

Concussion in Athletes

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Annotation: Treatment of the “ding” or “bellring” complaint continues to involve tough decision-making. Combined multispecialty research-based guidelines have now evolved to permit better handling of return to contact during practice and formal competition. Dr. Terrell brings first-hand experience and current evidence-based criteria and discussion to the reader.

Concussion or mild traumatic brain injury (MTBI) is a major sports medicine issue in the current medical literature and in the popular media. Well-publicized cases of careers ended by the results of multiple concussions as well as the potential for long-term neurocognitive deficits due to multiple concussions have raised concerns. This review will discuss the epidemiology, anatomy, and physiology of concussion, as well as the clinical evaluation, grading scales, risk of chronic traumatic brain injury, and research on genetic risk factors for concussion. The main focus of this article is to review the clinical evaluation and management of concussion, including return to play criteria and use of neuropsychological testing for the practicing physician.

An estimated 300,000 sports-related traumatic brain injuries of mild to moderate severity occur in the United States each year.¹ Football, ice hockey, soccer, and rugby are sports with the highest concussion prevalence. Some studies show that up to 15% of people diagnosed with MTBI experience persistent disabling symptoms.^{2,3}



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Definition

Various definitions of concussion have been reported in the literature. Cantu defined concussion as “a traumatically induced alteration of mental status.”⁴

The definition that most completely describes concussion was developed by the First International Conference on Concussion in Sport.⁵ However, all definitions have so far failed to adequately incorporate postconcussion symptoms.⁶

This Vienna conference defines concussion as “A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Concussion may be caused by either a direct blow to head, face, neck, or elsewhere on the body with an ‘impulsive’ force transmitted to the head. It typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously. It results in a graded set of symptoms that may or may not involve loss of consciousness. Resolution of clinical/cognitive symptoms typically follows a sequential course.”⁵

Concussion is characterized by a number of postconcussion symptoms, including headache, nausea, lightheadedness, amnesia, and confusion. Other symptoms are listed in Table 1. Headache may occur normally in football players following practice. There is some controversy about whether this is a normal posttraumatic phenomenon or represents a subconcussive event.⁷ Some studies have classified all injuries in which an athlete complains of headache as a “concussion” since it is a transient alteration of mental status that in itself can cumulatively lead to “subconcussive trauma”.

Anatomy

MTBI in animal studies is diffuse in nature,⁸ features little overt histologic damage,⁹ and involves no ischemia or hemorrhage.¹⁰ Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.^{5,6} This differentiates a concussion from moderate or severe TBI (ie, subdural hematoma). Diffusion-weighted magnetic resonance imaging (MRI)—largely a research tool—has shown abnormalities in frontal locations and in the corpus callosum in up to 20% of concussed subjects.¹¹

Physiology

Immediately following concussion, an alteration of the normal ionic fluxes in the cell and a decrease in cerebral blood flow and substrate delivery leads to “hyperglycolysis/cerebral

Table 1. Common symptoms of concussion

Headache
Amnesia
Loss of consciousness
Blurred vision
Nausea
Attention problems
Dizziness
Drowsiness
Balance problems
Sleep disturbance
Photophobia
Lethargy
Emotional lability
Irritability
Vacant stare

hypoperfusion mismatch."¹² As this mismatch corrects, clinical symptoms abate. Single photon emission computed tomography (SPECT) and positron emission tomography (PET) may show defects in brain physiology that are not identifiable as structural lesions on traditional CT or MRI scanning.¹³

Sideline Evaluation

Certainly, one of the most critical roles of the covering physician is proper evaluation of potential head and cervical spine injuries. Agreeing upon head injury management protocols with the staff before the game is critical.

Concussions sometimes go unrecognized by staff and are often underreported by athletes. A study of professional football players showed that 80% did not recognize that they had had a concussion.¹⁴ Thus, it is important to maintain a high index of suspicion for concussion, while educating the athlete, athletic trainer, parents, and coach about its signs and symptoms.

The sideline examination is an effective way to evaluate an individual who has sustained a head injury during an athletic contest. If the environment and location do not allow for an appropriate examination, then the athlete may be examined in the locker room or in another suitable facility.

The mechanism of injury assists in determining the potential severity of the head injury. Contact with another athlete, the playing surface, or some sport-specific part of the playing field (eg, soccer goal post) are the most common mechanisms for a concussion. In soccer, head to head contact is the most common mechanism.¹⁵

The typical presentation of a concussion is characterized by a period of confusion, amnesia, and possible disorientation. The amnesia may be posttraumatic or anterograde and of varying length. Posttraumatic amnesia is defined as "a partial or total loss of the ability to recall events that have occurred during the period immediately preceding brain injury."¹⁶ An-

terograde amnesia is a deficit in forming new memory after the injury ("do you remember being evaluated after the injury?").

First, it is important to carefully evaluate whether a major form of blunt head trauma such as an epidural or subdural hematoma has occurred. A subdural hematoma classically presents with loss of consciousness (LOC) and focal neurologic abnormalities. An epidural hematoma has transient LOC followed by a "lucid" interval. Unilateral pupillary dilation and subsequent decerebrate posturing and contralateral weakness are present. These cases require emergent transfer.

The athlete who sustains a concussion may have a very obvious presentation, with a directly observed blow to the head, loss of consciousness, and amnesia. A large number, however, are more subtle. Some individuals may experience "seeing stars" or "hearing bells ring" for a few seconds to a minute; this has been termed a "ding" in the past and considered by most as a subconcussive impact.

Table 2. Sideline evaluation of concussion^a

1. Initial evaluation—Assess "ABC's." Assess general level of consciousness, mental status
2. Clinical history
 - A. Mechanism of injury
 - B. Obtain some clinical history from observers (ATCs, teammates, parents)
 - C. Assess for concussion if head, face, or neck injuries present
 - D. Prior concussion history—inquire about previous symptoms of concussion (individual may never have been diagnosed with a concussion) and number of concussions.
3. Evaluate for symptoms (see Table 1)
4. Physical exam
 - A. Neurological exam—assess pupillary reflexes, cranial nerves, motor strength; *perform gait/station tests for balance (Romberg)*
 - B. Mental status
 1. Attention span (immediately repeat *four* numbers forwards; at 2 minutes, repeat months of year backwards or repeat *five* digits forwards and backwards),
 2. Short-term memory (*three* objects at 5 minutes)
 3. Assess for posttraumatic (retrograde) amnesia
 - i. "What team are you playing?"
 - ii. "What is the score of the game?"
 - iii. "What are the colors of the uniforms of the team you are playing?"
 4. Assess for anterograde amnesia
 5. Assess long-term memory
5. Neuroimaging
 - A. Emergent CT scan—if prolonged loss of consciousness or signs/symptoms concerning for epidural/subdural hematoma;
 - B. Nonemergent CT scan/MRI—if post concussion symptoms persist *two* weeks after injury or are worsening. Use your own clinical judgment.

Important Note: The evaluation presented is a suggested format. It does not represent a standard of care or clinical guidelines. It has been developed through review of the recommendations in the literature and from the experience of the author.

Table 3. Referral

Indications for acute injury referral ^a	
Evidence of a subdural or epidural hematoma	
Suspected cervical spinal injury	
Deteriorating level of consciousness	
Focal motor weakness not suspected to be a "stinger"	
Transient quadriparesis	
Seizure activity	
Indications for referral ^a	
Persistent headache and other symptoms one week after concussion	
Post concussion syndrome two weeks following the concussion	
Abnormal neuropsychological test scores that are difficult to interpret	
Previous history of multiple high-grade (Cantu grade 2 or 3) concussions in same season	
History of multiple high-grade concussions in a playing career (eg two grade 3 concussions in 2 years)	
Potential for career termination	
Clinical discretion of the physician	

***Important Note:** These are suggested indications for referral and do not represent a standard of care or treatment guidelines.*

A number of sideline assessment tools have been developed, primarily for athletic trainers, to more accurately diagnose concussion. Referral to a physician for further evaluation is recommended for all cases of concussion. In addition to taking a good clinical history and performing a focused neurologic examination, tests that assess orientation, immediate memory, delayed recall, concentration, and attention are important to perform.¹⁷ One validated instrument is the Standardized Assessment of Concussion (SAC), a series of tests that assess the cognitive domains listed above. Studies have shown that the SAC is sensitive in differentiating injured from noninjured players in cases of mild (ie, Cantu grade 1) concussion. The SAC has merit for immediate evaluation on the field. Test takers improve from repeated testing due to the learning effect.

If a player has LOC on the field, one must assume that a cervical spine (C-spine) injury has occurred and follow the standard "ABC's." The LOC may be so brief that the player may regain consciousness in a few seconds and be quite

lucid. At that point, if they are lucid and have a normal C-spine examination, it is not necessary to immobilize their C-spine with a C-collar and a spinal board. If the LOC is prolonged for more than a few seconds, and an adequate C-spine examination is impossible, then appropriate immobilization and emergent transport is indicated. The indications for emergency and urgent referral are outlined in Table 3.

Colorado guidelines (Table 4) recommend emergency spine boarding and transport to the hospital for any occurrence of unconsciousness. Even if the athlete is asymptomatic within a few seconds to minutes of the period of unconsciousness, transport is recommended. Return to play is not recommended until the athlete is asymptomatic for 2 weeks.

The American Academy of Neurology practice parameter is another concussion-management guideline.²³ The individual who sustains loss of consciousness for any length of time has sustained the highest grade (3) concussion possible. Recommendations using the AAN approach suggest the athlete's returning to participation once they are asymptomatic for one week.

Though standard of care^{18,24} or clinical guideline vary, a typical sideline evaluation is included in Table 2. Serial neurologic and mental status evaluations should be conducted at 5 to 10 minute intervals (depending on the clinical status) so that any deterioration in status may be detected early.

Management Guidelines

Over 18 different concussion grading scales have been promulgated in the literature.⁵ Unfortunately, none of the grading scales for concussion severity are solidly evidence-based, but do represent expert clinical opinion.^{5,19} Familiarity with one set of guidelines to help organize the evaluation and management of concussion is important to all sports medicine caregivers. The most common guidelines utilized include those developed by Dr. Robert Cantu, an authority on concussion,^{4,20,21} the Colorado Medical Society,²² and the American Academy of Neurology (AAN) practice parameters.²³ Grading scales are shown in Table 3. Each scale has based severity of concussion on the presence and length of any period of unconsciousness. Cantu has recently modified his

Table 4. Classification schemes for concussions

	Mild: grade I	Moderate: grade II	Severe: grade III
Cantu	No loss of consciousness and post-traumatic amnesia <30 min	Loss of consciousness <5 minutes or post-traumatic amnesia >30 minutes but <24 hours	Loss of consciousness >5 minutes or post-traumatic amnesia >24 hours
Colorado Consortium	No loss of consciousness; confusion without amnesia	No loss of consciousness; confusion with amnesia	Loss of consciousness
American Academy of Neurology	Transient confusion; no loss of consciousness; symptoms or abnormalities resolve in less than 15 minutes	Transient confusion; no loss of consciousness; symptoms or abnormalities last more than 15 minutes	Any loss of consciousness

grading scale to emphasize the increased importance of post-traumatic amnesia in concussion severity.²¹

Unique in this regard, the newer evidence-based Cantu grading scale classifies a concussion as a Grade 2 if an athlete experiences 30 minutes of posttraumatic amnesia, or if an athlete has less than 5 minutes of LOC.

A recently concussed individual who is still symptomatic is at risk of a severe life-threatening condition called second impact syndrome. This syndrome occurs when an athlete who has not yet recovered from a concussion sustains another blow to the head, often seemingly minor, with the result being a rapid deterioration of neurologic status, brain herniation, and death. Although there are some unanswered questions related to this syndrome, most physicians recognize its presence and risk.²⁵

Return to Play

Deciding on return to play following a concussion may be very challenging. There may be external pressure to return an individual to competition early. The inviolate principle that must be followed is that no athlete may return to play until he or she is asymptomatic. The Cantu guidelines on return to play following different types of concussion are widely recognized in the medical community as the most useful.^{4,20}

The most common concussion managed is a Cantu grade 1 in which there is no loss of consciousness and a brief period (less than 30 min) of confusion or amnesia. Can this person return to play on the day of the concussion? If this individual is asymptomatic with exercise with a normal neurologic/cervical spine examination, then it is possible to return them to athletic competition on the same day. Most experts, however, recommend return to play in 7 days following a grade 1 concussion.^{4,20,21}

Physicians who are not very experienced in concussion management should carefully weigh all the options before returning someone to play. The author is comfortable in allowing an athlete with a Cantu grade 1 concussion who is asymptomatic with exercise to return to activity at 5 days postconcussion and to play in a football game on day 7 postconcussion.²⁶ This should be an isolated single grade 1 concussion in a single season.

Forthcoming research should provide some outcome data on long-term outcomes using different guidelines to manage concussions of varying severity. Recent studies have been limited methodologically.²⁷ Some studies suggest that long-term participation in certain sports (soccer) may lead to reduced neurocognitive function.¹⁵

Perhaps the most difficult question is, "When should one recommend that an individual terminate their playing career?" For the higher-level collegiate or professional athlete, two scenarios that may lead to a recommendation to terminate a playing career include two or three grade 3 concussions in a career.

Concussion has proven in numerous studies to cause a number of acute short-term deficits in neurocognitive function, including disturbances of planning and the ability to switch mental "set",²⁸ new learning and memory,^{28,29} reduced attention and speed of information processing,³⁰ and tasks involving visuospatial construction ability.^{28,31}

The gold standard for deciding return to play continues to be clinical evaluation of symptoms and a complete neurologic examination. Neuropsychological testing (NP) is a valuable tool for objective evaluation of cognitive function following a concussion.³²

The NP test results may be factored into deciding on return to play, although it has not been conclusively demonstrated that NP testing, used in isolation, is an appropriate way to decide when an athlete is safe to return to competition. The scope of this article does not allow for a complete review of the NP literature.

Traditional NP tests have been validated for many years, but application to large groups is limited by the time required to perform baseline testing. The latest computerized NP testing software packages enable one to do baseline testing on large groups in an efficient manner. One web-based validated NP battery called "headminder" is available commercially at <http://www.headminder.com>.³³ Other batteries such as "Impact" are also used. They are in the process of being validated.³² The NP scores obtained following a concussion are compared with baseline scores to detect any significant differences. Retesting is usually done at 48 hours, 3 days, 5 days, and 7 to 10 days after injury.^{32,33}

The computerized NP testing programs have not yet been completely validated against the traditional NP testing methods.³³ The use of NP testing to make return to play decisions, in isolation from other clinical findings, is not recommended. The instrument has not been validated in any prospective outcome-based studies which would support the return to play option. However, it does provide an additional objective measure of neurocognitive function that enables the clinician to better assess recovery from concussion.

When to Refer

Some suggested indications for referral are listed in Table 4. The reader must base the need for referral on his or her own clinical judgment.

Whether there is long-term neurocognitive impact on athletes from concussion suggestive of chronic traumatic brain injury (CTBI) has been studied with no clear answer. Some studies provide some support for CTBI.³⁴⁻³⁶ For example, a small group of former professional soccer players who were compared with crossmatched noncontact athletes had cognitive impairment (memory, planning, and visuospatial tasks) from concussion that was cumulative and suggestive of CTBI.³⁴ The implications of these studies were limited due to the lack of control for factors such as alcohol intake and unreported head

injury in the soccer population, which may account for the deficits in NP scores demonstrated in the population.^{35,36} In contrast, another study comparing the postconcussion NP scores of soccer players with or without a history of concussion showed no impaired NP performance in the concussed group.³⁷ Long-term prospective studies are necessary to truly answer the question of whether CTBI occurs as a result of concussion or is simply related to sports participation.

The Latest Research

Historically, loss of consciousness has been considered a core feature of concussion. Of course, LOC is not required for a concussion to occur. The numerous concussion grading scales, with the exception of the Cantu scale,²¹ have all graded concussion severity based on the presence and length of any associated LOC. Today's clinician may safely manage concussion by utilizing the guidelines described above.

Recent data has raised significant questions about the relative importance of LOC as a predictor for concussion severity and outcome (as measured by NP test scores).^{31,21} A study of 78 athletes divided those who experienced concussions into "good" and "bad" presentation groups. The latter was defined as a significant increase in symptoms and significant decrease in memory functioning at 2 days. Athletes with poor NP performance at 2 days after injury were 10 times more likely to have experienced postinjury retrograde amnesia and 4 times more likely to have experienced post-traumatic amnesia. No differences existed in NP performance for the LOC group. Brief LOC, therefore, is not predictive of concussion injury severity.³¹ Two other independent studies have corroborated these findings.^{37,38} As detailed in the Vienna conference, the sports medicine community must carefully consider what measures are used to define concussion severity.

Preliminary functional MRI studies show that decreased working memory in an individual with multiple concussions correlates with decreased activity visible on MRI in the frontal areas of the brain.⁶ Additional methods to assess the impact of and recovery from concussion include postural stability testing, which is advocated by some.^{37,39}

Genetic risk factors for concussion may increase an individual's risk for concussion. Previous work on boxers with CTBI demonstrated that the group with higher chronic brain injury scores was more likely to possess one APOE4 allele.⁴⁰ Additional studies have suggested a possible role of APOE4 in negatively affecting NP test scores in older football players, but methodological problems in this study and small sample size limit the conclusions that may be drawn.⁴¹ Forthcoming work by the author, using a large sample of elite athletes, will provide the first published results, to our knowledge, on whether APOE4 and other genetic polymorphisms influence the risk for sustaining a concussion and the risk for sustaining a more severe (higher grade) one.

Additional Information and Guidance

It is highly recommended that the primary care or other physician or nonphysician provider develop a strong working relationship with a "concussion-experienced" primary care sports medicine physician, neurologist, or neurosurgeon who may serve as consultant. Additional resources include the Vienna conference proceedings, the American Academy of Neurology Practice parameter, and thematic issues on Concussion in *Clinical Sports Medicine*, July 2003 and *Journal of Athletic Training*, 36:3, 2001. The CDC has distributed an informational packet entitled "Heads Up: Brain injury in your practice" on MTBI for primary care physicians and has a helpful web site, www.cdc.gov/ncipc/tbi. The Brain Injury Association may be reached at (1-800-444-6433) or at <http://www.biausa.org>.

References

- Centers of Disease Control and Prevention. Sports related recurrent brain injuries-United States. *MMWR* 1997;46:224-227.
- Kushner DS. Mild traumatic brain injury. *Arch Internal Medicine* 1998; 158:1617-1624.
- Alexander MP. Mild traumatic brain injury: pathophysiology, natural history, and clinical management. *Neurology* 1995;45:1253-1260.
- Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Physician and Sportsmedicine* 1986;14:75-83.
- Concussion in Sport Group: Johnston, K, Aubry, M, Cantu, R et al. Summary and agreement statement of the first international conference on concussion in sport, Vienna 2001. *Physician and Sportsmedicine* 2002;30.
- Johnston KM, McCrory P, Mohtadi NG, et al. Evidence based review of sport-related concussion: clinical science. *Clin J Sports Med* 2001;11: 150-159.
- Sallis RE. Prevalence of headache in football players. *Med Sci Sports Exerc*, 2001;32(11):1820-1824.
- Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. *Brain* 1974;97:633-654.
- Marion, DW. *Traumatic Brain Injury*. New York, Thieme Medical Publishers, 1999, pp 32-33.
- Cantu RC. Return to play guidelines after concussion, in *Neurologic Athletic Health and Spine Injuries*. Philadelphia, W.B. Saunders Company, 1999: 117-122.
- Hofman PAM, Kemerink GJ, Jolles J, et al. Quantitative analysis of magnetization transfer images of the brain-effect of closed head injury, age, and sex on white matter. *Magn Reson Med* 1999;42:803-806.
- Hovda D, Lee S, Smith M, et al. The neurochemical and metabolic cascade following brain injury; moving from animal models to man. *J Neurotrauma* 1995;12:903-906.
- Mitchener A, Wyper DJ, Patterson J, et al. SPECT, CT, and MRI in head injury: acute abnormalities followed up at six months. *J Neurol Neurosurg Psychiatry* 1997;62:6336.
- Delaney SJ, Lacroix VJ, Leclerc S, et al. Concussions during the 1997 Canadian Football League season. *Clin J Sports Med* 2000;54:1488-1491.
- Matser JT, Kessels AG, Jordan BD, et al. Chronic traumatic brain injury in professional soccer players. *Neurology* 1998;51:791-796.
- Cartledge, NEF and Shaw, DA. *Neurologic Sequelae of Head Injury, in Head Injury*. London, WB Saunders, 1981, pp 28-45.
- McCreary M, Kelly JP, Kluge J, et al. Standardized assessment of concussion in football players. *Neurology* 1997;48:586-588.

18. Graham DI. Neuropathology of Head Injury, in Narayan RK, Wilberger JE Jr, Povlishock JT, (eds): *Neurotrauma*. New York, McGraw-Hill, 1996, pp 46–47.
19. Leclerc S, Lassonde M, Delaney, JS et al. Recommendations for grading of concussions in athletes. *Sports Med* 2001;31:629–636.
20. Cantu RC. Return to play guidelines after a head injury. *Clin Sport Med* 1998;17:45–60.
21. Cantu, R. Posttraumatic retrograde and anterograde amnesia: pathophysiology and implications in grading and safe return to play. *J Athl Training* 2001;36:244–248.
22. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. *Neurology* 1997;48:575–580.
23. Report of the Quality Standards Subcommittee. Practice parameter: the management of concussion in sports. *Neurology* 1997;48:581–585.
24. McCrory P. Does Second impact syndrome exist? *Clin J Sports Med* 2001;11:144–149.
25. Terrell T, Tucker A. Concussion in Sports (editorial) *American Family Physician* 1999;60:738–739.
26. Hinton-Bayre, A, Geffen, G. Severity of sports-related concussion and neuropsychological test performance. *Neurology* 2002;59:1068–1070.
27. Lenninger B, Gramling S, Farrell A, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *J Neurol Neurosurg Psych* 1990;53:293–296.
28. Collins M, Grindel S, Lovell M, et al. Relationship between concussion and neuropsychological performance in college football players. *JAMA* 1999;282:964–970.
29. Barth JT, Macciocchi SN, Giordani B, et al. Neuropsychological sequelae of minor head injury. *Neurosurg* 1983;13:529–533.
30. Collins M, Iverson G, Lovell M, et al. On-field predictors of neuropsychological and symptom deficit following sports-related concussion. *Clin J Sports Med* 2003;13:222–229.
31. Grindel S, Lovell M, Collins M. The assessment of sports-related concussion: the evidence behind neuropsychological testing and management. *Clin J Sports Med*, 2001;11:134–143.
32. Erlanger D, Saliba E, Barth J, et al. Monitoring resolution of postconcussion symptoms in athletes: preliminary results of a web-based neuropsychological test protocol. *J Ath Training*, 2001;36:280–287.
33. Matser J, Kessels A, Jordan B. Chronic traumatic brain injury in professional soccer players. *Neurology* 1998;51:791–796.
34. Tysvaer AT, Lochen E. Association football injuries to the brain: a preliminary report. *Br J Sports Med* 1981;15:163–166.
35. Tysvaer AT. Soccer injuries to the brain: a neuropsychologic study of former soccer players. *Am J Sports Med* 1991;19:56–60.
36. Guskiewicz K, Marshall S, Broglio S. No evidence of impaired neurocognitive performance in collegiate soccer players. *Am J of Sports Med* 2002;20:157–162.
37. Lovell M. Does brief loss of consciousness define concussion severity in athletes? Abstracts from 11th annual meeting of the American Medical Society for Sports Medicine, Orlando, Florida, April 6–7, 2002.
38. Peterson, C, Ferrara, M, Mrazik et al. Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. *Clin J Sports Med* 2003;13:230–237.
39. Jordan B, Relkin N, Ravdin L. Apolipoprotein E epsilon 4 associated with chronic traumatic brain injury in boxing. *JAMA* 1997;278:136–140.
40. Kutner KC, Erlanger DM, Tsai J, Jordan B, Relkin NR. Lower cognitive performance of older football players possessing apolipoprotein E epsilon-4. *Neurosurgery* 2000;47(3):651–658.
41. Kutner KC, Erlanger DM, Tsai J, Jordan B, Relkin NR. Lower cognitive performance of older football players possessing apolipoprotein E epsilon-4. *Neurosurgery* 2000;47(3):651–658.

I find that the harder I work, the more luck I seem to have.

—Thomas Jefferson

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