing the patient's eyes and motor and verbal responses, one can quantify the level of consciousness. A GCS of 13 or greater is classified as a mild head injury and usually has an excellent prognosis for recovery. By far, the most common sports-related head injury is the mild variety known as concussion.[15] Some authors have objected to the term "mild head injury," stating that no injury to the brain should be considered minor.[2,41,53] On the other hand, a GCS of 8 or less is considered severe.

Management of Concussion

The guidelines for the management of concussion in sports adapted by the American Academy of Neurology (AAN) specifically called for the development of a standardized, systematic sideline evaluation for the immediate assessment of concussion in athletes.[37,54] The AAN guidelines, in line with the concussion literature, stress that a sideline examination should test orientation and probe for deficits in attention, concentration, and memory.[54] *(See Table 4.)*

The player also should be questioned about the symptoms of dizziness, light-headedness, vertigo, blurring or double vision, photophobia, ringing in the ears, headache, nausea, and vomiting. Many of these symptoms may be present initially after an acute head injury, while headache, nausea, and vomiting may not become evident for several minutes after the precipitating trauma. Vomiting is not very common after athletic injuries, but when it is present, it suggests significant injury with elevated ICP.

A player initially should be observed for a minimum of 15 minutes on the sidelines and reevaluated as needed. If any symptoms develop, the athlete should not return to competition that day. If the player has not lost consciousness, is oriented, and is asymptomatic, provocative testing should be performed to determine whether symptoms will occur with physical stress. *(See Table 4.)*

The following recommendations for management of concussion are based on the AAN guidelines.[37,53] Recommendations by Cantu and the Colorado Medical Society have been discussed and are presented in Table 2.

Grade 1 Concussion. Grade 1 concussions are defined by symptoms of transient confusion without amnesia, no LOC, and concussion symptoms and/or mental status abnormalities that resolve in fewer than 15 minutes. This is both the most common and most difficult form of concussion to recognize, as the individual is not rendered unconscious and experiences only momentary confusion. The majority of concussions sustained in sports and recreational activities are this type. Although this type of concussion is often downplayed and referred to in terms of "being dinged" or "having their bell rung," an athlete who sustains a Grade 1 concussion should be removed from the game and reevaluated before reentering the playing area.

A second Grade 1 concussion on the same day will result in the player being removed from the contest for the remainder of the day and undergoing evaluation by a physician. It also is recommended that a player who has sustained three Grade 1 concussions not be allowed to return to play until he or she is symptom-free for at least one week. In addition, no further contact sports are permitted for at least three months, and then only if the individual is asymptomatic at rest and with exertion. CT or MRI scanning is recommended in all instances in which headache or other associated symptom either worsens or persists longer than one week after the concussion.

Grade 2 Concussion. Grade 2 concussion is defined by the presence of transient confusion with amnesia and concussion symptoms and/or mental status abnormalities lasting more than 15 minutes, although there is no LOC. With this type of concussion, the individual exhibits confusion and may have post-traumatic amnesia of the events following the impact. In more severe cases, the individual may experience retrograde amnesia of events preceding the injury. After an individual has sustained a Grade 2 concussion, he or she should be removed from play and not allowed to return to the playing area.

After the first Grade 2 concussion, the individual should refrain from playing any contact sport for at least one week, again only after the player is asymptomatic at rest and during exertion. Additionally, a physician should perform a neurological exam before the individual is allowed to return to play. If the

Table 3. Emergency Removal of a Football Helmet

- After arrival at a medical facility, protective equipment should be removed before x-ray and must follow strict protocol.
- A minimum of two people, preferably three, are needed to assist.
- Person A stabilizes the head and neck while person B removes the chin strap and cheek pads, and if applicable, deflates the helmet's inner surface air cells.
- Person B then stabilizes the head and neck while person A spreads the helmet open and gently slides the helmet off the head. Person B carefully supports the head to prevent it from dropping posteriorly.
- Person A then resumes control of the head and neck, the head being maintained at the same level of the torso.

Adapted from: Gastel JA, Palumbo MA, Hulstyn MJ, et al. Emergency removal of football equipment: A cadaveric cervical spine injury model. *Ann Emerg Med* 1998;32:411-417.

Table 4. Sideline Evaluation

MENTAL STATUS TESTING

Orientation:	The individual is questioned about the time, place, and people involved, and the situation (circumstances of the injury).
Concentration:	The individual can recite numbers (i.e., a telephone number) backward. Individual can recite the months of the year in reverse order.
Memory:	The individual can recall, for example, teams played in prior contests; the name of the President or other public figures; and recent news-worthy events. The athlete can recite three words and three objects, and then remember and repeat the same three words and three objects five minutes later. The individual can remember details of the contest (i.e., plays, moves, strategies), if applicable, and can recall all events since the injury (i.e., how he/she got off the field).

EXERTIONAL PROVOCATIVE TESTS

Individual can perform

- a 40-yard sprint
- five push-ups
- five sit-ups
- five knee bends

(Any appearance of associated symptoms is abnormal; e.g., headache, dizziness, nausea, photophobia, blurred or double vision, emotional lability, and/or mental status change.)

Coordination:	Athlete can perform finger-nose-finger exercise and tandem walking.
Sensation:	Individual performs finger-nose-finger exercise with eyes closed (Romberg test).
Strength:	Individual is fully strong in all muscle groups.

individual experiences worsening headaches and other associated symptoms, and/or these symptoms last longer than a week, a CT or MRI is recommended. Return to contact play after a second Grade 2 concussion should be deferred for at least two weeks or, in most cases, a month. After three or more Grade 2 concussions, the individual seriously should consider terminating play for the remainder of the season, and perhaps beyond when any abnormality (i.e., brain swelling or contusions) appears on the CT or MRI scan.

Grade 3 Concussion. It usually is quite easy to recognize a Grade 3 concussion, and this level of concussion is applied to anyone who experiences LOC no matter how brief. Initial treatment for Grade 3 concussion—the most severe and serious of the grades—calls for the individual to be transported to the nearest hospital for neurological evaluation and observation.

One month should be the minimum period the individual is barred from contact sports after sustaining a Grade 3 concussion. With multiple Grade 3 concussions, the individual must remain out of play for the remainder of the season and should be strongly discouraged from returning to playing any contact sports.

Management of Moderate and Severe Head Injury

The priority in managing the patient who has moderate (GCS 12-9) or severe (GCS < 8) head injury is to minimize secondary brain injury, with evaluation and treatment occurring simultaneously. Maintenance of adequate hemodynamics and oxygenation are of utmost importance. If hypoxic or ischemic injury occurs in addition to the traumatic injury, the prognosis worsens.[47,55] The ABCs (airway, breathing, circulation) of emergency medical care apply as in every life-threatening situation. Endotracheal intubation and mechanical ventilation should be performed in patients who have a GCS of 8 or less, hypoventilation, apnea, cardiorespiratory arrest, or other significant injury.[55,56] Hypoxia and hypercarbia should be avoided through the use of supplemental oxygen and controlled ventilation, because even mild-to-moderate hypoxia or hypercarbia can affect cerebral blood flow and result in further brain injury. Patients who exhibit signs of impaired cardiac output, such as tachycardia or borderline or low blood pressures, should receive a bolus of isotonic fluid intravenously to ensure adequate circulating volume to maintain blood flow to the compromised brain. The neurologic status of the patient must be followed during the period of resuscitation using the GCS to evaluate the response to therapy. The use of hyperventilation (PCO_2 of 30-35 torr) and mannitol (0.25-0.50 mg/kg) is appropriate for patients in whom herniation is impending until the neurologic evaluation is completed and the patient is stabilized. Seizures are common after traumatic and hypoxic brain injury and should be treated with benzodiazepines, phenytoin, and phenobarbital as needed.

After initial evaluation and stabilization, all athletes who have moderate or severe head injury require CT of the head. Significant SDH or EDH requires urgent or emergent evacuation to prevent secondary injury. Severe cerebral edema identified by CT may require placement of an ICP monitor, which necessitates consultation with a pediatric neurosurgeon. In the patient whose mental status is altered, there is a significant possibility of a spinal cord injury that requires appropriate immobilization. Spinal cord immobilization must be continued until proper evaluation is completed.

Conclusion

Head injuries in sports cause acute—and in some instances long-term—serious problems. The symptoms of mild head injury may be transient, but the cumulative effect may have permanent sequelae. Appropriate acute and subacute management can prevent secondary potentially disabling or fatal injury.

It is essential that all individuals who participate in sports and recreational activities take into consideration the risk of traumatic brain injury and make every effort to prevent such potentially catastrophic injury. Physicians, athletic trainers and coaches must work together as partners and become attuned to the problems and recognize even the mildest forms of concussion in ath-

letes. Most importantly, athletes need to be educated and aware of the recognition and management of concussions so they can participate in their own care from an informed perspective.

References

1. Landry GL. Central nervous system trauma: Management of concussions in athletes. *Pediatr Clin N Am* 2002;49:723-741.
2. Poirier MP, Wadsworth MR. Sports-related concussions. *Pediatr Emerg Care* 2000;16:278-283.
3. Perriello VA, Barth JT. Sports concussions. *Contemp Pediatr* 2000;17:132-140.
4. Cantu RC. Second-impact syndrome. *Clin Sports Med* 1998;17:37-44.
5. Loiselle JM, Poirier MP, Schutzman S. Closed head injury in children. *Pediatr Emerg Care* 2002;18:48-52.
6. Moynihan JA, Perkin RM. Current concepts in the management of minor closed head injury in children. *Trauma Reports* 2001;2:1-12.
7. American Academy of Pediatrics, Section on Sports Medicine and Fitness. Guidelines for Pediatricians: Head Injuries. *Sports Shorts* 2000;1:1.
8. Macciocchi SN, Barth JT, Littlefield LM. Outcome after mild head injury. *Clin Sports Med* 1998;17:27-36.
9. Bernhardt DT. Football: A case-based approach to mild traumatic brain injury. *Pediatr Ann* 2000;29:172-176.
10. American Academy of Pediatrics. The management of minor closed head injury in children. *Pediatrics* 1999;104:1407-1415.
11. McCrory PR. Were you knocked out? A team physician's approach to initial concussion management. *Med Sci Sports* 1997;29:207-212.
12. American Academy of Pediatrics, Committee on Sports Medicine and Fitness. Medical conditions affecting sports participation. *Pediatrics* 2001;107:1205-1209.
13. Barth J, Alves W, Ryan T, Macciocch S, et al. Mild Head Injury in Sports: Neuropsychological Sequelae and Recovery of Function. In: Levin H, Eisenberg H, Benton A, eds. *Mild Head Injury.* New York: Oxford Press; 1989: 257-275.
14. Buckley W. Concussion in college football: A multivariate analysis. *Am J Sports Med* 1988;16:51-56.
15. Warren WL, Bailes JC. On the field evaluation of athlete head injury. *Clin Sports Med* 1998;17:13-26.
16. Gerberich SG, Priest JD, Boen JR, et al. Concussion incidences and severity in secondary school varsity football players. *Am J Public Health* 1983;73: 1370-1375.
17. Bailes JE, Lovell MR, Maroon JC. *Sports Related Concussion.* St. Louis: Quality Medical Publishing, Inc.; 1999:1.
18. Chorley JN. Sports-related head injuries. *Curr Opin Pediatr* 1998;10:350-355.
19. Mueller FO. Fatalities from head and cervical spine injuries occurring in tackle football: 50 years' experience. *Clin Sports Med* 1998;17:169-182.
20. Lackstead EF, Patel DR. Catastrophic pediatric sports injuries. *Pediatr Clin N Am* 2002;49:581-591.
21. Davis PM, McKelvey MK. Medicolegal aspects of athletic head injury. *Clin Sports Med* 1998;17:71-82.
22. Collins MW, Lovell MR, McKeay DB. Current issues in managing sports-related concussion. *JAMA* 1999;282:2283-2285.
23. Genuardi FJ, King WD. Inappropriate discharge instructions for youth athletes hospitalized for concussion. *Pediatrics* 1995;95:216-218.
24. Collins MW, Grindel SH, Lovell MR, et al. Relationship between concussion and neuropsychological performance in college football players. *JAMA* 1999; 282:964-970.
25. Patel DR, Pratt HD, Greydanus DE. Pediatric neurodevelopment and sports participation. When are children ready to play sports? *Pediatr Clin N Am* 2002;49:505-531.
26. Powell JW, Barber-Foss KD. Traumatic brain injury in high school athletes. *JAMA* 1999;282:958-963.
27. Ponsford J, Willmott C, Rothwell A, et al. Impact of early intervention on outcome after mild traumatic brain injury in children. *Pediatrics* 2001;108: 1297-1303.
28. Proctor MR, Cantu RC. Head and neck injuries in young athletes. *Clin Sports Med* 2000;19:693-715.
29. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. *Neurology* 1997;48:575-580.
30. Kay T, Harrington DE, Adams R, et al. Definition of mild traumatic head injury. *J Head Trauma Rehabil* 1993;8:86-87.
31. Johnston KM, McCrory P, Mohtadi NG, et al. Evidence-based review of sport-related concussion: Clinical science. *Clin J Sport Med* 2001;11:150-159.
32. Wojtys EM, Horda D, Landry G, et al. Concussion in sports. *Am J Sports Med* 1999;27:656-687.
33. Zeman A. Consciousness. *Brain* 2001;124:1263-1289.
34. Roos R. Guidelines for managing concussion in sports. *Phys Sports Med* 1996;24:67-74.
35. Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Phys Sports Med* 1986;14:75-83.
36. Colorado Medical Society. *Report of the Sports Medicine Committee: Guidelines for the Management of Concussions in Sports* (revised). Denver:Colorado Medical Society;1991:1.
37. American Academy of Neurology. Practice parameter: The management of concussion in sports (summary statement). *Neurology* 1997;48:581-585.
38. Gronwall D, Wrightson P. Duration of post-traumatic amnesia after mild head injury. *J Clin Neuropsychology* 1985;2:51-60.
39. Gronwall D, Wrightson P. Memory and information processing capacity after a closed head injury. *J Neurol Neurosurg Psychiatry* 1981;44:889-895.
40. Russell WR, Nathan PW. Traumatic amnesia. *Brain* 1946;69:280-300**.**
41. Cantu RC. Return to play guidelines after a head injury. *Clin Sports Med* 1998;17:45-60.
42. Clarke KS. Epidemiology of athletic head injury. *Clin Sports Med* 1998;17:1-11.
43. McCrory PR, Berkoric SF. Secondary impact syndrome. *Neurology* 1998;50: 677-683.

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

44. Saunders RL, Harbaugh RE. The second impact in catastrophic contact sports head trauma. *JAMA* 1984;252:538-539.
45. Schneider RC. *Head and Neck Injuries in Football: Mechanisms, Treatment and Prevention.* Baltimore:Williams and Wilkins;1973.
46. Cantu FL, Voy R. Second impact syndrome: A risk in any contact sport. *Phys Sports Med* 1995;23:27-34.
47. Gedeit R. Head injury. *Pediatr Rev* 2001;22:118-123.
48. Atkinson JLD. The neglected pre-hospital phase of head injury: Apnea and catecholamine surge. *Mayo Clin Proc* 2000;75:37-47.
49. Zuckerman GB, Conway EE. Accidental head injury. *Pediatr Am* 1997;26: 621-632.
50. Swenson T, Lauerman N, Blanc R. Cervical spine alignment in the immobilized football player: Radiographic analysis before and after helmet removal. *Am J Sports Med* 1997;25:226-230.
51. Warren WL, Bailes JE. On the field evaluation of athletic neck injury. *Clin Sports Med* 1998;17:99-110.
52. Podolsky S, Baratt LJ, Simon PR, et al. Efficacy of cervical spine immobilization methods. *J Trauma* 1983;23:461-465.
53. Stein SC. Minor head injury: 13 is an unlucky number. *J Trauma* 2001;50: 759-760.
54. McCrea M, Kelly JP, Kluge J, et al. Standardized assessment of concussion in football players. *Neurology* 1997;48:586-588.
55. Perkin RM, Moyniham JA, McLeary M. Current concepts in the emergency management of severe traumatic brain injury in children. *Trauma Reports* 2000;1:1-16.
56. Kaide CG, Hollingsworth JC. Current strategies for airway management in the trauma patient. *Trauma Reports* 2003;4:1-12.

Physician CME Questions

61. Most acute head injuries are caused by a combination of rotational and translational acceleration.
    A. True
    B. False

62. What is the most common type of head injury that occurs in athletes?
    A. EDH
    B. Concussion
    C. SDH
    D. Intracerebral hematoma
    E. SAH

63. Which of the following is a common feature of a concussion?
    A. It is caused by either a direct blow to the head or elsewhere on the body with an impulsive force transmitted to the head.
    B. It results in an immediate and short-lived impairment of neurologic function.
    C. It may result in neuropathologic changes.
    D. It may result in a graded set of clinical syndromes that may or may not involve LOC.
    E. All of the above

64. Post-traumatic amnesia is defined as the period following the head injury in which the athlete is unable to remember anything.
    A. True
    B. False

65. Which of the following symptoms following a concussion may be indicative of a post-concussion syndrome?
    A. Impaired memory
    B. Impaired concentration
    C. Impaired attention
    D. All of the above

66. SIS refers to a condition in which fatal brain swelling occurs after a minor head injury in a patient who was still symptomatic from a previous concussion.
    A. True
    B. False

67. A CT scan obtained on an athlete following a head injury shows a crescent-shaped bleed with underlying brain injury. This most likely represents which of the following?
    A. EDH
    B. SDH
    C. SAH
    D. DAI

68. In a patient with normal neurologic status, what type of injury results in a full recovery in the majority of patients if the clot is evacuated immediately?
    A. EDH
    B. SDH
    C. SAH
    D. DAI

69. Which of the following is true regarding an SAH?
    A. It is usually cortical in nature.
    B. It represents a combination of necrosis, hemorrhage, infarction, and edema.
    C. These lesions may be coup or contrecoup.
    D. All of the above

70. A GCS of 12 is classified as a minor head injury.
    A. True
    B. False

Answer Key

61. A	66. A
62. B	67. B
63. E	68. A
64. A	69. D
65. D	70. B

In Future Issues: **Status Epilepticus**

PEDIATRIC Emergency Medicine Reports
The Practical Journal of Pediatric Emergency Medicine

Sports-Related Head Injuries

Diagnostic Grading Scales for Sports-Related Concussion

Guideline	Severity of Grade 1	2	3
Cantu	1) No loss of consciousness 2) Post-traumatic amnesia lasts fewer than 30 min	1) Loss of consciousness lasts fewer than 5 min OR 2) Post-traumatic amnesia lasts longer than 30 min	1) Loss of consciousness lasts longer than 5 min OR 2) Post-traumatic amnesia lasts longer than 24 hr
Colorado	1) Confusion without amnesia 2) No loss of consciousness	1) Confusion with amnesia 2) No loss of consciousness	1) Loss of consciousness (of any duration)
Practice Parameter, American Academy of Neurology	1) Transient confusion 2) No loss of consciousness 3) Concussion symptoms or mental status change resolve in fewer than 15 min	1) Transient confusion 2) No loss of consciousness 3) Concussion symptoms or mental status change last longer than 15 min	1) Loss of consciousness (brief or prolonged)

Examples of Differing Concussion Classification Systems and Return-to- Play Recommendations

CANTU GUIDELINES

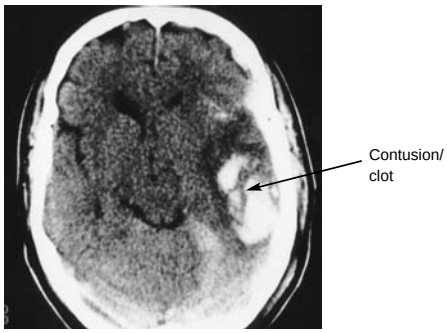
Severity	First Concussion	Second Concussion	Third Concussion
Grade 1 (mild): No loss of consciousness; post-traumatic amnesia < 30 min	May return to play if asymptomatic	May return in 2 weeks if asymptomatic at that time for 1 week	Terminate season; may return next year if asymptomatic
Grade 2 (moderate): Loss of consciousness < 5 min or post-traumatic amnesia > 30 min	Return after asymptomatic for 1 week	Wait at least 1 month; may return if asymptomatic for 1 week; consider terminating season	Terminate season; may return next year if asymptomatic
Grade 3 (severe): Loss of consciousness > 5 min or post-traumatic amnesia > 24 hr	Wait at least 1 month; may return then if asymptomatic for one week	Terminate season; may return next year if asymptomatic	

COLORADO MEDICAL SOCIETY GUIDELINES

Severity	First Concussion	Second Concussion	Third Concussion
Grade 1 (mild): Confusion without amnesia; no loss of consciousness	May return to play if asymptomatic for at least 20 min	Terminate contest or practice for the day	Terminate season; may return in 3 months if asymptomatic
Grade 2 (moderate): Confusion with amnesia; no loss of consciousness	Terminate contest/practice; may return if asymptomatic for at least 1 week	Consider termination of season, but may return if asymptomatic for 1 month	Terminate season; may return to play next season if asymptomatic
Grade 3 (severe): Loss of consciousness	May return after 1 month if asymptomatic for 2 weeks at that time; may resume conditioning sooner if asymptomatic for 2 weeks	Terminate season; discourage any return to contact sports	

Cantu guidelines adapted from: Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Phys Sports Med* 1986;14:75-83.
Colorado Medical Society guidelines adapted from: Colorado Medical Society. *Report of the Sports Medicine Committee: Guidelines for the Management of Concussions in Sports* (revised). Denver:Colorado Medical Society;1991.

Intraparenchymal Hemorrhage



Emergency Removal of a Football Helmet

- After arrival at a medical facility, protective equipment should be removed before x-ray and must follow strict protocol.
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- Person B then stabilizes the head and neck while person A spreads the helmet open and gently slides the helmet off the head. Person B carefully supports the head to prevent it from dropping posteriorly.
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Sideline Evaluation

MENTAL STATUS TESTING

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- five knee bends

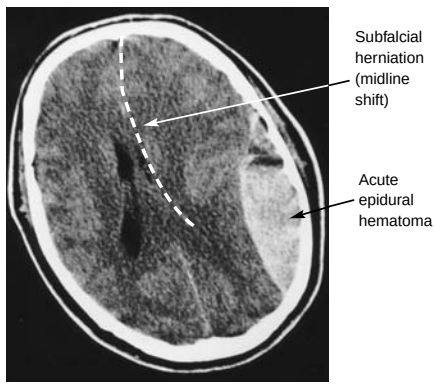
(Any appearance of associated symptoms is abnormal; e.g., headache, dizziness, nausea, photophobia, blurred or double vision, emotional lability, and/or mental status change.)

Coordination: Athlete can perform finger-nose-finger exercise and tandem walking.

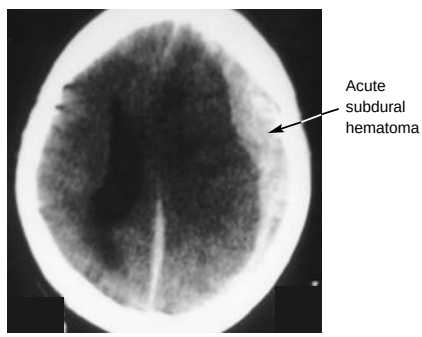
Sensation: Individual performs finger-nose-finger exercise with eyes closed (Romberg test).

Strength: Individual is fully strong in all muscle groups.

Epidural Hematoma



Subdural Hemorrhage



Supplement to *Pediatric Emergency Medicine Reports*, July 2003: "Sports-Related Head Injuries: Learn the Rules of this Serious Game." *Authors:* **Don W. Penney, MD, MSc, FACEP,** Neurological Surgeon; Professor, Department of Emergency Medicine, Medical College of Georgia, Augusta; and **Ronald M. Perkin, MD, MA, FAAP, FCCM,** Professor and Chairman, Department of Pediatrics, The Brody School of Medicine at East Carolina University, Greenville, NC.

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Pediatric Cervical Spine Injuries: Avoiding Potential Disaster

Author: **William A. Woods, MD, FAAP,** Assistant Professor of Emergency Medicine and Pediatrics, University of Virginia Health System, Charlottesville.

Peer Reviewer: **Larry B. Mellick, MS, MD, FAAP, FACEP,** Chairman and Professor, Section Chief, Pediatric Emergency Medicine, Department of Emergency Medicine, Medical College of Georgia, Augusta.

Although cervical spine injuries (CSIs) are uncommon in children, a missed or delayed diagnosis may have devastating consequences for the patient. A thorough understanding of normal pediatric anatomy, injury patterns, and children who are at increased risk for injury is critical for the physician caring for the acutely injured child. The author provides an overview of the unique features of the pediatric spine, and fracture patterns that occur commonly in children. The author also offers guidelines on instances when a child is at increased risk for sustaining a CSI.

—The Editor

Introduction

The diagnoses of CSIs in children deserves special attention and distinction from injuries in adults. The anatomy, biomechanics, and injury pattern of the infant are different than those of the school-age child and the adolescent. By adolescence, the cervical spine has assumed the same mechanical response as the adult spinal column. This review will describe the anatomy of the cervical spine from infancy through adolescence and discuss the varying injury patterns that may occur through childhood. A general sense of the biomechanical tolerance of the pediatric spine will be developed through a review of injury-producing impacts. Additionally, limitations of data will be acknowledged that will necessitate practical management recommendations.

Anatomy of the Cervical Spine

The cervical spine is composed of seven vertebrae, each separated by an intervertebral disc. The cranium rests upon the atlas (C_1) while C_7 rests upon the first thoracic vertebra (T_1). See Figure 1 for the landmarks of the lower cervical vertebrae.

Tethering ligaments and their associated attachments are listed in Table 1. The transverse ligament is unique to C_1 and maintains the relationship between C_1 and C_2. This ligament attaches to the posterior dens (odontoid process of C_2) from the inner, lateral aspect of C_1. The other tethering ligaments, as well as the facets and interfacet joints, limit horizontal motion between the vertebral bodies. Additionally, the tethering ligaments limit axial, or vertical, elongation of the spinal column. When the mechanical properties of these structures are overcome by the forced neck motion of impact, an injury will result.

Developmental Biology of the Cervical Spine

The ossification centers of the vertebral bodies appear during the second month of gestation as sclerodermal mesoderm

migrates and thickens into the vertebral column. The vertebral bodies are formed by two lateral neural arches and a centra. The centra and the anterior portion of the lateral arches fuse to form the vertebral body. The posterior portions of the neural arches fuse to form the transverse processes, facets, and spinous process. The anterior and posterior arches are radiographically evident at birth, while the spinous, articular, and transverse processes do not fuse until approximately 8 years of age.

Ossification of the dens is not complete until 6-8 years of age. The dens is formed by union of two lateral globular masses, achieving a conical shape at an age of viability.[1] This ossified dens retains a cartilaginous association with the body of C_2 until fusion occurs at age 6-8 years. The tip of the dens is formed by a separate ossification center. The superior portion of the dens is calcified by age 4 years.

In the newborn cervical spine, the facet joints are flatter than those in the mature teenage spine, therefore, anterior-posterior relative motion is not limited in the newborn's cervical spine as well as it is in the mature spine. One study found that the facet joint angle does not assume an adult angle until age 10 years.[2] Additionally, potential voluntary anterior-posterior motion of one vertebral body upon another is increased until age 12 years.

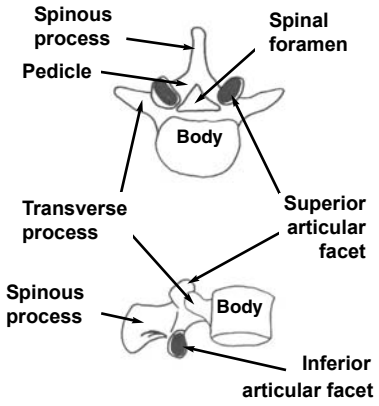
Although no biomechanical data exist to quantify the difference, the musculature supporting the pediatric cervical spine is assumed to be laxer than in adults. In addition to a weaker neck, infants have a relatively larger head, per body weight, than adults do. In summary, in young children, a weaker neck that has less restriction to mechanical motion must support a heavier head. Being at an anatomic disadvantage, the cervical spine in infants and children may be at risk for injury at lower impact energies than would cause injury in adults.

Cervical Spine Malformations and Anomalies. As described above, embryologic formation of the vertebral column starts during the third to fourth week of gestation. Additionally, the major structures of the face and neck develop between weeks 4 and 12.[3] Children who have abnormal embryologic development of facial or neck features are at risk for abnormal cervical spine development as embryologic development of these two structures occurs simultaneously.[4] See Table 2 for a list of associated abnormalities. While the emergency physician often will not be able to identify the specific malformation syndrome, patients with face and neck anomalies should be considered at risk of having a congenitally abnormal cervical spine.

When examining cervical spine radiographs, it is necessary to look for any signs of congenital abnormality. Fusion of adjacent posterior elements commonly is associated with fusion of the vertebral bodies. Identification of the posterior fusion will appear earlier, as the complete ossification of the vertebral bodies is delayed in children. Fusions are most common above C_4, yet can occur throughout the cervical spine. Other common abnormalities include occiptoatlantal fusion, hypoplastic or anomalous portions of the atlas, ligamentous laxity, and malformations of the dens.[3,5]

Studies of U.S. football players have demonstrated that congenital cervical stenosis commonly is detected in athletes with transient neuropraxias. However, this anomaly does not predispose athletes to catastrophic neurological injury.[6]

Figure 1. Anatomy of the Cervical Vertebra



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Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
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The intended audience for this publication is emergency, family, osteopathic, and general practice physicians and nurses who have contact with trauma patients.

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Table 1. Tethering Ligaments in the Cervical Spine

Vertebral bodies and discs	Anterior and posterior longitudinal ligaments
Facets	Interfacet capsular ligaments
Spinous processes	Interspinous and supraspinous ligaments
Transverse processes	Intertransverse ligaments
Adjacent lamina	Ligamentum flavum

Even though it would seem logical, neuromuscular disorders typically do not affect the cervical spine. Instead, neuromuscular disorders tend to result in thoracolumbar scoliosis.[7] Examples of these disorders include cerebral palsy, muscular dystrophy, spinal muscular atrophy, and Rett's syndrome.

Cervical Spine Injury Distribution (Age/Location)

CSIs are less common in pediatric trauma patients than in adult patients. The NEXUS group found 30 injuries in 3065 (1%) pediatric patients, compared to injuries in 2.5% of adult patients (788/31,004).[8] Approximately two-thirds of pediatric CSI occur in patients older than 8 years of age.[9] These data reflect patients who arrived to emergency departments, and do not include those who died prior to transport. In a series of 102 patients, 42% of those younger than 10 years of age arrived neurologically intact, 42% had an incomplete spinal cord injury and 16% had a complete lesion.[10] Of those between 10 and 16 years, 41% (26 of 64 patients) arrived neurologically intact, 47% had partial spinal cord lesions, and 12% had complete spinal cord injury on arrival.

Types of injuries to the pediatric cervical spine include fractures, dislocations, or SCIWORA (Spinal Cord Injury Without Radiographic Abnormality). In one review of the National Pediatric Trauma Registry, 25-30% of all CSI were SCIWORA in children younger than 11 years.[11] In patients between 11 and 18 years, 15-20% of all CSI were SCIWORA. Fractures accounted for 35-40% of CSI in children younger than 7 years, 45-50% between 7 and 11 years, 60-70% from 12 to 16 years, and 70-75% in those older than 16 years. Dislocations caused 30-40% of the injuries in those younger than 7 years, 20-30% in those 7-11 years, and 15-25% in those 12-16 years. In summary, fractures are the most common injury seen and increase in frequency through childhood. Dislocations also are more common in younger children, but the difference between young and old is not as dramatic. SCIWORA is common in younger children, but may occur in any age group.

Younger children with CSI most commonly are injured in the upper cervical spine. *(See Figure 2.)* By the end of the teenage years, injuries are distributed more evenly between the upper and lower cervical spine. In children younger than 11 years of age, 15-20% of all CSI are below C_4. In the ages 11-15, 35-40% of CSI are below C_4. Upper and lower CSI are equally as likely in the child older than 15 years.[11] Series published through individual institutions have presented differing distribution of injuries, but one paper[11] included 408 children, the largest data set of pediatric CSI.[12-22]

Some data exist regarding the prevalence of CSI as an isolated injury. In a group of 72 children with CSI from Utah, one study notes that the median Glasgow Coma Score (GCS) was 15.[22] Fourteen percent of the children had a GCS less than 13. The mean injury severity score was 15, with a range of 4-54.[22] In another series, 37 children younger than 9 years had a mean injury severity score of 26, while the 36 children older than 8 years had a mean injury severity score of 12.1.[18] These data suggest that children may sustain an isolated CSI .

Combining the data presented in two studies allows some description of outcomes in children with CSI.[12,19] Eleven of 18 children younger than 5 years with CSI died. Fourteen of 19 children between the ages of 5 and 10 died. Of those older than 10 years, four of 42 children died. In one of the two studies, all children survived if they had an isolated cervical spine fracture. One child out of nine with a fracture/subluxation died. All 15 children with distraction injuries died (eight of which were occipito-cervical injuries).

Injury Etiology

CSI in young children tends to result from motor vehicle collisions (MVCs), falls, pedestrian injuries, or child abuse. Older children are more active in sports and activities and may endure a CSI due to MVCs, sports, falls or bicycle accidents.[9-12,16-18,20-22]

To further clarify the force of impact necessary to cause CSIs in children, it is tempting to read injury reports regarding specific mechanisms of trauma. Many authors have published series of papers dealing with the injuries seen after specific types of trauma, including falls,[24-27] crashes,[28-31] and animal attacks.[32] These data series tend to reinforce to the clinician that a specific impact may be an injury-producing impact. Potentially more useful are series that emphasize mechanisms that did not result in a CSI from a specific type of trauma. CSI did not occur in any child in a series of 432 falls down stairs reported in two series.[33,34] One child died after a fall down stairs in a walker after suffering a CSI, skull fracture, and subdural hematoma.[35,36]

Similarly, a CSI did not occur in any child after falling from bed (207 children),[37] high chairs (103 children),[38] or shopping carts (62 children).[39] One author reported 151 falls from heights, none resulted in CSI.[40] Another group reported a series of 101 children admitted to the hospital with a skull fracture. None of these children had a concurrent CSI.[41]

One author published a series of eight children who sustained CSI after short falls.[42] All were symptomatic at the time of presentation (although the time from injury to symptom onset is not reported). In this series, a 4-year-old had a C_1-C_2 subluxation after falling out of bed. A 9-month-old fell while pulling herself up and suffered a subluxation of C_1-C_2 with an odontoid fracture. A 3½-year-old fell while running and suffered a fracture of the neural arch of C_2 with subluxation of the inferior articulating facet. The other children had rotary subluxations after falling or somersaulting.

Table 2. Malformation Syndromes with Associated Cervical Spine Abnormalities

SYNDROME	SIGNS	CERVICAL SPINE ABNORMALITIES
Klippel-Feil	Short, webbed neck; low posterior hairline; sensorineural hearing loss	Cervical fusion, cervical ribs
Turner Syndrome	Female; short stature; webbed neck; low posterior hairline; small mandible; epicanthal folds; high arched palate; broad chest, cardiac abnormalities	Cervical hypoplasia
Goldenhar Syndrome	Hyperplasia of malar, maxillary, and mandibular face; ear abnormalities; hearing loss; cleft lip or palate	Cervical fusion, vertebral malformations (including posterior fusion); Chiari malformation
Crouzon Syndrome	Premature craniosynostosis; hypoplasia of maxilla; hypertelorism; proptosis; cleft lip and palate	Cervical fusion; foramen magnum stenosis w/Chiari malformation
Apert's Syndrome	Asymmetric face; proptosis; syndactyly of hands and feet; hypoplastic midface; beak nose; cleft palate	Cervical fusion; progressive calcifications; foramen magnum stenosis
VATER*	Anal atresia; tracheo-esophageal fistula; radial side malformations; renal defects	Cervical defects
Larsen's	Prominent forehead; flat face; hypertelorism; multiple dislocations; cleft palate; kyphosis	
Mucopolysaccharidosis	Coarse features; corneal clouding and other ophthalmologic abnormalities; short stature; hirsutism; hydrocephalus; macroglossia	Joint laxity with cervical instability-(especially MPS type IV); vertebral anomalies
Ehlers-Danlos	Skin hyperelasticity; fragile skin and blood vessels	Cervical ligament instability
Fetal Alcohol Syndrome	Short palpebral fissures; epicanthal folds; maxillary hypoplasia; micrognathia; thin upper lip; cardiac defects; delayed cognitive development	Cervical fusion

* VATER = Vertebral defects, Anal atresia, Tracheo-esophageal fistula with Esophageal atresia, and Radial and renal anomalies.

A general summary of the above data is necessary to give the clinician a sense of the type of impact that may cause a spine injury. Collisions in which a child impacts an object with a closing speed faster than a child can run or more than twice the child's height are greater-risk impacts. This is especially true in situations where the child may initiate contact with the skull, as is common in sports injuries. While falls can result in CSI, the injury-producing falls tend to be higher, or there are extenuating circumstances. One such circumstance occurs when a child falls down stairs with the extra weight of a walker attached. Unfortunately, as demonstrated by one group,[42] these guidelines are not completely reliable, and CSI may result from minimal trauma.

Diagnostic Evaluation

After understanding the epidemiology of CSI and the pertinent anatomy, it is necessary to discuss the appropriate evaluation of the potentially injured child. The evaluation of a child with a potential neck injury involves a clinical and radiographic evaluation.

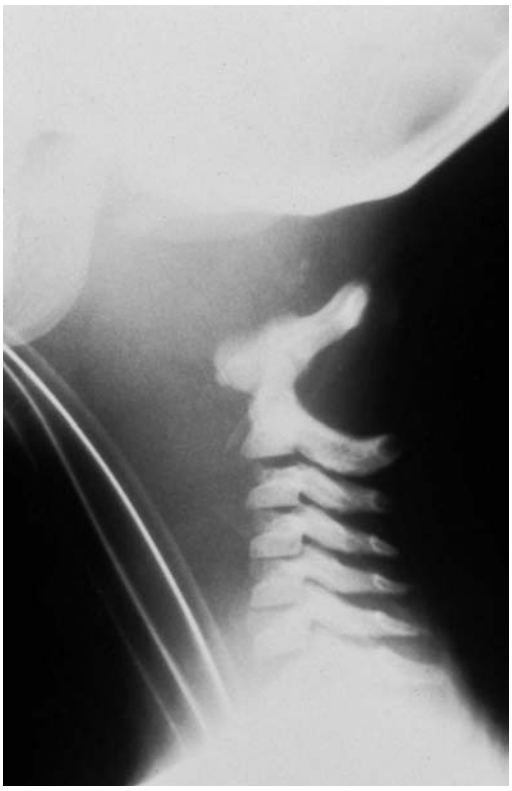
Clinical Evaluation. When faced with a child who has sustained a traumatic impact, the clinician must determine whether the child is at risk for having sustained a CSI. The clinician must make a decision regarding the need for immobilization of the child and the need to pursue diagnostic radiographic evaluation of the cervical spine. Recent work has narrowed the focus of physical examination findings that are present in adults with CSI.[43,44] Two published series identify low-risk criteria that include a normal physical examination of the neck and no history from the patient of neck complaints. The Canadian group characterized low-risk clinical criteria as being the victim of a low-speed rear end crash, being ambulatory prior to transport, or sitting in the department. The U.S. group included criteria requiring a clear sensorium and absence of a distracting injury (as defined by the treating physician). These clinical criteria did well in excluding the likelihood of an unstable cervical spine fracture in adults.

Unfortunately, there are no data in children to identify low-risk patients who do not require cervical spine radiographic evaluation. The Canadian study did not enroll children. The U.S. study identified 30 children with CSI (none younger than 2 years old, and four children younger than 9 years), and thus, lacks the power to support clinical guidelines. Therefore, while these clinical criteria are very similar to published guidelines and recommendations made after retrospective study of injured children, there are no prospective data to validate these recommendations.[15,25,26,45]

Emergency physicians continuously are faced with injured children and must make decisions regarding immobilization, imaging, and management, despite a paucity of clear criteria to use to evaluate these patients. Therefore, careful clinical judgment and evaluation are required. It is hoped that the emergency physician can refine clinical judgment by reviewing the aggregate of clinical presentations of published series of children with CSIs.

In the 30 pediatric patients with CSI from the U.S. trial, all

Figure 2. Severe Upper Cervical Spine Injury (AO and C_1-C_2)



were not low-risk by clinical criteria.[8] Twenty-one of 25 (five were unable to be evaluated for this criterion) patients had midline neck tenderness. Eleven of 28 had a distracting injury. Only eight of 27 had neurologic findings, while none had SCIWORA.

A retrospective review of 72 patients included information on the neck examination of 61.[22] Thirty had radiographically apparent CSI (RACSI) and 30 had SCIWORA. Sixteen of the 31 (51%) with RASCI reported midline neck tenderness, while 24 of 30 (80%) of those with SCIWORA had midline neck tenderness documented.

One review of 50 children with CSI noted that all 30 of the children awake at the time of ED arrival had neck pain or tenderness.[12] Another author reported the retrospective review of 25 children with CSIs.[23] She reported that the criteria of any history of neck pain or vehicle crash with head injury identified all children with CSI.

The retrospective review of children with low falls described eight children who had CSI after a low fall.[42] All eight patients had neck symptoms at the time of diagnosis. Unfortunately, the time of onset of these symptoms was not reported in the series.

Additionally, the data on SCIWORA reveal that any neurologic symptoms can be markers of SCIWORA. The data from one group report that all 32 patients in that series with SCIWORA had symptoms upon initial presentation, although some were isolated sensory deficits.[22] The data by another author note a percentage of children with SCIWORA that had a normal examination on presentation yet had neurologic deterioration days later.[46,47]

Table 3. Suggestions for Radiographic Evaluation

PRE-VERBAL OR PRE-COOPERATIVE CHILD AT RISK OF CSI

High Risk

- Fall in which the body weight lands on the head
- Head-on motor vehicle crash with child in a forward-facing seat
- Abnormal posture of the head and neck
- Anomaly of the face, head, or neck
- Any suspicion of non-accidental trauma
- Evidence of intracranial injury or significant facial trauma
- High speed, rear-end impact with an infant in a rear-facing seat
- Risky mechanism with distracting pain
- Neck tenderness
- Neurologic deficit
- Fall while in an infant walker

Low Risk

- Head-on motor vehicle crash with child in a rear-facing seat
- Short fall in which impact is evenly distributed between trunk and head
- Unwitnessed short fall with no scalp hematoma or soft-tissue injury
- Lateral impact motor vehicle crash with the child in appropriate restraint and no evidence of intracranial injury or concussion

VERBAL AND COOPERATIVE CHILD AT RISK FOR CSI

- Neck tenderness
- Neurologic abnormality
- Distracting pain with adequate mechanism
- Altered mental status
- High-energy impact involving a child younger than 8 years

In summation, a review of all the significant published series of children with CSI does not identify any criteria or criterion that will assure that a child, especially a pre-verbal or pre-cooperative child, is at low risk of an unstable CSI. Because SCIWORA exhibits a spectrum of presentations, the clinician must maintain diagnostic vigilance in any child with any neurologic symptoms. This is in contrast to the adult, in which one may consider transient, painful, radicular symptoms to be markers of peripheral nerve injury due to neck loading (i.e., the football player with a "stinger").

While noting from the above information that neck tenderness and possibly pain suggest a high-risk group, the converse is not true. How shall the clinician identify a high-risk group deserving radiographic evaluation among those without neck symptoms, those without neurologic symptoms, or those who are pre-verbal or pre-cooperative? The clinician must have a rough idea of the type of impact necessary to cause CSI, especially in the infant and toddler. Table 3 combines an arbitrary definition of high impact with somewhat arbitrary clinical criteria to produce guidelines for identifying children who may need radiographic evaluation of the cervical spine.

Radiographic Evaluation. Once the decision is made to obtain radiographic imaging, what are the appropriate studies to obtain? Authors vary on the routine studies for evaluation of the

Figure 3. C_6-C_7 Ligamentous Injury

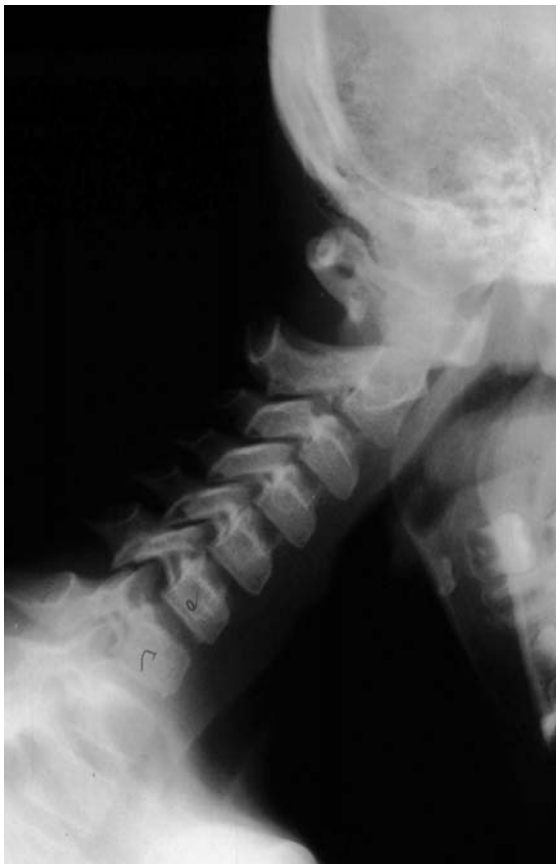
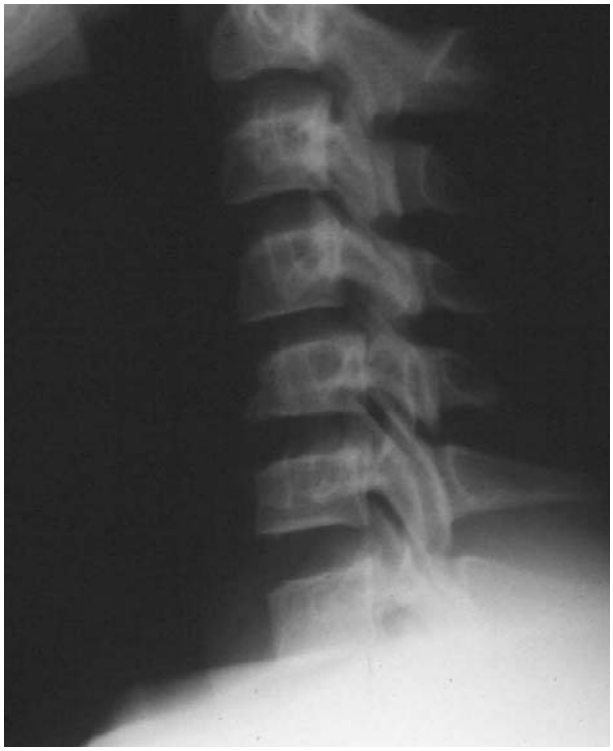


Figure 4. C_5 Fracture with Subluxation



A lateral cervical spine radiograph identifies a C_5 fracture with subluxation.

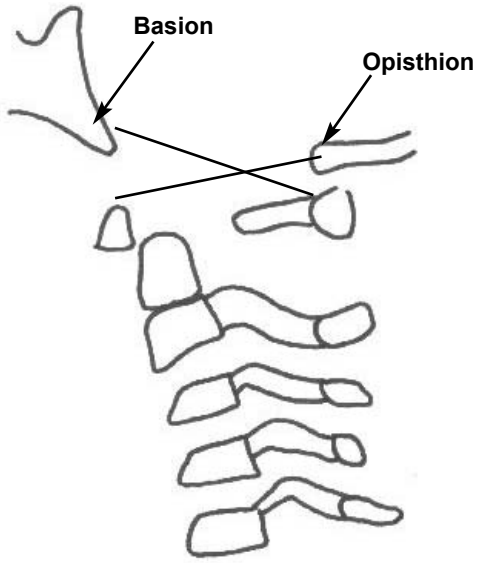
cervical spine. Some authors recommend a lateral view that visualizes the atlanto-occipital joint to the C_7-T_1 joint, an antero-posterior (AP) view, and an open-mouth odontoid in the cooperative patient.[12,48] Others advocate the three-view series for all patients.[10] A recently published survey of 432 pediatric radiologists notes that 40% of responders do not obtain the odontoid view in children younger than 5 years of age.[49] Another 25% only make one attempt at obtaining that view. Older recommendations suggested a five-view series for all patients, which included oblique views in addition to the standard three views.[50]

New data are available that suggest that computed tomography (CT) scanning of the cervical spine may be faster than obtaining plain films, especially in the patient who is to have a post-traumatic CT scan of another body region.[51-53] However, the role and accuracy of this technique have not been defined outside the multiple trauma patient.

The use of flexion-extension (FE) radiographs in evaluating alert trauma patients remains controversial. The FE views are imaging techniques that are used to delineate the endpoints of the patient's active neck flexion and extension that would radiographically identify any ligamentous injury. *(See Figure 3.)* The concern with FE views is that the patient will injure his or her spinal cord during performance of the test. Therefore, the utility of and indications for FE views are not clear. Data by two authors demonstrate that static radiography is adequate to diagnose CSI in 95% of patients.[12,22] *(See Figure 4.)* Three retrospective series suggest that FE films do not identify injuries in patients with normal static radiographic series.[48,54,55] In patients with subtle spine abnormalities, there was some diagnostic value to performing FE radiographs. In the only study of injury caused during FE studies, one patient of 129 had transient tingling in the upper extremities during positioning that resolved spontaneously after relaxation.[48]

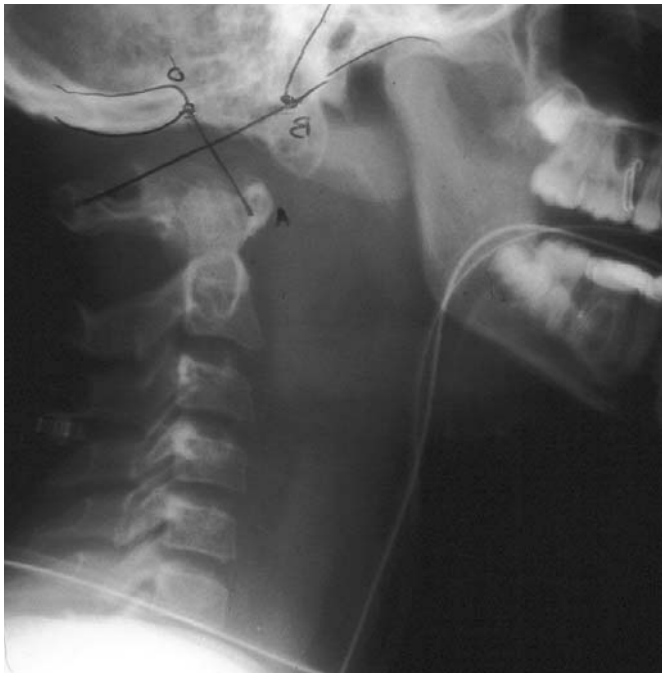
When ordering FE radiographs in the trauma patient, it is important to clarify whether the patient demonstrated adequate neck motion (FE) during the study.[56] In case of an inadequate study, a patient should be immobilized pending repeat radiographic evaluation in five days. Providing time for pain and muscle spasm to resolve should allow for a repeat, adequate radiographic study. However, it is unclear how often an FE study is inadequate. Up to one-third of FE radiographs ordered in adults acutely after trauma may be inadequate.[57] These data note that angular motion of approximately 40º is necessary between C_2 and C_7 for an adequate study. There is no clear definition of adequate motion on pediatric FE films. Specifically, the normal degree of tilting and relative motion between vertebral bodies changes throughout childhood. One author has demonstrated that tilting angles during flexion decrease with age at the C_{2-3} and C_{3-4} junctions.[2] Extension tilting increases with age at the C_{4-5} and C_{5-6} joints. These tilting changes are changes of approximately 3-5º throughout childhood. Sliding motion during flexion decreases with age at the C_{2-3}, C_{3-4}, and

Figure 5. Calculating Powers Ratio



Powers ratio is determined by the ratio of distance from the basion to posterior arch of C_1 to the distance from the opisthion to the anterior arch of C_1.

Figure 6. An AO Injury Diagnosed by Powers Ratio



C_{4-5} joints. This motion decreases from 18-25% down to 5-10% of vertebral width.

Acute FE magnetic resonance imaging (MRI) may have a role in identifying pediatric operative candidates sustaining ligamentous injury.[58]

Specific Injuries that Occur in Children

Occipitoatlantal Dislocation. The outcome of children with occipitoatlantal dislocation is uniformly poor.[12] This injury usually results from a high-energy impact, as seen in motor vehicle crashes. Examination of the cervical spine radiographs must include assurance of an appropriate relationship between the occiput and the atlas. The distance between the basion and the dens should not exceed 10 mm in children and 5 mm in adults.[59] It is not uncommon for normal patients to exceed these criteria.[60] The Powers ratio can be calculated. The ratio of the distance from the basion to the anterior edge of the posterior arch of the atlas divided by the distance from the opisthion to the posterior portion of the anterior arch of the atlas should be less than 0.9. A ratio greater than 1.0 is abnormal. The Wackenheim clivis line is another technique to inspect for integrity of the atlanto-occipital joint. In this test, a line drawn along the posterior clivus should intersect or be tangential to the odontoid.[61-63] *(See Figures 5 and 6.)*

Atlas Fractures. Burst fractures of the C_1 ring can occur in children, just as in adults. The fracture may occur through the synchondroses, which may remain unfused until age 7.[64] CT scan may assist in securing the diagnosis.

Atlantoaxial Injuries. Authors differ over the relative frequency of transverse ligament injuries compared to dens fractures. Although fractures through the base of the dens do occur at the synchondrosis, transverse ligament tears also are commonly seen. An atlantodens interval of 5 mm may suggest injury to the transverse ligament.

Surprisingly, children with dens fractures frequently are neurologically intact upon presentation. One series quotes seven of 15 children without weakness on presentation. Four of 15 had thoracic level symptoms, and three had low cervical symptoms (C_6 and C_7).[65] Thirteen of the 15 had anterior displacement ranging from 10-100% (mean 40%). One patient had a fracture, C_2 tetraplegia, and no displacement of the dens. Two patients with 20% displacement had a delay in diagnosis of four and six months. Tomograms may show widening of the growth plate not evident on plain radiographs.[7] CT scans require coronal and sagittal plane reconstruction because axial scanning may miss fractures.[66,67] As the growth plates close, it is important not to confuse the epiphyseal scar at the base of the dens with an acute fracture.

Os Odontoideum. Os odontoideum refers to an oval or round ossicle of variable size with a smooth cortical border located in the position of the odontoid process. Authors differ, but suspect that this is an acquired lesion after an undiagnosed odontoid fracture. These lesions commonly are unstable.[68,69]

Hangman's Fracture. Hangman's fracture, or C_2 pedicle fractures, can occur in children with a hyperextension injury, just as with adults. *(See Figure 7.)* The diagnosis of a hangman's fracture may be confused with physiologic subluxation of C_2 on C_3. Evaluating the alignment of the posterior laminar line can assist with clarifying the diagnosis.

Atlantoaxial Rotary Subluxation. Atlantoaxial rotary subluxation may occur spontaneously (Grisel's syndrome) or after minor trauma. The classic clinical presentation is torticollis in the "cock-robin" position, with the head rotated to one side and tilted

Figure 7. Hangman's Fracture

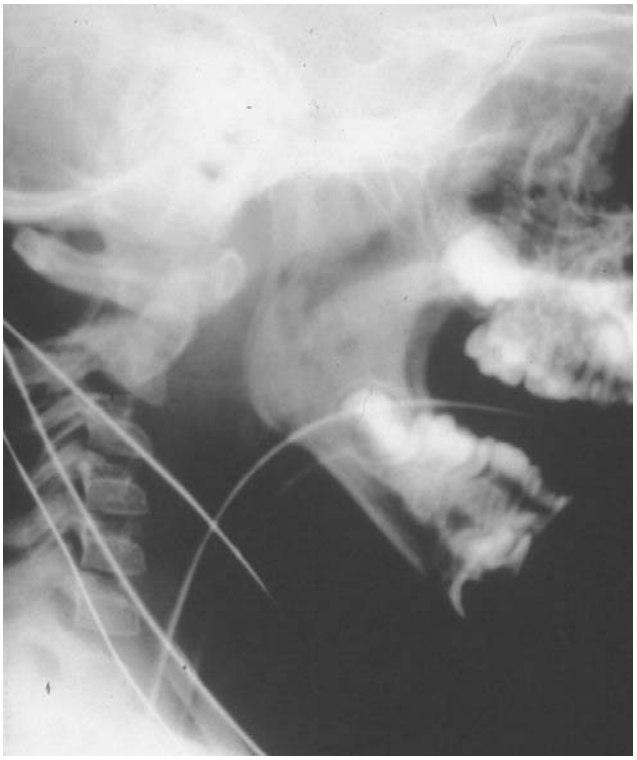
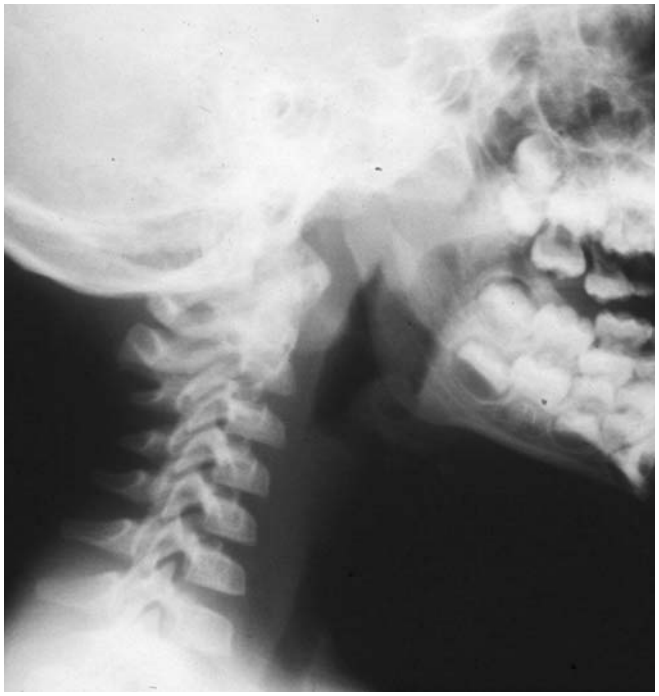


Figure 8. Apparent Anterior Vertebral Wedging



to the other, like a bird listening for a worm.[70] This usually stable injury is truly a pediatric injury, as up to 80% of these injuries occur in children younger than 13 years of age.[71,72] This type of rotary subluxation occurs most frequently at the C_1-C_2 joint, where most of the rotation of the neck occurs. The facets are flatter at this joint than at any other joint.

The diagnosis of rotary subluxation is difficult to secure with plain films alone. The abnormalities seen can be present in patients with torticollis not due to subluxation and in volunteers holding their head in the "cock-robin" position.[7,73,74] CT scanning, with 3D reconstruction, often is necessary to make the diagnosis.

Normal Radiographic Variants Simulating Injury

There are several common variations in the pediatric cervical spine radiograph that may simulate injury. This section will describe the pattern of variation and describe how to distinguish it from pathology.

Pseudosubluxation. Anterior displacement of C_2 on C_3 of up to 4 mm is common in children younger than 7 years, but frequently can be noted in children up to 16 years of age. A line drawn from the anterior cortex of the spinous process of C_1 to C_3 should come within 1.5 mm of the anterior spinous process of C_2. This misalignment may improve with FE views, but certainly isn't exaggerated by FE study. Pseudosubluxation also can occur at the level of C_3-C_4.

Apparent Anterior Vertebral Wedging. Anterior wedging of the vertebral body may be seen on pediatric radiographs. This wedging represents non-uniform calcification of the vertebral body, not an asymmetric shape. Wedging is most common at C_3 and may account for up to 3 mm difference between the anterior and posterior height of the vertebral body. The vertebral bodies should assume an adult shape by age 8. This variant can be noted in Figure 8 at C_3 and C_4.

Overriding Anterior Arch of C_1. Up to two-thirds of the anterior arch of C_1 may override the tip of the dens. This occurs in up to 20% of children younger than 7 years.[75]

Increased Predental Space. The predental space in children can be up to 5 mm. Ligamentous laxity (of the transverse and anterior atlanto-axial ligaments) may allow for an increased gap compared to the adult measurement of 3 mm.

Apical Odontoid Epiphysis. The odontoid tip has an epiphysis that usually is present at age 7, but may persist through age 16.[75,76]

Persistent Synchondrosis of the Dens. The growth plate at the base of the dens persists beyond age 7. This linear scarring may be confused with a fracture. This line is typically linear, occurs in a predictable location, and may have associated sclerosis. A fracture more commonly presents in an unpredictable location without sclerosis and with irregular edges.

Non-uniform Angulation During Flexion. There may be non-uniform angulation between adjacent vertebral bodies during flexion. This may appear as marked flexion at a single joint. Although this can be a normal variant, it is difficult to distinguish from acute trauma in the correct clinical setting.[75]

Asymmetric Odontoid. The odontoid may be centered asymmetrically between the lateral masses of C_1. Ligamentous laxity may cause this variant. While this can be a normal variant, it may be difficult to distinguish from an acute fracture in the correct clinical setting.[77]

Delayed Calcification of Anterior Arch of Axis. The anterior arch of C_1 frequently is not visible on plain film radiographs until 6 months of age. Before this, the axis is calcified insufficiently to be visible radiographically.

Treatment and Disposition

When treating patients with CSIs, emergency medicine physicians must remain focused on three goals. First, the emergency medicine physician must identify all significant injuries, including the CSI. The emergency physician, as the initial physician contact, must keep the complete patient clinical picture in mind to allow subspecialists to concentrate on specific injuries. Second, the emergency medicine physician must take steps to prevent worsening of any neurologic function associated with the spinal cord injury. Basic fundamentals must be addressed to complete this goal. Spinal immobilization of unstable injuries is imperative. Care must be taken during intubation to prevent further cord injury. Identification and treatment of shock is important to maintain adequate perfusion to the injured spinal cord. Finally, the emergency physician must expedite therapy for any CSI and associated spinal cord injury. Adult data suggest that urgent release of spinal cord compression may improve outcome. Urgent MRI may be necessary to identify cord compression. Intravenous steroids may be indicated for treatment of a spinal cord injury.

Steroids in Children. The use of steroids has been advocated for the treatment of patients with acute spinal cord injuries.[78,79] The indication for treatment in the quoted NASCIS trials was "having a spinal cord injury" as defined by study physicians. Approximately 8% of patients had normal neurologic function on enrollment in NASCIS 3.[79] No patient younger than age 14 was enrolled in this series. Patients with gunshot wounds were excluded, but patients with other forms of penetrating trauma were not excluded from study. Outcomes were not reported according to the type of cord injury sustained.

While steroid dosing in adults with acute spinal cord injuries may or may not provide benefit, the benefit and indications of this therapy in young children has not been proven. Centers should establish a consensus for the treatment of children after spinal trauma. Communication between emergency physicians and accepting subspecialists should be done so that all members of the treatment team will have a common understanding when a patient arrives in the ED. Controversy may arise about the treatment of a child with sensory deficits of an isolated cervical level or the treatment of a very young child with an apparently normal neurological examination with an abnormal cervical spine radiograph.

Conclusion

Evaluation of the pediatric patient with a potential CSI is a complicated process. The emergency physician must exercise thoughtful clinical judgment in evaluating a pre-cooperative patient at a low risk of a potentially catastrophic injury. A complete understanding of the pertinent anatomy, radiographic features, and biomechanical tolerance of the pediatric spine is necessary to provide this care.

References

1. Castellana C, Kosa F. Morphology of the cervical vertebrae in the fetal-neonatal human skeleton. *J Anat* 1999;194:147-52.
2. Kasai T, Ikata T, Katoh S, et al. Growth of the cervical spine with special reference to its lordosis and mobility. *Spine* 1996;21: 2067-2073.
3. Manaligod JM, Bauman NM, Menezes AH, et al. Cervical vertebral anomalies in patients with anomalies of the head and neck. *Ann Otol Laryngol* 1999;108:925-933.
4. Ugar DA, Semb G. The prevalence of anomalies of the upper cervical vertebrae in subjects with cleft lip, cleft palate, or both. *Cleft Palate-Craniofacial Journal* 2001;38:498-503.
5. Guille JT, Sherk HH. Congenital osseous anomalies of the upper and lower cervical spine in children. *J Bone Joint Surg* 2002;84: 277-288.
6. Torg JS, Naranja RJ, Pavlov H, et al. The relationship of developmental narrowing of the cervical spinal canal to reversible and irreversible injury of the cervical spinal cord in football players: An epidemiological study. *J Bone Joint Surg* 1996;78:1308-1314.
7. Weinstein SL, ed. *The Pediatric Spine: Principles and Practice.* 2nd ed. Baltimore: Lippincott Williams & Wilkins; 2001:1.
8. Viccellio P, Simon H, Pressman BD, et al. A prospective multicenter study of cervical spine injury in children. *Pediatrics* 2001;108:e20.
9. Brown RL, Brun MA, Garcia VF. Cervical spine injuries in children: A review of 103 patients treated consecutively at a level 1 pediatric trauma center. *J Pediatr Surg* 2001;36:1107-1114.
10. Eleraky MA, Theodore N, Adams M, et al. Pediatric cervical spine injuries: Report of 102 cases and review of the literature. *J Neurosurg* 2000;92:12-17.
11. Kokoska ER, Keller MS, Rallo MC, et al. Characteristics of pediatric cervical spine injuries. *J Pediatr Surg* 2001;36:10-105.
12. Dietrich AM, Ginn-Pease ME, Barkowski HM, et al. Pediatric cervical spine fractures: Predominately subtle presentation. *J Pediatr Surg* 1991;26:995-1000.
13. Bohn D, Armstrong D, Becker L, et al. Cervical spine injuries in children. *J Trauma* 1990;30:463-469.
14. Ruge JR, Sinson GP, McLone DG, et al. Pediatric spinal injury: The very young. *J Neurosurg* 1988;68:25-30.
15. Patel JC, Tepas III JJ, Mollitt DL, et al. Pediatric cervical spine injuries: Defining the disease. *J Pediatr Surg* 2001;36:373-376.
16. Hill SA, Miller CA, Kosnik EJ, et al. Pediatric neck injuries: A clinical study. *J Neurosurg* 1984;60:700-706.
17. Hadley MN, Zabramski JM, Browner CM, et al. Pediatric spinal trauma: Review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg* 1988;68:18-24.
18. Orenstein JB, Klein BL, Gotschall CS, et al. Age and outcome in pediatric cervical spine injury: 11-year experience. *Pediatr Emerg Care* 1994;10:132-137.
19. Givens TG, Polley KA, Smith GF, et al. Pediatric cervical spine injury: A three-year experience. *J Trauma* 1996;41:310-314.
20. Apple JS, Kirks DR, Merten DF, et al. Cervical spine fractures and dislocations in children. *Pediatr Radiol* 1987;17:45-49.
21. Orenstein JB, Klein BL, Ochsenschlager DW. Delayed diagnosis of pediatric cervical spine injury. *Peds* 1992;89:1185-1188.
22. Baker C, Kadish H, Schunk JE. Evaluation of pediatric cervical

spine injuries. *Am J Emerg Med* 1999;17: 230-234.
23. Rachesky I, Boyce WT, Duncan B, et al. Clinical predictors of cervical spine injuries in children. *Am J Dis Child* 1987;141:199-201.
24. Tarazi F, Dvorak MF, Wing PC. Spinal injuries in skiers and snowboarders. *Am J Sports Med* 1999;27:177-180.
25. Floyd T. Alpine skiing, snowboarding, and spinal trauma. *Arch Orthop Trauma Surg* 2001;121:433-436.
26. Lee KS, Doh JW, Bae HG, et al. Delayed central cord syndrome after a handstand in a child: Case report. *Paraplegia* 1996;34: 176-178.
27. Brown PG, Lee M. Trampoline injuries of the cervical spine. *Pediatr Neurosurg* 2000;32:170-175.
28. Wolfe JM, Anderson R. Human body surfing: A new way to incur injury at spectator events. *Am J Emerg Med* 1997;15:97-98.
29. Schmitt H, Gerner HJ. Paralysis from sport and diving accidents. *Clin J Sports Med* 2001;11:17-22.
30. all-Sebai M, al-Zahrani S. Cervical spinal injuries caused by collision of cars with camels. *Injury* 1997;28:191-194.
31. Russell A, Boop FA, Cherny WB, et al. Neurologic injuries associated with all-terrain vehicles and recommendations for positive measures for the pediatric population. *Pediatr Emerg Care* 1998; 14:31-35.
32. Kadesky KM, Manery C, Blair GK, et al. Cougar attacks on children: Injury patterns and treatment. *J Pediatr Surg* 1998;33:863-865.
34. Joffe M, Ludwig S. Stairway injuries in children. *Pediatrics* 1988; 82:457
35. Chiaviello CT, Christoph RA, Bond GR. Stairway-related injuries in children. *Pediatrics* 1994;94:679-681.
36. Chiaviello CT, Christoph RA, Bond GR. Infant walker-related injuries: a prospective study of severity and incidence. *Pediatrics* 1994;93:974-976.
37. Lyons TJ, Oats RK. Falling out of bed: A relatively benign occurrence. *Pediatrics* 1993;92:125-127.
38. Mayr JM, Seebacher U, Schimpl G, et al. Highchair accidents. *Acta Paediatrica* 1999;88:319-322.
39. Smith GA, Dietrich AM. Garcia CT, et al. Injuries to children related to shopping carts. *Pediatrics* 1996;97:161-165.
41. Greenes DS. Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. *Pediatrics* 1999;104:861-867.
42. Schwartz GR, Wright SW, Fein JA, et al. Pediatric cervical spine injury sustained in falls from low heights. *Ann Emerg Med* 1997;30: 249-252.
43. Hoffman JR, Mower WR, Wolfson AB, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. *N Engl J Med* 2000;343:94-99.
44. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian c-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001; 286:1841-1848.
45. Dormans JP. Evaluation of children with suspected cervical spine injury. *J Bone Joint Surg* 2002;84:124-132.
46. Pang D, Wilberger JE. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982;57:1114-1129.
47. Pang D, Pollack IF. Spinal cord injury without radiographic abnormality in children—The SCIWORA syndrome. *J Trauma* 1989; 29:654-664.
48. Ralston ME, Chung K, Barnes PD, et al. Role of flexion-extension radiographs in blunt pediatric cervical spine injury. *Acad Emerg Med* 2001;8:237-245.
49. Swischuk LE, John SD, Hendrick EP. Is the open-mouth odontoid view necessary in children under 5 years? *Pediatr Radiol* 2000;30: 186-189.
50. Freemyer B, Knopp R, Piche J, et al. Comparison of five-view and three-view cervical spine series in the evaluation of patients with cervical trauma. *Ann Emerg Med* 1989;18:818-821.
52. Barba CA, Taggart J, Morgan AS, et al. A new cervical spine clearance protocol using computed tomography. *J Trauma* 2001;51: 652-657.
53. Keenan HT, Hollingshead MC, Chung CJ, et al. Using CT of the cervical spine for early evaluation of pediatric patients with head trauma. *Am J Roentgenol* 2001;177:1405-1409.
54. Woods, WA, Brady WJ, Pollock G, et al. Flexion-extension cervical spine radiography in pediatric blunt trauma. *Emerg Radiol* 1998;5: 381-384.
55. Dwek JR, Chung CB. Radiography of cervical spine injury in children: Are flexion-extension radiographs useful for acute trauma? *Am J Roentgenol* 2000;174:1617-1619.
56. Kuhns LR, Strouse PJ. Cervical spine standards for flexion radiograph interspinous distance ratios in children. *Acad Radiol* 2000; 7:615-619.
57. Wang, JC, Hatch JD, Sandhu HS, et al. Cervical flexion and extension radiographs in acutely injured patients. *Clin Orthoped Rel Res* 1999;364:111-116.
58. Weng MS, Haynes RJ. Flexion and extension cervical MRI in a pediatric population. *J Pediatr Orthoped* 1996;16:359-363.
59. Wholey MH, Bruwer AJ, Baker HL. The lateral roentgenogram of the neck. *Radiol* 1958;71:350-356.
60. Powers B, Miller MD, Kramer RS, et al. Traumatic anterior atlanto-occipital dislocation. *Neurosurg* 1979;4:12-17.
61. Abroms IF, Bresnan MJ, Zuckerman JE, et al. Cervical cord injuries secondary to hyperextension of the head in breech presentation. *Obstet Gynecol* 1973;41:369-378.
62. Allen BL, Ferguson RL. Cervical spine trauma in children. In: Bradford DS, Hensinger RN, eds. *The Pediatric Spine.* New York: Thieme Medical Publishers;1985:1.
63. Kewalramani LS, Tori JG. Spinal cord trauma in children, neurologic patterns, radiologic features, and pathomechanics of injury. *Spine* 1980;5:11-18.
64. Galindo MJ, Francis WR. Atlantal fracture in a child through congenital anterior and posterior arch defects: A case report. *Clin Orthop* 1983;178:220-222.
65. Lebwohl NH, Eismont FJ. Cervical spine injuries in children. In: Weinstein SL, ed. *The Pediatric Spine: Principles and Practice.* 2nd ed. Baltimore: Lippincott Williams & Wilkins;2001:1.
65. Odent T, Langlais J, Glorion C, et al. Fractures of the odontoid process: A report of 15 cases in children younger than 6 years. *J Ped Ortho* 1999;19:51-54.
66. Murphey MD, Batnitzky S, Bramble JM. Diagnostic imaging of spinal trauma. *Radiol Clin North Am* 1989;27:855-872.
67. Pathria MN, Petersilge CA. Spinal trauma. *Radiol Clin North Am* 1991;29:847-865.

68. Harris JH Jr, Mirvis SE. *The Radiology of Acute Cervical Spine Trauma* 3rd ed. Baltimore: Williams & Wilkins;1996:421-474.
69. Fielding JW, Hensinger Rn, Hawkins RJ. Os odontoideum. *J Bone Joint Surg Am* 1980;62:376-383.
70. Roche C, Carty H. Spinal trauma in children. *Pediatr Radiol* 2001; 31:677-700.
71. Lukhelc M. Atlanto-axial rotatory fixation. *S Afr Med J* 1996;86: 1549-1552.
72. Mathern G, Batzdorf U. Grisel's syndrome. *Clin Orthop Rel Res* 1989;244:131-146.
73. Kowalski M, Cohen W, Cooper P, et al. Pitfalls in the CT diagnosis of atlantoaxial rotatory subluxation. Am J Neuroradiol 1987;8: 697-702.
74. Kawabe N, Hirotani M, Tanaka O. Pathomechanism of atlantoaxial rotatory fixation in children. *J Pediatr Orthop* 1989;9:569-574.
75. Cattell HS, Filtzer DL. Pseudo-subluxation and other normal variations in the cervical spine in children. *J Bone Joint Surg Am* 1965; 47:1295-1309.
76. Bonadio WA. Cervical spine trauma in children. I. General concepts, normal anatomy, radiographic evaluation. *Am J Emerg Med* 1993;11:158-165.
77. Lee S, Joyce S, Joachim S. Asymmetry of the odontoid-lateral mass interspaces: a radiographic finding of questionable clinical significance. *Ann Emerg Med* 1986;15:1173-1177.
78. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. *N Engl J Med* 1990;322:1405-1411.
79. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methyprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. *JAMA* 1997; 277:1597-1604.

CE/CME Questions

1. At what age is ossification of the dens usually complete in a pediatric patient?
   A. 1 year
   B. 3 years
   C. 4 years
   D. 5 years
   E. 6-8 years

2. Which of the following increases a child's risk for abnormal cervical spine development?
   A. Neuromuscular diseases
   B. Cerebral palsy
   C. Apert's syndrome
   D. Muscular dystrophy
   E. Rett's syndrome

3. A 4-year-old child presents after falling off a horse and landing on his head. The child has midline cervical pain with a normal neurologic examination. What would be your radiographic recommendation?
   A. No radiographs are necessary
   B. Flexion extension views only
   C. MRI of the spine
   D. Lateral cervical spine radiograph

4. Significant CSI does not occur as an isolated injury.
   A. True
   B. False

5. Which of the following is true regarding occipitoatlantal dislocations?
   A. Outcome usually is poor.
   B. The injury usually results from a high-energy impact.
   C. The Powers ratio is useful for assessing the existence of this type of injury.
   D. The Wackenheim clivus line also may be used to diagnose this injury.
   E. All of the above

6. An atlantodens interval of 5 mm may suggest injury to the transverse ligament.
   A. True
   B. False

7. Which of the following is *not* true regarding flexion extension films in pediatric patients?
   A. There is no clear definition of adequate motion of pediatric flexion extension films.
   B. Up to one-third of adult flexion extension films may be inadequate.
   C. In adults, approximately 40 degrees of motion between C_6 and C_7 is necessary for an adequate film.
   D. Flexion extension films may result in spinal cord injury.
   E. The normal degree of tilting and relative motion between vertebral bodies changes through childhood.

8. A Powers ratio of greater than 1.0 is abnormal.
   A. True
   B. False

9. Which of the following is true regarding Os odontoideum?
   A. It usually is a rectangular-shaped, irregularly margined bone fragment.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

B. It usually is located in the position of the odontoid process.
C. It is a congenital lesion.
D. These lesions commonly are stable.

10. Which of the following is *not* true regarding atlantoaxial rotary subluxation?
A. It usually is associated with major trauma.
B. Patients classically present with torticollus.
C. Up to 80% of these injuries occur in children younger than 13 years of age.
D. The injury most frequently occurs at the C_1-C_2 joint.
E. The diagnosis may be difficult to make with plain radiographs.

Answer key:

1. E
2. C
3. D
4. B
5. E
6. A
7. D
8. A
9. B
10. A

CME Objectives

Upon completing this program, the participants will be able to:
a.) Quickly recognize or increase index of suspicion for occult spinal trauma in pediatric patients;
b.) Be educated about how to correctly and quickly stabilize, and then to manage, cervical spine trauma;
c.) Understand various diagnostic modalities for cervical spine injuries; and
d.) Understand both likely and rare complications that may occur.

In Future Issues: **Recognizing Child Abuse**