Management of Common Neurologic Conditions in Sports

Elliot L. Dimberg, MD, Ted M. Burns, MD*

Department of Neurology, University of Virginia, Box 800394 Charlottesville, VA 22908, USA

Neurological conditions are common in athletes. Trauma can cause direct central (eg, concussion or hemorrhage) or peripheral (eg, stinger) injury. Also, as neurological conditions in athletes become better understood, more people who have pre-existing conditions are becoming involved in organized sports. This article reviews assessment and initial management of head injury, stingers, seizures, and headaches. Return-to-play criteria are also discussed.

CONCUSSION AND CATASTROPHIC INJURY

The American Academy of Neurology (AAN) has defined concussion as “a trauma-induced alteration in mental status that may or may not involve loss of consciousness” [1]. This definition underscores that the injury may not necessarily involve a direct blow to the head, nor loss of consciousness. The Center for Disease Control has reported that approximately 300,000 sports related concussions occur annually [2]. Actual incidence may be higher because of potential underreporting of concussion symptoms by athletes [3]. Concussions may occur in sports and situations not typically thought to put the athlete at risk, and not necessarily during games [4]. Such information should put the athletic trainer and physician at high suspicion for such an injury. The sports medicine practitioner must be able to recognize, assess, and manage the acute setting, determine appropriate return-to-play (RTP) time frames, and understand the long-term sequelae of this injury.

Preinjury Education and Assessment
Concussion management begins with preinjury education. It is important that the athlete be educated on symptoms of concussion, because recent reports have shown that athletes often are not aware that they have suffered a concussion [3]. The potential long-term and cumulative sequelae should be emphasized [5], because disregard for the seriousness of concussion can also lead to athlete
underreporting [3]. The AAN has released a “Patient Page” on concussion at www.neurology.org/cgi/reprint/63/8/E15 that can assist with the education of athletes at any level and their parents.

A total concussion history should be taken after concussion education. Previously concussed athletes have been shown to be at significantly higher risk for repeat concussions than teammates who have no prior concussions [6]. Previous concussions may have already caused neurocognitive and neuropsychological consequences that can affect postinjury assessment accuracy; this underscores the need for preinjury testing [7]. Previously concussed athletes have also been shown to report fewer symptoms at 2 hours postinjury, although they report more symptoms at baseline and at 1 week postinjury [8]. This also has implications for postinjury assessment and RTP decisions.

Practitioners should decide which assessment tool will be used once contact begins, and athletes should undergo baseline testing for accurate comparison postinjury. Baseline testing establishes accurate preinjury level of functioning and allows for effective correction for confounding factors such as learning disabilities, behavioral disorders, education level, and intelligence. Learning

1) ORIENTATION:

<table>
<thead>
<tr>
<th>Month</th>
<th>Date</th>
<th>Time (within 1 hr)</th>
<th>Orientation Total Score</th>
</tr>
</thead>
<tbody>
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</table>

2) IMMEDIATE MEMORY: (all 3 trials are completed regardless of score on trial 1 & 2; total score equals sum across all 3 trials)

<table>
<thead>
<tr>
<th>List</th>
<th>Trial 1</th>
<th>Trial 2</th>
<th>Trial 3</th>
</tr>
</thead>
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<tr>
<td>Word 3</td>
<td>0</td>
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</tr>
</tbody>
</table>

Immediate Memory Total Score / 15

(Note: Subject is not informed of delayed recall testing of memory)

3) CONCENTRATION:

Digits Backward (If correct, go to next string length. If incorrect, read trial 2. Stop after incorrect on both trials.)

<table>
<thead>
<tr>
<th>Word 1</th>
<th>Word 2</th>
<th>Word 3</th>
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<tbody>
<tr>
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<td>6-2-9</td>
<td>0 1</td>
</tr>
<tr>
<td>3-8-1-4</td>
<td>3-2-7-9</td>
<td>0 1</td>
</tr>
<tr>
<td>6-2-9-7-1</td>
<td>1-5-2-8-6</td>
<td>0 1</td>
</tr>
</tbody>
</table>

Months in Reverse Order: (entire sequence correct for 1 point)

<table>
<thead>
<tr>
<th>Month</th>
<th>Date</th>
<th>Year</th>
<th>Orientation Total Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dec-Nov-Oct-Sep-Aug-Jul</td>
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</tbody>
</table>

Concentration Total Score / 5

4) DELAYED RECALL:

<table>
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<th>Word 1</th>
<th>Word 2</th>
<th>Word 3</th>
<th>Word 4</th>
<th>Word 5</th>
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</thead>
<tbody>
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<td>0 1</td>
<td>0 1</td>
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<td>0 1</td>
<td>0 1</td>
</tr>
</tbody>
</table>

Delayed Recall Total Score / 5

SUMMARY OF TOTAL SCORES:

<table>
<thead>
<tr>
<th>ORIENTATION</th>
<th>IMMEDIATE MEMORY</th>
<th>CONCENTRATION</th>
<th>DELAYED RECALL</th>
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</thead>
<tbody>
<tr>
<td>/ 5</td>
<td>/ 15</td>
<td>/ 5</td>
<td>/ 5</td>
</tr>
</tbody>
</table>

OVERALL TOTAL SCORE / 30

Fig. 1. Standardized Assessment of Concussion (SAC). (From McCrea M. Standardized mental status testing on the sideline after sport-related concussion. J Athl Train 2001;36(3):276; with permission.)
disability in itself is associated with lower baseline neuropsychological functioning, but also may be associated with significantly worse long-term neurocognitive sequelae in conjunction with multiple concussions [7]. The National Athletic Trainer’s Association (NATA) Position Statement [9] currently recommends that sideline screening measures, such as the Standardized Assessment of Concussion (SAC) [10,11] (Fig. 1), the Balance Error Scoring System (BESS) [12] (described in Box 1 below) or symptom checklists [9] (Table 1), be used in conjunction with more extensive neuropsychological testing or vestibular balance testing. The use of computerized neuropsychological testing is gaining acceptance by some for these purposes due to time, cost, and ease of testing [13]. Should these or like measures be used, baseline testing should be obtained. If they are not used, RTP may be delayed, or worse, premature.

**Box 1: Balance error scoring system (BESS)**

**Test procedure**

- Test in three different stances (both feet, nondominant foot, tandem), two times each (once on firm surface, once on medium-density foam 45 cm² × 13 cm thick, density 60 kg/m³, load deflection 80–90 kg).
- Place hands on iliac crests, close eyes.
- Once eyes are closed, test for 20 seconds.
- On one foot, elevated leg is maintained at 20° to 30° hip flexion and 40° to 50° knee flexion.
- Stand as quietly as possible in position.
- If balance is lost, make any necessary adjustments to return to position.
- One error point for each error committed
- Incomplete test if unable to maintain stance for more than 5 seconds.
- Maximum score of 10.

**Scoring system**

- Errors
- Hands lifted off iliac crest
- Opening eyes
- Step, stumble, or fall
- Moving hip into more than 30° of flexion or abduction
- Lifting forefoot or heel
- Remaining out of testing position for more than 5 seconds
- Add 1 point for each error during a 20-second trial

Finally, a thorough neurological examination should be performed. This should include assessment of mental status, cranial nerves, motor and sensory systems, coordination, deep tendon reflexes, and gait. This not only serves as a baseline examination, but also alerts the practitioner to potential pre-existing conditions that may warrant further specialist investigation and may prevent inclusion in competition.

**Clinical Manifestations**
The clinical manifestations of concussion, either as witnessed by the observer or reported by the athlete, are highly varied. As noted in the AAN definition of

### Table 1
**NATA graded symptoms checklist (GSC)**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Time of injury</th>
<th>2–3 hours postinjury</th>
<th>24 hours postinjury</th>
<th>48 hours postinjury</th>
<th>72 hours postinjury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blurred vision</td>
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<td>Dizziness</td>
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<tr>
<td>Drowsiness</td>
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<tr>
<td>Excess sleep</td>
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<tr>
<td>Easily distracted</td>
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<tr>
<td>Fatigue</td>
<td></td>
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<tr>
<td>Feel “in a fog”</td>
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<tr>
<td>Feel “slowed down”</td>
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<tr>
<td>Headache</td>
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<tr>
<td>Inappropriate emotions</td>
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<tr>
<td>Irritability</td>
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<tr>
<td>Loss of consciousness</td>
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<tr>
<td>Loss or orientation</td>
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<tr>
<td>Memory problems</td>
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<tr>
<td>Nausea</td>
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<tr>
<td>Nervousness</td>
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<td>Personality change</td>
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<tr>
<td>Poor balance/coordinaton</td>
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<td>Poor concentration</td>
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<tr>
<td>Ringing in ears</td>
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<tr>
<td>Sadness</td>
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<td></td>
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<tr>
<td>Seeing stars</td>
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<tr>
<td>Sensitivity to light</td>
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<td></td>
<td></td>
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<tr>
<td>Sensitivity to noise</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Sleep disturbance</td>
<td></td>
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<tr>
<td>Vacant stare/glassy eyed</td>
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<tr>
<td>Vomiting</td>
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</tbody>
</table>

The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the ATC can ask the athlete to grade or score the severity of the symptom on a scale of 0–6, where 0 = not present, 1 = mild, 3 = moderate, and 6 = most severe.

concussion, loss of consciousness is not a necessary manifestation [1], and its presence as a benchmark for concussion severity and prognosis has come into question [14,15]. The most common symptoms, in decreasing frequency, are: headache, dizziness, confusion and disorientation, nausea, loss of consciousness, retrograde amnesia, and vomiting [16] (Table 2). The athlete may be unable to answer questions relating to contest specifics, date, place or time. He or she may suffer from imbalance or transient visual phenomena, including photophobia. Amnesia may also be anterograde. Observers may note a vacant stare, latent response times, poor focus of attention, difficulties of speech, incoordination, or emotional lability. If an athlete presents any signs or symptoms consistent with concussion, then he or she has almost certainly suffered a concussion and should be treated as such.

Postinjury Assessment
There is no universally accepted standard for concussion assessment, but basic guidelines are definable. Initial on-field care should focus on assessing level of

<table>
<thead>
<tr>
<th>Table 2</th>
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<tbody>
<tr>
<td>Frequency of symptoms observed at the sideline evaluation and observed or reported at the initial follow-up examination</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms of injured athletes</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>93.6</td>
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<tr>
<td>Dizziness</td>
<td>85.1</td>
</tr>
<tr>
<td>Confusion/disorientation</td>
<td>83.0</td>
</tr>
<tr>
<td>Nausea</td>
<td>53.2</td>
</tr>
<tr>
<td>LOC</td>
<td>25.5</td>
</tr>
<tr>
<td>Retrograde amnesia</td>
<td>13.0</td>
</tr>
<tr>
<td>Vomiting</td>
<td>4.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Initial follow-up examination</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>57.4</td>
</tr>
<tr>
<td>Cognitive impairment</td>
<td>55.3</td>
</tr>
<tr>
<td>Fatigue</td>
<td>44.0</td>
</tr>
<tr>
<td>Memory problems</td>
<td>37.2</td>
</tr>
<tr>
<td>Nausea</td>
<td>31.9</td>
</tr>
<tr>
<td>Concentration problems</td>
<td>29.8</td>
</tr>
<tr>
<td>Dizziness</td>
<td>23.4</td>
</tr>
<tr>
<td>Weakness</td>
<td>17.0</td>
</tr>
<tr>
<td>Irritability</td>
<td>17.0</td>
</tr>
<tr>
<td>Impaired vision</td>
<td>14.9</td>
</tr>
<tr>
<td>Sleep problems</td>
<td>14.9</td>
</tr>
<tr>
<td>Sensitivity to light</td>
<td>12.8</td>
</tr>
<tr>
<td>Depression</td>
<td>10.6</td>
</tr>
<tr>
<td>Nervousness</td>
<td>8.5</td>
</tr>
<tr>
<td>Vomiting</td>
<td>0.0</td>
</tr>
<tr>
<td>Other sensory problems</td>
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</table>

Abbreviation: LOC, loss of consciousness.

consciousness and other parameters appropriate for traumatic injury. If at any
time there is concern for a more immediately serious cerebral or spinal injury,
the athlete should be removed from the playing environment in a stable manner
by qualified personnel and transported immediately to a facility capable of
appropriate neurological and neurosurgical evaluation and treatment. Once
other potential or real injuries have been evaluated and managed, the athlete
should be removed from the contest and evaluated immediately and at regular
(ie, 5-minute) intervals for improvement or deterioration. Assessment should
include mental status testing for orientation, concentration, and perhaps most
important, memory, because amnesia has been reported to be an important
prognostic factor [14,15]. Sample sideline questions are listed in Box 2.

Neurological testing should include that of pupillary reflexes, extraocular
movements, cranial nerves, strength, sensation, coordination, and gait. Ab-

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**Box 2: Sample mental status assessment questions**

**Orientation**
- What is your name?
- What are you doing?
- Where are you/where are you playing?
- Who are you playing for?
- Who are you playing against?
- What year is it?
- What month is it?
- What day is it?

**Concentration**
- Spell “world.”
- Spell “world” backwards.
- What do a penny, a nickel, a dime and a quarter make?
- Subtract 7 from 100 (with serial subtractions to 65).
- Recite the months of the year in reverse, starting with December.

**Memory/amnesia**
- Repeat these three words (list three simple unrelated words).
- What just happened to you?
- What were you doing just before it happened?
- Were you winning or losing just before it happened?
- What was the score?
- What is the last thing you remember?
- Repeat the same three words again.
normalities in any of these domains of the neurological examination may herald the signs of a concussive injury. There are many standardized tools that exist for acute detection and assessment of concussion, but none supplant a thorough neurological examination, although many may be used adjunctively [9–12,17]. Repeated evaluation should continue until there is clinical improvement and stabilization. Off-field assessment should include formal neuropsychological and postural stability evaluation [9,17,18]. Neuroimaging may be reserved for athletes in whom structural lesions (ie, subdural hematoma) are considered. If a grading system such as that provided by the AAN [1] is used to rate severity of concussion, the particular system should be named and criteria met should be specified. The circumstances surrounding the injury, including time and place, type and direction of impact, reported symptoms, physical examination, and results of repeat testing and any provocative measures used should be documented.

Return to Play and Follow-Up

There is at present no universally accepted algorithm for follow-up evaluation and RTP decision-making. It is clear, however, that RTP decisions should be made upon an individual basis and should follow a standardized assessment, with reintroduction of physical exertion to evaluate persistence of symptoms [1,9,17,18]. Some groups advocate removal of the athlete from competition, with no RTP for the current contest [18]. Others allow for RTP if concussion is very mild and completely resolves at rest, as long as the athlete is assessed and gradually introduced to RTP by exertional provocative maneuvers over at least 10 to 15 minutes [1,9]. Otherwise, the athlete should be removed from the contest with no same-day RTP. Loss of consciousness (LOC) or amnesia should absolutely disqualify the athlete from same-day RTP; LOC and amnesia signify more severe concussion and longer symptom duration [14,15]. The practitioner should be comfortable removing an athlete from competition after any severity of concussion. If there is same-day RTP, the athlete should be frequently re-evaluated during the contest and for the next several days.

Athletes’ reported symptoms have been demonstrated to be accurate indicators of concussion severity [16] and have become the mainstay of RTP decision-making in many guidelines [1,9,18]. The Concussion in Sports (CIS) Group released its “Return to Play Protocol” in 2002 [18]. It entails a six-step process, described in Box 3, each step taking 1 day at a minimum. Athletes may progress a step once asymptomatic at the current level. Once asymptomatic with no activity, light aerobic exercise may be undertaken, then sport-specific training, then noncontact training, then contact, then game play. Elicitation of symptoms at any level necessitates a drop in step with reattempts to progress after 24 hours.

If possible, athletes should undergo formal neuropsychological evaluations as well, because this may unmask subtle continued deficits when compared with baseline testing. Such deficits have been shown to correlate with duration of symptoms [16]. This has become an increasingly important tool in concussion
evaluation. Postural stability testing may also be undertaken for adjunctive data in determination of concussion severity.

Symptoms, cognitive impairment, and postural impairment tend to resolve by day 7 postinjury [19,20], lending support for the 7-day sitting-out period necessitated by adhering to the CIS group RTP Protocol [18]. Previously concussed athletes tend to report fewer symptoms early after a concussion but more after 1 week, complicating their assessment [8]. There should be a very low threshold to withhold previously concussed athletes from play if there is suspicion for a new concussion. Ultimately, the decision to RTP is an individual one based on initial concussion severity, duration of symptoms, and repeated examinations, and assisted by formal neuropsychological testing and postural testing.

Long-Term Sequelae
Although cognitive impairment has been shown to resolve within about 7 days for most concussions [19,20], cognitive impairment has been shown to persist, particularly for athletes suffering multiple concussions. In one study, a single concussion did not correlate with long-term neuropsychological or cognitive complications, but multiple concussions resulted in long-term neuropsychological abnormalities, particularly in executive functioning and information-processing speed [7]. Moreover, athletes who have had previous concussions are more likely to have future concussions, and are more likely to have a longer recovery time per concussion [5]. Athletes who have a history of concussion should be treated more carefully; the NATA has recommended that athletes who have a history of more than two concussions and prolonged recovery time should consider retiring from contact sports either temporarily or permanently [9].

Emergent and Catastrophic Injury
Catastrophic injury has been defined in some epidemiologic studies as an injury occurring while participating in high school- or college-sponsored sport, and may lead to fatality, severe disability, or involve a severe injury but result in no
permanent disability [21,22]. Injuries may cause epidural hematoma (EDH), subdural hematoma (SDH), intracerebral hematoma (ICH), cerebral contusion, subarachnoid hemorrhage (SAH), or diffuse axonal injury (DAI). In high school and college football, 69% of reported fatalities between 1945 and 1999 were due to head injuries, with a further 16.3% attributed to cervical spine injuries [22]. Of fatal head injuries, 74.5% were caused by subdural hematomas [22]. Catastrophic head injuries have been reported in a number of sports, including track and field, baseball, cheerleading, gymnastics, softball, field hockey, volleyball, soccer, basketball, boxing, during weight training, and following aerobic and anaerobic exercise [22–28]. This is clearly a partial list, and the sports medicine practitioner must be prepared to evaluate and recognize potentially catastrophic injuries so as to properly triage the athlete, regardless of the sport.

Epidural hematomas frequently result from skull fractures with subsequent laceration of the middle meningeal artery. Classically, EDH leads to an initial loss of consciousness, which is followed by recovery of consciousness and a lucid period. The athlete then progressively deteriorates neurologically, with development of headache, a decline in mental status, and focal neurological findings such as contralateral weakness or numbness, pupillary reflex abnormalities, or facial asymmetry. Seizures may occur. The classic clinical progression, however, has been reported to develop in only one third of observed cases [29]. Diagnosis is usually made by CT scanning of the head.

SDH is thought to result from tearing of bridging veins running between the brain and dural sinuses. In the young, the classic large subdural fluid collection with local mass effect on underlying brain causing neurological deterioration is less common than in the elderly. More often, there is a small amount of subdural blood, but it is associated with underlying contusion, edema, and elevated intracranial pressure [29]. These patients may or may not have exhibited a lucid interval, frequently lose consciousness at the time of injury, and often exhibit focal neurological signs. Diagnosis is again usually made by CT scanning of the head.

ICH involves blood and hematoma formation within the substance of the brain parenchyma. A hemorrhagic contusion is similar, but is associated with more significant edema rather than localized hematoma formation. Loss of consciousness is reportedly rare; common clinical features include worsening headache, confusion, and persistent amnesia [29]. Diagnosis is usually made by CT scanning of the head.

SAH can result from trauma or intracerebral aneurysm rupture. Either can occur in the athlete. Traumatic SAH is related to direct arterial injury, whereas aneurysmal SAH may be spontaneous, but has been reported to result from varying forms of exercise, usually those associated with significant Valsalva maneuvers, such as weight lifting [27]. Diagnosis is made by CT scanning of the head or by lumbar puncture.

DAI is a severe form of closed head injury associated with acceleration-deceleration or rotational injuries that result in diffuse shearing injury to
neuronal axons. When severe, it is associated with focal abnormalities in the corpus callosum, dorsolateral rostral brainstem, and throughout the cerebral white matter, and may include intraventricular hemorrhage [30]. Diagnosis is suggested by CT scanning of the head, but may require MRI of the brain and longer-term observation.

On-field assessment should focus on recognition of the potential for catastrophic injury. It needs to be remembered that apparently significant initial neurological impairment, even with loss of consciousness, may be the consequence of a concussion, but more severe head injury may present with abnormalities that initially appear mild. Any athlete who suffers any kind of head trauma or acceleration-deceleration injury should initially be suspected to have suffered a closed head injury, and the possibility of cervical spine injury should be entertained as well, because the two are closely pathomechanically related. The athlete should be evaluated for level of consciousness and mental status and, if there is concern for cervical spine injury, undergo cervical spine stabilization with a hard cervical collar and spine board until cervical spine clearance can be obtained [31]. If the athlete is unconscious or exhibits an altered level of consciousness, cervical spine injury should be presumed and the cervical spine stabilized [30]. The athlete should be managed according to advanced trauma life support and advanced cardiac life support guidelines [30,31]. Once severe head or neck injury is considered to be a possibility, the athlete should be removed from the field of play after appropriate stabilization by qualified personnel, and transported immediately to a facility capable of appropriate neurological and neurosurgical evaluation and treatment.

CERVICAL NEURAPRAXIA (“STINGER”)
The stinger, or “burner,” was initially described in 1965 [32] and was eventually given the name “cervical nerve pinch syndrome” [33]. Incidence is reportedly near 50% in college contact football players [34], and has been shown to be the most common symptomatic upper extremity nerve injury in athletes [35]. The mechanism of injury may involve trauma to the brachial plexus or cervical nerve roots; it is not a cervical cord injury. Although most signs and symptoms are transient, some athletes may suffer prolonged symptoms or recurrent injury.

Injury Type, Pathomechanics, Symptoms and Signs
A stinger may be precipitated by a blow to the head, neck, or shoulder causing downward pressure on the shoulder on the affected side, with contralateral neck flexion or neck extension, such as in tackling or blocking in football. Although common in football, stingers can occur in other sports [32]. Site of injury has been localized to the upper trunk of the brachial plexus or the fifth and sixth cervical nerve roots. Proposed mechanisms of injury include traction injury to the upper trunk of the brachial plexus [34,36], compressive injury to the upper trunk of the brachial plexus [36–38], traction injury to the cervical nerve root [32,36,40], and compressive injury to the cervical nerve root in the neural foramen [39–42]. Brachial plexus injuries are more common in high
school athletes [40,43] and are more likely to result from lateral neck flexion with shoulder depression. Cervical nerve-root compression is more common in college and professional football players [40,43], particularly those who have recurrent or chronic stingers. It may be associated with cervical disk disease, cervical canal stenosis, and neural foraminal stenosis [40–42], and may result from neck extension or forceful lateral neck flexion. It is possible that any or all of these mechanisms may account for a particular athlete’s symptoms.

Symptoms involve a single upper extremity; bilateral or lower extremity symptoms should raise the possibility of a cervical spinal cord injury rather than a stinger syndrome. There may be rapid onset of burning pain in the shoulder [34,39], or from the neck [32] or supraclavicular area [33] down the arm to the hand [32,33,37,43] or fingers [39,44,45]. Paresthesias or anesthesia may occur throughout the upper extremity [32,33,39], but more commonly occurs in a C5/C6 dermatomal distribution [43]. Alternatively, there may be no sensory deficit, only pain [37,38]. The athlete may shake the arm [33] or exhibit a dropped shoulder [37,38]. Weakness following a stinger is variable, ranging from mild weakness in a myotomal pattern [43–45] to inability to move the entire arm [32,33], and the athlete may exit the contest supporting the affected arm with the contralateral arm [33,45,46]. There may be no evidence of weakness immediately after the injury, only to develop later [33,37]. Muscles commonly involved include the deltoid, biceps, supraspinatus, and infraspinatus [43,45], causing weakness of shoulder abduction, elbow flexion, and upper arm external rotation. Symptoms are usually transient, lasting only seconds to minutes. There is usually no associated neck pain or limitation of neck movement.

Assessment and Management

On-field evaluation begins with establishment of the mechanism of injury—either lateral flexion/shoulder depression or extension/compression. The athlete should describe the location of symptoms and their duration if they have resolved. Otherwise the athlete should be monitored for resolution of symptoms. The cervical spine should be evaluated by palpation for tenderness, edema, evidence of structural damage, or muscle spasm. The supraclavicular fossa should also be examined in like fashion. If there is no concern for cervical fracture, active range of motion within the limitation of pain should be assessed to include rotation, lateral flexion, anteroflexion, and extension. A full neurological examination should be performed, with specific attention to strength testing in all muscle groups, full sensory examination, and muscle stretch reflex testing. The contralateral extremity should serve as a normal control.

If symptoms or deficits involve the bilateral upper extremities or include one or both lower extremities, or if there is concern for other serious structural damage, the cervical spine should be immobilized and spine precautions instituted. The athlete should then be transported in a stable fashion by qualified personnel to a facility capable of appropriate neurological and neurosurgical evaluation and treatment.
If symptoms persist past several minutes, there is limitation of neck range of motion, or there is neck pain, the athlete should undergo MRI of the cervical spine [33,43,44] before any consideration of RTP. A high incidence of cervical disk disease or neural foraminal narrowing has been reported in athletes who have chronic or recurrent stingers [40]. Also, some authors have reported a correlation between cervical canal and neural foraminal stenosis and stinger occurrence [41,42], although its significance remains unclear [46]. Cervical spine MRI may reveal other pathology contraindicating return to sport [44,45], but these are more likely to cause transient quadriplegia than a stinger syndrome.

Electromyography (EMG) may be undertaken if abnormalities persist beyond 2 or 3 weeks [33,43,46]. Weakness persisting past 72 hours has correlated to positive EMG findings [36]. EMG assists with injury localization (nerve root versus brachial plexus), and determination of extent and severity.

Return to Play and Prevention
RTP decisions are somewhat controversial, although many authors rely upon clinical and electrodiagnostic criteria in the absence of clear radiographic abnormalities or contraindications [33,43,45–47]. If symptoms resolve within minutes, the athlete has returned to full strength and sensation, and there is no limitation of neck range of motion or increase in neck pain, the athlete may return to play in the same contest. In this situation, the athlete should be re-examined at least once during the current contest, then during each of the next 2 weeks [33] to assess for delayed onset of weakness necessitating further evaluation with neuroimaging or electrodiagnostics. In the event of persistent symptoms, EMG may play a role in RTP decision-making, but this is also controversial. Some recommend that fibrillation potentials in the setting of clinical weakness necessitate withdrawal from play [44–46]. If weakness persists, repeated studies are recommended. If there is resolution of fibrillation potentials or presence of only scattered positive waves and polyphasic motor unit potentials, the athlete may return to play [46]. This should, however, only occur if the athlete has attained full strength and sensation, with full neck range of motion and no neck pain [44,45]. Others have noted that clinical weakness and EMG abnormalities have not correlated [36], and recommend that the normalization of the physical examination and lack of contraindications to play on neuroimaging dictate RTP timing. The authors recommend that regardless of EMG findings, the athlete only return to play after clinical return to baseline, and after an evaluation that may include appropriate neuroimaging in the setting of prolonged symptoms. EMG is most helpful in the setting of a stinger to assist in localization and to assess changes over time, and to assess for changes in the setting of repeated injury.

One or two stingers, even in the same season, are not an absolute contraindication to RTP. Three or more stingers, especially within the same season or after implementation of equipment adjustment and blocking/tackling technique adjustment, should lead to a consideration of cessation of play.
Following a stinger, a comprehensive rehabilitation program to include neck and shoulder strengthening should be instituted [33,46,47]. High-riding shoulder pads and neck rolls with or without chest orthosis can be added to football uniforms to offer protection against all proposed pathomechanisms [33,38,43,47]. Furthermore, blocking and tackling techniques should be examined and proper technique taught to avoid dropping the head and shoulder [43,46,47]. Because athletes who have suffered a stinger have been shown to be more likely to experience another stinger [41,48], and repeated stingers may lead to long-term weakness and discomfort [36], prevention is essential after even a single injury.

SEIZURES AND EPILEPSY
Seizures and epilepsy have long been an area of debate concerning athletic participation. Historically, people who have epilepsy, particularly children, have been discouraged from sports participation or exercise on the basis that it may lead to seizure-related injuries, precipitate seizures, or worsen the person’s epilepsy [49–52]. Considering the long medical history of epilepsy, it is only relatively recently that restrictions on participation in certain sports have been eased explicitly [50,53–56]. There are reports of seizures associated with exercise [57–61], but as evidence grows that not only are exercise and sports participation rarely harmful to people who have epilepsy, but in many instances beneficial [52,58,60–66], it is likely that increasing numbers of people who have epilepsy will be active participants in sports, including organized sports. Accordingly, the sports medicine practitioner should have a basic understanding of seizures, epilepsy, seizure first aid, the relationship of exercise and sports to seizures and epilepsy, and participation recommendations.

Seizures, Epilepsy, and Classification
A seizure is a paroxysmal stereotyped event of acute onset resulting from hypersynchronous, rhythmic, neuronal discharges. A seizure may occur as a result of a specific insult to the brain, such as an electrolyte abnormality, alcohol withdrawal, or hemorrhage. A seizure may also occur due to an intrinsic characteristic of the brain that may or may not be immediately identifiable. Seizures have been systematically classified by the International League Against Epilepsy (ILAE) into partial, or focal, seizures and generalized seizures [67]. Partial seizures originate in a relatively small part of the brain, rather than involving both hemispheres at the outset as in generalized seizures. Partial seizures are classified according to clinical manifestations and whether or not there is an impairment of consciousness. Simple partial seizures do not alter consciousness, and may be motor, somatosensory/special-sensory (ie, hallucinations or smells), autonomic (ie, piloerection), or psychic (ie, déjá-vu). Complex partial seizures may or may not begin as simple partial seizures, but they do involve alteration, but not loss, of consciousness, either eventually or at the outset. They are usually followed by a short period of “post-ictal confusion,” after which the person returns to normal cognition, although with no memory
of the event itself. Partial seizures may also secondarily generalize when the focal neuronal discharge spreads to involve both hemispheres.

Generalized seizures originate in both hemispheres and necessarily lead to alteration and loss of consciousness. They may be absence seizures (ie, brief relatively motionless staring spells immediately followed by a return to normal cognition, usually occurring in children), myoclonic (ie, small isolated jerks), tonic-clonic (ie, generalized convulsions), tonic (ie, tonic limb muscle contraction), clonic (ie, jerking movements), or atonic (ie, sudden loss of muscle tone with falls) [67]. In all but absence and myoclonic seizures, the post-ictal recovery period may be more pronounced and prolonged than after a complex partial seizure. It is simple to see how various manifestations of seizures hold very different implications for different activities, particularly depending upon effects on level of consciousness and the ability to protect oneself and react to the environment.

A person who has a seizure does not necessarily have epilepsy. Epilepsy is a disease manifested as the tendency toward recurrent seizures. The ILAE has also classified the epilepsies according to syndrome type [68]. Epilepsy syndromes vary according to clinical and electrographic characteristics, as well as according to potential etiology. A full discussion of the epilepsy syndromes is beyond the scope of this article; please see the ILAE’s published classification for details [68].

Assessment and Management

Before an athlete who has epilepsy participates in sports, the sports medicine practitioner should become familiar with the athlete’s seizure history. The typical seizure type, clinical manifestations, frequency, duration, and post-ictal recovery characteristics should be known. Any history of the athlete having been in status epilepticus (ie, a seizure or series of seizures that did not stop) should be clearly understood. There should be some understanding of the adequacy of seizure control, use of anticonvulsant medications, and the athlete’s compliance with those medications. Many people who have epilepsy have known precipitants [69], and these should be known so as to be avoided if possible. The more knowledge held about an athlete’s seizures, the better equipped the practitioner will be if the athlete seizes.

If an athlete has a seizure, basic seizure first aid applies, regardless of whether the athlete is known to have epilepsy or not. Most importantly, medical personnel should stay calm and urge those around them to do so as well. As with any potentially unstable situation, attention should be given to basic life support principles—ensure adequacy of airway, breathing, and circulation. During a seizure, respiration can be compromised, and this should be monitored and treated appropriately. The athlete should be assisted to the ground in the event of a generalized convolution, potentially harmful objects should be moved away, the head cushioned if possible, and restrictive clothing, uniforms, or equipment should be loosened or, if necessary, removed. The athlete should not be actively restrained. The athlete should only be moved if in a potentially
dangerous place (ie, at the top of a staircase or at the side of a pool of water). Always prevent injury. Never place anything in the mouth or between the teeth of an athlete having a seizure—the tongue will not be “swallowed,” but teeth can easily be broken off by an object placed in the mouth and swallowed or aspirated. If the seizure is complex partial, the athlete should be attended to, but not forcibly interfered with unless entering into potentially harmful activity.

With an absence seizure, the athlete may persist in the activity he or she was engaged in at the onset of the seizure, and should be gently guided away from dangerous situations. Several characteristics of the seizure should be at least mentally recorded if possible. Seizure characteristics are listed in Box 4. Most seizures stop without intervention. Once the seizure has stopped, the athlete should be rolled to the side in case there is post-ictal vomiting. Nothing should be given by mouth until the athlete is fully alert. The athlete should be attended to for the duration of the seizure and recovery.

There are several situations that should prompt activation of emergency medical services and subsequent stable transport to a facility capable of neurological or neurosurgical evaluation. If it is the athlete’s first seizure of life, it needs to be evaluated for etiology and possible treatment. If the seizure is prolonged—lasting for more than 2 to 5 minutes—depending upon the athlete’s typical seizure duration if known, or if there are repeated seizures without recovery to baseline mental status, this meets criteria for status epilepticus and is a medical emergency requiring prompt treatment. If the athlete has been injured by the seizure or circumstances surrounding it, if there is any evidence of respiratory compromise following the seizure, or if anything is unusual about a seizure occurring in an athlete who has epilepsy, he or she should be evaluated more extensively. This is not an exhaustive list; if in any circumstance the sports medicine practitioner is uncomfortable or concerned, the athlete should

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**Box 4: Seizure characteristics to note**

- Activity at the time of the seizure onset
- Athlete premonition, aura, or warning of seizure onset
- Time of seizure onset
- Initial clinical manifestation of seizure (ie, focal twitching of the right arm versus generalized convulsion)
- Changes in clinical manifestation during seizure evolution
- Absence or presence of alteration of consciousness
- Presence of cyanosis
- Automatic activities (ie, lip smacking, playing with clothing)
- Seizure duration
- Presence of tongue biting or incontinence
- Mental state after seizure cessation
- Duration and character of post-ictal state
be evaluated by a physician knowledgeable and experienced in seizure management and epilepsy. Special note should be made regarding the head-injured athlete and seizures. Despite some evidence that head injury causes seizures, particularly in severe head injury, there is little to support the notion that sport-related head trauma (such as concussion) predictably leads to seizure onset or epilepsy [52, 70–72]. Nonetheless, there is evidence that the occurrence of a seizure up to a week after even mild head injury (ie, resulting in a Glasgow Coma Scale of 13 to 15) may herald intracranial pathology, including that requiring neurosurgical intervention, albeit in a low percentage of patients [73–75]. Following even mild head injury, new onset of seizures should lead to prompt evaluation and neuroimaging via CT scan or MRI to rule out catastrophic, treatable conditions.

Exercise, Seizure Exacerbation and Improvement, and Participation Recommendations

As stated above, there are reports of exercise exacerbating seizures and generating epileptiform electroencephalographic (EEG) activity [57–61]. In most series, however, these patients represent the minority of those studied [58,60,61]. Other studies have revealed a higher proportion of patients who experienced an attenuation of seizure frequency or epileptiform EEG changes with regular exercise [58,60–64,66]. It has even been shown that hyperventilation following muscular exercise does not lead to the induction of absence seizures as

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voluntary hyperventilation does [62,64]. Furthermore, positive psychological, social, and physical effects of exercise and participation in sports for patients who have epilepsy have been asserted and demonstrated [49,50,53–56,58,65,66].

Other than true exercise-induced seizures, exercise seems to have an overall positive effect for people who have epilepsy. Any decision to participate in sport, however, must be individualized. Age of the athlete, seizure type, seizure frequency, predictable seizure timing, anticonvulsant compliance, and sport of choice all factor into the decision to participate [50]. The ILAE published recommendations for the restriction of activities in children who have epilepsy in 1997 [50], and the only “activities that should be avoided” are scuba diving and sky diving. In their excellent review of current data, Fountain and May [52] outline several recommendations that are specific to particular sports and recreational activities. Full details can be found in the original article. A summary of their recommendations can be found in Table 3.

HEADACHE
Headache remains an extremely prevalent condition in the general population. One-year prevalence of all headache has been reported to be as high as 46% in women and 30% in men [76], with frequent headache of at least 180 headaches per year occurring in up to 4.1% of randomly selected people in one study [77]. Migraine headache alone has been estimated to have a 1-year prevalence of 6.0% in men and 17.2% in women in one survey [78], but up to 38.8% in women and 19.6% in men when pooling patients including criteria for both strict migraine and probable migraine [79] as defined under the revised International Classification of Headache Disorders (ICHD-2) [80]. Sport- and exercise-related headache has been reported in 35% of university student respondents [81]. Although the epidemiology of sport- and exercise-related headaches is less well defined, athletes are susceptible to the same maladies that affect the general population. Athletes also suffer from exacerbations of underlying headache disorders due to exercise, and are susceptible to headaches due to sports-related causes such as trauma and high altitude. Although it is not necessary for the sports medicine practitioner to be able to treat and prevent many of the primary headache disorders, it is necessary that the practitioner is able to assess the athlete who has headache, is able to recognize “red flags” indicating a potentially more serious condition, is familiar with some of the more common headache syndromes as classified in the ICHD-2, and knows when to refer an athlete to a specialist for evaluation, treatment, and prophylaxis.

Assessment
A complete history and physical examination, both systemic and neurological, begin the assessment of the athlete who has headache. The focus is not only on determining headache characteristics to aid in diagnosis, but also on delineating signs of possible secondary headaches, such as those caused by intracranial hemorrhage or other mass lesion, infection or inflammation, or substance abuse or withdrawal. Important headache history notes can be found in Box 5. The
physical examination should include, but not be limited, to: inspection for rashes or other signs of infection; cranial and cervical palpation for tenderness, trigger points, evidence of structural abnormality, and cervical range of motion; mental status, including assessment of level of consciousness and speech and language abilities; all cranial nerves, including ophthalmoscopy for fundoscopic examination; motor and sensory testing; muscle stretch reflexes; and coordination and gait. If at any time the sports medicine practitioner is concerned about any potential abnormalities in an athlete’s history or examination, a referral to a neurological specialist is warranted.

Of particular import are certain signs and symptoms that may denote serious underlying pathology as the etiology of an athlete’s headache. There have been few studies validating these red flags, but they stand as important warning signs. In one study investigating certain clinical features as predictors of abnormal neuroimaging, only paralysis, papilledema, and the symptom complex of drowsiness, confusion, memory impairment, or loss of consciousness were statistically significant predictors, although asymmetrical pupillary response and progressive visual or neurological changes also correlated. Presence of three or more red flags also showed a strong correlation to abnormal imaging [82]. Such indicators, however, do not only indicate the possibility of abnormal imaging; the presence of any red flag warrants not only neuroimaging, but also prompt neurological evaluation to assess for conditions that may

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**Box 5: Headache history**

Current headache
- Time of onset
- Acuity of onset
- Pain severity
- Pain location
- Pain character
- Preceding injury
- Associated symptoms
- Exacerbating and alleviating maneuvers
- Treatment attempts/medication use/drug use

Past history
- History of prior headaches
- Age of onset of prior headaches
- Characteristics of prior headaches (as above)
- Changes in headaches
- Past medical history
- Family history of headaches

Screen for red flags (see Box 6, below)
or may not be found on neuroimaging studies. A partial list of these red flag warning signs can be found in Box 6.

**Migraine Headache**

Migraine headache is common generally, and the same is true in athletes. There are many types of migraine classified by the ICHD-2 that may commonly occur in the athlete, such as migraine without aura, migraine with aura and its variants, and retinal migraine. Diagnostic criteria for migraine include at least five attacks lasting 4 to 72 hours (without successful treatment) with at least two of the four characteristics of unilaterality, pulsatile quality, moderate to severe pain, and aggravation with routine activity. Nausea with or without vomiting, or photophobia and phonophobia must be present. An aura is a reversible, focal, often visual neurological abnormality that occurs before or during the headache. The headache may not be attributable to another disorder. A retinal migraine consists of monocular vision changes followed by a typical migraine headache [80].

Migraine headache has many potential triggers, including menstruation, tannins, chocolate, aspartame, and stress [83,84], to name a few. Migraines have also been associated with exertion and trauma [84,85]. If known triggers exist, prevention focuses on avoidance or reduction, although this may not be realistically possible for the athlete who has exertion-exacerbated migraines. Certain nutritional adjustments or supplementations such as magnesium and riboflavin may be helpful [84,86]. Pharmacologic prophylaxis should be considered when headaches are interfering with activities, when two headaches per week require abortive therapy, when at least two headaches per month are not effectively aborted, or when abortive therapy cannot be used [87]. In the United States, common prophylactic medications include tricyclic antidepressants, beta-blockers, and certain anticonvulsants. Alternative regimens may include calcium channel blockers, selective serotonin reuptake inhibitors, and indomethacin [84]. Medications given to abort acute migraines include anti-
inflammatories, ergot derivatives, and triptans; some have reported utility of oxygen, lidocaine nasal drops, and isometheptene [84].

Tension-Type Headache
Tension-type headache (TTH) is probably the single most common primary headache type. Episodic TTH (ETTH) has been shown to have a 1-year prevalence of 38.3% in the United States, with chronic TTH (CTTH) having a 1-year prevalence of 2.2% [88], although some studies have rated it much higher. It may or may not be associated with pericranial tenderness. TTH may last from minutes to days, is generally bilateral, has a tight nonpulsatile quality, is mild to moderate in intensity, and is usually not exacerbated by routine activity. ICHD-2 criteria do not allow for nausea or vomiting, and allow photophobia or phonophobia, but not both [80]. In practice, TTH can be fairly severe and may involve nausea, photophobia, or phonophobia. Muscular tenderness to palpation over the head and neck is common. Non-narcotic, over-the-counter analgesics such as aspirin and acetaminophen have been shown to be effective in the acute setting [89,90], as have nonsteroidal anti-inflammatory drugs [91–93], which have also been shown to be equivalent to [94,95] or superior to [96,97] acetaminophen. Physiotherapy [98–101] and certain antidepressants [101–105] are useful in TTH prophylaxis. A recent well-constructed trial of botulinum toxin A, the first of its kind, did not find a significant effect on TTH [106], but this remains a controversial issue.

Post-Traumatic Headache
Headache is a common sequela of even mild head or neck injury, and a very common manifestation of concussion [16]. In a study of 443 high school and college football players, 85% reported experiencing a headache at some time as a result of hitting, 21% in the preceding contest; and only 16% of those experiencing headache overall informed a coach, physician, or trainer [107]. The true incidence of acute post-traumatic headache (PTH) is difficult to determine for this reason. The ICHD-2 indicates that for a headache to be considered acute PTH, it must have an onset within 7 days of any severity of head trauma (or of regaining consciousness), and must resolve within 3 months (or it is known as chronic PTH), but PTH has no typical characteristics [80]. Almost any headache type may result from head injury, but TTH is most common, occurring in up to 85% of patients [108]. The single most important consideration in acute PTH is to exclude other more serious pathology. This may require neuroimaging or neurological or neurosurgical evaluation. Treatment is focused on addressing any underlying pathology, and PTH is treated generally with prophylactic medication and abortive medication appropriate to the primary headache syndrome of which the PTH is reminiscent. Treatment may involve physiotherapy or biofeedback [108,109].

Primary Exertional Headache
Previously known as benign exertional headache, this headache has been described as being the most common type of exercise-related headache in athletes,
although not with strict criteria applied [85]. It is considered exclusive of migraine induced by exercise. It is precipitated by exercise, described as throbbing, and lasts 5 minutes to 48 hours. Upon first occurrence of such a headache, subarachnoid hemorrhage must be excluded [80] by CT of the head or cerebrospinal fluid analysis. When benign, it usually occurs in the young, typically in men, is less explosive, and is often bilateral. When associated with underlying pathology (usually due to subarachnoid hemorrhage), age of occurrence is older, there is no gender predominance, and the headache is explosive and persistent and is more reliably bilateral [110]. Indomethacin has been described as beneficial for the benign exertional form [111,112].

Cautions and Warnings
Headaches are extremely variable in presentation, characteristics, evaluation, and management. There is a vast array of headache syndromes, far beyond what can be addressed in this article. In addition to the few relatively common types described here, other headache syndromes may result from neuralgiform pain, vascular etiologies, inflammatory disorders, or structural abnormalities. Concern has even been raised that dietary supplements, herbal preparations, and sports energy drinks may lead to headache, sometimes as a symptom of intracranial hemorrhage (Bradford Worrall, MD, Charlottesville, Virginia, personal communication, January 2005).

Treatment can be complex, particularly in the athlete. A number of common headache abortive regimens and prophylactic medications can be found on the World Anti-Doping Agency’s Prohibited List for 2005 [113], although there is a process for therapeutic exemptions. Many headache medications have the potential for overuse as well, and this can lead to analgesic overuse, or rebound, headache, which can be quite difficult to manage and treat. Newer recommendations regarding the safety of certain nonsteroidal anti-inflammatory medications have only served to make treatment of the patient who has headache more difficult. Anyone treating athletes who have headaches should have familiarity with the medications being used, knowledge of the indications, effects, maximal doses, potential complications, and contraindications to usage. If at any time the sports medicine practitioner is not comfortable with the evaluation or management of an athlete who has headache, it is appropriate to refer the athlete to a specialist with experience in the field of headache management.

SUMMARY
Neurological conditions are relatively common in sports, either as a result of injury suffered during play (eg, head injury, stingers, or post-traumatic headache), or as a pre-existing condition such as epilepsy or common headache syndromes. Sports medicine practitioners need to be prepared to assess the injured athlete, appropriately triage the athlete according to possible severity of injury, and be aware of RTP issues. Moreover, they need to be sufficiently aware of the appropriateness of referral to a neurological specialist for evalu-
tion and management of certain conditions, and be comfortable with certain modes of management that such specialists may implement.

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