HOUSE JUDICIARY COMMITTEE ANSWERS

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TAB 1A

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TACKLING CONCUSSIONS IN SPORTS

The safety of our players is an important priority for all of us in the National Football League (NFL). We have taken many steps in recent years to enhance player safety, including several rules changes aimed at reducing head injuries.

In the early 1990s, as we looked more deeply into the specific area of concussions, we realized that there were many more questions than answers. In 1994, I appointed an NFL committee on mild traumatic brain injury with experts from inside and outside our league. It consisted of NFL team doctors and trainers, plus a neurologist, neurosurgeon, neuropsychologist, biomechanical engineer, and epidemiologist.

One of the key recommendations of the committee was that the NFL should support independent scientific research to foster better understanding of the causes, diagnosis, treatment, and prevention of concussions. In response to this recommendation, the NFL and NFL Charities funded important research projects at leading universities in the United States and Canada.

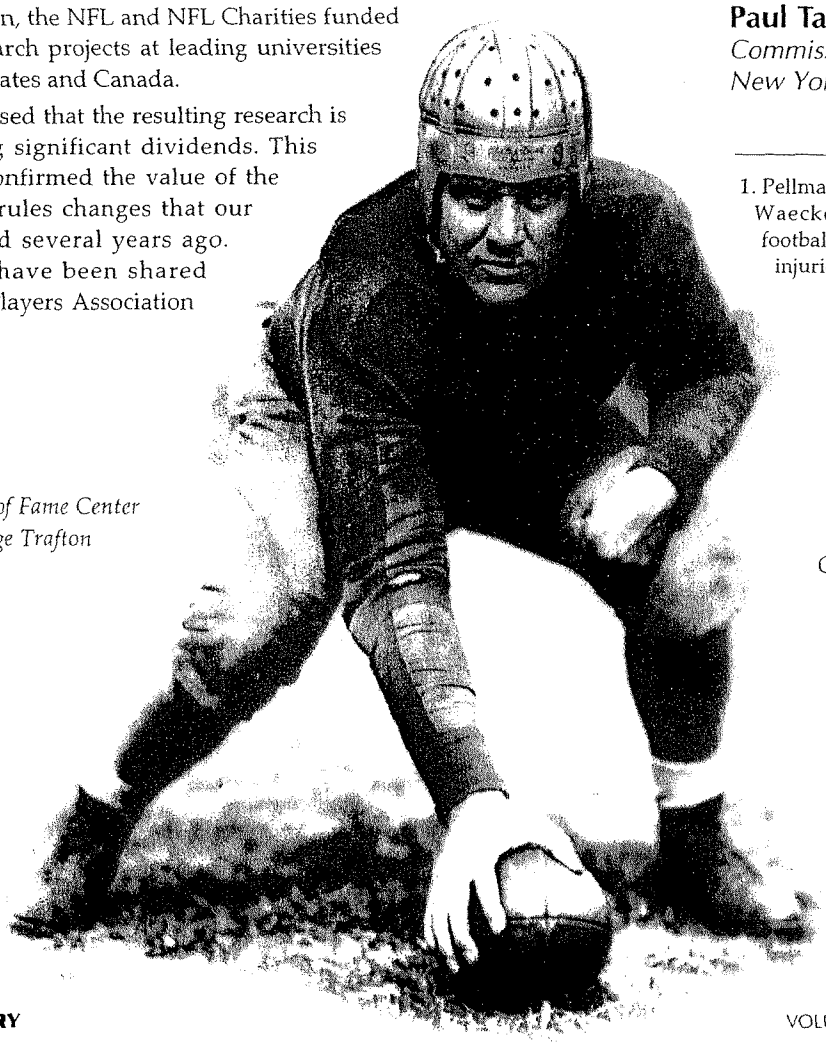
We are pleased that the resulting research is already paying significant dividends. This research has confirmed the value of the safety-related rules changes that our clubs approved several years ago. The findings have been shared with the NFL Players Association and independent equipment testing groups and helmet manufacturers, one of which, Riddell, has designed a new helmet. The research has also contributed to advancing our understanding of the science of concussions, which is a concern for everyone involved in competitive sports and recreational activities.

The accompanying article (1) confirms the groundbreaking character of this research. We expect to publish further reports and look forward to positive reactions from the medical and scientific communities.

We salute our Mild Traumatic Brain Injury Committee for its leadership and the individuals involved in this project who put in so many hours of work. We are confident that this important new information will continue to advance the cause of improving the safety of professional and amateur athletes on all levels.

Paul Tagliabue
Commissioner, National Football League
New York, New York

1. Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF: Concussion in professional football: Reconstruction of game impacts and injuries. **Neurosurgery** 53:799-814, 2003.



Hall of Fame Center George Trafton

Chicago Bears, c. 1927

BACKGROUND ON THE NATIONAL FOOTBALL LEAGUE'S RESEARCH ON CONCUSSION IN PROFESSIONAL FOOTBALL

In 1994, as the associate team physician and internist for the New York Jets, I was responsible for the diagnosis and treatment of concussions. I had been with the New York Jets since 1987, having trained as an internist and rheumatologist. Although published information existed, most of what I—like other team physicians—knew about concussions was from on-field anecdotes passed on from other team physicians and athletic trainers who had been treating professional football players for many years. During my years of medical school, internal medicine training (including an extra year as chief medical resident), and fellowship, from 1975 to 1986, I had never received a single lecture on concussions. As I learned later, this was typical of physician training for what was then an often underdiagnosed and little-understood clinical condition.

Al Toon, a Pro Bowl receiver for the New York Jets, had been with the team since 1985 after playing college football at the University of Wisconsin. As a player for the New York Jets, Mr. Toon was recognized as one of the finest receivers in the National Football League (NFL). From the beginning of his professional career, Mr. Toon began to incur what we now recognize as concussions. These "dings," as they were referred to then, were minor, often causing no more than mild headaches and some dizziness. Unrecognized by everyone, including myself, these concussions began to worsen in the later years of his career. Mr. Toon began to experience severe headaches, malaise, intolerance of loud noises, depression, and emotional lability after what were viewed as mild, inconsequential blows to the head. Mr. Toon was experiencing what we now call postconcussion syndrome, which would eventually lead to the premature retirement of this great athlete in 1992. He was the first documented NFL player that I know of to retire as a result of this problem.

Mr. Toon's retirement, which received some attention in the public news media but none in the medical world, was overshadowed by the injury to Dennis Byrd, a defensive lineman who sustained a fracture of the cervical spine while also playing for the Jets. It was not until the following year, when another NFL player, Merrill Hoge, a player for the Pittsburgh Steelers, was also forced to retire because of postconcussion syndrome, that both medical personnel and football executives took notice.

Shortly after the retirement of Mr. Hoge, NFL Commissioner Paul Tagliabue began to inquire about this medical issue. On the basis of my experience with Mr. Toon, I was invited to the Commissioner's office to offer my limited insight into this problem. The Commissioner and I realized that we had many more questions than answers. Was this a new problem or just an often misdiagnosed or unrecognized one? Was the premature retirement of these men a statistical anomaly or the beginning of an epidemic? I was asked to mount an effort to answer these questions.

During my treatment of Mr. Toon, I quickly realized how few experts and how little prospective, scientific medical information were available regarding concussions. I decided that a novel approach would be necessary to gather information, particularly for a professional sports league, and with the encouragement and support of Commissioner Tagliabue, in 1994 I formed the NFL Committee on Mild Traumatic Brain Injury. This Committee was composed of experts from inside and outside the NFL, consisting of team physicians, team athletic trainers, a team equipment manager, a neurologist who had considerable previous clinical experience with boxers, a neurosurgeon who had experience in treating NASCAR drivers, a neuropsychologist who was a pioneer in using neuropsychological testing to evaluate athletes, a biomechanical engineer to help us understand safety equipment, and an epidemiologist.

It became apparent to the Committee that there was no single accepted definition of concussion and that, if we were to begin asking questions regarding the problem, we would need a single definition that would be used league-wide by the medical staffs of all the teams. The first several months of meetings were dedicated to defining concussion, or as we quickly decided, the more academically appropriate term, *mild traumatic brain injury*, which is more commonly referred to as MTBI. After a great deal of discussion, we decided that our definition would be broad, realizing that we would rather overidentify injuries than potentially exclude milder ones. A reportable MTBI was defined as a traumatically induced alteration in brain function that is manifested by 1) alteration of awareness or consciousness, including but not limited to loss of consciousness, "ding," sensation of being dazed or stunned, sensation of "wooziness" or "fogginess," seizure, or amnesic period; and 2) signs and symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, light-headedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive

dysfunction, memory disturbance, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities.

Once there was agreement on the definition of MTBI, we quickly realized that the first order of business was to gather information, in the knowledge that this would need to be a meticulous and lengthy process, and to educate and sensitize the League's medical and coaching staffs to this medical problem. With the help of the epidemiologist on the Committee, we devised a questionnaire that team physicians and trainers would be required to fill out after a player sustained an MTBI. A memo was sent to the physicians, athletic trainers, and team executives by the Commissioner emphasizing the importance of this information and mandating compliance in filling out these detailed forms.

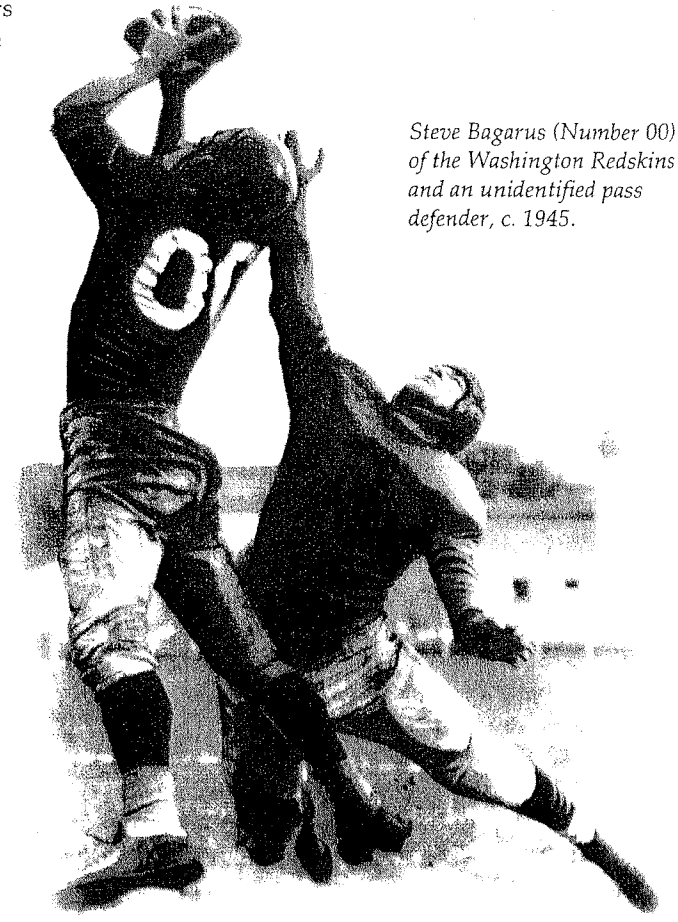
After there was agreement on the form and a basis for collecting MTBI data, the Committee decided to begin to examine the available safety equipment. Many team physicians had similar experiences with regard to the sales personnel of helmet manufacturers, who made claims about helmets and their potential to decrease the risk of MTBI. Players and medical staff were told to increase the air in the inflatable bladders in the helmets or to change the type of helmet to reduce the risk of MTBI. When manufacturers were invited to present the scientific data to support these claims, it became apparent to the Committee members that these claims were not based on scientific data but rather were "sales pitches" made by overzealous salespeople.

Contacts with representatives and biomechanical engineers of the National Operating Committee on Standards for Athletic Equipment additionally made it obvious to the Committee that the testing methods used to quantify football helmet head protection were unable to predict the amount of protection afforded to prevent MTBI. Furthermore, this lack of understanding of the physics and biomechanics of the injury was retarding helmet manufacturers from making changes to helmets that could lead to greater MTBI protection.

After lengthy discussion and many Committee meetings, it was decided that the committee would recommend to Commissioner Tagliabue that the NFL should independently fund scientific research that would enable scientists to better understand the cause(s) of MTBI; that this research should be funded to independent scientific researchers; and that the NFL Mild Traumatic Brain Injury Committee should be charged with oversight of the project. It was also decided that all research results would be given to the National Operating Committee on Standards for Athletic Equipment, to helmet manufacturers, and to as many researchers and clinicians as possible in an attempt to promote a better understanding of MTBI and methods that might, directly and indirectly, prevent and treat these injuries.

From the current perspective, the enormous investment in research on MTBI is paying dividends in an improved understanding of the causes and prevention of the injury. Although much more research is needed, the results of the Committee's research thus far will be presented as a series of articles in **NEUROSURGERY**. The intention is now, as it was originally, to contribute scientific articles on the NFL's research on the biomechanics of concussion, the epidemiology of the injury, its symptoms and treatment, neuropsychological evaluations, and other aspects of MTBI. Thanks to the support of the NFL, NFL Charities, owners, executives, team medical staffs, players, and Committee members, the NFL's approach to funding scientific research on the problem is a model for the approach needed by other sport leagues when medical issues of player health and safety emerge.

Elliot J. Pellman
Chairman, Mild Traumatic Brain Injury Committee, National Football League, New York, New York



Steve Bagarus (Number 00) of the Washington Redskins and an unidentified pass defender, c. 1945.

CONCUSSION IN PROFESSIONAL FOOTBALL: RECONSTRUCTION OF GAME IMPACTS AND INJURIES

Elliot J. Pellman, M.D.
ProHEALTH Care Associates, LLP, Lake Success, New York

David C. Viano, Dr. med., Ph.D.
ProBiomechanics, LLC, Bloomfield Hills, Michigan

Andrew M. Tucker, M.D.
University of Maryland School of Medicine, Timonium, Maryland

Ira R. Casson, M.D.
Department of Neurology, Long Island Jewish Medical Center, New Hyde Park, New York, and Department of Neurology, New York University Medical Center, New York, New York

Joe F. Waeckerle, M.D.
Acute Care and Emergency Specialists, Leawood, Kansas

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics, LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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OBJECTIVE: Concussion in professional football was studied with respect to impact types and injury biomechanics. A combination of video surveillance and laboratory reconstruction of game impacts was used to evaluate concussion biomechanics.

METHODS: Between 1996 and 2001, videotapes of concussions and significant head impacts were collected from National Football League games. There were clear views of the direction and location of the helmet impact for 182 cases. In 31 cases, the speed of impact could be determined with analysis of multiple videos. Those cases were reconstructed in laboratory tests using helmeted Hybrid III dummies and the same impact velocity, direction, and head kinematics as in the game. Translational and rotational accelerations were measured, to define concussion biomechanics. Several studies were performed to ensure the accuracy and reproducibility of the video analysis and laboratory methods used.

RESULTS: Concussed players experienced head impacts of 9.3 $\pm$ 1.9 m/s (20.8 $\pm$ 4.2 miles/h). There was a rapid change in head velocity of 7.2 $\pm$ 1.8 m/s (16.1 $\pm$ 4.0 miles/h), which was significantly greater than that for uninjured struck players (5.0 $\pm$ 1.1 m/s, 11.2 $\pm$ 2.5 miles/h; $t = 2.9$, $P < 0.005$) or striking players (4.0 $\pm$ 1.2 m/s, 8.9 $\pm$ 2.7 miles/h; $t = 7.6$, $P < 0.001$). The peak head acceleration in concussion was 98 $\pm$ 28 g with a 15-millisecond half-sine duration, which was statistically greater than the 60 $\pm$ 24 g for uninjured struck players ($t = 3.1$, $P < 0.005$). Concussion was primarily related to translational acceleration resulting from impacts on the facemask or side, or falls on the back of the helmet. Concussion could be assessed with the severity index or head injury criterion (the conventional measures of head injury risk). Nominal tolerance levels for concussion were a severity index of 300 and a head injury criterion of 250.

CONCLUSION: Concussion occurs with considerable head impact velocity and velocity changes in professional football. Current National Operating Committee on Standards for Athletic Equipment standards primarily address impacts to the periphery and crown of the helmet, whereas players are experiencing injuries in impacts to the facemask, side, and back of the helmet. New tests are needed to assess the performance of helmets in reducing concussion risks involving high-velocity and long-duration injury biomechanics.

KEY WORDS: Biomechanics, Concussion, Football helmets, Head injury criteria, Impact tolerances, Sport injury prevention, Traumatic brain injury

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In 1973, the National Operating Committee on Standards for Athletic Equipment (NOCSAE) established standards for the impact performance of football helmets (14). This followed earlier work by Wayne State University Department of Neurosurgery, which was contracted by NOCSAE in 1971 to develop voluntary standards for football helmets. Preliminary tests of representative football helmets on cadavers and the Z-90 metal headform demonstrated that a more realistic head model was required before helmet performance could be specified. Further research yielded an improved headform made of synthetic materials, which approached human cadaver performance and was rugged and reusable. The NOCSAE standard limited the severity index (SI), which is based on resultant head acceleration. All new football helmets available for use in high school and college football were then certified to the NOCSAE standard, and the wearing of such

helmets was made mandatory for college players in 1978 and for high school players in 1980. The certified helmets yielded one-half the SI, compared with those before establishment of the standard.

By 1980, significant reductions in injuries were observed with the voluntary adoption of NOCSAE standards by helmet manufacturers. In youth football, a 51% reduction in fatal head injuries, a 35% reduction in concussions, and a 65% reduction in cranial fractures were observed (15). Between 1981 and 1985, there were further reductions in fatal head injuries. Although rule changes were also implemented, most of the reductions were considered to be related to helmet design, because neck injury rates increased, indicating that significant head impacts were still occurring.

Hodgson and Thomas (16) reported on the performance of pre- and poststandard helmets in NOCSAE tests; 84% of the prestandard helmets exceeded a SI of 1450, whereas the poststandard helmets exhibited an average SI of 1064. Mertz et al. (26) converted the SI values to head injury risks and concluded that the NOCSAE standard and helmet design changes had reduced the risk of head injury from 55% to 12%. Cantu and Mueller (6) recently summarized the reductions in fatal head injury risks in football, and others have addressed the topic (3, 7, 28).

While continued improvements in the NOCSAE performance of helmets were being made through the late 1980s and 1990s, attention focused on the incidences of concussions in organized sports (42). Professional football had experienced the retirement of key skill players after repeated concussions, and the need for further improvements in the safety performance of helmets was becoming apparent. Concussions were observed to be associated with a range of symptoms and variations in recovery (23). Some individuals experienced postconcussion syndrome (5), and others experienced even more serious consequences after repeated concussions (45). An uncommon and controversial complication is the second-impact syndrome, involving often-fatal cerebral edema after a second or repeated concussion before the resolution of an initial injury (40). This proposed entity has been linked to boxing, ice hockey, and high school and college football but not professional football. Some authors have questioned the verifiability of this syndrome (25).

In 1994, the commissioner of the National Football League (NFL) formed a committee on mild traumatic brain injuries (MTBIs), in response to safety concerns regarding concussions in professional football (37). The committee included representatives from the NFL Team Physicians Society and the NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the areas of traumatic brain injury, basic science research, and epidemiology. The mission of the committee was to scientifically investigate MTBIs (concussions) in the NFL.

For this research, a reportable MTBI was defined as a traumatically induced alteration in brain function manifested by an alteration of awareness or consciousness, including but not limited to a loss of consciousness, "ding," sensation of being dazed or stunned, sensation of "wooziness" or "fogginess," seizure, or amnesic period, and by symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, light-headedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbances, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities. This definition of concussion, which was used by the MTBI committee, is consistent with that of the American Congress of Rehabilitation Medicine (2) and is a natural extension of a much earlier definition by the Congress of Neurological Surgeons, which in 1966 defined concussion as "a clinical syndrome characterized by immediate transient impairment of neural function such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces" (7a). At that time, concussion was generally thought to be fully recoverable; however, investigators later reported the occurrence of permanent brain injury with MTBIs (18, 24, 39).

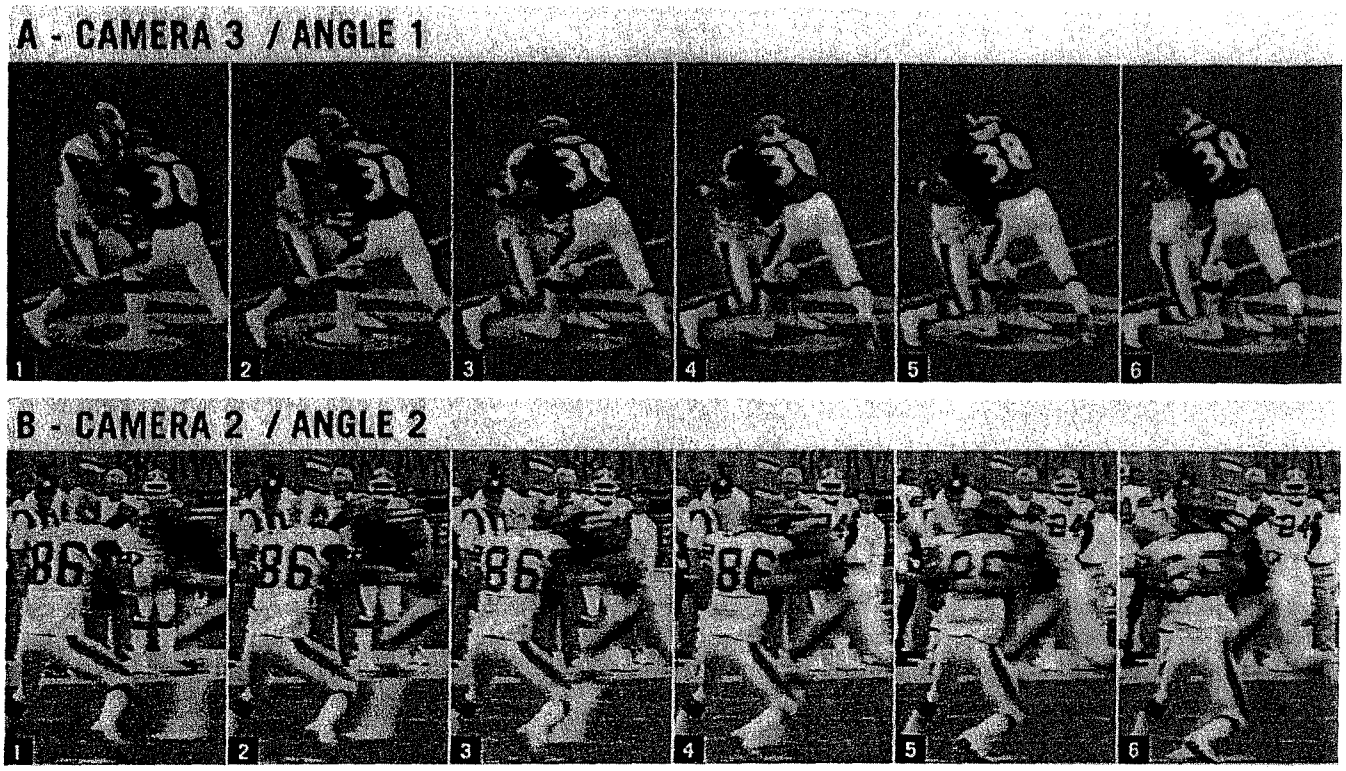
Between 1996 and 2001, MTBI cases in professional football games were systematically recorded and data on the player, the impact, the equipment worn, the initial and follow-up treatments, and the return to play were tabulated. Cases involving hospitalization were also recorded. Nearly 900 MTBI cases have been recorded and will be reported in future articles. The subset of cases with multiple-video coverage of the impact was evaluated in this study and was determined to be a representative sample of the total group of cases.

The MTBI Committee planned to perform a series of research projects aimed at defining the biomechanics of concussive impacts in professional football. After consideration of various alternatives, the effort focused on analyses of game videos of concussions. Experts proposed that, with multiple views of the impact and line markings on the field, the direction and speed of concussive impacts could be determined. This allowed laboratory reconstructions (reenactments) of the game impacts with the use of instrumented test dummies to simulate the helmeted players, closely matching the situations on the field. With transducers in the head of the dummy, the translational and rotational accelerations of the head could be determined in concussive and noninjurious impacts. This article discusses the results of the analyses of videotapes from NFL games with significant head impacts and MTBIs and the laboratory reconstructions of head biomechanical responses related to concussion.

MATERIALS AND METHODS

Overall Process for Video Analyses and Laboratory Reconstructions

When an MTBI occurred on the field during an NFL game, it was evaluated by a physician and a trainer, who completed forms describing the impact and injury. MTBIs were also reported to Biokinetics and Associates, Ltd., the engineering group contracted to analyze and reconstruct game impacts.



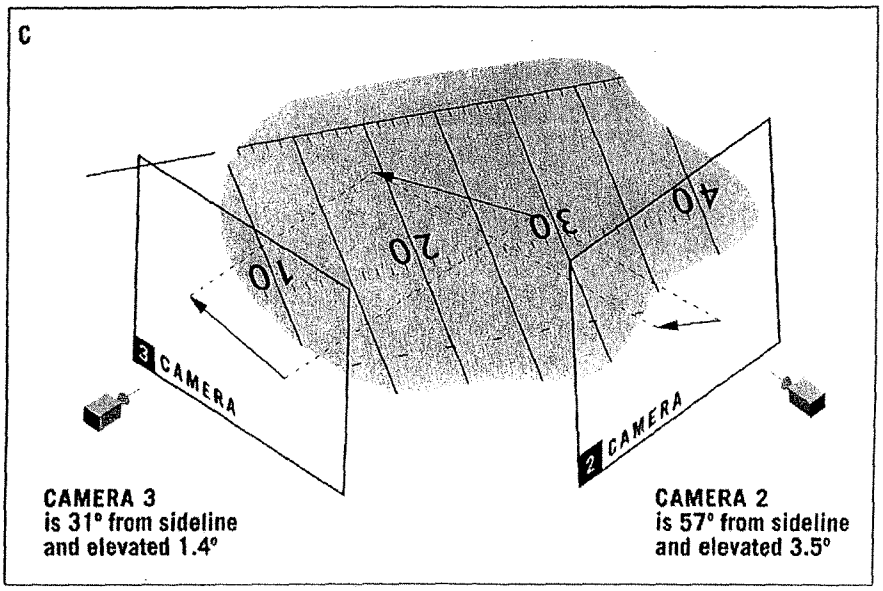


FIGURE 1. *Video analysis of game impacts, determining the velocity of helmet impact on the basis of two camera views. A and B, impact sequence from two views. C, camera locations and perspectives of the two views of the game impact.*

Network tapes of games were obtained from the NFL and analyzed. In addition to concussive impacts, other cases of significant head impacts were selected for analysis; these cases were identified on NFL films. Between 1996 and 2001, 182 cases were recorded on videotape for analysis. The initial analysis determined the helmet location involved in each impact, classified as the facemask region or the helmet shell. Also, the striking object was classified as the ground or another player's helmet, shoulder or arm, or leg, knee, or hip.

Biokinetics and Associates, Ltd., determined the feasibility of analysis and subsequent reconstruction of the impact speed. This analysis involved determining the three-dimensional impact velocity, orientations, and helmet kinematics. At least two clear views were necessary for this analysis. For videos in which the three-dimensional impact velocity could be analyzed, a laboratory model with crash dummies was prepared for reenactment of the game impact. Helmets were placed on the dummies in the laboratory reconstructions and the velocity and orientation of impact were simulated, with the subsequent helmet kinematics. A number of significant impacts in which MTBIs did not occur were also reconstructed, to establish injury risk functions for concussion. For this study, emphasis was placed on helmet-to-helmet and helmet-to-ground impacts, because the video views of other impacts were more obscured.

Video Analyses

The reconstruction of head impacts in professional football was based on video recordings made by several broadcast cam-

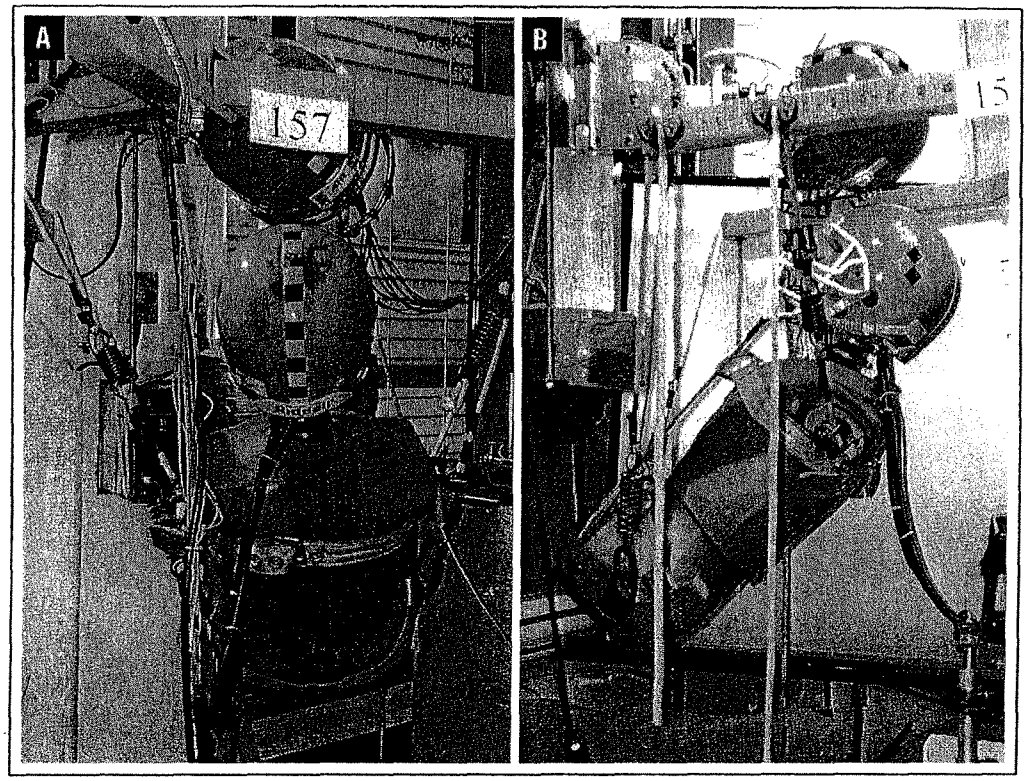


FIGURE 2. *Reconstruction of game impacts in laboratory tests with instrumented dummies. The upper torso of the struck player was supported from below, and the striking player was simulated with the head and neck attached to a 25-kg drop weight. Adjustments were made in the configuration to duplicate the helmet kinematics in a game impact.*

eras during NFL games. A cinematographic analysis was developed to determine the speed at which the players were moving, relative to each other, before colliding. The method computed the two-dimensional relative velocity in the plane of one camera view and combined it with the two-dimensional component from another camera view to yield the relative velocity vector. The method is illustrated diagrammatically in *Figure 1*, which demonstrates the principle of reconstructing the three-dimensional relative velocity of impact from two camera views. Each video view must include sufficient line markings on the field to allow determination of the camera angle and the distance from the impact. This is essential to establish a scale factor and thus determine distance and velocity. Details of the analytical approach were described by Newman et al. (30, 31).

When video recordings were examined, the orientation and distance of the cameras from the impact were not known. It was necessary to develop a method to determine the camera angles and evaluate the sequential motion of the players up to helmet contact, to determine the impact velocity (32).

Laboratory Reconstruction Techniques

Laboratory reconstructions typically involved two Hybrid III male anthropometric test devices (crash test dummies; First Technology Safety Systems, Plymouth, MI). *Figure 2* illustrates the reconstruction model. A helmeted head/neck assembly was guided in freefall from a height sufficient to achieve the same impact velocity as that determined from the video analysis of the game impact. Impact was against another helmeted head/neck assembly attached to a freely suspended anthropometric test device torso or against a simulated ground surface.

Acceleration was measured in the dummy's head. A point in space is defined by its position on three orthogonal axes. The motion of the point is defined by three orthogonal components of velocity and acceleration. The acceleration is translational, although the trajectory of the point can be curvilinear. In head impacts, the point selected for motion tracking is the head center of gravity. As the head center of gravity moves in space under translational acceleration, the head can also rotate about the head center of gravity. This involves rotational acceleration, and there are three orthogonal axes for rotational acceleration and velocity. When the head is assumed to be rigid, as is the case for the dummy, the three axes of translational and rotational accelerations define the motion sequence of the impact.

Each headform was equipped with standard accelerometers at the head center of gravity and nine linear accelerometers in a so-called 3-2-2-2 configuration, for determination of rotational acceleration (35). Processing of the nine signals determined the complete three-dimensional motion of each head. Rotational accelerations were computed from linear accelerations at various sites on the headform. The equations used to calculate rotational accelerations involved all nine accelerometers. The analysis is valid for accelerometers coincident with the origin of the system or coincident with one of the axes. Deviations from those positions were required for the headform configurations used in these tests, and corrections for centripetal and Coriolis accelerations were performed as described by DiMasi (8).

Two high-speed videos recorded head kinematics in the reconstructions. The cameras were positioned just as in the views from the game coverage. This allowed one-to-one comparisons of the game and reconstruction kinematics and facilitated fine adjustments in the impact orientation and alignment of the laboratory impact to closely match the helmet kinematics in the game situation. *Figure 3* presents an example

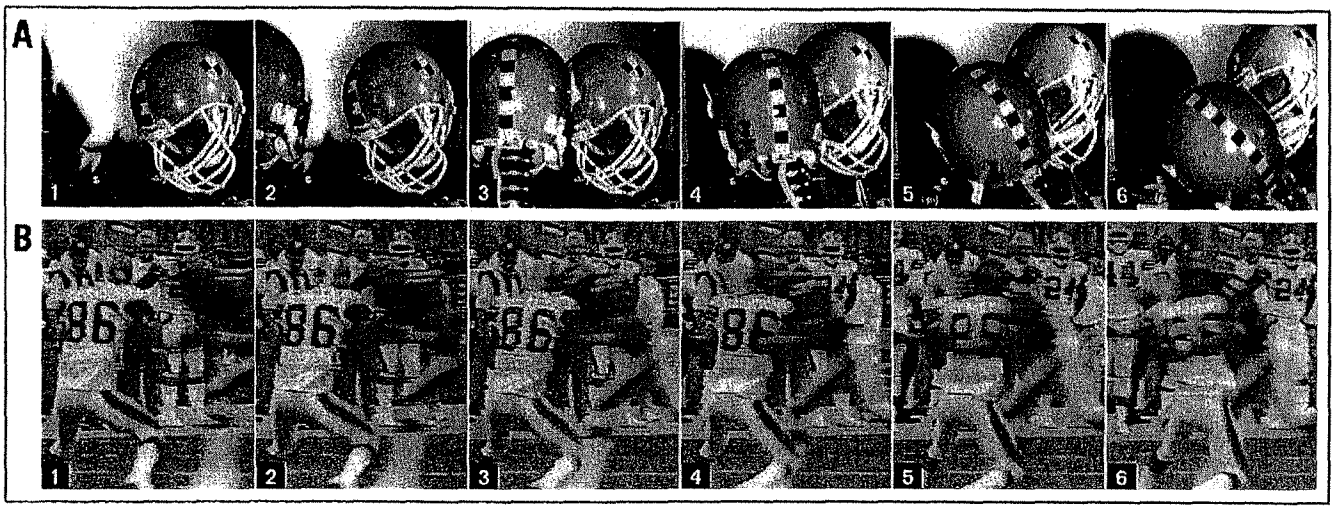


FIGURE 3. *Comparison of the laboratory reconstruction (A) and game impact (B) for a concussion case. Helmet kinematics were refined in the laboratory test until they duplicated the game impact sequence.*

of the high-speed video from a laboratory reconstruction, with a video view similar to that of the game impact causing concussion. Details of this approach were described by Newman et al. (30, 31).

Error Analyses

The Hybrid III dummy was developed for assessment of injury risks in automotive crash tests (4). It typically does not "wear" a helmet, although earlier studies used it for evaluation of head and neck injury risks in football. Two categories of potential error were investigated. The first involved error sources specific to the use of crash test dummy instrumentation (accelerometers) and associated data-processing techniques to determine the translational and rotational head accelerations. The second involved the use of broadcast video data and crash test dummies to replicate the complex head impacts in whole-body collisions between football players. Details of the validation studies and error analyses were described by Newman et al. (32).

All acceleration measurements and processing techniques were based on well-established Society of Automotive Engineers practices and standards. Potential errors in the translational accelerations were attributable to electrical or mechanical noise but did not exceed 2.2% for the three noise sources investigated. Potential errors in the rotational accelerations could be as high as 6.7%, because of error accumulation with multiple linear acceleration measurements.

The accuracy of the video analysis method for determining the camera angles and the players' relative helmet velocities was examined by analyzing staged events in which the values of the computed parameters were compared with direct measurements of the same parameters. Access to the stadium of an NFL football team was obtained and a broadcast video crew collected recordings of a series of events in which the relative velocities of two helmeted subjects could be directly measured.

After positioning, the locations of the cameras required for video analysis were surveyed in relation to a regulation football field. This information was used to create a computer-aided design representation of the field and camera orientations. A high end-zone camera and a high 50-yard-line camera were selected for the analysis. With the computer-aided design representation, the angle of the camera line of sight, relative to any point on the field, could be determined.

Three events at different locations on the field were videotaped. The events involved helmeted subjects moving past each other. Reproducible constant velocities were obtained by having the subjects drive small utility vehicles. The speed of these vehicles was measured directly. Tape switches were placed on the ground with a known separation, and a stopwatch was used to measure the time between activations of the switches as the vehicles drove over them. The potential error in the relative impact velocity between colliding heads could be as high as 11.3% and was observed to be the largest error source among the techniques used to reconstruct the game impacts. However, this error is a theoretical maximum, and precautions were taken to reduce potential errors during the reconstructions.

The overall accuracy of the reconstruction method was determined by using four different staged impacts. The error for a typical reconstruction was less than 15% of the peak values. Despite the complexity of the complete reconstruction method, including establishing impact locations, directions, and velocities from 30-Hz video data, measuring translational accelerations of crash test dummies representing the athletes in game impacts, and processing the data with combinations of translational and rotational head accelerations, the overall accuracy was well within the generally accepted ranges of accuracy and reproduc-

ibility for research of this type, and the error was much less than the theoretical maximum (32).

Biomechanical Responses and Injury Risk Functions

The primary response of the head is the resultant translational acceleration of the head center of gravity. This acceleration was determined from three orthogonal accelerations measured in the dummy. Although translational acceleration was measured in units of meters per second squared, it was reported in grams, with the measured acceleration being normalized to the acceleration of gravity (1 g = 9.8 m/s^2). Integration of the resultant acceleration yielded the change in the velocity of the head during impact.

Lissner et al. (21) developed the Wayne State University concussion tolerance curve, relating the tolerable peak translational acceleration of the head to pulse duration. The early work emphasized cranial fractures, because concussions occurred in 80% of the clinical cases with fractures. The tolerance curve was determined from cadaveric head drops onto a flat steel plate, causing high-acceleration impacts of 1- to 6-millisecond duration (*Fig. 4*). Animal experiments were conducted with dynamic pressure on the brain causing injury (12). Similar tests in cadavers established 6- to 10-millisecond acceleration tolerance levels. Tolerance to longer-duration accelerations was estimated from human volunteer test results reported by Stapp (41), which established a 42-g asymptote. However, Patrick et al. (36) suggested a higher asymptote of 80 g. Progress in understanding head injury biomechanics since the 1960s has been summarized (19, 29, 44).

The NOCSAE football helmet standard (27) uses the resultant translational acceleration measured in a biofidelic headform placed in the helmet. The helmet is dropped onto a stiff flat rubber pad from a height of 1.52 m (5 feet), yielding an impact velocity of 5.47 m/s (12 miles/h). Several drops are performed, with impact sites around the periphery and crown of the helmet. For each impact, the SI is calculated by using the method of Gadd (9),

$$SI = \int_{t}^{T} a(t)^{2.5} dt \qquad (1)$$

where $a(t)$ is the resultant translational acceleration at the head center of gravity and T is the duration of the acceleration pulse. SI depends on the time history of the resultant translational acceleration. For the helmet to be acceptable, the SI at each impact site cannot exceed 1200.

The National Highway Traffic Safety Administration uses a variation of SI to assess head safety in car crashes. The head injury criterion (HIC) has been in effect since 1975 and is a variation of SI. It is determined as

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)]^{2.5}\}_{max} \qquad (2)$$

where t_1 and t_2 are determined to give the maximal value for the HIC function and $a(t)$ is the resultant translational acceleration of the head center of gravity. In practice, a maximal limit of $T = t_2 - t_1 = 15$ milliseconds was used.

The second type of biomechanical response of the head involves rotational acceleration and velocity. Many researchers have speculated that rotational acceleration is a key response associated with head injury (10, 13, 17, 34); therefore, it was measured in the game impact reconstructions.

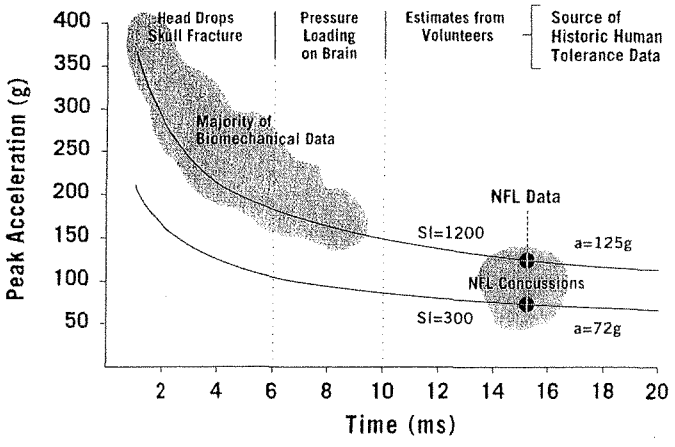


FIGURE 4. *Peak head acceleration (a) and impact duration for head injuries. The historical data involve early biomechanical experiments on cranial fractures, intracranial pressure loading, and estimates of longer-duration exposures. NFL concussions provide unique biomechanical data.*

Concussion Risk Functions

Concussion risk functions were computed by using the logist function in the Statistical Analysis System program (SAS Institute, Cary, NC). This function relates the probability of injury, $p(x)$, to a response parameter x on the basis of a statistical fit to the sigmoidal function $p(x) = [1 + \exp(\alpha - \beta x)]^{-1}$, where α and β are parameters fit to the responses from the laboratory reconstructions of game impacts. The goodness-of-fit was quantified with the -2 log-likelihood ratio parameter, P value, and correlation coefficient (r).

Verification of MTBIs

The concussion cases reconstructed in the laboratory were independently verified as MTBIs by two members of the MTBI Committee (NFL team physicians) who reviewed the clinical information. The clinical information reviewed originated from three sources, i.e., team physician forms, athletic trainer forms, and direct contact of the member of the MTBI Committee with the medical staff for the injured player (independent verification).

Statistical Analyses

The significance of differences in responses for players struck and concussed, compared with those not injured and striking players, was determined by using the standard *t* test, assuming equal variance and a single-tail distribution. The *t* test was performed by using the standard analysis package in Excel (Microsoft, Seattle, WA). The regression analysis from Excel was also used, which determined the average and 95% confidence interval for a linear fit of response data.

RESULTS

Analyses of NFL Game Impacts

Table 1 presents the 182 cases of concussive and severe helmet impacts in NFL games. The impact was indeterminate in 8 cases, leaving 174 cases for analysis. Twenty-nine percent of the head impacts involved loading of the facemask, with the remainder on the helmet shell. Sixty-one percent of the cases involved impact by another player's helmet, with 16% involving impact by the shoulder pads or arm. Ground impacts occurred in 16% of the cases.

Table 2 presents the 31 impacts reconstructed in laboratory tests with Hybrid III dummies. The categories of impact types represent 78% of the video analysis cases. The cases were limited to helmet or ground impacts, with a slightly greater proportion of facemask impacts than occurred in the overall video sample.

Figure 5 indicates the points of helmet contact in the reconstructions for cases of concussion in NFL games. *Figure 5A* indicates the locations for the struck players who were either concussed or not, and *Figure 5B* indicates those for the striking players. All of the impacts are shown on the right side of the helmet to demonstrate the impacts, although the contacts occurred on both sides of the helmet. More than one-half of these impacts involved the facemask or the area where the facemask is attached to the helmet shell for the struck player, whereas virtually all impacts involved the front crown of the helmet for the striking player.

There were 25 concussions in the 31 videos analyzed for closing speed at impact. On offense, six quarterbacks, five wide receivers, three tight ends, one flanker, and one split end were involved in helmet impacts leading to concussion. As these players focused on throwing or catching the ball, a defensive player closed at an angle to tackle the player or disrupt the play. On defense, two cornerbacks, two linemen, and one linebacker were involved in high-speed collisions during tackling. On special teams, one ball carrier and three players from the kicking or receiving units were involved in open field impacts. In some cases, players from the same team inadvertently collided during tackling; in other cases, there was clear targeting of the struck player.

Laboratory Reconstructions of Game Impacts

Table 3 summarizes the results from 31 laboratory reconstructions of game impacts, including the biomechanical responses of struck players who were concussed or not concussed and the responses of striking players. Blank rows for the striking player indicate that the reconstruction was of a ground impact, for which there was only a struck player in the reconstruction. There were 25 struck players who experienced concussions from helmet impacts and 6 who were struck but not injured. No striking player experienced a concussion. In four cases, the struck player fell to the turf; therefore, there was no striking player in the reconstruction with a helmet impact to the ground. In all of those cases, the player fully flexed his neck while falling backward, so that the impact was on the back of the helmet, at a downward angle of 30 degrees toward the neck.

The typical impact involved a player running toward another player, who was generally unaware of the closing angle. With concussion, the average impact speed was 9.3 ± 1.9 m/s (20.8 ± 4.2 miles/h), as determined in video analyses of game impacts. The striking player lined up his head, neck, and torso and struck the other player. The strike was oblique on the facemask or facemask attachment to the helmet, usually below the head center of gravity, or on the side of the helmet, above the head center of gravity. Because the struck player was unaware, only his head was initially involved in the impact.

TABLE 1. Number of facemask or helmet impacts by striking object in 182 cases of significant head impact and concussion in National Football League games

Player struck on the	No. impacted by					
	Helmet	Ground	Shoulder/arm	Hip/knee/leg	Torso	Total
Facemask	45	1	3	1	1	51
Helmet	62	27	24	8	2	123
Subtotal	107	28	27	9	3	174
Indeterminate						8
Total						182

TABLE 2. Number of facemask or helmet impacts in laboratory tests of game impacts and concussion biomechanics in professional football

Player struck on the	No. impacted by:					
	Helmet	Ground	Shoulder/arm	Hip/knee/leg	Torso	Total
Facemask	17	0	0	0	0	17
Helmet	10	4	0	0	0	14
Total	27	4	0	0	0	31

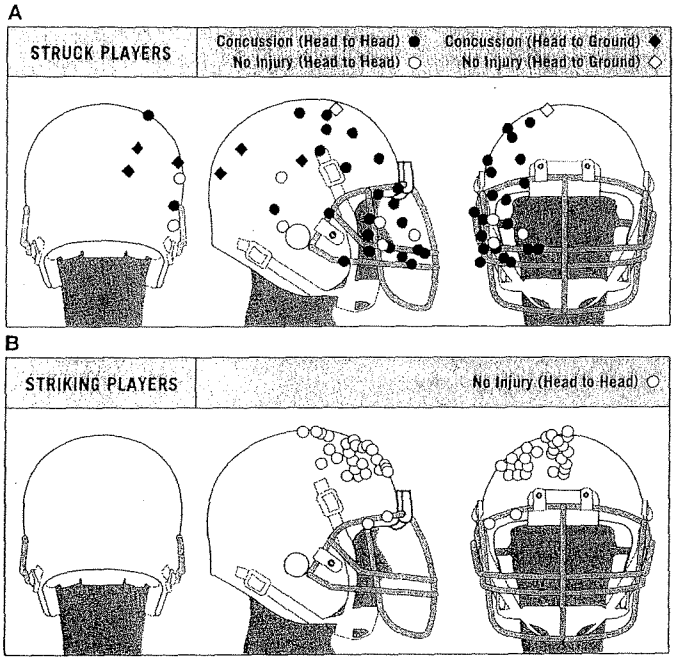


FIGURE 5. *Locations of initial helmet contacts for the struck players (including both concussed and nonconcussed impacts and falls to the ground) (A) and striking players (none of whom were concussed) (B). All of the strikes are shown on the right side of the helmet to demonstrate the impacts, although the game impacts occurred on both sides of the helmet.*

Because this yielded a higher effective mass for the striking player, more momentum was transferred to the struck player. This caused a rapid change in head velocity of 7.2 ± 1.8 m/s (16.1 ± 4.0 miles/h), which was statistically greater ($t = 2.9$, $P < 0.005$) than the 5.0 ± 1.1 m/s (11.2 ± 2.5 miles/h) for uninjured struck players. The striking players experienced a head velocity change of only 4.0 ± 1.2 m/s (8.9 ± 2.7 miles/h), which was a reduction in head velocity from the initial striking speed. The peak head acceleration for concussed players averaged 98 ± 28 g, with 15-millisecond duration, and was significantly greater than the 60 ± 24 g for uninjured struck players ($t = 3.1$, $P < 0.005$).

Figure 6 summarizes the peak biomechanical responses from the reconstructed game impacts. The average values are presented for struck concussed players, players struck without injury, and striking players. Concussion was primarily related to translational head acceleration resulting from helmet impacts to the facemask at an oblique or lateral angle, strikes by other body regions (such as the shoulder pads or knee), and falls to the back of the head. Concussion was strongly correlated with SI ($t = 3.0$, $P < 0.005$), HIC ($t = 3.2$, $P < 0.005$), peak translational acceleration, and head velocity change. There was a lower correlation with peak rotational acceleration ($t = 2.7$, $P < 0.01$) and no correlation with peak rotational velocity. The conventional measures of head injury risk, namely, SI and HIC, were effective in assessing concussion risks. On the basis of the responses measured, nominal tolerance levels for concussion were SI of 300 and HIC of 250 for helmet impacts.

Table 4 summarizes the results of the logist analysis of injury risk functions. The primary results are for the struck players, because they experienced nominally similar impacts. Results are also given for all players involved, mixing the striking players, who typically struck the other player with the crown of the helmet, with players struck on the facemask, side, or back. Because concussion risk may depend on impact orientation, the mixture of loading orientations is presented only for completeness. The strongest correlations with concussion involved HIC and SI, followed by head velocity change and translational acceleration.

Figure 7 presents the average head translational and rotational acceleration time histories for concussed players, players struck without injury, and striking players. These findings demonstrate the 15-millisecond duration of impact and typical levels causing concussion. A 2-g trigger was used to establish time 0 in the helmet impacts. The peak in the average time history was lower than the maximal values in *Table 3* because of differences in the timing of peak values. However, there was considerable similarity among the responses within each group and between the responses for nonconcussed struck and striking players.

Figure 8A presents the head velocity change versus peak translational acceleration for concussed and nonconcussed struck players and striking players. Head velocity changes increase with translational acceleration, and there was good delineation from impacts without concussion for impacts of more than 7 m/s and 90 g. On the basis of velocity changes, energy transfer to the head was calculated by assuming a Hybrid III head mass of 4.55 kg. Concussion occurred with an

TABLE 3. Peak biomechanical responses for struck and striking players in 31 National Football League game impact reconstructions[a]

Case	Struck players								Striking players						
	MTBI	Velocity (m/s)	SI	HIC	Peak translational acceleration (g)	Peak velocity change (m/s)	Peak rotational acceleration (rad/s^2)	Peak rotational velocity (r/s)	MTBI	SI	HIC	Peak translational acceleration (g)	Peak velocity change (m/s)	Peak rotational acceleration (rad/s^2)	Peak rotational velocity (r/s)
7	Yes	6.9	120	93	61	4.6	6266	28.1	No	65	51	50	2.2	2832	9.8
9	Yes	10.3	843	600	134	10.1	7428	27.4	No	275	217	79	2.3	6719	18.7
38	Yes	9.5	736	554	118	9.7	9678	50.8	No	157	127	60	4.0	5205	28.2
39	Yes	10.9	656	522	129	8.4	5921	36.1	No	60	43	44	2.3	4487	10.4
57	Yes	8.8	253	206	77	6.0	6514	37.0	No	48	38	32	4.1	4151	33.2
67	Yes	8.1	756	632	135	8.0	5957	13.8							
69	Yes	10.3	177	153	61	5.0	4381	19.9	No	55	50	38	3.1	2620	23.0
71	Yes	10.3	658	510	123	7.3	5400	35.0	No	512	434	102	6.6	5541	32.4
77	Yes	9.9	226	185	80	5.2	5148	36.4	No	65	53	35	4.2	2714	25.5
84	Yes	9.4	276	222	82	6.3	9193	80.9	No	96	78	45	4.4	3169	26.5
92	Yes	11.1	630	508	107	10.0	6878	44.2	No	204	164	60	5.6	6070	43.0
98	Yes	9.6	351	301	91	6.2	7548	43.4	No	241	187	84	4.8	4487	38.5
113	Yes	7.0	163	140	59	5.1	3965	12.8	No	101	75	61	3.7	3700	31.2
118	Yes	10.7	492	378	101	9.6	7017	42.9	No	122	73	56	3.7	3687	23.4
123	Yes	6.3	866	730	121	8.3	4727	30.3							
124	Yes	11.4	380	282	81	7.5	7138	34.8	No	105	73	56	3.1	4086	16.1
125	Yes	11.7	817	633	113	9.1	7716	63.3	No	132	111	47	4.2	3366	28.1
133	Yes	6.0	648	557	113	8.4	5012	16.0							
135	Yes	10.0	751	566	138	8.6	7540	41.0	No	230	179	81	3.8	5005	29.3
148	Yes	6.6	117	99	48	5.1	3476	23.9	No	47	37	33	3.9	2466	26.5
155	Yes	9.1	418	341	100	6.6	6940	37.0	No	76	61	45	4.2	4217	29.5
157	Yes	10.8	545	472	103	8.1	6750	33.5	No	215	180	79	5.0	4662	15.7
162	Yes	5.5	94	77	52	4.2	2615	18.4	No	34	30	29	3.2	1672	17.2
164	Yes	10.8	451	370	124	6.0	9590	26.6	No	243	202	89	5.1	6136	30.8
181	Yes	11.7	423	382	93	7.1	8011	36.5	No	402	333	85	7.3	6613	55.8
Concussed															
Average		9.3	474	381	98	7.2	6432	34.8							
SD		1.9	252	197	28	1.8	1813	15.2							
48	No	9.7	155	130	57	4.7	5617	42.4	No	44	37	32	3.2	2939	28.0
59	No	5.3	205	138	82	5.6	5387	26.9	No	32	26	32	2.3	2087	13.1
142	No	3.1	12	9	19	2.9	1170	7.4							
154	No	6.6	136	114	53	5.1	4167	24.0	No	35	31	29	3.1	3159	23.1
175	No	9.6	158	125	62	5.6	3555	39.2	No	81	62	47	3.9	2535	19.3
182	No	8.1	256	208	85	5.9	5512	17.8	No	272	213	87	4.7	3206	27.2
No injury															
Average		7.0	154	121	60	5.0	4235	26.3		146	117	56	4.0	3983	26.1
SD		2.6	82	64	24	1.1	1716	13.1		121	101	22	1.2	1402	10.0
t statistic		2.45	3.04	3.16	3.10	2.91	2.69	1.26		6.05	6.14	6.03	7.56	5.47	2.46
P value		0.0104	0.0025	0.0018	0.00215	0.0034	0.0059	0.1089		9.0E−08	6.5E−08	9.9E−08	4.0E−10	7.1E−07	0.0088

[a] MTBI, mild traumatic brain injury; SI, severity index; HIC, head injury criterion; SD, standard deviation.

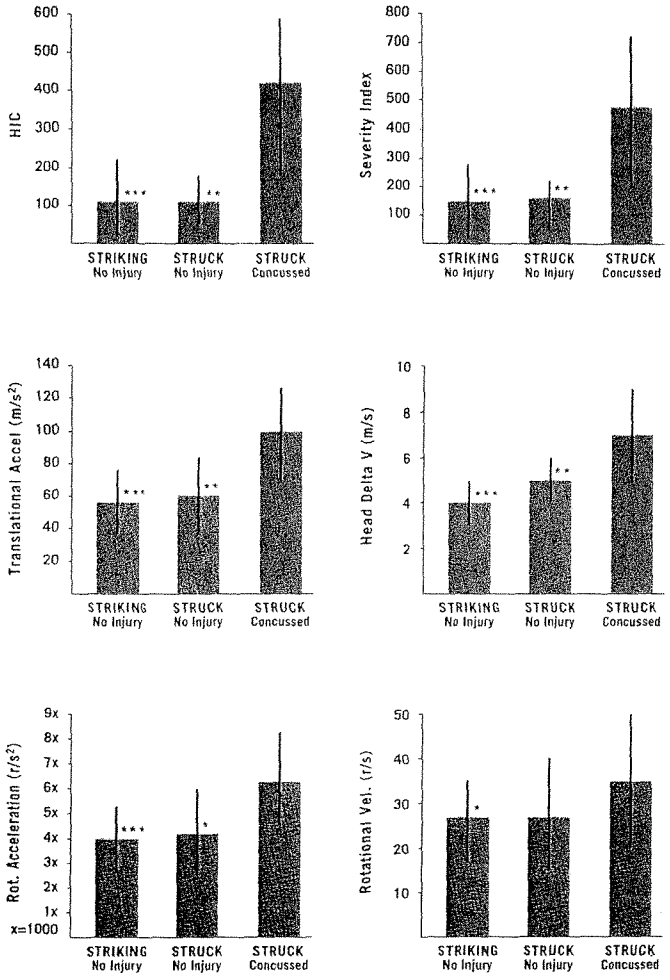


FIGURE 6. *Peak values (average ± 1 standard deviation) of key biomechanical responses in the reconstruction of game impacts, including the translational and rotational* (Rot.) *acceleration* (Accel) *and velocity* (Vel.) *and functions used to assess injury risks.* ***, $P < 0.001$; **, $P < 0.005$; *, $P < 0.01$, *significant differences from concussed players.*

average of 118 J (66–184 J for ±1 standard deviation in velocity change). For players hit but not concussed, the average energy was 57 J (35–85 J for ±1 standard deviation in velocity change). *Figure 8B* presents the peak rotational velocity versus the rotational acceleration of the head. There was more overlap in the peak values for the concussed and nonconcussed players, but concussions regularly occurred at more than 7000 radians/s^2. There was no correlation with rotational velocity.

Figure 9 demonstrates the strong correlation between the peak translational and rotational accelerations in the reconstructed game impacts. Regression analysis of all impact data yielded an average slope of 47.7 radians/s^2/g (95% confidence interval, 36.8–58.6 radians/s^2/g; $t = 3.45$, $P < 0.001$) and an intercept of 1515 radians/s^2 (95% confidence interval, 636–2394 radians/s^2; $t = 8.78$, $P < 0.0001$; model fit, $r = 0.76$, $F = 77.2$, $P < 0.0001$). Because the strongest correlation of concussion was with translational acceleration, that was considered the primary response for assessment of helmet performance, with rotational acceleration closely tracking in proportion to translational acceleration.

Verification of MTBIs

The clinical data for the laboratory reconstructions of concussion were independently reviewed. Of the 25 reconstructed cases of MTBI, 23 were judged to be definite cases of MTBI. Physician data forms were the source of clinical information for 18 cases of MTBI. Athletic trainer-generated information was the source of clinical information for two cases, and independent verification was accomplished for three cases of concussion. The physician reviewers judged two cases with laboratory reconstructions to be probable cases of MTBI, on the basis of a thorough review of the clinical information available.

Detailed clinical information was available for review for 21 of 25 reconstructed cases. Memory problems (12 of 21 cases) and cranial nerve symptoms (12 of 21 cases) were the most common symptoms in the reconstructed MTBI cases. Dizziness was the most common nonspecific complaint (9 of 21 cases). The next most common symptoms were headaches (9 of 21 cases), abnormal immediate recall (8 of 21 cases), and nonspecific cognition problems/complaints (8 of 21 cases). A number of less common symptoms were also observed, and most players exhibited multiple symptoms.

DISCUSSION

A striking observation from this study is that concussion in NFL football involves an average impact velocity of 9.3 m/s (20.8 miles/h), head velocity change of 7.2 m/s (16.1 miles/h), head acceleration of 98 g, and duration of 15 milliseconds. These are exceptionally high velocities and accelerations and long duration. Because the mass of the Hybrid III head is 4.55 kg, the average peak force acting on the head is 4.4 ± 1.2 kN (980 ± 280 lb) with concussion. This is within the experimental range for cranial fractures with short-duration impacts by rigid structures (1). The helmet shell and padding are functioning well in distributing the load and lowering the risks for more serious brain injuries and cranial fractures with impacts in NFL games.

The NFL concussion data are plotted in *Figure 4*, demonstrating the relationship of those data to the existing body of biomechanical data on human tolerance to head impacts. The NFL data are unique and represent new scientific information on human tolerance. Automotive crashes typically involve impact durations of less than 6 milliseconds for head impacts with vehicle rails, pillars, and structures. Head impacts into airbags with seat belt restraints have durations of more than 40 milliseconds. The NFL results establish new information on tolerances in the 15-millisecond range, where there has been a virtual absence of scientific data on human tolerance. Historically, the acceleration tolerance for 15-millisecond head impacts has been estimated to be 42 to 80 g, on the basis of extrapolations from studies with military volunteers. The NFL

TABLE 4. Injury risk functions and statistical fits from reconstruction of helmet impacts in professional football[a]

	HIC	SI	Translational acceleration	Rotational acceleration	Velocity change	Rotational velocity
Striking and struck players						
α	2.7335	2.7366	4.8971	4.9391	7.2886	2.0220
β	0.0114	0.0092	0.0606	0.0009	1.2809	0.0582
−2LLR	47.8	48.1	50.9	54.4	41.4	72.7
R correlation	0.75	0.75	0.72	0.68	0.80	0.38
P value	0.0002	0.0002	0.0001	0.0001	0.0001	0.0220
Struck players only						
α	1.5861	1.4094	2.9223	2.3441	4.8341	0.0868
β	0.0146	0.0109	0.0559	0.0007	1.0568	0.0501
−2LLR	19.2	19.8	21.9	23.8	21.7	28.6
R correlation	0.70	0.68	0.62	0.56	0.63	0.31
P value	0.054	0.052	0.025	0.031	0.037	0.210

[a] HIC, head injury criterion; SI, severity index; LLR, log-likelihood ratio.

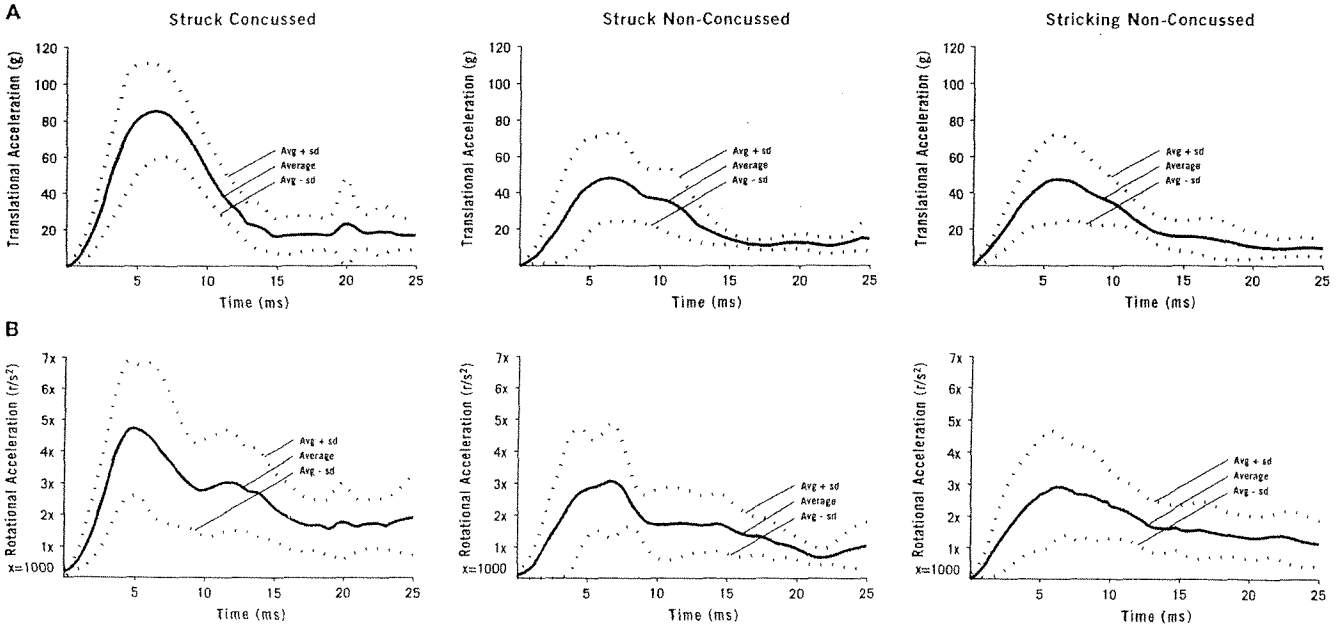


FIGURE 7. *Head translational (A) and rotational (B) accelerations (average [Avg] ± standard deviation [sd]) for concussed and nonconcussed struck players and striking players, as determined from reconstructed game impacts.*

reconstruction data suggest a value of 70 to 75 g for concussion in padded impacts, which is at the high end of the earlier tolerance ranges but is consistent with the Wayne State University concussion tolerance curve (21).

MTBIs involve translational and rotational head accelerations. The two accelerations are inextricably coupled by the vector of impact, head-neck anatomic features, and musculoskeletal structures. Although concussion exhibited the stron-

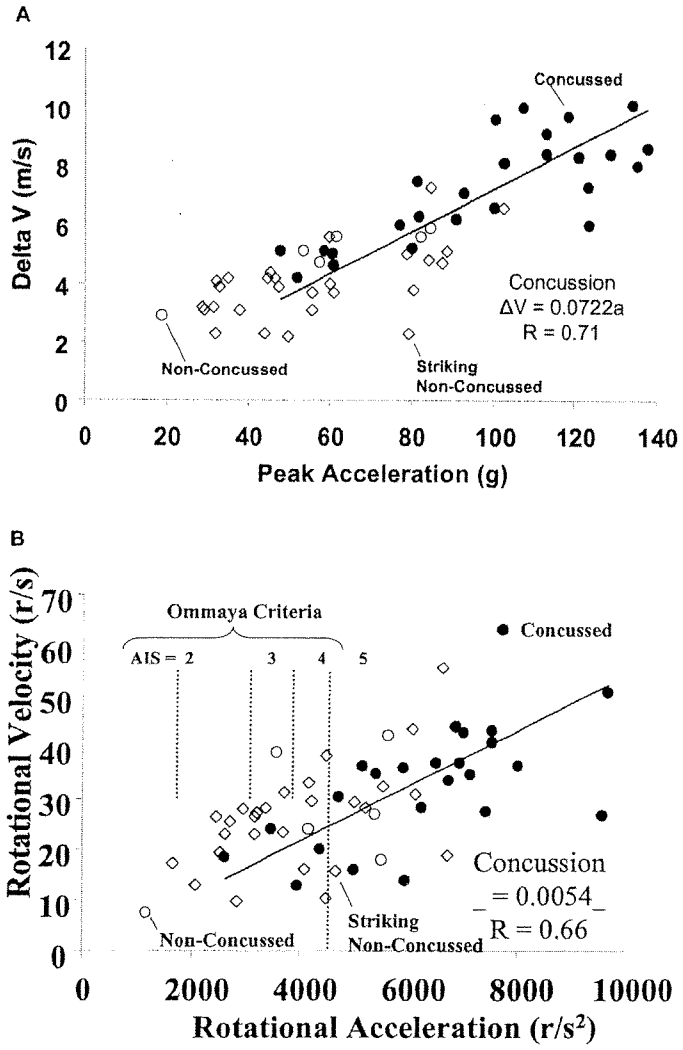


FIGURE 8. *Correlations between peak translational acceleration and velocity change* (Delta V) *(A) and peak rotational acceleration and rotational velocity (B) for helmet impacts with concussion (●) and without concussion (○) of struck players and without concussion of striking players (△).* AIS, *Abbreviated Injury Scale.*

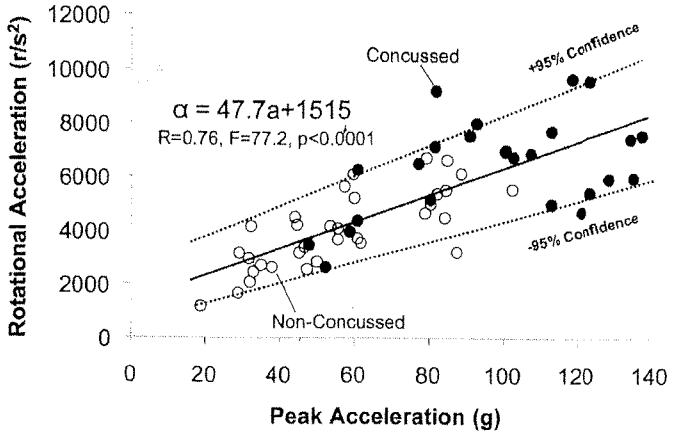


FIGURE 9. *Correlation between the peak translational and rotational accelerations in helmet impacts for concussed (●) and nonconcussed (○) players in professional football.*

gest correlation with translational acceleration, there is a strong possibility of significant effects of rotational acceleration on injury occurrence. This study cannot determine the underlying cause of concussion, except to demonstrate the stronger correlation of MTBIs with translational acceleration.

There has been much speculation that head injuries and concussion are related to rotational acceleration of the head. The helmet impacts demonstrated a strong correlation of concussion with translational acceleration, indicating that translational acceleration should be the primary measure for assessment of the performance of helmet protection systems. Moreover, *Figure 9* demonstrates the relationship between peak translational and rotational accelerations in the reconstructed game impacts. These findings indicate that efforts to reduce the peak translational acceleration, SI, or HIC involve proportional reductions in the rotational responses.

A conclusion of these studies is that the current NOCSAE SI and the more widely accepted HIC are adequate performance measures for helmet standards and that the added complexity of measuring rotational acceleration may not be needed for the first implementation of improved or supplemental NOCSAE helmet standards. However, measurement of rotational acceleration is encouraged for experimental research on concussion and helmet development, until there is a better understanding of the underlying biomechanical causes of concussion.

Ommaya (33) and Goldsmith and Ommaya (11) proposed concussion tolerance criteria based on peak rotational acceleration and head velocity. The criteria are demonstrated in *Figure 8B*. Although Ommaya indicated that the criteria were "highly speculative," on the basis of scaled primate data, the criteria are widely reported. The NFL data demonstrate tolerable exposures well beyond the proposed limits. The 4500 radians/s^2 limit is for Abbreviated Injury Scale 5 brain injuries, whereas the NFL exposures demonstrate both concussed and nonconcussed players above the proposed life-threatening injury limit. NFL impacts typically exceed 7000 radians/s^2 for concussion. Pincemaille et al. (38) demonstrated tolerable impacts with rotational acceleration of 13,600 radians/s^2 and velocity of 48 radians/s among volunteer boxers. The NFL helmet impacts do not approach the level of 13,600 radians/s^2. They also do not approach the level of 16,000 radians/s^2 proposed by Margulies et al. (22) for severe diffuse axonal injury, although even that level seems conservative on the basis of the NFL concussion data.

Experimental animal models used to study head injuries typically involve much lower velocities and higher accelerations than those experienced by the helmeted players in professional football (20). It is important that the average biomechanics of the head be replicated in animal models used to study brain injuries and treatments for concussion. Some tests have demonstrated that brain tissue injury depends on the

strain rate and the product of strain and strain rate (43). This indicates the importance of high impact velocities and large velocity changes for the neurophysiological effects of concussion. New experimental models are needed to investigate MTBI brain responses, treatment, and prevention. Such models should simulate the scaled responses for concussion in NFL football, which involve high impact velocities, large head velocity changes, high acceleration, and long duration, without cranial fractures.

Finally, new helmet designs and tests are needed to improve the protection of players against concussion. Because it has been proposed that human tolerance is lower after repeated concussions, vulnerable players might want to seek out helmets with the greatest protection. Furthermore, game rules must be strictly enforced for targeted head impacts of unsuspecting players, to reduce the number of injuries occurring on the field. The use of publicized fines for obvious cases is another way to increase attention on NFL adherence to the rules.

There are some limitations of this study. For the reconstructed cases, there were clear views of the head impact in various sequences from the game video and a firm diagnosis of concussion. This diagnosis was established after play and was verified with follow-up medical testing and treatment of the player. Every effort was made to ensure proper identification of the play, player, and injury. However, concussion Cases 148 and 162 involved unusually low-severity blows to the head. Each step in the analysis was checked for those cases, and each was determined to accurately depict the impact.

Every effort was made to ensure accuracy in determining the impact velocity and head kinematics from the various video views and in reconstructing game impacts with helmeted Hybrid III dummies. However, the helmet coupling to the Hybrid III head is a factor. In the testing, the vinyl skin of the head was covered with a nylon stocking, to ensure some sliding between the helmet liner and the head. The motion sequences of the laboratory simulations and the actual game impacts indicated that close agreement was achieved, but there are many factors in the game situations that cannot be replicated with laboratory tests using crash dummies.

For example, the Hybrid III mid-sized male dummy was used. This dummy is lighter than the typical NFL player. Also, the facemask impacts involved the chin strap as a load path to the head. In the laboratory tests, there was evidence of strain in the adjustment buckles, but the dummy has a fixed jaw. There may be compliance differences between a player wearing a mouthguard and the dummy. However, these effects are considered to be second-order influences on the head responses.

With the careful validation of each step in the methods used (32), there is assurance of accurate replication of the biomechanical responses associated with concussion in NFL football. These results provide a basis for new helmet evaluation methods, new helmet designs, and future approaches for preventing concussions in football. Another limitation of the study is that only a minority of the total number of concussions that occurred during the study years were analyzed with video analyses and laboratory reconstructions. However, the data provide a statistically relevant understanding of the circumstances associated with concussion in the NFL.

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Acknowledgments

The NFL MTBI Committee is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society and the NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the areas of traumatic brain injury, biomechanics, basic science research, and epidemiology. The authors of this article are members of the committee. The efforts of other committee members are gratefully acknowledged, including Ronnie Barnes, A.T.C., Henry Feuer, M.D., Mark Lovell, Ph.D., John Powell, Ph.D., A.T.C., and Doug Robertson, M.D. None of the committee members has a financial or business relationship that poses a conflict of interest for the research conducted on concussion in professional football. The MTBI Committee gratefully acknowledges the insight of Commissioner Paul Tagliabue in forming the committee and issuing a charge to scientifically investigate concussion and the means to reduce injury risks in football. Also, the efforts of Dorothy Mitchell, former counsel for the NFL, are most gratefully acknowledged. She worked tirelessly to initiate the MTBI research. Although she left the NFL in the middle of the program, her efforts paved the way for successful completion of the research. The encouragement and support from Jeff Pash and Peter Hadhazy of the NFL are also appreciated. The committee further acknowledges the efforts of Dr. James A. Newman, Nicholas Shewchenko, Marc C. Beusenberg, Ed Fournier, Chris Withnall, Cameron Barr, and Matthew Keown of Biokinetics and Associates, Ltd. (Ottawa, Ontario, Canada). These individuals and the organization led the effort to analyze and reconstruct the game impacts, under the direction of the committee. The committee also appreciates the contributions of all of the NFL team physicians and trainers who completed the MTBI report forms and the players who consented to participate in the surveillance via blinded identification in the MTBI database. The efforts of Dean Blandino of the NFL Officiating Department are appreciated. He identified, assembled, and made available for analysis game impact videos from NFL games from 1996 through 2001. Funding for this research was provided by the NFL and the NFL Charities. The NFL Charities are funded by the NFL Player's Association and League. Their support and encouragement for research on concussion are greatly appreciated.

COMMENTS

This research effort represents the most sophisticated attempt at analysis of the mechanisms involved in concussion in professional football to date. With the support of the National Football League (NFL) and the NFL Players Association, the authors' goal was to define the biomechanics of concussive impacts in professional football. Through the use of game video footage of actual cerebral concussions in NFL players, they biomechanically evaluated the three-dimensional impact velocities, orientation, and helmet kinematics and then took the additional step of reconstructing the game impact in the laboratory with crash dummies. By use of cinematographic analysis, the speed, direction, and velocity at which the players were moving relative to each other before colliding were determined. Of 382 cases studied, only 31 were available for complete analysis and laboratory reconstruction.

Some of the observations are not surprising. Sixty-one percent of the concussions occurred from helmet-to-helmet impact, 16% from ground impact, and 16% from shoulder pads or arms. An observation not previously reported, however, is that the primary cause of concussion involved translational head acceleration forces from helmet impact to the facemask at an oblique to lateral angle. Current National Operating Com-

mittee on Standards for Athletic Equipment (NOCSAE) standards for evaluating football helmets, however, measure primarily impacts to the periphery and crown of the helmet. Also of interest is the calculation that a concussion in NFL football involves an average impact velocity of 20.8 miles per hour (mph), a change in head velocity of 16.1 mph, and 98 g head acceleration of 15-millisecond duration. With these exceptionally high parameters, it is apparent that the existing helmet, shell, and padding are effective in the distribution of load and obviate more serious brain and cranial injuries. Another important contribution is the new scientific information the authors gleaned on human tolerance. The data accumulated by Gurdjian et al. (1) in the 1960s with regard to head impacts in cadavers and the work of Ommaya (2) on the biomechanics of head injury, as well as others, are significantly extended by the data accumulated in this study.

Although this study was limited to professional football players, the data certainly may be extrapolated to the more than 1.3 million students who participate annually in high school and college football in the United States. The call for new helmet designs to further improve the protection of players, now based on sound biomechanical data, is extremely important. Efforts have already been made by one major helmet manufacturer to redesign the helmet to diminish direct impact to the facemask and subsequent translational forces. Evaluation of the effectiveness of this endeavor is currently under way. Perhaps of greatest importance is the fact that the NFL and the NFL Players Association fully appreciate the impact of concussion not only on the sport but on the players themselves and have committed major resources to study the problem and limit the serious consequences of traumatic brain injury that, at times, is not "minimal." The authors and the engineers involved have provided a sophisticated analysis and presentation.

Joseph C. Maroon
Mark R. Lovell
Michael W. Collins
Pittsburgh, Pennsylvania

1. Gurdjian ES, Lissner HR, Evans FG, Patrick LM, Hardy WG: Intracranial pressure and acceleration accompanying head impacts in human cadavers. **Surg Gynecol Obstet** 113:185–190, 1961.
2. Ommaya AK: Biomechanics of head injury: Experimental aspects, in Nahum AM, Melvin J (eds): *The Biomechanics of Trauma*. East Norwalk, Prentice-Hall, 1985, pp 245–270.

Using both game video analysis and reconstruction of concussive events in a biomechanical laboratory with crash test dummies, Pellman et al. provide a unique and highly detailed look at the biomechanics of concussion in professional football players. As the authors show, concussive impacts occurred with an average impact velocity of 21 mph and head acceleration of 16 mph and correlated most strongly with translational acceleration. Also important was the finding that the majority of concussions occurred from strikes on the helmet facemask or on the side or back of the helmet, whereas strikes to nonconcussed players made impact on the front crown of the helmet. It may be hoped that this detailed assessment of concussion in NFL players and the ongoing efforts of the NFL Mild Traumatic Brain Injury Committee will lead to helmet design modifications and rule changes that further reduce the frequency and severity of these debilitating high-velocity injuries.

Daniel F. Kelly
Los Angeles, California

Although sports-related brain injuries frequently take place in very public arenas, their low rate of occurrence during a given game makes it difficult to study them in a scientific manner. Moreover, the emphasis during games is, quite understandably, on competition and on winning. Conducting randomized, prospective, double-blind studies is not practical in such a setting.

This fascinating report takes advantage of the fact that traumatic brain injuries sustained during professional football games are usually captured by video cameras located at different positions around the field. Cinematographic analysis of the same event filmed from different vantage points allowed the authors to calculate such parameters as head velocity at impact and rotational and translational head acceleration. Impacts that resulted in a mild traumatic brain injury were compared with other significant head impacts that did not produce brain injury. A subset of these events was reconstructed in a laboratory that used crash test dummies equipped with helmets containing multiple accelerometers. The authors found that concussion sustained during professional football games is characterized by head impact of relatively high duration, velocity, and acceleration. A significantly more rapid change in head velocity during impact was seen in concussed players as opposed to uninjured players, and peak head acceleration was also significantly higher in concussed players. Concussion was related primarily to translational acceleration, especially from impacts to the facemask, side, and back of the helmet.

Although it is reassuring that the authors' results fall within the same broad ranges as biomechanical data reported in other studies, some important discrepancies between this and previous work are of interest. For example, the strong association of concussion with translational head acceleration but not with rotational head acceleration is surprising. It would be interesting to establish whether other investigators can provide independent corroboration of these results. Alternatively, an inability to demonstrate a relationship between concussion and rotational acceleration may be attributable to limitations of the authors' methodology, some of which they discuss in detail in this article.

Pellman et al. have provided data that may offer improved helmet design for American football players, and they contribute interesting information to the existing body of knowledge about the biomechanics of concussion. Perhaps this methodology could be expanded to increase the safety of a great many

other athletic and recreational pursuits in which the participant is subjected to an increased risk of brain injury.

Alex B. Valadka
Houston, Texas

This is the first of several articles to be published in *Neurosurgery* regarding the epidemiology and biomechanics of concussion during the period 1996 to 2001 in NFL games. Pellman and his colleagues have provided a unique prospective study of concussion in our most popular televised professional sport.

Although this article addresses only 31 of 787 game-related concussions because of the requirements of two camera angles and yard markers so as to calculate collision speeds and forces, subsequent articles confirm that players at highest risk, i.e., quarterbacks, wide receivers, and defensive backs, are not unique to this open-field study.

Furthermore, although this article presents the most extensive study to date on the biomechanics of athletic concussion in football, the earpieces of race car drivers' helmets are now custom-fitted with accelerometers that are providing real-time acceleration forces imparted to the head as opposed to calculated forces imparted to the helmet. It is expected that data from the use of such earpiece accelerometers in football and other collision sports, such as boxing, ice hockey, and lacrosse, will soon become available. Again, I highly recommend this article and commend both Pellman and his collaborators for this exciting, innovative, unique study and the NFL for funding this research on a topic very critical to its athletes.

Robert C. Cantu
Concord, Massachusetts

The NFL Mild Traumatic Brain Injury Committee and the authors have performed a novel and important work. In 31 instances of on-field concussions, reconstruction of the impacts and forces involved could be determined from analysis of multiple game videotapes. Subsequent reconstruction in laboratory experiments allowed correlation between the findings on the game tapes and application in a test dummy model.

Although Schneider, Torg, and a few others have previously analyzed game films or videotapes for information concerning the mechanism of action of head and spinal injuries in football, none of these reports have provided the degree of detail with regard to the various viewing angles, allowing determination of velocity and directional vectors. In addition, a laboratory reconstruction with numerous data points is unprecedented in such a study of neurological injury. This study documents not only the changes in head velocity in concussed players but also the fact that concussion was most often related to translational head acceleration occurring at an oblique to lateral angle on the facemask or the side of the helmet or to falls on the back of the helmet. Although the authors acknowledge that the test mannequin method may have certain shortcomings—data collection was performed by use of accelerometers; the torso alone may not be representative of the entire weight and inertia of a moving human body; and the broadcast video may not accurately depict the entire data set involved in a complex collision—this work nevertheless represents our most detailed information to date.

A limitation of this study is that only a minority of the overall number of concussions during the study period were analyzed by this method, thus introducing a possible selection bias, which the authors admit and, we may hope, have mitigated by choosing a representative sample of concussed players. Further study should focus on correlating the clinical picture and experience of these players with the biomechanical aspects of these injuries. This research adds a great deal of new information regarding the velocity and acceleration forces in athletic mild traumatic head injury, human tolerance, and measures of future performance standards for helmet protection systems. It also reinforces the need for rules enforcement on the field by officials to limit or protect the unaware player against a targeted head collision. We eagerly await further study from this research group concerning their findings with subsequent analysis of their data and game material.

Julian E. Bailes
Morgantown, West Virginia

This article represents an important addition to the literature regarding closed-head trauma and concussive injury. It has been appropriately submitted to a neurosurgical peer audience for review. From the early work initiated by NOCSAE at Wayne State University to the subsequent integral contributions by Hodgson (1, 2), the establishment of quantitative analysis of headgear has resulted in significant reductions of head injuries to athletes participating in "America's sport." This is even more important given the potential that both single and repeated concussive injuries may be associated with permanent compromise (3).

The contribution of variants of linear, angular, and rotational acceleration as opposed to velocity in head injury cannot be overstated. The integration of novel models to supplant those initially devised by NOCSAE must occur so as to promote safety through a more complete understanding of the biophysics of football-related head injury.

I am not certain that the actual correlation of videotapes documenting the injury and the extent of injury is of significance. In my previous experience while associated with the football program at the University of Southern California, such video is interesting when one tries to make such correlations, yet it does not contribute to management in the short term. In addition, an important corollary of preventing such injuries must flow to participants at all levels of play, most of whom are not under the scrutiny of videotape.

Michael L. Levy
San Diego, California

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TAB 1B

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CONCUSSION IN PROFESSIONAL FOOTBALL: LOCATION AND DIRECTION OF HELMET IMPACTS—PART 2

Elliot J. Pellman, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProHEALTH Care Associates, LLP, Lake Success, New York

David C. Viano, Dr. med., Ph.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Andrew M. Tucker, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and University of Maryland School of Medicine; Timonium, Maryland

Ira R. Casson, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park, New York; and Department of Neurology, New York University Medical Center, New York, New York

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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OBJECTIVE: National Football League game video was analyzed for the typical locations of severe helmet impacts in professional football. By use of selected cases that were reconstructed in laboratory tests and reported previously, the magnitude and direction of force causing concussion was determined for these locations.

METHODS: Multiple video views were obtained for 182 severe helmet impacts that occurred between 1996 and 2001. From a top view, the helmet was divided into 45-degree quadrants with 0 degrees eyes forward. From a side view, it was divided into seven equal levels, four (+Q1 to +Q4) above the head center of gravity and three below (−Q1 to −Q3). The initial helmet contact was located in these regions. Thirty-one impacts were reconstructed with helmeted Hybrid III dummies involving 25 concussions. Measurement of head translational and rotational acceleration was used to determine the average and ±1 standard deviation in responses, with impacts reflected to the right side.

RESULTS: From video, the majority (71%) of impact is to the helmet shell primarily from a striking player's helmet, arm, or shoulder pad to the side (45–135 degrees) or from ground contact to the back (135–180 degrees). Most impacts were high on the helmet at +Q2 to +Q4. The remainder (29%) were primarily from helmet contact on the facemask at an oblique frontal angle (0–45 degrees) and −Q3 to +Q1 height. From reconstructions, concussion occurred with the lowest peak head acceleration in facemask impacts at 78 ± 18 g versus an average 107 to 117 g for impacts on other quadrants ($t = 2.90$, $P < 0.005$). There was a significantly higher head acceleration for concussed versus nonconcussed players ($t = 2.85$, $P < 0.05$). The vector of peak force was essentially horizontal for facemask impacts and downward at 12 to 27 degrees for impacts to the helmet side and back. Concussion in professional football involves four typical conditions, as follows: A, 0- to 45-degree quadrant, −Q3 to +Q3 level, peak force 49 ± 18 degrees from front and horizontal; B, 45- to 90-degree quadrant, −Q2 to +Q3 level, peak force 73 ± 12 degrees and horizontal; C, 90- to 135-degree quadrant, +Q1 to +Q4 level, peak force 97 ± 9 degrees and 12 degrees downward; and D, 135- to 180-degree quadrant, +Q1 to +Q4 level, peak force 157 ± 1 degrees and 27 degrees downward. Concussed players averaged 3.6 ± 2.7 initial signs and symptoms. The most common were headaches, dizziness, immediate recall problems, and difficulty with information processing.

CONCLUSION: The location, direction, and severity of helmet impacts causing concussion in the National Football League have been defined from analysis of game video and laboratory reconstruction. These conditions define the circumstances in which helmets need to reduce head injury risks in professional football.

KEY WORDS: Biomechanics, Concussion, Football helmets, Head injury criteria, Impact tolerances, Sport injury prevention, Traumatic brain injury

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In 1994, the Commissioner of the National Football League (NFL) formed a Committee on Mild Traumatic Brain Injuries (MTBI) in response to safety concerns regarding concussion in professional football (9). After a number of years of multifaceted research on concussion biomechanics, injury epidemiology, neuropsychological testing, brain response modeling, and treatment, the Committee has started publishing its scientific findings.

The first published study involved the initial findings on concussion biomechanics in professional football (10). It analyzed game video of concussion and severe head impacts and selectively reconstructed impacts with Hybrid III dummies to measure head accelerations with concussion. The work showed a considerable severity of concussion impacts in NFL football, with an average 9.3-m/s (20.8-mph) impact velocity, 7.2-m/s (16.1-mph) change in head velocity (head ΔV), 98 g head acceleration, and 15-ms duration. The peak force of concussion averaged 4.4 ± 1.2 kN (980 ± 280 lb) on the head.

The findings of the first study are extended by further analysis of the laboratory reconstruction data and the video database of severe helmet impacts in NFL games from 1996 to 2001. The game video showed impact locations on the helmet, which were classified by dividing the helmet into four quadrants from a top view. Data from left-side impacts were reflected to the right side of the helmet for analysis. This used 45-degree quadrant angles from the front (0 degrees eyes forward) to the rear (180 degrees). From the side, the helmet was divided into four equal levels above and three below the head center of gravity (CG).

The purpose of classifying the initial helmet contacts was to determine whether impact clusters could be defined for the most typical conditions causing concussion. This would allow the determination of average responses for the most important clusters and refine the understanding of typical conditions causing concussion in NFL players. By adding biomechanical response data, the picture of helmet impacts would be further clarified.

The aim of this work was to define conditions representing the majority of concussion in professional football so that current helmet performance can be assessed and efforts taken to reduce risks of concussion in game impacts. This would motivate improvements in helmet performance for the critical impact conditions, help the National Operating Committee on Standards for Athletic Equipment (NOCSAE) establish new standards to evaluate helmets, and improve the safety of professional football players and others.

MATERIALS AND METHODS

Overall Process

An earlier article (10) defined the research methods for obtaining and evaluating game video of concussion with helmet impacts in professional football. Briefly, network tape was obtained of games from 1996 to 2001 that included impacts causing concussion and other severe impacts. In all, 182 cases were available for analysis of the location of initial helmet contact. The helmet was divided into the facemask region and the helmet shell. The striking object was classified as the ground or another player's helmet; shoulder or arm; leg, knee, or hip; or torso.

Biokinetics and Associates, Ltd., reconstructed the impact speed for 31 cases using at least two clear video views of the collision. Laboratory tests were then set up to reconstruct or reenact the game impacts with crash dummies and measure head responses. The reconstructions emphasized helmet-to-helmet and helmet-to-ground impacts, because the video of other impacts proved to be more obscured from clear view.

Video Analysis

A cinematographic analysis method was developed to determine the actual speed at which the players were moving before colliding (5, 6). It computed the two-dimensional velocity in the plane of one camera and combined it with the two-dimensional component from another camera view to yield the relative velocity vector. An error analysis of the method found a worst-case inaccuracy of 15% in determining impact velocity (7). Because the nominal error was much less and efforts were taken to reduce or eliminate the worst-case conditions, the method was considered satisfactory.

In this study, the location of initial head contact was categorized. *Figure 1* shows the top and side views of the helmet. With eyes forward being the 0-degree reference, four quadrants from front to back of the helmet were defined, with left-side impacts reflected to the right side. The categories included 0 to 45, 45 to 90, 90 to 135, and 135 to 180 degrees. From a side view, the helmet was divided into seven equal levels. There were four levels above the head CG, +Q1 to +Q4, with each being one-quarter the distance from the head CG to the top of helmet. There were three equal levels below the head CG, −Q1 to −Q3.

Helmet contact of the struck player was categorized by impact quadrant and level for helmet contacts. The source of contact by quadrant included ground impacts and strikes by another player's helmet; shoulder pads or arm; or knee, hip, or leg. A second analysis tabulated the impact level on the helmet to determine clusters with the greatest number of severe impacts grouped by quadrant and level. Similar definitions were used for the striking players. The head CG is 20 mm (0.79 inch) above the center of the ears, or just above the eyes. The initial location of the helmet impact was classified for each struck player. Eight cases were indeterminate, leaving 174 cases for this analysis. Although there was an attempt to include video analysis of all positions involving severe head impact, the available video coverage emphasizes plays around the ball and key skill positions of quarterback, ball carrier, receivers, and secondary. There is less clear video coverage of linemen, which introduces some selection bias. However, the speed of impacts is greatest in open-field tackling and blocking, where the more severe impacts often occur and video coverage is good.

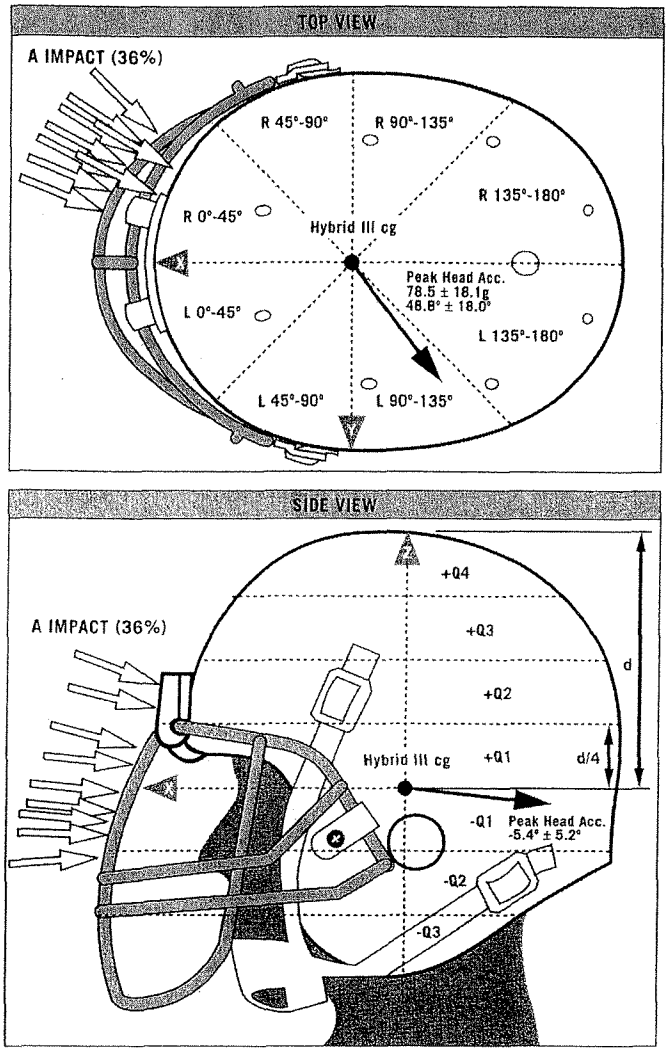

FIGURE 1. *Chart showing quadrants of impact from the top view and levels from the side of the helmet used to categorize NFL impacts from video analysis. Also shown are the results of impact Condition A with nine front-quadrant impacts on the facemask, with the average and ±1 SD in orientation and direction of maximum head acceleration of the struck player.*

Laboratory Reconstruction Techniques

Reconstruction of game impacts typically involved two Hybrid III anthropometric test devices (crash test dummies). The techniques and error analysis for these methods have been published (7, 10). Briefly, the helmeted head and neck of a dummy were guided in free fall from a height sufficient to match the velocity determined from video analysis of the game impact. Another helmeted dummy was freely suspended to simulate the striking player, or ground from the injury site was used.

Two high-speed videos recorded head kinematics in the reconstruction. The cameras were positioned identically to the views from the game video to allow a one-to-one comparison. This allowed a fine tuning of the impact orientation and alignment between the laboratory kinematics and game situation. Each Hybrid III head was equipped with standard triaxial accelerometers to record the translational acceleration of the head CG. Nine additional linear accelerometers were set up in the so-called "3-2-2-2 configuration" (8) in the dummy head to determine three-dimensional rotational acceleration using a correction for centripetal and Coriolis accelerations (1).

For analysis and clustering, all impacts were reflected to the right side of the helmet to determine average and ±1 standard deviation (SD) in responses. Impacts in each quadrant were averaged as a function of time using a 2 g trigger to establish time zero for each test. Thirty-one cases were reconstructed in laboratory tests with helmeted Hybrid III dummies. Head translational and rotational accelerations were averaged, and ±1 standard deviation was computed, for the four quadrants (0–45, 45–90, 90–135, and 135–180 degrees) involving concussed and nonconcussed players.

The average vector of maximum head acceleration (impact force) was also determined for each quadrant of helmet impact for the concussed and nonconcussed players. This is a three-dimensional vector with components angled from front to back and top to bottom of the helmet. This vector defines the peak head response, impact force, and neck loading. The information completes the definition of impacts associated with concussion in NFL games according to the quadrants from facemask to back of the helmet and impact speed, head ΔV, and acceleration response. Preliminary analysis of the data showed this to be the most representative way to cluster the impacts for future standards and testing of helmets.

Biomechanical Responses and Injury Risk Functions

The primary response of the head is the resultant translational acceleration of the head CG. Integration of the resultant acceleration gave the ΔV. NOCSAE (4) uses the Severity Index (SI) to determine safety performance. It is calculated as follows:

$$SI = \int^{T} a(t)^{2.5} dt \qquad (1)$$

where $a(t)$ is the resultant translational acceleration at the head CG, and T is the duration of the acceleration pulse (2). The National Highway Traffic Safety Administration uses a variation of SI to assess head safety in car crashes. The head injury criterion (HIC) has been in effect since 1975 and is determined as follows:

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)]^{2.5}\}_{max} \qquad (2)$$

where t_1 and t_2 are determined to give the maximum value to the HIC function, and $a(t)$ is the resultant translational accel-

eration of the head CG. In practice, a maximum limit of $T = t_2 - t_1 = 15$ ms is used.

The second type of head biomechanical response is rotational acceleration and velocity, which may be a factor in head injury and concussion. These responses are calculated about the head CG.

Clinical Aspects of MTBI

The previous article (10) discussed the verification of concussion for the reconstructed cases from NFL games. In this study, the signs and symptoms of the concussed players were grouped into five categories, including general symptoms, cranial nerve symptoms, memory problems, cognition problems, and somatic complaints. All of the commonly accepted symptoms and signs of cerebral concussion were included. These were somewhat arbitrarily divided into the above five categories to assist in data analysis and presentation. No sensory or motor abnormalities were reported in any player. The only cerebellar abnormality reported was nystagmus in a few cases. The 33 possible recorded symptoms were evaluated by impact type to determine injury patterns.

Statistical Analyses

The significance of differences in responses for players struck and concussed was determined from those not injured and from the striking players by use of the standard t test assuming unequal variance and a single-sided tail distribution. The t test was performed with the standard analysis package in Excel (Microsoft, Seattle, WA). The proportion of symptoms was calculated with standard deviations to compare differences by impact orientation.

RESULTS

Analysis of NFL Game Impacts

Table 1 shows the 182 cases of severe helmet impact in NFL games. In 8 cases, the impact was indeterminate, leaving 174 cases for analysis. Twenty-nine percent (51 of 174) of the impacts involved loading of the facemask, with the remainder on the helmet shell. All facemask impacts fell into the front two quadrants, with 67% (34 of 51) in 0 to 45 degrees. For impacts of the helmet shell, 22% (27 of 123) involved the ground, with the remainder the other player's helmet (50%); shoulder pad or arm (20%); or hip, knee, or leg (7%). In the total sample, 61% (107 of 174) of the impacts involved the other player's helmet.

Table 2 shows the impacts by quadrant and level on the struck player's helmet. Of the facemask impacts, 76% (39 of 51) were to levels −Q1 to −Q3, which are below the head CG. In contrast, 79% (49 of 62) of impacts by a helmet and 89% (54 of 61) of impacts by other body regions or ground involved loading of the shell at levels +Q2 to +Q4, above the head CG. This indicates an upward shift in impact height from the facemask to the rear of the helmet. *Table 3* shows the impacts by quadrant and level for the striking player's helmet. There were 107 impacts involving the helmet of the striking player: 57% (61 of 107) involved the +Q4 and 21% (23 of 107) the +Q3 levels, the top portions of the helmet; 48% (51 of 107) of the strikes involved the 0- to 45-degree and 30% (32 of 107) the 45- to 90-degree quadrants.

On the basis of the video analysis and clustering, the majority of helmet impacts to the struck player in NFL games involved four conditions (quadrants). These were defined as

TABLE 1. Number of facemask or helmet impacts by quadrant and striking object for 182 cases of significant head impact and concussion in National Football League games

Struck players	Impacted by:					Total
	Helmet	Ground	Shoulder, arm	Hip, knee, leg	Torso	
Facemask impacts						
0–45 degrees	31	0	2	0	1	34
45–90 degrees	14	1	1	1	0	17
Subtotal	45	1	3	1	1	51
Helmet shell impacts						
0–45 degrees	8	1	4	0	2	15
45–90 degrees	23	4	7	4	0	38
90–135 degrees	22	4	7	1	0	34
135–180 degrees	9	18	6	3	0	36
Subtotal	62	27	24	8	2	123
Total	107	28	27	9	3	174
Indeterminate						8
Total						182

TABLE 2. Number of facemask or helmet impacts by quadrant and level of initial contact of the struck player in National Football League games

Struck players	Impact height							Total
	−Q3	−Q2	−Q1	+Q1	+Q2	+Q3	+Q4	
Facemask impacts								
By helmet								
0–45 degrees	14	2	10	2	3	0	0	31
45–90 degrees	0	4	4	6	0	0	0	14
Subtotal	14	6	14	8	3	0	0	45
By other								
0–45 degrees	2	1	0	0	0	0	0	3
45–90 degrees	0	1	1	1	0	0	0	3
Subtotal	2	2	1	1	0	0	0	6
Helmet shell impacts								
By helmet								
0–45 degrees	0	0	0	0	3	5	0	8
45–90 degrees	2	1	0	0	6	12	2	23
90–135 degrees	0	1	2	4	6	3	6	22
135–180 degrees	0	1	0	2	4	1	1	9
Subtotal	2	3	2	6	19	21	9	62
By other								
0–45 degrees	0	1	0	0	1	3	2	7
45–90 degrees	0	0	0	0	2	10	3	15
90–135 degrees	0	0	0	0	7	2	3	12
135–180 degrees	0	0	0	6	15	3	3	27
Subtotal	0	1	0	6	25	18	11	61
Total	18	12	17	21	47	39	20	174
Indeterminate								8
Total								182

TABLE 3. Number of facemask or helmet impacts by quadrant and level of initial contact by 107 striking players in National Football League games

Striking players	Impact height							Total
	−Q3	−Q2	−Q1	+Q1	+Q2	+Q3	+Q4	
Facemask impacts								
0–45 degrees	0	0	0	1	5	4	14	24
45–90 degrees	0	0	0	0	0	1	7	8
90–135 degrees	0	0	0	1	1	0	4	6
135–180 degrees	0	0	0	1	1	0	5	7
Subtotal	0	0	0	3	7	5	30	45
Helmet shell impacts								
0–45 degrees	1	0	1	1	3	5	16	27
45–90 degrees	0	2	0	3	0	12	7	24
90–135 degrees	0	1	0	1	0	1	3	6
135–180 degrees	0	0	0	0	0	0	5	5
Subtotal	1	3	1	5	3	18	31	62
Total	1	3	1	8	10	23	61	107

Conditions A to D, based on the initial quadrant of helmet contact. The nominal range in impact level (or height) was as follows: A, 0- to 45-degree quadrant, −Q3 to −Q1 level on facemask and +Q2 to +Q3 on helmet shell; B, 45- to 90-degree quadrant, −Q2 to +Q1 level on facemask and +Q2 to +Q3 on helmet shell; C, 90- to 135-degree quadrant, +Q1 to +Q4 levels; and D, 135- to 180-degree quadrant, +Q1 to +Q4 levels. There were two conditions, E and F, defining the quadrant and level of impact by striking players: E, 0- to 45-degree quadrant, +Q2 to +Q4 levels; and F, 45- to 90-degree quadrant, +Q3 to +Q4 levels.

Laboratory Reconstruction of Game Impacts

There were 31 impacts reconstructed by laboratory testing with Hybrid III dummies; 25 cases involved concussion. *Table 4* shows the type of concussion cases reconstructed. They were limited to helmet or ground impacts, with a higher proportion of facemask impacts than occurred in the overall video sample. They represented 78% (135 of 174) of the video analysis cases.

Table 5 summarizes the average and ±1 SD responses from the laboratory reconstruction of game impacts by impact Conditions

TABLE 4. Type of reconstructed cases of concussion in professional football

Struck players	Impacted by:					Total
	Helmet	Ground	Shoulder, arm	Hip, knee, leg	Torso	
Facemask impacts						
0–45 degrees	8	0	0	0	0	8
45–90 degrees	6	0	0	0	0	6
Helmet shell impacts						
0–45 degrees	1	0	0	0	0	1
45–90 degrees	4	0	0	0	0	4
90–135 degrees	3	1	0	0	0	4
135–180 degrees	0	2	0	0	0	2
Total	22	3	0	0	0	25

TABLE 5. Peak biomechanical responses from reconstruction of 31 helmet impacts involving 25 cases of concussion[a]

Case	Velocity (m/s)	ΔV (m/s)	Translational acceleration				SI	HIC	Rotational		Resultant direction		Helmet contact	
			Linear x (g)	Linear y (g)[b]	Linear z (g)	Resultant (g)			Accel (rad/s²)	Velocity (r/s)	Front to back (deg)[2c]	Up + down (deg)	Top view[d]	Side view median[e]
25 Concussed struck players														
A Condition 36%														
Average	9.8	6.6	−50.9	53.9	−7.3	78.5	311	257	6120	35.7	48.8	−5.4	R 0–45	−Q1 to +Q2
SD	1.8	1.6	25.6	15.7	7.0	18.1	154	129	1964	19.4	18.0	5.2		
B Condition 40%														
Average	9.6	7.6	−27.6	100.1	−10.7	107.6	558	431	6730	38.9	72.6	−6.1	R 45–90	−Q1 to +Q3
SD	1.7	1.9	19.6	27.8	14.7	26.1	260	188	1854	12.2	12.1	8.0		
C Condition 16%														
Average	9.2	7.2	8.2	101.3	−25.8	106.8	489	401	7173	28.2	96.5	−12.4	R 90–135	+Q1 to +Q3
SD	2.0	2.4	9.3	32.3	22.5	32.7	276	231	1656	12.5	8.8	11.7		
D Condition 8%														
Average	6.1	8.4	96.1	41.3	−52.4	117.1	757	644	4870	23.2	156.7	−26.7	R 135–180	+Q2 to +Q3
SD	0.2	0.1	8.6	1.1	4.0	5.7	154	122	202	10.1	1.3	3.6		
6 Nonconcussed struck players														
A Condition														
Average	8.6	5.1	−33.9	44.6	0.7	57.5	150	123	4446	35.2	53.4	0.5	R 0–45	−Q1
SD	1.8	0.5	13.0	5.2	8.2	4.1	12	8	1059	9.8	13.6	7.9		
C Condition														
Average	5.5	4.8	11.8	55.8	−1.4	61.9	158	118	4023	17.4	117.0	−6.9	R 90–135	
SD	2.5	1.7	19.4	42.6	8.0	37.3	129	101	2472	9.8	37.5	14.2		
27 Nonconcussed striking players														
E Condition 59%														
Average	8.9	3.7	−27.9	23.6	−20.9	48.3	112	90	3470	22.8	35.5	−27.4	R 0–45	+Q2 to +Q4
SD	2.0	1.1	23.4	16.3	16.2	21.8	123	104	1113	7.6	22.4	18.4		
F Condition 30%														
Average	10.2	4.4	−1.9	10.9	−23.5	70.4	211	169	4629	29.6	57.6	−22.3	R 45–90	+Q3 to +Q4
SD	1.2	1.6	34.7	61.7	17.7	20.8	117	96	1651	12.7	28.8	21.7		
Other conditions 11%														
Average	9.2	4.4	11.7	32.7	−37.5	60.1	154	122	4992	34.4	102.0	−40.4	R 90–180	+Q3 to +Q4
SD	2.1	1.0	31.5	18.5	13.0	0.6	52	45	1199	8.3	51.3	18.1		

[a] SI, severity index; HIC, head injury criterion; SD, standard deviation.
[b] The average was determined from the absolute value of the *y* acceleration.
[c] The vector is adjusted to the right side of the helmet for analysis of averages.
[d] The impacts are reflected to the right side for analysis but occurred on both sides of the helmet.
[e] This shows the typical levels of the impacts evaluated.

A to F. The components of the peak resultant translational acceleration are given, and the full data are given in the Appendix (*Tables A1* and *A2*). Twenty-five struck players experienced concussion, and 6 were struck hard but experienced no injury. The biomechanical responses from the striking players are also shown. None of the 27 striking players were concussed. In four cases, the struck player fell to the turf, so there was no striking player in the reconstruction of the helmet impacts to ground. In all of these cases, the player flexed his neck while falling backward, so the impact was high on the helmet, because the angle of fall and shoulder pads prevented a lower level of impact.

Figure 2 shows selected peak impact and biomechanical responses with ±1 SD for struck players in Conditions A to D and striking players in Conditions E and F and other. The facemask impacts in Condition A involve the highest average impact velocity but the lowest ΔV with concussion. In contrast, falls to the back of the helmet (Condition D) involve the lowest impact speed but the highest ΔV. This is a result of the closing velocity coming from only one player, and the ΔV is increased by rebound off the ground. The lowest resultant head acceleration with concussion occurred with the facemask impacts and the highest with falls to the back, followed by the side, of the helmet. Condition A was statistically lower than other concussion conditions ($t = 2.90$, $P < 0.005$). Falls to ground resulted in the lowest rotational accelerations and velocities, which were statistically similar for all conditions.

For the striking players (Conditions E and F), the impact speeds were as high as for the struck players, but the ΔV and

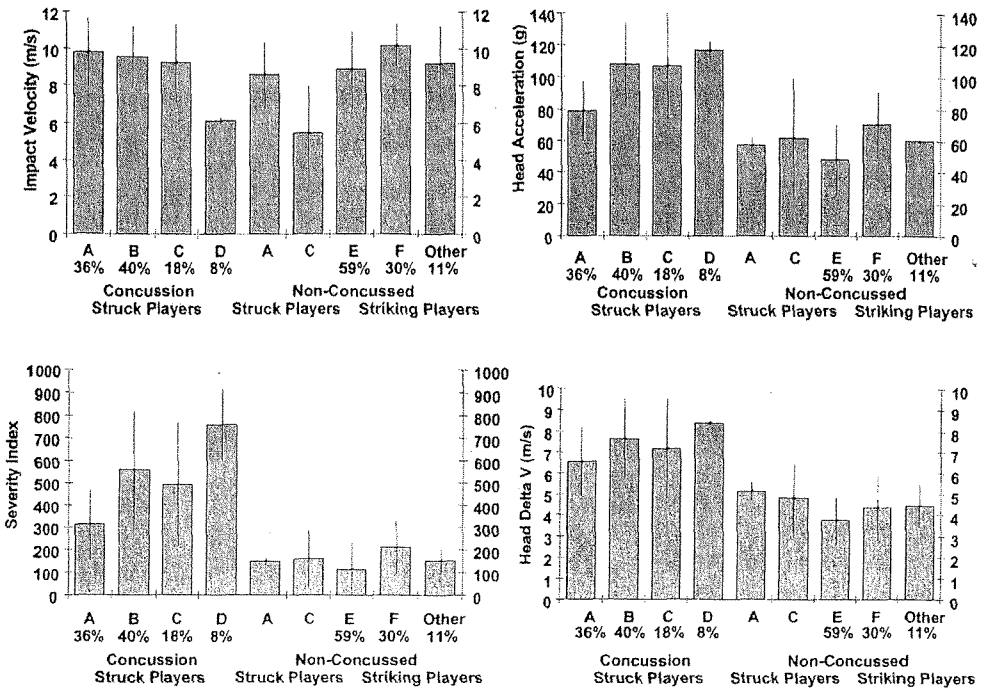

FIGURE 2. *Bar graphs showing peak impact velocity* (top left), *head acceleration* (top right), *severity index* (bottom left), *and head* ΔV (bottom right) *for the four conditions of concussive impact and nonconcussive impact, including the two conditions for the striking players. The height of each* bar *is the average, and the* vertical line *shows the* ± 1 *SD.*

peak resultant accelerations were lower than for concussion cases ($t = 2.85$, $P < 0.05$). The effective mass of the striking player is much greater than that of the struck player. The impact level is near the top of the helmet, because the striking player lowers his head to drive through the other player. In these cases, the striking player aligns his head, neck, and torso to impact the head of the other player. This gives more effective mass and energy transfer to the struck player, whose head and neck initially react to the loading. The rotational accelerations and velocities were statistically similar to those of concussed players.

The direction of maximum head acceleration (and impact force by multiplying head mass and acceleration) was summarized by the average and ± 1 SD for the vector from front to back of the helmet (related to the quadrant in the top view, with 0 degrees being eyes forward) and the angle of impact with respect to an upward or downward angle on the neck. Although impact Condition A involves the 0- to 45-degree quadrant helmet contact, the orientation of the maximum head acceleration (force on the head) is at 48.8 ± 18.0 degrees. *Figure 1* shows the eight concussion and one nonconcussion impacts for Condition A and the average head acceleration vector from the top and side view of the helmet. At peak head acceleration, the vector is outside the initial contact in the 0- to 45-degree quadrant. As force builds up on the facemask, the head rotates counterclockwise in the direction of the impact vector. This increases the lateral component of maximum acceleration and force. Facemask impacts involve a nearly horizontal direction of loading.

A similar but less significant effect is seen for the striking player, with some vectors of maximum acceleration falling outside the initial 0- to 45-degree quadrant in Condition E (*Table A2*). The opposite rotation is seen for Condition C impacts, with initial contact in the 90- to 135-degree quadrant, as the head rotates clockwise, reducing the vector angle at peak acceleration. For these impacts, there is a downward vector of load on the neck, consistent with the high initial impact on the helmet.

On the basis of the laboratory reconstructions, the majority of helmet impacts involve four conditions for struck players in the NFL. The following provides a further definition for Conditions A to D based on the quadrant and nominal level (or height) of helmet contact and nominal orientation of maximum head acceleration (force) from the reconstructed cases: A, 0- to 45-degree quadrant, −Q3 to +Q3 level, peak force 49 ± 18 degrees from front and horizontal 9.8 m/s impact velocity, 6.6 m/s ΔV, and 79 g; B, 45- to 90-degree quadrant, −Q2 to +Q3 level, peak force 73 ± 12 degrees and horizontal 9.6 m/s impact velocity, 7.6 m/s ΔV, and 108 g; C, 90- to 135-degree quadrant, +Q1 to +Q4 level, peak force 97 ± 9 degrees and 12 degrees downward 9.2 m/s impact velocity, 7.2 m/s ΔV, and 107 g; and D, 135- to 180-degree quadrant, +Q1 to +Q4 level, peak force 157 ± 1 degrees and 27 degrees downward 6.1 m/s impact velocity, 8.4 m/s ΔV, and 117 g.

There are two conditions, E and F, defining the striking players by quadrant, level (or height), and orientation of maximum head acceleration: E, 0- to 45-degree quadrant, +Q2 to +Q4 level, peak force 36 ± 22 degrees and 27 degrees downward 8.9 m/s impact velocity, 3.7 m/s ΔV, and 48 g; and F, 45- to 90-degree quadrant, +Q3 to +Q4 level, peak force 58 ± 29 degrees and 22 degrees downward 10.2 m/s impact velocity, 4.4 m/s ΔV, and 70 g.

Figure 3 shows the average and ± 1 SD head translational acceleration time history for concussed players, those struck without injury, and striking players. *Figure 4* shows the rotational acceleration time histories. Not all conditions are shown because of a lack of sufficient response data. The responses demonstrate the 15-ms duration of impact and typical levels causing concussion, which are rather similar in response among the concussed players, except for somewhat lower

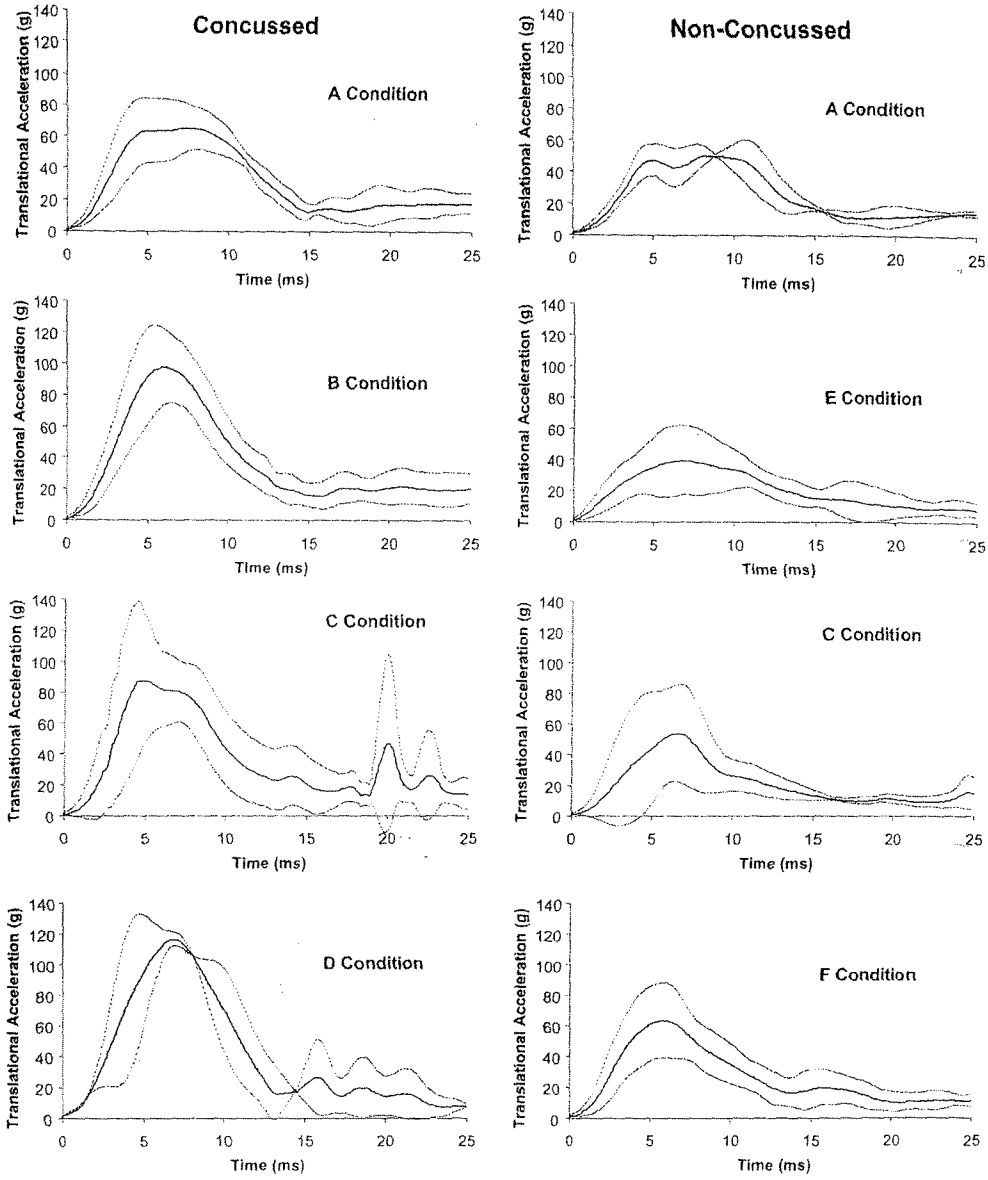

FIGURE 3. *Graphs showing average and ±1 SD in translational acceleration of the head for various impact conditions for concussed and nonconcussed players.*

values for Condition A. The peak average response in the time histories is lower than that of the individual impacts in *Table 5* because of differences in timing of peak values.

MTBI Symptoms by Impact Condition

Table 6 shows the signs and symptoms for the 25 concussed players, which were grouped by impact condition and category of injury. In four cases, no detailed information was available. On average, a concussed player exhibited 3.6 ± 2.7 signs and symptoms, with a slightly higher number for facemask and back-of-helmet impacts. The highest incidences were dizziness (43%), headache (43%), immediate recall problems (38%), retrograde amnesia (33%), and difficulty with information processing (29%). There were fewer cases of neck pain, fatigue, photophobia, and antegrade amnesia. There was no statistical difference between the proportion or type of symptoms for facemask (Condition A) and helmet-shell (Condition C and D) impacts. One player was hospitalized (Patient 135) with symptoms of dizziness, fatigue, neck pain, and somatic complaints. Two other players had 10 and 11 symptoms, respectively. One player experienced seizure.

DISCUSSION

The MTBI Committee is unaware of any study identifying the location and direction of helmet impacts associated with concussion in professional football. Also, the laboratory reconstruction of game impacts provides unique biomechanical data on head responses associated with concussion. Although the 31 laboratory reconstructions may seem a small number, they contain a very large amount of data for impact biomechanics research and represent a substantial effort to carefully match field conditions involving concussion.

The response data are clustered by quadrant from front to back of the helmet. This study shows the importance of facemask impacts at an oblique angle, with the majority of contacts below the head CG. Players seem to be aware of a pending impact when the closing angle is within ±30 degrees and take action to avoid significant helmet impacts. Outside the area of sight, the facemask impacts can be severe when the struck player is unaware of the closing speed. The facemask impacts involve the highest impact velocity, lowest ΔV, and lowest acceleration associated with concussion.

Impacts to the facemask generally twist the head while accelerating it. This increases the lateral component of acceleration. The combination of kinematics and shifting vector of acceleration may be a factor influencing concussion tolerance. Obviously, the facemask, chin strap, brow, and side padding all contribute to the

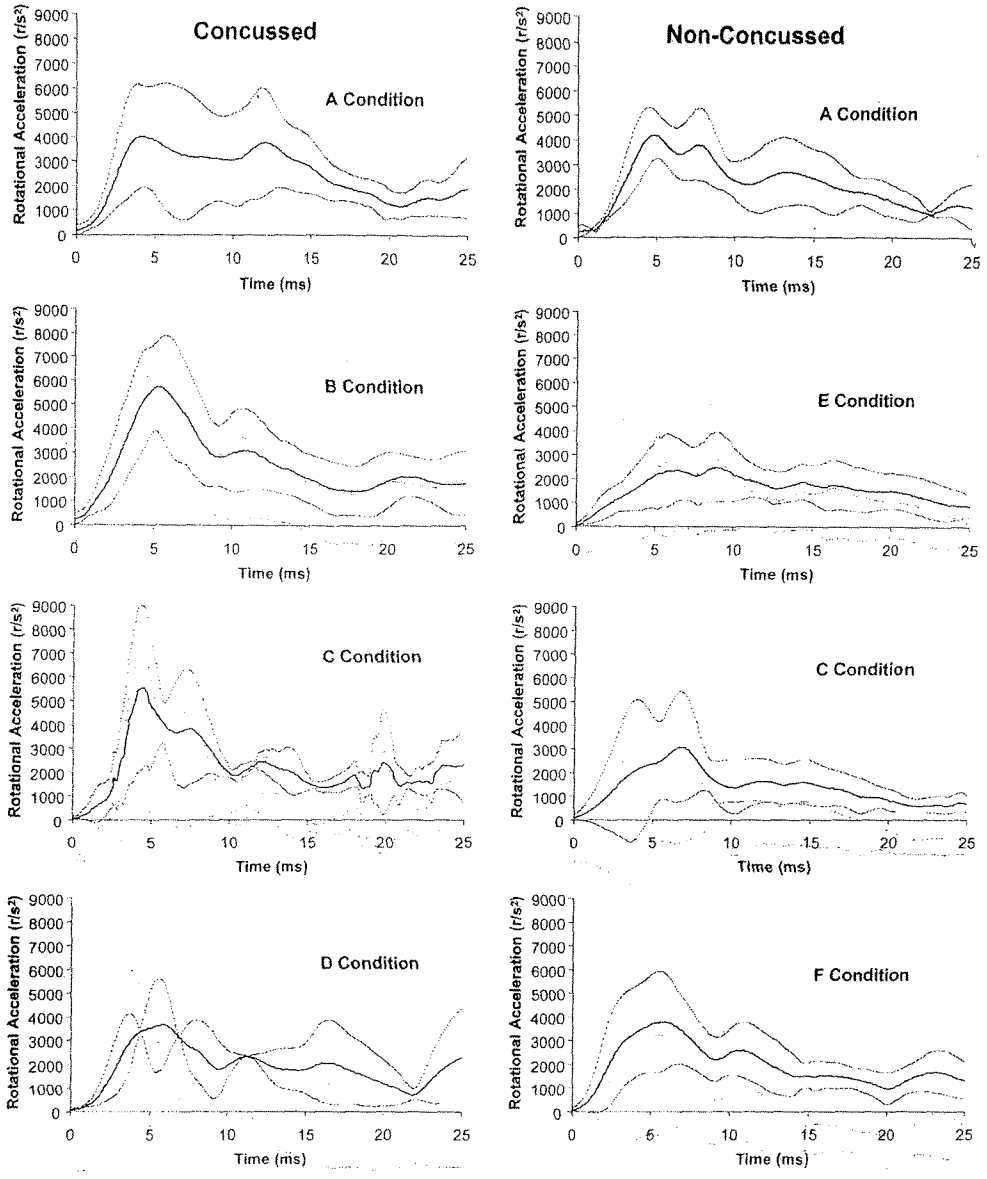

FIGURE 4. *Graphs showing average and ±1 SD in rotational acceleration of the head for various impact conditions for concussed and nonconcussed players.*

head loading. They are also the means to reduce risks in this exposure. Further study is needed on energy-absorbing designs to reduce concussion risks with oblique facemask impacts. The role of the chin strap and mouth guard in load transfer and concussion risks is unknown.

Because impacts occur on the helmet shell and toward the back two quadrants, they are primarily at levels +Q2 to +Q3, with some at +Q4 at a downward angle compressing the neck. Falls to the ground involve the highest ΔV and most severe head responses because of rebound from the ground. Obviously, the compliance of the turf is a factor, as well as the interaction between the helmet and ground. It seems that higher energy absorption may be needed for these impacts. Regarding the neck loads, there are no field data indicating a problem with any of the helmet impacts.

There was no specific finding about the role of rotational acceleration and velocity with concussion in NFL impacts. This finer analysis of the data did not identify a relevant correlation, consistent with the earlier study (10). It seems that translational acceleration remains the single most important response to address in future helmet designs. However, the measurement of rotational acceleration is encouraged in research and helmet development to further explore its potential influence on concussion.

This study describes quadrants on the helmet in which future NOCSAE standards may establish performance requirements. Clearly, head responses with facemask impacts are different from those involving the helmet shell. This aspect needs further investigation. By defining relevant quadrants, greater performance may be ensured over a segment of the helmet in which risks for concussion are high in professional football. This study defined biomechanical responses for the various impact conditions. They show a substantial severity of impact speed and head response with concussion impacts.

Table 6 was an attempt to link impact conditions with the initial clinical signs and symptoms of concussion. The small sample size for each impact condition and the extremely variable clinical nature of MTBI makes this tenuous on the basis of the information thus far accumulated. It would be of interest to associate certain impact locations with specific combinations of signs and symptoms. It would also be useful to link certain impact locations and conditions with severity of MTBI. This type of information would be useful for future helmet design changes. Also, with reference to *Table 6*, the one case of seizure is uncommon with concussion (3).

The video analysis in *Table 3* reveals that striking players use the top portion of the helmet (+Q2 to +Q4) when impacting the concussed player. These impact conditions approach, and probably include, the act of spearing. Spearing was banned in competitive football many years ago to decrease the risk of severe cervical spine

TABLE 6. Signs and symptoms from players concussed in professional football and impacts reconstructed in laboratory tests

	Impact condition				Total	%
	A	B	C	D		
Reported cases	7	9	3	2	21	100%
No information	2	1	1	0	4	
General symptoms	5	6	1	1	13	62%
Headaches	5	3	0	1	9	43%
Neck pain	1	3	0	0	4	19%
Nausea	1	1	0	0	2	10%
Syncope	0	0	1	0	1	5%
Vomiting	0	0	0	0	0	0%
Back pain	0	0	0	0	0	0%
Seizures	0	1	0	0	1	5%
Cranial nerve symptoms	5	4	1	2	12	57%
Dizziness	4	3	1	1	9	43%
Blurred vision	1	0	0	0	1	5%
Vertigo	0	1	0	0	1	5%
Photophobia	2	2	0	0	4	19%
Tinnitus	0	0	0	1	1	5%
Diplopia	0	0	0	0	0	0%
Nystagmus	0	0	0	0	0	0%
Pupil response	0	1	0	0	1	5%
Pupil size	0	0	0	0	0	0%
Hearing loss	0	0	0	0	0	0%
Memory problems	4	5	1	2	12	57%
Retrograde amnesia, delayed	2	2	1	2	7	33%
Information processing	1	3	1	1	6	29%
Attention problems	0	2	0	0	2	10%
Antegrade amnesia delayed	3	0	1	0	4	19%
Cognition problems	3	3	1	1	8	38%
Immediate recall	3	3	1	1	8	38%
Not oriented to time	1	1	1	0	3	14%
Not oriented to place	0	1	0	0	1	5%
Not oriented to person	0	0	0	0	0	0%
Somatic complaints	4	1	1	0	6	29%
Fatigue	3	1	0	0	4	19%
Anxiety	0	0	0	1	1	5%
Personality change	2	0	0	1	3	14%
Irritability	0	0	1	0	1	5%
Sleep disturbance	1	0	0	0	1	5%
Loss of appetite	0	0	0	0	0	0%
Depression	0	0	0	0	0	0%
Loss of libido	0	0	0	0	0	0%
No. of symptoms	30	28	8	9	75	
No. of symptoms/player	4.3	3.1	2.7	4.5	3.6	

injury. This raises the question of whether the rate of MTBI would be affected by continuing to reinforce proper tackling techniques, which emphasize the "face-up" position of the tackler.

This study has for the first time delineated the locations and directions of helmet impacts causing most concussions in professional football. Together with impact speed and change in head velocity, these results define the biomechanical factors associated with concussion in the NFL. The results should spur improvements in protective equipment and ultimately lead to increased safety for all football players.

APPENDIX

TABLE A1. Impact location and reconstruction results for the struck players[a]

Case	Velocity (m/s)	ΔV (m/s)	Translational acceleration				SI	HIC	Rotational		Resultant direction		Facemask Helmet contact		
			Linear x (g)	Linear y (g)[b]	Linear z (g)	Resultant (g)			Accel (rad/s²)	Velocity (r/s)	Front to back (deg)[2c]	Up + down (deg)	Facemask	Top view	Side view
25 Concussed struck players															
A Condition 36%															
113	7.0	5.1	−12.8	−55.1	−15.3	58.6	163	140	3965	12.8	77.0	−15.1	N	L 0–45	+Q2
84	9.4	6.3	−64.5	−49.1	−11.6	81.9	276	222	9193	80.9	37.3	−8.1	Y	L 0–45	+Q2
148	6.6	5.1	−28.5	−38.4	−2.8	47.9	117	99	3476	23.9	53.4	−3.4	Y	L 0–45	+Q1
157	10.8	8.1	−63.4	78.4	−18.8	102.6	545	472	6750	33.5	51.0	−10.6	Y	R 0–45	+Q1
69	10.3	5.0	−36.2	48.6	−2.7	60.7	177	153	4381	19.9	53.3	−2.5	Y	R 0–45	−Q1
118	10.7	9.6	−93.6	36.5	−5.0	100.6	492	378	7017	42.9	21.3	−2.8	Y	R 0–45	−Q1
124	11.4	7.5	−72.8	35.4	−9.6	81.5	380	282	7138	34.8	25.9	−6.7	Y	R 0–45	−Q1
181	11.7	7.1	−65.0	65.9	−5.6	92.8	423	382	8011	36.5	45.4	−3.4	Y	R 0–45	−Q1
77	9.9	5.2	−21.5	77.2	5.6	80.3	226	185	5148	36.4	74.4	4.0	Y	R 0–45	−Q2
Average	9.8	6.6	−50.9	53.9	−7.3	78.5	311	257	6120	35.7	48.8	−5.4			
SD	1.8	1.6	25.6	15.7	7.0	18.1	154	129	1964	19.4	18.0	5.2			
B Condition 40%															
135	10.0	8.6	22.5	129.5	−41.3	137.8	751	566	7540	41.0	99.8	−17.4	N	R 45–90	+Q4
9	10.3	10.1	−24.3	−130.2	−21.9	134.3	848	600	7428	27.4	79.4	−9.4	N	L 45–90	+Q3
162	5.5	4.2	−28.4	39.7	−18.2	52.1	94	77	2615	18.4	54.4	−20.4	N	R 45–90	+Q3
98	9.6	6.2	−38.8	−81.4	−12.1	91.0	351	301	7548	43.4	64.5	−7.6	N	L 45–90	+Q2
71	10.3	7.3	−42.5	114.1	−20.5	123.5	658	510	5400	35.0	69.6	−9.6	Y	R 45–90	+Q1
38	9.5	9.7	−20.0	−116.4	−9.4	118.5	736	554	9678	50.8	80.3	−4.5	Y	L 45–90	−Q1
57	8.8	6.0	−29.6	−71.3	−0.9	77.2	253	206	6514	37.0	67.4	−0.7	Y	L 45–90	−Q1
125	11.7	9.1	−57.4	−97.1	8.1	113.1	817	633	7716	63.3	59.4	4.1	Y	L 45–90	−Q1
155	9.1	6.6	−27.6	−96.4	2.1	100.3	418	341	6940	37.0	74.0	1.2	Y	L 45–90	−Q1
39	10.9	8.4	−29.4	125.1	7.5	128.7	656	522	5921	36.1	76.8	3.3	Y	R 45–90	−Q2
Average	9.6	7.6	−27.6	100.1	−10.7	107.6	558	431	6730	38.9	72.6	−6.1			
SD	1.7	1.9	19.6	27.8	14.7	26.1	260	188	1854	12.2	12.1	8.0			
C Condition 16%															
92	11.1	10.0	4.8	−97.0	−45.9	107.4	630	508	6878	44.2	92.8	−25.3	N	L 90–135	+Q4
164	10.8	6.0	10.1	−122.2	−16.4	123.7	451	370	9590	26.6	94.7	−7.6	N	L 90–135	+Q2
67	8.1	8.0	−2.1	128.6	−42.5	135.4	756	632	5957	13.8	89.1	−18.3	N	R 90–135	+Q2
7	6.9	4.6	20.0	57.4	1.5	60.8	120	93	6266	28.1	109.2	1.4	N	R 90–135	+Q1
Average	9.2	7.2	8.2	101.3	−25.8	106.8	489	401	7173	28.2	96.5	−12.4			
SD	2.0	2.4	9.3	32.3	22.5	32.7	276	231	1656	12.5	8.8	11.7			
D Condition 8%															
133	6.0	8.4	90.0	40.6	−55.2	113.1	648	557	5012	16.0	155.7	−29.2	N	R 135–180	+Q2
123	6.3	8.3	102.2	42.1	−49.6	121.1	866	730	4727	30.3	157.6	−24.2	N	R 135–180	+Q3
Average	6.1	8.4	96.1	41.3	−52.4	117.1	757	644	4870	23.2	156.7	−26.7			
SD	0.2	0.1	8.6	1.1	4.0	5.7	154	122	202	10.1	1.3	3.6			
6 Nonconcussed struck players															
A Condition															
154	6.6	5.1	−21.2	49.0	1.6	53.4	136	114	4167	24.0	66.5	1.7	Y	R 0–45	−Q1
175	9.6	5.6	−47.2	−38.8	8.4	61.7	158	125	3555	39.2	39.5	7.8	Y	L 0–45	−Q1
48	9.7	4.7	−33.2	−46.1	−7.9	57.3	155	130	5617	42.4	54.2	−7.9	Y	L 0–45	−Q1
Average	8.6	5.1	−33.9	44.6	0.7	57.5	150	123	4446	35.2	53.4	0.5			
SD	1.8	0.5	13.0	5.2	8.2	4.1	12	8	1059	9.8	13.6	7.9			
C Condition															
182	8.1	5.9	28.7	−79.2	7.6	84.6	256	208	5512	17.8	109.9	5.2	N	L 90–135	−Q1
59	5.3	5.6	−9.4	81.6	−4.7	82.3	205	138	5387	26.9	83.4	−3.3	N	R 90–135	+Q2
142	3.1	2.9	16.1	−6.7	−7.2	18.8	12	9	1170	7.4	157.5	−22.6	N	L 90–135	+Q4
Average	5.5	4.8	11.8	55.8	−1.4	61.9	158	118	4023	17.4	117.0	−6.9			
SD	2.5	1.7	19.4	42.6	8.0	37.3	129	101	2472	9.8	37.5	14.2			

[a] SI, severity index; HIC, head injury criterion; SD, standard deviation; N, no; Y, yes.
[b] The average was determined from the absolute value of the *y* acceleration.
[c] The vector is adjusted to the right side of the helmet for analysis of averages.

TABLE A2. Impact location and reconstruction results for the striking players[a]

Case	Velocity (m/s)	ΔV (m/s)	Translational acceleration				SI	HIC	Rotational		Resultant direction		Helmet contact		
			Linear x (g)	Linear y (g)[b]	Linear z (g)	Resultant (g)			Accel (rad/s²)	Velocity (r/s)	Front to back (deg)[c]	Up + down (deg)	Facemask	Top view	Side view
E Condition 59%															
71	10.3	6.6	−76.7	67.1	−10.5	102.4	512	434	5541	32.4	41.2	−5.9	N	R 0–45	+Q2
57	8.8	4.1	−13.8	23.0	−18.0	32.3	48	38	4151	33.2	58.9	−33.9	N	R 0–45	+Q3
77	9.9	4.2	−19.7	28.8	1.4	34.9	65	53	2714	25.5	55.7	2.3	N	R 0–45	+Q3
154	6.6	3.1	−24.6	10.9	−11.1	29.1	35	31	3159	23.1	23.9	−22.3	N	R 0–45	+Q3
7	6.9	2.2	−28.9	22.1	−34.0	49.8	65	51	2832	9.8	37.4	−43.1	N	L 0–45	+Q4
59	5.3	2.3	−21.1	−2.8	−23.9	32.0	32	26	2087	13.1	7.6	−48.2	N	L 0–45	+Q4
135	10.0	3.8	−46.5	−30.8	−61.2	82.8	230	179	5005	29.3	33.5	−47.6	N	L 0–45	+Q4
39	10.9	2.3	−15.9	−1.2	−40.9	43.9	60	43	4487	10.4	4.4	−68.7	N	R 0–45	+Q4
48	9.7	3.2	−23.9	19.1	−8.0	31.6	44	37	2939	28.0	38.6	−14.6	N	R 0–45	+Q4
118	10.7	3.7	−54.1	−11.2	−5.4	55.5	122	73	3687	23.4	11.6	−5.6	N	R 0–45	+Q4
125	11.7	4.2	18.1	35.7	−23.7	46.5	132	111	3366	28.1	26.9	−30.6	N	R 0–45	+Q4
148	6.6	3.9	−11.7	28.7	−11.1	32.9	47	37	2466	26.5	67.8	−19.7	N	R 0–45	+Q4
155	9.1	4.2	−28.6	30.5	−15.1	44.5	76	61	4217	29.5	46.9	−19.9	N	R 0–45	+Q4
157	10.8	5.0	−66.2	18.7	−38.5	78.9	215	180	4662	15.7	15.7	−29.2	N	R 0–45	+Q4
162	5.5	3.2	−26.1	7.5	−9.6	28.8	34	30	1672	17.2	16.1	−19.5	N	R 0–45	+Q4
175	9.6	3.9	−6.2	39.6	−25.1	47.3	81	62	2535	19.3	81.1	−32.0	N	R 0–45	+Q4
Average	8.9	3.7	−27.9	23.6	−20.9	48.3	112	90	3470	22.8	35.5	−27.4			
SD	2.0	1.1	23.4	16.3	16.2	21.8	123	104	1113	7.6	22.4	18.4			
F Condition 30%															
98	9.6	4.8	22.9	−77.5	−24.0	84.3	241	187	4487	38.5	16.5	−16.5	N	L 45–90	+Q4
181	11.7	7.3	25.7	−79.3	−15.3	84.8	402	333	6613	55.8	18.0	−10.4	N	L 45–90	+Q4
9	10.3	2.3	−20.4	51.6	−56.7	79.4	275	217	6719	18.7	68.4	−45.6	N	R 45–90	+Q4
69	10.3	3.1	−0.5	18.4	−33.0	37.8	55	50	2620	23.0	88.6	−60.8	N	R 45–90	+Q4
84	9.4	4.4	−19.2	40.4	6.3	45.2	96	78	3169	26.5	64.6	8.0	N	R 45–90	+Q4
124	11.4	3.1	49.2	−8.8	−24.3	55.6	105	73	4086	16.1	79.9	−26.0	N	R 45–90	+Q4
164	10.8	5.1	−10.2	86.5	−16.6	88.7	243	202	6136	30.8	83.3	−10.8	N	R 45–90	+Q4
182	8.1	4.7	−62.8	55.8	−24.0	87.4	272	213	3206	27.2	41.6	−16.0	N	R 45–90	+Q3
Average	10.2	4.4	−1.9	10.9	−23.5	70.4	211	169	4629	29.6	57.6	−22.3			
SD	1.2	1.6	34.7	61.7	17.7	20.8	117	96	1651	12.7	28.8	21.7			
Other conditions 11%															
92	11.1	5.6	−17.5	23.2	−52.1	59.7	204	164	6070	43.8	53.0	−60.9	N	R 90–135	+Q4
113	7.0	3.7	7.4	54.0	−27.1	60.9	101	75	3700	31.2	97.8	−26.4	N	R 90–135	+Q3
38	9.5	4.0	45.2	20.8	−33.3	59.9	157	127	5205	28.2	155.2	−33.8	N	L 135–180	+Q4
Average	9.2	4.4	11.7	32.7	−37.5	60.1	154	122	4992	34.4	102.0	−40.4			
SD	2.1	1.0	31.5	18.5	13.0	0.6	52	45	1199	8.3	51.3	18.1			

[a] Twenty-seven nonconcussed striking players. SI, severity index; HIC, head injury criterion; SD, standard deviation; N, no; Y, yes.
[b] The average was determined from the absolute value of the *y* acceleration.
[c] The vector is adjusted to the right side of the helmet for analysis of averages.

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Acknowledgments

The NFL has a Committee on Mild Traumatic Brain Injury (MTBI). It is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society, NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the area of traumatic brain injury, biomechanics, basic science research, and epidemiology. The authors of this article are members of the Committee. The efforts of other Committee members are gratefully acknowledged, including Ronnie Barnes, ATC, Henry Feuer, M.D., Mark Lovell, Ph.D., A.B.P.N., John Powell, Ph.D., A.T.C., Doug Robertson, M.D., and Joe Waeckerle, M.D. None of the Committee members have a financial or business relationship posing a conflict of interest to the research conducted on concussion in professional football.

The MTBI Committee gratefully acknowledges the insights of the Commissioner, Paul Tagliabue, for forming the Committee and issuing a charge to scientifically investigate concussion and means to reduce injury risks in football. Also, the efforts of Dorothy Mitchell, former Counsel for the NFL, are most gratefully acknowledged. She worked tirelessly to set up and initiate the MTBI research. Although she left the NFL in the midst of the program, her efforts paved the way for a successful completion of the research. The encouragement and support from the NFL's Jeff Pash and Peter Hadhazy are also appreciated.

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COMMENTS

It is difficult to solve a problem if we cannot even define what the problem is. Sports-related brain injuries receive a great deal of attention that frequently fuels demands for better equipment or for changes in rules, but lack of hard data about the nature of these injuries means that attempts at reform amount to little more than stumbling around in the dark. In this article, Pellman et al. continue their series of reports analyzing concussion in players on professional American football teams. This unique study has made significant strides toward identifying factors associated with football-related concussion so that modifications in equipment and changes in rules can now proceed on a more rational basis.

Alex B. Valadka
Houston, Texas

It is said that the role of a physician is threefold: the first is to prevent disease; if that be impossible, to cure it; and if that also be impossible, to relieve pain. In the second of a three-part series by members of the National Football League (NFL) Committee on Mild Traumatic Brain Injuries (MTBI), the authors extend their analysis of previously reported laboratory reconstruction data and use a sophisticated video database acquired from NFL game footage during a 5-year period to define the conditions of impact to the helmet that result in the majority of concussions in professional football. Their specific purpose in this analysis is to assess current helmet performance in an effort to reduce or prevent injuries to the brain in the high-speed-collision sport of football.

This sophisticated acquisition of high-speed video impact data and subsequent biomechanical derivation of impact velocity, change in head velocity, and acceleration forces is the first of its kind to be correlated with the direction of helmet impact leading to cerebral concussion in football. After culling 182 game impacts, the authors concluded that the most likely scenario for a cerebral concussion was a helmet impact to the facemask in the 0- to 45-degree position. Their graphs clearly show that this impact results in the greatest peak impact velocity and translational and rotational acceleration, as compared with nonconcussed helmet impacts. As one might believe intuitively and now is confirmed biomechanically, impacts to the facemask essentially twist the head while rapidly accelerating it. Thus, the authors conclude that these movements and forces significantly influence concussion tolerance. Their attempt to link impact conditions with the symptoms and signs of concussion was limited by the relatively small sample size.

Significant potential areas of investigation for preventing MTBI in football include the following: 1) the application of improved energy-absorbing materials in helmet construction and in the padding and facemask design; 2) reevaluation of National Operating Committee on Standards for Athletic Equipment (NOCSAE) standards for performance require-

ments of helmets; 3) rigid enforcement of already outlawed spearing techniques; and 4) continued improvement in the energy absorption properties of the various playing surfaces.

Both the leadership of the NFL and the members of the MTBI Committee are to be commended on their efforts to prevent head injuries not only in the NFL but also in the more than 1.2 million high school and college players as well who will be the beneficiaries of this research.

Joseph C. Maroon
Pittsburgh, Pennsylvania

Pellman et al. follow with the second in a series of reports based on analysis of network videotape evidence related to concussion in NFL players. This study focuses on factors such as the velocity, vectors of acceleration, location of head (helmet) impacts, source of contact, and other parameters. Laboratory analysis using anthropometric mannequins was used to reconstruct the forces involved in impacts and the resultant head responses. Although one may debate the methodology and classification of symptoms in their definitions, it is difficult not to be impressed with the quality of documentation of concussive forces, locations, vectors, and players' responses and the authors' subsequent attempts at analysis.

In addition to representing a novel approach to the study of biomechanical and force characteristics of MTBI, this research elucidates several new and important facts concerning such injuries in athletic endeavors. First, there was documentation that a higher head acceleration was present in concussed players and that the majority sustain impacts to the helmet shell. Players who were approached and struck at an acute angle, unaware of the closing speed of their opponents, who were outside of their peripheral vision, sustained severe impacts to the facemask area. This research thus establishes the fact that the helmet shell and facemask impart different force vectors to the head of the athlete sustaining the blow. It affirms that translational acceleration is the most important feature of athletic concussion in football. The fact that impacts with opponents and with the ground each have characteristic locations may also assist researchers and helmet manufacturers to improve design in the future.

We await further data from the Committee's investigation that may help us in correlating type and area of impact and the resultant symptoms, severity of MTBI, and possible preventive measures through equipment or rule changes. In having access to unprecedented multiangle video documentation of the physics of concussion in football, the authors have ushered in a new era in the study and analysis of the many nuances of these high-speed bodily collisions. To paraphrase Richard Schneider, they are studying by darkroom analysis of an important laboratory for head injury, the football field.

Julian E. Bailes
Morgantown, West Virginia

By further painstakingly analyzing the biomechanics of helmet impacts in NFL players, Pellman et al. have shown that concussions occur with several predominant helmet impact patterns related to whether the concussed player is struck in the facemask or on the helmet shell. This analysis points toward several areas of the helmet architecture, including the facemask, chin strap, and padding, in which design modifications could potentially reduce the risk of concussion. Given the nature of the game and the ever-increasing size and strength of the players, reducing the risk of concussion in these human projectiles by effective helmet modifications will certainly be a challenge. The NFL MTBI Committee is to be commended for their effort.

Daniel F. Kelly
Los Angeles, California

This article represents an important step in the right direction with regard to enabling the neurosurgical readership to better identify those factors related to concussive injury in sport. With the resources of the NFL, important studies such as this could be of significant benefit in preventing further injury at all levels of play. The authors have reported the locations and directions of helmet impact that are most likely to result in concussion in addition to the potential relationship of impact speed and changes in head velocity.

Using game video analysis and laboratory reconstruction, the authors were able to record helmet impact by quadrant in 96% of injuries evaluated. The importance of such an analysis is to potentially evaluate and modify the NOCSAE standards on the basis of a game-oriented or clinical interface. An additional point of interest was the grouping of signs and symptoms into five categories on the basis of their experience with head injury. Of interest is that cranial nerve function was included, whereas motor, sensory, and cerebellar function were not. Although surprising, this methodology was based on a consensus of experience that allowed the authors to separate the most salient variables into five groupings. In retrospect, their groupings assisted in data analysis and presentation and were found to be appropriate.

Interesting points concern the relationship of injury to the potential speed of injury. This is supported by the fact that with higher closing speeds in the open field, one tends to see an increased rate of injury. Although the video resources available tend to evaluate play at the line of scrimmage less than in the open field or respective backfields, the authors have supported the contention that linemen sustain fewer concussive injuries than other players.

Future goals include evaluating the relationships between helmet impact location and injury. I anticipate that with continued accumulation of data, modification of the current NOCSAE standards will be forthcoming.

Michael L. Levy
San Diego, California

TAB 1C

Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProHEALTH Care Associates, Lake Success, New York

John W. Powell, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University, Lansing, Michigan

David C. Viano, Dr. med., Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics, Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park, New York; and Department of Neurology, New York University Medical Center, New York, New York

Andrew M. Tucker, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and University of Maryland School of Medicine, Timonium, Maryland

Henry Feuer, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Indianapolis Neurosurgical Group, Indianapolis, Indiana

Mark Lovell, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

Joseph F. Waeckerle, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Acute Care and Emergency Specialists, Leawood, Kansas

Douglas W. Robertson, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and American Health Network, Carmel, Indiana

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics, 265 Warrington Road, Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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CONCUSSION IN PROFESSIONAL FOOTBALL: EPIDEMIOLOGICAL FEATURES OF GAME INJURIES AND REVIEW OF THE LITERATURE—PART 3

OBJECTIVE: A 6-year study was performed to determine the circumstances, causes, and outcomes of concussions in the National Football League.

METHODS: Between 1996 and 2001, the epidemiological features of concussions were recorded by National Football League teams with a standardized reporting form. Symptoms were reported and grouped as general symptoms, cranial nerve symptoms, memory or cognitive problems, somatic complaints, and loss of consciousness. The medical actions taken were recorded. In total, 787 game-related cases were reported, with information on the players involved, type of helmet impact, symptoms, medical actions, and days lost. Concussion risks were calculated according to player game positions.

RESULTS: There were 0.41 concussions per National Football League game. The relative risk was highest for quarterbacks (1.62 concussions/100 game-positions), followed by wide receivers (1.23 concussions/100 game-positions), tight ends (0.94 concussion/100 game-positions), and defensive secondaries (0.93 concussion/100 game-positions). The majority of concussions (67.7%) involved impact by another player's helmet. The remainder involved impact by other body regions of the striking player (20.9%) or ground contact (11.4%). The three most common symptoms of mild traumatic brain injury were headaches (55.0%), dizziness (41.8%), and blurred vision (16.3%). The most common signs noted in physical examinations were problems with immediate recall (25.5%), retrograde amnesia (18.0%), and information-processing problems (17.5%). In 58 of the reported cases (9.3%), the players lost consciousness; 19 players (2.4%) were hospitalized. A total of 92% of concussed players returned to practice in less than 7 days, but that value decreased to 69% with unconsciousness.

CONCLUSION: The professional football players most vulnerable to concussions are quarterbacks, wide receivers, and defensive secondaries. Concussions involved 2.74 symptoms/injury, and players were generally removed from the game. More than one-half of the players returned to play within 1 day, and symptoms resolved in a short time in the vast majority of cases.

KEY WORDS: Concussion, Injury epidemiology, Sports injury prevention, Traumatic brain injury

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Mild traumatic brain injury (MTBI) or concussion has been defined as a traumatically induced alteration in neural function, which may or may not involve loss of consciousness (8, 39). For the purposes of this study, a broad but specific definition of concussion was adopted, as stated below.

MTBI is a major public health problem in the United States, with an estimated annual incidence of 160 to 375 cases/100,000 persons (23). The Centers for Disease Control and Prevention (4) estimated that the number of MTBIs has reached 300,000 cases/yr in all sports. Mild head injury has received a great deal of publicity in recent years because of its prevalence in many sports with contact or the potential for collisions; it has become a major issue in athletics. Powell and Barber-Foss (39) estimated that 3.9 to 7.7% of high school and college athletes sustain MTBIs each year, in all sports.

At the professional level, publicity has increased attention regarding MTBIs, because of repeated concussions among a number of high-profile professional football players. Until recently, however, little was known regarding the long-term sequelae of MTBIs. Although research has been conducted and progress has been made, some research has involved anecdotally based information and there have been few scientific data available regarding the symptoms of MTBI (concussion) among professional football players. There is also a paucity of epidemiological information available regarding the natural history of concussion in this population.

This article is the result of a multiyear effort by the National Football League (NFL) to address the deficiencies in our knowledge regarding concussion. The specific purpose is to analyze patterns of concussions and trends with a special epidemiological database, in the hope of identifying the most vulnerable positions and most frequent symptoms. The research efforts are ultimately aimed at improving the safety of football for players at all levels (professional, college, high school, and pee-wee), the safety of other contact sports for all participants, and the general public safety during nonathletic endeavors that place individuals at risk of head injury.

In 1994, the NFL formed the MTBI Committee in response to safety concerns regarding head injuries. Background information on the committee was reported by Pellman (36). The committee consisted of medical and scientific experts in the areas of traumatic brain injury, basic science research, and epidemiology. The mission of the committee was to scientifically investigate the subject of MTBIs in the NFL. The committee examined the existing literature and spent time interviewing a variety of experts to identify what was known and accepted regarding the natural history of MTBIs and to determine what areas required additional research. Committee members were on-field physicians and athletic trainers with experience recognizing and treating MTBIs.

After an initial review, the committee identified two areas that required attention. First, there was a strong need to be able to monitor the frequency of MTBIs in the NFL and to more specifically identify the clinical symptoms associated with concussions. Second, the MTBI Committee undertook a series of research projects aimed at defining concussion biomechanics in professional football. On the basis of analyses of game videos of MTBIs and laboratory reconstructions of the impacts with instrumented test dummies, the biomechanical features of concussions were determined for professional players. Those findings included the acceleration severity of the injuries (38) and the location and direction of the impacts (37). This article discusses the epidemiological features of MTBI in professional football and combines those data with the clinical symptoms present at the time of injury.

The NFL has collected epidemiological data on player injuries since 1980. The data included "concussions," regardless of time lost from participation. The committee analyzed those data and identified a need to collect clinically oriented information regarding head injuries among the league's players. In addition to the data being recorded by the NFL Injury Surveillance System, the specific clinical symptoms evident at the time of injury needed to be documented.

Information regarding the circumstances at the time of injury and the clinical nature of the injury was also essential for determining the natural history of MTBIs. The committee agreed that data on the diagnosis and management of MTBIs should be recorded by the team physicians, with a standardized form. The program began with the 1995 NFL season and has continued in the subsequent years.

At the beginning of the project in 1995, the committee recognized a need to develop an all-inclusive definition of MTBI, so that all of the team physicians and athletic trainers could easily recognize the symptoms of reportable injuries. It was understood that any player with a recognized symptom of head injury, no matter how minor, should be included in the study. In the initial year of the project, a reportable MTBI was characterized by an "altered mental state regardless of duration and/or altered memory, regardless of duration or content, that resulted from trauma and occurred in an NFL practice or game." Feedback from physicians and the committee's analysis of the project data during the first year of the project resulted in the development and modification of report forms and enhancement of the definition of reportable MTBIs.

The definition introduced by the committee in 1996 and used for the remainder of the study was as follows. A reportable MTBI was defined as a traumatically induced alteration in brain function, manifested by 1) alteration of awareness or consciousness, including but not limited to being dinged, dazed, stunned, woozy, foggy, or amnesic or, less commonly, being rendered unconscious or experiencing seizures, and 2) signs and symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, lightheadedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbances, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality changes, drowsiness, lethargy, fatigue, and inability to perform usual daily activities. The definition of concussion used by the MTBI Committee was a natural extension of a much earlier definition proposed by the Ad Hoc Committee to Study Head Injury Nomenclature of the Congress of Neurological Surgeons, which in 1966 defined concussion as "a clinical syndrome characterized by immediate transient impairment of neural function such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces" (8a).

PATIENTS AND METHODS

Data Collection

Since 1980, the NFL Injury Surveillance System has provided a mechanism for athletic trainers and team physicians to record data on injured players and circumstances surrounding injuries. The system requires that each team record data on all concussions that occur, regardless of the amount of time lost to participation because of the injury. The recorded data include

the player time lost, the player position, the activity of the player at the time of the injury, and the nature of the activities of the team. For the current research project, the data set was modified to include the equipment being worn, the mechanism of injury (e.g., head-to-head impact), the facemask, the chin strap, the type of mouthpiece (if worn), and the approximate location of the impact on the helmet.

In addition to data recorded by the athletic trainers, the committee devised a simple form regarding observed and reported symptoms for the individual team physicians to complete when they evaluated, in initial and follow-up visits, players who had sustained MTBIs. Players' names were not included on the forms, to maintain confidentiality. The players were identified with the last six digits of their Social Security numbers; with this coding system, it was possible to merge the NFL Injury Surveillance System data with data on the clinical evaluations of injuries.

Symptoms

MTBI is a clinical syndrome that may present with a wide range of symptoms, many of which are rather nonspecific and can be associated with other clinical diagnoses. The categories in *Table 1* represent groupings of 34 of the most common symptoms of concussion, including unconsciousness. The list was developed by MTBI Committee members who are NFL team physicians, as well as MTBI Committee consultants with special expertise in the fields of sport neuropsychology and sport neurology. Symptoms of concussion were grouped into six categories, as follows: 1) general symptoms, 2) cranial nerve symptoms, 3) memory problems, 4) cognitive problems, 5) somatic complaints, and 6) unconsciousness. These symptoms are consistent with findings previously noted after traumatic brain injury (3, 14, 20, 21, 28, 33, 41, 45).

TABLE 1. Signs and symptoms recorded on the mild traumatic brain injury form[a]

General symptoms	Memory problems
Headaches	*RGA delayed*
Neck pain	*Information-processing problems*
Nausea	*Attention problems*
Syncope	*AGA delayed*
Vomiting	
Back pain	Cognitive problems
Seizures	*Immediate recall*
	Not oriented with respect to time
Cranial nerve symptoms	*Not oriented with respect to place*
Dizziness	*Not oriented with respect to persons*
Blurred vision	
Vertigo	Somatic complaints
Photophobia	*Fatigue*
Tinnitus	*Anxiety*
Diplopia	*Personality changes*
Nystagmus	*Irritability*
Pupil response	*Sleep disturbances*
Pupil size	*Loss of appetite*
Hearing loss	*Depression*
	Loss of libido
	Loss of consciousness

[a] RGA, retrograde amnesia; AGA, anterograde amnesia.

Players spontaneously reported many of the symptoms, but the complete symptom complex, including mental status (retrograde amnesia, anterograde amnesia, and problems with information processing, attention, and immediate recall), was assessed with physician questions. The committee did not distribute uniform testing instruments to the team physicians and instead left the assessment of these symptoms to the discretion of individual team physicians.

Efforts to Improve Compliance

The NFL, through the commissioner, strongly encouraged all team physicians to complete and return the project's forms whenever they examined players with head injuries. The project was designed to record information regarding the injuries. The initial form contained questions regarding the initial symptoms of the MTBI, the physical examination findings, the initial treatment, the tests ordered, and the disposition regarding the return to play. Physicians completed follow-up forms that documented the symptoms noted, the tests ordered and their results, and the time before return to full participation. Physicians used their own evaluation procedures to treat the injuries. The committee had neither the authority nor the inclination to impose outside medical decision-making on the medical staffs of the individual teams. The individual team physicians were to complete the initial and follow-up forms on the basis of their clinical findings.

To improve compliance, the forms were designed for ease of completion and the data were limited to the points that would provide the strongest and most consistent information on MTBI. After being completed, the data forms were sent to the NFL epidemiologist and entered into a database with a blinded coding system, to maintain the anonymity of the players. When an initial evaluation form was submitted but the follow-up forms were not, committee members contacted the team athletic trainers and doctors directly, to remind them to submit the follow-up forms.

In biannual meetings, the committee monitored the data and discussed the findings. Approximately 1.5 to 2 years after the initiation of the project, the forms were modified according to the findings of the analysis. For example, Glasgow Coma Scale scores were removed after the first year and data on loss of consciousness were added. Before the 1999 season, the forms were modified again, to remove fields that were determined to be of little value in the analysis. For example, the physical examination fields were modified so that the physicians could better describe their findings. To enhance other aspects of the committee's work, the identification and dates of neuropsychological tests were included on the physician's form.

Educational symposia based on the medical literature and the clinical backgrounds of committee members were held throughout the NFL, to increase awareness of the implications of even the mildest MTBIs and to promote compliance, to ensure the longevity of players' careers. With promulgation of the definition throughout the NFL, the committee considered it more likely that all players with MTBIs would be included in the data collection efforts. The committee held numerous educational symposia on MTBI for medical personnel for all NFL teams, thus increasing awareness of MTBI and compliance with the study of MTBI.

Final MTBI Epidemiological Form

After a review of the data from the 1995 season, the recording forms were modified to improve clarity and the definition of a reportable event was extended. Because of these significant changes in the program, the 1995 data were considered pilot data and were not included in the analysis. With the specific limitations of the data, the project has complete injury data, initial clinical evaluation data, and follow-up evaluation data for 787 MTBIs that occurred in preseason, regular, or playoff games during the six professional football seasons from 1996 to 2001. There were 100 additional MTBI cases from practice sessions, which are included in the database but are not analyzed here because the risks are being considered according to player position and play type in games. Furthermore, unconsciousness data were not initially collected and, when such data were added to the standardized form, there were cases in which no determination was made by the presiding physician. Determinations were made in 623 cases.

Quality Assurances

The clinical evaluation forms were designed to include an initial evaluation form, to be completed at the time of injury, and a follow-up evaluation form, to be completed each time the physician evaluated the player until his return to full participation. The data forms were sent to the NFL epidemiologist and entered into the database. The MTBI evaluation forms were logged in and scanned into a database file with a commercial software program (Teleforms, Cardiff, CA). During the data logging, the individual forms were manually reviewed. Each form was scanned into a temporary database and verified before it was entered into the final database. Fields that were incomplete or inconsistent triggered a follow-up contact with the team athletic trainer or physician for data verification. When the MTBI evaluation data were merged with the injury surveillance data, the data were again reviewed and verified. The final database includes information from the initial and follow-up evaluation forms submitted by the team physicians.

Data Analyses

This analysis involved data on MTBIs that occurred during games in the 1996 to 2001 NFL seasons. The data reflect only injuries that occurred during preseason, regular, or playoff games. The elimination of injuries that occurred during practice sessions allowed the analysis to focus on NFL games and the activities of the players at the time of injury. It also allowed a calculation of risks on the basis of player positions.

Each MTBI was analyzed as an independent event, as a first step toward understanding concussions. Summary measures are reported with 95% confidence intervals. Nonoverlapping intervals may be interpreted as significantly different at $\alpha = 0.05$. Exploratory analyses were performed to determine factors associated with concussion outcomes (i.e., number of days out of play and hospitalization). Factors associated with the number of days out of play were identified with nonparametric analyses (Spearman correlation, Mann-Whitney test, and Kruskal-Wallis test), because of the highly skewed distribution. For multivariate analyses, both multiple regression and logistic regression were used with stepwise selection of variables that were significant at $\alpha = 0.10$ in the bivariate analysis. Factors associated with hospitalization were identified with χ^2 tests for bivariate analysis and logistic regression for multivariate analysis. Only variables that were significant at $\alpha = 0.10$ were used in the logistic regression.

Injury rates per 100 game-positions were calculated to provide information on the risk of injury among the position categories. The denominator for these rates reflects the number of standard position players multiplied by the number of team-games (3826 team-games) during the study period. For example, there is one quarterback position for each team in each game (game-position exposure = 1 × 3826). For the offensive line, there are five positions (one center, two guards, and two tackles) per team (game-position exposure = 5 × 3826). Injury rates per 1000 plays were also calculated for rushing, passing, kickoff, and punt plays, on the basis of the number of plays in the NFL for the study period (34). When type-of-play data were considered, proportions for the various types were based on regular-season games (16 games), because the NFL tracks the number of rushing, passing, kickoff, and punt plays. This allowed a comparison of the relative risks of injury for the four most common types of plays in the NFL.

RESULTS

Concussion in the NFL

During the 1996 to 2001 NFL seasons, there were 787 reported cases of MTBI in 3826 team-games (1913 games). This total included all preseason, regular-season, and playoff game-related concussions. Concussions occurred with an average incidence of 131.2 ± 26.8 concussions/yr and a rate of 0.41 concussion/game.

Table 2 presents the incidence of concussions according to player position in professional football. As a group, the offensive team experienced the highest frequency of concussions in the NFL. Individually, the position group most often associated with concussion was the defensive secondary (18.2%), followed by the kicking unit (16.6%) and the wide receivers (11.9%). When the injury rates per 100 game-positions were

TABLE 2. Incidence of mild traumatic brain injury according to player position in National Football League games

Position	No. of cases	Incidence (%)	No. of game positions	Risk per 100 game-positions[a]
High risk				
Offensive				
Quarterback	62	7.9	3826	1.62 (1.22–2.02)
Wide receiver	94	11.9	7652	1.23 (0.98–1.48)
Tight end	36	4.6	3826	0.94 (0.63–1.25)
Running back	69	8.8	7652	0.90 (0.69–1.11)
Defensive				
Secondary	143	18.2	15,304	0.93 (0.78–1.08)
Moderate risk				
Offensive				
Offensive line	56	7.1	19,130	0.29 (0.21–0.37)
Defensive				
Linebacker	52	6.6	11,478	0.45 (0.33–0.57)
Defensive line	67	8.5	15,304	0.44 (0.34–0.54)
Special team				
Return ball carrier	22	2.8	3826	0.58 (0.34–0.82)
Kick unit	131	16.6	38,260	0.34 (0.28–0.40)
Low risk				
Special team				
Punter	7	0.9	3826	0.18 (0.05–0.31)
Return unit	33	4.2	38,260	0.09 (0.06–0.12)
Kicker, FGA	1	0.1	3826	0.03 (−0.02–0.08)
Kicker, PAT	1	0.1	3826	0.03 (−0.02–0.08)
Holder	1	0.1	3826	0.03 (−0.02–0.08)
Unknown/undesignated	12	1.5		
Total	787	100		8.08

[a] Risk per 100 game-positions is the number of concussions divided by the number of times the position was played during the observed period of 3826 games, multiplied by 100. Values in parentheses are 95% confidence intervals. FGA, field goal attempt; PAT, point after touchdown. Risk strata are approximate, because there is some overlap of confidence intervals.

adjusted for the number of persons in each position group, the relative risk of concussion was highest for quarterbacks (1.62 concussions/100 game-positions), followed by wide receivers (1.23 concussions/100 game-positions), tight ends (0.94 concussion/100 game-positions), and defensive secondaries (0.93 concussion/100 game-positions). The positions at low risk were punter, return unit, kicker, and holder. When the quarterback, running backs, wide receivers, and defensive secondary were grouped as "backs" and the offensive and defensive linemen were grouped as "linemen," the backs demonstrated nearly 3 times the relative risk of concussion.

Table 3 presents the types of plays in which concussions occurred. The highest frequency of injury was in passing plays (35.8%), followed by rushing plays (31.3%), kickoffs (15.9%), and punts (9.5%). *Table 4* indicates that, when the injury rates per 1000 plays in regular-season games were considered, the relative risk of concussion in kickoff plays (9.29 concussions/1000 plays) was more than 4 times the risks in rushing and passing plays and 2.5 times the risk in punt plays (3.86 concussions/1000 plays). Kickoffs and punts were associated with significantly higher injury rates than were rushing or passing plays.

Whereas *Tables 3* and *4* summarize team activities at the time of the concussion, *Figure 1* indicates the player activities during injury. The highest injury frequency was noted for players who were tackling (31.9%) or being tackled (28.6%). When data were combined, concussions were more often associated with tackles (60.5%) than with blocks (29.5%).

Figure 2 presents the percentages of concussions associated with different objects delivering the blow, from the epidemiological sample. The majority of concussions involved a strike by another player's helmet (67.7%). The remainder involved impact with the ground (11.4%) or impact by other body regions of a striking player (20.9%).

TABLE 3. Types of play for concussed players in National Football League games

Team activity	No. of cases	Incidence (%)[a]
Passing	282	35.8 (32.5–39.1)
Rushing	246	31.3 (28.1–34.5)
Kickoff	125	15.9 (13.3–18.5)
Punt	75	9.5 (7.5–11.5)
Unknown	47	6.0 (4.3–7.7)
Change of possession	9	1.1 (0.4–1.8)
Field goal attempt	2	0.3 (−0.1–0.7)
Point after touchdown	1	0.1 (−0.1–0.3)
Total	787	100

[a] Values in parentheses are 95% confidence intervals.

Table 5 summarizes the symptoms for concussed players in the 1996 to 2001 seasons. The median number of symptoms recorded for a concussion was 2 (range, 0–12). The three most common symptoms were headaches (55.0%), dizziness (41.8%), and blurred vision (16.3%). The least common symptoms were somatic complaints. There was considerable overlap between symptoms related to cognitive and memory problems, with 21.2% of the concussed players demonstrating both cognitive and memory problems. Overall, 45.9% of the concussed players experienced either cognitive or memory problems or both. No sensory or motor abnormalities were reported for any player, and nystagmus was the only cerebellar abnormality.

Table 6 summarizes the initial medical actions performed after injury. Only 16.1% of the players returned immediately to the game, including players who were evaluated on the field and returned to play and players who withdrew from the game for a few plays and then returned to play. The rest of the players rested for an extended period of time and returned in the same game (35.6%) or were removed from play and did not return to the game (44.0%). Nineteen players (2.4%) were removed from the game and hospitalized. Further analysis of data for the nonhospitalized players revealed strong associations between the presence and number of each symptom category and the action taken. Logistic regression was used to identify independent associations between the presence of categories, the number of symptoms, and the action taken. The model identified three variables as being independently associated with the action taken. The factors initially associated with resting of a player included the number of symptoms, any general symptom, any memory problem, any cognitive problem, and any somatic complaint. The multivariate logistic regression analysis identified the number of symptoms (odds ratio [OR] = 1.32 for each additional symptom), any memory problem (OR = 2.09), and any cognitive problem (OR = 3.41) as independent risk factors for resting of a nonhospitalized player.

Figure 3 presents the percentages of players returning to practice and games as a function of days lost after concussion in NFL games. Ninety-two percent of concussed players experienced ≤6 days lost and 97% experienced ≤9 days lost after MTBIs; 56.5% of concussed players experienced no days out of play. However, during the study period, 7% of concussed players experienced ≥7 days and 1.8% experienced >14 days out of play. Loss of consciousness occurred in 58 of 623 reported MTBI cases (9.3%) and involved more lost days, with 69% of players experiencing ≤6 days lost and 88% experiencing ≤9 days lost. Concussed players who sustained unconsciousness averaged 5.0 ± 7.5 lost days, which was 2.6 times longer than the time for players with MTBIs without loss of consciousness (1.9 ± 5.3 d).

Factors initially associated with increased days out of play included the number of symptoms, any general symptom, any memory problem, any cognitive problem, and any somatic complaint. The position played, type of play, action, or object of impact was not associated with days out of play. Although the results of the multivariate regression model suggested that only the number of symptoms was associated with an increased number of days out of play [$F(1,785) = 46.27$, $P < 0.001$], the analysis comparing any days missed with no days missed identified the number of symptoms (OR = 1.22 for each additional symptom), any general symptom (OR = 1.62), any somatic symptom (OR = 1.63), and any cognitive problem (OR = 2.35) as independent risk factors.

Comparison with the Video Analysis Cases

Figure 2 also indicates the percentages of concussions associated with different objects delivering the blow in the 182 video analysis cases reported by Pellman et al. (37, 38). The comparison demonstrated almost identical distributions of objects delivering the blow, except for a lower incidence of impacts by the knee. There was greater involvement of quarterbacks, flankers, and split ends in the video cases, which is related to the greater chances of multiple video views of those players in game coverage. In addition, a greater proportion of video cases involved hospitalization.

DISCUSSION

This study is unique in many ways. Data were prospectively collected during a 6-year period, and a broad definition of MTBI was used. The pilot epidemiological work in the 1995 season helped refine the methods, and further efforts were taken to maximize the consistency and accuracy of MTBI reporting for this population. The study used a standardized reporting form to improve the team-to-team consistency of findings. The committee monitored the results reported with these standardized forms and adjusted the forms as needed to improve data collection. All subjects were evaluated by phy-

TABLE 4. Injuries according to type of play for concussed players during regular-season National Football League games

Team activity	No. of cases	Incidence (%)	No. of plays	Risk per 1000 plays[a]
Kickoff	109	18.2	11,738	9.29 (7.55–11.03)
Punt	57	9.5	14,767	3.86 (2.86–4.86)
Rushing	183	30.5	81,692	2.24 (1.92–2.56)
Passing	206	34.3	96,447	2.14 (1.85–2.43)
Other	45	7.5		
Subtotal	600	72.3		
Pre- or postseason	187			
Total	787			

[a] Risk per 1000 plays is the number of injuries during an activity divided by the number of times the activity was performed, multiplied by 1000. Values in parentheses are 95% confidence intervals.

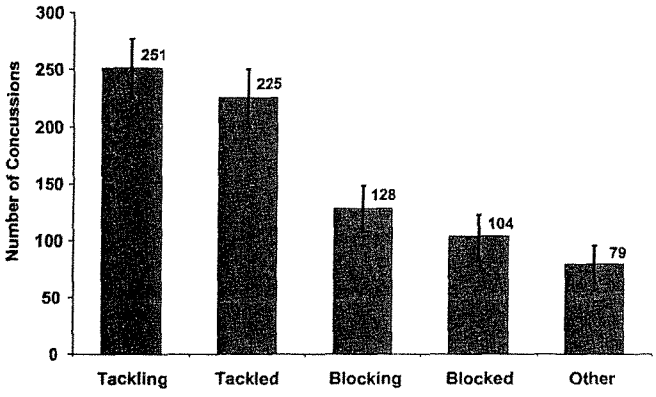

FIGURE 1. *Numbers of concussions associated with tackling, blocking, and other activities in NFL plays.*

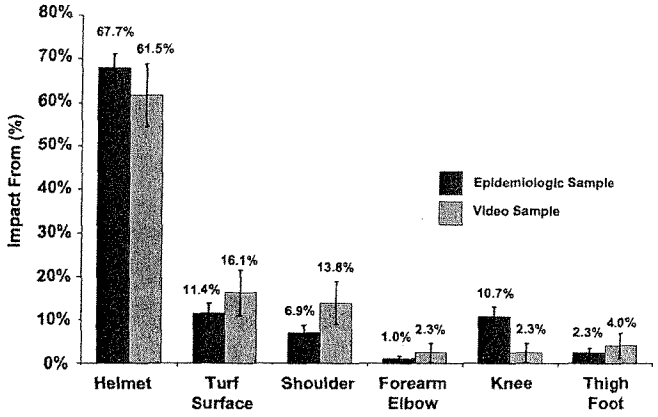

FIGURE 2. *Impacts from various objects associated with concussions in NFL games.*

sicians and underwent follow-up physician evaluations until they returned to play. The forms were completed by physicians; therefore, there is increased confidence in the medical validity and reliability of the information collected. In addition, all patients underwent certified athletic trainer evaluations, and follow-up reports were included in the results. The report forms were completed contemporaneously with the examinations and were therefore not subject to the vagaries of recall at a later date.

As with any research project that includes epidemiological data from multiple recording sites, there are specific limitations to the data. Numerous individual team physicians evaluated the injured players and completed the data collection forms. Whenever there are multiple data recording sites and multiple data recorders, the question of interobserver reliability must be addressed. The documentation process was designed prospectively and included specific criteria for the inclusion of an MTBI, to minimize the effect of interobserver reliability. With these procedures in place, the overall effect of the diversity of the physicians and recording conditions for each team is not known. Despite efforts to ensure a standardized definition of MTBI and to increase the team physicians' and athletic trainers' knowledge of MTBI, it remains unclear whether the project identified all cases of MTBI during the study period. The well-known reluctance of professional athletes to report their injuries to medical personnel might have prevented the reporting of some MTBI cases.

Every player in the NFL was a potential subject for the study. Every player with a reported MTBI was evaluated and underwent follow-up evaluations if unable to return to play on the day of the injury. None of the players with MTBIs in the study was lost to follow-up monitoring. Another factor that makes this study unique is the fact that the number of players exposed to head injury during the 6-year period is known; therefore, relative risks could be determined according to player positions. There are no other studies with precise ex-

TABLE 5. Initial symptoms for concussed players in National Football League games[a]

Symptoms	No. of cases	Incidence (%)[a]
General symptoms (median, 1; range, 0–5)	487	61.9 (58.5–65.3)
Headaches	433	55.0 (51.5–58.5)
Neck pain	99	12.6 (10.3–14.9)
Nausea	62	7.9 (6.0–9.8)
Syncope	13	1.7 (0.8–2.6)
Vomiting	9	1.1 (0.4–1.8)
Back pain	3	0.4 (0.0–0.8)
Seizures	1	0.1 (−0.1–0.3)
Cranial nerve symptoms (median, 1; range, 0–4)	416	52.9 (49.4–56.4)
Dizziness	329	41.8 (38.4–45.2)
Blurred vision	128	16.3 (13.7–18.9)
Vertigo	31	3.9 (2.5–5.3)
Photophobia	32	4.1 (2.7–5.5)
Tinnitus	21	2.7 (1.6–3.8)
Diplopia	16	2.0 (1.0–3.0)
Nystagmus	8	1.0 (0.3–1.7)
Pupil response	5	0.6 (0.1–1.1)
Pupil size	0	0.0 (0.0–0.0)
Hearing loss	0	0.0 (0.0–0.0)
Memory problems (median, 0; range, 0–4)	311	39.5 (36.1–42.9)
RGA delayed	142	18.0 (15.3–20.7)
Information-processing problems	138	17.5 (14.8–20.2)
Attention problems	102	13.0 (10.7–15.3)
AGA delayed	74	9.4 (7.4–11.4)
Cognitive problems (median, 0; range, 0–4)	217	27.6 (24.5–30.7)
Immediate recall	201	25.5 (22.5–28.5)
Not oriented with respect to time	63	8.0 (6.1–9.9)
Not oriented with respect to place	40	5.1 (3.6–6.6)
Not oriented with respect to persons	23	2.9 (1.7–4.1)
Somatic complaints (median, 0; range, 0–4)	158	20.1 (17.3–22.9)
Fatigue	71	9.0 (7.0–11.0)
Anxiety	41	5.2 (3.6–6.8)
Personality changes	39	5.0 (3.5–6.5)
Irritability	25	3.2 (2.0–4.4)
Sleep disturbances	6	0.8 (0.2–1.4)
Loss of appetite	2	0.3 (−0.1–0.7)
Depression	1	0.1 (−0.1–0.3)
Loss of libido	0	0.0 (0.0–0.0)
Total (median, 2; range, 0–12)	2158	
Unconsciousness (623 reported cases)	58	9.3 (7.0–11.6)

[a] Values in parentheses are 95% confidence intervals. RGA, retrograde amnesia; AGA, anterograde amnesia.

posure data. The importance of the exposure data is that they enable the calculation of MTBI incidences in this population. The incidence data are not based on estimates or extrapolations, as in previous reports of incidences of MTBI, cerebral concussion, or serious head injury.

Because case management was not mandated by the committee, the medical course of the subjects reflects the true natural history of MTBIs among professional football players during this 6-year period. These epidemiological data and the report of the natural history of MTBIs among professional

TABLE 6. Initial actions taken by team physicians for concussed players in National Football League games

Action taken	No. of cases	Incidence (%)[a]
Removed from play	346	44.0 (40.5–47.5)
Rested and returned	280	35.6 (32.3–38.9)
Returned immediately	127	16.1 (13.5–18.7)
Hospitalized	19	2.4 (1.3–3.5)
Unknown	15	1.9 (0.9–2.9)
Total	787	100

[a] Values in parentheses are 95% confidence intervals.

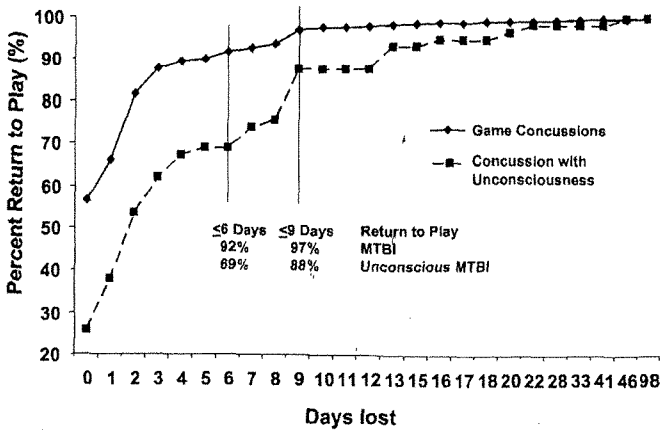

FIGURE 3. *Percentages of players returning to practice and games after concussions with or without unconsciousness.*

football players can serve as baseline findings for future monitoring. Because the monitoring of MTBIs will continue for the foreseeable future, future trends in MTBI incidence and severity in the NFL will be determined. This continuing effort will permit a study of the effects of newer protective equipment, rule changes, and perhaps even new medical treatments for head injuries on the incidence and severity of MTBIs. Such information should be of great value to professionals and students involved in brain injury. The applicability of these findings to athletes playing football at other levels (college, high school, or pee-wee), athletes participating in other contact sports, and/or the general population might be questioned. Certainly the players in this study are in a unique situation. They are highly trained, highly motivated, highly skilled athletes in excellent physical condition. They play with the best protective equipment available, which is maintained in the most efficient professional manner possible. They have the best training staff available to them on a daily basis. High-quality, personalized, medical care is also available. The players are monitored on a daily basis by athletic trainers and physicians who know them personally. However, despite these unique conditions, the players sustained MTBIs that seemed to be generally similar to the MTBIs experienced by other athletes and by the general population. This suggests that many of these findings are applicable to other athletes and the general population.

There have been previous attempts to study the epidemiological features of MTBI in the general population, among football players, and among other athletes. A brief review of those previous studies indicates the unique nature of this study. There have been many studies of head injury incidences in the general population (14). Those studies all yielded estimates of incidences, because the data were retrospectively collected from reviews of hospital records or insurance records; the values were then extrapolated to approximate incidences in the entire population. Such reported annual incidences of mild head injuries ranged from 131 cases/100,000 population to 511 cases/100,000 population. None of those studies relied on data collected from treating physicians, and none presented detailed physical findings.

There have been numerous attempts to epidemiologically study traumatic brain injury among football players. Gerberich et al. (16) reported on the incidence of football concussions in high schools in Minnesota. The data were collected with questionnaires sent to high school coaches and players. There were no physician reports. The data were all collected retrospectively. Torg et al. (43, 44) reported on the National Football Head and Neck Injury Registry. The criteria for inclusion in the registry were not those of MTBI. The criteria designated serious injuries involving hospitalization, surgery, paralysis, or death. Data collection was clearly retrospective, from reports completed by high school principals, athletic trainers, and members of the American College of Sports Medicine at the end of each season. Information was also collected by a news-clipping service. The later years of the study reportedly used prospective data collection, but most of the data collection was still performed at the end of the season, with forms completed by the persons noted above, and the inclusion criteria still designated only serious brain injuries. Torg et al. (44) mentioned earlier work by Schneider (41a), who collected cases of intracranial hemorrhage, not MTBI, among high school and college football players between 1959 and 1963. There were no detailed reports from physicians. There were no reports of symptoms of MTBI, and obviously the criterion of intracranial hemorrhage is not indicative of MTBI.

Powell and Barber-Foss (39) reported on brain injuries among high school athletes, in a prospective study. The data were provided by athletic trainers who volunteered for the study. A standardized reporting form was used. Of the athletic trainers who volunteered, a fraction were selected to participate in the study, on the basis of representation of high schools of different sizes and geographical distribution throughout the United States. Reportable injuries included those that caused the cessation of customary participation in the current session of play. This criterion is somewhat broad

and clearly includes many cases of MTBI. The reports were based on evaluations by athletic trainers, not physicians. An important strength of the study by Powell and Barber-Foss (39) was that incidents that occurred during practice and those that occurred during games were included. There was no report of clinical symptoms experienced by the players in the study. The total number of high school players in the United States was estimated from various sources; therefore, the incidence of traumatic brain injury could only be estimated from the data in the study.

Clarke (5) reported on the National Athletic Injury Report System, which was established in 1975. This report system was retrospective in nature. Concussion was defined as an incident of disorientation caused by trauma that required cessation of play. There were no reports of clinical symptoms. The reports were not based on physician examinations. There have been two reports of the incidence of concussions among Canadian football and soccer players (9, 10). Both studies were based solely on retrospective survey questionnaires completed by the players themselves. There were no physician reports or examinations. Both studies reported very high incidences of concussion symptoms among the athletes.

There have been a few attempts to study brain injury in other sports. Gerberich et al. (15) reported an epidemiological study of high school hockey players in Minnesota during the 1982/1983 season. Only 12 high school teams participated. The study was retrospective and was not based on physician reports. There was no report of clinical symptoms observed among the injured players. Boxers have also been studied on a few occasions. Larsson et al. (25) examined 44 Swedish amateur boxers before and after matches. The boxers underwent neurological examinations, including brief bedside cognitive testing. That was a very small study that did not establish the incidence of concussion in the population of boxers. The report did not provide much detail regarding the clinical symptoms that were observed for the participating boxers.

McCown (31) retrospectively analyzed results from New York State Athletic Commission records on professional boxers who were examined between 1950 and 1958. The report indicated that 11,103 boxers were examined, 325 boxers were knocked out, 789 experienced technical knockouts, and 10 required hospitalization. The article included a few detailed case reports of injured boxers but did not include any systematic review of symptoms observed among the boxers who were evaluated. There was also no indication of the course of the patients' symptoms after knockouts or technical knockouts.

Kaplan and Browder (22) evaluated 1043 professional boxers with electroencephalographic studies, ringside examinations, and evaluations of fight films. The authors did not specifically list the clinical findings, but they indicated that no neurological abnormalities were noted during ringside assessments or examinations in the training room after the fight, even among the fighters who lost in knockouts. No additional details regarding the clinical findings were provided.

Enzenauer et al. (12) reported on the incidence of boxing injuries in the United States Army between 1980 and 1985. That report was based on a retrospective review of Army hospital records. The only data available to the authors were the diagnoses, lengths of stay, and procedures performed. There were no clinical details regarding the symptoms. Because the data were collected from hospital records, only more serious head injuries were included and the report undoubtedly did not detect most cases of MTBI among Army boxers. Reviews of those earlier studies support the conclusion that our study is unique.

The results of this study help validate the information presented in the earlier study of the biomechanical features of concussions determined with video analyses and laboratory reconstructions of selected NFL MTBIs and severe head impacts (37, 38). One concern regarding the selection of the cases for video analysis was that the cases might not have been representative of most, if not all, concussions observed among NFL players. The analytical method required multiple camera angles and clear yard line and sideline markers for accurate determination of impact conditions. Therefore, the cases analyzed with the video techniques tended to be those that occurred in the open field. Such cases tended to involve impacts between wide receivers and defensive backs, impacts between wide receivers and the ground, players striking the quarterback, and special-team players running in the open field and striking other players in kicking plays. This study indicates that these are the same players who have the highest risk of sustaining MTBIs among all NFL players. The data indicate that quarterbacks, wide receivers, defensive backs, and special-team players on kicking units are more likely to sustain MTBIs than are players such as offensive and defensive linemen. This indicates that the cases selected for video analysis in the earlier studies were representative of many, if not all, NFL concussive events. Furthermore, comparison of the symptoms indicated that the video sample was reasonably representative.

Some of the results in this report can be understood in the context of the biomechanical data determined in the earlier studies (37, 38). The data indicate that quarterbacks have the highest risk of MTBI among all NFL players. Quarterbacks are representative of immobile or slowly moving players who are struck at high velocity by other players, often in situations in which the quarterback is unaware of the approaching player. The biomechanical data indicate that, in such situations, the high velocity of the striking player is transferred to the struck player (the quarterback), causing large changes in velocity and acceleration of the quarterback's head.

Defensive backs and wide receivers are the next most likely groups of NFL players to sustain clinical MTBIs. These players are moving at high speeds. They are often struck by more than one teammate or opposing player in high-velocity, high-acceleration impacts during tackling or blocking. Wide receivers are often struck in midair and fall backward, landing with the back of the helmet against the ground. As indicated by the biomechanical data, these high-velocity impacts often produce

head accelerations exceeding the tolerance levels and thus frequently result in clinical MTBIs. Offensive and defensive linemen have relatively lower risks of MTBI than do the aforementioned players. Offensive and defensive linemen tend to move at slower velocities for shorter distances; therefore, when they are struck in the head, the velocities and accelerations are lower than those observed among quarterbacks, wide receivers, and defensive backs. Because of these lower-acceleration impacts, lineman are less likely to experience impacts exceeding the tolerance levels and are less likely to sustain clinical MTBIs, except when they are running at full speed during kickoffs and punts.

One of the results might seem inconsistent with the biomechanical data. These data indicate that tackling players are somewhat more likely to sustain MTBIs than are players being tackled. Our biomechanical data indicated that the striking player does not sustain MTBI but the player being struck sustains MTBI because of the velocity being transferred from the striking player to the struck player. It might initially be presumed that the tackling player is always the striking player, but closer analysis indicates that this is often not the case. The tackling player, especially a relatively smaller defensive back, is often struck by a quickly moving, charging, offensive ball carrier, who becomes the striking player although he is being tackled. Therefore, the higher accelerations and velocities are often transferred to the tackling player. Furthermore, the tackling player is often struck by other tackling players from his own team or by blocking players from the opposing team during the act of tackling.

The tackler is usually focused on the ball carrier and often does not see the blocking opponent or his teammate who is also attempting to make a tackle. In these collisions, the tackling player often experiences a high-velocity impact, sustaining high accelerations to the head that result in concussion. Therefore, even this apparent inconsistency can be understood in terms of the biomechanical data presented by Pellman et al. (37, 38).

As indicated in *Table 5*, the most common initial symptoms for concussed players were headaches, dizziness, memory problems, cognitive problems, and somatic complaints. Headaches were observed for 55.0% of concussed NFL players. These results are consistent with the results observed in previous studies. Headaches occur among 30 to 90% of patients after concussions (14, 33). In recent studies of MTBIs among athletes, posttraumatic headaches were noted in 40 to 86% of cases (7, 18, 32). Our results were within that range and are consistent with those findings.

Dizziness, including vertigo, was observed for 45.7% of the NFL players with concussions. Dizziness has been frequently reported in studies of nonathletes with closed-head injuries. One study demonstrated that 53% of nonathletes complained of dizziness after mild head injuries (14, 27). Dizziness and vertigo are usually indicative of vestibular system dysfunction. This dysfunction is often thought to be of peripheral origin, but it is not clear whether some of the dizziness and vertigo might be of central origin. Earlier reports suggested that mild head injuries could cause benign positional vertigo. Nystagmus was infrequently noted among the NFL players. On the basis of examinations of boxers in the ring immediately after knockouts, nystagmus might be more frequently observed among concussed NFL players. The low incidence of nystagmus in this study could be related to the fact that very few of the NFL players experienced loss of consciousness or the fact that the players were not examined immediately after impact.

Blurred vision was observed for 16.3% of the study population. A previous study demonstrated a 14% incidence of blurred vision among nonathletes with concussions (33). Double vision was observed for 2% of the concussed NFL players. No additional details regarding the double vision were recorded in the survey, but double vision attributable to IVth cranial nerve palsy is well known to occur after mild head injury in the nonathlete population (24). Photophobia (sensitivity to light) was observed for 4.1% of the NFL players with concussions. In an earlier study, photophobia was observed for 7.2% of nonathletes examined 14 days after mild head injuries (14, 17). Photophobia may be part of a posttraumatic migraine syndrome.

Memory and cognitive problems were observed for a total of 45.9% of the NFL players after concussion. Memory problems were observed for 39.5% of the players and cognitive problems for 27.6%. Other sources indicated that retrograde amnesia is one of the most common forms of memory dysfunction with MTBI (27, 28, 30). Diagnostic approaches for assessment of memory function and cognitive problems were not uniform among the team physicians, which represents a potential error in the accuracy of reporting of such signs. Of the overall sample, 25.5% of players demonstrated difficulties with immediate recall in the initial examination, 8.0% were disoriented with respect to time, 5.1% were disoriented with respect to place, and 2.9% were disoriented with respect to person. Impairment of immediate recall was thus much more frequent than disorientation among the NFL players. These results confirm our clinical experience that it is not enough to ask a concussed athlete what year, month, or day it is to ascertain whether he is experiencing cognitive difficulties. It is necessary to specifically test for immediate recall in the initial patient examination after MTBI, to accurately assess the nature and extent of the injury. This is an important message for athletic trainers, sports medicine physicians, and others who examine athletes on the sidelines after mild head injuries. Team physicians were not given standardized questions or formats for assessment of information-processing ability or memory.

The 39.5% incidence of memory problems was approximately equally divided between retrograde amnesia, information-processing problems, attention problems, and anterograde amnesia. These incidences are consistent with the findings of previous studies of nonathletes and athletes after concussions. In one study of nonathletes examined 4 weeks after mild head injuries, 19% complained of memory losses and 21% complained of concentration difficulties (14, 33).

There is extensive literature on the neuropsychological sequelae of MTBIs among high school and college football players, as well as other athletes (6, 8, 11, 13, 14, 19, 46). Those articles reported significant abnormalities on cognitive and memory tests among concussed athletes. Our results are consistent with those earlier reports.

Somatic complaints were observed for 20.1% of concussed NFL players. The most common somatic complaints were fatigue, anxiety, personality changes, irritability, and sleep disturbances. These results are consistent with previous studies of symptoms noted after mild head injuries among nonathletes. It has been reported that 50 to 84% of patients experience these types of symptoms after cerebral concussion (14, 40–42). Fatigue was noted for 29% of patients evaluated 4 weeks after mild head injuries (14, 33). Disrupted sleep patterns have also been reported (14, 35). We are not aware of studies of the incidences of somatic complaints among other groups of athletes after mild head injuries. However, the MTBI results are consistent with findings reported for the nonathlete population, indicating the importance of inquiries about somatic complaints during evaluations of athletes after concussions. Complaints of fatigue, anxiety, personality changes, irritability, and sleep disturbances can be significantly disabling to the patient and must be taken seriously by the evaluating physician.

There have been a few studies of some symptoms after acute head injuries among amateur and professional boxers. Kaplan and Browder (22) reported normal neurological examination results for boxers after knockouts but noted that the "loss of awareness of one's surroundings" was often observed for fighters after concussions. Blonstein and Clarke (2) evaluated 29 amateur boxers who had been knocked out during the 1955/1956 boxing season in England. They did not report specific values, but they indicated that amnesia was frequently observed. Kaplan and Browder (22) and Blonstein and Clarke (2) also performed electroencephalographic examinations of fighters after cerebral concussion, but the comments regarding those findings did not mention clinical symptoms. In another report addressing head injuries in boxing, Winterstein (47) reported that nearly one-half of the boxers he examined described instances of anterograde amnesia. No additional details regarding the number of boxers examined or the nature or extent of the evaluations were provided in that report. It seems clear that the symptoms observed among concussed NFL players were generally consistent with the symptoms previously noted for athletes and nonathletes after MTBIs. These findings for a group of professional athletes are certainly more detailed and comprehensive than those reported in prior studies.

Only one NFL player experienced a seizure after MTBI. The seizure occurred in the locker room approximately 30 minutes after the injury and could therefore be classified as an early posttraumatic seizure. The patient made a full recovery. Posttraumatic seizures represent a rare occurrence in the general population after MTBI. They occur for 0.8 to 2.3% of patients during the first 1 week after mild head injuries (1, 14, 26).

For the great majority of concussed NFL players, MTBIs did not cause prolonged disability or prolonged absence from play. *Figure 3* indicates that 56.5% of the players (445 players) returned to play on the day of the injury and 97.1% (764 players) returned to play by day 9 after the injury. Only 23 players (2.9%) missed more than 9 days before returning to play. This indicates that most MTBIs in the NFL are self-limiting and players recover fully and spontaneously in a short time. *Table 6* indicates that more than one-half of the concussed players returned to play during the same game, either immediately (16.1%) or after a period of rest (35.6%). Conversely, 44.0% did not return to the same game. The data did not reveal some clinically pertinent information, such as the number of players who returned to play with symptoms, but they allowed an analysis of the types of symptoms that were more likely to be associated with removal from play. The multivariate logistic regression analysis of nonhospitalized players identified the number of symptoms (OR = 1.32 for each additional symptom), any memory problem (OR = 2.09), and any cognitive problem (OR = 3.41) as independent risk factors for resting of a player. The MTBI Committee did not intend to interfere with clinical decision-making by the individual medical staffs, and it must be assumed that there were variations in the treatment of the injuries. Because a significant percentage of players returned to play in the same game and the overwhelming majority of players with concussions were kept out of football-related activities for less than 1 week, it can be concluded that MTBIs in professional football represent mild injuries, in the context of the wide spectrum of diffuse brain injuries. This suggests that MTBIs in the NFL represent injuries in which symptoms resolve within a short time in the vast majority of cases.

Only 9.3% of the NFL players in this study experienced loss of consciousness as a result of severe concussive head impacts. This small number is consistent with clinical experience indicating that loss of consciousness is not a common occurrence with football-related MTBIs. It is important for all physicians who care for athletes with head injuries to know that most concussions they treat are not associated with loss of consciousness. Our data also demonstrated that concussed football players who sustained a loss of consciousness returned to play significantly later than did those who did not sustain a loss of consciousness. There are a few possible explanations for this finding. It could be that players who experience a loss of consciousness exhibit more severe symptoms or different patterns of symptoms than do those who do not lose consciousness. Players who lose consciousness may exhibit more abnormalities in examinations than do their counterparts who do not lose consciousness. It also may take longer for the symptoms of concussion to resolve among players who experience a loss of consciousness, compared with players who do not sustain a loss of consciousness.

It is also possible that the symptom of loss of consciousness itself is enough to make the treating physician more concerned regarding the potential seriousness of the injury and thus delay return to play longer, although the symptoms are not

different from those for players who did not sustain a loss of consciousness. The available data do not allow differentiation between these possibilities. However, a more detailed analysis of the subgroup of athletes who sustained a loss of consciousness is planned for a subsequent report.

MTBIs are relatively common injuries sustained by professional football players in game situations. As expected, the risk of injury is greatest for players whose positions more frequently involve high-velocity impacts (wide receivers and defensive backs). The data demonstrated that the quarterback is the most at-risk position, after adjustment for the number of positions on the field (*Table 2*). Rule changes have been implemented in the past to help protect this position from the potentially increased risk (e.g., pre- and post-throwing positions and blind-side impacts).

These data do not differentiate between initial and recurrent concussions. Repeated concussions are of concern because of the potential risks of chronic deficits and prolonged postconcussion syndrome. NFL players have occasionally been forced to retire because of prolonged postconcussion syndrome. However, most players return to play very soon after concussion and long-term sequelae after MTBI are quite rare in the NFL.

The results of a unique, prospective, 6-year study of the epidemiological features, natural history, and clinical features of MTBIs in professional football were presented. The results were analyzed with respect to player position and type of football activity (i.e., blocking, being blocked, tackling, or being tackled). Details of the clinical findings for MTBI were reported for 787 cases, and an attempt was made to correlate those findings with the biomechanical data gleaned from video analyses and impact reconstructions of concussions, as previously presented by Pellman et al. (37, 38). These findings should assist physicians in the diagnosis, treatment, and counseling of patients who sustain MTBIs.

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COMMENTS

In this article by Pellman et al., the third in a series, the authors detail the circumstances, causes, symptoms, and outcomes of game-related concussions in National Football League (NFL) players during a 6-year period. The authors found that quarterbacks, wide receivers, tight ends, and defensive secondary positions had the highest relative risk for concussion, and kickoffs and punt returns were the types of play that carried the highest relative risk for concussion. Importantly, they also showed that only 9% of concussed players lost consciousness, emphasizing the fact that in the great majority of cases, concussion does not equate with unconsciousness. Instead, the most common signs and symptoms were headache, dizziness, memory problems, and cognitive complaints. The authors have provided a comprehensive and in-depth analysis of this complex clinical problem. This work will allow focus to be placed on the most vulnerable players in the game to reduce the incidence and severity of concussion in football players at all levels. Further study is warranted on the long-term neurobehavioral effects of repeated concussions relative to concussion frequency, injury severity, and time interval between concussions.

Daniel F. Kelly
Los Angeles, California

This article is the third in a series of research observations supported by the NFL and performed through the NFL Committee on Mild Traumatic Brain Injury (MTBI) in response to safety concerns regarding head injuries in the NFL. The present observations represent the institutionalization of change in the former understanding of a concussion as an episode associated with loss of consciousness to the currently recognized definition as "an altered mental state regardless of duration and/or altered memory, regardless of duration or content that results from trauma." The authors' description and categorization of symptoms and signs commonly associated with concussion clearly establish these as the hallmarks in defining MTBI in sports.

During the 6-year study period, 3826 team games were analyzed for the incidence and severity of MTBI by player position. From preseason, regular, and playoff game-related concussions, there were 787 reported cases of MTBI. The incidence was only 0.41 concussion per NFL game. This seemingly low incidence perhaps highlights one of the few limitations of the study: the known reluctance of some athletes to report symptoms and signs. Also, with the numerous individual team physicians and trainers filling out the collection forms, interobserver reliability and lack thereof remains a question. In these incidents, 16.1% of players returned immediately to the game, 35.6% returned later in the game, and 44% were removed from further contact that day; 2.4% were subsequently hospitalized.

As one would expect, the risk of injury is greatest for players involved in high-velocity impacts, such as quarterbacks, wide receivers, and defensive backs. Because of the high vulnerability of quarterbacks, rule changes were implemented to help protect against blind-side impacts and prethrowing and postthrowing positions.

The great value in this study is the promulgation of a standard definition, the delineation of players at greatest risk, and the detailed analysis of symptoms and signs associated with MTBI. It is a prospective study that used a standardized reporting form in which all subjects were evaluated by physicians, and there was 100% participation from all players in the NFL, with none lost to follow-up. The NFL and the members of the MTBI Committee of the NFL are to be commended for their concise and succinct summary of 5 years of work.

Joseph C. Maroon
Pittsburgh, Pennsylvania

In their latest article investigating concussion during professional football games in the United States, Pellman et al. change their focus from biomechanics to an analysis of the positions most likely to sustain concussion and the most common signs and symptoms. The fact that quarterbacks were found to have the highest relative risk of concussion should come as no surprise to anyone who reads the sports pages between August and January. However, the finding that is perhaps most important for all practitioners (even those who are not sports fans) is that fewer than 10% of all concussions were complicated by loss of consciousness. It cannot be stated often enough to the general public and even to other medical practitioners that concussion can occur without loss of consciousness and, in fact, does so quite frequently.

Fortunately, in the vast majority of cases, MTBI in football players seems to resolve quickly. Most players are able to return to competition after a relatively brief period. The subset of patients who sustain loss of consciousness may experience more significant sequelae, and the authors report that they plan to present a more detailed analysis of these athletes in a future article. I look forward to reading that article. The authors also state that they plan to investigate the effects of recurrent concussions in players. Concerns about the effects of repeated concussions seem to have played a role in the retirement of some of the most prominent football players of recent years. The high visibility and high stakes of such decisions guarantee that the publication of that article will also be eagerly awaited.

Alex B. Valadka
Houston, Texas

This article represents the third part of an informative series regarding concussive injury in the NFL. A significant contribution is the documentation that players in the NFL with MTBI tend to return to play quickly without increasing the chance for subsequent sequelae. This counters an existing literature that tends to suggest that even minimally symptomatic players with MTBI have serious injuries. It is unfortunate that, to date, only a few articles discussing MTBI in general have been prospective in nature, especially given the burden of a 3.9 to 7.7% rate of MTBI in high school and college athletes each year in all sports. We agree that the use of standardized criteria for recording variables related to the injury is a significant contribution, especially as determined by a small, select group of trainers and/or physicians. The realization that 182 severe game impacts were the result of spearing should support the need for a more aggressive stance on the part of the NFL with regard to penalization of the offending player. The authors will be pursuing repeat concussion in a future article, given the significance of such injuries with regard to long-term and even career-ending sequelae.

As with any study of this sort, a number of issues remain problematic. Our experience has been that motivated players will universally underrepresent their injuries, given the will to return to play. Given the caliber and motivation of elite athletes, there is little chance that this will change. Thus, criteria to assess players with potential MTBI need to account for this variable. Close relationships between the trainers, players, and physicians are necessary to further reduce the impact of a player minimizing his injuries. In addition, close adherence to strict criteria of evaluation will further diminish such issues with regard to return-to-play considerations.

Our greatest concern is that the physicians and trainers truly represent the well-being and long-term health of the players, as opposed to the specific ball club or the NFL. It is unusual that neurosurgical coverage tends to be limited in the NFL, especially given the significant knowledge base regarding head injury that we harbor. In a recent Monday Night Football game, Jeff Garcia (quarterback of the San Francisco 49ers) probably sustained a concussion as the result of a open-field tackle. Replay analysis represented the tackle itself and Garcia's hesitancy to leave the field. Without the benefit of performing an examination of the player, any consideration of the degree of his injury is only conjecture. Conversely, the visual evidence was enough to suggest that he did receive a significant impact that was associated with a concussive injury. In any case, the rapidity with which he returned to play was of concern. It has been only a few years since Troy Aikman of the Dallas Cowboys was rendered unconscious on the playing field, only to return to play later in the game.

Burak Ozgur
Michael L. Levy
San Diego, California

The NFL's Committee on MTBI has analyzed clinical data regarding what they believe is a comprehensive capture mechanism of virtually all instances of concussion in the league during the 1996 to 2001 football seasons. This study has yielded information from 787 MTBIs that occurred throughout the playing seasons in 1913 games and consisted of both initial and follow-up evaluations. The findings included the fact that although the positions most often associated with concussion are the defensive secondary, followed by the kicking team members and wide receivers, the quarterback position has the highest relative risk for sustaining concussion. This confirms our previous conviction that those involved in high-speed collisions, that is, the quarterbacks, running backs, wide receivers, and defensive secondary players, are at greatest risk for sustaining concussion. However, depending on size, ve-

locity, and other factors, the tackler may not always be the one imparting the greater force and may himself sustain the concussion. In addition, the quarterback is often the victim of a "blind-side" hit, being unaware of the closing angle, speed, and presence of the tackler, and is often a stationary target absorbing and not delivering a force vector to the opponent.

The findings also showed that most concussed football players had two symptoms, with headaches, dizziness, and blurred vision being the most common. Either cognitive or memory problems or both were seen in 46%, whereas, interestingly, there was no documentation of motor or sensory abnormalities in any players. It is also of interest to note that only a minority (16.1%) of MTBI players returned to play immediately within the same game, whereas 35.6% rested for an extended period before returning to the same game, and 44% were removed for the remainder of that contest. Loss of consciousness was present in 9.3%, and only 19 players (2.4%) required hospitalization. The return to play was rapid, because 92% of concussed players had 6 or fewer days away from play, and 97% were absent for fewer than 10 days.

This report contributes much to our understanding of the incidence and characteristics of football-related MTBI, at least at the highest level of competition. The methodology was inclusive, allowing exposure and incidence to be determined accurately, as well as presenting symptomatology. Although there are some limitations to their data, as the authors admit, this study involved examinations by physicians and good follow-up information, including the athletes' ability to return to competition. It did not differentiate between initial and multiple concussions. The large number of concussions in highly skilled and conditioned athletes, the data related to playing position, physical examination and presentation, and completeness make this study unique. The findings also underscore the importance of assessing memory by testing for immediate recall in the sideline evaluation. The authors have presented a thorough study that documents many important features of athletic MTBI at the professional level.

Julian E. Bailes
Morgantown, West Virginia

TAB 1D

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Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProHEALTH Care Associates, LLP, Lake Success, New York

David C. Viano, Dr. med., Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park, New York; Department of Neurology, New York University Medical Center, New York, New York; and Department of Neurology, Albert Einstein College of Medicine, Bronx, New York

Andrew M. Tucker, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and University of Maryland School of Medicine, Timonium, Maryland

Joseph F. Waeckerle, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Acute Care and Emergency Specialists, MD, PC, Leawood, Kansas

John W. Powell, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University, Lansing, Michigan

Henry Feuer, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Indianapolis Neurosurgical Group, Indianapolis, Indiana

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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CONCUSSION IN PROFESSIONAL FOOTBALL: REPEAT INJURIES—PART 4

OBJECTIVE: A 6-year study was conducted to determine the signs, symptoms, and management of repeat concussion in National Football League players.

METHODS: From 1996 to 2001, concussions were reported by 30 National Football League teams using a standardized reporting form filled out by team physicians with input from athletic trainers. Signs and symptoms were grouped by general symptoms, somatic complaints, cranial nerve effects, cognition problems, memory problems, and unconsciousness. Medical actions taken and management were recorded.

RESULTS: Data were captured for 887 concussions in practices and games involving 650 players. A total of 160 players experienced repeat injury, with 51 having three or more concussions during the study period. The median time between injuries was 374.5 days, with only six concussions occurring within 2 weeks of the initial injury. Repeat concussions were more prevalent in the secondary (16.9%), the kick unit on special teams (16.3%), and wide receivers (12.5%). The ball return carrier on special teams (odds ratio [OR] = 2.08, P = not significant) and quarterbacks (OR = 1.92, $P < 0.1$) had elevated odds for repeat injury, followed by the tight end (OR = 1.24, P = not significant) and linebackers (OR = 1.22, P = not significant). There were similar signs and symptoms with single and repeat concussion, except for a higher prevalence of somatic complaints in players on their repeat concussions compared with their first concussion (27.5% versus 18.8%, $P < 0.05$). More than 90% of players were managed by rest, and 57.5% of those with second injuries returned to play within a day. Players with three or more concussions had signs, symptoms, and treatment similar to those with only a single injury.

CONCLUSION: The most vulnerable players for repeat concussion in professional football are the ball return carrier on special teams and quarterbacks. Single and repeat concussions are managed conservatively with rest, and most players return quickly to play.

KEY WORDS: Concussion, Epidemiology, Injury surveillance, Repeat or multiple concussions, Second-impact syndrome, Traumatic brain injury

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Physicians have been concerned for many years about the possible deleterious effects of multiple concussions on the brains of athletes. The areas of concern range from chronic mild or moderate cognitive and memory impairments to a full-blown chronic encephalopathy syndrome and from an increased risk of repeat concussion to prolonged postconcussion syndrome and the rare but lethal so-called second-impact syndrome (SIS). Martland (25) first described a syndrome of chronic brain damage in boxers, presumably related to multiple blows to the head.

In 1945, Quigley expressed concern about repeat head injuries in other sports by stating that "three concussions in one season dictated discontinuing participation in that sport" (41–43). The influence of this statement persists to the present time, as indicated by the recommendations of concussion management guidelines that players who sustain three concussions in one season should be withdrawn from play for the remainder of that season (1, 6, 11, 37). In the 1970s and 1980s, a number of articles raised the specter of the so-called SIS and related it to "additive or compounding

effects of minor impact injuries" (41). This rare syndrome has been defined as occurring when "an athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before symptoms associated with the first are fully cleared" (7).

The SIS has been characterized by rapid clinical deterioration resulting from cerebral hyperemia and an increase in intracranial pressure, with a high rate of mortality, after a second impact to a brain still symptomatic from an initial head injury. The existence of SIS, as it has been described and defined, has been questioned (28, 30). However, persistent concerns about the SIS still influence the authors of current guidelines, who recommend rigid time intervals that delay return to play after many grades of concussion (1, 6, 11, 37). Starting in the 1970s (17) and continuing in recent years, numerous studies have documented the increased cognitive impairments seen after repeat concussions compared with the first injury (9, 18, 24, 45). These studies have prompted concerns about more subtle chronic brain damage caused by multiple concussions than that seen in the full-blown chronic encephalopathy of boxers.

There have been numerous clinical studies defining and describing the chronic encephalopathy of boxers (8, 31, 32, 39, 40). Clinical studies and relevant animal studies all indicate that the pattern of chronic brain damage is a result of a large number of subconcussive blows to the head over a relatively long period of time (44). However, this injury has not been observed in professional football.

In the past 10 years, a number of highly publicized retirements of well-known professional athletes after they sustained multiple concussions have raised the issue of prolonged or persistent postconcussion syndrome occurring as a result of multiple concussions (15). Over the years, there have also been numerous reports indicating that players who sustain one concussion are at significantly increased risk of sustaining a repeat concussion as well as being more likely to sustain a more serious injury as a result of the repeat concussion (6, 9, 16, 18).

In dealing with all of these appropriate concerns, treating physicians until now have had to rely on personal experience, anecdotal information, case reports, and studies of small numbers of players with repeat mild traumatic brain injury (MTBI) to aid in their decision-making. The purpose of the present study is to analyze in detail the results of a 6-year prospective study of a relatively large number of repeat concussions in the National Football League (NFL). It is hoped that this will help the medical community to address its numerous concerns regarding the effects of multiple concussions in athletes.

In 1994, the NFL formed the Committee on Mild Traumatic Brain Injury in response to safety concerns regarding head injuries. Background on the committee has been provided by Pellman (33). Its mission was to investigate MTBI in the NFL by various scientific methods.

The MTBI Committee undertook a series of research projects aimed at defining concussion biomechanics in professional football. On the basis of analysis of game video and laboratory reconstruction of severe impacts using instrumented test dummies, the biomechanics of concussion has been determined for professional players. This has included data on the head acceleration of injury (36) and the location and direction of impacts (35).

The committee also determined a strong need to monitor the frequency and to have physician evaluation of MTBI in the NFL and at the same time to identify the clinical signs and symptoms associated with concussion. The initial study focused on 787 concussions during regular-season NFL games (34). It addressed the signs and symptoms of concussion and determined the relative risk by player position. The present study involved all 887 concussions reported over a period of 6 years from 1996 to 2001 in practice and all games. During this period, 787 (89%) of 887 concussions occurred in regular-season games. This study discusses repeat concussions in professional football, including the clinical signs and symptoms, medical actions taken, and lost days with single and repeat injury.

PATIENTS AND METHODS

The MTBI Committee devised a simple form for team physicians to complete on observed and reported signs and symptoms on initial and follow-up examinations whenever they evaluated a player who sustained concussion. At the NFL level, there is close cooperation between team physicians and athletic trainers on player medical issues, and they worked together to collect cases and data for this study. All players were examined by team physicians, and all management decisions were made by physicians. During the study period, two teams were added to the NFL. This registry of concussions involved MTBI data from 30 teams in the NFL. The median number of concussions reported by the teams was 26 (range, 6–72) during the study period. Players' names were not included on the forms to maintain confidentiality. They were identified by a six-digit number.

Operational Definitions

The definition introduced by the committee in 1996 and used for the study is as follows. A *reportable MTBI* is a traumatically induced alteration in brain function, which is manifested by alteration of awareness or consciousness, including but not limited to being dinged, dazed, stunned, woozy, foggy, amnesic, or, less commonly, rendered unconscious, or even more rarely, experiencing seizure; or by signs and symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, light-headedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbance, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities.

The definition is a natural extension of a much earlier one from the Ad Hoc Committee to Study Head Injury Nomen-

clature of the Congress of Neurological Surgeons (12) and is consistent with a more recent definition by the American Congress of Rehabilitation Medicine (2).

Alteration of Awareness

There may be occasional difficulty in eliciting a history of loss of consciousness or a transient alteration in awareness in professional football players. Loss of consciousness may be very short-lived (a few seconds or less) and thus may not be directly witnessed by the athletic trainer or medical staff. In addition, the player may not want to admit that such an event occurred because of a concern that he may lose playing time, although this tendency is waning with a loss of stigma associated with MTBI. Any player who met the all-inclusive criteria as determined by the medical staff and athletic trainers was included as an MTBI case.

Signs and Symptoms

MTBI is a clinical syndrome that may present with a broad spectrum of signs and symptoms, many of which are nonspecific and can be associated with other clinical diagnoses. The MTBI Committee members who are team physicians in the NFL, as well as MTBI Committee consultants with special expertise in the fields of sport neuropsychology and sport neurology, developed a list of the most common signs and symptoms with concussion. They were grouped into six categories: 1) general symptoms, 2) somatic complaints, 3) cranial nerve findings, 4) cognitive abnormalities, 5) memory problems, and 6) unconsciousness. The checklist was filled out for each player with a concussion.

A purposely large and inclusive list was selected so as to capture all of the possible clinical signs and symptoms with MTBI in professional football players. The signs and symptoms that were recorded are consistent with previous medical literature on the postconcussion syndrome and the symptoms and signs seen after traumatic brain injury. Most of the items are symptoms the player may complain of or the physician may elicit by history. Some items are mental status findings (retrograde amnesia, anterograde amnesia, or problems with information processing, attention, and immediate recall). The committee did not distribute uniform testing instruments to the team physicians but rather left the assessment of these parameters to the discretion of the individual team physicians. The rationale for the various signs and symptoms can be found in a report by Pellman et al. (34).

The form also contained questions about physical examination findings, initial management, tests ordered, and disposition regarding return to play as well as information on the equipment worn, impact types, and conditions of the field and play. A form was generated for each player's MTBI, including the initial evaluation and all subsequent follow-up visits until the player was cleared for return to play. Team physicians and their consultants used their own evaluation procedures to manage the injury. The committee did not impose outside medical decision-making on the medical staffs of the individual teams. The individual team physicians were to complete the initial and follow-up forms on the basis of their clinical findings.

Return to Play

The following definitions apply to the return-to-play aspect of the medical report.

- *Return immediately:* The player returns after an evaluation by the team physician demonstrates that the player is asymptomatic. The key here is that the player, because of his relative position on the team, may not be called to action for several minutes. For example, if the player was on the kickoff team and sustained an MTBI, the physician would perform an evaluation and determine that the player is ready to return, yet, depending on the game, it may be minutes or possibly an hour or so before he actually gets back on the field.
- *Rest and return:* The player is evaluated, and it is determined that there should be some protracted time before a decision is made to return. The key would be that the player did eventually return to the same game or practice. An example might be that the injury occurs in the last 5 minutes of the second quarter. Because it is close to halftime, the decision to return is not made until the third quarter.
- *Removed from play*: This means that the player was not allowed to return to the game or session in which he was injured.
- *Hospitalized:* The player was admitted to the hospital; this is generally is characterized as 18 hours or more of hospitalization. This would mean that going to a local hospital for an x-ray, head computed tomographic scan, or the like and then going home would not be classified as "hospitalized."

Days Out

The definition of *days out* is the time between the date of injury and the date that the player was permitted to return to full and unlimited participation (38). *Full and unlimited participation* means that the player must be able to perform all the activities of the session at the same intensity as his teammates. If a player were in a practice session and was not allowed to participate in contact drills, he would not be considered to be returned to participation, because he was not able to perform all of the activities expected of his teammates. In essence, this tells us that on the date of return, the player was expected to participate fully in all of the activities that were planned for the team practice or game.

Efforts to Improve Compliance

The Commissioner of the NFL encouraged all team physicians to complete and return forms whenever they examined a player with a head injury. The project was designed to record information about the injury. The forms were designed for ease of completion, and the data were limited to those points that would provide the most relevant information on MTBI to improve compliance. The data forms were sent to the NFL epidemiologist and entered into a database with a

blinded coding to maintain anonymity of the players. When an initial evaluation form was submitted but the follow-up visit form was not, committee members contacted team athletic trainers and doctors directly to remind them to submit the follow-up forms. During biannual meetings, the committee monitored the data and discussed findings.

Quality Assurances

The MTBI evaluation forms were logged in by the committee's epidemiologist and scanned into a database using a commercial software program (Teleforms, Cardiff, CA). During the data logging, the individual forms were manually reviewed. Each form was then scanned into a temporary database and verified before being entered into the final database. Any fields that were incomplete or inconsistent triggered a follow-up contact with the team athletic trainer or physician to verify the data. The final database includes information from the initial and follow-up evaluation forms submitted by team physicians.

Statistics

Descriptive statistics were used to characterize those players who had a single concussion and those who had repeated concussions during the 6-year study. χ^2 analyses were used to compare the presence of individual signs and symptoms, medical action, and management of those with repeat concussions with those with a single concussion. *t* tests were used to compare the mean number of signs and symptoms for those with single and repeated concussions.

Odds ratios (ORs) with 95% confidence intervals (CIs) were used to summarize the magnitude of associations. Paired *t* tests and the McNemar test were used to compare those same items for players on their first and second concussions within the 6-year study. Because the number of signs and symptoms may accumulate in closely spaced injuries, the correlation between the change in number of signs and symptoms and time interval was calculated. A separate analysis was conducted on the 51 players with three or more concussions. This analysis included a review of their signs and symptoms for each injury and days out. Differences between injuries for the same player were evaluated with the McNemar test. The results for the 15 players with four or more concussion (46 concussions) were summarized separately.

RESULTS

Repeat Concussion

Table 1 shows the prevalence of single and repeat concussions during the 1996 to 2001 NFL seasons. There were 887 reported MTBI cases in practice and preseason, regular-season, and postseason games. This involved 650 players, 160 (24.6%) of whom had repeat concussions. Fifty-one concussed players (7.9%) experienced three or more injuries, and one player had seven concussions during the study period.

TABLE 1. Single and repeat concussions in the National Football League, 1996–2001

No. of concussions	No. of players	Percentage	Total concussions
1	490	75.6%	490
2	109	16.8%	218
3	36	5.6%	108
4	8	1.2%	32
5	4	0.6%	20
6	2	0.3%	12
7	1	0.2%	7
Total	650	100%	887

The median duration between the first and second concussions was 374.5 days (range, 0–1693 d). Of those occurring within a 90-day window (n = 38), the median was 31.5 days. Of these 38 players, 36 received their injuries during regular games in the same season. Only six concussions occurred within 2 weeks of the initial injury.

Table 2 shows the prevalence of concussion by player position. It includes the frequency of single and the first of repeat concussions over six seasons. As a group, the offensive team has a slightly higher frequency of single and repeat concussions. Individually, the position groups most often associated with the first of repeat concussions are the defensive secondary (16.9%), kick unit (16.3%), wide receivers (12.5%), and defensive line (10.0%). The defensive secondary includes the safety and cornerbacks, and wide receivers include flankers and split ends.

The ORs in *Table 2* show that only two position groups have statistically elevated odds of repeat concussions. Quarterbacks (OR = 1.92; 95% CI, 0.99–3.74; $P < 0.1$) have higher odds of repeat concussion, and the offensive line (OR = 0.54; 95% CI, 0.27–1.08; $P < 0.1$) has lower odds. Ball carriers on special teams (OR = 2.08; 95% CI, 0.73–5.93) have the highest point estimate of repeat injury, but the numbers are very small. The other positions at elevated odds include tight ends (OR = 1.24; 95% CI, 0.58–2.64) and linebackers (OR = 1.22; 95% CI, 0.63–2.38). There was no difference in proportion of injuries that occurred in games versus practice across single concussions (85.9%), first of repeat concussions (87.5%), and second of repeat concussions (94.4%).

Table 3 summarizes the signs and symptoms for single and repeat concussions during the 1996 to 2001 seasons. The presence of signs and symptoms for the 490 players with a single concussion and 160 players with repeat injuries (on their first injury) were compared by use of χ^2 analysis. The prevalence of signs and symptoms was similar for players experiencing one concussion and the first of repeat concussions, except for a higher prevalence of diplopia ($P < 0.01$) and lower prevalence

TABLE 2. Incidence of single and first of repeat concussions in six National Football League seasons[a]

	Single concussion		First of repeat concussions		Odds ratio	95% confidence interval	Three or more concussions	
	No.	%	No.	%			No.	%
Offense								
Wide receiver	57	8.6%	20	12.5%	1.08	0.63–1.87	8	15.4%
Running back	42	8.6%	14	8.8%	1.02	0.54–1.92	6	11.5%
Quarterback	25	5.1%	15	9.4%	1.92[b]	0.99–3.74	5	9.6%
Offensive line	54	11.0%	10	6.3%	0.54[b]	0.27–1.08	0	
Tight end	25	5.1%	10	6.3%	1.24	0.58–2.64	4	7.7%
Subtotal	203	41.4%	69	43.1%	1.07	0.75–1.53	23	44.2%
Defense								
Secondary	80	16.3%	27	16.9%	1.04	0.64–1.67	6	11.5%
Defensive line	43	8.8%	16	10.0%	1.15	0.63–2.11	5	9.6%
Linebacker	33	6.7%	13	8.1%	1.22	0.63–2.38	5	9.6%
Subtotal	156	31.8%	56	35.0%	1.15	0.79–1.68	16	30.8%
Special team								
Kick unit	92	18.8%	26	16.3%	0.84	0.52–1.35	8	15.4%
Return unit	18	3.7%	2	1.3%	0.33	0.08–1.44	1	1.9%
Return ball carrier	9	1.8%	6	3.8%	2.08	0.73–5.93	2	3.8%
Punter	8	1.6%	0				0	
Kicker, FGA	1	0.2%	0				0	
Kicker, PAT	1	0.2%	0				0	
Holder	1	0.2%	0				0	
Subtotal	130	26.5%	34	21.3%	0.75	0.49–1.16	11	21.2%
Unknown	1	0.2%	1	0.6%	3.07	0.19–49.35	1	1.9%
Total	490	100%	160	100%			51	100%

[a] FGA, field goal attempt; PAT, point after touchdown.
[b] $P < 0.10$ from χ^2 analysis comparing positions having repeat versus single concussion versus all other positions.

of personality change ($P < 0.01$) in those who later incurred a repeat concussion.

The prevalence of somatic complaints was higher at the second concussion ($P < 0.05$) by use of an analysis that compares the presence of signs and symptoms on the initial and repeat concussions with the McNemar test. Loss of consciousness for 1 minute or more ranged from six (1.2%) in the single-concussion group to three (1.9%) in the initial and four (2.5%) in the second concussion groups (P = not significant). With a paired t test, there was no statistical difference in the mean number of signs and symptoms from the initial to the repeat concussion. Likewise, there was no correlation between the change in number of signs and symptoms and the time interval between the initial and repeat concussions.

Table 4 summarizes the medical action taken after injury. The most common action with the player who has a single concussion is to remove him from play (50.4%), as it is with the initial (45.6%) and repeat concussion (41.3%). Of the 36 players with a second injury during regular games in the same season, 50% were removed from play.

Table 5 shows the management of concussion. Overall, 97.4% of known cases of the players with repeat concussion were managed with rest. This is slightly higher than 94.3% for those with a single concussion. These frequencies are essentially similar, because only a few players were given various drug therapies or medical procedures, which were not further defined in the medical form.

Figure 1 shows days out from play after single and repeat concussion. Of the concussed players, 91.9% returned to play within a week. The proportions of players who were not cleared for play for 7 days or more were 7.8% (single injury), 5.0% (initial), and 10.0% (repeat concussions). Of these players, 13.0% were out 7 days or longer with three or more concussions. Not shown in *Figure 1* is that 50% of the players with same-season regular-game second injury were not cleared for play within 6 days.

Three or More Concussions

Table 6 gives the signs and symptoms and days out for the 51 players experiencing three or more concussions in the

TABLE 3. Signs and symptoms of single and repeat concussions in the National Football League (data on repeat concussions for first, second, and third injury)[a]

Signs and symptoms	Single concussion (n = 490)	Repeat concussions		
		First (n = 160)	Second (n = 160)	Third (n = 51)
General symptoms	65.1%	61.3%	65.0%	64.7%
Headaches	58.0%	56.3%	56.3%	58.8%
Nausea	9.0%	11.9%	6.9%	5.9%
Vomiting	2.4%	0.6%	0.6%	
Neck pain	12.9%	11.3%	14.0%	15.7%
Back pain	0.6%			
Syncope	1.4%	1.9%	1.9%	
Seizures		0.6%		
Somatic complaints	18.0%	18.8%[b]	27.5%[b]	17.6%
Irritability	2.2%	3.1%	5.0%	2.0%
Anxiety	3.5%	5.6%	10.6%	5.9%
Depression	0.2%		1.3%	
Personality change	5.7%[b]	1.9%[b]	6.9%[b]	2.0%
Fatigue	9.0%	8.8%	10.0%	7.8%
Sleep disturbance	1.0%			2.0%
Loss of libido				
Loss of appetite	0.4%			
Cranial nerve effects	50.8%	53.1%	60.0%	56.9%
Dizziness	42.9%	39.4%	44.4%	41.2%
Vertigo	3.7%	4.4%	3.1%	3.9%
Tinnitus	2.7%	1.9%	2.5%	5.9%
Nystagmus	0.8%	1.3%	1.3%	2.0%
Hearing loss				
Diplopia	1.0%[c]	5.0%[c]	2.5%	
Photophobia	2.9%	4.4%	6.9%	3.9%
Blurred vision	13.9%	19.4%	18.8%	11.8%
Pupil response	0.6%	1.3%		
Pupil size	0.2%			
Cognition problems	26.7%	29.4%	27.5%	19.6%
Not oriented to person	2.9%	1.9%	3.8%	2.0%
Not oriented to place	5.5%	3.8%	6.9%	
Not oriented to time	7.1%	7.5%	11.9%	2.0%
Immediate recall	24.3%	26.3%	26.3%	17.6%
Memory problems	37.8%	39.4%	41.9%	23.5%
Attention problems	13.5%	12.5%	15.6%	7.8%
Information processing	16.1%	16.3%	21.3%	5.9%
AGA delayed	9.8%	10.6%	9.4%	
RGA delayed	17.1%	18.8%	17.5%	11.8%
Unconsciousness (>1 min)	6	3	4	0
All loss of consciousness	25	10	16	5
All reported as zero	154	55	64	15
Mean no. of signs and symptoms	2.7	2.8	3.1	2.2
Range	0–12	0–11	0–12	0–12

[a] AGA, anterograde amnesia; RGA, retrograde amnesia. Comparisons were made between single concussion and first of repeat concussions using χ^2 test or Fisher's exact test. Comparisons were made between first and second concussions of repeat concussions using the McNemar test.
[b] $P < 0.05$.
[c] $P < 0.01$.

TABLE 4. Action taken with players experiencing single and repeat concussions in the National Football League[a]

Action taken	Single concussion (n = 490)	Repeat concussion	
		First (n = 160)	Second (n = 160)
Return immediately	16.9%	15.6%	11.9%
Rest and return	29.6%	34.4%	40.6%
Removed from play	50.4%	45.6%	41.3%
Hospitalized	1.4%	1.3%	4.4%
Unknown	1.6%	3.1%	1.9%

[a] Comparison of single versus first of repeat concussions was not significant by χ^2 analysis.

TABLE 5. Management of players experiencing single and repeat concussions in the National Football League[a]

Management	Single concussion (n = 490)	Repeat concussion	
		First (n = 160)	Second (n = 160)
Rest	88.2%	92.5%	94.4%
Prescription drug therapy	1.4%	1.3%	0.6%
Proprietary prescription	0.6%	0.6%	1.3%
Therapeutic modality	1.0%	3.1%	0.6%
Medical procedures	1.6%		
Immobilization	0.6%	0.6%	
Not recorded	6.5%	1.9%	3.1%

[a] Comparison of single versus first of repeat concussions was not significant by χ^2 analysis.

6-year study period. The average duration between concussions was 1.05 years for the first and second injuries and 1.20 years for the second and third injuries in the study period. Overall, there was a slight decline in the number of signs and symptoms with increasing number of concussions, but a very similar pattern is seen for the first through third injuries. Only one symptom/sign was significantly different between the injuries. Players were more likely to be reported as having blurred vision on their first concussion than on their third ($P < 0.05$). The median number of days out is 0 days out for each group. A review of medical action was made, and the proportions of removed from play, rest and return, and hospitalization were similar for the first through fourth concussions. On average, 46% were rested and returned, 37% were removed from play, 13% returned immediately, and 4% were hospitalized.

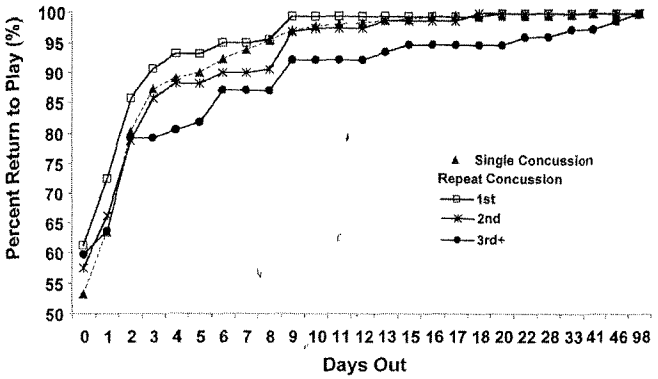

FIGURE 1. *Graph showing percentage of NFL players returning to practice and games after single (only one) and repeat concussions during the 6-year study period versus the days lost from full participation in practice and games (note: the x-axis does not show equally spaced time intervals past 13 d lost).*

DISCUSSION

In this article, the clinical picture of repeat MTBIs is described in professional football players during a 6-year period from 1996 to 2001. This is the first study that prospectively collected clinical data from the treating physicians and athletic trainers of such a large number of repeat concussion cases. This report represents a comprehensive, detailed, scientifically based attempt to provide the physician who treats athletes with objective data on which to build evidence-based management and treatment plans, although care must be taken in applying the information to other age groups and levels of competition. Until recently, there had been few prospective reports that attempted to look at the effects of repeat MTBIs in athletes. With the increased use of standardized sideline assessment tests and user-friendly neuropsychological tests at the high school and collegiate levels, more evidence-based studies are now appearing in the sports medicine literature (18, 21, 27). These recent reports complement our study, although there are differences in the approaches taken and the findings. Before the general discussion of this study and its implications are approached, limitations for the research are described. This provides a framework within which to consider the study of repeat concussions in the NFL.

Limitations

As with all research, there are limitations to this study. We did not collect retrospective data on previous concussion history as part of this study. Some of the players may have had previous concussions either in the NFL in the years before the study began or during their playing careers in high school, college, or other levels of football. It is also possible that some of the players sustained cerebral concussions at earlier times in their lives in nonfootball athletic or nonathletic endeavors.

TABLE 6. Analysis of 51 players with three or more concussions in the 6-year National Football League study[a]

Signs and symptoms	Repeat concussions				
	First (n = 51)	Second (n = 51)	Third (n = 51)	Fourth (n = 15)	Fifth+ (n = 11)
General symptoms	60.8%	66.7%	64.7%	66.7%	63.6%
Headaches	56.9%	58.8%	58.8%	60.0%	54.5%
Nausea	9.8%	5.9%	5.9%	6.7%	
Vomiting		2.0%		6.7%	
Neck pain	7.8%	9.8%	15.7%	13.3%	18.2%
Back pain	0.0%				
Syncope	2.0%				
Seizures					
Somatic complaints	11.8%	15.7%	17.6%	26.7%	27.3%
Irritability	3.9%	2.0%	2.0%	0.0%	9.1%
Anxiety	3.9%	3.9%	5.9%	6.7%	
Depression					
Personality change	2.0%	2.0%	2.0%	13.3%	
Fatigue	2.0%	7.8%	7.8%	6.7%	18.2%
Sleep disturbance			2.0%		
Loss of libido					
Loss of appetite					
Cranial nerve effects	54.9%	58.8%	56.9%	53.3%	63.6%
Dizziness	41.2%	39.2%	41.2%	26.7%	45.5%
Vertigo	5.9%	2.0%	3.9%	6.7%	
Tinnitus	2.0%		5.9%		
Nystagmus			2.0%		
Hearing loss					
Diplopia	3.9%	2.0%		6.7%	
Photophobia	3.9%	9.8%	3.9%	6.7%	9.1%
Blurred vision[b]	27.5%	21.6%	11.8%	26.7%	27.3%
Pupil response				6.7%	
Pupil size					
Cognition problems	27.5%	17.6%	19.6%	20.0%	18.2%
Not oriented to person		3.9%	2.0%		
Not oriented to place	2.0%	7.8%		6.7%	
Not oriented to time	7.8%	9.8%	2.0%	6.7%	
Immediate recall	25.5%	15.7%	17.6%	13.3%	18.2%
Memory problems	39.2%	25.5%	23.5%	26.7%	45.5%
Attention problems	11.8%	9.8%	7.8%	6.7%	9.1%
Information processing	11.8%	7.8%	5.9%	13.3%	27.3%
AGA delayed	2.0%	3.9%			
RGA delayed	23.5%	13.7%	11.8%	13.3%	18.2%
Average no. of signs and symptoms	2.5	2.4	2.2	2.5	2.5
Range	0–8	0–12	0–12	0–7	1–5
Unconsciousness (>1 min)	0	0	0	1	0
All loss of consciousness	3	6	5	2	1
All reported as zero	17	26	15	3	4
Days out average	1.0	0.9	4.9	3.2	2.3
Standard deviation	2.3	2.2	15.6	5.9	4.6
Range	0–9	0–12	0–98	0–22	0–15
% days = 0 or 1	78.4%	78.4%	64.7%	60.0%	63.6%
% days = 7+	5.9%	2.0%	13.7%	13.3%	9.1%

[a] AGA, anterograde amnesia; RGA, retrograde amnesia.
[b] $P < 0.001$ for first versus third concussion by the McNemar test.

Previous concussion history may affect our conclusions regarding repeat concussions, because a certain number of the concussions that we labeled as initial concussions may in fact have been repeat concussions for some individual players. In this article, the "initial" MTBI does not necessarily mean "first MTBI." It means that the case represents the "initial" MTBI during the study period. The same applies to repeat injuries that occur during the study period.

The authors also realize that some MTBIs were not reported by the affected player to team medical personnel and therefore were not included in this database. Such unreported injuries most likely were very mild in nature and associated with rapid recovery to escape detection by very involved NFL athletic trainers and physicians. In addition, although 160 (24.6%) of 650 repeat concussions reflects the proportion of repeat injuries in this sample, the population of NFL players changes year to year: new players enter the league, older players leave the league, and we do not know what number of players who constitute the 1996 population are still in the league in subsequent years.

There was difficulty collecting data on loss of consciousness. The initial data collection sheet did not ask for data regarding loss of consciousness. Once this was corrected, we found that many of the reports that were submitted did not answer the question in the loss-of-consciousness part of the form; therefore, we do not have definitive loss-of-consciousness data on a certain number of players. What has been reported are the cases with a known time of unconsciousness and those cases that reported a zero or no loss of consciousness. Interestingly, there was a failure to find any relationship between loss of consciousness and neuropsychological function in one study, calling into question the assignment of primary importance to loss of consciousness in grading severity of concussion (22). The difference between the sum of these two numbers and the overall size of the group is the unknown or indeterminate cases.

It is also important to note that in a multisite study such as this, there are numerous different examiners. In some cases, different examiners from a given medical staff may evaluate that team's players. There was no uniform method of evaluation of concussion in this study, which will give rise to variability in assessments among the 30 teams and, on occasion, within the same team. It must be emphasized that players were not cleared to return to play until they were asymptomatic, with normal medical examinations, although some players may return with headaches.

As noted previously, many of the players in the database had neuropsychological testing at baseline and/or after MTBI. The results of these neuropsychological test batteries are not currently in this database and therefore not part of this report. It is possible that including the results of the neuropsychological testing on the patients may add information on the cognitive effects of repeat concussions. Return-to-play data were collected on players with initial and repeat concussions. Although the medical condition of the player certainly is the most important factor in determining return to play by team physicians, there are many other factors that go into the decision of when the player should return to play. The importance of the player to the team; the importance of the upcoming game to the team; and pressure from owners, players and their families, coaches, agents, and media certainly may influence the final decision of when the player returns to play. The authors believe, however, that the medical factors regarding the patient's recovery are and should be the overriding facts that guide the team physicians' decision-making on return to play. Furthermore, our results apply to NFL-level players, and extrapolation to younger athletes has not been demonstrated. It is clear that differences may exist between MTBI in high school and professional athletes.

Repeat Concussions

The present study found that 160 (24.6%) of 650 players who sustained an MTBI in the NFL went on to sustain one or more repeat MTBIs during a 6-year period. There were 490 players who sustained only one MTBI. The 160 players who had a repeat MTBI represent only a small percentage (5.0%) of the 3228 NFL players in regular-season games during the 6-year study. In addition, only 51 of the 160 players had a third MTBI, 15 had four MTBIs, 7 were reported with five MTBIs, 3 with six MTBIs , and 1 with seven MTBIs. Overall, there were 397 repeat injuries of 160 players in practices and all games during a period of 6 years. These results indicate that, despite public and media perceptions, repeat MTBI affects only a relatively small number of NFL players.

Nearly all of the NFL players had been evaluated before their entrance into the league at the NFL Combine, which is a predraft evaluation of 330 elite college athletes invited by the 32 NFL teams. The Combine is held every February in Indianapolis, and it is uncommon to see a prospective professional player who had two or more concussions during his previous high school and college experience. However, no effort was made to capture the MTBI history of players involved in this 6-year NFL study.

One of the most frequently quoted articles in the older literature is by Gerberich et al. (16). They used questionnaires regarding head injuries mailed to coaches and players representing 103 high school football teams. This retrospective study used no athletic trainer or physician reports and included no detailed reports of signs and symptoms. The authors found that players with a history of previous traumatic loss of consciousness were 4 times more likely than those without such a previous history to sustain a second MTBI with loss of consciousness. Delaney et al. (13, 14) published the results of two separate retrospective self-report questionnaires based on Canadian professional football players and university football and soccer players. They found that 69% of the professionals and more than 80% of the college athletes who sustained a concussion reported a second concussion as well. They indicated that a history of previous concussion increased the risk of sustaining another MTBI (13, 14). An attempt was made to determine whether there was an increased risk of

repeat concussion after an initial MTBI in the NFL, but that aspect was not covered in this study. Numerous statistical means of approaching the problem have been investigated, but exposure data were not available to determine relative risk. If we had used exposure data, we would have had to limit the analysis to game injuries during the regular 16-game season. This would miss the intervening MTBIs that occur in practice and complicate the calculation.

Powell and Barber-Foss (38) reported data on the occurrence of multiple MTBIs in high school athletes of both sexes competing in football, wresting, basketball, baseball, softball, field hockey, volleyball, and soccer. The data were collected prospectively by athletic trainers over a 3-year period. There were no reports of clinical signs and symptoms or return-to-play data. A total of 92 players with more than one MTBI were found, 65 of which occurred during football. Guskiewicz et al. (19) surveyed athletic trainers at high school and college programs over three seasons. There was a total number of 17,549 players in these programs. They reported that 131 repeat concussions occurred during the same season as the initial concussion (881 initial concussions). Not all players were examined by physicians, and there were no detailed reports of signs and symptoms. Only 69% of athletic trainers who were initially asked to participate ultimately completed the study (19). The very high incidence of repeat concussions in the above studies is not consistent with the results of other studies, including the present one, which found that 24.6% of NFL players (160 of 650 players) who sustained one MTBI later sustained repeat MTBIs during a 6-year period (an average annual incidence of 4.1%).

Some recent studies have used standardized concussion tests and computerized neuropsychological tests. There have been five reports of neuropsychological test results in athletes with multiple cerebral concussions (9, 10, 23, 26, 45). In these studies, the authors relied solely on the athletes' self-reports of previous concussion history to determine their inclusion in the study. The studies noted above did not report clinical signs and symptoms, nor did they report physician findings. Four of the studies found that athletes with a history of self-reported previous concussions performed worse on postconcussion neuropsychological testing than did their counterparts who had not reported a history of previous concussions. One study found no difference in the neuropsychological test results between the two groups (23). The report did not contain more detailed clinical information other than the results of these few neuropsychological tests.

One recent study analyzed the results of a general health questionnaire completed by 2488 retired NFL players (3). It reported that 24% of former players had sustained three or more concussions during their careers. The study was retrospective, relied completely on unverified self-reports, and did not involve physician evaluations. The results of Bailes' study (3) are in marked contrast to the findings of the present study that only 160 (5%) of 3228 NFL players in the league during the 6-year study sustained multiple MTBIs. Although the number of players entering and leaving the league complicates determination of a precise risk, this value gives a general estimate over a recent 6-year period. The marked variability of conclusions in the above-mentioned studies points to one of the many strengths of this present study, in that the concussion history was obtained from medical documentation of prospective events, not from retrospective self-reports, in which answers depend on how the questions are asked, the context of the questioning, and the selective memory of the athletes (30).

Guskiewicz et al. (18) reported a prospective National Collegiate Athletic Association concussion study of 2905 football players over three seasons, of whom 184 (6.3%) had a concussion and 12 had a repeat concussion in the same season. The data were collected from questionnaires filled out by athletic trainers. No reports from treating physicians were included in the results. They concluded that there may be an increased risk of repeat concussive injuries and there may be a slower recovery of neurological function after repeat concussions in those who have a history of previous concussions. The results of this present NFL study do not support those conclusions. There was concern that after a concussion, there may be a 7- to 10-day window of increased susceptibility to sustaining another concussion. Again, the results of this present NFL study do not support that conclusion. In fact, the present NFL study found that the average time interval between MTBIs was 1 year and that relatively few repeat events occurred within the first 10 days after injury. Also, although approximately one-half of players went back to play during the same game/session and approximately 90% returned within 1 week, recurrent injury caused by an increased vulnerability in the immediate postconcussion period does not seem to be a factor in our population.

Second-impact Syndrome

No cases of SIS were detected during the 6-year period of this study in the NFL. One hundred sixty players sustained a total of 397 repeat concussions, ranging from 2 to 7 per individual. There were no deaths, prolonged comas, or any evidence of diffuse cerebral edema in the patients. Furthermore, there have been no case reports of SIS in the history of the NFL, despite a relatively common occurrence of concussion and the likelihood that some players may fail to report symptoms of MTBI and thus play despite being symptomatic with MTBI. The anecdotal experience of team physicians is that, occasionally, the medical staff does not learn about a player's concussion until after a game.

There are a number of possible explanations for the absence of SIS in NFL players. Most obvious is the small sample size versus the expected incidence rate. The incidence of SIS in high school and college football is 1 to 2 per 1,500,000 players. Thus, one would need 375 to 750 years to expect to see a case of SIS, assuming 2000 players involved per year. In addition, patients who are reported to have had SIS are usually adolescents or young adults (5, 7, 29, 41). This implies that the brain of a pediatric patient or adolescent may respond to head

acceleration differently from that of an adult. Cerebral edema after brain injury in the pediatric population has been well documented (4). The overwhelming majority of players in the NFL are more than 22 years of age. Because SIS is thought to be a very rare occurrence, our sample size of 397 repeat concussions in 160 players may have been too small to detect such a rare event. A number of the case reports of SIS document autopsy findings showing cerebral contusions or small subdural hematomas that have been deemed "insignificant" (7, 41). Perhaps the phenomenon of SIS represents a complication of a focal brain injury more so than true concussion. Fortunately, focal brain injury in the NFL seems to be exceedingly rare. It is also possible that the results of the present study demonstrate that SIS does not truly exist in this population of players.

Concussion Management and Guidelines

Many of the currently promulgated guidelines for the management of sports-related concussion have established exclusion periods for multiple reasons, one of which is the prevention of SIS. This is based at least partially on the belief that everyone with a symptomatic MTBI is at risk for developing SIS (1). The absence of any cases of SIS in the NFL during this 6-year study period or in the history of the NFL suggests that such arbitrary return-to-play guidelines may be too conservative for professional football.

Another often-expressed concern of the authors of concussion management guidelines is the occurrence of chronic brain damage as a result of multiple head injuries (1). The chronic encephalopathy of boxers is a well-accepted and documented clinical and pathological syndrome (8, 31, 39, 40, 44). The clinical features include a combination of cerebellar, extrapyramidal, and pyramidal dysfunction, along with cognitive and personality changes. This is accompanied by a well-defined neuropathological picture. The full-blown clinical picture is present in approximately 17% of retired boxers (39), but partial or more subtle abnormalities are seen in a much higher percentage (8, 40). The present study was admittedly not the best vehicle to search for evidence of chronic encephalopathy in professional football players, but it is important to note that no signs or symptoms were found of significant extrapyramidal, cerebellar, or pyramidal dysfunction in NFL players with repeat concussions. This was not a surprising result, because chronic traumatic encephalopathy has been reported only in boxers and a few steeplechase jockeys. We are not aware of any cases of this syndrome in football players. Furthermore, numerous studies have shown that the occurrence of chronic encephalopathy of boxers is related to length of career and number of professional bouts, not to the number of knockouts (concussions) sustained (8, 31, 39, 40). It is well accepted that chronic encephalopathy of boxers results from the accumulation of damage from multiple subconcussive blows to the head over a prolonged period of time, not the number of concussions sustained (8, 31, 39, 40, 44).

There was no evidence of chronic encephalopathy in this group of football players who had sustained a relatively small number of multiple concussions, although, with years of practice and play from high school, college, and professional play, the length of participation in full-contact football is usually more than 10 years in most professional athletes. The authors realize that there have been a small number of NFL players who have retired with prolonged or permanent postconcussion syndrome. These players did not have evidence of pyramidal, extrapyramidal, or cerebellar dysfunction. They did not have clinical dementia. They clearly did not have chronic encephalopathy such as that seen in boxers.

Our questions regarding management (*Table 5*) were broad in nature and did not allow us to carefully determine specific management strategies of the treating physician. It is still noteworthy that only approximately 5% of players with a first concussion or multiple concussions received therapeutic modalities, drug treatments, or medical procedures. It is possible that managing physicians will provide more definite management in the future because of the ever-expanding knowledge on this subject and the availability of more sophisticated testing modalities.

In analyzing these data, an attempt was made to determine whether there were any features of a player's initial concussion that might indicate an increased risk of his going on to sustain multiple MTBIs. There were no differences in management, including time interval until return to play, between those who did not have another MTBI and those who did. Regarding clinical signs and symptoms, there were two statistically significant differences between the groups. Diplopia was reported more often in players who later went on to sustain a repeat MTBI. Personality change was reported less often in players who later went on to sustain a repeat MTBI. It is uncertain how valuable these differences will be to physicians who treat athletes with head injuries. Diplopia, for example, was a very infrequent symptom in both groups and is a well-known but infrequent occurrence after MTBI (20, 34). In this setting, it is often related to IVth or VIth cranial nerve dysfunction, but it may also be an indicator of transient brainstem or ocular muscle dysfunction. This could suggest increased severity of injury and thus help explain an increased risk of repeat MTBI. Diplopia was reported in 1% of the 490 players who had sustained only one concussion (5 players) and 5% of the 160 players who subsequently sustained a repeat MTBI (5 players). Of the 10 players who complained of diplopia after an initial MTBI, 5 (50%) subsequently sustained a repeat MTBI.

These results raise the remote possibility that players with diplopia after an MTBI may be at increased risk of sustaining a subsequent MTBI, but the small number of players involved suggests caution in accepting this finding. More likely, there was another factor involved. Nine of the 18 cases with diplopia occurred in the 1997 season in players from seven different teams (1996, 2 cases; 1998, 1 case; 1999, 2 cases; 2000, 3 cases; 2001, 1 case). Because the cases are not evenly distributed in time, there may have been some increased sensitization to diplopia on the part of some physicians in 1997. Also, two players accounted for four cases. In addition, it is difficult to

understand why players with personality change were less likely to sustain a repeat MTBI. In view of the small number of players involved, this association is probably clinically insignificant. In considering these findings, it may be more important for clinicians to note that none of the other signs and symptoms evaluated in this study occurred at any significantly different frequency between the two groups.

Neuropsychological Effects

There have been previous studies using neuropsychological testing that demonstrate subtle cognitive, attention, and memory impairments that are more prominent in players with multiple concussions than in those with only one concussion (9, 10, 24, 26, 45). Although many of the players in our study did undergo neuropsychological testing, these results are not currently part of this study. However, the purely clinical examinations did not find cognitive and memory impairments with greater frequency in players who experienced repeat versus single MTBIs (*Table 3*). In fact, the data show that cognitive and memory problems were present almost equally in players with more than three concussions compared with their first and second concussions in the study period.

There may be a "practice effect" occurring. This is a well-known phenomenon in neuropsychological testing whereby subjects perform better on subsequent administrations of the same tests because of learned responses rather than because of any true improvement in their abilities. However, it has also been reported that athletes with multiple concussions differ from those with only one concussion specifically by not demonstrating the practice effect on follow-up neuropsychological testing (45). Another possible explanation could be that treating physicians did not aggressively test for cognitive and memory impairments after the third concussion because they thought it would be present as it had been in previous concussions. If the examiner does not specifically pursue cognitive and memory questioning, these signs will often be overlooked. It may be that if the results of neuropsychological testing were available on all of the players in our study, then we would be able to confirm the findings of earlier studies that players with multiple concussions perform worse on this type of testing than their counterparts.

Three or More Concussions

Another part of the data analysis evaluated players who sustained three or more concussions. *Table 6* shows the signs and symptoms of the earlier and later concussions of these 51 players. In this analysis, the player serves as his own control for the history of successive concussions. With the fourth or later concussion, there was a higher incidence of personality change and fatigue, but the difference did not reach statistical significance. The average numbers of signs and symptoms were somewhat lower in the group with four or more MTBIs, but again the difference did not reach statistical significance. The incidence of loss of consciousness at the time of MTBI was not different with successive concussions. The time intervals to return to play were not significantly different between the two groups, although they were longer with repeat concussion (*Fig. 1*). What is most striking to the authors is the marked similarity of the signs and symptoms of each of the successive MTBIs for these players, although there was a trend to more days out based on the average and percentage of players out 7 days or longer with three or more concussions.

Overall, the signs and symptoms reported in our study were very similar between players with single and multiple MTBIs (*Tables 3* and *6*). The total number of symptoms per case was essentially the same, except for a slight decrease in those with three or more concussions. There was a slightly increased frequency of anxiety in the second MTBI compared with the initial event. This may reflect the athlete's appropriate concerns regarding the effect of repeat injury on his health and career, rather than being an indicator of cerebral damage. Although there is a trend toward repeat injuries having more time missed, the symptoms and signs of repeat injuries are so similar to those of single injuries that the longer return to play for repeat injuries may be a result of more conservative care by the medical staff rather than more severe injury.

It is possible that more subtle abnormalities in personality or behavior occurred after multiple MTBIs but that these were too vague and nonspecific to be adequately recorded on the standardized forms. Certainly, experienced athletic trainers and team physicians often report that a post-MTBI athlete "does not seem right," even though by all accounts (clinical examination, history, neuropsychological testing) he has returned to normal (R Barnes, personal communication, 2003). It is also the perception of some team physicians that less biomechanical force is needed to cause the fourth to seventh concussions in a small percentage of these players. This seems to be supported by the game video, which shows lower collision velocities and less severe helmet impacts. However, a thorough analysis of this possible effect has not been made. It would be interesting to study the susceptibility to injury after many concussions. Obviously, the time between injuries and the cumulative effects are additional factors to consider.

Incidence of Repeat Concussion by Player Position

The present study found that ball return carriers, quarterbacks, tight ends, and linebackers had a higher incidence of repeat MTBIs than their counterparts (*Table 2*). The higher incidence was statistically significant only for quarterbacks, even though kick return ball carriers on special teams had the highest OR of repeat MTBI. Both of these positions are often subjected to high acceleration from open-field or blindside head impacts at high velocity. The positions at increased risk of multiple MTBIs are the same ones that were found to have increased risk of sustaining any MTBI in an earlier study in this series (34). In recent years, the NFL has implemented and reinforced several rule changes aimed at protecting players from head injury. In addition, helmet manufacturers have developed newer protective equipment that is now in use. Future analysis of ongoing MTBI data may determine what, if

any, effect these initiatives will have on the incidence of repeat MTBIs in the years to come.

CONCLUSION

This study presents an analysis of MTBI in professional football with a focus on repeat injuries. Clinical data were collected prospectively from treating physicians and athletic trainers during a 6-year period. The results indicate that repeat MTBIs were very similar to first MTBIs. The overall clinical symptoms and signs were similar in both groups of players. No cases of SIS and no evidence of chronic encephalopathy were detected in the clinical evaluations. Players with multiple MTBIs returned to play within intervals similar to those with a single MTBI, and there were no differences in the management of players with multiple MTBIs. Players with multiple MTBIs did not sustain loss of consciousness at a frequency any different from those with single concussions. There was no evidence of increased severity of injury in multiple versus single MTBI cases. The data also indicate that repeat MTBI occurred relatively infrequently during this 6-year period.

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Acknowledgments

The NFL's Committee on MTBI is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society, NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the area of traumatic brain injury, biomechanics, basic science research, and epidemiology. The authors of this article are members of the committee. The efforts of other committee members are gratefully acknowledged, including Douglas Robertson, M.D., Mark Lovell, Ph.D., A.B.P.N., Ronnie Barnes, A.T.C., and Jay Brunetti. None of the committee members have a financial or business relationship posing a conflict of interest to the research conducted on concussion in professional football.

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We also appreciate the contributions of all the NFL team physicians and athletic trainers who filled out the MTBI report forms and those of the players who consented to participate in the study through a blinded identification in the MTBI database. We thank the staff at Med Sports Systems for their efforts in managing the data flow among the various aspects of the project. Without their support, the project could not have been completed.

After publication of Part 3 of the NFL MTBI series (34), the committee became aware of two players with the same six-digit identification number. The last six digits of a player's Social Security number were used and were thought to be a unique identifier. This increased the number of players with concussion by one and slightly affected the statistics. The data reported in this article reflect the accurate count of players who experienced concussion during the 6-year collection of data.

The committee extends its appreciation to Cynthia Arfken, Ph.D., at Wayne State University, who was our epidemiological consultant for statistical analysis of the concussion data. Her insights and involvement were instrumental to the interpretation of the data.

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COMMENTS

Pellman et al. have provided us with another analysis of the data derived from the National Football League (NFL) Mild Traumatic Brain Injury Committee study. In this report, they have studied 160 repeat mild traumatic brain injuries (MTBIs) out of an initial group of 887 concussions. These were reported in practices and games for 650 players during the 1996–2001 football seasons. Their findings corroborated previous studies showing that players involved with high-speed and often unsuspected collisions sustained concussions and that repeat concussions occurred at a higher incidence in the defensive secondary, special team tacklers, ball return specialists, quarterbacks, tight ends, and linebackers. The clinical expression of repeat concussion was similar to that of players with an initial or single MTBI except for a higher incidence of somatic complaints. There were no findings indicating a difference in the number of signs or symptoms between the initial and repeat concussions. The vast majority (92%) of the NFL players with MTBI returned to play within 1 week, and the average duration between concussions was slightly greater than 1 year. They found that 160 of 650 of their players (24.6%) sustained one or more repeat concussions during the 6-year period. This study used team physicians to complete an evaluation form on those players identified as having sustained an MTBI.

The limitations of this study include the fact that neuropsychological evaluations were not included in the evaluation of the patient or in reaching their management strategies or conclusions. It has been well documented that the clinical examination or reported neurological symptoms or signs are not as sensitive as neuropsychological testing in a concussed athlete. The latter has been shown to be sensitive to the presence of significant cognitive and mental processing deficits, even in a reportedly asymptomatic football player with a normal neurological examination. Also, the players' concussion histories were not known. The fact that they report a 5% concussion rate does not indicate a true ongoing incidence, because there is an annual turnover rate in team personnel. The assessment of long-term consequences is likewise limited in this brief follow-up in players who continue to be actively used by the NFL. For several reasons, there may be disincentives to report mild cognitive disturbance or symptoms, or it may be too early for a chronic syndrome to have developed. I would urge caution in extrapolation of these data to include a management scheme with rapid return of players at other levels, as was their practice, in which approximately 60% returned to play during the same game after sustaining MTBI. In regard to the lack of second-impact syndrome (SIS) in NFL players, this probably reflects the fact that it is primarily a phenomenon of younger contact athletes and a rare occurrence.

Julian E. Bailes
Morgantown, West Virginia

Pellman et al., working through the NFL Committee on MTBI, with this article have made another significant contribution to the medical literature dealing with repeat concussions in athletes. Their data clearly show that special teams, ball return athletes, and quarterbacks are the players most vulnerable to repeat concussion, and single and repeat athletes with concussion in more than 90% of the cases have a good recovery and quick return to play.

Several other important observations are made. Under prospective physician-reported circumstances, the actual incidence of concussions in the NFL is surprisingly low: approximately 3% per year. Furthermore, although repeat concussions occurred to approximately one-quarter of the players who sustained an initial concussion, the interval for this second concussion was approximately 1 year.

The authors also emphasize that there were no cases of SIS during the 6-year period of the study. Indeed, there has never

been a case report of SIS in an NFL football player, although this syndrome must be considered in any athlete with a cerebral concussion. The fact that it is exceedingly rare, indeed unreported, in the NFL also needs to be considered in the judgment-making process.

The apprehension of chronic encephalopathy developing in football players from multiple concussions is also somewhat tempered by the fact that in the present study, there were no athletes with documented extrapyramidal, cerebellar pyramidal, or pyramidal dysfunction in players with repeat concussions.

Neuropsychological testing is now considered the most sensitive means of assessing cognitive and memory function after MTBI. This form of testing is now available and is used by all NFL teams in patients with cerebral concussion. This heightened awareness clearly is preventive in reducing serious sequelae from returning to play too soon. Using these tests, we have discussed definite personality changes and cognitive impairment in athletes with multiple concussions, but in most of them, almost all symptoms and signs clear given an adequate time with contact participation.

Although the authors well document concussions in the NFL, a note of caution must be made in extrapolating these data to college and high school athletes. We have found, for example, that high school athletes with three or more concussions are nine times more likely to have a repeat concussion and more significant abnormalities in the on-field presentation compared with those athletes with no history of previous concussion. This supports the increased vulnerability of the brain to repeat concussive blows. In addition, we have found that high school athletes take longer to recover, as determined by neurocognitive testing, than college athletes, which suggests that the "younger" brain may be even more vulnerable to concussive brain injuries (1).

Finally, the relatively low incidence of concussion in the NFL may indicate a selective bias in that those athletes who make it to that level are more resistant to concussive injuries and those who are not have already been "weeded out" because of previous injuries. Overall, the authors have made another significant contribution to the medical literature on athletic concussive injuries.

Joseph C. Maroon
Pittsburgh, Pennsylvania

1. Field M, Collins MW, Lovell MR, Maroon JC: Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. **J Pediatrics** 12:546–554, 2003.

At first glance, the NFL experience with single and repeat concussion (no difference) and management (more than 50% of players return to the same game, including more than 25% of those with loss of consciousness) seems to be at odds with virtually all published guidelines and consensus statements on managing concussion.

In truth, all those guidelines and consensus statements were generated from data on high school and college athletes. Those in the NFL are not only the most skilled football players but also probably the best physically and genetically equipped to withstand the trauma of football, or they never would have been accepted on an NFL team. Thus, these authors may be studying unique individuals.

Other explanations can be found by reading and reflecting carefully on the Limitations section of this article, which is found at the beginning of the Discussion section. Because the athletes' previous concussion histories were not obtained, we really do not know how many concussions these athletes received, and therefore, comparisons between first, second, and third concussions in this study do not necessarily reflect the athlete's first, second, or third concussion. It can be argued that this makes all comparisons suspect.

Another concern is this study, understandably, is a 6-year window during which there was turnover of players; thus, comments about not seeing SIS or traumatic encephalopathy could reflect the limited period of exposure.

All of the above being said, I find that this article comes at a most interesting time. In the past several years, multiple articles have been published that place the incidence of concussion, based on direct questioning of the athlete, to be 40 to 70% annually, not less than 10% as routinely reported by trainers (*Table C1*) (2–4; Woronzoff, personal communication, 2001). Thus, the incidence of concussion is clearly much higher than that seen by the medical team on the sidelines, with most athletes with minor concussions continuing to play. The fact that the athletes questioned did not seem to have problems, as evidenced by their behavior, suggests that further reflection on the management of minor concussions might be appropriate. This article also supports the concept that concussion severity should be determined not on the day of injury but rather only after concussion symptoms have cleared (1). I am sure these findings will be deliberated at the second international concussion conference in Prague in November 2004.

TABLE C1. Incidence: player survey data

Series (ref. no.)	Level	Incidence
Langburt et al., 2001 (4)	High school	47.2%
Delaney et al., 2002 (3)	College	70.2%
Delaney et al., 2000 (2)	Canadian Football League	47.8%
Woronzoff, 2001 (personal communication)	College	61.2%

Robert C. Cantu
Concord, Massachusetts

1. Cantu RC: Concussion severity should not be determined until all post concussion symptoms have abated. **Lancet** 3:437–438, 2004.

2. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM: Concussions during the 1997 Canadian Football League season. **Clin J Sport Med** 10:9–14, 2000.
3. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM: Concussions among university football and soccer players. **Clin J Sport Med** 12:331–338, 2002.
4. Langburt W, Akhthar N, O'Neill K, Lee JC: Incidence of concussion in high school football players of Ohio and Pennsylvania. **J Child Neurol** 16: 83–85, 2001.

The authors have presented a 6-year surveillance program that prospectively collects data describing the signs, symptoms, and outcome of repeat concussion in the American NFL. This report addresses an important issue in contact sport that is not restricted to American football.

The prospective collection of data is particularly useful, but it is important to note that the definition of MTBI used in this study is very broad and perhaps overinclusive of minor nonspecific clinical features. Nevertheless, the authors note that the definition put forward by the MTBI Committee of the NFL is an extension of the definition proposed by the ad hoc committee established by the Congress of Neurological Surgeons in 1966 and is consistent with the definition proposed by the American Congress of Rehabilitation Medicine. A more strict definition commonly used by sports authorities in other countries describes concussion as a period of definite alteration in consciousness, with the severity being defined as the length of posttraumatic amnesia.

It was of interest that only a relatively small percentage of players in the NFL were reported to have sustained an MTBI, although 25% of those having an MTBI had repeat concussion, with a rate of 26.6 per year. It is difficult to extrapolate these data for other contact sports, but the other codes could well take a lesson from the MTBI Committee and commence prospective data collection.

A major concern has been whether multiple minor brain injuries could lead to chronic brain damage characterized by the chronic encephalopathy of boxers. It is well accepted that the chronic encephalopathy of boxers does result from the accumulation of damage from the multiple subconcussive blows to the head over a period of time, and there have been studies using neuropsychological testing that demonstrated subtle cognitive and memory impairment in other sportsmen who have sustained multiple concussions. Although the neuropsychological assessment was not part of this present study, the authors should be encouraged to continue with their prospective collection of data and include the neuropsychological evaluation in future reports.

A further contentious issue has been the timing of return to play after a concussive injury. A particular concern has been that too early a return to the sporting field renders the player more vulnerable to a second concussive episode, with the possible sequelae as mentioned, and also specifically the possibility of SIS. This has been described after a second impact to the brain that is still symptomatic from the initial head injury and is characterized by rapid clinical deterioration because of cerebral hyperemia and an increase in intracranial pressure, with a high rate of mortality. I have personally never seen such a case, despite the relatively common occurrence of mild head injury and concussion in Australian Rules football an my association with junior football over a period of man years.

The authors report a very rapid return to the sporting fiel after an MTBI, but the usual management protocol in Austra lian sport, and specifically for Australian Rules football, dic tates that a player should generally not resume full contac sport for at least 1 week after the injury, and then only if he o she is completely asymptomatic. The difference may be in th definition of "concussion."

Andrew H. Kaye
Melbourne, Australi

I want to preface my comments by saying that I have enjoyed reading the biomechanics studies from last year in *Neuro surgery* by members of the NFL's MTBI Committee. This group undertook a series of important research projects aimed at gaining a better understanding of impact biomechanics associated with concussive injuries in the NFL. These articles have made significant contributions to the literature on the biomechanics of concussion in professional football, which has laid the foundation for several new studies aimed at studying impact biomechanics in football.

Unfortunately, the present article (Part 4) in this series o studies on professional football players has several flaws with respect to the study design, data collection, and data analyses Physicians and certified athletic trainers managing athletes with concussion should be aware of the limitations of the study, most of which are outlined by the authors, and be aware that these limitations probably could have affected the findings. Thus, the interpretation of the findings and conclusions should be viewed with caution. There is a potentially dangerous message sent through the authors' conclusion: that single and repeat concussions are managed conservatively and most players return quickly to play.

The more important and more meaningful variables for clinicians and researchers are symptom duration and symptom severity. However, the authors collected data on how many days physicians withheld players from competition ("days lost"). They state in the article, "It must be emphasized that players were not cleared to return to play until they were asymptomatic, with normal medical examinations, although some players may return with headaches." My first concern is that a headache is a postconcussive symptom, which means that players in the NFL are cleared in some cases before they are asymptomatic. The article therefore sends a message that it is acceptable to return players while still symptomatic, which contradicts the literature published over the past 20 years suggesting that athletes be returned to play only after they are asymptomatic, and in some cases for 7 days.

Second, the analysis on days lost has a potentially ambigu ous meaning, because although the authors provide a defini tion of days lost, the reader in reality has no idea as to wha days lost refers to. Without knowing whether the team phy sician returned the player immediately upon becomin

TAB 1E

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CONCUSSION IN PROFESSIONAL FOOTBALL: INJURIES INVOLVING 7 OR MORE DAYS OUT—PART 5

Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, and ProHEALTH Care Associates LLP, Lake Success, New York

David C. Viano, Dr. med., Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park; Department of Neurology, New York University Medical Center, New York; and Department of Neurology, Albert Einstein College of Medicine, Bronx, New York

Cynthia Arfken, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Department of Psychiatry and Behavioral Neurosciences, Center for Health Effectiveness Research, Wayne State University, Detroit, Michigan

John Powell, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University, Lansing, Michigan

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952. Email: dviano@comcast.net

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OBJECTIVE: A 6-year study was conducted to determine the signs, symptoms, and outcome of concussions with 7 or more (7+) days out from play or extended postconcussion recovery in the National Football League (NFL).

METHODS: From 1996 to 2001, reporting of concussion was performed by NFL teams using a special standardized reporting form filled out by team physicians. Signs and symptoms were grouped by general symptoms, somatic complaints, cranial nerve effects, cognition problems, memory problems, and unconsciousness. Medical action taken and management were recorded. In all, 887 concussions were reported in practices and games.

RESULTS: There were 72 concussions (8.1%) involving 7+ days out from play. The highest frequency occurred in quarterbacks (14.8%), the return unit on special teams (11.8%), and secondary (10.8%). Quarterbacks had the highest odds ratio (OR) of 7+ days out with concussion (OR = 2.10, $P = 0.049$), whereas running backs had the lowest relative risk (OR = 0.13, $P = 0.021$). The greatest fraction of 7+ days out occurred in passing plays (36.1%) and kickoffs (22.2%). Many signs and symptoms occurred at a greater frequency on initial examination in players 7+ days out; the average number per player was 4.64 with 7+ days out versus 2.58 with fewer days out ($t = 6.02$, $df = 77.1$). The signs and symptoms with the highest incidence for 7+ days out were disorientation to time ($\chi^2 = 51.2$, $P = 001$), retrograde amnesia ($\chi^2 = 33.2$, $P = 0.001$), fatigue ($\chi^2 = 28.1$, $P = 0.001$), and the general category of cognition problems ($\chi^2 = 21.7$, $P = 0.001$). Loss of consciousness for more than 1 minute was a predictor of 7+ days out ($\chi^2 = 33.5$, $P = 0.001$), although it occurred in only 7.9% of cases. Of players with 7+ days out, 72.2% were removed from the game and 12.5% were hospitalized. These frequencies were significantly greater than for players with fewer than 7 days out ($\chi^2 = 68.03$, $df = 3$, $P < 0.0001$). Approximately 90% of players were managed by rest, irrespective of days out, but a greater fraction were given drug or medical therapies with prolonged days out.

CONCLUSION: The most vulnerable players for 7+ days out with concussion were quarterbacks and the secondary in professional football. Although 8.1% of concussions involved 7+ days out, only 1.6% involved a prolonged postconcussion syndrome. They recovered from symptoms and had a consistent return to play in the NFL.

KEY WORDS: Concussion, Concussion guidelines, Epidemiology, Injury surveillance, Postconcussion syndrome, Sport injury, Traumatic brain injury

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The great majority of athletes who sustain concussion (mild traumatic brain injury [MTBI]) make a relatively quick recovery and return to play in a short period of time. However, a relatively small number of athletes develop persistent postconcussion symptoms and are unable to return to play for an extended period. Often, the persistent postconcussion symptoms are seen 1 or more weeks after the injury. The postconcussion syndrome follows head injury that is usually mild and consists of any combination of symptoms and signs that occur after MTBI (19; see also the Operational Definitions section of this article).

Although physicians have known of postconcussion symptoms for hundreds of years, the term "postconcussion syndrome" was first coined in 1934 to describe the "subjective posttraumatic syndrome ... due directly to the blow on the head" (45). Some authors have divided the postconcus-

sion syndrome into an early and a late postconcussion syndrome when it persists for more than 6 months (7, 19). However, it is not exactly clear at what point in time the symptoms that characterize cerebral concussion (MTBI) come to be those of postconcussion syndrome. In other words, when does cerebral concussion end and postconcussion syndrome begin? Very few data are available on the evolution from head injury to postconcussion syndrome in athletes. Detailed analysis of the cohort of athletes who are out 7 or more (7+) days after MTBI can provide such data and help shed light on the recovery from concussion in professional football players.

Over the years, there have been numerous attempts to determine the severity of concussion in athletes. These have generally appeared in the form of guidelines for the evaluation and management of MTBI (1, 4, 9, 11, 12, 31, 40). With the exception of a recent revision of Cantu's earlier work (5), the symptoms for the grading of concussion severity have relied on events immediately surrounding the head impact to determine or predict the severity of injury. Their focus has been on the presence or absence of loss of consciousness after impact, the presence or absence of posttraumatic amnesia, and the presence or absence of symptoms such as confusion, dizziness, and headaches. These observations are made within a few minutes of concussion to determine the grade or severity of the injury. The MTBI Committee and many physicians who treat athletes with concussion have been uncertain of these grading systems because they often do not seem to correlate with the patients' clinical course after concussion in National Football League (NFL) players.

Clinicians, including the present authors, frequently treat athletes with a prolonged and/or severe postconcussion syndrome who by the grading criteria had only very mild concussions and conversely, often treat patients who make a complete, rapid recovery after experiencing what was "graded" as a severe concussion. This raises questions about the sensitivity and specificity of the grading criteria.

The purpose of this article is to compare the small group of NFL players who do not return to play for 7+ days after an MTBI with the majority of NFL players who do return within 7 days. By analyzing this cohort with 7+ days out, the authors hope to determine the demographic and clinical differences between the two groups that might account for the extended period of missed playing time. Such data may shed light on possible prognostic factors that can aid treating physicians in the early evaluation of athletes who sustain concussion. These data may also be useful in the refinement of existing concussion grading and management criteria.

The 7-day dividing line between the groups does not reflect an arbitrary distinction. Because NFL teams play games only once a week, the players in this study cohort all missed at least one game. NFL teams play only 16 games per season. Therefore, missing one game implies a significant loss of playing time. Therefore, the study cohort all had significant functional impairment secondary to MTBI, although the recovery from many of the signs and symptoms may have occurred a few days after concussion.

BACKGROUND

In 1994, the NFL formed the Committee on MTBI in response to safety concerns regarding head injuries. Background on the committee has been provided by Pellman (35). Its mission was to investigate MTBI in the NFL by various scientific methods.

The MTBI Committee undertook a series of research projects aimed at defining concussion biomechanics in professional football. On the basis of analysis of game video and laboratory reconstruction of severe impacts using instrumented test dummies, the biomechanics of concussion has been determined for professional players. This has included data on the head acceleration of injury (38) and the location and direction of impacts (39).

The committee also determined a strong need to monitor the frequency of MTBI in the NFL and at the same time to identify the clinical signs and symptoms associated with concussion. The initial study focused on the characteristics of 787 concussions during regular-season NFL games, its signs and symptoms, and the relative risk by player position (36). A more recent study by Pellman et al. (37) addressed 887 concussions occurring over a period of 6 years, from 1996 to 2001, in practice and in all games. It evaluated repeat concussions in professional football, including the clinical signs and symptoms with repeat and multiple (three or more) concussions. The second impact syndrome was not found in this population.

PATIENTS AND METHODS

The MTBI Committee devised a simple form for team physicians to complete on observed and reported signs and symptoms on initial and follow-up examinations whenever they evaluated a player who sustained concussion. At the NFL level, there is close cooperation between team physicians and athletic trainers on player medical issues, and they worked together to collect cases and data for this study. All players were examined by team physicians, and all management decisions were made by physicians. During the study period, two teams were added to the NFL. This registry of concussions involved MTBI data from 30 teams in the NFL. The median number of concussions reported by the teams was 26 (range, 6–72) during the study period. Player's names were not included on the forms to maintain confidentiality. They were identified by a six-digit number.

Operational Definitions

The definition introduced by the committee in 1996 and used for the study is as follows. A reportable MTBI is a traumatically induced alteration in brain function, which is manifested by 1) alteration of awareness or consciousness, including but not limited to being dinged, dazed, stunned, woozy, foggy, amnesic, or, less commonly, rendered unconsciousness or, even more rarely, experiencing seizure; and 2) signs and symptoms commonly associated with postconcussion symptoms, including persistent headaches, vertigo, lightheadedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbance, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities. The definition is a natural extension of a much earlier one from the Ad Hoc Committee to Study Head

Injury Nomenclature of the Congress of Neurological Surgeons (13) and is consistent with a more recent definition by the American Congress of Rehabilitation Medicine (2).

Alteration of Awareness

There is some difficulty in eliciting a history of loss of consciousness or a transient alteration in awareness in professional football players. MTBIs may be very short-lived and thus not witnessed by the athletic trainer or medical staff. In addition, the player may not want to admit that such an event occurred because of a concern that he may lose playing time. Accordingly, any player who met any of the above criteria as determined by the athletic trainer or medical staff was included as an MTBI. Despite this, it is possible that a player could have sustained an MTBI but not have been included because of a lack of cooperation or a very transient and unrecognized episode.

Signs and Symptoms

MTBI is a clinical syndrome that may present with a broad spectrum of signs and symptoms, many of which are nonspecific and can be associated with other clinical diagnoses. The MTBI Committee members who are team physicians in the NFL, as well as MTBI Committee consultants with special expertise in the fields of sport neuropsychology and sport neurology, developed a list of the most common signs and symptoms with concussion. They were grouped into six categories: 1) general symptoms, 2) somatic complaints, 3) cranial nerve findings, 4) cognitive abnormalities, 5) memory problems, and 6) unconsciousness. The checklist was filled out for each player with a concussion.

A purposely large and inclusive list was selected to capture all of the possible clinical signs and symptoms with MTBI in professional football players. The signs and symptoms that were recorded are consistent with previous medical literature on the postconcussion syndrome and the symptoms and signs seen after traumatic brain injury. Most of the items are symptoms the player may complain of or that the physician may elicit by history. However, some items are mental status findings (retrograde amnesia, anterograde amnesia, and problems with information processing, attention, and immediate recall). The committee did not distribute uniform testing instruments to the team physicians but rather left the assessment of these parameters to the discretion of the individual team physicians. The rationale for the various signs and symptoms can be found in Pellman et al. (36).

The form contained questions about physical examination findings, initial management, tests ordered, and disposition regarding return to play. A form was generated for each player's MTBI, including the initial evaluation and all subsequent follow-up visits until the player was cleared for return to play. The individual team physicians were to complete the initial and follow-up forms on the basis of their clinical findings. In the final analysis, the signs and symptoms were designed to be interpreted by the physicians in the context of the case. In a few cases, the initial examination form may have been completed the next day, which would have allowed a sleep disturbance finding on the form. Team physicians and their consultants used their own evaluation procedures to manage the injury. The committee did not impose outside medical decision-making on the medical staffs of the individual teams. The individual team physicians were to complete the initial and follow-up forms on the basis of their clinical findings.

Return to Play

The following definitions apply to the return-to-play aspect of the medical report. NFL team physicians clear a player for return to play only after he becomes asymptomatic (with the exception of a mild headache) and has a normal neurological examination.

Return immediately: The player returns after an evaluation by the team physician demonstrates that the player is asymptomatic. The key here is that the player, because of his relative position on the team, may not be called to action for several minutes. For example, if the player was on the kickoff team and sustained an MTBI, the physician would perform an evaluation and determine that the player is ready to return, yet, depending on the game, it may be minutes or possibly an hour or so before he actually gets back on the field.

Rest and return: The player is evaluated, and it is determined that there should be some protracted time before a decision is made to return. The key would be that the player did eventually return to the same game or practice. An example might be that the injury occurs in the last 5 minutes of the second quarter. Because it is close to halftime, the decision to return is not made until the third quarter.

Removed from play: This means that the player was not allowed to return to the game or session in which he was injured.

Hospitalized: The player was admitted to the hospital, generally characterized as more than 18 hours. This would mean that going to a local hospital for an x-ray, head computed tomographic scan, etc., and then going home would not be classified as "hospitalized."

Days Out

The definition of days out is the time between the date of injury and the date that the player was permitted to return to full and unlimited participation (41). Full and unlimited participation means that the player must be able to perform all the activities of the session at the same intensity as his teammates. If a player were in a practice session and were not allowed to participate in contact drills, he would not be considered to be returned to participation, because he was not able to perform all of the activities expected of his teammates. In essence, this tells us that on the date of return, the player was expected to participate fully in all of the activities that were planned for the team practice or game.

Efforts to Improve Compliance

The Commissioner of the NFL encouraged all team physicians to complete and return forms whenever they examined a player with a head injury. The project was designed to record information about the injury. The forms were designed for ease of completion, and the data were limited to those points that would provide the most relevant information on MTBI to improve compliance. The data forms were sent to the NFL epidemiologist and entered into a database with a blinded coding to maintain anonymity of the players. When an initial evaluation form was submitted but the

follow-up visit form was not, committee members contacted team athletic trainers and doctors directly to remind them to submit the follow-up forms. During biannual meetings, the committee monitored the data and discussed findings.

Quality Assurances

The MTBI evaluation forms were logged in by the committee's epidemiologist and scanned into a database by use of a commercial software program (Teleforms, Cardiff, CA). During the data logging, the individual forms were reviewed manually. Each form was then scanned into a temporary database and verified before being entered into the final database. Any fields that were incomplete or inconsistent triggered a follow-up contact with the team athletic trainer or physician to verify the data. The final database includes information from the initial and follow-up evaluation forms submitted by team physicians.

Statistics

Descriptive statistics were used to characterize those players who were out fewer than 7 (<7) days and those who were out 7+ days from play during the 6 years of surveillance. χ^2 and t tests were used to compare the signs and symptoms, medical action, management, and time loss to the team of those players who were out <7 days and those who were out 7+ days from play. Combinations and presence of signs and symptoms that might predict 7+ days out were explored in multiple ways. The final screening was derived from a multiple logistic regression model with forward conditional selection using variables that were at least twice as common in those 7+ days out than in those with <7 days out. Cantu's (5) suggestion of 1 minute or more of unconsciousness was used to categorize loss of consciousness for statistical analysis, because of many unknown, missing, or indeterminate cases. Data are presented on all known cases of loss of consciousness, those reported as zero (or no loss of consciousness), and the unknown cases.

The median duration between the first concussion and concussion in those with 7+ days out was more than 1 year. The longest interval between the first concussion and concussion in those with 7+ days out was 4.6 years, but seven players experienced the second injury during the same week.

Signs and Symptoms from Initial and Follow-up Examinations

The physicians were asked to record at least one follow-up per case at the time of return to play. If a player did not return on the same day, they were to complete a follow-up each time they performed an evaluation until the player was cleared to return to play. The follow-up examination dates and times were recorded on the individual forms. All cases with 7+ days out were searched for follow-up medical forms, and each was coded by the order of occurrence using the date and time recorded by the physicians. Using the onset time of the case, the initial examination date and time and each follow-up date and time were calculated, along with the time between the initial examination and the first follow-up, then the second, and so on. The median and percentiles were determined. This information showed the change in signs and symptoms during recovery.

Video of 182 NFL Game Impacts

Video of severe and concussive impacts in NFL games was evaluated for impact types and locations by Pellman et al. (38, 39). In a further analysis, it was possible to use the date, team, and player identifications to match cases with concussions in the MTBI database. In all, 89 matches (49%) were made with verified concussions and medical evaluations. There were 18 concussions with 7+ days out caught on game video, typically with multiple views of the impact and with clear visual evidence of the injury; one case was obscured from view. This allowed an analysis of the type of plays and collisions associated with lengthy stays out from play. The general characteristics of the impacts was observed and compared with other concussion blows involving fewer days out. This included the type of play, tackle, and speed of impact.

RESULTS

Table 1 shows the number of days out from practice and play in the NFL with single and repeat concussion. For this study, the cases involving 7+ days out were investigated. They represent 7.8% of single concussions, 10.0% of those with a second injury, and 14.1% ($P = 0.09$) of players experiencing the third to seventh concussion in the 6-year study period. On average, concussions with 7+ days out represent 8.1% of all MTBIs in the NFL, but it is an important group if there is a way to differentiate the likelihood of having 7+ days out at the time of the initial medical evaluation.

There were 72 cases with 7+ days out involving 68 players. The average age of the players was 27.6 ± 3.6 years, similar to the average of players with 0, 1, 2, and 3 to 6 days out with concussion. Of these injuries, 38 were single concussions in the study period, 8 were the first of repeat concussion, 16 were the second, 7 the third concussion, and so on in the study period. The median duration between first injury and 7+ days out was 364 days, and the median duration between last injury and 7+ days out was 329 days, statistically similar.

Four players had two concussions with 7+ days out in the study period. Two of these players had successive concussions in the same season. For one player, it was the second and third concussions. The third injury occurred 38 days after the second. The second injury involved 13 days out, and the third, 32 days out. For the other player, it was the first and second concussions. The second injury occurred 40 days after the first, and each injury involved 9 days out. Two players had concussions with 7+ days out in successive seasons. For one player, it was the fourth and fifth concussions, involving 22 and 15 days out. That player had a sixth concussion, involving 6 days out in the study period. For the other player, it was the first and third injuries, involving 9 and 46 days out. The intervening concussion occurred 18 days before the third injury and involved 3 days out. The player with 98 days out with the third concussion had experienced the second injury in the previous season, and that injury involved 0 days out.

Table 2 shows the incidence of concussion by player position for all concussed players in the six NFL seasons. There were 650 players who experienced 887 concussions during the study period, and

TABLE 1. Days out with single and repeat concussions in the National Football League, 1996–2001

Days out	Single concussion	Repeat concussions							Total concussions
		1st	2nd	3rd	4th	5th	6th	7th	
0	261	98	92	32	7	5	1	1	497
1	50	18	14	1	2				85
2	82	21	20	8	2	1	1		135
3	34	8	11						53
4	9	4	4	1					18
5	5				1				6
6	11	3	3	2	1		1		21
7	8								8
8	8	1	1						10
9	8	6	10	3	1				28
10	3		1						4
11	2								2
12	1								1
13	2		2	1					5
15	1					1			2
16	2								2
17	1								1
18			2						2
20		1							1
22					1				1
33	1			1					2
41	1								1
46				1					1
98				1					1
No. of concussions	490	160	160	51	15	7	3	1	887
No. of 7+ days out	38	8	16	7	2	1	0	0	72
% 7+ days out	7.8%	5.0%	10.0%	13.7%	13.3%	14.3%	0.0%	0.0%	8.1%

their position for each concussion was counted in this analysis. The left columns show the number of concussions by player position with <7 and 7+ days out. As a group, special teams (29.2%) experienced fewer concussions with 7+ days out than the defensive (34.7%) and offensive (36.1%) teams. Individually, the position groups most often associated with 7+ days out are the defensive secondary (23.6%), kick unit (19.4%), quarterbacks (12.5%), and wide receivers (12.5%).

The fraction of players in a position with 7+ days out versus all in that group was highest for the quarterback (14.8%), the return unit on special teams (11.8%), the secondary (10.8%), followed by the kick unit (10.4%) on special teams. Also included is the odds ratio (OR) for those positions statistically different for 7+ days out with concussion versus other positions on the team. Quarterbacks had the highest odds ratio (OR = 2.1, $P = 0.049$) of 7+ days out with concussion, whereas running backs had the lowest relative risk (OR = 0.13, $P = 0.021$). As a position group, special teams have a higher incidence of 7+ days out (OR = 1.34, P = not significant [NS]). The punter in the 7+ days out group sustained his injury during practice, early in the season. He had a "collision with the ground" in some game-like scrimmage on Astroturf.

Table 3 shows the team activity associated with concussion and the number of days out from the NFL. The highest incidence of 7+ days out occurs in passing plays (36.1%), followed by kickoffs (22.2%). The frequency of 7+ days out for concussions in an activity category is also included. The highest frequency of 7+ days out by activity versus all concussions is in kickoffs (12.4%), runs inside tackle (8.6%), and passing plays (8.5%). There was no statistical difference in team activity for comparison of <7 and 7+ days out (P = NS).

Table 4 summarizes the signs and symptoms of concussion by days out during the 1996 to 2001 seasons. The players experiencing 7+ days out have more signs and symptoms than those with MTBIs involving fewer days out. The symptoms with the highest incidence are headaches (70.8%), dizziness (52.8%), immediate recall problems (44.4%), and delayed retrograde amnesia (41.7%) with concussion having 7+ days out. Many of the symptoms occur at a statistically higher rate. On the basis of χ^2 analysis, the symptoms most frequently

TABLE 2. Incidence of concussions involving <7 and 7+ days out by player position in six National Football League seasons[a]

Player position	Days out			Odds ratio[b]	95% confidence interval
	<7	7+	% 7+		
Offense					
Wide receiver	101	9	8.2%	1.01	0.49–2.09
Running back	77	1	1.3%	0.14[c]	0.02–0.99
Quarterback	52	9	14.8%	2.10[d]	0.99–4.45
Offensive line	71	5	6.6%	0.78	0.31–2.00
Tight end	45	2	4.3%	0.49	0.12–2.06
Subtotal	346	26	7.0%	0.77	0.46–1.26
Defense					
Secondary	140	17	10.8%	1.49	0.84–2.65
Defensive line	73	5	6.4%	0.76	0.30–1.94
Linebacker	62	3	4.6%	0.53	0.16–1.73
Subtotal	275	25	8.3%	1.04	0.63–1.73
Special team					
Kick unit	121	14	10.4%	1.38	0.75–2.56
Return unit	30	4	11.8%	1.54	0.53–4.50
Return ball carrier	20	2	9.1%	1.14	0.26–4.96
Punter	7	1	12.5%	1.63	0.20–13.40
Kicker, FGA	1	0	0.0%	0.00	
Kicker, PAT	1	0	0.0%	0.00	
Holder	1	0	0.0%	0.00	
Undesignated	11	0	0.0%	0.00	
Subtotal	192	21	9.9%	1.34	0.78–2.28
Unknown	2	0	0.0%	0.00	
Total	815	72	8.1%		

[a] FGA, field goal attempt; PAT, point after touchdown.
[b] Unit of observation is concussion. Also tested using unit of observation as the player, with virtually the same results.
[c] $P = 0.021$, $\chi^2 = 5.36$.
[d] $P = 0.049$, $\chi^2 = 3.87$.

occurring with 7+ days out are not oriented to time ($\chi^2 = 51.2$, $P = 0.001$), loss of consciousness >1 minute ($\chi^2 = 33.5$, $P = 0.001$), retrograde amnesia ($\chi^2 = 33.2$, $P = 0.001$), fatigue ($\chi^2 = 28.1$, $P = 0.001$), and the general category of cognition problems ($\chi^2 = 21.7$, $P = 0.001$). There was also a higher incidence of problems with immediate recall ($\chi^2 = 17.2$, $P = 0.001$) and the general category of memory problems ($\chi^2 = 18.0$, $P = 0.001$). On average, there were 4.64 signs and symptoms with 7+ days out compared with 2.58 for concussions with <7 days out ($t = 6.0$, $df = 77.7$, $P < 0.001$).

For the <7 days out group, of 326 players with an entry recorded at the time of concussion, there were 40 players who experienced loss of consciousness. The other cases were listed as indeterminate, unknown, or unrecorded. The duration of unconsciousness ranged from 1 second to 15 minutes. Seven players were unconscious 1 minute or more. For the 7+ days out group, of 28 recorded cases, 19 players experienced loss of consciousness. The duration of unconsciousness ranged from 6 seconds to 22.5 minutes, and there were 7 cases with 1 minute or more of unconsciousness. Seven of the 19 players with loss of consciousness were hospitalized, and the remainder were removed from play. Although the typical duration of unconsciousness was less than 1 minute, the four longest ranged from 3 minutes 20 seconds to 22.5 minutes, but interestingly, none of these patients were hospitalized. The nine hospitalized patients usually showed loss of consciousness and many signs and symptoms; however, one player had only two. It was his fourth concussion. On the basis of reported cases, the rate of unconsciousness was 5.5 times greater in the 7+ days out group than in those out <7 days ($P < 0.001$).

Knowing which players will experience prolonged removal from play would be valuable to team physicians and players. The simple assumption that all players will return to play in <7 days would miss all those players out 7+ days; that is, this assumption has a sensitivity of 0%. To increase the sensitivity of that estimate, signs and symptoms occurring at least twice

TABLE 3. Team activity at time of injury and days out with concussion in the National Football League[a]

Team activity	Days out								Total	% <7	% 7+	% 7+ versus all
	0	1	2	3	4	5	6	7+				
Passing	167	27	42	26	10	3	4	26	305	34.2%	36.1%	8.5%
Interception	5	0	0	1	0	1	3	0	10	1.2%	0.0%	0.0%
Run inside tackle	88	15	23	5	1	1	6	13	152	17.1%	18.1%	8.6%
Run outside tackle	75	11	26	8	1	0	0	8	129	14.8%	11.1%	6.2%
Fumble	3	0	0	0	0	0	0	0	3	0.4%	0.0%	0.0%
Punt	40	9	15	6	1	0	1	5	77	8.8%	6.9%	6.5%
Field goal attempt	1	1	0	0	0	0	0	0	2	0.2%	0.0%	0.0%
Point after touchdown	1	0	0	0	0	0	0	0	1	0.1%	0.0%	0.0%
Kickoff	71	12	19	5	2	1	3	16	129	13.9%	22.2%	12.4%
Other	28	5	5	2	3	0	2	1	46	5.5%	1.4%	2.2%
Unknown	18	5	5	0	0	0	2	3	33	3.7%	4.2%	9.1%
Total	497	85	135	53	18	6	21	72	887	815	72	8.1%

[a] P = not significant for comparison of <7 and 7+ on team activity.

as frequently in those players experiencing 7+ days out were entered into a multivariate logistic regression with forward selection to arrive at a parsimonious group of signs and symptoms. The presence of fatigue, photophobia not oriented to time, or retrograde amnesia correctly identified 50 of the 72 players experiencing 7+ days out, for a sensitivity of 69%, and correctly identified 599 of the 815 players who did not experience 7+ days out, for a specificity of 73%. Unfortunately, the presence of any one of those signs and symptoms also identified 216 players who did not experience 7+ days out, for a positive predictive value of 19%. Adding loss of consciousness for 1 minute or more to the screening increased the sensitivity to 72% but decreased the specificity.

Table 5 summarizes the medical action taken after concussion by the number of days out. The majority of players with 7+ days out with concussion are removed from the game (72.2%). The next highest fraction is players who are hospitalized (12.5%). Only 6.9% of the players who experienced 7+ days out returned to the game immediately. In terms of percentage of players with 7+ days out versus those with <7 days out by action taken, a higher fraction (45.0%) were hospitalized and removed from play (12.6%). The differences in action taken are statistically significant ($\chi^2 = 68.03$, $df = 3$, $P < 0.0001$ for comparison of <7 versus 7+ days out). For players who returned immediately or rested and returned to the game, the median time was 15 minutes (mean, 30.1 min) for those out <7 days. For the 10 players out 7+ days, it was not possible to determine the time to return to the same game, but the median time to the next return to play was 10 days (mean, 12.1 d).

Table 6 shows the management of players with concussion by number of days out. The majority of players with concussion (88.9%) are rested with 7+ days out, compared with 90.7% with <7. In terms of percentage of players with 7+ days out versus fewer days by management, a greater fraction are given medical procedures (22.2%), immobilized (20.0%), and given therapeutic procedures (18.2%) with 7+ days out. Overall, the data show a conservative treatment of concussion. There was no statistical difference in player management for comparison of <7 and 7+ days out (P = NS).

Signs and Symptoms from Initial and Follow-up Examinations

Figure 1 shows the time to initial and follow-up examinations for the players with 7+ days out. The initial examination was typically made within a couple of minutes for the majority of players. The median first follow-up examination was within 19.3 hours (3.2 and 43.1 h were the 25th and 75th percentiles). Typically, the fourth median follow-up examination was made in 4.7 days and the seventh at 13 days for the most severely injured players.

Between the initial examination and first follow-up, most of the signs and symptoms started to decrease, except for increases in the general category of memory problems (24–39 cases with problems), fatigue (17–22 cases), irritability (1–3 cases), and sleep problems (3–4 cases). Curiously, the large increase in the general category of memory problems was not seen in the specific symptoms of attention problems, retrograde amnesia, antegrade amnesia, and information processing, which decreased 30 to 60% by the first follow-up examination.

By the fourth follow-up examination, all memory and cognition problems had cleared. However, the following signs and symptoms were present in some players. Nine (17.7%) still showed a general symptom, which was exclusively headaches. Eight (18.2%) of the initial 44 players with cranial nerve symptoms remained. The two specific symptoms were dizziness in 5 (16.7%) and photophobia in 2 (25%) from the initial examination. Somatic complaints remained in 4 players (16%), including 1 (14.3%) with personality change and another with fatigue. By the

TABLE 4. Initial signs and symptoms of concussion by days out from the National Football League[a]

Signs and symptoms	Days out; no. of cases (n = 887)								% <7 (815)	% 7+ (72)	7+ injuries versus all (8.1%)	Comparison of <7 and 7+	
	0 (497)	1 (85)	2 (135)	3 (53)	4 (18)	5 (6)	6 (21)	7+ (72)				*P* value	χ^2
General symptoms	281	56	106	37	16	5	15	55	63.3%	76.4%	9.6%	0.026	4.93
Headaches	243	52	95	34	15	4	15	51	56.2%	70.8%	10.0%	0.016	5.80
Nausea	32	7	18	7	2	1	5	6	8.8%	8.3%	7.7%		
Vomiting	5	2	4	1	1	0	2	0	1.8%	0.0%	0.0%		
Neck pain	44	8	33	5	3	2	7	14	12.5%	19.4%	12.1%		
Back pain	3	0	0	0	0	0	0	0	0.4%	0.0%	0.0%		
Syncope	9	1	0	1	0	0	1	1	1.5%	1.4%	7.7%		
Seizures	0	0	0	0	1	0	0	0	0.1%	0.0%	0.0%		
Somatic complaints	65	15	33	17	8	3	8	29	18.3%	40.3%	16.3%	0.001	19.95
Irritability	14	1	6	3	1	0	0	1	3.1%	1.4%	3.8%		
Anxiety	17	6	12	2	3	0	0	7	4.9%	9.7%	14.9%		
Depression	0	0	1	0	0	0	1	1	0.2%	1.4%	33.3%		
Personality change	14	4	7	3	2	2	5	8	4.5%	11.1%	17.8%	0.015	5.93
Fatigue	26	5	11	11	4	2	3	19	7.6%	26.4%	23.5%	0.001	28.12
Sleep disturbance	1	1	1	0	0	0	0	3	0.4%	4.2%	50.0%	0.001	14.21
Loss of libido	0	0	0	0	0	0	0	0	0.0%	0.0%	—		
Loss of appetite	1	1	0	0	0	0	0	0	0.2%	0.0%	0.0%		
Cranial nerve effects	258	47	70	31	6	3	9	50	52.0%	69.4%	10.5%	0.005	8.07
Dizziness	208	32	56	26	4	2	8	38	41.2%	52.8%	10.2%		
Vertigo	13	3	10	1	0	1	0	5	3.4%	6.9%	15.2%		
Tinnitus	11	1	5	1	0	0	1	4	2.3%	5.6%	17.4%		
Nystagmus	2	4	0	1	0	0	1	1	1.0%	1.4%	11.1%		
Hearing loss	0	0	0	0	0	0	0	0	0.0%	0.0%	—		
Diplopia	11	0	1	2	0	1	0	3	1.8%	4.2%	16.7%		
Photophobia	12	4	3	3	1	1	1	9	3.1%	12.5%	26.5%	0.001	14.34
Blurred vision	69	17	26	10	1	2	1	16	15.5%	22.2%	11.3%		
Pupil response	3	1	0	1	1	0	0	0	0.7%	0.0%	0.0%		
Pupil size	1	0	0	0	0	0	0	0	0.1%	0.0%	0.0%		
Cognition problems	83	23	50	24	11	3	7	36	24.7%	50.0%	15.2%	0.001	21.69
Not oriented to person	6	1	6	2	2	1	0	6	2.2%	8.3%	25.0%	0.002	9.43
Not oriented to place	11	3	12	5	2	2	0	10	4.3%	13.9%	22.2%	0.001	12.65
Not oriented to time	22	3	10	7	3	2	0	21	5.8%	29.2%	30.9%	0.001	51.17
Immediate recall	76	20	45	23	11	2	7	32	22.6%	44.4%	14.8%	0.001	17.17
Memory problems	144	32	60	32	11	2	11	44	35.8%	61.1%	13.1%	0.001	17.97
Attention problems	45	12	23	11	4	0	4	18	12.1%	25.0%	15.4%	0.002	9.54
Information processing	63	13	30	15	4	0	4	18	15.8%	25.0%	12.2%	0.045	4.03
AGA delayed	23	9	16	12	5	1	2	12	8.3%	16.7%	15.0%	0.018	5.59
RGA delayed	60	11	24	17	4	1	5	30	15.0%	41.7%	19.7%	0.001	33.21
Unconsciousness (≥1 min)	0	0	3	1	2	1	0	7	1.7%	9.7%	50.0%	0.001	33.46
All loss of consciousness	14	6	9	6	3	2	0	19	4.9%	26.4%	32.2%		
All reported as zero	193	27	38	12	7	2	7	9	36.2%	12.5%			
Mean no. of symptoms	2.10	2.61	3.37	3.85	4.22	4.50	3.48	4.64	2.58	4.64		0.001	$t = 6.02$, $df = 77.7$, $P < 0.001$

[a] AGA, antegrade amnesia; RGA, retrograde amnesia.

TABLE 5. Action taken with players experiencing concussion by days out from the National Football League[a]

Action taken	Days out								Total	% <7	% 7+	% 7+ versus all
	0	1	2	3	4	5	6	7+				
Removed from play	137	55	95	37	14	4	18	52	412	44.2%	72.2%	12.6%
Rest and return	241	19	28	9	1	1		5	304	36.7%	6.9%	1.6%
Hospitalized	1	1	2	3	1	1	2	9	20	1.3%	12.5%	45.0%
Return immediately	111	5	8	3	2		1	5	135	16.0%	6.9%	3.7%
Unknown	7	5	2	1				1	16	1.8%	1.4%	6.3%
Total	497	85	135	53	18	6	21	72	887	815	72	8.1%

[a] $\chi^2 = 68.03$, $df = 3$, $P < 0.0001$ for comparison of <7 versus 7+ days out on action taken.

seventh examination, only headaches remained to clear, because all other signs and symptoms had returned to normal.

Video of 182 NFL Game Impacts

There were 18 concussions involving 7+ days out that were caught on video and showed clear evidence of the collision. They fell into three general types. The first type involved 6 helmet impacts (33.3%) on the facemask of the concussed player in the 0- to 45-degree quadrant (see Pellman et al. [39] for a description of the quadrant and level). The blows were mostly low on the facemask (3 at −Q3 and 2 at −Q1), because the player's head was pushed up and backward. The second type involved 6 helmet (33.3%) (4 cases) or shoulder pad (2 cases) impacts to the side of the helmet in the 45- to 135-degree quadrants. These were usually high on the helmet (2 at +Q2 and 3 at +Q3) at a downward angle toward the neck. The third type involved 6 impacts to the back of the helmet (135–180 degrees) (33.3%) either from a fall to the ground (3 cases) or helmet impact (3 cases). The majority (4 cases) of these impacts were at level +Q2 on the back of the helmet. The general characteristics of the impacts were observed and compared with other concussion blows involving fewer days out. This included the type of play, tackle, and speed of impact.

The review of 18 cases involving 7+ days out caught on game video supports this point. Most of the cases involve open-field impacts in which the players are running at high speeds before the collision. The most severe impacts seem to occur on kickoffs and passing plays, in which the offensive and defensive players are running at full speed. In a few cases, players from the same team converge on the ball carrier or receiver, missing their target and colliding together. In others, the collision involves a targeted impact with players lowering their helmets and driving through the other player, or the player is thrown hard to the ground. In a few cases, the collision involved a double impact, which may be a factor in the severity of injury. These cases involved different directions of loading in the collision sequence.

Two of the cases of 7+ days out caught on game video were also reconstructed in the laboratory with crash test dummies. They are cases 69 and 124 in Pellman et al. (38, 39) and involved very high impact speeds, 10.3 m/s (23.0 mph) and 11.4 m/s (25.5 mph), respectively. These speeds are in the highest range of the collision severities for concussion in the NFL, close to average plus 1 standard deviation in collision speed. Pellman et al. (38) found that the average concussion impact in the NFL was at 9.3 ± 1.9 m/s (20.8 ± 4.3 mph), so the average plus 1 standard deviation is 11.2 m/s (25.0 mph). Interestingly, the two reconstructed cases did not produce the highest head accelerations or velocity changes, even though the collisions were in the upper extreme in impact severity.

DISCUSSION

The present study involved specifically a large cohort of athletes with relatively extended return to play after MTBI. Data are presented on 72 cases involving 68 players in a 6-year study. It must be emphasized that this represents only a small percentage (8.1%) of all NFL MTBIs and an even smaller percentage (2.1%) of the approximately 3200 players involved in the NFL during the study years. However, this is a very important group, which represents the most severely injured of the NFL concussion cases.

Postconcussion Syndrome

Prolonged postconcussion syndrome has been reported in a significant percentage of the general population of mild head injury patients (30). The various symptoms that are part of the postconcussion syndrome also persist in many of these patients. There have been a number of previous studies in nonathletes on the persistence of postconcussion symptoms for extended periods of time after mild head injury. Headaches have been reported in between 36 and 71% of patients at 1 week, between 31 and 90% at 1 month, between 47 and 78% at 3 months, between 8 and 35% at 1 year, and in 24% of patients 4 years after head injury (7, 14, 17, 19, 25, 34, 42–44). Dizziness is reported to occur in 53% of patients in the first week and persists after 2 years in 18% of patients (7, 19, 25). Photophobia was reported in 7% of patients 14 days after head injury (19, 22).

Psychological and somatic complaints such as personality change, anxiety, irritability, and depression have been reported

TABLE 6. Management of players experiencing concussion by days out from the National Football League[a]

Management	Days out								Total	% <7	% 7+	% 7+ versus all
	0	1	2	3	4	5	6	7+				
Immobilization			1		1	1	1	1	5	0.5%	1.4%	20.0%
Therapeutic modality	3	2	3		1			2	11	1.1%	2.8%	18.2%
Prescription drug therapy	5	2	1				1	1	10	1.1%	1.4%	10.0%
Proprietary drug therapy	4		2					1	7	0.7%	1.4%	14.3%
Rest	449	78	121	52	15	5	19	64	803	90.7%	88.9%	8.0%
Medical procedures	5		1		1			2	9	0.9%	2.8%	22.2%
Unknown	31	3	6	1				1	42	5.0%	1.4%	2.4%
Total	497	85	135	53	18	6	21	72	887	815	72	8.1%

[a] P = not significant for comparison of <7 and 7+ days out on management.

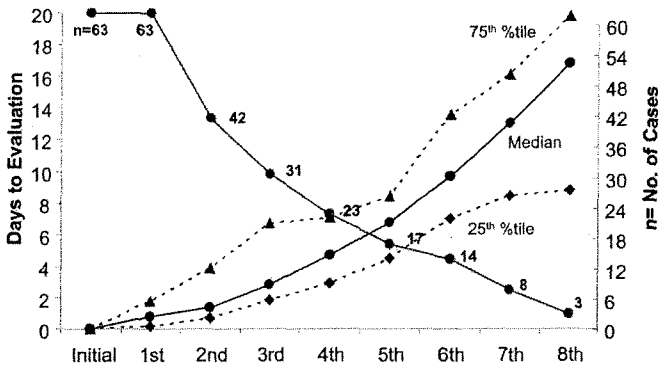

FIGURE 1. *Median time to initial and follow-up examinations for players with 7+ days out based on 63 cases with at least a first follow-up examination (n = the number of patients with follow-up examinations).*

in 51 to 84% of patients after 3 months and persist in 15 to 33% at 1 year and 15% after 3 years (14, 19, 42–44). One study reported persistent fatigue in 23% of patients 6 months after mild head injury (19, 34). Disrupted sleep patterns have been found in 15% of patients 6 weeks after injury (19, 44). Memory problems reportedly occur in 19% of patients at 1 month after head trauma, 59% at 3 months after injury, and 15% at 6 months after injury (19, 34, 42, 44). Persistent cognitive impairments (impaired attention, slowed reaction times, and slowed processing speeds) have been described in a significant percentage of patients 3 months after MTBI (3, 15, 19, 24).

Another way to evaluate persistent postconcussion impairments is by assessing ability to return to work after mild head injury. There have been three prospective studies of time interval to return to work after mild head injury (15, 19, 42). All patients were hospitalized after their injury, so it is not clear that these are truly representative of MTBI patients. One study found that 57 to 100% had returned to work at 3 months. A second study found that 20% had not returned to work at 2 months. The third study reported that 75% had not returned to work at 1 month, 37% had not returned at 6 months, and 20% had not returned to work at 1 year after the injury.

A review of these previous studies suggests that there are clearly a significant minority of general population patients with persistent postconcussion syndrome after "mild head injury." However, there is a great deal of variability in the actual percentages reported. This most likely reflects the variability of inclusion criteria, definitions of mild head injury, means of data collection, and extent of neurological, neuropsychological, and radiological investigations of the patients.

The results of the present NFL study are very different from those of the previously cited studies. Postconcussion symptoms and signs resolved much more quickly in NFL players than in nonathletes. Furthermore, only a very small percentage of NFL players missed 7+ days, and even fewer were impaired for more prolonged periods. After MTBI, a large percentage of NFL players recover fully within minutes to 1 hour. A smaller percentage have persistent postconcussion symptoms for a period of hours up to 2 days before becoming asymptomatic.

These athletes can be considered to be in the recovery phase after MTBI with postconcussive symptoms but do not truly have a postconcussion syndrome. These players return to practice within a few days and play in the next week's game. Only 8.1% of the NFL players with concussions in this study had persistent symptoms and/or signs for 3 to 5 days or longer after injury and were out 7+ days (they missed at least one game). This small group can truly be seen as experiencing a postconcussion syndrome. The great majority of these players (80.5%) become asymptomatic in 7 to 11 days and return to play before the second game after injury (14 d after injury). Thus, almost all the NFL players who experience postconcussion syndrome recover in less than 2 weeks. Only a very small number (19.5% of the 7+ days out group) have symptoms persisting for longer than 2 weeks and thus miss more than two games.

This study defines four groups of NFL MTBI patients, classified by time course of recovery (*Table 7*). The immediate recovery group represents 56.0% of the NFL concussions and becomes asymptomatic within minutes to 1 hour after MTBI. They return

to play on the day of the injury. The early recovery group is 35.9% of the cases and has postconcussive symptoms and signs for between 1 hour and 2 days after injury before becoming asymptomatic. These players return to play in <7 days. They do not develop a postconcussion syndrome. The small number (6.5%) of players who fall into the short-duration postconcussion syndrome are symptomatic for 3 to 10 days before making a full recovery and are kept out of play for more than 7 but fewer than 14 days (they miss only one game after injury). The extremely small number (1.6%) of players who are symptomatic for more than 10 days and thus miss two or more games after injury constitute the prolonged postconcussion group.

There are multiple possible explanations for these differences between MTBI recovery in nonathletes and NFL players. Patients more than 40 years old reportedly have a worse prognosis after MTBI than younger patients (15, 19–21, 42). Because virtually all NFL players in this study are under 40 years old (most are in their 20s), one would expect an overall quicker recovery in the NFL players. The cited studies on nonathletes with concussion involved patients with multiple causes of injury and variable definitions of concussions. In contrast, the present study involved MTBI patients with specific, definite inclusion criteria and known football-related causes only. The nature of the MTBI injuries may also have been different.

One of the other processes that may account for some of these differences between NFL players and the general population might be deemed to be a type of artificial selection. Most NFL players have been involved with organized football since junior or senior high school and on through college. It is well known that MTBIs occur at all these levels of the sport. For whatever reasons, certain individuals undoubtedly are more prone to MTBI than others. Some individuals are more prone to delayed or poor recovery after MTBI. These groups may overlap. It is likely that many of these individuals will stop playing organized football before reaching the professional level. They are "selected out" either of their own volition or because their head injuries prevent them from continuing to participate in the sport. As a result of this winnowing process, those players who ultimately play in the NFL are probably less susceptible to MTBI and prolonged postconcussion syndrome than the general population.

Previous work from the present authors (38, 39) has documented that the accelerations and durations of impacts in NFL head injuries are much different from those seen in automobile accidents and other common causes of MTBIs in nonathletes. The protective benefits of football helmets may also improve the prognosis of MTBI in NFL players compared with the general nonhelmeted population. The nature of the injured patients may also play a significant role in improved prognosis. NFL players are a highly conditioned, physically fit population accustomed to playing with pain and highly motivated to return to play as soon as possible. In these regards, they are much different from the general population. In addition, NFL players receive a high level of individualized medical care from the day they reach training camp that is often not available to the general public. All of these factors probably play a role in the differing clinical courses seen in NFL versus nonathlete MTBI patients.

Previous studies on nonathletes have found that age is a significant prognostic factor in MTBI patients (15, 19, 21, 42). The evidence showed that patients more than 40 years old have an increased risk of prolonged postconcussion symptoms and delayed return to work. The authors are unaware of any such data in athletes. The results of this present study indicate that there is no difference in median age between the groups with 7+ days out and those with <7 days out. This demonstrates that within the age groups of 20 to 40 years, age differences are not associated with an alteration in prognosis after MTBI.

Very few data are available regarding persistent postconcussion symptoms in athletes after MTBI. There are a number of studies reporting the results of neuropsychological testing for 7 to 10 days after injury (8, 9–11, 16, 18, 26, 27, 29). These generally indicate that the neuropsychological test performance falls off from baseline immediately after MTBI but almost always returns to baseline by 7 to 10 days after injury. A 4-year prospective study of football players at one university also recorded subjective symptoms of 200 players after MTBI (40). Memory disturbances were reported by 34% of players at 1 day, 27% at 5 days, and 8% at 10 days. Complaints of dizziness were reported by similar percentages of the players at similar time intervals.

Another recent study of 196 concussions in collegiate football players collected data from questionnaires filled out by team athletic trainers (23). This study reported that 167 players had headaches at the time of injury, 149 (89%) had headaches at 3 hours, 110 (65.9%) had headaches 1 day after injury, 41 (24.5%) had headaches 5 days after injury, and 23 (13.8%) had headaches at Day 7. It was also reported that 12.2% of the players did not achieve full symptom resolution within 1 week. The same group also reported on recovery time after concussion in collegiate football players (32). The study reported the results on the basis of symptom checklists, standard-

TABLE 7. National Football League concussion recovery groups by signs and symptoms and return to play[a]

Concussion recovery group	Signs and symptoms	Return to play	% NFL MTBI
Immediate recovery	<1 h	On the day of injury	56.0%
Early recovery	1 h–2 d	1–6 d out	35.9%
Short-duration postconcussion syndrome	3–10 d	7–14 d out	6.5%
Prolonged postconcussion syndrome	>10 d	>14 d out	1.6%

[a] NFL, National Football League; MTBI, mild traumatic brain injury.

ized concussion assessment scores, and results of standardized balance testing along with results of a neuropsychological test battery. The study reported a pattern of more severe dysfunction in all spheres immediately after injury, followed by a gradual improvement over the next few days. Neuropsychological cognitive deficits persisted up to Day 5 and usually resolved by Day 7. None of these previous studies focused on the more severely injured of the MTBI patients.

Game Video of Concussion Impacts

The present study found that players at certain positions (quarterbacks, defensive backs, wide receivers, return team players) have a higher incidence of delayed recovery compared with other position players. Earlier studies from the NFL MTBI Committee (36, 37) found that these same position players were at increased risk of sustaining any concussion and repeat concussions compared with players at other positions. Players at higher-risk positions are subjected to the highest impact speeds and blindside hits, thus accounting for the increased risk of sustaining concussion.

Running backs have a statistically lower (OR = 0.14) incidence of 7+ days out with concussion. There are several reasons why they have a much lower rate of concussion causing 7+ days out. In part, their running stance helps them avoid very high-speed collisions when they are unprepared, their peripheral vision allows them to make lateral movements avoiding direct impacts, and their experience allows them to get ready for impacts by aligning their head, neck, and torso to present a greater effective mass when a head impact collision occurs. The opposite is true with blindside impacts of the quarterback and some collisions of the secondary and during kickoffs and punts.

These cases and the game video of the 7+ days out impacts point to the possible need for special helmets for the most vulnerable players and plays in the NFL and for testing the performance of protection systems in up to 11.2-m/s (25.0-mph) impacts. This is one way to address the risks of concussion with prolonged recovery and the means to reduce concussion risks in the most severe NFL collisions. High-speed testing may lead to new innovations in helmet design.

Predictors of 7+ Days Out

Some previous studies have indicated that loss of consciousness is correlated with severity of MTBI, whereas other studies have disagreed with this position (14, 24, 28, 33). The results of this present study indicate that loss of consciousness is a risk factor for missing 7+ days after MTBI, although few players in the 7+ days out group experienced loss of consciousness for 1 minute or more. This is the threshold suggested by Cantu (5). Loss of consciousness occurs across all severities of concussion in the NFL, so it is not, per se, a specific hallmark of the most severely injured. The results of this study nevertheless support those who believe that loss of consciousness is a factor that is related to grading the severity of concussion.

There are multiple other factors that are of prognostic significance; loss of consciousness alone is certainly not the only factor that determines prognosis. A number of researchers have suggested that the presence and duration of posttraumatic amnesia is an even better predictor of prognosis after MTBI than is loss of consciousness (6, 17, 19, 33, 34). The present study found that the presence on initial evaluation of retrograde amnesia, difficulties with immediate recall, and memory problems in general were significantly correlated with ultimate inclusion in the group of players who were out 7+ days. This certainly lends support to the notion that amnesia after MTBI has prognostic significance.

The present results also indicate that disorientation to time and general cognitive difficulties are also correlated with inclusion in the study cohort. Thus, it may be more accurate to state that the presence of disorientation, amnesia, or other cognitive problems after MTBI is very predictive of a more delayed recovery. In fact, one could generalize even further and state that it is the absolute number of postconcussion signs and symptoms reported on the initial evaluation that are as important as specific signs and symptoms in assessing prognosis. The present data showed a highly statistically significant difference between the number of signs and symptoms seen in the study group and in the players who were out <7 days. On initial evaluation, the players who ultimately would end up in the 7+ days out group had an average of 4.64 signs and symptoms versus an average of 2.58 signs and symptoms in the players who would ultimately end up in the <7 days out group. Players with more signs and symptoms presumably have more widespread central nervous system dysfunction, which translates into a slower recovery.

The present study also indicates that hospitalizations after injury and the removal from play after injury are statistically associated with ultimately ending up in the group of players who are out 7+ days. A very revealing finding is that only 6.9% of players who eventually were out for 7+ days were allowed to return to play on the day of the injury. This indicates that NFL team physicians and athletic trainers are extremely effective in screening out the most severely injured players on the sidelines within a short period of time after injury. It is important to note that team medical personnel were making these decisions on an individual case-by-case basis without the imposition of any formal management or evaluation guidelines. Team physicians used their clinical judgment along with whatever ancillary testing (neuropsychological testing, magnetic resonance imaging scans, etc.) they deemed necessary to make these decisions. Many of the NFL players included in this study had neuropsychological testing, which will be reported later. Those results are consistent with the clinically based findings presented in this article.

Players with repeat concussion were more likely to end up in the study cohort than players with only a single concussion. Although this could indicate a cumulative effect of previous injury, a previous study (37) did not find evidence of such cumulative effects. It is certainly possible that team physicians are more cautious in treating patients with repeat injuries and are thus more likely to delay their return to play. The results of this present study indicate that having a concussion that results in 7+ days out does not result in an increased risk of sustaining a repeat injury after the player has returned to play. Only 11 players who were out 7+ days went on to

sustain a repeat concussion. However, 4 of them were out 7+ days on the repeat concussion. Over the 6 years of this study, there were 152 players who sustained an MTBI, returned to play in <7 days, and subsequently went on to sustain a repeat concussion at some point. Of these players, 19 were out 7+ days after the repeat concussion. These data would argue against there being any long-term predisposition to repeat MTBI in players who experience delayed recovery after a first MTBI. However, there was one player who sustained his fourth and fifth concussions in the 7+ days out group.

Headaches were seen more frequently in the study cohort than in other players. However, because headache is such a common symptom in both groups of MTBI patients, its presence alone does not have prognostic significance to the clinician. Photophobia was noted in a statistically significantly increased percentage of players in the study cohort. It is well known that photophobia is frequently seen in migraine-type headaches. It is likely that the photophobia seen in these study patients was part of a postconcussion migraine-type syndrome. The results suggest that players with postconcussion migraine-type syndromes are at increased risk of experiencing delayed return to play. This is not surprising, given the well-known propensity of migraine headaches to be debilitating and disabling to victims in the nonathlete general population.

Sleep disturbance is also statistically correlated with delayed return to play. However, the very small absolute number of players who experience this symptom suggests a very cautious approach to its prognostic use. Fatigue is another symptom prominently associated with delayed return to play. Although it is by nature a nonspecific, vague complaint, the listlessness, tiredness, and lack of energy that are implied certainly represent a major challenge to return to play in such a physical contact sport as professional football. The data suggest that the presence of fatigue on initial evaluation has prognostic significance that might be of value to treating physicians. It is interesting to note that fatigue was more commonly noted on the first follow-up evaluation the day after injury than it was on the initial evaluation. It should be noted that dizziness was also seen frequently in players in the study cohort. However, it is also seen frequently in players who return to play in <7 days and therefore has no prognostic significance.

Follow-up Examinations

Analysis of the evaluation of signs and symptoms over time reveals some interesting results. Especially notable is the increased frequency of memory and cognitive impairments at first follow-up (usually the day after the injury) compared with the initial evaluation, usually on the day of the injury. This may suggest that the MTBI sets off intracranial processes that result in worsening cognitive functioning over the first 24 to 48 hours after injury. It is also of interest to note that these clinical cognitive and memory impairments almost always resolve over the next few days, suggesting that the intracranial processes are self-limited and short-lived. These results also confirm the clinical impression of team physicians and athletic trainers that persistence of headaches is the most common reason for extended delays in return to play.

There are multiple factors that help determine return-to-play decisions in this group of athletes. Most of the players who will ultimately miss 7+ days are identified as having significant MTBI shortly after the traumatic event. They have multiple symptoms and signs at initial evaluation, especially signs of cognitive and memory impairments. As a result, they are very rarely allowed to return to the game on the day of injury. When seen in the first follow-up evaluation the next day, most of these players still have multiple symptoms and signs, with even more frequent memory impairments and complaints of fatigue. Headaches and photophobia are often prominent as well. Over the next few days, the signs and symptoms generally improve to a point at which headaches alone are the most common isolated residual symptom. Almost all the players are asymptomatic by 5 to 6 days after the injury.

Why, then, are the players in this group kept out of play for 7 or more days? First, they have missed practice all week long, and most teams will not allow players who miss so many practices to participate in a game. Second, most team physicians adopt a cautious approach and want these players to practice for a few days without a reemergence of symptoms before being allowed to play in a game situation. Third, some of these players may have had abnormalities on neuropsychological testing (not part of the database for this article but certainly part of the database available to the team physicians as part of their decision-making process) that may suggest a delay in return to play.

Players with persistent signs and symptoms are not allowed to return to play until they become asymptomatic. It seems that of all the signs and symptoms, persistent headaches are the most common reason for a delayed return to play. However, players who have had newly apparent cognitive and memory impairments and/or fatigue at first follow-up visit also account for a large number of the players in the study cohort, even though these signs and symptoms resolve within the next few days. Worsening of symptoms and signs over the first day suggests that the affected players had sustained an MTBI on the more severe end of the spectrum.

General Discussion

The results of the present analysis stand in contrast to those presented in an earlier study of concussions in college football players (23). The present study shows a statistically significant increase in the absolute number of signs and symptoms as well as in the frequency of many specific signs and symptoms, such as photophobia, fatigue, disorientation to time, anterograde amnesia, and cognitive and memory impairments on initial evaluation in patients who ultimately will miss 7+ days of play. The study of college players reported that those with a "moderate to severe concussion" had a lower frequency of all these symptoms compared with the overall group of players with MTBI (23). There are a number of plausible explanations for these different results. The two studies used different means of collecting data. The college study collected data on the symptoms from a standardized symptom checklist filled out by the athletic trainer on the basis of the athletes' self-rated reports on the presence and severity of 17 symptoms.

In contrast, the present NFL study collected data through physician reports of their history and physical examination findings on the athletes. Furthermore, the two studies differed in the definition of moderate to severe concussion. The college study classified players into this category by grading them using criteria in one of the standardized grading scales. In contrast, this present NFL study used the criteria of being held out of play for 7+ days (a functional impairment) for inclusion in the study group. The inadequacies of using standardized scales to grade concussion severity are illustrated by the inability to correlate patients' clinical picture (at least their signs and symptoms) with their group classification. Conversely, the strength of using a functional approach to classifying concussion is confirmed by the strong correlation with the clinical picture seen in this present study.

The data presented in this article allow for the development of profiles of two groups of concussed NFL patients. The first group is the small minority of players who ultimately do not return to play for 7+ days after injury. Members of this group are more likely to be quarterbacks, defensive backs, wide receivers, and special teams return players injured in high-speed, high-acceleration collisions often occurring during passing plays or kick returns. They are more likely to experience loss of consciousness as a result of the head injury. The significance of their injuries is usually quickly recognized on the field and sidelines by team physicians and athletic trainers, who therefore only rarely allow them to return to play on the day of the injury. They are more likely to be hospitalized on the day of the injury. At the time of initial evaluation, these players complain of and exhibit a multitude of postconcussion signs and symptoms and have a significantly increased number of signs and symptoms compared with the other group. They are likely to complain of migraine-type headaches with photophobia, fatigue, and perhaps sleep disturbance. On initial examination, they are very likely to be disoriented to time and to have retrograde amnesia, difficulties with immediate recall, and overall difficulties with cognition and general memory.

It must be emphasized that the cognitive and memory impairments were noted on clinical mental status testing and that the results of this study do not include results of neuropsychological evaluations. The day after the injury, these players are more likely to exhibit fatigue and overall memory disturbances. There is no evidence, however, that when they do return to play, these players are at increased risk of sustaining another MTBI. The <7 days out group includes the large majority of NFL players who return to play in less than 1 week after MTBI. These players are more likely to be running backs than players at other positions, and, if not running backs, more likely to be linemen or linebackers. Their injuries are more likely to occur on running plays with presumably lower-speed and -acceleration impacts. They are less likely to sustain loss of consciousness as a result of the injury. They are much more likely to be cleared by medical personnel to return to play on the same day of the injury. They are less likely to be hospitalized on the day of the injury. On initial evaluation, they exhibit a significantly lower total number of signs and symptoms than their counterparts. Although they also frequently complain of headaches, the headaches are more often nonmigrainous in nature, without associated photophobia. These players are much less likely to experience fatigue and sleep disturbance. On examination, they are much less likely to exhibit disorientation, difficulties with memory, or cognitive impairments. The above information should be of great value to clinicians who treat athletes after MTBI. By focusing on the factors that distinguish between these two groups, physicians may be able to make an accurate prognostic evaluation soon after MTBI.

Key symptoms and signs that team physicians should be aware of include fatigue, photophobia, disorientation to time, and retrograde amnesia. The presence of any one of these symptoms and signs increases the risk of prolonged removal from play. Loss of consciousness for 1 minute or more adds some additional predictive power, but it occurred only rarely in the NFL and infrequently (9.7%) in players out 7+ days. However, the slight increase in sensitivity with it was offset by a decrease in specificity.

There are different ways to interpret these findings. Players who miss 7+ days may do so because their MTBI was on the more severe end of the spectrum of concussion. The recovery is delayed because they sustained a more severe brain injury. The statistically significantly increased absolute number of symptoms and signs and the increased frequency of many specific postconcussion symptoms and signs certainly support this position. The fact that team medical personnel were able to quickly determine that these players should not return to play on the day of the injury again points to the increased severity of the injury. The finding that certain player positions are predisposed to miss 7+ days and that these players' positions are those most susceptible to the highest-velocity acceleration impacts also supports the view that these players sustained more severe MTBIs than their counterparts. The data showing the time course for resolution of symptoms also indicate that these players had symptoms for at least 3 to 5 days after injury, which suggests that they could not be allowed to return to play until at least 6 to 7 days.

However, some of the data are also consistent with a different viewpoint, namely, that team physicians took an overly conservative and cautious approach to players who they thought were more severely injured and thus were kept out longer even though some of the players might have been able to return to play sooner. The data on time course of resolution of symptoms argue somewhat against this position. It is likely that there are elements of both of these viewpoints at work in trying to correctly interpret the data from this study and apply them to management of MTBIs.

In addition to the recorded signs and symptoms from the database, physicians on the MTBI Committee have a consistent impression of players who will experience a lengthy recovery. These impressions appear on the first or second day after concussion. Players who develop prolonged postconcussion syndrome often complain of "feeling hung over" and/or of trying to "look out past a fog." These feelings often linger after their other complaints have abated. In other cases, the player reports a feeling of fatigue, blurred vision, or dizziness, and the treating physician notes a change in personality. Players often complain of migraine-type symptoms, including pounding, throbbing headaches with nausea and photophobia. These feelings are common and tend to persist in players eventually out 7+ days. In

other cases, the player complains of "just not feeling right," not feeling ready to work out or even practice with a no-contact shirt on and "feeling queasy." In some cases, conditioning exercises increase the player's symptoms.

Concussion Evaluation and Management Guidelines

The results of this present study and previous studies from the MTBI Committee (36, 37) prompted the authors to critically analyze the widely promoted guidelines for the evaluation and management of concussion in sport. It must be emphasized that this critique arises from data obtained exclusively from professional football. This 6-year study indicates that no NFL player experienced the second-impact syndrome or cumulative chronic encephalopathy from repeat concussions. While the study did not follow players who left the NFL, the experience of the authors is that no NFL player has experienced these injuries. This finding may lead to future research aimed at challenging two of the expressed rationales for developing management guidelines to prevent the second-impact syndrome and cumulative chronic brain injury from repeat concussions.

The most widely used guidelines propose that concussion severity be graded by use of a limited number of criteria, such as presence or absence of loss of consciousness and posttraumatic amnesia at the time of injury, presence or absence of confusion and other postconcussion symptoms, and presence or absence of mental status changes very soon after injury (1, 9, 11, 12, 31, 40). The guidelines then make clinical management recommendations on the basis of the grade of concussion diagnosed by these criteria. The proponents of these guidelines clearly believe that the grade of concussion severity is linked to prognosis after MTBI. They also assume that delaying return to play for a prolonged time interval after injury can somehow prevent or alter poor outcomes, repeat injury, or catastrophic brain injury.

The results of this NFL study confirm that loss of consciousness and posttraumatic confusion and/or cognitive or memory impairments are predictors of longer recovery after MTBI than a few days. However, the data also reveal that there are other prognostic factors of equal importance that are not included in the grading systems. These include photophobia, fatigue, and increased absolute numbers of signs and symptoms. Furthermore, the grading systems do not consider factors such as the position played by the injured athlete and the type of play during which the injury occurred, both of which have been demonstrated in this study to have prognostic value. This NFL study also found that the presence of signs and symptoms such as fatigue, sleep disturbance, irritability, and/or cognitive or memory impairments on examination the day after injury also has significant prognostic usefulness; yet, none of the grading systems incorporate any results from examinations other than on the day of injury. Although the grading systems use some important prognostic findings, they are limited in their scope and fail to incorporate a number of other factors that have been demonstrated to be predictors of delayed recovery.

The present study also suggests that grading concussions immediately after injury is prone to error. A number of players with signs and symptoms suggesting a poor prognosis in fact recovered very quickly and returned to play on the day of or within a few days of injury. Conversely, there were two players with minimal signs or symptoms suggesting good prognosis who ultimately were kept out of play for 7+ days after MTBI. None of the prognostic factors or combination of factors uncovered by this present analysis was 100% accurate in predicting delayed recovery. The presence of any of the five signs or symptoms (loss of consciousness for 1 min or more, fatigue, photophobia, not oriented to time, or retrograde amnesia) correctly identified 72% of the players with delayed recovery. However, it also identified 216 players who did not have a delayed recovery. Using these signs and symptoms as a prognostic tool would mean that only 19% of those identified would actually have delayed recovery. The authors believe that, if one insists on grading concussion severity, the best way is retrospectively, on the basis of how long it actually takes the player to become asymptomatic, with a normal neurological examination.

It follows from this analysis that the current attempts to link prospective concussion grading symptoms to arbitrary rigid management decisions are not consistent with the scientific data. For example, current guidelines indicate that all players who sustain loss of consciousness should be removed from play for at least 7 days. The present data show that although loss of consciousness is one factor related to prognosis, it is certainly not the only factor, and most players have an early return to play with loss of consciousness. Photophobia, fatigue, sleep disturbance, and cognitive and/or memory impairments and/or disorientation have also been shown to be predictors of delayed recovery. There were many players in this NFL study who sustained an MTBI with loss of consciousness and subsequently returned to play on the day of the injury or within 1 to 2 days after injury without any cases of adverse outcome. Current guidelines also indicate that players who sustain three MTBIs in a single season should be removed from play for the remainder of that season. There were only two players who experienced three concussions in the same season, so this aspect of the guidelines cannot be addressed with the NFL data.

The authors of the guidelines must believe that there is some increased risk associated with three MTBIs in one season that requires removal from play for the remainder of the season. Certainly, it is reasonable for physicians to take a cautious approach to such clinical situations, but there are no data from any of the NFL studies indicating that there is any difference between a second MTBI and a third, or between three MTBIs in one season and three MTBIs occurring over a few seasons. Thus, there are no data to support that guideline recommendation.

One of the prominent guidelines (1) states that players who have symptoms and/or signs for more than 15 minutes after MTBI should be removed from further play that day and not be allowed to return until at least the next game (7 d later in NFL football). The 15-minute threshold guideline is inconsistent with the results of the NFL study. Many players (35.9% of all NFL MTBIs, *Table 7*) who were symptomatic shortly after injury made full recoveries within 24 to 48 hours and returned to full practice when they had recovered without incident. The guideline recommendation is therefore inconsistent with the natural history of MTBI in the NFL. The presence of multiple postconcussion

symptoms and/or mental status changes and/or specific symptoms such as fatigue and photophobia at the time of initial evaluation is linked to delayed return to play not because of the imposition of arbitrary restrictions but rather because the athletes who exhibit these signs and symptoms are more likely to have persistent symptoms and signs for 3 to 5 days after injury.

It is apparent that the guidelines have strengths and weaknesses. Although their aim is to assist the treating physician in making clinical management decisions on concussed athletes, the rationale for their development seems not to be based on sufficient scientific evidence about patient recovery and outcome. The grading criteria and their linkage to management recommendations are not consistent with the current NFL data. The guidelines focus on some findings that are not clinically relevant and ignore other factors that are. Most physicians would agree that players should not return to play until they are asymptomatic, with normal neurological and mental status examinations. There is no evidence from the NFL data that keeping players out of play for longer arbitrarily determined time periods results in any improved outcomes. The only way to determine when injured players are asymptomatic with normal clinical findings is for physicians to do what they are trained to do: to take histories and examine their patients as part of regular follow-up visits after injury. One cannot make these determinations by relying solely on rigid and possibly inappropriate guidelines for professional football players. The currently available guidelines therefore should not be used in the evaluation and management of MTBI in professional football until a thorough refinement is made to bring consistency with the NFL data. It is the recommendation of the authors and the MTBI Committee that team physicians continue to manage their players on an individual case-by-case basis, using their best clinical judgment based on the most relevant, objective medical data obtained from this and other studies.

Limitations

These limitations follow those described in Pellman et al. (37). The MTBI Committee did not collect retrospective data on previous concussion history as part of the study. Some of the players may have had previous concussions either in the NFL in the years before the study began or during their playing careers in high school, college, or other levels of football. It is also possible that some of the players sustained cerebral concussions at earlier times in their lives in nonfootball athletic or nonathletic endeavors. Previous concussion history may affect our conclusions regarding repeat concussions because a certain number of the concussions that we labeled as initial concussions may in fact have been repeat concussions for some players.

The authors also realize that some MTBIs were not reported by the affected player to team medical personnel and therefore were not included in this database. Such unreported injuries most likely were very mild in nature and associated with rapid recovery to escape detection by very involved NFL athletic trainers and physicians. There was also difficulty collecting data on loss of consciousness. The initial data collection sheet did not ask for data regarding loss of consciousness. Once this was corrected, we found that many of the reports that were submitted did not answer the question in the loss-of-consciousness part of the form; therefore, we do not have definitive loss-of-consciousness data on a certain number of players. What has been reported are the cases with a known time of unconsciousness and those cases reporting a zero or no loss of consciousness.

It is also important to note that in a multisite study such as this, there are numerous different examiners. In some cases, different examiners from a given medical staff may evaluate that team's players. There was no uniform method of evaluation of concussion in this study, which will give rise to variability in assessments among the 30 teams and, on occasion, within the same team. It must be emphasized that players were not cleared to return to play until they were asymptomatic, with normal medical examinations, and able to return to full, unrestricted participation in a team practice or game. There were some players who returned with headaches.

Many of the players in the database had neuropsychological testing at baseline and/or after MTBI. The results of the neuropsychological test batteries are not in this database and therefore not part of this study. It is possible that including the results of the neuropsychological testing on the players will provide more information on the injury and recovery.

There are a number of possible explanations for the absence of second-impact syndrome in NFL players. Most obvious is the small sample size versus the expected incidence rate. The incidence of second-impact syndrome in high school and college football is 1 to 2 in 1,500,000 players. Thus, one would need 375 to 750 years to expect to see a case of second-impact syndrome, assuming 2000 players involved per year.

Although the medical condition of the player is certainly the most important factor in determining return to play by team physicians, many other factors go into the decision of when the player should return to play. The importance of the player to the team; the importance of the upcoming game to the team; and pressure from owners, players and their families, coaches, agents, and media certainly may influence the final decision of when the player returns to play. The authors believe, however, that the medical factors regarding the patient's recovery are and should be the overriding facts that guide the team physicians' decision-making on return to play. Furthermore, our results apply to NFL-level players, and extrapolation to younger athletes has not been demonstrated. It is clear that differences may exist between MTBI in high school and professional athletes.

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Acknowledgments

The NFL's MTBI Committee is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society, NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the area of traumatic brain injury, biomechanics, basic science research, and epidemiology. None of the committee members have a financial or business relationship posing a conflict of interest to the research conducted on concussion in professional football. The committee involved Cynthia Arfken, Ph.D., at Wayne State University to assist in the research and provide epidemiological consultation on the statistical analysis of the concussion data. She is an associate for the study but not a committee member. Funding for this research was provided by the NFL and NFL Charities. The Charities is funded by the NFL Players' Association and League. Their support and encouragement to conduct research on concussion is greatly appreciated.

COMMENTS

In Part 5 of the National Football League (NFL) Committee on Mild Traumatic Brain Injury (MTBI) study, the authors have studied those players with greater injury and extended recovery times lasting 7 days or more after the traumatic event. They analyzed 72 concussions that represented 8.1% of athletes injured in the NFL during the years 1996 to 2001. The data were collected by team physicians who completed evaluation forms on the injured players. Several interesting aspects emerged from the

study. First, it affirms the widespread observation by previous researchers that those players injured are usually involved with high-speed collisions, such as defensive secondary, kicking unit, quarterbacks, and wide receivers. These players had more signs and symptoms compared with players who missed less playing time, including the symptoms of headaches, dizziness, fatigue, photophobia, and amnesia. Between the initial and subsequent examinations, they found trends indicating diminishment in symptoms with the exception of persistent memory disturbance, fatigue, irritability, and insomnia. In their series, only one-fifth of this prolonged-injury group required removal from participation for more than 2 weeks. They thought that their players experiencing a lengthy recovery were often discerned early after their brain injury with feelings of being "hung over," also with fatigue, blurred vision, dizziness, or a change in personality noted.

Although the above findings are noteworthy and of interest to all those participating in the care of contact or collision athletes, several aspects bear discussion. First, as the authors acknowledge, this represents a group of highly conditioned, motivated, financially incentivized, and skilled athletes. At this level of play, they have most likely already undergone an intensive selection process for various characteristics, some of them germane to the phenomenon of MTBI. This study does not use any ancillary testing, whether it be routine neuroradiological studies, neuropsychological testing, balance testing, or formal neurological evaluation. In addition, no studies of cerebral metabolism were performed. Numerous researchers have previously demonstrated that the reporting of symptoms and the clinical signs on physical examination may not be reliable indicators of ongoing cerebral metabolic function. Capturing the true incidence of MTBI has always been and continues to be difficult in football players. A recent study of professional athletes in the Canadian Football League has shown that 45% sustained a concussion during a single season, whereas only one of five (19%) realized that cerebral injury had occurred (1). There is also evidence that MTBI is more common in football than previously estimated, with the incidence in high school athletes being reported to be as high as 47% annually (3, 4). Another study in active professional football players showed that those older players who possessed the *APOE4* allele had poorer performance on cognitive assessment than players who did not have this allele or who were younger with any genotype (2). Thus, MTBI is a complex clinical issue in contact athletes that is still incompletely understood.

The authors state that their data refute two of the expressed rationales for development of guidelines, that is, to prevent the second-impact syndrome and to avoid cumulative brain injury from repeat concussions. The former would not be expected in this age group and in a relatively small number of exposures, and the latter could not be assessed without long-term studies, including after retirement. They go on to criticize currently accepted concussion management guidelines that use only a limited number of criteria as opposed to their series with findings of multiple symptoms in concussed players with prolonged loss of playing time.

However, the conventional guidelines have been promoted in great part for usefulness and brevity in the recognition and diagnosis of an athlete with MTBI. Certainly, no clinician would base the ultimate management solely on an initial examination and not consider subsequent clinical data relevant. Although the playing position of the injured athlete is of interest, those regularly treating patients with MTBIs do not consider the human brain to react, recover, and have a propensity for cumulative effects on the basis of playing position per se. Current management guidelines have been developed on the basis of the largest number of exposed athletes, developed primarily for the amateur or scholar contact athlete, and therefore a more conservative position and management scheme has always been known to be better for recovery of cerebral function. If there is any doubt, most authorities and neurological specialists have not felt a great rush to return a player several days earlier to exposure to repetitive brain impacts. The authors state that they cannot make clinical return-to-play decisions by using rigid and possibly inappropriate guidelines in NFL players, and current clinical guidelines should not be used in the evaluation and management of MTBI in professional football. We eagerly await results and publications of findings in their players, including long-term outcomes, for further elucidation concerning proper head-injury management.

Julian E. Bailes
Morgantown, West Virginia

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This is the fifth article emanating from the NFL Committee on MTBI, which analyzed 887 concussions occurring during a 6-year period from 1996 to 2001 in practice and games in NFL athletes. The purpose of this study is to compare and analyze concussions in 68 players who did not return to play for 7+ days after an MTBI.

The wealth of data is analyzed and subsequently extrapolated to management guidelines in the NFL. It is no surprise that quarterbacks, defensive backs, and wide receivers have the highest incidence of 7+ days out. Similarly, most concussions occur among the defensive secondary, the kick units, and then the quarterback, which appears to be the most susceptible position for MTBI. The greatest incidence of concussions occurred during passing plays. The most significant symptoms include headache, dizziness, problems with immediate recall, and retrograde amnesia. Headaches are the primary symptom resulting in delay of return greater than 7 days. The authors emphasize that fatigue, photophobia, disorientation to time, and retrograde amnesia are the most frequent symptoms and signs of a delayed nature.

Unfortunately, neuropsychological test data were not used in this analysis, but the results confirm what previously has

been documented by such tests: the great majority of athletes with MTBI quickly recover from their symptoms. In fact, in this study, athletes out for 7+ days included only 8% of all MTBIs in the NFL. Furthermore, 80% of these athletes returned to play within 14 days, so only 1.6% of concussions resulted in a prolonged postconcussion syndrome.

Significant negative findings include the absence of any identifiable cases of second-impact syndrome and also the failure, in this study, to document cumulative brain damage from multiple concussions. To further substantiate this latter observation, I believe neuropsychological test data would need to be included in future prospective studies.

Most pertinent is the authors' discussion of existing management guidelines for MTBI. These have been promulgated primarily to avoid repeat and/or cumulative chronic brain damage from reinjury. Arbitrary criteria including loss of consciousness, mental status changes, and various symptoms associated with postconcussion syndrome are used. It is now generally appreciated that loss of consciousness, previously thought to be the most significant prognosticating factor of subsequent brain injury, may have minimal long-term consequences, and yet a seemingly minor head injury with associated photophobia, fatigue, and amnesia can lead to a prolonged postconcussion syndrome. The authors make four points that support neuropsychological testing as the standard of care to replace the guidelines presently being used: 1) none of the grading systems incorporate any results from examinations other than on the day of injury; 2) the present study suggests that grading concussions immediately after injury is prone to error; 3) current attempts to link prospective concussion grading systems to arbitrary rigid management decisions are not consistent with the scientific data; and 4) the management guidelines are too simplistic and too rigid for the NFL. Serial neuropsychological testing is the only way, presently, to immediately and longitudinally document the symptoms of postconcussion syndrome, avoid grading concussions immediately after injury, provide objective scientific data for management decisions, and avoid the simplicity and rigidity of current guidelines. Indeed, the authors state that, "the currently available guidelines therefore should not be used in the evaluation and management of MTBI in professional football . . . ." For this reason, the majority of NFL teams have evolved to using neuropsychological test batteries as the primary instrument for making return-to-play decisions. The authors have presented a superb prospective study, which brings new information concerning MTBI in athletics.

Joseph C. Maroon
Pittsburgh, Pennsylvania

With all contact sports, especially American football, we now are recognizing MTBI as a serious phenomenon with potential long-term side effects to our athletes. The study by Pellman et al. demonstrates the significance of these consequences to athletes who sustain an MTBI. The purpose of this study and its goals are well stated. The data collected are excellent and need to be published. The authors' points could be made with less peripheral text, but otherwise it is extremely valuable information. Dr. Pellman, and the NFL MTBI Committee collectively, are to be commended for the work they have done and continue to do. From these data, we can establish guidelines for returning our athletes safely after they have sustained an MTBI. It is imperative that this type of research continue, for it directly affects how we can provide better care for our athletes.

Michael A. Colello
Head Athletic Trainer
Cleveland Browns
Cleveland, Ohio

Concussion management in sports is a hot topic right now. The National Athletic Trainers Association will release a position statement on concussion management in the summer of 2005. At the recent PAC 10 Student Athlete Health Conference, the topic of computerized neuropsychological testing was discussed. All 10 schools plan to use Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) or Automated Neuropsychological Assessment Metrics (ANAM) to assess baseline and evaluate postconcussion under their protocol in the upcoming football season.

I appreciate that *Neurosurgery* has provided a forum to evaluate and debate the series of NFL concussion articles. In Part 5, Pellman et al. investigate and differentiate the concussed athletes who were held out of participation for more than 7 days versus those who returned in less than 7 days. Although this particular study design was limited to NFL players in a 6-year period, there were interesting findings with regard to the most vulnerable positions, types of plays in football, correlating symptoms, and how these concussions were managed. The authors challenge concussion grading scales and management recommendations.

In my experience at the University of Southern California, we have a team approach to evaluation and management of concussion. We use the Standard Assessment of Concussion (SAC), the Balance Error Scoring System (BESS), the Post Concussion Symptom Scale (PCSS), and ANAM for both baseline and postconcussion evaluation. After injury, a sideline evaluation is performed. Depending on the signs and symptoms and any change of direction in which the signs and symptoms are heading, emergency medical services may be used or an athlete may be admitted to the hospital for observation. Team physicians may order diagnostic studies to further assess injury. Concussion grading scales and management recommendations are merely reference guidelines. Before we clear an athlete to return to play, even after he becomes asymptomatic, we consider his physical stature and style of play and discuss the importance of honesty in reporting symptoms. The "bottom line" is that, after all of the baseline testing, sideline evaluation, postconcussion evaluations, results from diagnostic studies, opinions from other team physicians, staff athletic trainers, and coaches, and references to acceptable medical guidelines are accounted for, the

final decision rests with the clinical judgment of our team neurosurgeon.

Russ Romano
Director of Sports Medicine, Head Athletic Trainer
University of Southern California
Los Angeles, California

As I commented in Part 4 of this series of articles, I believe that the NFL's MTBI Committee's original articles on *impact biomechanics* and concussive injuries made significant contributions to the literature on sport concussion. However, I have some of the same concerns with the current article as I had with Part 4. The current study (Part 5) on "Concussion in Professional Football: Injuries Involving 7 or More Days Out" is flawed with respect to the study design and the interpretation of the findings. Although the authors outline the study's limitations, they unfortunately make conclusions that are very suspect given the potential impact of the limitations on the findings.

The authors' choice of "days lost" to categorize concussed players, rather than *symptom duration* and *symptom severity*, complicates the interpretation of the findings. This is especially problematic since the authors state in Part 4 that "players were not cleared to return to play until they were asymptomatic, with normal medical examinations, although some players may return with headaches," and again, in Part 5, that "NFL team physicians clear a player for return to play only after he becomes asymptomatic (with the exception of a mild headache) and has a normal neurological examination." Sending the message that it is acceptable to return players while still symptomatic with a headache—regardless of whether or not the clinician thinks the headache is related to the concussion—is the wrong message to send. The authors' findings do not necessarily support their conclusions that "they [concussed NFL players] recovered from symptoms and had a consistent return to play in the NFL."

The authors go on to state essentially that concussion grading scales and return-to-play guidelines are not useful to the NFL team physician and that they should "not make these determinations by relying solely on rigid and possibly inappropriate guidelines for professional football players." I definitely agree with the authors' opinion that more variables should be considered than loss of consciousness and amnesia and that each athlete should be evaluated and managed on an individual basis. But I disagree with their accompanying opinion that there is no difference between a first, second, and third concussion. This is where the concussion grading scales become even more valuable.

First, there are sufficient data in the literature to support the notion that athletes with repeat injuries respond differently than those with initial injuries and that previous concussive injuries likely predispose players to future injuries (2–5). Interestingly, the authors of the current study did not even factor concussion history (before the study period) into their analyses. Secondly, within-season repeat concussions have been found to occur 75% of the time within 7 days of the first injury (3). These findings would seem to lend some credence to the proposed concussion guidelines that recommend a 7-day asymptomatic waiting period after an initial concussion.

Most people would agree that concussions should be managed on an individual basis. However, to suggest that the proposed concussion grading scales—especially those, such as the revised Cantu guidelines (1), that consider symptom duration—should be ignored is again sending the wrong message. The professional football players in the present study are not very different from the players studied at the collegiate level. Is this not where they came from? Hopefully, future NFL studies based on more comprehensive clinical evaluations of symptom duration and neuropsychological function will provide team physicians and athletic trainers with better guidance for making return-to-play decisions. These studies should consider both the acute and chronic effects of recurrent concussion in NFL players.

Kevin M. Guskiewicz
Director, Sports Medicine Research Laboratory
University of North Carolina
Chapel Hill, North Carolina

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TAB 1F

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CONCUSSION IN PROFESSIONAL FOOTBALL: NEUROPSYCHOLOGICAL TESTING—PART 6

Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, and ProHEALTH Care Associates, LLP, Lake Success, New York

Mark R. Lovell, Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Department of Orthopaedics and Sports Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

David C. Viano, Dr. med., Ph.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park; Department of Neurology, New York University Medical Center, New York; and Department of Neurology, Albert Einstein College of Medicine, Bronx, New York

Andrew M. Tucker, M.D.
Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and University of Maryland School of Medicine, Timonium, Maryland

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952. Email: dviano@comcast.net

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OBJECTIVE: The National Football League (NFL) neuropsychological testing program is reviewed, and neuropsychological test data are presented on various samples of NFL athletes who sustained concussion (mild traumatic brain injury, MTBI).

METHODS: This study evaluated post-MTBI neuropsychological testing of NFL players from 1996 to 2001. All athletes completed a standardized battery of neuropsychological tests and underwent postinjury neuropsychological testing within a few days after concussion. Test scores were compared with baselines using analysis of variance for athletes having on-field memory dysfunction, three or more concussions, or 7+ days out from practice and play.

RESULTS: The MTBI group did not display significant neuropsychological dysfunction relative to baseline scores within a few days of injury. However, a subsample of the injured athletes who displayed on-field memory dysfunction performed significantly more poorly on two of the memory tests. The neuropsychological test results of a group of athletes with a history of three or more MTBIs did not differ significantly compared with a group who had fewer than three concussions or compared with league-wide normative data. The neuropsychological performance of athletes who were out from full participation 7+ days was not significantly different from the group who returned to play within 7 days or the norms.

CONCLUSION: Neuropsychological testing is used within the overall medical evaluation and care of NFL athletes. Players who experience MTBI generally demonstrate rapid recovery of neuropsychological performance, although poorer neuropsychological test results were related to on-field memory dysfunction. NFL players did not demonstrate evidence of neurocognitive decline after multiple (three or more) MTBIs or in those players out 7+ days. The data show that MTBI in this population is characterized by a rapid return of neuropsychological function in the days after injury.

KEY WORDS: Concussion, Neuropsychological testing, Sport injury, Traumatic brain injury

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Over the past decade, mild traumatic brain injury (MTBI) has become a major area of interest in sports medicine. Interest in sports-related MTBI has led to programs aimed at improving both the diagnosis and clinical management of athletes. As part of this overall effort, neuropsychological testing is being used to aid in the evaluation and management of players with MTBI in the National Football League (NFL). The development and use of neuropsychological testing in the NFL has been rapid and has contributed to the implementation of neuropsychological testing in other professional sports, including ice hockey (25), automobile racing (31), and Australian Rules football (17). When used in concert with other medical information, neuropsychological test data contribute quantitative information regarding neurocognitive processes, such as attentional, memory, and cognitive processing speed. Neuropsychological testing provides information about changes in cognitive processes that may be missed through sideline or on-field examinations of the athlete and also provides objective information regarding the recovery process during the acute recovery period (15). The testing process also compares the athlete's performance against established normative data and the individual athlete's preinjury level of performance.

The program developed for the NFL has influenced the development of neuropsychological testing at the high school (3, 14, 27) and college levels (10, 26, 30). In published studies that have used the NFL test battery, Collins et al. (10) found that mild cognitive impairment in a group of collegiate athletes generally resolved within a week. More recently, McCrea et al. (30) found neuropsychological deficits in college athletes that were evident at 2 days after injury but dissipated within 7 days. Echemendia et al. (12) documented a decline in neuropsychological functioning 2 hours after MTBI and 1 week after injury. Working with high school athletes and using a computer-based test battery, Lovell et al. (27, 28) have found impairments in memory processes and elevated symptoms in concussed athletes relative to baseline test results. These deficits resolved within 1 week of injury.

Recent neuropsychological studies of nonprofessional athletes have demonstrated a strong relationship between on-field markers of MTBI, such as amnesia and specific neuropsychological tests of memory administered days after injury. Collins et al. (11) found that the presence of anterograde and retrograde amnesia was linked to poor outcome as determined by neuropsychological testing and symptoms (using the ImPACT computer-based neuropsychological test battery). Lovell et al. (27, 28) linked poor neurocognitive outcome as measured by the ImPACT battery to on-field memory impairment. Finally, McCrea et al. (30) found that on-field disruption of memory was related to severity of injury. The association of on-field markers of memory dysfunction with formal neuropsychological test results is predictable, because MTBI is known to disrupt the memory system (40, 43, 45). These initial studies emphasized the importance of conducting a careful evaluation of the neurocognitive status of athletes immediately after injury. However, no such study has been completed with professional football players. This is an important issue, because NFL athletes represent a cohort distinctly different from high school and collegiate athletes in age and skill level. Generalization from the results of studies with younger athletes may lead to inaccurate and misleading expectations regarding the severity of injury and expected pattern of recovery after MTBI in professional athletes.

In a series of additional studies of collegiate athletes, Lovell and Collins (26) and Collins et al. (10) documented memory impairment and a decline in speed of information processing on the Symbol Digit Modalities and Trail Making Tests, which resolved within 5 to 7 days after injury. Working with professional rugby athletes in Australia, Hinton-Bayre et al. (17) found deficits in speed of information processing, with a return to baseline levels of performance within 1 to 2 weeks of injury. More recently, Field et al. (14) have documented impairments in memory processes in high school and collegiate athletes with MTBIs using the Hopkins Verbal Learning Test (HVLT) and the Brief Visuospatial Memory Test–Revised (BVMT-R).

The issue of whether or not the effects of sports-related MTBIs are cumulative has also been an important and highly controversial area of study. Some authors have suggested that multiple MTBIs result in discernible decrements on neuropsychological testing in high school (11) and collegiate athletes (10, 16). Other studies have not supported this finding (12). This issue has yet to be investigated with neuropsychological testing in professional football athletes.

This article describes the neuropsychology program in the NFL from 1996 to 2001. It also investigates neuropsychological test performance during the initial follow-up evaluation within several days of concussion. The article addresses the following questions. 1) Do professional football players display neuropsychological dysfunction in the days after their injury? 2) Is neuropsychological dysfunction related to on-field diagnosis of injury by team medical staff? 3) Do athletes with three or more MTBIs in the study period display greater neuropsychological dysfunction than professional football athletes who have not had three or more MTBIs? and 4) Do athletes who miss 7 or more (7+) days after MTBI perform more poorly on neuropsychological testing than those who return to play earlier?

THE USE OF NEUROPSYCHOLOGICAL TESTING IN PROFESSIONAL FOOTBALL: HISTORICAL ROOTS

The use of neuropsychological assessment procedures in sport is a recent phenomenon. The first large-scale study of MTBI in football players was performed at the college level and involved the cooperative efforts of the University of Virginia, the Ivy League schools, and the University of Pittsburgh (4). The University of Virginia study was conceptualized primarily as a research study, and data were not initially used to make clinical return-to-play decisions (4). However, these efforts helped to establish a model for neuropsychological assessment that was later adapted for clinical use.

In an effort to improve the overall care of professional football athletes, a neuropsychological evaluation program was instituted by the Pittsburgh Steelers in the early 1990s with the active participation of the Steelers management and medical/athletic training staff (23, 24, 29). This represented the first clinically oriented project in a professional sport to use neuropsychological testing to assist team medical personnel in making return-to-play decisions after concussion. The Steelers' program involved the baseline evaluation of each athlete before the season. This provided a basis for comparisons when an injury occurred during the season. Testing was then repeated within 24 to 48 hours after head injury and again before the player's return to contact.

During the 1993 to 1994 seasons, the Steelers project involved the baseline evaluation of approximately 60 NFL athletes who volunteered to undergo preseason testing. The initial two seasons used neuropsychological testing to assist in determining player readiness to return to play after concussion. Postinjury neuropsychological testing results were compared with preseason baseline results, and this information, in addition to player symptoms, was used by the team medical

staff to make return-to-play decisions. The project expanded to other athletes on the Steelers' roster after the 1994 season, and testing was used effectively to evaluate a number of injured athletes during that season.

In 1994, the NFL Committee on MTBI was created with the support of the Commissioner, Paul Tagliabue. The committee was formed and chaired by Dr. Elliot Pellman of the New York Jets (32). The scientific activities of the NFL MTBI committee have been described in publications by Pellman et al. (33, 35–37), and the committee has overseen multiple projects in the NFL designed to better understand and manage concussion. In addition to supporting the neuropsychology program discussed in this article, the committee has spearheaded research on the epidemiology of MTBI, the biomechanical forces involved in concussion, and studies designed to evaluate current helmet safety testing standards (36, 37). The most recent studies involve the epidemiology of players with three or more concussions and those out from play 7+ days (33–35).

GOALS OF THE NFL NEUROPSYCHOLOGY PROGRAM

The NFL neuropsychological testing program was established as a clinical research program with the goal of investigating the use of neuropsychological testing in assisting team physicians in the return-to-play decision. The program was designed to assist in the gathering of neuropsychological outcome data in the broader context of the MTBI committee's epidemiological and biomechanics projects. Initial funding for the project was provided through a grant from NFL Charities in 1995. Participation in the project has not been mandatory but has been strongly encouraged throughout the league by the MTBI committee, the NFL Injury Panel, and the Commissioner's office. Since the inception of the neuropsychological testing program, participation has grown to include all NFL teams.

PATIENTS AND METHODS

Data Collection and Analyses

All participating subjects in this study were NFL athletes who underwent neuropsychological testing during the 1996 to 2001 seasons. Preseason normative data were collected on 655 NFL athletes. Although many participants in this study had undergone preseason baseline testing before their injury, athletes were also included who had completed only postinjury evaluations. The overall sample of injured players who underwent neuropsychological testing consisted of 143 athletes. This sample represents 22% of the 650 NFL athletes who experienced 887 concussions in the study period from 1996 to 2001 (33–35). Because participation in the neuropsychological study was voluntary, not all athletes with MTBI completed neuropsychological testing.

Within the group of 143 athletes with completed neuropsychological testing, four subsamples were subjected to statistical analyses depending on the specific research question being addressed and the availability of completed physician and neuropsychological test data, as follows.

The postconcussion performance of NFL players was compared with preseason baseline assessments in 95 athletes.

The neuropsychological test performance of a subsample of athletes with and without documented on-field amnesia was contrasted.

The potential role of multiple MTBIs on neuropsychological test performance was examined through a preliminary study in which a sample of injured NFL players, some of whom had experienced more than three concussions, were compared with a sample who had not experienced three concussions.

The neuropsychological test performance of athletes who lost 7+ days of playing time was compared with that of a sample of players who had returned to play within the week.

Neuropsychological testing was completed by trained neuropsychologists or by closely supervised psychology technicians or interns. The administration and test procedures were standardized and uniform from team to team. After data collection by individual team neuropsychologists, the data were stripped of identifying information in compliance with Health Insurance Portability and Accountability Act standards and stored at the University of Pittsburgh.

Procedures

The NFL neuropsychology program involves the participation of a network of neuropsychological consultants in each NFL city. The consultants complete preseason baseline testing and postinjury follow-up testing. Neuropsychological consultants were selected for this program on the basis of their expertise in dealing with MTBI. All neuropsychological consultants held Ph.D. degrees and were licensed in the state of their practice.

Before each testing session, the athletes were given a brief explanation of the purpose of neuropsychological testing. All athletes volunteered for the project, and no player was required to participate. Participating players represented a relatively homogeneous group. All were men 21 to 35 years old involved in all offensive and defensive positions.

After neuropsychological testing of the player with a reported concussion, the team physician discussed the results with the club's consulting neuropsychologist. The information from the testing was used in conjunction with the player's clinical status to determine return to play. The team physician has the ultimate responsibility for this decision. As mentioned in previous articles by Pellman et al. (33–35), the Committee on MTBI did not impose guidelines or recommendations on the club's medical staffs regarding concussion evaluation, testing, treatment, or return-to-play criteria.

The NFL Test Battery

Table 1 lists the neuropsychological tests that were used for this study. The test battery was based on those used with the Pittsburgh Steelers (23) and by a limited number of teams

TABLE 1. National Football League neuropsychological test battery (1996–2001)[a]

Series (ref. no.)	Ability evaluated	Test
Brandt, 1991 (7)	Memory for words (verbal memory), immediate recall	HVLT
Brandt, 1991 (7)	Delayed memory for words (20-min delay from presentation)	Delayed recall from HVLT
Benedict, 1997 (5)	Memory for designs (visual memory)	BVMT-R
Benedict, 1997 (5)	Delayed memory for designs (20-min delay from presentation)	Delayed recall from BVMT-R
Reitan, 1958 (38)	Visual scanning, mental flexibility	Trail Making Test
Smith, 1982 (41)		Symbol Digit Modalities
Benton and Hamsher, 1978 (6)	Word fluency, word retrieval	Controlled Oral Word Fluency
Wechsler, 1997 (44)	Attention span, forward and backward conditions	Digit Span

[a] HVLT, Hopkins Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised.

from 1993 to 1995. The battery was selected to assess the brain processes most often affected by MTBI. Some of these tests have been used with collegiate (4) and professional athletes (23). More recently, the test battery has undergone revision to include additional test instruments.

The NFL test battery evaluates multiple aspects of cognitive functioning, while being relatively brief. The entire battery takes approximately 30 minutes to administer. The battery is heavily oriented to the evaluation of attentional processes, visual scanning, and information processing, although it also evaluates visual memory, verbal memory, visual-motor coordination, and speech fluency. Past research in neuropsychology has identified these as the cognitive functions most likely to be affected by MTBI. For example, Barth et al. (4) described mild deficits in psychomotor speed in a large sample of collegiate athletes, with the majority of these difficulties resolving within 5 days of injury.

Each test in the NFL battery has been validated with brain-injured patients and has been administered according to standardized instructions to avoid variation in test results across testing sessions and teams. More details can be found in the individual test manuals and the review by Lezak (22), which includes supporting psychometric data. The tests are described briefly as follows.

The HVLT (7) consists of a 12-word list that is presented to the athlete on three consecutive trials. In its revised version, the athlete is assessed for recall after each presentation and again after a 20-minute delay period (maximum of 12 words). The HVLT total score represents the total words produced over the three trials (maximum of 36 words).

The BVMT-R (5) evaluates visual memory (e.g., memory for designs) and involves the presentation of six abstract spatial designs on three consecutive trials. As in the HVLT, the athlete's recall after each trial and his delayed recall are evaluated. The total score represents the total number of design elements recalled (maximum of 36), and the delayed recall condition represents the number of elements recalled after approximately 20 minutes (maximum of 12). Both the HVLT and the BVMT-R have six equivalent forms, which minimize practice effects and makes them ideal for use with athletes who are likely to undergo evaluation on multiple occasions throughout the course of their careers. The BVMT-R was added to the protocol in 1998 after its publication.

The Trail Making Test (38) consists of two parts and requires the athlete to use spatial scanning, speed, and cognitive flexibility skills. For this protocol, the time to complete Parts A and B were analyzed, and a lower score reflects better (faster) performance.

The Controlled Oral Word Association Test (COWAT) (6) requires the athlete to recall as many words as possible that begin with a given letter of the alphabet within a 60-second time period. This is completed for three separate letters and provides a measure of verbal fluency. A higher score reflects better performance.

The Digit Span (44) subtest represents a test of attention span in which the subject is required to repeat increasingly longer strings of numbers in both forward and backward order. A higher score represents better performance.

The Symbol Digit Modalities Test (SDMT) (41) requires the player to use visual scanning and processing speed to match a series of numbers and symbols while under time pressure. A higher score reflects better performance.

Wherever possible, tests were used that had multiple forms to avoid the improvement in test performance secondary to practice effects. Practice effects represent improvements in test performance as a result of previous exposure to the test and may cloud the interpretation of the recovery process. Past studies of athletes using tests that make up the NFL battery have demonstrated that the Trail Making Test, the SDMT, and the COWAT demonstrate significant practice effects. For instance, Lovell and Collins (26) reported mean improvements on the Trail Making Test, Part B, of 2 seconds in a sample of 40

nonconcussed college football players tested at the beginning and end of the season. Nearly identical scores were found for the HVLT at preseason and postseason. Working with uninjured high school athletes, Barr (3) reported an improvement of approximately 4 seconds on the Trail Making Test, Part B. No significant change was found on the HVLT. Both of these studies referenced above reported improvements of two words across two administrations for the COWAT test. To date, practice effects have not been studied in an NFL sample. Unfortunately, acceptable equivalent forms of these tests did not exist at the time of initiation of the study.

Timeline of the Neuropsychological Evaluations

After a reported MTBI, the initial neuropsychological evaluation of the athlete usually took place within 24 to 48 hours, although this varied somewhat depending on the readiness of the athlete to engage in cognitive exertion after injury. Even when athletes denied initial symptoms, a neuropsychological evaluation was recommended to evaluate subtle aspects of cognitive functioning, such as information processing speed and memory. If the athlete displayed cognitive deficits on testing or continued to exhibit postconcussive symptoms, a follow-up neuropsychological evaluation was recommended 5 to 7 days after injury. This time interval represents a useful and practical time span and is consistent with brain metabolism studies, which have demonstrated metabolic changes that persist several days after injury in animals (19) and humans (27).

Determination of abnormal test performance was made by the individual team neuropsychologist and was based on a comparison of postinjury test results with established normative data, which were provided to each team consultant by the program director (MRL). Abnormal performance was defined as performance that deviated negatively from the athlete's baseline performance. If baseline testing was not completed, league-wide normative values were used to aid in interpretation. Clinical interpretations were made by team neuropsychologists and not by the program director. If baseline testing had been completed, the player's preinjury performance was used as the basis for comparison. Ultimately, return-to-play decisions were made by team physicians after consultation with the team's athletic trainer, consulting neuropsychologist, and other personnel.

Statistics

Because of limitations in sample sizes, analysis of variance (ANOVA) models were performed using Statistica software (42). No adjustments were made for multiple comparisons.

RESULTS

Normative Values for NFL Players Test Performance

Baseline neuropsychological test data were gathered to determine average performance, or "norms," for NFL athletes. The data establish the normal range for performance in each test score in the battery and also provide the basis for comparisons if an athlete is injured during the season. Baseline evaluations were conducted at preseason, before contact.

Table 2 summarizes the baseline performance of NFL athletes. These results fall within expected limits for the general population and are similar to data on collegiate athletes (10). Data were collected on up to 655 active NFL athletes, depending on the neuropsychological test. The NFL players have a mean age of 25.4 years (range, 20–44 yr; median age, 24 yr). The college data represent a sample of 386 collegiate athletes with a mean age of 20.4 years (10).

Acute Effects of Concussion: Do Concussed Athletes Display Abnormal Neuropsychological Test Results Compared with Preseason Baseline Results?

The neuropsychological performance of a large sample of NFL athletes who had experienced MTBI was compared with the group's baseline performance by use of a series of repeated-measures ANOVAs. This addresses the question of whether professional football players experienced measurable neuropsychological decline after documented MTBI. Athletes completed neuropsychological testing an average of 1.4 days after injury (range, 1–10 d; standard deviation, 1.29 d). The average time from baseline to postinjury testing was 531 days (range, 4–2190 d). This wide range in time between baseline testing and postconcussion is characteristic of athletes and reflects the uncertainty of when each injury will occur. Although it is conceivable that an athlete tested days after injury

TABLE 2. Normative neuropsychological test data for National Football League athletes[a]

Test	No.	Mean (SD)	College sample
Hopkins VLT (Total)	653	26.0 (3.9)	24.6 (4.0)
Hopkins VLT (Delay)	630	9.0 (2.1)	7.8 (2.0)
BVMT-R (Total)[b]	479	27.4 (5.5)	
BVMT-R (Delay)[b]	478	10.4 (2.0)	
Trail Making Test, Part A	651	21.4 (7.4)	21.0 (5.9)
Trail Making Test, Part B	654	55.6 (17.1)	55.4 (17.3)
COWAT	655	41.9 (11.4)	37.5 (9.3)
Symbol Digit Modalities Test	155	56.6 (8.4)	56.8 (8.9)
Digit Span-Total	646	14.9 (4.0)	15.8 (3.9)

[a] VLT, Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised; COWAT, Controlled Oral Word Association Test; SD, standard deviation.
[b] The BVMT-R was not used in the study by Collins et al. (10).

might demonstrate a larger practice effect, the number of athletes who were retested within 2 weeks of baseline testing was quite small (n = 4) and therefore is not likely to have had a significant impact on the overall group data. Each neuropsychological test score was treated as a within-subjects factor. This allowed analysis of changes on each score over time. *Table 2* shows that most athletes had completed the HVLT, Trail Making Test, and COWAT, whereas fewer had completed the BVMT-R and SDMT because of changes in the test battery during the course of the study.

Table 3 shows that athletes with MTBI did not perform significantly more poorly on any of the individual tests that make up the NFL battery. In fact, the only significant differences between baseline and postinjury performance were on the Trail Making Test, Part A ($F = 4.97$, $P < 0.03$), the Digit Span Forward test ($F = 10.53$, $P < 0.001$), and the SDMT ($F = 8.13$, $P < 0.006$). For these tests, the postinjury group performed significantly better than the overall performance of the group at baseline. However, these differences are in the "normal range," on the basis of the NFL norms presented in *Table 2*. The differences are likely to reflect the influence of practice effects on these tests, as have been documented by previous studies using the NFL or similar test batteries (3, 10, 18, 26). Practice effects refer to improvements in performance on neuropsychological testing based on repeated exposure to a given test. Practice effects vary widely, depending on the nature of the test and whether or not alternative equivalent forms of the test are used. For this study, alternative forms were used for the memory tests (HVLT and BVMT-R), whereas no alternative forms were used for the Trail Making Test, SDMT, or COWAT. Therefore, larger practice effects were expected for the Trail Making test, the SDMT, and the COWAT compared with the HVLT and the BVMT.

Does Postinjury Neuropsychological Dysfunction Relate to On-field Memory Dysfunction?

Next, a series of analyses was completed to evaluate the relationship between neuropsychological testing within days of injury and on-field memory dysfunction. Athletes were evaluated by the team medical staff after injury, and the results were reported on a standard NFL physician form (33).

Athletes were selected according to the following criteria: 1) completion of postinjury neuropsychological testing before return to play and 2) a completed physician evaluation detailing the on-field sequelae of the concussion. Athletes who did not have completed physician data were excluded from the analysis. A series of one-way ANOVAs was completed, with the individual neuropsychological tests representing the dependent variables and the absence or presence of on-field memory dysfunction as the independent variable. Follow-up evaluations were completed an average of 1.3 days after injury (range, 1–9 d), with 90% of the athletes being evaluated 1 day after injury. The diagnosis of on-field memory dysfunction was determined by team physicians (33, 35). There was no significant difference between the memory dysfunction and no memory dysfunction groups with regard to whether or not they were rendered unconscious ($\chi^2 = 0.008$, $P = 0.926$).

Table 4 shows that athletes who were diagnosed as having memory dysfunction after injury perform more poorly on memory tests than injured athletes who did not display on-field memory dysfunction. For example, the on-field memory dysfunction group performed significantly more poorly on immediate memory ($F = 6.1$, $P < 0.02$) and delayed memory ($F = 5.4$, $P < 0.03$) aspects of the BVMT-R. In addition, the on-field memory dysfunction group also displayed relatively poorer performance on immediate memory and delayed

TABLE 3. Baseline and postinjury test performance for National Football League concussions in 1996–2001[a]

Test	N	Baseline	Follow-up	*F, P*
HVLT (Total)	95	25.8 (4.4)	26.6 (4.6)	$F = 2.97$ ($P < 0.10$)
HVLT (Delay)	95	8.8 (2.2)	8.7 (2.4)	$F = 0.02$ ($P < 0.89$)
BVMT-R (Total)	47	29.0 (4.3)	28.7 (6.8)	$F = 0.14$ ($P < 0.71$)
BVMT-R (Delay)	47	11.1 (1.3)	10.8 (1.7)	$F = 0.70$ ($P < 0.41$)
Trail Making Test, Part A	93	21.2 (8.4)	18.9 (6.2)	$F = 4.97$ ($P < 0.03$)
Trail Making Test, Part B	89	52.8 (14.3)	51.9 (30.3)	$F = 0.09$ ($P < 0.80$)
COWAT	90	39.9 (9.7)	41.1 (10.1)	$F = 1.42$ ($P < 0.23$)
SDMT-Total	59	59.2 (9.1)	62.2 (11.4)	$F = 8.1$ ($P < 0.006$)
Digit Span-Forward	87	9.8 (2.3)	10.6 (2.4)	$F = 10.5$ ($P < 0.001$)
Digit Span-Backward	87	7.9 (2.8)	8.0 (2.5)	$F = 0.71$ ($P < 0.40$)

[a] HVLT, Hopkins Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised; COWAT, Controlled Oral Word Association Test; SDMT, Symbol Digit Modalities Test.

TABLE 4. Neuropsychological test performance at follow-up for athletes with and without on-field memory dysfunction[a]

Test	Memory dysfunction	N	No memory dysfunction	N	*F*, *P*
HVLT (Total)	25.7 (5.0)	35	27.2 (4.4)	25	$F = 1.52$ ($P < 0.25$)
HVLT (Delay)	8.5 (2.6)	35	9.4 (2.1)	24	$F = 1.70$ ($P < 0.20$)
BVMT-R (Total)	24.6 (6.8)	27	29.5 (5.7)	17	$F = 6.1$ ($P < 0.02$)
BVMT-R (Delay)	9.4 (2.2)	24	10.9 (1.6)	14	$F = 5.4$ ($P < 0.03$)
Trail Making Test, Part A	19.3 (7.1)	35	18.9 (6.6)	24	$F = 0.04$ ($P < 0.85$)
Trail Making Test, Part B	47.6 (13.3)	35	44.6 (21.4)	24	$F = 0.48$ ($P < 0.50$)
COWAT	43.0 (10.7)	35	43.0 (10.6)	24	$F = 0.0001$ ($P < 0.99$)
SDMT-Total	60.9 (9.5)	31	60.8 (6.9)	23	$F = 0.52$ ($P < 0.49$)
Digit Span-Forward	10.6 (2.5)	33	10.0 (3.2)	24	$F = 0.49$ ($P < 0.50$)
Digit Span-Backward	8.4 (2.5)	33	7.7 (3.1)	24	$F = 0.77$ ($P < 0.39$)

[a] HVLT, Hopkins Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised; COWAT, Controlled Oral Word Association Test; SDMT, Symbol Digit Modalities Test.

memory components of the HVLT, although this did not reach statistical significance. Differences in test performance seem to be specific to memory and not, more generally, to cognitive dysfunction, because test performance on speed tests such as the SDMT and the Trail Making Test were nearly identical for both groups.

Effect of Multiple MTBIs on Neuropsychological Testing: Are There Cumulative Effects of Injury in Professional Football Athletes?

Two subject groups were formed to investigate the potential cumulative effect of multiple MTBIs on neuropsychological test performance. One group consisted of athletes with a history of three or more MTBIs during the course of the study, and the other was made up of athletes who had fewer than three concussions. All athletes underwent neuropsychological testing within 2 days of their third injury (mean, 1.04 d; range, 1–2 d). A series of ANOVAs was completed with individual neuropsychological testing scores as dependent variables and multiple concussion status as independent variables. Multiple ANOVAs were completed because not all of the athletes had completed all of the tests that made up the test battery because of changes in test procedures over the course of the neuropsychological testing program.

Table 5 shows that the two groups did not differ on any of the neuropsychological tests in the battery. Players with three or more concussions did not display statistically significant differences on the 10 individual scores that made up the neuropsychological test battery. It should be noted that the completion of multiple ANOVAs created an increased potential for finding significant differences between groups. However, despite the use of this very liberal research design, no such differences were found between groups.

Do Athletes Who Miss 7+ Days after MTBI Perform More Poorly on Neuropsychological Testing?

Next, the neuropsychological test performance of a group of athletes who missed 7+ days after MTBI was compared with that of an MTBI group who returned to play within a week of injury. This particular group had undergone previous study (34, 36, 37). The 7+ days out group was evaluated neuropsychologically relatively soon after injury (an average of 2.2 d after injury; range, 1–9 d). Group performance was analyzed statistically via multiple ANOVAs, with the individual neuropsychological measures gathered after the injury representing the dependent variables. Neuropsychological test results from the BVMT delayed memory data were not sufficient to allow meaningful statistical analysis. Consistent with our analysis of the cumulative effects of injury, the use of multiple ANOVAs served to increase the risk of finding significant differences in test performance between groups.

Table 6 shows that the 7+ days out group did not perform more poorly on any of the neuropsychological test measures compared with athletes who had been returned to play without missing 7 days. None of the group differences reached statistical significance. In addition, the mean values for all of the scores were comparable to NFL normative values presented in *Table 2* and to age expectations for a large sample of collegiate athletes who had been tested by the NFL test battery.

Figure 1 shows the means and standard deviations in BVMT-R (Total) neuropsychological test scores for various

TABLE 5. Neuropsychological test performance at follow-up for athletes with and without a history of three or more concussions from 1996 to 2001[a]

Test	Baseline for concussed group		Three or fewer concussions	N	Three or more concussions	N	*F, P*
HVLT (Total)	25.8 (4.4)	95	26.9 (4.7)	49	27.2 (5.0)	20	$F = 0.74$ ($P < 0.40$)
HVLT (Delay)	8.8 (2.2)	95	9.0 (2.4)	49	9.6 (2.5)	21	$F = 0.093$ ($P < 0.80$)
BVMT-R (Total)	29.0 (4.3)	47	26.7 (7.2)	36	27.9 (5.9)	17	$F = 0.29$ ($P < 0.59$)
BVMT-R (Delay)	11.1 (1.3)	47	10.1 (2.1)	30	10.3 (1.9)	15	$F = 0.040$ ($P < 0.84$)
Trail Making Test, Part A	21.2 (8.4)	93	19.5 (6.7)	49	17.6 (7.8)	20	$F = 1.03$ ($P < 0.32$)
Trail Making Test, Part B	52.8 (14.3)	89	46.4 (12.8)	49	44.7 (23.0)	20	$F = 0.16$ ($P < 0.70$)
COWAT	39.9 (9.7)	90	41.9 (8.7)	49	46.2 (13.5)	20	$F = 2.5$ ($P < 0.13$)
SDMT-Total	59.2 (9.1)	59	61.5 (11.5)	28	65.5 (8.2)	12	$F = 1.21$ ($P < 0.28$)
Digit Span-Forward	9.8 (2.3)	87	10.6 (2.1)	49	10.6 (3.6)	20	$F = 0.0001$ ($P < 0.98$)
Digit Span-Backward	7.9 (2.8)	87	8.2 (2.5)	47	8.5 (3.2)	20	$F = 0.136$ ($P < 0.72$)

[a] HVLT, Hopkins Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised; COWAT, Controlled Oral Word Association Test; SDMT, Symbol Digit Modalities Test.

TABLE 6. Neuropsychological test performance at follow-up for athletes who missed 7 or more days of practice/playing after concussion[a]

Test	<7 Days Out	N	7+ Days Out	N	*F, P*
HVLT (Total)	26.3 (4.8)	74	28.3 (4.7)	17	$F = 2.6$ ($P < 0.11$)
HVLT (Delay)	8.8 (2.4)	73	9.6 (2.0)	17	$F = 1.9$ ($P < 0.18$)
BVMT-R (Total)	27.4 (6.6)	54	26.3 (6.4)	15	$F = 0.32$ ($P < 0.60$)
Trail Making Test, Part A	19.3 (6.9)	73	17.0 (4.2)	17	$F = 1.3$ ($P < 0.26$)
Trail Making Test, Part B	48.2 (16.5)	73	49.6 (20.7)	17	$F = 0.09$ ($P < 0.76$)
COWAT	41.2 (10.9)	72	44.3 (8.4)	18	$F = 1.1$ ($P < 0.33$)
SDMT-Total	61.5 (11.5)	28	65.5 (8.2)	12	$F = 1.21$ ($P < 0.28$)
Digit Span-Forward	10.2 (2.6)	71	11.4 (2.5)	17	$F = 2.5$ ($P < 0.12$)
Digit Span-Backward	7.7 (2.6)	71	8.8 (2.6)	17	$F = 2.3$ ($P < 0.14$)

[a] HVLT, Hopkins Verbal Learning Test; BVMT-R, Brief Visuospatial Memory Test–Revised; COWAT, Controlled Oral Word Association Test; SDMT, Symbol Digit Modalities Test.

groups of NFL players for the purposes of comparison. The baseline scores are compared with those of players with memory problems, three or more concussions, and 7+ days out from play. Only the memory problem group showed a significant difference from the comparison group; however, the differences are subtle when the standard deviations are considered. Also shown are data from a sample of high school players (14) and college players at Penn State University (unpublished data; 12) showing scores similar to those in the professional players.

DISCUSSION

This study represents the first on neuropsychological function of NFL athletes in the United States. Numerous studies have been published on the neurocognitive recovery of high school (3,

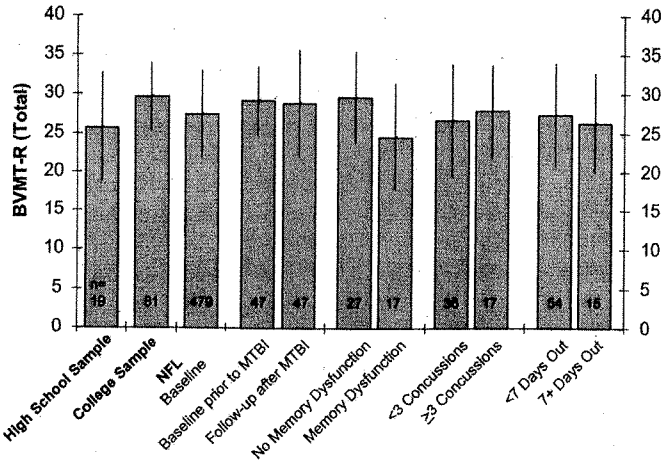

FIGURE 1. *Graph showing average and standard deviation in BVMT-R (Total) neuropsychological test scores for NFL baselines, players with memory problems, three or more concussions, and out 7+ days from play. Also shown are data in the first two columns from a sample of high school players (*from, *Field M, Collins MW, Lovell MR, Maroon J: Does age play a role in recovery from sports-related concussion? A comparison of high school and college athletes.* ***J Pediatr*** *142:546–553, 2003 [14]) and college players at Pennsylvania State University (unpublished data; and* from, *Echemendia R, Putukian M, Mackin S, Julian L, Shoss N: Neuropsychological test performance prior to and following sports related mild traumatic brain injury.* ***Clin J Sports Med*** *11:23–31, 2001 [12]).*

27, 28) and collegiate (10, 26, 30) athletes. The NFL neuropsychological test battery has been used extensively over the past decade both in and outside of the NFL. In addition to gaining general acceptance by NFL team medical staff and athletic trainers, neuropsychological testing has also been well accepted by the athletes. There is currently general agreement that neuropsychological testing provides quantitative information regarding recovery from injury, thereby improving the evaluation and management of MTBIs in athletes (2). More specifically, neuropsychological testing may provide diagnostic information regarding subtle disruptions of cognitive processes such as attention, memory, and speed that may not be detected by a cursory sideline evaluation.

On-field Cognitive Impairment

This study supports previous research that has shown that on-field signs of cognitive impairment, such as amnesia, are useful in determining the severity of brain injury (13, 27, 28). Consistent with studies of high school and collegiate athletes, professional athletes who were diagnosed by team physicians as having impaired memory function after concussion performed significantly more poorly on memory testing, while displaying generally intact performance on other measures requiring cognitive speed. These findings suggest that neuropsychological testing in combination with other diagnostic information helps track recovery from MTBI in professional football players. The results of this study also suggest that team physician diagnosis of "memory impairment" on the sideline has predictive value. Athletes who display on-field memory impairment have measurable memory impairment within 1 to 2 days after injury. Neuropsychological testing evaluates disruptions in cognitive processes such as attention, memory, and cognitive speed and adds quantitative information for the evaluation and management of concussion.

This suggests that only those players identified as having cognitive and memory disturbances are likely to have neuropsychological test impairments on follow-up testing. Athletes with no clinical cognitive and memory impairments on physician examination did not, as a group, have more subtle changes in cognitive processes that were missed on sideline clinical examination. This suggests that the on-field evaluation used by NFL team physicians is effective with regard to the identification of cognitive and memory impairments immediately after injury. The authors attribute this success to the training and knowledge of the NFL team physicians and the thorough nature of the league's injury documentation process. Furthermore, it must be pointed out that even the players with on-field memory impairments and neuropsychological test memory impairments 1 to 2 days later went on to make rapid, complete recoveries.

NFL Athletes Recover Quickly from Concussion

The finding of no overall significant neuropsychological testing differences between a group of injured NFL athletes who had previously undergone baseline neuropsychological testing suggests that NFL athletes with MTBIs recover quickly after injury. In contrast to previous studies that have suggested cognitive difficulties lasting 1 week or more, NFL athletes demonstrated generally intact performance within several days relative to baseline performance levels. There are a number of potential hypotheses for the apparent difference between professional and amateur athletes.

First, athletes selected for inclusion in this study were diagnosed as having experienced concussion based on multiple diagnostic criteria that were not limited to on-field neurocognitive dysfunction. In other words, a number of athletes in the sample did not necessarily demonstrate amnesia, confusion, or disorientation but rather noncognitive difficulties such as headache, nausea, or balance dysfunction. Within the population of NFL player injuries from which this sample was drawn (n = 887), approximately 50% did not have memory or other cognitive findings on the field (33). Therefore, it is not surprising that a significant number of injured athletes would not display signs of cognitive impairment days after concussion. Studies on high school and collegiate athletes have relied heavily on on-field amnesia and/or confusion, which is more predictive of neuropsychological sequelae. Lovell et al. (28) found group differences between baseline and postinjury performance in high school athletes who had a large proportion of on-field amnesia or confusion.

Second, recently published evidence suggests that there may be a recovery gradient, with younger athletes (high school age and below) displaying more pronounced injury and longer recovery times than older athletes (college age and above). Several lines of evidence support this hypothesis. Chil-

dren are known to exhibit more diffuse and prolonged cerebral swelling than adults (1, 21), and the majority of fatal brain injuries in contact sports have occurred in children below the age of 18 years (8). There is also evidence from animal research suggesting that the immature brain may be more sensitive to the negative effects of glutamate-mediated *N*-methyl D-aspartate than the brains of older animals. This has been hypothesized to play a role in the detrimental effects of excitatory amino acids after brain injury. Finally, recent clinical data have documented neuropsychological deficits lasting days after injury and a differing rate of recovery depending on the age of the athlete (14). A sample of collegiate athletes improved more rapidly on standardized tests of memory compared with a group of high school athletes with MTBI. The authors suggest that neurodevelopmental differences between older and younger subjects play a role in the recovery differences between the groups.

Finally, there may be a different tolerance for concussion between professional and nonprofessional athletes. The level of conditioning and skill necessary for success in the NFL may result in an overall sample pool of athletes who are less prone to injury than younger and less talented or well-conditioned individuals. There may be a "natural selection" process in professional football whereby athletes who are easily injured do not successfully rise through the ranks of high school to collegiate football to the NFL without resilience to cerebral concussion. This hypothesis is supported, albeit indirectly, by current NFL data suggesting that a relative minority of athletes develop postconcussion syndrome (34, 35).

Relevance to Previous NFL Concussion Studies

In previous articles, the authors have presented the results of clinical evaluations in professional football players after MTBI (33–35). Those studies demonstrated that NFL players, as a group, made relatively quick recoveries after cerebral concussion. As a group, players who sustained multiple MTBIs fared no differently from those with single injuries. Analysis of the group of players with the most prolonged recoveries after MTBI revealed no evidence of permanent brain dysfunction or other long-term effects on the basis of clinical examination. One possible limitation of those earlier studies was the absence of any neuropsychological test results from the database and its subsequent analysis. This article presents the neuropsychological testing results that were not included in those earlier articles.

The issue of the potential cumulative effects of sports-related MTBI has been a particularly controversial one, and many studies have offered different views on the significance of multiple injuries. This study did not find a pattern of poorer neuropsychological test scores in a group of professional athletes who were followed up closely for 6 years. The interpretation of these findings is complicated and may relate to a variety of factors, including potential differences in response to MTBI between professional and younger athletes as well as the general resiliency of highly conditioned athletes who have largely escaped serious consequences of brain injury throughout their high school, college, and professional careers.

As a group, NFL athletes who were held from play 7+ days did not display poorer neuropsychological performance than a group who returned within a week. Although based on a somewhat limited sample size, the results suggest that these athletes did not display significant cognitive impairment at the time of their postinjury follow-up evaluation within days of injury. Athletes are held from participation by a number of factors, which include both medical and nonmedical considerations. As suggested by Pellman et al. (34), it is possible that athletes who made up this group were treated more conservatively relative to other athletes. The overrepresentation of "skill position" players such as quarterbacks may also help explain why these athletes were held out of competition for more than one game. However, the 7+ days out group represented the most severely injured players on the basis of signs and symptoms (34). It is noteworthy that they had normal neuropsychological test performance and medical evaluation of cognitive and memory function within several days of injury.

The neuropsychological testing results presented here corroborate earlier purely clinical reports. As noted previously, the athletes who were held out of play for 7+ days displayed neuropsychological test performances equivalent to those of the group who returned to play in less than 1 week. This supports the findings of Pellman et al. (34) that even those players in the more seriously injured group in terms of number of signs and symptoms made good recoveries, apparently without discernible residual neurocognitive effects. Furthermore, the neuropsychological test performances did not differ between the group with three or more concussions and the group with fewer than three concussions. The strong correlation between the results of clinical and neuropsychological evaluations also provides supportive evidence for the position that there is no evidence in this study of widespread permanent or cumulative effects of single or multiple MTBIs in professional football players.

In other words, the results of this present study support the authors' previous work, which indicated that there was no evidence of worsening injury or chronic cumulative effects of multiple MTBIs in NFL players. Critics might contend that the specific neuropsychological test battery used in this study was not sensitive enough to detect chronic brain injury. However, the evidence suggests otherwise. Similar (frequently the same) neuropsychological test batteries were used to detect chronic brain dysfunction in retired boxers (9, 39). In both studies, neuropsychological tests uncovered evidence of cognitive and memory impairments, which were much more widespread than was suspected from purely clinical evaluations. Thus, the failure to find abnormalities in NFL athletes even after multiple and/or more serious MTBIs is not suggestive of a cumulative effect of injuries in NFL players.

Critics might also suggest that the paper-and-pencil neuropsychological test battery used in this present study was not sensitive enough to detect subtle cognitive and memory impairments. However, similar test batteries have been used in numerous studies of high school and college players and have been found to be sensitive to cognitive dysfunction (3, 10–12, 14, 16, 26, 30). The authors' interpretation of the present re-

sults, which found a strong relationship between on-field signs and symptoms and neuropsychological test results, would be that a thorough clinical, mental status examination of the players after MTBI by a well-trained physician is perhaps more sensitive than had previously been thought. Therefore, the thorough on-field evaluation of signs and symptoms is encouraged in all concussed athletes.

Importance of Clinical Evaluation by Team Physicians and Role of Neuropsychological Testing

The standard practice of NFL team physicians is to perform thorough, not cursory, sideline evaluations. This explains the strong correlation between on-field clinical findings and neuropsychological test results reported in this article. Given the established league-wide neuropsychological database, the test performance of the injured athlete can be compared with appropriate league standards, or ideally, the athlete's performance can be compared with his preinjury baseline levels of performance. In addition, given the tendency for some athletes to underreport symptoms in an effort to hasten their return to play (27), neuropsychological testing provides performance-based indicators of recovery that help ensure resolution of cognitive difficulties before return to play.

This study points out the overriding importance of the clinical evaluation by the team physician in determining the appropriate management of athletes who sustain MTBI. Had the team physicians relied solely on the neuropsychological test results in making their decisions, many of these more severely injured players would have returned to play sooner than was warranted by the clinical picture. Furthermore, of the 32 signs and symptoms of MTBI that were evaluated by NFL team physicians (33), *Table 7* shows that at most 13 (most likely only 8) are evaluated by neuropsychological testing. Therefore, if one relies solely on neuropsychological testing when managing MTBI, then one will almost certainly overlook more than half of the signs and symptoms that may occur. It clearly follows from the above that neuropsychological testing alone cannot be relied on, in and of itself, to make return-to-play decisions. The authors strongly caution against the growing trend of substituting neuropsychological testing for decision making based on physician evaluations in the treatment of sports-related MTBI.

As has been noted, all NFL teams use neuropsychological testing in the evaluation of concussion. The extent of testing varies greatly among teams. Likewise, the role of testing in the management of MTBI may differ. The data from earlier reports (33–35) reinforce that the vast majority of concussions in NFL players are mild, with little time lost from play. The neuropsychological data corroborate the rapid clinical recovery made by the players in routine cases. In these instances, the testing would seem to play a minor role, serving to confirm neuropsychological test recovery that correlates with the rapid clinical recovery.

In general, team physicians find neuropsychological testing most helpful in the management of more severe concussions. In cases of prolonged postconcussion symptoms, neuropsychological testing may help to "objectify" the neurocognitive changes after injury, allow monitoring of the recovery process, and assist the medical team and player regarding return-to-play decisions. NFL team physicians are relying on neuropsychological testing as a corroborative diagnostic modality when trying to quantify whether injury results in cognitive dysfunction. As refinements in testing are made, the role of testing in the evaluation and

TABLE 7. Signs and symptoms of concussion[a] and whether neuropsychological testing can evaluate changes

Signs and symptoms	Evaluated by neuropsychological testing
General symptoms	
Headaches	No
Neck pain	No
Nausea	No
Syncope	No
Vomiting	No
Back pain	No
Seizures	No
Cranial nerve symptoms	
Dizziness	No
Blurred vision	No
Vertigo	No
Photophobia	No
Tinnitus	No
Diplopia	No
Nystagamus	No
Pupil response	No
Pupil size	No
Hearing loss	No
Memory problems	
Retrograde amnesia, delayed	Yes
Information processing problems	Yes
Attention problems	Yes
Anterograde amnesia, delayed	Yes
Cognition problems	
Immediate recall	Yes
Not oriented to time	Yes
Not oriented to place	Yes
Not oriented to persons	Yes
Somatic complaints	
Fatigue	Possibly
Anxiety	Possibly
Personality change	Possibly
Irritability	Possibly
Sleep disturbance	No
Loss of appetite	No
Depression	Possibly
Loss of libido	No
Loss of consciousness	No

[a] From, Pellman et al. (33).

management of concussions in NFL players is likely to evolve and remain a diagnostic tool for team physicians.

Neuropsychological tests represent one tool that can be used to assist treating physicians when diagnosing and managing cerebral concussion. It should be used in conjunction with the clinical evaluation, brain imaging, and other diagnostic studies, where appropriate. However, neuropsychological testing should not be used alone to determine diagnosis or make management decisions such as return to play. In addition to having diagnostic value, the quantitative nature of the results of neuropsychological testing is often reassuring to injured players and their treating physicians and offers an objective means of tracking recovery. The present study has clearly indicated that the results of neuropsychological testing are strongly correlated with the results of clinical mental status testing on the sideline by the team physician. This does not, however, minimize the usefulness of such testing. The usefulness of neuropsychological testing can be explained best in parallel to the clinical neurological examination (excluding mental status). The clinical neurological examination is almost always normal after MTBI. The examining physician expects the neurological examination to be normal in these athletes. The normal neurological examination is reassuring to the physician that their diagnosis is correct.

If the neurological examination is abnormal, this raises a red flag for the treating physician and alerts him or her to the possibility that some other process may be going on. Even though the neurological examination is almost always normal in the setting of MTBI, no one is suggesting that the neurological examination is unnecessary or should be discarded. Similarly, the neuropsychological test results most often correlate with the clinical evaluation findings and almost always confirm the clinical evaluation findings after MTBI in the NFL. When the neuropsychological test results confirm the clinical examination, this is reassuring to the treating physician that his or her diagnosis is correct. In the rare instances in which the neuropsychological test results do not correlate with and/or confirm the clinical findings, this should also serve as a red flag warning to the treating physician to reevaluate the situation to be certain that no other abnormal processes are occurring. Thus, neuropsychological testing should be a part of the evaluation of athletes after MTBI.

On the basis of the results of this study, the MTBI Committee makes the following recommendations regarding the proper role of neuropsychological testing in the NFL. Neuropsychological testing is a tool that can assist the physician in evaluating and managing MTBI. It is definitely not to be used in isolation and cannot and should not be used to replace the clinical judgment of the treating physician in the diagnosis and management of MTBI. The main value of neuropsychological testing in this setting is its ability to confirm and corroborate the results of the clinical and mental status evaluation. Just as a normal clinical neurological examination (excluding mental status) helps confirm the diagnosis of MTBI, so neuropsychological test results consistent with the clinical findings help confirm the clinical impression regarding the presence or absence of cognitive and/or memory impairments.

Similarly, just as the occasional focal abnormality on the neurological examination alerts the physician to the possibility of a more serious underlying intracranial pathological condition, so the occasional neuropsychological test result that is incongruous with the clinical findings should alert the physician to investigate further. Neuropsychological testing is also useful because it provides quantifiable results regarding neurocognitive processes. These "objective" data can help both physicians and athletes realize the validity and the organic basis of cognitive and memory dysfunction after MTBI. It is also sometimes easier to track recovery using such quantifiable results as those of purely clinical examinations. The Committee believes that neuropsychological testing can be of value for the treating physician when used in the appropriate manner outlined above.

The Neuropsychological Test Battery

It is apparent that some of the tests in the NFL test battery are more effective in discriminating NFL concussions than others. This is similar to what has been found in other studies using the NFL test battery on collegiate and high school player samples (10, 26, 30). Future studies in the NFL will continue to implement tests that reflect the "state of the art" and have high sensitivity in detecting subtle brain injuries. As this refinement occurs, there will be attention to using tests with more reliability and fewer practice effects.

Although regarded as being sensitive to MTBI, tests such as the Trail Making and the Symbol Digit Modalities may eventually be replaced by tests that are less contaminated by practice effects when used on multiple occasions. Specifically, tests should be used that have high sensitivity to subtle deviations in neurocognitive processes, while also being reliable across multiple evaluation sessions. As *Figure 1* demonstrates, efforts are needed to reduce the normal deviation in responses for baseline evaluations and injured players to better differentiate the effects of injury. The goal of developing increasingly reliable and sensitive neuropsychological tests has been the impetus for the development of computer-based testing protocols over the past 5 years.

Transition to Computer-based Neuropsychological Testing

Although the program described in this article represents the standard for neuropsychological testing within the NFL during 1996 to 2001, many professional football teams are currently transitioning to a computer-based neuropsychological testing protocol. Approximately two-thirds of NFL teams are now using computer-based testing. Computer-based neuropsychological testing has become increasingly popular, for several reasons. First, computer-based testing evaluates many athletes with limited manpower (29). This has increased the participation in baseline testing throughout the league. Because the number of athletes evaluated depends only on the number of computers available, teams can potentially complete baseline testing during a 1-day session. Second, computer testing provides a more effective evaluation of certain neurocognitive areas, such as reaction time, which can be measured to within 0.01 second. In addition, the computer ensures uniformity of administration across teams and elimi-

nates differences in administration because of human error or differences in administrator style. There is also some evidence that computer testing may be less subject to the vagaries of practice effects than standard pen-and-paper testing (20).

Limitations of the Study

This research is an observational study of NFL players within the context of six seasons. The neuropsychology program was designed to operate with minimum disruption to the NFL teams and athletes. The study relied on a convenience sample of injured athletes without assurance that the sample is representative of the overall population of injured NFL athletes from 1996 to 2001. Furthermore, because this study was initiated with only a few participating teams in 1996 and the use of neuropsychological testing became more accepted toward the end of the study period, the overall number of participating athletes is relatively small compared with the 650 injured athletes that made up the MTBI sample (33). This somewhat limited sample size led to the use of completion of multiple individual ANOVAs rather than a more elegant multiple ANOVA design that is usually preferable in studies with multiple dependent variables.

Currently, all NFL teams are using neuropsychological testing, but not all use it in the same manner. Some teams perform baseline testing on all players, whereas others select baseline tests to be performed on players in higher-risk positions or players who have a history of previous concussion in high school and/or college. As a result, the overall number of NFL players participating in the neuropsychological testing program is much higher at present than it was during the early years of this study. The lack of completed baseline assessments in all athletes resulted in smaller sample sizes, which limits the statistical power of some of the analyses completed in this study. The authors expect that future studies on this much larger database will yield more information on the neurocognitive recovery process after concussion.

All neuropsychological testing was conducted by trained neuropsychology personnel using standardized administration protocols and test scoring rules. Test results are thought to accurately reflect the athlete's level of performance on any given test. However, as is true of any study involving multiple personnel, it is possible that scores may have differed somewhat from team to team, thereby resulting in a variation in test scores. The future use of computer-based neuropsychological protocols should minimize this factor. Along similar lines, the diagnosis of injury on the field was based on the clinical judgment of the team medical staff and did not necessarily involve the use of standardized on-field mental status examinations. Therefore, the diagnosis of memory impairment or other cognitive difficulties may have varied from team to team.

Another potential limitation of this study is the lack of established exact standards for the determination of abnormal test performance. If preseason baseline testing had been completed on a particular player, abnormal performance was determined by comparison of the athlete's postinjury score to this baseline. If the score was poorer than baseline levels, the score was determined to be "abnormal." If a preinjury baseline test battery had not been completed, the athlete's score was compared with existing norms. Although this represented the standard for analysis of neuropsychological test scores in the NFL in the 1990s, more recently, Reliable Change Index (RCI) scores have evolved to provide a more objective determination of abnormal performance (3, 20). RCIs are based on the test-retest reliability of the particular test and adjust for practice effects. RCIs provide confidence intervals for determining whether or not a test score is reliably different from baseline. RCIs have not been calculated for professional football athletes, although they do exist for high school (3, 20) and professional rugby athletes (18). As the NFL neuropsychology program develops, the implementation of RCIs of other statistical standards will aid in future clinical interpretations.

DISCLOSURE

None of the Committee members have a financial or business relationship posing a conflict of interest to the research conducted on concussion in professional football. MRL has no financial interest in the neuropsychological tests used in this study but does have a financial interest in the ImPACT computer-based neuropsychological test battery used by many NFL teams.

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Acknowledgments

The NFL has a Committee on MTBI. It is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society, NFL Athletic Trainers Society, NFL equipment managers, and scientific experts in the area of traumatic brain injury, biomechanics, basic science research, and epidemiology. The authors of this article are members of the Committee. The efforts of other Committee members are gratefully acknowledged, including John Powell, Ph.D., Henry Feuer, M.D., Douglas Robertson, M.D., Joseph Waeckerle, M.D., Ronnie Barnes, ATC, and Jay Brunetti.

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COMMENTS

This study by Pellman et al. analyzes the neuropsychological testing profile of 143 National Football League (NFL) players who experienced concussions, out of a total of 650 athletes. An additional analysis was performed on four subgroups consisting of the postconcussion performance compared with preseason baseline assessments in 95 athletes. They assessed the neuropsychological test performance of 70 athletes in relation to the presence of on-field amnesia, the potential role of multiple concussions on neuropsychological test performance in 70 athletes, and the neuropsychological testing profile of 18 players who lost 1 week or more of playing time compared with 71 who returned to play within the same week. Their findings showed that the presence of cognitive and memory abnormalities correlate with neuropsychological test impairment, although they point out the limitations of neuropsychological testing. This includes particularly the lack of established standards, the use of neuropsychological testing being uniform among all teams and neuropsychologists, and the occurrence of practice effects. Neuropsychological testing has been established as a very valuable

ancillary test to help objectify the extent of injury and recovery in athletic mild traumatic brain injury (MTBI). The authors postulate an interesting hypothesis that the younger athletes sustain more serious MTBIs; however, I do not believe that this has yet been proved. In addition, I do not believe that this study, with correlation between clinical and neuropsychological evaluation, proves that there are no widespread permanent or cumulative effects of single or multiple MTBI in NFL players. I think that it is premature to conclude that there are no long-term consequences of MTBI in football while players are still active, for many reasons. Further studies using neuropsychological testing, balance testing, neuroimaging, metabolic studies, functional magnetic resonance imaging, or other ancillary objective measures will be welcome as we attempt to understand this phenomenon further.

Julian E. Bailes
Morgantown, West Virginia

In this sixth in a series of articles on MTBI in NFL players, neuropsychological data from concussed players were analyzed. The sample included 143 athletes, representing 22% of 650 players who experienced 887 concussions during six seasons. Although the majority of players showed rapid recovery after concussion, a subset with on-field memory disturbance showed persistent memory problems within 2 days of the concussive event. The authors conclude that MTBI is associated with a rapid return to normal functioning in the great majority of players. The findings are somewhat surprising, but at first glance, they seem encouraging for the athletes in terms of short-term neuropsychological risk from concussion. However, these results should be interpreted with caution. Further follow-up of players sustaining MTBI is needed to better determine the cumulative effect of multiple concussions. In addition, it would be important to better identify specific players at risk or particular injury mechanisms that result in more severe concussions with on-field memory loss. Finally, as the authors acknowledge, a major shortcoming of this study is that a relatively small sample size of concussed athletes, only 22%, actually underwent neuropsychological testing, and participation was voluntary. Why did 78% of concussed players choose not to participate? This study design creates potential for significant sampling bias. The authors are encouraged to continue and expand this important work to better characterize the short- and long-term consequences of MTBI in professional athletes.

Daniel F. Kelly
Los Angeles, California

This study addresses important questions and uses a highly interesting group of subjects; in short, it is an irresistible read. It also is ambitious, because studies based on an absence of findings ("absence of proof is proof of absence") are inherently difficult and controversial.

An article's experimental sample defines the outer boundaries of what that article can find, conclude, and recommend. It is perplexing that the authors chose to include athletes 1 to 10 days after injury in their MTBI group. As the authors note in the introduction of the article, previous studies with the same test battery have found recovery "within a week." The authors' choice to include subjects more than 7 days after injury, therefore, increases the likelihood that their experimental group will contain subjects who have already recovered. This would dilute the overall cognitive symptoms of the experimental group and increase the variability within it, both of which make it more difficult to obtain statistically significant findings.

Most of the conclusions and recommendations of this article are based on statistical analyses in which no differences were found. Therefore, methodological choices that decrease the likelihood of obtaining statistically significant differences are worrisome. In the present study, they also are perplexing. As the authors note, the MTBI group's range of days after injury was 1 to 10, with a mean of 1.40 days and a standard deviation of 1.29 days. This indicates that, even if the experimental sample had been defined to include subjects as far as 2 standard deviations from the mean, most subjects would have been 4 days or less after injury, and that subjects at the 10-day end of the range almost certainly are outliers. The authors' data thus demonstrate that they had a large corpus of subjects within 4 days after injury. Why not use this group? Or why not apply their own understanding of the literature and use a group 7 days or less after injury? The authors' choice to dilute their experimental sample by including outliers weakens the impact of their findings, conclusions, and recommendations.

The authors possess a remarkable data set. My strongest impression after reading the article was that the data set was so important that it deserved additional analysis and that a good place to start would be to remove the outliers and see the results.

Joseph Bleiberg
Neuropsychologist
Washington, District of Columbia

This article provides a review of the use of neuropsychological testing in the NFL from the inception of the program in the mid 1990s to the present time. This is an important article, because the NFL's neuropsychological testing program has become an important model for the neuropsychological assessment of athletes in sports.

Overall, the article is very well written, provides an excellent review of research in the area, and makes an important contribution to the literature. In addition, the article provides normative values that have never before been published. This will provide useful data for the interpretation of neuropsychological test results in professional football players and may also help in the interpretation of emerging data in other professional athletes (e.g., ice hockey, rugby, and automobile racing). The article also presents new data from subsamples of injured NFL athletes to investigate the following research questions. 1) Do concussed NFL athletes display abnormal test results when tested days after injury? 2) Does postinjury neuropsychological dysfunction relate

to on-field memory dysfunction? 3) Is there evidence of the cumulative effect of injury in professional athletes? and 4) Do athletes who miss 7+ days after MTBI perform more poorly on testing? These are all extremely interesting questions that have been addressed to some extent by previous articles in younger athletes. However, this is the first article to attempt to address these issues in a group of professional athletes.

The strength of this article is its ambitious attempt to address multiple aspects of the concussion issue. To answer these research questions, the article provides analyses of several different subsamples of athletes. Although some of these groups are quite large (e.g., the baseline group of more than 600 athletes) and provide a very sound statistical foundation for analysis of the data, the analyses of the 3+ concussions and 7+ days concussion groups are based on relatively small sample sizes. Therefore, although extremely interesting and worthy of discussion, the conclusions based on these analyses should be tempered somewhat, particularly in the Discussion section. For example, the authors seem to imply that the data reported in this article unequivocally indicate that there is diminished risk of long-term injury in professional athletes relative to younger players. Although this may turn out to be true in the long run, this study, in and of itself, does not allow a strong conclusion regarding this issue. Clearly, further research is needed in this area. It is specifically recommended that the statement that there are no widespread permanent or cumulative effects of single or multiple MTBIs in professional football players be softened somewhat.

The article makes the important point that neuropsychological testing, at least in its present form, does not detect all postconcussive symptoms, particularly those that may be noncerebral or vestibular in nature. This point is often overlooked. The best approach to concussion diagnosis and treatment clearly should involve attention to both neuropsychological and non-neuropsychological signs and symptoms. However, the authors seem to suggest that the role of neuropsychological testing is "minor." Such a strong statement does not seem to be justified. Regarding this point, it is important to note that this present study uses "traditional" testing procedures rather than computer-based tests that have demonstrated increased sensitivity to concussion. Therefore, any broad generalizations regarding the limitations of this particular neuropsychological test battery do not seem to be warranted. This point should be clearly made within the article.

The comparison of test results on the basis of different samples of athletes (e.g., high school, college, professional) is particularly interesting. This represents the first "cross-sample" comparison of test results across professional and amateur ranks. However, *Figure 1* could be more clearly labeled so that it is clear that Columns 4 to 8 represent NFL athletes.

Overall, this article presents very interesting new data that will enhance the overall understanding of concussion. The authors have drafted an ambitious article.

Joseph C. Maroon
Pittsburgh, Pennsylvania

The NFL's MTBI Committee began the neuropsychological testing program several years ago, and we can only hope that the league and the players' union will find a way to mandate (sooner, rather than later) the program for all players in the league. My understanding is that while nearly all teams "participate" in the program, not all players on every team choose to participate. This is one of the obvious problems with the most recent article by Pellman et al. It is unfortunate that only 22% of the concussed players (16% of the overall concussions sustained) are represented because of incomplete data. In fairness to the authors, it is challenging to capture data on these injured players at consistent time points. Still, the study is based on only a small subsample of players who experienced MTBI and agreed to participate in the neuropsychological testing program. The reader is left wondering about those concussed players who chose not to participate in the neuropsychological testing program. Could there be players with three or more concussions who were experiencing cognitive difficulties and whose outcomes would have changed the findings if included in the data analyses? Thus, a major limitation of the study is that it could involve a biased sample.

In addition to the sampling issues, the authors have overinterpreted some of their findings. Given the methods and statistical design used, it is difficult to understand how they can comment that "the strong correlation between the results of clinical and neuropsychological evaluations also provides supportive evidence for the position that there is no evidence in this study of widespread permanent or cumulative effects of single or multiple MTBIs in professional football players." They only studied the acute neuropsychological effects of single and repeat cuncussion, and the data presented tell us nothing about potential "permanent" or long-term complications. The authors cannot assume that there could not be chronic effects, especially since they have only looked at a brief window of time.

The authors use of the word "preliminary" at one point within the text of the article was appropriate, because—at the very best—it is a preliminary study. "Preliminary" should have been used in the title and abstract as well. I think that future studies on this topic will capture a clearer picture of what is really occurring, given that there is now more widespread use of neuropsychological testing in the NFL. Neuropsychological testing is very important in helping physicians and athletic trainers to manage sport-related concussion. Hopefully, it can soon be mandated in the NFL so that players participate in the program; in time, such participation will lead to more complete data. I hope that the findings will be consistent with these preliminary findings.

Kevin M. Guskiewicz
Director, Sports Medicine Research Laboratory
University of North Carolina
Chapel Hill, North Carolina

TAB 1G

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CONCUSSION IN PROFESSIONAL FOOTBALL: PLAYERS RETURNING TO THE SAME GAME—PART 7

Elliot J. Pellman, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, and ProHEALTH Care Associates, LLP, Lake Success, New York

David C. Viano, Dr. med., Ph.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Ira R. Casson, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York; Department of Neurology, Long Island Jewish Medical Center, New Hyde Park; Department of Neurology, New York University Medical Center, New York; and Department of Neurology, Albert Einstein College of Medicine, Bronx, New York

Cynthia Arfken, Ph.D.

Department of Psychiatry and Behavioral Neurosciences, Center for Healthcare Effectiveness Research, Wayne State University, Detroit, Michigan

Henry Feuer, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and Indianapolis Neurosurgical Group, Indianapolis, Indiana

Reprint requests:
David C. Viano, Dr. med., Ph.D., ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952. Email: dviano@comcast.net

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OBJECTIVE: A 6-year study was conducted to determine the signs, symptoms, and outcome of players who were concussed and either returned immediately or were rested and returned to the same game in the National Football League (NFL).

METHODS: From 1996 to 2001, concussions were recorded by NFL teams by use of a special standardized reporting form filled out by team physicians. Signs and symptoms were grouped by general symptoms, somatic complaints, cranial nerve effects, cognition problems, memory problems, and unconsciousness. Action taken after concussion was recorded for 887 patients.

RESULTS: There were 135 players (15.2%) who returned immediately and 304 (34.3%) who rested and returned to the same game after concussion. There were few differences by player position or team activity about the injury or action taken. However, the mean number of signs and symptoms progressively increased from those who returned immediately (1.52), rested and returned to play (2.07), were removed from play (3.51), or were hospitalized (6.55). Immediate recall problems (odds ratio [OR], 1.93; confidence interval [CI], 1.26–2.94), memory problems (OR, 1.52; CI, 1.06–2.19), and the number of signs and symptoms (OR, 1.39; CI, 1.25–1.55) were predictive of removal from play or hospitalization. There was no statistical association between return to play in the same game and a subsequent concussion or a more serious concussion involving 7+ days out.

CONCLUSION: Players who are concussed and return to the same game have fewer initial signs and symptoms than those removed from play. Return to play does not involve a significant risk of a second injury either in the same game or during the season. The current decision-making of NFL team physicians seems appropriate for return to the game after a concussion, when the player has become asymptomatic and does not have memory or cognitive problems.

KEY WORDS: Concussion, Concussion guidelines, Epidemiology, Injury surveillance, Sport injury, Traumatic brain injury

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Earlier articles in this series have documented the National Football League (NFL) experience with repeat mild traumatic brain injuries (MTBIs), the more serious MTBIs that resulted in players being removed from play for more than 7 days, and the entire cohort of all NFL players in the MTBI registry who sustained concussion during a recent 6-year period (12–14). Analysis of the results uncovered a small number (n = 10) of players who sustained an MTBI, returned to play on the same day as the injury, and subsequently were kept out of play for more than 7 (7+) days (13). This raised concerns that perhaps some players were being returned to play too soon after MTBI, thus resulting in more prolonged postconcussion syndrome and perhaps risk of more severe brain injury. The authors undertook this present analysis of the NFL players who returned to play on the same day as their MTBI to investigate these concerns.

One of the primary expressed rationales for developing concussion management guidelines has been to prevent head-injured athletes from being cleared to return to play too soon after MTBI (1, 4). The authors of these guidelines raise the specter of second-impact syn-

drome, subdural or other intracranial hemorrhage, and/or the increased risk of repeat concussion when players are prematurely returned to play. The guidelines make recommendations that specifically define criteria to limit the number of players who will be cleared to return to play on the day of injury (1, 4). NFL physicians often clear players to return to the game in which they were injured on the basis of the clinical judgments of those team physicians rather than the recommendations of these guidelines. A close examination of the real-life NFL experience with return to play after MTBI was another reason for this present study.

In 1994, the NFL formed the Committee on MTBI in response to safety concerns regarding head injuries. Background on the Committee has been described by Pellman (10). Its mission was to investigate MTBI in the NFL by various scientific methods.

The MTBI Committee undertook a series of research projects aimed at defining concussion biomechanics in professional football. On the basis of analysis of game video and laboratory reconstruction of severe impacts using instrumented test dummies, the biomechanics of concussion has been determined for professional players. This has included data on the head acceleration of injury (16) and the location and direction of impacts (15).

The Committee also determined a strong need to monitor the frequency of MTBI in the NFL and at the same time identify the clinical signs and symptoms associated with concussion. The initial study focused on the epidemiology of 787 concussions during regular-season NFL games, its signs and symptoms, and the relative risk by player position (12). Pellman et al. (14) evaluated repeat concussions in professional football, including the clinical signs and symptoms with three or more concussions over 6 years from 1996 to 2001. A history of concussions before participation in the NFL was not recorded; however, on the basis of NFL Combine experience, it is rare to see a player with two or three concussions during his entire high school or college career.

More recently, concussions involving 7+ days out from play were evaluated (13). This involved an 8.1% subset of the 887 concussions in the NFL and addressed the most severely injured players. The most recent study covered neuropsychological testing of the recovery of players with concussions, repeat concussions, and injuries with 7+ days out of play evaluated (11). The present study was motivated by a question on the safety of returning concussed professional players to the same game, either immediately or after a period of rest, considering that return to the same game may be ill-advised at the high school level. Because this involves approximately half of the concussions in the NFL registry, a thorough analysis was conducted.

MATERIALS AND METHODS

The MTBI Committee devised a simple form for team physicians to complete on observed and reported signs and symptoms on initial and follow-up examinations, whenever they evaluated a player who sustained concussion. At the NFL level, there is close cooperation between team physicians and athletic trainers on player medical issues, and they worked together to collect cases and data for this study. All players were examined by team physicians, and all management decisions were made by physicians. During the study period, two teams were added to the NFL. This registry of concussions involved MTBI data from 30 teams in the NFL. The median number of concussions reported by the teams was 26 (range, 6–72 concussions) during the study period. Player's names were not included on the forms so as to maintain confidentiality. They were identified by a six-digit number.

Operational Definitions

The definition introduced by the Committee in 1996 and used for the study is as follows. A reportable MTBI is a traumatically induced alteration in brain function, which is manifested by 1) alteration of awareness or consciousness, including but not limited to being dinged, dazed, stunned, woozy, foggy, amnesic, or, less commonly, rendered unconsciousness or, even more rarely, experiencing seizure; or 2) signs and symptoms commonly associated with postconcussion symptoms, including persistent headaches, vertigo, lightheadedness, loss of balance, unsteadiness, syncope, near syncope, cognitive dysfunction, memory disturbance, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities.

The definition is a natural extension of a much earlier one from the Ad Hoc Committee to Study Head Injury Nomenclature of the Congress of Neurological Surgeons (5) and is consistent with the definition by the American Congress of Rehabilitation Medicine (2).

Alteration of Awareness

There is occasionally some difficulty in eliciting a history of loss of consciousness or a transient alteration in awareness in professional football players. MTBIs may be very short-lived and not witnessed by the athletic trainer or medical staff. In addition, the player may not want to admit that such an event occurred because of a concern that he may lose playing time. Accordingly, any player who met any of the criteria above as determined by the athletic trainer or medical staff was included as having an MTBI.

Signs and Symptoms

MTBI is a clinical syndrome that may present with a broad spectrum of signs and symptoms, many of which are nonspecific and can be associated with other clinical diagnoses. The MTBI Committee members who are team physicians in the NFL, as well as MTBI Committee consultants with special expertise in the fields of sport neuropsychology and sport neurology, developed a list of the most common signs and symptoms with concussion. They were grouped into six categories: 1) general symptoms, 2) somatic complaints, 3) cranial

nerve findings, 4) cognitive abnormalities, 5) memory problems, and 6) unconsciousness. The checklist was filled out for each player with a concussion.

A purposely large and inclusive list was selected so as to capture all of the possible clinical signs and symptoms with MTBI in professional football players. The signs and symptoms that were recorded are consistent with previous medical literature on the postconcussion syndrome and the symptoms and signs seen after traumatic brain injury. Most of the items are symptoms that the player may complain of or that the physician may elicit by history. However, some items are mental status findings (retrograde amnesia, anterograde amnesia, or problems with information processing, attention, and immediate recall). The Committee did not distribute uniform testing instruments to the team physicians but rather left the assessment of these parameters to the discretion of the individual team physicians. The rationale for the various signs and symptoms can be found in Pellman et al. (12).

The form contained questions about physical examination findings, initial management, tests ordered, and disposition regarding return to play. A form was generated for each player's MTBI, including the initial evaluation and all subsequent follow-up visits until the player was cleared for return to play. The individual team physicians were to complete the initial and follow-up forms on the basis of their clinical findings. In the final analysis, the signs and symptoms were designed to be interpreted by the physicians in the context of the case. Sometimes, the initial examination form was completed the next day, which allowed a sleep disturbance finding. Team physicians and their consultants used their own evaluation procedures to manage the injury; however, their practices are all very similar to previously published sideline assessments. The Committee did not impose outside medical decision-making on the medical staffs of the individual teams.

Return to Play

The following definitions apply to the return-to-play aspect of the medical report. For this study and statistical analyses, the "return immediately" and "rest and return" terms designate "returned to the same game or session." These categories are compared with players who were removed from the game or session or were hospitalized. It should be stressed that NFL team physicians cleared a player for return to play only after he became asymptomatic and had a normal neurological examination.

Return immediately: The player returns after an evaluation by the team physician demonstrates that the player is asymptomatic. The key here is that the player, because of his relative position on the team, may not be called to action for several minutes. For example, if the player was on the kickoff team and sustained an MTBI, the physician would perform an evaluation and determine that the player is ready to return, yet, depending on the game, it may be minutes or possibly an hour or so before he actually gets back on the field. The injury could also occur near the end of the game.

The American Academy of Neurology (1) guidelines say that if the player's symptoms clear within 15 minutes, he can be judged able to return. In many cases, the return-to-play decision was made earlier than 15 minutes. In a practical sense, this might characterize "return immediately." By the same token, the player may be called to action immediately after the physician's declaration of "ready to return."

Rest and return: On initial evaluation, the player is symptomatic, but the symptoms then clear, and it is determined that there should be some protracted time before a decision is made to return. The key would be that the player did, in fact, return to the same game or practice. An example might be that the injury occurs in the last 5 minutes of the second quarter. Were it within the first 5 minutes, he might have been judged to return in the second quarter. But because it is close to halftime, the decision to return is not made until after halftime.

Removed from play: The player is not allowed to return to the game or session in which he was injured.

Hospitalized: The player was admitted to the hospital. Over the years, we have continually characterized this as greater than 18 hours. This would mean that simply going to a local hospital for an x-ray, head computed tomographic scan, etc., and then going home would not be classified as "hospitalized."

NFL players who have been identified as having had an MTBI during a game and having been cleared to return to play that same day have several physicians (at least three) and athletic trainers (at least three) who continue to monitor the players for any subtle signs or symptoms that suggest MTBI relapse, repeat MTBI, or misdiagnosis. This plethora of team medical staff available for observation is admittedly unique compared with most high schools, colleges, and many universities.

Days Out

The definition of days out is the time between the date of injury and the date that the player was permitted to return to full and unlimited participation (18). Full and unlimited participation means that the player must be able to perform all the activities of the session at the same intensity as his teammate. If a player were in a practice session and were not allowed to participate in contact drills, he would not be considered to be returned to participation because he was not able to do all of the activities expected of his teammates. In essence, this tells us that on the date of return, the player was expected to participate fully in all of the activities that were planned for the team practice or game.

Efforts to Improve Compliance

The Commissioner of the NFL mandated all team physicians to complete and return forms whenever they examined a player with a head injury. The project was designed to record information about the injury. The data forms were sent to the NFL epidemiologist and entered into a database with a

blinded coding to maintain anonymity of the players. When an initial evaluation form was submitted but the follow-up visit form was not, Committee members contacted team athletic trainers and doctors directly to remind them to submit the follow-up forms. During biannual meetings, the Committee monitored the data and discussed findings.

Quality Assurances

The MTBI evaluation forms were logged in by the Committee's epidemiologist and scanned into a database using a commercial software program (Teleforms, Cardiff, CA). During the data logging, the individual forms were manually reviewed. Each form was then scanned into a temporary database and verified before being entered into the final database. Any fields that were incomplete or inconsistent triggered a follow-up contact with the team athletic trainer or physician to verify the data. The final database includes information from the initial and follow-up evaluations submitted by team physicians.

Statistics

Descriptive statistics were used to characterize those players who were returned immediately to play or rested and returned versus those players who were removed from the game or hospitalized during the 6 years of study. χ^2 and t tests were used to compare the signs and symptoms, medical action, management, and time loss to the team for these two groups of injured players. For subanalyses with small sample sizes, Fisher's exact test was used. Combinations and presence of signs and symptoms that may predict returning to play were explored in multiple ways. The final screening was derived from multiple logistic regression models with forward conditional selection using variables that were at least twice as common in returning to play versus those who were removed or hospitalized. Cantu's (3) suggestion of 1 minute or more of unconsciousness was used to categorize loss of consciousness for statistical analysis, because of many unknown, missing, or indeterminate cases. Data are presented on all known cases of loss of consciousness, those reported as zero (or no loss of consciousness), and the unknown cases.

RESULTS

Table 1 shows the action taken with concussion in the NFL for single and repeat injuries. It shows that 49.5% of players returned immediately or rested and returned to the same game. The median time interval between concussion and immediate return to the game was 5 minutes; the median time between injury and rest and return to the game was 17 minutes. Of the 300 injuries with recorded return date of the same day or the next day after injury, 41% of players returning to the same game or session (return immediately or rest and return) were out more than 15 minutes. Of the 95 who returned immediately, 17.9% were out more than 15 minutes. Of the 205 who rested and returned, 51.7% were out more than 15 minutes. There is no trend between repeat concussions and the action taken. The linear $\chi^2 = 2.65$, $df = 1$, and $P = 0.10$ for repeat concussion Numbers 1 to 3 and action taken.

Of the 45 players with same-season concussion, 20 were removed from play or hospitalized versus 25 who returned to the same game either immediately or after resting. For those players returning to the same game, only 12% had a second concussion in the same season involving 7+ days out. This is similar to the 10% of cases involving removal and hospitalization (Fisher's exact test, 1.00). When controlling for days between injury and immediate return to play, logistic regression showed no association ($P = 0.99$), and the odds ratio for a second concussion was similar (OR, 1.01; 95% CI, 0.15–7.09) irrespective of the action taken to return to play.

TABLE 1. Action taken with concussed players in the National Football League, 1996–2001

Action taken	Single concussion	Repeat concussions							Total concussions
		1st	2nd	3rd	4th	5th	6th	7th	
Return immediately	83	25	19	8	0	0	0	0	135
Rest and return	145	55	65	25	8	4	1	1	304
Removed from play	247	73	66	16	5	3	2	0	412
Hospitalized	7	2	7	2	2	0	0	0	20
Unknown	8	5	3	0	0	0	0	0	16
No. of concussions	490	160	160	51	15	7	3	1	887
% Return immediately	16.9%	15.6%	11.9%	15.7%	0%	0%	0%	0%	15.2%
% Rest and return	29.6%	34.4%	40.6%	49.0%	53.3%	57.1%	33.3%	100%	34.3%
Either	46.5%	50.0%	52.5%	64.7%	53.3%	57.1%	33.3%	100%	49.5%

Table 2 shows the action taken with concussion by player position for the six NFL seasons. There were minimal differences in action taken by player position, whether position is considered individually or by groups. The defensive linemen and punters had a higher rate of immediate return or rest and return to play versus all other positions. Overall, approximately half of the players, irrespective of position, returned to the same game after concussion in the NFL.

Table 3 shows the team activity at the time of the injury and the action taken after concussion. Concussion occurring during interceptions had the highest rate of return to the same game, whereas injuries during kickoffs and passing plays had the lowest rate. When considering passing, runs inside or outside the tackle, punts, and kickoffs, there is no statistical difference between the groups with immediate return and rest and return to the same game and the other actions taken by the play causing the injury ($\chi^2 = 5.88$, $df = 4$, $P = 0.21$).

Table 4 shows the signs and symptoms by action taken with concussion in the 1996 to 2001 NFL seasons. Players returning immediately to the game have the lowest mean number of signs and symptoms (1.52), followed by those rested and returned to the same game (2.07). Players removed from the game or hospitalized had much higher mean number of signs and symptoms, showing a progression in the severity of injury and action taken. The comparison of either immediate return or rest and return to the same game versus other actions is statistically significant ($t = 1.29$, $df = 717$, $P = 0.000$). Players who return to the same game have significantly lower incidences of cognitive ($\chi^2 = 91.37$, $P = 0.001$) and memory ($\chi^2 = 84.48$, $P = 0.000$) problems than players removed from play or hospitalized.

The risk factors for not returning to the game are the number of signs and symptoms (OR, 1.39; CI, 1.25–1.55), immediate recall problems (OR, 1.93; CI, 1.26–2.94), and memory problems (OR, 1.52; CI, 1.06–2.19). None of the players who returned to the game experienced loss of consciousness for 1 minute or more. However, nine injuries (2.1%) in eight players caused some reported loss of consciousness, and they were listed as returned to the game. All but two of these players had injuries near the end of the game. Although the sample size is

TABLE 2. Action taken with concussion by player position in the National Football League, 1996–2001[a]

Player position	Action taken								
	Return immediately	Rest and return	Removed from play	Hospitalized	Unknown	Total	% Return immediately	% Rest and return	% Either
Offense									
Wide receiver	9	49	51	1	0	110	8.2%	44.5%	52.7%
Running back	13	28	34	2	1	78	16.7%	35.9%	52.6%
Quarterback	14	12	28	5	2	61	23.0%	19.7%	42.6%
Offensive line	14	25	37	0	0	76	18.4%	32.9%	51.3%
Tight end	4	15	25	1	2	47	8.5%	31.9%	40.4%
Subtotal	54	129	175	9	5	372	14.5%	34.7%	49.2%
Defense									
Secondary	24	44	81	5	3	157	15.3%	28.0%	43.3%
Defensive line[b]	13	36	25	1	3	78	16.7%	46.2%	62.8%
Linebacker	12	20	28	3	2	65	18.5%	30.8%	49.2%
Subtotal	49	100	134	9	8	300	16.3%	33.3%	49.7%
Special team	1	4	6	0	0	11	9.1%	36.4%	45.5%
Kick unit	23	44	65	1	2	135	17.0%	32.6%	49.6%
Return unit	4	12	17	1	0	34	11.8%	35.3%	47.1%
Return ball carrier	2	10	10	0	0	22	9.1%	45.5%	54.5%
Punter[c]	2	5	1	0	0	8	25.0%	62.5%	87.5%
Kicker, FGA	0	0	1	0	0	1	0.0%	0.0%	0.0%
Kicker, PAT	0	0	1	0	0	1	0.0%	0.0%	0.0%
Holder	0	0	0	0	1	1	0.0%	0.0%	0.0%
Undesignated	0	0	2	0	0	2	0.0%	0.0%	0.0%
Subtotal	32	75	103	2	3	215	14.9%	34.9%	49.8%
Total	135	304	412	20	16	887	15.2%	34.3%	49.5%

[a] χ^2 for player position between either and all others. FGA, field goal attempt; PAT, point after touchdown.
[b] $\chi^2 = 7.32$, $P = 0.007$.
[c] $\chi^2 = 4.45$, $P = 0.035$.

TABLE 3. Team activity versus action taken with concussion in the National Football League, 1996–2001

Team activity	Action taken								
	Return immediately	Rest and return	Removed from play	Hospitalized	Unknown	Total	% Return immediately	% Rest and return	% Either
Passing	48	99	143	11	4	305	15.7%	32.5%	48.2%
Interception	1	4	1	0	0	6	16.7%	66.7%	83.3%
Run inside tackle	30	43	68	2	6	149	20.1%	28.9%	49.0%
Run outside tackle	19	56	54	5	2	136	14.0%	41.2%	55.1%
Fumble	0	2	1	0	0	3	0%	66.7%	66.7%
Punt	13	33	29	1	1	77	16.9%	42.9%	59.7%
Field goal attempt	0	1	1	0	0	2	0%	50.0%	50.0%
Point after touchdown	0	1	0	0	0	1	0%	100%	100%
Kick off	18	41	68	1	1	129	14.0%	31.8%	45.7%
Other	1	14	30	0	0	45	2.2%	31.1%	33.3%
Unknown	5	10	16	0	3	34	14.7%	29.4%	44.1%
Total	135	304	411	20	17	887	15.2%	34.3%	49.5%

too small for conclusive results, six of the eight players did have injuries in later seasons.

Of the 439 injuries with the player returning to the game, 10 players did not return to practice or play for 7+ days. Five players returned immediately, and 5 were rested and returned (*Table 5*) (13). These players included seven different positions. Five of the players had only one injury during the study period, and the other injuries were their first (of two, n = 2), second (of two), third (of three), and fourth (of four). Of the players with multiple MTBIs over time, only one had another injury during the same season (6 wk later). He was removed from play during that second injury. The 10 players who returned to the game and were out for 7+ days had, on average, more signs and symptoms compared with other injuries with the player returning to the game [3.10 versus 1.87, $t = 2.52$, $P = 0.012$]. Somatic complaints were more frequent in this group (50 versus 11.7%, $P = 0.004$), especially fatigue (30 versus 4.2%, $P = 0.009$). However, there was a low incidence of cognitive and memory problems in these 10 players.

Table 5 shows the management of players after concussion. The vast majority of players are rested after injury, although the period out from play can be short for those returning immediately to the game. The standard practice is that a player must stay out at least one play after the game is stopped for an on-field injury. A comparison of return to the same game and those removed from play or hospitalized showed no statistical significance to the action taken ($\chi^2 = 0.66$, $df = 4$, $P = 0.96$).

DISCUSSION

Return to Same Game

The results of this study demonstrate that the safety concerns regarding return to play on the same day as the MTBI may be unfounded at times. None of the players identified with MTBI who returned to play on the same day as they were injured experienced any intracranial catastrophe (subdural hematoma or other hemorrhage, cerebral edema, etc.) or developed second-impact syndrome. Compared with the players who did not return to play on the day of injury, those who returned to play on the day of injury had no statistically increased risk of subsequently developing a prolonged postconcussion syndrome (being out of play for 7+ days) or a repeat concussion. The data thus suggest that, in the NFL environment, it may be safe for athletes who sustain MTBI to return to play on the same day if they become asymptomatic, have normal neurological examinations, and are cleared to return to play by their team physician.

The authors used a very broad and inclusive definition of MTBI to capture as many cases as possible. Such mild cases would be expected to often clear completely in a brief period of time. Thus, the large number of injured players who returned to play on the same day as the injury did not come as a surprise to the authors. Despite efforts to include all cases of MTBI, some very mild cases were probably not included because the injured players did not report their symptoms to the team physicians or trainers. Thus, there were probably an

TABLE 4. Signs and symptoms of concussion by action taken in the National Football League, 1996–2001[a]

Signs and symptoms	Action taken, no. of cases (n = 887)						χ^2 or FET comparison	
	Return immediately (n = 135)	Rest and return (n = 304)	Removed from play (n = 412)	Hospitalized (n = 20)	Unknown (n = 16)	Total		
General symptoms	68	176	296	19	12	571	28.46	0.000
Headaches	55	160	267	16	11	509	24.31	0.000
Nausea	11	21	40	4	2	78	2.29	0.130
Vomiting	2	2	11	0	0	15	3.44	0.060
Neck pain	11	24	68	11	2	116	20.36	0.000
Back pain	0	2	1	0	0	3	FET	1.000
Syncope	3	4	4	2	0	13	0.06	0.080
Seizures	0	0	1	0	0	1	FET	0.500
Somatic complaints	11	44	108	10	5	178	29.91	0.000
Irritability	0	10	14	1	1	26	1.11	0.290
Anxiety	2	12	28	3	2	47	7.06	0.008
Depression	0	1	2	0	0	3	FET	0.620
Personality change	3	9	30	3	0	45	10.69	0.001
Fatigue	8	13	52	6	2	81	19.72	0.000
Sleep disturbance	2	0	3	1	0	6	FET	0.450
Loss of libido	0	0	0	0	0	0	FET	
Loss of appetite	1	1	0	0	0	2	FET	0.500
Cranial nerve effects	68	157	227	13	9	474	1.62	0.200
Dizziness	57	127	176	11	3	374	0.17	0.680
Vertigo	4	6	21	2	0	33	5.54	0.019
Tinnitus	2	7	13	1	0	23	1.20	0.270
Nystagamus	0	1	6	1	1	9	4.64	0.031
Hearing loss	0	0	0	0	0	0	FET	
Diplopia	3	7	7	0	1	18	0.49	0.483
Photophobia	4	8	20	3	1	36	3.79	0.052
Blurred vision	8	41	83	5	5	142	13.93	0.000
Pupil response	1	1	3	1	0	6	FET	0.450
Pupil size	0	0	1	0	0	1	FET	0.500
Cognition problems	6	49	164	14	4	237	91.37	0.001
Not oriented to person	0	4	18	2	0	24	11.24	0.007
Not oriented to place	0	7	31	7	0	45	23.05	0.000
Not oriented to time	1	13	45	9	0	68	26.22	0.000
Immediate recall	5	43	151	13	4	216	86.38	0.000
Memory problems	17	84	215	15	5	336	84.48	0.000
Attention problems	5	26	76	9	1	117	30.01	0.000
Information processing	11	26	103	6	1	147	44.06	0.000
AGA delayed	4	17	54	4	1	80	19.71	0.000
RGA delayed	2	35	105	8	2	152	48.01	0.000
Unconsciousness (≥1 min)	0	0	12	2	0	14	14.46	0.000
All loss of consciousness	2	7	40	9	1	59		
All reported as zero	62	95	125	4	9	295		
Mean no. of symptoms	1.52	2.07	3.51	6.55	2.50	2.76	$t = 1.29$	$df = 717$
								$P = 0.000$

[a] FET, Fisher's exact test; AGA, anterograde amnesia; RGA, retrograde amnesia. Under comparison, if no χ^2 is given, FET was used.

TABLE 5. Management of players with concussion by action taken in the National Football League, 1996–2001

Management	Action taken								
	Return immediately	Rest and return	Removed from play	Hospitalized	Unknown	Total	% Return immediately	% Rest and return	% Either
Immobilization			5			5	0%	0%	0%
Therapeutic modality		4	6		1	11	0%	36.4%	36.4%
Prescription drug therapy		5	5			10	0%	50.0%	50.0%
Proprietary drug therapy	1	3	3			7	14.3%	42.9%	57.1%
Rest	121	279	369	20	14	803	15.1%	34.7%	49.8%
Medical procedures		5	4			9	0%	55.6%	55.6%
Unknown	13	8	20	0	1	42	31.0%	19.0%	50.0%
Total	135	304	412	20	16	887	15.2%	34.3%	49.5%

even larger number of players who returned to play (or continued to play without being evaluated by medical personnel) on the day of injury than are reported here. No subdural hematomas or other catastrophic brain injuries, second-impact syndrome, or any other serious intracranial sequelae were reported in any NFL players during this 6-year period.

All players who reported any symptoms of MTBI to the team athletic trainers or physicians were included in this study. They were all examined by team physicians soon after the injury. Only players who were asymptomatic and had normal examination findings were allowed to return to play.

The general scenario for the immediate return-to-play group was as follows. The player sustained a blow to the head resulting in signs and/or symptoms of MTBI. The player was removed and evaluated by team medical personnel. Over a very brief period of time, the player's symptoms resolved. The physician's examination, including neurological and mental status testing of the player, was normal. The player was cleared to return to the game and resumed play the next time he was due back on the field. The general scenario for the rest-and-return-to-play group differs only in degree, i.e., the players returned to the game on the day of injury but only after a longer time interval had elapsed. When these players were initially evaluated, they still had symptoms of MTBI and/or manifested abnormalities on the neurological examination. Because they were not asymptomatic and/or had abnormal physical signs, they were not allowed to return to play immediately. They continued to be monitored by team medical personnel. At some point in time, while the game was still in progress, they became asymptomatic with a normal neurological examination. They were then medically cleared to return to play.

The symptoms that these players reported (and/or the signs they manifested) immediately after the injury were recorded on the examination form for reporting of MTBI events. These players were monitored by medical personnel over the ensuing hours and days. If, at a later time, the player reported that the MTBI symptoms had recurred, he was again evaluated, and the findings were reported. In this scenario, the player was not allowed to return to play again until these recurrent symptoms and/or signs had resolved. If the time interval for this total resolution was 5 days or more, it is very likely that the player was held out of the next week's game, thus joining the 7+ days out group. It is hoped that this detailed explanation of the process will help the reader understand how, if only asymptomatic players were cleared to return to play, a player can return to play on the same day as the injury yet subsequently end up in the 7+ days out group. It is also hoped that this explanation will help the reader to understand how players can be reported as having certain signs and/or symptoms of MTBI in the database but were allowed to return to play, asymptomatic with normal examinations, on the same day as the injury.

This article describes a unique group of NFL players who sustained MTBI. This is the only report of which the authors are aware that documents the signs and symptoms and outcomes of a large group of professional football players who sustained what could be considered to be the very mildest forms of MTBI. The fact that these players became asymptomatic and had normal examinations shortly after injury is what allowed them to be medically cleared to return to play that same day. This also attests to the relatively mild nature of their concussive injury. The data demonstrate that these players had fewer signs and symptoms than players who were kept out of play for longer periods of time (*Table 4*). This is yet another sign of the relatively mild nature of their injuries.

Nevertheless, even among this group at the mildest end of the MTBI spectrum, there were a few players (n = 10) who later developed a recurrence of symptoms that lasted long enough to result in their missing more than 7 days of play

after the initial injury and quick recovery. Analysis of this small group of players suggests that they had a higher incidence of somatic complaints, such as fatigue, on their initial evaluations than the players who did not develop recurrent symptoms resulting in missing more than 7 days of play. The authors suggest caution in interpreting this finding. Fatigue is a vague, nonspecific symptom, especially when reported on initial evaluation shortly after MTBI. It is doubtful that the complaint of fatigue in this setting has any prognostic significance. There were no other significant differences between this group of 10 players and the other players who returned to play on the same day as the MTBI. The present results point to the importance of follow-up medical evaluation in the days after initial MTBI, even in those players who had returned to play on the day of the original injury.

Signs and Symptoms

This article describes in detail the group of NFL players who returned to play on the same day as their MTBI. The description of the signs and symptoms experienced by this group of athletes does not necessarily lend prognostic significance to these signs and symptoms. Nevertheless, there are a number of differences between this group and the group of NFL players who did not return to play on the same day as their MTBI. The mean number of signs and symptoms reported is lowest in the players who returned to play immediately, somewhat higher in the players who rested and returned later in that game, and highest in those who did not return to play that day. This suggests that the mean number of total MTBI symptoms can be used to differentiate between various levels of MTBI severity. This finding is consistent with results of an earlier article from the present authors, which demonstrated that players in the 7+ days out group had a statistically significantly higher mean number of signs and symptoms on initial evaluation than those players who were out for less than 7 days (13). This present article indicates that the statistical association holds up for the milder end of the MTBI spectrum as well. In other words, players who returned to play immediately after MTBI had a statistically lower mean number of symptoms and signs on initial evaluation than those players who did not return immediately and those who did not return that day. There were also some differences in the incidence of specific signs and symptoms on initial evaluation between players who returned to play the same day and those who were removed from all play on the day of injury.

The players who returned to play on the day of injury had a statistically significantly lower incidence of cognitive and memory impairments than those who did not return to play that day. This suggests that cognitive and memory impairments are less likely to resolve completely within minutes to hours than many of the MTBI symptoms that were evaluated. This could indicate that cognitive and memory impairments are indicators of more serious injury than the other signs and symptoms evaluated. It could also indicate that cognitive and memory impairments do not usually occur in isolation and thus are more likely to be associated with a higher mean number of signs and symptoms than such symptoms as headaches or dizziness.

A review of the list of signs and symptoms of MTBI included in this analysis indicates that there are eight different signs and symptoms included under categories of cognitive and memory dysfunction. Clinical experience has shown that players who sustain MTBI who have cognitive and memory dysfunction will have many (not just one or two) of these eight specific signs and symptoms. Therefore, they will probably fall into the group with a relatively higher mean number of signs and symptoms. Athletes who experience headache or dizziness, conversely, will be recorded as having only one abnormal symptom, because the subcategories of different headache or dizziness subtypes are not included on the report forms. Thus, the finding that players with the smallest mean number of signs and symptoms and without cognitive/memory impairments are most likely to return to play on the day of injury may actually be a different way of expressing the same result.

Players at Higher Odds Ratio for Return to Play in the Same Game

Punters and defensive linemen are more likely to return to play on the day of injury than players in other positions. This may be related to the players in these positions being less likely to be involved in the very-high-speed-acceleration impacts that cause the most serious MTBIs (12–14). NFL defensive linemen are less likely to sustain MTBI than other players such as wide receivers, defensive backs, quarterbacks, or special team players, presumably because they are less likely to be involved in high-speed impacts. Punters are also less likely to be involved in high-velocity-acceleration head impacts. Although they sometimes are blocked or make a tackle well after they have punted the ball, many of their impacts are sustained while they are kicking the ball. Opposing players are charging at the punter to block the ball and thus aim at the lower part of the punter's body or at an area in front of the punter rather than at the punter's head. In addition, contact with the punter while he is kicking the ball is usually penalized. As a result of these factors, head impacts are inadvertent and unlikely to involve high speeds. The punter's low risk of more serious MTBI stands in contrast to that of the quarterback, who is also often a relatively stationary player being charged at by opponents. The one punter with an MTBI and out 7+ days from play experienced his injury during practice and after the punt on the kick return.

Although it is illegal to hit quarterbacks after they throw the ball, they are fair game before the ball is released. Thus, unlike punters, quarterbacks are often hit while they are in a defenseless position. Furthermore, defensive players are attempting to disrupt the passing play by hitting the quarterback, blocking the ball, or at least obstructing the quarterback's line of sight. Because the quarterback holds the ball at or above shoulder

level while attempting a pass, defensive players aim their charge at the upper part of the quarterback's body. Even though blows to the head of the quarterback are illegal and penalized, the nature of the game nevertheless predisposes the quarterback to high-velocity-acceleration head impacts. The present results also indicate that MTBIs occurring during kickoffs and passing plays are the least likely to result in the affected player's being cleared to return to play on the same day. Passing and special teams plays involve the highest-speed-acceleration impacts of all NFL plays and the highest frequency and most severe MTBI of all NFL plays.

One result is more difficult to explain. MTBIs occurring during interceptions are more likely to result in return to play on the day of injury than those occurring during any other type of play. Interception plays are nothing more than passing plays gone awry and should involve high speeds and accelerations. However, this is not really the case. When an interception occurs, players suddenly change direction, often after coming to a full stop or at least slowing down. Player velocities thus diminish and gradually reaccelerate. In addition, after an interception, players must change their usual manner of play. Offensive players become tacklers and defensive players become blockers and ball carriers. Players are less likely to be as efficient and hard hitting in these unaccustomed roles. Furthermore, because interceptions usually occur well after the initial play has begun, players' initial bursts of speed have diminished, and some fatigue may have developed. For all of the above reasons, head impacts after interceptions occur at lower velocities than during most passing plays, thus resulting in milder MTBIs.

Concussion Management Guidelines

Widely used concussion management guidelines state that athletes can return to play on the day of injury if they become asymptomatic and have normal examinations within 15 minutes of their injury (1, 4). In the NFL database, 41% of players returning to the same game either immediately or after resting were out from play more than 15 minutes, 17.9% of those who returned immediately were out more than 15 minutes, and 51.7% of those who rested and returned were out more than 15 minutes. Because no adverse effects were found with the return to play in the same game, it may be safe for NFL players who are asymptomatic and have normal neurological examinations to return to play on the day of injury, irrespective of the time taken to become asymptomatic.

The data showed no increased risk of repeat MTBI, prolonged postconcussion syndrome, delayed return to play (7+ days out), second-impact syndrome, or catastrophic intracranial event in this group of players. These results seem to confirm the validity of the above guideline recommendations for players' symptoms that clear within 15 minutes; however, many of the NFL players who returned to the same game were symptomatic longer than 15 minutes. The median time to return to play for players rested and returned was 17 minutes; 51.7% of these players did not become asymptomatic with normal examinations for more than 15 minutes after injury. The present data suggest that NFL players in this category also did not have any increased risk of repeat MTBI, prolonged postconcussion syndrome, delayed return to play (7+ days out), or catastrophic intracranial events compared with the immediate-return-to-play group or the group who were not cleared to return to play on the day of injury.

The NFL experience thus suggests that players who become asymptomatic with normal examinations at any time after injury, while the game is still in progress, have been and can continue to be safely returned to play on that day. This indicates that the "15-minutes rule" in the current guidelines may be too conservative for the NFL. Many of the currently accepted guidelines also indicate that any player who experiences loss of consciousness with MTBI should not be allowed to return to play that day (1, 4, 17). Although the numbers were small, there were a few players in this study who had recorded loss of consciousness as a result of MTBI and later returned to play in the same game. There was no evidence of any adverse effect of this action. These data suggest that these players were at no increased risk of repeat MTBI or prolonged postconcussion syndrome compared with other players. None of these players developed a catastrophic central nervous system event or second-impact syndrome. Whether the guideline recommendations, which clearly state no return to same-day action after loss of consciousness, are too conservative for NFL players will require further analysis because of the limited cohort and the difficulty to determine the actual duration of unconsciousness in this study. It may also be an indication that loss of consciousness does not predict the severity of a concussion (8).

The ultimate goal, as noted in our previous articles as well as in the extensive literature on concussion in sports, is to help formulate more definitive guidelines for the safe and timely return of an athlete to a collision or contact sport. The recent literature, which seems to be the most evidence-based to date, suggests that it is inadvisable to return a high school football player with any concussive symptoms or signs to a game on the same day or within a week for fear of causing a more serious brain injury (7). An article from the National Collegiate Athletic Association Concussion Study suggests similar concerns with college-level players (9). Another recent article with prospective data suggests that at the college level, recurrent concussions during the same season seem more prevalent and recovery slower from a recurrence (6). Our study, which was prospective and NFL team physician-monitored from 1996 to 2001, did not reveal the concerns found at the high school and college levels.

Limitations

These limitations follow those described by Pellman et al. (11, 13, 14). The MTBI Committee did not collect retrospective data on previous concussion history as part of the study. Some of the players may have had previous concussions either in the NFL in the years before the study began or during their

playing careers in high school, college, or other levels of football. It is also possible that some of the players sustained cerebral concussions at earlier times in their lives in nonfootball athletic or nonathletic endeavors.

There was difficulty collecting data on loss of consciousness. Many of the reports did not answer the question on loss of consciousness, so we do not have definitive loss-of-consciousness data on a certain number of players. What has been reported are the patients with a known time of unconsciousness and those patients reporting a zero or no loss of consciousness.

In a multisite study such as this, there are numerous different examiners. In some cases, different examiners from a given medical staff may evaluate that team's players. There was no uniform method of evaluation of concussion in this study, which will give rise to variability in assessments among the 30 teams and, on occasion, within the same team. It must be emphasized that players were not cleared to return to play until they were asymptomatic with normal neurological examinations and able to return to full, unrestricted participation in team activities. There were some players who returned with headaches.

Although the medical condition of the player certainly is the most important factor in determining return-to-play decisions by team physicians, there are many other factors that go into the decision of when the player should return to play. The importance of the player to the team; the importance of the game to the team; and pressure from owners, players and their families, coaches, agents, and media certainly may influence the decision of when the player returns to play. The authors believe, however, that the medical factors regarding the patient's recovery are and should be the overriding factors that guide the team physicians' decision-making on return to play. Furthermore, our results apply to NFL-level players, and extrapolation to younger athletes has not been demonstrated; thus, differences may exist between MTBI in high school and professional athletes.

There are a few other limitations that are more specific to this analysis. Some players with very mild MTBIs may not have come out of the game or reported the event to team trainers or physicians. These players would have continued to play in the game. The fact that this group of players did not come to medical attention indicates that they did not develop any adverse events as a result of continuing play. Had such players been involved in this analysis, the number of players who returned or continued to play on the day of the injury would have been even higher. This would only have strengthened the conclusions reached, and this potential limitation does not detract from the results.

Most of the players were examined only on the sideline before returning to play. By their very nature, such sideline evaluations are not as comprehensive as formal neurological examinations performed in the training room or in the physicians' office. Nevertheless, these were not cursory examinations and, on the basis of the outcomes reported in this article, were more than adequate to allow the physicians to accurately determine which players could safely return to play. This potential limitation also does not detract from the conclusions.

A final potential limitation is that the data do not reveal how many plays the returning players participated in after being cleared to return to the game on the day of the injury. In other words, there are no data on the actual exposure to repeat MTBI or other injury of the players who were cleared to return to play on the same day as an MTBI. A regular offensive or defensive player who was injured early in the first quarter of a game and cleared to return to play immediately would be exposed to almost a complete game's worth of risk of repeat injury. Conversely, a purely special teams player who was injured in the fourth quarter and cleared to return to play with only a few minutes remaining in the contest might have been involved in only a few or no further plays before the game ended, thus having only a very small or no risk of repeat MTBI that day. The authors believe that because of the large number of players involved in this study, these variations in risk of subsequent MTBI on the day of injury would tend to average out, thereby not significantly affecting the results.

CONCLUSION

The results of this article indicate that many NFL players can be safely allowed to return to play on the day of injury after sustaining an MTBI. These players had to be asymptomatic, with normal clinical and neurological examinations, and be cleared by a knowledgeable team physician. There were no adverse effects, and the results stand in contrast to the recommendation of published guidelines (1, 4) and the standard practice of most college and high school football teams. The authors are not suggesting that college and high school football players be cavalierly allowed en masse to return to play on the same day that they sustain an MTBI. The authors are aware of significant differences between professional and college/high school football players (e.g., age differences, possible differences in propensity to MTBI, differences in training, etc.).

We are also aware of differences in the extent and level of medical supervision between the professional and college/high school games. However, it is also true that, to the best of our knowledge, there have been no articles published analyzing the outcomes of college/high school players who return to play on the day of MTBI. It is possible that such studies might find results similar to those presented here. Thus, under the right circumstances, specifically with regard to final decision making on return to play being solely at the clinical discretion of a knowledgeable team physician, it might be safe for college/high school football players to be cleared to return to play on the same day as their injury. The authors suggest that, rather than blindly adhering to arbitrary, rigid guidelines, physicians keep an open mind to the possibility that the present analysis of professional football players may have relevance to college and high school players.

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COMMENTS

This additional report from the National Football League's (NFL's) Committee on Mild Traumatic Brain Injury (MTBI) studies the characteristics of those players who were returned to play on the same day as their concussive brain injury. During a 6-year period in which a total of 887 concussions were identified, they analyzed 439 players, of whom 135 (15.2%) were returned immediately and 304 (34.3%) rested and later returned to the same contest. Overall, there were no substantial differences in those athletes who were returned to play, but there were progressively fewer symptoms compared with those players who did not return to play or those who subsequently required hospitalization.

A study of this magnitude has some inherent limitations, as the authors acknowledge. However, this is an interesting analysis that demonstrates that, at least in the acute phase and during their active playing years, these athletes seem to perform well without a risk for intracranial hemorrhage or a later high incidence of recurrent concussion or postconcussion symptoms. Only 12% of those football players who returned to play the same day had a second concussion within the same season, and the propensity for such was similar regardless of the management decisions. It is of interest to note that of eight players who experienced a loss of consciousness (LOC) and later returned to the same game, six sustained similar injuries in subsequent seasons; this constitutes an insufficient sample size to draw definitive conclusions about the implications of LOC and same-day return.

There were 10 players who, even in this most mild form of brain injury, developed recurrent symptoms. They were unable to be identified by presenting symptoms, with fatigue being the only complaint on initial evaluations, a symptom that the investigators caution about using. The authors are telling us that their management protocols are valid and appropriate for use in professional football players, including even returning the athlete to play without a 15-minute observation period or with having had an episode of LOC. As they point out, application of such policies at other levels of play for amateur or scholar athletes is currently unproven and may or may not be similarly recommended. In addition, no detailed neuropsychological testing or assessment of status after playing for years is reported. This is a provocative study that has been undertaken with great attention to detail. Its findings should be assessed and considered by all who take care of athletes in contact sports.

Julian E. Bailes
Morgantown, West Virginia

This is the seventh article in the most widely published and richly funded football concussion study to date. That such a long time, 6 years of collecting data and then 3-plus years of

analyzing data and writing, has occurred is highly commendable.

The conclusions cited in this article are supported by the data presented. However, I caution all readers to read very carefully the nearly two pages of limitations of the study that the authors, to their credit, candidly cite. I will not repeat them here, but after reflecting on them, one needs to ask oneself, I believe, are we dealing with a unique population and situation in these articles? Is it correct to extrapolate these data to the many previous concussion articles and consensus statements involving the high school and college scholar-athlete? Although the answer in my mind is no, not at this time, I believe that these data should make all of us participating in concussion research and consensus statements view data with an energized critical eye.

Multiple studies in the past several years have indicated that the incidence of concussion cited by the athlete questioned after the season is over is many times higher, four to seven times, than that currently reported by the team medical personnel (1–5). That most athletes do play through most minor concussions is supported by these studies. Although this does not change current consensus statements regarding management of concussion, such as the recent "Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004," which states that "when a player shows ANY symptoms or signs of a concussion, the player should not be allowed to return to play in the current game or practice," we realize that in the real world, this often does not happen.

Robert C. Cantu
Boston, Massachusetts

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This article represents the seventh and final contribution by the NFL Committee on MTBI on the various aspects of head injury in professional football. The present study evaluated the safety of returning concussed professional football players to the same game immediately or after a period of rest. Somewhat surprisingly, 49.5% of players were returned to the same game either immediately or after a period of rest. For those players returning to the same game, only 12% had a second concussion in the same season. As would be predicted, players who returned to the same game have significantly lower incidences of cognitive and memory problems than players removed from play or hospitalized. This is in keeping with the observations of Collins et al. (1), who found that impairments in these two areas were the most predictive factors confirmed by subsequent psychological testing of cerebral malfunction. They observed that athletes with on-field retrograde amnesia are 10 times more likely to have "poor" acute outcomes and athletes with on-field anterograde amnesia are 4 times more likely to have "poor" acute outcomes as measured by neuropsychological testing.

Additional observations suggest that players who become asymptomatic and have normal neurological examinations at any time after an MTBI may safely return to the game without waiting 15 minutes, as suggested by some guidelines. Contrary to most established guidelines, there were a few players who had recorded LOC and returned to play in the same game after regaining complete neurological and neurocognitive function with no subsequent neurological consequences.

This article essentially confirms that the practice by team physicians and trainers in the NFL of not allowing symptomatic or neurologically abnormal athletes to return to play in the same game is a safe practice. This policy did not result in any detectably higher incidence of repeat concussions, postconcussion syndrome, or cognitive impairment. This practice seems to be sound and is contingent upon a thorough neurocognitive and neurological assessment of the sidelined athlete and the experience and judgment of the team personnel in detecting at times subtle cues concerning neurological impairment or self-serving denial of symptoms by the athlete. In summary, the NFL Committee on MTBI, under the direction of Dr. Pellman, has made another significant contribution to the medical literature and confirmed the safety of the current management of concussions at the professional level.

Joseph C. Maroon
Pittsburgh, Pennsylvania

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Return-to-play decisions regarding athletes who sustain concussion can be difficult for the sports medicine team. Pellman et al., in Part 7, describe signs, symptoms, and management of NFL players who sustained concussion and returned to the same game during the 6-year period. The authors of this study conclude that the results of this NFL study differ from previous articles and did not reveal the same return-to-play concerns. The sports medicine team that provides medical care to high school and college athletes should probably still adopt a conservative approach. Second-impact syndrome, although not evident in the 6-year NFL study, is a concern with a younger population. I agree that an athlete who has sustained a concussion should not return to play until

asymptomatic. The main problem with a sideline evaluation is how sensitive the test is to determine whether the athlete is really asymptomatic, especially in adolescent athletes. Part of the sideline evaluation is subjective. A football player with a headache after MTBI is symptomatic regardless of how hard he tries to persuade you to allow him back into the game. The sideline examination should be thorough and should include a clinical examination, cognitive testing, and balance assessment. If at all possible, baseline scores should be accessible on the sideline for comparison. The team physician bears a heavy load of responsibility in making same-day return-to-play decisions with athletes who sustain MTBI, regardless of the level of play.

Russ Romano
Director of Sports Medicine, Head Athletic Trainer
University of Southern California
Los Angeles, California

TAB 1H

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CONCUSSION IN PROFESSIONAL FOOTBALL: BIOMECHANICS OF THE STRIKING PLAYER—PART 8

David C. Viano, Dr. med., Ph.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York, and ProBiomechanics LLC, Bloomfield Hills, Michigan

Elliot J. Pellman, M.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, and ProHEALTH Care Associates, LLP, Lake Success, New York

Reprint requests:
David C. Viano, Dr. med., Ph.D.,ProBiomechanics LLC, 265 Warrington Road, Bloomfield Hills, MI 48304-2952. Email: dviano@comcast.net

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OBJECTIVE: Concussive impacts in professional football were simulated in laboratory tests to determine the collision mechanics resulting in injury to the struck player and the biomechanics of the striking players, who were not concussed or neck-injured in the tackle.

METHODS: Twenty-seven helmet-to-helmet collisions were reconstructed in laboratory tests using Hybrid III dummies. The head impact velocity, direction, and kinematics matched game video. Translational and rotational head accelerations and six-axis upper neck loads and moments were used to evaluate how the striking player delivered the concussive blow. The neck injury criterion, *Nij*, was calculated to assess neck injury risks in the striking player.

RESULTS: The time-averaged impact force reached 6372 ± 2486 N at 7.2 milliseconds because of 46.8 ± 21.7*g* head acceleration and 3624 ± 1729 N neck compression force in the striking player. Fifty-seven percent of the load was contributed by neck compression. The striking players had their heads down and lined up the impact axis through their necks and torsos. This allowed momentum transfer with minimal neck bending and increased the effective mass of the striking player to 1.67 times that of the struck player at peak load. The impact caused 94.3 ± 27.5*g* head acceleration in the concussed players and 67.9 ± 14.5*g* without concussion ($t = 2.06$, $df = 25$, $P = 0.025$). The striking player's *Nij* was greater than tolerance in 9 of 27 cases by exceeding the 4000 N neck compression limit. For these cases, the average neck compression force was 6631 ± 977 N (range, 5210–8194 N). *Nij* was 1.25 ± 0.16 for eight cases above the tolerance *Nij* = 1.0.

CONCLUSION: In the NFL, striking players line up their heads, necks, and torsos to deliver maximum force to the other player in helmet-to-helmet impacts. The concussive force is from acceleration of the striking player's head and torso load through the neck. Even though neck responses exceeded tolerances, no striking player experienced neck injury or concussion. A head-up stance at impact would reduce the torso inertial load in the collision and the risk of concussion in the struck player.

KEY WORDS: Biomechanics, Concussion, Football, Impact tolerances, Neck injury, Spearing, Sport injury prevention

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Since the 1970s, there has been concern for head-down tackling, or spearing, which can result in catastrophic neck injuries in the striking player. This concern is generally related to head impacts into the tackled player's torso, in which the mass of the struck player's body increases the load in the striking player's neck. Neck flexion and lateral bending increase injury risks. This type of tackle can lead to compression-flexion or other compression-bending injuries in the striking player, with quadriplegia and death the most serious consequences (1, 7, 21, 25, 26, 49, 52).

The incidence of catastrophic neck injuries has been tracked for more than 30 years in a national registry of cervical spine injuries in football and other databases (4, 13, 20, 47, 51–55). These epidemiological and cinematographic analyses of neck injuries have shown that the majority of cervical fracture-dislocations are caused by axial loading. This has resulted in rule changes in high school, college, and professional football banning deliberate spearing and the use of the top of the helmet as the initial point of contact in a tackle (11, 12, 27, 28). The rule changes significantly reduced the incidence of cervi-

cal spine injuries by the late 1970s, with a continued decline until the present (14).

The injury statistics have also led to the use of isometric and resistance exercises to develop strong neck musculature and reduce injury risks. These exercises are part of preseason conditioning to prevent catastrophic head and neck injury. With stronger necks, more impact force can be delivered in tackles without injury; however, the greater tolerance of the striking player can have negative consequences in those struck. Furthermore, training in proper blocking and tackling techniques is given to reduce head-down spearing (9, 21).

Biomechanical studies have been conducted to assess neck loads causing fracture-dislocations during head-down impacts in tackling-dummy practice (24) and game collisions (3, 15, 16, 48). Neck compression forces greater than 4000 N are considered sufficient to seriously injure a player because of axial compression of the cervical spine. These and other studies have established injury tolerance criteria for neck loading that are used with test dummies to study injury risks in sports, automotive crashes, and other impacts (5, 18, 22, 43, 50).

This study is part of a larger series on concussion in professional football. The National Football League (NFL) has a Mild Traumatic Brain Injury (MTBI) Committee, which has undertaken research aimed at defining the biomechanics of concussive impacts in professional football (37). One aspect of the effort focused on the analysis of multiple views of concussive impacts from game video to determine the speed of impact. Laboratory reconstructions of the collisions were performed using instrumented test dummies to simulate the helmeted players.

The laboratory re-enactments closely matched the field situation. With transducers in the dummy, the translational and rotational accelerations of the head and neck loads in the striking player allowed an evaluation of biomechanical responses during concussive impacts. This article evaluates the impact biomechanics of the striking players who are not concussed or neck-injured in the collisions; it also describes the collision biomechanics resulting in concussion of the struck player.

This study points out a new concern with head-down tackling, which is concussion of the struck player. This type of tackle can lead to injury in the struck player with little risk of head or neck injury in the tackler, because only the head and neck of the struck player initially resist the impact, and the striking player lines up his head, neck, and torso. This study addresses the biomechanics of the striking and struck players with head-down tackling in NFL helmet-to-helmet collisions causing concussion.

MATERIALS AND METHODS

Video Analysis of NFL Game Impacts

Details of the game film selection and analysis can be found in the studies by Pellman et al. (38, 39). For this study, a short overview of the laboratory methods is provided. When an MTBI occurred on the field during an NFL game, it was reported to Biokinetics and Associates, Ltd. (2470 Don Reid Drive, Ottawa, ON K1H 1E1, Canada), the engineering group contracted to analyze and reconstruct game impacts. Network tape of games was obtained from the NFL and subsequently analyzed. In addition to concussion impacts, other cases of significant head impact were selected for analysis. These were determined by NFL films. During the period 1996 to 2001, 182 cases were obtained on video for analysis. The initial analysis determined the impact location on the helmet and the contact source (helmet, ground, shoulder, etc.); 61% of the collisions involved helmet-to-helmet contacts (39).

Biokinetics determined the feasibility of determining the three-dimensional impact velocity, orientation, and helmet kinematics. At least two clear views were necessary to make this analysis. For those videos in which the three-dimensional impact velocity could be analyzed, a laboratory setup with crash dummies was made to re-enact the game impact. Helmets were placed on the dummies in the laboratory reconstructions, and the velocity and orientation of impact were simulated along with the subsequent helmet kinematics. A number of significant impacts were also reconstructed in which MTBI did not occur to study nonconcussion impacts. In total, 27 NFL helmet-to-helmet collisions were reconstructed; 22 involved concussion of the struck player, and 5 involved no injury. There was no injury to the striking players.

Laboratory Reconstruction Techniques

Figure 1 shows the reconstruction setup, which involved two Hybrid III male dummies (2). A helmeted head-neck assembly representing the struck player was attached to a 7.1

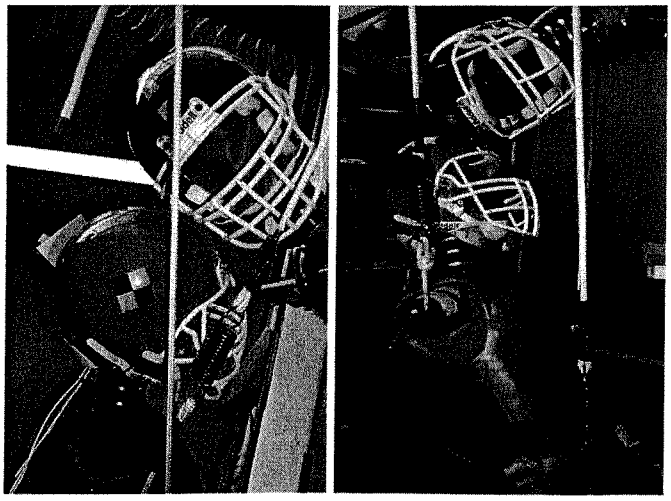

FIGURE 1. *Photographs showing reconstruction of game impacts in laboratory tests with instrumented dummies* (left, *Test 39, and* right, *Test 162). The torso and pelvis of the striking player were suspended from below, and the struck player was simulated with the head and neck attached to a 7.1 kg drop weight. Adjustments were made in the setup to duplicate the helmet kinematics in the game impact. The tests involved VSR-4 helmets by Riddell.*

kg mass simulating the struck player's torso and guided in free fall from a height to match the impact velocity determined from video analysis of the game collision. The Hybrid III head and neck weighed 4.38 kg with instrumentation. The helmet and face mask weighed 1.92 kg, and the falling mass was 15.1 kg. Impact was against another helmeted head-neck assembly attached to the torso and pelvis of the Hybrid III dummy. This dummy weighed 46.4 kg without arms and legs and was suspended by flexible cables.

Acceleration was measured in both dummy heads. The center of gravity (cg) of the head is a reference point, which is defined by its position in three orthogonal axes. The motion of the head cg is defined by three orthogonal components of velocity and acceleration. The acceleration is translational, even though the trajectory is curvilinear. As the head cg moves in space under translational acceleration, it can also rotate about the head cg. This involves rotational acceleration, and there are three orthogonal axes for rotational acceleration and velocity. When the head is assumed to be rigid, as in the dummy, the three axes of translational and rotational acceleration define the motion sequence of the head during impact. The sign convention used in this study has neck compression as $-Fz$ and neck tension as $+Fz$, because the positive z axis is from the neck upward through the top of the head (46). The positive x axis is forward, and the positive y axis is through the left ear. Neck extension is $-My$, and flexion is $+My$.

Each head form was equipped with standard accelerometers at the head cg and nine linear accelerometers set up in a so-called "3-2-2-2 configuration" to determine rotational acceleration (36). The analysis is valid for accelerometers coincident with the origin of head cg or coincident with one of the axes. Deviations from this were required in the head-form configuration used in these tests, and a correction for centripetal and Coriolis acceleration was made (6).

The dummy representing the striking player had a six-axis neck transducer installed between the head and the top of the neck. The transducer measured three axes of neck force (Fz, compression-tension; Fx, fore-and-aft shear; and Fy, left-to-right shear) and three axes of neck moment (My, flexion-extension; Mx, lateral bending; and Mz, rotation about the z axis).

High-speed video recorded head kinematics in the reconstruction. The camera was positioned similarly to one of the views from the game video. This allowed a one-to-one comparison of the game and reconstruction kinematics and facilitated fine adjustments in the impact orientation and alignment of the laboratory impacts to closely match the helmet kinematics in the game (39).

Extensive testing was conducted to isolate and quantify sources of error and variability in the reconstructions (30). This work showed the reconstructions to be repeatable and with minimal error for this type of testing. In the laboratory reconstructions, every effort was made to reduce potential sources of error.

Game Impacts

Figure 2 shows the initial helmet contact points determined from game video. The *top* shows the location for the struck players, who were either concussed or not concussed, and the *bottom* shows the striking players, none of whom were injured in the collisions. The impacts are shown on the right side of the helmet, although contacts occurred on both sides. More than half of the impacts involved the face mask or area in which the face mask attaches to the helmet shell of the struck player, whereas virtually all striking players (26 of 27 cases) involved the front crown or top portion of the helmet, because their head was down and the axis of impact was through their head

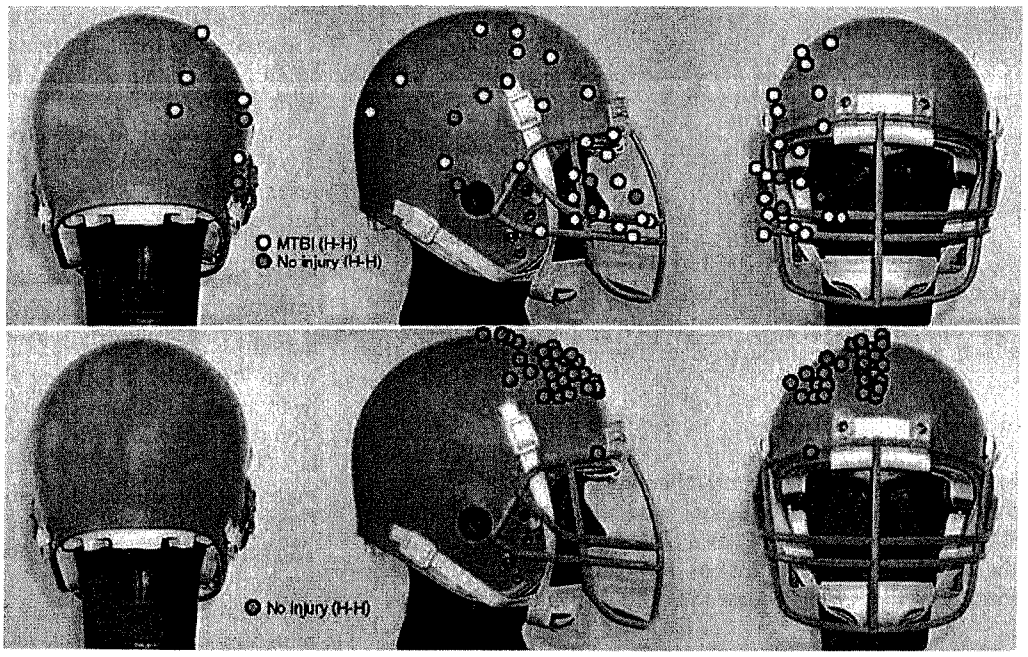

FIGURE 2. *Photographs showing location of initial helmet contacts for the struck players* (top, *both concussive and nonconcussive impacts) and striking players* (bottom, *none of whom were concussed). All of the strikes in the plot are shown on the right side of the helmet to visualize the impacts, although the game impacts occurred on both sides of the helmet.* MTBI (H-H) *indicates a concussed player involved in a helmet-to-helmet impact (modified* from, *Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF: Concussion in professional football: Reconstruction of game impacts and injuries.* ***Neurosurgery*** *53:799–814, 2003 [39] by removing the helmet-to-ground impacts).*

cg, neck, and torso. Deviations from this alignment in game impacts and laboratory tests caused the helmets to slide off because of the smooth plastic shell of the helmets. This dramatically lowered the impact responses.

Collision Biomechanics

Impact force (F) from the striking player was determined by adding the head inertia force of the striking player and neck compression force:

$$F = m_{\text{Striking}} a_{\text{Striking}} + F_N \quad (1)$$

where a_{Striking} is the resultant acceleration of the striking player's head, F_N is the resultant neck compression force, and m_{Striking} is the mass of the striking player's head above the neck load cell. The mass was $m_{\text{Striking}} = 5.90$ kg and included the Hybrid III head (3.64 kg), the load cell above the sensing element (0.34 kg), and the helmet with face mask (1.92 kg). Mass below the sensing element is not included in Equation 1, because the striking player's neck load was measured. In the collision, the striking player used the top or crown portion of the helmet. This area is substantially stiffer than the side or face mask region of the helmet.

The impact force is equilibrated by the struck player. His head, neck, helmet, and a portion of the torso are involved:

$$F = m_{\text{Struck}} a_{\text{Struck}} \quad (2)$$

where a_{Struck} is the resultant acceleration of the struck player's head. The mass of the struck player is $m_{\text{Struck}} = 8.40$ kg and includes the head (4.38 kg), neck (1.06 kg), helmet and face mask (1.92 kg), and a portion of the torso mass (1.04 kg). The difference in the Hybrid III head mass between the striking and struck players reflects the full weight of a bracket that is used in place of the neck load cell.

Head acceleration of the striking player is lower than that of the struck player, so the effective mass of the striking player is greater than that of the struck player. The neck load cell measures the contribution from the torso mass in the collision, which adds to the impact force. The effective mass of the striking player is

$$m_{\text{Eff.Striking}} = F / a_{\text{Striking}} \quad (3)$$

On the basis of the average head acceleration and impact force, $m_{\text{Eff.Striking}} = 14.0$ kg, indicating a mass ratio of $m_{\text{Eff.Striking}} / m_{\text{Struck}} = 1.67$, or a 67% greater effective mass of the striking player than that of the struck player during peak force. The mass ratio equals the ratio of head accelerations:

$$a_{\text{Struck}} / a_{\text{Striking}} = m_{\text{Eff.Striking}} / m_{\text{Struck}} \quad (4)$$

where the relationship assumes that a single mass is involved in the head impact for each player. The impact force and other biomechanical responses, including head accelerations and changes in velocity (ΔV), describe the collision mechanics leading to concussion in the struck player.

Head Injury Tolerances

The primary response of the head is the resultant translational acceleration of the head cg. This was determined from three orthogonal accelerations measured in the dummy. Although translational acceleration is measured in units of m/s^2, it is reported in units of gravity (g), where the measured acceleration is normalized by the acceleration of gravity ($1g = 9.8\ m/s^2$). Integration of the resultant acceleration gave the change in head velocity, or ΔV, during impact.

For the head impacts, the resultant acceleration is used to calculate two head injury criteria. The National Operating Committee on Standards for Athletic Equipment (29) football helmet standard uses the severity index (SI), which is determined by the method of Gadd (10):

$$SI = \int^{T} a(t)^{2.5} dt \quad (5)$$

where $a(t)$ is the resultant translational acceleration at the head cg and T is the duration of the acceleration. The National Highway Traffic Safety Administration (NHTSA) uses a variation of SI to assess head injury risks in car crashes. The head injury criterion (HIC) is determined by

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)]^{2.5}\}_{\max} \quad (6)$$

where t_1 and t_2 are determined to maximize the HIC function and $a(t)$ is the resultant translational acceleration of the head cg. In practice, a maximum limit of $T = t_2 - t_1 = 15$ milliseconds is used.

The second type of biomechanical response of the head involves rotational acceleration and rotational velocity. Many researchers have speculated that rotational acceleration is a key response associated with head injury (35).

Neck Injury Tolerances

The early neck tolerance for axial compression was estimated by using a Hybrid III dummy to measure neck loads when struck by a tackling block that had produced serious head and neck injuries in football players (24). The compression tolerance varied with load duration but was estimated to be 4000 N. Neck tension and shear load tolerances were estimated using the Hybrid III dummy in reconstructions of three-point belted occupant injuries in frontal car crashes (34). The limits for tension and shear force were 3300 and 3000 N, respectively.

Tolerance levels for neck flexion and extension were estimated by use of sled tests of volunteers and cadavers (23, 45). Volunteer tests provided data up to the pain threshold for extension bending moments, and cadaver tests estimated tolerance limits for serious injuries at 57 Nm. The maximum voluntary flexion moment of 190 Nm was set as the tolerance limit. The bending moments were based on human responses, rather than dummy measurements. Cadaver tests on neck

tension showed failure at 3373 N (56); however, lower forces were found for combined loading conditions of tension-extension (44).

Kleinberger et al. (19), working with the NHTSA, reviewed earlier studies and developed a neck injury criterion, *Nij*, in which the "*ij*" indices represent four injury mechanisms: tension-extension (*Nte*), tension-flexion (*Ntf*), compression-extension (*Nce*), and compression-flexion (*Ncf*). The criterion emphasizes injury risks for sagittal plane motion. Crash tests using a six-axis upper neck load cell in the Hybrid III dummy in frontal crashes established limits for flexion-extension bending (*My*) and tension-compression force (*Fz*). Shear load (*Fx*) was used to calculate the effective moment at the occipital condyles by multiplying the shear load by the height of the load cell above the condyles and subtracting this value from the measured *My*.

Nij is calculated as a function of time by normalizing *My* and *Fz* with intercept tolerances for extension, flexion, tension, and compression. The normalized flexion-extension moments are added to the normalized axial loads to give *Nij*:

$$Nij = (Fz/Fzc) + (My/Myc) \quad (7)$$

where *Fzc* is the critical intercept for axial neck loading and *Myc* the critical intercept for flexion-extension bending moment at the occipital condyles. The critical intercepts are *Fzc* = 6806 N for tension, *Fzc* = 6160 N for compression, *Myc* = 310 Nm for flexion, and *Myc* = 135 Nm for extension. The neck extension intercept is substantially higher than the earlier 57 Nm estimate by Mertz and Patrick (23).

During an impact, all four combinations of neck response need to be below *Nij* = 1.0. In addition, peak neck tension cannot exceed 4170 N and compression 4000 N. The *Nij* criterion is consistent with information from experimental and laboratory studies (17, 31–33, 40–42).

Establishing Time Zero to Align the Data

The following procedure was used to align time zero for the individual cases, because the orientations of the collisions and timing varied between tests. A "soft trigger" was used to determine the start of head acceleration. For most cases, a 1*g* trigger was used to determine the start of the impact; however, some tests had noise on the responses requiring a 3*g* (Tests 7, 38, 39, 48, 59, 69, 84, and 92) or 5*g* trigger (Test 77). The responses presented here were based on the time-zero adjustments to align the impact responses by assuming that time zero occurs when the head acceleration surpasses the soft trigger.

Statistical Analyses

The significance of differences in responses for the striking player causing concussion and no concussion were determined using *t* tests assuming unequal variance and a single-sided tail distribution. If Levine's test suggested inequality of variances, a *t* test with adjustment for unequal variances was used. The *t* tests were performed using SPSS 11.5 for Windows (SPSS, Inc., Chicago, IL). The regression analysis was also used from Excel (Microsoft, Seattle, WA), which determined the average and 95% confidence interval for a linear fit between response data.

RESULTS

Biomechanics of the Striking Player

Table 1 shows the peak responses for the 27 NFL helmet-to-helmet collisions reconstructed in laboratory tests. The average impact speed for these collisions was 9.3 ± 1.9 m/s. The peak resultant head acceleration for the striking player was 56.1 ± 22.1*g*, and impact resulted in a 4.1 ± 1.2 m/s change in head velocity. HIC was 117 ± 101 in the striking player. More momentum was transferred to the struck player than the striking player in the collision, because the ΔV of the struck player was 6.8 ± 1.8 m/s. The mass ratio based on ΔV was $m_{Eff.Striking}/m_{Struck}$ = 1.67 (6.8/4.1). The average peak impact force was 7191 ± 2352 N. The calculated impact force and neck responses for the striking player, including *Nij*, are new information, whereas the head accelerations of the players and collision speeds are re-reported from Pellman et al. (39) and shown in *Table 2* for the struck players. This study excludes the helmet-to-ground impacts.

The NFL game impacts involved the striking player hitting either the right or the left side of the opponent's helmet. A notation is included in *Table 1* about the side of helmet impact. This is one factor in the direction of neck forces and moments. Head kinematics was complex, and variations in the direction of neck responses occurred during impact. The primary neck load was axial compression (*Fz*) in the striking players. The average peak neck compression force was 4227 ± 1888 N in the striking player. *Table 1* also includes the average and standard deviation in positive and negative neck responses. The average positive fore-and-aft neck shear (*Fx*) was 767 ± 327 N, and lateral shear (*Fy*) was 504 ± 217 N. The average peak neck bending moment (*My*) was 47.2 ± 38.7 Nm in flexion and 35.7 ± 20.5 Nm in extension.

The average *Nij* was 0.79 ± 0.33. However, eight of the tests involved *Nij* greater than 1.0, which is the NHTSA human tolerance level for neck loading. For these tests, *Nij* was 1.25 ± 0.16. Also shown is the type of *Nij* associated with the peak value. All cases involved neck compression, but there were 23 cases of compression-flexion (*Ncf*) and 4 cases of compression-extension (*Nce*). Nine cases exceeded the neck compression force of 4000 N and averaged 6631 ± 977 N (range, 5210–8194 N).

Head-Neck Impact Kinematics

Figure 3 shows the kinematic sequence of the helmet impacts from two reconstructions (Cases 38 and 39) out of the 27 cases in this study. Both struck players were concussed in NFL games. The sequence from the high-speed video progresses from the top down and shows the striking player on the *left* and the struck player on the *right*. The laboratory tests re-

TABLE 1. *Peak responses from the laboratory reconstruction of the striking player in National Football League collisions*[a]

Case no.	Side (L or R)	Impact velocity (m/s)	SI	HIC	Head responses				Neck response						*Nij*		Impact force (N)
					Transl. accel (g)	ΔV (m/s)	Rotat. accel (rad/s^2)	Rotation velocity (r/s)	*Fx* (N)	*Fy* (N)	*Fz* (N)	*Mx* (Nm)	*My* (Nm)	*Mz* (Nm)	Value	Type	
7	L	6.9	65	51	50	2.2	2832	9.8	−750	285	−3822	12	42	2	0.73	*Ncf*	6030
9	R	10.3	275	217	79	5.2	6719	18.7	643	703	−7657	53	−66	13	1.64	*Nce*	11680
38	L	9.5	157	127	60	4.0	5205	28.2	662	270	−6406	−18	−38	−8	1.09	*Nce*	9776
39	R	10.9	60	43	44	2.3	4487	10.4	−707	−275	−6660	54	50	−9	1.21	*Ncf*	7889
48	R	9.7	44	37	32	3.2	2939	28.0	−690	347	−2327	−18	29	4	0.46	*Ncf*	4108
57	R	8.8	48	38	32	4.1	4151	33.2	−521	444	−3558	−50	33	14	0.67	*Ncf*	5333
59	L	5.3	32	26	32	2.3	2087	13.1	−741	−70	−3784	8	33	−3	0.72	*Ncf*	4913
69	R	10.3	55	50	38	3.1	2620	23.0	347	419	−3243	−14	−20	−5	0.56	*Ncf*	4796
71	R	10.3	512	434	102	6.6	5541	32.4	−1212	755	−2079	43	80	17	0.53	*Ncf*	8258
77	R	9.9	65	53	35	4.2	2714	25.5	−1027	440	−3164	−9	58	12	0.68	*Ncf*	5612
84	R	9.4	96	78	45	4.4	3169	26.5	−663	520	−3875	−19	25	4	0.71	*Ncf*	6431
92	R	11.1	204	164	60	5.6	6070	43.8	−392	889	−8194	−53	45	5	1.45	*Ncf*	11510
98	L	9.6	241	187	84	4.8	4487	38.5	140	−851	−3633	50	14	−6	0.63	*Ncf*	7953
113	R	7.0	101	75	61	3.7	3700	31.2	152	575	−3343	42	5	−4	0.56	*Nce*	6323
118	R	10.7	122	73	56	3.7	3687	23.4	1351	−303	−6269	−10	−59	7	1.23	*Nce*	8937
124	R	11.4	105	73	56	3.1	4086	16.1	−651	−306	−5056	−22	47	7	0.84	*Ncf*	7959
125	R	11.7	132	111	47	4.2	3366	28.1	−584	746	−6391	−42	49	−10	1.06	*Ncf*	9015
135	L	10.0	230	179	81	3.8	5005	29.3	1244	−463	−7395	−53	−97	−16	1.72	*Nce*	11490
148	R	6.6	47	37	33	3.9	2466	26.5	470	−465	−2193	21	−24	−6	0.53	*Nce*	4065
154	R	6.6	35	31	29	3.1	3159	23.1	767	−169	−2099	12	−37	−6	0.60	*Nce*	3774
155	R	9.1	76	61	45	4.2	4217	29.5	943	−396	−3591	30	−41	−11	0.87	*Nce*	6247
157	R	10.8	215	180	79	5.0	4662	15.7	1317	−286	−5494	−28	−117	−4	1.53	*Nce*	9568
162	R	5.5	34	30	29	3.2	1672	17.2	645	−242	−2894	−17	−40	−2	0.74	*Nce*	4505
164	R	10.8	243	202	89	5.1	6136	30.8	373	−682	−2629	38	−25	−12	0.61	*Nce*	7872
175	R	9.6	81	62	47	3.9	2535	19.3	501	−819	−2518	−50	−35	−12	0.61	*Nce*	5011
181	L	11.7	402	333	85	7.3	6613	55.8	−632	905	−2312	−56	25	−8	0.38	*Ncf*	6877
182	R	8.1	272	213	87	4.7	3206	27.2	977	−717	−3386	−39	−60	−12	0.96	*Nce*	8239
Average		9.3	146	117	56.1	4.1	3983	26.1	702	561		33.0	38.2	8.5	0.86		7191
SD		1.9	121	101	22.1	1.2	1402	10.0	395	219		17.3	19.0	5.1	0.38		2352
								−Average	−714	−432	−4221	−31.1	−50.7	−7.9			
								SD	227	246	1885	17.3	28.9	3.9			

[a] L, left; R, right; SI, severity index; HIC, head injury criterion; Transl., translational; accel, acceleration; Rotat., rotational; *F*, neck force; *M*, neck movement; *x*, *y*, *z*, axes; *Nij*, neck injury criterion; SD, standard deviation; *Ncf*, compression-flexion; *Nce*, compression-extension.

TABLE 2. Peak responses from the laboratory reconstruction of the struck player in National Football League collisions[a]

Case no.	MTBI	Velocity (m/s)	SI	HIC	Peak transl. accel (g)	Peak ΔV (m/s)	Peak rotat. accel (rad/s^2)	Peak rotation velocity (r/s)
7	Yes	6.9	120	93	61	4.6	6266	28.1
9	Yes	10.3	848	600	134	10.1	7428	27.4
38	Yes	9.5	736	554	118	9.7	9678	50.8
39	Yes	10.9	656	522	129	8.4	5921	36.1
48	No	9.7	155	130	57	4.7	5617	42.4
57	Yes	8.8	253	206	77	6.0	6514	37.0
59	No	5.3	205	138	82	5.6	5387	26.9
69	Yes	10.3	177	153	61	5.0	4381	19.9
71	Yes	10.3	658	510	123	7.3	5400	35.0
77	Yes	9.9	226	185	80	5.2	5148	36.4
84	Yes	9.4	276	222	82	6.3	9193	80.9
92	Yes	11.1	630	508	107	10.0	6878	44.2
98	Yes	9.6	351	301	91	6.2	7548	43.4
113	Yes	7.0	163	140	59	5.1	3965	12.8
118	Yes	10.7	492	378	101	9.6	7017	42.9
124	Yes	11.4	380	282	81	7.5	7138	34.8
125	Yes	11.7	817	633	113	9.1	7716	63.3
135	Yes	10.0	751	566	138	8.6	7540	41.0
148	Yes	6.6	117	99	48	5.1	3476	23.9
154	No	6.6	136	114	53	5.1	4167	24.0
155	Yes	9.1	418	341	100	6.6	6940	37.0
157	Yes	10.8	545	472	103	8.1	6750	33.5
162	Yes	5.5	94	77	52	4.2	2615	18.4
164	Yes	10.8	451	370	124	6.0	9590	26.6
175	No	9.6	158	125	62	5.6	3555	39.2
181	Yes	11.7	423	382	93	7.1	8011	36.5
182	No	8.1	256	208	85	5.9	5512	17.8
	Average	9.3	389	308	89.4	6.8	6272	35.6
	SD	1.9	240	182	27.5	1.8	1851	14.2

[a] MTBI, mild traumatic brain injury; SI, severity index; HIC, head injury criterion; transl., translational; accel, acceleration; rotat., rotational; SD, standard deviation.

versed the collision by giving all of the impact velocity to the struck player. The collision was reconstructed by dropping the head, neck, and torso mass of the struck player into the suspended Hybrid III dummy simulating the striking player.

A *white line* has been added to the photographic sequence to help visualize the motion and compression of the striking player's head-helmet and neck in the impact and eventual neck bending as the helmets separate. In the top three photographs of the sequence, the striking player's head and helmet are pushed to the left by compressing the head-helmet interface and then the neck. The last three photos in each sequence show neck bending and eventual release after impact.

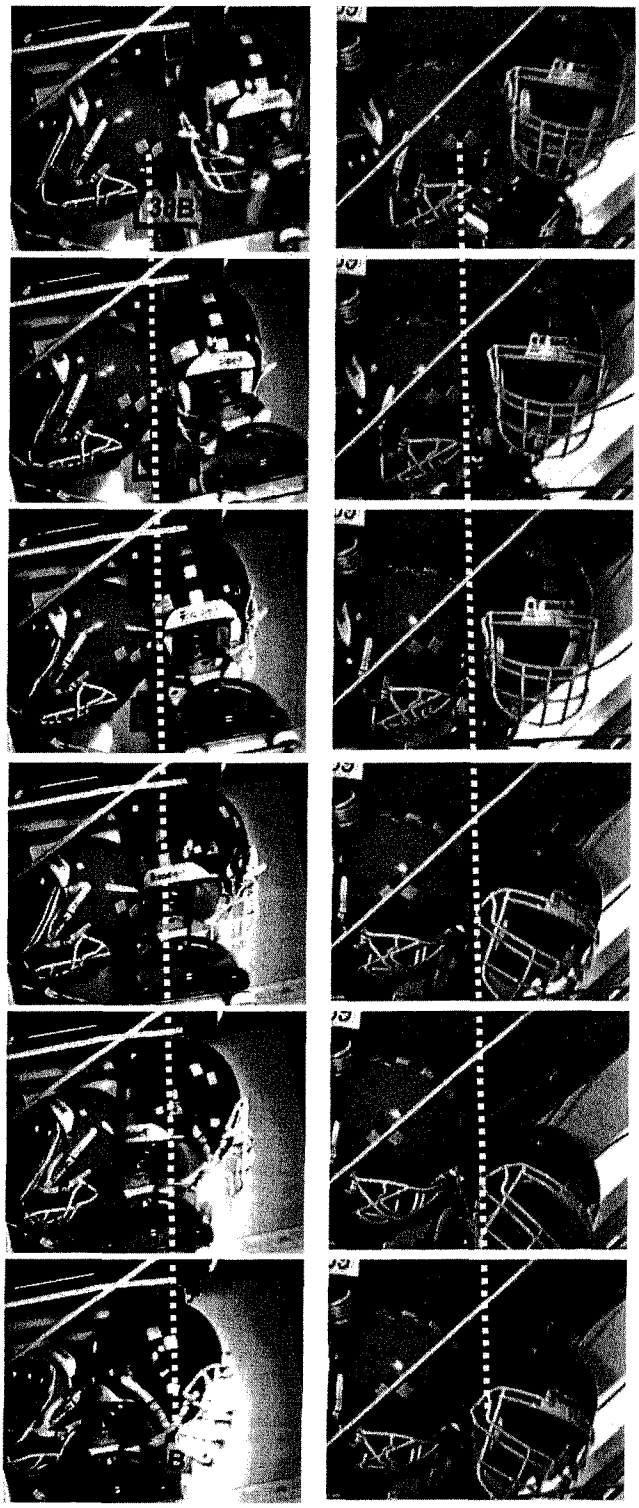

FIGURE 3. *Sequence from high-speed video of laboratory reconstruction of NFL concussion cases* (left, *Case 38, and* right, *Case 39). The sequence is from* top *to* bottom, *with the striking player on the* left *and struck player on the* right. *The* vertical white line *helps visualize the movement of the helmet and neck of the striking player.*

Figure 4 shows the time-average head acceleration and impact force of the striking players for the 27 NFL collisions reconstructed in laboratory tests with the Hybrid III dummies. The peak values in these plots are lower than the values in *Table 1* because the average response was determined as a function of time. Because peak values occur at different times for each reconstruction, there are lower values in the time histories, which smooth the peaks. At impact, there was an increase the striking player's head acceleration, which reached an average of 46.8 ± 21.7*g* at 7.2 milliseconds. The struck player's head acceleration reached 76.9 ± 26.2*g*. The peak impact force was 6372 ± 2486 N acting on the struck player. The neck compression force was 3624 ± 1729 N and contributed 57% of the impact load. The peak head rotational acceleration was 4289 ± 2156 r/s^2. Double integration of the struck player's head acceleration indicated a 48 mm displacement at peak force.

The impact biomechanics was consistent for the cases reconstructed from the NFL. The biomechanical responses of the striking and struck players demonstrated a 67% higher effective mass of the striking player than the struck player at peak force. There were also high neck compression forces in the striking player, causing concussion. Neck shear forces were considerably lower in amplitude than the neck compression force, and the bending moments were moderate and primarily flexion and lateral bending. This is consistent with the alignment of the impact axis through the head cg, neck, and torso of the striking player to effect a solid blow on the struck player.

Head-Neck Impact Biomechanics

Figure 5 shows the neck shear and compression responses, which occurred during the loading. In the reconstructions, the forces are either positive or negative, depending on the orientation of the heads at impact. The average and standard deviation in fore-and-aft and lateral neck shear are shown for the cases with positive values along with the identification of the particular tests making up the response. The neck shear forces were less than one-fifth the level of the neck compression force in the collisions. Also shown are the average and standard deviation in neck compression force for the nine highest responses. This gives an indication of the most severe impacts by the striking players and levels of neck loading tolerated by the striking players without head or neck injury.

Figure 6 shows the neck bending responses, which peak somewhat later than the impact force, head accelerations, and neck shear forces. Neck bending can be seen occurring later in the impact sequences shown in *Figure 3*. Depending on the orientation of the heads at impact, the bending moments vary from the positive to the negative in value. The average and standard deviation in responses for the positive-moment cases are shown, along with the identification of the particular tests making up the responses. The *Mz* bending moment was much lower in amplitude.

Figure 7 shows the neck compression force and flexion-extension bending moment at peak *Nij* for the reconstructions. Superimposed on the plot is the NHTSA neck tolerance criterion. *Nij* combines the normalized neck compression force and flexion-extension moment as a function of time to estimate the significance of neck loading for serious cervical injury. The *lines*

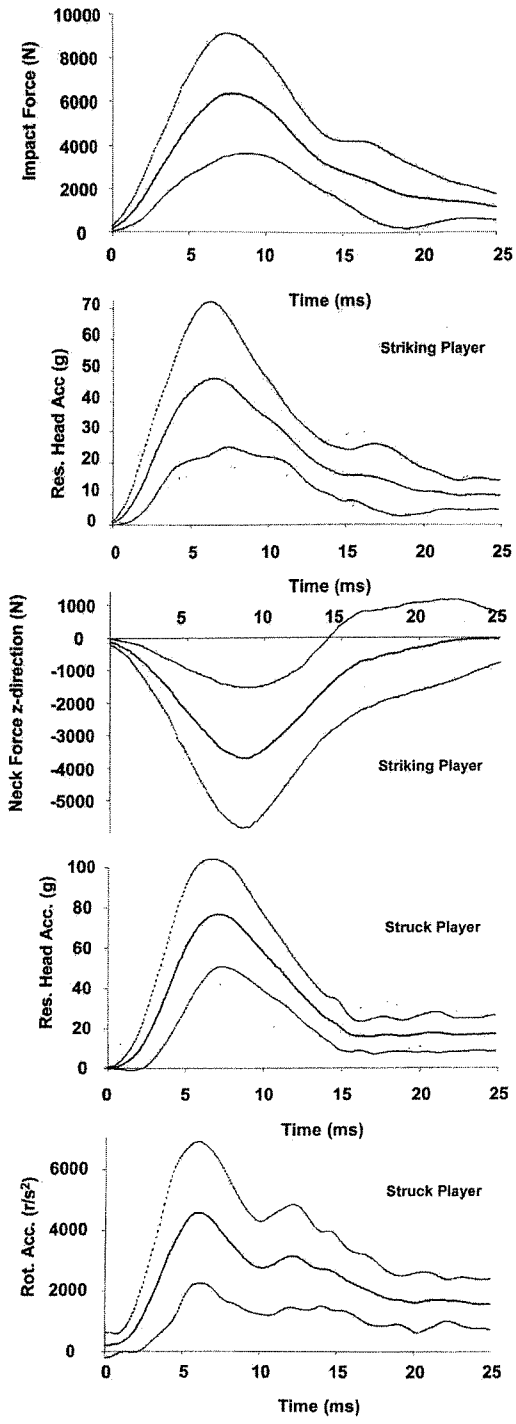

FIGURE 4. Top trace, *graph showing average and ±1 standard deviation in the impact force of the striking player. The lower traces show the resultant head acceleration* (second trace) *and the neck compression force* (third trace) *of the striking player (the sign of the neck compression force is reversed and added to the head inertial load to obtain the impact force in the* top trace). *The* bottom traces *are the resultant head translational and rotational accelerations of the struck player.*

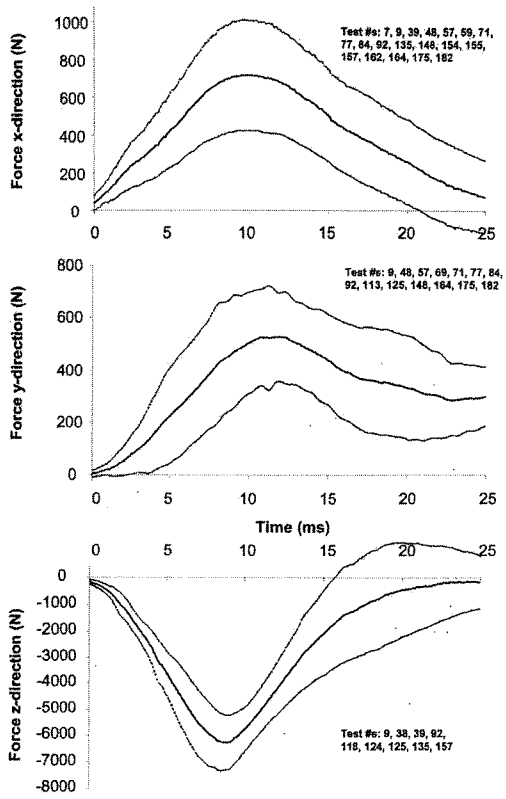

FIGURE 5. *Graphs showing average and ±1 standard deviation in the upper neck forces for the striking player. Because some impacts involved positive and others negative responses, depending on the impact orientation, the averages are shown for the group with positive neck* x *and* y *responses.*

in *Figure 7* show *Nij* = 1.0 and a 4000 N limit on peak neck compression force. These values are the current human tolerance limits for neck loading. The NFL reconstructions show nine cases outside the tolerable limits. In all nine cases, the neck compression force exceeded the tolerance limit of 4000 N, and in a few cases, it exceeded it by almost a factor of two. For these cases, the average neck compression force was 6631 ± 977 N (range, 5210–8194 N), and *Nij* was 1.25 ± 0.16 for those greater than 1.0. It is also interesting that there was a relatively moderate level of neck flexion-extension moment. The primary impact response was neck compression.

Figure 8 shows the peak neck compression force versus the initial impact speed and the computed change in head velocity at peak *Nij*. These data give an impression of the speed of neck compression in the striking player in the NFL impacts. The average collision speed for the reconstructions was 9.3 ± 1.9 m/s, and the average head velocity change was 2.8 ± 0.9 m/s at peak *Nij*, which is 30% of the initial collision speed. Most of the impact velocity (73% = 6.8/9.3) was transferred to the struck player in the collision.

Impact Conditions with Concussion

Five of the reconstructions involved hard hits in the game but no concussion to the struck player. The remaining 22 collisions

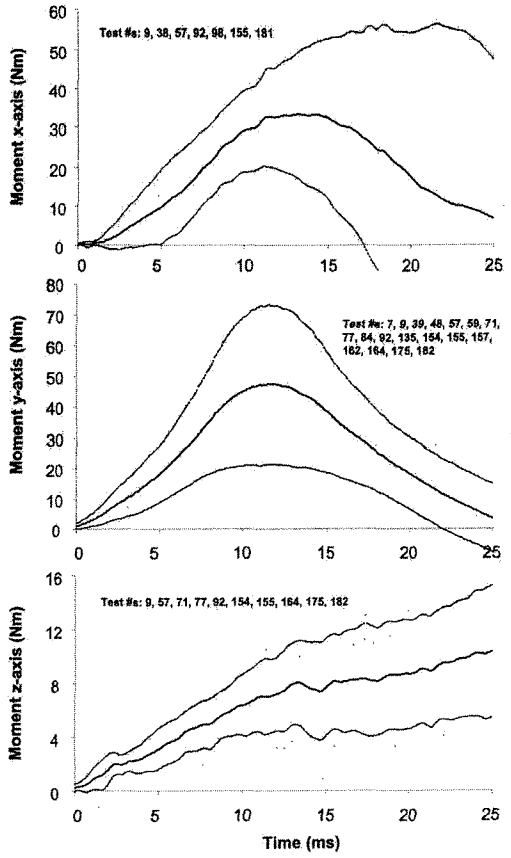

FIGURE 6. *Graphs showing average and ±1 standard deviation in the upper neck moments for the striking player. Because some impacts involved positive and others negative responses, depending on the impact orientation, the averages are shown for the group with positive responses.*

involved concussions. When the data were analyzed for impact conditions that caused concussion, the impact biomechanics of the struck player causing concussion was higher than for the players without injury. Peak head acceleration was 94.3 ± 27.5*g* with concussion and 67.9 ± 14.5*g* without ($t = 2.06$, $df = 25$, $P = 0.025$). The peak impact force averaged 7642 ± 2259 N with concussion and 5209 ± 1774 N without injury ($t = 2.24$, $df = 25$, $P = 0.017$). The head ΔV was 7.08 ± 1.88 m/s with concussion and 5.38 ± 0.48 m/s without ($t = 3.75$, $df = 24$, $P = 0.0005$). The striking player experienced a 4.26 ± 1.23 m/s head ΔV with concussion and 3.44 ± 0.90 m/s without injury ($t = 1.39$, $df = 25$, $P = 0.088$). These differences are statistically significant. The average peak neck compression force in the striking player was 4539 ± 1931 N with concussion compared with 2823 ± 725 N without concussion ($t = 3.27$, $df = 18$, $P = 0.002$).

DISCUSSION

In these helmet-to-helmet impacts, the striking player lowers his head and lines up his head, neck, and torso to deliver maximum force to the struck player, whose head and neck resist the

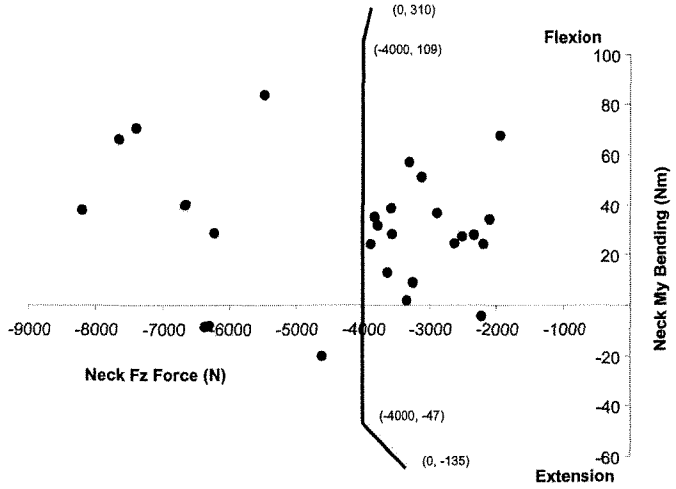

FIGURE 7. *Scatterplot of neck compression force and flexion-extension moment at the time of peak* Nij. *The lines show the tolerance criterion of* Nij = *1.0 and limit of 4000 N on neck compression force.*

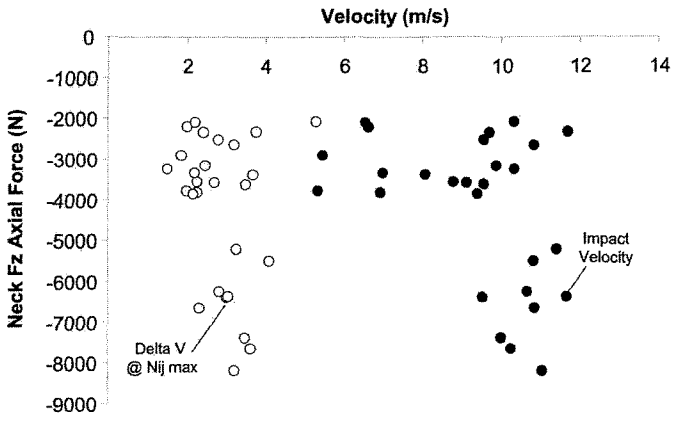

FIGURE 8. *Scatterplot of neck compression force for the 27 reconstructed collisions in the NFL showing the impact velocity of the helmets and the change in velocity of the striking player's head at peak* Nij.

impact. This is the typical situation when the struck player does not see the tackle coming and does not prepare for the collision. *Figure 9* shows an example (Player 38) of this tackling technique in a helmet-to-helmet impact causing concussion in an NFL game. With greater inertia of the striking player behind the impact, the average peak acceleration of the struck player's head reached 94.3 ± 27.5*g*, causing concussion. This acceleration was significantly higher than the 67.9 ± 14.5*g* in the nonconcussed players ($t = 2.06$, $df = 25$, $P = 0.025$). The striking player had even lower peak head accelerations of 56.1 ± 22.1*g* because of the added mass through neck compression. HIC and SI in the striking player were very low, so there was minimal risk of concussion in the striking player.

The key to the concussive blow is the head-down position, which involves a 67% greater mass of the striking player by coupling his torso into the collision. This kinematic transfers

more momentum to the struck player. In the situation in which the struck players see the impending tackle, they have a chance to line up their bodies and prepare for the collision. In this case, they have a greater effective body mass and are better prepared to resist the momentum transfer, particularly if they can lean into the tackle and line up their body.

Even though there are high compressive forces in the struck player's neck in the collisions, no NFL player, to the best of our knowledge, has experienced serious neck injury or concussion in this type of tackle. In fact, in 9 of the 27 NFL collisions reconstructed in this study, the compressive neck force exceeded current tolerance criteria for serious neck injury. The avoidance of neck injury is primarily by maintaining an axial alignment of the impact-force vector through the neck and torso; by minimizing neck extension and lateral bending, which increase the risk of injury; and by engaging the helmet of the struck player. This delivers more momentum to the struck player.

In this study, neck compressive forces were recorded above the current tolerance for serious neck injury. Using the neck injury criterion of the NHTSA, the *Nij* averaged 1.25 ± 0.16 in eight cases above the tolerance limit of *Nij* = 1.0. The average neck compression force was 6631 ± 977 N (range, 5210–8194 N) for the nine cases above the tolerance limit of 4000 N neck compression force. The neck tolerance criterion represents a risk of serious injury in 30- to 35-year-old men. However, the NFL data are from a population of players with superior physical conditioning and training. Strengthening exercises for neck muscles give these players far greater tolerance to neck compression, and the players have an ability to maintain an axial alignment of their cervical spine during head-down tackle.

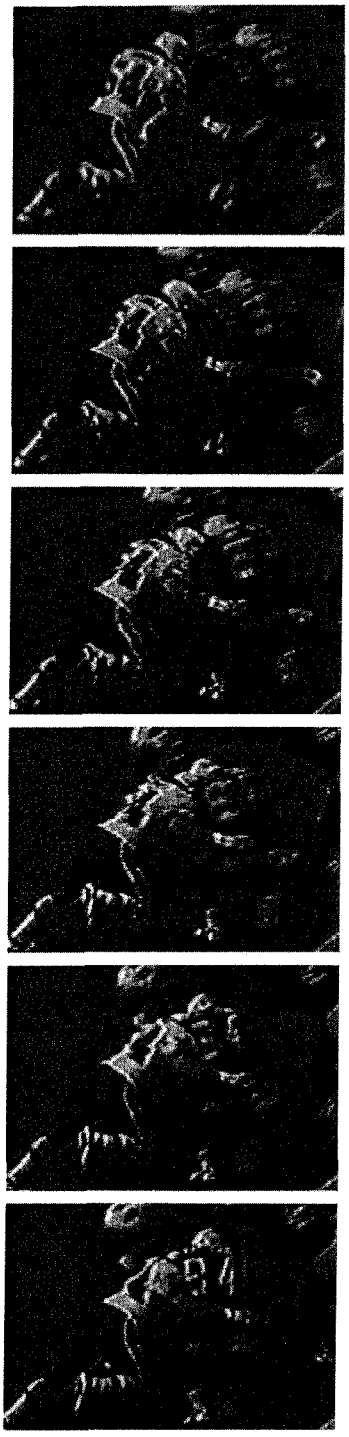

FIGURE 9. *Sequence from a game tackle showing the head-down position of the striking player as his helmet impacts the other player's head and his body drives forward. This is Case 38. Sequences from the laboratory reconstruction of this case are shown in Figure 3* (left sequence).

There is another reason why the NFL players are not experiencing neck injuries in concussive impacts. NFL players are typically bigger than the 50th percentile dummy used in the game reconstructions. It is known that the larger the player, the greater the tolerance to impact force. By using established scaling procedures, the NHTSA has found that the tolerable neck compression force for the 95th percentile man is 5440 N, compared with 4500 N for the 50th percentile man (8). However, even if the 95th percentile level is used, 8 of 27 NFL reconstructions exceeded the higher tolerance limit. Obviously, NFL players have a unique ability to sustain impact forces. Nonetheless, the NFL experience reported here provides new tolerance information relevant to a wide range of safety assessments.

Only 4 of 27 of the NFL tackles involved neck compression-extension (*Nce*), with relatively moderate extension moments of 35.7 ± 20.5 Nm. For the majority of cases with neck compression-flexion (*Ncf*), the peak flexion moment was 47.2 ± 25.7 Nm. These values are low in comparison to human tolerance levels. This indicates that the striking players control the impact alignment to limit bending moments and shear forces in their tackling technique. Experience has probably taught this lesson to the players.

The collision mechanics indicate that concussion occurs during the peak load when the highest head accelerations occur in the struck player. Head accelerations in the concussed players are statistically higher than in those not injured or in the striking players. Because the striking players have lower head accelerations, they have more mass in the impact. Their effective mass is 67% greater than that of the struck player. Because neck loads are high at this time, the mass includes torso inertia. Neck forces couple torso mass into the collision, which contribute to the higher effective mass of the striking player.

Reducing Concussion Risks

There are several ways to potentially lower the risk of concussion in helmet-to-helmet collisions. The primary means would be to enforce head-up tackling techniques. This would reduce the torso inertia involved in the striking player's collision and reduce the impact force. Helmet impacts are 61% of the concussive collisions in the NFL. In this tackle, the striking player delivers more of his momentum to the struck player by impacting the helmet. This lowers the deformation of the striking player's neck, because he initially loads only the struck player's head. A head-up tackling position would reduce the torso mass and lower the force on the struck player. This gives a new reason to reinforce antispearing rules and thereby decrease the risk of concussion in struck players.

The prevention of concussion in the struck player provides another reason to enforce rules against head-down tackling or

spearing in football. The most commonly reported reason for this rule has been the risk of catastrophic neck injuries in the striking player when the players tackle or block the torso of an opponent. The biomechanics of that type of tackle involve initial head acceleration in the striking player and a buildup of compressive forces in the neck; but the mass of the struck player's torso is substantial, so the striking player's neck eventually buckles in flexion, lateral bending, or another mode, leading to cervical fracture-dislocations and spinal cord injury.

A second means to lower concussion risks may be to reduce the stiffness of the top-crown region of the helmet. For reasons of durability and to gain performance in National Operating Committee on Standards for Athletic Equipment testing, the top of the helmet is the stiffest part of the plastic shell, much stiffer than the side of the helmet. The striking player uses the top of his helmet to strike the more flexible side of the struck player's helmet, leading to an incompatibility in the deformations of the two shells. It may be possible to include a load-limiting capability in the top of the shell, which would limit the impact force, lower head accelerations, and lengthen the duration of impact if the top of the helmet is used in a tackle. The impact force averaged 7642 ± 2259 N with concussion, so a load limit at the average minus 1 standard deviation would be 5383 N, or approximately 1200 lb. This level would limit the load close to the average for nonconcussion impacts in the NFL reconstructions. A local load-limiting function in the shell may also decouple the helmet mass in the collision. This approach is hypothetical and would require development to ensure an overall performance of any new helmet design to ensure comparable play in all situations and durability.

A third but potentially much less effective means would be to reduce the mass of the helmet, because this would lower the inertia of the striking player in the impact. Football helmets weigh 1.9 kg, compared with the 4.38 kg mass of the head and neck. If the helmet were reduced 20% in weight, there would be a 6% reduction in mass of the striking player's head and an even smaller reduction in the collision force if a head-down impact were used in the tackle. The determination of collision mechanics causing concussion may offer insights for innovators to consider in the development of new safety equipment. In the meantime, enforcement of head-up tackling offers the best means of reducing concussions in helmet-to-helmet collisions.

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COMMENTS

Football and the National Football League (NFL) have arguably become the most popular sport and professional league, respectively, in the United States. Among 32 professional teams in the NFL and 117 Division IA teams in the National Collegiate Athletic Association ranks, there are thousands of individuals participating every autumn in football at or near the highest level. This number balloons when one includes other professional leagues (e.g., Canadian Football League, Arena Football League, NFL Europe) and non-Division IA collegiate teams.

The number of mild traumatic brain injuries (MTBIs) occurring during participation in football every year is likely underreported. The NFL, as the premier league, has taken the responsibility to fund important research in the biomechanics of injuries leading to MTBI. Viano and Pellman reconstructed from actual game footage 27 helmet-to-helmet collisions that occurred within a 5-year period. Twenty-two of the 27 collisions resulted in concussions to the struck player. Hybrid III male dummies were equipped with standard and linear accelerometers to measure the acceleration and calculate the force occurring during impact. Transducers were placed in the neck to measure the force received by the striking model during collisions. The data provided in this article are necessary to help guide the development of future helmets and to assist the NFL in making and enforcing new rules governing play. Care should be taken, however, when attempting to transfer the data to clinical situations. The research used uniform models and situations during the reconstruction of the impacts. The mass of the striking and struck models was idealized in the authors' experiments. In the NFL, the mass of players can range from 200 lb for defensive backs to 300 lb or more for linemen. Because mass is an important component of the calculation for force, this makes it more difficult to translate the laboratory data to clinical situations. The medical history of the players involved in the original collisions was not examined.

These individuals may have had previous MTBI, which may have made them more susceptible to future MTBI. The authors also state that only helmet-to-helmet collisions were studied.

They did not examine the force sustained during helmet-to-ground collisions, which may contribute to MTBI in the NFL.

This article by Viano and Pellman is an important addition to the series on the biomechanics of MTBI in professional football. This ongoing research is likely to contribute to increased safety in football at all levels.

Min S. Park
Michael L. Levy
San Diego, California

In Part 8 of the NFL concussion series, Viano and Pellman describe the biomechanics of the striking player in 27 helmet-to-helmet collisions in which the struck player sustained a concussion in 22 of the collisions. Videotape analysis was used to recreate the collisions for biomechanical analysis in the laboratory. The authors describe how the striking player uses a head-down position to transfer momentum maximally, which causes MTBI to the struck player. Although none of the striking players in the study sustained a head or neck injury, one-third of the collisions that were reconstructed in the laboratory produced excessive compression forces in the cervical spine that exceeded current tolerance criteria for serious neck injury. Viano and Pellman recommend a head-up tackling position and advocate the enforcement of antispearing rules in football. Since 2001, Bob Watkins has advocated a "see what you hit" approach to tackling in football. Dr. Watkins has made his videotape available free of charge through his foundation (available at: http://www.spineinsports.org/programs.htm). Football helmet companies, such as Riddell with the Revolution helmet and Schutt with the DNA helmet, have made changes to the size, weight, and face masks of helmets. Even with these changes, collisions in football should be initiated with proper technique so as to reduce injury to the striking player and the one being struck.

Russ Romano
Director of Sports Medicine, Head Athletic Trainer
University of Southern California
Los Angeles, California

Viano and Pellman have provided objective data regarding football head and neck impact biomechanical and loading parameters. These data were acquired by using dummy models to recreate actual game videotaped scenarios. In so doing, the authors observed that supramaximal loads are occasionally encountered. From this important information, they derived recommendations for injury prediction and prevention. This study model may become the "gold standard" in the future. The authors have presented an objective scientific approach to football injury and injury prevention.

Edward C. Benzel
Cleveland, Ohio

The authors have addressed the biomechanics of the "striking and struck players" with head-down tackling in NFL helmet-to-helmet collisions causing concussion. In this study, 27 helmet-to-helmet collisions were reconstructed in the laboratory using dummies. Although limited to American football, this study does have implications in other football codes that have tackling as a major component of the game, including Australian Rules Football, Rugby Union, and Rugby League codes. However, it should be noted that the descriptions of the biomechanics described apply to the situation in which the player being "hit" does not see the tackle approaching.

This type of study does provide important data to sports administrators that would lower the risk of injury from helmet-to-helmet collisions. The authors suggest enforcing "head-up tackling" techniques and reducing the "stiffness" of the top crown region of the helmet as well as a "potentially less effective" means by reducing the mass of the helmets, because this would lower the inertia of the striking player in the impact. It is noted that helmet impacts comprise 61% of concussive collisions in the NFL, and it would be of interest to compare this study with those that are undertaken in other football leagues that do not use helmets. Devotees of other football codes often wonder whether what seems to be the excessive padding and armor worn by American footballers paradoxically might increase the risk of serious injury by providing a larger target and allowing the player to use the "armor" as a weapon.

The authors have provided an excellent study that continues the series dealing with neurosurgical injuries in professional football. These types of studies can only help us to understand the game better and provide a rational basis for the introduction of rule changes to prevent devastating injuries in the sport.

Andrew H. Kaye
Melbourne, Australia

This Part 8 study by the NFL Committee on Mild Traumatic Brain Injury focuses on the important issue of examining the impact biomechanics of players involving the generation of concussive injuries. Specifically, Viano and Pellman have analyzed the factors implicated in imparting sufficient forces to the struck player such that he sustains a concussion, with special attention to the characteristics related to the potential for serious or catastrophic cervical spine injury. Using game videotape analysis, 27 helmet-to-helmet collisions were reconstructed using laboratory test mannequins, in which 22 struck players incurred concussions and 5 did not, although there were no injuries to the striking players (tacklers).

Their findings included that the majority of the impacts to the struck players occurred to the face mask or its area of attachment, whereas in 93% of instances, the striking players made contact with the top or crown of their helmet. This positioning allowed the latter to align their torso with their head and neck, not only striking with the hardest portion of the headgear but generating more mass into the collision. The struck player, in contrast, characteristically did not have sufficient time or warning to bring his body mass into the crash,

leading to a 67% greater effective mass of the striking player at the moment of peak force. Regarding concussion, peak head acceleration, peak impact force, and the change of head velocity were all greater in those players sustaining such injuries than in those who did not. The average peak neck compressive force was also greater in the striking players than in the struck players who incurred concussions.

There are several characteristics of NFL players that make them unique and resistant to neck injuries, but this relative safety does not translate to lower of levels of play for the scholar athlete. Once again, rules, customs, and methods of play are usually admired and emulated by younger football players and coaches. The NFL player is ordinarily larger and stronger and has had additional years in which to develop hypertrophied and conditioned neck musculature and supporting ligamentous structures. Professional players have also learned to align their head center of gravity and control impact alignment in their favor to limit cervical flexion or extreme hyperextension movements, as the authors' data demonstrate. Because of the extreme influence that the NFL has on the lower levels of play, it would seem that their changing or evoking greater enforcement of the rules, which are supposed to limit or prohibit initial contact with the top or crown of the helmet, would be most beneficial to scholar athletes, who are at greater risk of sustaining catastrophic cervical spinal injuries. As the authors mention, there are numerous issues involved with helmet redesign that make it a less attractive or viable option to prevent such catastrophic injuries. This study is a valuable analysis of the biomechanical forces and issues involved in cervical spine injuries and should assist in future efforts to limit such occurrences.

Julian E. Bailes
Morgantown, West Virginia

TAB 1I

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CLINICAL STUDIES

CONCUSSION IN PROFESSIONAL FOOTBALL: BRAIN RESPONSES BY FINITE ELEMENT ANALYSIS: PART 9

David C. Viano, Dr. med, Ph.D.
ProBiomechanics, LLC,
Bloomfield Hills, Michigan,
Bioengineering Center,
Wayne State University,
Detroit, Michigan

Ira R. Casson, M.D.
Department of Neurology,
Long Island Jewish Medical Center,
New Hyde Park, New York,
and Department of Neurology,
Albert Einstein College of Medicine,
Bronx, New York

Elliot J. Pellman, M.D.
ProHEALTH Care Associates, LLP
Lake Success, New York

Liying Zhang, Ph.D.
Bioengineering Center,
Wayne State University,
Detroit, Michigan

Albert I. King, Ph.D.
Bioengineering Center,
Wayne State University,
Detroit, Michigan

King H. Yang, Ph.D.
Bioengineering Center,
Wayne State University,
Detroit, Michigan

Reprint requests:
David C. Viano, Dr. med, Ph.D,
ProBiomechanics, LLC,
265 Warrington Road,
Bloomfield Hills, MI 48304–2952.
Email: dviano@comcast.net

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OBJECTIVE: Brain responses from concussive impacts in National Football League football games were simulated by finite element analysis using a detailed anatomic model of the brain and head accelerations from laboratory reconstructions of game impacts. This study compares brain responses with physician determined signs and symptoms of concussion to investigate tissue-level injury mechanisms.

METHODS: The Wayne State University Head Injury Model (Version 2001) was used because it has fine anatomic detail of the cranium and brain with more than 300,000 elements. It has 15 different material properties for brain and surrounding tissues. The model includes viscoelastic gray and white brain matter, membranes, ventricles, cranium and facial bones, soft tissues, and slip interface conditions between the brain and dura. The cranium of the finite element model was loaded by translational and rotational accelerations measured in Hybrid III dummies from 28 laboratory reconstructions of NFL impacts involving 22 concussions. Brain responses were determined using a nonlinear, finite element code to simulate the large deformation response of white and gray matter. Strain responses occurring early (during impact) and mid-late (after impact) were compared with the signs and symptoms of concussion.

RESULTS: Strain concentration "hot spots" migrate through the brain with time. In 9 of 22 concussions, the early strain "hot spots" occur in the temporal lobe adjacent to the impact and migrate to the far temporal lobe after head acceleration. In all cases, the largest strains occur later in the fornix, midbrain, and corpus callosum. They significantly correlated with removal from play, cognitive and memory problems, and loss of consciousness. Dizziness correlated with early strain in the orbital-frontal cortex and temporal lobe. The strain migration helps explain coup-contrecoup injuries.

CONCLUSION: Finite element modeling showed the largest brain deformations occurred after the primary head acceleration. Midbrain strain correlated with memory and cognitive problems and removal from play after concussion. Concussion injuries happen during the rapid displacement and rotation of the cranium, after peak head acceleration and momentum transfer in helmet impacts.

KEY WORDS: Concussion, Finite element modeling, Football helmets, Head injury, Injury mechanisms, Sport injury

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Computer models are now capable of simulating fine anatomic detail and tissue-level characteristics for impacts leading to injury (74, 75). The current study uses this powerful tool to study the mechanisms of concussion signs and symptoms by investigating strain and strain rate in the brain. Accelerations measured in Hybrid III dummy reconstructions of National Football League (NFL) game concussions were used as input to the cranium of the finite element (FE) model (122, 123).

FE models have assisted researchers studying brain contusion (25, 150), subdural hematomas (64, 65), brain edema (101), and brain deformation (94, 152, 153, 155, 165, 167, 168). However, there has been limited FE analysis of concussion and diffuse axonal injury. Ruan et al. (134, 135) and Zhou et al. (175, 176) indicated that shear stress and strain re-

sponses could be the injury indicator for diffuse axonal injury and subarachnoid hematoma. Bandak and Eppinger (17) suggested that an accumulated volume of elements exceeding a tolerable principal strain, referred to as the cumulative strain damage measure, was related to diffuse axonal injury.

The Wayne State University Head Injury Model (WSUHIM) Version 2001 is the most sophisticated FE model of the brain (171, 172, 173). It has a long history of development and validation since 1993, and it has been subjected to many investigations and improvements (134, 135, 136, 170, 171, 174, 175, 176). Al-Bsharat et al. (7) modified an earlier FE model developed by Zhou et al. (176) to simulate large relative motion between the brain and the cranium, showing that proper modeling of the interface between cranium and brain was a factor in predicting brain injury. This paralleled work by Bandak et al. (18), although the National Highway Traffic Safety Administration model only simulated the upper portion of the brain.

All FE numerical simulations rely on experimental studies of brain injury to determine material responses and predict failures. There have been investigations into brain contusion (3, 72, 76, 96, 115, 132), subdural hematoma (91), diffuse axonal injury (4, 5, 10, 15, 16, 43, 70, 86, 88, 107, 125, 133, 146, 147), concussion (1, 49, 50, 112, 156), brain displacement (35, 47, 57, 81, 128, 129), directional sensitivity of injury (42, 60), and head impact tolerances (108, 110, 113). Physical models have been used to bridge information from cadaver and animal research to FE modeling. These studies provide insights into brain injury mechanisms (8, 20, 34, 52, 53, 55, 59, 61, 66, 67, 68, 86, 163). However, none of the above studies have used injury data from in-depth study of on-field football concussions to determine brain responses related to injury.

Sports-related mild traumatic brain injury (MTBI) has received attention because of the large number of people affected at the high school and college levels; however, sport injury is just a portion of the overall incidence of mild to serious brain injury. With the advent of airbags and safety belt use in automobile crashes and the introduction of improved protective devices in sport, the incidence of serious-fatal brain injury has been decreasing (6, 21, 77, 78, 87, 149, 151, 157). Football helmets have developed an effective padding system, which has prevented severe head injuries in NFL players, however, they have only recently started addressing designs to prevent concussion, and much less has been understood of the biomechanics of concussion. The recent work of the NFL MTBI Committee has aimed at advancing the understanding of concussions (117–123, 162) and promoting improved helmet designs to prevent concussion.

Current standards for head injury risk assessment use the Gadd Severity Index (SI) and Head Injury Criterion (HIC), which are based on the resultant translational acceleration at the center of gravity (cg) of the head (39, 158). These criteria are based on the Wayne State University tolerance curve, which was based on head accelerations from animal concussions and cadaver cranium fractures from rigid, flat surface impacts on the forehead (80). These criteria are used in safety standards to assess head protection against automobile and sport injuries. However effective these criteria may be, it is believed that they cannot account for the complex motion of the brain in a deformable cranium. Foremost, the criteria neglect the contribution of head angular acceleration to injury and the directional sensitivity of brain injuries, and local failure modes of brain tissue are not currently specified (43–45).

On the basis of research conducted over the past 4 decades, a widely held belief is that angular acceleration is more damaging than linear acceleration (44, 45), even though in any head impact, both forms of acceleration occur. Research on the effects of angular acceleration has been pursued more vigorously than that on the effects of linear acceleration in an attempt to find a tolerance limit for angular acceleration and velocity (43, 86, 126). Although there has been considerable research on brain injury, the biomechanical focus has been on which of the two types of head acceleration cause injury. There has been much less attention on specific brain responses that are the underlying cause for neural tissue injury. Tissue related strain, strain-rate, and product of strain and strain-rate are relevant to neural injury (161).

This article studies brain response at the tissue level. The responses include strain, strain rate, and product of strain and strain rate, which were determined for NFL football impacts causing concussion or severe helmet loadings without injury. Brain responses were simulated using the 2001 version of the WSUHIM FE model, which simulates the essential anatomic features of a human head and brain and has been validated with cadaver data (172, 173). The FE model determines intracranial pressure, stress, and strain in the brain from head accelerations measured in NFL impact reconstructions (104–106, 121, 122). Statistical analyses have been conducted to determine initial concussion predictors and to estimate injury probability. The strongest correlation of concussion was with the product of strain and strain rate (174).

In this study, brain responses with concussion were studied through the use of NFL head impact data to establish relationships between brain responses and injury. Concussion in NFL football provided a unique living "laboratory" to study mechanisms of injury and human tolerance with possible extrapolation to the general population. Performing game impact reconstructions using the Hybrid III dummy (13) in laboratory reconstructions provided inputs to the anatomically detailed FE model to predict the extent and severity of brain responses for each game impact. The goal was to determine brain responses associated with specific signs, symptoms, and outcomes of concussion. This approach is unlike previous studies, which determined tolerance limits for human head injury based on head input kinematics either scaled from animal data or noninjurious volunteer test results. The ultimate goal was to improve the understandings of helmet designs to reduce concussion in football and other contact sports.

A case-by-case computer simulation was carried out using the head kinematics obtained from laboratory reconstructions of game impacts to drive a FE model. Brain tissue responses were calculated to identify sites of high deformation. These tissue-level responses were used to study injury mechanisms. A validated FE model along with tissue tolerance data offered a means of identifying and quantifying the incidence of con-

cussion. Concussion is an injury with many signs and symptoms, which can have their origin in specific regions of the brain as well as from diffuse injury. It is also a threshold injury in the spectrum of brain injuries involving contusion, diffuse axonal injury, and coma.

Twenty-two NFL concussions were known to interrupt brain function, whereas an additional six cases of severe impact without injury were used to determine a threshold for loss of brain function. Head kinematics of the helmeted Hybrid III headform was defined by three translational and three rotational acceleration components in an anatomic, body-fixed reference frame. The data were measured directly from the laboratory reenactment of the head collisions and were used as input to the WSUHIM. By defining the outer surface of the cranium as a rigid body and applying the head kinematics at the head cg, the loading boundary conditions of the head were prescribed.

MATERIALS AND METHODS

Finite Element Model of the Head and Brain

This study used the latest version of the WSUHIM (Version 2001). This model has a fine mesh representing the human cranium, membranes, brain, and ventricles in three-dimensional (3D) FEs (172–174). The average characteristic length of the brick or shell elements was approximately 2 mm. Such a fine mesh size enabled more detailed and accurate modeling of anatomic features of the human head. The gross geometry of the head was taken from the model by Al-Bsharat et al. (7), whereas the boundaries between anatomic structures were based on anatomic drawings (90). The model simulates the essential anatomic features of a 50th percentile male head, including the scalp, cranium with an outer table, diploë, and inner table, dura, falx cerebri, tentorium, pia, sagittal sinus, transverse sinus, cerebrospinal fluid (CSF), hemispheres of the cerebrum with distinct white and gray matter, cerebellum, brainstem, lateral ventricles, third ventricles, and bridging veins. The 50th percentile Hybrid III head is used on the 50th and 95th percentile Hybrid III dummy, so it is relevant to average and larger sized males.

For this study, the cranium was assumed rigid, and the translational and rotational acceleration from laboratory reconstructions of concussion impacts in the NFL were used to load the head (122, 123). The accelerations define the motion of the head cg, and they were transferred to the cranium boundary nodes to load the brain in a manner identical to the motion of the Hybrid III dummy in the game reconstruction. The face, scalp, and three-layer cranium assume rigid bodies while maintaining the head weight and moment of inertia. This also reduced computational time because cranium accelerations were the means to load the brain. The cranium-brain model consisted of 281,800 nodes and 314,500 elements and a mass of 4.5 kg. The mesh of the model was developed using a preprocessor (HyperMesh, Version 3.1, Altair Engineering, Inc., Troy, MI). A nonlinear explicit FE solver (PAM-CRAS, Version 2000, ESI Group, Paris, France) was used for model analysis. The results were plotted using an interactive graphics postprocessor (PAM-VIEW Version 2000, ESI, Paris, France). For this FE model, it takes approximately 36 hours to complete a 20 ms simulation using a PC with 2.7 GHz Pentium IV using a Linux operating system.

Skull and Dural Partition

Figure 1 shows the sagittal and coronal section through the cranium of the WSUHIM version 2001. The dimensions of the interior volume include a 171 mm length, 138 mm width, and 152 mm height. The cranium resembles a sandwich structure containing cancellous and cortical bones. The cranium model was meshed with three layers to represent an outer table, diploë, and inner table. The base of the cranium includes the foramen magnum, which has a characteristic diameter of 31 mm. The falx cerebri, tentorium, and sinus (superior sagittal sinus, transverse sinus, and straight sinus) are also shown. The falx has a maximum depth of 40 mm and thickness of 1 mm, whereas the tentorium has depth of 51 mm. The cranium weighs 1.3 kg.

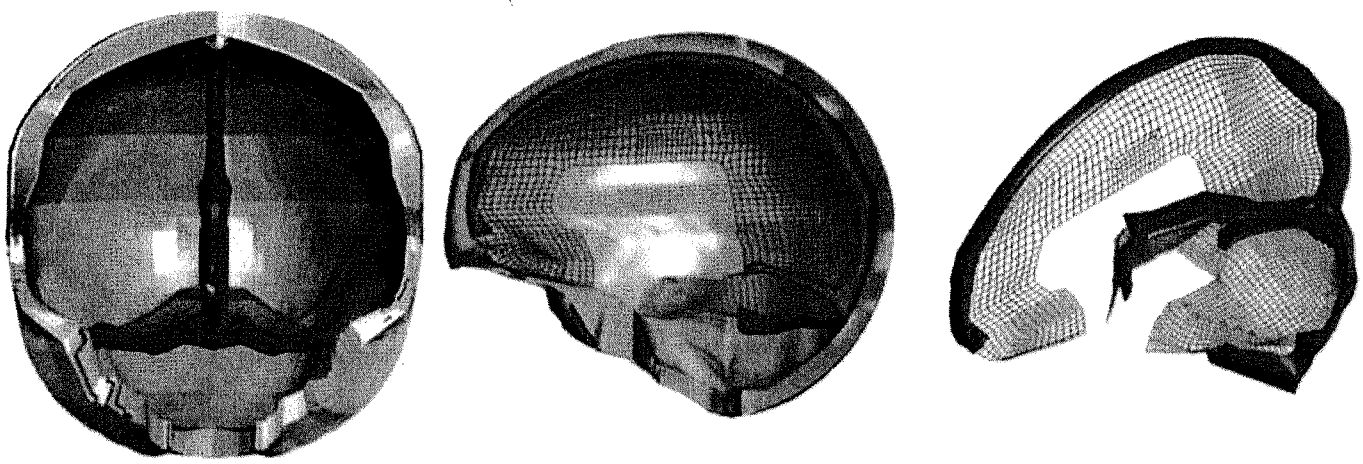

FIGURE 1. *Skull, falx cerebra, tentorium, and dura-sinus.*

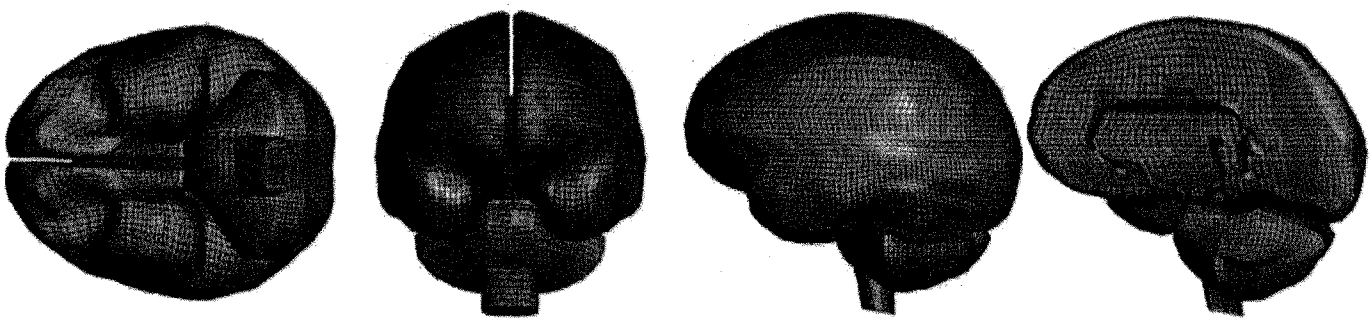

FIGURE 2. *Representation of the brain.*

Brain Model

Figure 2 shows the representation of the brain. It includes cerebrum with gray and white matter, brainstem, and cerebellum (90). The right and left hemispheres of cerebrum are connected internally by a corpus callosum. The length and transverse diameter of the brain are 167 mm and 134 mm, respectively. The midbrain of the brainstem is located in the opening of the tentorium. The medulla oblongata is represented in the posterior cranial fossa just superior to the level of the foramen magnum. The brainstem diameter measures 7 mm. The brain weighs 1.4 kg.

Subarachnoid Space

The dura mater is firmly attached to the internal surface of the cranium. The outer layer of the arachnoid membrane slides freely with respect to the innermost layer of the dura. The subarachnoid space is filled with CSF. The CSF and the arachnoid trabaculae suspend the brain in the cranium.

Several algorithms were reviewed by Al-Bsharat et al. (7) to determine which one was the most suitable to model the CSF layer and brain-cranium interface. The best approach was to model the CSF with solid elements in addition to the definition sliding interface between the dura and the CSF. The model used a low shear modulus solid, which allowed relative displacement between the brain and the cranium as well as intracranial pressure to match that obtained experimentally.

Pudenz and Shelden (128), and Meaney (91) suggested that pure-sliding condition was a realistic interface for the simulation of head-brain kinematics. According to Ueno et al. (155), separation of the brain from the dura mater is unlikely to occur in blunt head impacts because of nearly incompressible nature of the brain tissue.

Bridging Veins and Ventricles

Figure 3 shows the bridging veins and lateral and third ventricles. Ten pairs of bridging veins were included with length of 8 to 17 mm and orientation of 160 to 1100 on the basis of published data (108a). The lateral ventricle has a length of 102 mm and width of 66 mm, whereas the third ventricle has a length, width, and height of 33, 20, and 5 mm, respectively. The intracranial membranes, CSF, ventricles, and blood vessels weigh 0.26 kg.

The lateral and third ventricles are explicitly modeled using solid elements because fluid elements are not available in the current simulation code. A shear modulus that is lower than that of the subarachnoid CSF was assumed for the material properties of the ventricles. No sliding was defined in the interface between the ventricular CSF and the ventricular cavity.

Face Model

Because the input acceleration was to the cranium, which was assumed rigid, the face was not a factor except for its mass in the simulations. However, for completeness, it will be described. The FE face model consists of 14 bones, two zygomatic bones in the cheek, two maxillae forming the upper jaw, most of the side walls of the nose, and front part of the hard palate, two palate bones forming the rest of the hard palate and part of the floor and side walls of the nasal cavity, two lacrimal bones (one in each eye socket or orbit), two nasal

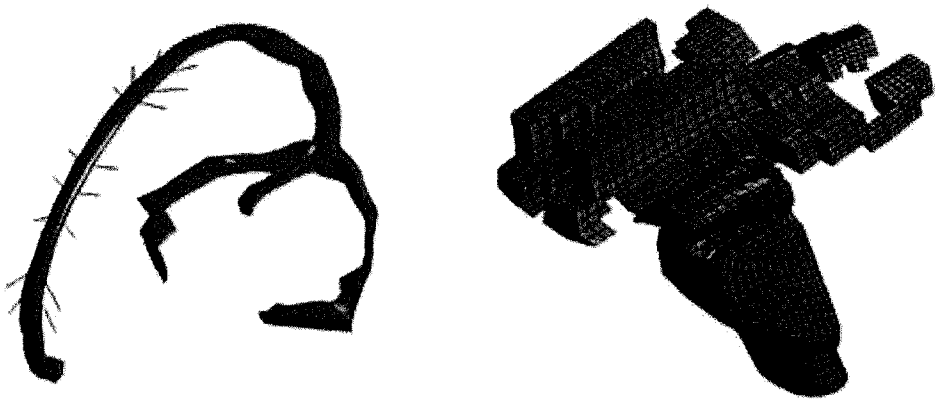

FIGURE 3. *Bridging veins, ventricle, and brainstem.*

TABLE 1. Viscoelastic material properties simulating the brain

Brain tissue	Density g/cc	Shear modulus		Decay constant s^{-1}	Bulk modulus GPa
		G_∞ kPa	G_8 kPa		
Gray matter	1.06	10.0	2.0	80	2.19
White matter	1.06	12.5	2.5	80	2.19
Brainstem	1.06	22.5	4.5	80	2.19
Cerebellum	1.06	10.0	2.0	80	2.19

bones forming the bridge of the nose, two turbinate bones inside the nose, one vomer forming the posterior and inferior part of the nasal septum, and one mandible forming the lower jaw. The face geometry was based on magnetic resonance imaging and computed tomography scans from the Visual Human Project database available from the National Library of Medicine (Bethesda, MD). The images were transverse sections scanned at 1 mm intervals along the vertical axis of the body and scaled to the cranial size of the 50th percentile male head model. Because of the irregularity and complexity of the facial bony structures, the bone geometry was simplified and smoothed while preserving the structural features of the human face. The facial model consisted of 36,400 elements with a mass of 0.78 kg and was integrated with the cranial model.

Material Properties

Table 1 gives the material properties used to simulate the viscoelastic brain. Biological materials display both elastic and viscous properties. Brain tissue is a hydrated soft tissue consisting of approximately 78% water. It is similar to a soft gel when defined as an engineering material (61). Experimental characterization of brain materials from a variety of species suggests that brain tissue exhibits incompressible viscoelastic behavior (38, 41). The bulk modulus of brain tissue was similar to water, with a value of approximately 2.00 GPa according to Stalnaker (145) and 2.10 GPa according to McElhaney et al. (89). Traditionally, the material behavior of the brain has been approximated by a Kelvin (viscoelastic) model, which is a combination of linear springs and dashpots. The behavior of this material is characterized as viscoelastic in shear with the deviatoric stress rate dependent on the shear relaxation modulus, whereas the hydrostatic behavior of the brain was considered elastic. The shear modulus of the viscoelastic brain material was expressed by:

$$G(t) = G_\infty + (G_0 - G_\infty)e^{-\beta t} \quad (1)$$

where G_0 is the short-term shear modulus, G_∞ is the long-term shear modulus, β is a decay constant, and t is the duration (170).

The cerebral hemispheres are composed of white matter and gray matter. The neuroarchitecture of brain tissue is naturally inhomogeneous and anisotropic. The shear modulus of the white matter was assumed to be 25% higher than that of the gray matter because of the fibrous nature of the white matter to incorporate different material compositions of white and gray matter. The brain was modeled as isotropic because anisotropic properties of the brain are not available at present. The material properties assigned to the brain did consider the regional differences between the cerebrum white matter and the brainstem.

Pieropaoli and Basser (124) indicated that some regions of white matter could be modeled as a transversely isotropic structure and gray matter as isotropic structures on the basis of magnetic resonance diffusion tensor images of the brain neuroarchitecture. Experimental results obtained from oscillatory shear tests of pig brain tissue revealed that the complex shear modulus for the brainstem region was 80% larger than that of the cerebrum tissue at large strain (11). These regional differences can affect the mechanical stress or strain response during an impact. In addition, the upper brainstem is considered by many researchers as a critical anatomic substrate responsible for maintaining the state of consciousness (3, 28, 29, 54, 70, 85, 125, 132). The short-term shear modulus assumed for the brainstem was 80% higher than that for the cerebral white matter.

Studies have characterized the brain shear properties (38, 41, 143, 144). Reported shear moduli varied from one study to another. Recently, Arbogast et al. (12) reported that the instantaneous shear moduli were 1040 Pa for white matter in the brain stem and 680 Pa for gray matter in an experimental study using pig brain. In their study, the brain tissues were dissected from adult pigs, and vibratory shear forces were applied while the brain sample was placed between two parallel plates. Shear moduli were calculated for these tissues as a function of excitation frequency. However, the experimental results did not fully reflect the tethering effect of the blood vessels in an intact brain because of the dissection of the brain tissue alters the property of the complex network of blood vessels.

In the current model, the material properties for the solid elements representing the CSF have the bulk modulus of water and a very low shear modulus. This considered a combined effect of fibrous trabaculae and the fluid nature of the CSF. The average thickness of the CSF layer was less than 2 mm. A low-friction slip algorithm is used to model the interface between the dura and the arachnoid membrane. In addition, the interface algorithm allows no separation of the two layers in contact and therefore prevents the formation of a gap in the arachnoid membrane-dura interface. From the numerical stability point of view, allowing separation is prone to result in an excessive distortion of the soft brain elements and to generate unrealistic intracranial pressures when a gap is developed.

Dobrin (33) reported that the vascular wall of small arteries (<4 mm in diameters) contain mainly smooth muscles with some collagen fibers. Collagen contributes to the load bearing role under large tensions (131). Although complete and detailed information regarding the material properties of the cerebral vessels are lacking, Viano (159) reported that the Young's modulus of the human aorta ranged from 360 kPa to 400 kPa. This value is significantly higher than that measured from dissected brain tissues. In a recent simulation of a two-dimensional brain model with explicit modeling of cerebral vessels, Bae et al. (14) reported that the maximum principal stress was higher with the inclusion of the vasculature in the brain. Both studies indicate that the cerebral blood vessels contribute to the overall stiffness of the brain tissue in vivo, particularly when the vessels are closely attached to brain tissue and anchored or tethered to structures outside of the brain.

The shear moduli used to model the brain were assumed to be approximately one order of magnitude higher than those reported by Arbogast et al. (11) to compensate for the effect of vascular structures. The values used for brain materials were approximately one third of those defined in previous versions (7, 172, 173). They are the lowest that can be used without causing permanent deformation to the brain structure and were selected to match the experimental results.

Table 2 gives the Young's modulus of 31.5 MPa and other properties for the fax cerebri and tentorium membranes. The values used for the membranes were in the range found in the literature (41, 89, 93). An elastic-plastic material model was used for cortical and cancellous bones of the face. Damage elements available in the PAM-CRASH material model were introduced to predict bony fracture. The element elimination option was used. This option removes any element with a strain that exceeds a preset ultimate strain magnitude in each time step. A Young's modulus of 560 MPa was assumed for the cancellous bone of the face. The value used for cancellous bone was in the range found in the literature (mandibular condyle: 431 MPa [46], Mandible body: 273 MPa [23], vertebral body: 316 MPa [63], proximal femur: 441 MPa [82], and proximal tibia: 635 MPa [32]) and [97].

TABLE 2. Elastic material properties of the head[a]

Elastic material	Density g/cc	Poisson's ratio	Modulus (GPa) stiffness N/mm
Falx, tentorium	1.13	0.45	E = 0.0315[b]
CSF[a]	1.04		G = 1.0E-06[c]
Ventricle	1.04		K = 2.19[d]
Bridging Vein	1.13	0.48	0.219

[a] CSF, cerebral spinal fluid.
[b] E: Young's modulus.
[c] G: shear modulus.
[d] K: Bulk modulus.

A Young's modulus of 13 GPa was reported for the human mandible (23). When using a Young's modulus of over 10 GPa, the model responses were very stiff. One possible reason is that the highly porous characteristic of the human facial bony structure was not explicitly implemented in the current model. As such, the stiffness of the cortical bone was adjusted and assumed to be approximately 50% (Young's modulus of 6 GPa) lower than those reported in literature or those used previously that did provide a realistic deflection (7, 172, 175). An ultimate strain and stress of 1.6% and 4.5 MPa were assumed for the cortical bone of the face, respectively (46). An ultimate strain of 4.5% and an ultimate stress of 4.9 MPa were defined for cancellous bone (169).

Nasal cartilage (hyaline cartilage) was defined as an elastic material under dynamic loading, even though it has been characterized as a biphasic viscoelastic material by Mow and Hayes (98). Its Young's modulus was assumed to be 30 Mpa, and its Poisson's ratio was taken to be 0.45 (58, 98, 99). The facial skin and underlying superficial muscle were assumed to be elastic, with an assumed Young's modulus of 1 MPa. The stress-strain relation was derived based on data reported by Yamada (169), Melvin and Evans (92), and McElhaney et al. (89). Material properties of the remaining components of the WSUHIM used in this study were consistent with those of previous studies.

Table 3 gives the facial material properties. Facial bone resembles a sandwich structure containing cancellous and cortical layers except in the nasal bone where the cancellous layer is not well developed. The cortical layer of the bone in the face other than the mandible is thinner than the inner and outer tables of the cranium. The thin layer of the facial bone was modeled with 1-mm-thick shell elements, whereas the cancellous bone was modeled with solid elements of varying thickness. The maxillary sinuses and ethmoid cells were modeled.

The nasal cartilages form the external and internal framework of the nose. The modeled cartilages included septal, lateral, and alar cartilage of the nose and were connected and attached to the adjacent facial bones. During impact, soft tissues such as muscle, skin, and fatty tissue of the face serve primarily to distribute applied loads over the irregularly shaped bony structure. The soft tissues were modeled as solid elements covering the face and were directly attached to the outer surface of the facial bones.

TABLE 3. Elastic material properties for the face

Material	Density g/cc	Elastic modulus Mpa	Poisson's ratio
Cortical	2.10	6000	0.25
Cancellous	1.00	560	0.30
Cartilage	1.50	30	0.45
Soft Tissue	1.10	1.0	0.45
Ligament	1.00	32	0.40

Their thickness varied from 3.0 mm at the nose to 8.0 mm at the zygoma, 8.75 mm at the maxilla, and 10 mm at the mandibular regions. The temporomandibular articulation (jaw joint) was also included and stabilized by ligaments. The facial masses, skin, and tissues move with the cranium because of the imposed acceleration in these simulations.

Model Validation with Experimental Data

Nahum intracranial pressure data

Cadaver head impact tests were used to validate intracranial and ventricular pressures in the FE brain model. Nahum et al. (102) conducted forehead impacts at 4.4 to 13.0 m/s using flat, rigid 5 to 6 kg mass covered with padding. Pressure transducers were placed in five regions of the brain to measure intracranial pressure changes during impact in the frontal region adjacent to the impact area, posterior and inferior to the coronal and squamosal suture, parietal areas inferior to the lambdoidal suture on both sides, and the posterior fossae in the occipital area. The padding material was not defined, so padding stiffness was adjusted until the difference in coup pressure was within ±10% of the experiment. This resulted in an elastic modulus of 49 MPa. Predicted coup and contrecoup pressures correlated in time history and peak for five of the six tests, indicating a model response generally within ±15% (170).

Trosseille intracranial pressure data

The FE model was also subjected to intracranial and ventricular pressure validation using data reported by Trosseille et al. (152). In their study, cadaver head impacts were carried out at 7 m/s with a 23.4 kg mass loading the facial region. A 12-accelerometer array was attached to the occipital region to measure 3D head kinematics. Miniature pressure transducers were placed in the subarachnoid space and ventricles. Validation was carried out for one test with complete three translational and three rotational accelerations at the head cg, intracranial pressures measured in the frontal and occipital lobes, and ventricular pressures in the lateral and third ventricles. The cranium in the FE model was assumed rigid, so the head accelerations could be transferred to the brain boundary bones. The calculated pressures matched in time and peak values up to 14 ms (170).

Brain motion data

The validity of the FE model to simulate brain motion with respect to the cranium was tested using cadaver head impacts. Relative motion between the cranium and the brain was determined at different regions of the brain with high-speed biplanar x-rays combined with radio-opaque neutral-density targets (NDTs) embedded in the cranium and brain (57, 75). Two columns of five to six NDTs were implanted in the temporoparietal and occipitoparietal regions approximately 10 mm apart.

The head and neck was removed at the second thoracic vertebra (T2), inverted, and attached to a trolley fixture. A 152 mm diameter impactor struck the occiput at 2 to 4 m/s, and brain target motion was videoed by x-ray contrast at 250 frames/second. Skull kinematics were determined using the 3 to 2–2 to 2 accelerometer array (114) mounted on the apex of the cranium.

Motion analysis was limited to the resultant displacement in a laboratory coordinates. Some aspects of this validation are described by Al-Bsharat et al. (7). Hardy et al. (57) transformed the target motion onto an anatomic coordinate system with its origin at the head cg. Skull accelerations were applied to the FE model cg to validate it against cadaver brain motion for a 2 m/s impact. The peak resultant linear and angular accelerations were 24 g and 1,813 rad/s^2, respectively. The overall brain motion in FE showed the same figure-eight motion as in the experiment, reaching 3 to 6 mm excursion at 24 to 26 ms. The excursions of brain targets at different locations in the model resembled the experimental data with some exceptions (170). Coronal and sagittal sections were selected from the model to map displacement contours in other regions of the brain. The superior corner of the lateral ventricle, the caudate nucleus, the amygdala, and the hippocampus experienced displacements of 4 to 5 mm as did the anterior commissure, midbrain, and temporal lobes. The brain cortex sustained displacements of 3 mm at the precentral gyrus and postcentral gyrus.

Figure 4A compares the three components (x, y, and z) of brain displacement between the FE model and experiment for two targets (a3 and p3). These two targets were located at the transverse plane through the anterior commissure and pineal body. The model predicted greater y-displacement (3 mm) but less z displacement than that measured at a3. For p3, both y and z displacements matched experimental measurements. The brain excursion trends predicted by the model resemble those patterns observed in experiments.

When brain displacements were compared for each NDT, the FE y-displacement was lower than the experiment (3 mm), whereas the z-displacement was greater, up to 25 ms for the anterior targets 1 and 2. For anterior target 3 and 4, the model predicted greater y-displacement (3 mm) than that measured. For the posterior targets, the FE model predictions matched the experimental data at target 3 and over predicted at target 2 and 4. The geometrical and structural features may affect the degree of brain motion at certain targets location. For instance, relative motions predicted for targets a1 and p5 (targets located near the top of the brain and lateral ventricle) may be affected by the brain-cranium interface and ventricular fluid.

Validation of Brain Motion in Higher-Speed Sagittal Impacts

Cadaver test C383-T1 was a frontal head impact generating sagittal plane motion with a peak resultant linear acceleration of $62 \times g$ and rotational acceleration of 2739 rad/s^2 (57). *Figure 4B* compares the brain-cranium relative motions between model predictions and experimental measurements at two target (a3 and p4) locations. These two targets were located at the same position as those for the coronal motion analysis. The displacements compared were in x (anterior-posterior) and z (inferior-superior) directions because the head moved mainly in the sagittal plane. The first 50 ms of displacements is of interest as it is

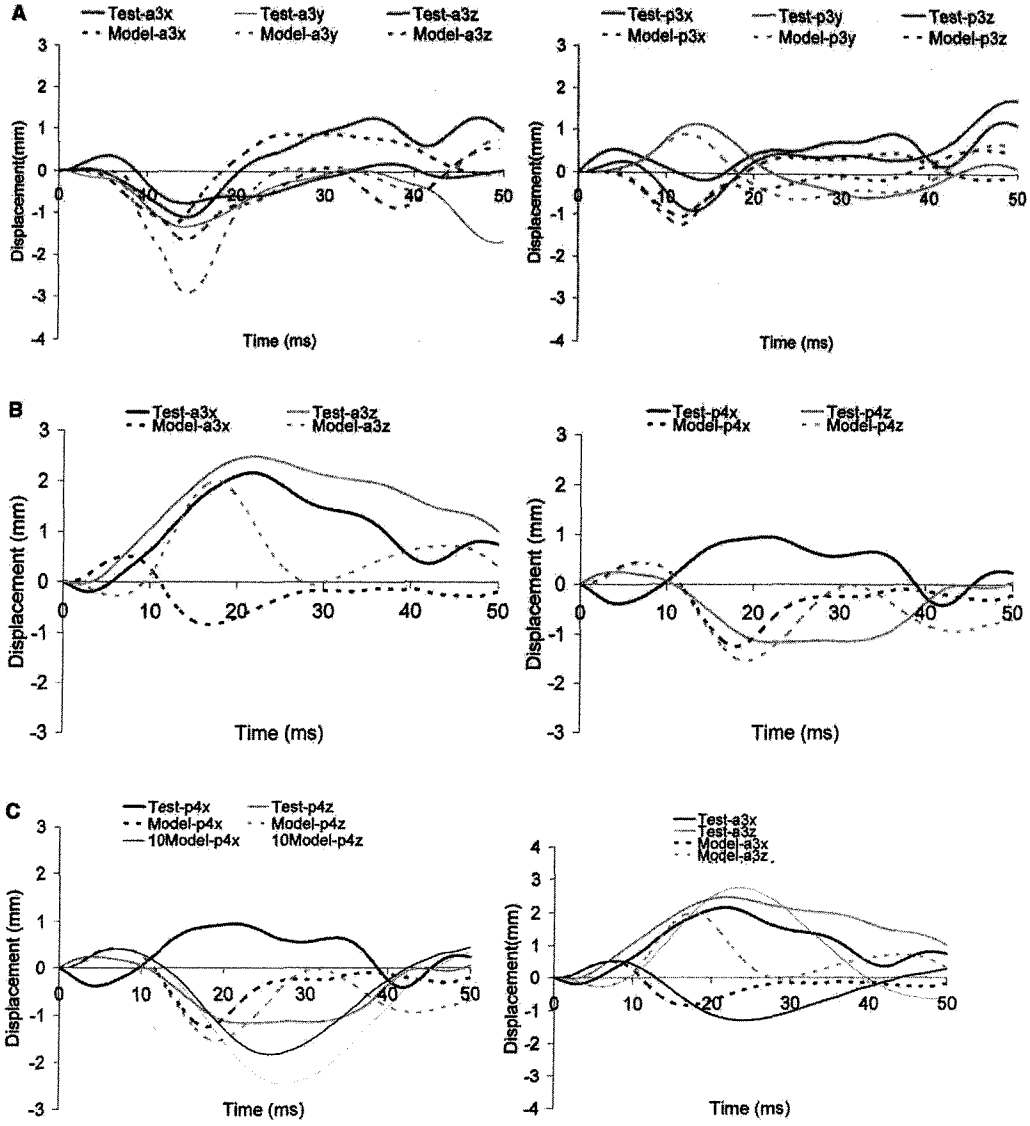

FIGURE 4. A, *comparison of the brain/cranium relative motion for two targets (a3 and p3) between model predictions and experimental measurements for Test 291-T1. These two targets were located at the tranverse plane through the anterior commissure and pineal body. A2 and p3 were approximately 10 mm anterior and 30 mm posterior to the cg of the head, respectively.* B, *comparison of the brain/cranium relative motion for target a3 and p4 between model predictions and experimental measurements for Test C383-T1.* C, *relative displacements in the x- and z-directions observed experimentally and from baseline model or from the 10B Model for targets a3 and p4.*

relevant to the duration of collision sustained by football players. The model displacements in z-direction were comparable with the test data in terms of magnitude. However, x-displacements were under predicted by the model, and the predicted directions were opposite to the direction of x-displacement seen in experimentally. The inconsistency in displacement direction may be a consequence of the difference in the cg location between the cadaver head and the FE model.

When displacements are compared for each NDT, the x-direction relative displacements for anterior and posterior targets 1, 2, and 3 predicted by the model were less than those observed experimentally. On the other hand, displacements for both anterior and posterior targets 4, 5, and 6 predicted by model were comparable with the experimental values. The average maximum brain excursion was approximately 3 mm predicted by the model and 5 mm obtained experimentally.

Parametric Study of Brain Responses

Whereas pressure responses validated well, the correlation between model-predicted relative displacements and experimental measurements was less satisfactory at some NDT locations. Several factors may contribute to the variations.

First, the geometry and material properties used by the WSUHIM might be different from those cadaver heads tested. Therefore, it is unrealistic to expect an exact match between experimental measurements and model predictions. Second, the model assumed a rigid cranium, whereas the cadaver head may deform during impact. Third, the test setup we used was quite complex, and therefore experimental artifacts might exist. Fourth, the experimental methods used to derive material properties are complex and subject to many influencing factors. Fifth, the volume compliance of FE model is assumed equal to that of water and is several orders in magnitude great than that of the human (160). The last factor alone contributes to lower brain displacements with translational acceleration.

A series of parametric studies was conducted to confirm the sensitivity of brain response to varying viscosity defined for the shear relaxation modulus. Three new simulations, namely, the 0.1BModel, 0.5BModel, and 10BModel, were conducted in this parametric study using a decay constant that was 0.1 (β = 8 s-1), 0.5 (40 s-1), and 10 (800 s-1) times that used in the

baseline model, respectively. *Figure 4C* shows the relative displacements in the x- and z-direction observed experimentally and from baseline model or from the 10BModel for targets a3 and p4. The results show that both the peak relative displacement and time predicted by the 10BModel increased as compared with the original model. As a result of this increase, model predictions correlated better with experimental data (z-components at a3 and p4). A decay constant that is 10 times that of the original value increased the peak relative displacement by as much as 40% at all target locations. Results based on this cadaver appear to indicate that the magnitude of the decay constant has an effect on brain deformations and should be measured experimentally in the future to identify an appropriate value for use in modeling long-duration head impacts. Although it may be advisable to use decay constant of 600 to 1000 seconds-1 for the simulation of brain displacements for long-duration impacts, this study uses the commonly used value of 80 seconds-1. This appears to give a good balance in the validity of brain displacements, deformation, and pressure for the loading conditions in the helmet impacts.

Head Accelerations from NFL Game Reconstructions of Concussion Impacts

Details of the game film selection and analysis of concussive impacts can be found in Pellman et al. (122, 123). For this study, a short overview of the laboratory methods is provided. When an MTBI occurred on the field, it was reported to Biokinetics and Associates Ltd. (Ottawa, Ontario, K1H 1E1, Canada), the engineering group contracted to analyze and reconstruct game impacts. Network tape of games was obtained from the NFL and subsequently analyzed. In addition to concussion impacts, cases of significant head impact were also selected for analysis. During 1996 to 2001, 182 cases were obtained on video for analysis. The initial analysis determined the impact location on the helmet and contact (helmet, ground, shoulder, etc.). Sixty-one percent of the collisions involved helmet-to-helmet impacts (122).

Biokinetics determined the feasibility of determining the 3D impact velocity, orientation, and helmet kinematics. At least two clear views were necessary to make this analysis. For those cases where the 3D impact velocity could be analyzed, a laboratory setup with crash dummies was made to re-enact the game collision. Helmets were placed on the dummies in the laboratory reconstructions, and the velocity and orientation of impact was simulated along with the subsequent helmet kinematics. A number of significant impacts were also reconstructed where MTBI did not occur to study nonconcussion impacts. In total, 28 NFL helmet-to-helmet collisions were reconstructed, 22 involved concussion of the struck player, and 6 no injury.

Laboratory Reconstruction Techniques

The laboratory reconstruction involved two Hybrid III male dummies. A helmeted head-neck assembly representing the struck player was attached to a 7.1 kg mass simulating the struck player's torso and guided in freefall from a height to match the impact velocity determined from video analysis of the game collision. Impact was against another helmeted head-neck assembly attached to the torso and pelvis of the Hybrid III dummy. This dummy was suspended by flexible cables.

Acceleration was measured in both dummy heads. The cg of the head is a reference point, which is defined by its position in three orthogonal axes. The motion of the head cg is defined by three orthogonal components of velocity and acceleration. The acceleration is translational even though the trajectory is curvilinear. Because the head cg moves in space under translational acceleration, it can also rotate about the head cg. This involves rotational acceleration, and there are three orthogonal axes for rotational acceleration and velocity. When the head is assumed rigid, as in the dummy, the three axes of translational and rotational acceleration define the motion sequence of the head during impact. The sign convention had positive z-axis from the neck upward through the top of the head (138). The positive x-axis is forward, and the positive y-axis is through the left ear.

Each headform was equipped with standard accelerometers at the head cg and nine linear accelerometers set up in a so-called 3 to 2–2 to 2 configuration to determine rotational acceleration by the method of Padgaonkar et al. (114). The analysis is valid for accelerometers coincident with the origin of head cg or coincident with one of the axes. Deviations from this were required in the headform configuration used in these tests, and a correction for centripetal and Coriolis acceleration was made according to DiMasi (30, 31).

High-speed video recorded head kinematics in the reconstruction. The camera was positioned identical to one of the views from the game video. This allowed a one-to-one comparison of the game and reconstruction kinematics and facilitated fine adjustments in the impact orientation and alignment of the laboratory impact to closely match the helmet kinematics in the game. An extensive analysis was conducted to isolate and quantify sources of variability in the reconstructions (105). This work showed the reconstructions to be repeatable and with minimal error for this type of testing. Every effort was made to reduce potential errors in the laboratory reconstructions.

FE Model Simulations

The three translational and three rotational accelerations at the cg of the head were used as input into the WSUHIM to simulate the game impact. By defining the cranium as a rigid body, the FE cranium is moved in the same way as the Hybrid III in the laboratory reconstruction. The primary duration of NFL impacts was 15 to 20 ms, and the duration of the simulation was 30 ms to capture brain displacements and deformation. Twenty-eight cases of struck players were analyzed in this study including helmet-facemask, helmet-helmet, and helmet-ground impacts. Biomechanical responses in the brain included intracranial pressure, maximum shear stress, first principal strain, and first principal strain rate to assess the association between FE model responses and injury, signs, and symptoms and other on-field outcomes.

Grouping of Game Impacts and FE Simulations

The reconstructed game collisions were grouped in four types (A–D) depending on the orientation of the helmet contact. With eyes forward being the 0° reference, four quadrants from front to back of the helmet were defined with left side impacts reflected to the right side. The categories included A: 0° to 45°, B: 45° to 90°, C: 90° to 135°, and D: 135° to 180°. Details of this classification and grouping of head accelerations can be found in Pellman et al. (122).

Correlation with Concussion Signs and Symptoms

The individual brain responses for the reconstructed cases of concussion were used to compare the sites of high-strain response, with the clinical signs and symptoms occurring in the players as reported by the team physicians. Although some of the 32 signs and symptoms cannot be localized to sites in the brain, a number may arise from specific brain regions. "Hot spots" were identified as regions of the brain with high relative strain responses in comparison with surrounding areas. They were visualized as colored regions in the FE simulation at various times in the strain and strain-rate response of the brain. Because of consistency in the simulated brain responses, "hot spots" in various regions of the brain were shown during the early response, which coincided with the peak in translational acceleration. The mid and late responses occurred after the primary impact acceleration during the rapid displacement of the cranium. The mid and late responses were combined for this analysis because the sites and level of strain and strain-rate were found to be similar. This provides two brain deformations (strain and strain-rate) at two times (early and mid-late). The initial interest was to also report on the product of strain and strain-rate; but the FE code does not have that as a standard output, so it could not be systematically determined from the simulation.

Head Injury Tolerances

Head accelerations were used to determine the SI and HIC head injury criteria (137). In addition, the peak rotational acceleration and rotational velocity was used to assess injury risks. Details of these parameters can be found in Pellman et al. (122, 123).

Statistical Analyses of Concussion

The significance of differences in responses for the struck player with concussion or no injury was determined using the standard t test with two-tail distribution. Before this analysis, Levene's test for variance was run to determine whether the t test could assume equal variance for a comparison (62). For those cases with $P < 0.05$ in the Levene's test, the t test assumed unequal variance. The t test was performed using the standard analysis package in Excel. The regression analysis was also used from Excel, which determined the average and 95% confidence interval for a linear fit between response data.

RESULTS

Head and Brain Responses: Example Case 124

Case 124 from the NFL game reconstructions is presented to illustrate head and brain responses in the helmet impacts. *Figure 5* shows the head dynamics measured in the Hybrid III dummy. Plots on the left show the translational response and those on the right, the rotational response. The primary impact causes a rise in translational acceleration to a peak in 8 ms, and the impact is essentially over by 15 ms. This causes a delta V of 6.2 m/s and 42 mm displacement of the head cg at 15 ms. The initial rotation is about the z-axis and then it shifts to the y and x direction, causing a resultant rotational velocity of 26 r/s and rotation of 110 at 15 ms. Between 15 and 25 ms, head displacement increases to 115 mm and the rotation to 240. These responses are secondary to the momentum exchange in the impact, but the cranium motion continues to deform the brain because of the rapid motion.

Figure 6 shows the distortion of the brain at 15 ms and 25 ms. The impact displaces and rotates the cranium, producing curvature in the midline between the hemispheres at 15 ms. This focuses deformation in the midbrain at 25 ms, with substantial distortion of brain tissues. The displacement time histories show the lag in brain motion at the midbrain (center), particularly in the y-displacement, which approaches 6 mm at 25 ms. The displacements under the impact point (impact) and along the axis of loading (remote) show earlier and lower responses.

Figure 7 shows "hot spots" of strain and strain-rate at three times in the impact. The early pattern occurs at 8 ms during peak head acceleration while strain is gradually increasing in magnitude. The "hot spots" are in the temporal lobe adjacent to the impact site in this oblique lateral collision. The mid response is at 18 ms and occurs after the primary impact. It shows a shift in "hot spots" to the temporal lobe in the far side of the brain. By the late response at 22 ms, the "hot spots" have moved to the midbrain regions including the fornix and Ammon. Nine of the 22 concussions showed this specific migration of strain from the near to far temporal lobes and then to the midbrain. When condition D was excluded, it occurred in approximately half of the cases. All 22 concussions showed the later strain "hot spots" in the regions of the fornix, midbrain, and corpus callosum.

Correlation of Concussion with Head and Brain Responses

Table 4 shows the significant correlations between the occurrence of concussion and Hybrid III head and FE brain responses. Only correlations with $P < 0.10$ are given from the many possible analyses conducted with the 28 laboratory reconstructions and FE modeling of the brain responses of struck players. The full data is given in the Appendix. The strongest correlation of concussion was with HIC and SI, which are measures of head injury severity from the Hybrid III dummy. There was also significant correlation with head delta V and peak translational and rotational acceleration, but the P values were lower in significance. For the FE simulations, mid-late strain and strain-rate in the midbrain and fornix showed the strongest correlation with the

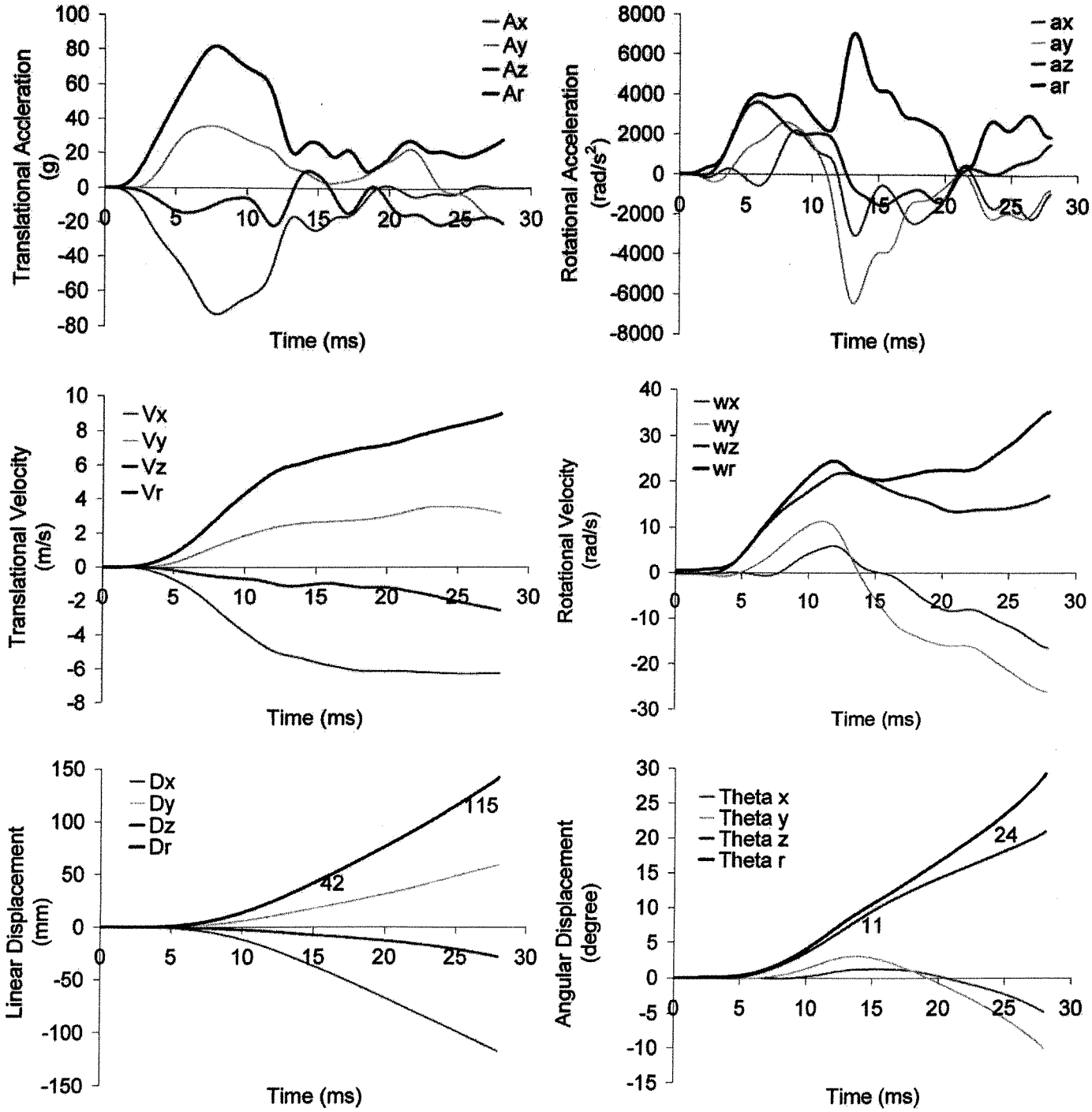

FIGURE 5. *Hybrid III head dynamics for Case 124, including 3D translational and rotational acceleration of the cranium. The x, y, and z components of the response and resultant are given. Integration gives the component translational and rotational velocities and displacements of the head.*

occurrence of concussion of all regions of the brain. This was consistent with the "hot spots" moving to the midbrain region later in the brain response, as shown in the example case. The average mid-late strains are in the range of 0.35 to 0.45 with concussion and they are statistically lower without injury. The average strain-rates are in the range of 60 to 80 1/s with concussion and are statistically lower without injury. These correlations indicate that concussion is related to brain deformations occur-

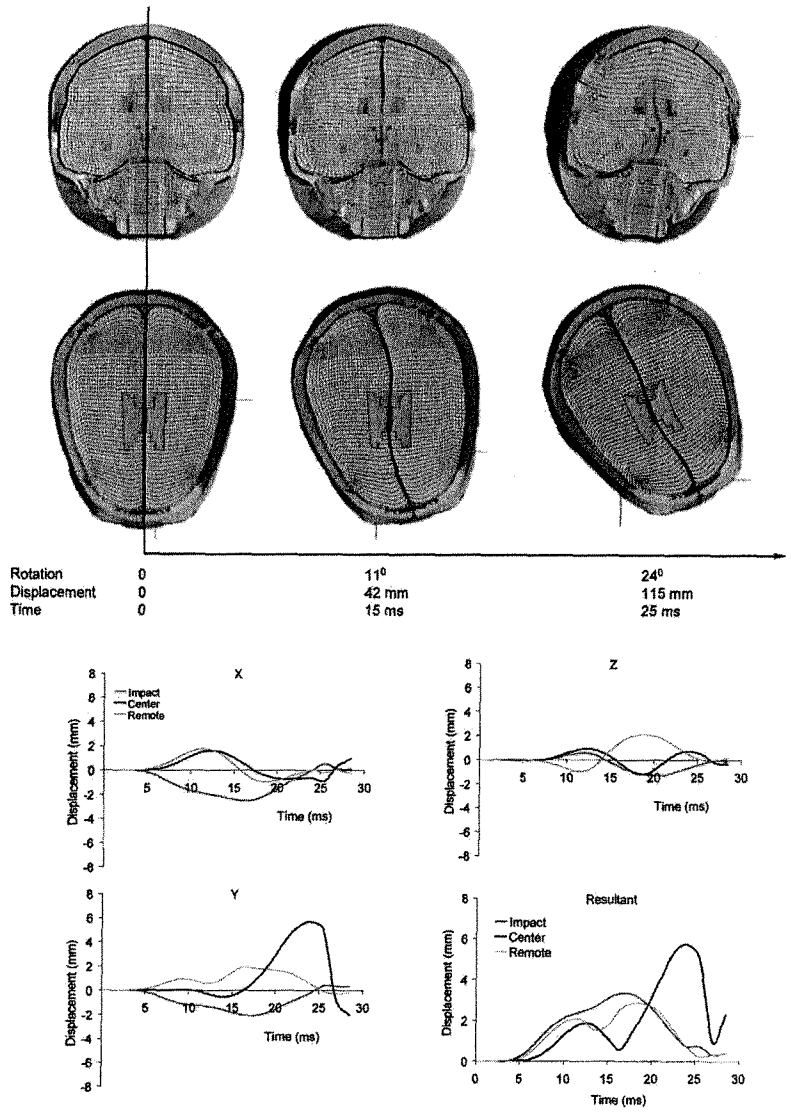

FIGURE 6. *Deformation pattern of the FE brain elements from a superior view of the hemispheres at 15 and 25 ms for Case 124, along with brain displacements along the axis of impact adjacent to the contact (impact), central, and remote (contrecoup).*

ring after the primary head impact and momentum transfer. This phase of the response involves rapid displacement and rotation of the head once the delta V and rotational velocity have occurred.

Correlation of Signs, Symptoms and Outcomes with Head and Brain Responses

Table 5 shows the significant correlations between specific signs, symptoms, and outcomes of concussion and Hybrid III and FE brain responses. Again, only correlations with $P < 0.10$ are shown from the many possible analyses conducted. There were only 11 significant correlations. Interestingly, none of the Hybrid III responses correlated with specific signs, symptoms, or outcomes. Return to play is a measure of the severity of concussion in terms of signs and symptoms affecting memory, cognition, and somatic function (119). Mid-late strains of 30% in the fornix correlated with not returning to play. Mid-late strain-rate of 78 1/s in the corpus callosum also correlated with removal from play.

Memory and cognition problems correlated with mid-late strains of 30 to 50% in the fornix and midbrain and strain-rates over 90 1/s in the midbrain. Loss of consciousness occurred in only four concussed players but correlated with mid-late strain-rates over 100 1/s in the midbrain. Dizziness was common after concussion. It correlated with early strains of 14 to 18% in the orbital frontal cortex and temporal lobe.

Correlation of Direction of Helmet Impact with Head and Brain Responses

Table 6 shows the significant correlations between the direction of helmet impact and early and mid-late FE brain and Hybrid III responses. Only correlations with $P < 0.10$ are shown. There were only 10 significant correlations. Six of the differences were between direction B and C with primarily mid-late strain and strain-rate in regions of the midbrain. There was a significant difference in rotational velocity. Three difference were found between direction D and the others in HIC and delta V, which are consistent with direction D being falls to the ground with loading of the back of the helmet versus directions A–C being essentially helmet-to-helmet impacts either oblique on the face mask (A) or lateral on the helmet shell (B and C).

DISCUSSION

FE Strain "Hot Spots" Versus the Signs, Symptoms, and Outcome of Concussion

This study is unique. It is the first of its kind to correlate the physical forces occurring in concussion (biomechanics) with the clinical characteristics (neurobiology) of actual injured players. How the physical forces acting upon specific brain regions are manifested as clinical signs and symptoms is unknown, but deformation of brain tissue is the underlying biomechanical cause for neurobiological affects on function. It can be postulated that the strain or strain-rates measured by the FE model can cause purely physiological dysfunction, including biochemical changes, without anatomic correlates, submicroscopic anatomic injury, microscopic anatomic lesions, or gross anatomic lesions in the affected brain regions.

Clinical symptoms or signs can arise from the injury to the affected brain areas by many possible means. The affected brain region may be responsible for a certain brain function, and this

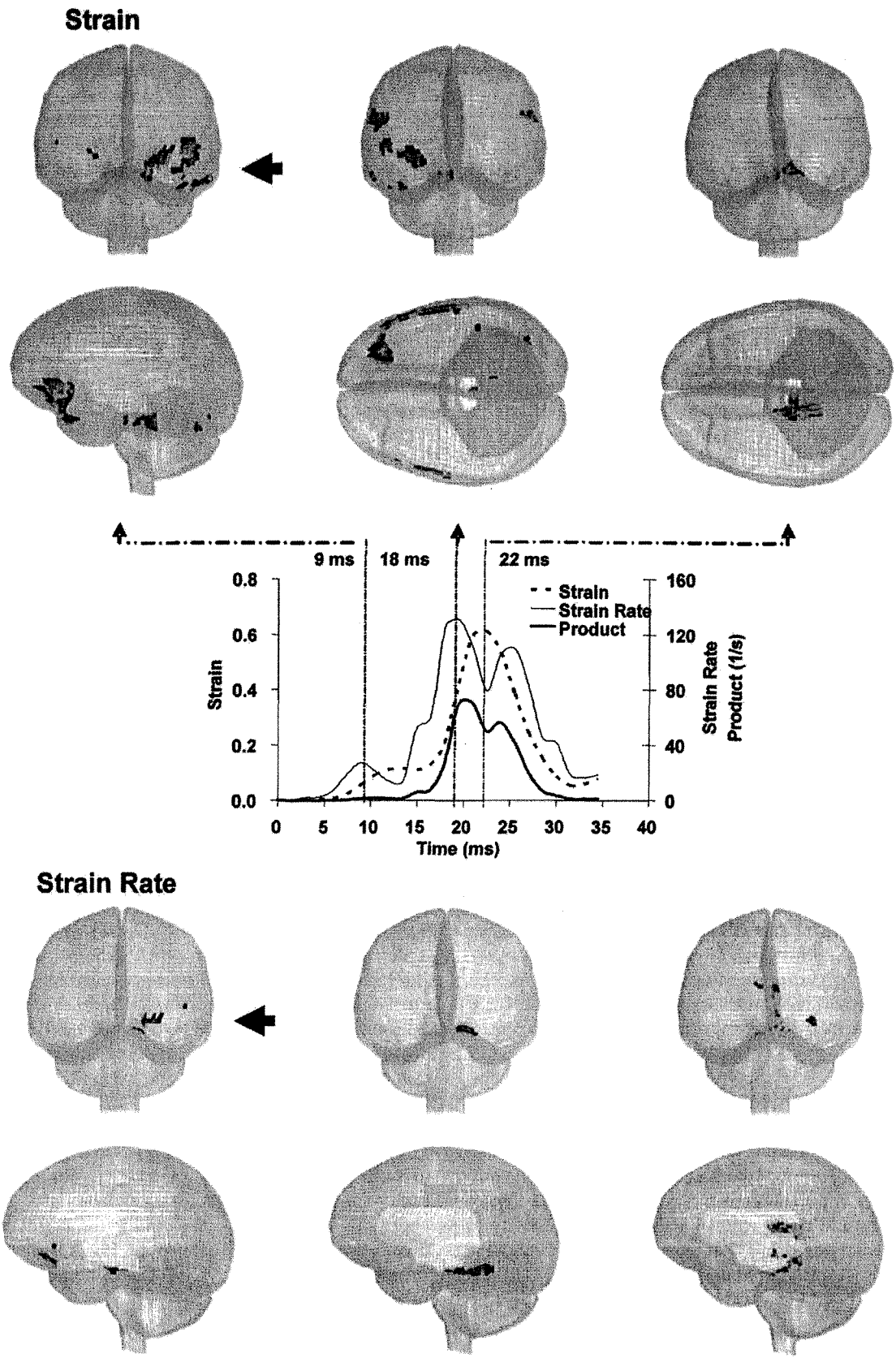

FIGURE 7. *Strain, strain-rate, and product of strain and strain-rate "hot spots" early (8 ms) during the head acceleration, and mid (18 ms) to late (22 ms) during the free-motion displacement and rotation of the head. "Hot spots" are areas of the brain with high levels of response relative to the surrounding tissues of the brain. They show regions of the brain with proportionally greater deformation. The arrow shows the direction of impact in the early response.*

TABLE 4. Significant *t* test results $P < 0.10$ for the occurrence of concussion versus the hybrid III and finite element responses. Finite element brain responses are for strain and strain rates in regions of the brain early or late[a]

		Concussed (n = 22)		No injury (n = 6)		*t*	*t* test *df*	*P*
		Mean	SD	Mean	SD			
Hybrid III responses								
HIC[a,b]		339	215	121	64	4.14	26	0.000
SI[a,b]		422	270	154	82	4.03	25	0.000
Translational acc (g)		90	32	60	24	2.18	26	0.039
Delta V[b] (m/s)		6.9	2.0	5.0	1.1	3.07	15	0.008
Rotational acc (r/s2)[a,b]		6106	1984	4235	1716	2.10	26	0.046
FE modeling strain[a]								
Parahipp, uncal regions[a,b]	Early	0.108	0.094	0.043	0.037	2.57	22	0.017
Midbrain	Mid-late	0.344	0.140	0.226	0.111	1.91	26	0.067
Thalamus, hypothal[a]	Mid-late	0.376	0.193	0.208	0.122	2.01	26	0.055
Fornix and/or midbrain	Mid-late	0.448	0.176	0.217	0.153	2.92	26	0.007
Hypothalamus	Mid-late	0.373	0.178	0.210	0.138	2.07	26	0.049
Ammon, parahipp[a]	Mid-late	0.416	0.165	0.170	0.111	3.42	26	0.002
Orbito-frontal-temporal	Mid-late	0.317	0.139	0.193	0.149	1.90	26	0.069
FE modeling strain rate[a]								
Orbito-frontal temporal	Early	60.0	26.8	33.7	19.6	2.23	26	0.034
Temporal	Early	55.0	29.0	30.7	15.5	1.96	26	0.061
Parahipp[a,b]	Early	34.5	28.2	17.0	9.3	2.46	25	0.021
Midbrain	Mid-late	79.3	37.4	36.3	16.3	2.72	26	0.012
Thalamus	Mid-late	74.4	37.6	43.8	25.7	1.86	26	0.074
Fornix and/or midbrain	Mid-late	81.5	39.7	45.2	25.5	2.11	26	0.044
Ammon, parahipp[a]	Mid-late	70.4	29.9	43.2	21.5	2.08	26	0.048
Orbito-frontal-temporal	Mid-late	61.4	27.6	36.7	18.0	2.06	26	0.049

[a] Acc, accelerations; FE, finite element; SI, Gadd Severity Index; HIC, head injury criterion; hypothal, hypothalamus; Parahipp, parahippocampal.
[b] Based on Levene's test for equality of variances. Sig < 0.05 and unequal variance is assumed for the *t* test.

function may be directly impaired or lost by injury to that region. The affected brain region may have an inhibitory effect upon the function(s) of one or many other brain structures, and the injury may result in the over-expression of those other functions. The affected brain area may serve as a relay station between other brain regions, and injury may disrupt the communication between multiple brain structures.

When analyzing the clinical effects of brain lesions, including in this article those purely physiological dysfunctions caused by physical forces, it must be remembered that symptoms from lesions of a part of the nervous system are not to be equated with the function of that part. As Adams and Victor (2) stated, "symptoms of a lesion in a region are the product of both a loss of certain functions and the functional activity or over activity of portions of the nervous system that remain intact." Although the results of this study may aid in the understanding of MTBI and perhaps general brain functioning, one must be cautious in interpreting the findings.

Return to Play

The clinical severity of MTBI was correlated with the strain-strain rates in the FE model. In earlier articles, the authors have used a functional approach to determining concussion severity (119, 120). Those studies demonstrated that players who were out of play for more than 7 days had more MTBI signs and symptoms than those who were out for less than 7 days (120) and that players who were removed from play but returned later that game had more signs and symptoms of MTBI than those who returned to the game immediately after injury (120). Only two of the players in this present FE analysis were kept out of play for more than 7 days. This was too small a number to allow for statistical analysis. The ability or inability to return to play on the day of the injury was used as a marker of concussion severity. Players who returned to play on the day of the injury (immediately returned and rested and returned) were considered to have sustained a less severe concussion than those who did not return to play on the day of the injury. This obviously includes the two players who were out for more than 7 days.

Statistical analysis of these two groups is summarized in *Table 5* and showed a correlation between high strain or strain-rates in the mid-late time frame in the fornix and the corpus callosum and not returning to play on the day of the injury because of a more severe concussion. High strains in the fornix also correlated with memory impairments. The authors' previous studies demonstrated that clinical memory or cognitive impairments were correlated with delayed return to play (119, 120). This may explain why high strain in the fornix is related to this marker of concussion severity. The correlation with high strain-rates in the corpus callosum may have another explanation. The corpus callosum is the largest of the white matter brain structures that contain fibers linking the left and right cerebral hemispheres (9). Disruption of these pathways might result in impairment of many brain functions and delay return to play after MTBI.

TABLE 5. Significant *t* test results $P < 0.10$ for the occurrence of specific signs symptoms and outcomes from concussion versus finite element brain responses. Finite element brain responses are strain and strain rates in regions of the brain early or late[a]

			n	Mean	SD	n	Mean	SD	*t* test	*df*	*P*
Return to play			No			Yes					
Fornix	Strain	Mid-late	16	0.318	0.125	12	0.199	0.136	2.393	26	0.024
Corpus callosum[b]	Strain rate	Mid-late	16	78.1	40.5	12	54.9	24.0	1.886	25	0.071
Loss of consciousness			Yes			No					
Midbrain	Strain rate	Mid-late	4	105.8	40.9	24	64.2	35.2	2.145	26	0.042
Memory problems			Yes			No					
Fornix	Strain	Mid-late	10	0.334	0.150	18	0.229	0.124	1.99	26	0.057
Fornix and/or midbrain	Strain	Mid-late	10	0.491	0.210	18	0.347	0.170	1.97	26	0.060
Cognitive problems			Yes			No					
Midbrain	Strain rate	Mid-late	6	93.8	38.6	22	63.6	36.3	1.783	26	0.086
Cranial nerve problems			Yes			No					
Orbito-frontal temporal	Strain	Early	12	0.164	0.103	16	0.104	0.066	1.866	26	0.073
Hypothalamus	Strain	Mid-late	12	0.410	0.190	16	0.284	0.158	1.917	26	0.066
Ammon, parahipp[a]	Strain	Mid-late	12	0.443	0.189	16	0.303	0.161	2.103	26	0.045
Dizziness			Yes			No					
Orbital-frontal cortex	Strain	Early	9	0.176	0.099	19	0.108	0.075	1.994	26	0.057
Temporal lobe	Strain	Early	9	0.146	0.070	19	0.095	0.069	1.800	26	0.083

[a] FE, finite element; Parahipp, parahippocampal.
[b] Based on Levene's test for equality of variances. Sig < 0.05 and unequal variance is assumed for the *t* test.

TABLE 6. Significant *t* test results $P < 0.10$ for the direction of impact versus hybrid III dummy and finite element brain responses. Finite element brain responses are strain and strain rates in regions of the brain early or late[a]

			n	Mean	SD	n	Mean	SD	*t* test	*df*	*P*
Impact directions			A			C					
Midbrain[a]	Strain rate	Mid-late	11	83.6	50.0	6	48.2	22.9	1.99	15	0.064
			B			C					
Rotational velocity (r/s)	—	—	9	38.9	12.8	6	25.2	12.2	2.07	13	0.058
Fornix and/or midbrain	Strain	Mid-late	9	0.477	0.153	6	0.292	0.182	2.14	13	0.052
Ammon plus parahipp[a]	Strain	Mid-late	9	0.436	0.168	6	0.272	0.176	1.82	13	0.092
Temporal	Strain rate	Early	9	64.2	24.1	6	42.2	20.5	1.84	13	0.089
Midbrain	Strain rate	Mid-late	9	72.8	27.2	6	48.2	22.9	1.82	13	0.091
Orbito-frontal-temporal	Strain rate	Mid-late	9	70.4	32.4	6	42.5	22.3	1.83	13	0.090
			D			A + B + C					
HIC[a]	—	—	2	644	122	26	265	193	2.71	26	0.012
Delta V[b]	—	—	2	8.4	0.1	26	6.3	2.0	5.19	26	0.000
			D			B + C					
HIC[a]	—	—	2	644	122	15	306	228	2.02	15	0.062

[a] HIC, head injury criterion; Parahipp, parahippocampal.
[b] Based on Levene's test for equality of variances. Sig < 0.05 and unequal variance is assumed for the *t* test.

Earlier studies have shown that the corpus callosum is a common site of axonal injury and white matter lesions after closed head injury; and white matter lesions in the corpus callosum are a part of the spectrum of diffuse axonal injury

after head trauma (48, 146, 164). Such lesions are a sign of more severe injury. High strain rates in the mid-late time frame in the corpus callosum may be related to the presence of such white matter lesions, perhaps by direct causation. This could explain the link between high strain rates in the corpus callosum and the clinical marker of severity of MTBI.

The present study demonstrated a significant correlation between high strain or strain-rates in the mid-late time frame in the midbrain, the fornix, and the corpus callosum and cognitive/memory problems, loss of consciousness, or interval to full functional recovery (i.e., as manifested by ability to return to play). All of these structures are in the deep midline brain regions. This suggests that protective devices that could lower the mid-late time frame strain-strain rates in these midline brain regions might lessen the severity of MTBI.

Loss of Consciousness

Loss of consciousness occurs infrequently with concussion in the NFL (118). Many authors have suggested that it is a result of upper brainstem dysfunctions (109, 139, 142, 164). The results of the present study confirm these earlier statements. High strain rates during the mid-late time frame in the midbrain are statistically correlated with the occurrence of loss of consciousness. This is most likely caused by dysfunction in the ascending reticular activating system, which plays a major role in the maintenance of consciousness (109, 139, 142, 164).

Memory and Cognitive Problems

For the purposes of this analysis, cognitive problems included difficulties with immediate recall and disorientation, whereas memory problems included retrograde or anterograde amnesia, information processing difficulties, and altered attentional processes. These probably are artificial, arbitrary distinctions. The authors therefore will discuss cognitive and memory problems together. Cognitive and memory functions are subserved by complex interactions between many areas of the brain. It is generally accepted that the functions of short-term memory, including immediate recall, are subserved by the medial temporal lobes (hippocampus, parahippocampus) and medial diencephalic structures (22, 56, 71, 164). It is therefore of great interest to note that in this study there was no correlation between high strains and/or strain-rate in these anatomical structures and clinical cognitive or memory dysfunctions. This study did reveal a correlation between cognitive and memory problems and high strain and/or strain-rate in the midbrain and fornix during the mid-late timeframes. The relationship with high strain in the fornix can be understood because the fornix is one of the main outflow pathways from the hippocampus (69).

Dysfunction in the outflow from one of the regions specifically associated with short-term memory function could be expected to result in clinical memory impairments. The correlation between high midbrain strain and strain-rate, and clinical memory and/or cognitive impairments is not so easily explained. One of the clinical symptoms included in this analysis was "attention problems." This function is at least partially related to levels of arousal and alertness. Its is well known that the ARAS arises in the midbrain and connects with widespread areas of the limbic lobes and cerebral hemispheres, and modulates consciousness, arousal and alertness (139, 164). Perhaps high strain and/or strain-rate in the midbrain impair the functioning of the ARAS, secondarily resulting in clinical difficulties with attention.

The midbrain is interconnected by multiple pathways to the diencephalon, the temporal lobes, the limbic lobes, and multiple other areas of both cerebral hemispheres (139, 164). High strain or strain-rates in the midbrain could affect some or all of these pathways, resulting in clinically apparent dysfunction in some or all of those other brain areas. In this way, cognitive or memory impairments of seemingly medial temporal lobe and medial diencephalic origin could arise secondarily to midbrain dysfunction. Another possible way to explain the correlation with midbrain strain and strain-rate is more biomechanical and less neurobiological.

Cranial Nerve Problems

Symptoms and signs listed under "cranial nerve problems" in this study included dizziness, blurred vision, vertigo, photophobia, tinnitus, diplopia, nystagmus, abnormal pupils, and hearing loss. In the entire cohort of NFL MTBIs over a 6 year period, dizziness was by far the most common of these symptoms with blurred vision, vertigo, photophobia, tinnitus, and diplopia occurring less frequently (118). Among the 28 cases which are included in this FE model analysis, dizziness occurred in nine cases, photophobia in four cases, and none of the other symptoms occurred in more than one case each. Thus, most of the cranial nerve problems in this study were dizziness.

The correlation between cranial nerve problems and high early strain-rate in the orbital frontal cortex is most likely a reflection of the correlation between dizziness and high early strains in the orbital frontal cortex. The correlation between cranial nerve problems and high strain in the mid-late time frame in the hypothalamus and parahippocampal/Ammon's horn regions of the medial temporal lobes requires further explanation. There are numerous connections between the limbic lobes, including the parahippocampal gyri and Ammon's horn, the hypothalamus, and the midbrain (56, 69, 164). It is possible that high strains in the hypothalamus and limbic structures could disrupt these pathways and result in brainstem symptomatology. There was no correlation between cranial nerve symptoms and high strain or strain-rates in posterior fossa structures (brainstem, cerebellum). It is possible that many of the MTBI symptoms ascribed to the brainstem by clinical neurologists are really of cerebral origin.

Dizziness

Dizziness is a nonspecific complaint that encompasses a wide variety of sensory experiences including lightheadedness, wooziness, "faint" feeling, unsteadiness, feelings of ab-

normal movement, rotary sensations of true vertigo, and "not feeling right" in the head (164). True vertigo most likely indicates vestibular dysfunction of central or peripheral origin (37, 164). The other dizziness symptoms noted above are notoriously difficult to localize. All of the above sensations can be experienced by MTBI patients.

The results of the present study indicate that there is at least some role of central nervous system dysfunction involved in the generation of dizziness after MTBI. The correlation between early strain in the orbitofrontal cortex and the temporal lobe cortex and dizziness suggests that cerebral injury plays a role in the development of sensations of dizziness after MTBI. It is possible that orbitofrontal and temporal lobe injury directly results in the symptoms of dizziness, but it is also possible that early high strain in these regions is only a marker of high strains in more diffuse areas of the brain resulting in dizziness.

It is of some interest to note that high strain and strain-rates in the brainstem or cerebellar regions did not correlate with the presence of dizziness. Many of the sensations that fall under the rubric of dizziness (i.e., unsteadiness, sensations of abnormal movement, true vertigo) are often considered to be indicators of brainstem-cerebellar-vestibular dysfunction. The present results indicate that high strain or strain-rates in these areas are not correlated with dizziness. This, however, does not rule out the possibility that the early high strains in the orbitofrontal and temporal cortices disinhibit brainstem cerebellar regions from higher cortical control and thus result in dizziness. The present study cannot address the role of inner ear dysfunction in the generation of dizziness after MTBI because of technical difficulties in evaluating strain and strain-rates in that area with the FE model.

Biomechanics

Migration of "hot spots"

The FE model shows a delayed response of the brain for impact acceleration of the cranium. An early, low strain response occurs during the primary impact. This is focused to the regions of the brain adjacent to the impact site (coup site). Because the NFL concussion impacts are primarily oblique or lateral, the early region of "hot spot" strain is in the temporal lobe. "Hot spots" during the mid-response move to the opposite side on the brain from the impact loading (contrecoup site). This is the far-side temporal lobe. Late in the response, the "hot spots" move to the midbrain above the brainstem and near midline.

This migration of "hot spots" is not a wave propagation phenomena as that would occur at much shorter durations than occur in the FE simulation. The migration appears to be driven by the motion of the head secondary to the delta V and during the rapid free-motion displacement and rotation of the cranium. To our knowledge, this type of timing for the migration of strain has not been reported for concussions and offers insights into the possible underlying mechanisms of injury related to head kinematics and the various signs and symptoms of MTBI in players.

Coup-Contrecoup Injury

Much has been written about coup-contrecoup injuries to the brain. The classical definition of coup injuries are those adjacent to the site of head impact and have been thought to be related to the local deformation of the cranium and brain directly under the impact. Contrecoup injuries have often been restricted to brain contusions on the opposite side of the brain in line with the axis of impact; however, the original definition was for any brain contusions away from the direct site of impact. There have been many theories for coup and contrecoup injury, including wave propagation, cavitation, and cranium deformation (26, 27, 49, 51, 111). The FE modeling offers a new mechanism for contrecoup injury associated with rather long-duration head impacts involving helmets and padding. This is related to the migration of relatively high strain and strain-rate from the coup region during the primary impact to the contrecoup regions of the brain during the rapid motion of the cranium after the primary impact. Clearly, depending on the tolerance to strain, brain injuries can occur at different times in the near and far temporal lobes and midbrain regions over the 30 ms brain response.

Injury Criteria

The correlation of Hybrid III responses with concussion and not the specific signs, symptoms, and outcomes is particularly interesting. The initial analyses showed the strongest correlation of concussion with HIC and SI. These are the traditional measures of head injury risk that have widespread use in all kinds of automotive, sport, and defense safety evaluations. At the time, it was comforting to see that the risk for concussion in the NFL could be accurately predicted by traditional measures of translational acceleration. The measurement of rotational accelerations was encouraged for research purposes, even though there was an inherent relationship between translational and rotational acceleration (123).

Surprisingly, the Hybrid III head responses did not correlate with any of specific signs, symptoms, and outcomes of concussion. There was no correlation with return to play, which is arguably a good indicator of the more severely injured players. No Hybrid III measures correlated with players experiencing memory, cognitive, or somatic problems after injury.

It was interesting that strain and strain-rate responses in specific regions of the brain and phases of the brain response correlated with return to play, cognitive, and memory problems (*Table 5*). The mid-late strain and strain-rate in the midbrain, fornix, parahippocampus, Ammon horn, and corpus callosum correlated with the more severe signs and symptoms of concussion. These correlations imply that FE models and tissue-level injury criteria may provide effective means of addressing specific concussion injuries, particularly the ones responsible for removal from play and longer recovery times. Clearly, the prevention of memory and cognitive problems

with concussion would be an important new focus for research.

The use of FE models and strain-related injury criteria may offer new insights into the timing of concussion injuries and affected locations. It is further possible that new means of preventing concussion can be devised if attention is given to reducing the mid-late strain and strain-rate in the midbrain. Currently, helmet designs are evaluated for the performance in reducing HIC and SI, which assess the loading during the primary impact. Little attention has been paid to means of reducing strain effects occurring mid-late or after the primary impact. The role of the neck and neck musculature may be additional factors, particularly the change in orientation of the head with respect to the neck and deformation of the brainstem.

Obviously, more study will be needed to develop concepts to reduce mid-late strains in the midbrain. Improved padding in the helmet is one means of reducing the energy transfer in the impact and head dynamics early and mid-late or the migration of strain to the midbrain late in the response. There may be other means of changing the mid-late response by interventions that work after the primary impact.

Tissue Level Injury Criteria

Previous studies have shown that the product of strain and strain-rate is an important predictor of brain and spinal cord injury, including contusion and loss of function (161). The product of strain and strain-rate is proportional to the energy absorbed by tissue deformation. It is a cellular level formulation of the Viscous criterion, which has been shown to be a good predictor of soft tissue injury to the heart, lungs, and liver, including interruption of cardiac function and ventricular fibrillation (19). It is unfortunate that modern FE codes do not give the product of strain and strain-rate as a standard or optional output from simulation. In the future, this important measure of injury risk should be determined because it tracks brain injury risks, including concussion.

Concussion and the Direction of Impact

The statistical analyses showed that mid-late strain and strain-rate were higher in the midbrain and fornix for A (0°–45°) and B (45°–90°) direction impacts than C (90°–135°). This was an unexpected difference. A and B impacts are frontal oblique on the facemask and helmet, whereas C in laterally oblique from the rear. The practical implications of this finding are unclear. HIC and SI were similar for the B and C impact directions, and the mid-late strain was statistically higher for the B direction. This implies a greater vulnerability for midbrain strain for the same HIC or SI. Because mid-late strain in the midbrain correlated with the more severe signs, symptoms, and outcomes of concussion, the assessment of injury risk may ultimately be more complicated than just evaluating HIC and SI.

Limitations

Laboratory reconstructions

Extra effort was taken to determine the validity and accuracy of measuring the 3D impact velocity from the NFL game video and rotational acceleration in the Hybrid III dummy (105). However, there is an additional limitation in how realistically the response of the Hybrid III dummy simulates the actual player's head in the helmet. Although care was taken to closely match helmet kinematics before and after the impact, there has been no means to determine possible difference in the head interaction with the helmet padding and face mask. This is thought to be a small source of error but one that has not been quantified.

FE head model volume compliance of the brain-cranium system

One of the possible limitations of the FE model is the representation of the volume compliance of the brain in the cranium. The material property of brain is close to the bulk modulus of water and therefore incompressible. The foramen magnum is the only opening in the cranium, which does not allow much volume compliance during impact. Physiological studies have shown a bilinear compliance with an initial low response rising to a stiffer level after fluid volume is added (160). However, even the stiffer level is more than two orders in magnitude lower than the bulk modulus of water.

In the initial modeling, various simulations were made with a bulk modulus of brain tissue reduced by two orders from that of water to better mimic the volume compliance in situ. However, even with these reductions, the brain still behaved as an incompressible fluid. Simulations in this study used the previously used properties, which makes the model behave as if there was a fluid contained in a relatively closed container. This substantially reduces the brain displacement and deformation caused by translational acceleration, which may limit the realism of the overall simulation.

Additional runs were made with case 124 using either the translational or rotational acceleration input to compare the responses. *Figure 8* is consistent with earlier FE studies (154) and shows that the brain response is dominated by the rotational acceleration because the high bulk modulus prevents appreciable displacement and strain for translational acceleration.

Because concussion was strongly correlated with measures of translational acceleration and delta V, the appropriate modeling of the volume compliance of the brain-cranium system may lead to different "hot spots." There are reasons to believe the model is reasonably representative of the human response, and there is a strong correlation between rotational and translational acceleration. However, this remains unproven and an unquantified source of error, which requires additional evaluation and study.

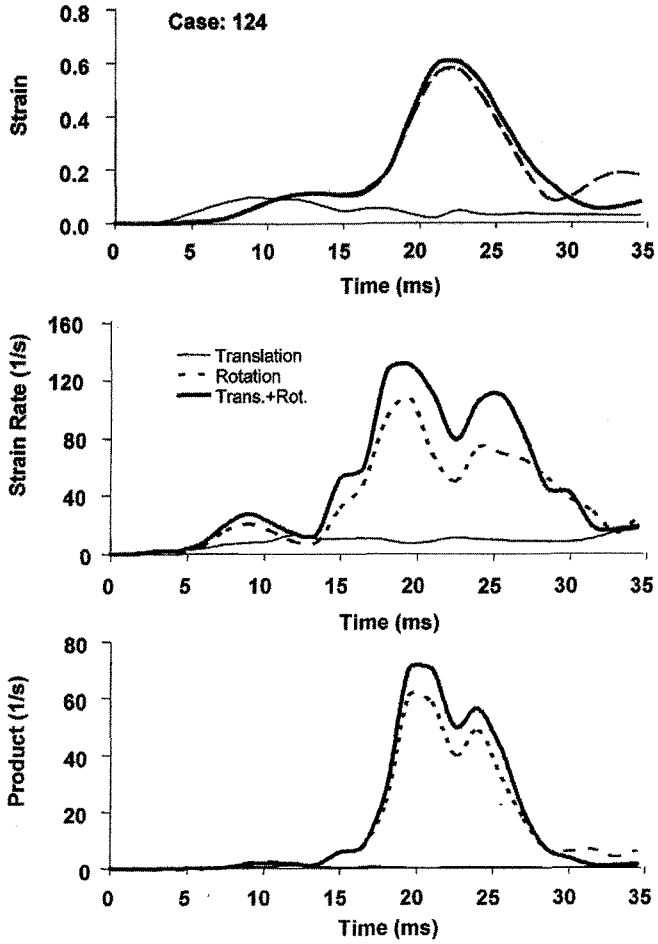

FIGURE 8. *Determination of brain responses for translational, rotational, and combined head acceleration for Case 124.*

Model material properties and geometry

There is another possible limitation with the FE model in assuring validity in the various material properties used to simulate the brain and model the detailed geometry of the brain. In total, 15 different tissue types were modeled and the properties taken from the literature. However, there is quite variability in the reported properties of various tissue, and much of the data involved animal studies and in vitro preparations after death of animals and humans. It is unclear how these properties actually mimic the in vivo properties of human brain. Some parametric analysis was conducted to explore the sensitivity of the model responses to variations in the material properties, but an exhaustive study has not been made. These aspects may be important to the specific levels of strain and strain-rate associated with injury but would not affect the relative comparison between the simulated cases. It is felt that the correlations with concussion and the signs and symptoms would *not* be greatly influenced and are reliable.

Skull response in helmet impacts

The cranium in the FE simulation is assumed to be rigid so that the measured accelerations from the Hybrid III dummy in the reconstruction can be a applied to the boundary as the input load to the model. However, the cranium has compliance and can deform under load. This is not considered a significant limitation because the helmet shell and padding distribute forces over a broad area of the cranium, reducing the concentration of stress that might locally bend cranium bone. In addition, the forces in these impacts would only cause small deformations of the cranium, which are not considered sufficient to alter the response of the brain.

Bridging veins

The helmet impacts caused sufficient brain motion that the FE simulation of the bridging veins was not stable in some cases. This required the bridging vein calculation to be turned off for several runs because of excessive deformation at the brain insertion points. Future simulation efforts need to modify this aspect for improved, stable performance.

Vestibular effects

It was not possible to study deformation in the vestibular region because the main anatomic structures of the auditory system are extracranial, and the cranium was assumed rigid. The vestibulocochlear nerve (VIIth cranial nerve) is a sensory nerve that is responsible for the sense of hearing and the sense of balance and body position. Clinically, problems with the vestibulocochlear nerve may result in deafness, dizziness, tinnitus, vertigo, and vomiting. Anatomically, the VIIIth cranial nerve travels through the internal auditory meatus and enters the brainstem at the junction of the pons and medulla lateral to the facial nerve. The auditory component of the nerve terminates in a sensory nucleus (cochlear nucleus), which is located at the junction of the pons and medulla. The strain and strain-rate response predicted by the model at these locations may alter the sensory nerve function and be responsible for the signs and symptoms sustained by the player if the magnitude is sufficiently high. On the basis of the model prediction, the level of the strain and strain-rate induced at the junction area was quite low as compared with those at midbrain region.

Concussion

The NFL research used a very broad and inclusive definition of concussion to capture as many possible cases as possible. From 1996 to 2001, only 7% of the concussion involved players out 7+ days, and even these most severely affected players showed a rapid recovery of function in a few days. Forty-nine percent of players were removed from play, which represents another subsample of the more severely injured players with concussion. Removal from play often involved memory, cognition, or somatic problems that lead team physicians to prohibit return to player on the day of injury.

Obviously, concussion is a term covering a broad spectrum of mild brain injury. It is interesting that the Hybrid III and mid-late strain and strain-rate in the midbrain correlated with concussion. However, it may be more interesting that no Hybrid III response correlated with any specific sign, symptom, or outcome of concussion. It is obvious that FE model and Hybrid III correlations depend on the definition of concussion and the particular signs and symptoms that make it up. When return to play was considered, no Hybrid III response and only two FE responses correlated with that subgroup. A larger collection of cases and FE modeling would be needed to shed more light on this aspect because these results are limited by the 28 NFL cases (22 concussions) reconstructed.

Furthermore, there may be a natural selection of athletes that make it to the NFL because players more prone to concussion may have been weeded out during high school and college play. Brain responses shown here may represent those of players who are most resistant to the damaging effects of neural deformation during head impact.

Research Methodology

This is the first time, to our knowledge, that local deformations of the brain have been used to correlate with the signs and symptoms of concussion. We believe there is a limitation in not being able to validate the research methods, which involved the integration of laboratory and analytical approaches. Each step builds upon the previous. The steps involved analysis of game video to determine impact velocity and helmet kinematics, laboratory reconstructions using instrumented Hybrid III dummies wearing helmets, and FE modeling of brain responses using the measured accelerations from the laboratory tests. Time will tell how useful and valid these observations prove to be in setting directions for the future design of helmets and protective systems. One obvious conclusion is that the more severe deformations of the brain occur after the primary impact acceleration. This offers an interesting challenge in considering safety equipment that reduces the mid-late responses and whether interventions applied at the end of the primary impact may influence the more significant signs and symptoms of concussion.

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TAB 1J

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David C. Viano, Dr. Med., Ph.D.
Mild Traumatic Brain
Injury Committee,
National Football League,
New York, New York
Sports Biomechanics Laboratory,
Bioengineering Center,
Wayne State University
Detroit, Michigan,
Pro Biomechanics LLC,
Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Mild Traumatic Brain
Injury Committee,
National Football League
New York, New York,
Department of Neurology,
Long Island Jewish Medical Center,
New Hyde Park, New York,
Department of Neurology,
Albert Einstein College of
Medicine,
Bronx, New York

Elliot J. Pellman, M.D.
Mild Traumatic Brain
Injury Committee,
National Football League
New York, New York,
ProHEALTH Care Associates, LLP,
Lake Success, New York

Cynthia A. Bir, Ph.D.
Sports Biomechanics Laboratory,
Bioengineering Center,
Wayne State University
Detroit, Michigan

Liying Zhang, Ph.D.
Sports Biomechanics Laboratory,
Bioengineering Center,
Wayne State University
Detroit, Michigan

Donald C. Sherman, M.S.
Sports Biomechanics Laboratory,
Bioengineering Center,
Wayne State University
Detroit, Michigan

Marilyn A. Boitano, M.D.
Safety and Equipment Committee,
USA Boxing,
US Marine Corp,
United States Naval Reserves,
Trauma Division,
Hamilton Health Sciences,
McMaster University
Hamilton, Ontario, Canada

Reprint requests:
David C. Viano, Dr. Med., Ph.D.,
ProBiomechanics LLC
265 Warrington Rd.
Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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CONCUSSION IN PROFESSIONAL FOOTBALL: COMPARISON WITH BOXING HEAD IMPACTS—PART 10

OBJECTIVE: This study addresses impact biomechanics from boxing punches causing translational and rotational head acceleration. Olympic boxers threw four different punches at an instrumented Hybrid III dummy and responses were compared with laboratory-reconstructed NFL concussions.

METHODS: Eleven Olympic boxers weighing 51 to 130 kg (112–285 lb) delivered 78 blows to the head of the Hybrid III dummy, including hooks, uppercuts and straight punches to the forehead and jaw. Instrumentation included translational and rotational head acceleration and neck loads in the dummy. Biaxial acceleration was measured in the boxer's hand to determine punch force. High-speed video recorded each blow. Hybrid III head responses and finite element (FE) brain modeling were compared to similarly determined responses from reconstructed NFL concussions.

RESULTS: The hook produced the highest change in hand velocity (11.0 ± 3.4 m/s) and greatest punch force (4405 ± 2318 N) with average neck load of 855 ± 537 N. It caused head translational and rotational accelerations of 71.2 ± 32.2 g and 9306 ± 4485 r/s^2. These levels are consistent with those causing concussion in NFL impacts. However, the head injury criterion (HIC) for boxing punches was lower than for NFL concussions because of shorter duration acceleration. Boxers deliver punches with proportionately more rotational than translational acceleration than in football concussion. Boxing punches have a 65 mm effective radius from the head cg, which is almost double the 34 mm in football. A smaller radius in football prevents the helmets from sliding off each other in a tackle.

CONCLUSION: Olympic boxers deliver punches with high impact velocity but lower HIC and translational acceleration than in football impacts because of a lower effective punch mass. They cause proportionately more rotational acceleration than in football. Modeling shows that the greatest strain is in the midbrain late in the exposure, after the primary impact acceleration in boxing and football.

KEY WORDS: Boxing, Concussion, Impact biomechanics, Sport equipment testing, Sport injury

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Closed head injury is an occupational hazard of many sports, and specifically in boxing and football. Participants in both sports are at risk for sustaining concussions ("dinged," "knocked out," cerebral concussion, MTBI). Zazryn et al. (101) found 107 injuries to 427 professional boxing participants, 89.8% of the injuries were to the head, neck and face with 15.9% concussions. For amateur boxers, the incidence of concussion is 4.0% to 6.5% (19, 38, 98). The difference in concussion rates between professional and amateur boxing may be due to differences in safety gear, and there have been recommendations to analyze professional and amateur boxers' injury rates separately (41).

The clinical picture of more severe brain injury is different in football and boxing (60–64, 75, 88, 89). The pattern of brain injuries in boxing has been extensively studied (2, 6, 7, 12, 13, 15, 18, 20, 22, 25, 26, 29–31, 36, 37, 44, 47, 51, 67–69, 72–74, 82, 85, 98). The pattern of brain injury in professional football has been recently studied and reviewed (60–64).

The medical literature is clear on the difference in the acute phase. Boxers are much more likely to develop subdural hematomas and brain-injury deaths than professional football

players (40, 42, 66, 88, 89). Boxers are also prone to develop, over the long term, a characteristic pattern of chronic brain injury (chronic encephalopathy of boxers, pugilistic dementia, "punch-drunk") that has never been reported in professional American football players (12, 39, 47, 75, 87, 89).

The biomechanical forces affecting the brain of professional football players have been recently reported as part of the ongoing studies of the NFL (58, 59, 64, 93–95). This study of the biomechanical forces in boxing was undertaken to help explain the similarities and differences between the clinical overview of brain injuries in the two sports. A longer term goal of this research is to study the effectiveness of protective headgear and sport equipment, including helmets in football and headgear and gloves in boxing. An understanding of the biomechanical forces causing injury is the first step in addressing improved protection. Boxing gloves and headgear are currently required in amateur boxing to prevent head injury (98). The equipment may reduce some injuries, but it does not eliminate the risk of knockouts (101).

The effectiveness of boxing safety equipment has been addressed by Schmidt-Olsen et al. (81) in a three-year study of amateur boxers in Denmark. No decrease in injuries was found with an increase from 8- to 10-ounce gloves, unlimited hand-wrap and use of helmets for heavier boxers. The lack of other data on this topic leaves boxing officials, athletes and trainers uncertain as to what specific safety equipment is most effective and what areas of improved safety needed additional study.

An improved understanding of the mechanisms of brain injury and biomechanics of head responses in amateur boxing is needed to lay the foundation for improvements in the effectiveness of protective equipment in boxing. This study of boxing and the biomechanics of the punch involves several factors, including how much force is exerted during a punch type, how that force is transferred to the opponent's head, how the opponent's head responds to the punch and how the opponent's head responds to different punches.

Punch Forces in Boxing

The biomechanics of punches has been studied using surrogates simulating the opponent (3, 83, 99). Atha et al. (3) and Smith et al. (84) analyzed different surrogates that have been used, including a ballistic pendulum, a uniaxial strain gauge platform, instrumented punching bags, water-filled elastic bags and boxing dynamometer. A consideration when choosing a surrogate is its ability to mimic the human body in both shape and impact response.

Atha et al. (3) used a single boxer and an instrumented ballistic pendulum to evaluate a single straight punch. The professional boxer punched the surrogate with 4096 N, which the author estimated translated into 6320 N of force to a human head. This force produced peak acceleration for 53 g's on the 7 kg ballistic pendulum.

Joch et al. (34) placed 70 boxers into one of three categories, including 24 elite, 23 national and 23 intermediate boxers. The force of straight right punches was measured with a water-filled punching bag fit with a pressure transducer. The average maximum punch force was 3453 N, 3023 N and 2932 N, respectively. In addition to the lack of biofidelity, there was a need to stabilize the surrogate and de-gas the bag, which made testing cumbersome (84).

Smith et al. (84) developed a boxing dynamometer to measure punch force. Twenty-three boxers were sorted into elite, intermediate, and novice boxer categories. Boxers were instructed to punch the head region of a pear-shaped bag mounted to a wall. Boxers threw straight punches using both their lead and rear hands. Punches were thrown singularly and in two and three punch combinations. The elite boxers had a mean rear-hand punch force of 4800 N and a front-hand punch force of 2847 N. The intermediate boxers' rear and front hand punch forces were 3722 N and 2283 N, respectively, and the novice boxers' mean rear and front hand punch forces were 2381 N and 1604 N (84). The researchers developed a surrogate, but the faceless pear-shaped device lacked the human response provided by the neck.

Walilko et al. (97) recently studied the biomechanics of straight punches to the jaw causing translational and rotational head acceleration. This was a precursor study to the present investigation. Seven Olympic boxers from five weight classes delivered 18 straight punches to the compliant face of the Hybrid III dummy. Instrumentation included hand acceleration and pressure distribution on the jaw. The punch force averaged 3427 ± 811 N, hand velocity 9.14 ± 2.06 m/s and the effective punch mass 2.9 ± 2.0 kg. The jaw load was 876 ± 288 N. The peak translational acceleration was 58 ± 13 g, rotational acceleration 6343 ± 1789 r/s^2 and neck shear 994 ± 318 N. They found that boxers deliver straight punches with high impact velocity and energy transfer to head rotation. The severity of the punch increased with weight class primarily due to a greater effective mass of the punch.

Assessing Head Injury Risks

Following the methods of Pellman et al. (58), the risk of head injury was determined by the methods of Gadd (21) and Hodgson et al. (32) using a human surrogate that has biofidelity in its impact response. Biofidelity reflects the ability of the surrogate to simulate the essential biomechanical characteristics of the human impact response. The Hybrid III dummy used in this effort is currently the most advanced, validated biomechanical surrogate, particularly for the head and neck areas. Sensors placed in the surrogate collect biomechanical data that are related to risk of injury. Previous studies have developed criteria to estimate the risk of head injury from various impacts (24, 28, 55).

One of the earliest head injury criteria was based on research conducted at Wayne State University (46). By investigating the relationship between the level of acceleration and duration of the impact, the Wayne State Tolerance Curve (WSTC) was developed (27). Impacts to the head that were

lower in acceleration required a longer pulse duration to cause the same injury as those higher in acceleration.

From this initial research, Gadd (21) expanded the analysis by including other human tolerance data from Eiband (17) and plotted the effective or average acceleration versus duration of impact on a log-log scale. The result was a straight line that had a slope of –2.5. Based on this result, the Severity Index (SI) was developed relating head acceleration to risk of injury:

$$SI = \int^{T} a(t)^{2.5} dt$$

where a(t) is the resultant translational acceleration at the head center of gravity (cg) and T is the duration of the acceleration. SI depends on the time history of the resultant translational acceleration. From the existing data, an SI tolerance of 1000 was established.

Versace (90) presented a new method for determining a head impact injury that took the SI one step further by optimizing the formula over the duration of impact. The final result was the Head Injury Criterion (HIC):

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)]^{2.5}\}_{max}$$

where t_1 and t_2 are determined to give the maximum value to the HIC function and a(t) is the resultant translational acceleration of the head cg. In practice, a maximum limit of $T = t_2 - t_1 = 15$ milliseconds is used.

Risk of head injury is calculated from accelerations at the head cg in three orthogonal directions. The resultant acceleration is calculated from these measurements and is used to determine HIC. As summarized by Walilko et al. (97), the final value provided a maximum acceptable value. The US delegation to Working Group (WG) 6 provided an estimate of the percent of the adult population expected to experience a life-threatening brain injury (AIS 4) for various HIC levels due to frontal head impacts (70). The delegation's best estimate is that 16% of the adult population would experience a life-threatening brain injury at a HIC level of 1000. In a recent study of concussions in the NFL, Pellman et al. (58, 59) recommended a value below 250 to minimize the risk of Mild Traumatic Brain Injury (MTBI or concussion).

Holbourn (33) worked with gel models of the brain and showed that rotational acceleration was an important mechanism in head injury. Ommaya and Hirsch (55) scaled primate head injury data to humans and predicted that a level of head rotational acceleration in excess of 1,800 r/s^2 would have a 50% probability of cerebral concussion in man. Analysis of injuries produced in rhesus monkey experiments resulted in Gennarelli et al. (23) estimating a 16,000 r/s^2 rotational acceleration tolerance threshold in man.

In a recent survey of rotation head injury criteria, Ommaya et al. (54) found that the rotational acceleration of 4,500 r/s^2 was required to produce concussion in an adult and that severe diffuse axonal injuries (DAI) occurred at 18,000 r/s^2. The range is from scaling of animal impact data and indicates the difficulty in developing a precise injury-prediction criterion for rotational motion, since the shape and mass of the animal brains are different from human and scaling laws assume geometric similarity. This makes extrapolating animal data to humans difficult. Also, the low mass of the animal brain requires very high rotational accelerations to produce closed head injuries (55). The combination of these factors makes predicting injuries in humans difficult. Furthermore, Pellman et al. (58, 59) found concussion was related to translational acceleration of the head.

In an effort to understand the relationship between forces delivered in boxing and risk of head injury from linear and rotational accelerations, the biomechanics of four different types of boxing punches was studied. The punches included straight punches to the forehead and jaw, a hook and an uppercut. Olympic boxers threw punches at an instrumented Hybrid III headform with their dominant hand, except for the hook. Correlations were made between the biomechanics of the Hybrid III head responses for the boxing punch and helmet impacts in professional football with attention to concussion. This study shows the similarities and differences between the head impacts.

Methodology

Eleven Olympic boxers weighing 51 kg (112 lb) to 130 kg (285 lbs) were included in the study. They were tested while participating in the 2004 United States Boxing National Championships. The research received approval from the Wayne State University's Human Investigation Committee and each boxer read and signed an informed consent prior to testing. Since their involvement was voluntary, they could withdraw from the study at any time. A certified boxing trainer was present for the tests even though the risk of injury was minimal. The boxers were not compensated for their participation.

Each boxer was evaluated for four punches. After a boxer warmed-up, they were instructed to strike the instrumented Hybrid III head with their gloved fist two times with four different punches, straight punches to the forehead and jaw and a hook and uppercut. Based on the method of Walilko et al. (97), three of the four punches were delivered with the dominant hand, including a straight punch to the jaw, a straight punch to the forehead and an upper cut to the jaw. For the fourth punch, the boxers' non-dominant hand was instrumented and they were asked to deliver a hook to the temple. Impact location of the punch was determined by high-speed video.

Measurement of Effective Hand-Arm Mass

Height and weight of each boxer were measured and anthropometric data for the dominant hand was collected. Volume measurements were obtained by submerging the dominant fist up to the styloid process in a water bath (97). The displaced volume of water was measured. Boxers then submerged their fist and forearm up to the epicondyles of the humerus bone. The effective mass of the fist and forearm were

calculated from the anthropometric and volumetric measurements by estimating the density of the body and converting densities of the forearm and hand. The segmental forearm and hand densities were multiplied by the segmental volumes to determine mass. Density estimates (d) were made using $d = 0.6905 + 0.0297c$ with $c = h/w^{1/3}$, where h = height (inches), w = weight (lbs), conversion factor for hand density (1.08) and conversion factor for forearm density (1.06). The equations have been shown to be suitable for estimating segmental body masses (8, 10, 100).

Test Setup

The test methods follow those described by Walilko et al. (97). A Hybrid III dummy with a frangible face was used to represent the response of the jaw and realistically transfer acceleration to the head. For the tests, a cork insert was used to give facial compliance for the straight jaw punches. The straight blow to the forehead and hook to the temple were on regions of the Hybrid III with known biofidelity. The uppercut was to the jaw, which has less biofidelity.

The tests used the compliant face of the Hybrid III head (50). This design has an improved biomechanical response in the facial region over the standard molded Hybrid III and more accurately reproduces the force and acceleration of the head for impacts in the frontal, zygomatic, maxillary and mandibular regions. Other devices used either a stiff load-measuring face or deformable structures in regions other than the jaw (1, 52, 92). The head and neck of the dummy were attached to the upper torso, which was fixed to the table by the flexible lumbar joint. This gave realistic head and upper body motion.

For the straight punch to the forehead, hook and uppercut, the punches were directly to the Hybrid III head. The straight punch to the jaw loaded the compliant face. Headgear was not placed on the dummy. The upper torso was attached to a rigid table with a foam pad placed below the Hybrid III abdomen insert so that the dummy remained in an upright position after each punch. Scaffolding was used to adjust the height of each boxer to a minimum 175 cm (69″) to ensure the punches were in the horizontal plane.

The Hybrid III simulates a tensed neck so the head is normally upright. The segmented neck includes flexible polymer discs to simulate the flexion-extension and lateral bending responses. A cable inside tightens the assembly to give the right neck response in calibration testing and during head acceleration (77, 80). While the Hybrid III neck was utilized in the study, it is unknown how it represents the strength of a boxer's neck. Boxers undergo extensive training to develop the neck muscles necessary to resist punch forces from an opponent. However, Johnson et al. (35) demonstrated that neck muscle tension has little effect on the oscillation of the head under sinusoidal excitation from a shaker.

Instrumentation

Instrumentation was placed in the boxer's clenched hand. Two Endevco (San Juan Capistrano, CA) 7264-2k accelerometers were secured to a semicircular cylinder, which was wrapped with the boxer's hand to measure hand acceleration in a biaxial arrangement. Integration of acceleration gave the velocity change of the hand during the punch. The Hybrid III was equipped with the standard triaxial accelerometers (Endevco 7264-2k) at the head center of gravity (cg) and six more accelerometers in a "3-2-2-2 configuration" to determine rotational acceleration (56). Processing of the nine accelerations determined the complete three-dimensional motion of the head. Rotational accelerations were computed from linear accelerations in the head. The analysis is valid for accelerometers coincident with the origin of the system or coincident with one of the axes. Deviations from this were required in the Hybrid III head, and a correction for centripetal and Coriolis acceleration was made according to DiMasi (16). Data was collected at 10,000 Hz using the TDAS PRO (DTS, Inc.) data acquisition system (SoMat, Co, Urbana, IL) and post processed according to SAE J211-1 (78).

Video Film Analysis and Target Location

A target was placed on the glove to digitize its motion and calculate impact velocity. Additional targets were attached to the head of the Hybrid III to measure the overall kinematics of the dummy during impact. Images were captured with a Kodak HG2000 high-speed video camera. The camera recorded the event at 4500 images per second. Digitization of the data was completed using the Image Express for video recording and processed according to SAE J211-2 (79).

Data Collection Procedure

After an appropriate warm-up period, the boxer was asked to lightly punch the head of the instrumented dummy with their wrapped and gloved hand. If there was no pain or discomfort, they were asked to increase their punch strength until they reached a point where they were throwing "normal" punches. Once the boxer was comfortable throwing punches, they were asked to deliver four different punch types to the dummy. Each punch type was performed twice for a total of eight punches per boxer. The order of punch placement was varied randomly; however, all of the punches for a particular hand were completed before the alternate hand was tested.

Punch and Head Inertial Forces

Impact forces were determined by two methods. First, the hand acceleration was measured for each punch and multiplied by their effective punch mass, which was determined separately. This estimated the impact force for the punch. Second, the resultant head acceleration of the Hybrid III was multiplied by the head mass of 4.45 kg to estimate the inertial force on the head. The punch force includes the inertial force on the head and neck loads, so it was always higher than the inertial force. The severity of the impacts was further quantified using translational and rotational acceleration, head injury criterion (HIC), severity index (SI) and change in head velocity (ΔV).

Concussion Risks from Boxing Punches

Pellman et al. (58) determined concussion risks using the Logist function in the Statistical Analysis Package (SAS). This function relates the probability of concussion p(x) to a response parameter x based on a statistical fit to the sigmoidal function $p(x) = [1+\exp(\alpha-\beta x)]^{-1}$, where α and β are parameters fit to the NFL response experience from the laboratory reconstruction of game impacts. The risk of concussion was determined for Olympic boxer punches using the NFL risk functions based on all football players exposed to helmet collisions. The parameters for the Logist functions were α = 2.677 and β = 0.0111 for HIC, α = 4.678 and β = 0.0573 for translational acceleration and α = 5.231 and β = 0.000915 for rotational acceleration.

FE Modeling of Brain Responses

Head accelerations from the Hybrid III dummy were used as input to a finite element (FE) model of the boxer's brain. This analysis follows the approach reported by Viano et al. (95) in the study of brain responses in NFL concussions. The brain responses for three punches from the heaviest boxer were simulated and compared with the patterns of brain deformation determined with NFL concussions. Early and mid-late strain responses and brain displacement were determined to show timing and areas of greatest brain deformation from the punches.

Effective Impact Radius

During a punch, the head experiences translational and rotational acceleration from the impact force. The accelerations are coupled. The impact can be resolved into a force at the head cg and a moment. The moment is related to the punch force times a radius between the impact axis and head cg. The radius (r) of impact causing rotational acceleration can be approximated by a simplified 2D relationship: $r = \alpha I/F = (\alpha/a)(I/m)$, where the head mass (m) is 4.45 kg and moment of inertia (I) about a lateral axis through the head cg is 0.022 kg m^2 (45). The impact radius is proportional to the ratio of rotational to translational acceleration, where the constant of proportionality is the ratio of head moment of inertia to mass, or 0.0049 m^2. If the simplified analysis assumes the head and neck are acting together to resist the impact, the mass is 5.80 kg and the average moment of inertia is 0.035 kg m^2 (4). This gives a ratio of head-neck moment of inertia to mass of 0.0059 m^2 and the effective radius increases 20%.

RESULTS

Boxer Anthropometry and Effective Punch Mass

Table 1 shows anthropometric data on each boxer. It also gives the volume of the hand and forearm, which were used to determine the effective punch mass. The average weight of the 11 boxers was 76.5 ± 22.1 kg (167.7 ± 48.5 lb) and their height was 177.2 ± 9.2 cm (69.8 ± 3.6 inch). The hand mass increased with boxer weight and averaged 1.67 ± 0.28 kg.

Punch Force and Biomechanical Responses

Figure 1 shows an example of the punch kinematics for the four different blows. These are images from the high-speed video and show the progression of the punch to the head of the Hybrid III dummy. The left column shows the straight punch to the jaw, which initially causes flexion of the upper neck and extension of the lower cervical region (third image). This happens because the punch is below the head cg. The

TABLE 1. Anthropometry of the Olympic boxers[a]

Boxer #	Height (cm)	Weight (kg)	Hand volume (mL)	Forearm volume (mL)	Forearm circumference (cm)	Wrist circumference (cm)	Wrist width (cm)	Wrist thickness (cm)	Wrist to knuckle (cm)	Fist width (cm)	Fist thickness (cm)	Hand mass (kg)
2	165	50.9	320	1130	26.2	16.2	5.3	3.8	10.2	8.5	6.7	1.26
11	168	55.0	350	1270	26.2	16.1	5.6	3.7	10.7	8.3	6.4	1.41
13	168	57.7	350	1260	27.1	16.5	5.5	3.7	9.7	8.5	6.1	1.39
4	180	65.9	360	1300	27.5	17.0	6.0	4.0	8.0	8.5	6.5	1.45
9	173	70.9	470	1730	29.5	17.0	5.4	4.5	10.9	8.5	6.9	1.88
10	178	70.9	460	1660	27.8	16.5	5.7	4.2	12.2	8.9	7.0	1.83
14	175	74.5	490	1840	30.4	18.5	6.3	4.7	9.8	9.4	7.5	2.00
8	177	84.1	440	1540	30.0	18.0	5.7	4.4	10.2	9.1	6.8	1.66
7	188	87.3	540	1520	31.2	18.7	6.4	4.6	12.7	9.5	7.5	1.68
12	183	91.8	440	1590	30.8	18.5	6.1	4.6	11.8	9.2	6.7	1.72
6	196	129.5	470	2030	32.7	19.0	6.0	4.3	10.8	9.0	7.0	2.15
Average	177.2	76.2	426	1534	29.0	17.5	5.8	4.2	10.6	8.9	6.8	1.67
SD	9.2	22.1	70	276	2.2	1.1	0.4	0.4	1.3	0.4	0.4	0.28

[a] SD, standard deviation.

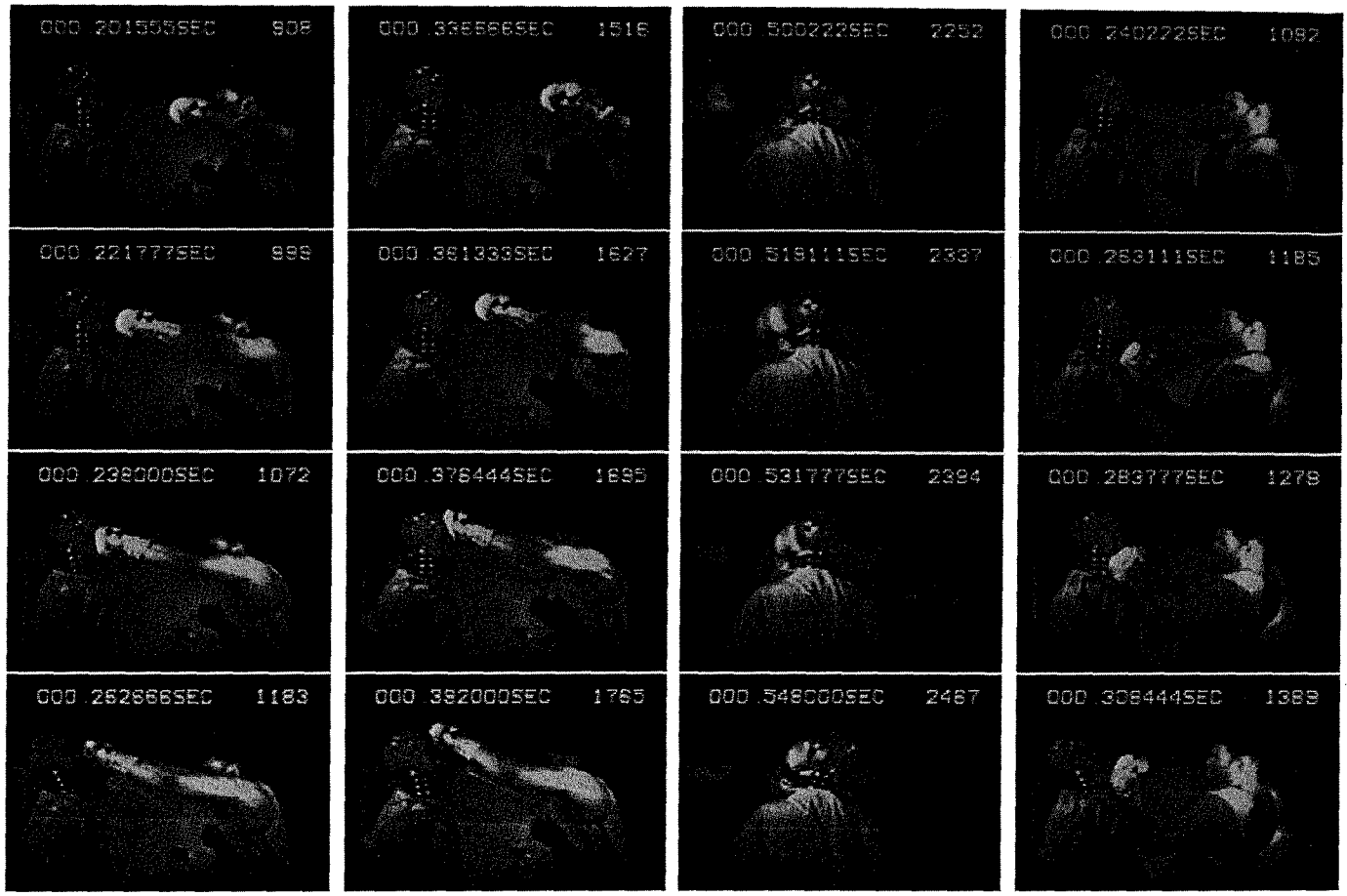

FIGURE 1. *Sequences from high-speed video of a boxer throwing a straight punch to the jaw* (left column) *and forehead* (second column), *a hook* (third column) *and an uppercut* (right column). *Time is shown in the top left of each image and the number in the top right is the frame count.*

hook produces lateral bending of the neck and twists the head about its vertical axis. The uppercut is shown in the last sequence at the right.

Table 2 summarizes the average biomechanical responses for the four different punch types. The full data is given in the *Appendix*. The hook produced the greatest impact force (4405 ± 2318 N) and inertial load on the head (3107 ± 1404) with an average neck load of 855 ± 537 N. It also had the highest change in hand velocity (11.0 ± 3.4 m/s). The lowest forces occurred with the uppercut to the jaw. Because of the range in boxer weight, the overlap in responses does not produce significant differences when the punches are grouped by type.

The hook also produced the largest head translational and rotational accelerations, reaching an average 71.2 ± 32.2 g and 9306 ± 4485 r/s^2, respectively. The straight punch to the jaw resulted in the largest neck loads (1088 ± 381 N) as the neck flexes as the jaw is driven rearward. This also resulted in the greatest bending moment of 81.9 ± 23.8 Nm about the y-axis (flexion-extension bending).

Even though the hand velocity change was in the range of 6.7 to 11.0 m/s on average, the change in velocity of the Hybrid III head was only 2.8 to 3.1 m/s on average for the various punches. This reflects the relatively low punch mass during the momentum exchange in the punch, although the change in hand velocity includes some effects of rebound from the punch.

Comparing Boxer Punches to NFL Concussions

Figure 2 shows the average and standard deviation in head inertial force (head mass times acceleration) for the four punches from the Olympic boxers and three conditions from NFL helmet impacts. The highest force is for NFL players experiencing concussion. Lower forces were measured in the NFL reconstructions for players struck without injury and for the striking players in helmet-to-helmet tackles. The force from the boxer's hook exceeded that of the non-injured NFL players and was within the statistical range for concussion. The jaw and forehead impact forces were lower and the uppercut produced the lowest inertial loads on the Hybrid III head.

Figure 3 compares the head biomechanical responses for the NFL game reconstructions and the boxer punches to the Hy-

TABLE 2. Summary punch forces and biomechanical responses of the Hybrid III[a]

Punch type	HIC15	SI	Res. head acc. g	Head delta V m/s	Res. rot. acc. r/s^2	Res. rot. vel. r/s	Res. neck load N	X-neck moment Nm	Y-neck moment Nm	Z-neck moment Nm	Res. hand acc. g	Hand delta V m/s	Peak force using head N	Punch force using hand N
Forehead														
Average	58	72	47.8	3.1	5452	22.9	664	−8.0	39.6	4.8	206.7	8.2	2085	3419
SD	44	53	20.1	0.7	2107	5.9	199	8.9	26.6	3.0	75.2	1.5	876	1381
Hook														
Average	79	99	71.2	3.1	9306	29.3	855	34.6	8.4	−14.8	263.4	11.0	3107	4405
SD	70	87	32.2	1.0	4485	6.2	537	21.1	6.6	8.1	105.4	3.4	1404	2318
Jaw														
Average	52	66	48.8	2.9	6896	20.7	1088	−21.1	81.9	10.3	145.8	9.2	2127	2349
SD	42	53	20.9	1.0	2848	5.6	381	22.2	23.8	6.3	57.1	1.7	910	962
Uppercut														
Average	17	23	24.1	2.8	3181	17.5	1486	−12.0	−21.0	6.5	92.9	6.7	1051	1546
SD	19	25	12.5	0.9	1343	5.0	910	5.2	6.1	3.4	39.9	1.5	547	857

[a] HIC15, head injury criterion for 15 ms duration; SI, severity index; Res, resultant; Acc, acceleration; Rot, rotation; SD, standard deviation; Vel, velocity.

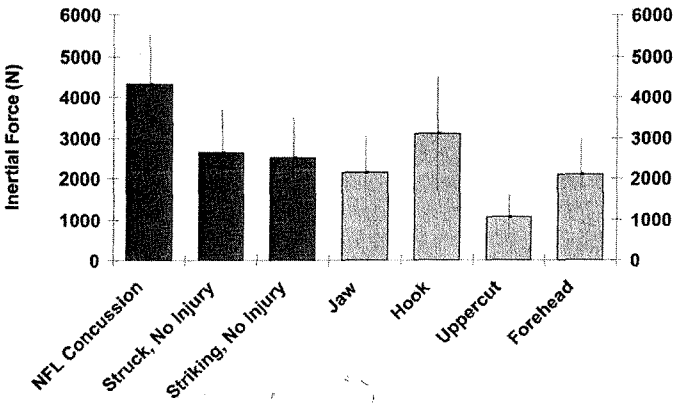

FIGURE 2. *Inertial force on the Hybrid III head for NFL game impacts and four different boxing punches.*

brid III dummy. The HIC was higher for NFL players experiencing concussion. The NFL players not injured or striking without injury had HICs slightly higher than the range of boxer punches. A similar trend can be seen in the translational acceleration, but the hook shows levels in the range for a risk of concussion based on the NFL experience. Interestingly, the boxers deliver more rotational acceleration to the Hybrid III dummy head for the hook and jaw punches than occurred in NFL concussions. However, the duration of impact is shorter for the boxing punch, so the rotational velocity of the head is similar to that in NFL concussion impacts with longer duration but lower rotational acceleration.

Figure 4 shows the peak rotational and translational accelerations for the NFL concussions and the boxers punches to the Hybrid III dummy. The closed circles represent concussed players in the NFL and the open symbols biomechanical data from players struck without injury or the striking players. The boxer data is included showing that jaw and hook punches have impacts in the range of the concussions experienced in the NFL. On average the boxers produce more rotational than translational acceleration with their punches.

Concussion Risks in Boxing and NFL Head Impacts

Table 3 shows the average and standard deviation in concussion risk using biomechanical responses from the Hybrid III dummy. Risk functions were used for concussion, where HIC had the strongest statistical correlation with NFL concussions (58). Based on HIC, the hook had a 13.8% ± 14.3% risk of concussion. On average, the predicted risks were in the range of 7 to 14% for the various punches. Similar concussion risks were predicted by peak translational acceleration, which was also a good predictor of concussion for the NFL impacts. The risk averaged 11 to 97% based on the peak rotational acceleration.

For comparison, the average and standard deviation in NFL concussion risk is shown from Pellman et al. (58, 59). Based on 28 players struck with 22 concussions, the average risk of concussion was 58.2% ± 33.0% based on HIC. While no striking player experienced concussions, the head responses were high enough to estimate a risk of 23.4 ± 20.7%, which obviously overstates the incidence based on the field experience. Nonetheless, the Logist risk functions were determined as a

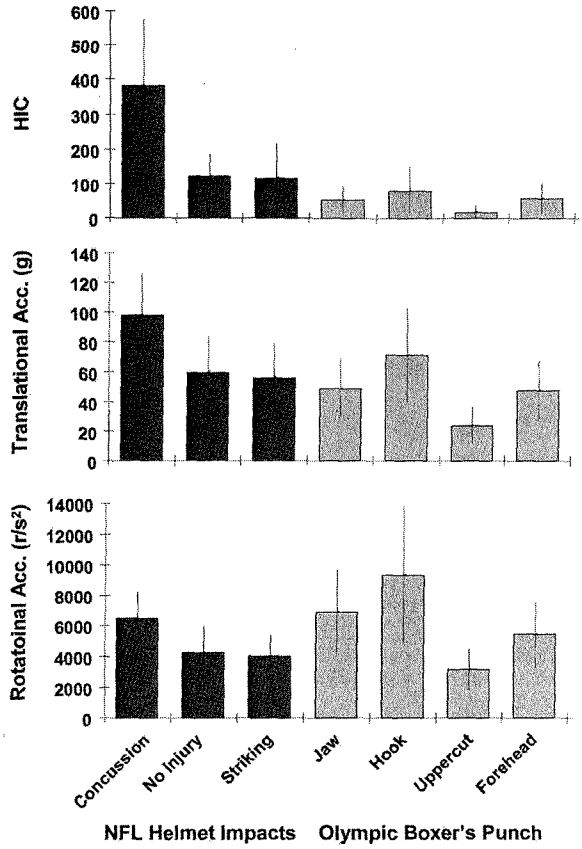

FIGURE 3. *HIC and peak translational and rotational acceleration for NFL game impacts and four different boxing punches.*

probability relationship between measured biomechanical responses and physician observed concussions.

FE Modeling of Brain Responses

Figure 5 shows the simulated brain responses for a hook from the heaviest Olympic boxer (boxer #6). This was a substantial blow and the strain "hot spots" show an early pattern in the temporal lobes with the highest strain occurring late in the midbrain. The maximum strain occurred about 10 milliseconds after the peak impact force.

Effective Impact Radius

Figure 6 shows groupings of peak accelerations for NFL concussions, striking players without concussion, struck players without injury and the four boxer punches. The lines are based on the relationship: $r = \alpha I/F = (\alpha/a)(I/m)$. The average radius between the punch axis and head cg was 57 to 71 mm with 57 mm for the forehead punch, 65 mm for the hook, 66 mm for the uppercut and 71 mm for the jaw punch. In contrast, NFL concussions occur at a higher translational acceleration and force on the head, but lower rotational acceleration. The average radius varied from 32 to 36 mm for the NFL

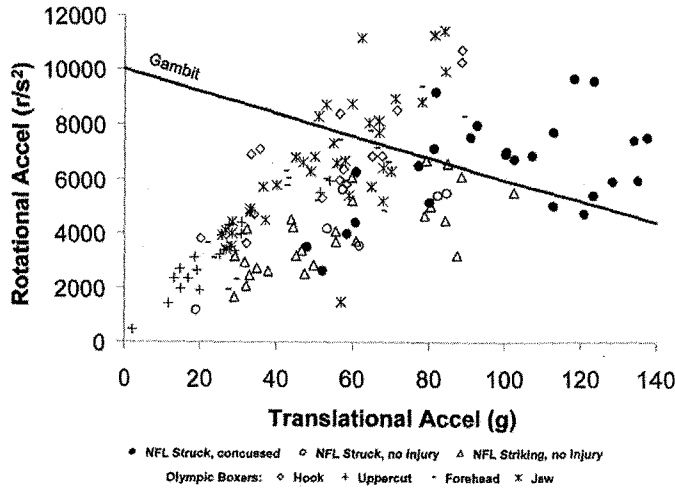

FIGURE 4. *Individual data points for translational and rotational acceleration of the Hybrid III head for NFL game impacts and four different boxing punches.*

players struck and injured (35 mm), those struck and not injured (36 mm) and the striking players (32 mm).

The ratio of head rotational to translational acceleration is higher in boxing than in NFL helmet impacts and results in a larger effective radius. The effective radius in football is 48% smaller than in boxing on average (34 mm v 65 mm). If the simplified analysis assumed the head and neck was acting together to resist the impact, the effective radius increases 20%.

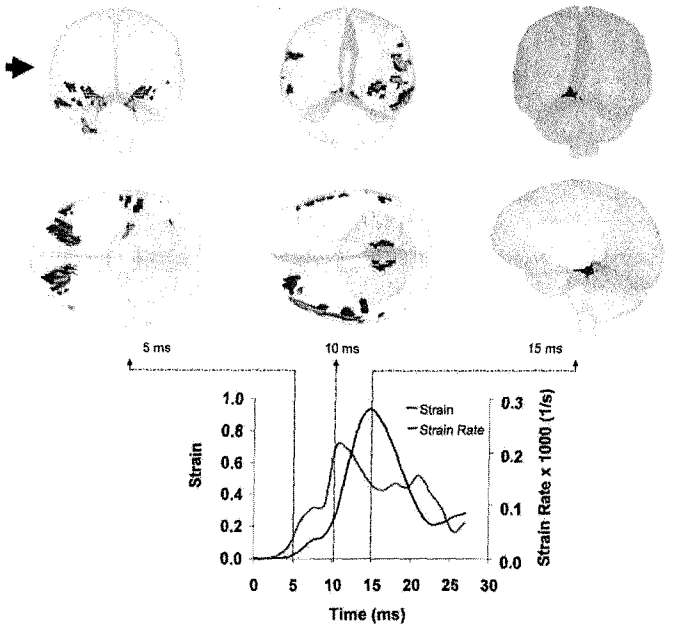

FIGURE 5. *Strain "hot spots" in the brain for a hook (Test 6h) showing the early, mid and late response pattern. The punch is to the right side of the Hybrid III head. The peak translational acceleration occurred at 5 milliseconds and the duration was about 8 milliseconds. The strain and strain-rate responses are shown for tissue in the brain.*

TABLE 3. Estimated risk of concussion for boxer punches based on the National Football League concussion experience[a]

		Risk of concussion		
		HIC	Trans acc. g	Rot. acc r/s^2
Olympic boxer punches	Forehead			
	Average	11.2%	11.9%	49.2%
	SD	5.7%	16.5%	32.5%
	Hook			
	Average	13.8%	35.9%	96.9%
	SD	14.3%	31.9%	29.3%
	Jaw			
	Average	10.5%	12.5%	78.0%
	Standard Deviation	5.2%	18.1%	35.2%
	Uppercut			
	Average	7.3%	3.1%	11.1%
	SD	1.8%	4.1%	15.9%
NFL Helmet Impacts	Struck players			
	Average	58.2%	57.7%	61.8%
	SD	33.0%	29.9%	28.8%
	Striking players			
	Average	23.4%	24.6%	26.0%
	SD	20.7%	22.7%	22.1%
	All players			
	Average	40.8%	41.2%	43.9%
	SD	32.5%	31.2%	31.2%

[a] HIC, head injury criterion; Trans acc, translational acceleration; Rot acc, rotational acceleration; SD, standard deviation.

DISCUSSION

This study compares the biomechanical forces affecting the head and brain from boxing punches with football helmeted impacts occurring in the NFL. There were three significant differences noted. The boxers' punches resulted in lower translational accelerations in the struck head, as compared to the football impacts. The boxers' punches applied a higher moment to the struck head than did the football impacts. This necessarily resulted in higher rotational accelerations in the head struck by the boxers' punch. Boxers therefore sustain brain injury by two mechanisms, translational and rotational accelerations of the brain, with a preponderance of the rotational component. Professional football players, on the other hand, sustained MTBI mostly by translational accelerations. These differences in the biomechanical forces may help explain the clinical differences between head injury in boxing and professional football.

Brain injury resulting in death is mostly due to acute subdural hematomas, which is much more common in boxing than in professional football. This difference can be explained by the differing effects on the bridging veins and other fragile brain structures resulting from the biomechanical forces. In addition, boxers are susceptible to a specific, unique pattern of chronic brain damage that has never been seen in American football players. The relative preponderance of rotational accelerations and the lower translational accelerations seen in boxing impacts may set the stage for boxers to sustain this type of long term brain injury.

This study also demonstrates one significant similarity between the head impacts in boxing and professional football. In both cases, the FE modeling indicates that the highest strain and strain-rate occur in the midbrain in the late time frame after the peak head acceleration. This suggests that high midbrain strain in the late timeframe might be a final common pathway in the development of concussion from a variety of head impact conditions.

Difference Between Boxing and NFL Brain Injuries

Acute head injuries in boxing can be more serious and devastating than those seen in professional football. There were over three hundred boxing deaths recorded in England due to brain injury before 1937 (66). Between 1945 and 1980, there were over 335 documented fatalities due to boxing (38–40, 66, 86–89). There have been many more deaths due to boxing in the years since 1980.

Approximately 75% of brain injury deaths from boxing are due to subdural hematoma (40, 66, 75, 89). Most of the other 25% are due to other traumatic intracerebral hemorrhages. Most acute subdural hematomas that account for the majority of boxing deaths are due to tearing and rupture of the bridging veins that run between the dura and the surface of the brain (40, 66, 89). These fragile structures are easily torn by head trauma. Studies in animals as well as clinical experience in humans indicate that almost all of the subdural hematomas are due to the effects of rotational forces stretching the bridging veins (89). One would expect that tearing of the bridging veins and subsequent subdural hematoma would be seen more commonly after head blows in a sport such as boxing with a preponderance of rotational acceleration as compared to professional football in which translational forces predominate.

The data from these tests on boxers shows proportionately higher rotational accelerations than translational acceleration in boxing. There is another possible explanation for tearing of the bridging veins in boxing. The present study indicates that

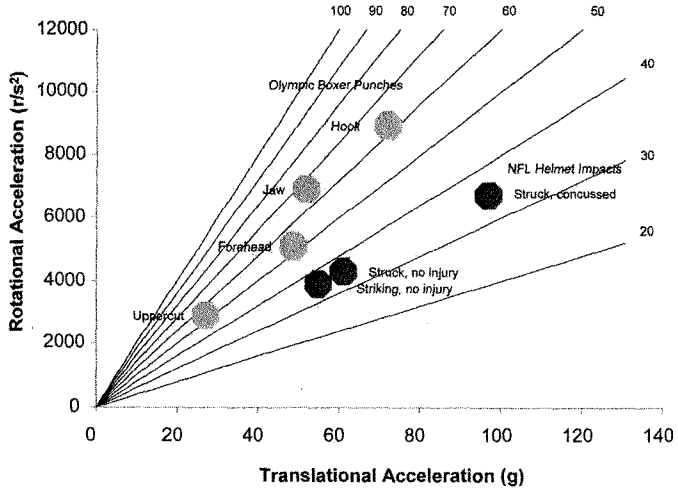

FIGURE 6. *Peak translational and rotational acceleration for Olympic boxing punches and NFL helmet impacts. The lines are a constant distance between the axis of impact force and head center of gravity (cg) using a simplified formula linking translational and rotational acceleration.*

punches directly to the jaw cause flexion of the head and neck resulting in stretching (high strains) of the bridging veins. In contrast, punches impacting the forehead cause extension of the head and neck with resultant compression (low strains) of the bridging veins.

Although there have been 433 fatalities due to head injury in football between 1945 and 1984 (337 cases due to subdural hematoma), almost all of these have occurred in high school or college players (66), where neck musculature and abilities are not as well developed as in the professional athlete. The authors are aware of one head injury related death in a professional Canadian football player and no brain injury related deaths in American professional football players since 1945. There was one case of subdural hematoma occurring in an American professional football player during those years but this was successfully removed without fatality.

The occurrence of subdural hematoma is consistent with the relatively larger translational accelerations and inertial force on the head in professional football than in boxing. This also raises the possibility that rotational head accelerations may be more predominant among college and high school players than in professional football players. It is also possible that the younger high school and college athletes' brains are more susceptible to tearing of the bridging veins than the more mature adult brains of the professional players. Although the absolute number of head injury related fatalities are similar in boxing and amateur levels of football, the incidence is in fact much higher in boxing because of the much larger number of annual participants in football at all levels compared to the number of annual participants in boxing.

Another clinical difference between the patterns of brain injury seen in boxing and football is the chronic brain damage seen in boxers but not in football players. A chronic encephalopathy of boxers has been well known to physicians since its initial description in 1928 (39, 47, 49, 75, 89). The clinical syndrome of pyramidal, extra pyramidal and cerebellar dysfunction combined with organic mental syndromes with cognitive and memory impairments and personality changes has been well documented (75, 89). The chronic encephalopathy may range from very mild to very severe.

During the past 25 years, studies have documented a pattern of cognitive and memory impairments in boxers ranging from subclinical to clinical dementia of varying degrees from chronic brain injury (5, 76). Studies have also defined a specific pattern of neuropathology which constitutes the chronic encephalopathy of boxers. This consists of abnormalities of the septum pellucidum, the cerebellum, the substanta nigra and the cerebral hemispheres (11, 89). The abnormalities of the septum pellucidum region include tears and fenestrations with resultant CSF leakage into the septum resulting in cavum septum pellucidum. The cerebellar findings consist of scarring and loss of Purkinje cells. In the substantia nigra there is depigmentation and loss of neurons. There is cerebral scarring as well as the presence of neurofibrillary tangles without senile plaques. There is enlargement of the third and lateral ventricles. This distinct neuropathological pattern is diagnostic of chronic encephalopathy of boxers. Studies have demonstrated that this chronic encephalopathy of boxers is related to the accumulation of multiple subconcussive blows to the brain over a long period of time. Its occurrence is directly related to the length of a boxer's career and the number of bouts fought, not to the number of times the boxer had been knocked out (5, 75, 76).

Roberts' (75) study suggested that chronic encephalopathy was more prevalent in heavier weight class boxers. This certainly would be consistent with the findings of the present study that heavier fighters delivered blows with higher forces than those generated by the lighter boxers. Critchley's (14) earlier paper, however, found that chronic encephalopathy occurred equally across all weight classes thus raising a cautionary note to interpreting the findings of this present study. This syndrome has never been reported in American football players. The present study may give some insight into why this syndrome is seen in boxers but not in other athletes such as professional football players. The present results indicate that boxers' brains sustain translational forces which are largely at or below the threshold for MTBI in NFL players and are likely to be subconcussive in nature when encountering an alert opponent rather than the stationary Hybrid III dummy. As a result, boxers are infrequently knocked out and thus able to continue fighting even though there may be substantial force in the punches landed.

In the course of training or a bout, a boxer may sustain large numbers of such blows in a repetitive manner. Repetitive head impacts with relatively high translational and rotational acceleration sustained over a period of time may cause tearing of structures such as the septum pellucidum resulting in cavum septum pellucidum, damage to the deep midline structures of the brain such as the substantia nigra and damage to the cerebellum and cerebral hemispheres. The nature of the forces impacting the boxer's brain may ultimately make him susceptible to

long term chronic brain damage (39, 71). The professional football player's head, on the other hand, is occasionally subject to much higher translational accelerations which are more likely to result in cerebral concussion and more likely to result in the player being removed from play or at least limited in his game activities for at least a short period of time. Professional football players do not sustain frequent repetitive blows to the brain on a regular basis. In addition, the relative preponderance of translational forces in professional football players may make them less susceptible to chronic injury than does the relative preponderance of rotational accelerations in boxing.

Difference Between the Biomechanics of Boxing and NFL Head Impacts

Figure 2 shows the inertial force on the head (58, 59) from reconstruction of helmet impacts in the NFL. They collected data from Hybrid III dummies simulating impacts recorded on game video. The laboratory reconstructions provided data on the biomechanical responses associated with recorded concussions in the players, and other severe impacts without injury. No concussion occurred in six struck players, and none of the striking players was injured. Interestingly, the boxers in this study generated impact forces that are similar to the non-concussion forces on the helmeted heads of NFL players.

The super-heavy weight boxer generated inertial forces of 3633 ± 1196 N and punch forces of 5352 ± 2775 N, which is at the average impact force causing concussion in NFL players. Since a majority of NFL players are injured by facemask or lateral impacts on the helmet, the loading direction is consistent with directions of the boxing punches, although straight anteroposterior impacts are an uncommon cause for NFL concussions. Boxer weight correlated with punch force, HIC and head acceleration in tests on Olympic boxers (97). While weight was a good predictor, punch force had a stronger correlation with HIC and translational acceleration. This means the effective mass of the boxer's punch is more important in increasing the severity of a blow.

There are probably two means by which boxers deliver concussive blows. The first means involves the boxer delivering enough translational acceleration. The hook involves a blow to the temple, which is just above the head cg. The forehead punch delivers force frontally above the head cg and the jaw impact applies force below the head cg. These impacts translate the head cg, and the forces can reach levels consistent with NFL concussions. The damaging mechanism is translational acceleration where the greater the mass of the punch, the greater the head HIC and translational acceleration. The second means involves rotational acceleration, which occurs with the impacts taking advantage of the offset from the head cg. During the punch, the axis of impact moves away from the head cg and introduces proportionately more rotational acceleration during the punch. The hook, for example, is always thrown with the elbow bent (43). This necessarily results in the axis of impact moving away from the cg after impact, thus imparting a significant amount of rotational acceleration to the opponent's head. The rotational nature of the hook has led sports writers to describe this punch as "whirling" and "tornadic" (43).

Increasing the effective mass behind a punch is the best way to increase the force of the punch. Increased acceleration of the punch may also result in increased force but it would seem difficult to increase the acceleration in the actual scenario of throwing a punch. Boxers and trainers intuitively realize that increasing the effective mass behind the punch increases its force. Champion boxer Jack Dempsey wrote that when he threw a straight left hand punch he began his attack "with a falling step forward toward the target with his left foot" (43). This "started the weight transfer which was the power source." He continued, "as you take your falling step forward, you shoot a half open left hand straight along the power line chin high" (43). The emphasis on keeping the arm and hand straight is consistent with the results from Walilko et al. (97) indicating that keeping the wrist straight and not flexed increases the force of the punch. By falling forward into the punch, Dempsey was increasing the effective mass behind the punch and thus increasing its force. Dempsey also knew that it was very difficult to deliver a knockout punch without throwing the entire weight of his body behind the punch. He wrote that it would be very difficult to throw a knockout punch by just turning the shoulders (43).

Presumably, other champion fighters and trainers have learned the same lessons that were known to Dempsey (96). The results of the present study lend scientific validity to this intuitive knowledge.

Figure 3 shows the HIC and translational and rotational accelerations from Pellman et al. (58, 59) on the biomechanics of NFL concussion. The boxers cause HIC and peak translational accelerations in the lower range of concussions in the NFL, but the head rotational accelerations can be higher from boxing punches. This means the boxers do not transfer as much energy in their punch as the collisions in the NFL. *Figure 4* shows more of the trend in the peak translational and rotational acceleration. In this case, there is a greater overlap in the peak rotational accelerations of the boxing impacts with concussion levels found for NFL players. However, the peak translational accelerations are lower than what occurs in the NFL. GAMBIT is a head injury criterion that limits the combination of rotational and translational acceleration (53). The tolerance line is shown.

With the use of football helmets, the striking player must line up his impacts closely with the head cg of the other player. This allows the impact to transfer energy. If the impact vector is at an angle, the blow will glance off due to the smooth plastic shell of the helmets. Players realize that they need to align their impact through the head cg to deliver a solid blow and maximize energy transfer to the other player. Severe helmet impacts that cause concussion involve high translational acceleration and change in head velocity (ΔV). NFL concussions involve an average impact velocity of 9.3 ± 1.9 m/s; and, the ΔV is 7.2 ± 1.8 m/s for the concussed player. Since the duration of impact is nominally 15 milliseconds, the peak head acceleration is high at 98 ± 28 g. In football, there is a strong correlation between translational and

rotational acceleration due to the impact alignment and subsequent head-helmet motion.

In boxing, the punch and glove conform more to the head of the opponent allowing punches to induce high rotational acceleration without high translational acceleration. The effective mass of the boxer's fist is 1.67 ± 0.28 kg, which is more than an order of magnitude lower than the 25 kg effective mass of the helmeted football player who strikes an opponent (64, 93). With concussion, the striking player lines up their head, neck and torso so their effective mass is considerable, and only the head and part of the opponent's neck resist the blow. In boxing, the most efficient energy transfer involves more rotational acceleration than translational acceleration.

The punch velocity of the boxers averaged 6.7 to 11.0 m/s for the four different punches. These levels are essentially similar to the impact speed in football concussions; but, the head ΔV after a punch was only 2.8 to 3.1 m/s on average, well below half that with NFL concussion. This reflects the much lower effective mass of the punch. Boxers cannot deliver high translational acceleration and ΔV to the opponent because of the low punch mass in comparison. Obviously, the effectiveness of punches is greater when the opponent is dazed and their neck muscles are more relaxed, since this lowers the effective head mass resisting the punch. Many of the well known boxing fatalities in the modern era have involved a fighter who has been dazed and stunned by multiple blows from his opponent. He is in a defenseless state with resultant marked diminution of muscle tone in the cervical paraspinal muscles (89). The resultant decrease in effective head mass results in increased translational and rotational accelerations of the head with every further punch. These increased accelerations are more likely to result in high strains to the brain, including the bridging veins, leading to severe injury or death.

While this discussion is theoretical, it is based on the mechanics of two different sports that can deliver neurocognitive effects to the brain in the form of memory, cognitive and functional problems. What is critical to the logic is that striking players in the NFL do not experience concussion even though they have head ΔV of 4.0 ± 1.2 m/s and the same impact velocity as the concussed player. Their ΔV and peak translational acceleration are above what the boxer can deliver in their punches. This indicates that rotational accelerations may be a factor in boxing knockouts, since translational effects are low.

Obviously, boxers can deliver rotational accelerations in and above the range where NFL players are concussed. However in both sports, we have not determined the root cause of concussion or knockouts. The underlying injury mechanism may depend on yet unknown combinations of translational and rotational acceleration, or factors of the brain response to skull accelerations associated with the impact. It is clear that head accelerations displace the skull in a complex kinematic, which loads the brain and causes internal stresses that deform neural tissues (45, 93). Brain and spinal cord tissues are sensitive to the rate and extent of strain in an impact (91). A sufficient combination of strain and strain rate can bruise the tissue and cause dysfunctions in neural function.

FE modeling of the brain response to a hook is shown in *Figure 5*. The responses are similar to the patterns of strain "hot spots" found in NFL concussions (95). Strain migrates from the temporal lobes early in the response to the midbrain, where the largest strain occurs late after the primary impact force of the punch. Since most of the NFL concussions involve lateral acceleration of the head, the hook has a similar direction of head loading. The boxers claim the hook is their knockout blow, but it is hard to deliver in a bout. Obviously, more analysis of the FE responses is needed to determine strategies for improving head protection in boxing and football; but, these results offer new insights into the biomechanical responses of the brain during head impact.

Effective Impact Radius

Figure 6 shows typical NFL concussion conditions and those of the striking and struck players who were not injured. The average radius was 34 mm for the three groups (range 32-36 mm). The average radius for the four boxer punches was 65 mm (range 57–71 mm). The lines are constant radii. The impact radius is proportional to the ratio of rotational to translational acceleration with the ratio of moment of inertia to mass a constant of proportionality. The ratio of head rotational to translational acceleration is larger in boxing than in NFL impacts. This simplified analysis seems to point to rotational acceleration as a possible factor in the severity of knockout punches, whereas the NFL concussion studies found the strongest correlation with translational acceleration and that the impacts had to be aligned with the head cg to prevent the helmets from sliding off. The duration of impact is shorter for boxing punch.

This analysis points to two different biomechanics of head injury in boxing. One associated with high translational acceleration and HIC, and another related to high rotational acceleration with low translational acceleration and HIC. Obviously, more study is needed to determine the underlying causes of boxing knockouts and football concussions. The simplified analysis assumed average values for a complex three dimension event. Also, the radius varies with time, the punch can be at varying orientation to the head cg and the flexibility of the neck is a factor. Nonetheless, the simplified analysis shows that a punch produces proportionately larger rotational than translational accelerations than in football by having a larger effective radius. The analysis also shows that rotational acceleration depends on the translational acceleration; they are inextricably coupled in an impact.

The data generated by this present study has been compared to the data from MTBI in the National Football League. The present study indicates that boxer punches cause relatively lower translational accelerations to the Hybrid III dummy head than the impacts seen in the NFL; whereas, the rotational accelerations are similar or higher to those seen in NFL concussions. This indicates that there may be a greater role for rotational acceleration in boxing blows compared to translational accelerations in helmet impacts in professional football. These results suggest that rotational acceleration of the head may be a factor in chronic

brain damage from repetitive impacts in boxing, since the pattern of brain injury is not seen in football players.

Comparing Concussion Risks in Boxing with NFL Head Impacts

Previous studies have primarily determined the force of a boxer's punch using a heavy bag or instrumented pendulum. The current study is a continuation of the effort started by Walilko et al. (97) to collect head impact responses using the Hybrid III dummy to determine head injury risks from boxer punches. The head-neck assembly of the Hybrid III closely represents the mass and compliance of the human head and neck. With this system, the risk of injury in terms of HIC, and translational and rotational acceleration can be explored from the momentum transfer of a punch to the head.

Because the dummy has humanlike impact responses and there are risk functions for concussion, the results of this study are relevant to determining the concussion risks from the punches of boxers. Although the knockout punch is a dramatic part of the sport, it is a relatively uncommon event. Studies have shown that knockouts occur in less than 5% of professional fights and probably in less than 2 to 3% of amateur level fights and in less than 1% of all amateur fights (40, 48).

The present results are consistent with this data. The translational accelerations resulting from these punches were at levels that one would expect to see relatively few clinical concussions, particularly for the uppercut and forehead impacts. The rotational accelerations are relatively higher and perhaps closer to the levels that one might see with clinical concussion. Most of the rotational accelerations were at or above levels expected to cause cerebral concussion in football. The uppercut is the exception. Therefore the laboratory results are consistent with a measurable but low incidence of knockout in the sport of boxing.

High rotational accelerations were found. The average peak rotational acceleration varied from a high of 9308 rad/s^2 for the hook to 3181 to 6898 rad/s^2 for the other punches. Ommaya et al. (54) indicated a rotational acceleration of approximately 4500 rad/s^2 were required to produce concussion. He also stated severe DAI occurs at 18,000 rad/s^2, and moderate and mild DAI occur at 15,500 r/s^2 and 12,500 r/s^2, respectively. Earlier studies by Pincemaille et al. (65) measured rotational accelerations of 13,600 rad/s^2 and rotational velocities of 48 rad/s during boxing. There were no cases of concussion in the tests. The current tests with the Hybrid III also show high rotational accelerations, however, the data reflect higher tolerances than specified in the literature otherwise knockouts would be much more common in boxing matches. Since the rotational acceleration tolerances are based on scaling of animal data, a question may be raised about the adequacy of the technique, which assumes similar geometry and equivalent material characteristics between animal and man.

Using risk functions for concussion in NFL players (58), the boxer impacts in this study show the highest risks with peak rotational acceleration (*Table 3*). The average risk was 11 to 97% for the four boxing punches. However, the NFL data showed the most significant correlation of concussion with HIC and peak head acceleration. Based on those biomechanical responses, the boxing data indicate a concussion risk of 7 to 14% for HIC and 3 to 36% for translational acceleration. Interestingly, the boxers do not generate enough head ΔV to reach much of a concussion risk based on the NFL data. HICs were low for the four punches with a risk of severe traumatic brain injury <2% (70). The average HICs were 17 to 79 also well below the proposed NFL concussion threshold of 250 (58, 59).

Limitations

Force delivered to the jaw loads the dummy in an area with responses that are similar to the human, so the reported translational acceleration and HIC reflect what occurs in boxing (50). However, it is uncertain how force from the uppercut to the jaw is related to risk of MTBI or if the Hybrid III dummy, in its current form, has sufficient similarity to the human response to measure risks with this punch. Development of human surrogate with an articulating jaw may improve the response of the head in this region and may show different biomechanical responses for the uppercut.

Before applying the results of this study to actual boxing experience in the ring, one must be aware of other limitations. The boxers that participated in this study were Olympic level amateur boxers. Although these boxers are at the higher echelon of amateur boxers, they most likely have not attained the proficiency or power levels of professional boxers. It is probable that more accomplished professional boxers can deliver punches with significantly higher force than those that were generated by these boxers.

It also must be pointed out that only four specific punches were evaluated in this study, including a straight punch to the jaw, uppercut to the jaw, hook to the temple and forehead punch. Other punches and uppercuts, hooks and crosses to other regions of the head may have different characteristics than the punches studied here; and, therefore may result in different translational and rotational accelerations in the opponent's brain than were seen in this study.

Furthermore, the data in this study was collected in a controlled laboratory setting, not during an actual boxing match. Factors such as fatigue, excitement and the effects of "adrenaline" on the boxers may significantly alter the forces of the punches delivered. Also, in an actual boxing match, the movements and defensive maneuvers of the opponent may affect the forces of the punches delivered to the opponent's head. In the present study, the punches were delivered to a stationary dummy head. The punch forces that were measured may be different than those that are seen in an actual boxing match.

APPENDIX: Measured and determined results from testing of Olympic boxers[a]

Boxer No. punch ID punch type	HIC15	SI	Res. head acc. g	Head delta V m/s	Res. rot. acc. r/s ^{2}	Res. rot. vol. r/s	Res. neck load N	X-neck moment Nm	Y-neck moment Nm	Z-neck moment Nm	Res. hand acc. g	Hand delta V m/s	Peak force using head N	Punch force using hand N
Forehead														
2f	15	18	23.1	2.5	3062	24.1	453	−18.3	29.5	3.0	87.4	8.4	1006	1082
11c	66	86	55.9	3.4	7448	21.6	825	−23.2	71.0	9.4	219.4	9.0	2438	3037
11f	87	106	66.0	3.4	7159	21.9	711	−10.8	41.5	7.9	298.7	9.4	2878	4134
13b	56	69	52.5	2.9	6017	10.6	789	−28.8	79.5	2.6	234.2	9.1	2286	3198
13e	82	101	67.8	3.4	4835	20.7	1105	−3.0	11.4	5.0	239.6	10.0	2955	3272
4c	9	13	21.3	2.1	3622	19.7	561	−2.3	60.1	10.2	165.1	8.2	930	2350
4f	16	23	29.2	2.5	3938	28.1	491	−1.9	40.3	7.0	159.7	7.2	1273	2273
9c	16	19	26.8	2.1	1928	16.5	376	−1.8	11.7	2.7	91.0	6.1	1171	1682
9f	18	22	28.9	2.3	2313	16.8	349	−1.3	13.7	3.2	90.7	6.2	1259	1676
10e	47	57	50.2	2.6	5202	20.3	701	−1.3	53.0	7.1	184.5	9.2	2187	3306
10h	53	61	42.7	3.1	6060	20.1	570	−9.6	38.6	4.6	221.4	7.0	1861	3968
14c	55	69	42.2	3.9	5764	28.6	683	−22.0	55.8	—	309.1	8.3	1842	6049
8c	15	19	28.2	2.1	2971	21.1	515	−0.7	7.0	0.6	87.4	5.0	1230	1422
7d	121	148	68.7	4.3	6663	25.0	920	−1.3	18.2	2.4	263.0	9.1	2994	4321
7g	123	145	64.2	4.1	7793	38.9	747	−10.4	53.3	3.4	273.2	9.1	2801	4488
12d	26	35	32.2	3.0	4747	23.0	556	−1.8	47.6	3.2	284.4	6.3	1405	4784
12g	31	42	42.1	2.8	6327	19.8	946	−9.1	98.9	10.0	290.4	9.4	1837	4886
6b	120	150	77.5	3.9	9421	30.1	585	−2.2	11.1	2.6	207.8	9.8	3379	4376
6e	145	181	88.9	3.9	8307	25.8	739	−1.9	10.4	1.7	221.3	9.9	3875	4661
Average	58	72	47.8	3.1	5452	22.9	664	−8.0	39.6	4.8	206.7	8.2	2085	3419
SD	44	53	20.1	0.7	2107	5.9	199	8.9	26.6	3.0	75.2	1.5	876	1381
Hook														
2a	57	70	57.6	3.2	6386	27.1	579	20.2	7.7	−5.7	181.6	10.7	2513	2247
2b	72	87	65.1	3.5	6862	27.8	780	19.6	5.6	−6.2	275.4	11.7	2839	3407
11g	111	142	92.2	3.7	12562	19.4	1081	54.3	13.5	−20.9	294.7	11.3	4022	4079
11h	77	96	71.4	3.2	8536	28.8	641	26.9	5.6	−13.6	310.1	10.6	3114	4292
13g	58	69	58.4	3.0	5804	24.3	448	13.4	3.2	—	293.0	9.3	2545	4001
13h	52	66	56.6	2.7	5967	22.1	446	8.6	5.7	−9.2	212.6	8.5	2470	2904
4a	74	90	67.7	3.3	6863	28.8	476	23.5	4.2	−4.6	313.7	14.0	2952	4466
4b	102	142	126.6	3.8	17487	36.0	1763	22.1	3.3	−6.2	281.5	19.8	5519	4007
9g	20	25	32.1	2.1	3635	21.5	491	18.8	1.9	−9.6	111.6	7.2	1400	2063
9h	55	64	52.0	2.8	5282	24.9	550	14.5	3.3	−6.0	272.2	10.7	2267	5030
10a	16	19	34.1	2.1	4693	27.9	494	30.7	3.6	−18.1	214.4	8.5	1488	3843
10b	11	14	33.2	1.6	6907	46.3	709	56.8	18.0	−27.1	55.4	5.9	1447	994
14a	87	116	102.3	3.5	12083	22.5	36	98.4	31.7	−27.8	270.9	12.6	4462	5302
14b	107	137	114.7	3.1	13337	37.0	1929	49.8	11.3	−21.4	443.0	12.4	5001	8671
8i	5	8	20.1	1.4	3804	36.6	315	21.2	6.2	−24.8	110.0	6.4	875	1791
8j	54	72	88.6	2.2	10756	35.2	840	27.8	8.4	−27.5	301.5	9.7	3862	4907
7a	96	118	92.9	3.6	19925	26.9	2167	43.6	7.9	−9.1	296.1	10.2	4052	4865
7b	95	116	80.4	3.5	12381	32.0	1304	62.3	9.5	−22.2	197.2	12.6	3508	3241
12a	19	26	35.6	2.5	7117	28.8	975	56.3	6.0	−13.7	200.2	9.2	1552	3368
12b	52	67	56.6	2.7	8396	32.0	652	34.6	11.1	−18.6	231.0	9.0	2470	3887
6g	187	221	88.6	4.6	10301	29.4	950	29.6	4.7	−8.3	472.4	14.4	3863	9950
6h	330	415	140.6	5.7	15626	28.4	1178	27.4	13.2	−9.0	455.7	17.7	6131	9597
Average	79	99	71.2	3.1	9305	29.3	856	34.6	8.4	−14.8	263.4	11.0	3107	4405
SD	70	87	32.2	1.0	4485	6.2	537	21.1	6.6	8.1	105.4	3.4	1404	2318

APPENDIX: Continued

Boxer No. punch ID punch type	HIC15	SI	Res. head acc. g	Head delta V m/s	Res. rot. acc. r/s ^{2}	Res. rot. vol. r/s	Res. neck load N	X-neck moment Nm	Y-neck moment Nm	Z-neck moment Nm	Res. hand acc. g	Hand delta V m/s	Peak force using head N	Punch force using hand N
Jaw														
2e	11	14	28.3	1.5	4422	19.6	842.3	−22.8	46.9	12.5	82.0	10.2	1232	1015
2h	20	25	36.5	2.2	5706	11.0	1020.4	−12.3	68.3	10.0	134.1	8.7	1590	1659
11d	49	64	53.0	3.0	8706	26.0	966.2	−21.1	97.4	16.9	190.1	10.3	2311	2632
13c	66	82	64.3	3.5	8097	16.8	954.4	−5.4	95.5	10.0	227.9	9.7	2804	3113
13f	66	81	55.8	3.1	6605	14.4	812.1	−23.2	80.7	—	250.8	10.3	2435	3424
4e	15	29	32.9	2.1	4775	19.0	899.2	−14.8	60.1	12.6	100.9	7.7	1433	1436
4h	15	21	28.3	2.3	3979	22.4	768.0	−9.4	73.5	15.4	124.4	8.0	1236	1771
9a	12	16	25.8	2.2	3946	18.6	706.7	−24.5	63.4	5.8	54.7	7.2	1124	1010
9e	15	21	26.8	2.5	4108	19.8	823.8	−15.2	77.0	6.3	68.2	6.5	1168	1260
10d	76	95	62.2	3.9	11215	25.8	2101.2	−104.3	50.7	26.8	113.1	9.2	2715	2028
10g	10	13	26.6	2.0	3399	13.5	664.0	−6.5	58.6	2.5	95.2	7.1	1159	1707
8a	77	100	55.0	4.4	7334	23.9	1350.5	−25.7	107.6	8.1	95.4	7.0	2399	1553
7e	120	153	84.1	3.8	11492	28.8	1711.6	−10.8	117.8	11.5	203.3	11.3	3666	3340
7h	80	103	59.7	4.3	8738	28.2	1453.6	−34.2	116.6	15.3	175.0	10.6	2603	2876
12c	12	16	28.1	1.7	3511	11.3	906.8	−12.1	59.1	9.9	174.2	8.6	1224	2930
12f	32	40	45.0	2.0	6805	24.5	1082.0	−18.7	96.7	8.8	172.4	12.0	1961	2901
6a	130	168	81.5	4.1	11321	25.5	1427.7	−3.8	120.1	2.4	168.2	10.9	3553	3543
6d	121	149	84.2	3.4	9969	24.6	1101.4	−14.1	83.7	0.8	194.2	11.0	3671	4090
Average	52	66	48.8	2.9	6896	20.7	1088.4	−21.1	81.9	10.3	145.8	9.2	2127	2349
SD	42	53	20.9	1.0	2848	5.6	381.4	22.2	23.8	6.3	57.1	1.7	910	962
Uppercut														
2d	19	24	28.8	2.8	3338	24.4	1496.0	−18.5	−18.5	10.3	96.1	8.9	1257	1189
2g	18	24	25.5	3.6	3946	22.0	1729.2	−8.2	−25.6	5.7	96.9	8.8	1113	1198
11b	6	0	2.1	2.8	472	5.5	73.6	−1.9	−4.0	1.6	33.1	4.6	92	458
11e	9	14	19.1	3.5	2632	15.8	1424.9	−9.3	−27.6	5.7	55.8	5.7	831	773
13a	12	18	26.1	2.8	3381	20.3	1865.2	−12.2	−27.8	4.8	87.6	6.7	1140	1197
13d	20	27	30.7	2.9	4387	10.0	2175.7	−20.3	−15.1	5.8	98.4	8.5	1339	1343
4d	10	14	18.6	3.1	3125	22.6	1291.9	−10.1	−28.3	8.3	48.2	6.7	809	686
4g	3	4	14.9	3.6	1973	14.8	819.0	−6.1	−22.5	4.9	70.2	5.9	651	999
9b	4	5	13.1	1.9	2359	15.8	919.6	−13.0	−22.7	4.4	63.1	4.4	572	1167
9d	8	9	19.9	1.7	1919	12.7	909.9	−8.7	−16.0	6.8	107.3	6.4	867	1983
10c	4	7	14.7	2.1	2707	15.4	815.6	−15.8	−26.3	11.8	67.0	5.1	640	1201
10f	12	17	25.1	2.4	3218	18.2	1383.6	−13.9	−25.6	7.3	112.3	5.5	1096	2012
8b	23	30	27.7	3.4	3478	21.6	1499.2	−14.1	−21.6	12.6	74.7	6.1	1209	1216
7c	5	7	16.6	1.7	2354	19.8	850.9	−5.3	−16.1	2.1	74.4	7.5	725	1223
7f	19	27	30.6	3.3	3964	17.2	2044.5	−19.3	−22.2	7.7	78.5	8.0	1337	1291
12e	2	3	11.6	1.1	1431	15.0	419.8	−11.1	−17.0	8.3	95.1	4.2	507	1600
12h	14	20	27.5	3.0	4288	15.7	1347.0	−20.3	−15.1	11.8	192.9	7.2	1200	3246
6c	73	94	53.9	4.2	5950	20.2	3658.7	−11.3	−21.9	2.9	156.2	8.0	2350	3270
6f	64	85	51.3	4.2	5515	25.5	3500.2	−9.3	−25.6	1.4	158.1	8.5	2238	3330
Average	17	23	24.1	2.8	3181	17.5	1485.6	−12.0	−21.0	6.5	92.9	6.7	1051	1546
SD	19	25	12.5	0.9	1343	5.0	910.5	5.2	6.1	3.4	39.9	1.5	547	857

[a] HIC15, head injury criterion for 15 ms duration; SI, severity index; g, gravity; Res acc, resultant acceleration; Res rot acc, resultant rotational acceleration; N, newton; SD, standard deviation.

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COMMENTS

Viano et al. continue their fascinating biomechanical studies of sports-related traumatic brain injury by investigating boxing punches and comparing them to helmet impacts in professional football. Rotational acceleration of the head is proportionately greater after boxing punches, whereas translational acceleration tends to predominate in football impacts. As the authors suggest, this fundamental biomechanical difference may account for the higher incidence of both acute injuries, such as acute subdural hematoma, and chronic brain injury in boxing.

Alex B. Valadka
Houston, Texas

Viano et al. carefully studied the biomechanics of boxing injuries using finite element modeling, and compared the physical characteristics of the impact to the head from an amateur boxer's punch with the physical characteristics of head injuries sustained in professional football. As they point out in the discussion section, the clinical applicability of their results is limited. For example, the forces applied to the head as measured in their laboratory would be expected to cause a much higher incidence of concussion than is actually seen on the playing field. It also is likely that professional boxers sustain significantly greater translational and rotational displacement of the head than do amateur boxers. Because of the more severe rotational forces sustained by boxers compared with football players, and the much higher incidence of lethal brain injuries in boxing, I completely agree with the statement by Viano et al. that "boxers are susceptible to a specific, unique pattern of chronic brain damage that has never been seen in American football players." Their study underscores the need for studies that clearly define the histopathology of chronic traumatic encephalopathy in professional football players, and cautions against generalizing the autopsy findings in boxers to other sports.

Donald W. Marion
Boston, Massachusetts

In sports, such as American football, ice hockey, boxing, rugby, lacrosse, and martial arts, in which contact is an integral part of the game, athletes develop hitting strategies to gain an advantage. In many cases, this strategy involves head impacts. This article provides insight into what, until now, has largely been ignored. Depending on the situation, athletes are able to use different strategies to create concussions. Even though it is obvious that a number of factors contribute to head injuries in sport, helmet designs have, for the most part, focused on preventing subarachnoid bleeds. This has primarily been accomplished by managing a 40 to 80 joule impact under 275 gs of linear acceleration. Recently, cerebral concussion has become more of a concern, whereas subarachnoid bleeds have become relatively rare. Although the threshold for protecting the brain against concussive injuries is not well established, it has been estimated at approximately 4500 r/s^2 for angular acceleration and 75 to 80 gs of linear acceleration. Football helmets are primarily designed to manage impacts to prevent subarachnoid bleeds and although they do provide some protection against concussion, they are not designed to do so.

The objective of these and many other sports is to manage physical interactions in such a way as to enrich the competitive environment of the activity without undue risk of injury to participants. This thin line is managed by game rules, coaching and training programs, protective equipment, and player integrity. A breakdown in any one of these elements increases the risk of player injury. Coaches, players, and game officials all have incentives to allow increased hitting. Examples of this are numerous and include coaches having to gain an advantage over other teams, especially when the team is under pressure to win, players fighting to win a spot on the team, and league officials under pressure to increase fan attendance. Professional sports are extremely vulnerable to the pressures of producing a product that is attractive to a broad audience.

This article demonstrates the need for research to better understand the mechanism underlying head injuries in specific activities. There is little doubt athletes become extremely skilled in gaining an advantage and, if necessary, can use any number of strategies to take advantage of their opponents. Just how athletes receive concussions in sport is still not well understood. The recent National Football League study identified linear acceleration as the primary mechanism for concussions in professional football. This included a very limited data set and should be interpreted accordingly. More extensive research directed at understanding the mechanism of head injuries in individual sports would be extremely valuable in managing head injuries in all sports.

T. Blaine Hoshizaki
Ottawa, Ontario, Canada

In a further contribution from the National Football League (NFL) Mild Traumatic Brain Injury Committee, the authors sought to evaluate the potential relationships between the impact biomechanics of boxing and football. To do so, 11 Olympic-level boxers (weight range 112–285 lbs) delivered 78 blows (inclusive of four types including hooks, uppercuts, and jabs) to the head of a Hybrid III Dummy. Recorded variables included translational and rotational head acceleration and neck load. Punch force was measured using a biaxial acceleraton model. The most significant change in hand velocity was in the hook (11.0 m/s) in addition to the greatest punch force (4405 ± 2318 N). The authors found that boxing punches have proportionately more rotational than translational acceleration than what is observed in football cuncussions.

This is useful information and indirectly addressed a number of questions. It is interesting to note that the NFL Mild Traumatic Brain Injury Committee has yet to identify an example of dementia pugilistica (or variants thereof) in retired NFL players. Obviously, this is quite the contrary in professional level boxing. Exposure obviously differs significantly both with regard to the quantity of head impacts and the likely associated force in the absence of headgear.

The importance of head-gear will need to be defined at the amateur level before any consideration at the professional level. Even then, the more spurious elements involved in the management and promotion of the sport will likely be slow in adopting any suggested guidelines regarding headgear use at the professional level. The perception is the possible impact on the fan base and the associated income. Initial work continues at the Olympic Training Facility in Colorado to evaluate responses in the presence and absence of headgear in boxing. Evaluation of impact at varying distance will also be required.

The consideration of headgear is important at the professional level. A lack of understanding of the components of injury that are most significantly related to central nervous system injury and short- or long-term disability remain unknown. Conceptually, the understanding of the force associated with "Heavy Hands," or lack thereof, in athletes susceptible to concussion (Glass Jaw) will also need to be determined. An important first step would be to evaluate the potential myth of the big punch. Obviously, the only chance an individual may have in a fight in which, based upon points, the individual has no chance of winning the fight. The damage sustained by individuals who are losing in these competitions tends to be consistent. We would suspect that the suggestion of an early end to a 12-round championship fight given an inability to win based on the 10 point scale would be as well received as the use of head gear. A final caveat. There are a number of things about the sport of professional boxing that we cannot and should not change.

'I am going to kill him good. I do not care about styles. Styles do not mean anything. I have seen every style in the world. I have been in this game for 18 years. I have been a world champ for 12. He cannot even touch that. I am going to be the WBA heavyweight champ of the world. I am ready to go no matter what. I do not care: you want to play rough, I will play rough. Boy, you have no idea. I tell you, I will hurt you.'

Min Park
James Toney
John Arthur
Michale L. Levy
San Diego, California

This study by Viano et al. investigates the biomechanical forces involved in boxing punches in comparison with football impacts. Using a Hybrid III test dummy and accelerometers, 11 Olympic boxers were studied delivering hooks, uppercuts, and straight punches to the instrumented Hybrid III test mannequin. Using finite element analysis, the kinetic energy and head responses were compared with similarly determined impacts from video documented and reconstructed concussions in NFL players. Any study that analyzes the biomechanical forces imparted during contact sports is a welcome addition to the literature. As the authors note, closed head injury is an occupational hazard of many sports, particularly in boxing and football, in which neurocognitive effects to the brain can occur in the domains of memory, cognitive, and functional injury. The ability to carefully visualize and measure the various aspects of the contact athlete's torso and head response to high velocity impacts increases our understanding of the issues involved.

This study also adds insight and helps clarify the fact that translational and rotational accelerations are major components of the human body's response in contact sports. In my opinion, the effective radius of the impact and the relative contribution of the supporting musculature in the thorax, head and neck are most important factors.

Our computerized video analysis of various types of boxing matches has led to the conclusion that there is little difference between the fights which are considered "classic" versus those that result in a lethal outcome. Unquestionably, the most significant contributing factor in lethal boxing outcomes is the absorption of multiple blows to the cranium, especially during long-duration fights and with a fighter who is progressively impaired, resulting in relaxation of the supporting neck musculature and lowers the effective head mass against the punch.

Surprisingly, the dramatic single-punch knockout rarely results in a lethal outcome or significant brain injury in a boxer. The chronic effect seen in boxers is related to an accumulation of blows to the cranium, accentuated with years in the sport and in heavier weight classifications. the authors note the limitations of their study, which was performed in a controlled laboratory setting with Olympic-level amateur boxers, looking at a limited number of punches, and those delivered to a stationary test mannequin. This is an important study with comparative data between two contact sports which are associated with potential for brain injury.

Julian E. Bailes
Morgantown, West Virginia

TAB 1K

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CONCUSSION IN PROFESSIONAL FOOTBALL: HELMET TESTING TO ASSESS IMPACT PERFORMANCE—PART 11

Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee,
National Football League,
New York, New York,
ProHEALTH Care Associates, LLP,
Lake Success, New York

David C. Viano, Dr. med., Ph.D.
Mild Traumatic Brain Injury Committee,
National Football League,
New York, New York,
ProBiomechanics, LLC,
Bloomfield Hills, Michigan

Chris Withnall, B.S.
Biokinetics and Associates, Ltd.,
Ottawa, Ontario, Canada

Nick Shewchenko, B.S.
Biokinetics and Associates, Ltd.,
Ottawa, Ontario, Canada

Cynthia A. Bir, Ph.D.
Sport Biomechanics Laboratory,
Bioengineering Center,
Wayne State University,
Detroit, Michigan

P. David Halstead
National Operating Committee on
Standards for Athletic Equipment,
Overland Park, Kansas,
University of Tennessee,
College of Engineering,
Sports Biomechanics,
Impact Research Lab,
Knoxville, Tennessee

Reprint requests:
David C. Viano, Dr. med., Ph.D.,
ProBiomechanics LLC,
265 Warrington Road,
Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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OBJECTIVE: National Football League (NFL) concussions occur at an impact velocity of 9.3 ± 1.9 m/s (20.8 ± 4.2 mph) oblique on the facemask, side, and back of the helmet. There is a need for new testing to evaluate helmet performance for impacts causing concussion. This study provides background on new testing methods that form a basis for supplemental National Operating Committee on Standards for Athletic Equipment (NOCSAE) helmet standards.

METHODS: First, pendulum impacts were used to simulate 7.4 and 9.3 m/s impacts causing concussion in NFL players. An instrumented Hybrid III head was helmeted and supported on the neck, which was fixed to a sliding table for frontal and lateral impacts. Second, a linear pneumatic impactor was used to evaluate helmets at 9.3 m/s and an elite impact condition at 11.2 m/s. The upper torso of the Hybrid III dummy was used. It allowed interactions with shoulder pads and other equipment. The severity of the head responses was measured by a severity index, translational and rotational acceleration, and other biomechanical responses. High-speed videos of the helmet kinematics were also recorded. The tests were evaluated for their similarity to conditions causing NFL concussions. Finally, a new linear impactor was developed for use by NOCSAE.

RESULTS: The pendulum test closely simulated the conditions causing concussion in NFL players. Newer helmet designs and padding reduced the risk of concussion in 7.4 and 9.3 m/s impacts oblique on the facemask and lateral on the helmet shell. The linear impactor provided a broader speed range for helmet testing and more interactions with safety equipment. NOCSAE has prepared a draft supplemental standard for the 7.4 and 9.3 m/s impacts using a newly designed pneumatic impactor. No helmet designs currently address the elite impact condition at 11.2 m/s, as padding bottoms out and head responses dramatically increase.

CONCLUSIONS: The proposed NOCSAE standard is the first to address helmet performance in reducing concussion risks in football. Helmet performance has improved with thicker padding and fuller coverage by the shell. However, there remains a challenge for innovative designs that reduce risks in the 11.2 m/s elite impact condition.

KEY WORDS: Concussion, Helmets, Protective headgear, Recreation and sport, Sports equipment

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Football helmets used in the National Football League (NFL) are currently certified to National Operating Committee on Standards for Athletic Equipment (NOCSAE) standards, including NOCSAE 001-04m05, 002-98m03, 004-96m04a, 002-96m03, and 021-98m05 (*www.nocsae.org*) (19–24). These impact tests are designed to provide confidence that protective helmets are effective in reducing life-threatening head injuries. The test methodologies use a specialized headform that is mounted to a drop fixture (13). Tests are conducted at 36, 48, and 60 inches onto a flat padded surface, and impacts are directed at six locations on the helmet. A triaxial accelerometer is located at the center of gravity (cg) of the headform. Data from the accelerometer is used to calculate the shock attenuating properties of the helmet based on the head severity index (SI), where the risk of

serious head injury is determined from the SI (5). Additional standards are maintained by the American Society for the Testing of Materials (ASTM), including the tests ASTM F429, F717, and F1446 (*www.astm.org*) (1–3).

However, none of the current standards address helmet performance in reducing the risk for concussion. Pellman et al. (28, 29) and Viano and Pellman (34) analyzed reconstructions of on-field collisions causing concussion in the NFL to fill the gap in understanding injury biomechanics. These studies demonstrated that concussions occur in impacts low on the front, side, and back of the helmet with a combination of linear and rotational head acceleration. Because these impacts were not addressed by the current helmet standards, a new test and specification was needed to reduce the risk for concussion by improvements in helmet design and performance. This article addresses the background and methods evaluated and developed to assess concussion risks with football helmets. The research converged on a recommendation for a new methodology for standardized testing of helmets to reduce the risk for concussion.

Background on Biokinetics' Studies

In September 1997, Biokinetics (*www.biokinetics.com*) began research sponsored by the NFL to investigate the biomechanics of concussion in professional football players (26). Under the auspices and direction of the NFL Committee on Mild Traumatic Brain Injury (MTBI), the aim was "to gain a better understanding of how and why concussion occurs, and to devise better means to measure concussion, such that ultimately improved head protection can be offered." Game video of player collisions involving concussion was analyzed to determine the speed and direction of impact. By the end of 1999, 10 cases of helmet-to-helmet contact had been analyzed, reconstructed with Hybrid III dummies, and head accelerations were correlated with the player injury.

In March 1998, Biokinetics began working with Riddell, Inc. (Elyria, OH) to design a new football helmet. It was intended that Biokinetics would incorporate new engineering strategies based on its analysis of on-field head impacts. At the time, there was a widespread sentiment that rotational acceleration was an important contributor to concussion, and football helmet test standards did not address this head response. There was speculation about what contributed to head rotational acceleration in a football collision. For example, it was supposed that a glancing blow, directed tangential to the helmet, would introduce helmet rotation that would cause head rotation. Perhaps a smoother faired exterior would be beneficial or a looser chinstrap, a lighter facemask, or a slip layer between the helmet shell and the padding may lower rotational accelerations.

Biokinetics developed a pendulum with an impactor face designed to simulate helmet-to-helmet contacts between two players. It was used to investigate what helmet parameters would serve to promote or mitigate head rotational acceleration. A Hybrid III 50th percentile male head and neck were mounted on an adjustable platform and positioned in the path of a weighted pendulum that swung down and struck the helmet (4). An array of nine accelerometers was used to determine translational and rotational accelerations experienced by the impacted and impacting players. The face of the pendulum simulated the curvature and friction of another helmet because many impacts observed in the NFL video involved helmet-to-helmet collisions (29). The weight of the hammer was chosen as five times the weight of the head to account for torso mass of the striking player.

A substantial test matrix was initiated that involved many parameters of helmet fit, impact location, chinstrap fixation, surface friction, and facemask size and weight. Hundreds of tests were conducted on a host of impact sites, and the linear and rotational accelerations for each set-up were analyzed to determine what parameters increased or decreased rotational acceleration. Throughout the work, the pendulum test and the Hybrid III head-neck assembly proved to be robust and repeatable. However, football helmets performed poorly for lateral or oblique impacts to the jaw-pad region. This was an area not tested by the NOCSAE football standards. It was also discovered that rotational accelerations were consistently worse in hits directed squarely at the helmet where there was minimal glancing or deflecting action of the helmets. This was in contrast to earlier speculation that large rotational accelerations would be induced by hits that generated large helmet rotations.

A draft helmet test method was prepared for Riddell describing the test machine and how to use it. Riddell was later granted a United States patent for this testing methodology (35). There were also suggestions for helmet test speeds, impact sites, and pass/fail responses. Although these values were a draft, they were selected when the first injury predictor functions were emerging from the NFL study. The test methodology was presented as a supplement, not a replacement, of the existing NOCSAE football helmet standards.

The original pendulum and draft helmet test methodology were established at roughly the same time as the first biomechanical response and injury data were emerging on NFL player collisions. The aim was to simulate the characteristics of helmet-to-helmet collisions on the field. The draft methodology included impacts to eight sites to the facemask, side, and rear of the helmet. Helmet manufacturers Riddell and Schutt purchased the pendulum test machine and have used it in the development of new football helmets. There was some communication with NOCSAE about adopting the pendulum test to supplement existing helmet test protocols. Riddell and Schutt have referenced the pendulum test in the same breath as the NFL research program, and media reports have reinforced the supposed link. The first new helmet to emerge as a result of the NFL-sponsored research was Riddell's "Revolution," unveiled in early 2002.

The MTBI Committee investigated pendulum test data from Riddell on this new product and compared it to Biokinetics' full dummy reconstruction data. Biokinetics also identified the contact points of helmet impacts for 31 reconstructed game

collisions to compare with the suggested test sites of the earlier Riddell pendulum test protocol. The results of the review found that the impact pulse of the pendulum did not closely match the dummy reconstructions and that the impact sites in the protocol did not represent those observed in game plays involving concussion. For these reasons, and the fact that the original test protocol needed refinement before gaining more acceptance, the NFL Committee on MTBI directed work to improve the pendulum test. The pendulum test underwent refinement to its design to deliver impacts to a helmeted Hybrid III head that simulated full-scale dummy-to-dummy reconstructions of game collisions. While the original goal of the test apparatus was to study the effects of helmet design parameters on rotational head acceleration, it was used to guide new helmet designs.

Background on Wayne State University's Research

Since the 1960s, Wayne State University (WSU) (*http://ttb.eng.wayne.edu/sports/labintro.html*) has conducted research addressing the protection provided by football helmets and the mechanisms for brain injury from head impact (6–10, 12). The studies led to the development of a human-like headform and test methods for the evaluation of football helmets, which were adopted by NOCSAE in the 1970s to reduce the risks for serious head injury (11, 13, 15). The work with NOCSAE also involved biomechanical studies on neck loads causing fracture-dislocation during head down impacts in tackling-dummy practice and game collisions (14, 16). This led to a neck compression tolerance criterion of 4000 N to prevent serious axial-compression injury of the cervical spine.

WSU was contracted by the NFL to conduct linear impactor tests at higher speeds than possible at Biokinetics and to use the full upper torso of the Hybrid III dummy. This led to an integrated torso test (ITT) that allowed helmet interactions with neck collars, shoulder pads, and other protective equipment. It used the Hybrid III head, neck, shoulders, and upper torso in the helmet impact. A nine-accelerometer array was used in the Hybrid III head to assess translational and rotational acceleration. This procedure was used to replicate on-field impacts causing concussion. The pendulum and linear impactor tests were considered for a supplement to the current NOCSAE helmet standards. The evaluation and refinement in the testing methods and helmet response data formed a basis for a recommendation for a new NOCSAE standard. The overall goal of the research was to develop an appropriate test method to evaluate helmets for concussion risks in football.

NFL Concussion Impact Responses

Based on the reconstruction of NFL concussions from Pellman et al. (28, 29), the impact velocity (V) of helmet-to-helmet and helmet-to-ground impacts was V = 9.3 ± 1.9 m/s (20.8 ± 4.2 mph). *Figure 1* shows the risk of concussion based on peak translational head acceleration and SI from the reconstruction of NFL game impacts. There are two curves for concussion

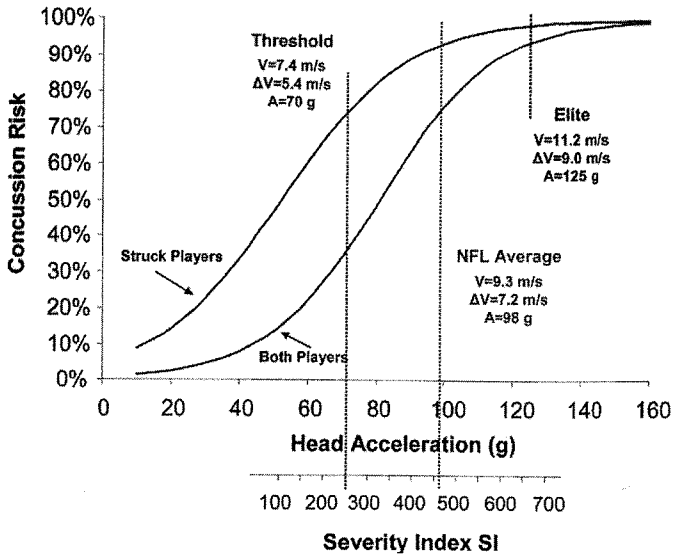

FIGURE 1. *Concussion risk for peak head acceleration and SI measured in the struck player's head in NFL reconstruction by Pellman et al. (28, 29). The lower two impact speeds represent the threshold and average condition for concussion in the NFL and are the basis for a draft supplemental NOCSAE standard. The highest impact speed represents an elite condition that is too severe for current helmets, but occurs in NFL games where concussed players are often out more than seven days from play.*

risk. One involves only the struck players and the other combines data on the striking and struck players in the collisions. The difference reflects the larger number of uninjured players when the striking players were included. No striking player experienced concussion, and the collision biomechanics have been recently described (34).

Figure 1 also identifies three test conditions representing the NFL concussion experience. One is the average impact velocity of V = 9.3 m/s (20.8 mph) and biomechanical responses causing concussion in NFL players. Another is the average impact velocity plus one standard deviation in biomechanical responses. This higher level represents the most severe impacts seen in the NFL with a velocity of V = 11.2 m/s (25.0 mph). It defines an elite impact condition that is representative of collisions causing the most severe concussion with players often out seven or more days from practice and play (27). Most of these collisions involve open field tackles during kickoffs or punt returns where both players are running towards each other at full speed, or during open field tackles during passing or running plays.

The average impact velocity minus one standard deviation is V = 7.4 m/s (16.5 mph) represents a threshold condition for NFL concussion. The plot shows the concussion risk for peak translational acceleration and SI for the three collision speeds. The lowest two impacts involved an SI = 220 and SI = 475, respectively. Since acceleration and duration are factors in determining SI, the change in head velocity (ΔV) of concussed players is equally important to the NFL experience. The aver-

FIGURE 2. *Kinematic sequence with tests at five impact sites on the helmet: 1* (left column) *through 5* (right column). *The top image is -10 ms before impact and the images under are at 0, 10, and 20 ms. The struck player is the one at the left. He is free-falling into contact with the striking player at the right. The images are rotated 90^0 counterclockwise to give a horizontal perspective to the collision.*

age $\Delta V = 7.2 \pm 1.8$ m/s (16.1 ± 4.0 mph) giving a threshold $\Delta V = 5.4$ m/s and elite at $\Delta V = 9.0$ m/s.

The testing presented in this paper involved helmet-to-helmet impacts simulating the threshold, average, and elite impact conditions with NFL concussion. When ground impacts are removed from the NFL concussion data, the average impact velocity increases. This is because ground impacts involve lower impact velocity, but they have substantial rebound of the head. The change in head velocity (ΔV) is higher for ground impacts than other helmet impacts. The average velocity for helmet-to-helmet impacts was $V = 9.6 \pm 1.7$ m/s (21.5 ± 3.8 mph). Based on this subset of NFL concussions, the threshold impact velocity was $V = 7.9$ m/s (17.7 mph) and the elite condition $V = 11.3$ m/s (25.3 mph). SI for the lowest two impacts was 200 and 450, respectively.

MATERIALS AND METHODS

The testing conducted in this study involved evaluations of the original and modified Biokinetics' pendulum and WSU's ITT. While the Biokinetics' pendulum impactor provided a useful simulation of NFL game concussions, it was necessary to determine the most appropriate test speeds and corresponding pass-fail criteria that would establish new performance goals for helmet standards to prevent concussion. The pendulum test provided a good starting point to evaluate headgear at energy levels consistent with the average condition for NFL concussions; however, it was necessary to investigate how state-of-the-art football helmets performed under these conditions before assigning pass/fail criteria. These tests determined that there was a need for refinement in the methodology based on new information from NFL concussion studies, helmet manufacturers' testing, and these evaluations.

Biokinetics Pendulum Impactor

Impact Conditions

The pendulum response was tuned to the initial reconstruction tests of NFL concussions. However, as the number of reconstructions increased and different test speeds and impact conditions emerged, it became cumbersome to match them with the existing pendulum protocol and setup. It was necessary to have helmet-to-helmet data at common test speeds for a standard. Furthermore, it was necessary to condense the broad cluster of game impact sites into a smaller number of standardized, but representative, areas of the helmet.

Helmet-to-helmet impacts are often glancing blows owing to the smooth rounded shape of the helmet. This was recognized in both the design of the pendulum arm and initial selection of impact sites. Impacts to a wide variety of sites on helmets revealed that hits high on the helmet, or nearly tangential to the outer profile, tended to be very minor in terms of head accelerations. Most hits received by a player are to the front half of the helmet. Hits to the rear involved impacts with the ground. In general, the pendulum delivered more substantial blows to sites in the lower regions of the helmet because it was difficult for the pendulum to clear the helmet. This caused more energy to be delivered to the Hybrid III head.

The NFL MTBI research revealed that many concussions occurred with lateral impacts to the jaw pad. This region is currently not required to offer impact protection in NOCSAE or ASTM standards. While football helmets offer padding in this region, it was primarily for comfort and fit, not impact attenuation. Eight impact sites were defined by a horizontal line and the centerline of the pendulum face that are measured while the pendulum is at rest at the base of its swing (35). Impacts were delivered to the rear, rear 45°, side, front 45°, and front of the helmet with additional impacts to the jaw pad region. These sites were later refined as information about the NFL concussions was being developed by Pellman et al. (28, 29).

Figure 2 shows the five test sites that were chosen to represent the 22 concussions from NFL helmet-to-helmet game collisions available at the time. For these cases, 9.5 m/s represented the average collision speed between players. Full-scale dummy reconstructions were staged with hits targeted on these five sites at 9.5 m/s as well as 6.7 m/s, which represented one-half the impact energy. The main reason for the second test was to observe performance differences at lower energy that would show helmet performance at the threshold for concussion. *Figure 1* can be used to see that a 6.7 m/s impact speed is below the average minus 1 standard deviation in NFL concussions and represents a below-threshold condition for injury. Tests were performed using a Riddell VSR 4 helmet, size L, which was the same model used in the NFL game reconstructions (28, 29).

Each of the test sites yielded a particular peak head acceleration and ΔV for the Hybrid III head at the threshold and average test speed. When these tests were duplicated using the pendulum, design parameters were adjusted so each of the same sites matched the peak acceleration and ΔV. A pendulum speed of 6.9 m/s nominally represented the average condition for NFL concussion from helmet-to-helmet impacts. A pendulum speed of 5.3 m/s represented approximately half the energy level, or a threshold concussion condition. These impact speeds were lower due to the larger pendulum mass.

The Original Biokinetics Pendulum Impact Test

A helmeted Hybrid III head with neck was impacted by the weighted pendulum (17, 18). The head was instrumented to measure translational and rotational acceleration and calculate SI and HIC. The head was machined to provide mounting locations for nine accelerometers in the 3-2-2-2 configuration (from *www.radenton.com*, reference drawing B-3623). The accelerometers were piezoresistive and capable of withstanding a 2000 G (Endevco model 7264A-2000, Endevco Corp., San Juan Capistrano, CA). The data was analyzed using the method of Padgaonkar et al. (25) to determine head rotational acceleration. The head was covered with the standard head skin. A six-axis upper neck load cell was installed for the testing. The internal neck cable was tightened to a torque of 1.1 Nm (10 in lb) before testing. The data acquisition system recorded transducer data at 10 kHz for 100 ms and preconditioned by CFC 1000 Hz filters after SAE J211-1 (32). A velocity trap trigger was used.

The impacting face was steel with a diameter of 152 mm. It was a domed spherical surface of radius 127 mm $\pm$ 5 mm. Under a lateral force of 350 N $\pm$ 25 N, the impact face deflected 100 mm $\pm$ 10 mm. The effective pendulum mass was 19.0 kg $\pm$ 0.5 kg measured at the centerline of the impacting face with the arm held horizontal. A minimum 75% of the effective mass was concentrated in the pendulum head. An arresting mechanism was used to catch the pendulum as it continued past the helmeted head.

The base supporting the Hybrid III head-neck was capable of adjusting in five degrees of freedom, including fore-aft (x), lateral (y), and up-down (z) translation, as well as fore-aft (y) and axial (z) rotation of the neck base. The adjustment provisions were lockable and remained fixed throughout the testing. The adjustment ranges for the base allowed the Hybrid III head to be impacted from any direction by the center of the pendulum face. The base was located relative to the pendulum so that the initial impact with the helmet was within 10 cm of the base of the pendulum's swing.

The Hybrid III vinyl skin has considerable friction against the vinyl lining of most football helmets. This makes donning the helmets difficult, and it introduced an unrealistic head-helmet interface. Nylon stockings on the head reduced the friction and provided a reasonable simulation of skin-to-helmet friction. Two layers of nylon stockings were stretched over the dummy head before installation of helmet, allowing relative motion between the helmet and head. The stockings remained tightly fit to the head skin.

The pendulum tests were conducted with a facemask installed using the manufacturer's recommended attachment method. The helmet test methodology included eight targets on the front, back, sides, and facemask. However, these points were selected before the availability of NFL research and were not necessarily relevant when compared with the later analysis of NFL collisions causing concussion (28). This led to refinements in the impact locations.

Grouping of NFL Game Impacts Causing Concussion

In Pellman et al. (28), the reconstructed game collisions were grouped in four types (A–D), depending on the site of the helmet contact. With eyes forward being the 0° reference, four quadrants from front to back of the helmet were defined with left side impacts reflected to the right side. The categories included A: 0–45°, B: 45–90°, C: 90–135° and D: 135–180°. Elevation of the impact site on the helmet was defined by seven horizontal sectors, four above the head center of gravity (cg) and three below. Head responses were further evaluated, including the determination of the average and standard deviation in velocity and displacement. Head accelerations were also used to determine SI and HIC criteria for head injury (30) and the peak rotational accelerations and velocities were also reported (28, 29).

Pendulum Design Modifications, Basic Goals and Strategies

Figure 3 shows a schematic of the pendulum impactor test, including several of the updated features. The objective of the test was to simulate helmet-to-helmet collisions in NFL games with a pendulum. It was necessary to investigate several features of the striking player that needed to be integrated into the pendulum system. The initial evaluation of the pendulum was based on comparison of peak head acceleration and ΔV with NFL game reconstructions. A design target of 10% difference in responses was established as a realistic goal.

Slider System

The original pendulum test had the dummy head and neck mounted rigidly to an adjustable base. When the pendulum

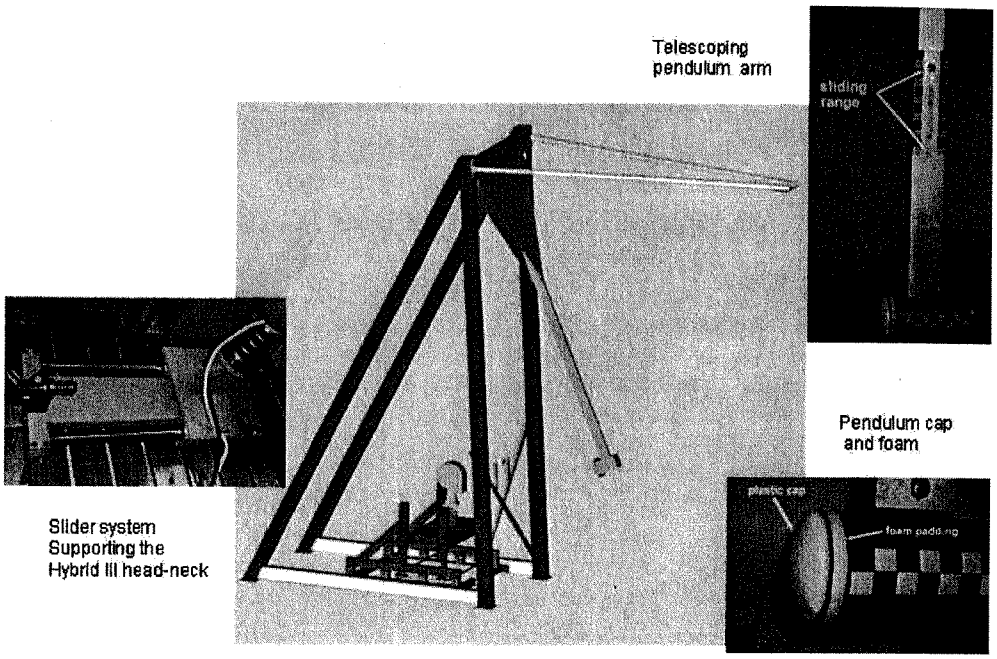

FIGURE 3. *Schematic and photographs of the pendulum impactor used at Biokinetics to simulate the helmet-to-helmet collisions resulting in concussion in the NFL. The slider system is shown in the photograph on the* left, *and the telescoping pendulum and impactor cap are shown in the photographs on the* right.

struck the head, it was free to move only as far as the neck was able to bend. High-speed video and acceleration data confirmed that head acceleration was over before the head had reached excessive neck bending. Although the data was not compromised, the stress on the dummy neck was too great and the head kinematics did not simulate those seen in the NFL video or the reconstructions.

The NFL reconstructions were done with a falling carriage on a guided rail. The pendulum did not have a rail supporting the head. *Figure 3* shows a slider system, which was designed and installed at the base of the dummy neck to allow motion of the base. This consisted of twin bearing rods mounted horizontally in the direction of the pendulum motion. Linear bearings mounted to the base of the dummy neck allowed the head-neck up to 45 cm (18 in) rearward translation. Use of the bearings resulted in a more natural motion after impact. A damper gently slowed the carriage at the end of the travel.

Pendulum Head Mass Versus Velocity

The striking dummy is typically oriented with the front boss of the helmet as the initial point of contact. From the dummy's perspective, the direction of impact is in line with its vertical axis with the neck and torso supporting the head (34). The struck dummy is typically hit from the side with little support provided by the neck or torso. For this reason, there is 67% more mass on average involved from the striking player than the struck player, and the biomechanical data shows that head acceleration in the striking player was always lower than in the struck player (34).

One of the areas investigated to achieve realistic pendulum impacts was that of momentum transfer. Typical impact speed was V = 9.5 m/s (21.3 mph) in full-scale reconstructions of NFL concussions. With gravity, this speed required a 4.6 m (15 ft) fall height, which could not be achieved by the existing pendulum arm. Accelerator boosting systems were a possibility, such as bungee cords or torsion springs, but they added a dimension of complexity and unreliability to the system.

By using the principles of momentum transfer, the pendulum head was made more massive so the struck head rebounded in a realistic manner, but with a lower pendulum speed. Momentum is the product of mass and velocity. During a collision between two objects, conservation of momentum requires that mass times velocity of the pendulum before collision must equal the sum of the mass times velocity of the struck head and pendulum after collision. It was necessary that the pendulum head have enough momentum to accelerate the Hybrid III head to the desired speed, and get the ΔV of the dummy head higher to match the target condition. In this manner, the pendulum could move at a slower speed than the original full-scale NFL reconstructions, yet achieve the desired head ΔV. The revised pendulum head mass was 28 kg, which was 50% more than the original 19 kg pendulum head.

Padded Face

In full-scale reconstructions, there were two helmets involved in the collision and both had energy-absorbing padding, which compressed during impact. This prolonged the duration of helmet contact. In helmet testing, the peak acceleration is related to the maximum force exerted on the head, but the duration of acceleration influenced the velocity change. It was necessary to add "helmet padding" to the pendulum face to make the system simulate the impact duration of the full-scale NFL reconstructions.

Football helmet padding is designed to attenuate energy and reduce momentum transfer in a collision. It is compressed by the force of impact and slowly recovers. In the pendulum, an elastic or spring-like padding material was used because a higher pendulum speed would be needed if the padding absorbed energy. An aerospace-grade polyurethane foam material was selected (Model 5070 13; General Plastics Manufacturing Company, Tacoma, WA), which had good restitution

and minimal damping properties. Sixteen mm of foam was inserted between the plastic impact cap, which was machined from ultra high molecular weight (UHMW) polyethylene. The cap and padding were fixed to the steel face of the pendulum.

Telescoping Arm

In a helmet-to-helmet collision in football, the players' heads are free to glance off each other followed by a torso collision that limits the overall head travel. In the pendulum test, the impactor represents the striking player. It had lateral compliance but minimal radial (up-down) compliance. The struck head with its new slider base could move fore-aft and laterally as far as the neck would allow, but could not move in the up-down direction. A new pendulum arm was designed with a telescoping feature that allowed the arm to shorten by up to 25 cm under radial load.

Verification Tests with the Revised Pendulum

The modifications to the pendulum were achieved through a series of iterations and refinements. The final design reflected a balance in the pendulum response for all test sites. The tests were performed using a Riddell VSR-4 size L, which was used in the NFL reconstructions. The pendulum was aligned with the five sites from the early helmet tests (*Fig. 2*). Tests were run at the low (threshold) and average condition for concussion in the NFL.

Biokinetics Helmet Testing

Up to this point, all tests were conducted with a Riddell VSR-4 helmet. It was necessary to verify the feasibility of testing other helmets with the refined pendulum. Five football helmets were included in the evaluation test series, including a Schutt Air Varsity Commander, an Adams Pro Elite, a Riddell Revolution, and two Riddell VSR-4s. The additional VSR-4 was included because Riddell changed the padding used in this model from a dual density stiff vinyl nitrile foam and soft polyurethane layer against the head to a single layer vinyl nitrile liner around 2000–2001. The VSR-4 helmet was used in all the reconstruction work of Pellman et al. (28, 29) and Viano and Pellman (34), and it had a dual density liner.

Figure 4 shows the three impact sites representing the helmet-to-helmet impacts from NFL game video. Sites A, B, and C were those described by Pellman et al. (28) and were similar to locations 5, 4, and 1 of the earlier pendulum testing. The five helmets were randomly assigned an alphabetical naming convention (V–Z) to blind the name of the manufacturer and model. However, Model V was the Riddell VSR-4 with original padding because this helmet has been the baseline for helmet testing in the NFL research on concussion.

WSU Linear Impact Test

Under sponsorship from the NFL, an ITT was developed to more thoroughly represent the upper body response of struck NFL players. It provides additional biofidelity (ability to mimic the essential biomechanical responses of the human) and better simulates helmet impacts observed in game collisions than the standard NOCSAE head drop or Biokinetics pendulum test. This approach includes neck loading and resulting motion of the upper body and potential interactions between the helmet and shoulder pads and neck collars, if used. It also has the proper inertia of the upper torso of the dummy.

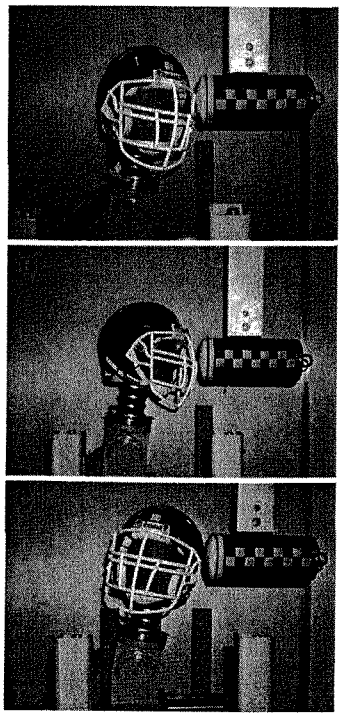

FIGURE 4. A–C, *photographs of NFL concussions demonstrating helmet impact conditions* A, B, *and* C. *These impact conditions are similar to locations 5, 4, and 1 in the earlier Biokinetics testing.*

Figure 5 shows the Hybrid III head, neck, and torso, which was mounted on a translating joint attached to an adjustable table. The translating joint allowed the upper torso to move laterally during impact. Helmet impacts were delivered by a linear, pneumatic impactor. The test fixture was designed to consistently reproduce impact speeds and energies experienced in NFL game collisions. The advantage of the linear pneumatic impactor was its ability to simulate the threshold, average, and elite impact conditions seen in NFL concussions.

The pneumatic impactor uses a thrust piston in a guide tube that is placed against a solenoid release valve and high-pressure accumulator that stores compressed air (33). By rapidly opening the solenoid, the set pressure acts against the end of the piston, which is rapidly accelerated to impact velocity. The impact mass is guided by linear bearings and has a controlled stop by crushing honeycomb Hexcel after a prescribed displacement of helmet contact. This type of pneumatic impactor can reach velocities above 12 m/s and is not limited by the drop height constraint of the pendulum. The accumulator was filled with compressed air to 20 pounds per square inch, producing an impact velocity of V = 9.5 m/s. The impactor was allowed to translate 10 cm after helmet contact to produce 10–15 ms impact duration. The level of set pressure could be varied to increase impact velocity above V = 12 m/s. This allowed testing at the elite impact condition from NFL concussions.

The impactor face was the same hemispherical surface used in the Biokinetics pendulum tests. It was used because it was designed and validated to simulate helmet-to-helmet contacts. The face included foam padding from a football helmet that was covered with the urethane helmet shell. It weighed 3.2 kg. The thrust piston was held in place by a small amount of

asymptomatic, or perhaps 1, 2, or 3 days after the player became asymptomatic, days lost could mean any number of things. We know that some physicians are more conservative than others, so days lost is not a standardized measure. As a result, *Figure 1* tells us only that NFL team physicians cleared approximately 85 to 90% of all players with a first, second, or repeat concussion to play within 3 days of their concussion. It does not tell us the more important information regarding how long they were symptomatic.

The authors' failure to capture information about concussion history before the study period could also have affected the findings. Recent peer-reviewed studies published in the medical literature (some of which the authors reference [2, 3]) suggest that previous concussions are a predisposition to future injuries. Several recent studies also suggest that there is a cumulative effect of concussion as measured by increased symptomatology or slowed recovery on symptom checklists and neuropsychological tests after subsequent injuries (1–4). The authors of the present study are quick to criticize some of these previous studies, many of which seem to have collected data on a more regular (daily) basis, using more sophisticated data collection methods, including neuropsychological screenings and postural stability assessments. Perhaps the players in the present study who were returned to play while still symptomatic with headaches were the same players who had a history of previous concussions, but we do not know this, because the authors had no knowledge of their concussion history.

Finally, the authors did not consider athlete exposure in reporting their findings, which would have allowed for a better understanding of the relative risk of initial concussion and repeat concussion, as well as by playing position. Previous studies, some of which the authors chose to compare and contrast their findings, have reported these injury data in a more epidemiologically accepted manner with respect to athlete exposure. This most likely explains some of the differences between the present study and previously published articles. For example, perhaps the concussed players in the present study played less after their injury, so they were not exposed to the same risk of injury as those who have not previously been injured. The authors state that the very high incidence of repeat concussions in the above studies is not consistent with the results of other studies, including the present one, which found that 24.6% of NFL players (160 of 650 players) who sustained one MTBI later sustained repeat MTBIs during a 6-year period (an average annual incidence of 4.1%). If the authors had reported these repeat injuries with respect to athlete exposure, the average annual incidence would have undoubtedly been higher than 4.1%. The denominator in the calculation used should be 650 only if all 650 players were enrolled in each of the study's 6 years. We know that this could not be the case, because players retire or are released by their team. Furthermore, rookies who entered the study in years 2 through 6 of the study had less total exposure and therefore a reduced chance of experiencing initial or repeat concussions during the remainder of the study period. Without capturing athlete exposures, which is not very difficult to do, the prevalence reported is suspect.

The NFL's MTBI Committee has done well to undertake this project, and I am sure that more valuable information will come from their work. However, in my opinion, the findings of the present study (as a stand-alone study) may not accurately reflect recovery from repeat concussion, and the authors' interpretation of the findings does not advance our understanding of how to safely manage sport-related concussion.

Kevin Guskiewicz
Athletic Trainer
Chapel Hill, North Carolina

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TAB 1M

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CONCUSSION IN PROFESSIONAL FOOTBALL: PERFORMANCE OF NEWER HELMETS IN RECONSTRUCTED GAME IMPACTS—PART 13

David C. Viano, Dr. Med., Ph.D.
Mild Traumatic Brain Injury Committee,
National Football League,
New York, New York and
ProBiomechanics LLC,
Bloomfield Hills, Michigan

Elliot J. Pellman, M.D.
Mild Traumatic Brain Injury Committee,
National Football League,
New York, New York
ProHEALTH Care Associates, LLP,
Lake Success, New York

Chris Withnall, M.S.
Biokinetics and Associates Ltd.,
Ottawa, Canada

Nick Shewchenko, M.S.
Biokinetics and Associates Ltd.,
Ottawa, Canada

Reprint Requests:
David C. Viano, Dr. Med., Ph.D.,
ProBiomechanics LLC,
265 Warrington Road,
Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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OBJECTIVE: The performance of five newer helmets was compared with the baseline VSR-4 helmet in 10 reconstructed cases of National Football League (NFL) collisions causing concussion. The laboratory reconstructions were conducted to determine changes in concussion risk with newer football helmets.

METHODS: In 60 laboratory tests, translational and rotational head accelerations were measured in the striking and struck players represented by Hybrid III dummies. Six-axis upper neck loads and moments were measured in five cases with the struck player and five with the striking player. Biomechanical responses and concussion risks were evaluated for each collision to determine changes with newer helmet designs.

RESULTS: Thirty-two out of 50 reconstructed cases showed greater than 10% reduction in severity index with newer helmets compared with the VSR-4; four cases increased. The average reduction in concussion risk with newer helmets was 10.8% (range, 6.9–16.7%) based on severity index. The reduction was 9.7% (range, 6.5–13.9%) based on translational acceleration and 18.9% (range, 10.6–23.4%) with rotational acceleration. Neck responses in the struck player showed a general reduction in moment and force with newer helmets.

CONCLUSION: With newer football helmets, there was a trend toward 10 to 20% lower risks of concussion in reconstructed National Football League game collisions. However, a few designs and cases showed increased responses. The evaluation of football helmets to the proposed National Operating Committee on Standards for Athletic Equipment concussion standard should lead to more uniform reductions in concussion risk with future football helmets.

KEY WORDS: Biomechanics, Concussion, Football, Impact tolerances, Neck injury, Spearing, Sport injury prevention

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Football safety equipment manufacturers have recently introduced newer helmet designs to reduce concussion risks. This has included the Adams USA Pro Elite, Riddell Revolution, and the Schutt Sport Air Varsity Commander (AVC), and DNA. National Football League (NFL), college, and high school players are starting to wear this newer head safety equipment. Most recently Collins et al. (2) concluded that high school students wearing one type of these newer helmets had a decreased risk of concussion. Although helmet testing to a new National Operating Committee on Standards for Athletic Equipment (NOCSAE) standard for concussion is still in development (3), an evaluation of the newer helmet models was undertaken to determine the performance of designs in reconstructed NFL game collisions causing concussion. The comparative data was used to determine the potential effectiveness of newer helmets in reducing the risk for concussion or mild traumatic brain injury (MTBI).

Several new helmet models have been introduced to the marketplace after a biomechanical study of concussion in NFL players (11). This study was the first to determine the speed of impact and biomechanical responses associated with NFL concussions. A second study showed a high proportion of low and

oblique impacts to the side and back of the helmet (10). Until that time, the side of the helmet was an area in which comfort foam had been used. These new understandings caused the helmet manufacturers to rapidly advance the design of helmets by providing more coverage and energy-absorbing foam on the perimeter of the helmet shell. These design changes were the first to specifically address helmet performance in reducing concussion risks. The NFL testing techniques addressing concussion have been extensively evaluated and shared with the manufacturers and NOCSAE (12).

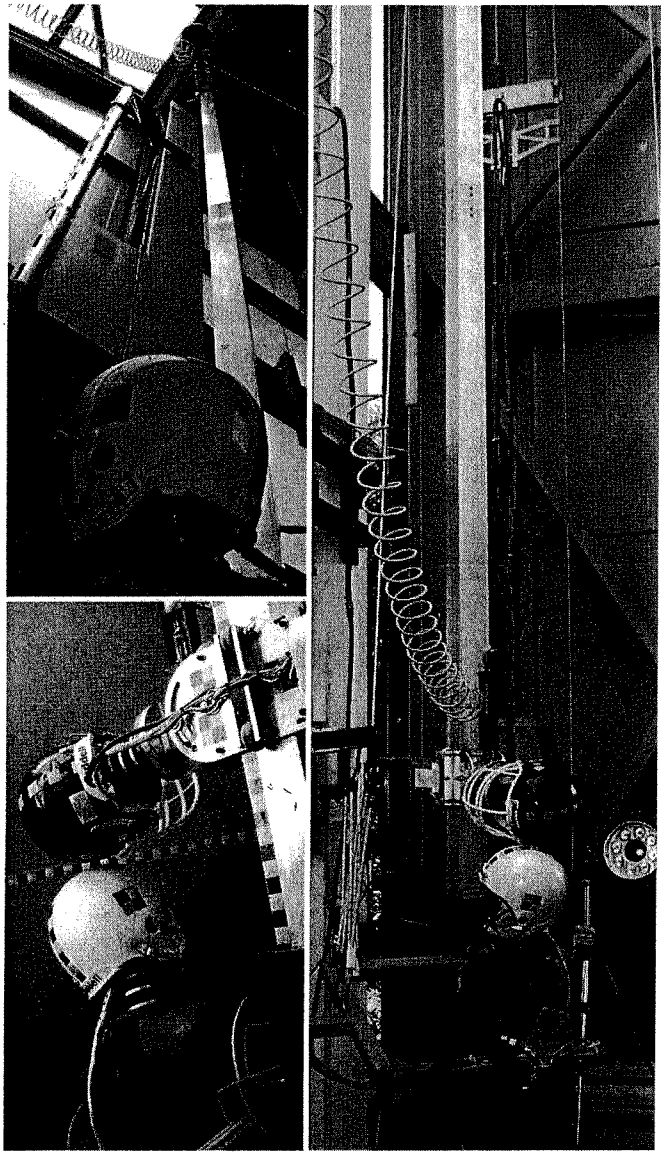

FIGURE 1. *Laboratory setup for NFL game impact reconstructions using a drop tower facility and instrumented Hybrid III dummies. The torso and pelvis of the striking player were suspended from below, and the struck player's head and neck was attached to a 7.1-kg drop weight. Adjustments were made in the alignment of the player's helmets to duplicate kinematics in the game impacts.*

The work of Pellman et al. (11) reconstructed 31 collisions in NFL games, 25 of which were associated with concussion from helmet-to-helmet impacts and falls to the ground. This study chose 10 representative reconstruction cases from the four typical impact orientations A to D causing injury (10). The 10 cases were reconstructed in new laboratory tests using the same methodology as in Pellman et al. (11). Five newer helmet designs were evaluated from the football helmet manufacturers. Baseline performance was determined by using the Riddell VSR-4 helmet from the original studies (10, 11). This allowed a verification of the test setup in repeating the earlier reconstructions and established a baseline of performance with an older helmet design.

MATERIALS AND METHODS

Brief Background

For this study, a short overview of previous laboratory methods is provided. Details of the game film selection and analysis have been published (8, 10, 11). When an MTBI occurred on the field during an NFL game, it was reported to Biokinetics and Associates Ltd. (Ottawa, Canada), the engi-

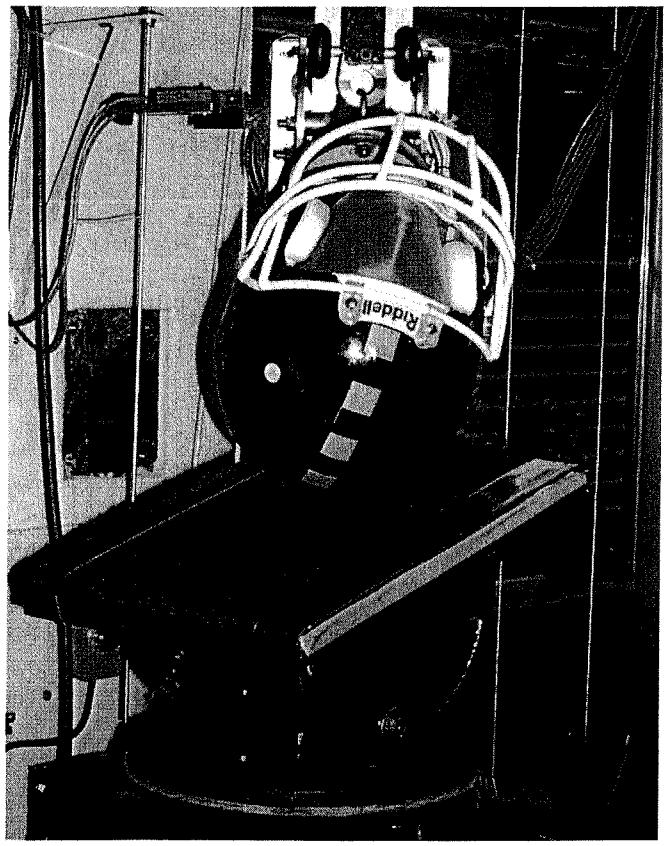

FIGURE 2. *Laboratory setup for NFL game reconstruction of ground impacts on artificial turf using the drop tower facility. Adjustments were made in the alignment of the dummy to duplicate the helmet kinematics in the ground impacts.*

TABLE 1. Original conditions and responses for 10 National Football League reconstruction tests selected to evaluate newer helmets (10, 11)[a]

	Case No.	Impact velocity (m/s)	Translational			Rotational			Resultant direction[b]		Helmet contact[c]			
			Delta V (m/s)	Accel (g)	SI	HIC	Accel (rad/s^2)	Velocity (r/s)	Front to back (degree)	Up +, down − (degree)	Facemask	Impact quad[d]	Top view[d]	Side view[e]
Helmet-to-helmet														
	84	9.4	6.3	81.9	276	222	9193	80.9	37.3	−8.1	Y	A	L 0–45	+Q2
	69	10.3	5.0	60.7	177	153	4381	19.9	53.3	−2.5	Y	A	R 0–45	−Q1
	118	10.7	9.6	100.6	492	378	7017	42.9	21.3	−2.8	Y	A	R 0–45	−Q1
	57	8.8	6.0	77.2	253	206	6514	37.0	67.4	−0.7	Y	B	L 45–90	−Q1
	155	9.1	6.6	100.3	418	341	6940	37.0	74.0	1.2	Y	B	L 45–90	−Q1
	38	9.5	9.7	118.5	736	554	9678	50.8	80.3	−4.5	Y	B	L 45–90	−Q1
	98	9.6	6.2	91.0	351	301	7548	43.4	64.5	−7.6	N	B	L 45–90	+Q2
	164	10.8	6.0	123.7	451	370	9590	26.6	94.7	−7.6	N	C	L 90–135	+Q2
Helmet-to-ground														
	67	8.1	8.0	135.4	756	632	5957	13.8	89.1	−18.3	N	C	R 90–135	+Q2
	123	6.3	8.3	121.1	866	730	4727	30.3	157.6	−24.2	N	D	R 135–180	+Q3
	Avg	9.3	7.2	101.0	478	389	7154	38.3						
	SD	1.3	1.6	23.7	235	192	1889	18.7						

[a] Accel, acceleration; SI, severity index; HIC, head injury criterion; Y, yes; N, no; Avg, average; SD, standard deviation.
[b] The impact vector is adjusted to the right side of the helmet for analysis.
[c] Helmet quadrants: A, 0-45 degree; B, 45-90 degree; C, 90-135 degree; D, 135-180 degree (9).
[d] Height of impact defined +Q1 to +Q4 are equal increments above the head cg, and −Q1 to −Q3 are below the cg (9).
[e] Resultant direction of peak head acceleration in the game reconstruction.

neering group who analyzed and reconstructed game impacts. Network tape of games was obtained from the NFL and subsequently analyzed. In addition to concussion impacts, cases of significant head impact were selected. From 1996 to 2001, 182 cases were obtained on video for analysis of the impact location on the helmet and the contact source (helmet, ground, shoulder, etc.). Sixty-one percent of the collisions involved helmet-to-helmet contacts (11).

Biokinetics determined the feasibility of determining the three-dimensional (3-D) impact velocity, orientation, and helmet kinematics. At least two clear views were necessary to make this analysis. A laboratory setup was made with instrumented crash dummies to reenact the game impacts for those videos where the 3-D impact velocity could be determined. Helmets were placed on the dummies in the laboratory reconstructions and the velocity and orientation of impact and subsequent helmet kinematics were matched to the game video. A number of significant impacts were also reconstructed where MTBI did not occur to study nonconcussion collisions. Helmet-to-ground reconstructions were limited to impacts involving artificial turf so that ground conditions could be simulated in the laboratory.

Laboratory Reconstruction Techniques

Figure 1 shows the reconstruction setup for helmet-to-helmet impacts. It involved two Hybrid III 50th percentile male dummies (1). A helmeted head-neck assembly representing the struck player was attached to a 7.1-kg mass simulating the struck player's torso and guided in free-fall from a height to match the impact velocity determined from video analysis of the game collision. The Hybrid III head and neck weighed 4.38 kg with instrumentation. Impact was against another helmeted head-neck assembly attached to the torso and pelvis of the Hybrid III dummy. This dummy represented the striking player, and weighed 46.4 kg without arms and legs and was supported by an adjustable yoke that was suspended by bungee cords to allow realistic rebound. The Hybrid III heads were covered with two layers of nylon stocking to ensure realistic movement in the helmet. *Figure 2* shows the test setup for head-to-ground impacts. A section of artificial turf and backing pad was positioned on a flat platform to simulate the field and the orientation was adjustable to simulate the angle of helmet impact.

Acceleration was measured in the dummy at the center of gravity (cg) of the head. Each headform was equipped with nine linear accelerometers (Endevco 7264a-2000, San Juan Capistrano, CA) set up in a "3-2-2-2 configuration" to determine rotational acceleration (9). This comprises three orthogonal accelerometers at the head cg and three pairs of accelerometers on the outboard x, y, and z axes. Translational acceleration at the outboard accelerometers is compared with the cg acceleration to calculate rotational acceleration (11).

In half of the cases, the dummy representing the striking player had the six-axis neck transducer installed between the head and top of the neck. The transducer measured three-axes of neck force (Fz, compression-tension; Fx, fore-aft shear; and Fy, left-right shear) and three-axes of neck moment (My, flexion-extension; Mx, lateral bending; and Mz, rotation about the z axis). The other cases had the neck transducer fit in the struck player. The sign convention used in this study has neck compression as

FIGURE 3. *Helmets used in the 10 reconstructions of NFL concussions. From* top *to* bottom *the helmets are the Adams Pro Elite, Riddell VSR-4, Riddell Revolution, Schutt DNA, and Schutt AVC.*

–Fz and neck tension +Fz, because the positive z axis is from the neck upward through the top of the head (14). The positive x axis is forward and the positive y axis is through the left ear. Neck extension is –My and flexion is +My.

High-speed video recorded head kinematics in the reconstructed game collisions. The camera (Motionscope 1000; Redlake Imaging, Tucson, AZ) was positioned similar to one of the views from the NFL game video. This allowed a one-to-one comparison of the game and reconstruction kinematics and facilitated fine adjustments in the impact orientation and alignment of the laboratory tests to match the helmet kinematics in the game (11).

An extensive series of earlier tests was conducted to isolate and quantify sources of error and variability in the reconstruction of NFL game collisions (7). This work showed the reconstructions to be repeatable and with minimal error for this type of testing.

Game Impact Selection

In previous work by Pellman et al. (11), 182 NFL game collisions were reviewed, 31 cases of which were analyzed and reconstructed. These cases comprised 27 head-to-head and four head-to-ground impacts. In 25 cases, the struck player was diagnosed with a concussion. The average impact speed of concussion cases was 9.3 ± 1.9 m/s. Video analysis revealed the impact location on the struck and striking players' helmets. From a top view, the helmet was divided into 45-degree quadrants with 0 degrees eyes forward. From a side view, it was divided into seven equal levels, four (+Q1 to +Q4) above the head center of gravity and three below (−Q1 to −Q3). Impacts to the struck helmet were resolved into four conditions, A to D: A, 0- to 45-degree quadrant, −Q3 to +Q3 level; B, 45- to 90-degree quadrant, −Q2 to +Q3 level; C, 90- to 135-degree quadrant, +Q1 to +Q4 level; and D, 135- to 180-degree quadrant, +Q1 to +Q4 level.

For this study, 10 cases of NFL game concussions were selected for repeat testing with newer helmets to investigate their effectiveness in reducing the risk of concussion. The range of impact speed was between 7.4 to 11.2 m/s for eight cases of helmet-to-helmet impacts. This was within one standard deviation of the average condition for concussion in the NFL. *Table 1* shows the cases selected from NFL concussion conditions A to D (10). The eight helmet-to-helmet cases averaged 9.8 ± 0.75 m/s. The two head-to-ground impact cases averaged 7.2 m/s.

Helmet Selection

The aim of this study was to investigate the performance of newer football helmets in conditions causing concussion in NFL players. There have been a number of newer helmets introduced during the past couple of years by the football helmet manufacturers: Adams USA (Cookeville, TN), Riddell (Elyria, OH), and Schutt Sport (Litchfield, IL). The newer helmet designs included

the Adams Pro Elite, Riddell Revolution, and Schutt Air Varsity Commander (AVC) and DNA. Because the VSR-4 padding material has recently changed, it was included as one of the newer helmets in the comparison testing. The helmets were provided directly from the manufacturers for this testing, along with generic standard issue facemasks that the manufacturer felt were commonly used.

Figure 3 shows the five helmets used in the reconstructions. The Riddell VSR-4 was the baseline helmet worn by both the struck and striking dummies in the original NFL reconstructions (10, 11, 16, 17). The outward appearance is the same as the one with newer padding. The earlier design of the VSR-4 was used to verify each of the 10 reconstructions against the results of Pellman et al. (10, 11).

For the reporting of results here, the identity of each of the newer helmets was blinded. The five newer helmets are identified as J to N. Ten NFL reconstructions of game concussion were repeated with newer helmets J to N. Helmet H is the original VSR-4, and its performance determined the baseline responses for each reconstruction. All helmets were size large and inflation fit systems were not filled. The specific helmets, facemasks, and chinstraps used in the tests are listed in *Table 2*. The materials and construction of each helmet are given in *Table 3*. Football helmets can accept a number of different facemask and chinstrap combinations. This allows a helmet to be adapted to different playing positions and player comfort. The facemask and chinstrap combinations were the same for each reconstructed case.

TABLE 2. Six football helmets, facemasks, and chinstraps used in the 10 reconstruction cases of National Football League player concussion[a]

Manufacturer	Helmet	Facemask	Chinstrap[b]
Adams	Pro Elite	FBFM-ANJOP-DW	4-pt high hookup deep cup
Riddell	VSR-4 original[c]	Z-2B	4-pt low hookup soft cup
	VSR-4 new	Z-2B	4-pt low hookup soft cup
	Revolution	G-2B	4-pt high hookup deep cup
Schutt	AVC	RJOP-DW	4-pt high hookup deep cup
	DNA	DNA-RJOP	4-pt high hookup deep cup

[a] *AVC, Air Varsity Commander.*
[b] The VSR-4 traditionally used a low hookup chinstrap, which refers to all anchor points being connected to the helmet in the lower half. High hookup refers to a triangulated chinstrap arrangement where two anchor points are low on the helmet, and two are high up, at the sides of the forehead.
[c] The baseline helmet is the Riddell VSR-4 original.

Setup Verification

For each case of helmet-to-helmet impact, the striking and struck dummies were oriented to match the original laboratory reconstructions of NFL concussions (10, 11). The exact Riddell VSR-4 helmets used in the original reconstructions were no longer available. A similar VSR-4 was fit on the dummies. Despite orienting the two dummies and camera positions relative to recorded measurements from the earlier work, several

TABLE 3. Overview of material and construction of the football helmets used in the 10 reconstruction cases of National Football League concussion[a]

	Riddell			Schutt		Adams
	Original VSR-4	New VSR-4	Revolution	Air Varsity Commander	DNA	Pro Elite
Forehead pad	Molded urethane	Molded urethane	Molded urethane	Dual density VN	Skydex + comfort foam	VN
Crown pad	VN + comfort foam	VN	VN + comfort foam	Co-molded EVA and PE	Skydex + comfort foam	VN / EPP + EVA comfort foam
Back and sides	VN + comfort foam	VN	VN + comfort foam	Co-molded EVA and PE	Skydex + comfort foam	VN / EPP + EVA comfort foam
Fit adjustment	Crown, back and sides padding encased in inflatable vinyl bladders	Crown, back and sides padding encased in inflatable vinyl bladders	Crown, back and sides padding encased in inflatable vinyl bladders	Halo-style tubular air bladder	Vinyl air bladder encasing comfort foam	Vinyl air bladder covering crown back and sides
Jaw pads	Interchangeable thickness comfort foam	Interchangeable thickness comfort foam	VN + comfort foam	VN + comfort foam	VN + comfort foam	Interchangeable thickness comfort foam

[a] VN, vinyl nitrile; EPP, expanded polypropylene; EVA, ethylene vinyl acetate.

preliminary tests were necessary to make small realignments in the setup until the postimpact kinematics, head accelerations, and velocity changes duplicated the game film and prior test data (10, 11). All verification impact tests were run at the same speed as the original game impact reconstructions.

The verification tests ensured that the 10 NFL reconstructed impacts were setup similarly to the original testing (10, 11). Cases 84 and 118 involved impacts on the facemask, which was replaced after each test. For these cases, the verification test facemask was the same model as used in the original reconstructions. This was done because the facemask's design, whether it be by the placement or number of wire bars, can affect the postimpact head kinematics. Correct reconstruction kinematics play a role in verifying that the dummy collision mimics the game video. For the other cases, a "standard" facemask was used in the comparison tests because the type of facemask would not influence the overall response.

As many as 6 to 10 tests were conducted for four reconstructions (Cases 38, 57, 84, and 118) to get the impact responses and helmet kinematics to satisfactorily match the results of Pellman et al. (10, 11). While each test involved small differences in the setup, the 32 tests were useful in determining the type of variability in repeating a test. The coefficient of variation (CV) was determined for each case by dividing the standard deviation by the average. Then, the average of the four cases was determined. For impact velocity, the CV was 1.4% (range, 0.4–2.9%). For translational and rotational acceleration, the CV was 8.9% (range, 8.1–9.8%), and 13.5% (range, 6.6–19.9%), respectively. The largest variations were in severity index (SI) and head impact criterion (HIC), which had a CV of 17.6% (range, 12.8–20.9%) and 18.1% (range, 13.1–23.6%), respectively.

Reconstruction of NFL Concussions

The testing involved repeating each case of concussion with the six different helmets fit to the struck player's head. For each case, one test was conducted for each helmet. The stationary dummy was always fit with a Riddell VSR-4 helmet having the old padding system. In this manner, one can interpret the test as though everything was exactly the same as the original reconstructions (10, 11); only the concussed player was wearing newer helmet designs. A NOCSAE nose gauge was used for repeatable fitting of the helmets to the dummy's head (5).

The position of the struck and striking heads relative to each other was maintained throughout a particular reconstruction case, even if the first point of contact on the helmets changed owing to different geometries of the newer helmet. For instance, the Revolution and Air Varsity Commander are physically larger than the original VSR-4. For a hit with some glancing, it moved the initial point of contact on the helmet and/or facemask. No adjustment was made to account for this difference. Care was taken to ensure that the orientation of the dummies and the position of the heads remained the same for each test. Measurements between the dummy and stationary laboratory landmarks were noted, and the tilt and pitch of the striking dummy support yoke were recorded to ensure repositioning of the dummy.

TABLE 4. Verification tests for the reconstruction of National Football League concussions including the original comparing the original and repeat tests with the VSR-4 (10, 11)[a]

	Case no.	Impact quad	Velocity (m/s)	Delta V (m/s)		Trans accel (g)		SI		HIC		Rot Accel. (r/s^2)	
				Original	Repeat	Original	Repeat	Original	Repeat	Original	Repeat	Original	Repeat
Helmet-to-helmet													
	84	A	9.4	6.3	7.0	81.9	73.0	276	283	222	242	9193	5914
	69	A	10.3	5.0	4.5	60.7	53.6	177	136	153	123	4381	4282
	118	A	10.7	9.6	7.0	100.6	94.0	492	273	378	179	7017	11,202
	57	B	8.8	6.0	5.5	77.2	75.0	253	226	206	196	6514	6861
	155	B	9.1	6.6	6.5	100.3	104.6	418	425	341	350	6940	7780
	38	B	9.5	9.7	10.2	118.5	109.7	736	715	554	564	9678	8354
	98	B	9.6	6.2	6.5	91.0	83.8	351	316	301	269	7548	5347
	164	C	10.8	6.0	5.5	123.7	111.9	451	369	370	296	9590	10,615
Helmet-to-ground													
	67	C	8.1	8.0	8.1	135.4	131.7	756	775	632	659	5957	4859
	123	D	6.3	8.3	7.3	121.1	115.3	866	633	730	551	4727	4300
		Avg	9.3	7.2	6.8	101.0	95.3	478	415	389	343	7154	6951
		SD	1.3	1.6	1.6	23.7	23.7	235	219	192	185	1889	2502
			Signif	NS		$P < 0.001$		NS		NS		NS	
			t =	1.53		4.11		2.17		1.78		0.31	
			P =	0.164		0.003		0.058		0.108		0.761	

[a]Trans accel, translational acceleration; SI, severity index; HIC, head injury criterion; Rot Accel, rotational acceleration; Avg, average; SD, standard deviation; Signif, significant; NS, not significant.

The struck dummy was fit with one of the six helmets, and the carriage was raised to a height that, when released, gave the desired impact velocity. The assembly was dropped, impacting the stationary dummy, which wore a traditional VSR-4. Impact data was recorded from both struck and striking dummies. The two dummy positions were checked, realigned if necessary, and the struck dummy was fit with the next helmet. For a particular reconstruction, all six helmets were tested before setting up for the next reconstruction and repeating the process. After each test, the helmets were removed and inspected for damage. Damage was only observed in facemask hits and from bent wires or, sometimes, torn chinstraps. When this happened, the facemask was replaced. The same helmet was then available for reuse in the next reconstruction case.

The two cases of ground impact followed the same procedures. The drop carriage was used to accelerate the helmeted head into impact with artificial turf placed on a rigid plate (*Fig. 2*). For head-to-ground cases, a fresh portion of artificial turf was positioned at the impact site for the six repeat impacts. This ensured that the turf backing padding did not deteriorate and influence the impact response.

Head Impact Responses and Injury Tolerances

The primary response of the head is the resultant translational acceleration of the head cg. This was determined from three orthogonal accelerations measured in the dummy. Although translational acceleration is measured in units of m/s^2, it is reported in g (1 g = 9.8 m/s^2). Integration of the resultant acceleration gave the change in head velocity or ΔV during impact. For the head impacts, the resultant acceleration was used to calculate two head injury criteria (13). NOCSAE football helmet standards use the SI:

$$SI = \int^{T} a(t)^{2.5} dt \qquad (1)$$

where a(t) is the resultant translational acceleration at the head cg and T the duration of the acceleration. An SI of 1200 is the tolerance for serious injury in helmet testing (2, 4, 6), but an SI of 300 has been proposed for concussions (11). The National Highway Traffic Safety Administration (NHTSA) uses a variation of SI to assess head injury risks in car crashes. The HIC is determined by:

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)]^{2.5}_{max}\} \qquad (2)$$

where t_1 and t_2 are determined to maximize the HIC function and a(t) is the resultant translational acceleration of the head cg. In practice, a maximum limit of $T = t_2 - t_1 = 15$ ms is used. A HIC of 700 for a duration of 15 ms is used in car crash safety evaluations. A HIC of 250 has been proposed for concussions (11). The second type of biomechanical response of the head involves rotational acceleration and rotational velocity. Although tolerances have been proposed, there is no consensus on limits at this time.

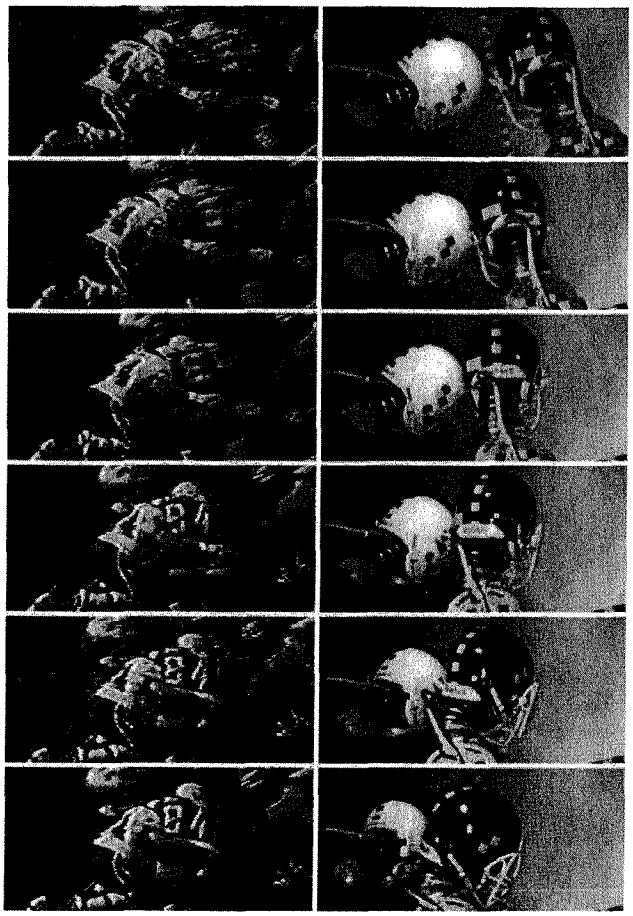

FIGURE 4. *Sequence from high-speed video of the laboratory reconstruction of NFL concussion Case 38* (right). *The sequence is from* top *to* bottom *with the striking player on the* left *and struck player on the* right *in the sequence. The* left *sequence is from game film.*

Neck Impact Responses and Injury Tolerances

Neck responses and tolerances were extensively reviewed in a recent study of the striking player in NFL concussions (17). Only a short overview will be given here. Impact of the head causes the neck to deform by forces and moments acting at the occipital condyles. For years, the axial compression tolerance has been 4000 N (13). Neck tension and shear tolerances are 3300 N and 3000 N, respectively. Until recently, the tolerance for sagittal bending of the neck has been 57 Nm in extension and 190 Nm in flexion.

Nij is a neck injury criterion used by NHTSA to address four different mechanisms: tension-extension (Nte), tension-flexion (Ntf), compression extension (Nce), and compression-flexion (Ncf). The criterion is only for sagittal plane motion based on flexion-extension bending (My) and tension-compression force (Fz). The formula for Nij is:

$$Nij = (Fz/Fzc) + (My/Myc) \qquad (3)$$

where Fzc is the critical intercept for axial neck loading and Myc the critical intercept for flexion-extension bending moment at the

TABLE 5. Repeat reconstruction of 10 National Football League concussions with newer football helmets (H is the original VSR-4 helmet and J–N are newer designs)[a]

Case no., impact velocity site	Helmet	SI	HIC	Transl accel (g)	Delta V (m/s)	Rot accel (r/s^2)	Res neck force (N)	Res neck moment (Nm)	
84[b]	H	283	242	73	7.0	5914	1436	90.8	Struck
9.4 m/s	J	292	252	75	7.3	6152	1410	100.5	
A	K	317	246	81	7.1	4793	1403	101.2	
	L	297	253	81	7.3	5844	1365	79.2	
	M	272	233	69	7.1	4939	1654	64.4	
	N	233	196	69	6.1	3234	1656	41.4	
	Avg	282	237	75	7.0	5146	1487	79.6	
	SD	28	21	5	0.5	1087	132	23.3	
69[c]	H	136	123	54	4.5	4282	2521	12.6	Striking
10.3 m/s	J	110	93	56	4.4	4503	2488	19.1	
A	K	107	92	58	3.6	4496	2657	12.7	
	L	90	80	48	4.1	4674	2036	16.6	
	M	83	74	44	3.9	3318	2036	15.1	
	N	110	97	54	4.2	3816	1849	11.6	
	Avg	106	93	53	4.1	4182	2265	14.6	
	SD	19	17	5	0.3	517	330	2.9	
118[b]	H	273	179	94	7.0	11,202	2975	144.5	Struck
10.7 m/s	J	140	113	51	6.5	5879	2223	180.6	
A	K	204	158	65	6.5	5023	2236	186.6	
	L	242	198	65	6.9	6175	2545	169.0	
	M	327	274	76	8.5	5898	2393	110.0	
	N	492	389	100	9.1	8909	4253	254.6	
	Avg	280	219	75	7.4	7181	2771	174.2	
	SD	122	99	19	1.1	2373	777	48.4	
57[b]	H	226	196	75	5.5	6861	1869	42.6	Struck
8.8 m/s	J	251	213	83	5.4	5943	2178	46.9	
B	K	235	201	79	5.3	5845	1379	91.5	
	L	217	190	70	5.6	5721	1758	51.1	
	M	226	190	75	5.7	5378	1741	31.0	
	N	281	233	79	6.0	8020	1598	100.3	
	Avg	239	204	77	5.6	6295	1754	60.6	
	SD	23	17	5	0.3	979	268	28.3	
155[c]	H	425	350	105	6.5	7780	4572	45.3	Striking
9.1 m/s	J	376	317	95	6.3	6939	4617	39.1	
B	K	296	242	79	6.3	5620	5199	48.2	
	L	451	369	109	6.6	6347	4730	39.9	
	M	298	249	79	6.6	4051	4843	53.5	
	N	387	312	91	6.7	5473	4931	59.5	
	Avg	372	307	93	6.5	6035	4815	47.6	
	SD	64	52	13	0.2	1295	231	7.9	

TABLE 5. Continued

Case no., impact velocity site	Helmet	SI	HIC	Transl accel (g)	Delta V (m/s)	Rot accel (r/s^2)	Res neck force (N)	Res neck moment (Nm)	
38[b]	H	715	564	110	10.2	8354	2598	63.0	Struck
9.5 m/s	J	633	511	95	10.5	5270	2821	70.3	
B	K	622	514	92	10.2	4824	2917	55.4	
	L	685	604	105	9.7	7312	2718	63.5	
	M	592	475	89	9.9	6338	2680	33.5	
	N	645	531	97	10.1	5152	2785	65.8	
	Avg	649	533	98	10.1	6208	2753	58.6	
	SD	45	45	8	0.3	1397	112	13.2	
98[c]	H	316	269	84	6.5	5347	3568	28.1	Striking
9.6 m/s	J	272	236	76	6.6	4186	3517	16.5	
B	K	285	246	80	6.1	5920	3300	32.9	
	L	232	209	65	6.4	4618	3120	38.0	
	M	272	235	82	5.8	5509	3343	38.2	
	N	217	186	73	5.6	3686	2402	34.6	
	Avg	266	230	76	6.2	4878	3208	31.4	
	SD	36	29	7	0.4	857	427	8.2	
164[c]	H	369	296	112	5.5	10,615	3002	30.3	Striking
10.8 m/s	J	282	220	93	5.9	8464	3503	31.0	
C	K	266	209	86	5.9	7816	3716	32.4	
	L	258	214	85	5.8	9517	3386	28.1	
	M	287	247	83	5.3	8031	3313	37.4	
	N	286	245	83	5.9	6466	3398	37.6	
	Avg	291	238	90	5.7	8485	3386	32.8	
	SD	40	32	11	0.2	1438	235	3.9	
67[b]	H	775	659	132	8.1	4859	3603	136.9	Struck
8.1 m/s	J	706	614	119	8.0	3416	3312	113.0	
C	K	593	521	105	8.0	4123	3401	125.8	
Ground	L	662	570	114	8.2	4012	3599	129.4	
	M	659	563	116	8.1	3758	3330	121.1	
	N	543	454	107	8.2	4642	2960	96.8	
	Avg	656	564	115	8.1	4135	3367	120.5	
	SD	82	71	10	0.1	540	237	14.1	
123[c]	H	633	551	115	7.3	4300	–[d]	–[d]	Striking
6.3 m/s	J	690	593	123	7.5	3931	–	–	
D	K	431	378	92	7.4	3463	–	–	
Ground	L	919	765	153	7.9	5169	–	–	
	M	540	474	99	7.9	3851	–	–	
	N	508	440	102	7.6	3533	–	–	
	Avg	620	534	114	7.6	4041	–	–	
	SD	173	137	22	0.3	629	–	–	

[a] SI, severity index; HIC, head injury criterion; Trans accel, translational acceleration; Rot Accel, rotational acceleration; Res, resultant; Avg, average; SD, standard deviation.
[b] Neck load cell placed in the struck player
[c] Neck load cell placed in the striking player
[d] No data obtained in these tests.

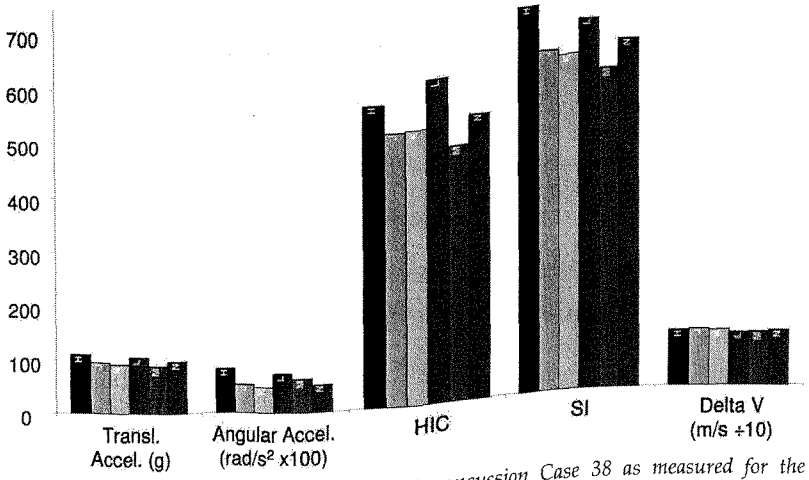

FIGURE 5. *Biomechanical responses for NFL concussion Case 38 as measured for the baseline helmet (H) and five newer football helmets (J–N).* Transl, *translational;* Accel, *acceleration;* HIC, *head injury criterion;* SI, *severity index.*

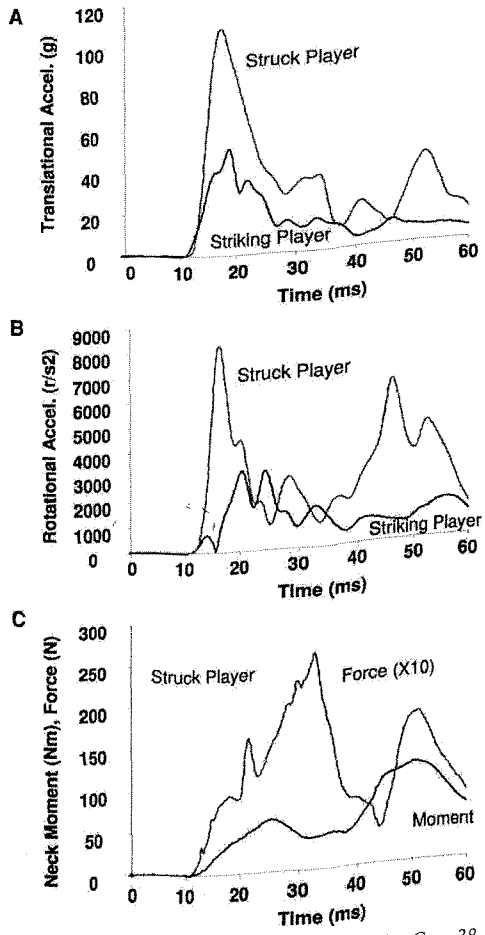

FIGURE 6. *Head and neck biomechanical responses for Case 38.* A, *graph showing the resultant translational acceleration of the struck and striking player.* B, *graph showing the resultant rotational acceleration.* C, *graph showing the resultant neck force and moment in the struck player.* Accel, *acceleration.*

occipital condyles. The critical intercepts are: Fzc = 6806 N for tension, Fzc = 6160 N for compression, Myc = 310 Nm for flexion, and Myc = 135 Nm for extension. During an impact, all four combinations of neck response need to be below Nij = 1.0. In addition, peak neck tension cannot exceed 4170 N and compression 4000 N. Although lateral bending tolerances are not specified, the sagittal criteria give an indication of tolerable responses.

Concussion Risk Functions

Pellman et al. (11) determined concussion risk functions, which relate the probability of injury, p(x), to a response parameter, x, based on a statistical fit to the sigmoidal function $p(x) = [1 + \exp(\alpha - \beta x)]^{-1}$, where α and β are parameters statistically fit to the responses from the NFL impact reconstructions. The NFL concussion parameters for the Logist function are: $\alpha = 2.677$ and $\beta = 0.0111$ for HIC, $\alpha = 2.737$ and $\beta = 0.0092$ for SI, $\alpha = 4.678$ and $\beta = 0.0573$ for translational acceleration, and $\alpha = 5.231$ and $\beta = 0.000915$ for rotational acceleration.

Statistical Analyses

The percent change in concussion risk is defined as: $\% = (p_o(x)-p_N(x))/p_o(x)$, where $p_o(x)$ is the probability of concussion with the original VSR-4 helmet and $p_N(x)$ is the probability of concussion with the newer helmet for biomechanical variable x. The increment in concussion risk is defined as: $\Delta = p_o(x)-p_N(x)$. The significance of differences between the reconstructions of Pellman et al. (11) and repeat testing of 10 cases was determined by a matched *t* test using two-sided tail distribution with the baseline VSR-4 helmet data. The *t* test was performed using the standard analysis package in Excel software (Microsoft, Seattle, WA). The regression analysis was also used from Excel, which determined the average and 95% confidence interval for a linear fit between response data.

RESULTS

Verification tests

Table 4 shows the verification test data for the 10 NFL re-reconstructed impacts. This was used to match each reconstruction as close as possible to the original results from Pellman et al. (10, 11). The old and new data are shown side-by-side. The impact velocity was the same for each case by matching the drop height. The head ΔV was within 9.0 ± 7.6% on average for the 10 reconstructed cases. This was determined by taking the absolute value of the percent change between the repeat and original response and determining the average and standard deviation for all cases.

The difference in ΔV was not statistically significant (t = 1.53, P = 0.16, NS) for the 10 reconstructions using a paired *t* test. The peak translational acceleration had a smaller difference of 6.6 ± 3.2%, but it was significantly lower (t =

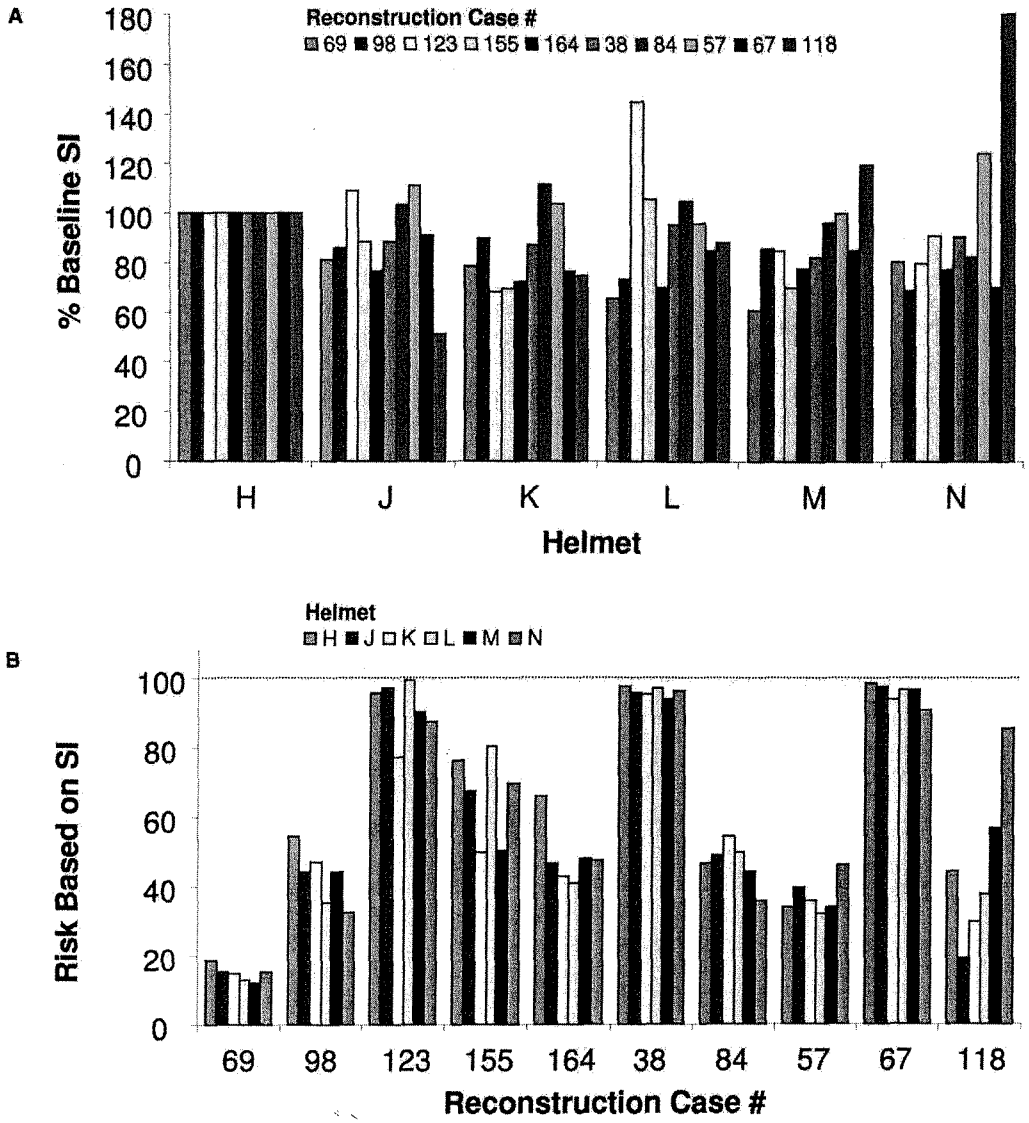

FIGURE 7. A, *graph showing the head severity index (SI) for the six helmets (H, J–N) tested in 10 NFL concussion reconstructions.* B, *graph showing the concussion risk for each game reconstruction and helmet for the 10 cases based on the SI injury risk function. Cases 123, 38, and 67 are the most severe, and Cases 69 and 57 are the least severe in terms of concussion risk.*

4.11, $P < 0.001$) in all reconstructed cases than the original NFL data. The head SI and HIC showed higher variations from the original reconstructions, but the differences were not significant. Based on a comparison of the head kinematics and biomechanical responses, it was determined that the 10 reconstructions were reasonably matched to the original data. Once the setup was verified for a case, the five newer helmets and baseline VSR-4 were run in sequence for direct comparison.

Newer Helmet Performance in Case 38

Figure 4 shows an example of the game film and laboratory reconstruction of an impact causing concussion in the NFL. It is a film clip for Case 38. The left sequence shows the NFL game collision; the right shows the laboratory reconstruction using the helmeted Hybrid III dummies. The collision caused concussion in the struck player. In the tackle, the striking player aligns his head, neck, and torso to deliver momentum to the struck player. The struck player's head absorbs the momentum as the neck bends and head displaces in the direction of the collision. The laboratory reconstruction shows similar head kinematics. The force of impact compresses the neck of the striking player, increasing the effective mass. As in the game video, the struck player's neck bends late in the collision, and the helmets eventually slide off each other.

Figure 5 summarizes the head responses for the concussed player with the baseline VSR-4 helmet (H) and the five newer helmet designs (J–N). In most cases, the differences are small, but there is a general reduction in head acceleration, SI, and HIC. The HIC with helmet L was higher than baseline; however, the head translational and rotational accelerations were lower, indicating a longer duration impact with the newer energy-absorbing liner. Similar ΔV indicate an equivalent energy transfer in the collisions with the six different helmets.

Figure 6 shows the time history of head responses for the struck and striking players in Case 38. This is the baseline VSR-4 performance. Time zero is before helmet contact, which occurs around 10 ms. The head translational and rotation accelerations rise rapidly because of the 9.5 m/s impact speed. The struck player experiences peak accelerations of 110 g and 8354 r/s^2, which are much greater than those in the striking player. The SI was 715. Neck tension and bending moment occur later in the impact as the neck is deformed by the follow through of the striking player (bottom two images in *Fig. 4*).

Newer Helmet Performance in All Reconstructions

Table 5 gives data on the struck player in the 10 reconstructions with six different helmets. SI and HIC values are calculated from the resultant translational acceleration, whereas the other responses are peak values for the resultant translational and rotational acceleration, head ΔV, and resultant neck tension and bending moment. Half of the neck responses are for the struck players and the other half are for the striking players.

Reduction in Concussion Risks with Newer Helmets

Figure 7 shows the percent change from baseline SI with the five newer helmets in each of the 10 reconstructed concussion impacts. Helmet H is the baseline, which is at 100% by definition. For newer helmets J to N, there is a trend for lower SIs, although an increase is noted in several cases with each of the newer helmets. Thirty-nine out of the 50 tests with the newer helmets had lower SIs than the baseline. The SI was greater for at least one case for every newer helmet, although most of the increases were within the expected standard deviation for repeat testing.

Only four cases with newer helmets had SI greater than 10% of baseline performance. The 10% level was chosen to look at differences beyond a reasonable level of test-to-test variability. Three of the four involved facemask impacts with different helmets (Cases 84, 118, and 57). In contrast, 32 cases showed more than 10% reduction in SI with the newer helmets. Case 57 had no reductions of greater than 10% in SI and Case 84 had only one helmet with more than 10% improvement. Many of the cases showed uniform reductions in SI with most of the helmets, including Cases 164, 69, 98, and 67. The number of reconstructed cases showing greater than 10% reduction in SI with the newer helmets ranged from five to eight. Helmet K showed improvements in eight cases and J in 5.

Figure 7 also shows the risk of concussion for each reconstruction and helmet. These values were determined using the SI logist parameters for concussion. Cases 123, 38, and 67 are the most severe, with concussion risks approaching 100% with all helmets. There was a general trend toward lower risks with newer helmets; however, there are some exceptions. *Table 6* shows the average reduction in concussion risk based on SI, HIC, and translational and rotational acceleration for all of the reconstructed cases. Both the average reduction in risk and the incremental reduction are given with the standard deviation in change.

Figure 8 summarizes the average and standard deviation in the percent reduction in concussion risk with the five newer helmets in the 10 NFL game reconstructions. Helmets K and M had the largest reductions at 16.7 ± 14.9% and 13.6 ± 16.3%, respectively. Helmets N and L had the smallest reductions at 5.4 ± 33.9% and 6.9 ± 23.1%, respectively, and the standard deviation involved negative performance. For all helmets, the percent reduction in concussion risk averaged 10.8% based on SI. The increment in risk reduction was also determined. This is the difference in risk with the baseline and newer helmet in the 10 reconstructions. Newer helmets M and K had the largest increment in lower risk at 13.3 ± 10.4% and 9.2 ± 11.1%, respectively. The average increment in lower risk was 7.2% for the five newer helmets. The largest reduction in risk was seen using rotational acceleration.

Figure 9 summarizes the average and standard deviation in resultant neck force and moment. For the struck player, the peak neck force is due to tension and shear late in the collision, after the head acceleration (*Fig. 6*). In contrast, neck loads in the striking player are primarily compression and occur during the primary impact accelerations. Neck tension was below the current limit set as part of the Nij crite-

TABLE 6. Reduction in head biomechanical responses converted to concussion risks with newer helmets tested in 10 National Football League game impacts[a]

		Helmet					
		J	K	L	M	N	Avg
SI	Avg (%)	11.4	16.7	6.9	13.6	5.4	10.8
	SD	17.5	14.9	23.1	16.3	33.9	
	Δ risk (%)	6.1	9.2	5.0	13.3	2.7	7.2
	SD	9.9	11.1	9.7	10.4	18.0	
HIC	Avg (%)	10.4	16.2	3.5	9.6	2.0	8.3
	SD	15.1	13.0	21.7	24.5	44.3	
	Δ risk (%)	5.1	8.5	3.4	11.9	1.6	6.1
	SD	8.1	10.1	9.3	13.0	20.5	
Translational Acceleration	Avg (%)	8.1	11.5	6.5	13.9	8.6	9.7
	SD	16.3	15.4	18.9	8.9	10.5	
	Δ risk (%)	9.6	14.4	9.7	18.1	9.3	12.2
	SD	18.7	18.1	18.0	12.8	12.2	
Rotational Acceleration	Avg (%)	18.0	20.4	10.6	23.4	22.0	18.9
	SD	16.9	19.6	17.5	15.3	18.9	
	Δ risk (%)	16.4	21.6	9.6	18.3	20.9	17.3
	SD	17.7	23.8	15.3	20.0	21.3	

[a]Avg, average; SI, severity index; SD, standard deviation; HIC, head injury criterion.

rion, but compression forces exceeded tolerance in one case. The resultant neck moment in the struck player has levels four to five times that of the striking player and occurs late as the head is pushed in the direction of the collision. The striking players align their head, neck, and torso through the collision minimizing neck bending.

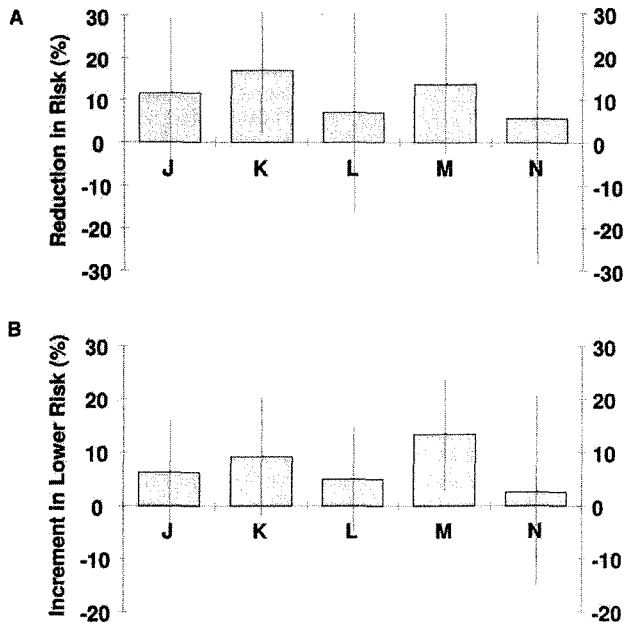

FIGURE 8. *A, graph showing the average reduction in concussion risk with newer helmets J to N as compared with the baseline VSR-4 in 10 NFL reconstructed collisions (±1 SD* bars *are shown). B, graph showing the incremental reduction in risk with the newer helmets.*

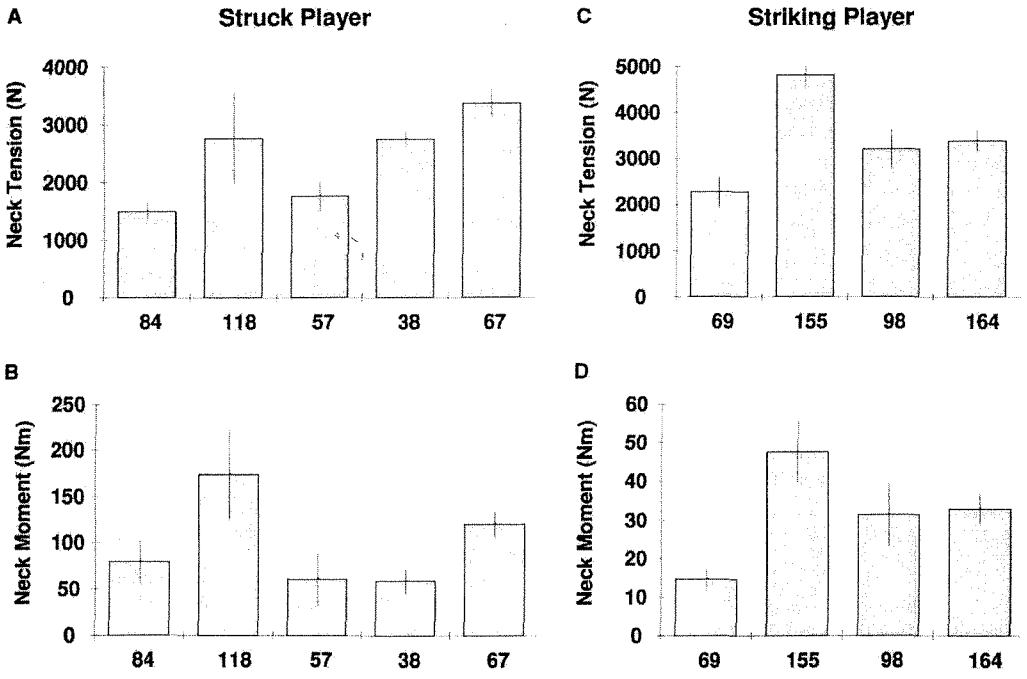

FIGURE 9. *Bar graphs showing average and standard deviation in peak resultant neck force and moment for six helmets tested in five cases (84, 118, 57, 37, 67) for the struck player* (A *and* B) *and four cases (69, 155, 98, 164) for the striking player* (C *and* D). *The reconstruction case number is shown on graphs* B *and* D.

DISCUSSION

It is hoped that these tests give perspective to helmet manufacturers, equipment managers, players, athletic trainers, and physicians on the progress being made in football helmet designs to reduce the risk of concussion. Newer football helmets reduce concussion risks in collisions representative of NFL player experiences. Depending on the biomechanical response, the reductions are in the range of 10 to 20% lower risk of concussion. This is an encouraging trend with newer helmets, and additional progress is expected as efforts focus on a new football helmet standard to reduce concussion (12). This study provides a perspective on the state of art in helmet performance in real-world game impacts in the NFL. The tests should help NOCSAE in its effort to finalize new helmet standards for concussion.

The newer football helmets reduce concussion risks by using thicker and more energy-absorbing padding low on the side and back of the helmets, and around the ears. These are the primary areas where NFL players are experiencing collisions causing concussion. Until now, helmets have used a mixture of comfort padding and energy-absorbing foam in these areas of the helmet. The size and coverage area of the side of the helmet are evolving. This is providing more space for sophisticated liner materials and constructions. The testing reported here shows that the selection of softer grades of vinyl nitrile (www.dertexcorp.com), coupled with increased thickness, can absorb impact energy, lowering head accelerations and reducing the risk of concussion in player collisions. Those collisions were defined by the reconstructions of Pellman et al. (10, 11), and they are the basis for a proposed NOCSAE concussion standard (12). Improved padding constructions are also being employed to distribute force and absorb energy. An example of an innovative padding construction is SKYDEX (Centennial, CO), which is a molded thickness of twin-hemispheres of polymer sheets that can be tuned for energy absorption.

Concussion often occurs to an unsuspecting player by impacts outside their line of sight and during open field tackles, which involve high collision speeds (10, 11, 17). The unsuspecting player does not take evasive action to protect himself, and the substantial forces of the collision rapidly displace the struck player's head. The striking players align their head, neck, and torso to provide more of their body mass in the collision. This causes the struck players to experience much greater head accelerations, as seen in *Figure 6*. Although targeted helmet impacts to the head are drawing penalties and fines in NFL games, there will remain unintentional and inadvertent helmet-to-helmet impacts with enough impact velocity to cause concussion with newer helmet designs. Helmet-to-ground impacts are another matter. Because of the continued exposure to significant head impacts, we expect to see further advances in the design of helmets as attention continues to focus on concussion prevention.

Limitations

The previous research by the NFL's MTBI Committee (10–12, 15–17) has described the limitations of laboratory testing of this type with crash test dummies and measurements of biomechanical responses. The obvious limitation is the realism of the dummy and injury criteria in mimicking the player's experience with concussion. We think the testing described here is the best approach to assess and reduce the risks of concussion by helmet testing to assess the performance of helmet designs. However, we again acknowledge the limitations in this type of methodology.

During the setup of the reconstructions, several of the repeat tests were used to determine variability in the impact and biomechanical responses of the dummy. Although these variabilities may seem large, they are typical of impact testing with crash test dummies used in automotive safety research. This series of tests involved single impacts with a particular newer helmet in each of 10 NFL game reconstructions. There was no intention of running repeat tests for each reconstruction with the same newer helmet. This type of testing would be needed to determine statistically significant differences among the helmets. As such, these tests provide information on the trends for reductions in biomechanical responses of the head and risks for concussion with newer helmet designs.

There remains a need to continue the development of information on the underlying mechanisms of concussion injuries. Our studies using finite element modeling of brain responses (16) and comparison of head dynamics in football and boxing (15) offer new insights into injury mechanisms, but these studies only scratch the surface on how and why concussion occurs. For example, the finite element studies show that the largest strains occur late in the midbrain well after the peak head accelerations of the impact. What implication that has on future helmet designs has not been explored. Even as new information emerges on concussion biomechanics, we are confident that the measured biomechanical responses reported here correlate with on-field conditions of player injury. As new insights are developed, these studies will provide a technical background upon which to judge the evolving science and development of new methods and technologies.

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Acknowledgments

Adams USA, Riddell, and Schutt Sport supplied helmets and facemasks without charge for evaluation in this study. We appreciate their cooperation with Biokinetics and the National Football League (NFL) Mild Traumatic Brain

Injury Committee. The manufacturers agreed with the identification of their product in the article, but the authors decided that the test results would be blinded to the specific manufacturer and model. The original VSR-4 has been identified in the testing because it served as the baseline helmet design. Funding for this research was provided by the NFL and NFL Charities. The Charities is funded by the NFL Player's Association and League. Their support and encouragement to conduct research on mild traumatic brain injury is appreciated. None of the authors has a financial or business relationship with the helmet manufacturers posing a conflict of interest to the research conducted on helmet performance and concussion in professional football. The authors appreciate the assistance of Cynthia Arfken, Ph.D., Wayne State University, in the statistical analysis of the data.

COMMENTS

Because of liability issues, football helmet manufacturers have essentially been reduced to three companies. Using biomechanical studies previously published in *Neurosurgery* and carried out under the auspices of the National Football League (NFL) committee that addresses mild traumatic brain injury, the authors have attempted to determine the risk of concussion with the use of new football helmet designs. Using reconstructed NFL game collisions in a laboratory setting, they concluded that the more recently designed and constructed helmets reduce the risk of concussion in reconstructed NFL laboratory simulated game collisions as much as 10 to 20%.

As a result of the sophisticated biokinetic analyses of head impacts in football, helmet manufacturers have redesigned helmets by using thicker and more energy-absorbing padding, particularly low on the side and back of the helmets where it is thought that most concussive blows occur.

We recently reported our results in terms of concussion rates and recovery times among more than 2100 high school football players in Pennsylvania (1). More than half of the players used the newer Revolution helmet evaluated by the authors in this study.

More than 5% of players with the Revolution helmet experienced concussion versus nearly 8% of those who wore traditional helmets. Recovery time, however, was similar in both groups. Approximately 50% of those concussed required more than 1 week to fully recover with roughly 15% requiring 3 weeks or longer.

Regardless of the innovations in technology, cerebral concussions will never be eliminated from the sport of football. The severity of the injuries, however, may be mitigated and with neurocognitive tests widely available for assessing the post-concussion syndrome and neurocognitive abnormalities, and return to play can be much more appropriately and scientifically determined. All of these factors should help to reduce the long-term disability that can result from traumatic brain injury.

The authors are to be commended for their 13th contribution to *Neurosurgery*, which will almost certainly be assembled with the other articles into a compendium that represents a major contribution by the NFL in this area of injury management and mitigation.

Mark Lovell
Neuropsychologist
Joseph C. Maroon
Pittsburgh, Pennsylvania

1. Collins M, Lovell MR, Iverson GL, Ide T, Maroon J: Examining concussion rates and return to play in high school football players wearing newer helmet technology: A three-year prospective cohort study. **Neurosurgery** 58:275–286, 2006.

I found this article to be of great interest. Although there are clear limitations to this kind of engineering science (as detailed in the study), the authors' ability to quantify changes in protection with helmet design modifications should translate into reduced injuries or reduction in certain kinds of injuries. We will anticipate data on head injury reports after new helmets are used on the playing field. I serve on the medical staff of a professional hockey team, where helmets often fit more loosely, chin straps are not as tight, and rotational head movements are much freer to occur. Thankfully, head collisions are less frequent in this sport than in football.

Douglas S. Kondziolka
Pittsburgh, Pennsylvania

The authors have built upon their earlier work, much of which was published by the same researchers working with the NFL Mild Traumatic Brain Injury Committee. Their research has led to football helmet design changes that may lower the risk of concussion in athletes. It is always gratifying when additional data supports the objectives under which new products are developed, and the athlete will be the ultimate beneficiary.

My employer, Riddell, carefully followed the earlier work of Viano et al., while developing one of the helmet models tested in this study, the Riddell Revolution. This work indicated that a high percentage of concussion-causing impacts were to the side of the head and face of the athlete. Laboratory testing that focused on the types of impacts and player responses known to have resulted in concussion of NFL players resulted in helmet design changes at the side of the head and face. This article seems to reinforce the data presented by Collins et al. (1), which strongly suggests that the newer helmet design reduces the risk of concussion in athletes.

It is understood that the purpose of this study was to comment generally on the performance of various helmet models when subjecting them to specific test criteria and not to describe the functions of specific product features or product design choices. We should keep in mind that subtle product features, such as face mask style or fit on the headform, can affect performance of a helmet in the laboratory as well as on the field. The helmets in this study might have been configured more equivalently. *Figure 3* of the article shows multiple faceguard styles, offering varying degrees of protection, used on the various test helmet models. Because the facemask was so often an initial point of impact, similar facemask styles should have been used in all tests. Although standardizing helmet fit on the Hybrid III test headform can be subjective, the air fit systems of the helmets might have been used in addition to using a National Operating Committee on Standards for Athletic Equipment nose gauge to achieve uniform fit to the head.

Inclusion of the Adams Pro Elite and newer Riddell VSR-4 in the test group raises interesting questions. What constitutes a newer helmet design? The Adams Pro Elite has been available since 1999; the newer Riddell VSR-4 since 2001. Should they be grouped with the Riddell Revolution, Schutt Air Varsity Commander, and Schutt DNA as newer designs? Riddell has aggressively marketed, first the design intent of the Revolution to reduce the risk of concussion, then as more data was generated, that it was reducing the risk of concussion on field. Schutt Sports clearly cites the NFL-sponsored work by Viano et al. as an influence in the design of their newer models. I point this out because laboratory data and on-field data suggest that all helmets do not offer the same degree of protection to the athlete against concussion. It is my hope that studies like this one will influence institutions that participate in football to accelerate replacement of older helmets with newer models that make use of the latest designs. There is a risk

that helmet users will opt for the familiar VSR-4 because it was included in the newer design group when it may not belong there.

Again, I commend the authors for more good work. Their efforts and the publication of data like this help increase the public awareness of concussion. Increased awareness reinforces the need for proper medical treatment when concussions do occur. The public should understand that lowering the risk of concussion to the athlete is an incremental process and that no helmet can prevent all concussions.

Thad Ide
Biomedical Engineer
Chicago, Illinois

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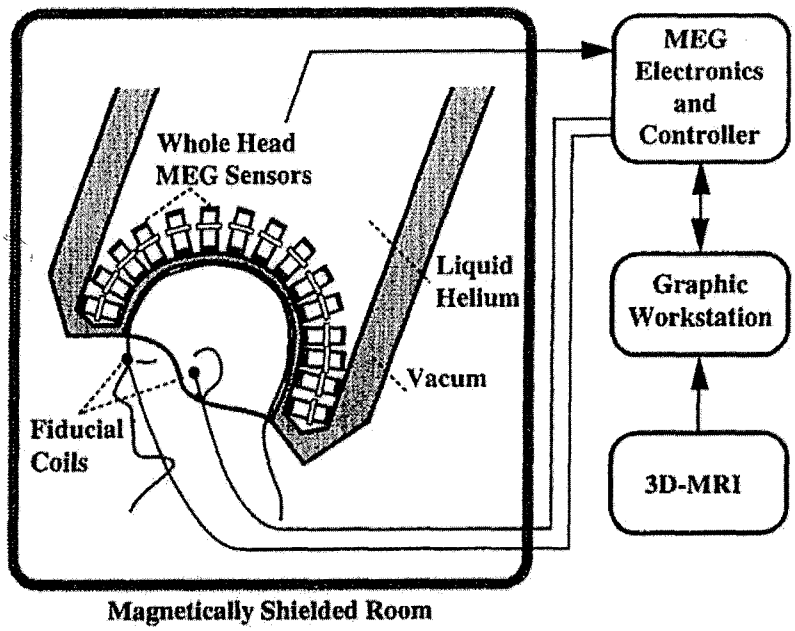
1. Collins M, Lovell MR, Iverson GL, Ide T, Maroon J: Examining concussion rates and return to play in high school football players wearing newer helmet technology: A three-year prospective cohort study. **Neurosurgery** 58:275-286, 2006.

In this study, the authors evaluated the performance of five of the more recently designed football helmet models compared with the commonly used VSR-4 helmet. The authors recreated 10 NFL collisions that resulted in concussions using Hybrid III dummies and appraised the performance of the various helmets in these reconstructed situations. The study broke down the elements of the collision between the "struck" and "striking" player and the evolution of head and neck positions during the collisions, which aids in illustrating the mechanism of many injuries.

Based on these findings, the authors converted the average reductions in head biomechanical responses to concussion risk based on injury probability functions, which were published in previous studies, and reported trends of a 10% reduction in concussion risks consistent with severity index and translational acceleration and a 19% reduction based on rotational acceleration findings. There are obvious limitations to such a study, including that the design precludes being able to demonstrate statistical significance. It is hoped that the relatively small number of collisions that were studied are representative of the biomechanical forces involved with football concussions, and that the results are able to be accurately extrapolated to lower levels of play, making helmet design changes applicable to mitigate injuries in high school and collegiate games. Although this study did not address specific design alterations and their influence on reduction of injury, future analysis should include such factors.

Because a large number of collisions involve oblique impacts to the front and side of the helmet in the areas of the facemask, studies should analyze the effect of newer designs on this and the role of the wide range of facemask designs should be measured. Likewise, the influence of mouthpiece design and usage should be considered. This is a welcomed study that should provide stimulation for future research to further define the characteristics and issues involved with improved design for football helmets. Given the nature of the game, there will continue to be mild traumatic brain injuries and occasional catastrophic injuries, and optimization of protective equipment is necessary for limiting these instances as size and speed continue to grow at all levels of play.

Julian E. Bailes
Vincent J. Miele
Morgantown, West Virginia



MRI-linked whole head magnetoencephalography (MEG) systems. *Schematic depicting a whole head, helmet-shaped 66-channel MEG device with integrated three-dimensional MRI and graphic workstation. This 1995 technology configuration was utilized in localizing the auditory cortices in 20 subjects in combination with superimposing auditory evoked magnetic field sources on three-dimensional MRIs in order to evaluate source estimation accuracy. (Nakasato N, Fujita S, Seki K, Kawamura T, Matani A, Tamura I, Fujiwara S, Yoshimoto T: Functional localization of bilateral auditory cortices using an MRI-linked whole head magnetoencephalography (MEG) system.* ***Electroenceph Clin Neurophysiol*** *94:183–190, 1995.) Please see addition material on MEG technology on pages 520, 544, 584, 590, 606, 620, 678, and 696.*

TAB 1N

CONCUSSION IN PROFESSIONAL FOOTBALL: BIOMECHANICS OF THE STRUCK PLAYER—PART 14

David C. Viano, Dr. med., Ph.D.
ProBiomechanics LLC,
Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Department of Neurology,
Long Island Jewish Medical Center,
New Hyde Park, New York, and
Department of Neurology,
Albert Einstein College of Medicine,
Bronx, New York

Elliot J. Pellman, M.D.
ProHEALTH Care Associates, LLP,
Lake Success, New York

Mild Traumatic Brain Injury Committee,
(DCV, IRC, EJP),
National Football League,
New York, New York

Reprint requests:
David C. Viano, Dr. med., Ph.D.,
ProBiomechanics LLC,
265 Warrington Road,
Bloomfield Hills, MI 48304.
Email: dviano@comcast.net

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OBJECTIVE: Impacts causing concussion in professional football were simulated in laboratory tests to determine collision mechanics. This study focuses on the biomechanics of concussion in the struck player.

METHODS: Twenty-five helmet impacts were reconstructed using Hybrid III dummies. Head impact velocity, direction, and helmet kinematics-matched game video. Translational and rotational accelerations were measured in both players' heads; 6-axis upper neck responses were measured in all striking and five struck players. Head kinematics and biomechanics were determined for concussed players. Head displacement, rotation, and neck loads were determined because finite element analysis showed maximum strains occurring in the midbrain after the high impact forces. A model was developed of the helmet impact to study the influence of neck strength and other parameters on head responses.

RESULTS: The impact response of the concussed player's head includes peak accelerations of 94 ± 28 *g* and 6432 ± 1813 r/s^2, and velocity changes of 7.2 ± 1.8 m/s and 34.8 ± 15.2 r/s. Near the end of impact (10 ms), head movement is only 20.2 ± 6.8 mm and 6.9 ± 2.5 degrees. After impact, there is rapid head displacement involving a four-fold increase to 87.6 ± 21.2 mm and 29.9 ± 9.5 degrees with neck tension and bending at 20 ms. Impacts to the front of the helmet, the source of the majority of National Football League concussions, cause rotation primarily around the *z* axis (superior-inferior axis) because the force is forward of the neck centerline. This twists the head to the right or left an average of 17.6 ± 12.7 degrees, causing a moment of 17.7 ± 3.3 Nm and neck tension of 1704 ± 432 N at 20 ms. The head injury criterion correlates with concussion risk and is proportional to $\Delta V^4/d^{1.5}$ for half-sine acceleration. Stronger necks reduce head acceleration, ΔV, and displacement. Even relatively small reductions in ΔV have a large effect on head injury criterion that may reduce concussion risks because changes in ΔV change head injury criterion through the 4th power.

CONCLUSION: This study addresses head responses causing concussion in National Football League players. Although efforts are underway to reduce impact acceleration through helmet padding, further study is needed of head kinematics after impact and their contribution to concussion, including rapid head displacement, z-axis rotation, and neck tension up to the time of maximum strain in the midbrain. Neck strength influences head ΔV and head injury criterion and may help explain different concussion risks in professional and youth athletes, women, and children.

KEY WORDS: Biomechanics, Concussion, Football, Impact tolerances, Neck injury, Sport injury prevention

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Figure 1 shows a sequence from a National Football League (NFL) game collision resulting in concussion to the struck player on the right. The striking player lines up his head, neck, and torso and drives through the other player. The collision first involves compression of the padded helmet liners as impact force increases. This causes the struck player's head to move to the right and rotate primarily about the z axis (superior-inferior anatomic axis). There is also lateral bending of the neck about the *x* axis (anterior-posterior anatomic axis). Near the end, the helmets slide off each other. These kinematics are similar to other video recorded concussions involving impact to the front of the helmet (28, 29, 37).

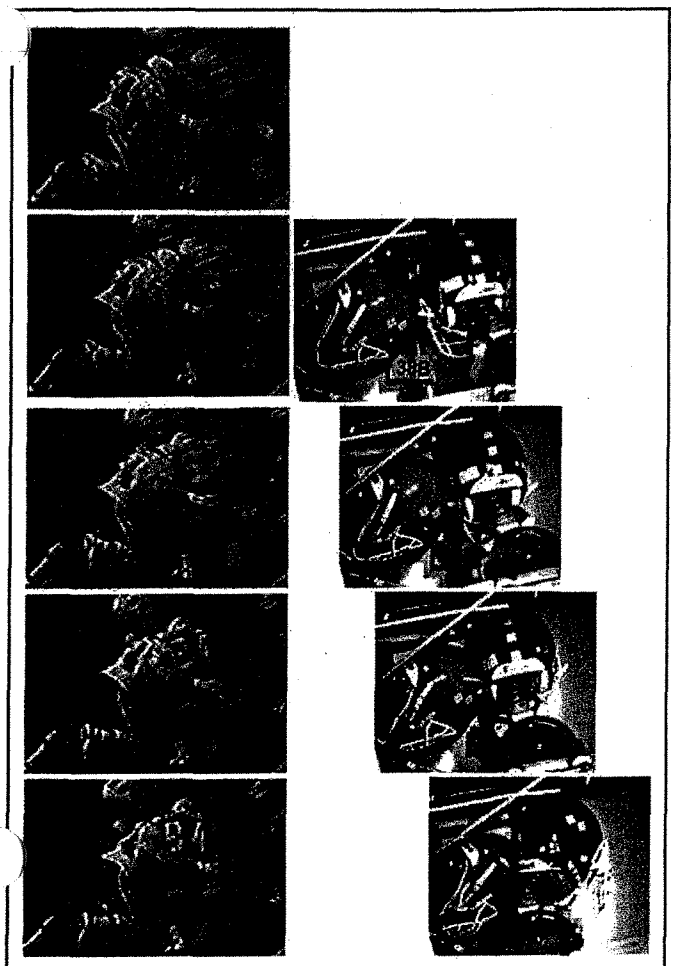

FIGURE 1. *The left sequence is from an NFL game tackle with the head-down position of the striking player as his helmet impacts the other player and his body drives forward (Case 38). The right sequence is from high-speed video of the laboratory reconstruction of the concussion. The striking player is on the left and struck player on the right. The reconstruction has the struck player moving into the impact, so the images have been shifted to duplicate the game kinematics.*

Concussions occur in most contact sports and even non-contact sports in which inadvertent head impacts occur. Between 2000 and 2004, concussions were the fifth most frequent injury in the NFL, averaging 184 incidents per year and causing 594 lost days per year. Quarterbacks have the highest risk of concussion (1.62/100 game positions [gp]), followed by wide receivers (1.23/100gp), tight ends (0.94/100gp), and the defensive secondary (0.93/100gp) (30). The plays with the highest risk for concussion are kickoffs (9.29/1000 plays) and punts (3.86/1000 plays), which have higher risks than rushing (2.24/1000 plays) and passing (2.14/1000 plays) plays.

There has been much publicity about NFL players who have retired after multiple concussions as of late. A recent study found minimal effects of multiple concussions in active NFL players (31); however, the potential usefulness of an elite-player helmet remains an option in the future. This may help players in the most vulnerable positions or plays, as well as players with multiple injuries who want to take extra precautions (37). This study explores the biomechanics of concussion in the struck player and considers head responses during and after the high accelerations of the helmet impact. A clearer understanding of injury mechanisms may lead to further improvements in safety equipment.

This study considers not only the head impact responses during the time of high collision forces, but also longer duration responses of the struck player during the follow-through of the striking player, who drives through the struck player. The greatest strains occur in the midbrain and have been shown to occur well after the high impact forces. Viano et al. (38, 39) simulated strains in the concussed player's brain using head accelerations from the NFL reconstructions (28, 29) and a sophisticated finite element model of the brain (43). *Figure 2* shows the principal strain in the midbrain for five NFL impacts to the front of the helmet involving concussion. Peak strain averaged 52.6 ± 8.6% at 16.4 ± 3.4 ms, which is well after the high impact force, which usually occurs at 6 to 8 ms (29). *Figure 2* also shows the average and one standard deviation in midbrain strain, confirming the consistently late occurrence of midbrain strain. Late occurring strain in the midbrain was found by Ivarsson et al. (14) using a physical model of brain responses

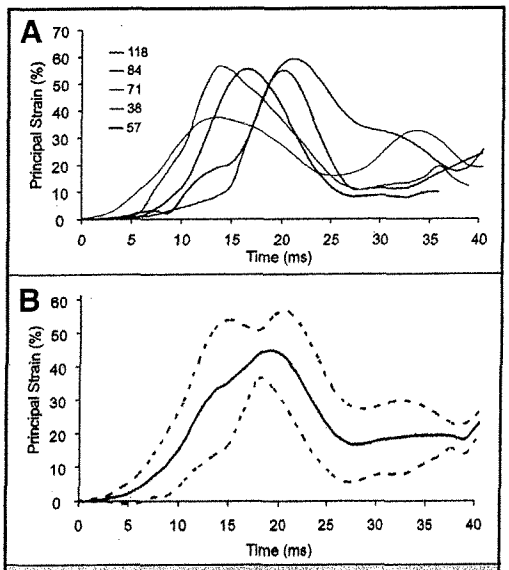

FIGURE 2. *Principal strain (A) in the midbrain for five reconstructions (Cases 38, 57, 71, 84, and 118) of impact to the front of the helmet causing concussion, and the average and one standard deviation (B) in midbrain principal strain as a function of time based on results from Viano DC, Casson IR, Pellman EJ, Zhang L, King AI, Yang KH: Concussion in professional football: Brain responses by finite element analysis—Part 9. Neurosurgery 57:891–916, 2005 (38).*

from head rotation. Similarly, the late occurring FE brain responses are a result of head rotation.

Consideration of longer duration responses is also motivated by observations of Denny-Brown and Russell (4), who concluded "in using the lighter pendulum, which does not crush the fixed skull, the animal's head is prevented from moving at the moment of injury, concussion could not be produced even with the maximum velocity obtainable with this pendulum (28.7 f/s, 8.8 m/second). Whereas, if even slight movement of the head was allowed (e.g. 3 mm), concussion occurs" (4). Although the animal tests of Gurdjian (8) are not conclusive to the observation of Denny-Brown and Russell, the possibility exists that rapid head displacement may be a factor in concussion. Denny-Brown and Russell (4) and others (6, 7, 10, 26) felt that rapid change in head velocity was a key factor in brain injury. A rapid change in velocity (ΔV) leads to head displacement.

Early animal tests (4, 8) used thinly padded impactors with one-eighth-inch hard rubber padding. Skull fracture occurred as the speed of impact increased, indicating a role of skull deformation in short duration head impacts causing brain injury. Initial efforts to reduce the risk of concussion involved preventing skull fracture. By the 1970s, thick padded liners, four-way chin straps, smoother outer shells, and a change from web suspension to internal padding were introduced in football helmets. This lengthened the pulse duration of impact and reduced the frequency of skull fractures and brain contusions (2, 11, 12, 21, 26). Padding changed the duration of impact and the loading of the brain. This situation is quite different from the conditions of the early animal tests. By the mid-1990s, concern emerged regarding concussion and efforts started to reduce concussion by further improving the design and performance of football helmets.

At the present time, helmet manufacturers are increasing the thickness and energy-absorption properties of helmet padding, as well as making other changes on the side and back of football helmets (40). New testing protocols are available to simulate the type of collision shown in *Figure 1* and other impacts with controlled laboratory tests (32). Head acceleration, severity index (SI), and head injury criterion (HIC) are being used to assess changes in the risk of concussion. However, the question remains of whether or not these measures are sufficient to define the essential aspects of injury risk.

For this study, kinematic responses (head displacement, rotation, and velocities) were determined for the struck player. A snapshot of head kinematics is presented near the end of impact (10 ms) and during rapid displacement (20 ms). Impact forces usually reach a peak in 6 to 8 ms and the high forces and head accelerations decline by 10 ms (29). This is approximately the time the head has achieved much of its ΔV and rapid head displacement and rotation begin to occur. By 20 ms, the head has experienced large displacements and rotations, and the neck is deformed. Responses at 20 ms were chosen to view the late responses of the head. Although the 10 and 20 ms comparisons of kinematic responses are somewhat arbitrary, they offer a means to visualize two time-points during the biomechanics of concussion.

Our question is, if concussion occurs by strains in the midbrain and the maximum strains occur late after the high impact forces, do late responses of the head and neck influence injury? Specifically, is rapid head displacement and rotation with neck tension, bending, and shear affecting cranial motion, loading the brain and influencing concussion? Although the information presented here may not be definitive, we hope it stimulates further study and discussion. If the late responses are a factor in injury, additional considerations may be needed to improve the protection of players, even though recent testing of newer helmets showed a reduction in head accelerations and neck responses (40).

This study is part of a larger series on concussion in professional football. The NFL has a Mild Traumatic Brain Injury Committee, which has undertaken research aimed at defining the biomechanics of concussion in professional football (28). One aspect of the effort focused on analysis of multiple views of concussive impacts from game video to determine the speed of impact. Laboratory reconstructions of the collisions were performed using instrumented test dummies to simulate the helmeted players. The laboratory reenactments closely matched the helmet motion of the field collisions. With transducers in the dummy, the translational and rotational acceleration of the head and neck loads in the struck player were determined, allowing an evaluation of the biomechanical responses causing concussion. This study evaluates the biomechanics of the struck players' concussion.

Analysis of NFL game video showed that 62% (107 out of 174) of NFL collisions involved helmet-to-helmet contact and that 16% (28 out of 174) involved helmet-to-ground contact (29). These collisions involved primarily open-field impacts of which two or more clear views of the collision were recorded. Collisions on the line are necessarily underreported. For the reviewed cases, 61% (82 out of 135) involved impact to the front of the helmet (0–90 degrees, Conditions A and B), including 34% (46 out of 135) to the facemask and 27% (36 out of 135) to the shell of the struck player's helmet (29). The remaining cases (53 out of 135, 39%) involved impact to the back of the helmet (Conditions C and D). For these, 59% (31 out of 53) involved helmet-to-helmet and 42% (22 out of 53) involved helmet-to-ground impacts. Virtually all of the striking players use the front crown or top of their helmet with their head down. This lines up their head, neck, and torso in the collision (37). Deviations from this alignment in game impacts and laboratory tests caused the helmets to slide off as a result of the smooth plastic shell of the helmets. For this study, emphasis was placed on impacts to the front of the helmet because of the high proportion of this type of impact in NFL concussion.

MATERIALS AND METHODS

Video Analysis of NFL Game Impacts

Details of the game film selection and analysis have been reported by Pellman et al. (28, 29) and Viano and Pellman (37). For this study, a short overview of the laboratory methods is provided. When a concussion occurred on the field during an NFL game, it was reported to Biokinetics and Associates, Ltd. (Ottawa, Canada); Biokinetics

reviewed 182 game impact videos from 1996 to 2001 and found 174 collisions that could be initially analyzed (28, 29).

Biokinetics determined the feasibility of determining the three-dimensional (3-D) impact velocity, orientation, and helmet kinematics. At least two clear views of the collision were necessary to make this analysis. Ground markings were used to determine a dimensional scale factor. A laboratory setup was made with crash dummies to reenact the game impact when a 3-D impact velocity could be determined. Helmets were placed on the dummies and the velocity, orientation and helmet kinematics were matched to the game video.

Thirty-one NFL collisions involving helmet-to-helmet and helmet-to-ground impacts were reconstructed involving 25 concussions of struck players. For this analysis, concussion cases were divided into impacts to the front or back of the helmet. Nineteen concussions occurred by impacts to the front of the helmet. Seventy-six percent (19 out of 25) of the helmet-to-helmet impacts occurred in Conditions A (0–45 degrees) or B (45–90 degrees) (29). The remaining cases involved three impacts with the ground and three helmet-to-helmet impacts for Conditions C (90–135 degrees) and D (135–180 degrees). This is slightly higher than the 61% of helmet-to-helmet or helmet-to-ground impacts from NFL game video analysis.

Laboratory Reconstruction of NFL Game Impacts:

Figure 1 shows a sequence from an NFL game video of a collision resulting in concussion to the struck player. The struck player's head is pushed to the right in the direction of the striking player's movement. The struck player's head also rotates about the z axis (superior-inferior axis) and bends laterally about the *x* axis (anterior-posterior axis).

Figure 1 also shows the helmet kinematics from the laboratory reconstruction. The impact initially involves compression of the helmet liners as force increases. The struck player's head is pushed to the right and the neck deforms. In this case, the head rotates about the z axis as the eyes turn to the right as the neck twists. The neck also bends laterally to the right as in the game video. Late in the collision, the helmets slide relative to each other and the striking player's helmet interacts with the carriage.

The laboratory reconstruction usually involved two Hybrid III male dummies following methods already described (28, 29, 37). A helmeted head-neck assembly representing the struck player was attached to a 7.1 kg mass simulating the struck player's torso and guided in freefall from a height to match the impact velocity determined from video analysis of the game collision. The Hybrid III head and neck weighed 4.38 kg with instrumentation. The helmet and facemask weighed 1.92 kg and the falling mass was 15.1 kg. Impact was against another helmeted head-neck assembly attached to the torso and pelvis of the Hybrid III dummy. This dummy weighed 46.4 kg without arms and legs and was suspended by flexible cables. Ground (turf) impacts were simulated in three of the tests by placing a segment of artificial turf on a flat plate under the falling helmeted head.

Acceleration was measured at the center of gravity (cg) in both dummy heads. As the head cg moves in space under translational acceleration, it can also rotate about the head cg. Each headform was equipped with nine linear accelerometers setup in a so-called "3–2–2–2 configuration" to determine rotational acceleration (28).

In the initial tests (28, 29), the dummy representing the striking player had a six-axis upper neck transducer measuring three axes of neck force (Fz, compression-tension; Fx, fore-aft shear; Fy, left-right shear) and three axes of neck moment (My, flexion-extension; Mx, lateral bending; Mz, rotation about the z axis). In these tests, the six-axis upper neck transducer was used in five of the struck players to provide more complete biomechanical data on the head-neck response during concussion. The data was merged for analysis assuming similarity in the repeat tests.

The sign convention had neck compression − Fz and neck tension + Fz because the positive z axis is from the base of the neck up through the top of the head (34). The positive *x* axis is forward and the positive *y* axis is through the left ear. Neck extension is −My; flexion is +My. The procedure described by Viano and Pellman (37) was used to align time zero for the reconstructed cases because the orientation and timing of the impact varied between test setups. A 1 *g* trigger was typically used to determine the start of the impact, although a 3 *g* trigger was used in Tests 7, 38, 39, 48, 59, 69, 84, and 92, and a 5 *g* trigger was used in Test 77 owing to noise in the acceleration (37).

High-speed video recorded head kinematics in the reconstruction using an orientation that was similar to one of the views from the game video. This allowed a one-to-one comparison of the game and reconstruction kinematics (see *Fig. 1*), and facilitated fine adjustments in the impact orientation and alignment in the laboratory to match game kinematics. A study was conducted to quantify sources of error and variability in the reconstructions (27). That work showed the tests to be repeatable and with minimal error. In the laboratory reconstructions, every effort was made to reduce potential sources of error.

Collision Biomechanics

Impact force (F) from the striking player is the sum of a head inertia force and neck compression acting in the collision:

$$F = m_{Striking}a_{Striking}+F_N \quad (1)$$

where $a_{Striking}$ is the resultant acceleration of the striking player's head, F_N is the resultant neck compression force and $m_{Striking}$ is the mass of the striking player's head above the neck load cell (37). Mass was $m_{Striking}$ = 5.90 kg and included the Hybrid III head (3.64 kg), load cell above the sensing element (0.34 kg), and helmet with facemask (1.92 kg). Mass below the sensing element is not included in Equation 1 because the striking player's neck load was measured. In the collision, the striking player used the top or crown portion of the helmet. This area is substantially stiffer than the side or facemask region of the helmet but involves a padded liner. Impact compresses the liners of both helmets as force increases.

Impact force from the striking player is equilibrated by acceleration of the struck player:

$$F = m_{Struck}a_{Struck} \quad (2)$$

where a_{Struck} is the resultant acceleration of the struck player's head. Mass of the struck player is m_{Struck} = 8.40 kg and includes the head (4.38 kg), neck (1.06 kg), helmet with facemask (1.92 kg), and a portion of the torso mass (1.04 kg). The resultant accelerations are assumed collinear for this analysis, and the masses were determined to equilibrate the peak impact force.

Head acceleration of the striking player is lower than that of the struck player because the effective mass of the striking player is greater than that of the struck player. The neck load cell in the striking player measures the contribution from the torso mass in the collision, which adds to the impact force. The effective mass of the striking player is:

$$m_{Eff.\ Striking} = F/a_{Striking} \quad (3)$$

where $m_{Eff.\ Striking}$ = 14.0 kg based on average head acceleration and impact force. This gives a mass ratio of $m_{Eff.\ Striking}/m_{Struck}$ = 1.67 or a 67% greater effective mass of the striking player than that of the struck player during peak force. The impact force, head acceleration and head ΔV describe the initial collision mechanics resulting in concussion of the struck player.

Impact causes a rapid increase in head velocity, which displaces and rotates the head after the primary impact force. As the head displaces, the struck player's neck develops load and the analysis of equilibrium becomes more complicated because head rotation changes the orientation of the x, y, and z axes with respect to an inertial frame of reference. This causes the axis system in one dummy head to continually change with respect to the orientation in the other head. The use of resultant values assumes the responses are collinear.

Modeling the Helmet-to-helmet Reconstruction Tests

In helmet-to-helmet collisions, the striking player lines up their head, neck, and torso to deliver force to the helmet of the struck player. The collision involves an initial velocity of the striking player $V_{striking}$ (0), who has an effective mass $m_{Eff.Striking}$ of 14.0 kg based on the NFL reconstruction tests (37). This mass includes the head, helmet, and added mass of the torso of the struck player through neck compression.

As the collision proceeds, the helmet padding and shells deform. Impact force (F) develops in proportion to the stiffness (k_H) of the interaction between the helmets:

$$F = k_H d_H \quad (4)$$

where d_H is the relative displacement between the heads of the striking and struck player. Based on modeling the reconstruction tests, k_H is 175 N/mm. Impact force decelerates the striking player and accelerates the head of the struck player, which is assumed to accelerate in the direction of impact. Deceleration of the striking player is:

$$a_{Striking} = F/m_{Eff.\ Striking} \quad (5)$$

Acceleration of the struck player is reduced by forces developed in the neck. Force in the struck player's neck (F_N) occurs as the head is moved by the follow-through of the striking player. Initially, it is shear force but as the head displaces and rotates, the neck force becomes primarily axial tension with the added effects of bending and rotation. Acceleration of the struck player's head is:

$$a_{Struck} = (F - F_N)/m_{Struck} \quad (6)$$

Force in the neck is proportional to the relative displacement between the struck player's head and torso (d_N). This includes the cumulative effects of neck shear, tension, and bending as the head is displaced and rotated in the collision. Neck stiffness (k_N) is related to anthropometry and muscle tension with the added effects of ligament and tendon stretching at the extremes of the natural range of motion. Neck stiffness is the cumulative effect of 3-D forces and displacements. Force in the neck is:

$$F_N = k_N d_N \quad (7)$$

Viano (36) found that the neck stiffness in extension was 5 N/mm for relaxed neck muscles supporting the head in an upright position subjected to inertial (whiplash) loading without direct contact on the head. For the forced response here, the effective neck stiffness is much greater. Based on four NFL reconstructions in which head displacement and neck force were measured, the effective stiffness of the Hybrid III dummy neck was k_N = 80 N/mm. This is based on a resultant neck force of 1548 ± 735 N and displacement of 18.0 ± 4.1 mm at 10 milliseconds.

Force in the struck player's neck accelerates the torso (a_{Torso}):

$$a_{Torso} = F_N/m_{Torso} \quad (8)$$

where the torso mass (m_{Torso}) is 6.06 kg for the reconstruction tests of Pellman et al. (28, 29, 32) and Viano and Pellman (37). This was determined by subtracting the 1.04 kg mass involved in the head acceleration from the 7.1 kg carriage attached to the guidewires of the drop fixture. Initial testing showed that the carriage mass did not influence the initial phase of high impact force. The torso mass does, however, influence head displacement as neck loads increase and the torso mass is accelerated. Neck strength is a factor by coupling the struck player's torso into the impact. A stronger, stiffer neck reduces head acceleration in the struck player according to Equation 6. Forces due to neck and helmet padding deformation were reduced during unloading by assuming a coefficient of restitution of 0.45.

A step-forward solution to the equations of motion was developed in Microsoft Excel (Microsoft Corp., Redmond, WA) to simulate the player's responses in NFL reconstruction tests. An impact speed of 9.7 m/s was used because it is the average of the laboratory reconstructions of concussions for Conditions A and B. A close match was found in the level and timing of the impact force, head acceleration and neck loads. The peak acceleration of the striking player's head was 70.5 g and the struck player was 102.9 g for a peak force of 9700 N (d_H = 55 mm) at 8.2 milliseconds. ΔV was 7.1 m/s for the struck and 5.6 m/s for the striking player. Force developed in the neck reached 4600 N (d_N = 49 mm) at 18.6 ms. The model was considered validated with the NFL reconstruction tests by matching the essential timing and amplitude of head impact responses.

Influence of Neck Strength (Stiffness)

The model was used to consider the influence of various parameters on the relative response of the head. Particularly, neck strength and its effect on head responses was investigated in the helmet collision by varying the neck stiffness while keeping the other conditions constant in the model, except the torso mass was increased to 17.2 kg to match the upper torso mass of the adult (19).

For this analysis, differences in neck strength (stiffness) for the 10-year-old, 5% female, 95% male Hybrid III dummies were determined from the literature (9, 19, 20, 22, 42) and used to adjust the 80 N/mm found for the 50% male Hybrid III in the reconstruction tests. This gave 28 N/mm for the 10-year-old, 39 N/mm for the 5% female, and 113 N/mm for the 95% male Hybrid III dummies. Because these values are calculated for a relaxed neck that is able to maintain the head upright, higher values of neck stiffness were considered to reflect the greater anthropometry and tensing capability of highly trained athletes. Values up to 240 N/mm were evaluated.

Head Injury Tolerances: Resultant head acceleration is used to calculate the Severity Index (SI) used by the National Operating Committee on a Standards for Athletic Equipment (NOCSAE) in its football helmet standard:

$$SI = \int_0^T a(t)^{2.5} dt \quad (9)$$

where a(t) is the resultant translational acceleration at the head cg and T the duration of the acceleration. NOCSAE limits SI to 1200. The National Highway Traffic Safety Administration (NHTSA) uses the Head Injury Criterion (HIC), which is a variation of SI:

$$HIC = \{(t_2 - t_1)[\int_{t_1}^{t_2} a(t)dt/(t_2 - t_1)^{2.5}\}_{max} \quad (10)$$

where t_1 and t_2 are determined to maximize the HIC function and a(t) is the resultant translational acceleration of the head cg. A limit of 15 milliseconds is typically used. NHTSA limits HIC to 700 for 15 milliseconds duration and 1000 maximum value. The studies of Pellman et al. (28) found the tolerance for NFL concussion was SI = 300 and HIC = 250. Head rotation is a second type of biomechanical response that influences head injury; however, there is no concurrence on tolerance limits.

Neck Injury Tolerances

For the Hybrid III dummy, the upper neck tolerance for axial tension is 4170 N and compression is 4000 N (20). Neck tolerance for shear is 3100 N. The extension bending moment tolerance is 96 Nm and lateral bending is 143 Nm. The tolerance for head rotation is 96 Nm about the z axis (20, 24). Neck injury criterion is used by the National Highway Traffic Safety Administration to evaluate the combined effects of sagittal extension-flexion and tension-compression. Neck injury criterion is not used here because the predominant modes of neck response are lateral and about the z axis in the NFL reconstructions of the struck player.

Statistical Analyses

The significance of differences in responses for the concussed player was determined using the *t* test with unequal variance and a two-sided distribution. The *t* test was performed using Microsoft Excel.

RESULTS

Biomechanics of the Struck, Concussed Player

Figure 3 shows biomechanical responses for the concussed player (Case 38) from a reconstructed NFL game impact. The collision and reconstruction are shown in *Figure 1*. It involves an impact to the side of the helmet and some aspects of the case have been previously described by Viano and Pellman (37).

The helmet collision develops impact force reaching 9780 N (2198 lb) at 8 ms and extends over a duration of approximately 15 ms. The force occurs as the striking player lines up his head, neck, and torso, coupling mass from his body into the impact. The striking player continues forward, driving through the other player.

Because the collision is oblique to the side of the helmet, the primary acceleration is lateral (*y* direction) to the struck player's head. The resultant acceleration reaches 106 *g* in 6 ms and then decreases. The impact also causes head rotational acceleration about the z and *x* axes. The rotational accelerations turn (twist) the head to the right around the superior-inferior axis (z axis) and laterally bend the neck to the right about the *x* axis.

Head acceleration causes a rapid increase in lateral and rotational velocity. By 10 ms, the lateral velocity has reached 6.7 m/s. By 20 ms, the *y* axis velocity has reached 8.8 m/s and the head has been displaced 104 mm laterally to the right. The head has also rotated 25.6 degrees around the z axis and 17.6 degrees around the *x* axis, resulting in a 40.1-degree change in the resultant head angle.

Head displacement and rotation load the neck. Lateral shear force develops during the impact; and, with head displacement to the right, the neck is put into axial tension. Neck tension increases to 2420 N (544 lb) at 21 ms as the head moves to the right and the neck bends. The combination involves z axis rotation to the right and lateral neck bending about the *x* axis.

There is minimal bending moment resisting the z axis rotation of the Hybrid III head, although the Mz moment continues to rise over 30 ms. The primary bending resistance is lateral, reaching 62 Nm at 14 ms. After the peak head impact force and during the phase of rapid head displacement, the neck is deformed, building up forces and moments that couple the torso mass of the struck player into the final aspects of the collision. Neck loads alter head kinematics and influence the early and late responses of the head.

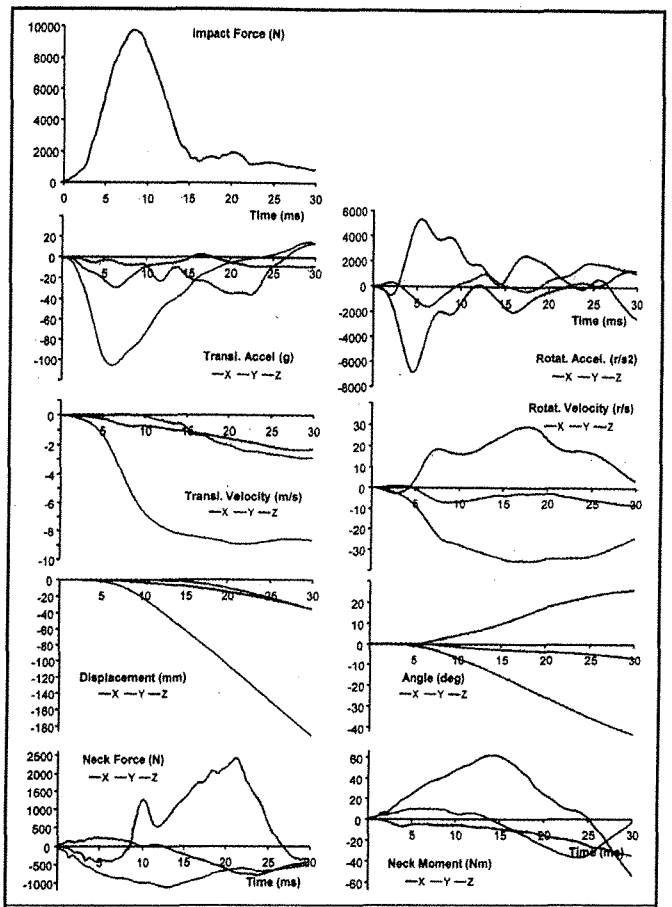

FIGURE 3. *Line graphs showing the biomechanical responses from the reconstruction of the struck NFL player (Case 38), including the impact force of the helmet collision, head accelerations, velocities and displacements, and neck forces and moments for the concussed player.*

Twenty-five Reconstructed NFL Collisions

Table 1 summarizes the peak head accelerations for the struck and striking players involved in impacts causing concussion. The average impact velocity was 9.3 ± 1.9 m/s. Three cases involved falls to the ground and did not include a striking player. Five cases were significant helmet impacts but did not result in concussion. The peak head acceleration for the struck player was 94 ± 28 *g* and 6432 ± 1813 r/s^2, resulting in a change in velocity of 7.2 ± 1.8 m/s and 34.8 ± 15.2 r/s. The data provided in *Table 1* are resultant values. HIC was 381 ± 197 (SI, 436 ± 242) for the struck player.

Impact Conditions A and B involve loading to the facemask and helmet shell within ± 90 degrees of eyes straight forward. This constitutes the front of the helmet. These impacts are forward of the head cg and the z axis of the neck. NFL game

TABLE 1. Peak biomechanical responses for the struck and striking National Football League players for 25 impacts causing concussion[a]

Case no.	Condition	Impact		Struck players				Striking, uninjured players				
		Velocity (m/s)	Force (N)	HIC	Peak translational accel (g)	Peak ΔV (m/s)	Peak rotation accel (r/s²)	Peak rotation velocity (r/s)	HIC	Peak translational accel (g)	Peak ΔV (m/s)	Peak rotation accel (r/s²)	Peak rotation velocity (r/s)
69	A	10.3	4796	153	61	5.0	4381	19.9	50	38	3.1	2620	23.0
77	A	9.9	5612	185	80	5.2	5148	36.4	53	35	4.2	2714	25.5
84	A	9.4	6431	222	82	6.3	9193	80.9	78	45	4.4	3169	26.5
113	A	7.0	6323	140	59	5.1	3965	12.8	75	61	3.7	3700	31.2
118	A	10.7	8987	378	101	9.6	7017	42.9	73	56	3.7	3687	23.4
124	A	11.4	7959	282	81	7.5	7138	34.8	73	56	3.1	4086	16.1
148	A	6.6	4065	99	48	5.1	3476	23.9	37	33	3.9	2466	26.5
157	A	10.8	9568	472	103	8.1	6750	33.5	180	79	5.0	4662	15.7
181	A	11.7	6877	382	93	7.1	8011	36.5	333	85	7.3	6613	55.8
9	B	10.3	11680	600	134	10.1	7428	27.4	217	79	2.3	6719	18.7
38	B	9.5	9776	554	118	9.7	9678	50.8	127	60	4.0	5205	28.2
39	B	10.9	7889	522	129	8.4	5921	36.1	43	44	2.3	4487	10.4
57	B	8.8	5333	206	77	6.0	6514	37.0	38	32	4.1	4151	33.2
71	B	10.3	8258	510	123	7.3	5400	35.0	434	102	6.6	5541	32.4
98	B	9.6	7953	301	91	6.2	7548	43.4	187	84	4.8	4487	38.5
125	B	11.7	9015	633	113	9.1	7716	63.3	111	47	4.2	3366	28.1
135	B	10.0	11490	566	138	8.6	7540	41.0	179	81	3.8	5005	29.3
155	B	9.1	6247	341	100	6.6	6940	37.0	61	45	4.2	4217	29.5
162	B	5.5	4505	77	52	4.2	2615	18.4	30	29	3.2	1672	17.2
7	C	6.9	6030	93	61	4.6	6266	28.1	51	50	2.2	2832	9.8
67	C	8.1		632	135	8.0	5957	13.8					
92	C	11.1	11510	508	107	10.0	6878	44.2	164	60	5.6	6070	43.8
164	C	10.8	7872	370	124	6.0	9590	26.6	202	89	5.1	6136	30.8
123	D	6.3		730	121	8.3	4727	30.3					
133	D	6.0		557	113	8.4	5012	16.0					
Average		9.3	7642	381	98	7.2	6432	34.8	127	58.5	4.1	4255	27.0
SD		1.9	2259	197	28	1.8	1813	15.2	104	21.4	1.3	1405	10.6

[a] HIC, head injury criterion; accel, acceleration; SD, standard deviation. Cases without a striking player involve helmet impacts to the turf. Cases 48, 59, 154, 175, and 182 were severe impacts that did not result in concussion.

impacts involve the striking player hitting either the right or the left side of the opponent's helmet. This analysis, however, assumes that all impacts are from the right. For impact Conditions C and D, the struck player experiences the highest translational accelerations as the load is closer to the head cg.

Table 2 demonstrates the average translational head responses at 10 and 20 ms for the struck player in Conditions A to D, including head acceleration, velocity, and displacement. *Table 3* provides similar averages for rotational kinematics. The average was determined using the absolute value of the responses; however, a negative sign is included in the tables when the majority of the responses had the same response direction. When the sign was a mix of positive and negative values, a positive average is shown.

Tables 2 and *3* show a fourfold increase in head displacement and rotation between 10 and 20 ms. For the B impacts, head acceleration dropped from peak values approaching 100 *g* to 19.8 ± 10.3 *g* at 20 ms, and the struck player's head has reached 8.3 ± 2.0 m/s ΔV. The resultant head displacement increased from 22.1 ± 5.6 mm at 10 ms to 97.7 ± 20.7 mm at 20 ms; head rotation increased from 7.8 ± 1.9 degrees to 33.8 ± 6.1 degrees. The primary rotation is about the z axis for impacts to the front of the helmet (Conditions A and B) because the impact is forward of the head cg and z axis of the neck.

Figure 4 shows the relatively large z axis rotations of the head for impacts to the front of the helmet. At 20 ms, *x* axis rotation was greater than 15 degrees in 18 (95%) of the 19 reconstructions for Conditions A and B and greater than 25 degrees in 15 (79%) cases. The data also show the importance of lateral displacement of the head with concussion. At 10 ms, the average translational and rotational displacements are 20.2 ± 6.8 mm and 6.9 ± 2.5 degrees. By 20 ms, displacements have increased

TABLE 2. Average translational responses of the struck player's head at 10 and 20 ms with concussion[a]

	Acceleration (g)				Velocity (m/s)				Displacement (mm)			
	A_x	A_y	A_z	A_r	V_x	V_y	V_z	V_r	D_x	D_y	D_z	D_r
Condition A, 10 ms												
Average	−37.8	42.0	4.2	60.2	−3.1	2.9	−0.4	4.4	−11.7	10.3	−1.8	16.2
SD	19.1	13.5	3.6	9.8	1.3	0.9	0.3	1.2	5.2	4.8	1.3	6.3
Condition A, 20 ms												
Average	4.8	5.9	−10.8	14.9	−4.5	4.0	−0.7	6.9	−50.7	46.4	−6.0	73.8
SD	5.1	5.7	7.8	8.0	2.0	1.0	0.3	1.5	21.2	11.0	4.2	14.6
Condition B, 10 ms												
Average	−15.7	49.5	−6.1	54.0	−1.7	5.3	−0.7	5.8	−6.9	20.1	−2.9	22.1
SD	15.8	18.9	7.6	21.6	0.7	1.4	0.8	1.2	2.3	6.3	3.3	5.6
Condition B, 20 ms												
Average	5.6	7.6	−15.3	19.8	−2.3	6.7	−1.7	8.3	−27.9	84.0	−13.3	94.7
SD	6.0	5.1	11.0	10.3	1.5	1.8	1.2	2.0	13.4	22.0	13.1	20.7
Condition C, 10 ms												
Average	4.3	36.2	−10.4	39.8	0.8	5.0	−1.7	5.5	3.5	20.8	−7.0	22.9
SD	5.9	26.6	7.7	24.6	0.5	1.5	1.2	1.6	1.0	7.8	4.7	7.9
Condition C, 20 m												
Average	3.5	6.6	−22.1	24.0	0.7	6.4	−2.4	7.7	11.0	81.0	−26.9	90.6
SD	3.5	5.6	14.2	14.3	0.7	2.0	1.7	2.3	7.0	20.7	20.1	25.3
Condition D, 10 ms												
Average	55.8	31.3	−32.8	73.0	5.2	2.5	−3.0	6.7	18.1	8.4	−11.3	24.0
SD	20.1	6.5	29.0	31.2	1.7	0.9	0.4	1.3	10.6	4.7	0.2	9.0
Condition D, 20 ms												
Average	−8.6	6.1	−8.8	14.3	6.0	3.2	−4.6	9.4	78.7	39.7	−50.3	107.7
SD	11.5	3.9	6.3	12.5	0.1	0.2	0.5	1.1	18.1	10.2	8.9	15.7

[a] SD, standard deviation.

fourfold to 87.6 ± 21.2 mm and 29.9 ± 9.5 degrees. *Figure 5* shows the resultant displacement and rotation for the individual cases at 10 and 20 ms. The responses are clustered below 35 mm and 15 degrees at 10 ms but significantly increase by 20 ms.

For impacts to the back of the helmet, the head experiences a high ΔV, particularly with rebound off the ground. For Condition D, ΔV was 9.4 ± 1.1 m/s at 20 ms and head displacement was 107.7 ± 15.7 mm. The head experiences rotations primarily about the *z* axis for Condition C and about the *y* and *z* axes for Condition D.

Figure 6 shows the neck force and moment measured in four struck players. The largest force is neck tension, which reaches its maximum at around 20 ms. The largest neck moments are about the *x* axis. They reach maxima after the primary impact force and are consistent with the lateral bending of the neck seen in *Figure 1*. The moment about the *z* axis increases steadily through 40 ms and is associated with increasing head rotation angles. At 20 ms, the twist moment around the *z* axis averaged 17.7 ± 3.3 Nm and neck tension was 1704 ± 432 N for the four tests with the struck player having the upper-neck load cell.

Geometry of the Helmeted, Hybrid III Head and Neck

Figure 7 is a drawing of a helmet on a player with axes through the cg of the head. The facemask extends approximately 160 mm forward, and the front of the chin is 95 mm forward of the head cg. For Condition A impacts, contact is 120–160 mm forward of the head cg and an additional 50 mm forward of the *z* axis of the neck. For Condition B impacts, the contact ranges from 0 to 120 mm forward of the head cg. Impacts to the back of the helmet (Conditions C and D) are closer to the head cg and *z* axis of the neck. The forward projection of the helmet, chinstrap, and player's face from the *z* axis of the neck and head cg promotes *z* axis rotation for impacts to the front of the helmet.

Depending on the initial axis of impact and subsequent helmet-head motion, the moment arm may be as large as 70 to 140 mm forward of the *z* axis of the neck. *Figure 8* shows the Hybrid III head and neck. The neck axis is leaning forward 5 degrees in the Hybrid III dummy. The centerline of the upper neck bracket is 18 mm rearward of the *z* axis through the head cg; it is 30 mm rearward at the lower neck bracket.

TABLE 3. Average rotational responses of the struck player's head at 10 and 20 ms with concussion[a]

	Rotational acceleration (rad/s²)				Rotational velocity (rad/s)				Angle (degree)			
	α_x	α_y	α_z	α_r	ω_x	ω_y	ω_z	ω_r	θ_x	θ_y	θ_z	θ_r
Condition A, 10 ms												
Average	1322	1419	2155	3266	6.2	4.7	19.7	26.2	1.2	1.1	4.6	6.0
SD	1169	1436	1639	1888	5.1	4.5	14.2	14.2	1.5	0.7	3.2	3.2
Condition A, 20 ms												
Average	857	−693	−939	1645	12.6	−13.5	23.7	47.8	5.7	−3.9	17.8	27.0
SD	540	468	745	619	6.7	6.9	31.6	22.1	4.4	3.2	13.7	13.2
Condition B, 10 ms												
Average	1794	427	−2007	3149	14.1	−4.7	20.8	32.8	3.4	−1.0	4.8	7.8
SD	1631	321	1580	1580	7.8	3.7	11.5	7.4	2.1	0.8	2.5	1.9
Condition B, 20 ms												
Average	1514	583	976	2248	21.1	−6.8	25.9	54.7	14.3	−4.2	17.4	33.8
SD	1510	466	1235	1546	9.7	4.4	14.3	12.0	8.1	2.9	12.5	6.1
Condition C, 10 ms												
Average	1904	1143	−472	2354	16.6	5.4	11.8	29.0	4.2	1.4	3.8	7.6
SD	513	528	430	457	12.7	3.7	8.4	6.2	2.9	1.0	3.0	2.4
Condition C, 20 ms												
Average	1177	−754	406	1537	21.6	3.6	8.0	46.9	15.9	4.3	8.9	29.8
SD	835	454	282	810	15.4	2.1	5.6	4.5	11.2	1.1	7.8	5.2
Condition D, 10 ms												
Average	−607	1486	477	1707	2.3	8.3	−11.6	21.8	0.6	1.4	−3.4	5.0
SD	762	729	249	975	3.2	3.8	4.7	3.4	0.4	0.8	0.2	0.9
Condition D, 20 ms												
Average	556	867	607	1212	9.4	18.3	−5.0	40.8	4.0	7.5	−8.0	23.2
SD	276	850	452	961	7.0	20.8	6.6	12.8	3.6	8.2	2.6	4.9

[a] SD, standard deviation.

Concussion in NFL Players

Table 4 shows the most common signs and symptoms of concussion in NFL players during the 1996 to 2001 seasons (30). The most common symptom is a headache (55.0%), followed by dizziness (41.8%), and then a series of cognitive and memory problems. Neck pain occurs in 12.6% of concussions. This sample included 787 reported concussions in 3826 team games (1913 games) during preseason, regular, and playoff games. The annual incidence of concussion was 131.2 ± 26.8 per year, or 0.41 per game, in this sample.

The reconstruction data was analyzed for impact responses causing concussion (37). Peak head acceleration was 94.3 ± 27.5 *g* with concussion and 67.9 ± 14.5 *g* without (t = 3.02; df = 12; *P* = 0.005). The peak impact force averaged 7642 ± 2259 N with concussion and 5209 ± 1774 N without injury (t = 2.62; df = 7; *P* = 0.017). The head ΔV was 7.08 ± 1.88 m/s with concussion and 5.38 ± 0.48 m/s without (t = 3.75; df = 24; *P* = 0.0005). These differences are statistically significant. Similar response comparisons have not been made for head displacement and rotation.

Relationships for Concussion Risks

Chou and Nyquist (3) assumed a half-sine head acceleration and determined various relationships for head impacts and HIC. For example, Equation 10 simplifies to:

$$HIC = 0.4146A_p^{2.5}T \quad (11)$$

where A_p is the peak head acceleration (in *g*) and T is the duration of the half-sine pulse (in s). HIC can also be related to head impact variables, such as the head ΔV (in m/s) and peak acceleration A_p:

$$HIC = 0.0132\Delta VA_p^{1.5} \quad (12)$$

They also determined that displacement of the head is:

$$d = 0.1246\Delta V^2/A_p \quad (13)$$

where d (in m) is head displacement at the end of the half-sine acceleration. One relationship they did not determine can be found by combining Equations 12 and 13. This gives a relationship between HIC, head ΔV and head displacement (d):

$$HIC = 0.0058\Delta V^4/d^{1.5} \quad (14)$$

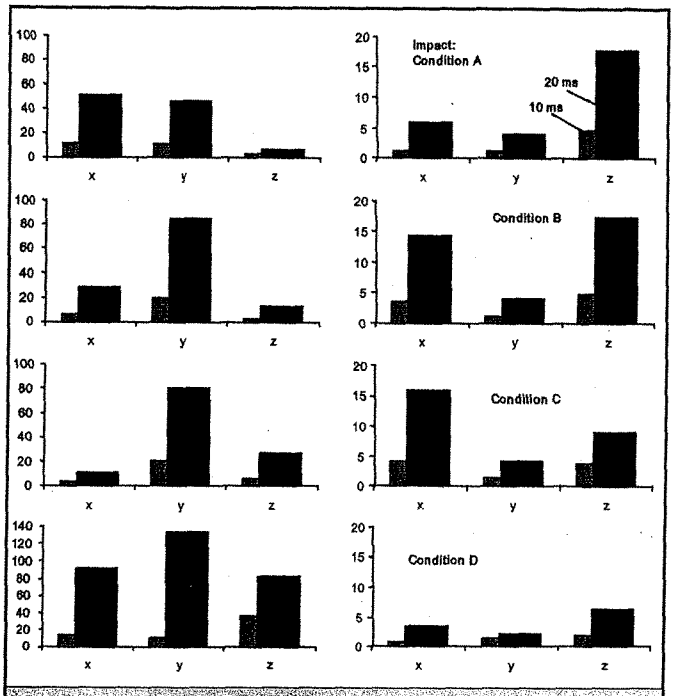

FIGURE 4. *Bar graphs showing the resultant head displacement and rotation at 10 and 20 ms for the struck player. The average responses are shown for impacts to the helmet (Conditions A–D). The x axis is the anterior-posterior direction, the y axis is the lateral direction, and the z axis is the superior-inferior direction. Rotation about the x axis is lateral bending, rotation around the y axis is flexion-extension, and rotation around the z axis is head rotation where the eyes move right or left.*

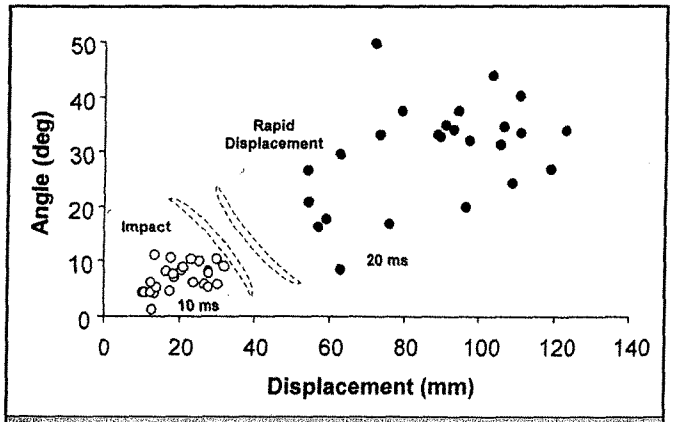

FIGURE 5. *Resultant displacement and rotation of the head for NFL concussions averaging 20.2 ± 6.8 mm and 6.9 ± 2.5 degrees at 10 ms, and 87.6 ± 21.2 mm and 29.9 ± 9.5 degrees at 20 ms.*

HIC is proportional to the fourth power of the head ΔV and inversely related to head displacement to the 1.5 power.

Kinetic energy (E) transferred to the head of the struck player is related to head ΔV:

$$E = 0.5 m_{Struck} \Delta V^2 \quad (15)$$

so HIC can be considered proportional to the square of the kinetic energy transferred to the struck player's head in a collision divided by head displacement to the 1.5 power. Equation 14 is helpful in considering relationships and influences on head injury, including the influence of neck strength on concussion risk.

Concussion is correlated with HIC (28). Equation 14 indicates that HIC are related to ΔV^4. This means that a 10% reduction in head ΔV gives a 34% reduction in HIC assuming a constant head displacement (d). ΔV has a significant influence on concussion risk. Equation 14 also shows the influence on increased head displacement (i.e., padding thickness on the side of the helmet). A 10% increase in head displacement reduces HIC 15%. The benefit of head displacement increases by the 1.5 power. However, head ΔV and d are interdependent and the actual benefits of head displacement are less because of the coupling of the parameters.

Influence of Neck Strength

Figure 9 shows the results of helmet impact simulations where neck stiffness was varied from 30 to 240 N/mm and all other parameters were held constant, except torso mass. The responses are shown normalized to that of the 50% Hybrid III dummy that was used in the laboratory reconstructions. Data points represent the simulated responses and the line is a curve fit using a power function. Increasing neck stiffness reduces peak head acceleration and ΔV. The effect makes a large difference in HIC because the difference in ΔV is raised to the fourth power. Because HIC is the strongest correlate with concussion in the NFL reconstructions (28), this analysis indicates an important role for neck strength controlling head responses and concussion risk.

For example, an increase in neck tension from 80 to 180 N/mm causes a 14% reduction in head ΔV, but a 46% reduction in ΔV^4 and only a 11% reduction in head displacement at 15 ms. Using Equation 14, the effect on HIC is a 35% reduction. By introducing early forces in the neck, head acceleration and ΔV are lower and HIC is substantially reduced by coupling more of the torso mass into the collision. The weaker necks of females and children cause a disproportionate increase in head ΔV and HIC and would significantly increase the risk of concussion. The ΔV^4 response of the 10-year-old boy and 5% female are about twice that of the 50% male.

DISCUSSION

Role of the Neck in Concussion

This study has shown that neck force influences head rotation and displacement and may play a role in the development of brain dysfunction after closed head injury. These results are consistent with the earlier observations of Denny-Brown and Russell (4) that concussion in animal models depended on movement of the head and neck and that head ΔV was a significant factor in brain injury. They are also consistent with observations from the world of boxing that knockouts and many

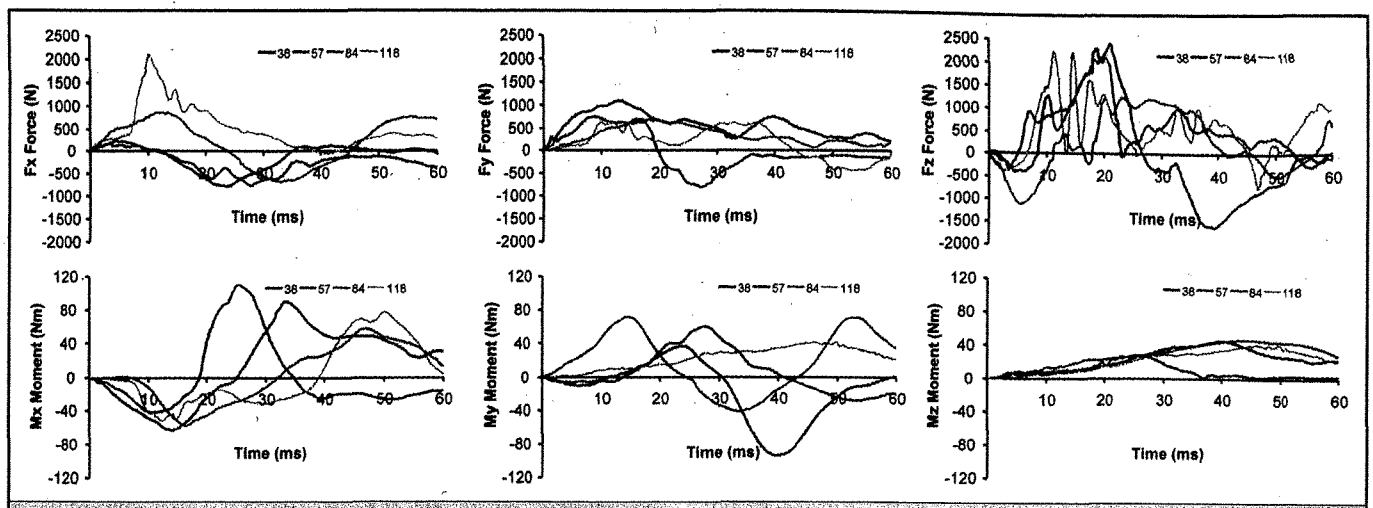

FIGURE 6. *Line graphs showing neck forces and moments measured at the upper neck for four struck players (Cases 38, 57, 84, and 118). The signs of some responses have been reversed so the trends are in the same direction.*

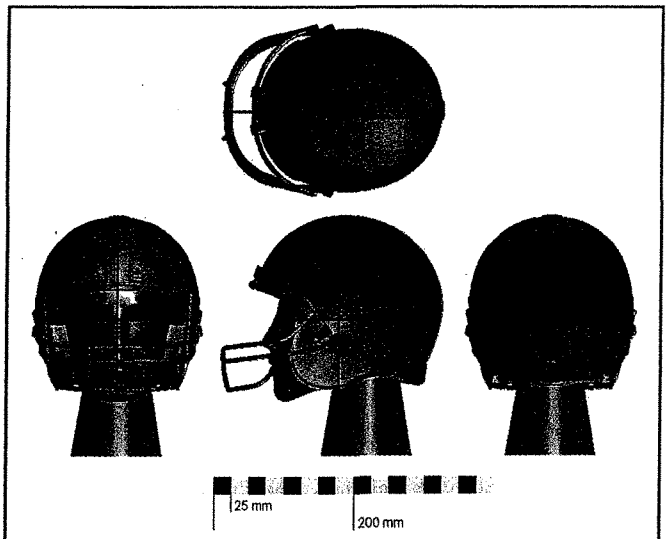

FIGURE 7. *Representation of the head with helmet showing the location of the cg of the player's head and typical dimensions of the facemask and shell.*

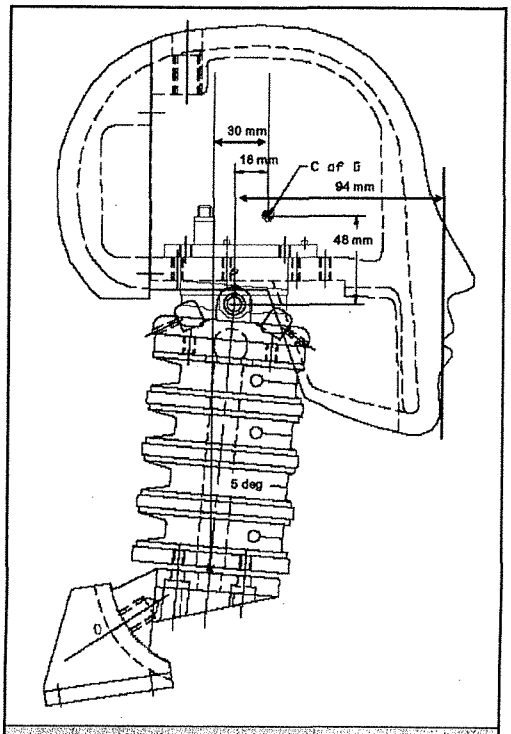

FIGURE 8. *Schematic of the Hybrid III dummy head and neck showing the head cg and offset of the centerline of the upper and lower neck and chin.* Modified from, *Backaitis SH, Mertz HJ (eds): Hybrid III: The First Human-like Crash Test Dummy. Society of Automotive Engineers, PT-44, Warrendale, 1994 (1).*

serious brain injuries occur as a result of diminished tone of the neck muscles (35). A series of blows to the head may result in a "groggy state" in which "the muscle tone, most obviously that of the neck, is reduced, and the head moves like a pendulum under the blows. It sustains, therefore higher accelerations and, consequently, more severe effects" (35).

The findings of the present study are also consistent with the authors' previous observations using finite element models that the highest strains and strain rates in the brain occur more than 10 ms after the high impact forces and that the greatest strains and strain rates were seen in deep midline structures, such as the midbrain, which are near the head cg (38, 39). Late effects are influenced by not only the early neck forces but rapid head displacements and ΔV of the collision.

Much of the clinical symptomatology of concussion is related, at least in part, to dysfunctions in deep midline brain

TABLE 4. Initial signs and symptoms for 787 reported concussions in National Football League games from 1996 to 2001[a]

Signs and symptoms (n = 2166)	No.	Percentage	95% confidence interval
Headache	433	55.0	(51.5%, 58.5%)
Dizziness	329	41.8	(38.4%, 45.2%)
Immediate recall	201	25.5	(22.5%, 28.5%)
RGA delayed	142	18.0	(15.3%, 20.7%)
Problems processing information	138	17.5	(14.8%, 20.2%)
Blurred vision	128	16.3	(13.7%, 18.9%)
Attention problems	102	13.0	(10.7%, 15.3%)
Neck pain	99	12.6	(10.3%, 14.9%)
AGA delayed	74	9.4	(7.4%, 11.4%)
Unconsciousness[b]	58	9.3	(7.0%, 11.6%)
Fatigue	71	9.0	(7.0%, 11.0%)

[a] RGA, retrograde amnesia; AGA, anterograde amnesia.
[b] Based on 623 reported cases.

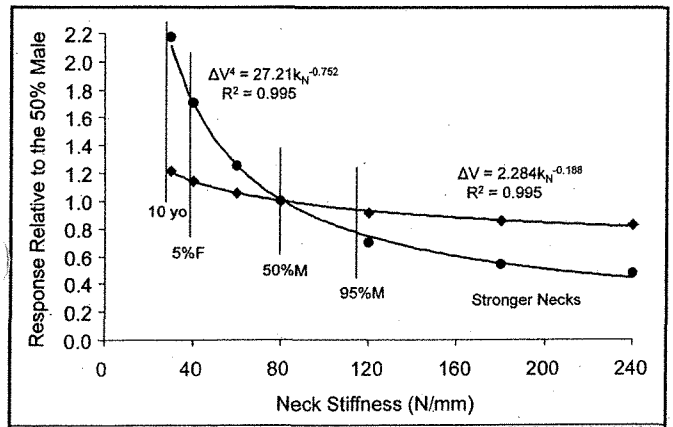

FIGURE 9. *Line graph showing the response of the head relative to the average male (50% male Hybrid III), including the head ΔV and ΔV⁴, which is proportional to HIC (see Equation 14). Data on neck strength are taken from the literature and are identified for various sized people with normal neck tension to maintain the head upright. Stronger necks represent maximum tension for well-conditioned athletes.*

structures. In a previous study (38), finite element modeling was used to demonstrate correlations between late occurring shear strain in deep midline structures such as the midbrain, the fornix and the corpus callosum to concussion-related cognitive and memory dysfunction, loss of consciousness and delayed return to play. Many of the frequently seen symptoms and/or signs of concussion in NFL players are included in these categories, including dysfunction of immediate recall, retrograde amnesia, difficulties with information processing, difficulties with attention, anterograde amnesia, and loss of consciousness.

The most common symptom, headache, did not correlate with FE modeling results. Although headache is a nonspecific symptom in terms of localization, it is not unreasonable to speculate that the brainstem region may serve as a generator for some types of migraine-like headaches after concussion. It is also not unreasonable to suggest that two other common symptoms after concussion, namely dizziness and blurred vision, which did not correlate with late high strains in the deep midline structures on FE modeling, may be at least partially the result of brainstem dysfunction. It is, therefore, possible that lessening the late high shear forces in the deep midline brain structures might also lessen the pattern and severity of the clinical syndrome of concussion. These strains occur late, after the high forces of the helmet collision, during the period of time during which the neck is being deformed by head displacement.

These findings may help to explain some of the clinical phenomena of concussion in sports. A number of studies have suggested that younger athletes (children and adolescents) have a higher susceptibility to concussion and a more delayed recovery from concussion than those in their 20s and 30s (5, 15–18, 23, 33, 41). The reasons for these differences are not clear, although some type of increased susceptibility to trauma in the developing brain seems like a plausible explanation.

The results of this present study suggest that the answer may, in part, lie in the relatively weaker, developing neck musculature of the younger athlete. High school and college football players have weaker neck muscles than professional football players based on scaling in various publications (7, 13, 19–22). As a result, they may exhibit higher head displacements than their professional counterparts. This could result in higher strains and strain rates in the midline brain structures occurring late in the collision sequence. This could also explain why head impacts resulting from velocities and accelerations of lower magnitude have a more deleterious effect upon the brains of younger athletes than those of higher velocities and accelerations seen in the professional game upon those more mature brains (*Fig. 9*). As a result of their relatively weaker neck muscles, younger athletes may develop higher ΔV, which

results in a magnified increase in HIC and, thus, an increased risk of clinical concussion.

If a crucial difference is between the strength of neck musculature and not between the levels of brain development, then it follows that increasing the strength of those neck muscles might diminish the frequency and severity of concussion in younger athletes. In fact, our model shows that increasing the strength of the neck muscles diminishes head ΔV, resulting in a significant lowering of the HIC because of the powerful mathematical relationship between the two, see Equation 14. This indicates that even relatively small changes in the strength of neck muscles may have significant effects on the forces related to the development of clinical concussion. This effect needs to be verified.

Weight training or other exercise programs aimed at specifically increasing the strength of neck muscles that resist rotation (z axis rotation of the head) might be of special value in this regard. It is our understanding that strength training focuses on neck muscles resisting flexion-extension and to a lesser degree lateral bending. We are unaware of any exercise program to develop muscles resisting head rotation (twist). It also may be possible to engineer protective equipment that limits head displacement and rotation in younger football players, thus lowering their susceptibility to concussion.

In a similar vein, it is possible that the incidence of concussion in female athletes could be lowered if head ΔV and displacement could be lessened. Female athletes generally have weaker neck muscles than males (9, 19–22). Females are, therefore, more likely to experience higher ΔV and displacements after impact than their male counterparts (*Fig. 9*). Exercise and/or weight training programs that specifically increase the strength of neck muscles that resist rotation and head displacement might lower the incidence of concussion in women's sports. A similar benefit for female athletes might also result from protective equipment design changes that limit head movement after impact.

The helmet collision model was used to determine impact velocity as a function of neck stiffness (strength) that gave the same head ΔV as for the 50% male. This resulted in a power fit relationship: $V_{Striking} = 0.436k_N^{0.189}$, $R^2 = 0.995$. The 5% female has a 13% lower impact velocity for an equivalent head ΔV as the 50% male. Head acceleration for the struck player is influenced by forces in the neck. The relationship can be seen in a variation of Equation 6:

$$a_{Struck} = (F - k_N d_N)/m_{Struck} \quad (16)$$

where the neck stiffness (k_N) and head displacement with respect to the torso (d_N) determine force in the neck. Force in the neck resists head displacement and is proportional to the relative displacement between the struck player's head and torso. Becasue k_N and m_{Struck} are smaller in the 5% female than the 50% male, head acceleration will be greater for the same impact force. The greater the strength of the neck, the lower the head acceleration and ΔV for a given helmet impact force.

Based on the present results, one could suggest that a specific individual difference that may account for the variability in susceptibility to concussion is strength of neck musculature, specifically in regard to the ability to resist head rotation and displacement. These differences may be genetic or developmental in origin and could possibly be altered by specific exercise programs. Some players with increased susceptibility (e.g., perhaps those few NFL players who experience multiple concussions) as a result of relative neck musculature weakness might benefit from preventative exercise programs specifically aimed at increasing the strength of the neck muscles that resist rotation and head displacement and/or by custom designed protective equipment aimed at limiting delayed head rotation and displacement.

The authors' previous studies have shown that head impacts exceeding certain velocities and accelerations are correlated with the occurrence of concussion in NFL players (28, 29). The present study shows that head ΔV may be a more important factor in concussion risk than the absolute impact velocities and peak head accelerations. This can help to explain why some NFL players who sustained head impacts that exceeded the velocity-acceleration thresholds did not experience clinical concussion and some who sustained head impacts below those thresholds did develop clinical concussion (28, 29). The authors have tried to explain these cases as being the result of "individual differences" between players, but there may be underlying biomechanical factors influencing concussion risk. This study has quantified some of those factors but has not quantified the effects of differing head rotation because there is no accepted head injury criterion for rotation. It seems logical that the high z axis rotations may be a factor in concussion.

Impacts to the Front of the Helmet

Another interesting finding of this study is that impacts to the facemask resulted in larger head rotations than impacts directly to the helmet shell. Because the facemask juts far out in front of the face, it is significantly further from the head's cg and the z axis of the neck than the helmet shell itself. As a result, impacts to the facemask have a greater moment arm than those to the shell, resulting in significantly higher rotations. This may result in higher forces affecting the midbrain and other deep midline structures, thus increasing the risk of clinical concussion. Similarly, blows directly to the chin or lateral loads through the chinstrap may also increase risk of clinical concussion because the chin protrudes forward of the head cg. This may also explain why boxing blows directly to the chin from a hook often result in knockouts. It may be possible to engineer different facemasks and different chinstraps that limit rotations and head displacements occurring from loads forward of the head cg and z axis of the neck. Such equipment improvements might lessen the incidence and/or severity of concussion in all football players.

Concussion Relationships

Pellman et al. (28) determined an HIC tolerance of 250 for concussion based on the reconstruction of NFL player injuries. Using an HIC of 250 in Equation 14, the allowable head ΔV is related to the distance moved by the head during impact:

$\Delta V = 14.4d^{0.375}$. For a d value of 0.05 m (50 mm), the allowable ΔV is 4.7 m/s. The allowable ΔV increases gradually with head displacement. A 20 mm increase in distance gives a 0.63 m/s increase in head ΔV; this is only a 13% increase. This is a relatively small increase in head ΔV for a large increase in the distance the head is displaced during impact. Head ΔV is the dominant factor in Equation 14. This is consistent with the observation of Denny-Brown and Russell (4) that the rapid change in head velocity (ΔV) is a key factor in brain injury. The most effective means to reduce the risk of concussion may be to reduce head ΔV in the response region of most susceptibility to injury.

Because of the highly nonlinear nature of head ΔV, HIC, and the probability of concussion, a 10 to 20% reduction in head ΔV or HIC may mean anything from virtually no change in concussion risk to something substantial. This depends on the particular value of the biomechanical response in relation to concussion risk. Logist risk functions have been defined for NFL concussions (28) and they are sigmoidal in shape with a region of increasing risk between plateaus at 0 and 100% risk. Any reduction in a biomechanical response needs to be considered in regard to its effect on reducing concussion risk in the transition region between the plateaus of low and high risk. Furthermore, there remains debate over the most meaningful biomechanical parameters associated with head injury (25, 26, 28).

New Football Helmets

By 1980, significant reductions in serious head injuries were observed with the voluntary adoption of National Operating Committee on Standards for Athletic Equipment (NOCSAE) standards by helmet manufacturers. In youth football, a 51% reduction in fatal head injuries, a 35% reduction in concussions, and a 65% reduction in cranial fractures were observed (12). Between 1981 and 1985, there were further reductions in fatal head injuries. Although rule changes were also implemented, most of the reductions were considered to be related to helmet design because neck injury rates increased, indicating that significant head impacts were still occurring. Hodgson and Thomas (11) reported on the performance of pre- and poststandard helmets in NOCSAE tests; 84% of the prestandard helmets exceeded an SI of 1450, whereas the poststandard helmets exhibited an average SI of 1064. Mertz et al. (21) converted the SI values to serious head injury risks and concluded that the NOCSAE standard and helmet design changes had reduced the risk of head injury from 55 to 12%. Cantu and Mueller (2) have summarized reductions in fatal head injuries in football.

In the early 2000s, football helmets were specifically modified to reduce concussion risks. This included the Adams USA Pro Elite, Riddell Revolution, and the Schutt Sport Air Varsity Commander (AVC) and DNA. These designs have been shown to reduce concussion risks in collisions representative of NFL player experiences (40). Depending on the biomechanical response, the reductions are in the range of 10% for HIC and peak head acceleration and up to 20% based on rotational acceleration. This is an encouraging trend with the newer helmets as NOCSAE pursues the development of football helmet standards for concussion (32). This study describes additional factors that may influence concussion and may point to further means to improve player safety.

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COMMENTS

Perhaps the most important take-home message from this latest in the series of remarkable articles by the National Football League's (NFL) Mild Traumatic Brain Injury (MTBI) Committee is that increasing a player's neck strength may be an effective way to minimize the risk of future concussions. Another interesting result is that some interventions that seem intuitively obvious, such as increasing the amount of padding in helmets, may not be as effective as one might think at preventing concussions.

Alex B. Valadka
Houston, Texas

This study, Part 14 of the NFL MTBI Committee's studies, focuses on head impact velocity and the various force vectors involved with football collisions in reference to concussive injuries. Their findings showed characteristic head displacement, rotation, acceleration, and neck loads depending on several factors. A total of 31 collisions that involved 25 concussions in players who were struck, the majority of whom sustained frontal impacts, were reconstructed and analyzed. Much data is presented regarding the biomechanical forces sustained; for instance, the peak head acceleration for the struck player was 94 ± 28 g and a velocity change of 7.2 ± 1.8 m/s. Head displacement, rotation, and neck tension were all computed using their modeling methods. Another key finding was that progressive increases in neck stiffness reduced peak head acceleration and change in velocity, emphasizing the influence of an effectively strong neck to mitigate head rotation, displacement, and, presumably, the forces transmitted to the brain.

They report that the highest strain forces and rates occur more than 10 m/s after a high-impact force, with impartation by rapid head displacement of these forces to the deep brain structures. They postulate that the midbrain is the anatomical site of concussion origin. It is not surprising that the forces continue to propagate to the midbrain level, but I disagree with the authors' assertion that many of the clinical manifestations of concussion represent mesencephalic dysfunction. Rather, in my experience, the majority of concussion symptoms are cortical in origin, and headaches do not originate at the brainstem region but are vascular and supratentorial. Although the relative contribution of neck musculature is undoubtedly of vast importance, I don't think weak necks have been a major contributing cause of the majority of concussions in football, especially at the collegiate and professional levels. Rather than being caused by having a weak neck, I believe there is sufficient experiential and experimental evidence to conclude that being struck with high velocity forces and/or in an unprepared state (e.g. being "blindsided") is what leads to most traumatic brain injuries in sports. On another matter, it is also not accurate to infer the degree of long-term injury potential by analysis of active players.

As reported, the newer helmet designs have reduced head injury criterion by 10% and peak head acceleration by up to 20% in rotational acceleration. Viano et al. state that concern for concussion in football began in the mid-1990s. However, it actually began with the work of Richard Schneider and others in the late 1960s when football helmet modifications first began in earnest. As noted, by the early 1970s, significant helmet improvements had occurred. In my personal experience, the change from the old web suspension to the thick internal padding and four-way chin strap were the biggest advances thus far, notwithstanding the changes in recent helmet models, some of which were driven by commercial reasons. Given the nature of the sport and its participants, a progressively linear reduction of head injuries may not be possible with further helmet modifications, as acceleration-deceleration forces will continue to be operative regardless of head protection. They suggest that future modifications in the face mask and chin strap system may be possible. However, I am not certain whether or not the lever arm potential of the face mask could ever be substantially reduced. Currently, even the buckling of all four fixation points of the chin strap is not enforced by officials.

Within the limitations of the authors' case study selection and two-dimensional model, this study adds to our knowledge and is replete with data of the physics involved in football and those biomechanical forces associated with concussion. Future researchers should continue to measure these factors and build upon the work which Viano et al. have begun.

Julian E. Bailes
Morgantown, West Virginia

Viano et al. have presented their 14th article in their series examining MTBI in professional football. Using analysis from video replays and simulators in the laboratory setting, they reconstructed a total of 31 collisions that resulted in concussions in 25 players. They were able to measure the translational and rotational acceleration of the head and loads placed on the neck during these collisions. Furthermore, the authors developed a model examining the role of increasing neck stiffness on the biomechanics of concussions.

This work by Viano et al. is another important contribution toward better understanding the forces sustained by the head and neck during blunt head trauma. By reconstructing and analyzing specific instances, they provide us with a glimpse at the forces necessary to cause concussions in the human brain. Although these insights are from the study of impacts in professional football, the information gathered can be used as a model and database for the study of MTBI in general.

Another interesting aspect of this article was their examination of neck strength and its ability to minimize the displacement and acceleration of the head during impact, thus decreasing the chances of sustaining a concussion. It will be interesting to see if new training techniques (i.e., additional neck strengthening exercises) or equipment designs (i.e., modifications to the shoulder pads and helmets) can be developed to take advantage of this information.

Although there have been some discussions in the past regarding findings from previous articles in this series, this particular addition serves as a necessary and thorough biomechanical study of MTBI. Besides providing us with more insight into the biomechanics of concussions, studies such as these allow product manufacturers to tailor their equipment designs to minimize and better dissipate these forces and to better protect the tens of thousands of individuals who play some form of organized football every year.

Min S. Park
Michael L. Levy
San Diego, California

This biomechanical model of head impact in professional football suggests that stronger neck muscles will reduce the likelihood of concussion. Certainly, this makes anatomical sense. However, the model fails to account for a player's relative awareness or unawareness of an impending impact. Many concussions occur after a "blindside" hit that the concussed player never saw coming and which occurred without any preemptory neck flexion on the part of the concussed victim. So, although this biomechanical modeling is a useful exercise in the abstract, it may have less relevance on the field. Nonetheless, safer helmets are needed to further reduce the incidence of concussion in these professional athletes given the deleterious and cumulative impact of repeated concussion. Of note, the authors' assertion, citing their own work, that there are minimal effects of multiple concussions in active NFL players is in stark contrast with much of the recent concussion literature (1–4).

Daniel F. Kelly
Los Angeles, California

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Viano et al. and the NFL MTBI Committee are to be congratulated on the 14th contribution in a superb series of the analysis of concussions in NFL players. The subject of helmets and neck strength are an important part of this discussion. It should be stated again that the 1980s National Operating Committee on Standards for Athletic Equipment standard and subsequent significant helmet changes made for a huge drop in fatalities, fractures, and concussions in football players everywhere. The current study will add more specific knowledge on how to better protect the NFL player and, hopefully, by extrapolation, protect all who play the sport. However, the true incidence of intracranial injury in NFL players is not known. There is no prospective Class I or II data from the NFL tracking each player with a concussion. Although each player now receives extensive neuropsychological examinations as part of their baseline examination, which is a major positive step in protection and management of the players, there have been no guidelines introduced on how to best manage each concussed player. This decision is left to the team physician. The data presented in Part 7 of this fascinating series of articles revealed that approximately 15% of players return to play immediately and approximately 34% rest and return to the same game after concussion. According to the authors in Part 7, return-to-play did not appear to present an increased risk of a second injury. However, until the team doctors, players, and their agents request that all concussed players undergo a routine scan, we shall never fully grasp the true sequelae of these concussions and how best to protect the players in addition to designing better helmets.

Richard G. Ellenbogen
Seattle, Washington

TAB 1P

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Concussion in professional football

Summary of the research conducted by the National Football League's Committee on Mild Traumatic Brain Injury

ELLIOT J. PELLMAN, M.D., AND DAVID C. VIANO, DR. MED., PH.D.

Mild Traumatic Brain Injury Committee, National Football League, New York, New York; ProHEALTH Care Associates, Lake Success, New York; and ProBiomechanics, Bloomfield Hills, Michigan

✓In 1994 the National Football League (NFL) initiated a comprehensive clinical and biomechanical research study of mild traumatic brain injury (TBI), a study that is ongoing. Data on mild TBIs sustained between 1996 and 2001 were collected and submitted by NFL team physicians and athletic trainers, and these data were analyzed by the NFL's Committee on Mild Traumatic Brain Injury. At the same time, analysis of game videos was performed for on-field mild TBIs to quantify the biomechanics involved and to develop means to improve the understanding of these injuries so that manufacturers could systematically improve and update their head protective equipment. The findings and analysis of the Committee have been presented in a series of articles in *Neurosurgery*.

KEY WORDS • **traumatic brain injury** • **sports-related concussion** • **neuropsychological assessment**

In 1992 Al Toon, who was a wide receiver for the New York Jets, was the first NFL player known to have retired because of postconcussion syndrome.[2] The year after Mr. Toon's retirement, another player, Merrill Hoge of the Chicago Bears, retired because of the same problem. Commissioner Paul Tagliabue, team physicians, and many others raised questions: was this a new problem or a misdiagnosed or unrecognized one? Was this a statistical anomaly or the beginning of an epidemic?

It was decided that a rigorous, scientific approach was necessary to gather the data to answer these questions for this high-profile professional sports league. In 1994, Commissioner Tagliabue approved the creation of the NFL's Committee on Mild Traumatic Brain Injury.[12] The Committee was composed of experts inside and outside the NFL. It was decided by the Committee that protection against injury as well as collection and analysis of injury data would be critical to the success of their mission. For the study, a reportable mild TBI was defined as a traumatically induced alteration in brain function manifested by an alteration of awareness and consciousness, including but not limited to an LOC, a "ding," a sensation of being dazed or stunned, a sensation of "wooziness" or "fogginess," a seizure or amnesic period, and by symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, lightheadedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbances, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities.[10] The research summarized here was developed, supervised, and completed in response to the stated goals of the NFL's Committee on Mild Traumatic Brain Injury.

Abbreviations used in this paper: CI = confidence interval; ImPACT = Immediate Postconcussion Assessment and Cognitive Testing; LOC = loss of consciousness; mph = miles per hour; NFL = National Football League; NOCSAE = National Operating Committee on Standards for Athletic Equipment; SI = severity index; TBI = traumatic brain injury.

Protection Against Mild TBI

I: Helmet Standards

Next to impact avoidance, football helmets are the most important factor in protecting a player from mild TBI. In 1973, the NOCSAE established standards for the impact performance of football helmets.[9] The NOCSAE standard limited the SI, which is based on resultant head acceleration. All new football helmets available for use in high school and college football were then certified to the NOCSAE standard, and the wearing of such helmets was made mandatory for college players in 1978 and for high school players in 1980.

The certified helmets cut the SI score by half compared with the headgear worn before the establishment of the standard. By 1980, significant reductions in injuries were observed after the voluntary adoption of the standards by helmet manufacturers. The injury reduction was believed to be the result of the helmet design changes, which targeted serious brain injuries such as brain contusion. Despite the improvement of helmet design for the prevention of serious brain injury, little was known regarding the effectiveness of football helmets in protecting against mild TBI at the time of the initial research conducted by

the NFL's Committee on Mild Traumatic Brain Injury. Therefore, the Committee planned a series of research projects aimed at defining the biomechanics of concussive impacts in professional football.

After consideration of various alternatives, the effort focused on analyses of game videos of plays that had resulted in concussions. Experts in biomechanics proposed that, with multiple views of the impact and line markings on the field, the direction and speed of concussive impacts could be determined. Cinematographic analysis methods were developed to determine the actual speed at which players were moving before colliding (Fig. 1). This would allow laboratory reconstructions (reenactments) of the game impacts by using instrumented test dummies to simulate the helmeted players. The reenactments closely matched the situations on the field.

Reconstruction of the game impacts involved two Hybrid III anthropometric test devices. Two high-speed videos recorded head kinematics in the reconstruction. The cameras were positioned identically to the views from the game video to allow one-to-one comparison (Fig. 2). With the aid of transducers placed in the head of the dummy, the translational and rotational accelerations of the head could be determined in concussive and noninjurious impacts. Matching the available on-field injury video to clinically confirmed mild TBI made determination of an appropriate "event" possible and verifiable. All mild TBI events in the players were examined, confirmed, and recorded by NFL team physicians.

When a mild TBI occurred on the field, it was evaluated by a physician and athletic trainer, who completed forms describing the impact and the injury. Mild TBIs were also reported to a biomechanical engineering group contracted to analyze and reconstruct game impacts. Television network tapes of games were obtained from the NFL and analyzed.

The most striking observation in this study is that concussion in professional football involves a mean impact velocity of 9.3 m/second (20.8 mph) and a head velocity change of 7.2 m/second (16.1 mph). These are exceptionally high velocities and accelerations and long durations. Automotive crashes typically involve impact durations of less than 6 msec for head impacts with vehicle rails, pillars, and structures. The NFL results established new information on tolerances in the 15-msec range; there had been a virtual absence of scientific data on human tolerance. The NFL reconstruction data also supported a value of 70 to 75 G for concussion in padded impacts, which is at the high end of earlier tolerance ranges but is consistent with the Wayne State University concussion tolerance curve. Most important, the initial study demonstrated the strong correlation of concussion with translational acceleration, which should therefore be the primary measure for assessment of the performance of helmet protection systems.[10]

One conclusion of the initial biomechanical study was that the current NOCSAE SI and the more widely accepted Head Injury Criterion are adequate performance measures for helmet standards and that the added complexity of measuring rotational acceleration may not be needed

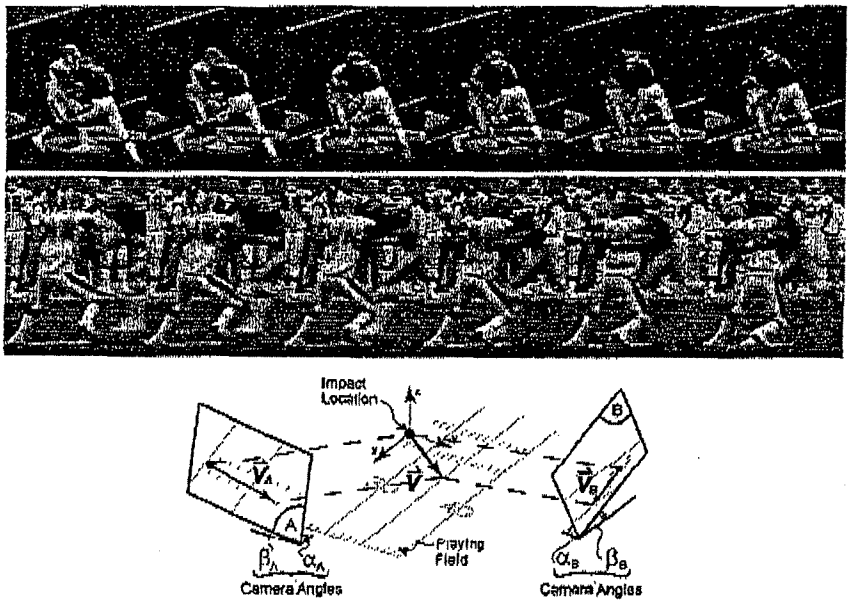

FIG. 1. Still photographs from films showing game action and mathematical calculations of the vector of impacts. The impact velocity of game hits was determined by analysis of two camera views of the collision. *Upper:* The photos show the impact sequence from two views. *Lower:* Graph showing the camera locations and the perspective of the two video images of the game impact. The two perspectives are mathematically merged as vectors that change with each time-step of the video. (Reprinted in modified form with permission from Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF: Concussion in professional football: reconstruction of game impacts and injuries. Neurosurgery 53: 799–814, 2003.)

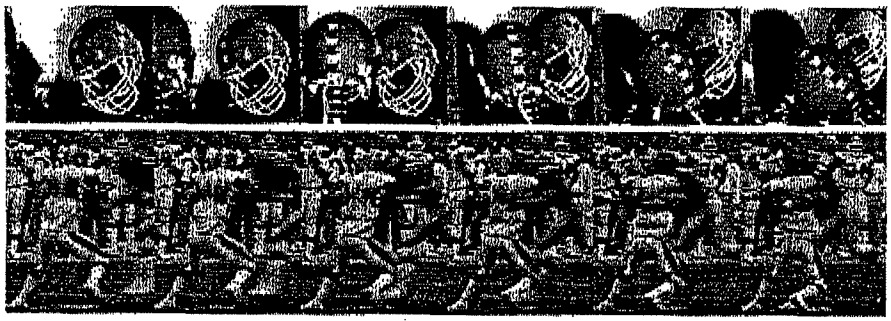

FIG. 2. Comparison of the laboratory reconstruction *(upper)* and still photos of the game impact *(lower)* from a case of concussion sustained in an NFL game. Refinements in the test setup were done until the helmet kinematics matched the game impact sequence. (Reprinted in modified form with permission from Pellman EJ, Viano DC, Tucker AM, Casson IR, Waeckerle JF: Concussion in professional football: reconstruction of game impacts and injuries. **Neurosurgery 53:** 799–814, 2003.)

for an improved or supplemental NOCSAE helmet standard. The results of this study provided a basis for new helmet evaluation methods, new helmet designs, and the prevention of concussions in football.

II: Biomechanical Testing

It was recognized by the Committee that a greater understanding of the location and direction of helmet impacts was needed to give manufacturers the ability to develop newer, improved mild TBI–resistant helmets. Therefore, NFL game videos were further analyzed for the typical locations of severe helmet impacts in professional football. The magnitude and direction of force causing concussion were determined by the use of selected cases that were reconstructed in laboratory tests.

A request was made to have a biomechanical testing contractor reconstruct the impact in 31 cases by using at least two clear video reviews of the collision. Laboratory tests would then be set up to reenact the game impacts with crash dummies and to measure head responses. The reconstruction emphasized helmet-to-helmet and helmet-to-ground impacts, because the video of other impacts was more obscured from clear view. Helmet contact of the struck player was categorized by the impact quadrant and head level for helmet contacts.

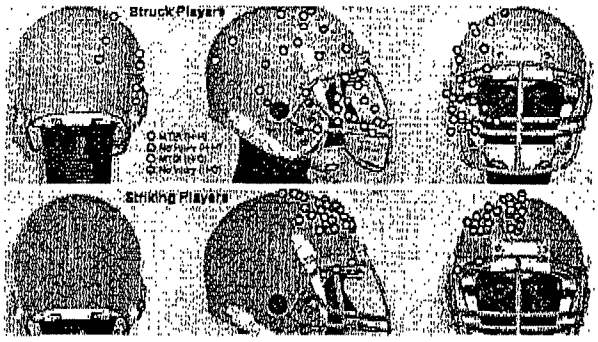

FIG. 3. *Upper:* Photographs of dummy heads showing location of initial helmet contacts for the struck players. Both concussive and nonconcussive impacts and falls to the ground are shown. *Lower:* The impact location for the striking players involved no concussions. The impact locations are all shown on the right side of the helmet, although the game impacts occurred on both sides. H-G = helmet-to-ground impact; H-H = helmet-to-helmet impact; MTBI = mild TBI. (Reprinted with permission from Viano DC, Pellman EJ: Concussion in professional football: biomechanics of the striking player—part 8. **Neurosurgery 56:** 266–280, 2005.)

The study demonstrated the importance of face-mask injuries at an oblique angle, with the majority of contacts occurring below the head's center of gravity.[9] Another important aspect was that it described the quadrants on the helmet for which future NOCSAE standards may establish performance requirements (Fig. 3). By defining relevant quadrants, greater performance may be ensured over a segment of the helmet in which risks of concussion are higher in professional football, particularly low on the side and back and oblique to the face mask.

The laboratory reconstruction of game impacts provided the Committee with data identifying the location and direction of helmet impacts associated with concussion in NFL players. It also provided unique biomechanical data on head responses associated with concussion. The response data also allowed the determination of injury risk functions for concussion.

Using the Logist function, the probability of concussion p(x) was related to various biomechanical parameters (x) measured in the reenactment tests by using the following formula: $p(x) = [1 + \exp(\alpha - \beta x)]^{-1}$ where α and β are parameters fit to the NFL data. The parameters determined for NFL concussion were as follows: $\alpha = 2.677$ and $\beta = 0.0111$ for the Head Injury Criterion; $\alpha = 4.678$ and $\beta = 0.0573$ for translational acceleration; and $\alpha = 5.231$ and $\beta = 0.000915$ for rotational acceleration.[10]

III: Head-Down Tackling

For decades head-down tackling (or so-called spearing) has been a concern because it can result in catastrophic neck injuries in the striking player. The epidemiological and cinematographic analyses of neck injuries have shown that axial loading with flexion or extension causes the majority of cervical fracture–dislocations. This evidence has resulted in rules changes in high school, college, and professional football banning deliberate spearing

and the use of the top of the helmet as an initial point of contact in a tackle. It was observed that players who suffered concussions were sometimes struck by players who were using head-down tackling techniques. The Committee decided to study the biomechanics of this form of injury both in the striking player ("nonconcussed") and the player who was struck ("concussed").

Once again, game film and video were collected from the NFL and correlated with clinical mild TBI data supplied by each club's team physicians. Laboratory reconstruction was performed using Hybrid III male dummies. In the dummy representing the striking player, a six-axis neck transducer was installed between the head and the top of the neck.

In helmet-to-helmet impacts, the striking player lowers the head, neck, and torso to deliver maximum force to the struck player, whose head and neck resist the impact.[15] This is the typical situation when the struck player does not see the tackle and does not prepare for the collision. The key to the concussive blow is the head-down position, which involves a 67% greater mass of the striking player by engaging his torso in the collision. Neck forces couple torso mass into the collision, which contributes to the higher effective mass of the striking player.

The prevention of concussion in the struck player provides another reason, besides preventing neck injuries in the striking one, to enforce rules against head-down tackling or spearing in football. Another means to lower concussion severity may be to reduce the stiffness of the top-crown portion of the helmet and to lower the mass of the helmet, although these changes may be less effective than enforcement of antispearing rules.

IV: Boxing

Because boxing entails considerable risk of closed head trauma, comparisons are often made between this sport and football regarding mechanisms of injury. The risk of concussion is considerably greater in professional boxing compared with professional football. The clinical picture of more severe brain injury is different in football and boxing. Boxers are much more likely to suffer subdural hematomas and deaths from brain injury than are professional football players. A better understanding of the biomechanics of head responses and mechanisms of brain injury would continue to lay the foundation for better protective headgear for sports.

Eleven Olympic boxers were included in this study.[13] These athletes were instructed to strike an instrumented Hybrid III head with their gloved fist two times with four different punches (to the forehead and jaw and with a hook and an uppercut). The height and weight of each boxer were measured and anthropometric data for the dominant hand were collected to allow the effective hand–arm mass to be calculated. Instrumentation was placed in the boxer's clenched hand as well as in the Hybrid III head. A camera recorded the event at a lens speed of 4500 images per second. The punch and head inertial forces were measured.

There were three significant differences noted between the biomechanical forces exerted on the head and brain by boxing punches and the football helmet impacts in the NFL. The boxer's punches resulted in lower translational accelerations in the struck head compared with the football impacts (Fig. 4). The boxer's punch applied a higher moment to the struck head than did the football impacts. This resulted in a higher rotational acceleration in the head that was struck than did the football impacts. Boxers sustain a brain injury by two mechanisms: translational and rotational accelerations of the brain, with a preponderance of the rotational component. Professional football players, on the other hand, sustain mild TBI mostly by translational forces because the shell of the helmet allows the players to slide relative to one another, limiting head rotational accelerations. These differences were further studied using finite element analysis of brain responses.[14] The localized strains in the brain and different biomechanical inputs help explain the clinical differences between head injuries in boxing and professional football.

Finite element modeling also showed that strains develop late, after the primary impact force, and focus on their response at the midbrain. This study shows a complicated interaction of the head kinematics, detailed geometrical and material properties of the brain, and the role of brain movement and deformation within the skull (Fig. 5).

V: Impact Velocity

In our earlier studies, we found that concussions in NFL players occur at an impact velocity of 9.3 ± 1.9 m/second (20.8 ± 4.2 mph) oblique on the face mask, side, and back of the helmet. There is a need for new testing methods to evaluate helmet performance in protecting against impacts causing concussion.

The NOCSAE certifies the helmets used by professional football players. The impact tests provide confidence that protective helmets are effective in reducing life-threatening head injuries. Data collected from the accelerometers used in the NOCSAE head drop test are used to assess the shock-attenuating properties of the helmet based on the head SI, in which the risk of serious head injury is

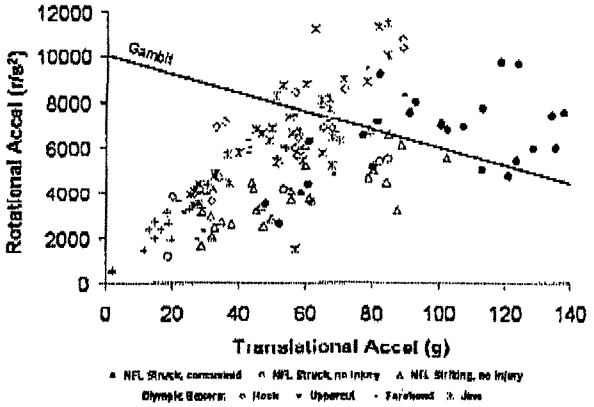

FIG. 4. Scatterplot showing individual data points for translational and rotational acceleration (Accel) of the Hybrid III head for NFL game impacts and four different Olympic boxing punches. (Reprinted with permission from Viano DC, Casson IR, Pellman EJ, Bir CA, Zhang L, Sherman DC, et al: Concussion in professional football: comparison with boxing head impacts—Part 10. **Neurosurgery 57**: 1154–1172, 2005.)

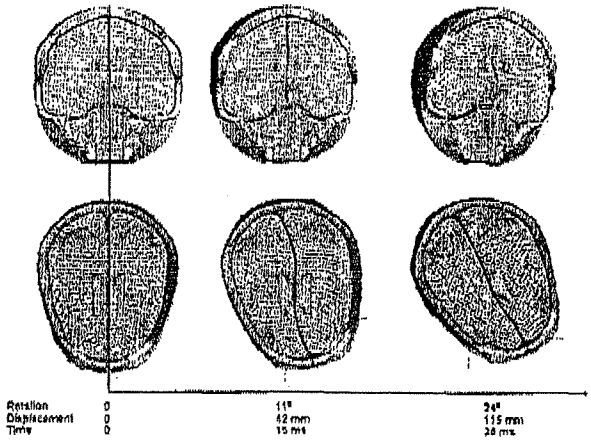

FIG. 5. Anatomical models showing the deformation pattern of the finite element brain from a frontal and superior view of the hemispheres at 0, 15, and 25 msec for one of the NFL concussion cases. The sequence shows the head kinematics and brain deformations. (Reprinted in modified form with permission from Viano *DC, Casson IR, Pellman EJ, Zhang L, King AI, Yang KH:* Concussion in professional football: brain responses by finite element analysis—part 9. Neurosurgery 57: 891–916, 2005.)

determined from the SI. This standard does not address helmet performance in reducing the risk of concussion.

It was believed by the members of the committee that the previous NFL mild TBI research findings would allow a recommendation to be made for a new methodology for testing helmets to reduce the risk of concussions.[11] The initial approach involved pendulum impactors that were used to simulate 7.4 and 9.3 m/second impacts causing concussion in NFL players. A helmet was placed on an instrumented Hybrid III head that was supported on the neck, which was fixed to a sliding table for frontal and lateral impacts. The testing evolved to a linear pneumatic impactor, which gives better control and a broader speed range for helmet testing. The NOCSAE has prepared a draft supplemental helmet standard for the 7.4- and 9.3-m/second impacts evaluated using the new impactor. The proposed NOCSAE standard is the first to address helmet performance in reducing the risk of concussion.

VI: Performance of the Newer Helmets

The new understanding of the biomechanics of concussion in NFL players has enabled football helmet manufacturers to make design changes, for the first time, specifically to reduce the risk of mild TBI. The NFL testing techniques addressing concussion were shared previously with the helmet manufacturers and NOCSAE. The Adams USA Pro Elite, Riddell Revolution, and Schutt Sport Air Varsity Commander and DNA helmets are examples of headgear designed using the new information. Using the new mild TBI testing methodology, the Committee believed it would be useful to test the performance of newer helmets in reconstructed game impacts to compare them with a more standard VSR-4 football helmet.

The aim of this most recent study[10] was to investigate the performance of newer football helmets under conditions causing concussion in NFL players. Ten cases of NFL game concussions were selected for reenactment testing with newer helmets to investigate the equipment's effectiveness in reducing the risk of concussion. The range of impact speed was between 7.4 and 11.2 m/second for eight cases of helmet-to-helmet impacts. This was within one standard deviation of the average condition for concussion in the NFL. The two head-to-ground impact cases averaged 7.2 m/second. For each case of helmet-to-helmet impact, the striking and struck dummies were oriented to match the original laboratory reconstructions of NFL players' concussions. Verification tests ensured that the 10 reconstructed impacts from NFL games were set up similarly to the original testing. Identification on all helmets was obscured, and random tests were conducted.

Testing revealed that newer football helmets reduce concussion risks in collisions that were representative of NFL player experiences. Depending on the biomechanical response, the reductions are in the range of a 10 to 20% lower risk of concussion. The newer headgear reduces concussion risk by using thicker and more energy-absorbing padding on the side and back of the helmets and around the ears. This demonstrates an encouraging trend with the newer headgear; and we expect additional progress. The tests should help NOCSAE in its effort to finalize new helmet standards for preventing concussions.

Injury Collection and Data Analysis

I: Prevalence of Mild TBI

Mild TBI is a major public health problem in the US, with an estimated annual incidence of 160 to 375 cases per 100,000 persons per year. Officials at the Centers for Disease Control and Prevention have estimated that the number of mild TBIs has reached 300,000 cases per year in all sports. In an attempt to better understand mild TBI in the NFL, the Committee supervised prospective collection of data on this condition in NFL players from 1996 to 2001.

All data were collected using standardized forms, and the information was assessed in a blinded fashion. In all, 787 game-related cases (1913 games) were reported, and all players were examined by team physicians, with information reported on player position, type of helmet, symptoms, medical actions, and playing days lost.[5] All patients were evaluated by physicians immediately after the injury and underwent follow-up physician evaluations until they returned to play. Forms were completed by the physicians, which increased the medical validity and reliability of the information collected. Because the Committee did not mandate case management for mild TBI, the patients' medical course reflects the true natural history of mild TBIs among professional football players during this 6-year period.

Mild TBIs are relatively common injuries sustained by professional football players. The data indicate that quarterbacks, wide receivers, defensive backs, and special-team players on kicking units are more likely to sustain these injuries than are offensive and defensive linemen (Table 1). The clinical information helped validate the biomechanical data on professional football–related mild TBIs derived from the earlier studies.

The most common initial symptoms for players who sustained concussions were headaches, dizziness, memory

TABLE 1

Incidence of mild traumatic brain injury according to player position in National Football League games

Position	No. of Cases	Incidence(%)	No. of Game Positions	Risk per 100 game-positions[a]
High Risk				
Offensive				
Quarterback	62	7.9%	3,826	1.62(1.22, 2.02)
Wide Receiver	94	11.9%	7,652	1.23(0.98, 1.48)
Tight End	36	4.6%	3,826	0.94(0.63, 1.25)
Running Back	69	8.8%	7,652	0.90(0.69, 1.11)
Defensive				
Secondary	143	18.2%	15,304	0.93(0.78, 1.08)
Moderate Risk				
Offensive				
Offensive Line	56	7.1%	19,130	0.29(0.21, 0.37)
Defensive				
Linebacker	52	6.6%	11,478	0.45(0.33, 0.57)
Defensive Line	67	8.5%	15,304	0.44(0.34, 0.54)
Special Team				
Return Ball Carrier	22	2.8%	3,826	0.58(0.34, 0.82)
Kick Unit	131	16.6%	38,260	0.34(0.28, 0.40)
Low Risk				
Special Team				
Punter	7	0.9%	3,826	0.18(0.05, 0.31)
Return Unit	33	4.2%	38,260	0.09(0.06, 0.12)
Kicker, FGA	1	0.1%	3,826	0.03(−0.02, 0.08)
Kicker, PAT	1	0.1%	3,826	0.03(−0.02, 0.08)
Holder	1	0.1%	3,826	0.03(−0.02, 0.08)
Unknown/Undesignated	12	1.5%		
Total	787	100%		8.08

[a]Risk per 100 game-positions is the number of concussions divided by the number of times the position was played during the observed period of 3,826 games, multiplied by 100. Values in parentheses are 95% confidence intervals. FGA = field goal attempt; PAT = point after touchdown. Risk strata are approximate, because there is some overlap of confidence intervals.

(Table reproduced with permission from Pellman EJ, Powell JW, Viano DC, Casson IR, Tucker AM, Feuer H, et al: Concussion in professional football: epidemiological features of game injuries and review of the literature—part 3. Neurosurgery 54:81–96, 2004.)

problems, cognitive problems, and somatic complaints. Headaches were observed in 55% (95% CI 51.5–58.5%) of NFL players who suffered concussions.[5] For the great majority of these players, mild TBIs did not cause prolonged disability or prolonged absence from play. In the NFL, 56.5% of players with concussions returned to play on the day of the injury and 97.1% returned to play by Day 9 after the injury. Only 2.9% of the players missed more than 9 days before returning to play. This indicates that most mild TBIs sustained in the NFL are self-limiting and that players recover fully and spontaneously in a short time. Because a significant percentage of players returned to play in the same game and the overwhelming majority of players with concussions were kept out of football-related activities for less than 1 week, it can be concluded that mild TBIs in professional football are not serious injuries.

Only 9.3% (95% CI 7–11.6%) of the NFL players experienced an LOC as a result of severe concussive head impacts (58 of 623 reported cases of mild TBI). It is important for all physicians who care for athletes with head injuries to know that most of the concussions they treat are not associated with LOC, and that when this symptom occurs it is for a relatively short duration.

II: Repeated Mild TBI

Despite the findings and conclusions in the preliminary clinical study, questions remained concerning NFL players who suffered repeated mild TBIs. Physicians have been concerned for many years about the possible deleterious effects of multiple concussions on the brains of athletes.[8] In this study, data on 887 concussions sustained in practices and games involving 650 players from all 30 NFL teams between 1996 and 2001 were prospectively collected and analyzed. A total of 160 players experienced repeated injury, with 51 suffering three or more concussions during the study period. The median time between injuries was 374.5 days, with only six concussions occurring within 2 weeks of the initial injury. Repeated concussions were more prevalent in the secondary, the kick unit on special teams, and in wide receivers.

There have been reports in which researchers have concluded that there may be an increased risk of repeated concussive injuries, and there may be a slower recovery of neurological function after repeated concussions in those who have a history of previous ones. The results of this study in professional football players do not support that conclusion. Although approximately one half of players returned to play during the same game or practice session, and approximately 90% returned within 1 week, recurrent injury caused by an increased vulnerability in the immediate postconcussion period does not seem to be a factor in professional football players.

No cases of "second-impact syndrome" were detected during the 6-year period of this NFL study. There were no deaths, prolonged comas, or evidence of diffuse cerebral

edema in any player. Furthermore, there have been no case reports of second-impact syndrome in the history of the NFL. It is possible that this syndrome does not truly exist in this population of athletes. Many of the mild TBI guidelines have established exclusion periods based at least partially on the belief that everyone who experiences a symptomatic mild TBI is at risk for the development of second-impact syndrome. The absence of the syndrome in this patient population supports the suggestion that such arbitrary return-to-play guidelines may be too conservative for professional football.

Another often-expressed concern underlying the development of mild TBI guidelines is the occurrence of chronic brain damage as a result of multiple head injuries. A recent letter to the editor in *Neurosurgery* addressed the case of an NFL player who was alleged to have died of complications of chronic traumatic encephalopathy, underscoring this concern.[1] Chronic traumatic encephalopathy in boxers is a well-accepted and documented clinical and pathological syndrome. The clinical features include a combination of cerebellar, extrapyramidal, and pyramidal dysfunction, along with cognitive and personality changes. In the NFL study, none of these features was identified in any player, including those with repeated injury. There were no signs of chronic traumatic encephalopathy in this group of active, contemporary football players.

In players with four or more concussions there was a greater chance of personality change and fatigue, but the number did not reach statistical significance. The incidence of LOC at the time of mild TBI was no different with successive concussions. Overall, however, the signs and symptoms reported in the NFL study were very similar in players with single and multiple mild TBIs. There was no evidence of increased severity of injury in multiple compared with single mild TBI cases.

III. Postconcussion Syndrome

In the data analysis there were a small number of athletes in whom persistent postconcussion symptoms developed, and these individuals were unable to return to play for an extended period. Often the postconcussion symptoms were seen 1 or more weeks after the injury. The postconcussion syndrome follows head injury that is usually mild, and consists of any combination of the symptoms and signs that occur after mild TBI. The Committee decided to analyze the data obtained in players in whom postconcussion syndrome was diagnosed.[7]

When does cerebral concussion end and postconcussion syndrome begin? Very few data are available on the evolution of head injury to postconcussion syndrome in athletes. The purpose of this part of the study was to compare the small group of NFL players who did not return for more than 7 days after a mild TBI with the majority of NFL players who do return within 7 days. The 7-day dividing line between the groups does not reflect an arbitrary distinction. Because NFL teams play games once a week, the players in this study cohort all missed at least one game. The NFL teams play only 16 games per season. Therefore, missing one game involves a significant loss of playing time. The study cohort all had significant functional impairment caused by mild TBI.

There were 72 cases with more than 7 days away from play among the 887 cases of mild TBI analyzed between 1996 and 2001. Of these injuries, 38 were single concussions experienced in the study period, eight were the first of repeated concussions, 16 were the second, seven were the third concussion, and so on in the study period. The median duration between the first injury and 7 or more days away from play was 364 days, and the median duration between the last injury and 7 or more days away was 329 days, which is statistically similar.

For the whole sample, there were 650 players who experienced 887 concussions during the study period, and the position they were playing was recorded in this analysis. Individually, the position groups most often associated with loss of 7 or more days are the defensive secondary (23.6%), kick unit (19.4 %), quarterbacks (12.5%), and wide receivers (12.5%). The fraction of players in a position with 7 or more days away from play compared with all in that group was highest for the quarterback (14.8%), the return unit on special teams (11.8%), and the secondary (10.8%), followed by the kick unit (10.4%) on special teams. Quarterbacks had the highest odds ratio of 7 or more days away from play with concussion, whereas running backs had the lowest relative risk.

The majority of players with concussions (88.9%) are rested, with 7 or more days out, compared with 90.7% with fewer than 7 days out. Overall, the data show a conservative treatment of concussion. There was no statistical difference in players' treatment in comparisons between the two groups.

Between the initial examination and the first follow-up review, most of the signs and symptoms started to decrease, except for increases in the general category of memory problems, fatigue, irritability, and sleep problems. By the fourth follow-up examination (median 4.7 days), all memory and cognitive problems had cleared. Nevertheless, some players still reported headaches, dizziness, and photophobia. Somatic complaints continued in some players, including personality change and fatigue. By the seventh examination (median 13 days) only headaches remained to clear.

The data analysis allowed the development of profiles for two groups of NFL players with concussions. The first group is the small minority of players who ultimately do not return to play for 7 or more days postinjury. They are more likely to experience LOC as a result of the head injury, and they are more likely to be hospitalized on the day of the injury. At the time of the initial evaluation, these players have a significantly increased number of the signs and symptoms of mild TBI. On initial examination, they are very likely to have retrograde amnesia, difficulties with immediate recall, and overall difficulties with cognition and general memory.

The results of this study and the previous ones prompted the Committee to perform a critical analysis of the widely promoted guidelines for the evaluation and management of concussion in sport. This 6-year study indicates that no NFL player experienced second-impact syndrome, chronic cumulative injury, or chronic traumatic encephalopathy from repeated injuries. These are a few of the expressed rationales for developing management guidelines. The proponents of these guidelines recommend grading the severity of concussion by a limited number of criteria, such as presence or absence of LOC, post-

traumatic amnesia at time of injury, confusion, and mental status changes soon after injury. The guidelines then make clinical management recommendations on the basis of the grade of concussion diagnosed using these criteria.

Analysis of the NFL data reveals that there are other prognostic factors of equal importance that are not included in the grading systems. These include photophobia, fatigue, and increased absolute numbers of signs and symptoms. Furthermore, the grading systems do not take into account factors such as the position played by the injured athlete and the type of play during which the injury occurred. In the NFL study we found that the presence of signs and symptoms such as fatigue, sleep disturbance, irritability, and/or cognitive or memory impairments on examination the day after the injury also has significant prognostic usefulness. None of the grading systems incorporate results from examinations performed other than on the day of the injury. Although the grading systems use some important prognostic findings, they are limited in their scope and fail to incorporate a number of other factors that have been demonstrated to be predictors of delayed recovery.

The NFL studies also support the contention that grading concussions immediately after injury is prone to error. A number of players with signs and symptoms suggesting a poor prognosis in fact recovered very quickly and returned to play on the day or within a few days of injury. Conversely, there were two players with minimal signs or symptoms, suggesting a good prognosis, who were ultimately kept out of play for 7 or more days after mild TBI. None of the prognostic factors or combinations of factors was 100% accurate in predicting recovery.

It followed from this analysis that the current attempts to link prospective grading of concussion symptoms to arbitrary, rigid management decisions are not consistent with scientific data. We believe that if one insists on grading concussion severity, the best way is retrospectively, on the basis of how long it actually takes the player to become asymptomatic, with normal results on neurological examination. It is the recommendation of the NFL's Committee on Mild Traumatic Brain Injury that team physicians treat their players on a case-by-case basis, using their best clinical judgment and basing their decisions on the most relevant, objective medical data obtained.

IV: Neuropsychological Testing in Evaluating Mild TBI

The development and use of neuropsychological testing in the NFL has been rapid, and it has contributed to the implementation of this testing in other professional sports organizations, including ice hockey, automobile racing, and Australian Rules football. When used in concert with other medical information, neuropsychological test data contribute quantitative information regarding neurocogni tive processes, such as attentional, memory, and cognitive processing speed. Neuropsychological testing can provide objective information regarding the recovery process and allows comparisons of the athlete's performance against normative data and the individual's preinjury level of performance.

The NFL's neuropsychological testing program was established as a clinical research program with the goal of investigating the use of such testing to assist team physicians in the return-to-play decision. Athletes in the NFL who underwent neuropsychological testing between 1996 and 2001 and who participated in the study were included.[4] Preseason normative data were collected in 655 NFL athletes. The overall sample of injured players who underwent testing consisted of 143 athletes. This sample represented 22% of the 650 NFL athletes who experienced 887 concussions during the study period. Because participation in the study was voluntary, not all athletes with mild TBI completed neuropsychological testing.

This study supports previous research that has shown that on-field signs of cognitive impairment, such as amnesia, are useful in determining the severity of brain injury.[4] Players identified as having cognitive and memory disturbances are likely to show neuropsychological impairments on follow-up testing. Athletes with no clinically recognized cognitive and memory impairments on physician examination did not, as a group, have more subtle changes in cognitive processes that were missed during the sideline clinical examination. This suggests that the on-field evaluation by team physicians is effective with regard to the identification of cognitive and memory impairments immediately after an injury.

On review of the data, the fact that there were no overall significant differences in test results between a group of injured NFL athletes who had previously undergone baseline neuropsychological testing suggests that NFL athletes with mild TBIs recover quickly after injury (Fig. 6). In contrast to previous studies in which cognitive difficulties lasting 1 week or more were suggested, NFL athletes demonstrated generally intact performance within several days relative to baseline performance levels.

The issue of the potential cumulative effects of sports-related mild TBI has been a particularly controversial one, and many studies have offered different views on the significance of multiple injuries. In this study we did not find a pattern of worse neuropsychological test scores in a group of professional athletes who received close follow-up care for 6 years. We also did not find worse neuropsychological performance in NFL athletes who were held back from play for 7 or more days compared with a group who returned within 1 week. It is noteworthy that in this group that was kept from play for 7 or more days, the results of neuropsychological tests and medical evaluation of cognitive and memory function were normal within several days of the injury. The results of this study indicate no evidence of worsening injury or chronic cumulative effects of multiple mild TBIs in NFL players.

On the basis of this study, the NFL's Committee on Mild Traumatic Brain Injury makes the following recommendations regarding the proper role of neuropsychological testing in the NFL. Neuropsychological testing is a tool that can assist the physician in evaluating and managing mild TBI. It definitely should not be used in isolation and cannot replace and should not be used to replace the clinical judgment of the treating physician in the diagnosis and management of mild TBI. The main value of neuropsychological testing in this setting is its ability to confirm and corroborate the results of clinical and mental status evaluation.

V: The ImPACT Program

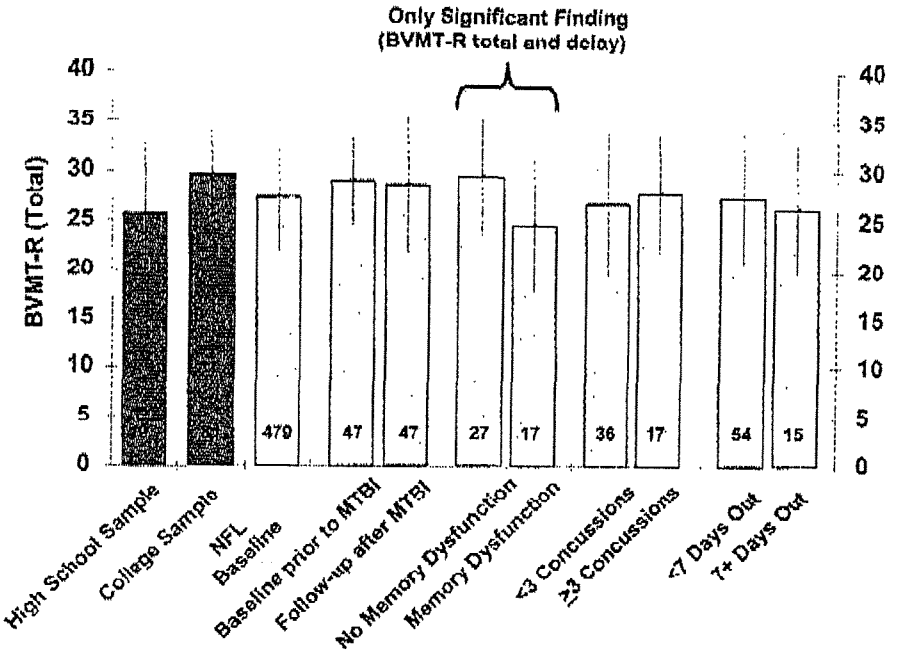

FIG. 6. Bar graph showing the mean and standard deviation in Brief Visuospatial Memory Test-Revised (BVMT-R) (Total) neuropsychological test scores for NFL baselines, players with memory problems, more than three concussions, and out 7 or more days from play. Also shown are data (first two bars) from a sample of high school and college players at Penn State University. (Reprinted with permission from Pellman EJ, Lovell MR, Viano DC, Casson IR, Tucker AM: Concussion in professional football: neuropsychological testing—part 6. **Neurosurgery 55**: 1290–1305, 2004.)

Despite the initial findings, based on neuropsychological testing, that NFL players had a rapid return to baseline after a mild TBI, the Committee was aware that this was in contrast to several studies that found more long-lasting neuropsychological decrements in high school athletes. Members of the committee decided to perform an additional study, in which professional and younger athletes were compared using the same protocol and identical neurocognitive test battery.[5]

The ImPACT program is a computerized neuropsychological testing instrument used by some within the NFL neuropsychology program. The ImPACT program (version 2.0) consists of six neuropsychological tests designed to target different aspects of cognitive functioning, including attention, memory, processing speed, and reaction time. The results of ImPACT testing on NFL and high school players were compared.

In this study,[5] we found no significant neurocognitive deficits in the NFL sample within the 1st week postinjury, suggesting that NFL athletes with mild TBIs recover relatively quickly after injury. In contrast, we found residual difficulties in reaction time and memory in the high school sample but not in professional players. This raises the question of differential response to mild TBI in professional and high school athletes.

VI: Return to Play

There were concerns based on the results of the earlier studies of mild TBI that perhaps some players were being returned to play too soon after injury, thus resulting in more prolonged postconcussion syndrome and perhaps creating the risk of more severe brain injury. The committee therefore decided to do a data analysis on NFL players who returned to play on the same day as their mild TBI.[6]

In the NFL players studied between 1996 and 2001, there were 135 (15.2%) who returned to play immediately after mild TBI and 304 (34.3%) who rested and returned to the same game after concussion. There were few differences in the player position or team activity related to the injury or action taken. However, players who suffered concussions and returned to the same game had fewer initial signs and symptoms than those who were removed from play.

Widely used concussion management guidelines state that athletes can return to play on the day of the injury if they become asymptomatic and if results of examinations performed within 15 minutes of their injury are normal. In the NFL database, 41% of players returned to the same game either immediately or after resting more than 15 minutes. Of those who returned immediately, 17.9% were out more than 15 minutes, and 51.7% of those who rested and returned were out for more than 15 minutes. The data showed no increased risk of repeated mild TBI, prolonged postconcussion syndrome, delayed return to play (≥ 7 days out), second-impact syndrome, or catastrophic intracranial event. The NFL experience thus supports the suggestion that players who become asymptomatic and have normal results on examinations performed at any time after injury, while the game is still in progress, have

been and can continue to be safely returned to play on that day. The data also support the proposition that players who experienced LOC had no increased risk of repeated mild TBI or prolonged postconcussion syndrome compared with other players.

The results of this study indicate that many NFL players can be safely allowed to return to play on the day of the injury after sustaining a mild TBI. These players had to be asymptomatic, with normal results on clinical and neurological examinations, and be cleared by a knowledgeable team physician. There were no adverse effects, and the results once again are in sharp contrast to the recommendations in published guidelines and the standard of practice of most college and high school football team physicians. This data analysis was performed on information obtained in adult professional football players, and these findings are not meant to be carried over to any other patient population, including high school and college football players.

Conclusions

The NFL study was conducted to increase the scientific information available to physicians and sports professionals based on prospective clinical information and contemporary, "real-time" biomechanical data. The NFL's Committee on Mild Traumatic Brain Injury is currently supervising work on an animal model of mild TBI, mouthpieces, and studies of retired players that we hope will continue to add information and shed light on the complicated clinical syndrome of mild TBI in athletes.

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Address reprint requests to: David C. Viano, Dr. Med., Ph.D, ProBiomechanics, 265 Warrington Road, Bloomfield Hills, Michigan 48304. email: dviano@comcast.net.

TAB 1Q

Report on ProCap Helmet Tests at Biokinetics

Prof. Dr. David C. Viano
Mild Traumatic Brain Injury (MTBI) Committee
National Football League

SUMMARY

Protective Sports Equipment markets ProCap and sponsored impact tests with the Hybrid III dummy to assess ProCap's potential effect on concussion risks. Two series of helmet impact tests were conducted using a matched test protocol where the helmeted head was struck by a pendulum impactor giving a baseline response and the identical condition re-run with ProCap in place. The baseline helmet was either the Riddell VSR-4 or Bike helmet. The impact locations were those determined by the NFL's MTBI research on concussion and were developed into a standard impact series by Biokinetics in Ottawa, Canada. The first test series involved the full array of 8 new impact locations that represent concussion exposures in NFL games. The second series involved three impact locations. Accelerometers were placed in the Hybrid III head to record translational and rotational acceleration. These responses provided information on peak accelerations and were used to calculate injury risk functions, including the NOCSAE Severity Index (SI) and the Head Impact Power Index (HIPI).

The results of the first series show a reduction in head acceleration and injury indices for 6 of the 8 impact locations. However, the average effect for all conditions is only a small improvement and is not statistically different from the baseline helmet response. For the 4 lateral impacts, ProCap reduced the head accelerations by 3%-12% and SI 3%-23%. The two conditions giving higher responses were the impacts on the rear of the helmet and the oblique front boss causing up to 25% increases. The second series of tests produced less favorable results. Increases in head acceleration were seen in 4 of the 6 matched test conditions. In the front boss impact, there was a 52% increase in head rotational acceleration.

A further analysis of the response data was made by converting the SI values to concussion injury risks using a Logist function obtained from the Biokinetics' reconstruction experiments of NFL game impacts. The eight impact conditions have an average concussion risk of 72% ± 28% with the Riddell helmet, and 63% ± 28% with ProCap on the helmet. In some cases, there is a substantial reduction in risk with ProCap, but others show equally large increases.

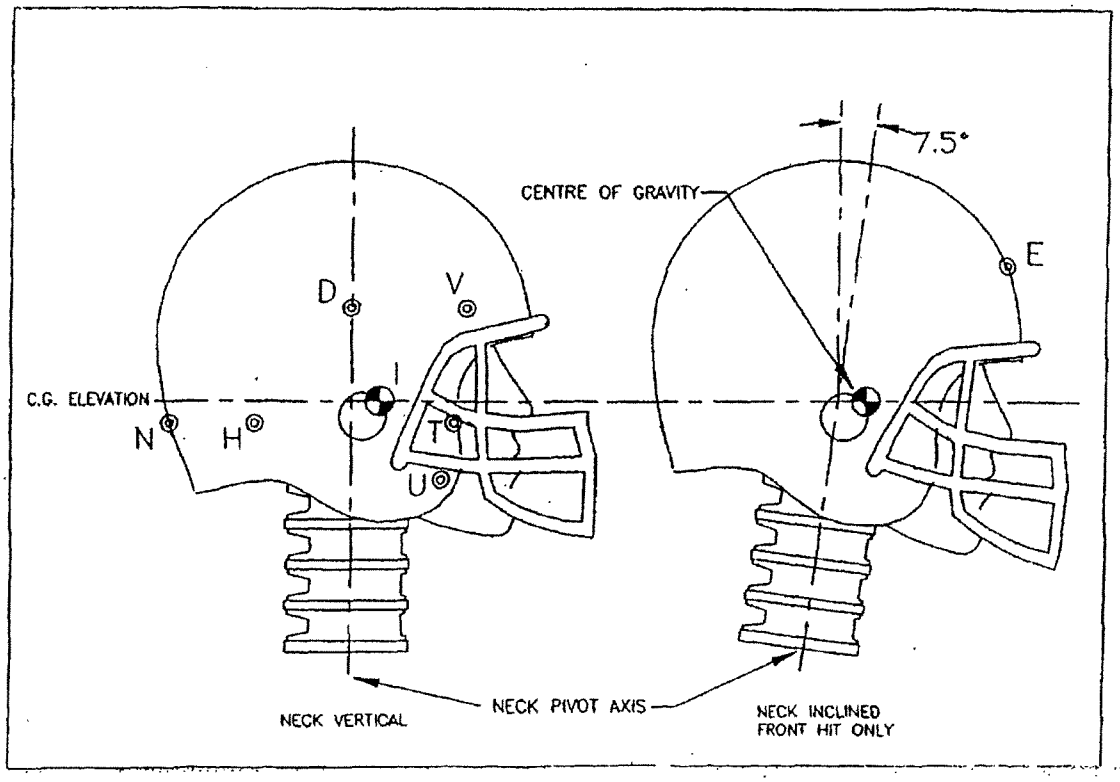
On balance, the tests do not demonstrate a consistent reduction in head impact acceleration or concussion risk with ProCap. In some cases, up to 37% reductions were seen in responses and 61% in concussion risk, but equally large increases were also observed. Until the increases are better understood and resolved, the tests should be viewed as equivocal results, some improvements and some contrary effects, and do not demonstrate consistent safety improvement against concussion with ProCap.

METHODOLOGY

Figure 1 shows eight new helmet impact locations used to evaluate concussion risks in football; these conditions are different from what NOCSAE routinely runs to assess helmet performance. For the Biokinetics tests, the Hybrid III head and neck are supported on a rigid plate. The head is tightly covered in two layers of nylon stocking and the helmet placed on the head. The chinstrap is fixed in place. Four of the impact locations (I, D, T and U) involve lateral blows low on the side of the helmet. There are two impacts at 45^0 between front and side with loading on the front or rear boss. The remaining two tests involve a front impact high on the helmet and a rear impact low at the back surface.

A pendulum impactor is used to deliver a blow at 5.4 m/s with a 19 kg mass. The impact interface is a section from a football helmet placed over a mating steel surface. The Hybrid III head is fit with conventional triaxial accelerometers and a 3-2-2 array of accelerometers to determine head rotational acceleration and velocity. In some tests, high-speed video was used to record the impacts. However, in this analysis no video was available for review.

Two series of tests were conducted using a matched test procedure. One test was run with the ProCap fixed to the helmet and an identical test run on the base helmet. For some of the impact locations, there was a primary impact on the ProCap, while in some others the impact was to the underlying helmet with ProCap interacting secondarily with the impactor. The first series was conducted around February 1, 2001 and involved all eight impact locations giving 16 tests, eight with ProCap added to the helmet



Site I (C of G) – Neck is oriented vertical. Lateral impact directed through the centre of gravity of the headform.

Site D (high side) – Neck is oriented vertical. Lateral impact directed 20 mm behind and 64 mm above centre of gravity.

Site T (front side) – Neck is oriented vertical. Lateral impact directed 52 mm forward and 15 mm below centre of gravity.

Site U (front earflap) – Neck is oriented vertical. Lateral impact directed 43 mm forward and 54 mm below centre of gravity.

Site N (rear) – Neck is oriented vertical. Rear impact, centred on the headform, directed 15 mm below centre of gravity.

Site H (rear boss) - Neck is oriented vertical. Neck is pivoted on its vertical axis 45° from site N.

Site V (front boss) – Neck is oriented vertical. Neck is pivoted on its vertical axis 45° from site D.

Site E (front) – Neck is inclined forwards 7.5°. Front impact, centred on headform, 103 mm above the centre of gravity.

Figure 1: Impact locations on the helmet used in the ProCap evaluation tests.

and 8 with the baseline VSR-4 Riddell Helmet. The second series of tests was conducted around May 7, 2001 and involved three impact locations and two different baseline helmets. Some tests were carried out with the Riddell VSR-4 helmet and others with a Bike helmet.

The impact responses were signal conditioned and A-D converted. Data was stored digitally using standardized practices from automotive crash testing. The key biomechanical results were summarized in tables of peak values. The key responses included the:

Peak Resultant Translational Acceleration
Peak Resultant Rotational Acceleration
Head Impact Power (HIPI)
Gadd Severity Index (GSI or SI)

The Gadd severity Index (GSI) or SI is used by NOCSAE to certify football helmets using a different test procedure and dummy head than that used in these tests.

Concussion injury risks were calculated for the tests with and without ProCap on the helmet. A Logist risk function was determined from the reconstruction experiments of NFL impacts that are being invested under the auspices of the NFL's MTBI Committee. For this analysis, the Severity Index (SI) was selected because it is the biomechanical measure used by NOCSAE and has an established history and acceptance by the safety community. Similar results are expected if the HIC or other response measures were used.

Biokinetics' database of NFL impact reconstructions includes 40 players, 17 with concussions. While the research is ongoing and all results will require verification, the Logist analysis yields the following relationship between SI and the risk of concussion (p):

$$p(SI) = 1/[1 + \exp(3.84 - 0.013*SI)].$$

This function has a correlation coefficient of 0.77.

RESULTS

Table 1 summarizes the key impact biomechanical responses in the first matched test series. On average, there is a slight reduction in head acceleration responses, but the standard deviations are considerable because 2 of the 8 conditions showed an increase in responses. There is no statistically significant difference between the baseline helmet and ProCap responses, based on the 8 impact locations evaluated with a standard t-test. Impact location V, which is an oblique impact on the front boss, showed the greatest increase in head accelerations, varying from 25%-47% higher levels than with only the VSR-4 helmet. It should be noted that this impact location produced the lowest head accelerations among the 8 impacts conditions. The other impact causing an increased response was to the rear low on the back of the helmet. It is likely that this impact did not directly load ProCap but most likely secondarily interacted with it later in the impact sequence.

Table 2 summarizes the key biomechanical responses in the second matched test series. There was a slight increase in head acceleration responses in 4 of the 6 matched test conditions. On average, the head responses were slightly higher with ProCap than with the baseline Riddell or Bike helmets, and there were no statistically significant differences using a t-test comparing the baseline and ProCap responses. There was only one test with a substantial reduction in head responses with ProCap; but in one condition, there was a 52% increase in head rotational acceleration.

Table 3 shows the risk of concussion for the paired helmet impacts in test series 1. The change in concussion risk varies from a 61% reduction for impact site E to a 57% increase for impact site V. On average, the change was an 8% ± 33% reduction with ProCap when all sites were considered, but the standard deviation was quite large. Only impact site D showed a substantial reduction from an initially high concussion risk.

Table 4 shows the risk of concussion for the paired helmet impacts in test series 2. Here, four of the six test conditions showed an increase in concussion risk with ProCap, but the average change for all conditions was a 2% ± 31% reduction. The average improvement was dominated by one test condition and again there was a large standard deviation in the results.

ADDITIONAL COMMENTS

Since ProCap does not cover the entire football helmet, some of the impact tests resulted in initial contact with the underlying helmet or face-guard, and potential secondary interactions with ProCap. The addition of ProCap displaces the initial contact by the thickness of the add-on. This slightly changes the impact conditions. Also, the effect of ProCap on the coefficient of friction between the impactor and helmet system was not determined. The possible effects of these factors on the helmet kinematics could not be assessed without high-speed video of the tests. Since only a few tests were videoed, these effects could not be determined. In addition, any effects on the neck response were not determined in the testing.

ProCap adds weight to the head-neck and helmet setup. On average, the higher mass should lead to lower accelerations in the type of pendulum test conducted by Biokinetics. These effects were also not considered in the comparisons.

Table 1: Results from the First Series of ProCap Helmet Tests.

Impact Site	Resultant Translational Acceleration g			Resultant Rotational Acceleration r/s/s			HIPI: Head Injury Power			SI: NOCSAE Severity Index		
	w/o	w PC	% diff.	w/o	w PC	% diff.	w/o	w PC	% diff.	w/o	w PC	% diff.
U	169.2	150.4	-11%	10408	9302	-11%	27910	24094	-14%	612	508	-17%
T	102.8	99.4	-3%	8684	8100	-7%	16126	14919	-7%	310	295	-5%
H	129.2	123.9	-4%	10177	9432	-7%	17310	15309	-12%	418	395	-6%
N	110.9	118.5	7%	5583	4952	-11%	19335	17904	-7%	470	489	4%
I	126.8	111	-12%	7892	7980	1%	19181	16028	-16%	442	352	-20%
D	137.6	124.3	-10%	11240	10333	-8%	18508	15426	-17%	492	378	-23%
V	62.3	77.6	25%	4273	6274	47%	8122	11286	39%	181	226	25%
E	97.7	74.5	-24%	5212	4918	-6%	14108	10097	-28%	298	188	-37%
Average	117.1	110.0	-4%	7934	7661	0%	17575	15633	-8%	403	354	-10%
sd	31.6	25.4	15%	2645	2072	19%	5567	4261	20%	135	114	19%

Table 2: Results from the Second Series of ProCap Helmet Tests.

Name of Helmet	Impact Site	Resultant Translational Acceleration g			Resultant Rotational Acceleration r/s/s			HIPI: Head Injury Power			SI: NOCSAE Severity Index		
		w/o	w PC	% diff.	w/o	w PC	% diff.	w/o	w PC	% diff.	w/o	w PC	% diff.
VSR-4	V	65.8	73.8	12%	3939	5989	52%	8804	10732	22%	171	194	13%
VSR-4	E	98.5	76.6	-22%	5598	5518	-1%	14574	10563	-28%	294	191	-35%
Bike	V	67.7	73	8%	5802	5904	2%	9401	9620	2%	212	203	-4%
Bike	E	60.3	68.9	14%	3850	2728	-29%	7599	8810	16%	158	175	11%
Bike	E	65.8	69.0	5%	3908	3279	-16%	7713	8628	12%	166	173	4%
Bike	High E	67.1	65.6	-2%	4865	4848	0%	8828	8156	-8%	153	154	1%
	Average	70.9	71.2	2%	4660	4711	1%	9487	9418	3%	192	182	-2%
	sd	13.8	4.0	13%	891	1394	28%	2588	1064	18%	54	18	18%

Table 3: Concussion Risks from the First Series of ProCap Helmet Tests.

Impact Site	SI: NOCSAE Severity Index			Concussion Injury Risk (%)		
	w/o	w PC	% diff.	w/o	w PC	% diff.
U	612	508	-17%	98%	94%	-4%
T	310	295	-5%	55%	50%	-9%
H	418	395	-6%	83%	78%	-6%
N	470	489	4%	91%	93%	2%
I	442	352	-20%	87%	68%	-22%
D	492	378	-23%	93%	75%	-20%
V	181	226	25%	18%	29%	57%
E	298	188	-37%	51%	20%	-61%
Average	403	354	-10%	72%	63%	-8%
sd	135	114	19%	28%	28%	33%

Table 4: Concussion Risks from the Second Series of ProCap Helmet Tests.

Name of Helmet	Impact Site	SI: NOCSAE Severity Index			Concussion Injury Risk (%)		
		w/o	w PC	% diff.	w/o	w PC	% diff.
VSR-4	V	171	194	13%	17%	21%	27%
VSR-4	E	294	191	-35%	50%	20%	-59%
Bike	V	212	203	-4%	25%	23%	-8%
Bike	E	158	175	11%	14%	17%	20%
Bike	E	166	173	4%	16%	17%	8%
Bike	High E	153	154	1%	14%	14%	1%
	Average	192	182	-2%	23%	19%	-2%
	sd	54	18	18%	14%	3%	31%

ACKNOWLEDGEMENT

The ProCap test data was given to D. Viano on November 26, 2001 during a visit at Biokinetics. This analysis solely addresses the head acceleration effects of ProCap in pendulum impacts representative of concussion exposures to NFL players.

I had several conversations with Bert Straus in January 2002 about the ProCap testing, analysis and interpretation of results. I appreciated the exchange and his insights.

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Albert Straus' letter to Marc Beusenberg, Biokinetics, authorizing ProCap test data review by the NFL. October 31, 2001.

Chris Withnall', Biokinetics, letter to Bert Straus summarizing test results from ProCap pendulum impacts. February 28, 2001.

Chris Withnall', Biokinetics, letter to Bert Straus indicating NFL interest to review new ProCap test data. January 10, 2001 (sic).

TAB 2

NEWS

NATIONAL FOOTBALL LEAGUE
280 Park Avenue, New York, NY 10017
(212) 450-2000 * FAX (212) 681-7573
WWW.NFLMedia.com

Joe Browne, Executive Vice President-Communications
Greg Aiello, Vice President-Public Relations

FOR IMMEDIATE RELEASE
MAY 22, 2007

NFL OUTLINES STANDARDS FOR CONCUSSION MANAGEMENT

Commissioner **ROGER GOODELL** reviewed with NFL clubs today consensus medical recommendations regarding the management of concussions.

The standards are based on extensive medical research and discussion. They were recommended by the NFL's independent committee on mild-traumatic brain injury (MTBI) chaired by **DR. IRA CASSON** of the Long Island Jewish Medical Center and the Albert Einstein School of Medicine, and **DR. DAVID VIANO** of Wayne State University.

"NFL clubs do an excellent job in the care and management of concussions that affect NFL players," Commissioner Goodell said. "It is important that we articulate for our clubs and players, and for the broader medical community, the standards our clubs are applying as we work together to protect the health of our players. The overriding principle governing management of concussions in the NFL is that medical considerations must always have priority over competitive situations."

The recommended standards were presented at the NFL's spring business meeting in Nashville, Tennessee, and include the following elements:

- Medical decisions must always override competitive considerations.

- Neuropsychological baseline testing will be required for all NFL players beginning this season, using a standardized test to establish an individual functional baseline. Neuropsychological testing is one tool a physician can use to assist in the management of MTBI. It cannot be used by itself to make clinical decisions. For players removed from games due to concussions, repeat testing will be done during the season to track recovery and to help decide when they can return to play. These players also will be re-tested against their baseline performance the following season at training camp.

- An NFL MTBI conference will be held on June 19 in Chicago for all NFL team physicians and athletic trainers to share the most up-to-date information on state-of-the-art care and management of concussions.

- The MTBI Committee will continue to operate as an independent group. Three non-NFL affiliated physician have been added to the committee (Joseph Maroon, Joel Morgenlander, and Thomas Naidich). The goal of the committee remains the advancement of scientific knowledge of MTBI through well-conceived research to protect the health of NFL players and improve the safety of the game.

- To promote the use of best practices by all teams, the evaluation procedures used by NFL teams will be shared among all medical and training staffs.

- A brochure will be developed and distributed to NFL players to help educate players and their families about concussions, including how to recognize the symptoms of a concussion and recommended treatment procedures.

- Return-to-play decisions should continue to be made by team medical personnel using their expertise and professional judgment.

- The NFL rule requiring every player to wear a chin strap that is completely and properly buckled to the helmet will be strictly enforced. Teams and players will not be permitted to modify the attachment of the chin strap to the helmet or improperly modify the helmet in any other way. The longstanding safety-related rules related to the use of the helmet also will be strictly enforced.

- The NFL will establish a "whistle blower" system so that anyone may anonymously report any incident in which a doctor is pressured to return a player to play from a concussion or that a player with a concussion is pressured to play. The NFL will investigate any such reports and take whatever action is necessary.

* * *

The NFL MTBI Committee, which was established in 1994, has launched a new study to determine if there are any long-term effects of concussions on retired NFL players.

To date, NFL-funded concussion research directed by the MTBI Committee has led to the publication of numerous peer-reviewed scientific papers and helped advance the understanding of concussions and player safety:

Analysis and Definition of Concussions

- Using 3D analysis of game video to define the velocity and orientation of impacts that cause concussion.

- Reconstructing game concussions to determine the head acceleration and forces causing injury.

- Determining game-actual impacts that cause concussion.

Development and Enforcement of Rules to Promote Player Safety

- Assisting the league in developing rules to prevent unnecessary helmet impacts and spearing of players with head-down tackles.

- Defining the positions and impacts most vulnerable to concussion and emphasizing the need for protecting quarterbacks, receivers and defensive backs.

Improved Testing and Other Helmet-Related Developments

- Defining standardized test procedures to evaluate new helmet design and providing information to the National Operating Committee on Standards for Athletic Equipment (NOCSAE) that led it to revise and update its testing standards.

- Developing test equipment and protocols for evaluating the safety performance of helmets for concussion. This was given at no cost to NOCSAE.

- Encouraging objective evaluation of new helmets designed to lower the risk of concussion and encouraging research to further improve helmets in the future.

- Sharing testing information with helmet manufacturers and NOCSAE for use in their efforts to improve helmet design and safety.

Understanding Effects and Management of Concussions

- Evaluating injury data to determine risks and treatment for concussion, repeat injury, player return to play, and other epidemiology of concussion.

- Defining post-concussion syndrome as well as investigating the recovery from the injury over time.

- Recommending that the management and evaluation of concussions be based upon the scientific medical assessment of physicians and not on arbitrary guidelines.

Role of Neuropsychological Testing

- Promoting neuropsychological testing as a useful part of player care and treatment.

  a) The NFL MTBI program was the first to formally investigate the use of neuropsychological testing in its athletes.

  b) Based on the NFL's work, neuropsychological testing now has been widely adopted by youth sports, 1,000 high schools nationally and internationally, Division I collegiate football and hockey programs, the NHL, MLS and NASCAR.

Trickle-Down Impact on College and High School Football

- Improved helmet safety for all levels of play.

- NFL MTBI Committee definition of concussions and NFL rule changes have been adopted by colleges and high schools.

Following are the members of the NFL MTBI Committee and the committee overseeing the retired player study:

MTBI Committee

Non-NFL:

- **David Viano, Dr. med., PhD** (Biomedical Engineer): *Co-Chair*; Adjunct Professor of Engineering, Wayne State University
- **Ira Casson, MD** (Neurologist): *Co-Chair*; Long Island Jewish Medical Center; Assistant Professor of Neurology, Albert Einstein School of Medicine
- **Mark Lovell, PhD** (Neuropsychologist): Director University of Pittsburgh Sports Concussion Program; Associate Professor of Neurological Surgery, University of Pittsburgh
- **Henry Feuer, MD** (Neurosurgeon): Indiana University Medical Center; Indianapolis Neurosurgical Group
- **Joseph Maroon, MD** (Neurosurgeon): Vice Chairman of Neurosurgery, University of Pittsburgh Medical Center; Clinical Professor of Neurosurgery, University of Pittsburgh
- **Joel Morgenlander, MD** (Neurologist): Professor of Neurology, Duke University Medical Center
- **Thomas Naidich, MD** (Neuroradiologist): Professor and Chief of Neuroradiology, Mount Sinai School of Medicine
- **John Powell, PhD** (Epidemiologist): Associate Professor of Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University

NFL:

- **Andrew Tucker, MD**, Sports Medicine (Ravens): Chief of Sports Medicine, Union Memorial Hospital
- **Doug Robertson, MD**, Sports Medicine (Colts)
- **Joe Waeckerle, MD**, Emergency Medicine (Chiefs): Editor Emeritus Annals of Emergency Medicine; Clinical Professor of Medicine, University of Missouri School of Medicine
- **Ronnie Barnes, ATC**, Head Athletic Trainer (Giants)
- **Rick Burkholder, ATC**, Head Athletic Trainer (Eagles)
- **Elliot Pellman, MD**, Associate Clinical Professor of Medicine and Orthopedics, Mount Sinai School of Medicine (Jets)

Retired Player Study Investigators:

- **David Viano, Dr. med., PhD** (Biomedical Engineering): Adjunct Professor of Engineering, Wayne State University
- **Mark Haacke, PhD** (Biomedical Engineering): Professor of Biomedical Engineering Wayne State University; Director of the MRI Institute for Biomedical Research
- **Thomas Naidich, MD** (Neuroradiologist): Professor and Chief of Neuroradiology, Mount Sinai School of Medicine
- **Victor Haughton, MD** (Neuroradiologist): Professor and Chief of Neuroradiology, University of Wisconsin (Madison)
- **Chi-Sing Zee, MD**, (Neuroradiologist): Director of Neuroradiology, USC School of Medicine; Professor of Radiology
- **Kathleen Finzel, MD**: Chief of Radiology, ProHEALTH Care Associates
- **Ira Casson, MD** (Neurologist): Long Island Jewish Medical Center; Assistant Professor of Neurology, Albert Einstein School of Medicine
- **Brian Hainline, MD** (Neurologist): Associate Clinical Professor, NYU School of Medicine; Chief of Neurology, ProHEALTH Care Associates
- **Joel Morgenlander, MD** (Neurologist): Professor of Neurology, Duke University Medical Center
- **Mark Lovell, PhD** (Neuropsychologist): Director University of Pittsburgh Sports Concussion Program; Associate Professor Of Neurological Surgery, University of Pittsburgh

- **Joseph Maroon, MD** (Neurosurgeon): Vice Chairman of Neurosurgery, University Pittsburgh Medical Center; Clinical Professor of Neurosurgery, University of Pittsburgh
- **Elliot Pellman, MD,** Associate Clinical Professor of Medicine and Orthopedics, Mount Sinai School of Medicine
- **Danielle LeStrange, RN** (Study Coordinator)

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TAB 3

Echocardiography characteristics in a sample of professional football players

Andrew M Tucker, Andrew E Lincoln, Elizabeth A Carter, Robert A Vogel, Anthony P Yates, Elliot J Pellman, Robert A Heyer, Peter WF Wilson. NFL Subcommittee on Cardiovascular Health, New York, NY. Medstar Research Institute, Hyattsville, MD.

Echocardiography is frequently used to screen for evidence of cardiovascular abnormalities in athletes. Cardiac dimensions that fall outside the normal ranges may be difficult to interpret because of possible confounding by large body size and extensive physical training. Normative data for echocardiograms in professional football players is generally lacking.

PURPOSE: To determine cardiac dimensions in a sample of professional football players, with special attention to wall thickness and chamber size, and to compare results to established norms for the population.

METHODS: Ninety-four National Football League players on 4 teams took part in a pilot study in 2006 examining cardiovascular risk factors. Portable echocardiograms were obtained on 90 (96%) players. Echoes for two of the teams were obtained by a contracted sonography company and read by their cardiologist. Cardiology consultants for the two other teams performed the echoes and interpreted the findings. Players were stratified into 4 position groups associated with body habitus: linemen (n=31), tight ends/linebackers (n=23), wide receivers/running backs/defensive backs (n=24), and quarterbacks/kickers (n=12). The student's t-test was used to compare mean echo parameters between linemen and the other palyer position groups combined.

RESULTS: The mean interventricular septum was 1.1 cm (range: 0.8-1.5cm) and 48 (53.3%) players had a septum thickness greater than 1.0 cm. The mean posterior wall thickness was 1.1 cm (range: 0.8-1.6cm) and the mean left ventricle internal dimension was 5.6 cm (range: 4.5- 7.1cm). Linemen had a higher mean left ventricle internal diameter than the other players (5.9 cm vs. 5.4 cm, $p<0.001$). The mean septum thicknesses among linemen and other players was 1.14cm and 1.09cm, respectively ($p=0.20$).

CONCLUSIONS: A significant percentage of professional football players who underwent screening echocardiography had wall thickness and chamber size values larger than established norms. These data reinforce the challenge of interpreting screening echocardiography in professional football players and suggest the need to develop normative data for large, physically fit individuals.

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DISCLOSURE

David C. Viano, Dr. med, Ph.D., Ira R. Casson, M.D., and Elliot J. Pellman, M.D. are members of the Mild Traumatic Brain Injury (MTBI) Committee, National Football League, New York, New York.

APPENDIX 1A

Player's Signs and Symptoms with Concussion, Reconstructed NFL Collisions and FE Modeling Results Used for the Statistical Correlation

Dir.	Case #	Position	Signs & Symptoms of Concussion																	
			Somatic Complaints						Memory Problems					Cognitive Problems			Cranial Nerve Symptoms			Others	
			Loss of Consciousness	Initial Symptoms	Headaches	Somatic Complaints	Personality Change	Fatigue	Memory Problems	Attention Problems	Info Processing Problems	AGA Delayed	RGA Delayed	Cognition Symptoms	Not Oriented to Time	Immediate Recall	Crainial Nerve Exam	Dizziness	Photophobia	Others	Return to Same Game
C	7	Quarterback															Yes	Yes			
B	38	Flanker							Yes		Yes			Yes		Yes					Yes
B	39	Tight End		Yes	Yes															Neck pain	Yes
B	57	Tight End	Yes	Yes	Yes				Yes	Yes	Yes			Yes	Yes	Yes	Yes	Yes	Yes	Seizure, not oriented to time-place	
A	69	Cornerback	Yes	Yes	Yes	Yes	Yes	Yes	Yes		Yes	Yes	Yes	Yes	Yes	Yes	Yes		Yes	Blured vision, Neck pain	
B	71	Cornerback		Yes	Yes												Yes	Yes	Yes	Vertigo	Yes
A	77	Special Team		Yes	Yes	Yes		Yes	Yes			Yes					Yes	Yes			Yes
A	84	Tight End							Yes			Yes					Yes	Yes			Yes
C	92	Center							Yes		Yes	Yes	Yes	Yes	Yes	Yes					
B	98	Wide Receiver		Yes	Yes												Yes			Nausea, Blurred vision	Yes
A	113	Wide Receiver		Yes	Yes				Yes				Yes	Yes		Yes	Yes	Yes			
A	118	Wide Receiver		Yes	Yes	Yes	Yes													Nausea	
D	123	Safety																			Yes
A	124	Kicking Unit	Yes	Yes	Yes	Yes		Yes									Yes	Yes	Yes	Sleep disorder	
B	125	Wide Receiver							Yes		Yes										Yes
D	133	Quarterback		Yes	Yes												Yes	Yes			
B	135	Tight End		Yes	Yes	Yes		Yes									Yes			Diplopia	
A	148	Quarterback		Yes	Yes												Yes	Yes			
B	155	Kicking Unit							Yes				Yes								
A	157	Ball Carrier, ret	Yes											Yes		Yes					Yes
B	162	Linebacker		Yes					Yes	Yes		Yes	Yes							Neck pain	
C	164	Quarterback		Yes		Yes														Syncope, Irratibility	Yes
A	154		Struck players without Injury/MTBI																		Yes
A	175																				Yes
A	48																				Yes
C	182																				Yes
C	59																				Yes
C	142																				Yes

APPENDIX 1B

Dir.	Case #	High Strain () Early Brain Responses				High Strain () Mid-Late Brain Responses								High Strain Rate (1/s) Early Responses			High Strain Rate (1/s) Mid-Late Responses					
		Orbital-Frontal Cortex	Temporal Lobe	Parahippocampal, Uncal Regions	Medulla	Fornix	Midbrain	Thalamus, Hypothalamus	Corpus Callosum	Fornix and/or Midbrain	Hypothalamus	Ammon plus Parahippocampal Region	Orbito-Frontal-Temporal Regions	Orbito-Frontal-Temporal Regions	Temporal	Parahippocampal	Midbrain	Thalamus	Corpus Callosum	Fornix plus Midbrain	Ammon plus Parahippocampal Region	Orbito-Frontal-Temporal Regions
C	7	0.16	0.15	0.18	0.12	0.00	0.34	0.41	0.35	0.36	0.45	0.43	0.30	68	56	66	54	79	69	78	66	60
B	38	0.20	0.20	0.33	0.10	0.46	0.60	0.45	0.37	0.45	0.28	0.50	0.40	76	90	98	98	126	128	126	103	70
B	39	0.02	0.01	0.02	0.09	0.40	0.49	0.48	0.38	0.44	0.42	0.45	0.19	23	52	7	80	77	48	88	60	70
B	57	0.23	0.13	0.15	0.04	0.31	0.45	0.55	0.36	0.56	0.48	0.24	0.31	74	73	26	94	88	98	96	82	96
A	69	0.05	0.02	0.01	0.01	0.14	0.24	0.20	0.12	0.31	0.23	0.38	0.17	28	10	5	75	40	43	52	34	32
B	71	0.11	0.13	0.14	0.06	0.43	0.42	0.36	0.42	0.47	0.40	0.45	0.25	54	33	52	67	69	56	66	70	62
A	77	0.14	0.12	0.05	0.05	0.43	0.28	0.15	0.22	0.54	0.59	0.64	0.38	53	44	54	100	58	84	88	74	50
A	84	0.39	0.29	0.14	0.14	0.50	0.50	0.85	0.66	0.85	0.75	0.70	0.62	128	131	79	154	160	162	155	143	119
C	92	0.08	0.10	0.06	0.04	0.45	0.52	0.50	0.45	0.56	0.58	0.49	0.28	48	56	23	75	82	44	72	64	57
B	98	0.28	0.26	0.28	0.13	0.38	0.28	0.44	0.41	0.51	0.60	0.68	0.30	77	86	88	68	69	114	78	101	80
A	113	0.12	0.13	0.18	0.12	0.21	0.24	0.22	0.15	0.28	0.17	0.25	0.08	28	25	38	55	35	31	36	40	31
A	118	0.20	0.16	0.02	0.16	0.11	0.52	0.48	0.12	0.48	0.21	0.22	0.40	84	80	14	138	98	49	96	105	43
D	123	0.05	0.04	0.04	0.03	0.20	0.30	0.27	0.26	0.38	0.23	0.40	0.30	32	31	15	54	60	51	47	69	46
A	124	0.24	0.21	0.21	0.04	0.26	0.44	0.29	0.50	0.52	0.49	0.61	0.40	68	48	7	86	63	96	90	72	57
B	125	0.12	0.12	0.02	0.10	0.38	0.36	0.75	0.51	0.75	0.59	0.61	0.67	87	72	10	114	139	115	160	88	137
D	133	0.05	0.04	0.01	0.05	0.15	0.12	0.30	0.35	0.19	0.14	0.17	0.22	40	23	7	45	48	59	51	70	38
B	135	0.06	0.05	0.05	0.06	0.20	0.44	0.48	0.42	0.50	0.40	0.52	0.24	72	75	20	58	60	56	64	45	51
A	148	0.14	0.11	0.10	0.05	0.10	0.12	0.12	0.21	0.12	0.22	0.24	0.18	32	21	29	30	26	35	26	32	44
B	155	0.14	0.12	0.23	0.11	0.40	0.22	0.19	0.36	0.45	0.24	0.28	0.44	86	77	57	52	30	60	52	33	42
A	157	0.08	0.07	0.03	0.11	0.26	0.33	0.35	0.22	0.62	0.37	0.35	0.35	62	49	23	166	131	98	158	111	72
B	162	0.05	0.07	0.07	0.03	0.06	0.15	0.07	0.06	0.16	0.11	0.19	0.19	21	20	24	24	21	19	24	24	26
C	164	0.08	0.04	0.05	0.11	0.32	0.21	0.36	0.30	0.36	0.25	0.34	0.30	79	57	16	56	77	106	90	62	68
A	154	0.08	0.08	0.05	0.07	0.16	0.21	0.18	0.14	0.11	0.10	0.09	0.08	23	22	20	32	21	46	21	54	37
A	175	0.22	0.18	0.11	0.08	0.24	0.19	0.33	0.26	0.25	0.28	0.2	0.3	38	33	32	41	49	43	57	38	50
A	48	0.11	0.08	0.02	0.04	0.37	0.35	0.37	0.40	0.47	0.42	0.36	0.4	60	45	13	41	80	87	80	60	63
C	182	0.18	0.15	0.05	0.11	0.25	0.29	0.18	0.24	0.27	0.26	0.2	0.27	51	48	21	60	59	70	51	68	26
C	59	0.02	0.03	0.01	0.04	0.27	0.28	0.15	0.25	0.17	0.16	0.12	0.06	22	30	9	34	45	26	52	28	32
C	142	0.04	0.03	0.02	0.02	0.03	0.03	0.04	0.04	0.03	0.04	0.05	0.05	8	6	7	10	9	15	10	11	12

COMMENTS

By collaborating with the bioengineering center at Wayne State University, the authors have conducted a unique study. They have correlated the biomechanics derived from previous three-dimensional studies from National Football League (NFL) concussions with the neurobiology of actual injured players previously reported. With finite element analysis and detailed anatomical modeling of the brain, they attempted to determine tissue level injury mechanisms at various anatomical levels. By looking at deformation and strain in modeled brain tissue, the authors discuss the signs, symptoms, and outcomes of concussion relative to the calculated generated biomechanical forces. In the case of loss of consciousness, memory and cognitive problems, and cranial nerve problems, correlative observations are made that have never been described before.

This integration of laboratory and clinical observations has not been seen before and, as the authors state, time will tell how useful and valid these observations prove to be in setting direction for the future design of helmets and protective systems. Based on some of the data provided, one helmet manufacturer has already attempted to redesign a football helmet to better protect the temporal and mandibular areas. The authors are commended for their clinical and biomechanical work.

Joseph C. Maroon
Pittsburgh, Pennsylvania

Part 9 of the Mild Traumatic Brain Injury Committee of the NFL analyzes cerebral concussion using 28 laboratory reconstructions of NFL video-documented head impacts which caused concussions in 22 athletes. Using a Wayne State University head injury model, the viscoelastic properties of the brain, including its various soft tissue components and bony support structures, were analyzed. The game video was used to calculate the characteristics of concussions, including impact velocity, direction, helmet kinematics, and other factors. The translational and rotational responses of the primary impact and the resultant displacement and deformation were calculated.

A particular contribution of this study is the correlation between the clinical manifestations of concussion and the calculated biomechanical forces of the injury. This work gives further evidence of the clinical correlation of concussion, including observations of high strain rates in deep white matter locations, which may well explain the common symptoms of concussion including loss of consciousness, cognitive memory problems, vertigo, and others. The issue of "hot spots," the regions of the brain adjacent to the primary impact site, is described and further elucidated, including the phenomena of "hot spot migration," which seems to be related to rotatory injury mechanisms.

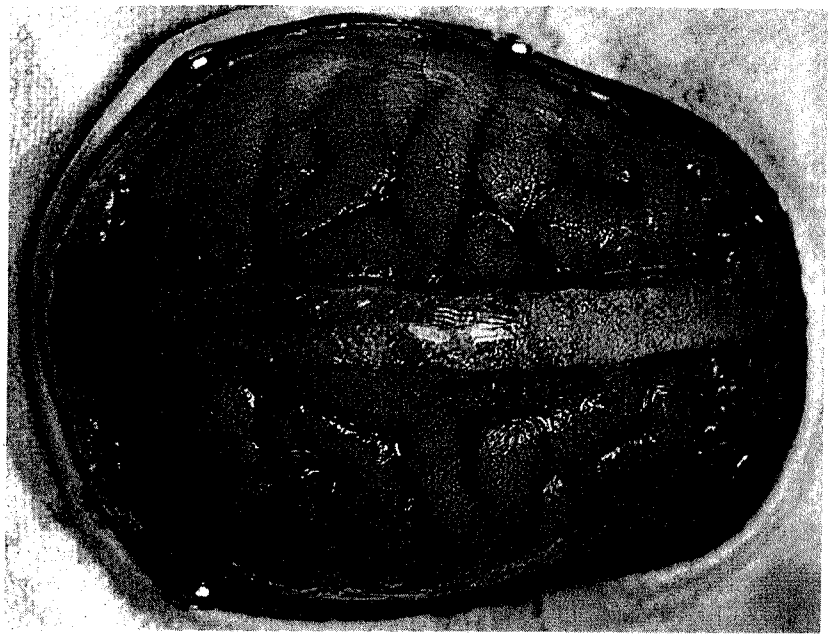
Although there are some inherent limitations of this study, as the authors acknowledge, this is novel research using sophisticated techniques analyzing local deformations of the brain to correlate with signs and symptoms of concussion. There was, for instance, no correlation with players' ability to return to play. Irrespective of the fact that the major mechanism of mild traumatic brain injury is thought to be caused by diffuse forces, focal origin may also occur. This model clearly demonstrates the deformation that occurs in these NFL impacts in a coup fashion, with transmission to remote areas, including the mesencephalon and brainstem. The authors are to be congratulated on continuing to further explore mechanisms in injury and brain responses to mild traumatic brain injury.

Julian E. Bailes
Morgantown, West Virginia

Much of this report is speculative. As the authors acknowledge, this article makes many assumptions, including those about the mechanical properties of the head and its contents, those about the responses of intracranial structures as calculated from mathematical models that incorporated laboratory findings and videotaped analyses of actual concussions, and those concerning the correlation of specific signs and symptoms in concussed patients with mechanical deformations of specific brain structures. However, the conclusions are intriguing. Future advances will allow us to decide if the findings reported here have to be modified or if they contribute to an improved way to conceptualize and prevent concussion.

Alex B. Valadka
Houston, Texas

Lexan polycarbonate calvarium fixed to a monkey skull. Lexan, a high-quality polymer, replaced the Lucite utilized by Pudenz and Shelden (1946) for the visualization of the brain deformation during trauma. (From, *Gosch HH, Gooding E, Schneider RC: An experimental study of cervical spine and cord injuries.* ***J Trauma*** *12:570–576, 1972.)*



TAB 5

NFL Subcommittee on Cardiovascular Health Membership and Affiliations
As of November 1, 2007

Andrew Tucker, M.D.,Co-Chairman	Head Team Physician, Baltimore Ravens; President, NFL Team Physicians Society; Member NFL Injury and Safety Panel and NFL MTBI Subcommittee; Medical Director Sports Medicine, Union Memorial Hospital, Baltimore
Robert A. Vogel, M.D., Co-Chairman	Professor of Medicine, Director of Clinical Vascular Biology, University of Maryland School of Medicine
Thomas W. Allen, D.O.	Emeritus Professor of Medicine, Oklahoma State University
Lon W. Castle, M.D.	The Cleveland Clinic Foundation; Staff Electrophysiologist, Director of Electrophysiology, Lakewood Hospital, OH
Robert A. Heyer, M.D.	Team Physician, Internal Medicine, Carolina Panthers; Chief of Pulmonary/Critical Care Medicine, Department of Internal Medicine, Carolinas Medical Center
Marty Lauzon, ATC	Head Athletic Trainer, Cleveland Browns
Ron Medlin, ATC	Head Athletic Trainer, Atlanta Falcons
Patrick J. Strollo, Jr., M.D.,	Medical Director, University of Pittsburgh Sleep Medicine Center
Peter W.F. Wilson, M.D.	Department of Medicine, Cardiology Division Emory School of Medicine, Atlanta, GA
Anthony P. Yates, M.D.	Medical Director, Team Physician, Pittsburgh Steelers; University of Pittsburgh Medical Center
Ex officio- Elliot Pellman, M.D.	NFL Medical Advisor; Medical Director, New York Jets; Member, NFL Injury and Safety Panel, NFL MTBI Subcommittee, NFL Foot and Ankle Subcommittee; Medical Director, ProHealth Care Associates; Associate Professor of Medicine and Orthopaedics, Mount Sinai School of Medicine

TAB 6

NFL INJURY & SAFETY COMMITTEE

The NFL's Injury and Safety Committee is in its 15th year of existence, having been formed in 1993. Dr. John Bergfeld served as the first chair of the committee and was succeeded by Dr. Elliott Hershman, team physician for the New York Jets, in 1999. Dr. Hershman continues to lead the committee. The committee is comprised of health care professionals whose primary objective is to provide medical advice to the NFL regarding the care and treatment of NFL players.

The committee created and manages an injury surveillance system, which was designed to provide the NFL with accurate information concerning the types and rates of injuries sustained by NFL players. A committee member serves as statistician for this report and furnishes a copy of the report mid-year and year end to the NFL's Competition Committee.

The committee also conducts epidemiological studies on an annual basis. Several committee members are affiliated with NFL member clubs by serving as team physicians, while other committee members have no affiliation with NFL member clubs. The vast majority of committee members hold medical degrees and have more than 250 years of health care and medical experience. The committee also oversees the work of a number of subcommittees, which are as follows:

- Mild Traumatic Brain Injury
- Foot and Ankle
- Cardiovascular
- Grant Review

The committee meets on a quarterly basis and holds it principal meeting during the annual Scouting Combine in Indianapolis, Indiana. The current committee membership can be found below.

Robert Anderson, M.D.
John Bergfeld, M.D.
James P. Bradley, M.D.
Charles L. Brown, Jr., M.D.
James T. Collins, Jr., ATC
Elliott Hershman, M.D., Chair
Robert J. Johnson, M.D.
John Lombardo, M.D.
Elliott Pellman, M.D.
John Powell, Ph.D., ATC
Kurt Spindler, M.D.
William Tessendorf, ATC
Andrew Tucker, M.D.
Edward Wojtys, M.D.

NFL Injury and Safety Panel Membership and Affiliations
As of October 30, 2007

Elliott Hershman, M.D., Chairman	Team Physician, New York Jets Associate Director of Orthopaedics, Lenox Hill Hospital; Chief of Orthopaedics, ProHealth Care Associate
Robert Anderson, M.D.	Team Physician/Orthopedics, Carolina Panthers; Co-Chairman, NFL Foot and Ankle Subcommittee
John Bergfeld, M.D.	Team Physician/Orthopedics, Cleveland Browns; Former Chief of Sports Medicine, The Cleveland Clinic Foundation
James P. Bradley, M.D.	Team Physician/Orthopedics, Pittsburgh Steelers; Member NFL Foot and Ankle Subcommittee; Associate Professor of Orthopaedics, University of Pittsburgh Medical Center
Charles L. Brown, Jr., M.D.	Professor of Medicine, LSU Health Sciences Center; Former Team Physician/Internal Medicine, New Orleans Saints
James T. Collins, Jr., ATC	Head Athletic Trainer, San Diego Chargers
Robert J. Johnson, M.D.	Professor of Orthopaedics & Rehabilitation, University of Vermont
John Lombardo, M.D.	NFL Advisor on Anabolic Steroids and Related Substances
Elliot Pellman, M.D.	NFL Medical Advisor; Medical Director, New York Jets; Member NFL MTBI Subcommittee; Member NFL Subcommittee on Cardiovascular Health; Medical Director, ProHealth Care Associates; Associate Professor of Medicine and Orthopaedics, Mount Sinai School of Medicine
John Powell, Ph.D., ATC	NFL Consultant — Injury Studies; Med Sports Systems; Epidemiologist: Associate Professor of Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University

NFL Injury and Safety Panel Membership and Affiliations
As of October 30, 2007 (Continued)

Kurt Spindler, M.D.	Vanderbilt Orthopaedic Institute; Professor of Orthopaedics, Vanderbilt University
William Tessendorf, ATC	VP of Medical Services/Head Athletic Trainer, Baltimore Ravens
Andrew Tucker, M.D.	Team Physician, Baltimore Ravens; President, NFL Team Physicians Society; Co-Chairman, NFL Subcommittee on Cardiovascular Health; Member NFL MTBI Subcommittee; Chief Sports Medicine, Union Memorial Hospital
Edward Wojtys, M.D.	Med Sport; Team Physician, University of Michigan; Professor of Orthopaedics, University of Michigan

TAB 7

NFL Foot and Ankle Subcommittee Membership and Affiliations
As of October 31, 2007

Robert Anderson, M.D., Co-Chairman	Team Physician/Orthopedics, Carolina Panthers; Member NFL Injury and Safety Panel; OrthoCarolina
Michael Coughlin, M.D., Co Chairman	Boise State University; Professor of Orthopaedics, University of Oregon
Ronnie Barnes, ATC	Head Athletic Trainer, New York Giants
Kevin Bastin, ATC	Head Athletic Trainer, Houston Texans
Tony Egues	Director of Operations, Miami Dolphins; Former Head Equipment Manager, Miami Dolphins
Patrick McKenzie, M.D.	Team Physician/Orthopedics, Green Bay Packers, Member NFL Injury and Safety Panel; Orthopedic Associates of Green Bay
Elliot Pellman, M.D.	NFL Medical Advisor, Medical Director, New York Jets; NFL Medical Consultant; Member NFL Injury and Safety Panel, Member NFL MTBI Subcommittee, Member NFL Cardiovascular Health Subcommittee; Medical Director, ProHealth Care Associates; Associate Professor of Medicine and Orthapaedics, Mount Sinai School of Medicine
John Powell, Ph.D., ATC	Statistician/NFL Consultant — Injury Studies; Med Sports Systems; Epidemiologist: Associate Professor of Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University
Mike Ryan, ATC PT	Head Athletic Trainer, Jacksonville Jaguars
Joe Skiba	Assistant Equipment Manager, New York Giants

TAB 8

2003-2007 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Year	Amount
2003	$695,993.00
2004	$862,825.00
2005	$1,182,900.00
2006	$1,154,875.00
2007	$1,230,073.00
Research Grants Total	**$5,126,666.00**

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

2003	$65,000.00
2004	$92,000.00
2005	$92,000.00
2006	$92,000.00
2007	$112,000.00
Education Grants Total	**$453,000.00**

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury sub-committee

2003	$200,000.00
2004	$180,000.00
2005	$200,000.00
2006	$345,900.00
2007	$100,000.00
MTBI Grants Total	**$1,025,900.00**

TOTAL GRANT AMOUNT 2003-2007 **$6,605,566.00**

2003 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Institution/Organization	Amount	Description of Medical Research
1.) Hospital for Special Surgery	$74,000.00	**Arthritis**--The use of an autogenous fibrin clot in the treatment of full-thickness chondral defects.
2.) Johns Hopkins University	$80,000.00	**Cardiac**--Effect of long-term training on right ventricular regional function in athletes.
3.) Massachusetts General Hospital	$100,000.00	**PCL**--Prevention of osteoarthritis following PCL injury: seeking an optimal technique for PCL reconstruction.
4.) University of Maryland-Baltimore	$86,019.00	**Neuro**--Assessment of brain blood flow following concussion.
5.) University of Wisconsin-Madison	$59,000.00	**Arthritis**--The effect of cytoprotective agents on the metabolic activity of articular cartilage exposed to defined levels of thermal and mechanical stress.
6.) Washington University School of Medicine	$14,000.00	**Surgery**--Mechanical properties of arthroscopic knots.
7.) Michigan State University	$50,569.00	**Injury Risk**-Relationship Among Players Risk Factors and Injuries in Youth Football.
8.) University of Florida	$89,000.00	**Muscle**-Can Muscle Heating Attenuate Atrophy Caused by Immobilization.
9.) University of Michigan	$82,158.00	**ACL**- ACL Injury Mechanisms
10.) University of Pittsburgh	$61,247.00	**Shoulder**- Study of Proprioception, Neuromuscular Activation, and Functional Characteristics Following Arthroscopic Thermal Capsularaphy v. Open Surgical Anterior Anterior Capsulorraphy in Chronic Recurrent
Research Grants Sub-Total	**$695,993.00**	

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

1.) National Athletic Trainers' Association (NATA)	$25,000.00	**Education**- Funding for Non-Medical Research and Scholarship Fund.
2.) NFL Physicians Society	$25,000.00	**Education**- Funding for the NFLPS Annual Scientific Meeting.
3.) Professional Football Athletic Trainers Society (PFATS) Foundation	$15,000.00	**Education**- Funding for Ethnic Minority Scholarship Program.
Education Grants Sub-Total	**$65,000.00**	

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury (MTBI) sub-committee as part of NFL's overall MTBI project

1.) Wayne State University Sports Lab	$200,000.00	Concussion Studies
MTBI Grants Sub-Total	**$200,000.00**	
OVERALL TOTAL	**$960,993.00**	

2004 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Institution/Organization	Amount	Description of Medical Research
1.) Children's Hospital	$89,500.00	**ACL**--Stimulation of Intra-Articular Defect Healing.
2.) Massachusetts General Hospital	$125,000.00	**Shoulder**--In-Vivo Fluoroscopic Analysis of Pre and Post-Operative Shoulder Kinematics.
3.) University of Buffalo (SUNY)	$125,000.00	**Ankle**--Effects of Electrical Stimulation on Time to Recovery Following Grade I & II Inversion Ankle Sprains in Intercollegiate and Professional Athletes.
4.)University of California Los Angeles	$113,636.00	**PCL**--PCL Reconstruction in Conjunction with Reconstructions of the Posterolateral Structures.
5.) University of South Florida College of Medicine	$19,312.00	**Heat Illness**--Prevention and Early Detection of Heat Illness.
6.) University of Wisconsin Medical School	$124,658.00	**Hamstring**--Effects of a Previous Strain Injury on Hamstring Muscle Mechanics During Sprinting: Implications for Preventing Re-Injury.
7.) Vanderbilt University Medical Center	$125,000.00	**ACL**--Prospective Multicenter Study of ACL Reconstruction Outcomes: Activity Level and Predictors.
8.) Cardiovascular Consultants	$50,865.00	**Heart**- A Comparison of Electrocardiographic and Echocardiographic Findings in Elite American Football Players.
9.) Steadman Hawkins Sports Medicine Foundation	$89,854.00	**Shoulder**-Mechanical Determinants of Upper Extremity Motion and Glenohumeral Stability During Dynamic Activities.
Research Grants Sub-Total	**$862,825.00**	

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

1.) NATA Research and Education Foundation	$30,000.00	**Education**- Funding for Non-Medical Research and Scholarship Fund.
2.) Professional Football Athletic Trainers Society Foundation	$32,000.00	**Education**- Funding for Ethnic Minority Scholarship Program.
3.) NFL Physicians Society	$30,000.00	**Education**- Funding for the NFLPS Annual Scientific Meeting.
Education Grants Sub-Total	**$92,000.00**	

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury (MTBI) sub-committee as part of NFL's overall MTBI project

1.) Wayne State University Sports Lab	$180,000.00	Concussion Studies
MTBI Grants Sub-Total	**$180,000.00**	
OVERALL TOTAL	**$1,134,825.00**	

2005 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Institution/Organization	Amount	Description of Medical Research
1.) Cleveland Clinic	$125,000.00	**ACL**-Effect of Neuromuscular Fatigue on Anterior Cruciate Ligament Injury Risk in NFL Athletes.
2.) Washington University School of Medicine at St. Louis	$18,400.00	**Meniscus Repair**- Meniscal Repair Outcome in Association with Anterior Cruciate Ligament Reconstruction: Two Year Follow-up of a Prospective Database.
3.) Ohio State University	$37,500.00	**Metabolic Syndrome**- Metabolic Syndrome in Football Lineman.
4.) University of Florida College of Medicine	$97,000.00	**Cooling Shoulder Pad**- Intermittent Cold and Dry Air Underneath Football Shoulder Pads as a Method to Assist in Temperature Homeostasis: Evaluation of Efficacy.
5.) University of Michigan	$125,000.00	**MRI**- The Effect of Geographic Bone Bruises on Biological Markers and MRI Findings of Osteoarthritis After Anterior Cruciate Ligament Injury.
6.) University of Iowa	$87,000.00	**ACL**- ACL Tunnel Variation: A Radiographic Analysis.
7.) Drexel University	$125,000.00	**MRI Dx L Spine**- MRI Diagnostics for Degenerative Disc Disease Based on Bound Water.
8.) University of Virginia	$125,000.00	**Ankle**- Biomechanics of Chronic Ankle Instability.
9.) University of California, San Diego	$125,000.00	**Articular Cartilage**- Treatment of Advanced Cartilage Defects.
10.) Steadman Hawkins Sports Medicine Foundation	$125,000.00	**Shoulder/Knee/Hip**- Mechanical Determinants of Upper Extremity Motion and Gleno-Humeral Stability During Dynamic Activities Part II-Virtual Shoulder Model to Rehabilitation Exercises.

11.) Michigan State University School of Medicine	$125,000.00	**High Ankle Sprain**- An Experimental Model and In Situ Assessment Tool for the Study of "High Ankle" Sprains.
12.) University of Pittsburgh	$68,000.00	**Lipid Profiles**- Evaluation of Lipid Profiles Inflammatory Markers and the Use of Omega-3 EFA in Professional Football Players.
Research Grants Sub-Total	**$1,182,900.00**	

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

1.) National Athletic Trainer's Association (NATA) Research Foundation	$30,000.00	**Education**- Funding for Non-Medical Research and Scholarship Fund.
2.) NFL Physicians Society	$30,000.00	**Education**- Funding for the NFLPS Annual Scientific Meeting.
3.) Professional Football Athletic Trainers Society (PFATS) Foundation	$32,000.00	**Education**- Funding for Ethnic Minority Scholarship Program.
Education Grants Sub-Total	**$92,000.00**	

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury (MTBI) sub-committee as part of NFL's overall MTBI project

1.) Wayne State University Sports Lab	$45,000.00	**Mouth guards**- Development of a Mandible and Teeth for the Hybrid III Dummy Head to Test the Influence of Mouth guards on Risk of Concussions.
2.) Institute for Injury Research	$155,000.00	**Concussion**- Comparing Injuries in the NFL Animal Model with those from an Established Head Injury Model by Marmarou.
MTBI Grants Sub-Total	**$200,000.00**	
OVERALL TOTAL	**$1,474,900.00**	

2006 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Institution/Organization	Amount	Description of Medical Research
1.) Cincinnati Children's Hospital	$85,780.00	**ACL**- Pre-Season Football Combine Testing To Isolate Neuromuscular Deficits Predictive of ACL Injury and Re-injury Risk.
2.) University of California Los Angeles	$125,000.00	**ACL**-Biomechanical Studies of Single v. Double Bundle ACL Reconstruction.
3.) Wake Forest University	$85,269.00	**Meniscus**- Creation of a Biologic Meniscus Replacement Scaffold.
4.) University of South Florida	$123,650.00	**Heat Stress**- Heat Illness Symptoms and Physiology in Exercising Athletes Under Heat Stress.
5.) Vanderbilt University	$124,778.00	**Rotator Cuff Tears**- Features to Predict Success with Nonoperative Treatment of Patients with Rotator Cuff Tears.
6.) Harvard University	$125,000.00	**Cardiac Disease**- Gene-based Diagnosis of Cardiac Diseases that Predispose to Sudden Death on the Athletic Field.
7.) University of Pennsylvania	$125,000.00	**Meniscus**- In Vivo Meniscus Repair with Anisotropic Biodegradable Nanofibrous Scaffolds.
8.) Oregon Health and Science University	$73,700.00	**Cartilage Injury**-Regeneration of Full Thickness Articular Cartilage Injury.
9.) Cleveland Clinic	$124,848.00	**Cartilage Injury**-Intraarticular Injection for Treatment of Impact Cartilage Injury in a Rabbit Model.
10.) Washington University	$50,667.00	**Knee Ligament**-Effects of Smoking on Knee Ligament Healing.
11.) University of Wisconsin-Madison	$111,183.00	**ACL**-The Effect of Early Hyaluronic Acid Delivery on the Development of Articular Cartilage Lesion in a Sheep Model.
Research Grants Sub-Total	**$1,154,875.00**	

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

1.) National Athletic Trainer's Associati (NATA) Research Foundation	$30,000.00	**Education**- Funding for Non-Medical Research and Scholarship Fund.
2.) NFL Physicians Society	$30,000.00	**Education**- Funding for the NFLPS Annual Scientific Meeting.
3.) Professional Football Athletic Train Society (PFATS) Foundation	$32,000.00	**Education**- Funding for Ethnic Minority Scholarship Program.
Education Grants Sub-Total	**$92,000.00**	

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury (MTBI) sub-committee as part of NFL's overall MTBI project

1.) Wayne State University Sports Lab	$170,000.00	Helmet and Mouth Guard- Concussion Studies
2.) Biokinetics Association	$175,900.00	Concussion Studies
MTBI Grants Sub-Total	**$345,900.00**	
OVERALL TOTAL	**$1,592,775.00**	

2007 NFL CHARITIES MEDICAL RESEARCH GRANTS

Scientific Research Grants- reviewed by the NFL Charities grants sub-committee

Institution/Organization	Amount	Description of Medical Research
1.) Cleveland Clinic Foundation	$125,000.00	**Meniscus Repair**-Activation of Adult Human Stem Cells for Meniscus Repair.
2.) University of Wisconsin-Madison	$125,000.00	**Hamstring**- Rehabilitation of Acute Hamstring Strains: Effects of Treatment on Muscle Morphology, Running Mechanics and Re-Injury Potential.
3.) Regents of the University of California (UCLA)	$125,000.00	**Biomechanics**-Biomechanical Studies of a New Double Bundle PCL and PLC Reconstruction.
4.) University of Pennsylvania	$125,000.00	**Disc Strain and Tears**- Noninvasive Quantification of Disc Strain and Propagation of Annular Tears.
5.) Cincinnati Children's Hospital Medical Center	$105,073.00	**ACL**- ACL Reconstruction and Athletes: Strength, Knee Mechanics, and Outcome.
6.) Columbia University	$125,000.00	**ACL**-Controlled Growth Factor Delivery for Interface Tissue Engineering and the Biological Fixation of ACL Reconstruction Grafts.
7.) Princeton University	$125,000.00	**Orthopedics**- Osseo integration of Soft Tissue Prostheses.
8.) University of Florida	$125,000.00	**ACL**-The Effect of Plyometric Exercise Intensity on Function & Articular Cartilage Metabolism After ACL Reconstruction.
9.) Massachusetts General Hospital	$125,000.00	**Cartilage Repair**-Engineering Human Tissue for Cartilage Repair.
10.) Children's Hospital of Pittsburgh Foundation	$125,000.00	**Orthopedics/ Sports Medicine**- The Use of Relaxin to Improve Sports Related Strain Injury in Skeletal Muscle.
Research Grants Sub-Total	**$1,230,073.00**	

Education Grants- Sports-related education grants reviewed by NFL Injury and Safety Panel

1.) National Athletic Trainer's Association (NATA) Research Foundation	$30,000.00	**Education**- Funding for Non-Medical Research and Scholarship Fund.
2.) NFL Physicians Society	$50,000.00	**Education**- Funding for the NFLPS Annual Scientific Meeting.
3.) Professional Football Athletic Trainers Society (PFATS)	$32,000.00	**Education**- Funding for Ethnic Minority Scholarship Program.
Education Grants Sub-Total	**$112,000.00**	

MTBI Grants- Scientific research grants reviewed by Mild Traumatic Brain Injury (MTBI) sub-committee as part of NFL's overall MTBI project

1.) Wayne State University Sports Lab	$25,000.00	Mouth guard and Helmet Testing
2.) Institute for Injury Research Bloomfield Hills, Michigan	$75,000.00	Concussions- Studying Protein Deposits in the Brain After Concussions
MTBI Grants Sub-Total	**$100,000.00**	
OVERALL TOTAL	**$1,442,073.00**	

TAB 9

NATIONAL FOOTBALL LEAGUE

ROGER GOODELL
Commissioner

MEMORANDUM

To: **Chief Executives**
Club Presidents
General Managers
Head Coaches
Team Physicians
Head Athletic Trainers

From: **Commissioner Goodell**

Date: **August 10, 2007**

Re: **Materials re Management of Concussions**

This will follow up on our discussions of this issue at the League Meetings in March and May. As we advised at that time, we were pursuing a number of initiatives, including holding a medical and scientific conference devoted to the subject of concussions. Based on the discussions at the conference, we have, in conjunction with our medical committee and the NFLPA medical advisor, developed additional material relating to the management of concussions. This information is attached and will be made public on Monday.

We will send this material to each club in sufficient quantities to permit you to distribute it to your players, your coaching, medical, athletic training, and public relations staffs, and other interested parties within your organization. The specific items are as follows:

First, a letter to all players from Gene Upshaw and me reviewing the work that has been done over the past several months and outlining important steps to continue managing concussions in a conservative way that emphasizes player safety.

Second, an informational pamphlet for players and others describing the symptoms of concussions, what they should look for in themselves or in a teammate, and what families should know about concussions.

Third, a summary developed by our medical committees of the key medical factors that will be used in making decisions regarding when it is safe for a player to

return to practice or play following a concussion. This summary confirms that concussions will continue to be managed in a conservative way, and that medical issues will take precedence over competitive concerns.

Fourth, we have established a toll-free hotline to allow people to report any instance in which a player who has suffered a concussion is being pressured to return to practice or play against medical advice.

Fifth, a copy of the press release that we will issue on this subject.

Please ensure that this information is shared with all coaches, players, and members of your athletic training staffs.

NFL PLAYERS
ASSOCIATION

August 2007

Dear NFL Player:

In the past year, we and our medical advisors have focused considerable time and attention on the care of players who suffer concussions. We would like to tell you about a number of steps that we have taken.

First, this past June, we held a medical and scientific conference devoted entirely to the subject of concussions. The conference was attended by physicians and athletic trainers from every NFL team, as well as by NFLPA President Troy Vincent, executive committee members Mark Bruener and Ernie Conwell, and NFLPA Medical Advisor Dr. Thom Mayer. The conference reviewed the most up-to-date medical and scientific research, and included presentations by doctors and scientists from within and outside the NFL.

Second, we have prepared a short informational pamphlet for all NFL players and their families concerning concussions. This pamphlet tells you what to look for in yourself, a teammate, or a family member. We encourage you to read this pamphlet and to talk with your team doctor or athletic trainer if you believe you have the symptoms of a concussion, or see symptoms in a teammate.

Third, we have continued to emphasize with clubs, including coaches, that player safety must take priority over competitive issues. We have established a hotline to allow anyone to report confidentially a case where a player has had a concussion and is being forced to practice or play against medical advice. The number for the hotline is

1-888-NFL-MTBI (1-888-635-6824)

This hotline will be available to you at all times.

Fourth, to share best practices throughout the League, the medical committee and Dr. Mayer have prepared a summary of the key factors used by team doctors and athletic trainers in

deciding when it is safe for a player who has had a concussion to return to practice or play. These factors have been identified in medical studies and are used by team medical staffs today. The goal is to continue to manage concussions conservatively and in a way that emphasizes player safety.

Fifth, we are expanding the use of neuropsychological testing so that all players will have a baseline test performed each year. This test is one way of determining the extent to which the brain has recovered following a concussion. Players who have been removed from a game and do not return due to a concussion will be re-tested as part of the medical staff's treatment of the player, and to assist in determining when players can return to practice and play.

Sixth, the League will continue closely to enforce rules that promote player safety. This year, for example, the requirement that chin straps on helmets be completely and properly buckled will be strictly enforced. Rules relating to the use of helmets in play will also be strictly enforced.

Seventh, we are continuing to research all elements of concussions, with a particular focus on long-term effects. The League has expanded the membership of its medical committee, and Dr. Mayer, the NFLPA Medical Advisor, will remain closely involved in these ongoing projects.

We wish you a healthy and successful 2007 season.

ROGER GOODELL GENE UPSHAW

What is a Concussion? It's More Than a "Ding."

Concussions are caused by a hard hit to the head. The hit is typically from another player's helmet, shoulder pad or knee or from a fall to the ground. The effects usually last a short time, but it's important that they are treated properly and promptly by you, your team doctors and your athletic trainers.

You shouldn't decide if it is just a "ding." Instead, you should report any symptom from the list below to your medical staff. This will help determine whether or not you have had a concussion.

"Ding" is not a medical term. It doesn't describe specific symptoms and won't help your medical staff. Try to describe your symptoms from the following list.

How do I know if I have had a concussion?

These are some of the symptoms you may experience immediately or within a few days of having a concussion. Every concussion is different, players may react differently and not all players will experience the same symptoms.

The most common symptoms are:

- **Imbalance:** You may feel a change in your sense of balance, feel dizzy, or unsteady on your feet.
- **Headache:** This is the most common symptom with concussion. It may be mild to severe in intensity and you may feel like there is pressure in your head. This may be accompanied by nausea and vomiting.
- **Confusion:** You may be confused about where you are, about a play, the score or game situation. You may not remember the play you are running.
- **Memory loss:** You may lose memory about things that happened BEFORE or AFTER you were hit. You may not remember what happened during the play or the quarter before your collision. Or you can't remember what happened on the field or on the sidelines after your hit. You may ask the same questions over and over again.
- **Loss of consciousness:** You may black out or get knocked out, even for a second or two.
- **Vision change:** You may become sensitive to light, have blurred vision, double vision or feel like lights seem brighter. Some athletes also report "seeing stars" or other objects following a hard hit.
- **Hearing change:** You may feel a change in your hearing so sounds suddenly seem very loud, or you may hear a high pitch tone in your ears.
- **Mood change:** You may have a sudden change in your mood or a teammate may notice a change in your mood following a collision. For example, you might suddenly start to laugh or cry for no reason. You may not know this is happening but teammates, coaches, or the medical staff may see it. After a game, you may feel more irritable, anxious, or cranky than usual.
- **Fatigue:** You may feel more exhausted than usual after a game when you had a hard hit to the head. Some athletes report that they need to sleep many more hours after a concussion.
- **Malaise:** You may just "not feel right" but can't point to a specific problem.

Not every hard hit to the head leads to a concussion and whether or not you have a concussion can only be determined by your team doctors and athletic trainers. If the team medical staff does not know that you are injured, it can't help you!

You may not always recognize your symptoms. But your teammates, coaches or family members may see a difference in you that you don't. If someone sees a change in you, take it seriously and report it to your team medical staff.

What should you report to your team medical staff?

Don't try to make a diagnosis yourself. A concussion needs to be diagnosed by your team medical staff. If you have had a hard hit to the head and have symptoms, you should immediately report your symptoms to your team doctors and athletic trainers, who will conduct a thorough evaluation on the sideline.

On occasions, symptoms from concussion will be more obvious or noticeable hours after the impact. Symptoms should be reported to your medical staff regardless when you become aware of them.

If you see any symptoms in a teammate, tell your team doctors or athletic trainers because your teammate may not always realize he has had a concussion.

When should I return to play following a concussion?

After a concussion, all return to play decisions should be made by your team medical staff. These decisions should never be made by players or coaches. You should be free of symptoms before you return to play.

If you have had a concussion and feel you are being pressured to return too quickly, or think that is happening to a teammate, you can call 1-888-635-6824 to make a confidential report.

Am I at risk for further injury if I have had a concussion?

Current research with professional athletes has shown that you should not be at greater risk of further injury once you receive proper medical care for a concussion and are free of symptoms.

If I have had more than one concussion, am I at increased risk for another injury?

Current research with professional athletes has not shown that having more than one or two concussions leads to permanent problems if each injury is managed properly. It is important to understand that there is no magic number for how many concussions is too many.

Research is currently underway to determine if there are any long-term effects of concussion in NFL athletes.

What is the treatment for a concussion?

The treatment for concussion usually consists of rest. Medication may sometimes be prescribed by your team doctors for symptoms such as headaches and dizziness. If your team doctor prescribes medication, be sure to follow his directions and those provided with the prescription.

It is important that you avoid drinking alcohol. Also, if you intend to use over-the-counter medication, vitamins or supplements, tell your team doctors. They may want you to stop taking them.

You should avoid caffeine and make sure that you do not become dehydrated.

Summary of Return-to-Play Considerations

Based upon the June 2007 Conference on NFL player health and safety, the MTBI Committee reaffirms the following:

- Team physicians and athletic trainers should continue to exercise their clinical judgment and expertise in the treatment of each player who sustains a concussion and to avail themselves of additional expert consultation when clinically indicated. We encourage team physicians and athletic trainers to continue to take a conservative approach to treating concussion.

- Team physicians and athletic trainers should continue to take the time to obtain a thorough history, including inquiring specifically about the common symptoms of concussion, and to conduct a thorough neurological examination, including mental status testing at rest and post-exertional testing, before making return to play decisions in a game or practice.

- The essential criteria for consideration of return to play remain unchanged. The player should be completely asymptomatic and have a normal neurologic examination, including mental status testing at rest and post-exertional testing, before being considered for return to play.

- Team physicians and athletic trainers should continue to take into account certain symptoms and signs that have been associated with a delayed recovery when making return to play decisions. These include confusion, problems with immediate recall, disorientation to time, place and person, anterograde and retrograde amnesia, fatigue, blurred vision and presence of three or more signs and symptoms of concussion.

- If the team medical staff determines a player was unconscious, the player should not be returned to the same game or practice.

- Team physicians and athletic trainers should continue to consider the player's history of concussion, including number and time between incidents, type and severity of blow, and time to recover.

- Team physicians and athletic trainers should continue to educate players about concussion and to emphasize the need for players to be forthright about physical and neurological complaints associated with concussion. Player input assists the medical staff and athletic trainers to render appropriate care.

NEWS

NATIONAL FOOTBALL LEAGUE
280 Park Avenue, New York, NY 10017
(212) 450-2000 * FAX (212) 681-7573
WWW.NFLMedia.com

Joe Browne, Executive Vice President-Communications
Greg Aiello, Senior Vice President-Public Relations

FOR IMMEDIATE RELEASE
August 14, 2007

NFL OUTLINES FOR PLAYERS STEPS TAKEN TO ADDRESS CONCUSSIONS

The National Football League has outlined for NFL players, coaches, and staff members the recent steps that have been taken to address the management of concussions in the NFL.

"We want to make sure all NFL players, coaches and staff members are fully informed and take advantage of the most up-to-date information and resources as we continue to study the long-term impact of concussions," Commissioner **ROGER GOODELL** said. "Because of the unique and complex nature of the brain, our goal is to continue to have concussions managed conservatively by outstanding medical personnel in a way that clearly emphasizes player safety over competitive concerns."

The recent steps were outlined in a memo that will be sent to all NFL players and team personnel with other information. They include the following:

- The NFL held a medical conference in June on the subject of concussions. It was attended by team physicians and athletic trainers from every NFL team and by active players and medical representatives of the NFL Players Association. The conference reviewed the current medical and scientific research and included presentations by doctors and scientists from within and outside the NFL.

- An informational pamphlet on concussions for NFL players and their families has been prepared (below). It describes the symptoms of concussions, what NFL players should look for in themselves or a teammate if they suspect a possible concussion, and what NFL families should know about concussions.

- The establishment of a hotline to report information on a confidential basis about an NFL player being forced to practice or play against medical advice. The hotline underscores the league's priority on player safety over competitive concerns.

- The NFL and NFLPA medical advisors prepared a summary of key factors in deciding when NFL players can safely return to the same game or practice. These factors have been identified in medical studies and are used by NFL team medical staffs. They emphasize that concussions in the NFL should continue to be managed conservatively and include the following specific points:

    1. The player should be completely asymptomatic and have normal neurological test results, including mental status testing at rest and after physical exertion, before returning to play.

    2. Symptoms to be taken into account include confusion, problems with immediate recall, disorientation to time, place and person, anterograde and retrograde amnesia, fatigue, and blurred vision.

3. If an NFL player sustains a loss of consciousness, as determined by the team medical staff, he should not return to the same game or practice.

4. NFL team physicians and athletic trainers will continue to exercise their medical judgment and expertise in treating concussions, including considering any history of concussions in a player.

- Neuropsychological testing has been expanded for all NFL players. NFL players who have been removed from a game due to a concussion will be re-tested during the season as part of the medical staff's treatment of the player and to assist in determining when players can return to practice and play. Each club will select the neuropsychological testing provider of its choice.

- Player safety rules relating to the use of the helmet will continue to be closely enforced. This will include strict enforcement of the requirement that chin straps on helmets be completely and properly buckled so that the helmet provides the maximum protection.

- The NFL will continue to research and study all elements of concussions with a particular focus on long-term effects.

(NFL PLAYER CONCUSSION PAMPHLET)

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Concussions are caused by a hard hit to the head. The hit is typically from another player's helmet, shoulder pad or knee or from a fall to the ground. The effects usually last a short time, but it's important that they are treated properly and promptly by you, your team doctors and your athletic trainers.

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"Ding" is not a medical term. It doesn't describe specific symptoms and won't help your medical staff. Try to describe your symptoms from the following list.

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The most common symptoms are:

- **Imbalance:** You may feel a change in your sense of balance, feel dizzy, or unsteady on your feet.
- **Headache:** This is the most common symptom with concussion. It may be mild to severe in intensity and you may feel like there is pressure in your head. This may be accompanied by nausea and vomiting.
- **Confusion:** You may be confused about where you are, about a play, the score or game situation. You may not remember the play you are running.
- **Memory loss:** You may lose memory about things that happened BEFORE or AFTER you were hit. You may not remember what happened during the play or the quarter

before your collision. Or you can't remember what happened on the field or on the sidelines after your hit. You may ask the same questions over and over again.

- **Loss of consciousness:** You may black out or get knocked out, even for a second or two.
- **Vision change:** You may become sensitive to light, have blurred vision, double vision or feel like lights seem brighter. Some athletes also report "seeing stars" or other objects following a hard hit.
- **Hearing change:** You may feel a change in your hearing so sounds suddenly seem very loud, or you may hear a high pitch tone in your ears.
- **Mood change:** You may have a sudden change in your mood or a teammate may notice a change in your mood following a collision. For example, you might suddenly start to laugh or cry for no reason. You may not know this is happening but teammates, coaches, or the medical staff may see it. After a game, you may feel more irritable, anxious, or cranky than usual.
- **Fatigue:** You may feel more exhausted than usual after a game when you had a hard hit to the head. Some athletes report that they need to sleep many more hours after a concussion.
- **Malaise:** You may just "not feel right" but can't point to a specific problem.

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You may not always recognize your symptoms. But your teammates, coaches or family members may see a difference in you that you don't. If someone sees a change in you, take it seriously and report it to your team medical staff.

What should you report to your team medical staff?

Don't try to make a diagnosis yourself. A concussion needs to be diagnosed by your team medical staff. If you have had a hard hit to the head and have symptoms, you should immediately report your symptoms to your team doctors and athletic trainers, who will conduct a thorough evaluation on the sideline.

On occasions, symptoms from concussion will be more obvious or noticeable hours after the impact. Symptoms should be reported to your medical staff regardless of when you become aware of them.

If you see any symptoms in a teammate, tell your team doctors or athletic trainers because your teammate may not always realize he has had a concussion.

When should I return to play following a concussion?

After a concussion, all return-to-play decisions should be made by your team medical staff. These decisions should never be made by players or coaches. You should be free of symptoms before you return to play.

If you have had a concussion and feel you are being pressured to return too quickly, or think that is happening to a teammate, you can call (the hotline number) to make a confidential report.

Am I at risk for further injury if I have had a concussion?

Current research with professional athletes has shown that you should not be at greater risk of further injury once you receive proper medical care for a concussion and are free of symptoms.

If I have had more than one concussion, am I at increased risk for another injury?

Current research with professional athletes has not shown that having more than one or two concussions leads to permanent problems if each injury is managed properly. It is important to understand that there is no magic number for how many concussions is too many.

Research is currently underway to determine if there are any long-term effects of concussion in NFL athletes.

What is the treatment for a concussion?

The treatment for concussion usually consists of rest. Medication may sometimes be prescribed by your team doctors for symptoms such as headaches and dizziness. If your team doctor prescribes medication, be sure to follow his directions and those provided with the prescription.

It is important that you avoid drinking alcohol. Also, if you intend to use over-the-counter medication, vitamins or supplements, tell your team doctors. They may want you to stop taking them.

You should avoid caffeine and make sure that you do not become dehydrated.

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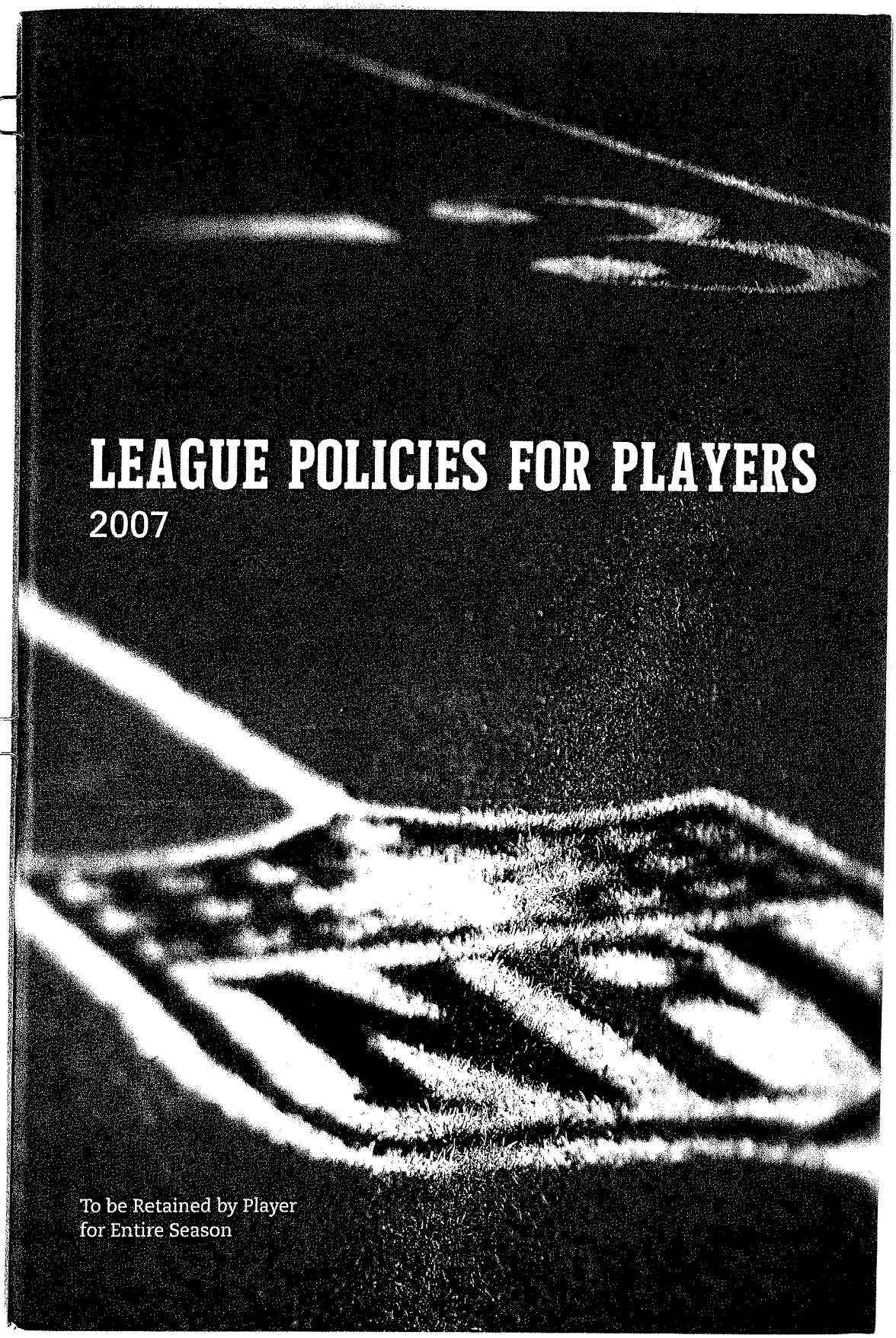


LEAGUE POLICIES FOR PLAYERS

2007

To be Retained by Player
for Entire Season

PLAYER EQUIPMENT SAFETY

Helmet Warning – No Helmet Can Prevent Serious Head Or Neck Injuries A Player Might Receive While Participating In Football.
Do not use the helmet to butt, ram or spear an opposing player. This is in violation of the football rules and such use can result in severe head or neck injuries, paralysis or death to you and possible injury to your opponent.

- Contact in football may result in **Concussion-Brain Injury** which no helmet can prevent. Symptoms include: loss of consciousness or memory, dizziness, headache, nausea or confusion. If you have symptoms, immediately stop playing and report them to your coach, trainer or parents. Do not return to a game or practice until all symptoms are gone and you have received **Medical Clearance.** Ignoring this warning may lead to another and more serious or fatal brain injury.

ProCap Helmet
The NFL Injury and Safety Panel holds the opinion that ProCap helmets have certain characteristics that contribute to catastrophic neck injury which could result in death. In addition, the panel also believes that the ProCap helmet insulates heat and as a result may contribute to heat-related injuries or illness.

- Players are encouraged to consult with their teams' medical staff if they have further questions about the ProCap helmet device.

Guidelines for Players During a Serious On-Field Player Injury
A serious on-field injury is an injury to a player's head, neck or spinal cord rendering him unconscious and/or unable to move all or part of his body.

- Players must go to and remain in the bench area
- Do not roll over an injured player
- Do not assist a teammate who is lying on the field
- Do not pull an injured teammate or opponent from a pile-up

Heads Up:
Preventing Concussion

A concussion is a brain injury. Concussions are caused by a bump, blow, or jolt to the head. They can range from mild to severe and can disrupt the way the brain normally works.

Most people will only experience symptoms from a concussion for a short period of time. But sometimes concussion can lead to long-lasting problems. The best way to protect yourself and your family from concussions is to prevent them from happening.

How to Prevent a Concussion

There are many ways to reduce the chances that you or your family members will have a concussion or more serious brain injury:

- Wear a seat belt every time you drive or ride in a motor vehicle.
- Buckle your child in the car using a child safety seat, booster seat, or seat belt (according to the child's height, weight, and age).
    - Children should start using a booster seat when they outgrow their child safety seats (usually when they weigh about 40 pounds).They should continue to ride in a booster seat until the lap/shoulder belts in the car fit properly, typically when they are approximately 4'9" tall.
- Never drive while under the influence of alcohol or drugs.
- Wear a helmet and make sure your children wear helmets that are fitted and maintained properly when:
    - Riding a bike, motorcycle, snowmobile, scooter, or all-terrain vehicle;
    - Playing a contact sport, such as football, ice hockey, lacrosse, or boxing;
    - Using in-line skates or riding a skateboard;
    - Batting and running bases in baseball or softball;
    - Riding a horse; or
    - Skiing, sledding, or snowboarding.
- Ensure that during athletic games and practices, you and/or your children:
    - Use the right protective equipment (should be fitted and maintained properly in order to provide the expected protection);
    - Follow the safety rules and the rules of the sport;
    - Practice good sportsmanship; and
    - Do not return to play with a known or suspected concussion until you have been evaluated and given permission by an appropriate health care professional.
- Make living areas safer for seniors by:
    - Removing tripping hazards such as throw rugs and clutter in walkways;
    - Using nonslip mats in the bathtub and on shower floors;
    - Installing grab bars next to the toilet and in the tub or shower;
    - Installing handrails on both sides of stairways;
    - Improving lighting throughout the home; and
    - Maintaining a regular exercise program to improve lower body strength and balance, if your health care professional agrees.
- Make living areas safer for children by:
    - Installing window guards to keep young children from falling out of open windows;
    - Using safety gates at the top and bottom of stairs when young children are around;
    - Keeping stairs clear of clutter;
    - Securing rugs and using rubber mats in bathtubs; and
    - Not allowing children to play on fire escapes or on other unsafe platforms.
- Make sure the surface on your child's playground is made of shock-absorbing material, such as hardwood mulch or sand, and is maintained to an appropriate depth.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

Over please >>

Atención:
Prevención de concusiones

Una concusión es una lesión del cerebro que surge a consecuencia de un golpe o una sacudida a la cabeza. Las lesiones cerebrales pueden ser desde ligeras hasta serias y pueden perturbar el funcionamiento normal del cerebro.

Si bien casi todas las concusiones causan síntomas durante un período relativamente corto, algunas veces pueden conducir a problemas duraderos. La mejor manera en que usted y su familia pueden protegerse de las concusiones es evitando que ocurran.

Cómo prevenir una concusión

Existen muchas formas de reducir las probabilidades de que tanto usted como su familia sufran una concusión:

- Utilice el cinturón de seguridad cada vez que conduzca o que viaje en un vehículo automotor.
- En el automóvil, sujete a su niño en una sillita de seguridad, un asiento elevado o con el cinturón de seguridad (de acuerdo a la estatura, el peso y la edad del niño).
    - Los niños deben comenzar a utilizar los asientos elevados en los automóviles cuando ya están muy grandes para las sillitas de seguridad (por lo general cuando pesan 40 libras / 18 kilos). Los niños deben seguir utilizando los asientos elevados hasta que los cinturones de seguridad del vehículo que van a los hombros y la cadera les ajusten bien, por lo general, cuando los niños alcanzan una altura de 4'9"/ 145 cm.
- Jamás conduzca un vehículo si está bajo los efectos del alcohol o las drogas.
- Utilice un casco y asegúrese que sus niños usan uno que les queda a la medida y que está en buen estado para:
    - montar en bicicleta, motocicleta, moto de nieve, monopatines o vehículos todo terreno
    - practicar deportes de contacto como fútbol americano, hockey sobre hielo, lacrosse o boxeo
    - utilizar patines sobre ruedas o patinetas
    - batear y correr a las bases en béisbol o softball
    - montar a caballo
    - esquiar, montar trineo o practicar snowboard.
- Asegúrese que durante los juegos atléticos y las sesiones de prácticas usted y/o sus niños:
    - utilizan el equipo protector adecuado (debe ajustarse a la medida y estar en buen estado para garantizar la protección esperada)
    - siguen las normas de seguridad
    - practican buena camaradería deportiva y
    - no vuelva a jugar si sospecha o sabe que tiene una lesión cerebral traumática o una concusión, hasta que haya sido evaluado por un profesional de la salud adecuado y éste le otorgue permiso para volver a jugar.
- Haga más seguras las áreas donde habitan los ancianos:
    - retire los materiales que puedan causar tropiezos, como alfombras pequeñas y obstáculos en los pasillos
    - use alfombras antirresbalantes en el suelo de la bañera y/o de la ducha
    - haga instalar barras de apoyo junto al inodoro y en la bañera o la ducha
    - instale pasamanos en ambos lados de las escaleras
    - mejore la iluminación en toda la casa y
    - mantenga un programa regular de actividad física, si su médico lo aprueba, para mejorar la fortaleza y el equilibrio de la parte inferior del cuerpo.
- Haga más seguras las áreas donde hay niños:
    - instale rejas en las ventanas para evitar que los niños pequeños salten de las ventanas abiertas
    - utilice rejas de seguridad en la parte superior e inferior de las escaleras cuando haya niños pequeños
    - mantenga las escaleras libres de obstáculos
    - utilice alfombras y tapetes de caucho antirresbalantes en las bañeras y
    - no permita que los niños jueguen en las chimeneas o en otras plataformas poco seguras.
- Asegúrese que las superficies de las áreas de juego infantiles estén recubiertas con materiales para amortiguar impactos, como arena o virutas de madera, y que sean de un espesor adecuado.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

Continua atrás >>

U.S DEPARTMENT OF HEALTH AND HUMAN SERVICES Public Health Service

Centers for Disease Control
and Prevention (CDC)
Atlanta, GA 30341-3724

Dear Colleague:

The Centers for Disease Control and Prevention (CDC) is pleased to provide you with the revised "Heads Up: Brain Injury in Your Practice" tool kit. This tool kit is part of a national initiative to prevent mild traumatic brain injury (MTBI) or concussion and to improve clinical management of patients with MTBI. This initiative is part of CDC's response to the Children's Health Act of 2000, which mandates attention to this public health concern. Physicians can play a key role in helping to prevent MTBI and in appropriately identifying, diagnosing, and managing it when it does occur.

MTBI symptoms may appear mild, but can lead to significant, life-long impairment affecting an individual's ability to function physically, cognitively, and psychologically. Research also indicates, that:

- An estimated 75%-90% of the 1.4 million traumatic brain injury (TBI)-related deaths, hospitalizations, and emergency department visits that occur each year are concussions or other forms of MTBI.
- Blasts are an important cause of MTBI among military personnel in war zones.
- Direct medical costs and indirect costs such as lost productivity from MTBI totaled an estimated $12 billion in the United States in 2000.

This tool kit was developed to provide physicians with a more individualized assessment of MTBI and to help guide the management and recovery of patients with MTBI. The tool kit contains:

- A booklet with information on diagnosis and management of patients with MTBI;
- A patient evaluation tool;
- An information sheet for patients who recently sustained an MTBI;
- A palm card with information about the on-field management of sports-related MTBI;
- Patient education materials in English and Spanish; and
- A CD-ROM with downloadable tool kit materials and additional resources.

We are pleased to be joined in this educational effort by a number of experts, as well as government and professional medical, sports, and voluntary organizations. We hope you will find this tool kit useful in your practice. **This tool kit can be ordered or downloaded free-of-charge at: www.cdc.gov/ncipc/tbi/Physicians_Tool_Kit.**

Your views about the tool kit are invaluable to us and we invite you to share them with us. Please email your comments to NCIPCDIRinfo@cdc.gov with a subject line "Tool Kit for Physicians" or fax comments to 770-488-4338, attention "Tool Kit for Physicians."

Sincerely,

Ileana Arias, PhD
Director
National Center for Injury Prevention and Control
Centers for Disease Control and Prevention

Richard C. Hunt, MD, FACEP
Director
Division of Injury Response
National Center for Injury Prevention and Control
Centers for Disease Control and Prevention

ACUTE CONCUSSION EVALUATION (ACE)

PHYSICIAN/CLINICIAN OFFICE VERSION

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name:_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____

A. Injury Characteristics **Date/Time of Injury**_____ **Reporter: __Patient __Parent __Spouse __Other**_____

1. **Injury Description** _____

1a. Is there evidence of a forcible blow to the head (direct or indirect)? __Yes __No __Unknown
1b. Is there evidence of intracranial injury or skull fracture? __Yes __No __Unknown
1c. Location of Impact: __Frontal __Lft Temporal __Rt Temporal __Lft Parietal __Rt Parietal __Occipital __Neck __Indirect Force
2. **Cause:** __MVC __Pedestrian-MVC __Fall __Assault __Sports (*specify*)_____Other_____
3. **Amnesia Before (Retrograde)** Are there any events just BEFORE the injury that you/ person has no memory of (even brief)? __Yes __No Duration
4. **Amnesia After (Anterograde)** Are there any events just AFTER the injury that you/ person has no memory of (even brief)? __Yes __No Duration
5. **Loss of Consciousness:** Did you/ person lose consciousness? __Yes __No Duration
6. **EARLY SIGNS:** __Appears dazed or stunned __Is confused about events __Answers questions slowly __Repeats Questions __Forgetful (recent info)
7. **Seizures:** Were seizures observed? No__ Yes___ Detail_____

B. Symptom Check List* Since the injury, has the person experienced any of these symptoms any more than usual today or in the past day?
Indicate presence of each symptom (0=No, 1=Yes). ***Lovell & Collins, 1998 JHTR***

PHYSICAL (10)		COGNITIVE (4)		SLEEP (4)	
Headache	0 1	Feeling mentally foggy	0 1	Drowsiness	0 1
Nausea	0 1	Feeling slowed down	0 1	Sleeping less than usual	0 1 N/A
Vomiting	0 1	Difficulty concentrating	0 1	Sleeping more than usual	0 1 N/A
Balance problems	0 1	Difficulty remembering	0 1	Trouble falling asleep	0 1 N/A
Dizziness	0 1	**COGNITIVE Total (0-4)** _____		**SLEEP Total (0-4)** _____	
Visual problems	0 1	**EMOTIONAL (4)**			
Fatigue	0 1	Irritability	0 1		
Sensitivity to light	0 1	Sadness	0 1		
Sensitivity to noise	0 1	More emotional	0 1		
Numbness/Tingling	0 1	Nervousness	0 1		
PHYSICAL Total (0-10) _____		**EMOTIONAL Total (0-4)** _____			
(Add Physical, Cognitive, Emotion, Sleep totals) Total Symptom Score (0-22)		_____			

Exertion: Do these symptoms worsen with:
Physical Activity __Yes __No __N/A
Cognitive Activity __Yes __No __N/A

Overall Rating: How different is the person acting compared to his/her usual self? (circle)
Normal 0 1 2 3 4 5 6 Very Different

C. Risk Factors for Protracted Recovery *(check all that apply)*

Concussion History? Y ___ N___	√	Headache History? Y ___ N___	√	Developmental History	√	Psychiatric History
Previous # 1 2 3 4 5 6+		Prior treatment for headache		Learning disabilities		Anxiety
Longest symptom duration Days__ Weeks__ Months__ Years__		History of migraine headache __ Personal __ Family_____		Attention-Deficit/ Hyperactivity Disorder		Depression
						Sleep disorder
If multiple concussions, less force caused reinjury? Yes__ No__				Other developmental disorder_____		Other psychiatric disorder _____

List other comorbid medical disorders or medication usage (e.g., hypothyroid, seizures)_____

D. RED FLAGS for acute emergency management: Refer to the emergency department with sudden onset of any of the following:

- * Headaches that worsen
- * Seizures
- * Focal neurologic signs
- * Looks very drowsy/ can't be awakened
- * Repeated vomiting
- * Slurred speech
- * Can't recognize people or places
- * Increasing confusion or irritability
- * Weakness or numbness in arms/legs
- * Neck pain
- * Unusual behavioral change
- * Change in state of consciousness

E. Diagnosis (ICD): __Concussion w/o LOC 850.0 __Concussion w/ LOC 850.1 __Concussion (Unspecified) 850.9 __Other (854) _____
__No diagnosis

F. Follow-Up Action Plan **Complete *ACE Care Plan* and provide copy to patient/family.**
___ **No Follow-Up Needed**
__ **Physician/Clinician Office Monitoring**: Date of next follow-up _____
_ **Referral:**
___ Neuropsychological Testing
___ Physician: Neurosurgery____ Neurology____ Sports Medicine____ Physiatrist____ Psychiatrist____ Other_____
___ Emergency Department

ACE Completed by:_____ **MD RN NP PhD ATC**

This form is part of the "Heads Up: Brain Injury in Your Practice" tool kit developed by the Centers for Disease Control and Prevention (CDC).

Acute Concussion Evaluation (ACE)
Care Plan

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name:_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____
Date of Injury:_____

You have been diagnosed with a concussion (also known as a mild traumatic brain injury). This personal plan is based on your symptoms and is designed to help speed your recovery. Your careful attention to it can also prevent further injury.

Rest is the key. You should not participate in any high risk activities (e.g., sports, physical education (PE), riding a bike, etc.) if you still have any of the symptoms below. It is important to limit activities that require a lot of thinking or concentration (homework, job-related activities), as this can also make your symptoms worse. If you no longer have any symptoms and believe that your concentration and thinking are back to normal, you can slowly and carefully return to your daily activities. Children and teenagers will need help from their parents, teachers, coaches, or athletic trainers to help monitor their recovery and return to activities.

Today the following symptoms are present (circle or check). ____**No reported symptoms**

Physical		Thinking	Emotional	Sleep
Headaches	Sensitivity to light	Feeling mentally foggy	Irritability	Drowsiness
Nausea	Sensitivity to noise	Problems concentrating	Sadness	Sleeping more than usual
Fatigue	Numbness/Tingling	Problems remembering	Feeling more emotional	Sleeping less than usual
Visual problems	Vomiting	Feeling more slowed down	Nervousness	Trouble falling asleep
Balance Problems	Dizziness			

RED FLAGS: Call your doctor or go to your emergency department if you suddenly experience any of the following

Headaches that worsen	Look very drowsy, can't be awakened	Can't recognize people or places	Unusual behavior change
Seizures	Repeated vomiting	Increasing confusion	Increasing irritability
Neck pain	Slurred speech	Weakness or numbness in arms or legs	Loss of consciousness

— SCHOOL VERSION —

Returning to Daily Activities

1. Get lots of rest. Be sure to get enough sleep at night- no late nights. Keep the same bedtime weekdays and weekends.
2. Take daytime naps or rest breaks when you feel tired or fatigued.
3. **Limit physical activity as well as activities that require a lot of thinking or concentration. These activities can make symptoms worse.**
   - Physical activity includes PE, sports practices, weight-training, running, exercising, heavy lifting, etc.
   - Thinking and concentration activities (e.g., homework, classwork load, job-related activity).
4. Drink lots of fluids and eat carbohydrates or protein to main appropriate blood sugar levels.
5. **As symptoms decrease, you may begin to gradually return to your daily activities. If symptoms worsen or return, lessen your activities, then try again to increase your activities gradually.**
6. *During recovery, it is normal to feel frustrated and sad when you do not feel right and you can't be as active as usual.*
7. Repeated evaluation of your symptoms is recommended to help guide recovery.

Returning to School

1. If you (or your child) are still having symptoms of concussion you may need extra help to perform school-related activities. As your (or your child's) symptoms decrease during recovery, the extra help or supports can be removed gradually.
2. Inform the teacher(s), school nurse, school psychologist or counselor, and administrator(s) about your (or your child's) injury and symptoms. School personnel should be instructed to watch for:
   - Increased problems paying attention or concentrating
   - Increased problems remembering or learning new information
   - Longer time needed to complete tasks or assignments
   - Greater irritability, less able to cope with stress
   - Symptoms worsen (e.g., headache, tiredness) when doing schoolwork

~Continued on back page~

This form is part of the "Heads Up: Brain Injury in Your Practice" tool kit developed by the Centers for Disease Control and Prevention (CDC).

Acute Concussion Evaluation (ACE)
Care Plan

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name:_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____
Date of Injury:_____

You have been diagnosed with a concussion (also known as a mild traumatic brain injury). This personal plan is based on your symptoms and is designed to help speed your recovery. Your careful attention to it can also prevent further injury.

Rest is the key. You should not participate in any high risk activities (e.g., sports, physical education (PE), riding a bike, etc.) *if you still have any of the symptoms below. It is important to limit activities that require a lot of thinking or concentration* (homework, job-related activities), as this can also make your symptoms worse. If you no longer have any symptoms and believe that your concentration and thinking are back to normal, you can slowly and carefully return to your daily activities. Children and teenagers will need help from their parents, teachers, coaches, or athletic trainers to help monitor their recovery and return to activities.

Today the following symptoms are present (circle or check). ___**No reported symptoms**

Physical		Thinking	Emotional	Sleep
Headaches	Sensitivity to light	Feeling mentally foggy	Irritability	Drowsiness
Nausea	Sensitivity to noise	Problems concentrating	Sadness	Sleeping more than usual
Fatigue	*Numbness/Tingling*	*Problems remembering*	*Feeling more emotional*	Sleeping less than usual
Visual problems	Vomiting	Feeling more slowed down	Nervousness	Trouble falling asleep
Balance Problems	Dizziness			

RED FLAGS: Call your doctor or go to your emergency department if you suddenly experience any of the following

Headaches that worsen	Look very drowsy, can't be awakened	Can't recognize people or places	Unusual behavior change
Seizures	Repeated vomiting	Increasing confusion	Increasing irritability
Neck pain	Slurred speech	Weakness or numbness in arms or legs	Loss of consciousness

— WORKING VERSION —

Returning to Daily Activities

1. Get lots of rest. Be sure to get enough sleep at night- no late nights. Keep the same bedtime weekdays and weekends.
2. Take daytime naps or rest breaks when you feel tired or fatigued.
3. **Limit physical activity as well as activities that require a lot of thinking or concentration. These activities can make symptoms worse.**
   - Physical activity includes PE, sports practices, weight-training, running, exercising, heavy lifting, etc.
   - Thinking and concentration activities (e.g., homework, classwork load, job-related activity).
4. Drink lots of fluids and eat carbohydrates or protein to main appropriate blood sugar levels.
5. **As symptoms decrease, you may begin to gradually return to your daily activities. If symptoms worsen or return, lessen your activities, then try again to increase your activities gradually.**
6. During recovery, it is normal to feel frustrated and sad when you do not feel right and you can't be as active as usual.
7. Repeated evaluation of your symptoms is recommended to help guide recovery.

Returning to Work

1. Planning to return to work should be based upon careful attention to symptoms and under the supervision of an appropriate health care professional.
2. Limiting the amount of work you do soon after your injury, may help speed your recovery. It is very important to get a lot of rest. You should also reduce your physical activity as well as activities that require a lot of thinking or concentration.

___Do not return to work. Return on (date)_____.
___Return to work with the following supports. Review on (date)_____.

Schedule Considerations
___Shortened work day _____ hours
___Allow for breaks when symptoms worsen
___Reduced task assignments and responsibilities

Safety Considerations
___No driving
___No heavy lifting or working with machinery
___No heights due to possible dizziness, balance problems

This form is part of the "Heads Up: Brain Injury in Your Practice" tool kit developed by the Centers for Disease Control and Prevention (CDC).

Concussion Information for NFL Players and Family

What is a concussion?

A concussion is an injury to the brain caused by a blow to the head. This injury causes the brain not to function normally for a period of time. Concussions may be referred to as mild traumatic brain injuries and get better with time. However, occasionally there can be a more significant problem, and it is important that the symptoms from a concussion be monitored. When you suffer this injury, you may have problems with concentration and memory, notice an inability to focus, feel fatigued, have a headache or feel nauseated. Bright lights and loud noises may bother you. You may feel irritable or have other symptoms.

What should I watch for?

After evaluation by your team physician, it may be determined that you are safe to go home. Otherwise, you may be taken to the hospital. **If you are sent home, you should not be left alone. A responsible adult should accompany you.**

Symptoms from your concussion may persist when you are sent home but should not worsen, nor should new symptoms develop. You should watch for such things as:

1. Increasing headache.
2. Increasing nausea or vomiting.
3. Increasing confusion.
4. Unusual sleepiness or difficulty being awakened.
5. Trouble using your arms or legs.
6. Garbled speech.
7. Convulsions or seizure.

If you notice any of these problems or have any other problem that appears worse as compared to how you felt at the time you left the stadium or practice, immediately call the physicians or athletic trainers. In an emergency, have someone transport you to a hospital.

Is it okay to go to sleep?

Concussion many times makes a player feel drowsy or tired. As long as you are not getting worse, it is all right for you to sleep. We do want the responsible adult to be at home with you in case any problems arise.

May I take something for pain?

Do not take any medication unless your team physician has told you to do so. Normally, we do not advise anything stronger than Tylenol. Avoid the use of aspirin, Motrin, Aleve, or any other anti-inflammatory medication that you may have been taking. We also ask that you not consume any alcohol and avoid caffeine and any other stimulants. If you are taking any supplements, we would suggest that you discontinue the use of them as well. The team physician will determine when you can restart medications and supplements.

May I eat after the game?

It is fine for you to eat if you are hungry. Remember, some players do have a sense of nausea and fatigue, and often find that their appetite is depressed immediately after a concussion. Do not force yourself to eat.

Do I need a CT scan or MRI examination?

If the team physicians have determined that you are able to go home after the game, these types of diagnostic tests are not necessary. If you are sent to the hospital with a more serious injury, a CT scan or MRI examination is likely. If your symptoms linger for several days after a concussion, CT scan or MRI examination may be a consideration.

How long will I be observed?

You are to report to the training room the morning after your concussion. You will be assessed by the team physicians and athletic trainers. You will take a neurocognitive test and your symptoms will be monitored. Return to play decisions vary by individual, and will be based on physical exam and a return to baseline on the neurocognitive test.

Telephone Numbers: Athletic Trainers: _____, Team Physicians_____

You are to report to the training room on: Day_____ Time_____

NFL Player Health and Safety Meeting With
Team Physicians and Athletic Trainers

9:15-9:20	Welcome Roger Goodell, Commissioner
9:20-9:35	Keynote Presentation Michael Apuzzo MD, Editor Neurosurgery
9:35-10:10	Summary of NFL Research Findings Ira Casson MD, David Viano Dr. med, PhD
10:10-10:20	What the Helmet Manufacturers and NOCSAE Are Doing? Michael Oliver
10:20-11:00	Assessing MTBI Cases and Return to Play in the Same Game Kevin Guskiewicz PhD, Rick Burkholder ATC, Andy Tucker MD
11:00-11:30	Are Existing Guidelines on Return to Play Applicable to NFL Players? Robert Cantu MD, Ira Casson MD
11:30-12:10	Role of Neuropsychological Testing in Return to Play Decisions William Barr PhD, Mark Lovell PhD, Cynthia Arfken, PhD
12:10-12:20	An Editor's View of the MTBI Research Joe Waeckerle MD
12:20-12:35	What the Player and Family Needs to Know and Can Expect? Stanley Herring MD, John Norwig ATC
12:35-13:15	Lunch
13:15-13:30	NFL Study of Retired Players Ira Casson MD, Thomas Naidich MD
13:30-14:15	Multiple Concussions, When is Enough, Enough? Robert Cantu MD, Henry Feuer MD, Ira Casson MD
14:15-14:45	Other Studies of Retired Players Julian Bailes MD, Mark Lovell PhD
14:45-15:30	Does Concussion Lead to Pugilistic Dementia and Alzheimers? Julian Bailes MD, Joseph Maroon MD, Ira Casson MD
15:30-16:15	Questions and Answers
16:15-16:30	Summary Points, Take-Home Messages and Topics for Study Elliot Pellman MD, Thom Mayer MD

Meeting moderator: David Viano

NATIONAL FOOTBALL LEAGUE

Memorandum

TO:	General Managers, Head Coaches, Trainers and Equipment Managers
FROM:	Ray Anderson, Executive Vice President of Football Operations
CC:	Ron Hill, Tim Davey, Gene Washington, Merton Hanks, Mike Pereira, Thad Ide
DATE:	May 1, 2007
RE:	Buckled Chin Straps

As a follow up to my memorandum dated April 18, 2007, attached for your review is a Technical Service Bulletin from Riddell dated April 30, 2007.

With respect to any modifications of Riddell helmets by equipment personnel, so long as the modifications comply with the instructions contained in the Technical Service Bulletin, they are authorized.

Please feel free to call Tim Davey, Ron Hill or myself if you have any questions.

280 Park Avenue, New York, New York 10017 (212) 450-2000 FAX (212) 681-7577

Riddell®

TECHNICAL SERVICE BULLETIN

01-07

TO: Riddell Helmet Customers

FROM: Thad Ide
VP R&D

DATE: April 30, 2007

SUBJECT: Chin Strap Attachment Modifications

Riddell football helmets are shipped with the following standard chin strap configurations:

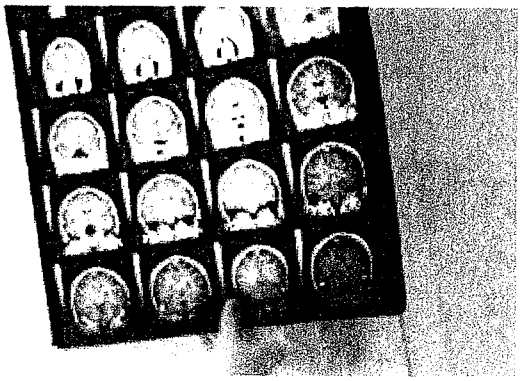
Revolution football helmets – 4 pt. Mid/High Hook-Up
Traditional football helmets incl./VSR-4 – 4 pt. Low Hook-Up

The following modifications are authorized provided that Riddell instructions and templates are used as appropriate:

- VSR-4 helmets maybe modified to accept a "High Hook-Up" chin strap (e.g. Riddell catalog no. 45747, 45708 or 45775) by using the ***Riddell High Hook-Up Drill Template and Instructions*** available through your Riddell representative at *1-800-275-5338.*

- Any Riddell helmet may be modified to accept the **Strap-Loc ™** (Riddell catalog no. 45970) upper chin strap attachment feature by following the instructions included with each **Strap-Loc ™** kit.

- Upper chin strap anchor points for Mid/High Hook-Up chin straps may be affixed using a screw and t-nut rather than the standard snap-buckle if desired.



Heads Up: Brain Injury in Your Practice A Tool Kit for Physicians

An estimated 75%-90% of the 1.4 million traumatic brain injury-related deaths, hospitalizations, and emergency department visits that occur each year are concussions or mild traumatic brain injuries (MTBI).

Many individuals who sustain an MTBI are not hospitalized or receive no medical care at all. An unknown proportion of those who are not hospitalized may experience long-term problems such as persistent headache, pain, fatigue, vision or hearing problems, memory problems, confusion, sleep disturbances, or mood changes. Symptoms of MTBI or concussion may appear mild, but can lead to significant, life-long impairment affecting an individual's ability to function physically, cognitively, and psychologically.

Physicians can play a key role in helping to prevent MTBI or concussion and improve a patient's health outcomes through early diagnosis, management, and appropriate referral.

In response, CDC, in collaboration with an expert work group, has recently updated and revised the "Heads Up: Brain Injury in Your Practice" tool kit. This tool kit is available free-of-charge and contains practical, easy-to-use clinical information and tools, including:

- a booklet with information on diagnosis and management of MTBI;
- a patient assessment tool (Acute Concussion Evaluation or ACE);
- a care plan to help guide a patient's recovery;
- fact sheets in English and Spanish on preventing concussion;
- a palm card for the on-field management of sports-related concussion; and
- a CD-ROM with downloadable kit materials and additional MTBI resources.

To order a free tool kit, click here.

View and download kit materials
(PDFs require Adobe Acrobat Reader)

Information for Physicians:	Information for Patients:

• Introductory Letter PDF
• Facts for Physicians booklet PDF
• Acute Concussion Evaluation (ACE) form PDF
• ACE Care Plan
  ○ Work Version PDF
  ○ School Version PDF
• Concussion in Sports palm card PDF
	• Heads Up: Preventing Concussion fact sheet
	  ○ English PDF
	  ○ Spanish PDF

Heads Up! Concussion Information for Physicians

Download as a podcast

"The Acute Concussion Evaluation or ACE is an innovative assessment tool that can be used for the initial evaluation and diagnosis of patients with known or suspected concussion."

Dr. Jean Langlois, CDC

CDC's National Center for Injury Prevention and Control-the Injury Center-invites your comments about the "Heads Up: Brain Injury in Your Practice" tool kit. We are especially interested in knowing which materials you found most useful and your recommendations for ways to improve the tool kit. To provide comments, please e-mail us at ncipcdirinfo@cdc.gov.

* Links to non-Federal organizations found at this site are provided solely as a service to our users. These links do not constitute an endorsement of these organizations or their programs by CDC or the Federal Government, and none should be inferred. CDC is not responsible for the content of the individual organization Web pages found at these links.

Please note: Some of these publications are available for download only as *.pdf files. These files require Adobe Acrobat Reader in order to be viewed. Please review the information on downloading and using Acrobat Reader software.

Content Source: National Center for Injury Prevention and Control, Division of Injury Response
Page last modified: June 07, 2007

Page Located on the Web at http://www.cdc.gov/ncipc/tbi/physicians_tool_kit.htm

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DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION
SAFER · HEALTHIER · PEOPLE™

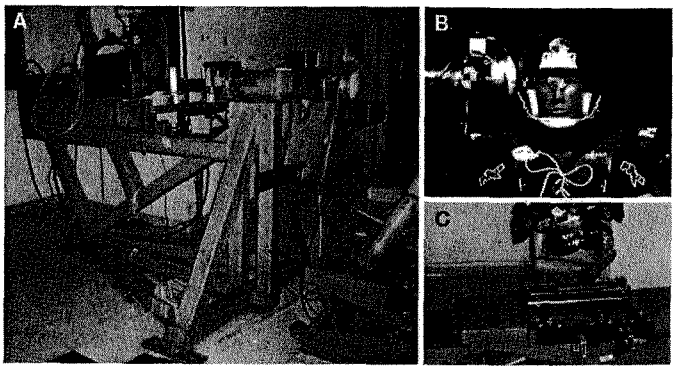

FIGURE 5. *Photographs of the linear impactor at WSU used to simulate the helmet-to-helmet collisions resulting in concussion in the NFL. A, the impactor face and charge cylinder supported on a steel framework next to the dummy. B, the impactor at contact with the helmet. The dummy is wearing shoulder pads and a helmet in the test. C, the Hybrid III dummy fixed to the sliding rails on an adjustable table via the lumbar spine (not visible).*

backpressure on the opposite side of the thrust piston. The trigger activated the electronic solenoid, which released pressure and allowed the higher accumulator pressure to propel the piston towards the helmeted Hybrid III dummy. The backpressure was simultaneously vented.

The Hybrid III dummy was instrumented with accelerometers, load cells, and potentiometers to record biomechanical responses. Head translational and rotational acceleration was measured using the established sign convention (31). The positive x axis is forward and the positive y axis is through the left ear. A "3-2-2-2" accelerometer array was mounted in the head to determine rotational acceleration of the head (25). The upper neck was instrumented with a six-axis load cell to measure moments and shear forces at the occiput. The chest was instrumented with a triaxial accelerometer located in the spine box. The data was sampled using the "tiny data acquisition system" (TDAS) at 20,000 Hz. Video data was gathered at 1000 frames per second. Targets were placed on the dummy head, impactor, and helmet to assess motion relative to ground. The camera was placed perpendicular to the impact axis.

WSU Helmet Testing

The four impact sites (A–D) described by Pellman et al. (28) were used in the evaluation of helmeted head responses at V = 9.5 and 11.2 m/s impact velocities. Two of the impact orientations required the neck to be vertical; the remaining impacts were conducted at a 12° and 24° neck flexion angle. Two different helmets were tested, the Riddell VSR-4 (Helmet V) and one of the new designs, Helmet X. Each helmet was impacted twice at sites A–D. The helmet bladder was pressurized to 3 pounds per second prior to impact. Each helmet was inspected after impact for damage to the faceguard or shell. Any damage was recorded with replacement of faceguard or helmet if required.

Sixteen tests were conducted at V = 9.5 m/s. Comparisons were made between the Hybrid III dummy responses in the integrated torso test (ITT) and the average response for four impact conditions of Pellman et al. (28). For impact condition D, one set of tests duplicated the 6.5 m/s head impact velocity with ground (called D). This led to a 4.1 m/s head ΔV that was not consistent with the rebound characteristics of the helmet in NFL ground impacts. A second set of tests was run at a V = 9.7 m/s impact velocity that matched the head ΔV (called D') of 7.7 m/s seen in the NFL reconstructions of falls to the turf.

Thirty-two additional tests were conducted at V = 11.7 m/s, higher than the elite impact condition to simulate the highest severity exposures for NFL concussions. The elite condition represents a challenge to current football helmets because the foam padding bottoms out causing high head accelerations. Two sets of impacts were run with the dummy torso attached to the sliding fixture by the curved lumbar spine or the pedestrian spine, which allows the dummy to stand more upright. The helmet for these tests was the new design identified as Helmet X.

Statistical Analyses

The average and standard deviation in responses are given for repeated tests. The significance of differences in responses for the various helmet tests was determined using the standard t test, assuming unequal variance and a two-sided distribution. The t test was performed using the standard analysis package in Excel assuming unequal variance and two-tailed probability distribution. The regression analysis was also used from Excel, which determined the average and 95% confidence interval for a linear fit between response data.

Biokinetics Development of a New Linear Impactor, Including Design Specifications

Although changes to the Biokinetics' pendulum were effective in simulating NFL helmet-to-helmet collisions up to 9.5 m/s, the speed of the swinging pendulum was limited by the length of the arm and the available laboratory ceiling height. An effort was undertaken to increase the capacity of the pendulum to reach the elite NFL impacts above 11.2 m/s. Several strategies were pursued to increase pendulum speed by energy assist devices or by increasing the swing radius. However, not all of the pendulum's energy is transferred to the struck Hybrid III head and the pendulum had considerable follow-through energy and higher speeds made it harder to arrest the pendulum posing a safety risk.

The pendulum was discontinued in favor of a WSU-type linear, pneumatic impactor for the evaluation of football helmets. A refinement in the WSU impactor design was undertaken under sponsorship from the NFL at Biokinetics to achieve collision velocities up to 15 m/s. There were several areas that needed refinement for a standardized test. Like the WSU impactor and earlier designs (33), the impactor was accelerated by applying high-pressure air on the thrust piston using a high-rate, high-flow solenoid valve. The linearly

guided impactor had to be moving at a constant speed when it struck the helmeted head. This was achieved by a 51-mm in diameter hollow bearing rod, which was constrained in high-speed linear bearings and propelled by the pneumatic thrust piston. The piston energized the impactor and then disengaged, allowing the rod to continue at constant velocity.

At the end of the impactor, a padded polymer cap was installed to simulate an opposing player's head and helmet. The impacting face was a 127-mm in diameter nylon disk with a curvature radius of 127 mm. A resilient layer of padding was placed between the impactor face and the thrust rod. It was made of DerTex VN600 vinyl nitrile (Detrex Corp., Southfield, MI) with a diameter of 127 mm and a thickness of 35 mm. The pad simulated the helmet padding of the striking player and was selected after evaluating different materials, stiffness, and thicknesses. The final selection balanced peak acceleration and ΔV for five impact sites at 9.5 and 6.7 m/s. The mass of the linear impactor was 14.5 kg, including the rod and impacting face. Finally, a new method of slowing the impact rod was developed by fitting the back end of the rod with a machined disk incorporating an O-ring that enters a tapered cylindrical brake. By venting the trapped air, the rod and impactor decelerate to a stop after impact.

NOCSAE Linear Impactor Helmet Standard

The mission of NOCSAE is to commission research and establish standards for protective athletic equipment. It is a non-profit organization with more than 30 years of experience establishing standards for athletic equipment using the NOCSAE headform developed by Hodgson et al. (13). It fulfills this mission in several ways. One is to prioritize the efforts on catastrophic, serious, and costly injuries. Another is to limit actions to those areas where there is science supporting a technical standard.

The NFL research was transferred to NOCSAE so they could develop a proposal for a new football helmet standard. This was an important milestone because there was no headgear standard establishing performance criteria to reduce the risk of concussion. After review of the NFL work reported here and previously (28, 29), NOCSAE initiated an effort to establish performance criteria for headgear used in football to reduce concussion risks.

NOCSAE has procedures for the drafting, proposing, and implementing of new standards. The key points are the drafting of a standard based on identified needs and available science. The draft must prescribe a method of testing and be exposed to public comment before finalization of the test method and performance requirements. Once drafted and approved, the document passes to proposed status and a mandatory feedback period of typically one year begins. Anyone can submit feedback and critique the proposal.

Typically, a meeting of interested parties is held to discuss the feedback. This input is considered by NOCSAE and modifications and/or revisions to the proposed methods and performance criteria may be made. Additional public comment time may be allowed, but the completed standard is passed to final status and can be mandated by governing bodies. While NOCSAE has no control over governing bodies in sport, it asks that final standards be adopted after 6 months to allow a smooth transition in manufacturing and distribution of helmets to the public.

In the case of concussion in football, NOCSAE has proposed 081-04m04 Standard Linear Impactor Test Method and Equipment Used in Evaluating the Performance Characteristics of Protective Headgear and Face Guards. The equipment is based on the data provided in this paper. While this is a test method containing the information needed to conduct helmet impacts, it does not include performance criteria. The test method describes laboratory equipment and basic requirements pertinent to linear impactor testing of protective headgear with face guards. The linear impactor delivers a controlled impact to a helmeted NOCSAE headform on a Hybrid III neck, which are free to move after impact. The linear impactor delivers an axial impact at 6–12 m/s with a precision ± 3% and emulates on field impacts causing concussion (28, 29).

The NFL sponsored the development of the linear impactor and has transferred its rights to NOCSAE with the agreement

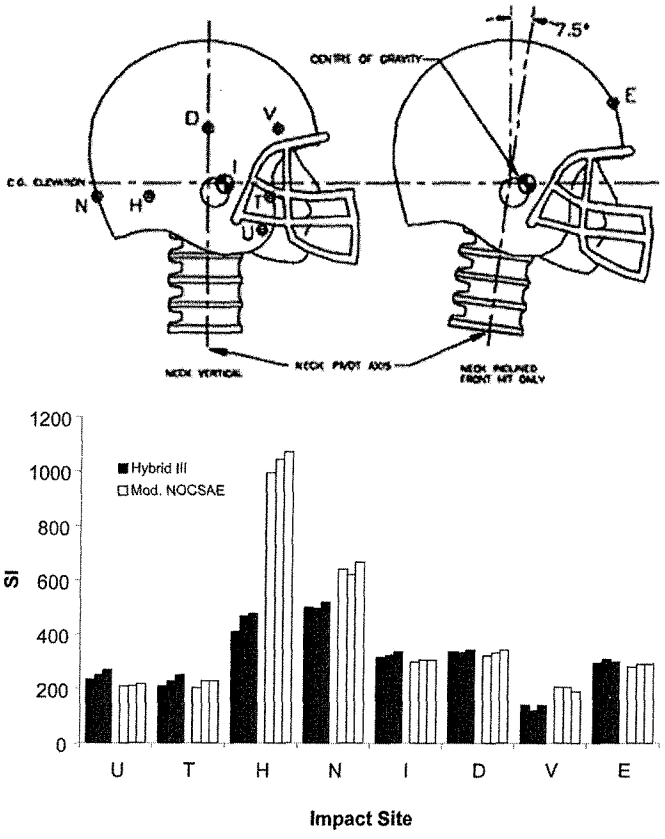

FIGURE 6. *Pendulum impact tests* (below) *of the modified NOCSAE headform and Hybrid III for the original eight helmet impact sites used by Biokinetics* (above). *Each test was repeated three times.*

that public access to the design and protocol is free. NOCSAE has agreed to manage all further development and finalization of specifications after public comment and round-robin testing. Round-robin testing will be conducted to determine if the proposed method complies with the purpose of the standard. The purpose is to provide reliable and repeatable measurements for the evaluation of various protective headgears and to establish a pass/fail criterion based on SI to reduce the risk for concussion. The test method uses the NOCSAE headform, which simulates facial anthropometry with improved harmonics and damping. A special NOCSAE headform was modified to measure rotational acceleration and to accept the Hybrid III neck. Pendulum impacts were conducted at 8 locations on the helmet with the modified NOCSAE headform and Hybrid III head. Testing was done using the original Biokinetics pendulum, and the sites were based on a draft pendulum test method for football helmets prepared for Riddell (35). NOCSAE headform accelerations were not adjusted per the normal test protocol.

The linear impactor delivers an axial impact with a mass of 12.0 ± 0.1 kg using a convex face padded with polyurethane foam of 13 lb/ft^3 density with mechanical properties yet to be specified. The impactor is capable of delivering impacts at 6–12 m/s with a precision ± 3%. Some specifications will be finalized after public comment and round-robin testing. *Figure 6* shows the pendulum impacts at 8 locations on the helmet with the NOCSAE headform and Hybrid III head. SI is significantly higher with the NOCSAE headform for Conditions H ($P < 0.000$), N ($P < 0.003$), and V ($P < 0.002$). This indicates greater sensitivity for those impact locations than the Hybrid III. The proposed test method uses the NOCSAE headform, which has been modified to accept the Hybrid III neck.

RESULTS

Verification Tests with the Revised Pendulum

Table 1 compares the revised pendulum with the NFL reconstructions. The data is organized by impact site, split into high and low speeds, and includes the average response from the NFL reconstructions. The impact speed describes either the collision speed for the NFL reconstruction or the pendulum velocity at impact. Peak accelerations, ΔV, and SI are shown. The high-speed pendulum simulates the 9.5 m/s NFL

TABLE 1. Peak responses from the laboratory reconstruction of NFL collisions with the Revised Pendulum Impactor[a]

Site	Test	9.5 m/s reconstructions				6.7 m/s reconstructions			
		Impact velocity (m/s)	Peak trans acc (g)	ΔV (m/s)	SI	Impact velocity (m/s)	Peak trans acc (g)	ΔV (m/s)	SI
1	NFL reconstruction	9.5	145	7.8	796	6.7	103	5.7	316
	Pendulum test	6.9	143	7.9	793	5.5	99	6.2	359
	Diff		−1.4%	1.3%	−0.4%		−3.9%	8.8%	13.6%
2	NFL reconstruction	9.5	125	6.7	563	6.7	70	5.2	176
	Pendulum test	6.9	126	7.3	633	4.8	75	5.2	204
	Diff		0.8%	9.0%	12.4%		7.1%	0.0%	15.9%
3	NFL reconstruction	9.5	143	7.1	711	6.7	78	5.8	233
	Pendulum test	6.9	146	7.0	800	5.4	76	5.5	231
	Diff		2.1%	−1.4%	12.5%		−2.6%	−5.2%	−0.9%
4	NFL reconstruction	9.5	105	7.6	486	6.7	70	5.9	219
	Pendulum test	7.0	104	7.7	518	5.5	71	5.8	237
	Diff		−1.0%	1.3%	6.6%		1.4%	−1.0%	8.2%
5	NFL reconstruction	9.5	76	6.4	343	6.7	44	4.7	112
	Pendulum test	7.0	74	6.6	300	5.5	39	4.8	99
	Diff		−2.6%	3.1%	−12.5%		−11.4%	2.1%	−11.6%

[a] Trans, translational; acc, acceleration; V, velocity; SI, severity index; NFL, National Football League; diff, difference.

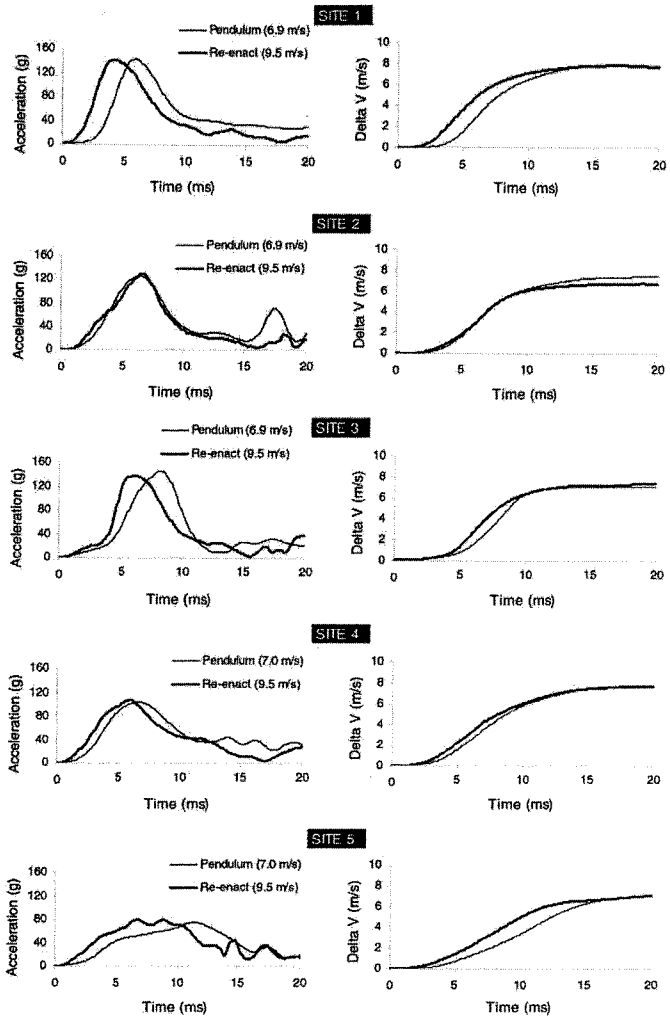

FIGURE 7. *Validation tests of the pendulum impact compared to NFL reconstructions from Pellman et al. (28).*

reconstructions and achieved peak acceleration responses within 5%. The corresponding ΔVs were also within 5%, except for site two, which was off by 9.0%. For the low-speed tests, peak acceleration simulating the 6.7 m/s NFL reconstructions for site two was 7.1% higher than target, and site five was 11.4% higher, but the other sites were below 5%. Corresponding ΔVs ranged from 8.8% above and 5.2% below target. *Figure 2* shows the kinematics of the head and neck of the struck player in the collisions. The last photo in each sequence shows neck bending, which occurs after the primary head acceleration.

Figure 7 compares the time histories for the five impact conditions. The timing of the pendulum versus reconstruction curves is offset because of different triggering and does not reflect a real difference. The shape and peak values are similar, and the primary goal was met that the pendulum system provides similar peak accelerations and ΔVs within ±10% of full-scale helmet-to-helmet from Pellman et al. (28, 29).

Biokinetics Pendulum Impact Tests

Table 2 shows the averages for pendulum impacts on five different helmets tested at 6.9 m/s representing a 9.5 m/s head-to-head game hit and 5.3 m/s representing half the energy. Although there is a trend for lower responses with the newer helmet designs, it was not consistent for all responses and helmets. The average responses of the newer helmets were consistently lower than the baseline VSR-4 helmet.

Figure 8 shows the peak translational acceleration, SI, and peak rotational acceleration with the corresponding 5%, 50%, and 95% risk level for NFL concussions. Risks were determined from the Logist functions derived from only helmet-to-helmet impacts. Head-to-ground cases were left out because the pendulum test was designed to simulate only helmet-to-helmet collisions. The Logist function was used to describe concussion risks. It relates the probability of concussion p(x) to a response parameter x based on a statistical fit to the sigmoidal function $p(x) = [1 + \exp(\alpha - \beta x)]^{-1}$, where α and β are parameters fit to the NFL data (26). The parameters for the Logist function were $\alpha = 2.671$ and $\beta = 0.00891$ for SI, $\alpha = 4.678$ and $\beta = 0.0573$ for translational acceleration and $\alpha = 5.231$ and $\beta = 0.000915$ for rotational acceleration.

These tests illustrate how newer helmets have improved performance in the threshold and average impact condition for NFL concussion. For the 5.3 m/s lower speed test, the concussion risk was between 5%–50% for all sites and models. The 5.3 m/s test represents the threshold severity for concussion 6.7 m/s helmet impacts, which is near the average minus 1 standard deviation in impact speed shown in *Figure 1*. Four of the 22 NFL concussions occurred at speeds of 5.5 to 7.0 m/s in the helmet-to-helmet reconstructions. These results show that even at the low test speed, there is a risk for concussion in professional football.

For the 6.9 m/s pendulum impact speed, the responses for site A are consistently lower for all helmets compared to sites B and C. Some helmets tested better at some sites than others. For site A, the higher-energy hits achieve responses around or below 50% concussion risk. For peak translational acceleration, all helmets tested below 50% risk. For SI, the original VSR-4 and Helmet Z scored above 50% risk; the remainder of the helmets were below 50% risk. This is in contrast to sites B and C where all responses were between 50-95% risk. There was an increased sensitivity of the responses at site A, and this may justify exploring separate injury risk functions for facemask hits to demonstrate equivalent injury risks.

The original VSR-4 helmet shows higher than average responses in all sites and at both speeds. The VSR-4 helmet with revised padding is also included and produced lower responses than the original design suggesting a benefit from improved padding on the side and chin areas. Helmet Z showed higher than average responses for all tests except the low speed site B exposure. Helmet X was poor at site B, but good at site C; Helmet Y was vice versa. Overall, the testing demonstrated that current helmet technology can handle impacts associated with the 6.9 m/s pendulum hit, which relates

TABLE 2. Pendulum test results for five football helmets in the National Football League A, B, and C impact condition[a]

Site	Helmet	6.9 m/s pendulum			5.3 m/s pendulum		
		Peak trans acc (g)	Peak rot acc (r/s^2)	SI	Peak trans acc (g)	Peak rot acc (r/s^2)	SI
A	V	76	5907	330	42	5325	106
	W	53	6689	162	32	3978	66
	X	50	5596	194	32	4307	68
	Y	69	3907	274	44	3054	100
	Z	80	4246	359	51	3010	136
	Average	66	5269	264	40	3935	95
	SD	14	1165	85	8	962	29
B	V	110	6810	598	72	5277	243
	W	77	3953	394	57	3243	167
	X	113	4438	588	62	3852	193
	Y	87	5069	405	65	3938	187
	Z	112	7745	662	60	4798	194
	Average	100	5603	529	63	4222	197
	SD	17	1613	122	6	809	28
C	V	126	10452	626	75	7652	239
	W	92	7114	407	60	5820	181
	X	89	6761	409	58	5403	156
	Y	115	9616	551	58	5080	165
	Z	109	7444	562	64	5407	194
	Average	106	8277	511	63	5872	187
	SD	16	1648	98	7	1029	33

[a] Trans, translational; acc, acceleration; rot, rotational; SI, severity index; SD, standard deviation.

to 9.5 m/s game collisions, and that refinements in helmet padding can reduce concussion risks.

WSU Linear Impact Tests

Figure 9 compares the ITT and NFL concussion responses for impact conditions A–D at 9.5 m/s, the average condition for NFL concussion. Various dummy responses are shown, including impact velocity, resultant head ΔV, and peak resultant translational and rotational acceleration for the average concussion severity.

Table 3 provides the responses. A *t* test determined if the NFL target impact conditions were satisfied by the ITT procedure. In all cases, the comparison showed statistical similarity with only a couple differences reaching $P = 0.01$ significance level for condition B. Conditions A and C showed similar responses. Impact condition D was significantly different from the NFL reconstruction of the ground impacts in terms of impact velocity, resultant head translational, and rotational acceleration and HIC. The additional tests identified as D′ were performed at higher impact velocity to simulate the effects of rebound off the ground. This increased the effective energy of the collision and ΔV of the player's head.

For the D′ impacts, the impact velocity was increased to 9.5 m/s, which is higher than the ground impact velocity in the NFL ground impacts. Although the impact velocity does not match the NFL reconstructions, impacted player's head ΔV, and accelerations more closely match the NFL reconstructions. The resultant translational acceleration of 115.3 G and HIC of 566 are consistent with the NFL reconstructions of 117.1 g and 644, respectively. The rotational acceleration obtained for D′, although closer than D, is still significantly different from the reconstruction data ($P = 0.02$). However, the rotational velocity was 26.4 ± 4.2 r/s and is within the level in the NFL reconstruction (23.2 ± 10.1 r/s). These findings support the increase in impact velocity for condition D′ to obtain a head impact severity seen by players impacting and rebounding off the ground.

Elite Test Condition

Table 4 shows the responses for the impacts above the elite condition with Helmet X, one of the newer designs. At this severity, the helmet padding bottoms out and head accelerations dramatically increase over those at the threshold and average NFL impact velocity. For condition A, the peak translational acceleration averaged 250 ± 56 g and the SI was 1922 ± 669. SIs were lower for impact conditions B–D, although the peak trans-

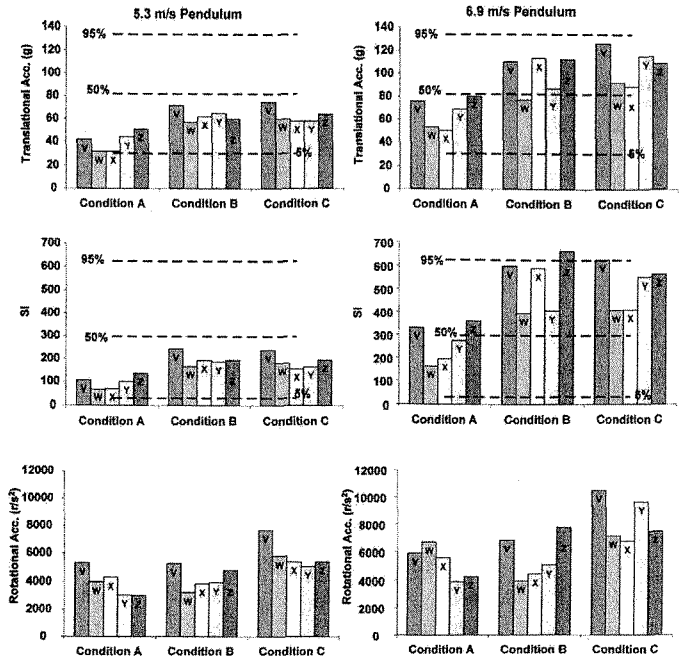

FIGURE 8. *Peak head acceleration, SI, and rotational accelerations for five helmets tested to Conditions A–C at 5.3 and 6.9 m/s, representing the threshold and average conditions for NFL concussion.*

lational accelerations were higher for condition B at 268 ± 36 g. The highest rotational accelerations averaged 15,707 ± 5348 r/s^2 and occurred for impact condition B. All of the biomechanical responses of the head showed the effects of the helmet padding bottoming out causing a substantial increase in head acceleration.

The ITT typically uses the standard Hybrid III pelvis, which has a molded contour representing the seated dummy. The additional tests with the standing pelvis did not show significant differences and so the results were merged. There was a trend for higher head translational acceleration, but overall the responses were similar and reflected the helmet padding bottoming out with a sharp increase in head acceleration.

New Linear Impactor Design Specifications

Table 5 shows tests comparing the response of the new linear impactor with the five staged NFL reconstructions using the same football helmet. Unlike the former pendulum tests, the linear impactor tests were conducted at the same speed as a NFL game collisions causing concussion. *Figure 10* shows a schematic and photo of the new linear impactor. The impactor face and padding material achieved reasonable performance over the impact range. For the 9.5 m/s tests, peak acceleration was 5.6% lower to 16.2% higher than the NFL target and ΔV ranged from 4.7% to 22.4% higher. The average error was 6.7% for acceleration and 10.5% for ΔV. For the 6.7 m/s tests, peak accelerations ranged from 4.3% to 34.6% higher and ΔV ranged from 14.9% lower to 13.5% higher. The average error was 18.6% for acceleration and −2.2% for ΔV.

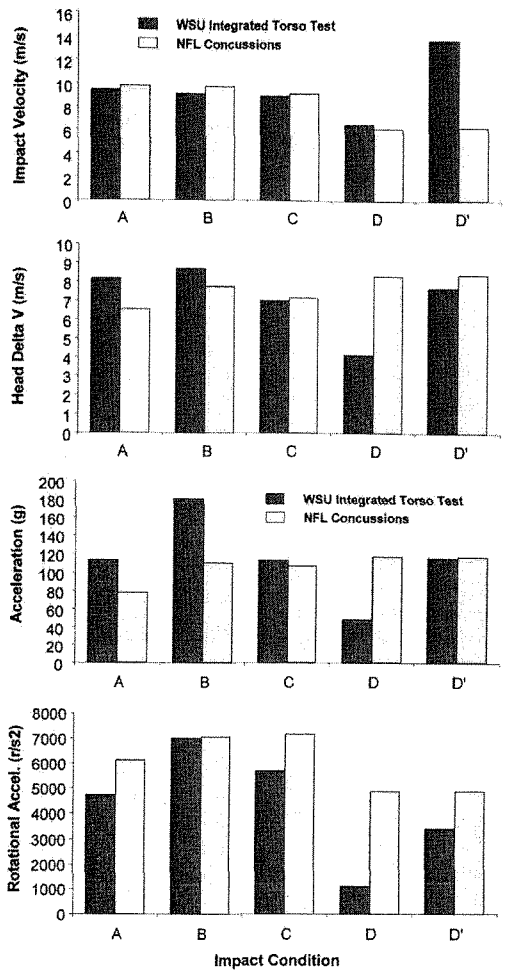

FIGURE 9. *Impact responses for the average NFL concussion impact for conditions A–D at 9.3 m/s using the WSU linear impactor. Two conditions were run for condition D, simulating helmet impacts on the ground. One matched the NFL impact velocity (D) and the other, the head ΔV (D').*

The reliability and repeatability of the NOCSAE prototype impactor has been excellent. At present, the prototype has been tested 700 times, including more than 20 tests at velocities greater than 14 m/s (1400 J). The pneumatic cylinder has been dismantled for inspection and showed no signs of damage or wear. The solenoid release valve, braking chamber, and linear bearings have also remained in good working condition. Minor modifications have been made to reinforce the cylinder connection to the frame reducing vibrations after impact.

NOCSAE Linear Impactor Helmet Standard

NOCSAE has drafted a revision to the current football helmet performance standard NOCSAE document 002. The draft will become proposed after round-robin testing of the equipment described in this paper and specified in NOCASE document 081. This would require all football helmets bearing the NOCSAE logo be subjected to additional linear impactor tests in

TABLE 3. Summary results of integrated torso testing with target data based on National Football League reconstructions[a]

Test no.	Impact		Helmet	Translational				Rotational		
	Condition	Velocity (m/s)		SI	HIC15	Res accel (g)	ΔV (m/s)	Accel (r/s^2)	Velocity (r/s)	Duration (ms)
NFL average	A	9.8		311	257	79	6.6	6120	35.7	—
SD		1.8		154	129	19	1.6	1964	19.4	
10	A	9.0	X	421	362	94	7.3	3049	41.9	18.3
11	A	9.5	X	794	668	140	8.4	6098	28.3	15.3
12	A	9.1	V	479	424	95	7.9	4443	22.6	18.0
13	A	10.0	V	692	553	124	9.1	5368	29.7	16.8
Avg		9.4		597	502	113	8.2	4740	30.6	17.1
SD		0.5		176	136	22	0.8	1315	8.1	1.4
t test		0.61		0.04	0.03	0.04	0.03	0.17	0.52	
NFL average	B	9.6		588	431	108	7.6	6730	38.9	—
SD		0.7		220	149	21	1.7	1390	7.3	
14	B	10.3	V	331	280	98	6.2	6254	34.0	15.8
15	B	9.7	V	995	810	161	8.8	7978	39.7	14.4
16	B	9.1	X	1062	873	175	8.4	6544	44.2	14.2
17	B	8.7	X	1382	1151	205	9.1	6471	48.6	14.5
Avg		9.2		1146	945	181	8.7	6998	44.2	14.4
SD		0.5		207	181	23	0.3	850	4.4	0.2
t test		0.50		0.01	0.02	0.01	0.10	0.74	0.29	
NFL average	C	9.2		489	401	107	7.2	7173	28.2	—
SD		2.1		276	231	33	2.4	1656	12.5	
6	C	9.4	V	579	490	122	7.6	6488	47.3	15.9
7	C	8.8	V	435	367	112	6.8	6227	42.4	15.4
8	C	8.9	X	510	418	117	7.4	4484	40.6	16.2
9	C	8.9	X	347	287	100	6.3	5547	41.9	16.7
Avg		9.0		468	391	113	7.0	5686	43.1	16.1
SD		0.3		100	85	9	0.6	895	2.9	0.5
t test		0.83		0.89	0.94	0.76	0.92	0.18	0.09	
NFL average	D	6.1		757	644	117	8.4	4870	23.2	—
SD		0.2		154	122	6	0.1	202	10.1	
18	D	6.3	V	106	96	49	4.3	1002	8.2	17.7
19	D	6.5	V	111	99	50	4.4	1013	8.2	17.2
20	D	6.8	X	84	73	49	4.1	1096	8.0	19.8
21	D	6.5	X	72	63	44	3.9	1300	10.0	18.2
Avg		6.5		93	83	48	4.1	1103	8.6	18.2
SD		0.2		18	18	3	0.2	138	0.9	1.1
t test		0.18		0.10	0.09	0.02	0.00	0.01	0.29	
26	D′	9.8	X	678	583	130	7.5	4020	32.0	12.8
27	D′	9.7	X	501	455	95	7.6	2452	23.4	15.0
28	D′	9.7	V	680	603	118	8.0	3307	27.2	14.1
29	D′	9.8	V	693	625	118	7.7	3855	23.0	12.8
Avg		9.7		638	566	115	7.7	3409	26.4	13.7
SD		0.1		92	76	15	0.2	707	4.2	1.1
t test		0.02		0.46	0.53	0.84	0.01	0.02	0.73	

[a] SI, severity index; HIC, head impact criterion; Res, resultant; Accel, acceleration; V, velocity; NFL, National Football League; Avg, average; SD, standard deviation.

TABLE 4. Elite impact performance of a newer helmet X[a]

Impact		SI	Translational		ΔV (m/s)	Rotational	
Condition (No. tests)	Velocity (m/s)		HIC15	Res accel (g)		Res accel (r/s^2)	Velocity (r/s)
A	Ave 11.7	2665	2068	316	8.4	8585	42.9
(n = 9)	SD 0.3	1066	846	86	0.7	1744	11.1
B	Ave 11.7	2068	1551	303	8.2	10951	55.9
(n = 8)	SD 0.2	555	480	46	0.4	6207	23.5
C	Ave 11.7	945	678	203	7.2	9105	53.6
(n = 8)	SD 0.2	96	62	31	0.2	549	3.9
D	Ave 11.9	948	751	141	8.2	3298	57.7
(n = 8)	SD 0.4	197	155	19	0.4	693	11.1

[a] SI, severity index; HIC, head injury criterion; Res, resultant; Accel, acceleration; V, velocity; Ave, average; SD, standard deviation.

TABLE 5. Initial testing responses with the new linear impactor and comparison with National Football League concussions[a]

Site	Test	9.5 m/s reconstructions				6.7 m/s reconstructions			
		Input velocity (m/s)	Peak trans acc (g)	ΔV (m/s)	SI	Input velocity (m/s)	Peak trans acc (g)	ΔV (m/s)	SI
1	NFL reconstruction	9.5	145	7.8	796	6.7	103	5.7	316
	Linear impactor	9.6	152	8.7	932	6.6	118	5.6	398
	Diff	0.8%	4.8%	11.5%	17.1%	−1.2%	14.6%	−1.8%	25.9%
2	NFL reconstruction	9.5	125	6.7	563	6.7	70	5.2	176
	Linear impactor	9.5	128	8.2	828	6.7	88	5.9	306
	Diff	−0.4%	2.4%	22.4%	47.1%	−0.7%	25.7%	13.5%	73.9%
3	NFL reconstruction	9.5	143	7.1	711	6.7	78	5.8	233
	Linear impactor	9.5	135	7.7	808	6.7	105	5.4	348
	Diff	0.3%	−5.6%	8.5%	13.6%	0.0%	34.6%	−6.9%	49.4%
4	NFL reconstruction	9.5	105	7.6	486	6.7	70	5.9	219
	Linear impactor	9.4	122	8.0	700	6.6	73	5.8	261
	Diff	−0.6%	16.2%	5.3%	44.0%	−1.3%	4.3%	−1.0%	19.2%
5	NFL reconstruction	9.5	76	6.4	343	6.7	44	4.7	112
	Linear impactor	9.4	88	6.7	370	6.7	50	4.0	87
	Diff	−1.4%	15.8%	4.7%	7.9%	0.1%	13.6%	−14.9%	−22.3%

[a] Trans, translational; acc, acceleration; V, velocity; SI, severity index; NFL, National Football League; diff, difference.

document 081 and demonstrate performance within the limits set in the revised helmet performance standard NOCSAE document 002 (*www.nocsae.org*).

The draft revision calls for linear impacts to be delivered at 325 Joules (J) and 520 J. The peak SI of any impact delivered at 325 J shall not exceed 200 SI. The peak SI of any impact delivered at 520 J shall not exceed 400 SI. This new criteria represents the first standardized effort to measure headgear response to these methods and impact magnitudes. The goal of the revised performance criteria is to reduce concussion risks and provide meaningful targets for helmet design and manufacture. Five specified areas of the helmet and facemask and one random impact area are tested at ambient and elevated temperatures with 325 and 520 J impactor energy. These impacts are in addition to the existing NOCSAE drop test methods.

Although much of the experimental data was developed using a 3-2-2-2 accelerometer set-up, NOCSAE concurred with the findings of Pellman et al. (28, 29) that SI was sufficient, at this time, for the pass/fail criterion. However, there remains a need for improvement in the NOCSAE calibration protocol, which tunes the headform response to target SI levels on a special elastomeric pad. This protocol needs to be refined for the linear impact testing.

DISCUSSION

When postconcussion syndrome forced the retirement of two prominent NFL players, Al Toon in 1994 and Merrill Hoge in 1995, the NFL, through its

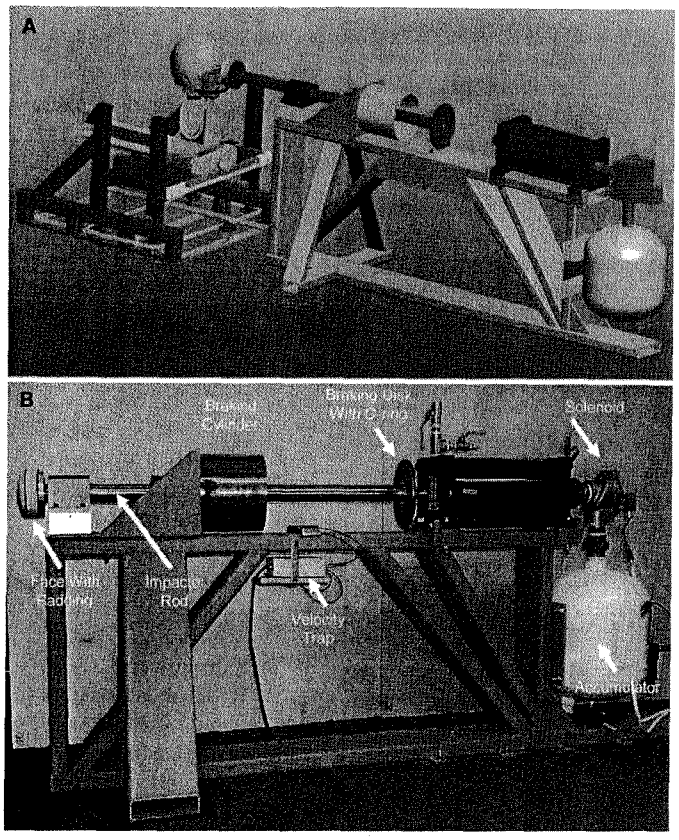

FIGURE 10. A, *schematic of the design of the new linear impactor to be used in the supplemental NOCSAE helmet standard. B, photograph showing the prototype design that was evaluated for use in the NOCSAE standard.*

Committee on MTBI, began to assess helmet performance with concussion (28, 29). Interviews with medical staff and equipment managers revealed the increasing use of air bladder pressure in helmets and their desire to make a change in helmet type to prevent additional injuries. These decisions were often based on anecdotal experience of players and staff, and often over-zealousness of sales personnel from helmet manufacturers.

It became apparent to the NFL MTBI Committee that neither NOCSAE nor the individual helmet manufacturers had the capability to predict the ability of headgear to protect NFL players from concussion. The helmets that were being used had done a superb job in protecting players from severe brain injuries, but there was no data available to assess the helmet's ability to protect from concussion. This lack of information was not only confounding clinicians in their ability to make equipment recommendations, but also was frustrating manufacturers and independent designers who were attempting to make innovative changes in helmets.

When the NFL was presented information on alleged innovative helmet protective changes, the MTBI Committee was frustrated in its inability to objectively grade the validity of the proposed changes. This lack of fundamental scientific data was causing helmet manufacturers to makes changes that seemed intuitive to lower risk; however, sometimes the changes were merely cosmetic. The gap in objective assessment of concussion risk in helmet testing was retarding the helmet industry from moving forward with effective new designs. It was apparent that research was necessary to help gather the data necessary to allow the design of testing modalities that addressed the questions of concussion prevention. The threshold had been reached where there was an obvious need for improved protection in impacts causing concussion in the NFL.

The research presented in this paper provides the foundation for a scientifically based test method and criterion to evaluate helmets for protection against concussion. It is based on field cases of concussion in the NFL and laboratory testing that reconstructed the injurious impacts and player's head responses. There existed a need to move forward with a standardized testing methodology to evaluate sporting equipment for the prevention of concussion. Due to the nature of the protective ability of helmets, these methodologies are based on principles of impact biomechanics and concussion data correlated to "real world" injuries seen in the NFL. The testing matches the impact velocity and head ΔV of concussions. With this correlation, the standardization of methodologies should lead to an effective evaluation of safety equipment.

This article provides the helmet response data quantifying the current state of performance in conditions causing concussion. This data was used to propose a new NOCSAE standard for impacts at the threshold and average severity of concussion in the NFL. The finalization of the standard and adoption will promote the development of new football helmets and refinement in older designs. Obviously, the addition of more energy-absorbing padding for the VSR-4 helmet has shown a reduction in head accelerations over the older design. This is encouraging and supports the effectiveness of the new testing to reduce forces causing injury. Further improvements can be realized, and it is hoped that the proposed helmet test will allow additional innovative changes to safety equipment as well as the critical assessment of the helmet's ability to reduce concussion risks.

Elite Impact Conditions

As the NFL impact data was collected and examined, it became clear that head protection of NFL players often involves very high speed impacts to the front and side of the helmet and that risk factors contributing to the severity of injury could be predicted from player position, type of play, and degree of open-field running before impact. There was also evidence that current helmet designs may be limited in their ability to protect NFL players from concussion in the most significant impacts that have been analyzed.

Figure 11 shows the sensitivity of the head SI to the velocity of impact. At the threshold and average velocity for NFL concussion, one of the newer helmets provides good control of SI. However, SI substantially increased as the padding bot-

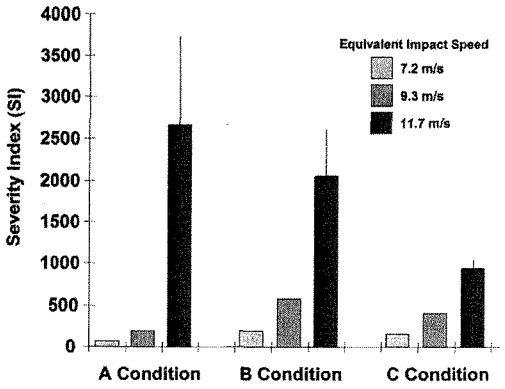

FIGURE 11. *Sensitivity of SI of head acceleration to impact velocity for NFL concussion Conditions A, B, and C with Helmet X, one of the newer designs.*

tomed out in the elite test condition. This indicates that an upper limit exists in the performance of football helmets that overlaps with game conditions of NFL concussion. It seems likely that a new helmet design will be needed to lower SIs in the elite impact condition. Such a helmet may also be particularly useful for the most vulnerable players and for use in plays where the most severe open field tackling and blocking occur.

Some of the highest impact speeds occur during kickoffs and punt returns in which opposing players are running at full speed towards each other. Vulnerable players include those at the highest risk of being concussed with more than 7 days out from play (27). This includes the quarterback, wide receivers, and the secondary. Such a helmet may also add protection for players who have experienced multiple concussions and want to extend their playing career. Although there is a need for this to be proven, there will also be a need for innovation in the future design of helmets that offers performance in elite impacts and for the most vulnerable players.

CONCLUSION

Implicit in this study is the assumption that the risk of cerebral concussion is related to biomechanical inputs to the head, specifically the head SI. In addition, head kinematics has been incorporated in these relationships by referring to previously analyzed NFL videos and reconstruction of concussions in NFL games. Although it is certainly understandable that this assumption is made and we are using what was measured biomechanically in the videos and relating that to what the players experience in terms of mild traumatic brain injury, the two may not be necessarily the same. While concussion is most likely based on SI biomechanical inputs to the head, the neurophysiological consequences may not be linearly related in terms of severity or degree of injury to the energy imposed on the head. While we are confident that our assumption is valid, it has not been proven and only will be proven if the incidence of concussion decreases with the use of modern helmets meeting the new performance standards outlined in this paper.

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COMMENTS

The challenge in obtaining field data that can provide significant insights into the mechanism of injury and subsequently determine better ways of measuring and protecting against those same injuries is daunting. The National Football League (NFL) study undertaken a number of years ago was one of the few projects with this as an objective. This alone characterizes the project and the subsequent studies as worthy scientific undertakings.

However, when one considers the present article in the context of the previous 10 articles from this project, the authors have clearly moved beyond the rather limited data. The authors have reconstructed impact parameters from data presented in a previous paper, creating a higher velocity test protocol. Without the benefit of statistical analysis revealing the reliability and validity of the resulting measures, a proper assessment of the methodology is limited. On a number of occasions the authors suggest that the new test method provides a means of evaluating the ability of a helmet to prevent or decrease the number of concussions. This is clearly not the case. In the broad context of the overall study, there may be some discussion regarding the advantages of using higher velocity impacts to better represent concussive impacts in football. This article is limited to describing a higher velocity impactor with a compliant anvil for the evaluation of football helmets. Further interpretation of the information presented in this article is purely speculative.

Blaine T. Hoshizaki
Biomedical Engineer
Ottawa, Ontario, Canada

Professional American football is characterized by some of the world's strongest, fastest, and most physically impressive athletes engaged in high velocity, aggressive contact. This naturally results in potentially severe injuries, both orthopedic and neurological in nature. Injury to the athletes remains the most undesirable aspect of American football. Over the course of its evolution, significant improvements in protective equipment and an understanding of the nature and etiology of impact injury have led to increased safety of the players. From the perspective of the neurosurgeon, the most important piece of protective equipment is the football helmet. In this article, the authors have provided a detailed description of helmet testing to assess impact performance in protecting against concussion. Pendulum impacts against phantoms were used to simulate impacts to real athletes, and high-speed video was used to analyze the impacts and helmet kinematics. The study showed that, although newer helmet designs reduced the concussion risks in most instances, there are no currently available designs that address the elite impact condition at 11.2 m/s. Systematic studies such as these are extremely useful in identifying shortcomings in current helmets and aiding in the design of more protective helmets in the future.

Charles Y. Liu
Los Angeles, California

Over the past several years, multiple meetings have been held to discuss the incidence rate, severity, and implications of mild traumatic brain injury in sports. In particular, these issues of concussion have been focused on American football, especially the NFL. From these meetings, recommendations and studies have been published regarding detection, when an athlete can return to play, and different types of protective strategies with very little hard, scientific support. Thankfully, this trend has recently changed and is exemplified by the excellent work reported by Pellman et al. This article, however, should be taken in context, as it reflects the continued efforts of the NFL to understand the extent of the problem of mild traumatic brain injury as it relates to its players.

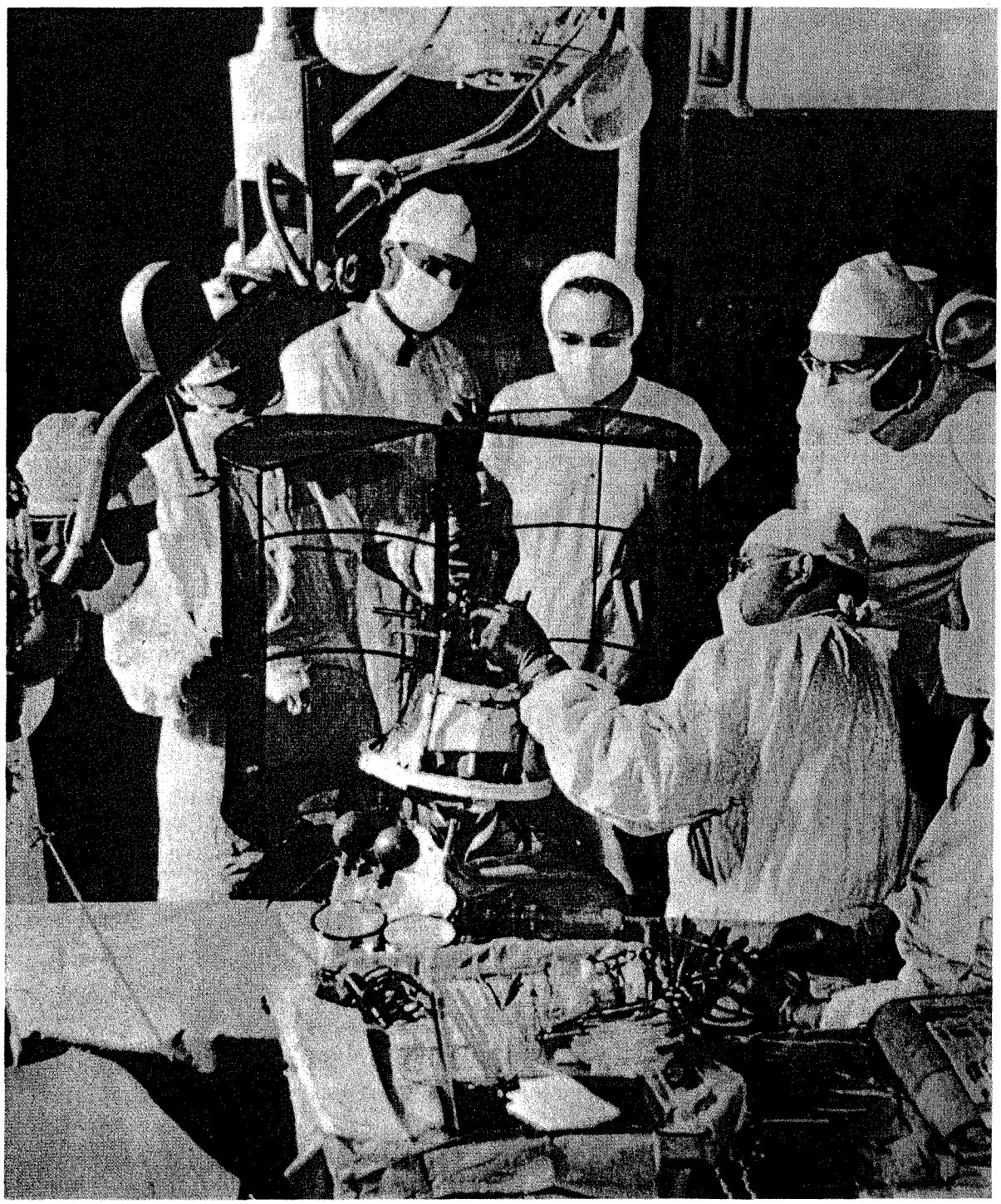
The systematic understanding of human concussion in any sport must first be approached from a biomechanical perspective. Here, Pellman et al. have taken advantage of previous efforts to document these injuries on the playing field, quantifying the extent of loads and forces via high-resolution videos. Through a modeling approach, they have not only developed and/or modified a series of experimental procedures replicating the severity, but, more importantly, the type of concussions sustained by players. Consequently, their work underscores the need to readdress current

accepted dogma regarding issues of severity and to accept that different biomechanical parameters produce different types of concussion, each of which may exhibit different symptoms.

It is no easy accomplishment to take film-documented events of concussion on the football field and translate this data to biomechanical modeling while maintaining such a high standard for validity and reliability. Now, there is ample opportunity to go forward with sound hypotheses for future helmet design as it applies specifically to professional football. However, just as important, today's strategies and/or helmet design, in conjunction with other protective gear, can be evaluated in terms of current prevention from mild traumatic brain injury. The authors and the NFL should be congratulated.

David A. Hovda
Neuroscientist
Los Angeles, California

Intraoperative application of stereoencephalatome by Spiegel and Wycis, circa 1960. (From, *Cooper IS*: Involuntary Movements Disorders. *New York, Harper & Row, 1969.)*



TAB 1L

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CONCUSSION IN PROFESSIONAL FOOTBALL: RECOVERY OF NFL AND HIGH SCHOOL ATHLETES ASSESSED BY COMPUTERIZED NEUROPSYCHOLOGICAL TESTING—PART 12

Elliot J. Pellman, M.D.
ProHEALTH Care Associates, LLP,
Lake Success, New York

Mark R. Lovell, Ph.D.
Department of Orthopaedics
and Sports Medicine,
University of Pittsburgh Medical
Center,
Pittsburgh, Pennsylvania

David C. Viano, Dr. Med., Ph.D.
ProBiomechanics LLC,
Bloomfield Hills, Michigan

Ira R. Casson, M.D.
Department of Neurology,
Long Island Jewish Medical Center,
New Hyde Park, New York
Department of Neurology,
Albert Einstein
College of Medicine,
Bronx, New York

Reprint requests:
David C. Viano, Dr. Med., Ph.D.,
ProBiomechanics LLC,
265 Warrington Road,
Bloomfield Hills, MI 48304-2952.
Email: dviano@comcast.net

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OBJECTIVE: Acute recovery from concussion (mild traumatic brain injury) is assessed in samples of NFL and high school athletes evaluated within days of injury.

METHODS: All athletes were evaluated within days of injury using a computer-based neuropsychological test and symptom inventory protocol. Test performance was compared to preinjury baseline levels of a similar but not identical group of athletes who had undergone preseason testing. Statistical analyses were completed using Multivariate Analysis of Variance (MANOVA).

RESULTS: NFL athletes demonstrated a rapid neuropsychological recovery. As a group, NFL athletes returned to baseline performance in a week with the majority of athletes having normal performance two days after injury. High school athletes demonstrated a slower recovery than NFL athletes.

CONCLUSION: Computer-based neuropsychological testing was used within the overall medical evaluation and care of NFL athletes. As found in a prior study using more traditional neuropsychological testing, NFL players did not demonstrate decrements in neuropsychological performance beyond one week of injury. High school players demonstrated more prolonged neuropsychological effects of concussion.

KEY WORDS: Concussion, Neuropsychological testing, Sport injury, Traumatic brain injury

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The last decade has witnessed the rapid development of new and innovative concussion (mild traumatic brain injury, MTBI) management strategies in the National Football League (NFL). These efforts have been endorsed by the Commissioner's office and have been supported by funding from the NFL charities. As part of this effort, the NFL has supported clinically based research designed to augment the evaluation and management of players with concussion. Neuropsychological testing has increasingly been recognized as a useful diagnostic technology that is used in combination with standard medical care. Numerous research studies have documented the utility of neuropsychological testing in measuring subtle changes in attentional processes, memory and cognitive speed following brain injury (11, 22, 23, 42). Neuropsychological testing provides unique information about changes in cognitive processes that may be missed through sideline or on-field examinations of the athlete and also provides objective information regarding the recovery process during the acute period (13).

There has been a large-scale implementation of neuropsychological testing in colleges (8, 10, 26) and high schools (22, 23, 27); and, numerous studies have examined neuropsychological recovery in high school and collegiate athletes. One published study that examined neuropsychological recovery in NFL athletes (30) suggested that NFL athletes, as a group, made a rapid return to baseline levels of neurocognitive performance following concussion. This finding was in contrast to several studies that found more long-lasting neuropsychological decrements in high school athletes (12, 22, 23, 25). However, none of these studies directly compared professional and younger athletes using the same protocol and an identical neurocognitive test battery. This

is the first study that provides a direct comparison of neurocognitive recovery in professional and younger athletes.

This paper describes the implementation of computer based neuropsychological testing in the National Football League from 2000 to 2004. The study directly compares neuropsychological test results in sport-matched samples of high school and NFL athletes, all tested within days of concussions. The paper specifically addresses the following questions: 1) What is the rate of neurocognitive recovery in a sample of NFL athletes? 2) Do professional and high school athletes display similar or different acute recovery following concussions? 3) Is the pattern of neurocognitive impairment and symptom reporting in these two athlete groups similar or different?

The Evolution of Computer-Based Neurocognitive Testing in Professional Football

The use of standardized neuropsychological/neurocognitive assessment protocols in sport is a recent phenomenon and has been discussed in detail elsewhere (23, 30). From 1993 to 2000, traditional "paper and pencil" neuropsychological testing was introduced in the NFL, and it has now been widely adopted in the league. Testing was introduced to provide additional diagnostic information to assist team physicians in making return to play decisions following concussion. This was one part of NFL MTBI Committee's scientific activities, which have been previously described (29–36, 39–41). The Committee has overseen multiple projects in the NFL designed to better understand and manage concussions.

Although the use of traditional neuropsychological testing in the NFL has been found to represent a useful adjunct to the postconcussion evaluation process (20, 30), limitations in the use of paper and pencil testing with athletes became evident. First, traditional testing proved to be limited with regard to its ability to evaluate neurocognitive difficulties following concussion, such as cognitive speed and reaction time. Additionally, neuropsychological consultants who conducted the assessments were limited to the use of less accurate or sophisticated technology (e.g., a stop watch) to assess the reaction-time related parameters. Furthermore, traditional testing proved to be time consuming and logistically difficult to implement for large groups of athletes. For instance, the implementation of preseason baseline testing proved too difficult because preseason team rosters have approximately eighty athletes. It took a team of neuropsychologists days to complete testing.

Computer-based neurocognitive testing was developed to improve player assessment and more accurately measure subtle neurocognitive deficits following mild concussions. Testing typically yields millisecond precision with regard to measurement of reaction time (5, 24). In addition, computer-based testing can be designed to minimize practice effects (e.g., improvement in test performance due to multiple exposures to the test) by randomizing the presentation of stimuli across administrations (22). Finally, computer-based testing can be administered on a computer network with minimal individual supervision and can be administered to groups of twenty or more athletes at a time, thus improving the ability of teams to complete baseline testing in a matter of hours rather than days.

In 2002, the MTBI Committee undertook an extensive internal review of computerized neuropsychological testing. Published literature was reviewed on the subject. Unpublished data was also reviewed with the consent and cooperation of authors of the four major computerized neuropsychological test batteries used in the evaluation of sports-related concussion: Automated Neuropsychological Assessment Metrics (ANAM), CogSport (www.cogsport.com), Headminders, and ImPact (www.impacttest.com). We determined that there were adequate reliability data on 1113 subjects and validity data on 1293 subjects. At the conclusion of this review, the Committee determined that there was enough evidence to support the use of computerized neuropsychological testing by NFL teams.

MATERIALS AND METHODS

Data Collection and Analyses

All players participating in the study underwent neuropsychological testing during the 2002 to 2004 seasons. As participation in the neuropsychological study was voluntary, not all NFL athletes with concussion completed neuropsychological testing. Furthermore, not all athletes evaluated postconcussion completed baseline neurocognitive testing prior to injury. Therefore, this study relied on a convenience sample of consecutive injured athletes. For professional and high school groups, only athletes who had completed ImPACT 2.0 or 3.0 were included. NFL and high school athletes who had completed earlier versions of the ImPACT test battery (Versions 1.0) from 2000 to 2002 were excluded from the study due to differences in the make-up of the test battery compared to the current versions.

Baseline Samples

Preseason baseline test data were collected on separate groups of professional and high school football players to form the basis for comparison of postinjury test performance to preinjury levels. *Table 1* shows the characteristics of high school and professional athletes who comprised the baseline samples. There were 68 NFL athletes who made up the baseline sample and 125 high school athletes. For the high school group, athletes were selected for inclusion in the baseline sample if they were male high school football players (ninth through twelfth grade) who had completed baseline testing, The baseline samples of high school and professional athletes were very similar to the postconcussion samples with regard to age, level of education and percentage of athletes in each position group. High school and professional players tested after concussion had a slightly higher mean number of past concussions and were similar with regard to the severity of prior concussions: the history of loss of consciousness was 13% for both groups. Forty percent of the high school sample and 44% of the NFL sample had a history of anterograde

TABLE 1. Sample characteristics of concussed high school and professional football players at first postinjury follow-up (n = 85)[a]

	Professional		High school	
	Baseline	**Postinjury 1**	**Baseline**	**Postinjury 1**
No.	68	48	125	37
Age	24.3 (range = 20–34)	26.3 (range = 20–33)	15.6 (range = 13–18)	15.8 (range = 13–18)
Education	15.8 (range = 14–22)	15.7 (range = 14–22)	9.4 (range = 9–11)	9.6 (range = 6–11)
Prior concussions	0.65 (range = 0–4)	1.2 (range = 0–4)	.59 (range = 0–6)	0.89 (range = 0–3)
Linemen	23 (34%)	12 (25%)	46 (37%)	14 (38%)
Offensive backs	9 (13%)	7 (15%)	37 (29%)	9 (24%)
Defensive backs	28 (41%)	18 (37%)	31 (25%)	11 (30%)
Receivers	6 (9%)	9 (19%)	11 (9%)	3 (8)
Kickers	2 (3%)	2 (4%)	0 (0%)	0 (0%)
LOC	N/A	77% none	N/A	65% none
		13% 1–20 s		32% 1–20 s
		2% 21–59 s		0% 21–59 s
		6% 1–2 min		3% 1–2 min
		2% 3–5 min		0% 3–5 min
Retrograde Amnesia	N/A	77% none	N/A	49% none
		2% 1–10 s		35% 1–10 s
		2% 11–59 s		0% 11–59 s
		0% 1–5 min		5% 1–5 min
		2% 6–15 min		3% 6–15 min
		17% > 15 min		8% > 15 min
Anterograde Amnesia	N/A	80% none	N/A	49% none
		8% 1–5 min		27% 1–5 min
		0% 6 15 min		11% 6–15 min
		4% 16–30 min		11% 16–30 min
		4% 31 min to 3 hr		0% 31 min to 3 hr
		4%> 3 hr		2% > 3 hr
Disorientation	N/A	29% none	N/A	30% none
		25% 1–59 s		25% 1–59 s
		19% 1–5 min		5% 1–5 min
		4% 6–15 min		11% 6–15 min
		2% 16–30 min		5% 16–30 min
		21% > 30 min		24% > 30 min

[a] LOC, loss of consciousness; N/A, not available.

amnesia. Nineteen percent of the high school sample and 21% of the NFL sample had a history of an episode of retrograde amnesia. The high school sample did have a higher rate of disorientation than the NFL sample. The average age of the NFL sample was 26.8 years and was similar to the overall league average of 26.5 years (28); and therefore, it is representative of the total population of NFL players with regard to age and experience.

Postconcussion Samples

Table 1 summarizes the sample characteristics. For the purposes of this study, only athletes who completed two postinjury follow-up evaluations were included in the postinjury analyses. Forty eight NFL athletes and 37 high school athletes completed one postinjury evaluation, while 30 NFL and 28 high school athletes completed a second evaluation. Whether an athlete was referred for a second neurocognitive evaluation was determined individually by the team athletic trainer or team physicians. These decisions were clinically based, because athletes who had normal performance following the first evaluation were not evaluated a second time. For the NFL sample, 37% of the athletes returned to play following the initial evaluation and 63% were evaluated a second time within five days of injury. For the high school group, 25%

returned to play prior to the second evaluation and 75% completed the second evaluation. Therefore, NFL and high school athletes who made a rapid recovery did not complete a second evaluation.

Procedures

The NFL neuropsychology program involves the participation of a network of neuropsychological consultants in each NFL city. The network has been described by Pellman et al. (30). For this study, all ImPACT test data was collected by the Director of the NFL neuropsychology program (MRL). It was obtained from team physicians, athletic trainers or team neuropsychologists for entry into a database and subsequent analysis. As the ImPACT battery is computer administered and scored, there were no differences between teams with regard to scoring of the test. Following data collection, the data was stripped of identifying information in compliance with HIPAA standards and stored at the University of Pittsburgh. For the high school sample, data was collected by the University of Pittsburgh Medical Center in cooperation with the high school that the student attended.

Prior to each testing session, the athletes were given a brief explanation of the purpose of neuropsychological testing. Participating players represented a relatively homogeneous group, with the exception of age and level of education. All were males aged 13 to 33 years involved in all offensive and defensive line positions as well as running back, quarterback, and kicking team positions (*Table 1*).

The ImPACT Computer-Based Test Battery

Table 2 shows the individual neuropsychological tests that make up the ImPACT test battery, which evaluates multiple aspects of cognitive functioning and is relatively brief. The battery takes under 30 minutes to administer, is automatically scored and generates a 6 page report. The test battery is heavily oriented towards the evaluation of attentional processes, visual scanning and information processing, although it also evaluates visual memory, verbal memory and visual motor reactions.

Many studies using the ImPACT have indicated that it is reliable and valid. For example, Iverson et al. (17) found no significant practice effects in a sample of non-injured high school athletes tested twice within several days. With regard to validity studies, ImPACT has been found to correlate with the Symbol Digit Modality test, an often used test of cognitive speed in research with athletes (16, 18). ImPACT also has the capability to discriminating mildly concussed high school athletes (22, 23, 37); and, it has been found to correlate with athlete's self-reporting of neurocognitive decline and "fogginess" (16).

Tests That Comprise ImPACT

ImPACT (version 2.0) consists of six neuropsychological tests, which are designed to target different aspects of cognitive functioning, including attention, memory, processing speed, and reaction time. *Table 2* lists the tests and gives a description of neurocognitive abilities assessed. From the six tests, four separate composite scores were created for verbal memory, visual memory, visuomotor speed and reaction time. In addition, an Impulse control composite score is calculated and provides an assessment of test validity. For this study, all athletes obtained valid baseline testing scores for the NFL and high school groups.

TABLE 2. The ImPACT neuropsychological test battery

Test name	Neurocognitive domain measured
Word memory	Verbal recognition memory (learning and retention)
Design memory	Spatial recognition memory (learning and retention)
Xs and Os	Visual working memory and cognitive speed
Symbol match	Memory and visual-motor speed
Color match	Impulse inhibition and visual-motor speed
Three letter memory	Verbal working memory and cognitive speed
Symptom scale	Rating of individual self-reported symptoms
Composite scores	**Contributing scores**
Verbal memory	Word memory (learning and delayed), symbol match, memory score, three letters memory score
Visual memory	Design memory (learning and delayed), Xs and Os (percent correct)
Reaction time	Xs and Os (average counted correct reaction time), symbol match (average weighted reaction time for correct responses), color match (average reaction time for correct response)
Visual motor	Xs and Os (average correct distracters)
Processing speed	Symbol match (average correct responses), three letters (number of correct numbers correctly counted)
Impulse control	Xs and Os (number of incorrect distracters), color match (number of errors)

The Post-Concussion Symptom Scale is also used because it is widely accepted by sports organizations as an accurate identifier of concussion symptoms (2, 20, 21). A 21-symptom checklist asks the injured athlete to rate each possible symptom on a seven-point scale, with zero indicating no experience of a symptom and 6 indicating a severe symptom. This scale is useful because "common" terms are used to describe symptoms and it avoids less common medical terminology (e.g., sensitivity to light is used instead of photophobia; for a review of the Post-Concussion Symptom Scale) (2, 20).

Initial On-Field Diagnosis of Concussion

All NFL teams had a physician and several certified athletic trainers present on the sideline at the time of injury. For high school athletes, concussions were witnessed and diagnosed by physicians and by certified athletic trainers, who were all trained in the diagnosis and management of concussion. In NFL athletes, concussions were diagnosed using standard criteria described previously in articles in this series and used throughout the league (30–34). This involved the identification of a number of neurocognitive (e.g., memory loss) or noncognitive (e.g., headache, nausea, dizziness) criteria identified on the field of play or sideline following a blow to the head. Criteria for high school athletes were highly similar and were based on the presence of mental status changes or player symptoms following a collision or blow to the head. Information regarding the preinjury concussion history was obtained from athletes and their parents. In NFL athletes, concussion history was obtained from the individual teams and medical staff.

Timeline of the Neuropsychological Evaluations

For the high school sample, the initial evaluation was generally conducted two to three days postinjury, as many injuries occurred on Friday nights and the first neurocognitive evaluation was not conducted until the following Monday. For the NFL group, most athletes were evaluated the day after injury, resulting in the completion of the first follow-up evaluation within 24 hours of injury Pellman et al. (30).

With regard to clinical interpretation, NFL players were compared to the normative database for NFL athletes. These data were available to neuropsychologists who served as consultants to the individual teams.

Return to play decisions in the NFL were made by team physicians based on the player's overall clinical evaluations. This included the results of neuropsychological testing and consultations with the team athletic trainers and neuropsychological consultants. Return to play decisions in high school athletes were based on the similar criteria, but decisions were made in consultation with team athletic trainers, the athlete's personal physicians and parents.

Statistics

All statistical analyses were completed using Statistica (38). Descriptive statistics were used to analyze sample characteristics. Z-score transformations were performed to allow comparison across the different composite scores from the ImPACT test battery. Multivariate Analysis of Variance (MANOVA) analyses were conducted to evaluate differences between professional and high school athletes in neuropsychological test results. Because of the different number of subjects evaluated at follow-up 1 and 2, separate MANOVAs were conducted for each of these follow-up periods. Differences between groups on specific indices of the test battery were analyzed with univariate F tests, as part of the MANOVA analyses. Effect sizes (Cohen's d) for differences between NFL and high school groups were calculated by dividing the difference between the postinjury and baseline groups for each sample by the pooled standard deviation (7).

RESULTS

Table 3 shows baseline ImPACT test data, which was gathered to establish average performance or "norms" for NFL (n = 68) and high school football athletes (n = 125). Baseline data came from a sample of athletes from those injured and provides a means of evaluating the postinjury data in the absence of individual baseline values. The preinjury baseline groups provide the basis for comparison if an athlete is injured during the season and also provide the basis for the calcula-

TABLE 3: National football league and high school neurocognitive test performance scores represent raw scores (average and standard deviation in raw score)

	Preseason	Follow-up 1	Follow-up 2
National Football League sample			
Verbal memory	85.7 (9.1)	79.6 (9.6)	84.4 (9.6)
Visual memory	77.3 (13.2)	71.7 (12.7)	77.9 (12.4)
Reaction time	0.58 (0.08)	0.61 (0.09)	0.60 (0.07)
Visual processing	35.5 (5.6)	34.07 (4.4)	35.6 (5.2)
Symptom score	1.34 (2.73)	9.97 (12.2)	6.2 (10.0)
High school sample			
Verbal memory	83.6 (8.2)	76.1 (11.2)	80.5 (12.1)
Visual memory	74.2 (13.2)	63.9 (13.3)	72.1 (11.9)
Reaction time	0.56 (0.07)	0.68 (13.3)	0.61 (0.10)
Visual processing	36.4 (6.3)	31.9 (5.4)	36.3 (6.3)
Symptom score	5.91 (8.65)	25.6 (22)	8.5 (10.1)

tion of Z-scores. Baseline evaluations for NFL and high school athletes were conducted preseason and prior to contact.

Table 3 also shows the mean test scores for postinjury samples for high school and NFL players. All high school athletes included in the postinjury study were initially evaluated within three days of injury (mean = 1.48 d, range = 0-3 d) and all NFL athletes within two days of injury (mean = 1.17 d, range = 1–2 d). For the second follow-up evaluation, the average time postinjury was five days for the high school athletes (range = 2–7 d) and 2.9 days for the NFL group (range = 2-4 d). Therefore, on average, NFL athletes were evaluated sooner postinjury compared to the high school athletes. For our two samples, this difference reflects the fact that NFL athletes routinely undergo neuropsychological testing on Monday or Tuesday following games on Sunday, while high school athletes in our program are most often evaluated on Monday or Tuesday following a game that took place on the previous Friday. As can be seen in *Table 3*, the high school and professional groups demonstrated a decline in raw test scores from baseline.

Table 1 provides demographic information regarding the high school and professional samples. It also presents information regarding on field markers of injury as collected by team medical and athletic training staff. The NFL and high school samples did not differ significantly with regard to loss of consciousness ($\chi^2 = 1.54$, $P = 0.15$) or disorientation ($\chi^2 = 0.00$, $P = 0.955$) following injury. With regard to the presence of retrograde amnesia, the high school sample displayed more amnesia ($\chi^2 = 7.38$, $P = 0.005$). Similarly, the high school group demonstrated greater retrograde amnesia than the professional group ($\chi^2 = 7.40$, $P = 0.007$).

Is There a Difference in Acute Neurocognitive Functioning between Professional and High School Athletes?

Figure 1 presents the results of MANOVA analyses that compare NFL and high school athletes on the four ImPACT composite measures at the first and second follow-up examinations. All scores were converted to standard (Z) scores to facilitate direct comparison using the same metric. Compared to preseason normative performance levels, NFL athletes displayed significantly better ImPACT performance compared to the group of high school football athletes (Wilke's Lambda, F = 4.00, $P < 0.005$). The univariate F tests reveal significant differences between NFL and high schools athletes on the Reaction time (F = 15.8, $P < 0.0001$, d = 0.91) and Processing speed indices (F = 5.29, $P < 0.02$, d = 0.51). NFL athletes performed better on Verbal Memory (F = 3.00. $P = 0.09$, d = 0.39) and the Visual Memory (F = 3.50, $P < 0.06$, d = 0.41) indices but these differences were not statistically significant. Declines in NFL players at follow-up 1 (relative to preseason levels) were not dramatic but did suggest an initial decline in performance on all four composite measures. Performance deficits were mild and generally on the order of 0.25 standard deviation units. In contrast, the high school group displayed significantly larger declines with a decline of almost 0.75 standard deviation on the Verbal Memory composite and a decline (increase in response latency) of 1.53 standard deviation units in reaction time. Less dramatic declines were demonstrated on the Visual Memory and Processing Speed composite scores for the high school sample.

Figure 2 shows the results of a second MANOVA analysis to compare professional and high school athletes at the time of the second follow-up. NFL athletes were evaluated several days sooner than high school players. With regard to an overall comparison of neuropsychological performance in the

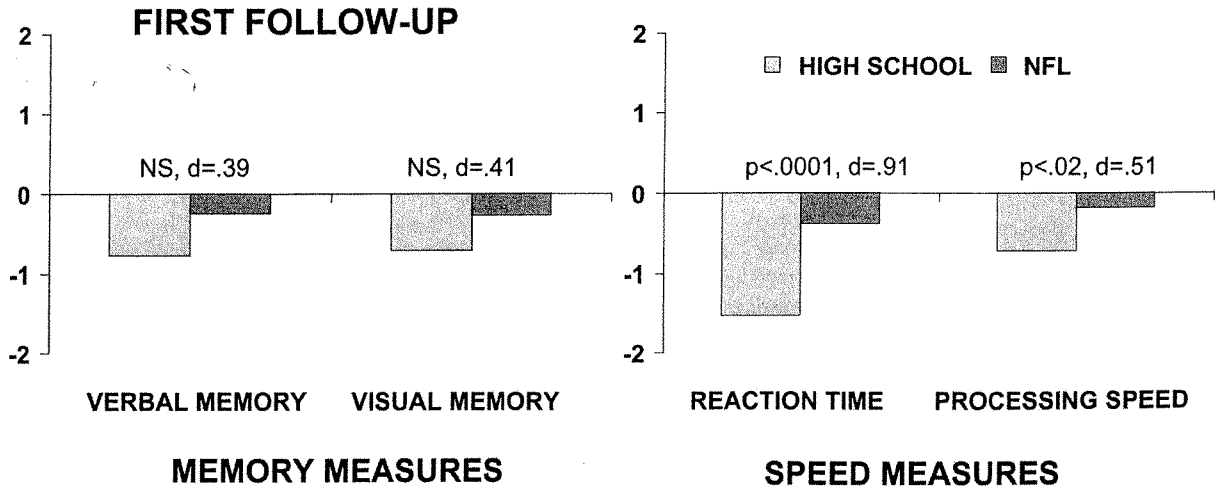

FIGURE 1. *Standard (Z) scores for 37 high school and 48 NFL players tested within five days of injury using ImPACT memory and speed scores at the time of first follow-up. High school athletes were tested within an average of 1.48 days of injury (range = 0–3 d) and NFL athletes within 1.1 day of injury (range = 1–2 d). MANOVA shows differences in performance between NFL and high school athletes on ImPACT composite scores (Wilke's Lambda, F = 4.00, P < 0.005). Univariate differences are represented on graphs.*

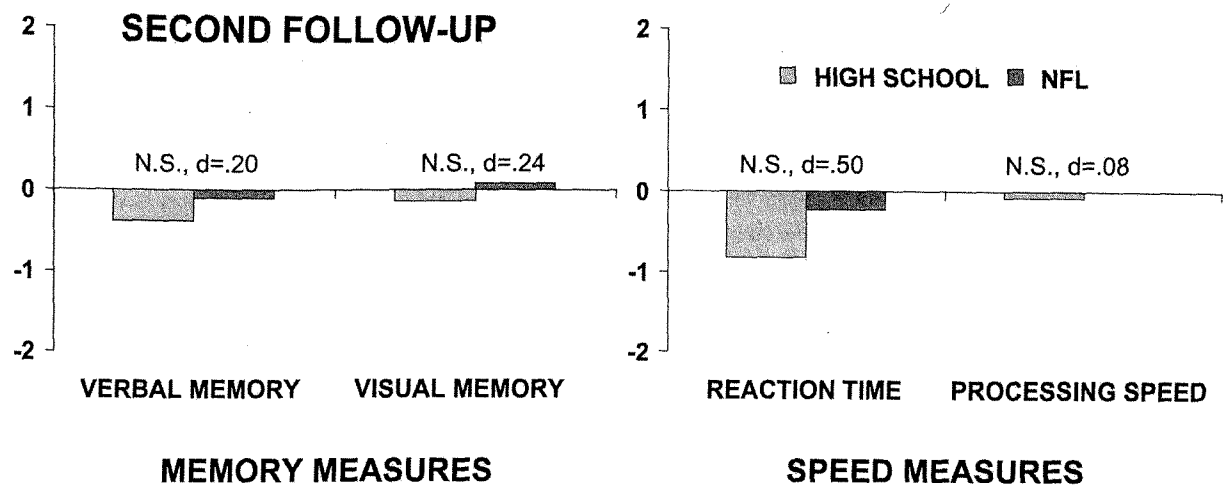

FIGURE 2. *Standard (Z) scores for 28 high school and 30 NFL players, tested within seven days of injury. High school athletes were tested within 5 days of injury (range = 2–7 d). NFL players completed their second evaluation within 2.93 days of injury (range = 2–4 d). MANOVA shows nonsignificant differences in performance between NFL and high school athletes on ImPACT composite scores (Wilke's Lambda, F = 1.09, P < 0.37). No univariate differences were significant, although there was a trend towards poorer performance in high school athletes.*

NFL and high school groups, the MANOVA was nonsignificant (Wilke's Lambda, F = 1.09, $P < 0.370$). At the second follow-up, approximately three days post injury, the NFL group demonstrated at or above the baseline sample levels on two of the composite indices (Visual Memory and Processing Speed) and very subtle decline on the Verbal Memory and Reaction time composite scores, relative to preseason levels. These standard scores were on the order of one tenth of a standard deviation for Verbal Memory and one fifth of a standard deviation for Reaction Time and are not of any clinical significance. In contrast, the high school group tested within seven days of injury demonstrated poorer performance relative to baseline sample levels. They had a drop of approximately 0.4 standard deviation unit is in Verbal Memory and a 0.83 standard deviation change in Reaction Time relative to preseason performance. There was a smaller decline on the Visual Memory composite and a slight (and not statistically significant) improvement in performance on the Processing Speed index.

The Role of Concussion History

An analysis was undertaken to investigate the potential role of concussion history with regard to neurocognitive recovery in the NFL and high school samples (*Table 1*). Athletes from both samples were classified based on the presence or absence of a concussion history prior to their current injury. Separate MANOVAs were then completed for both samples and yielded nonsignificant results for the NFL (Wilkes Lamda, F = 2.02, $P = 0.109$) and high school (Wilkes Lamda, F = 2.01, $P = 0.115$). Therefore, the presence or absence of a history of concussion was not related to neurocognitive test performance during the first week of recovery. The NFL and high school groups were then assigned to categories based on the absence of concussion history (Group 1), 1 prior concussion (Group 2) or 2 or more concussions (Group 3) to further investigate this issue. A MANOVA was completed with concussion history groups as the independent variable and the four ImPACT composite measures as dependent measures. This analysis did not yield significant results for either the NFL (Wilkes Lamda, F = 1.29, $P = 0.24$) or high school (Wilkes Lamda, F = 1.34, $P = 0.24$) groups.

DISCUSSION

Neuropsychological testing assists team physicians by providing quantitative information on recovery from concussion; and, it has been generally accepted as a component of the medical evaluation following sports-related concussion. More specifically, neuropsychological testing may provide diagnostic information regarding subtle disruptions of cognitive processes such as attention, memory and speed that may not be detected by a cursory sideline evaluation and may not be recognized by the injured athlete. Numerous prior studies have described the neurocognitive recovery of high school (4, 22, 23, 27) and collegiate (8, 9, 21, 26) athletes. These studies have shown neurocognitive recovery within weeks of injury. More recently, a study of NFL athletes found that athletes with MTBI did not perform significantly more poorly on a battery of traditional neuropsychological tests within the first week after injury (30). This study used non-computer based paper and pencil neuropsychological testing and there remains a question that it may have missed subtle cognitive changes in reaction time and cognitive efficiency.

This study represents the first of its kind to use computer-based neuropsychological testing in a group of professional athletes. It was designed to evaluate postinjury neurocognitive functioning in a consecutive sample of NFL and high school athletes who had all completed the ImPACT test battery twice

within the first week following their injury. It is the first study to directly compare professional and high school football players using a standardized on-field and postinjury computerized assessment protocol.

Recovery Rates in NFL and High School Athletes

The finding of no significant neurocognitive deficits in the NFL sample within the first week postinjury suggests that NFL athletes with MTBIs recover relatively quickly following injury. This finding supports the only other published study of neuropsychological performance in NFL athletes that did not find significant neuropsychological deficits within one week of injury (30). In contrast, this study found residual difficulties in reaction time and memory in the high school sample and raises the question of differential response to injury in professional and high school football players. There are a number of potential reasons for the apparent difference between professional and high school athletes (30).

First, several researchers have suggested that due to neurodevelopmental factors children are at greater risk of sustaining concussion after head impact. For instance, children are known to exhibit more diffuse and prolonged cerebral swelling after injury than adults (1, 19) and the majority of fatal brain injuries in contact sports have occurred in children younger than 18 (6). There is also evidence from animal research suggesting that the immature brain may be more sensitive to the negative effects of glutamate-mediated N-methyl D-aspartate (NDMA) than in older animal brains. This difference has been hypothesized to play a role in the detrimental effects of excitatory amino acids following brain injury. Finally, a recent clinical study has shown neuropsychological deficits lasting days after injury and a differing rate of recovery depending on the athlete's age (12).

Another hypothesis that may help to explain the differences between NFL and high school athletes is that there may be a different tolerance for concussion in professional and high school athletes. This hypothesis is based on the assumption that NFL athletes represent a highly select group with regard to skill level, size and injury tolerance. The level of conditioning and skill necessary for success in the NFL may result in athletes that are less prone to injury than younger and less talented or well-conditioned individuals. There may be a natural selection process as previously suggested by Pellman et al. (30, 32, 33). Although speculative, this hypothesis is supported indirectly, by current NFL data suggesting that a relative minority of athletes develop postconcussion syndrome (30, 32, 33). In addition, it may be the case that athletes, who have difficulties with concussion during their high school or college years, may choose noncontact sports that have a lower risk of concussion early in their sporting careers. Although this hypothesis is also speculative, it is in line with the observation that most athletes who enter the NFL do not have a significant history of concussion during their high school and college years. For this study, the mean concussion rate was only .65 for the NFL baseline sample (range of 0–4) and 1.2 for the injured NFL group. We also note that in the clinical concussion management program at the University of Pittsburgh Medical Center it is recommended that concussion-prone high school athlete's transition to noncontact sports. In our opinion, it is unlikely that athletes who rise to the level of the NFL are concussion prone.

The NFL and high school samples were not perfectly matched with regard to time of their first and second neuropsychological evaluations, postinjury. Based on when games are played in the NFL (usually on Sunday), most players were evaluated the day after injury. In the high school sample, athletes were more likely to be evaluated several days postinjury based on the fact that most games are played on Friday or Saturday and players do not routinely undergo neuropsychological testing until the following Monday. Since most individuals display neurocognitive deficit immediately following injury, it would be logically expected that the NFL group would demonstrate more dramatic neurocognitive decline since they were being evaluated more quickly after injury. However, this was not the case. In our opinion, this provides additional support for the hypothesis of a quicker recovery in NFL compared to high school athletes.

Relevance to Prior NFL Concussion Studies

In prior papers, the authors have presented the results of clinical evaluations in professional football players after concussion (30–34). However, one paper reviewed neuropsychological test performance using traditional measures (30). The earlier study demonstrated that NFL players, as a group, did not demonstrate neuropsychological deficits on traditional measures and players who sustained multiple concussions did not perform more poorly than those with a single injury. One possible limitation of the earlier study was a reliance on neuropsychological measures that were not specifically developed for sports and did not assess the importance of reaction time and neurocognitive speed. This present paper presents the data using a test battery that has proven to be reliable (17) and valid in detecting subtle changes in reaction time following mild brain injury (22, 23, 37). Therefore, we feel that we have addressed one of the limitations of our prior research.

Regarding the potential cumulative effects of concussion in NFL and high school athletes, this study was designed to evaluate the acute neurocognitive functioning of athletes following concussion. Therefore, we do not purport to present definitive data regarding more long-term outcome in either high school of professional athletes. Hopefully, additional studies will address this issue. However, we did assess the relationship of neuropsychological test results to test performance in our sample. The potential cumulative effect of sports-related concussion remains a controversial issue, and past published studies have differed in their findings (15). Most recently, in a sample of injured NFL athletes who completed traditional neuropsychological testing, Pellman et al. (30) did not find poorer neuropsychological test performance in athletes with a history of three or more concussions. Simi-

larly, the present study did not find a pattern of poorer neuropsychological test scores in a group of NFL athletes who underwent computer-based neuropsychological testing within days of injury. However, it is important to emphasize that the relatively limited sample size in the current study is by no means conclusive. As noted in previous studies, this is a complicated issue and may relate to threshold or age issues (30). This issue will require multiple studies designed to evaluate more long-term markers of outcome in both high school and professional groups. In addition to neuropsychological test results, these studies should involve the evaluation of school performance, activities of daily living and other age-appropriate and relevant measures of real life functioning.

Computer-Based Neuropsychological Testing

The development of the neuropsychological testing program in the NFL has been an incremental process that has involved the ongoing support and coordination of the NFL MTBI Committee. As noted by Pellman et al. (30), the program has evolved to the point where every NFL team is using some form of neurocognitive assessment. Over the past two years, the majority of the league has adopted computer-based neuropsychological testing. This study represents our evaluation of the first two years of data and involves the participation of a subset of eight teams who shared their test data.

Computer based neuropsychological tests have a number of advantages over more traditional testing protocols. One particular advantage is the ease with which athletes can complete baseline testing. This will lead to the completion of baseline testing in the next several seasons. Another advantage is the capability to evaluate subtle changes in reaction time and neurocognitive speed. This capability is highlighted in this present study. The most striking changes in postinjury test performance were evident on the Reaction Time Composite Index. Finally, previous research with computer-based neuropsychological tests has suggested minimal practice effects or improvements in test performance due to prior test exposure (17). This represents a significant improvement compared to more traditional approaches to testing.

The Role of Neuropsychological Testing in Sports

This study highlights the role of neuropsychological testing in professional and high school athletics. Neuropsychological testing represents one of the tools available to assist treating physicians when diagnosing and managing sports-related concussion. Its use is evolving as part of international standards on managing concussion in sports (2). Although a valuable tool, neuropsychological testing should only be used within the context of the clinical medical evaluation and should not be used in and of itself to determine diagnosis or make management decisions such as return to play (30). The overall treatment of the athlete should involve an assessment of the athlete's on-field signs and symptoms, subjective report of symptoms, the observations of the medical staff present at the time of injury, as well as neuropsychological test results. Furthermore, neuropsychological testing can assist in obtaining quantitative information regarding the cognitive status of an athlete that may help to confirm a diagnosis by the team medical staff. Involvement in the testing process also provides reassuring information to injured players and their treating physicians.

Limitations of the Study

As with most research with professional athletics, this research is of an observational nature. As noted in a prior study of NFL athletes, the neuropsychology program was designed to operate with minimum disruption to the NFL teams and athletes, while yielding useful clinical information (30). The study relied on a convenience sample of injured athletes without assurance that the sample is representative of the overall population of injured NFL athletes from 2002 to 2004. However, the sample did match the overall NFL sample in player age and NFL experience.

Currently, all NFL teams are using neuropsychological testing in some form. However, not all teams use the same test battery, conduct preseason baseline testing or use the results in the same manner. We anticipate that future studies of neurocognitive performance in NFL players will allow for the evaluation of larger injury samples and will allow the direct comparison of post injury functioning to individual performance on baseline testing.

Another potential limitation of this study is the lack of established standards for determining abnormal test performance. If preseason baseline testing has been completed on a particular player, abnormal performance was determined by comparing the athletes' postinjury score to their baseline. If the score was poorer than baseline levels, the score was determined to be "abnormal." However, since not all teams who supplied data for this study completed baseline tests, standard scores were calculated as the basis for analysis of deviations from preseason levels of performance. This allowed us to directly compare the athlete to his reference group.

Recently, Reliable Change Index (RCI) scores have evolved to provide an additional determination of abnormal performance (3, 5, 17). RCIs are based on the test-retest reliability of the particular test and they adjust for practice effects. RCIs provide confidence intervals for determining whether or not a test score is reliably different from baseline. However, RCIs have not yet been derived for professional football athletes, although they do exist for high school (3, 17) and Australian rules football athletes (14). As the NFL neuropsychology program develops, the implementation of RCIs should aid in clinical interpretation of neurocognitive recovery following concussion.

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Acknowledgments

This project was undertaken under the auspices of the National Football Leagues (NFL) Committee on Mild Traumatic Brain Injury (MTBI). The Committee is chaired by Dr. Elliot Pellman and includes representatives from the NFL Team Physicians Society, NFL Athletic Trainers Society, NFL equipment managers and scientific experts in the area of traumatic brain injury, biomechanics, basic science research and epidemiology. Authors of this paper are Committee members. With the exception of Dr. Lovell, none of the Committee members has a financial or business relationship posing a conflict of interest to the research conducted on MTBI in professional football. Dr. Lovell has a financial interest in the ImPACT computer based neuropsychological test battery used by many NFL teams. The Committee appreciates the contributions of all of the NFL team neuropsychologists, including Michael Collins, Gary Solomon, Thomas Sullivan, Steven Morgan, Thomas Hardey, Kathy Knee and Don Gerber. The Committee also commends the NFL players, who consented to participate in the study through a blinded identification. The Committee also appreciates the assistance of Cynthia Arfken, Ph.D., from Wayne State Universities, Department of Psychiatry and Behavioral Neurosciences, Center for Healthcare Effectiveness Research, in the statistical analysis of the neuropsychological data. In addition, the efforts of Michael McClincy of Dartmouth College are recognized. Funding for this research was provided by the National Football League and NFL Charities. The Charities are funded by the NFL Player's Association and League. Their support and encouragement to conduct research on MTBI is greatly appreciated.

COMMENTS

The role of neuropsychological testing to evaluate athletes with cerebral concussion has evolved over the past 12 to 15 years. Initially, "paper and pencil" testing was performed. But, it immediately became obvious that this was cumbersome, susceptible to practice learning, and time consuming. Approximately 5 years ago computer-based neuropsychological testing was introduced and was subsequently shown to be accurate, reliable, valid, quick to administer, and highly accepted by high school, college, and professional athletes. It has become a tool that is invaluable in assisting the physician in evaluating and managing an athlete with concussion.

To this end the authors compared neuropsychological test results in high school football players and National Football League (NFL) athletes all tested within days of cerebral concussion. They attempted to determine the rate of neurocognitive recovery, differences in the recovery process particularly as related to a previous history of concussions, and the pattern of impairment and symptoms reported in the two groups.

Their major finding was that NFL athletes had a more rapid recovery than high school athletes and had a rapid return to baseline usually within a few days after injury while high school athletes demonstrated a slower recovery pattern. The reasons for the residual difficulties in reaction time and memory in the high school athlete are speculative. Is the teenager brain really that much more susceptible than the athlete who is in the mid 20s or early 30s? Or, is it really a "natural selection process," as suggested by Pellman et al., with those athletes making it to the professional level being less susceptible to concussive injuries? These questions remain unanswered, but there does seem to be a differential risk of injury in the professional and younger football player.

The author's observation on the relationship of on field markers of injury reassert the now well recognized fact that loss of consciousness is not only not the sine que none of a concussion, but that most concussive injuries now occur without loss of consciousness. Retrograde amnesia at the time of injury seems to be the best on field marker to predict neurocognitive abnormalities. Subsequent demonstration of impaired reaction time on formal neuropsychological testing is another quantitative factor that seems reliable in predicting outcome.

The authors attempted to assess the affective cumulative concussions and increase susceptibility. Although previous studies support this belief, the present study did not reveal poorer performance in the high school athletes who had experienced two or more concussions before their current injury. Because of sample size, definitive statements could not be made by the present authors. With computer-based neuropsychological testing, the axiom "three concussions and you're out" has little use. One severe concussion with prolonged neuropsychological impairment may be sufficient to interdict play, whereas three or more very mild concussions with minimum abnormalities may be acceptable.

Before the introduction of neuropsychological testing, return to play decisions after a cerebral concussion were often subjective, judgmental, and not supported by quantitative data. We think that computerized neuropsychological testing as outlined in this study will become the standard of care to assist physicians with their clinical judgment in assessing and managing athletes with mild traumatic brain injury. As the authors note, all teams in the NFL now use some form of neurocognitivie assessment after concussion injury and 20 out of 32 use the computer-based system, as do approximately 800 colleges and high schools throughout the United States. This will allow much larger studies to be performed, comparing not only high school, but also college athletes, as well as those in the NFL looking at a host of parameters to better evaluate and quantify the affects of mild traumatic brain injury in athletics.

Michael W. Collins
Neuropsychologist
Joseph C. Maroon
Pittsburgh, Pennsylvania

The authors are to be commended for creating widespread implementation of standardized cognitive assessment within professional and high school sports. Standardized cognitive assessment has been difficult to implement within any area of health care, and doing so within professional and high school football is a significant and laudatory accomplishment.

As the authors note, gaining acceptance from sports teams for cognitive assessment involved making such assessment minimally disruptive to the teams' day to day operations. This resulted in numerous compromises, all seeming to be reasonable under the circumstances, but all also introducing deviations to optimal scientific methodology. The authors are to be commended for being explicit regarding many of these limitations, and explicitly labeling their samples as "convenience samples."

The end product, thus, is an ambitious and significant undertaking, impressive in many ways, but based on less than optimal scientific methodology. This is true of many naturalistic studies and is not offered as a criticism. Rather, it is offered as a caution: the methodology of the present study is not sufficient to provide definitive answers to any of the hypotheses. Rather, the present study, as the authors themselves note, contains intriguing findings in need of replication. By being explicit regarding some of the methodological limitations of their study, the authors provide guidance regarding methodologic improvements to be expected of future studies.

The authors frequently note that this is the "first" study of its kind. As such, it is entitled to more methodological leeway than would be tolerable in subsequent studies.

Joseph Bleiberg
Neuropsychologist
Washington, District of Columbia

The authors have provided an initial comparison of concussive induced cognitive sequelae in high school and professional athletes. As this comparison provides novel information, it adds new data to the scientific literature of sports related head injuries. The main concern of this study, and it is a major concern, is that postconcussion test performance was not compared with the athlete's actual baseline. Rather, the authors used a much weaker convenience group design. Therefore, results of this study must be interpreted with significant caution.

Kenneth C. Kutner
Neuropsychologist
Hackensack, New Jersey

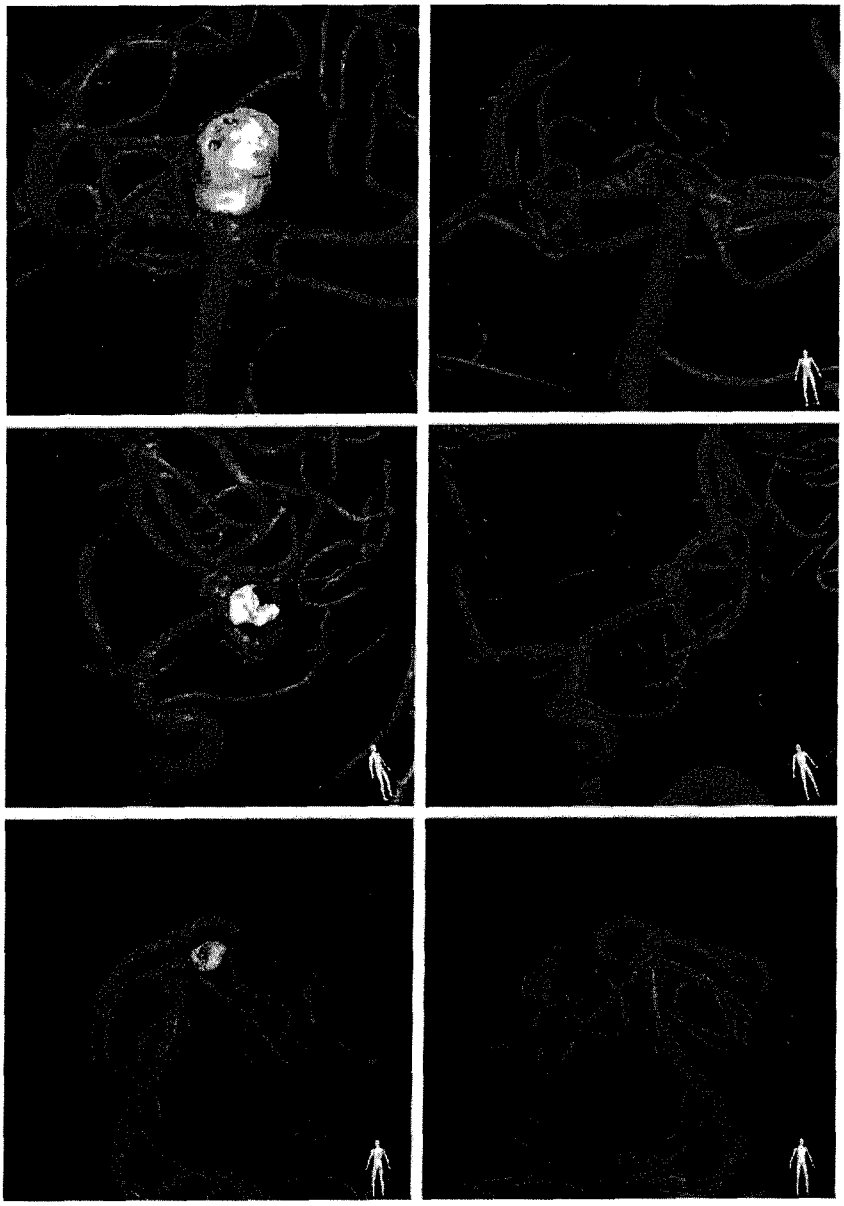
This study by Pellman et al. assesses the performance of concussed football players using computer-based neuropsychological tests and symptom inventory assessment compared with a similar, but identical, group and their preseason or baseline testing. This represents the implementation in the NFL of computer-based neuropsychological testing from 2000 and 2004. This study consisted of 68 NFL

and 125 high school football players using the ImPACT computerized neuropsychological testing methodology. They found that the NFL players had no significant neurocognitive deficits within the first week after injury. This was in contrast to residual neuropsychological deficits, particularly in reaction time and memory, in the high school population for a similar time course.

There is a good discussion regarding the development and implementation of neuropsychological testing at the NFL level which represents the first report of computer-based neuropsychological testing in professional athletes. They report their findings to help distinguish NFL versus younger athletes and discuss the "natural selection" process, as they have previously proposed. There does seem to be a difference between these particular age groups, and these findings may have bearing upon and should be considered by those caring for both the scholar and professional athletes. Further research will show whether these differences will continue to remain and whether there are inherent distinctions between mild traumatic brain injury occuring in younger age groups versus mature athletes with many years of contact sports experience. The authors are encouraged to continue their research with their unique patient population which should continue to add to our body of knowledge concerning athletic mild traumatic brain injury.

Julian E. Bailes
Morgantown, West Virginia

Three-dimensional rotational angiogram with coil mass depicted in white. After subtraction of the white coil mass, the angiogram shows no evidence of a residual neck. Twelve months after embolization, the wide-necked right superior cerebellar aneurysm remains completely occluded with preservation of the artery. (Feng L, Vinuela F, Murayama Y. Healing of intracranial aneurysms with bioactive coils. ***Neurosurg Clin N Am.*** *2005 Jul;16(3):487–99, v-vi).*



TAB 10

NFL PLAYERS
ASSOCIATION

NATIONAL FOOTBALL LEAGUE

POLICY AND PROGRAM

FOR SUBSTANCES OF ABUSE

2007

As Agreed by the National Football League Players Association
and the National Football League Management Council

As Amended June 1, 2007

TABLE OF CONTENTS

NATIONAL FOOTBALL LEAGUE POLICY AND PROGRAM FOR SUBSTANCES OF ABUSE

General Policy

The illegal use of drugs and the abuse of prescription drugs, over-the-counter drugs, and alcohol (hereinafter referred to as "substances of abuse") is prohibited[1] for players[2] in the National Football League ("NFL"). Moreover, the use of alcohol may be prohibited for individual players in certain situations where clinically indicated in accordance with the terms of this Policy.

Substance abuse can lead to on-the-field injuries, to alienation of the fans, to diminished job performance, and to personal hardship. The deaths of several NFL players have demonstrated the potentially tragic consequences of substance abuse. NFL players should not by their conduct suggest that substance abuse is either acceptable or safe.

The NFL and the National Football League Players Association ("NFLPA") have maintained policies and programs regarding substance abuse. In Article XLIV, Section 6(a) of the 1993 NFL Collective Bargaining Agreement, as extended and amended (the "CBA"), the NFL Management Council and the NFLPA (hereinafter referred to individually as "Party" and collectively as the "Parties") reaffirmed that "substance abuse is unacceptable within the NFL, and that it is the responsibility of the parties to deter and detect substance abuse . . . and to offer programs of intervention, rehabilitation, and support to players who have substance abuse problems." Accordingly, in fulfillment of this provision of the CBA, the Parties have agreed upon the following terms of a policy regarding substance abuse in the NFL (hereinafter referred to as the "Policy").

This Policy applies to all players who have not formally retired from the NFL. This Policy and its terms shall be binding on all NFL clubs and shall constitute the sole and exclusive means by which players will be tested or referred for treatment, and as to those players having problems with substances of abuse, the sole and exclusive means by which they will gain access to the benefits of this Policy. This Policy supersedes a previous policy that was effective on September 1, 1994, and shall continue until the expiration or termination of the CBA. This clarification of earlier versions that applied to all players as of February 14, 1997 and June 28, 1998 regardless of their status in the Intervention Program, shall apply to all players as of May 1, 2002 regardless of their status in the Intervention Program. *(See* Article I below.) Such terms that are not otherwise defined herein shall have the same meaning as set forth in the CBA.

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1 The National Football League prohibits players from the illegal use, possession, or distribution of drugs, including but not limited to cocaine, marijuana, opiates, methylenedioxymethamphetamine (MDMA) and phencyclidine (PCP). The abuse of prescription drugs, over-the-counter drugs, and alcohol is also prohibited. For example, the use of amphetamines and substances that induce similar effects, absent a verified and legitimate need for appropriate dosages of such substances to treat existing medical conditions, is prohibited.

2 Includes all present and future employee players in the NFL as described as being in the bargaining unit as set forth in the preamble to the CBA as well as all players who attend the Annual Player Combine while they are at the Combine.

The cornerstone of this Policy is the Intervention Program. Under the Intervention Program, players are tested, evaluated, treated, and monitored for substance abuse. Players who do not comply with the requirements of the Intervention Program are subject to established levels of discipline. The provisions of Article LV, Section 10 of the CBA are not applicable to the testing of players in the Intervention Program that is conducted pursuant to the terms of this Policy.

All discipline provided under the foregoing or following provisions of this Policy is imposed through the authority of the Commissioner of the National Football League ("Commissioner"). The Commissioner maintains the ability to impose other discipline as he deems necessary, provided, however, that the Commissioner may not modify the stated levels of discipline for violations of the requirements of the Intervention Stages absent additional circumstances amounting to conduct detrimental to the NFL. This Policy is not to be considered a grant of authority to discipline players but instead is an agreement to impose the stated discipline for violations of the requirements of the Intervention Stages. Discipline for violations of the law relating to use, possession, acquisition, sale, or distribution of substances of abuse, or conspiracy to do so, will remain at the discretion of the Commissioner.

The primary purpose of this Policy is to assist players who misuse substances of abuse, but players who do not comply with the requirements of the Policy will be subject to discipline. An important principle of this Policy is that a player will be held responsible for whatever goes into his body.

I. Intervention Program and Discipline for Violations of Its Terms.

A. Administration.

1. <u>Medical Director and Regional Teams</u>.

The Parties will select a Medical Director who will have the responsibility, among other duties, of selecting and overseeing physicians, psychologists, social workers and other counselors ("Evaluating Clinician(s)") who will be members of various Regional Teams headed by a Regional Psychiatrist. The Medical Director and the Evaluating Clinicians will work together in a collaborative manner to facilitate, coordinate, monitor, and assess players' compliance with their Treatment Plans. (For purposes of this Intervention Program, a "Treatment Plan" is defined as a written plan of interventions and requirements developed by a player's Treating Clinician to assist in the treatment of the player.) The Medical Director will be assisted by an Administrator to be selected by the Parties. The services of the Medical Director and/or the Administrator may be terminated by either Party at any time, or as otherwise delineated by the notice and termination provisions of their respective contracts, if any. The Parties agree that the Medical Director will have the sole discretion to make the various decisions assigned to him under the terms of this Policy, and such decisions shall be final and binding.

2. Medical Advisor.

The Commissioner will appoint a Medical Advisor for Substances of Abuse (the "Medical Advisor"). The Medical Advisor will be informed at all times of the identity and treatment status of all players in the Intervention Program with the exception of those entering the Intervention Stages through Self-Referral. The Medical Advisor may advise the Medical Director regarding a Treatment Plan for any player. The Medical Advisor will have the responsibility, among other duties, of overseeing substance abuse testing as required by the terms of this Intervention Program. The Medical Advisor shall direct the NFL collection teams and consult with the Chief Forensic Toxicologist. The Parties agree that the Medical Advisor will have sole discretion to make the various decisions assigned to him under the terms of the Policy, and such decisions shall be final and binding.

3. Treating Clinician.

A Treating Clinician for purposes of this Policy is a health care professional experienced and trained in the treatment of substance abuse and legally authorized to prescribe written plans of intervention and requirements designed to assist in the treatment of substance abuse. A health care professional who is not a psychiatrist and who wants to qualify as a Treating Clinician must establish a consulting relationship with an appropriately credentialed and experienced psychiatrist, as determined by the Medical Director. All Treating Clinicians shall be approved by the Medical Director.

4. Team Substance Abuse Physician.

Each NFL club will designate a physician as its Team Physician for Substance Abuse matters (the "Team Substance Abuse Physician"). With the exception of those players who enter the Intervention Stages through Self-Referral, the Team Substance Abuse Physician will be informed as to the participation of any player from his team in the Intervention Stages, the player's administrative status, and/or the nature of that player's treatment.

5. Chief Forensic Toxicologist.

The Commissioner will appoint the Chief Forensic Toxicologist. The Chief Forensic Toxicologist shall (1) be responsible for laboratory evaluation of urine samples produced pursuant to the terms of this Policy; (2) provide technical advice to the Parties, the Medical Director and the Medical Advisor; (3) be responsible for scientific interpretation of positive drug findings; and (4) provide forensic testimony as needed.

6. Club Physician.

Club Physicians are physicians designated by the clubs or selected by the player in accordance with Article XLIV of the CBA, "Players' Rights to Medical Care and Treatment."

7. Quarterly Review.

The NFL Management Council, the NFLPA, the Medical Director, the Medical Advisor, and the Administrator will meet quarterly to review the operation of the Intervention Program. To facilitate the review process, the Parties will have full access to all information relating to the implementation and operation of this Intervention Program, except to the extent that such access would conflict with the confidentiality provisions of this Policy. Modification of the Policy will require the mutual consent of the Parties.

8. Payment for Treatment.

Payment for treatment services rendered to players participating in the Intervention Program shall be governed by the terms and conditions set forth in the NFL Player Insurance Plan.

9. Treating Facilities and Clinicians.

Although it is the ultimate responsibility of the Medical Director in consultation with the Regional Team to designate suitable Treating Clinicians and treatment facilities at which players entering the Program will be treated ("Treatment Facilities"), at the time of the adoption of the initial Policy (September 1, 1994) many NFL teams had existing relationships with suitable Treating Clinicians and Treatment Facilities. In such cases, there was a presumption that the Treating Clinicians and Treatment Facilities being used by NFL clubs on September 1, 1994, were suitable. Notwithstanding the foregoing, the Medical Director may terminate the Program's relationship with any Treating Clinician or Treatment Facility if the Medical Director determines that such clinician or facility is unable to satisfy the medical requirements or other demands of this Policy. No Treatment Facility may be terminated until a replacement Treating Facility has been agreed upon. If the Parties are unable to agree upon a successor Treatment Facility within four (4) months of the notice from the Medical Director to the Parties of his desire to terminate a Treatment Facility, the matter shall be referred to the Medical Director and the Medical Advisor, who shall promptly select and consult with a third physician who is neither an Interested Party (as defined below) nor affiliated with an Interested Party; after consultation, the three physicians together will jointly choose a successor Treatment Facility as soon as practicable.

B. Confidentiality.

1. Program Information.

The Medical Advisor, Medical Director, Administrator, Team Substance Abuse Physician, Chief Forensic Toxicologist and all employees and consultants of the NFL, the NFL Management Council, the NFLPA (including its employees, members and Certified Contract Advisors), the Evaluating Clinicians, the Treating Clinicians and the NFL clubs ("Interested Parties") shall take reasonable steps to protect the confidentiality of information acquired in accordance with the

provisions of this Intervention Program, including but not limited to the history, diagnosis, treatment, prognosis, test results, or the fact of participation in the Intervention Program of any player or the club(s) who employs or has employed such player ("Intervention Program Information"). Any Intervention Program Information which becomes public information either by authorization of the player or through release by sources other than Interested Parties will, after authorization or release, no longer be subject to the confidentiality provisions of this section. Intervention Program Information about a player revealed by the player to an Interested Party shall not be subject to the confidentiality provisions of this section unless (a) it was disclosed by the player in the context of a clinical evaluation or treatment or was disclosed by the player to an Interested Party in accordance with the terms of this Policy; or (b) the player requests that the Intervention Program Information not be further disclosed by the Interested Party. Intervention Program Information, including but not limited to information learned on appeal, will be shared among Interested Parties only on a need-to-know basis and only in accordance with the terms of this Intervention Program.

2. Program Information Provided to Clubs.

An NFL club that:

a. has contacted a restricted or unrestricted free agent or that player's Certified Contract Advisor and is considering making an offer to and/or signing such player; or

b. has contacted another NFL club with regard to a potential acquisition of a player in a trade and is considering making the club an offer for such player; or

c. is contemplating acquiring a player through the waiver system;

may be informed by the Medical Advisor or the Management Council whether such player is subject to suspension the next time he fails to comply with any terms of the Intervention Program and whether or not in the opinion of the Medical Advisor or the Management Council a failure to comply with the terms of the Intervention Program has occurred at the time of the inquiry that could result in suspension. Both the Medical Advisor and the Management Council are authorized to disclose such information to the Team Substance Abuse Physician of the inquiring club or to the senior club executive responsible for signing restricted or unrestricted free agents who, in turn, shall share such information only with the club employee(s) or officer(s) who participate in the decision to sign such player. Any club employee or officer who, by reason of such inquiry, is in receipt of the information that the Medical Advisor or the Management Council is authorized to disclose pursuant to this section will immediately become subject to and be bound by the confidentiality provisions established by this Intervention Program.

3. **Discipline.**

a. **Fines:** Any Interested Party who violates the confidentiality provisions of this intervention Program may be fined a minimum of $10,000 up to a maximum of $500,000. Fines under this section shall be imposed by the Commissioner for individuals subject to his disciplinary authority and by the Executive Director of the NFLPA for individuals subject to his disciplinary authority. In the event the current disciplinary procedures of the NFLPA would not permit the levying of the fines prescribed herein, such procedures will be amended as soon as possible so as to authorize the disciplinary measures set forth in this paragraph.

b. **Determination:** Any player who believes that there has been a breach of the Intervention Program's confidentiality provisions as set forth in this section may report such violation to the Commissioner with respect to those individuals for whom he has authority to levy fines and the NFLPA's Executive Director with respect to those individuals for whom he has authority to levy fines to make a determination of whether a violation has occurred and the amount of the fine. The provisions of this section shall be the sole remedy available to a player aggrieved by an alleged violation of the Intervention Program's confidentiality provisions.

C. Testing for Substances of Abuse.

All testing for substances of abuse of NFL players either under contract with an NFL club or seeking a contract with an NFL club is to be conducted under the direction of the Medical Advisor pursuant to this Intervention Program ("Testing" or "Tests"). Before entering an Intervention Stage, players shall be tested only for benzoylecognine (cocaine); delta 9-THC-carboxylic acid (marijuana); amphetamine and its analogs; opiates (total morphine and codeine); phencyclidine (PCP); and methylenedioxymethamphetamine ("MDMA") and its analogues (the "NFL Drug Panel"). After a player enters any stage of the Intervention Program, Testing for substances of abuse, in addition to the NFL Drug Panel, shall be at the discretion of the Medical Director in accordance with the terms of this Intervention Program.

1. **Types of Testing.**

a. **Pre-Employment:** Unless otherwise required by this Policy, a pre-employment Test may be administered to:

(1) A rookie or veteran player desirous of signing a contract with an NFL club who:

(a) was not under contract to his last NFL club on the date of its last game of the immediately preceding season; and

(b) has not had a Test in the four month period prior to his pre-employment Test (excluding a test given at the annual combine for college players).

The Medical Advisor may inform any club contemplating signing a player who has been tested under the provisions of this subsection of the results of such pre-employment Test;

(2) A veteran player desirous of signing a contract with an NFL club who:

(a) was under contract with another NFL club on the date of its last game of the immediately preceding season; and

(b) agrees with the club with whom he is seeking employment, prior to the execution of a new NFL Player Contract to submit to a pre-employment Test.

(3) Draft-eligible players at the annual timing and Testing sessions.

b. **Pre-Season:** All players under contract with an NFL club will be tested once during the period beginning April 20 and continuing through August 9. Testing in the pre-season may be done on a team-wide basis or by position groups at the discretion of the Medical Advisor but not on an individual-by-individual basis. However, a player who is excused by the Medical Advisor from the scheduled team-wide or position's group Test may be tested individually but only if such Test takes place before the first regular season game absent a showing of extenuating circumstances. A player who is signed or otherwise acquired after the date of the pre-season Test that would have applied to him may be given the pre-season Test individually if such player has not already been given the preseason Test even though the Test will take place after August 9.

c. **Intervention Stages:** All players in an Intervention Stage will be required to provide a specimen when determined by the Medical Advisor. For players in Intervention Stage One, the Medical Director will determine the frequencies of Tests for each player; for players in Intervention Stages Two and Three, the Medical Advisor will determine the frequencies of Tests subject to the minimums and maximums for each Stage.

d. **Testing by Agreement:** An NFL club and a player may agree that such player will submit to unannounced Testing during the term of

said player's NFL Player Contract provided that the club has a reasonable basis for requesting such agreement. A Positive Test (as hereinafter defined) as a result of such Testing shall be reported to the Medical Director and shall result in the player's entering Stage One of the Intervention Program. Once a player enters an Intervention Stage the number of Tests that a player will be required to take will be determined by the Medical Director or the Medical Advisor, as set forth herein – not by the terms of the player's NFL Player Contract. Upon being dismissed from the Intervention Stages, a player's NFL Player Contract will govern the number of Tests that he is subjected to. All such individually negotiated Testing shall be conducted under the direction of the Medical Advisor and not the club. In cases of individually negotiated Testing, the Medical Advisor and other Interested Parties will continue to be bound by the confidentiality provisions established by this Policy.

2. Testing Laboratory.

A central laboratory certified by the Substance Abuse and Mental Health Services Administration ("SAMHSA Lab") will analyze all urine specimens for substances of abuse. NFLPA shall have a right to review the Policy's SAMHSA Lab annually. Although the NFLPA will have the right to terminate the SAMHSA Lab for cause at any time, no such termination will be effective unless and until the Parties have agreed on a successor laboratory. If no such successor laboratory is agreed upon within six months of the date on which the NFLPA gives notice of its desire to terminate, the matter shall be referred to the Medical Director and the Medical Advisor, who shall promptly select and consult with a third physician who is neither an Interested Party nor affiliated with an Interested Party; after consultation, the three physicians together will jointly choose a successor laboratory as soon as is practicable.

3. Testing Procedures.

a. Application: The following Testing procedures are applicable to all Testing performed in all Stages of the Intervention Program except certain unique Stage One procedures which are as follows:

(1) Unique Stage One Testing Procedures: A player in Stage One shall be tested as frequently as the Medical Director requests. In Stage One, there shall be no right to challenge the results of the Test and the Medical Director shall determine in his discretion whether failure or refusal to Test or an attempt to tamper with the results constitutes a player's failure to comply in Stage One subjecting the player to the discipline set forth in Article I, Section E.1.b.(2)(c). Except as set forth above, all other procedures

set forth in this Section C.3. shall be applicable to Stage One Testing.

b. **Collection of Sample:** At the time of his Test, the player will furnish a urine specimen to an authorized specimen collector, which specimen shall be split into an "A" bottle and a "B" bottle. To prevent evasive conduct, all specimens will be collected under observation. Specimens will be shipped in collection bottles sealed with tamper-resistant seals. All bottles will be identified by a control identification number, not by the player's name. The player will be asked to witness the entire procedure and then to sign the donor's statement on the chain-of-custody form. For more detailed information, refer to Appendix A ("Testing Procedures").

c. **Concentration Levels:** Tests will be deemed positive if they are confirmed by laboratory analysis at the following urine concentration levels: benzoylecognine (cocaine) – 150 ng/ml; delta 9-THC-carboxylic acid (*e.g.,* marijuana and marijuana by-products, including but not limited to hemp oil) – 15 ng/ml; amphetamine and its analogs – 300 ng/ml; opiates – 300 ng/ml (total morphine and codeine); phencyclidine (PCP) – 25 ng/ml; alcohol – .06 g/dl (%);and methylenedioxymethamphetamine (MDMA) and its analogues – 200ng/ml. Alcohol Testing will be conducted only in the context of clinical monitoring or as otherwise provided herein. Any Treatment Plan may include the provision for urine toxicology analysis for other substances not enumerated here and Tests will be deemed positive if they are confirmed by laboratory analysis at standard urine concentration levels recommended by the Chief Forensic Toxicologist and agreed to by the Parties. Any such positive test, as referenced in this subsection, shall hereinafter be referred to as a "Positive Test".

d. **Notification:** Once a Positive Test result is confirmed, the Medical Advisor will notify the player, the Medical Director and the Team Substance Abuse Physician. The Team Substance Abuse Physician may not disclose the fact of a Positive Test except in accordance with the terms of this Policy.

e. **Test of Split Sample:** Any player Testing positive from the "A" bottle of his split sample may, within two days of receiving notification of his Positive Test, ask the Medical Advisor for a Test of the other portion of his specimen from the "B" bottle. The "B" bottle Test is to be performed within ten working days of such request. The player may not be present at the "B" bottle Test, but, at his own expense, he may be represented at the "B" bottle Test by a qualified toxicologist not affiliated with a commercial laboratory. The "B" bottle Test will be performed at the same

laboratory that did the original Test. The "B" bottle Test need only show that the substance, revealed in the "A" bottle Test, is evident to the "limits of detection" to confirm the results of the "A" bottle Test.

f. **Failure or Refusal to Test:** The Medical Advisor will be responsible for scheduling all Tests and for ensuring that players are notified when individual Testing will take place. No test may be scheduled for a time that is more than 24 hours after notification. Players will have 4 hours from the time of the scheduled test to produce a specimen. A failure or refusal to appear for required Testing at the time selected by the Medical Advisor without a valid reason as approved by the Medical Advisor, or to cooperate fully in the Testing process as determined by the Medical Advisor, or to provide a dilute[3] specimen (as determined in accordance with the procedures set forth in Appendix A-1), will be treated as a Positive Test. In addition, a deliberate effort to substitute or adulterate a specimen; or to alter a Test result;[4] or to engage in prohibited doping methods[5] will be treated as a Positive Test and may subject a player to additional discipline by the Commissioner.

All players in an Intervention Stage who become unavailable for Testing due to travel, temporary or permanent change of residence, prior commitments, or otherwise, are required to notify the Medical Advisor in advance of such unavailability so that the Medical Advisor can schedule accordingly if such request is reasonable. If a player fails to provide the Medical Advisor with an address and telephone number where he can be contacted, and, as a result, such player cannot be contacted when the Medical Advisor requires that a Test be administered or the player cannot be contacted at the address and telephone number provided to the Medical Advisor, the player's failure to notify the Medical Advisor or inability to be contacted shall be considered a Positive Test.

D. Entrance Into the Intervention Stages.

1. Entrance.

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3 A "dilute specimen" shall be defined as a urine specimen which has a specific gravity value less than 1.003 and a creatinine concentration of less than 20 mg/dL.

4 Pharmacological, chemical or physical manipulation; for example, catheterization, urine substitution, tampering, or inhibition of renal excretion by, for example, probenecid and related compounds.

5 For purposes of this Policy, "prohibited doping methods" shall be defined as follows: Ingestion or injection of banned substances, or of products containing banned substances.

All NFL players shall be eligible for entrance into the Intervention Stages. Such eligibility will not be affected by termination or expiration of a player's contract subsequent to entry into the Intervention Stages. Players enter Stage One of the Intervention Program by a Positive Test, Behavior or Self-Referral more fully described as follows:

a. **Positive Test:** Urine or blood toxicology Tests that meet the concentration levels set forth in Article I, Section C.3.c.

b. **Behavior:** Behavior, including but not limited to an arrest related to an alleged misuse of substances of abuse, which, in the judgment of the Medical Director, exhibits physical, behavioral, or psychological signs or symptoms of misuse of substances of abuse.

c. **Self-Referral:** A player, who personally notifies the Medical Director of his desire to enter voluntarily Stage One of the Intervention Program prior to his being notified to provide a specimen leading to a Positive Test, or prior to behavior of the type described in Section D.1.b., above, becoming known to the Medical Director from a source other than the player, shall be a participant in Stage One as a Self-Referred player. Rather than notifying the Medical Director personally, the player may initiate such notice by first contacting a Club Physician for the purpose of Self Referral to Stage One of the Intervention Program. In order to effectuate a valid Self-Referral, the Club Physician must establish personal contact between the self-referring player and the Medical Director as soon as possible after being contacted by the player. Any information provided to the Club Physician by the player and disclosed by the Club Physician to the Medical Director for the purpose of establishing such contact will not be considered information from "a source other than the player" as described in subsection c(2) below. A Club Physician may not provide substance abuse treatment for any player or facilitate substance abuse treatment which is not provided by a Treating Clinician. Substance abuse treatment shall be provided only by a Treating Clinician in accordance with this Policy. A Self-Referred player will always remain in Stage One; however, a player will no longer be considered a Self-Referred player, but rather as a mandatory entrant into Stage One if:

(1) the player has a Positive Test (other than a Positive Test conducted pursuant to a Self-Referred player's Treatment Plan); or

(2) if the Medical Director is informed by a source other than the player that the player has engaged in behavior of the type described in Section D.1.b.,

above, regardless of the fact that the player has previously informed the Medical Director of this behavior; or

(3) the fact of a player's Self-Referral becomes public knowledge.

A Self-Referred player may not be fined under this Intervention Program prior to the time of his mandatory entrance into the Intervention Stages. Self-Referred players will be advised when the Medical Director determines that notification to the Team Substance Abuse Physician (if not previously notified by the player) is medically advisable, and the player will be given the option either to permit such notification or to withdraw from the Intervention Stages without such notification.

2. Continued Participation.

A player who enters the Intervention Stages will remain in the Intervention Stages until the player is dismissed or released in accordance with the terms set forth herein. All such players must continue to comply with the conditions of the Intervention Program. Notwithstanding the foregoing, (1) a player who is released and who has not been on a club roster for more than six consecutive regular or post season games ("Never-Rostered Player") is not required to comply with the terms of his Treatment Plan, if any, or submit himself for Testing until he resigns with a club; and (2) a veteran who is not under contract with a club ("Non-Contract Veteran") must comply with the conditions of the Intervention Program for a year after the expiration of his last contract or receipt by the Administrator of written notification of his retirement, whichever is sooner. After six months as a Non-Contract Veteran, testing shall cease unless the Medical Director or the Medical Advisor requests that testing be continued. After a veteran who is under contract with a Club ("Contract Veteran") or a Non-Contract Veteran notifies the Administrator of his retirement from football, he does not have to comply with the terms of his Treatment Plan. However, if after retiring from football, he signs a contract to play for an NFL club prior to the first anniversary date of (*i*) the expiration or termination of his last contract with an NFL club if a Non-Contract Veteran or (*ii*) the termination or tolling of his contract upon retirement if a Contract Veteran, he will be deemed not to have complied with the terms of his Treatment Plan and be disciplined for a violation of his Treatment Plan in accordance with the terms of this Policy. Non-Contract Veterans who either have not been under contract with an NFL club for a year or have notified the Administrator of their retirement; Contract Veterans who have notified the Administrator of their retirement; and released Never-Rostered Players who return to the NFL as a player, will re-enter the Intervention Program at the same place in the Intervention Program as when they left except as set forth above.

E. Intervention Stages.

1. **Stage One.**

a. **Procedures.**

(1) **Intervention Evaluation:** A player entering Stage One of the Intervention Program will be referred to a Regional Team which shall evaluate the player promptly. After receipt of the Regional Team's evaluation, the Medical Director, in his discretion, shall determine whether the player should be referred for appropriate clinical intervention and/or treatment (including inpatient treatment at a Treatment Facility selected by the Medical Director as being qualified to treat the player's particular needs) and subsequent development of a Treatment Plan. The Medical Director's determination is not dependent upon a finding that the player carries a diagnosis of substance abuse or dependence, but rather is dependent upon whether, in the Medical Director's judgment, participation in the Intervention Program may assist in preventing the player's potential future misuse of substances of abuse.

(2) **Treatment Plans:** If the Medical Director determines that a player should be referred for appropriate clinical intervention and/or treatment, the player shall be referred to a Treating Clinician. If the Treating Clinician determines the player requires a Treatment Plan, one shall be developed. The Medical Director shall determine whether the proposed Treatment Plan is an acceptable Treatment Plan for the purpose of affording the player the benefits of the Intervention Program.

Notwithstanding the foregoing, the Treating Clinician is solely responsible for the care of the player. A player who fails to adhere to his Treatment Plan approved by the Medical Director or fails to execute a Consent to Exchange Intervention Program Information shall be subject to the disciplinary and stage advancement provisions set forth below.

If the Medical Director determines that a player does not require clinical intervention and/or treatment, but should remain in the Intervention Program, the player will be subject to testing but will not have a Treatment Plan. However, if the Medical Director decides at any time while a player is in any stage of the Intervention Program that the player should be referred for clinical intervention and/or

treatment, a Treatment Plan shall be developed, if indicated.

(3) **Testing:** In Stage One, the Medical Director may require the player to submit to as many Tests for substances of abuse as, in his discretion, are required to adequately evaluate the player, and those Tests shall be administered under the direction of the Medical Advisor.

b. Duration.

(1) **Time Limitation:** Players will remain in Stage One for a period not to exceed 90 days; provided, however, that the Medical Director, in verbal consultation with the Medical Advisor, may extend the total time a player is in Stage One up to six months. If, due to unusual and compelling circumstances, the Medical Director determines that a period in excess of six months is required, the six-month period may be extended with the concurrence of the Medical Advisor.

(2) **Advancement to Stage Two:** Subject to the time limitations set forth in subsection (1) above, the Medical Director will determine how long a player will remain in Stage One. No player will be either advanced from Stage One to Stage Two or dismissed from the Intervention Stages without notification to the player by the Medical Director.

(a) **Behavior Evaluation:** A player who is referred to Stage One solely for Behavior and who upon evaluation in Stage One is deemed by the Medical Director not to require specific clinical intervention and/or treatment will immediately be released from any further obligations to participate in the Intervention Stages and will thereafter assume the same status as players who have never been referred to the Intervention Stages. However, a player who is referred to Stage One solely for Behavior, and who upon evaluation in Stage One, is deemed by the Medical Director to require specific clinical intervention and/or treatment, will be advanced to Stage Two upon notification to the player by the Medical Director. Notwithstanding the foregoing, a player who has a Positive Test while in Stage One shall be automatically advanced to Stage Two.

(b) **Positive Test Evaluation:** A player who is referred to Stage One by reason of a Positive Test, and who, upon evaluation in Stage One is deemed by the Medical Director not to require specific clinical intervention and/or treatment will advance to Stage Two upon notification to the player by the Medical Director, and will be subject to Stage Two Testing by the Medical Advisor but will not have a Treatment Plan. However, a player who is referred to Stage One by reason of a Positive Test and is deemed by the Medical Director to require specific clinical intervention and/or treatment, will be advanced to Stage Two upon notification to the player by the Medical Director.

(c) **Discipline:** If the Medical Director, after consultation with the Medical Advisor, determines in his discretion that a player in Stage One has failed to cooperate with the evaluation process or fails to comply with his Treatment Plan, both the NFL Management Council and the NFLPA shall be notified and the player will be subject to an immediate fine equal to three-seventeenths (3/17) of the amount in Paragraph 5 of the NFL Player Contract, and he will be placed in Stage Two upon notification by the Medical Director.

2. <u>Stage Two</u>.

a. **Procedures.**

(1) **Advancement:** A player will advance from Stage One to Stage Two after notification by the Medical Director or expiration of the Stage One time limitations.

(2) **Compliance with Treatment Plan:** A player in Stage Two must comply with the terms of his Treatment Plan, if one is developed, in Stage One and as may be amended in Stage Two.

(3) **Testing:** All players in Stage Two will be subject to unannounced Testing. At the sole discretion of the Medical Advisor, a player may or may not be tested; however, if he is tested, he may not be tested more than 10 times during any calendar month. Such Testing shall include Testing for the NFL Drug Panel and alcohol, but in addition Tests for other substances of abuse will be conducted if the player's

Treatment Plan requires abstention from and enumerates testing for such substances.

(4) **Evaluation:** A player, while undergoing Stage Two Testing, may be required to submit to further evaluation and subsequent treatment at the discretion of the Medical Director.

b. Discipline.

(1) **Discipline for First Failure to Comply in Stage Two:** A player in Stage Two who fails to comply with his Treatment Plan or fails to cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy, both as determined by the Medical Director, or has a Positive Test will be subject to:

(a) A fine of four-seventeenths (4/17) of the amount in Paragraph 5 of the NFL Player Contract if the player has successfully completed Stage One; and

(b) A suspension for the period of time to cover four consecutive regular season and post-season (including Pro Bowl, if selected) games without pay if he did not successfully complete Stage One.

(2) **Discipline for Second Failure to Comply in Stage Two:** A player who has two Positive Tests in Stage Two; or fails twice, as determined by the Medical Director, to comply with his Treatment Plan in Stage Two; or has a Positive Test and fails to comply with his Treatment Plan, as determined by the Medical Director; or fails to cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy, as determined by the Medical Director, will incur:

(a) A suspension for the period of time to cover four consecutive regular and post season games (including the Pro Bowl, if selected) without pay if the player was fined pursuant to Section E.2.b.(1)(a) above; and

(b) A suspension for the period of time to cover six consecutive regular and post season games (including the Pro Bowl, if selected) without pay if the player was suspended pursuant to Section E.2.b.(1)(b) above.

(c) The computation of the amount a player must forfeit and return to his Club as a result of a suspension under this section and banishment under Section 3.b(2) of this Article is set forth in Appendix D.

c. **Duration:** A player will remain in Stage Two for twenty-four months or two full seasons, whichever is shorter, beginning from the later of either the date of entry into Stage Two; or after entering Stage Two, the date upon which a player fails to comply with the Treatment Plan or fails to cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy, both as determined by the Medical Director or has a Positive Test. Notwithstanding the foregoing, without any notice a player will be automatically and immediately advanced to Stage Three if while in Stage Two he has any of the following:

(1) two Positive Tests; or

(2) two instances in which he fails to cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy or to comply with his Treatment Plan; or

(3) one Positive Test and one instance of a failure to comply with his Treatment Plan or cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy while in Stage Two.

d. **Completion of Stage Two:** A player who completes Stage Two without being advanced to Stage Three will be afforded the same status as a player who had never by Behavior or a Positive Test been referred to the Intervention Stages. Notwithstanding the foregoing, the Medical Director and the Medical Advisor may agree to extend the period of Stage Two for any player; provided, however, before extending the period of time in Stage Two, the Medical Director shall meet with the player (who shall attend either in person or by telephone at the option of the player), along with the Medical Advisor (who shall attend either in person or by telephone at the option of the Medical Advisor), at least 30 days before the expiration of the player's two year period in Stage Two (unless the justification for the extension occurs less than 30 days prior to the expiration of the normal term). The purpose of this meeting is to inform the player that his term in Stage Two is going to be extended and establish a time (no later than every six months after the normal expiration date) when the Medical Director and the Medical Advisor will confer with the player to review his

situation. At each conference, the player shall be informed what is expected of the player during each extension period.

3. **Stage Three.**

a. Procedures.

(1) **Term:** A player in Stage Three will remain in Stage Three for the remainder of his NFL career.

(2) **Compliance with Treatment Plan:** A player in Stage Three must comply with the terms of his Treatment Plan, as required in Stage Two and as may be developed and/or amended in Stage Three.

(3) **Testing:** A player in Stage Three will be subject to unannounced Testing. At the sole discretion of the Medical Advisor, a player may or may not be tested; however, if he is tested, he may be tested up to 10 times during any calendar month. Such Testing shall include Testing for the NFL Drug Panel and alcohol, but in addition Tests for other substances of abuse will be conducted if the player's Treatment Plan requires abstention from and enumerates testing for such substances. After being in Stage Three for three seasons, a player may request of the Medical Advisor that the number of tests that he is subject to be reduced. The Medical Advisor may, but is not required to, agree to the request. A player in Stage Three may not make this request more often than annually. The Medical Advisor, after consultation with the Medical Director, may extend or resume this period of Testing.

(4) **Evaluation:** A player, while undergoing Stage Three Testing, may be required to submit to further evaluation and subsequent treatment at the discretion of the Medical Director.

b. Discipline.

(1) **Discipline for Failure to Comply in Stage Three:** A player who fails to cooperate with testing, treatment, evaluation or other requirements imposed on him by this Policy or fails to comply with his Treatment Plan, both as determined by the Medical Director, or who has a Positive Test, will be banished from the NFL for a minimum period of one calendar year.

(2) **Banishment:** A player banished from the NFL pursuant to subsection (1) above will be required to adhere to his Treatment Plan and the provisions of this Intervention Program during his banishment. During a player's period of banishment, his contract with an NFL club shall be tolled.

c. **Reinstatement.**

(1) **Criteria:** After the completion of the one-year banishment period, the Commissioner, in his sole discretion, will determine if and when the player will be allowed to return to the NFL. A player's failure to adhere to his Treatment Plan during his banishment will be a significant consideration in the Commissioner's decision of whether to reinstate a player. A player seeking reinstatement must meet certain clinical requirements as determined by the Medical Director and other requirements as set forth in Appendix B.

(2) **Procedures After Reinstatement:** If a player is reinstated, he will remain in Stage Three for the remainder of his NFL career, subject to continued Testing and indefinite banishment. A player allowed to return to the NFL following a banishment must participate in continued treatment under this Intervention Program as required by the Medical Director.

4. **Notice to NFL Management Council and NFLPA.**

The NFL Management Council and the NFLPA shall be notified whenever an event occurs that will subject a player to discipline in any Intervention Stage.

F. Notice.

Players who are in any of the Intervention Stages are required to provide the Medical Advisor and the Medical Director with an address and telephone number where they can be reached at all times, and the Medical Advisor shall attempt to notify the player using the method that is reasonably calculated to provide notice to the player in a timely manner. Any notice required to be provided to a player under this Policy will be deemed to be delivered on the earlier of (1) four business days after mailing by regular mail to the address either provided by the player pursuant to this Section or maintained by the player's club, or (2) actual delivery or notice which for purposes of this Policy shall be deemed to have occurred at the time that a voice mail is left at the telephone number provided by a player or that a Federal Express or other similar means of overnight delivery, waiving signature, is delivered to the address provided by a player.

II. Discipline for Violation of Law Related to Substances of Abuse Other Than Alcohol.

A. Additional Commissioner Discipline.

Apart from and in addition to any other provisions of this Policy, players convicted of or admitting to a violation of law (including, within the context of a diversionary program, deferred adjudication, disposition of supervision, or similar arrangement including but not limited to *nolo contendere)* relating to use, possession, acquisition, sale, or distribution of substances of abuse other than alcohol, or conspiring to do so, are subject to appropriate discipline as determined by the Commissioner. Where appropriate, such discipline may include substantially longer suspensions than those set forth below.

B. Discipline for Violations of the Law.

A player will normally be subject to discipline up to and including suspension without pay for four regular and/or post-season games for a first violation of the law related to substances of abuse other than alcohol and for six regular and/or post-season games for a second violation of the law related to substances of abuse other than alcohol. A player's treatment history may be considered by the Commissioner in determining the appropriate degree of discipline. The suspension period may be extended if medically necessary, and, if extended, may involve mandatory treatment if required by the Medical Director.

III. Discipline for Alcohol-Related Violations of Law or Abuse of Alcohol.

A. Abusive Consumption.

Although alcoholic beverages are legal substances, when consumed abusively they can produce or contribute to conduct that is unlawful and threatens the health and safety of players and other persons. Such conduct is detrimental to the integrity of and public confidence in the NFL and professional football. In addition, the abusive consumption of alcoholic beverages may indicate a substance abuse problem that requires medical attention.

B. Alcohol-Related Offenses.

The Commissioner will review and may impose a fine, suspension, or other appropriate discipline if a player is convicted of or admits to a violation of the law (including within the context of a diversionary program, deferred adjudication, disposition of supervision, or similar arrangement including but not limited to *nolo contendere)* relating to the use of alcohol. Absent aggravating circumstances, discipline for a first offense will generally be a fine of one-half (1/2) of one-seventeenth (1/17) of the amount in Paragraph 5 of the NFL Player Contract to a maximum of $20,000. If the Commissioner finds that there were aggravating circumstances, including but not limited to felonious conduct or serious injury or death of third parties, and/or if the player has had prior drug or alcohol-related misconduct, increased discipline up to and including suspension may be imposed. Discipline for a second or subsequent offense is likely to be a suspension, the duration of which may escalate for repeat offenses.

IV. Imposition of Fines and Suspensions.

A. Fines.

1. Computation and Collection of Fines.

a. Computation: A club shall collect any player fine imposed pursuant to this Policy over the remaining pay periods of the season in which the fine is imposed in equal installments of no more than one-half of the player's gross salary; however, a player's salary shall not be reduced to such an extent that his per week take home pay would be less than $13,000 or thirty percent (30%) of one-seventeenth (1/17) of the amount in Paragraph 5 of the NFL Player Contract whichever is lower. The amount of the fine shall be calculated using the player's contract at the time of his failure to comply with the terms of the Policy or his last contract if not under contract to an NFL Club at the time of his failure to comply. If the incident giving rise to the fine occurs prior to the first day of the League Year, the contract for the season immediately prior to such first day of the League Year shall be used; if the incident giving rise to the fine occurs on or after the first day of the League Year, the player's contract, if any, for the season immediately following such first day of the League Year will apply. Any deferred compensation attributable to a game missed due to suspension or to a fine period shall be reduced or eliminated as appropriate. Any fines imposed for violations of this Policy shall be applied to the costs of the Policy.

b. Split Seasons/Different Clubs: Should a club be unable to collect the full amount of the fine during the season of its imposition, the

remaining portion of the fine shall be collected the following season(s). If, at the beginning of the next regular season, the player is under contract to the same club, the remainder of the fine imposed pursuant to this Policy will be collected by said club in equal installments of no more than one-half of the player's gross salary, if necessary, for each pay period until the fine is paid in full; however, a player's salary shall not be reduced to such an extent that his per-week take home pay would be less than $13,000 or thirty percent (30%) of one-seventeenth (1/17) of the amount in Paragraph 5 of the NFL Player Contract whichever is lower. If at the beginning of the next regular season, the player is under contract to a different club, the remainder of the fine imposed pursuant to this Policy will be collected by the new club in equal installments of one-half of the player's gross salary for each pay period until the fine is paid in full; however, a player's salary shall not be reduced to such an extent that his take home pay is less than $13,000 or thirty percent (30%) of the amount (or pro rata amount for players playing less than a full season) in Paragraph 5 of the NFL Player Contract, whichever is lower. If, at the beginning of the next regular season, the player is not under contract to any NFL club, the remainder of the fine imposed pursuant to this Policy may be recovered from any monies still owing from the NFL, or any of its clubs, to the player, including any salary or other form of compensation remaining owed pursuant to paragraphs 5 and 24 of a prior NFL Player Contract, any deferred compensation and any termination pay, but not including severance pay.

2. <u>Prohibition Against Club Payment of Fine</u>.

No club shall be permitted to pay any fine imposed pursuant to this Policy for or on behalf of a player so fined, nor shall a club be permitted to increase such player's compensation so as to cover, in whole or in part, the total amount of the fine.

B. Suspensions.

1. <u>Suspension Procedures</u>.

During any suspension, the player will not receive any pay, including pay for any post-season game that he misses because of his suspension, except as provided by Article XLII, Section 4 of the CBA. Notwithstanding the foregoing, if a bye week occurs during a suspension period, the player will receive his compensation for the bye week in equal installments over the remainder of the season after expiration of his suspension for as long as he is under contract and with the club that he was under contract with at the time of the commencement of his suspension. The disciplinary period will begin on the date set in the NFL's notification to the player of his suspension, subject to any appeal. If there are

fewer than the prescribed number of games remaining when the suspension begins, including any post-season games for which the club qualifies, the suspension will continue into the next regular season until the prescribed number of games has been missed.

In regard to all suspensions other than a banishment imposed pursuant to Stage Three of the Intervention Program, the player shall be eligible to participate in all off-season (not including post-season games) and pre-season club functions, training programs, practices, pre-season games and meetings, up to and including the date of the team's last pre-season game in the next NFL season. Additionally, the player shall be eligible, at the discretion of the club, to participate in all in-season club functions, training programs and meetings, but not in any in-season games or practices. Notwithstanding the foregoing, a player may not participate with his team in joint press conferences with team officials on or off club premises. A player banished pursuant to Stage Three of the Intervention Program may not participate with his club in any way except to see his Treating Clinician for treatment purposes on club property, but he must vacate the premises immediately following termination of the treatment session with the Treating Clinician. A free agent will serve his suspension as if he had a contract with a club. Any suspension period may be extended if medically necessary, and, if extended, may involve mandatory treatment if required by the Medical Director in his discretion.

2. Post-Season Treatment of Suspension or Fine.

Any suspension without pay imposed pursuant to the terms of this Policy shall include post-season games played by his team if, at the time of suspension, an insufficient number of games remain in the regular season to satisfy the period of suspension. Similarly, any fines remaining owed at the conclusion of the regular season will continue to be deducted from a player's post-season compensation, if any, in accordance with the provisions of subsection 1 above, except as provided below. If a player would otherwise qualify for a payment of post-season compensation pursuant to Article XLII, Section 4 of the CBA, such postseason pay shall not be affected by administrative actions imposed pursuant to the terms of this Policy.

3. Examination in Connection With Reinstatement.

A player seeking reinstatement from any suspension imposed under this Policy must be given a physical examination and physically cleared by the Team Substance Abuse Physician before he may participate in contact drills or in a game. Such examination shall not include drug Testing.

V. Appeal Rights.

Any dispute concerning the application, interpretation or administration of this Policy (hereafter "grievance") shall be resolved exclusively and finally through the following procedures.

A. Presentation of Grievances.

1. Disciplinary Appeals.

Any player who is notified by the NFL Management Council that he is subject to a fine or suspension (hereafter "discipline") for violation of the terms of this Policy may appeal such discipline directly to the Commissioner. A player must do so in writing within five (5) days of receiving notice from the NFL that he is subject to discipline. In his appeal, the player may not present grievances that have not been raised in a timely manner in accordance with this Article V, nor may he present defenses to the imposition of discipline that are the subject of "Other Appeals" set forth in Section A.2., below.

2. Other Appeals.

Any player who has a grievance over any aspect of the Policy other than discipline, including but not limited to claims of disparate treatment, must present such grievance to the NFLPA (with a copy to the NFL Management Council) within five (5) days of when he knew or should have known of the grievance. The NFLPA will endeavor to resolve the grievance in consultation with the NFL Management Council. Thereafter, the NFLPA may, if it determines the circumstances warrant, present such grievance to the Commissioner for final resolution. Such appeal must be presented to the Commissioner no later than thirty (30) days after the player's presentment of the grievance to the NFLPA.

3. Stays.

With the sole exception of the imposition of discipline, no other requirements of this Policy will be stayed pending the outcome of an appeal.

B. Conduct of Appeals Before the Commissioner.

1. Hearing.

The Commissioner will designate a time and place for a hearing (either in person or by telephone), at which either he or his designee will preside. A player may be accompanied by counsel and present relevant evidence or testimony in support of his appeal.

2. Effect of Pendency of Appeal.

A fine or suspension that has been timely appealed shall not take effect until completion of the player's appeal. However, the pendency of a grievance or appeal shall not excuse a player from compliance with any other aspect of the Policy.

3. Discovery.

In presenting a grievance or appeal concerning this Policy, the player shall be entitled to access only the information upon which the decision to impose disciplinary action was based; however, in no event will a player have access to records or reports concerning the participation in or application of this Policy to any other player.

4. Procedural Disputes.

The Commissioner shall have exclusive and final authority to resolve all issues affecting the presentation of grievances and the conduct of appeals, including the timing and location of the hearing, the timeliness of grievances and appeals, access to information, and the relevance of evidence. All issues affecting the presentation of grievances and the conduct of appeals (including disputes over discovery issues) that are known to either party to an appeal hearing must be resolved at least 48 hours prior to the commencement of the appeal hearing. Notwithstanding the foregoing, the player or his representative may argue such other issues if they become known to him after the time for resolution set forth above.

5. Witnesses.

Any professional who interacts with a player pursuant to the terms of this Program, including, but not limited to a Treating Clinician, an Evaluating Clinician, an authorized specimen collector, or a consulting psychiatrist, may not testify at an appeal hearing unless the professional will testify as to matters that only the professional has substantial knowledge. A player or his representative desirous of having a professional testify at a hearing must proffer to (i) the Medical Advisor in the case of a specimen collector or (ii) the Medical Director in the case of any other professional, the testimony that the professional is going to give and an explanation of why that professional is the only one who has substantial knowledge of that information. After the proffer, the Medical Advisor or the Medical Director, depending on to whom the proffer was made, shall determine whether to permit the professional to testify, but only after consulting with the Program's counsel and the Management Council and the NFLPA. The player and/or his representative may not communicate with any professional who interacts with the player pursuant to the terms of this program unless it is determined that the professional may testify at the appeal hearing.

6. Issues on Appeal, Witnesses, and Evidentiary Documents.

At least 96 hours prior to the hearing, the player or his representatives must present a statement of issues known to the player or his representatives at that time to be argued on appeal. Notwithstanding the foregoing, the player or his representatives may argue such other issues if they become known to him after submission of the statement of issues. Additionally, each side must present to the other a list of witnesses who are to testify at the appeal hearing and copies of evidentiary documents that they intend to introduce at the appeal hearing at least 96 hours prior to the appeal.

7. Commissioner Determination.

Within a reasonable period of time, following the hearing, the Commissioner will issue a written decision which will constitute a full, final, and complete disposition of the appeal and which will be binding on all parties.

VI. Miscellaneous.

A. Alcoholic Beverages and NFL Club Responsibility.

Alcoholic beverages are prohibited in club locker rooms. Clubs are responsible for taking appropriate measures to prevent abuse of alcohol on team flights to and from games.

APPENDIX A

Collection Procedures

Upon reporting to the collection site, a player shall be required to produce a government issued photo ID. Once identification is completed, the player will be asked to break through a heat-sealed plastic bag containing a urine specimen cup. To prevent evasive conduct, the player will then furnish a urine specimen under observation by a member of the collection team. Thereafter, in the presence of the player, the integrity seal on a sealed collection kit will be broken. This kit will be used to store and ship his urine specimen. The player will be asked to verify that the collection kit was sealed. After affording the player the opportunity to observe, the specimen will be split between an "A" bottle and a "B" bottle, and resealed with security seals. The player will be asked to sign the chain-of-custody form.

In the pre-season collection process, the player's urine specimen will be divided between two different Testing kits – one for anabolic steroid Testing and one for substances of abuse Testing.

Once the bottles for substances of abuse have been sealed and the chain-of-custody form has been completed, the bottles will be inserted into containers and placed back into the kit. The kit will then be sealed and sent by Federal Express or similar carrier to the Testing laboratory.

All bottles will be identified by a control identification number. The number on the bottles will be the same as the number on the chain-of-custody form. The Testing laboratories will be unable to associate any specimen with an individual player.

APPENDIX A-1

PROCEDURES FOR DILUTE SPECIMENS

The National Football League Players Association and the National Football League Management Council have agreed that, effective May 1, 2006, the following procedures and standards will be used to determine whether a "dilute" specimen is the equivalent to a Positive Test under Section I.C.3.f of the NFL Policy and Program for Substances of Abuse ("Program").

1. A dilute specimen will be tested to the "limits of detection" to determine if there is a presence of any substance banned by the Program or by an individual player's treatment plan. The presence of such substance, when the specimen is tested to the "limits of detection," shall be referred to as an "LOD Positive"; the absence of such substance shall be referred to as an "LOD Negative."

2. Any player who provides a dilute specimen during Pre-Employment Testing (Section I.C.1.a) or Pre-Season Testing (Section I.C.1.b) shall enter Stage One of the Intervention Program, as follows:

   a. Players who provide a dilute urine specimen that is an LOD Positive shall enter Stage One of the Intervention Program by Positive Test (Section I.D.1.a);

   b. Players who provide a dilute urine specimen that is an LOD Negative shall enter Stage One of the Intervention Program by Behavior (Section I.d.1.b)

3. A player who is in either Stage Two or Stage Three of the Intervention Program and provides a dilute urine specimen that is an LOD Positive shall be deemed to have had a Positive Test.

4. Each time a player enters the Intervention Program, he will be warned the first time he provides a dilute specimen that is LOD Negative after being advanced to Stage Two; however, after this one warning, a player in Stage Two or Stage Three who provides another dilute specimen that is LOD Negative shall be deemed to have produced a Positive Specimen.

Dress Code: The player's dress code for NFL drug testing is <u>BARE ABOVE THE KNEES</u>. No shirts or other upper body garments are to be worn for a test and all lower body garments are to be lowered to the knees.

APPENDIX B

Procedures for Making Application for Reinstatement by a Player Banned Under Stage Three of the Intervention Program

Any player who has been suspended under Stage Three may apply formally in writing for reinstatement no sooner than 60 days before the one-year anniversary date of the letter so suspending him.

The application should include all pertinent information about the player's

(a) Treatment;
(b) Abstinence from substances of abuse throughout the entire period of his suspension;
(c) Involvement with any substances of abuse related incidents; and
(d) Arrests and/or convictions for any criminal activity, including substances of abuse related offenses

Set forth below are the procedures to be used when an application is received by the Commissioner.

1. Within 45 days of receipt of the application, the player will be interviewed by the Medical Director and the Medical Advisor after which a recommendation will be made to the Commissioner with regard to the player's request for reinstatement.

2. The player will execute appropriate medical release forms that will enable the Commissioner's staff and NFLPA Executive Director's staff to review the player's substance abuse history, including but not limited to attendance at counseling sessions (individual, group and family); attendance at 12-step and other self-help group meetings; periodic progress reports; and all diagnostic findings and treatment recommendations.

3. The player will submit to urine Testing by an NFL representative at a frequency determined by the Medical Advisor.

4. The player will agree in a meeting with the Commissioner or his representative(s) to comply with the conditions imposed by the Commissioner for his reinstatement to the status of an active player.

5. All individuals involved in the process will take steps to enable the Commissioner to render a decision within 60 days of the receipt of the application.

APPENDIX C

Addresses and Phone Numbers

Medical Advisor

Lawrence S. Brown, M.D.
229A Carroll Street
Brooklyn, NY 11231

Business Phone:	718-522-7363
Business FAX:	718-596-5666
Email:	nflbrown@aol.com

Administrator

ERM Associates, Inc.
Suite C
221 Mount Hermon Road
Scotts Valley, CA 95066

	1-800-880-2376
	831-430-1533
	wcbrigham@cruzio.com

APPENDIX D

A Player who is suspended under this Policy shall forfeit and return to his Club (or forego entitlement to unpaid portions of) the proportionate amount of his signing bonus corresponding to the period of the suspension; provided that, if (a) the suspension is for a period of one year or more, (b) the Player's Contract is tolled during such suspension, and (c) the Player subsequently performs under the Contract during the expended period that results from the tolling, then the Player shall earn back the proportionate amount of forfeited or foregone signing bonus for the extended period in which he performs. For purposes of this Section, "proportionate amount" means $1/17^{th}$ of the signing bonus allocation for each regular season week or regular season game missed per League Year covered by the suspension, or $1/17^{th}$ of the forfeited or foregone signing bonus allocation for each regular season week or regular season game subsequently player per extended year of the Player's Contract, in the case of a Player earning back previously forfeited or forgone signing bonus.

By way of example, without limitation on any other example, if a Player with a four-year Player Contract for the 2006-2009 League Years that contains a signing bonus of $4 million is suspended for the 2007 and 2008 League Years for violation of the Policy, then the Player would forfeit and return to his Club $2 million in signing bonus allocation ($1 million for the 2007 League Year and $1 million for the 2008 League Year). If, after performing under the Player Contract in the 2009 League Year, the Player then performed one of his previously tolled years in the 2010 League Year, he would earn back $1 million. If the Player then performed for eight games of the second of his previously tolled years in the 2011 League Year and then retired, he would earn back an additional $470,588 (8/17 x $1 million).

TAB 11

NFL PLAYERS
ASSOCIATION

NATIONAL FOOTBALL LEAGUE

POLICY ON ANABOLIC STEROIDS AND RELATED SUBSTANCES 2007

as Agreed by the National Football League Players Association and the National Football League Management Council in the NFL Collective Bargaining Agreement, as Amended

June 21, 2007

NATIONAL FOOTBALL LEAGUE POLICY
ON ANABOLIC STEROIDS AND RELATED SUBSTANCES

TABLE OF CONTENTS

NATIONAL FOOTBALL LEAGUE POLICY
ON ANABOLIC STEROIDS AND RELATED SUBSTANCES

1. General Statement of Policy

The National Football League prohibits the use by NFL players of anabolic/androgenic steroids (including exogenous testosterone), certain stimulants, human or animal growth hormones, whether natural or synthetic, and related or similar substances. (See Appendix A). For convenience, these substances, as well as masking agents or diuretics used to hide their presence, will be referred to as "Prohibited Substances".[1] These substances have no legitimate place in professional football. This policy specifically means that:

-- **PLAYERS** may not, under any circumstances, have Prohibited Substances in their systems.

-- **COACHES, TRAINERS, OR OTHER CLUB PERSONNEL** may not condone, encourage, supply, or otherwise facilitate in any way the use of Prohibited Substances.

-- **TEAM PHYSICIANS** may not prescribe, supply, or otherwise facilitate a player's use of Prohibited Substances.

-- **All PERSONS**, including players, are subject to discipline by the Commissioner for violation of this Policy or of laws relating to possession and/or distribution of Prohibited Substances, or conspiracy to do so.

The League's concern with the use of Prohibited Substances is based on three primary factors. First, these substances threaten the fairness and integrity of the athletic competition on the playing field. Players use steroids for the purpose of becoming bigger, stronger, and faster than they otherwise would be. As a result, steroids and related

[1] An illustrative list of Prohibited Substances (see Appendix A) is attached to this Policy. Please note that, in addition to the substances specifically named, other categories and related substances can also violate the Policy.

substances threaten to distort the results of games and League standings. Moreover, players who do not wish to use these substances may feel forced to do so in order to compete effectively with those who do. This is obviously unfair to those players and provides sufficient reason to prohibit their use.

Second, the League is concerned with the adverse health effects of steroid use. Although research is continuing, steroid use has been linked to a number of physiological, psychological, orthopedic, reproductive, and other serious health problems.

Third, the use of Prohibited Substances by NFL players sends the wrong message to young people who may be tempted to use them. High school and college students are using these substances with increasing frequency, and NFL players should not by their own conduct suggest that such use is either acceptable or safe, whether in the context of sports or otherwise.

The NFL Player Contract specifically prohibits the use of drugs in an effort to alter or enhance performance. The NFL Player Contract and the League's Constitution and Bylaws require each player to avoid conduct detrimental to the NFL and professional football or to public confidence in the game or its players. Steroid use violates both these provisions. In addition, the Commissioner is authorized to protect the integrity of and public confidence in the game. This authorization includes the authority to forbid use of the substances prohibited by this Policy.

2. Administration of the Policy

As agreed in the 1993 Collective Bargaining Agreement, the program is conducted under the auspices of the NFL Management Council. The program will be directed by the NFL Advisor on Anabolic Steroids and Related Substances ("Advisor"). The Advisor shall have the sole discretion to make determinations regarding steroid-related matters, including medical evaluations and testing. He will also make himself available for consultation with players and team physicians; oversee the development of educational materials; participate in research on steroids; confer with the Consulting Toxicologist;[2]

[2] The Consulting Toxicologist on Anabolic Steroids and Related Substances ("Consulting Toxicologist") will consult on testing procedures and results, laboratory quality, and other issues referred to him by the Advisor. For more information, see Appendix B ("Personnel").

and serve on the League's Advisory Committee on Anabolic Steroids and Related Substances.[3]

3. Testing for Prohibited Substances

A. Types of Testing

All testing of NFL players for Prohibited Substances, including any pre-employment testing, is to be conducted pursuant to this Policy. All urine samples will be collected by an authorized specimen collector and tested at the appropriate laboratory (see Section 3D below). As is the case in the employment setting, players testing positive in a pre-employment setting will be subject to medical evaluation and clinical monitoring as set forth in Sections 3A, 4C and 12, and to the disciplinary steps outlined in Section 6.

Testing will take place under the following circumstances:

Pre-Employment: Pre-employment tests may be administered to free agent players (whether rookies or veterans). In addition, the League will conduct tests at its annual timing and testing sessions for draft-eligible football players.

Annual/Preseason: All players will be tested for Prohibited Substances at least once per League Year. Such testing will occur at training camp or whenever the player reports thereafter, and will be deemed a part of his preseason physical. In addition, random testing will be conducted during the weeks in which preseason games are played.

Regular Season: Each week during the regular season, ten (10) players on every team will be tested. By means of a computer program, the Advisor will randomly select the players to be tested from the club's active roster, practice squad list, and reserve list who are not otherwise subject to ongoing reasonable cause testing for steroids. The number of players selected for testing will be determined in advance on a uniform basis. Players will be required to test whenever they are selected,

[3] The Advisory Committee on Anabolic Steroids and Related Substances is appointed by the Commissioner and chaired by the Advisor.

without regard to the number of times they have previously been tested.

Postseason: Ten (10) players on every team qualifying for the playoffs will be tested periodically so long as their club remains active in the postseason. Players to be tested during the postseason will be selected on the same basis as during the regular season.

Off-Season: Players under contract who are not otherwise subject to reasonable cause testing may be tested during the off-season months up to 6 times at the discretion of the Advisor. Players to be tested in the off-season will be selected on the same basis as during the regular season, irrespective of their off-season location. Any player selected for testing during the off-season will be required to furnish a urine specimen at a convenient location acceptable to the Advisor. Only players who advise in writing that they have retired from the NFL will be removed from the pool of players who may be tested. If, however, a player thereafter signs a contract with a club, he will be placed back in the testing pool.

Reasonable Cause Testing For Players With Prior Positive Tests Or Under Other Circumstances: Any player testing positive for a Prohibited Substance, including players testing positive in college or at a scouting combine session, or with otherwise documented prior steroid involvement, will be subject to ongoing reasonable cause testing at a frequency determined by the Advisor. Such players will be subject to ongoing reasonable cause testing both in-season and during the off-season. Reasonable cause testing may also be required when, in the opinion of the Advisor, available information provides a reasonable basis to conclude that a player may have violated the Policy or may have a medical condition that warrants further monitoring. (See Section 12.)

B. Testing Procedures

In-season tests will ordinarily be conducted on two days each week, and each player to be tested will be notified on the day of the test. On the day of his test, the player will furnish a urine specimen to a DPA who will be present at the team facility.

To prevent evasive techniques, all specimens will be collected under observation by an authorized specimen collector. Specimens will be shipped in collection bottles with

tamper-resistant seals. Each bottle will be identified by a control identification number, not by the player's name. The player will be given an opportunity to witness the procedure and to sign the chain-of-custody form. For more detailed information, see Appendix C ("Collection Procedures").

C. Failure or Refusal to Test/Efforts to Manipulate Specimen or Test Result

An unexcused failure or refusal to appear for required testing, or to cooperate fully in the testing or evaluation process, will warrant disciplinary action. Any effort to substitute, dilute or adulterate a specimen, or to manipulate a test result to evade detection will be considered a violation of the Policy and likely will result in more severe discipline than would have been imposed for a positive test.

D. Testing Laboratories

The Advisor will determine the most appropriate laboratory or laboratories to perform testing under the Policy. Currently, the UCLA Olympic Analytical Laboratory in Los Angeles and the Sports Medicine Research and Testing Laboratory in Salt Lake City have been approved to analyze specimens collected for Prohibited Substances in a player's urine. These laboratories have been accredited by ISO and the World Anti-Doping Association for anti-doping analysis and testing for the NCAA, the United States Anti-Doping Agency and other sports organizations.

Screening and confirmatory tests will be done on state-of-the-art equipment and will principally involve use of GC/MS or LC/MS equipment. In addition, testing will be done for masking agents (including diuretics) as appropriate.

E. Unknowing Administration of Prohibited Substances

Players are responsible for what is in their bodies, and a positive test result will not be excused because a player was unaware that he was taking a Prohibited Substance. If you have questions or concerns about a particular dietary supplement or other product, you should contact Dr. John Lombardo at (614) 442-0106. As the NFL Advisor on Anabolic

Steroids and Related Substances, Dr. Lombardo is authorized to respond to players' questions regarding specific supplements. **Having your Club's medical or training staff approve a supplement will not excuse a positive test result.**

4. Procedures In Response to Positive Tests or Other Evaluation

(See Appendix D for a full outline of procedures normally followed after a positive test result.)

A. Notification

Once a positive result is confirmed, the Advisor will notify the player and the League Office.

B. Re-Test of Split Sample

Unless waived, any player testing positive from the first or "A" bottle will be afforded a test of the other portion of his specimen from the second or "B" bottle.

The player may not be present for the "B" test; however, except for pre-employment tests, at the player's request and expense the "B" test may be observed by a qualified toxicologist not affiliated with a commercial laboratory. The "B" test will be performed at the same laboratory that did the original test according to the procedures used for the original test and by a technician other than the one performing the original confirmation test on the "A" bottle. The player will be notified of the results in writing as soon as practicable.

C. Medical Evaluation

A medical examination such as outlined in Appendix E may be required of any player who tests positive. The Advisor will arrange for the evaluation, and the results of this evaluation will be reported to the player, the Advisor, and the team physician. If medical treatment (including counseling or psychological treatment) is deemed appropriate, it will be offered to the player. Players with a confirmed positive test result will also be placed on reasonable cause testing at a frequency to be determined by the Advisor.

The player is responsible for seeing that he complies with the arrangements of the Advisor for an evaluation as soon as practicable after notification of a positive test. This

requirement is in effect throughout the year.

5. Discipline for Violation of Law

Players or other persons within the NFL who: are convicted of or otherwise admit to a violation of law (including within the context of a diversionary program, deferred adjudication, disposition of supervision, or similar arrangement) relating to use, possession, acquisition, sale, or distribution of steroids, growth hormones, stimulants or related substances, or conspiring to do so; or are found through sufficient credible evidence (*e.g.*, authenticated medical or pharmacy records indicating receipt or use of banned substances; corroborated law enforcement reports) to have used, possessed or distributed performance-enhancing substances, are subject to discipline by the Commissioner, including suspension or, if appropriate, termination of the individual's affiliation with an NFL Club. Any suspension shall be without pay and served as set forth below. Longer suspensions may be imposed for repeat offenders. In addition, players violating this Policy by a violation of law will be appropriately placed or advanced within the three-step program. In this respect, players are reminded of federal legislation which criminalizes possession and distribution of steroids. (See Appendix H.)

6. Suspension and Related Discipline

Players with a confirmed positive test result will be subject to discipline by the Commissioner as outlined in the Policy below.

Step One:

The first time a player violates this Policy by testing positive; attempting to substitute, dilute or adulterate a specimen; manipulating a test result; or by violation of law (see Section 5), he will be suspended without pay for a minimum of four regular and/or postseason games. The suspension will begin on the date set in the League's notification to the player of his suspension, subject to any appeal (see Section 10). If fewer than four games remain in the season, including any postseason games for which the Club qualifies, the suspension will carry over to the next regular season, until a total of four regular and/or postseason games have been missed.

If the imposition of a player's suspension occurs prior to or during the preseason, the player

will be permitted to engage in all preseason activities. Upon the posting of final rosters, however, he will be suspended for four regular season games.

In addition, the player will be subject to evaluation and counseling if, in the opinion of the Advisor, such assistance is warranted.

Step Two:

The second time a player violates this Policy by testing positive; attempting to substitute, dilute or adulterate a specimen; manipulating a test result; or by violation of law (see Section 5), he will be suspended without pay for a minimum of eight regular and/or postseason games. The suspension will begin on the date set in the League's notification to the player of his suspension, subject to any appeal (see Section 10). If there are fewer than eight regular and/or postseason games remaining in the season, including any postseason games for which the Club qualifies, the suspension will continue into the next regular season until a total of eight regular and/or postseason games have been missed.

Step Three:

The third time a player violates the Policy by testing positive; attempting to substitute, dilute or adulterate a specimen; manipulating a test result; or by violation of law (see Section 5), he will be suspended without pay for a period of at least 12 months, subject to any appeal (see Section 10). Such a player may petition the Commissioner for reinstatement after 12 months. Reinstatement, and any terms and conditions thereof, shall be matters solely within the Commissioner's sound discretion.

Players who are suspended under this Policy will be placed on the Reserve/Commissioner Suspension list. During the period that he is on Reserve/Commissioner Suspension, the player will not be paid, nor may he participate in team activities, use the Club's facilities or have contact with any Club officials except to arrange off-site medical treatment. Before a player is reinstated following a suspension, he must test negative for all Prohibited Substances under this policy and must be approved as fit for play by his team physician.

In addition to the suspension imposed on him, any player suspended for a violation of the Policy will be ineligible for selection to the Pro Bowl, or to receive any other honors or awards from the League or the NFL Players Association, for the season in which the

violation is upheld (*i.e.,* following any appeals) and in which the suspension is served.

7. Procedures Regarding Testosterone

The Advisor is authorized to subject a percentage of all specimens to Carbon Isotope Ratio (CIR) testing to detect the use of exogenous testosterone.

If the introduction of testosterone or the use or manipulation of any other substance results in increasing the ratio of the total concentration of testosterone to that of epitestosterone in the urine to greater than 4:1, the test will be considered presumptively positive. Tests showing a ratio greater than 10:1 will be considered conclusively positive. Notwithstanding, when information available to the Advisor suggests but is not conclusive of testosterone use, the Advisor may require the player to submit to ongoing reasonable cause testing and shall order other medical procedures including Carbon Isotope Ratio Testing or other diagnostic tests to confirm whether exogenous testosterone has been used in violation of the Policy. In addition, the Advisor will be entitled to review any available past and/or current medical or testing records.

In addition, the use of epitestosterone to lower a player's T:E ratio is prohibited. When such use is detected or reasonably suspected by the Advisor, additional diagnostic tests may be required if the Advisor deems it necessary. If a player's epitestosterone level exceeds 300 ng/mL, it will be considered a positive test result regardless of the player's T:E ratio.

If on the basis of such follow-up tests, records, prior or subsequent test results, discussions with the player, or other studies, the Advisor subsequently concludes that the test results do in fact reflect the player's use of steroids, the player will be subject to discipline according to the terms of the Policy. Such discipline may be imposed within the season of the year in which the positive test occurred, or, if the Advisor prescribes follow-up measures that entail delay in the final determination, in a subsequent season.

8. Masking Agents and Supplements

The use of so-called "blocking" or "masking" agents is prohibited by this Policy. These include diuretics or water pills, which have been used in the past by some players to reach an assigned weight.

In addition, a positive test will not be excused because it results from the use of a dietary supplement, rather than from the direct use of steroids. Players are responsible for what is in their bodies. For more information concerning dietary supplements, see Appendix F.

9. Examination in Connection with Reinstatement

Prior to reinstatement from any suspension imposed under this Policy, a player must be examined by the team physician before he may participate in contact drills or in a game.

10. Appeal Rights

As is more fully outlined in Appendix D, any player who is notified by the League Office that he is subject to discipline for a violation of this Policy is entitled to an appeal.

The League will designate a time and place for a hearing, at which either the Commissioner or his designee will preside. The player may be accompanied by counsel and may present relevant evidence or testimony in support of his appeal. Additionally, the NFL Players Association may attend and participate notwithstanding the player's use of other representation.

After the record has been closed, the Commissioner or his designee will issue a written decision, which will constitute a full, final, and complete disposition of the appeal and which will be binding on all parties. (If appropriate, a summary ruling may be issued followed by a formal written decision as time permits.) Pending completion of this appeal, the suspension or other discipline will not take effect.

11. Burdens and Standards of Proof; Discovery

Upon appeal of a positive test result, the League shall have the initial burden to establish a prima facie violation of the Policy, and the specimen collectors, Advisor, Consulting Toxicologist and testing laboratories will be presumed to have collected and analyzed the player's specimen in accordance with the Policy. The player may, however, rebut that presumption by establishing that a departure from the Policy's stated protocols occurred during the processing of his specimen. In such case, the League shall have the burden of establishing that the departure did not materially affect the validity of the positive test or

other violation.

In presenting an appeal under this Policy, the player shall be entitled to access to only the information upon which the disciplinary action was based; in no event shall a player have access to records, reports or other information concerning the application of this Policy to any other player. Notwithstanding, this provision does not limit the Players Association's access to appropriate information concerning all violations under this Policy.

12. Reasonable Cause Testing

Reasonable cause testing procedures are more fully outlined in Section 3A of the Policy.

No Club may require any player to submit to reasonable cause testing without the agreement of both the team physician and the Advisor.

In addition, players on reasonable cause testing may be removed from their Club's active roster and placed in the category of Reserve/Non-Football Illness if, after consultation with the team physician, it is the Advisor's opinion that such a step is medically necessary.

13. Confidentiality

A. Scope

The confidentiality of players' medical conditions and test results will be protected to the maximum extent possible, recognizing that players who are disciplined for violating this Policy will come to the attention of and be reported to the public and the media.

B. Discipline for Breach of Confidentiality

Any Club or Club employee that publicly divulges, directly or indirectly, information concerning positive drug tests or other violations of this Policy (including numerical summaries or specific names of persons) or otherwise breaches the confidentiality provisions of this Policy is subject to a fine of up to $500,000 by the Commissioner.

14. Bonus Forfeiture

The computation of the amount a player must forfeit and return to his Club as a result of violating this Policy is set forth in Appendix J of the Policy.

15. Eligibility of Persons Suspended by Other Organizations

Any person who has been suspended from competition by a recognized sports testing organization based on: (a) a positive test result reported by a World Anti-Doping Agency accredited laboratory for a substance banned under this Policy; (b) an effort to substitute, manipulate or otherwise fail to cooperate fully with testing; or (c) a violation of law or admission involving the use of steroids or other performance-enhancing substances, shall be permitted to enter into an NFL Player Contract or Practice Contract. Such person, however, will be placed on reasonable cause testing and will be immediately advanced to Step Two of the Policy subject to a minimum eight-game suspension upon subsequent violation.

APPENDIX A

List of Prohibited Substances

The following substances and methods are prohibited by the National Football League:

I. ANABOLIC AGENTS

A. ANABOLIC/ANDROGENIC STEROIDS:

Generic Name	Brand Names (Examples)
Androstenediol	Androstederm
Androstenedione	Androstan, Androtex
1-Androstenedione	---
Bolasterone	Myagen
Boldenone	Equipoise, Parenabol
Calusterone	---
Clostebol	Turinabol, Steranabol
Danazol	Cyclomen, Danatrol
Dehydrochloromethyltestosterone	Oral-Turinabol
Dehydroepiandrosterone	DHEA
Desoxymethyltestosterone	DMT, Madol
Dihydrotestosterone	DHT, Stanolone
Dromostanolone	Drolban
Ethylestrenol	Maxibolin, Orabolin
Fluoxymesterone	Halotestin
Formebolone	Esiclene, Hubernol
Furazabol	Miotolon
Gestrinone	Tridomose
17-Hydroxypregnenedione	---
17-Hydroxyprogesterone	---
Hydroxytestosterone	---
Mestanolone	---
Mesterolone	Proviron
Methandienone	Danabol, Dianabol
Methandriol	Androdiol
Methandrostenolone	Dianabol
Methenolone	Primobolan
Methyltestosterone	Metandren
Mibolerone	Testorex
19-Norandrostenediol	19-Diol

I. Anabolic/Androgenic Steroids (cont'd)

Generic Name	**Brand Names (Examples)**
19-Norandrostenedione	19 Nora Force
Norbolethone	Genabol
Norclostebol	---
Norethandrolone	Nilevar
Normethandrolone	---
19-Nortestosterone (Nandrolone)	Deca-Durabolin
Oxandrolone	Anavar, Lonovar
6-Oxoandrosterone	6-Oxo
Oxymesterone	Oranabol
Oxymetholone	Anadrol
Progesterone	---
Stanozolol	Stromba, Winstrol
Stenbolone	---
Testosterone	Andronate
1-Testosterone	---
Tetrahydrogestrinone	THG
Trenbolone	Finaject

and related substances

B. HORMONES:

Generic Name	**Brand Names (Examples)**
Human Growth Hormone (hGH)	Saizen, Humatrope, Nutropin AQ
Animal Growth Hormones	---
Human Chorionic Gonadotropin (hCG)	Novarel, Menotropins
Insulin Growth Factor (IGF-1)	---
Erythropoietin (EPO)	---

and related substances

C. BETA-2-AGONISTS (Clenbuterol, etc.)

D. ANTI-ESTROGENIC AGENTS (Clomiphene [Clomid], Cyclofenil, Tamoxifen)

II. MASKING AGENTS

A. DIURETICS

Generic Name	Brand Names (Examples)
Acetazolamide	Amilco
Amiloride	Midamor
Bendroflumethiazide	Aprinox
Benzthiazide	Aquatag
Bumetanide	Burine
Chlorothiazide	Diuril
Cyclothiazide	Anhydron
Ethacrynic Acid	Edecrin
Flumethiazide	---
Furosemide	Lasix
Hydrochlorothiazide	Aprozide
Hydroflumethiazide	Leodrine
Methyclothiazide	Aquatensen
Metolazone	Zaroxolyn
Polythiazide	Renese
Probenecid	Benemid
Quinethazone	Hydromox
Spironolactone	Aldactone
Triamterene	Jatropur, Dytac
Trichlormethiazide	Anatran

and related substances

B. EPITESTOSTERONE

C. PROBENECID

III. CERTAIN STIMULANTS

Generic Name	Brand Names (Examples)
Amphetamine	Greenies, Speed
Ephedrine	Ma Huang, Chi Powder
Fenfluramine	Phen-Fen, Redux
Methamphetamine	---
Methylephedrine	---
Modafinil	---
Norfenfluramine	---
Pseudoephedrine *	Sudafed, Actifed
Phentermine	Fastin, Adipex, Ionamin
Synephrine	Bitter Orange, Citrus Aurantium

* Except as properly prescribed by Club medical personnel.

IV. DOPING METHODS

Introduction of a Prohibited Substance into the body by any means, including but not limited to the introduction of a Prohibited Substance, or the ingestion or injection of a supplement or other product containing a Prohibited Substance.

Pharmacological, chemical or physical manipulation by, for example, catheterization, urine substitution, tampering, or inhibition of renal excretion by, for example, probenecid and related compounds.

APPENDIX B

Personnel

The NFL Advisor on Anabolic Steroids and Related Substances is Dr. John Lombardo, Medical Director of the Max Sports Medicine Institute and Clinical Professor in the Department of Family Medicine at the Ohio State Medical School. He also was previously a member of the faculty at the Sports Medicine Center of the Cleveland Clinic and has served as team physician to the Cleveland Cavaliers of the NBA and as an adviser on steroid issues to both the NCAA and the Olympic Committee.

The Consulting Toxicologist on Anabolic Steroids and Related Substances is Dr. Bryan Finkle, a board-certified forensic toxicologist and Research Professor of Pharmacology-Toxicology in the College of Pharmacy and Department of Pathology in the College of Medicine at the University of Utah Health Sciences Center. He also serves as a consultant to the International Olympic Committee Medical Commission, World Anti-Doping Agency and United States Anti-Doping Agency.

Collection Procedures

Upon reporting to the collection site, the player to be tested shall be required to produce a government-issued photo ID. Once his identity is confirmed, the player will be given the opportunity to select a sealed urine specimen cup. The player will furnish a urine specimen under observation by an authorized specimen collector. Thereafter, the player will be given the opportunity to select a sealed collection kit which will be used to store and ship his urine specimen. In the player's presence, the specimen will be split between an "A" bottle and a "B" bottle and resealed with security seals. The specimen collector will note any irregularities concerning the specimen, following which the player will be given the opportunity to sign the chain-of-custody form.

Once the bottles have been sealed and the chain-of-custody form has been completed, the bottles will be inserted into containers and placed back into the kit. The kit will then be sealed and sent by Federal Express or similar carrier to the testing laboratories.

All bottles will be identified by a control identification number. The number on the bottles will be the same as the number on the chain-of-custody form. The testing laboratories themselves will be unable to associate any specimen with an individual player.

APPENDIX D

Procedures Following Positive or Presumptively Positive Tests

The following will outline the procedures to be used following the testing laboratory's notification to the Advisor of a positive "A" test:

A. Standard Tests

1. The Advisor will contact the laboratory for verification.
2. After verifying the result with the laboratory, the Advisor will match the control identification number with the player's name, and will then notify the player in writing of the positive result and request that the player call him to discuss the result.
3. If the player wishes to have the "B" sample test observed by a qualified toxicologist, he will notify the Advisor in writing within five (5) business days of receiving written notification of the positive test result. If observation is requested, the Advisor and toxicologist will schedule the test for the first mutually available date. Otherwise, in the absence of a reasonable basis for delay, the "B" sample test will be initiated within seven (7) business days following player's receipt of written notification of the positive test or as soon as possible following the Advisor's receipt of written notification by the player that he does not wish the test to be observed, whichever is sooner.
4. The laboratory will report the "B" sample test result to the Advisor, who may review the case with the Consulting Toxicologist and the laboratory director as appropriate.
5. The Advisor will report his findings to the player and, if confirmed positive or if reasonable cause testing is indicated, to the team physician and League Office.
6. If the player is subject to disciplinary action, the League Office will notify him in writing.
7. If the player decides to appeal, he must so indicate in writing to the Commissioner within five (5) business days after receiving a notice of discipline from the League Office. He should state in his notice of appeal whether or not he desires a hearing.
8. If a hearing is requested, the League will schedule it to take place within twenty (20) calendar days of the request absent mutual agreement or extenuating circumstances. The hearing may be conducted by conference call upon agreement of the parties.
9. Prior to the hearing, the League will provide the player and NFL Players Association with a laboratory documentation package prepared in accordance with Appendix I. In the absence of clear evidence to the contrary, such package will be deemed full and complete for the purpose of evaluating the integrity of the chain-of-custody and test results. Once the player has had sufficient opportunity to review the documentation package, he must provide to the League a written statement setting forth the specific grounds of his appeal. Additionally, no

later than two (2) business days prior to the hearing the parties will exchange copies of any documents or other evidence on which they intend to rely and a list of witnesses expected to provide testimony. Following the exchange, the parties may provide further supplementation as appropriate.

10. Once the record is closed, the Commissioner or his designee will evaluate the evidence and render a written decision with respect to disciplinary action within five (5) calendar days. (If appropriate, a summary ruling may be rendered, followed by a formal decision as time permits.)

B. Pre-Employment Tests

When notified of a positive test result obtained prior to employment (including Combine and Free Agent tests), the procedure set forth in Part A above shall apply, except that:

1. The "B" sample test will be conducted on the first available date without the opportunity for observation by an outside toxicologist.
2. Upon confirmation of the positive test result, the Advisor shall promptly notify the League Office and: all Clubs in the case of a Combine test, and the requesting Club(s) in the case of a Free Agent test.

The League will endeavor to conduct and conclude these procedures expeditiously, with appropriate regard to the possible need for follow-up tests or other measures required in the Advisor's judgment, or other extenuating circumstances.

APPENDIX E

Examples of Medical Evaluations Following a Positive Test

A. Initial Positive Test

History and Physical

Emphasize:
- Cardiovascular
- Abdominal
- Genitourinary (testicle, prostate, impotence, sterility)
- Psychological (aggressiveness, paranoia, dependency, mental status)
- Immune system (masses, infections, lymphadenopathy)

Testing

- CBC with Differential
- General chemistry panel
  - Electrolytes, BUN/Creatinine, Glucose, Liver enzymes
- Lipid Assay
  - Triglycerides/cholesterol, HDL-C, LDL-C
- Urinalysis
- Cardiovascular
  - EKG
  - Chest X-ray
  - Stress test
  - Echocardiogram
- Semen analysis
- Endocrine Profile
  - TSH, LH, FSH, T4, TBG, Testosterone, SHBG (TBG)
- Liver scan (either MRI or CT or Ultrasound or liver/spleen Scan)

B. Repeat Positive Test Evaluation

History and Physical - as above

Testing - Lab as above

CV } Liver scan } — As indicated by time since last test and by history and physical

POLICY ON ANABOLIC STEROIDS AND RELATED SUBSTANCES
-Use of Supplements-

Over the past several years, we have made a special effort to educate and warn players about the risks involved in the use of "nutritional supplements." Despite these efforts, several players have been suspended even though their positive test result may have been due to the use of a supplement. Subject to your right of appeal, **if you test positive or otherwise violate the Policy, you *will* be suspended.** You and you alone are responsible for what goes into your body. Claiming that you used only legally available nutritional supplements will not help you in an appeal.

As the Policy clearly warns, supplements are not regulated or monitored by the government. This means that, even if they are bought over-the-counter from a known establishment, there is currently no way to be sure that they:

(a) contain the ingredients listed on the packaging;
(b) have not been tainted with prohibited substances; or
(c) have the properties or effects claimed by the manufacturer or salesperson.

Therefore, if you take these products, you do so AT YOUR OWN RISK! For your own health and success in the League, we strongly encourage you to avoid the use of supplements altogether, or at the very least to be extremely careful about what you choose to take.

Take care and good luck this season.

Sincerely,

HAROLD HENDERSON

Executive Vice President
National Football League

GENE UPSHAW

Executive Director
NFL Players Association

APPENDIX G

To: All NFL Players
From: Dr. John Lombardo
Subject: Supplements
Date: November 10, 1998

Gene Upshaw and representatives from the NFLPA along with Harold Henderson and representatives from the NFL Management Council recently met with me and a number of my colleagues to discuss dietary supplements and their interrelationship with the NFL Policy and Procedures for Anabolic Steroids and Related Substances.

Upon the conclusion of the meeting all participants felt that I should advise you of both health and policy violation risks you may be faced with by adding over-the-counter supplements to your diet.

In 1994, the U.S government passed a law entitled "The Dietary Supplement Health and Education Act". As a result of this law, the supplement manufacturers and distributors do not have to prove the effectiveness or the safety of their products. Also, the ingredients of the supplements are not checked by any independent agency, such as the Food and Drug Administration (FDA), to certify the contents of the supplements. Therefore, the effectiveness, side effects, risks and purity of many products you can buy at the health food store are unknown.

This law also permits over-the-counter sale of products that violate the NFL Steroid policy. For example, androstenedione, a steroidal hormone that serves as a direct precursor for the synthesis of testosterone, is widely advertised. However, since this substance is found in some plants and animals, manufacturers are allowed to market it as a dietary supplement. This product, like many other supplements that contain substances that violate the policy, can be purchased at your local health food store and, when ingested, is no different than taking illegal anabolic steroids or related substances.

If you take a supplement that contains a substance that violates the policy it will subject you to discipline. More importantly, you run the risk of harmful health effects associated with steroid use.

I will continue to provide you with information on the subject throughout the year. In the meantime, if you have any questions about supplements or the steroid policy, please contact me.

JOHN A. LOMBARDO, M.D.
NFL Advisor for Anabolic/Androgenic
Steroids and Related Substances

APPENDIX H

U.S. Department of Justice
Drug Enforcement Administration
Washington, D.C. 20537
September 4, 2001

Mr. Paul Tagliabue
Commissioner
National Football League
410 Park Avenue
New York, New York 10022

Dear Commissioner Tagliabue:

Thank you for your concern regarding the policies of the Drug Enforcement Administration (DEA) in enforcing the Anabolic Steroids Control Act of 1990 and the National Football League's (NFL) policies to eliminate the use of anabolic steroids in the NFL.

Your program of random and reasonable cause testing for steroids reinforces the provisions of the Anabolic Steroids Control Act of 1990. Under this law, DEA has the responsibility to regulate all aspects of the legitimate steroid industry, including doctors and pharmacists.

To those who use anabolic steroids, including professional athletes, I should emphasize that under the Act, possession of even personal use quantities not validly prescribed by a doctor is a federal crime. The maximum penalty for simple possession (possession not for sale), is one year in a federal prison and a minimum $1,000 fine.

DEA will also investigate and prosecute violations involving the unlawful manufacture, distribution, and importation of anabolic steroids. Doctors who prescribe anabolic steroids for other than legitimate purposes will be prosecuted. Pharmacists who dispense anabolic steroids without a doctor's prescription or with one that they know is bogus, will also be prosecuted.

While DEA's primary focus is law enforcement, we also recognize the importance of public education on matters such as these. I would thus appreciate it if you would make this letter directly available to each NFL team, its players, physicians, trainers, and other personnel.

Sincerely,
[*Signature on file*]
Asa Hutchinson
Administrator

APPENDIX I

Standard Form of Documentation Package

Tab	Item(s)
1.	Cover Sheet
2.	Table of Contents
3.	General Overview of Laboratory Procedures
4.	Custody and Control Forms
	a. External Chain of Custody Form
	b. Specimen Chain of Custody
5.	Initial Test Information (A-Bottle)
6.	Confirmation Test Information
	a. Confirmation Test Description
	b. Chain of Custody Pull List
	c. Confirmation Aliquot Chain of Custody Log
	d. Specimen ID Verification Report
	e. Analytical Data
7.	Certification Information
	a. Pending Positive Report (Certifying Scientist Worksheet)
	b. Laboratory Report
8.	Re-Test Information (B-Bottle)
	a. Chain of Custody Pull List
	b. Confirmation Aliquot Chain of Custody Log
	c. Specimen ID Verification Report
	d. Analytical Data
9.	Re-Test Certification Information
	a. Pending Positive Report (Certifying Scientist Worksheet)
	b. Laboratory Report

APPENDIX J

Bonus Forfeiture

A Player who is suspended under this Policy shall forfeit and return to his Club (or forgo entitlement to unpaid portions of) the proportionate amount of his signing bonus corresponding to the period of the suspension; provided that, if (a) the suspension is for a period of one year or more, (b) the Player's Contract is tolled during such suspension, and (c) the Player subsequently performs under the Contract during the expended period that results from the tolling, then the Player shall earn back the proportionate amount of forfeited or forgone signing bonus for the extended period in which he performs. For purposes of this Section, "proportionate amount" means $1/17^{th}$ of the signing bonus allocation for each regular season week or regular season game missed per League Year covered by the suspension, or $1/17^{th}$ of the forfeited or forgone signing bonus allocation for each regular season week or regular season game subsequently played per extended year of the Player's Contract, in the case of a Player earning back previously forfeited or forgone signing bonus.

By way of example, without limitation on any other example, if a Player with a four-year Player Contract for the 2006-2009 League Years that contains a signing bonus of $4 million is suspended for the 2007 and 2008 League Years for violation of the Policy, then the Player would forfeit and return to his Club $2 million in signing bonus allocation ($1 million for the 2007 League Year and $1 million for the 2008 League Year). If, after performing under the Player Contract in the 2009 League Year, the Player then performed one of his previously tolled years in the 2010 League Year, he would earn back $1 million. If the Player then performed for eight games of the second of his previously tolled years in the 2011 League Year and then retired, he would earn back an additional $470,588 (8/17 x $1 million).

TAB 4

NFL Subcommittee on Mild Traumatic Brain Injury Membership and Affiliations
As of October 30, 2007

David Viano, Dr. med., Ph.D., Co-Chair	Outside Consultant; ProBiomecanics LLC, Biomedical Engineer; Adjunct Professor of Engineering, Wayne State University
Ira Casson, M.D., Co-Chair	Outside Consultant, Neurologist: Assistant Professor of Neurology, Albert Einstein School of Medicine & Long Island Jewish Medical Center
Ronnie Barnes, ATC	Head Athletic Trainer, New York Giants
Rick Burkholder, ATC	Head Athletic Trainer, Philadelphia Eagles
Henry Feuer, M.D.	Team Physician/Neurosurgeon, Indianapolis Colts; Neurosurgeon, Indiana University Medical Center, Indianapolis Neurosurgical Group
Mark Lovell, Ph.D.	Neuropsychologist, Director University of Pittsburgh Sports Concussion Program; Associate Professor of Neurological Surgery University of Pittsburgh; UPMC Center for Sports Medicine, Pittsburgh
Joseph Maroon, M.D.	Neurosurgeon, Vice Chairman Neurosurgery, University of Pittsburgh Medical Center; Clinical Professor of Neurosurgery, University of Pittsburgh
Joel Morgenlander, M.D.	Neurologist, Professor of Neurology, Duke University Medical Center
Thomas Naidich, M.D.	Neuroradiologist, Professor and Chief Neuroradiology, Mount Sinai School of Medicine
Elliot Pellman, M.D.	NFL Medical Liaison, Medical Director, New York Jets; Member NFL Injury and Safety Panel, NFL Subcommittee on Cardiovascular Health; NFL Foot and Ankle Subcommittee; Medical Director, ProHealth Care Associates; Associate Professor of Medicine and Orthopaedics Mount Sinai School of Medicine

NFL Subcommittee on Mild Traumatic Brain Injury Membership and Affiliations
As of October 30, 2007 (Continued)

John Powell, Ph.D., ATC	NFL Consultant — Injury Studies, Med Sports Systems; Epidemiologist: Associate Professor of Departments of Kinesiology and Physical Medicine and Rehabilitation, Michigan State University
Doug Robertson, M.D.	Team Physician/Family Practice, Indianapolis Colts
Andrew Tucker, M.D.	Team Physician, Baltimore Ravens; Co-Chairman, NFL Subcommittee on Cardiovascular Health; Member NFL Injury and Safety Panel; Chief Sports Medicine Union Memorial Hospital
Joe Waeckerle, M.D.	Emergency Medicine Physician, Kansas City Chiefs, Editor Emeritus Annals of Emergency Medicine, Clinical Professor of Medicine, University of Missouri School of Medicine

TAB 10

Reprinted from NEUROSURGERY
Vol. 59 No. 5 November 2006

10.1227/01.NEU.0000249027.33996.D5

Chronic Traumatic Encephalopathy in a National Football League Player

To the Editor:

We appreciate the response from Omalu et al. (6) to our original Letter to the Editor regarding the article "Chronic Traumatic Encephalopathy in a National Football League Player" (1). After much consideration of their arguments, as well as those of the reviewers, we are still troubled by key problems and new information that underscore the need for Omalu et al. to fully disclose all information and to present unbiased data before presenting "scientific" conclusions.

In their reply to our letter (6), Omalu et al. refer to another case of an "NFL player with similar neuropathological changes." Presumably, they are referring to the case of Terry Long.

Unfortunately, they failed to mention important information about this case. In January 2006, Drs. Wecht and Omalu announced to the press postmortem findings of a second football player, Mr. Terry Long, who they concluded died from repeated football-related head injuries. But, a revised death certificate, which was never released publicly, lists Mr. Long's cause of death as a result of drinking antifreeze. Ethylene glycol ingestion damages the brain and certainly clouds the pathologist's original conclusions. The conclusion that the ingestion of antifreeze was related to depression from repeated head injuries is, at a minimum, speculative. We think that the reporting of the Terry Long autopsy results and other examples cited below substantiate our concerns that Omalu et al. (5, 6) omit important data that readers need to make informed decisions, and that their original article reaches conclusions that cannot be relied upon.

Omalu et al.'s (6) reply to our letter continues the pattern of factual inaccuracies and important omissions that characterized their original article (5). Omalu et al. state that the absence of a cavum septum pellucidum (with or without fenestrations) does not preclude the diagnosis of chronic traumatic encephalopathy (CTE) because "Corsellis and others have never asserted that this finding is a sine qua non of dementia pugilistica" (6). Omalu fails to include the fact that all of Corsellis' cases had an abnormality of the septum pellucidum (2). Two of the original 15 patients had died of recent cerebral hemorrhage that might have affected the septal region, so Corsellis did not include their cava in the final results. All of the remaining 13 patients had abnormalities of the septum pellucidum (12 cava with or without fenestrations, one fenestrated septum without a cavum) (2). This stands in stark contrast to Omalu et al.'s case, which had no abnormalities of the septum (5).

Certain reviewers indicate that the nature of head injuries is very different in football than in boxing and suggest that we were essentially comparing apples and oranges (3, 4). We agree that there are significant differences between head trauma sustained in the two sports. The reviewers suggest that, because of these differences, chronic encephalopathy in football players would be expected to be different from that seen in boxers. This assumes that there is a chronic encephalopathy of football players. It seems that the end result of the difference we measured in head impacts sustained in the two sports (9) could be that there is no chronic encephalopathy in football players.

In another attempt to demonstrate that their findings are similar to Corsellis' results, Omalu et al. then states that "our case showed degeneration of the substantia nigra and, most importantly, widespread neocortical neurofibrillary tangle formation" (6). This statement is also inaccurate. With regard to "degeneration of the substantia nigra," Omalu et al. failed to mention that every one of Corsellis' cases with loss of pigment and neuronal dropout in the substantia nigra also had neurofibrillary tangles (NFTs) in some or many of the remaining neurons in the substantia nigra (2). This stands in clear contrast to Omalu et al.'s case, which had no NFTs in the substantia nigra (5).

With regards to widespread NFT formation, Omalu et al.'s choice of words in their original report is not the same as in their reply to our correspondence. In the abstract and body of their original article, Omalu et al. referred to their finding of "sparse neurofibrillary tangles" (5, p 130). The word "widespread" does not, in fact, appear in their original article. In their reply to our letter, Omalu et al. have replaced the word "sparse" with the word "widespread," seemingly adopted from our letter. The problem with this substitution is that "sparse" and "widespread" do not mean the same thing.

The adjective "widespread" is used to describe the occurrence of a phenomena in an area in two ways: 1) how it is distributed over the area, in which case it means that it occurs in many regions of the overall space, and 2) the density with which it is seen in the areas, in which case it is synonymous with the words "prevalent," "common," and "pervasive" (8). Remaining consistent with Corsellis' original description, we were clearly using "widespread" in its broadest sense to describe a pattern of numerous NFTs seen in many areas of the brain. It remains unclear to us how Omalu et al. are using the word in their reply to our letter. Are they replacing "sparse" with "widespread"? If so, they need to clearly state that they are revising their original report of meager, few NFTS (synonyms for "sparse") and are now stating that NFTs occurred frequently and commonly in their material (8). If that were the case, it would lead us to question the accuracy of the original neuropathological descriptions. Or, are they using "widespread" and "sparse" together to describe their findings? If they are using "widespread" only to describe the distribution of NFTs in many areas of the brain and "sparse" to describe the meager numbers of NFTs seen in these areas, then they are really stating that the occurrence of NFTs was few and far between, infrequent and scattered, in their material. This description is obviously not at all consistent with Corsellis' report of "an abundance" of NFTs and a "vast" number of neurons with NFTs (2). Omalu et al.'s adoption of the word "widespread" in their reply thus serves to further confuse, rather than clarify, the issues surrounding our points of concern regarding their original report.

Omalu et al. also write that "this pattern of neurofibrillary tangle formation would cause most neuropathologists to question whether the patient had a history of boxing and lead them to examine the clinical history for episodes of repetitive head trauma" (6). There are no citations or references given for this statement, and we think our findings further compromise the validity of the statement.

Omalu et al. also write that their case had "deposits of B/A4, which were also seen in 19 of 20 cases of dementia pugilistica," citing Roberts (7) as their reference. Once again, they failed to mention a critical point about these 20 cases: 19 of the 20 had a cavum septum and the 20th was a man with very limited exposure to boxing (fewer than 10 bouts during a 2-year boxing "career") that the authors described as having "probable" Alzheimer's disease (7). This is another example of Omalu et al.'s omission of information that we think is important if the reader is to reach an informed conclusion.

The reviewers who criticized us for bringing up the topic of CTE in boxers need to be reminded that it was Omalu et al. who tried to make the connection between their case and CTE in boxers. The entire first paragraph of the Discussion in their original article was devoted to reports of CTE in boxers (5). In this Discussion, Omalu et al. specifically mentioned the Corsellis study on "the neurohistological substrate in the brains of 15 retired professional and amateur boxers" (5). Omalu et al. referenced a number of other articles on CTE in boxers in their original report.

If Omalu et al. had accurately presented the findings of Corsellis and others regarding the neuropathology of CTE in boxers, there would have been no need for us to object to their statements. However, if they had done so, it would have been obvious to the readers and reviewers that Omalu et al.'s case was not at all consistent with the CTE of boxers. Omalu et al. clearly thought that they needed to connect their case to CTE in boxers, or they would not have discussed it so prominently in their report (5). Any criticism for trying to link the findings in one football player to the neuropathology of CTE in boxers should have been directed at Omalu et al.'s original report, where we think it belonged.

We are also concerned by Omalu et al.'s attempt to use an arbitrator's judicial decision to bolster their position (6). When did the courts become the arbiters of scientific truth? Legal opinions are irrelevant to scientific discussions in the forum of a medical journal such as *Neurosurgery*. In fairness to all of our colleagues, we think full disclosure of all facts by all parties is necessary for a full and open "scholarly discussion and understanding" regarding this important matter. We remain convinced that the authors should retract their paper or sufficiently revise it and its title.

Ira R. Casson
Elliot J. Pellman
David C. Viano
New York, New York

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