

Spinal Cord and Cerebral Concussion in a Professional Athlete: A Case Report

Ryan Grove, MS, ATC*; Mark Lovell, PhD†; Michael Collins, PhD†; John Norwig, MEd, ATC*; Ariko Iso, MA, ATC*; Joseph Maroon, MD*

*Pittsburgh Steelers Football Club, Pittsburgh, PA; †University of Pittsburgh, Pittsburgh, PA

Address correspondence to Ryan Grove, MS, ATC, Pittsburgh Steelers, 3400 South Water Street, Pittsburgh, PA 15203.
Address e-mail to grover@steelers.nfl.com.

Objective: To present the case of a professional football player who suffered a spinal cord and cerebral concussion. This case study presents an opportunity to discuss the evaluation, treatment, management and return-to-play decisions of an athlete with a spinal cord and cerebral concussion.

Background: A cerebral concussion is a traumatically induced metabolic and neurochemical phenomenon that results in no structural damage to the brain. Such pathophysiology may result in symptoms such as confusion, amnesia, and possibly loss of consciousness. A spinal cord concussion is analogous to a cerebral concussion in that a violent impact to the spinal column causes the function of the spinal cord to shut down due to the electrochemical imbalances. As in a cerebral concussion, there may be a transient loss of all spinal cord functions with no structural damage to the cord itself.

Differential Diagnosis: Spinal cord injury, cervical spine fracture, cervical spine dislocation, cervical spine sprain/strain.

Treatment: The athlete was admitted to the hospital for fur-

ther evaluation and follow-up care. Using the results of the diagnostic studies and the clinical examination, we addressed the athlete's subjective complaints by emphasizing a gradual progression to full activity based on complete symptomatic relief.

Uniqueness: A cerebral concussion is a common athletic injury that has been extensively investigated; however, a spinal cord concussion is a rare type of reversible injury to the central nervous system that can occur in competitive athletics.

Conclusions: Head and neck injuries are prevalent, especially in athletes who participate in collision sports. Therefore, team physicians and athletic trainers must take every precaution necessary when evaluating, treating, managing, and rehabilitating the athlete to prevent further damage.

Key Words: neuropsychological testing, athletic injury, head injury, brain injury, football injury

Cerebral concussion, or mild traumatic brain injury, is the most common form of transient and reversible state of neurologic disruption that occurs in the athletic environment. Over the past 20 years, the definition, epidemiology, clinical features, and pathophysiology of cerebral concussion have been extensively investigated and reviewed.¹⁻⁵ Spinal cord concussions, on the other hand, are extremely rare, and little is known about the condition due to the

small number of clinical cases.^{6,8} Spinal cord injuries are most commonly associated with permanent neurologic deficits; however, an athlete who suffers a spinal cord concussion can achieve complete recovery in a short period of time.⁷

Our purpose is to present the case of a professional football player who suffered a spinal cord and cerebral concussion during competitive play. This case study presents an opportunity to discuss the evaluation, treatment, management, and return-to-play decisions of an athlete with a spinal cord and cerebral concussion.

case study

During an away football game, a 31-year-old male professional football play-

er experienced a severe blow to the head and neck, leaving him motionless. The hit rendered by the opposing team's linebacker caused immediate unconsciousness and loss of sensory and motor function in all 4 extremities.

The injury occurred while the quarterback scrambled up field to avoid the pass rush from his right side. As the quarterback felt pressure by the pass rusher, who hit him in the lower extremity, he released the football while falling forward. As he fell, a second player delivered a blow with his forearm to the quarterback's left shoulder and cervical spine area. Before the quarterback hit the ground, the collision caused his head to flex slightly forward and rotate to the right. The

Drs. Lovell, Collins, and Maroon are company shareholders of ImPACT Applications, the company that manufactures the ImPACT Concussion Management Software.

anterior aspect of the quarterback's head and helmet made contact with the ground and was driven into the playing surface with the weight and momentum of the linebacker's body landing on top of the quarterback. In reviewing the film of the collision between the players, it was difficult to determine the magnitude of the force delivered.

As we approached the motionless athlete, our thoughts focused on the unknown. Thinking the worst, our primary management and assessment focused on immediately stabilizing the athlete's head and neck with manual fixation to prevent any neurologic injuries secondary to those produced by the initial traumatic event. Once the head and neck were stabilized, we performed a primary survey to assess level of consciousness, responsiveness of the athlete, airway status, respiratory status, and circulatory status.

Because the athlete was unconscious and unresponsive, a cervical spine fracture or spinal cord injury was suspected until it could be ruled out. The athlete was found lying in a prone position and was unresponsive, so we performed a log-roll maneuver in order to properly conduct a primary survey and assess the athlete's condition. Once the athlete was supine, our primary goal was to continue to provide adequate stabilization of the head and neck with manual in-line stabilization and slight traction by holding the head firmly with both hands. The helmet was left on with the chinstrap fastened to help stabilize the head and neck until radiographs confirmed the presence or absence of a cervical spine fracture or dislocation.

After several minutes, the athlete began to regain consciousness. Once he was responsive, the team physician performed a secondary survey. The secondary survey consisted of taking a history, followed by a head-to-toe examination to assess level of consciousness, cranial nerves, and motor and sensory function in all 4 extremities. The neurologic exam revealed that the athlete had motor and sensory loss in all 4 extremities. Based on these findings, it appeared that spinal cord injury was a distinct possibility.

After the primary and secondary sur-

veys, we placed the supine athlete on the spine board by performing a half log-roll maneuver. While rolling the athlete, we maintained manual in-line stabilization of the head, spine, torso, and pelvis at all times. According to our standard operating procedures, we removed the face mask before transport even though the athlete's respiratory status was normal. We then placed the athlete and spine board on a stretcher and positioned him in the ambulance feet first to prevent axial loading from deceleration and braking during transport. Because an acute spinal cord injury was suspected, methylprednisolone was administered while en route to the trauma center.

Immediately upon arrival at the trauma center, radiographs of the cervical spine, magnetic resonance imaging (MRI) of the brain and spinal cord, and computed axial tomography (CAT) scan of the cervical spine were obtained. The cervical spine MRI was performed for 3 reasons: first, to rule out a fracture or dislocation of the cervical spine or a discs that may have herniated into the spinal cord; second, to look for bruising or contusion of the spinal cord (with severe damage or injury to the spinal cord, an MRI shows hemorrhage or swelling within the cord); and third, to check for spinal stenosis or canal narrowing (if the spinal canal is narrowed, then significant implications exist regarding further participation in collision sports, even if return of function is complete). In collision sports, spinal stenosis increases the risk for catastrophic injury.⁹ The MRI showed no evidence of any spinal column or disc problem. The study demonstrated no evidence of fracture, dislocation, herniated disc, hemorrhage, spinal stenosis, or any intrinsic abnormalities within the spinal cord.

In addition to the cervical spine MRI, a brain MRI was ordered to identify any focal injury. This study was also normal, and no structural abnormalities were identified. The results of this study, along with those of the cervical spine MRI, helped to confirm the athlete's diagnosis. Because no abnormalities to the brain or spinal cord were identified, the athlete's loss of function

in all 4 extremities was attributed to an electrochemical abnormality and not a structural disruption of the cord.

Lastly, a CAT scan and plain radiographs with the head in flexion and extension were reviewed to help identify any cervical spine abnormalities that may not have shown on the MRI. The results of these studies were negative. Follow-up neurologic evaluations were conducted periodically throughout the testing process to assess changes in the athlete's sensory and motor function.

Based on the diagnostic tests, clinical evaluation, and signs and symptoms, the athlete was diagnosed with a cerebral and spinal cord concussion. A cerebral concussion was diagnosed because the athlete lost consciousness for at least 2 minutes and suffered concurrent retrograde amnesia for approximately 30 minutes.¹⁰ In addition, a spinal cord concussion was diagnosed as a result of the athlete's inability to feel and move his arms and legs for approximately 60 minutes and the fact that all diagnostic tests failed to reveal any anatomical, physiologic, or structural damage to the spinal cord or spinal column.⁷

The day after the injury, the athlete was evaluated by the team neurosurgeon and neuropsychological team, who conducted a complete history, physical, and neurologic examination. The complete neurologic examination, included testing of the cranial nerves, motor and, sensory function, reflexes, and cerebellum and was completely within normal limits.

Somatic sensory evoked potentials were performed to evaluate the transmission of impulses from the body to the brain. In this electrophysiologic study, the sensory receptors of the upper and lower extremities are stimulated with a natural stimulus, such as a tap on the skin. Once initiated, the stimulus is tracked from the extremity back to the spinal cord and is registered in the brain.¹¹ This test was used to assess the function of sensory systems within the athlete's body,¹² and all results were normal.

As part of the team's concussion program, the Immediate Post Concussion Assessment and Cognitive Test (ImPACT, Pittsburgh, PA) was administered the

same morning by the team neuropsychologist at the athlete's bedside.¹³⁻¹⁶ ImPACT is a tool that has been used extensively in examining recovery from sports-related concussions. Computerized neuropsychological testing has been covered comprehensively,¹⁷ and data regarding the reliability¹⁸ and validity^{13,14,16,19} of this instrument has been reported in the literature.

A base line study using ImPACT had been conducted during the athlete's initial preseason physical examination approximately 14 months before his injury. This baseline evaluation provided the standard for the athlete in the event of a concussion during the season. The Appendix contains the comprehensive clinical report that was generated by ImPACT. The postconcussion test results were compared with the athlete's individual baseline results to help determine recovery in the areas of verbal memory, visual memory, visual motor speed, reaction time (page 3 of clinical report) and symptom reporting (page 4 of clinical report). Higher scores indicate better performance on the verbal memory, visual memory, and visual motor speed composite scores, whereas lower scores indicate better performance on the reaction time and symptom composite scores. Impulse control is a validity scale indicating how many errors were committed during the battery of neurocognitive tests. The player had completed an earlier version of ImPACT at the time of baseline testing, so the Design Memory and X's and O's test were not available for comparison (see raw data on page 2 of report). Although there were no significant differences between among the 4 composite scores, a closer inspection of the ImPACT raw data revealed mild difficulties in cognitive speed and memory, as illustrated by the athlete's performance on the Symbol Match module. More specifically, his average reaction time score increased from 1.82 at baseline to 1.92 at the time of his first postconcussion evaluation. Furthermore, his memory score on Symbol Match (hidden symbols) dropped from 89% accuracy (8 of 9 correct) to only 56% accuracy (5 of 9 correct). At his next followup evaluation, 4 days postinjury, this score was better than his initial baseline perform-

ance. Overall, the test findings indicated the athlete's acute neurocognitive recovery was excellent.

The athlete was reevaluated in 2 days. His ImPACT test performance showed continued improvement in the area of reaction time. His memory scores continued to be excellent and did not indicate any neurocognitive difficulties. Within 48 hours of a significant blow to the head and neck, the athlete's neurocognitive functions had returned to a normal state. It is important to note the athlete did not report significant postconcussive symptoms at followup, such as headache, dizziness, or balance dysfunction. However, he did report some difficulties with fatigue and with sleep, which were thought to reflect the fact that he had been treated with large doses of corticosteroids after his injury.

It is difficult for athletic trainers to imagine what it is like to be lying on a football field unable to feel and move your arms and legs. To help the athlete, our first goal was to make sure he had a full understanding of his injury, current condition, and possible risk with future participation. With the help of the diagnostic studies and consultation with the team neurosurgeon and neuropsychologist, we presented the athlete with the information he needed to make an educated decision on whether or not to return to professional football. Once the athlete made a decision, it was our responsibility to help him become mentally and physically ready for competition. Our goal now focused on returning the athlete to competition in a safe manner by steadily building confidence while preventing setbacks.

The rehabilitation process addressed the athlete's subjective complaints, because objective studies were normal, and ImPACT test results returned to baseline within 48 hours. The rehabilitation program emphasized a gradual progression to full activity based on complete symptomatic relief. We instructed the athlete to rest and perform simple activities of daily living until he felt asymptomatic.

This gave him time to mentally and physically recover from the initial trauma and treatment administered in

intensive care. Due to the suspected spinal cord injury, we had administered pharmacological doses of methylprednisolone, according to the spinal cord protocol established by National Acute Spinal Cord Injury Study.^{20,21} Possible side effects of this medication are emotional instability, fatigue, and insomnia, among others. Many of the initial subjective complaints presented by the athlete may have been related to this treatment.

Once the athlete was asymptomatic at rest, he was permitted to begin a functional progression. We initiated a program of light exercise consisting of non-sport-specific aerobic activities (ie, stationary bicycle), and sport-specific activities (ie, throwing the football). We monitored the athlete's symptoms subjectively using a visual analog scale (0 to 10) and ImPACT to evaluate neurocognitive function. This stage also included mental preparation by reinstating off-field activities such as watching film, attending team meetings, interacting with players and coaches, and preparing for an opponent.

We had the athlete progress to strenuous exercise by gradually increasing the level of intensity and perceived exertion. Each stage consisted of a base of non-sport-specific exercises on which sport-specific activities were built. As the athlete advanced symptom free, we began to emphasize position-specific drills. We removed restrictions on team activities as the athlete felt comfortable with the return of his abilities. He successfully returned to full practice with no restrictions by carefully increasing the amount (time), type (noncontact versus limited contact versus full contact), and intensity of sport-related activities. During the entire rehabilitation process, neurocognitive function and the athlete's report of symptoms were closely monitored.

To ensure full physical and psychological recovery, the athlete needed to be asymptomatic at rest, with exertion, and with contact before being cleared for participation. Once he felt confident and comfortable with his level of function and sport-specific proficiency, he was able to return to competition at a high level for the remainder of the season.

discussion

Cerebral concussions have been extensively reviewed in the literature with clinical and long-term follow-up studies.¹⁻⁵ This research has resulted in a better understanding of the biomechanical and pathophysiological aspects of the injury. From this research many authors have proposed grading scales, management recommendations, and return-to-play guidelines for athletes.^{1,3,4}

In addition, permanent spinal cord injuries have also been studied comprehensively.²²⁻²⁴ However, spinal cord concussions are a rather unknown entity due to their infrequent occurrence and lack of clinical evidence. The term *spinal cord concussion* was first recorded by Obersteiner in 1879.²⁵ He coined the term to help describe a spinal cord injury involving complete recovery within 24-48 hours after acute trauma. Obersteiner hypothesized that spinal cord concussions were a result of "molecular disturbance of neurons due to trauma."²⁵ In 1941, Denny-Brown and Russel²⁶ theorized that spinal cord concussions were a result of a functional block of neural activity, similar to the disruption in neurologic functioning that occurs with a cerebral concussion. A few years later, Groat et al²⁷ further supported Obersteiner's beliefs that spinal cord concussions are a reversible condition and reiterated Denny-Brown and Russel's theories.

After these early reviews, few recent authors have discussed spinal cord concussions or transient spinal cord injuries. Torg et al,⁶ in 1984, reviewed 32 college and professional athletes diagnosed with transient spinal cord injuries and estimated that 1.3 of 10000 United States college and professional football players suffered a spinal cord concussion. Torg et al reported that all 30 subjects had a cervical spine abnormality consisting of spinal stenosis (26 patients) or posterior spinal ligament instability (4 patients). Given the high incidence of cervical spine abnormalities, they concluded transient spinal cord injuries are likely due to a compression of the spinal cord at the time of the acute injury.⁶

In 1990, Zwimpfer and Bernstein⁷ pre-

sented a clinical review consisting of 19 cases of spinal cord concussions in the general population. Of the 19 cases, 16 spinal cord concussions occurred in the cervical spine and 3 in the thoracolumbar region. Zwimpfer and Bernstein defined a spinal cord concussion based on clinical and radiologic findings of each subject that met the following criteria:

1. spinal trauma immediately preceded the onset of neurologic deficits,
2. neurologic deficits were consistent with spinal cord involvement at the level of injury; and
3. complete recovery occurred within 72 hours after injury.⁷

These criteria were also used in the Torg et al⁶ study. Signs of recovery from the sensory and motor deficits were observed in several patients within hours. Complete recovery in most cases was noted within 24 hours. Contrary to Torg et al's findings, radiologic and diagnostic studies were negative in all subjects except one who was diagnosed with an unstable cervical spine injury. Thus, 18 of 19 patients showed no evidence of predisposing factors such as ligamentous laxity, structural instability, or cervical stenosis.⁷ With the lack of radiological evidence of cervical stenosis and disruption of the cervical spine structural integrity, Zwimpfer and Bernstein⁷ concluded, "concussive injuries of the spinal cord may be the result of an indirect injury in which the injuring force is transmitted to the cord without direct cord compression."

Torg et al⁶ conducted the most recent clinical review found in the literature in 1997. They reviewed 110 cases of cervical cord neurapraxia. Cervical cord neurapraxia was defined as a transient neurologic phenomenon that an athlete experiences after acute trauma to the cervical spine, resulting in symptoms involving both arms, both legs, all 4 extremities, or an ipsilateral arm and leg. The neurologic symptoms include sensory changes with or without associated motor changes and typically last less than 15 minutes, fully resolving in most patients within 48 hours.⁸ Similar to Torg et al's⁶ earlier study in 1984, Torg et al⁶ concluded cervical cord neurapraxia is caused by "congenital or

degenerative narrowing of the sagittal diameter of the cervical canal" due to the high incidence of cervical spine abnormalities found on plain radiographs and MRI.⁸ Abnormal radiographs and MRIs demonstrated cervical stenosis, osteophytic ridging, loss of cervical lordosis, degenerative disc disease, disc protrusion, disc bulge, or neuroforaminal compromise. However, in the 110 subjects reviewed, 7% of the plain radiographs (7/104) and 6% of the MRI findings (4/53) were read as normal. Therefore, in a small number of clinical cases, cervical cord neurapraxia occurred even though diagnostic tests failed to establish anatomical, physiologic, or structural damage to the spinal cord or spinal column. The authors concluded, "individuals with uncomplicated cervical cord neurapraxia may be permitted to return to their previous activity without an increased risk of permanent neurological injury."⁸

conclusions

Acute trauma to the head and cervical spine can cause various degrees of damage to the brain, spinal cord, and neural elements, resulting in a life-threatening injury, transient paralysis, or permanent paralysis. In addition, athletic central nervous system injuries may result in sensory, memory, and vestibular deficits, with or without associated long-term cognitive effects. Spinal cord concussion remains a vague clinical condition with poorly defined findings.

Spinal cord concussion is a transient loss of motor and/or sensory function of the spinal cord that usually resolves within minutes to hours after the injury. In most patients, the initial clinical evaluation reveals rapidly diminishing signs and symptoms and yields a normal neurologic examination within 48 hours. The exact pathophysiology of spinal cord concussions is unknown, and it remains uncertain whether the injury model represents a biochemical, vascular, depolarization, or axonal injury as a result of direct mechanical injury. This case study along with the work of previous researchers has shown the short-term projections for athletes who suffer

spinal cord concussions are excellent. However, due to the small amount of clinical data reported in the literature, long-term results are difficult to predict. Because no clear management strategies exist, return-to-play decisions should be made on an individual basis by the athlete and the team physician using of the results of clinical evaluation and diagnostic studies.

references

1. Collins MW, Lovell MR, McKaeag DB. Current issues in managing sports-related concussion. *JAMA*. 1999;282:2283-2285.
2. Guskiewicz KM, Weaver NL, Padua DA, Garrett WE Jr. Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med*. 2000;28:643-650.
3. Maroon JC, Field M, Lovell M, Collins M, Bost J. The evaluation of athletes with cerebral concussion. *Clin Neurosurg*. 2002;49:319-332.
4. Maroon JC, Lovell MR, Norwig J, et al. Cerebral concussion in athletes: evaluation and neuropsychological testing. *Neurosurgery*. 2000;47:659-669.
5. Shetter AG, Demakas JJ: The pathophysiology of concussion: a review. *Adv Neurol*. 1979;22:5-14.
6. Torg JS, Pavlov H, Genuario SE, et al. Neuroapraxia of the cervical spinal cord with transient quadriplegia. *J Bone Joint Surg Am*. 1986;68:1354-1370.
7. Zwimpfer TJ, Bernstein M. Spinal cord concussion. *J Neurosurg*. 1990;72:894-900.
8. Torg JS, Corcoran TA, Thibault LE, et al. Cervical cord neurapraxia: classification, pathomechanics, morbidity, and management guidelines. *J Neurosurg*. 1997;87:843-850.
9. Giovanini M, Day A. Spinal injuries in athletes with cervical stenosis. *Techniq Neurosurg*. 1999;5:185-193.
10. Cantu RC. Posttraumatic retrograde and anterograde amnesia: pathophysiology and implications in grading and safe return to play. *J Athl. Train*. 2001;36:244-248.
11. Soliman E, Legatt A. Somatosensory evoked potentials: general principles. Available at <http://www.emedicine.com/neuro/topic640.htm>. Accessed April 24, 2004.
12. Burneo J, Barkley G. Somatosensory evoked potentials: clinical applications. Available at <http://www.emedicine.com/neuro/topic344.htm>. Accessed April 24, 2004.
13. Collins MW, Iverson GL, Lovell MR, McKaeag DB, Norwig J, Maroon J. On-field predictors of neuropsychological and symptom deficit following sports-related concussion. *Clin J of Sport Med*. 2003;13:222-229.
14. Collins MW, Field M, Lovell MR, Iverson G, Johnston KM, Maroon J, Fu FH. Relationship between postconcussion headache and neuropsychological test performance in high school athletes. *Am J of Sports Med*. 2003;31:168-173.
15. Collins MW, Lovell MR, Iverson GL, Cantu RC, Maroon JC, Field M. Cumulative effects of concussion in high school athletes. *Neurosurgery*. 2002;51:1175-181.
16. Lovell MR, Collins MW, Iverson GL, et al. Recovery from mild concussion in high school athletes. *J Neurosurgery*. 2003;98:295-301.
17. Podell, K. Computerized assessment of sports-related brain injury. In: Lovell M, Echemendia R, Barth J, Collins M. *Traumatic Brain Injury Sport: An International Neuropsychological Perspective*. 1st ed. Lisse, The Netherlands: Swets & Zeitlinger; 2004:375-396.
18. Iverson GL, Lovell MR, Collins MW. Interpreting change on ImPACT following sport concussion. *Clinical Neuropsychology*. 2003;17:460-467.
19. Lovell MR, Collins MW, Iverson GL, Johnston KM, Bradley JP. Grade 1 or "ding" concussions in high school athletes. *Am J Sports Med*. 2004;32:47-54.
20. Bracken, MB, Shepard, MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury: results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury. *JAMA*. 1997;277:1597-1604.
21. Kleiner DM, Almquist JL, Bailes J, et al. *Prehospital Care of the Spine-Injured Athlete: A Document from the Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete*. Dallas, TX: National Athletic Trainers' Association; 2001.
22. Osterholm JL. The pathophysiological response to spinal cord injury: The current status of related research. *J Neurosurg*. 1974;40:5-33.
23. Sandler AN, Tator CH. Review of the effect of spinal cord trauma on the vessels and blood flow in the spinal cord. *J Neurosurg*. 1976;45:638-646.
24. Schneider RC, Crosby EC, Russo RH, Gosch HH. Chapter 32: Traumatic spinal cord syndromes and their management. *Clin Neurosurg*. 1974;20:424-492.
25. Obersteiner H. Uber Erschutterung des Ruckenmarks. *Wein Med Jahrb*. 1879;34:531.
26. Denny-Brown D, Russel WR. Experimental cerebral concussion. *Brain*. 1941;64:93-164.
27. Groat, RA, Rambach WA Jr, Windle WF. Concussion of the spinal cord: an experimental study and a critique of the use of the term. *Surg Gynecol Obstet*. 1945;81:63-74.

ImPACT® Clinical ReportOrganization: **Pittsburgh Steelers**Subject ID#: **000-10-0023**Date of birth: **00/00/00**Age: **32**Gender: **Male**Height: **76 inches**Handedness: **Right**Weight: **218 lbs**Native country/region: **United States of America**Second language: **(None)**Native language: **English**Years speaking: **0**Years of education completed
excluding kindergarten: **16**Received speech therapy: **No**Diagnosed learning disability: **No**Problems with
ADD/Hyperactivity: **No**Attended special
education classes: **No**Repeated one or more
years of school: **No**Current sport: **Football**Primary position/event/class: **Quarterback**Current level of participation: **Professional**Years experience at this level: **10**Number of times diagnosed with a concussion (excluding current injury): **1**Concussions that resulted in loss of consciousness: **0**Concussions that resulted in confusion: **0**Concussions that resulted in difficulty remembering events that occurred immediately after injury: **0**Concussions that resulted in difficulty remembering events that occurred immediately before injury: **0**Total games missed as a result of all concussions combined: **0**Concussion history: **09/15/1989**Treatment for headaches by physician: **No** Treatment for psychiatric condition (depression, anxiety): **No**Treatment for migraine headaches by physician: **No** History of meningitis: **No**Treatment for epilepsy/seizures: **No** Treatment for substance/alcohol abuse: **No**History of brain surgery: **No**

ImPACT® Clinical Report

Exam Type:	Baseline	Post-concussion	Post-concussion
Date Tested:	08/07/2001	11/19/2002	11/21/2002
Last Concussion:	11/17/2002	11/17/2002	
Exam Language:	English	English	English
Test Version:	2.3.812	2.3.812	2.3.812

Word Memory	WG=1	WG=2	WG=3
Hits (immediate)	12	12	12
Correct distractors (immediate)	12	11	12
Learning percent correct	100%	96%	100%
Hits (delay)	8	10	10
Correct distractors (delay)	12	11	9
Delayed memory percent correct	83%	88%	79%
Total percent correct	92%	92%	90%

Design Memory

Hits (immediate)	12	11
Correct distractors (immediate)	11	12
Learning percent correct	96%	96%
Hits (delay)	12	12
Correct distractors (delay)	11	12
Delayed memory percent correct	96%	100%
Total percent correct	96%	98%

X's and O's

Total correct (memory)	10	9
Total correct (interference)	128	130
Avg. correct RT (interference)	0.38	0.38
Total incorrect (interference)	3	3
Avg. incorrect RT (interference)	0.35	0.27

Symbol Match

Total correct (symbols)	27	27	27
Avg. correct RT (symbols)	1.82	1.92	1.59
Total correct (symbols hidden)	8	5	7
Avg. correct RT (symbols hidden)	1.94	1.81	1.65

Color Match

Total correct	8	9	9
Avg. correct RT	0.87	0.54	0.82
Total commissions	0	18	0
Avg. commissions RT	0.00	0.00	0.00

Three Letters

Total sequence correct	5	5	5
Total letters correct	15	15	15
Percent of total letters correct	100%	100%	100%
Avg. time to first click	4.47	2.76	1.87
Avg. counted	13.0	14.8	16.4
Avg. counted correctly	13.0	14.2	13.6

ImPACT® Clinical Report

Exam Type:	Baseline	Post-concussion	Post-concussion
Date Tested:	08/07/02	11/19/2003	11/21/2003
Last Concussion:	11/17/2002	11/17/2002	

Composite Scores

Memory composite (verbal)	94%	82%	89%
Memory composite (visual)*	not given	90%	86%
Visual motor speed composite	35.88	37.30	36.95
Reaction time composite	0.62	0.62	0.58
Impulse control composite	11	4	3

* New clinical/research composite score for ImPACT version 2.0. All other composite scores are identical to ImPACT version 1.1.

Concussion Details

Loss of consciousness	3-5 minutes
Retrograde amnesia	1-10 seconds
Anterograde amnesia	16-30 minutes
Confusion/disorientation	> 30 minutes
Returned to play	Did not return
Taken to hospital	Yes
CT/MRI scan of head	Negative
Mouthguard type	
Mouthguard condition	
Symptoms	Numbness or tingling
Description of injury and additional information	

ImPACT® Clinical Report

Exam Type:	Baseline	Post-concussion	Post-concussion
Date Tested:	11/19/03	11/19/2003	11/21/2003
Last Concussion:	11/17/2002	11/17/2002	

Symptoms

Headache	0	0	0
Nausea	0	1	0
Vomiting	0	0	0
Balance Problems	0	1	0
Dizziness	0	1	0
Fatigue	0	1	2
Trouble falling asleep	0	0	3
Sleeping more than usual	0	0	1
Sleeping less than usual	0	0	0
Drowsiness	0	1	0
Sensitivity to light	0	0	0
Sensitivity to noise	0	0	0
Irritability	0	0	0
Sadness	0	0	0
Nervousness	0	0	0
Feeling more emotional	0	0	2
Numbness or tingling	0	0	0
Feeling slowed down	0	1	0
Feeling mentally foggy	0	0	0
Difficulty concentrating	0	0	0
Difficulty remembering	0	0	0
Visual problems	0	0	0
Total Symptom Score	0	6	8