

Heading in Soccer: Integral Skill or Grounds for Cognitive Dysfunction?

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Objective: To critically review the literature concerning the effect of purposeful heading of a soccer ball and head injuries on reported cognitive dysfunction in soccer players.

Data Sources: We searched MEDLINE (1965–2001) and SPORTDiscus (1975–2001) for refereed articles in English combining key words for soccer (eg, *soccer, football, association football*) with key words for head injuries (eg, *concussion, head injury*). In addition, literature on cognition and head injuries was obtained. We reviewed reference lists of current literature for pertinent citations that might not have been found in the search procedures.

Data Synthesis: The fact that soccer players (and other athletes) have selected cognitive deficits is not questioned, and the popular press is quick to publicize results of questionable

validity. The reasons for such deficits are many. Much of the early data implied that heading was the culprit; however, subsequent research has suggested that other interpretations and factors may be potential explanations for these deficits. The current focus is on concussions, a known factor in cognitive dysfunction and a common head injury in soccer.

Conclusions/Recommendations: It is difficult to blame purposeful heading for the reported cognitive deficits when actual heading exposure and details of the nature of head-ball impact are unknown. Concussions are a common head injury in soccer (mostly from head-head or head-ground impact) and a factor in cognitive deficits and are probably the mechanism of the reported dysfunction.

Key Words: concussion, mild traumatic brain injury

Soccer is one of the oldest games. References to “kick ball” can be found as far back as 200 BC in China and around 4 BC in Greece. Although various versions of games that used a ball propelled by the feet emerged throughout the centuries, the first set of formal rules was set down by Cambridge University in 1848. The Football Association of England was founded in 1863 and further defined the rules of the game. The international governing body, Federation Internationale du Football, was founded in 1904 and today has more member nations than the United Nations.

Soccer is unique among sports because of the purposeful use of the unprotected head to control and advance the ball. Obviously, this skill places the head in a vulnerable position for injury. The recent death of Algeria's top scorer, Hocine Gacemi, from complications of a skull fracture after he “crashed head first into the ground after a clash of heads,”¹ along with parental questions on the safety of heading have brought this skill to the attention of the media. Also, the use of helmets in soccer has been debated.² The US Consumer Product Safety Commission met in May 2000 to discuss head injuries and protective equipment.

From attempted heading of the ball to falls, soccer provides many opportunities for head contact with the ball, ground, opponent, goalposts, and off-the-field objects. In this brief review, we will examine the available data on head injuries in soccer and their mechanisms and consequences. Much is still to be learned, and the critical reader is asked to carefully examine the data on cognitive deficits in soccer players. Are

these deficits due to purposeful heading of the ball, or are the deficits a result of mild traumatic brain injury from head impacts other than routine heading of the ball?

MECHANICS OF IMPACT

The mechanics of impact are based on the relationship $F = ma$, where F is the force of impact, m is the mass of the object, and a is the acceleration of the object at any instant in time. Soccer balls come in 3 sizes and weights (Table 1). Before the 1970s, the ball was leather and could absorb considerable amounts of water when used on wet ground. In the mid 1970s, the leather ball was modified with a water-resistant coating, but it could still gain mass from water. The modern ball is synthetic and resistant to water absorption. When interpreting the literature, it is important to pay attention to the probable dates when soccer players might have competed using a leather ball.

Nearly every professional article and media presentation mentions that a kicked ball can travel more than 100 km/h. The implication is that these ball velocities occur across ages and both sexes. Yet, players rarely head that kind of shot voluntarily, even though accidental impacts are possible. The highest-velocity ball a player might voluntarily head would be from a punt (approximately 70 km/h), drop kick (approximately 85 km/h), or goal kick (also approximately 85 km/h). Maximum ball velocities according to age and sex have not been reported. Levensky et al³ suggested that most oppor-

Table 1. Dimensions of the Soccer Ball

	Size 3	Size 4	Size 5
Mass, g (oz)	312–340 (11–12)	312–369 (11–13)	396–453 (14–16)
Circumference, cm (in)	56–59 (23–24)	61–64 (24–25)	68–71 (27–28)
Age group, y	6–9	10–13	≥14

tunities for heading occur at ball velocities less than 65 km/h. A ball traveling 65 km/h hits a solid object for 10.23 milliseconds at a force of 850 to 912 N with an acceleration of 30 to 55 *g* (gravitational) forces.^{2,4} In American football, impacts last for 200 to 350 milliseconds at *g* forces ranging from 150 to 450. In boxing, impact duration ranges from 14 to 18 milliseconds at 6000 N and *g* forces of 100. Schneider et al⁵ estimated that the necessary concussive forces are about 22 N·s⁻¹. The estimated impact of a soccer ball (at 64.37 km/h) is between 12.4 and 13.7 N·s⁻¹, well below concussive levels.³

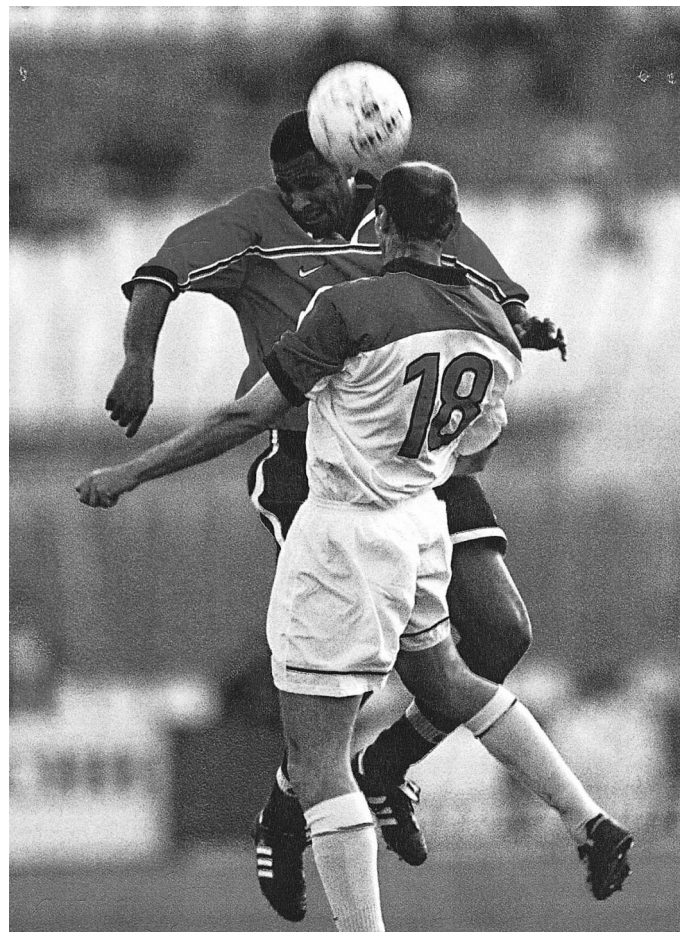
The head can withstand greater linear forces, whereas rotational impacts cause the most injury.⁶ Purposeful heading is largely linear. Boxing impacts are both linear (from “jabs”) and rotational (from “hooks”). In soccer, head injury from ball contact is mostly due to rotational impacts from accidental contact of the ball to the head. Head-ball contacts on the side of the head of an unprepared player can obviously lead to rotational impacts. Preparation of the head cannot be understated: strike an unprepared player on the forehead, and linear forces (pushing the head posteriorly) can be coupled with transverse rotation (as in a whiplash injury).

MECHANICS OF HEADING

Heading a soccer ball is a difficult task, which is further complicated by having to perform the skill during other activities (eg, standing, walking, running forward or backward, jumping, being challenged by an opponent, diving). Although coaching books describe teaching progressions, most do not discuss heading until the players are about 12 years or older. Younger players spend a modest amount of time practicing heading and even less time heading a ball in a game (making it more of a novelty than a polished skill). In youngsters, most heading opportunities occur after the ball has bounced. Any opportunity for heading requires the use of an age-appropriate ball (Table 1).

The ball is to be contacted on the forehead at or near the hairline (a coach will say to hit the ball on the forehead, where you put your hand to feel for a fever). Heading is active. Again, coaches will say “hit the ball, don’t let the ball hit you.” The trunk is hyperextended and the chin tucked in to the chest. The more trunk extension, the faster the forward velocity of the trunk and the harder the impact on the ball. The arms are usually extended for balance and protection. When the athlete jumps for a head ball, the legs are somewhat extended at the hip and flexed at the knee in preparation for striking the ball. Most of these positions are seen when 2 players compete for a head ball (Figure).

Successful heading is an exercise in precision timing. Flexing the hips brings the trunk and head toward the ball, while the arms are pulled back. Contracting the neck muscles fixes the head, making a firm surface for contact and effectively increasing mass of the contact surface. To contact the ball at the top of a jump (vertical velocity = 0), the trunk must be



When 2 players compete for a head ball, the arms and legs are extended and the knees are flexed (photograph courtesy of U.S. Soccer).

flexed to transfer force to the ball. Most ball-related injuries are due to the ball hitting an unprepared head (ie, the head and neck are not stabilized). This increases the ball mass-contact mass ratio and increases the risk of injury,⁷ because the force of the ball hitting the head can accelerate the head backward.

MECHANISMS OF HEAD INJURY IN SOCCER

Limited work is available on the mechanisms of head injury in soccer, and no reliable and valid statements exist on heading exposure in any level of soccer other than European (mostly English) professionals, where there is an average of 6 to 7 headers per game.⁸ The number of headers occurring during training is largely unknown.

Boden et al⁹ studied all Atlantic Coast Conference soccer teams for 2 years to determine concussion incidence. The most frequent mechanism of injury was head-to-head contact, followed by head to ground and then head to other body parts (eg, foot, knee, elbow). Importantly, purposeful heading was never a mechanism of injury, but injuries did occur when the player was accidentally struck by the ball. Although not discussed by Boden et al,⁹ a concussion is possible without contact. If the impulse of contact is strong enough to cause significant rotation of the head, mild traumatic brain injury is possible.

The experienced coach or observer of the game can direct

us to important factors in heading and head injuries. The penalty area (where players compete for a cross-corner kick) and the collision of an onrushing forward and goalkeeper are the most likely circumstances for a head injury. Goalpost collisions are possible but not common. Near the midfield line, where players compete for airballs (eg, punts, goal kicks), is another troublesome place on the field.

RATES OF HEAD INJURY IN SOCCER

Head injuries have been reported to account for 4% to 20% of all injuries in soccer.¹⁰ These statistics include nasal fractures, contusions, lacerations, concussions, and eye injuries. It is important to separate out the concussive injuries for this discussion. The added difficulty of grading a concussion is dealt with elsewhere in this issue.

Minimal published data are available on the frequency of head injuries in youths (12 to 14 years and younger). Powell and Barber-Foss¹¹ reported that mild traumatic brain injuries accounted for 3.9% and 4.3% of all injuries in boys' and girls' scholastic soccer, respectively. These injuries almost always occurred during games, and 80% or more of the injured athletes sat out for 1 week or less (median, 3 days). Powell and Barber-Foss¹¹ estimated 0.22 and 0.26 mild traumatic brain injuries per soccer team per season for boys and girls, respectively. Our ongoing survey of high-level youth soccer players (ages 12 to 18 years) in North Carolina shows that about 15% of all injuries were to the head (all head injuries, not solely concussions) and were due to player-player or player-ground contact. The survey of Boden et al⁹ of college-aged players (18 to 22 years old) demonstrated that each team could expect about 1 concussion per season. The reported concussions were largely game related and not due to purposeful heading of the ball. Barnes et al¹² surveyed soccer participants (n = 137) at a US Olympic Sports Festival. More than half the men and one third of the women had a concussion history. In comparison, Gerberich et al¹³ noted that 19% of secondary school football players had a concussion history, yet just fewer than half the Canadian Football League players (in 1997) reported a concussion.¹⁴ Powell and Barber-Foss's¹¹ survey of secondary school sports indicated that mild traumatic brain injury accounted for 7.3% of all injuries in football, and each football team could expect nearly 2 concussions a season.

One fact does appear evident: the higher and more competitive the play, the more frequent the incidence of concussion. More research at the recreational and professional levels, using many leagues and a consistent definition of concussion, is needed.

NEUROLOGIC AND NEUROPSYCHOLOGICAL FINDINGS IN SOCCER PLAYERS

The Norwegian Studies

The earliest description of heading injury was by Matthews,¹⁵ who diagnosed "footballer's migraine" in the days when a wet ball could increase in mass by 20%. Tysvaer et al,¹⁶⁻²⁰ who examined neurologic and neuropsychological sequelae in active and retired soccer players, directed the first projects on neurologic deficiencies in soccer players.

Their first report¹⁷ was a 1975 survey sent to 192 players in the 12 first-division Norwegian professional league teams. The subjects had a mean age of 25 years and had participated

in an average of 100 professional games, with nearly half having international experience. A total of 128 players met the inclusion criteria. Of those, 77 had full neurologic examinations; half of the players reported symptoms related to heading. The primary symptoms were disorientation, headache, and nausea. Seven players were rendered unconscious, and 3 were hospitalized. The mechanisms of head injury in this study were not reported. Given the publication date and the age and experience of these players, one would have to assume that many played in an era when water retention by the ball was a factor in impact.

Tysvaer et al^{16,18,19} published 3 articles in 1989 on neurologic and electroencephalographic (EEG) data on active and retired soccer players. The 69 active professional players (15 to 35 years of age and 128 professional games) were matched with a nonathletic, age-appropriate control group. All players with a history of head injury (4 concussions) reported acute symptoms. Eight players had persistent symptoms, and 2 had permanent complaints, but the neurologic examination results were normal in all players. Normal EEGs were obtained in 65% of the players and 87% of controls ($P < .001$). In addition, EEG abnormalities were most often detected in the youngest players. Forty-four percent of the players younger than 25 years had either a "slightly abnormal" or "abnormal" EEG, whereas 26% of the players older than 25 years had similar findings.

The 44 retired players (age range, 35 to 64 years; average length of retirement, 14 years) were compared with 37 men "in the same age range."¹⁸ The EEGs were read in a blinded manner. Seventy percent of the retired players had a history of head trauma (5 concussions). The neurologist's interviews with the players revealed prolonged symptoms from heading in 11 players, and 8 had permanent complaints. A variety of neurologic symptoms were reported in the interviews (nystagmus, n = 5; impaired coordination, n = 2; reflex disturbance, n = 1; impaired hearing, n = 2; unsteadiness, n = 1; reduced cervical spine motion, n = 15). No statistical differences were found in EEG readings for players with and without complaints or for players categorized as "headers" and "nonheaders." "No clear differences" were noted when the different age groups of players were compared with the control subjects. Unlike the previous study, age was not a factor in EEG disturbances.

Anatomical evidence of brain damage in a subset of the retired players was assessed by computed tomography¹⁶ in 33 of the retired professional players (age range, 39 to 60 years; range of retirement, 8 to 39 years). No control group was studied, which makes blinding a concern. The scans were read by one of the authors. Widened ventricles were found in 27% of the players assessed, indicative of central cerebral atrophy, and 18% had cortical atrophy. Players identified as "headers" had a significantly higher frequency of cortical atrophy. Only 7 players had a history of head injuries (with unconsciousness or amnesia), and they had no greater frequency of atrophy than those without a history of head injury. These findings of atrophy may or may not be associated with either reversible (eg, steroid therapy or starvation) or irreversible (eg, brain injury or aging) decline in intellectual function.

Finally, neuropsychological evaluation of these same retired players was reported in 1991.²⁰ The players were administered the Wechsler Adult Intelligence Scale, the Trail-Making Test Parts A and B, a modification of the Malstead-Wepman-Reitan aphasia screening test, tests of sensory-perceptual functions,

Table 2. The CAGE* Questionnaire of Alcohol Abuse

1. Have you ever *CUT* down on your drinking?
2. Have people *ANNOYED* you by criticizing your drinking?
3. Have you ever felt badly or *GUILTY* about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (ie, an *EYE OPENER*)?

*Acronym taken from the capitalized word in each question. Reprinted with permission from the *Journal of the American Medical Association* (1984;252:1905–1907). Copyright 1984, American Medical Association.

motor tests, tests of hemisphere dominance, and the Benton Visual Retention Test form C. For comparison, the authors chose 20 hospitalized patients with no history of head or neck injuries or evidence of brain damage. Group differences were most obvious in the Trail-Making Tests ($P < .01$). Impairment was severe to gross in 1 player, severe in 3 players, moderate in 14, mild in 12, and none in 7 compared with controls. Impairment was unrelated to subject age. Forty percent of the controls demonstrated mild impairment. The scores for nearly one third of the soccer players were low enough (both parts of the Trail-Making Test) to suggest physical damage to the brain. Throughout the full series of studies, the focus was on heading as a contributor to the findings.

Other Studies

The Norwegian studies brought attention to the problem and encouraged others to work in this area. Haglund and Eriksson²¹ compared amateur boxers with active or former professional soccer players (25 to 44 years of age) and track athletes. After clinical studies (medical and neurologic studies), personality trait assessment (Karolinska Scales of Personality), magnetic resonance imaging (MRI), and neurophysiologic and neuropsychological examinations, they “failed to demonstrate any signs of chronic brain damage in soccer players who were known to be frequent headers.” On one neuropsychological test (finger tapping), performance by the soccer players was lower than other athletes, but these results were still within normal limits.

Jordan et al²² compared the 20 players on the US men’s 1994 World Cup team with 20 track athletes and focused on symptoms of previous head injury and on MRI. Players were categorized as low- or high-frequency headers (player’s opinion), and the authors attempted to form a heading exposure index based on years of experience and level and location of competition. Alcohol abuse is a known factor in organic and neurologic brain damage, and, therefore, each player was screened with the CAGE scale for alcohol use (Table 2).^{23,24} No differences in symptoms were found between the 2 groups. Further, they “found no evidence that this exposure correlated with symptoms that might be attributed to chronic encephalopathy. In addition, increased heading exposure did not correlate with abnormalities on brain MRIs.” Symptoms were correlated with history of head injury, not with any playing parameter or their index of heading exposure.

Recently, Dutch researchers²⁵ performed comprehensive neurologic assessments on amateur and professional soccer players while controlling for heading exposure, prior head injury, and history of alcohol intake. “Regional-level” amateurs were compared with a control group of swimming and track athletes. These amateur players showed impaired planning ability ($P < .001$, Wisconsin Card-Sorting Task, Complex Figure Test), memory ($P < .004$, Complex Figure Tests, Wechsler

Memory Scale, 15-Word Learning Test), and motor ability ($P < .01$, fine motor behavior, nondominant hand puncture test). The 53 active professional soccer players were compared with a similar control group. The professional players scored lower on memory tests, planning, and visuosperceptual processing. Deficits in both studies were correlated with the head injury history. Matser et al²⁵ stated that “concussions incurred in amateur soccer may play a fundamental role in the development of cognitive impairment observed in these (soccer) players” and continued with “the neurological effects of amateur soccer appear to be related to soccer-related concussions caused by contact trauma.”

Matser et al^{25,26} grouped their subjects into controls and players. The player group actually contained 2 subgroups: players with a history of head injury and players without a history of head injury. Had the authors separated out these subgroups, some interesting findings would have been possible. If the results for the uninjured player group were similar to the control group, then purposeful heading would not have appeared to be a factor because they had a long history in the game without measurable deficits. However, if the uninjured player group was similar to the group with head injury history, then purposeful heading might indeed be a factor in the deficits. Grouping collegiate players this way does not support purposeful heading as a factor in cognitive deficits (K. M. Guskiewicz, unpublished data, 2000).

Short-term exposure to heading was studied in a prospective project on collegiate players.²⁷ One hundred male and female athletes were administered neuropsychological tests before and after 2 training sessions (1 with heading and 1 without heading). The tests included the Alphabet Backwards Test, Trail-Making Tests (Parts A and B), Stroop Color Word Test, and VIGIL/W. No significant differences were noted on any test when comparing the control (no-heading) condition with the experimental (heading) condition.

The parent of the very young soccer player should be relieved by the comments of Bijur et al,²⁸ who concluded, “there is no evidence. . . that multiple head injuries between birth and age 10 years have a deleterious effect on a global measure of intelligence or on academic achievement measured at 10 years of age.” If they showed no deficits after many concussions, then deficits from infrequent subconcussive events, such as the occasional heading by the youngest of players, are unlikely. However, more longitudinal work is necessary in this area. Differences in the findings on the young people²⁹ and adults^{16–20,25,26} may result from recall bias.

ASSIMILATING THE LITERATURE

The findings of the Norwegian studies are frequently cited as proof that heading is the cause of cognitive deficiencies. Other European and American investigators who mentioned decrements were not able to say that purposeful heading was the cause. Of interest is the low frequency of concussion in the Norwegian studies. According to Barnes et al¹² and Boden et al,⁹ the number of concussions seems quite small. Were there really so few concussions, or were concussions underreported due to definition difficulties? Some methodologic problems and other confounding factors must be considered before we blame purposeful heading for any negative findings.

First, consider the inclusion criteria for the Norwegian studies, especially the studies of retired players. The Norwegian studies began in the early 1980s, so the players likely were

participating in the 1960s and 1970s, before the advent of the water-resistant ball. Most players of that era freely admit that heading a wet, heavy ball could lead to the reported symptoms.

Second, other causes of impaired cognitive function were not considered in the earlier studies. Alcohol abuse and malnutrition are known to lead to cognitive deficiencies.^{23,24} Simply mentioning alcohol intake is not a valid way to explore alcohol intake or abuse.

Third, a previous head injury is a well-known cause of cognitive dysfunction. In the Norwegian studies, players with non-game-related head injuries were excluded, but the game-related head injuries were not described. Consciousness or unconsciousness was mentioned; however, few other details about concussion history or other head injuries were mentioned. Barnes et al¹² noted that 54% of the 72 men surveyed had a history of a diagnosed concussion, and 33% had multiple concussions. The authors estimated 50% odds of a male athlete playing for 10 years at this level sustaining at least one concussion. These results were for high-level amateur players, but we might be safe in assuming that professionals are more aggressive and may experience concussions at least at the same frequency, perhaps even more. Thus, concussion history is underreported in the Norwegian studies. Remember that the active professionals in the studies by Tysvaer et al¹⁶⁻²⁰ reported only 4 concussions in 69 players who averaged 128 professional games.

Part of the problem may be the definition of what constitutes a concussion. A common response by parents and players when questioned about a head injury is "there was no concussion—wasn't knocked out." Many physicians in Europe and South America believe concussions are not a problem, implying that concussions might be underdiagnosed.

Unconsciousness is not a requirement for cognitive dysfunction. Lovell et al³⁰ showed that loss of consciousness (LOC) was a poor predictor of cognitive deficits in patients with mild head trauma. The patients who met their inclusion criteria were divided into 3 groups: no LOC, undetermined LOC, or documented LOC. All patients with mild head injury showed "mildly decreased performance" on a variety of standard neuropsychological assessments, but LOC was not a factor in cognitive performance, because all 3 patient groups performed similarly. In addition, the effects of mild concussion can last well beyond the time when a patient might return to his or her regular activities. Macciocchi et al³¹ showed that concussed collegiate American football players improved during the succeeding 10 days but still were not fully recovered. In addition, more than half of the patients studied by Rutherford³² were still symptomatic up to 6 weeks after injury. Hugenholtz et al³³ showed that cognitive deficits from a "mild concussion" (grades 1 and 2) could still be documented up to 3 months after injury. A report by Rutherford et al³⁴ showed that nearly 15% of patients with "mild concussion" had symptoms for 1 year after injury. To ignore concussion history in soccer when reporting cognitive deficits could well bias conclusions and lead to the assumption that the neuropsychological deficits were due to purposeful heading.

A possible fourth factor is learning disabilities.³⁵ Children with dyslexia³⁵ and attention-deficit/hyperactivity disorders²⁹ tested poorly when compared with healthy controls. College students with learning disorders or mild brain injury performed below healthy students.³⁶

Another factor is that the overwhelming majority of research on mild traumatic brain injury and soccer is cross-sectional.

A longitudinal design with careful control of confounding factors would probably answer many of the questions raised by both the public and the medical community.

It is obvious that there are many factors to consider before attributing diminished cognitive function in soccer players to the subconcussive impacts of purposeful heading. The clinical significance of any reported dysfunction has yet to be clarified. Even though there are documented deficits, whether these deficits have any effect on activities of daily living is unknown.

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