Consequences of heading the ball in football

Consecuencias del golpeo de balón con la cabeza en el fútbol

Alberto López Moreno

Medical Specialist in Physical Education and Sports Medicine. Doctor of Real Valladolid Club de Fútbol.

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Introduction

Blows to the head pose a risk in sports and in particular in football. They can cause potentially life-threatening injuries. Many authors have long considered the number and consequences of head injuries to be underestimated[1,2]. Recent studies have raised concerns about injuries caused by repeatedly heading the ball in the long term[3].

Consequences of heading the ball in football

Immediate injuries: diffuse brain injury

Brain laceration

Decreased responsiveness occurs as an expression of alteration of the reticular formation. It is treated by trying to reduce the oedema: adequate ventilation and dexamethasone in high doses (50 mg/day). Urea and mannitol have also been used, monitoring the rebound effect.

Expanding contusion focus

This is characterised by an area of necrosis around which multiple haemorrhagic foci accumulate and, surrounding everything, a large area infarcted by vasodilation with stasis and anoxia which triggers a massive oedema in a few hours. This is the so-called pulping that Botterell describes.

Fibrillary rupture

This is the traumatic sectioning of fibre bundles which appears as a pure lesion, without bruising or associated haemorrhage. Extensive and diffuse location.

Cerebral concussion

The classical definition of concussion is a disorder of brain functions caused by trauma and characterised by a short loss of consciousness (minutes to a few hours). It is more a clinical than a pathological concept: brief loss of consciousness of traumatic aetiology.

In 1966, the *Committee of Head Injury Nomenclature of the Congress of Neurological Surgeons* defined concussion as "a clinical syndrome characterized by immediate and transient posttraumatic impairment of neural functions, such as alteration of consciousness, disturbance of vision or equilibrium".

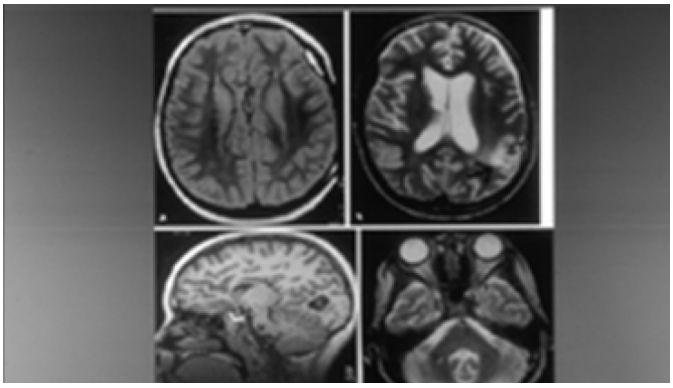
The genesis of cerebral concussion required a consensus document for the different degrees of injury, criteria for returning to competition, and level of severity[5,6].

Concussion is the most common diffuse brain damage in TBI[7]. It represents a spectrum of brain damage which is classified as follows:

- *Mild concussion*. These are very common injuries and sometimes go unnoticed. They involve confusion and disorientation without amnesia. It is a reversible syndrome without major sequelae.
- *Classical concussion*. This is an injury which involves a loss of consciousness, and is transient and reversible. It is accompanied by post-traumatic amnesia and the duration of this indicates the severity of the damage. There exists a post-concussion syndrome, which includes memory impairment, dizziness, nausea, anosmia and depression, and can be very disabling (Figure 1).
- *Diffuse axonal injury* (DAI) This is the term used to define prolonged post-traumatic coma which is not due to a lesion caused by the mass effect or ischaemic injury. Widespread damage to neuronal axons occurs. Those injured are often in a coma for long periods of time and are severely incapacitated if they survive. It is not easy

Correspondence: Alberto López Moreno
E-mail: alopezmoreno@hotmail.com

Figure 1. Diffuse axonal injury.



clinically to distinguish between DAI and hypoxic encephalopathy, and in fact they can coexist.

This classification of Concussion Severity Grades must be distinguished from the classification used to quantify the severity of neurological damage in TBIs (Glasgow Coma Scale)[8,9].

It is important to be extremely vigilant with these injuries, because sometimes the consequences can be catastrophic, as, for example, in the case of second impact syndrome[10].

In some cases, this type of injury leads to a loss of consciousness. This loss of consciousness implies inhibition of the Ascending Activating Reticular System (ARAS) and this leads to the tongue falling backwards, the cough and swallowing reflex disappearing, the inhalation of vomit and tracheobronchial oedema: a life-threatening situation due to obstruction of the airway[11].

Short-term injuries: delayed or focal

Extradural or epidural haematoma

This occurs when blood builds up between the outer layer of the dura mater and the skull. It is usually the result of tearing of the middle meningeal artery and affects the area irrigated by it: the temporoparietal area. The symptoms develop quickly with initial syncope, followed by a lucid period and renewed loss of consciousness. It requires immediate CT action to locate and drain the haematoma.

Subdural haematoma

This involves the accumulation of blood in the virtual space beneath the dura mater. The clinical evolution is slower because it is caused by venous bleeding. Those affected normally fall into a deep coma with clear signs of focality: anisocoria, hemiparesis, facial involvement and seizures.

Intracerebral or intraparenchymal contusions and haematomas

Simple brain contusions are relatively common. The frequency of this diagnosis has increased with the number of tomographs. These are usually associated with subdural haematomas. Contusions evolve and intracerebral haematomas are formed, which is a compact collection of blood located in the cerebral parenchyma.

Subarachnoid haemorrhage

Passage of blood to the subarachnoid space. Patients present with stiffness of the neck, headaches, photophobia, vomiting, hyperthermia, irritability. Diagnosis is by lumbar puncture and CT scan. The outcome is generally favourable. Hydrocephalus is a potential complication.

Cerebral oedema

Increased brain mass at the expense of:

- extracellular space (true oedema);
- the cells themselves (brain swelling);
- the increase in volume of the vascular bed (vasogenic oedema).

Treatment consists of proper ventilation, dexamethasone and diuretics (acetolamide, furosemide).

Brain congestion

This is the abnormal accumulation of blood in the cerebral vascular bed, producing paralytic vasodilation. The intimate mechanism is not known, but acidifying agents: CO_2, hypoxia and excess acidic metabolites cause massive vasodilation. In TBI, these factors are very frequently linked to respiratory complications. Treatment is similar to that for cerebral oedema: maintain proper respiratory function, hyperventilation, fluid retention, osmotic diuretics, glucocorticoids, hypothermia, barbiturates and surgical decompression.

Long-term Injuries: Chronic brain damage from repeated blows to the head: CTE (Chronic traumatic encephalopathy)

Repeated concussions and repeatedly heading the ball appears to be a risk factor for mental health and cognitive impairment[12-14]. Further studies are needed to understand traumatic conditions, such as traumatic encephalopathy and other neurological diseases, and the extent to which they are related to repetitive neurotrauma, such as repeatedly heading a football[15].

A study by Norwegian researchers on electroencephalograms (EEGs) reported abnormalities and a higher incidence of brain atrophy in both active and retired footballers compared to people who do not actively play football, finding cognitive and emotional symptoms typical of post-concussion syndrome[16,17]. However, fewer abnormalities were found in the EEGs of those football players more accustomed to heading the ball, due to their better technical adaptation when heading and strengthening of the neck musculature[18,19].

The results of most of Kirkendall's studies[12,20] showed that the electroencephalograms performed on professional footballers playing in the Norwegian first division revealed dysfunctions. Retired players exhibited abnormalities including reduced cortical tissue and increased lateral ventricles[21]. Another study by Matser *et al.*[22] reinforces the idea

that playing professional football has adverse effects on certain aspects of cognitive functions, indicating that most medical researchers seem to accept the fact[23]. Mc Crory[24] does not back up this idea: he admits the possibility that head-to-head impact may leave sequelae but is more sceptical about chronic damage being caused by repeatedly heading the ball.

EEG abnormalities were much more evident in younger soccer players and could be attributed to neuronal injury to a still-developing brain, caused by subconcussive injuries[25-27]. Meanwhile, in studies which included cranial imaging tests with computed and magnetic tomography, resonance showed no reliable correlation between post-contusion or post-concussion symptoms and signs of possible encephalopathy[28].

Other studies conclude that the cumulative effects of repeated heading in football are still not fully known despite computer simulations[29].

A recent study by the University of Glasgow (Scotland) has become a focus of social attention due to the conclusions it reached from its results. The research stemmed from a request from the English Football Association and the PFA (Professional Footballers' Association) given concerns regarding a link between brain diseases and players heading the ball. The study confirmed that the ex-professional football players group was three and a half times more likely to suffer from dementia, with greater neurodegenerative disease mortality than the control group, and that mortality from other common diseases was lower among Scottish ex-professional football players than in the control group[30].

CTE (Chronic traumatic encephalopathy)

This is a form of brain degeneration usually caused by repeated head trauma which is normally diagnosed at autopsy by studying sections of the brain. Although it is not an immediate consequence of TBIs, it has a complex relationship with them[31], specifically with:

- post-concussion syndrome.
- second impact syndrome.

Experts are still investigating how repeated head trauma, the number of head injuries and their severity, and other factors may contribute to changes in the brain which lead to chronic traumatic encephalopathy[32].

Chronic traumatic encephalopathy is not diagnosed during life, except in rare cases of individuals with high-risk exposure. The frequency of chronic traumatic encephalopathy in the population is also unknown and the relationship between sports and the development of CTE and neurodegenerative diseases is unclear. Should such a connection exist, it would be necessary to identify the nature and strength of that relationship[33].

According to the provisional consensus guidelines published in 2016[34], CTE is characterised by the abnormal accumulation of hyperphosphorylated tau protein (p-tau) within neurons, astrocytes, and cellular processes around small blood vessels in an irregular pattern, deep in cortical grooves. For identification of the p-tau protein using autopsy tissue, it is necessary to use immunohistochemical stains. P-tau protein accumulations are also identified in conditions secondary to other pathologies, such as Alzheimer's disease, Down syndrome, prion diseases, post-encephalitic parkinsonism and Niemann-Pick type C. P-tau protein is almost universally detected in autopsies performed on elderly patients. Although work is in progress[35], we do not at present have criteria to diagnose CTE in a living person. Consequently, the incidence of CTE in the general population is unknown[36,37].

No symptoms have been specifically related to chronic traumatic encephalopathy, because the possible signs and symptoms are commonly identified in many other conditions as well. In the few cases in which it has been possible to diagnose chronic traumatic encephalopathy, symptoms have included[38]: difficulty thinking (cognitive impairment), impulsive behaviour, depression or apathy, short-term memory loss, difficulty planning and performing tasks (executive function), emotional instability, substance abuse and suicidal thoughts or behaviours. The symptoms of chronic traumatic encephalopathy do not develop immediately after head trauma but appear years or decades after repeated head trauma.

There is no treatment for chronic traumatic encephalopathy. However, since it appears to be associated with a history of recurrent head trauma, the current recommendation to prevent it is to reduce mild traumatic brain injuries. We know that patients who have sustained concussion are more likely to develop another neurodegenerative disorder, so avoiding mild head trauma would prevent other post-concussive injuries[39-41].

Bibliography

1. Junge A, Dvorak J, Graf-Baumann T. Football Injuries During the World Cup 2002. A*m J Sports Med.* 2004;32:23-27.
2. Fuller CW, Smith GL, Junge A, Dvorak J. An Assessment of Player Error as an Injury Causation Factor in International Football. *Am J Sports Med*. 2004;32:28-35.
3. Head Injury in Soccer: From Science to the Field; summary of the head injury summit held in April 2017 in New York City, New YorkPutukian M, Echemendia RJ, Chiampas G, *et al. Br J Sports Med.* 2019;53:1332.
4. Committee of Head Injury Nomenclature of the Congress of Neurological Surgeons: Glossary of head head injury, incluiding some definitions of injury to the cervical spine. *Clin Neurosurg*. 1966;12:286-294.
5. Cantu RC. Return to play guidelines after a head injury. En: Neurological Athletic head and neck injuries. Philadelphia: *Clin Sports Med*. 1998;17(1):45-60.
6. Concussion is Treatable: Statements of Agreement from the Targeted Evaluation and Active Management (TEAM) Approaches to Treating Concussion Meeting held in Pittsburgh, October 15–16, 2015. Michael W. Collins, Anthony P. Kontos, David O. Okonkwo, Jon Almquist, Julian Bailes, Mark Barisa, Jeffrey Bazarian, O. Josh Bloom, *et al. Neurosurgery.* Author manuscript; available in PMC 2017 Dec 1. Published in final edited form as: *Neurosurgery*. 2016 Dec;79(6):912–929.
7. López A. Cranifacial Traumas (CFT) in football. En: 3rd European Congress of Sport Traumatology; Madrid, April 1-3, 2004. Madrid: EFOST;2004. p. 47.
8. Drent E, *et al. Programa Avanzado de Apoyo Vital en Trauma para Médicos* (ATLS). 6ª ed. Chicago, Illinois. American College of Surgeon Committee Trauma.1997.
9. Marion DW, Carlier PM. Problems with the initial Glasgow Coma Scale assessment caused by prehospital treatment of patients with head injuries: results of a national survey. *Journal of Trauma*. 1994;36(1):89-95.
10. Cantu RC. Second-Impact Syndrome. En: Neurological Athletic head and neck injuries. Philadelphia: *Clin Sports Med.* 1998;17(1):37-44.
11. Barberá J. Traumatismos Craneoencefálicos. En: Balibrea JC (dir). *Tratado de Cirugía* Vol. 1. Madrid: Marbán; 1994. p. 775-804.

12. Kirkendall DT, Jordan SE, Garrett WE. Heading and Head Injuries in soccer. *Sports Med.* 2001;31(5):369-86.
13. Tysvaer AT. Head and neck injuries in soccer: impact of minor trauma. *Sports Med.* 1992;14(3):200-13.
14. Kirkendall DT, Garrett Jr WE. Heading in soccer: integral skill grands for cognitive disfunction?. *Journal of Athl Training Dallas.* 2001;36(3):328-33.
15. Manley G, Gardner AJ, Schneider KJ, *et al.* A systematic review of potential long-term effects of sport-related concussion. *Br J Sports Med.* 2017;51:969–77.
16. Tysvaer AT, Storli OV. Soccer injuries to the brain. A neurologic and electroencephalographic study of former players. *Acta Neurol Scand.* 1989;80(2):151-6.
17. Pellman EJ, Powell JW, Viano DC, *et al.* Concussion in profesional football: epidemiological features of game injuries and review of the literature-part 3. *Neurosurgery.* 2004;54(1):81-96.
18. Sortland O, Tysvaer AT. Brain damage in former association football players. An evaluation by cerebral computed tomography. *Neuroradiology.* 1989;31(1):44-8.
19. Barnes BC, Cooper L, Kirkendall DT, McDermott TP, Jordan BD, Garrett WE Jr. Concussion history in elite male and female soccer players. *Am J Sports Med.* 1998;26(3):433-8. Doi: 10.1177/03635465980260031601
20. Kirkendall DT, Garrett Jr WE. Heading in soccer: integral skill grands for cognitive disfunction?. *Journal of Athl Training Dallas.* 2001;36(3):328-33.
21. Baroff GS. Is heading a soccer ball injurious to brain function?. *J Heda Trauma Rehabil.* 1998;13(2):45-52.
22. Matser J, Kessels AG, Jordan BD, Lezak MD, Troost J. Chronic traumatic brain injury in professional soccer players. *Neurology*. 1998;51(3):791-6.
23. Bailes JE, Cantu RC. Head injury in athletes. *Neurosurgery.* 2001;48(1):26-45.
24. Mc Crory PR. Brain injury and heading in soccer. *British Med Journal.* 2003;327:351-2.
25. Levy ML, Ozgur BM, Berry C, Aryan HE, Apuzzo ML. Analysis and evolution of head injury in football. *Neurosurgery*. 2004;55(3):649-55.
26. Bunc G, Ravnik J, Velnar T. May Heading in Soccer Result in Traumatic Brain Injury? A Review of Literature. *Med Arch.* 2017;71(5):356-9.
27. Viano DC, Casson IR, Pellman EJ. Concussion in profesional football: biomechanics of the struck player-part. *Neurosurgery.* 2007;61(2):313-28. doi:10.1227/01.
28. Straume-Naesheim TM, Andersen TE, Dvorak J, Bahr R. Effects of heading exposure and previous concussions on neuropsychological performance among Norwegian elite footballers. *Br J Sports Med*. 2005;39(1):70-7. doi:10.1136/bjsm.2005.019646
29. Naunheim RS, Standeven J, Bayly P. Cumulative effects of soccer heading are not fully known. *BMJ.* 2003;327:1168.
30. Mackay DF, Rusell ER, Stewart K, MacLean JA, Pell JP, Stewart W. Neurodegenerative Disease Mortality among Former Professional Soccer Players. *N Engl J Med.* 2019;381:1801-8. DOI:10.1056/NEJMoa1908483
31. Montenigro PH, Baugh CM, Daneshvar DH, *et al*. Clinical subtypes of chronic traumatic encephalopathy: literature review and proposed research diagnostic criteria for traumatic encephalopathy syndrome. *Alzheimers Res Ther.* 2014;6:68.
32. Ling H, Morris HV, Neal JW, Lees AJ, Hardy J, Holton JL, *et al*. Mixed pathologies including chronic traumatic encephalopathy account for dementia in retired association football (soccer) players. *Acta Neuropathol.* 2017;133:337–52.
33. McKee AC, Cantu RC, Nowinski CJ, *et al*. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. *J Neuropathol Exp Neurol.* 2009;68:709–35.
34. McCurry P, Meeuwisse WH, Dvorak J, *et al.* Consensus statement on concussion in sport: The 5th international conference on concussion in sport, Berlin, October 2016. *Bri J Sport Med.* 2017;51:838-47.
35. Bieniek KF, Ross OA, Cormier KA, *et al.* Chronic traumatic encephalopathy pathology in a neurodegenerative disorders brain bank. *Acta Neuropatholm.* 2015;130:877–89.
36. Grinberg LT, Anghinah R, Nascimento CF, *et al.* Chronic traumatic encephalopathy presenting as alzheimer's disease in a retired soccer player. *J Alzheimers Dis.* 2016;54:169–74.
37. Guthrie RM. Emerging data on the incidence of concussion infootball practice at all levels of amateur play. *Phys Sportsmed.* 2015;43(4):333-5. doi:10.1080/00913847.201.1081552.
38. Zuckerman SL, Brett BL, Jeckell A, *et al.* Chronic traumatic encephalopathy andneurodegeneration in contact sports and american football. *J Alzheimers Dis*. 8;66:37–55.
39. Schneider DK, Grandhi RK, Bansal P, *et al.* Current state of concussion prevention strategies: a systematic review and meta-analysis of prospective, controlled studies. *Br J Sports Med*. 2017;51:1473–82.
40. Kroshus E, Babkes Stellino M, Chrisman SPD, *et al.* Threat, pressure, and communication about concussion safety: implications for parent concussion education. *Health Educ Behav*. 2018;45:254–61.
41. Manley G, Gardner AJ, Schneider KJ, *et al.* A systematic review of potential long-term effects of sport-related concussion. *Br J Sports Med.* 2017;51:969–77.