

The History of Neurosurgical Treatment of Sports Concussion

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 Congress of Neurological Surgeons.

Concussion has a long and interesting history spanning at least the 5 millennia of written medical record and closely mirrors the development of surgery and neurosurgery. Not surprisingly, much of the past and present experimental head injury and concussion work has been performed within neurosurgically driven laboratories or by several surgically oriented neurologists. This historical review chronicles the key aspects of neurosurgical involvement in sports concussion as related to the diagnosis, treatment, mitigation, and prevention of injury using the example of American football. In addition, we briefly trace the developments that led to our current understanding of the biomechanical and neurophysiological basis of concussion.

KEY WORDS: Biomechanics, Concussion, Football, Head injury, History, Neurosurgery, Sports

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“If you had a son would you encourage or permit him to play football? The carefully considered answer has been: If the boy was of high school age, or older, and with the proper physique, conditioning, and balanced mental capacity; if the boy was supplied with the proper protective equipment; if he were to be taught the proper coaching techniques with the avoidance of stick-blocking or spearing; if his coach was a perceptive thoughtful man, who regarded each lad as his own son; if the boy had the desire—I would encourage him to play football.”

—Richard C. Schneider, 1973

The modern field of sports medicine and the importance of the intercollegiate athletic trainer in the prevention and management of injuries had their roots after World War I.¹ Samuel E. Bilik (1891-1972), a physician pioneer in sports medicine, published in 1917 the first edition of his book later titled *The Trainer's Bible* (1948), a major text on athletic training and care

of athletic injuries.² Between 1938 and 1962, the surgeon Augustus Thorndike (1896-1989) published 5 editions of *Athletic Injuries* (1948), a book primarily for physicians. A graduate of Harvard, for >3 decades he was chief of surgery, worked with Harvard athletes beginning in the 1920s, designed protective equipment for football and hockey, and was instrumental in athletic health policy.² Neurological surgeons have treated head and spinal sports injuries since the specialty was formed in the first decades of the 20th century. However, formal neurosurgical involvement with the mitigation of sports injury dates to 1931 at Yale (see below), and in 1941, Walter Dandy (1886-1946) patented the first protective baseball cap insert liner.^{3,4} Neurosurgeons in the past 50 years have become progressively more involved in the growing sports concussion problem, especially in American football and ice hockey, with the former having the greatest number of participants.⁵⁻⁸ The American College of Sports Medicine, founded in 1954, is a physician-led, multidisciplinary body dedicated to maintaining an individual's functional capacities, including the prevention and treatment of diseases and injuries related to exercise and sports.⁹ Thus, the American College of Sports Medicine is represented by a number of neurological surgeons and maintains much interest in concussion and advances in the field.

Since antiquity, the distinction of concussion as a clinical entity required surgical differentiation from other more severe head injuries also

ABBREVIATIONS: **AFCA**, American Football Coaches Association; **CTE**, chronic traumatic encephalopathy; **EEG**, electroencephalography; **LOC**, loss of consciousness; **RTP**, return to play

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FIGURE 1. College football, 1905. Reproduced from *Torg*³¹ with permission from the publisher. Copyright © 1982 Lea & Febiger.

characterized by a sudden primary alteration in the level of consciousness of immediate onset after injury. Throughout medical history, related important diagnostic and treatment concerns included differential diagnosis of the presence or absence of a skull fracture with or without depression, cerebral contusion, or the onset of delayed deterioration of consciousness secondary to cerebral compression from delayed accumulation of hemorrhage or infection (pus). Several of the above might warrant a surgical procedure or follow a prolonged concussion and be missed. Furthermore, the outcome after any head injury was complicated by a lack of understanding of the infective cause of inflammation and pus formation until acceptance of Louis Pasteur's (1822-1895) findings, followed by Joseph Lister's (1827-1912) antiseptic methods in the late 19th century.¹⁰⁻¹³

The belief that concussion and contusion can be accounted for on the basis of somewhat similar mechanisms, in addition to overlapping clinical syndromes, persisted into the 20th century.¹⁴ In adult humans, the impact force to cause concussion or cerebral contusion approximates that which causes a linear skull fracture.¹⁵ However, cerebral contusions alone, unless very large at onset, are not generally considered responsible for loss of consciousness (LOC).¹⁶

In the first quarter of the 20th century, many accepted the theory that explained cerebral concussion and possibly cerebral contusions by deformation of or a temporary change in the form or shape of the skull, causing an anemia of the brain.

AN ANATOMIC SUBSTRATE OF CONCUSSION

Progressive thoughts on consciousness would come in the later 19th and early 20th century writings of the esteemed British neurologist John Hughlings Jackson (1835-1911).¹⁷⁻¹⁹ Jackson adopted the ideas of social philosopher Herbert Spencer (1820-1903) on "evolution and dissolution," which Jackson applied to brain function.¹⁸⁻²⁰ Jackson was aware that a concussion, if the injurious physical forces were severe enough, could be fatal as

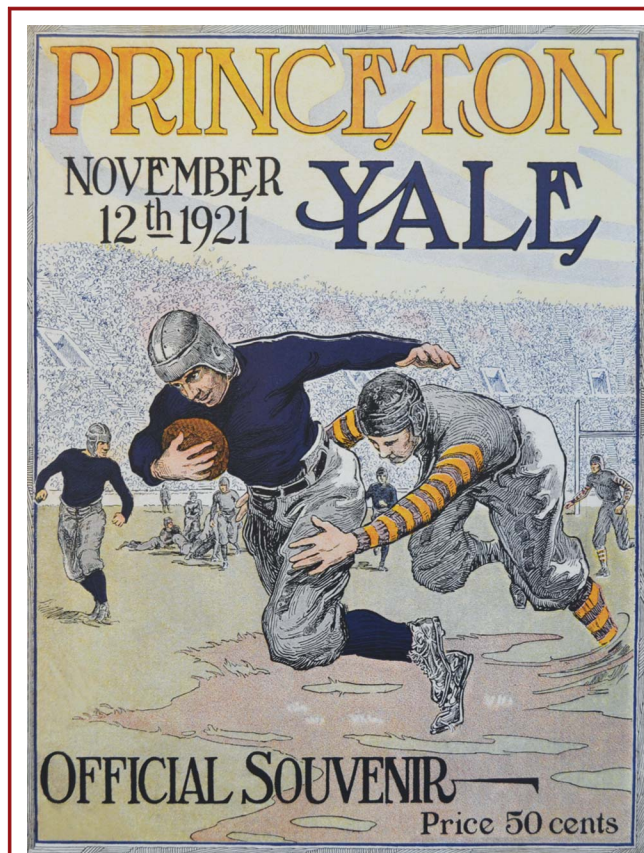
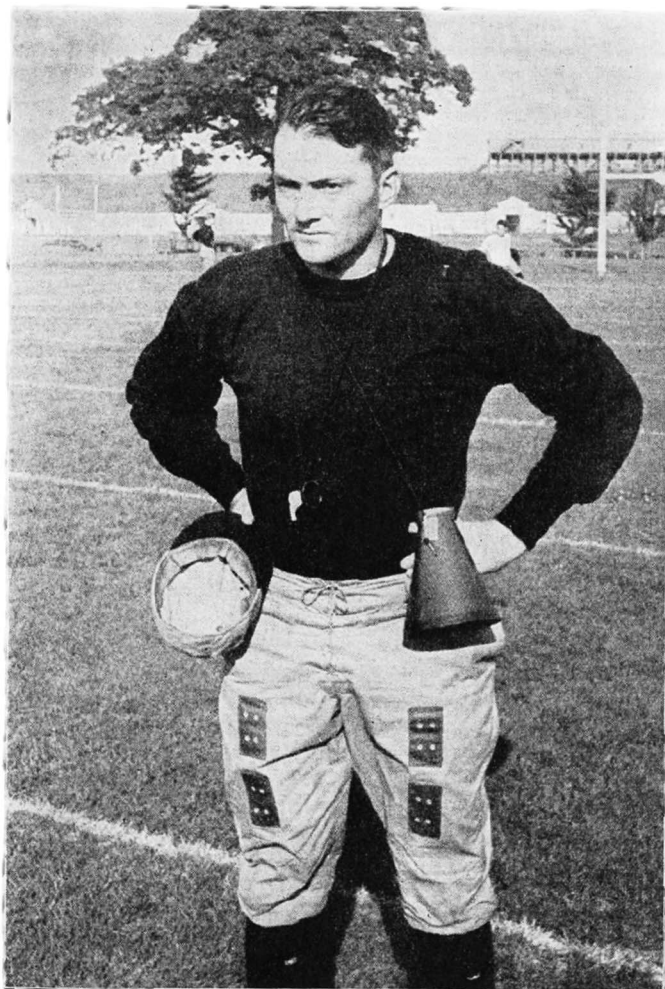


FIGURE 2. Yale/Princeton football program (1921) depicting the leather helmets then in use.

a result of cardiovascular and respiratory failure, and at autopsy, the brain in such cases may have a normal or near-normal gross appearance. Jackson expanded on a statement that the surgeon Sir Charles Bell made a half-century earlier in reference to the "degrees of drunkenness."¹⁹ Jackson deduced:

The first stage of concussion would be one of sudden dissolution. In these cases the whole nervous system is reduced, but the different centres are not equally affected. An injurious agent such as alcohol, taken into the system flows to all parts of it; but the highest centers, being the least organized, give out first and most; the middle centers, being more organized, resist longer; and the lowest centers, being most organized, resist longest. Did not the lowest centers for respiration and circulation resist much more than the highest do, (then) death by alcohol would be a very common thing.^{18,19}

The prefrontal cerebral cortex constituted Jackson's "highest centres" and continues to represent human's most integrated, complex, and evolutionary advanced centers. These highest centers of executive nature are associated with the most numerous interconnections and are not as simply "organized," or hard-wired, as Jackson's cardiorespiratory medullary, the "lower centres." In regard to Jackson's hypothetical intermediate or "middle centres,"



MAL" STEVENS IN A CHARACTERISTIC POSE

THE CONTROL OF FOOTBALL INJURIES

BY

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1933

FIGURE 3. Title page of *The Control of Football Injuries* by Stevens and Phelps.³²

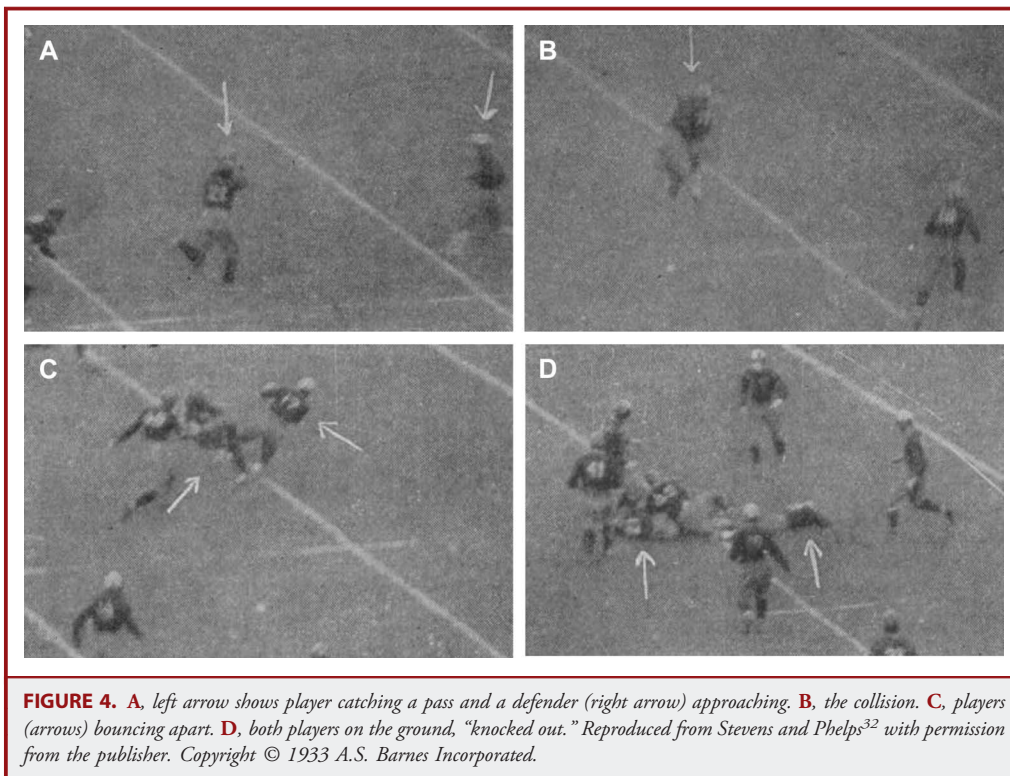
the dissolution of which contributes to concussion, today we believe this would likely be the midbrain-diencephalic region, including the upward projections of the reticular activating system and associated thalamocortical projections that maintain arousal and alertness.²¹

It is our opinion that Jackson's alcohol analogy of dissolution in concussion, combined with his conception of a graded complexity of brain centers, is compatible with the clinical findings in concussion. Any period of LOC (paralytic coma, unresponsiveness) would likely implicate a significant transverse mechanical shearing force (more easily produced by angular than linear accelerating forces), producing a physiological or variable anatomic disconnection within both cerebral hemispheres or at the midbrain-diencephalic ascending arousal system or lower brainstem/cervicomedullary junction. This may occur as a result of several mechanisms: secondary to motion-induced movements of the cerebral hemisphere(s) in relation to tethering or fixation at the upper midbrain-diencephalon or perhaps

more direct impact forces resulting in transient cerebellar tonsillar herniation/lower brainstem impaction, complicated by rigid tethering of the upper cervical cord by the dentate ligaments.^{15,22-26} Milder forms of concussion as commonly found in helmeted sporting injuries such as brief stunning and more minor impairments in cerebral functioning would more likely involve less severe, partial physiological or anatomic unilateral or bilateral cortical/hemispheric involvement.

A 20TH CENTURY PROBLEM: CATASTROPHIC BRAIN AND CERVICAL SPINAL CORD INJURIES IN AMERICAN FOOTBALL

The game of American football increased in popularity after the Civil War (1861-1865) but was very dangerous because no



protective equipment was used and the sport was inadequately officiated and regulated. According to Schneider,²⁶ “In the early days of football, 1890, the best protection afforded a football player was not a helmet but a bushy head of hair which not only cushioned the blow to the cranium but also afforded a fine handle

for tackling” (Figure 1). The American college football season of 1905 was characterized by 18 deaths and 159 serious injuries, which came to the attention of President Theodore Roosevelt. Roosevelt had been a strong advocate of the game, although in 1893 he stated, “The brutality must be done away with and the danger minimized.”²⁷

In late 1905, Roosevelt invited the college coaches, including those from Harvard, Princeton, and Yale, to the White House to discuss the brutality of the game. He told them, “Football is on trial. . . [and] . . . because I believe in the game, I want to do all I can to save it.”²⁷ Roosevelt asked that they adjust the rules to minimize injuries.²⁸⁻³⁰ Several meetings of college presidents followed and resulted in 1906 in the formation of the Intercollegiate Athletic Association of the United States (renamed the National Collegiate Athletic Association in 1910). Important rule changes that emphasized safety were instituted. These included rules against personal fouls, unnecessary roughness, tackling below the knees, and unsportsmanlike conduct; a rule for the passer to be 5 yards behind the scrimmage line and more protected (leading to the future passing aspect of the game); and the rule that the tackling player must have 1 foot on the ground (eliminating the flying tackle).²⁹

The origin of the leather football helmet was in preparation for the Army-Navy game of 1893, when a Navy player was advised by a physician that “he would be facing death if he took another hit to the head” and a local shoemaker fabricated a leather helmet to



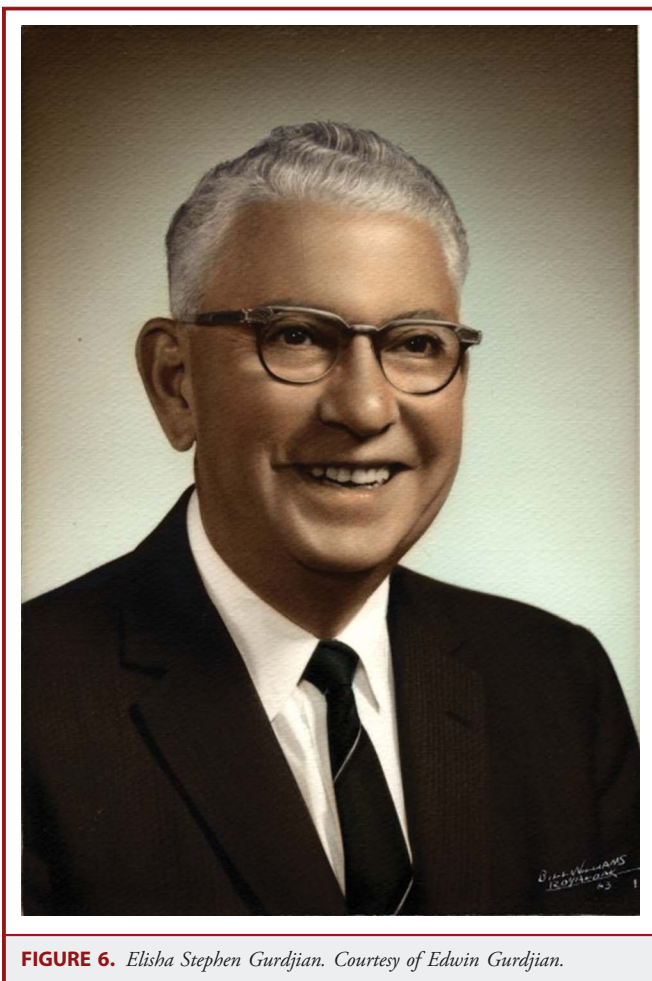


FIGURE 6. Elisha Stephen Gurdjian. Courtesy of Edwin Gurdjian.



FIGURE 7. Ayub K. Ommaya.

protect his skull.³⁰ Beginning in 1906, protective leather headgear, only occasionally used previously, began to be used more; however, the initial helmets were “essentially a pair of heavy-duty leather ear-muffs.” In about 1918, a helmet was developed consisting of an outer leather cover and inner felt lining (Figure 2).³¹

By 1931, American football was gaining popularity, attendance at games rapidly increased, and colleges and universities realized that substantial amounts of money could be made if they fielded a winning team. However, once again, the game came under considerable criticism because of concern about the great number of injuries and fatalities.²⁷

A pivotal individual during this period was Dr Marvin A. (Mal) Stevens (1900-1979), an orthopedic surgeon and head football coach at Yale. Stevens chaired the American Football Coaches Association (AFCA) Committee to Investigate Football Injuries and Fatalities, which he organized at the end of the 1931 season.³² Samuel C. Harvey (1886-1953) of Yale, a founding member of the Society of Neurological Surgeons and a Cushing trainee, was a member of this committee. The AFCA, with the

help of the National Safety Council and other concerned bodies, issued in 1931 the first annual report on football-related fatalities. Tackling was the activity with the greatest number of injuries (42% of fatalities), followed by blocking and being tackled (14% of fatalities).³² The report from 1931 to 1934 indicated that head and spine injuries accounted for about 68% of fatalities, with abdominal and internal injuries accounting for 24%. The major causes of death were “cerebral hemorrhage, spinal cord laceration, and internal hemorrhage.”³² Recommendations included preseason training; proper equipment for all players, including head protectors or helmets, to also be used in practice; better coaching of blocking and tackling techniques; preseason medical examinations; stricter officiating; and having a physician available at all practices and games. An additional recommendation was that an association of high school coaches be formed comparable to the AFCA for college football.²⁹

In 1932, a more modern helmet appeared with an early form of webbed suspension, and improvements of the suspension system progressed through the 1930s.³⁰ Helmets became mandatory at the high school level in 1935 and at the college level in 1939.²⁹

In 1933, Drs Stevens and Winthrop M. Phelps, also an orthopedic surgeon at Yale, published a 241-page book of much historical interest titled *The Control of Football Injuries* (Figure 3).³² This book very likely played a major role in the reducing football fatalities and catastrophic injuries. They had carefully analyzed football films over a period of 12 years to determine the causes of injuries and their prevention. Concussion was discussed

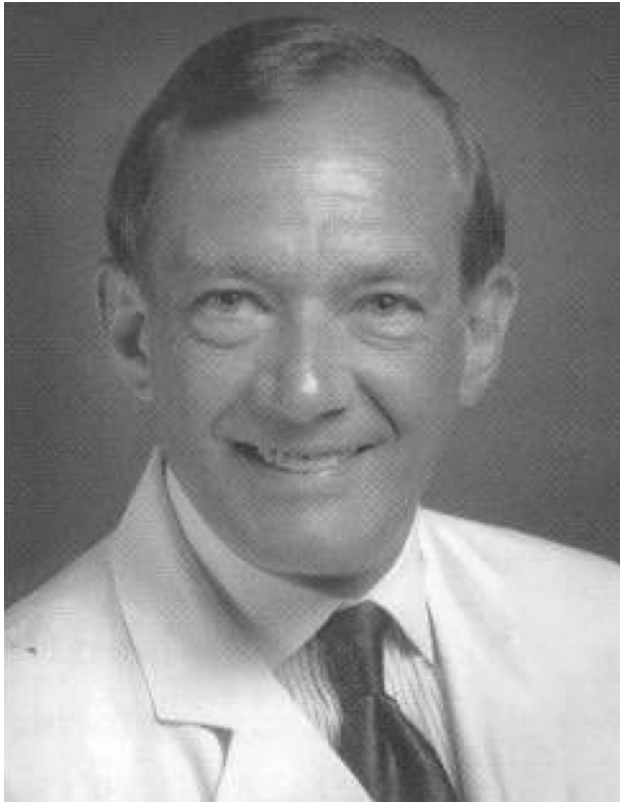


FIGURE 8. *Thomas A. Gennarelli.*

extensively. We quote that discussion here because much remains pertinent:

More concussions occur in football than are generally recognized. . . . Concussion is the result of a blow on the head. . . . sufficiently hard to cause a period of temporary disturbance of the proper functioning of the brain. . . . usually apparent either from a period of unconsciousness or. . . a period during which the player is dazed or unaware of what is going on. He may seem to continue to play normally but will not remember. . . events afterward. This period of amnesia may last from a few minutes to a few hours. A mild concussion may often be determined by asking the player questions. . . what is your name. . . the day of the month. . . or the signal of the last play. Inability to answer these questions promptly and accurately should result in removal of the player from the game for further examination. A differentiation must be made between inaccurate answers due to concussion and those due to pain or excitement. Headache or vomiting are usually diagnostic of a more severe concussion. . . treatment of a concussion must depend entirely upon the severity of the condition.

It may be said that a player who receives repeated concussions should consider very seriously withdrawing from football. The

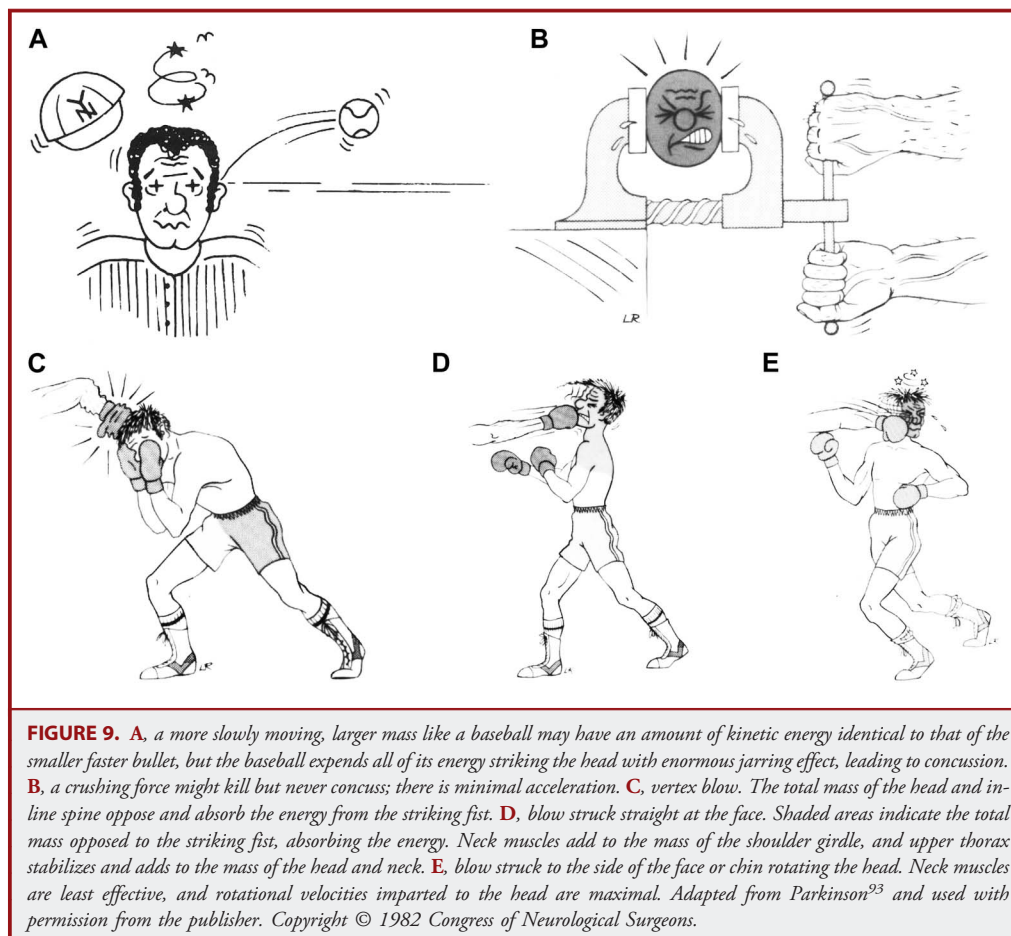
danger. . . with a concussion is that of a coincident intracranial or intracerebral hemorrhage which may be delayed in its appearance. The neurological examination which follows a concussion, therefore, should be made not only immediately after the player's removal from the game but he should be watched for at least twenty-four or 48 hours for any evidence of increased intracranial pressure, signifying bleeding. The symptoms which are referred to in connection with concussion are usually termed "dazed," "goofy," "nuts," "ringing in the head," "knocked out," etc. . . . These terms are all significant of temporary cerebral disturbances which must be considered in connection with concussion. The ability to continue playing the game is not necessarily evidence that a concussion has not occurred. A player with a concussion may frequently continue through the game, carrying through the plays in an automatic manner which appears to be perfectly normal. Close observation however, usually will show definite deviations, but the more highly trained the player and the more thoroughly he has learned his particular jobs in connection with the game, the more difficult it will be to determine any deviation from normal behavior. The criterion would be inability to remember at the end of the day, or the next day, activities during or following the game. In other words the usual type of concussion does not particularly interfere with the motor function of the brain but only with the memory and consciousness.

Probably the most important piece of equipment for the football player from the standpoint of preventing serious injury is his headguard. There had been a tendency to make headgear heavier, larger, and harder in the past few years in a vain attempt to curb an increasing number of head injuries. This has resulted, in many cases, in the helmets becoming offensive weapons on the heads of powerful, heavy, hard-hitting line plungers and yet not acting as shock absorbers for the cranium of the wearer. The heavy, molded, padded leather is stiff enough to prevent scalp wounds and contusions. However, while it will not collapse under the blow of a hammer, it fails to absorb the shock of such blows as kicks from shoes. . . knees or. . . the head striking the ground. . . . Despite the inadequate protection offered by the present day headguards, every player should be required to wear a helmet in practice and games.³²

Stevens and Phelps were very astute in their motion picture identification of factors leading to concussion and catastrophic brain and cervical spine injuries (Figure 4).³² They recommended prevention by rule changes combined with changes in the techniques taught by coaches. Further progress in the mitigation of head and spinal injuries would again come in the 1950s through the 1970s, especially from the landmark work of Schneider in football and Tator in ice hockey (see below).

Bilik's section on concussion in his book *The Trainer's Bible* (1948), unchanged from his 1934 edition, makes several points.^{2,33} After a mild concussion.

If he shows normal muscular control, complete orientation, does not complain of dizziness or headache, the conclusion must be that the jarring of the brain was quite mild. If the physician approves, the boy may resume play. If there is no urgent need of him, take him out for a rest and further observation. . . . The seriousness of the



concussion is not always proportionate to the violence of the blow. A severe fall or blow may result in nothing worse than a momentary sensation of “seeing stars,” whereas an apparently mild trauma may do untold organic damage. The explanation lies in individual susceptibility, which as I have repeatedly stressed, is a vital factor in a great number of athletic injuries. Some athletes are susceptible to concussion and may have a number of them. One must seriously consider the advisability of having them quit football. What’s that story of going to the well once too often. . . .³³

However, by 1939, it was stated that college players no longer left school “punch drunk.”³⁴

Another important AFCA recommendation made in 1937 was in response to the reporting of catastrophic death as due to “cerebral hemorrhage or old concussion.” The AFCA declared, “During the past 7 years the practice has been too prevalent of allowing players to continue playing after a concussion” and “this practice must be eliminated.”² Notably, from 1931 to 1940, the number of football fatalities dropped from 33 to 11, a reduction of 66%.²⁹

During this period of the 1930s and 1940s, athletic trainers were highly experienced in and knowledgeable about the diagnosis and dangers of concussion and clearly knew when to call for the

help of a physician. “Every case of concussion must be considered as a severe one. Someone on the coaching or training staff will have seen the injury as it occurred and will know about what to expect from experience. . . there is no occasion for giving stimulants in conditions of simple concussion.” (Decades ago, ammonium smelling salts were administered as a stimulant to unconscious or drowsy patients but could lead to agitation, combativeness, and secondary injury. They are no longer recommended.) “In the great majority of so-called concussions involved in athletics, the athlete returns to consciousness even before there is time to call the team physician, and harmful after effects are few. The team physician must be consulted, however, when recovery is delayed for more than a few minutes.”³⁵

A significant design improvement of the football helmet was the introduction of the internally padded rigid plastic shell outer helmet in about 1950, and progressively improved inner suspension systems were developed.^{26,31} Single-bar face protectors were added to helmets by 1955. Later, the double-bar face protector was developed. More recently, helmets have included birdcage-type face protectors attached to a polycarbonate shell with a variety of pneumatic, hydraulic, and web suspension systems.^{31,36}

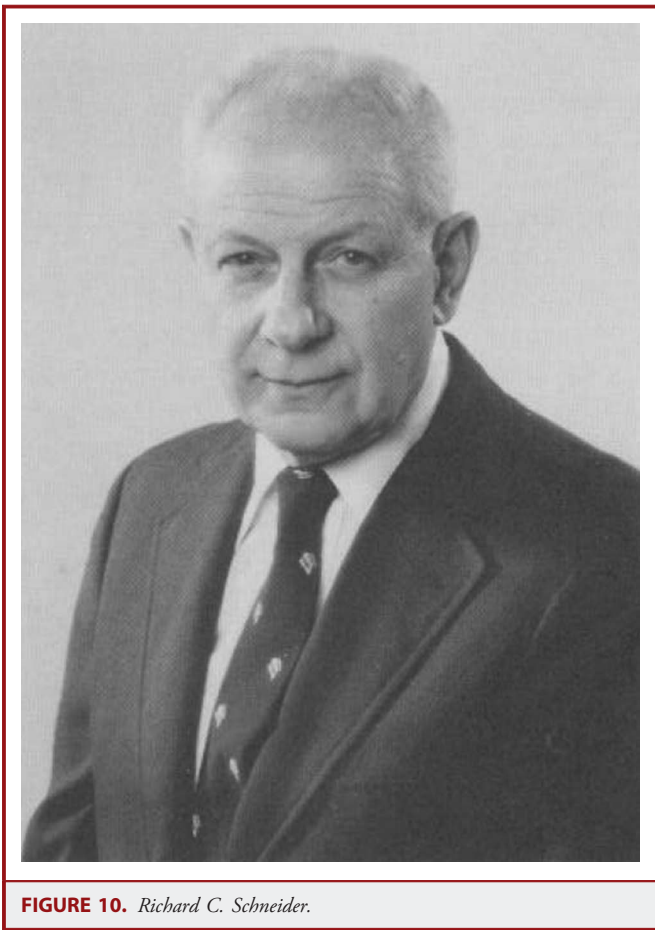


FIGURE 10. *Richard C. Schneider.*

Unfortunately, as predicted by Stevens and Phelps (1933), the modern helmet afforded such a high degree of protection that it permitted the head to be used as a battering ram, placing the head and cervical spine at significant risk of injury by the early 1960s.^{31,32} In fact, it is sadly ironic that coaches had been teaching players to make initial contact using their helmeted head or face (spearing or stick blocking) either into an opposing player’s chest (numbers tackling) or abdomen (“butt tackling” and “butt blocking”) or making initial contact with the head and face in tackling and blocking. In addition, the players were not taught to keep their heads up during blocking, tackling, or being tackled to avert cervical compression fracture/dislocation, as importantly pointed out by Stevens and Phelps (1933) and later by Schneider (1973), and Tator and Edmonds (1979).^{26,32,37}

Between 1961 and 1970, the largest number of catastrophic head and neck injuries and fatalities (90% of the 244 total fatalities) at the high school and college levels was seen, eclipsing the 1931 figures, with cerebral hemorrhages accounting for about 75% of deaths.²⁹ Fortunately, once again rule changes, especially the 1976 rule that prohibited initial contact with the head and face when blocking and tackling, and the National Operating Committee on Standards for Athletic Equipment helmet standard took effect at the college level in 1978 and high schools in 1980. These factors have significantly reduced major catastrophic brain and spinal cord injuries in American football.²⁹

PROGRESS IN UNDERSTANDING CONCUSSION BIOMECHANICS

During and after World War II, neurosurgeons Hugh W.B. Cairns (1896-1952; Figure 5) in Oxford, England, and

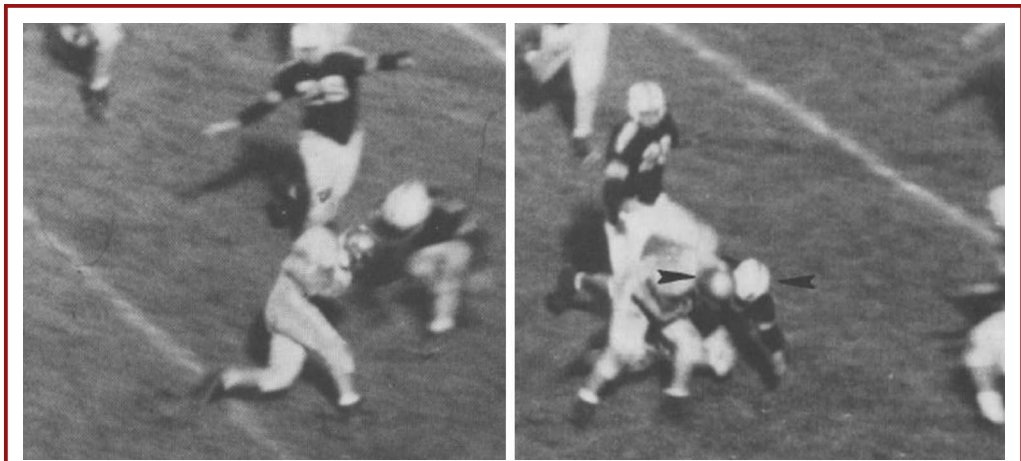


FIGURE 11. *Head butting” by a high school tackler (black jersey, right arrow) with no attempt to use arms. The tackler was immediately unconscious with dilated and fixed pupils on arrival at the hospital and died despite drainage of an acute subdural hematoma. Reproduced from Schneider²⁶ with permission from the publisher. Copyright © 1973 Williams & Wilkins.*



FIGURE 12. Lexan calvarium attached to a monkey skull used by Gosch and Schneider to study brain movements after impact. Reproduced from Schneider²⁶ with permission from the publisher. Copyright © 1973 Williams & Wilkins.

E. Stephen Gurdjian (1900-1986; Figure 6) in Detroit, Michigan, developed particular clinical and research interest in brain injury mechanisms and mitigation of injury. In addition to military and civilian casualties, escalating motor vehicular injuries led both Cairns and Gurdjian to engage the services of theoretical physicists and engineers.^{15,38-41} This resulted in original ideas and research to address the vexing mechanical substrates of cerebral concussion, skull fracture, brain contusion, and helmet design. Cairns enlisted the help of Oxford physicist A.H.S. Holbourn (1907-1962), who constructed 2-dimension gel-filled models of the head emphasizing the shear strains secondary to angular acceleration.⁴²⁻⁴⁴ Commissioned war-related research was performed by British neurologists Derek Denny-Brown (1901-1981) and W. Ritchie Russell (1903-1980) on acceleration-induced concussion in cats and monkeys.^{45,46} Their studies proved that with sufficient impact, it was the forces of acceleration or deceleration that were responsible for concussion (LOC) or a stunned or dazed condition, neither with apparent acute macroscopic or microscopic lesions. However, hemorrhagic

lesions were produced by forces exceeding the concussion threshold. Concussion or subconcussion did not occur if the head was held rigidly when the blow was delivered.

As Europe recovered from the devastation of World War II, American neurosurgically and neurologically driven investigation of experimental concussion significantly advanced. E. Stephen Gurdjian began work on clinical neurotrauma in the 1920s, and his neurosurgical research team formed in the 1940s performed original work on skull fracture, acceleration injury, intracranial pressure, and inertial brain movements and extensive work on concussion in experimental animals, including primates.¹⁵ Gurdjian believed that impact produced intracranial pressure gradients transmitted through the brain, combined with inertial brain movements, and that shear strains generated in the region of the brainstem resulted in concussion.^{15,26,47} Such basal strains were visually evident in a number of Gurdjian's gel models and sliced cadaver head models of the brain that, unlike Holbourn's models, included the foramen magnum.¹⁵ Under Gurdjian, a biomedical engineering group to study head and spine trauma, likely the first of its kind, was set up in Detroit, where General Motors is located. Gurdjian and his group's mitigation interests went well beyond automotive injuries and beginning in the 1950s included sports injuries and sports helmets.^{15,48-56}

Additional investigative neurosurgical teams in the 1940s and 1950s who worked in experimental primate concussion (either "acceleration concussion" or "fluid percussion/compressive concussion") or recorded cortical or subcortical electrical potentials in studying the reticular activating system in relation to consciousness included A. Earl Walker, John D. French, Arthur A. Ward, Eldon L. Foltz, neurologist Derek Denny-Brown who was then in Boston, and neurophysiologist Horace W. Magoun's group then at Northwestern.^{22,57-67} Many of these investigators, including Gurdjian, demonstrated nerve cell loss and chromatolysis within the brainstem reticular activating system and less so cerebral cortex after experimental primate and quadruped concussion.¹⁵ Opinions varied as to the pathological substrate of concussion from normal gross and microscopic findings in a very occasional rapidly fatal case to scattered petechial hemorrhages in the white matter and brainstem or the presence of hemorrhages within the corpus callosum and superior cerebellar peduncles.^{68,69}

In 1956, the British neuropathologist Sabina Strich characterized the extensive white matter axonal injury she found in clinically diagnosed severe concussion patients continually unconscious or poorly responsive after injury.^{70,71} These patients had survived as a result of more modern methods of resuscitation and nursing. Strich attributed these white matter findings to rotational or angular shear forces, as hypothesized by Holbourn, and felt a clinical/pathological continuum of severity likely existed. Notable British neurosurgeons came to believe that although concussion used to be regarded as a functional condition unaccompanied by structural changes and associated with rapid recovery, "it is never a totally reversible disorder of function, and structural damage always occurs even if only of minimal

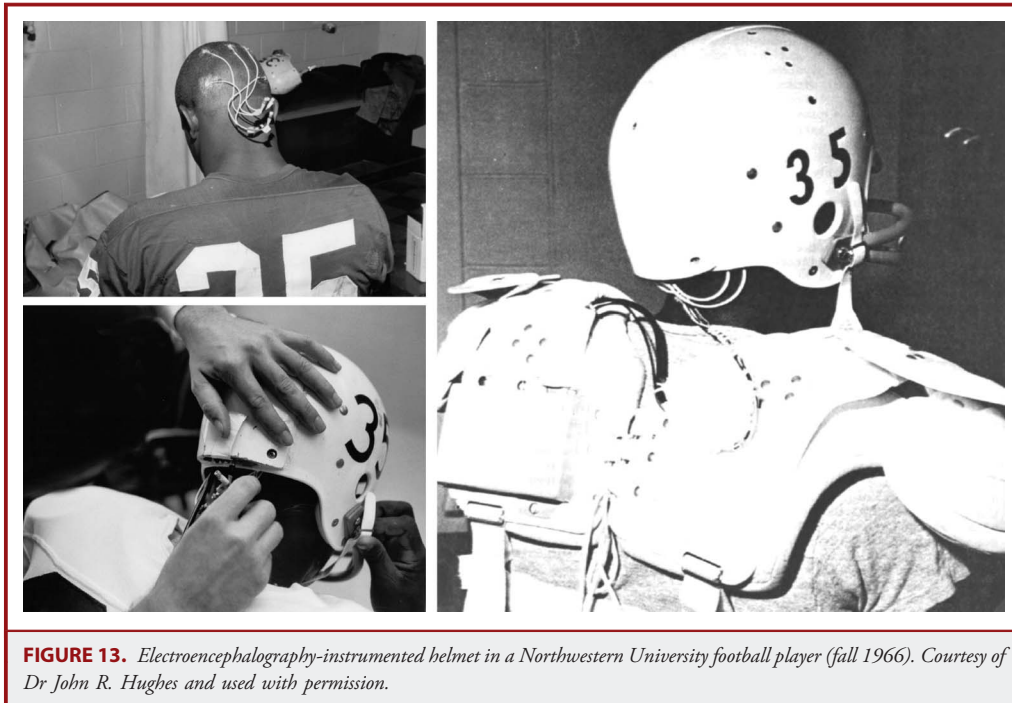


FIGURE 13. Electroencephalography-instrumented helmet in a Northwestern University football player (fall 1966). Courtesy of Dr John R. Hughes and used with permission.

degree.”⁶⁹ Bryan Jennett (1926-2008) concurred and added that “the difference between mild and severe brain injuries of a diffuse kind may be quantitative rather than qualitative” and “probably accounts for the observation that concussion injuries are cumulative (ie in boxing).”⁷²⁻⁷⁷

Beginning in 1963, the investigative neurosurgeon Ayub K. Ommaya (1930-2008; Figure 7), working at the National Institutes of Health, began the study of brain injury in primates, including whiplash without impact. He sought to test Holbourn’s hypotheses that angular (rotational) acceleration was more damaging to the brain as it resulted in more shearing or sliding forces because of the anatomy and tissue constituents of the brain, compared to linear (translational) acceleration and impact forces, which caused less harmful, largely compressive and tensile (pulling apart) forces.^{44,78,79}

Ommaya was joined in the early 1970s by neurosurgeon Thomas A. Gennarelli (Figure 8) and bioengineer Lawrence E. Thibault (1943-2011). By the mid-1970s, they completed a number of carefully designed brain injury studies in subhuman primates.⁷⁹ In particular, they investigated non-impact, acceleration forces, both angular (rotational) and translational (linear).^{80,81} Nonimpact angular (rotational) acceleration led to a reproducible spectrum of injury severity or “centripetal continuum” from physiological concussion to prolonged unconsciousness and death from severe widespread white matter shearing (Strich lesion).⁸²⁻⁸⁴ Nonimpact, isolated translational (linear) acceleration had a much greater threshold to produce concussion, and resulted in predominantly focal lesions as predicted by Holbourn.^{5,81,85,86} Gennarelli and Thibault moved

to Philadelphia where further work was completed, delineating angular coronal or oblique acceleration as most harmful and prone to concussion, as well as the biomechanics of acute subdural hematoma.^{87,88} Their primate autopsy findings were corroborated by a team of neuropathologists in Glasgow and called diffuse axonal injury in 1982.^{83,84} Ommaya and Gennarelli, like Gurdjian and others before them, addressed sports injury and helmet design in their writings.⁸⁹⁻⁹²

The forces related to motion or kinetic energy are defined as $\frac{1}{2}$ mass times velocity squared ($KE = \frac{1}{2} M \times V^2$). Velocity equaling distance divided by time squared ($(D/T)^2$) tells us that as time becomes of shorter duration (brief, impulsive loading), kinetic energy becomes greater, and the type of damage is dependent on the specific nature of the injurious forces and injured tissue.^{93,94} A more slowly moving, larger mass like a baseball may have an amount of kinetic energy identical to that the faster moving bullet, but the baseball expends all of its energy striking the head with enormous jarring effect, leading to concussion, as opposed to the much smaller penetrating bullet (Figure 9A). It has been known for centuries that it is virtually impossible to knock out an opponent whose head is fixed against a wall or the ground (Figure 9B), and boxers are well aware of the futility of pounding on the vertex of their opponent’s head (Figure 9C).^{93,95} In these cases, the head becomes an extension of the mass of wall, ground, or in-line spinal column and cannot be rapidly set in motion by a blow (Figure 9B and 9C).⁹³ Thus, kinetic energy is transferred through the immobile skull to the larger masses and the brain does not suffer a jolting concussive force. Figure 9D shows how the energy of a blow straight to the face is absorbed by the neck muscles and the

TABLE 1. The Evolving Definition and Grading of Sports Concussion

Thorndike¹²⁶ grading system for concussion, 1948	
Mild type	Athlete is “momentarily knocked unconscious” and immediately recovers or regains all of his intellectual functions of initiative, planning, thinking and attention.
Mild (boxing) type	Athlete is “knocked out on his feet” but never loses consciousness completely and retains some of his intellectual and all of his vegetative consciousness.
Severe type	The “period of unconsciousness is obvious and exists for a minute or longer,” and the patient suffers from the residual symptoms of headache, dizziness, and complete or incomplete loss of intellect.
Schneider and Kriss¹²⁹ grading system for concussion, 1969	
Mild	No loss of consciousness but other symptoms or signs
Moderate	Loss of consciousness for 3-4 min and mild posttraumatic amnesia
Severe	Loss of consciousness for >5 min and prolonged posttraumatic amnesia
Cantu¹⁵⁴ grading system for concussion, 1986	
Grade I	No loss of consciousness; posttraumatic amnesia lasting <30 min
Grade II	Loss of consciousness for <5 min or posttraumatic amnesia lasting >30 min but <24 h
Grade III	Loss of consciousness for >5 min or posttraumatic amnesia lasting >24 h
Kelly et al (Colorado)¹²⁸ grading system for concussion, 1991	
Grade I	Confusion without amnesia; no loss of consciousness
Grade II	Confusion with amnesia; no loss of consciousness
Grade III	Loss of consciousness
American Academy of Neurology Practice Parameters (Kelly and Rosenberg)¹³¹ grading system for concussion, 1997	
Grade I	Transient confusion; no loss of consciousness; concussion symptoms or mental status abnormalities on examination resolve in <15 min
Grade II	Transient confusion; no loss of consciousness; concussion symptoms or mental status abnormalities on examination last >15 min
Grade III	Any loss of consciousness, either brief (seconds) or prolonged (minutes)
Cantu¹³² grading system for concussion (revised) 2001	
Grade I	No loss of consciousness; posttraumatic amnesia/postconcussion signs or symptoms <30 min
Grade II	Loss of consciousness <1 minute or posttraumatic amnesia >30 min but <24 hours, or postconcussion signs or symptoms >30 min but <7 d
Grade III	Loss of consciousness ≥1 min or posttraumatic amnesia ≥24 h; postconcussion signs or symptoms >7 d

shoulder girdle, and the stability of the upper thorax adds to the mass of the head and neck. However, a rapid-velocity blow struck at the side of the head, face, or chin is most effective in producing concussion as a result of the rotational component of the blow, as evident in films of boxing ring knockouts (Figure 9E).⁹³ Recent work in our laboratory suggests that internal jugular vein compression by a collar, and perhaps in vivo by deeper cervical neck muscle contraction, reduces brain injury in animals (slosh mitigation) somewhat akin to that shown in Figure 9D.^{96,97}

THE CONTRIBUTIONS OF RICHARD C. SCHNEIDER

In the 1950s and 1960s, Richard C. Schneider (1913-1986; Figure 10), a neurosurgeon in Ann Arbor, Michigan, reviewed and analyzed the injury patterns involved in a personally treated

serious or fatal neurosurgical football injuries, and collected a larger series of 619 serious or fatal football injuries from 1959 to 1963 from American neurological surgeons (Congress of Neurological Surgeons and American Association of Neurological Surgeons).^{98,99} Schneider’s experience included several cases of primary pontine and primary central spinal cord hemorrhages at and just below the cervicomedullary junction. These injuries were in most cases caused by (photographically verified) vertex impact head injuries in helmeted players and suggested certain mechanisms of injury to Schneider (Figure 11).^{8,26,100,101}

Schneider’s research group next performed studies in anesthetized rhesus monkeys fitted with a transparent frontoparietal (at times including the occipital and temporal regions) clear, lexan calvarium (Figure 12) to directly visualize with high-speed motion pictures the degree and nature of cerebral hemispheric movements and marked cortical impaction subsequent to frontal and vertex

TABLE 2. Second International Symposium on Concussion in Sport, Prague, Czech Republic, in 2004 Proposed a New Classification of Concussion in Sport (in Place of Previous Grading Systems)

Simple concussion

The most common form of this injury can be appropriately managed by primary care physicians or by certified athletic trainers working under medical supervision. The athlete suffers from an injury that progressively resolves without complication over 7-10 d and typically resumes sport without further problem. The cornerstone of management is rest until all symptoms resolve and then a graded program of exertion before return to sport. All concussions mandate evaluation by a medical doctor. Formal neuropsychological screening does not play a role in these circumstances, although mental status screening should be a part of the assessment of all concussed athletes.¹³⁷ The Sport Assessment Concussion Tool or similar tool for onsite athlete symptom listing, athlete information, signs, memory cognitive assessment, and neurological screening should be filled out by the athlete if he/she is capable.¹³⁷

Complex concussion

In these cases, athletes suffer persistent symptoms, including persistent symptom recurrence with exertion, specific sequelae such as concussive convulsions (impact seizures), prolonged loss of consciousness for >1 min, or prolonged cognitive impairment after the injury. This group may include athletes who suffer multiple concussions over time or repeated concussions with apparently less impact force. This group may have additional management concerns beyond simple return to play decisions. "Formal neuropsychological testing and other investigations should be considered in the group, and it is envisioned that such athletes would be managed in a multidisciplinary manner by doctors with specific expertise in concussive injury management, such as a sports medicine doctor with experience in concussion, sports neurologist, or neurosurgeon."¹³⁷



FIGURE 14. *G. Robert Nugent of Morgantown, West Virginia, was a National Collegiate Athletic Association collegiate football sideline team physician for 40 years. During this period, he helped define the essence and role of sideline participation and clinical evaluation. He emphasized practical clinical management, added to the characterization of cervical neuropraxias such as burners and stingers, and joined Schneider in voicing concern over evolving dangerous techniques such as spearing related to the use of helmets and facemasks as weapons. His clinical points on concussion and other injuries remain exceptionally poignant today.*¹⁴⁵

decelerating impacts.^{26,102-104} Similar cerebral movements had been documented by others using different techniques.^{15,105,106}

His group concluded that significant vertex deceleration impacts resulted in direct transmission of force through the rigid, nonresilient helmet and intact skull to the brain. "Initially the brain moves en masse in a cephalad direction but then on impact—rebounds, forcing the cerebral hemispheres in a caudal direction. . . ."³⁶ He felt the result was acute, massive, downward cerebral displacement of the brain causing herniation at the tentorium and the foramen magnum.^{26,36} Schneider believed that urgent efforts should be made to dissipate these forces with improved protective equipment and elimination of spearing and stick-blocking.

After experimental vertex impact in the monkey, with the animal's neck maintained in a straight position, marked telescoping of the foramen magnum was noted over the upper cervical cord segments. They documented the marked movement of the visualized cerebral hemispheres compared with the grossly visualized rigidity and nonmovement of the upper cervical cord

resulting from the dentate ligaments, as also noted by others.^{28,47,107} Schneider also believed that cases of instantaneous respiratory or cardiac arrest may be secondary to compression of the vertebral arteries between the occipital condyles and C1; or direct transaxial transmission of force to the skull, cervicomedullary junction, and high cervical cord; or shear force damage to the above areas caused by tethering of the upper cervical cord by the dentate ligaments.³⁶ Hemorrhagic damage to the upper cervical cord and cervicomedullary junction was prevented in impacted monkeys by sectioning of the cervical dentate ligaments.^{36,107}

Schneider believed that the cause of cerebral concussion likely involved the mechanisms detailed above in that devastating upper cervical spinal injuries from spear tackling were associated with both concussion in football players and experimental primate concussion after vertex impact.^{26,101-104}

Schneider referred to experimental work by others in the late 1960s regarding the placement of early-generation accelerometers and 2-channel electroencephalography (EEG) scalp recording

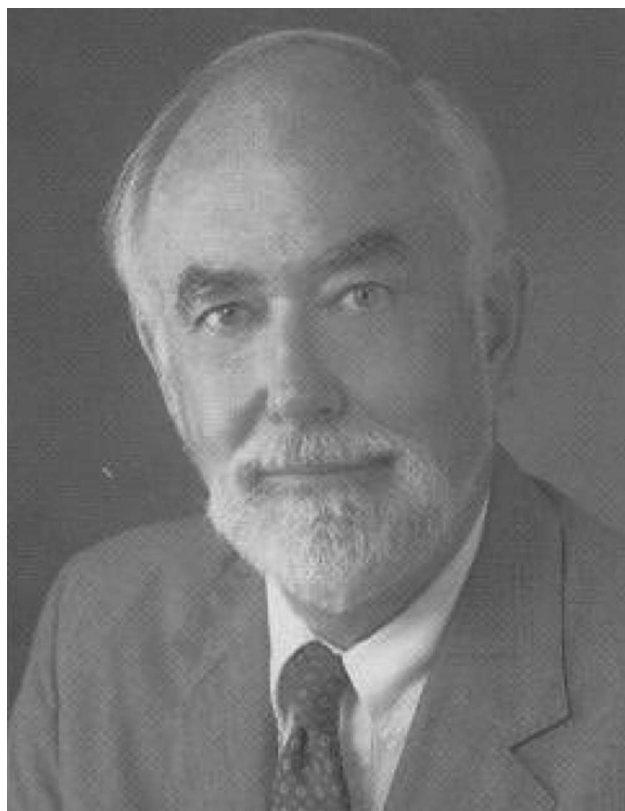


FIGURE 15. Horace Norrell was a football team physician at the University of Kentucky and defined his role as a “protector of athletes and educator of other physicians, coaches, and trainers.” A student of Schneider, he believed in the importance of critical observation, timely rule changes, and implementation (eg, spearing).¹¹⁵



FIGURE 16. Charles H. Tator of Toronto, Canada, in the early 1970s began an extensive clinical and research program in spinal cord injury. His continued dedication to concussion and the epidemiology and prevention of head and spinal cord injury in sports, especially ice hockey, has been quite remarkable. Additionally, his work has proved the effectiveness of preventative programs and he has been recognized internationally as a member of the Concussion in Sport Group. Dr Tator is a firm believer that more neurosurgeons should be involved in the field of sports and recreational injuries and can make a significant impact in their community. Several decades ago, Dr Tator and his engineering associate, Patrick Bishop, were contacted after a catastrophic cervical cord injury in a teenaged Chicago-area ice hockey player. Discussions centered on the causation of such injuries (head-down position near the boards) and how they can be prevented. We presented this beneficial, very much welcomed material to the local hockey organizations to institute more stringent rule changes, preferential coaching, and conditioning techniques.^{37,146-151.}

electrodes in the helmet of a filmed Northwestern University football player.²⁶ This allowed the telemetric capture of both the physical forces and EEG changes encountered during a full season.¹⁰⁸ Maximum tolerable forces were recorded between 188g to 230g with long durations of 310 to 400 milliseconds, indicating tremendous forces transmitted to the brain. One concussion was captured that was caused by an 188g left frontal force over 310 milliseconds, resulting in right-sided EEG showing θ slowing and amplitude suppression.¹⁰⁸ A 1966 study examined the dynamic aspects of EEG during play using 2-channel EEG telemetered helmet during a full season (Figure 13). A concussion was not captured, but after several severe impacts, a few seconds of θ slowing appeared, but the paucity and short duration of the slowing suggested insignificant head trauma.¹⁰⁹ Quantitative impact characterization with simultaneous physiological correlation is a very promising avenue of research in light of present concerns about cumulative concussive injuries, the emerging concept of subconcussion, and the potential for improvement in safety and player management.¹¹⁰

Schneider’s patented “Michigan helmet” had a double, inner crown pneumatic system and firm plastic outer shell with 4 parasagittal “islands” of more resilient material over relatively silent cortical areas. The helmet also had a collar of protective foam extending inferior to the posterior rim of the helmet to minimize a possible “guillotine effect” after a cervical hyperextension injury. Comparison with other helmets revealed the delayed and lower peak impact forces.^{26,36,111} The helmet was field tested by the University of Michigan football team but never came into general use.^{26,36} Schneider even advocated complete removal of the face

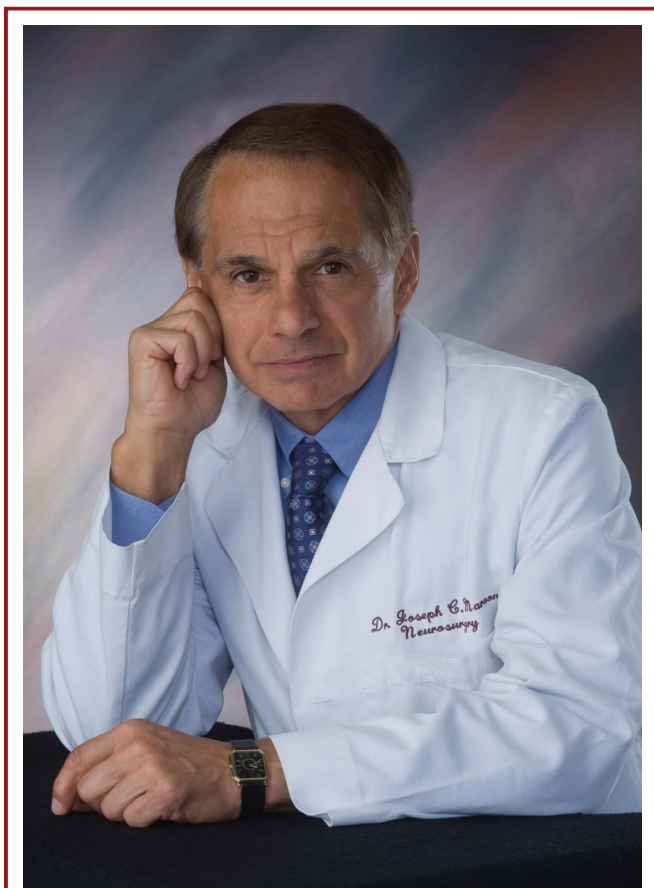


FIGURE 17. Joseph C. Maroon of Pittsburgh, Pennsylvania, was a collegiate scholastic all-American running back at Indiana and has been much involved with sport and fitness. He was a key figure in the modern era of sideline football assessment and the grading of concussion and was the first full-time National Football League sideline physician. Dr Maroon developed innovative programs for the management of athletes with cerebral concussions that have been extensively used throughout the National Football League and National Hockey League. He has acquired extensive clinical and operative experience in cervical spine injuries in athletes. Dr Maroon has been instrumental in the development of computerized onsite neuropsychological testing of athletes with possible or definite mild traumatic brain injury.^{130,152-154}

mask to improve protective vision by about 30%, to encourage proper skills, and thereby to lessen brain injuries.³⁶

In 1973, Schneider was the first to mention the second impact syndrome of malignant brain swelling by returning to contact sports before recovery from concussion.¹¹²⁻¹¹⁴ Schneider's presence continues to be felt in the neurosurgical recognition and management of injuries of the head and neck. He taught several generations of neurosurgeons to be proactive within our individual communities in regard to sports injury and to assume accountability as the experts and leaders in brain and spinal cord injuries who can make a great difference in the lives of young athletes.¹¹⁵



FIGURE 18. Robert C. Cantu of Boston was an early advocate of practical concussion grading scales with implications for return to play and gained extensive clinical experience in the management of athletic concussion. He has been a national and international leader in sports organizations for concussion and was an original Concussion in Sport Group member. He is a consultant for numerous amateur and professional athletes, has been a prolific writer on various major topics in neurological sports medicine, and actively serves numerous organizations related to sports medicine. In recent years, Dr Cantu established and now codirects the Center for the Study of Traumatic Encephalopathy at Boston University, which has helped define the pathophysiology of chronic traumatic encephalopathy.^{94,112,113,131,132,155,156}

In recent years, football has been made safer, thanks to the continued injury registries, oversight, rules changes, responsive changes in coaching techniques, and standardization of protective equipment, including helmets for football and other sports in the 1970s. From 2003 to 2008, *Neurosurgery* published an insightful series of articles on concussion in professional football to address the above topics, including impact video analysis, biomechanics, helmet technology, neuropsychological testing, and RTP decisions.^{116,117} Direct football fatalities combined for sandlot, high school, college and professional players were in single digits for the years 1987 to 2008.²⁹

THE EVOLVING DEFINITION AND GRADING OF SPORTS CONCUSSION

As traditionally defined, concussion denoted an immediate and full LOC of variable duration accompanied by posttraumatic amnesia at least for the event. The duration of both features was believed to be related to the magnitude of the injurious physical forces.^{76,77,118-123} Lesser grades of concussion severity such as



FIGURE 19. *Arthur L. Day of Houston Texas was president of the Sports Medicine Section of the American Association of Neurological Surgeons/Congress of Neurological Surgeons, a National Collegiate Athletic Association sideline physician at the University of Florida, and established protocols for the sideline evaluation of injured athletes. He has particular interest and experience in lumbar spine syndromes in athletes. He continues to be active in neurological sports medicine and serves on the Medical Advisory Board of Pop Warner youth football.*^{157,158}

a stun or brief disorientation have also been described throughout the ages.

Because cerebral contusions by themselves, unless very large, rarely cause LOC, “the criterion of unconsciousness and post-traumatic amnesia as evidence of brain damage is reliable only for the diffuse brain damage due to acceleration-deceleration injury.”¹¹⁸ It should be noted that the Scottish neurosurgeon Bryan Jennett (1926-2008), a modern pioneer in head injury, and his associate Graham Teasdale often addressed sports injury in their writings.^{16,124}

Agustus Thorndike, the American surgeon and sports physician, presented what may have been the first sports concussion

grading scale with corresponding indications for RTP or treatment indications in 1948 (see Table 1).¹²⁵ Regarding treatment, for the mild types, he advocated:

Often after a short rest, a football player can make a complete mental recovery and only then if nystagmus, dizziness, and headache are absent, should he be permitted to return to the game. The treatment of the more severe type of concussion, in which loss of consciousness is more than momentary and residual headache and dizziness continue for as much as an hour of recumbent rest, is hospitalization.¹²⁵

Thorndike was quite concerned about the risks of recurrent concussion and stated that individuals “with cerebral concussion that has recurred more than three times or with more than momentary loss of consciousness at any one time should not be exposed to further body contact trauma. . . authorities are conscious of the pathology of the ‘punch drunk’ boxer.”¹²⁶

Our current definitions of concussion stem from the Congress of Neurological Surgeons and have been subsequently applied to sports concussion.¹²⁷ Concussion of the brain is “a clinical syndrome characterized by immediate and transient impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces.”¹²⁷ The major feature is that “the term concussion no longer required the patient to have had a complete LOC and presented a much more realistic approach to the problem.”²⁶ Concussion includes a brief alteration of consciousness (stunned or dazed state, vacant stare), transient confusion (inattention, inability to maintain a coherent stream of thought and carry out goal-directed movements), slurred or incoherent speech, incoordination, delayed verbal and motor responses, memory loss, visual loss, and tinnitus.¹²⁸

Concerned with concussion in football, Schneider obtained permission from the Congress of Neurological Surgeons to publish its definition of concussion, along with a Sports Concussion Grading System useful to sports physicians, coaches, and trainers.^{8,26,129} In 1980, Maroon, an experienced team physician, and his group simplified sport concussion into 3 similar grades.¹³⁰ Cantu had a 1986 grading system modified in 2001.^{131,132}

In 1990 to 1991 James Kelly, a neurologist working with neurosurgeons (J.S. Nichols and K.O. Lillehei) in Colorado, devised a sports concussion classification,¹²⁸ which was modified in 1997 after meetings in Pittsburg, Pennsylvania, organized by neurosurgeons Bailes and Maroon and was supported by the American Academy of Neurology.¹³³ The player can RTP (first concussion) that day if all mental status abnormalities or postconcussive symptoms or signs clear at rest and with exertion within 15 minutes. Other grades are removed from contest that day, and various RTP recommendations are made. They make reference to the second impact syndrome, the possibility of cumulative concussion injury, and the Standardized Assessment of Concussion.¹³³

Once a concussion is recognized or brought to the attention of coaches or trainers, the player is removed from the game and observed. If the athlete is without headache or dizziness, has

apparently full cognition and orientation, and is completely asymptomatic at rest and after exertion with usual dexterity and speed, his or her return to the game may be considered.¹³²⁻¹³⁴ However, recent consensus statements have tended to become more conservative, not recommending most grade I concussion players to RTP that day (see the section consensus statements below).¹³⁵

The postconcussion syndrome consists of headache, especially with exertion, dizziness, fatigue, irritability, and impaired memory and concentration. It may last several weeks or (uncommonly) longer.^{29,136} Persistence of postconcussion syndrome usually correlates with the duration of posttraumatic amnesia and “suggests that the athlete should be evaluated with a CT scan and neuropsychological testing” (see the section Consensus Statements below).¹³⁷

An athlete is not returned to competition unless he or she has recovered fully from the symptoms or effects of concussion, the athlete is asymptomatic at rest and after moving with his or her usual dexterity and speed after exertion, and any diagnostic studies that may have been obtained are normal (see the section Consensus Statements below).

21ST CENTURY INTERNATIONAL SPORTS CONCUSSION CONSENSUS STATEMENTS

In 2001 and 2004, the First and Second International Symposia on Concussion in Sport were held in Vienna, Austria, and Prague, Czech Republic, respectively.^{134,138} The original aims of the symposia were to provide recommendations for the improvement of safety and health of athletes who suffer concussive injuries in American football, ice hockey, rugby, football (soccer), and other sports. The definition that arose from these meetings is as follows: “Sports concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.” Several common features include the following:¹³⁸

1. Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an “impulsive” force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
4. Concussion results in a graded set of clinical syndromes that may or may not involve LOC. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is typically associated with grossly normal structural neuroimaging studies.
6. In some cases, postconcussive symptoms may be prolonged or persistent.

The Vienna (First International Conference) recommendation was that injury grading scales be abandoned in favor of combined measures of recovery to determine injury severity or prognosis and

hence individually guide return-to-play (RTP) decisions.¹³⁴ Thus, a new Classification of Concussion in Sport (in place of previous grading systems) was proposed at the 2004 meeting (see Table 2).¹³⁷

The presence of a brief loss of consciousness as a symptom would not necessarily classify the concussion as complex...[and]...In regard to post traumatic amnesia—evidence suggests that the nature, burden, and duration of the clinical post-concussive (signs and) symptoms...may be more important than the presence or duration of amnesia alone...Further it must be noted that retrograde amnesia varies with the time of measurement after the injury and hence is poorly reflective of injury severity.¹³⁸

The 3rd International Conference on Concussion in Sport was held in Zurich in November 2008, and the consensus statement was subsequently published.¹³⁹ Although there was agreement about the principal consensus messages, the authors acknowledged that the science of concussion is evolving and therefore management and RTP decisions remain in the realm of clinical judgment on an individualized basis.¹³⁹ The definition and management of concussion are as in the previous consensus statements. Instead of the Sports Concussion Assessment Tool (first or second edition), the Standardized Assessment of Concussion or similar tools may be used.¹³⁹ “The cornerstone of concussion management is physical and cognitive rest until symptoms resolve and then a graded program of exertion prior to medical clearance and RTP.”¹³⁹

“A player with diagnosed concussion should not be allowed to return to play on the day of injury. Occasionally in adult athletes, they may return to play on the same day as the injury, as there is data that some American professional football players RTP more quickly, with even same day RTP without a risk of recurrence or sequelae.”¹⁴⁰ It was again emphasized that the young (<18 years of age) elite athlete should be treated more conservatively.¹³⁹

Objective balance assessment studies have identified postural stability deficits lasting about 72 hours after sports-related concussion, and this appears to be a valid addition to the assessment of concussed athletes, particularly when symptoms or signs indicate a balance component.¹³⁹

Again, neuropsychological testing in concussion has been shown to have clinical value and contributes significant information in concussion evaluation. Cognitive recovery may occasionally precede or more commonly follow clinical symptom resolution, suggesting that neuropsychological assessment should be an important part of any RTP protocol.¹³⁹

Consensus discussion determined that prolonged (>1-minute duration) LOC would be considered a factor that may modify concussion management.¹³⁹ Because published evidence suggests that retrograde amnesia varies with the time of measurement after the concussion, “it is poorly reflective of injury severity” compared with “the nature, burden and duration” of the clinical postconcussion syndrome.¹³⁹

In regard to chronic traumatic encephalopathy (CTE), epidemiological studies and case reports have presented neuropathological evidence of CTE that has suggested an association between repeated sports concussions during a career and late-life cognitive impairment. After panel discussion, “no consensus was reached on the significance of such observations at this stage. Clinicians need to be mindful of the potential for long-term problems in the management of all athletes.”¹³⁹

Regarding concussion prevention, there is no good clinical evidence that currently available equipment will prevent concussion, although mouth guards can prevent dental and orofacial injury. Although the use of helmets and headgear results in a reduction in biomechanically measured impact forces to the brain, “these findings have not been translated to show a reduction in concussion incidence.” Helmets are now recommended for skiing, snowboarding, and related alpine sports, and in sports such as “cycling, motor and equestrian sports, protective helmets may prevent other forms of head injury (eg, skull fracture)... related to falling on hard road surfaces; these may be an important injury prevention issue for those sports.”¹³⁹ Finally, “The issue of sports concussion management is continually evolving, and the usefulness of expert consensus in establishing a standard of care has been demonstrated...”¹³⁹

The 4th International Conference on Concussion in Sport was held in Zurich in November 2012.¹⁴¹ The Concussion in Sport Group concluded that “although the terms concussion and mild TBI [traumatic brain injury]... are often used interchangeably in the sporting context... Concussion is a subset of TBI...”¹⁴¹ They continued, “The majority (80%-90%) of concussions resolve in a short (7-10 day) period, although the recovery time may be longer in children and adolescents... Persistent symptoms (>10 days) are generally reported in 10% to 15% of concussions... and should be managed in a multidisciplinary manner by healthcare providers with experience in sports-related concussion.”¹⁴¹

A group of concussion modifiers that may influence investigation and management and may predict the potential for prolonged symptoms after concussion was assembled. LOC (>1 minute) and posttraumatic amnesia are discussed above. Although research evidence is not yet conclusive and there was no unanimous agreement among the Concussion in Sport Group, “it was accepted that the female gender may be a risk factor for injury and/or influence injury severity.” Motor and convulsive phenomena (eg, tonic posturing) “may accompany a concussion but these features are generally benign and require no specific management beyond the standard treatment of the underlying concussive injury.”¹⁴¹

Depression can be a clinically challenging problem after any traumatic brain injury, including concussion, and may be multifactorial. Functional magnetic resonance imaging studies “suggest that a depressed mood following concussion may reflect an underlying pathophysiological abnormality consistent with a limbic-frontal model of depression.”¹⁴¹

Neuropsychological assessment remains a cornerstone of concussion management. “Brief computerized cognitive evaluation tools are the mainstay of these assessments worldwide, given the logistical

limitations, but are not substitutes for formal neuropsychological assessment. At present, there is insufficient evidence to recommend the widespread routine use of baseline preinjury neuropsychological testing.”¹⁴¹

According to a recent consensus statement of the 4th International Conference on Concussion in Sport held in November of 2012, “It was unanimously agreed that no Return-To-Play (RTP) on the day of concussive injury should occur.”¹⁴¹ Published collegiate and high school data demonstrated that athletes allowed to RTP on the same day as the injury may demonstrate neuropsychological deficits after injury that are not apparent on sideline examinations, and they are more likely to have delayed onset of symptoms.¹⁴¹ Recently published concussion guidelines from the American Academy of Neurology (2013) concur with the above 4th International Conference consensus statement.¹³⁵ For grade II and III concussion with any LOC, the player is removed from the game on a fracture board if he/she cannot be cleared of an additional neck injury and is taken to a medical facility to be evaluated by a neurosurgeon, neurologist, or sports-minded physician.

A number of new technological platforms exist to assess concussion: quantitative EEG, sensory evoked potentials, eye-tracking technology, virtual reality tasking, functional and advanced neuroimaging, and head impact accelerometers. “At this stage only limited evidence exists for their role in this setting and none have been validated as diagnostic. It will be important to reconsider the role of these technologies once evidence is developed.”¹⁴¹

The consensus statement published in 2013 felt that risk strategies for concussion must be sport-specific, well-designed studies.

Rule changes in specific sports should always be considered. For example, in soccer, “research studies demonstrated that upper limb to head contact in heading contests accounted for approximately 50% of concussions.”¹⁴¹

Helmets in professional American football need to protect from impacts resulting in a head change in velocity of up to 10 m/sec, and up to 7 m/sec in professional Australian football (soccer). It also appears that helmets must be capable of reducing head-resultant linear acceleration to below 50 g and angular acceleration components to below 1500 rad/s² to optimize their effectiveness.¹⁴⁰

There is a strong need for studies evaluating the effects of a resting period, pharmacological interventions, rehabilitation techniques, and exercise in individuals who have sustained a sports-related concussion. Persistent symptoms (>10 days) are found in 10% to 15% of individuals with concussions and may be higher in certain sports (eg, elite ice hockey) and populations (eg, children, females).¹⁴¹

Finally, in regard to CTE, it was agreed that it represents a distinct tauopathy with an unknown incidence in athletic populations and that CTE was not related to concussions alone or simply exposure to contact sports.

At present there is no published epidemiological, cohort or prospective studies relating to modern CTE... It is not possible to determine the causality or risk factors with any

certainty...speculation that repeated concussion or subconcussive impacts cause CTE remains unproven. The extent to which age-related changes, psychiatric or mental health illness, alcohol/drug use or co-existing medical or dementing illnesses contribute to this process is largely unaccounted for in the published literature.¹⁴¹

They recommend that interpretation of causation in modern CTE case studies should “proceed cautiously...and it is important to address the fears of parents/athletes from media pressure related to the possibility of CTE.”¹⁴¹

The Future

Lessons learned from the history of concussion may foretell the future direction. Concussion remains a complex and incompletely understood form of brain injury. In American football, boxing, ice hockey, rugby, soccer, and many other sports and recreational activities, recent attention has appropriately been placed on the problem of concussion. Helmets cannot prevent all concussions, although their adoption is becoming more widespread and signifies realistic concern for brain injury. The International Consensus statements (above) have greatly aided our treatment decisions and decisions on when a player can safely return to a contact sport, especially after a complicated concussion with prolonged posttraumatic symptoms, lessening the risk of the secondary impact syndrome. Neurosurgeons have recently voiced concern about the emerging concepts of subclinical cranial impacts or subconcussion, as well as the possible cumulative effect of thousands of cranial impacts over several seasons or a full college or professional career. It also remains unclear why certain athletes are more predisposed to concussion than others. This year, reports in the neurosurgical literature suggest that advanced brain magnetic resonance images taken before and after a single season of college hockey suggest the presence of scattered cerebral microhemorrhages and white matter injury in those who have experienced concussion.¹⁴²⁻¹⁴⁴ These results, if substantiated by additional studies, would tell us what many have suspected, that sports concussion not only includes a transient functional disturbance or physiological dysfunction but may also, in a minority of instances, include a structural pathological injury and more likely cumulative injury in predisposed individuals. Sports concussion can be expected to remain a challenge to athletes, parents of child athletes, and physicians concerned with their care. As neurosurgeons and leaders in the management of brain injuries, we continue our present efforts at injury investigation, mitigation of injury, focused research, and treatment development.

THE CONTRIBUTIONS OF CONTEMPORARY NEUROLOGICAL SURGEONS TO CONCUSSION AND SPORTS INJURY

The example set by a handful of neurosurgeons (Figures 14-19), and many more we do not have space to mention, has stimulated generations of neurosurgeons, neurologists, sports medicine

physicians, psychologists, basic neuroscientists, engineers, athletic coaches, trainers, and others involved in the monitoring and mitigation of sports concussion. We extend our appreciation and thanks to our past, present, and future sports injury teachers and role models.

A podcast associated with this article can be accessed online (<http://links.lww.com/NEU/A664>).

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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