Concussion in youth athletes is a growing problem worldwide. During the past decade, significant progress has been made in standardization of the assessment of young athletes, and a growing appreciation of metabolic vulnerability, activity, and cognitive challenges has led to guidelines and suggestions for rest from the field as well as cognitive rest from school. Outcome data have begun to establish groups linked to symptom class, genetics, and sex who are at risk of worse outcomes from concussions. Decisions regarding return to activity are now based on at-rest symptoms, graded increases in activity, and neuropsychological testing. Using the case of Ms X, a 15-year-old otherwise healthy high school student who fell while skiing, evaluation, prognosis, and management of concussion are discussed.

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Concussion may be caused by many factors. In sports-related concussions, acceleration-deceleration processes typically cause the injury, although focal impact may play a role. At a neurophysiologic level, the forces associated with these processes result in the stretching and shearing of axons. Axonal stretching has important effects: it causes the pathological release of excess neurotransmitters, which leads to fluctuations in ion levels. These phenomena are commonly neurophysiologic distortions that cannot be seen on standard static brain imaging. The “ionic flux” that results from the disorganized neurotransmitter and ionic release in turn causes changes in the ion gradients across the membrane. Giza and Hovda have shown metabolic mismatch between energy needs and production in the neurometabolic cascade that occurs in vitro following an experimentally simulated and induced concussion. Cells typically recover from this multilayered neurometabolic cascade, although under certain circumstances a small number degenerate and die. These changes in metabolism and, thus, in blood flow play a crucial role in the pathophysiology of brain injury.

Metabolic changes enhance sodium-potassium pump activity, increasing cell glucose use in the face of unchanged cerebral perfusion, creating a mismatch between need for and availability of glucose. Vespa et al used positron emission tomography to demonstrate that in concussion, a “metabolic crisis” can occur without concomitant brain ischemia. Thus, a period of metabolic vulnerability exists that relates to physiologic function and clinical care.

Within a few days to weeks of initial concussion, depending on severity, neurophysiology begins to return to normal. Ionic imbalances begin to resolve, energy use and metabolism begin to normalize, blood flow is restored to acceptable levels, and axonal functioning and neurotransmitter release revert closer to normal levels. Although clinical practice has assumed that the course of metabolic stress is reversed in 1 to 2 weeks, magnetic resonance spectroscopy, electrophysiologic data, and neuropsychological assessments suggest a more prolonged timeline, with physiologic metrics returning to baseline after 30 to 45 days as measured by magnetic resonance spectroscopy. In addition, the developing brain differs from the adult brain regarding factors such as degree of myelination, brain/water volume, elastic properties, and blood-brain barrier properties and may be more vulnerable to a challenged recovery, in part related to less well-established sets of cognitive and motor skills.

In one study, N-acetylaspartate levels measured through spectroscopy were significantly lower for a mean of 15 days after concussion, compared with control patients, and returned to normal after 30 days. Lovell et al found that if “hyperactivation” was seen on the initial postinjury functional magnetic resonance image (fMRI), the athlete took longer to achieve a full recovery, suggesting that longer periods of clinical rest and observation may be needed.

AT THE CROSSROADS: QUESTIONS FOR DR ZAFONTE

What is the epidemiology of concussion associated with sporting activity? How should patients be evaluated after a fall and a loss of consciousness (LOC)? After a first concussion, how should patients be treated and counseled? When can patients like Ms X return to athletic activities? What is the prognosis after a first concussion? Which patients should be referred to neurologists after first concussion? If Ms X has a second concussion, what is her prognosis and what are the long-term effects? How can head injuries be prevented in adolescent athletes? What do you recommend for Ms X?

Definition and Pathophysiology of Concussion

Dr Zafonte: Concussion is a complex pathophysiologic process induced by external traumatic forces and capable of long-term functional disturbance. The term concussion describes the mild end of the traumatic brain injury (TBI) continuum. Typically, concussion results in rapid-onset neurophysiologic and neurologic dysfunction that resolves in a spontaneous manner over a relatively short period. Sports-related concussion is generally due to functional or metabolic rather than structural dysfunction, and standard neuroimaging results usually appear normal. Concussion can be graded by clinical manifestations and does not require LOC. Symptoms of concussion may include headaches, fatigue, mood swings, neck pain, nausea and vomiting, dizziness, blurred vision, balance difficulty, photosensitivity, phonosensitivity, cloudiness, difficulty concentrating, memory problems, slowed reaction times, cognitive impairment, confusion, drowsiness, insomnia, emotional lability, irritability, anxiety or nervousness, sadness or depression, and amnesia. Although most patients with concussion improve rapidly, a small percentage (approximately 10%-20%) of those with sports-related concussion may remain symptomatic, particularly children and adolescents.
Epidemiology of Concussion Associated With Sports

While motor vehicle crashes cause about 40% of mild TBI in the United States, sporting-related incidents cause a large and increasing proportion.14 However, many injuries are not reported, especially those that do not result in LOC and do not involve a visit to the emergency department. Many concussions are first identified by coaches or family members. Although experienced coaches can recognize signs of concussions, some misconceptions prevail and many coaches believe LOC is necessary for a concussion.15-17

In the United States, skiing, horseback riding, and football appear responsible for the greatest number of injuries,16 while wrestling, rugby, and soccer are more rare causes. According to the Centers for Disease Control and Prevention (CDC), the most frequent nonfatal sports-associated TBIs that resulted in an emergency department visit (from highest to lowest) were bicycle crash injuries followed by football, basketball, playground, soccer, baseball, all-terrain vehicle, hockey (all types), skateboard, swimming, and horseback riding injuries.19 Skiing was not included in that report. Patients aged 10 to 19 years visit emergency departments with a diagnosis of TBI more frequently, at a rate of roughly 350 per 100,000 visits. In the NCAA Concussion Study,18 98.6% of concussions were mild and 77.8% of concussions resulted in LOC or amnesia. In skiing, as in the case of Ms X, head injuries are responsible for only 3% to 15% of all injuries but are responsible for 50% to 88% of all skiing-related fatalities.20,21

Risk of concussion is associated with age, sex, and possibly some genetic traits. Field et al22 found that high school athletes took longer to recover based on neuropsychological testing compared with college athletes. Female sex is associated with greater risk and symptom severity.23 The apolipoprotein E ε4 allele has been associated with a greater risk of and more severe concussion.24 A cross-sectional multicenter investigation found a 3-fold greater risk of concussion in athletes with the APOEε4 promoter G-219T TT genotype.24 Another study found no increased risk but did find worse outcomes associated with the gene.25 Other associated genotypes have been implicated, including COMT, DRD2, ACE, COMT, CACNA1A, and p53, but none as yet conclusively.20,27

Evaluation After a Fall and LOC

Numerous concussion guidelines and criteria are available. The most prominent of these, the consensus statement from the International Conference on Concussion in Sport, was most recently updated in Zurich in 2008 and addresses the proper identification, care, and treatment of concussion injuries. The consensus statement suggests that clinicians categorize concussion by severity of presentation rather than by arbitrary grade.2 A revised version of the Sport Concussion Assessment Tool (SCAT2) was developed; posttraumatic amnesia, altered sensorium, and LOC are all included as significant metrics. Most researchers agree that LOC is not a requirement for a concussion, although at least 1 prominent case study asserted otherwise.18 According to the Zurich guidelines, a player who shows any features of a concussion should be evaluated by a health care professional on-site using a concussive injury tool such as SCAT2. Cervical spine injury should be excluded. If no health care professional is available, the player should be removed from practice or play and urgent follow-up with a health care professional arranged. The player should be observed and monitored for possible deterioration for several hours following injury. A youth or adolescent player with a concussion should not return to play on the day of the injury.2

The CDC concurs that coaches should not evaluate players for concussion; the coach’s role is to remove the athlete from play, ensure that the athlete is evaluated by a health care professional, inform the athlete’s parents or guardians about possible concussion, and keep the athlete out of play on the day of injury and until a health care professional states that the player is symptom free and may return to play. The coach should note important information for the health care professional including the cause of the injury, the force of the hit, any LOC and for how long, any memory loss or seizures immediately following the injury, and any previous concussions.28

The evaluation of concussion first focuses on the initial on-field/on-slopes or “sideline” evaluation. Evaluation typically involves a questionnaire testing memory and orientation, along with various physical maneuvers. Additionally, it is important to determine the presence or absence of LOC, posttraumatic amnesia, or retrograde amnesia. The 2 widely used formal systems of on-field clinical assessment are the Standardized Assessment of Concussion and the SCAT (versions 1 and 2).18,20 The SCAT2 incorporates exertional and balance testing. Patients with concussion are recommended to adopt “cognitive rest” from daily activities before undergoing graded exertion to return to play in a conservative manner. Clinicians should also use balance testing as a tool to objectively measure postural stability. However, neurological evaluation is often normal. Easily ascertainable stigmata of neurological deficit, such as cranial nerve abnormalities and focal neurological deficits, are more typically seen in cases involving multitrauma-related hemorrhage than in sports-related concussion.30

The patient history is most important because concussion is often self-reported; it is most useful when obtained as soon as possible after the initial injury. Relevant history includes prior concussion, headache history, and a detailed history of academic and athletic performance. Headache history is important because patients with posttraumatic migraines have been noted to experience increased neurocognitive dysfunction compared with those without headache.31-34 As with the sideline evaluation, clinicians can use a formal postconcussion history scale.30 Clinicians should assess for LOC, defined as the patient’s eyes being closed and the patient being unresponsive to external stimuli.
The presence of LOC is also considered posttraumatic amnesia (PTA) for the duration of the LOC. Ms X had LOC and PTA with her head injury; she does not remember what happened or how she fell, no witnesses were available, and the length of her LOC is not known.

Posttraumatic amnesia may have prognostic importance. Collins et al found that athletes with poor postinjury outcomes were 4 times more likely than those with good postinjury outcomes to have exhibited PTA. Brief LOC appeared to be less relevant. However, other studies have challenged the statistical significance of the correlations involving PTA because the relationship with duration of symptoms did not appear to be as robust.36-38

Cognitive and neuropsychological testing, including several formats of computerized testing, are important aspects of the evaluation. One commonly used test is the Immediate Post Concussion Assessment and Cognitive Testing (ImPACT). Athletes in high-risk sports have a baseline measurement taken in the preseason, then undergo subsequent event-related testing, each individual serving as his or her own control.39 According to Van Kampen et al, neuropsychological testing via ImPACT added 19% to 29% to the sensitivity of clinical examination for the diagnosis of concussion or mild traumatic brain injury. However, the reliability of assessments of verbal learning and memory in younger athletes40 and even in collegiate athletes may be suboptimal.41 In an investigation of the test-retest reliability of 3 primary concussion assessments, Broglio et al found a lower reliability than previously determined (intraclass correlation coefficient estimates from baseline to day 45 assessments ranged from 0.15 to 0.39 for the ImPACT, 0.23 to 0.65 for the Concussion Sentinel, and 0.15 to 0.66 for the Concussion Resolution Index) and suggested that clinicians be cautious in their interpretations of computerized assessments of concussion. Of interest, Lau et al recently demonstrated that identifying a series of symptom clusters (migraine, cognitive, sleep, and neuropsychiatric) combined with neuropsychological testing enhanced the ability to predict which patients will have a prolonged recovery.

The physical examination involves assessment of oculomotor function, which may be disturbed after concussion42; balance assessment, such as with the Balanced Error Scoring System43; and exercise-related stress testing. In the latter case, an athlete undergoes exercise-related stress using a bicycle, treadmill, or stair climbing when cognitive, headache, and neurobehavioral symptoms have resolved. If the athlete is able to withstand these activities at progressive degrees of difficulty (related to a percentage of maximal heart rate) without symptoms, then return to sport-specific activity and eventual play may be considered.44 Recently, Leddy et al have advocated that the Balke protocol has strong retest and interrater reliability in the evaluation of physiologic symptom exacerbation after concussion. Many other systems of various and increasing complexity (both human- and computer-based) can be found in the literature.45-48

Management of First Concussion

Although no prospective clinical trial has defined proper treatment, the consensus of experts suggests that after concussion, an athlete should undergo physical and cognitive rest until asymptomatic. Based on the Zurich guidelines, initial treatment entails avoiding all stimuli requiring concentration, including playing video games, text messaging, online browsing, schoolwork, and extracurricular activities.45 While watching television has been acknowledged as a possible stressor by some practitioners, there are few data on this subject; thus, whether to recommend avoiding television is up to the individual clinician. Physiologic stress (eg, high altitude) and alcohol consumption should also be avoided. In adults, a program of early education intervention has been shown to produce fewer symptoms and less stress among the treatment group 3 months after mild traumatic brain injury (eAppendix 1; available at http://www.jama.com).49

After symptoms fully resolve, an athlete may return to a trial of activity per a graded protocol of exertion with 24 hours in between each stage, subsequently followed by medical clearance prior to allowing the athlete to return to play. The graded protocol comprises 6 stages from no activity to return to play, in which athletes can proceed to the next level only if they remain asymptomatic for a predetermined duration. If they become symptomatic, then they must return to the previous level. During this testing period, they should not take medications that can alter the concussive symptoms.46

Management of postconcussive symptoms is based on the particular symptoms. Most athletes recover and will be symptom free and, therefore, do not require pharmacological management. Sleep hygiene is helpful; however, some may experience persistent symptoms. The evidence regarding specific medications in patients with concussion is limited and no standard recommendations exist.50 In the case of Ms X, posttraumatic headaches remain a concern. Posttraumatic headaches commonly have been treated with nonsteroidal anti-inflammatory drugs. However, some clinicians are concerned about the potential for headaches due to medication overuse.51 For persistent posttraumatic migraine-type headaches, abortive-type drugs such as sumatriptan and prophylactic medications such as amitriptyline may help with sleep, or propranolol may be useful.52 Valproic acid, other anticonvulsants, antidepressants, and calcium channel blockers have been used in some cases. For tension-type headaches, antidepresants and rare use of muscle relaxants have been considered by some practitioners.53-56 Other strategies include behavioral therapy, biofeedback, relaxation, anesthetic nerve block, and avoiding overuse of medications to prevent potential headache, tolerance, or dependency.57 For persistent postconcussive cognitive symptoms, cognitive rehabilitation is somewhat controversial, but a systematic review of 17 randomized controlled trials suggested that cognitive behavioral therapy may be effective in the treatment of postconcussive symptoms.58
**Prognosis After First Concussion**

Concussion can result in altered function, and prognosis relates to severity, initial management, and biological profiles that have yet to be completely defined. In animal models, a single concussion is associated with behavioral and subcellular alterations that can be exacerbated for several days. Lovell et al demonstrated a correlation between IMRI profile, neuropsychological testing, and clinical course in high school athletes (28 patients with concussions and 13 control patients). The presence of “hyperactivation” on IMRI 1 week after injury correlated with slower recovery. Thus, there appears to be a period of biological alteration after exposure to concussion. Vagnozzi et al demonstrated a period of prolonged metabolic vulnerability that appears to have cleared 30 days after injury using magnetic resonance spectroscopy. Data regarding the exact duration of physiologic change after adolescent concussion is lacking, but first-time concussion is usually a “good news story” when managed properly. Although some 90% of those with concussion show clinical neuropsychological deficits 1 day after injury, Collins et al and others demonstrated that most with a single concussion recover to baseline within 7 to 10 days and approximately 80% recover within 3 weeks. Ms X describes her symptoms as having been worst in the first month after her injury and subsequently resolving slowly, as expected.

Patients with posttraumatic migraine and cognitive symptoms have a more guarded early prognosis. Thus, Lau et al suggested that specific symptom complexes may have prognostic value when evaluating recovery from concussion. Iverson et al identified the symptom of “fogginess” as a risk factor for poorer neuropsychological performance after concussion. While Ms X’s headache pattern does not fit the pattern of posttraumatic migraine, the presence of headaches and cognitive symptoms in her case predicts a longer recovery period.

Fazio et al reported that neuropsychological deficits persist in many patients after other clinical symptoms have resolved. Activity level after concussion appears to affect symptoms, with those engaging in high-intensity activity having worse neurocognitive performance and reporting worse symptoms. Thus, in Ms X’s case, the early recommendation for rest was appropriate and should include both physical and cognitive rest. No athlete should return to play while still experiencing symptoms. In such cases, careful interaction with the parents and school are necessary to provide appropriate accommodation. While early rest makes both physical and metabolic sense and is the standard of care, some controversy does exist, as McCrea et al reported that the symptom-free waiting period did not intrinsically influence clinical recovery or repeat concussion risk.

The Zurich conference report includes a specific set of guidelines for return to play; these guidelines are based on observational data and consensus expert opinion. In this activity-based algorithm, if an athlete is asymptomatic at one “level” of activity, the athlete may then proceed to the next level, until he or she is finally ready for “game play or activity,” which is the sixth and final step. In all cases, return to sporting activity must be preceded by a resolution of all residual clinical symptoms and a lack of symptoms with exertion testing as well as a normalization of neuropsychological testing data.

**Prognosis After Second Concussion**

Most data regarding second concussion exposure are from contact sports rather than skiing. Those exposed to a second concussion during the so-called vulnerability period (when the athlete is still symptomatic from the first concussion) may be at risk of second-impact syndrome; while rare, second-impact syndrome can produce devastating consequences.

Second-impact syndrome is defined as a second concussion that occurs soon after the initial concussion, resulting in profound brain swelling and vascular dysregulation. The incidence of this syndrome is difficult to measure precisely, but at least 17 deaths have been documented in the literature and it is considered to carry a high mortality (approximately 50%) and morbidity (approximately 100%).

In a study of recurrent concussions and their cumulative effects in collegiate football players, players with multiple concussions were found to have slower recoveries from subsequent head injuries. More specifically, 30% of players with 3 or more previous concussions experienced symptoms that lasted more than 1 week.

An athlete with a history of concussion appears to be at increased risk of a second concussion by a factor of 3 to 5. Collins et al found that high school athletes with a self-reported history of 3 prior concussions were more than 9 times more likely than those with no previous concussion to exhibit 3 or 4 on-field abnormal markers of injury when they experienced a subsequent concussion. The cumulative risk of multiple concussions, especially more than 3 concussions, may represent a distinct risk factor for depression and neurocognitive sequelae. Many believe, however, that in addition to the number of concussions, the time between concussions and the management of the concussions is important. Guskiewicz et al found that athletes with concussions had significantly worse balance scores than athletes in the control group. Cognitive testing between the 2 groups did not yield as clear a difference, although mild defects in “concentration, working memory, immediate memory recall, and rapid visual processing” were seen acutely. In a study of soccer players, athletes with a history of concussion scored worse on neurocognitive assessments and also reported more symptoms. These results were seen to a greater degree in female than male athletes, despite similar body mass indexes.

In contrast to the findings that adverse consequences of concussion persist were the results of a prospective study.
by McCrea et al, in which 1631 players completed a graded symptom checklist, baseline neuropsychological tests, cognitive assessment, and posturography. Players with concussions were found to have more significant symptoms, a greater degree of neurocognitive problems, and more difficulties with balance than the control players. However, by 90 days after injury, there were no significant disparities between the injured patients and the controls.

While not a focus of Ms X’s case, another problem associated with multiple concussions is chronic traumatic encephalopathy/posttraumatic encephalopathy, which can cause cognitive, motor, and behavioral problems. There is also a risk of mild cognitive impairment, which as a tauopathy shares some pathological features with Alzheimer disease. A genetic predisposition may place athletes at higher risk of this phenomenon. In fact, several former National Football League (NFL) players have some occult manifestations of dementia.

In a prospective cohort study, Guskiwich et al found that 1 in 15 players with a concussion may have a second head injury in the same season and may have a slower neurological recovery period. Compared with noninjured controls, patients with 3 or more concussions were 5 times more likely to experience mild cognitive impairment and 3 times more likely to experience memory deficits. Furthermore, retired football players appeared to have an earlier onset of Alzheimer disease compared with male patients in general; however, this was not found to be associated with a history of multiple concussions.

A study conducted at the University of Michigan found that in a population of 1063 retired NFL football players who had played 3 or more seasons, based on a cross-sectional questionnaire, the prevalence of a dementia-related diagnosis in retired players older than 50 years was 6.1%, which is 5 times greater than the national average (1.2% in US men of the same age). Retired players with traumatic brain injury had a significantly higher risk of developing dementia. In men aged 30 to 49 years, the retired NFL players had a 1.9% rate of dementia compared with a 0.1% rate in the general population of US men of the same age.

De Beaumont et al found that athletes sustaining concussions in early adulthood, compared with controls, performed worse on neuropsychological tests of memory and response and exhibited bradykinesia, delayed P3a/P3b components of motor-evoked potentials, and prolonged cortical silent periods after single- and paired-pulse transcranial magnetic stimulation, with deficits persisting for 3 decades after concussion. The eTable (see also eAppendix 2 for review search criteria) describes studies of long-term sequelae following minor traumatic brain injury.

Preventing Head Injuries

Helmets are now standard in both skiing and contact sports such as hockey and football, at all levels, although Ms X was not wearing a helmet at the time of her fall. Sulheim et al noted that helmet use was associated with a reduced risk of head injury among snowboarders and alpine skiers. This perhaps should be extended or standardized for other sports. However, adequate structural integrity may be required to deliver the helmets’ protective effect. Novel helmet designs carry promise and may allow better displacement and assessment of the forces delivered but as yet are not proven to prevent injury or substantially dampen its effects. An assessment of adolescent soccer players found that headgear use was associated with a lower incidence of concussion (53% with no headgear vs 27% with headgear). Female athletes had a greater percentage of concussions. Rule changes, such as prohibitions against “leading with the head” or “spearing” in football, must also be considered.

Data from ice hockey, in which hitting is permitted, suggest that the degree of neck muscle development also may represent a significant risk.

RECOMMENDATIONS FOR MS X

Ms X’s physician appropriately prescribed physical and cognitive rest as first-line treatment. This rest should preclude any stressful school-based or other activities. Additional treatment involves adequate hydration, avoidance of excess medications that could result in rebound symptoms, and normalization of sleep patterns. Fortunately, if early rest is pursued, most individuals’ symptoms resolve in the first 3 to 4 weeks. In my experience and based on the data above, Ms X should expect excellent recovery. If she has persistent postconcussive symptoms, a program of slowly progressive exercise may benefit her. The long-term risk of multiple concussions and contact sports should be reviewed with Ms X and her family, and emphasis on proper technique and protection should apply to all sports. In sum, while progress has been made in the understanding of the course of recovery from concussion, evidence for treatment and novel diagnostics is as yet inadequate and, aside from ensuring ample time for recovery and avoiding additional injury, management of concussion continues to be based largely on expert opinion.

QUESTIONS AND DISCUSSION

QUESTION: You suggested that high schools perhaps institute baseline testing for athletes, so that they could do follow-up testing if any of the athletes had a head injury. How much does that testing cost per person? Is it easily available? And, if you had limited resources, are there specific sports that you can identify as being most important to think about in terms of that?

DR ZAFONTE: I think it is critical in high school, and it is now mandated by the National Collegiate Athletic Association. It is currently our best measure to evaluate if an athlete is back to baseline. Although flaws exist even in computerized testing, it may still add as much as 29% to the diagnostic yield of clinical examination. The expense is not

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prohibitive for most school districts and the importance is clear; however, if financial constraints are the issue, schools should use it where the yield appears highest: in football, ice hockey, lacrosse, and wrestling.

**QUESTION:** I've heard about a very sophisticated helmet that's now used in Division I colleges. Coaches are able to monitor head impacts on the field, even if injury is not reported by the player. Do you know about that helmet and how widely used is it?

**Dr ZAFONTE:** Several studies are under way evaluating pressure sensors and accelerometer data regarding force of impact and rotational concerns in football. The question would be: Can one say a certain force produced an injury in a certain person who is completely asymptomatic? The Summation of Tests for the Analysis of Risk (STAR) equation relates field exposure to a series of 24 drop tests performed at 4 impact locations and 6 energy levels; such technology is being incorporated into helmets to integrate head exposure.84 A recent article has suggested that detection of a high number of asymptomatic head blows was associated with demonstrable deficits in visual working memory and neurophysiologic change.1,12 Or, can this be used to screen individuals in a more intense manner? In addition, can such information be used to design helmets that distribute force in a more elegant manner, especially if high-risk regions of injury are able to be identified? But, one thing I am not sure is possible is to design a concussion-proof helmet. A critical complement to the helmet is education. Young athletes often focus on returning to the playing field, so underreporting of symptoms is a concern. Part of this is educating players about the potential deleterious effects of head injury. Young athletes need to learn that if they do not get proper rest, they may be affected both in sport and in academic work.

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**Online-Only Material:** eAppendices 1 and 2 and the eTable are available at http://www.jama.com.

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