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MONOGRAPH ON HEAD INJURY AND CONCUSSION IN SPORTS

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Return to training and play following concussion or traumatic brain injury

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The last chance. Bringing back training for the Sports Medicine specialisation

La última oportunidad. Recuperación de la formación de la especialidad de Medicina del Deporte

Sports Medicine Work Group*

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A long process began 17 years ago, in 2005, that we are convinced will soon end in applying the Royal Decree R.D. regulating cross-discipline training for Specialists (BOE 20 July 2022)[1]. This process originated from the European standard outlining training in all medical specialisations that must be completed during residency, full-time and in a hospital environment, replacing the training system using specific specialist colleges.

In Spain, the law on healthcare professions 44/2003[2] is set by the 2008 legislation to transform the specialisation, including the European Directive on Medical Specialisations.

This will not be easy. The conditions set by this standard are meticulous, demanding and require implication from anyone interested in giving this specialisation the prestige that we all think it deserves in our society.

The work has been relentless ever since. An enormous effort has been put into this eagerly-awaited recovery of the specialisation and, although not the subject of this editorial, it is essential to mention some significant moments in this process.

The Spanish Society of Sports Medicine has never shied away from its responsibility and has always spearheaded this claim and the tasks to put it into place. At the start, this was led by FEDAMEFYDE, working with the Specialisation Commission and in 2014, the Inter-regional Sports Medicine Commission was set up, comprising official representatives from the Autonomous Regions, AEPSAD and SEMED.

At several points, it seemed likely that the specialisation training was about to be brought back.

Minister Pajín stated, in the Congress plenary session on 13 April 2011[3], that "the physical education and sports medicine specialisations plus legal and forensic medicine, currently taken in the degree programme, will be reinforced by considering them as specialisations to be included in the MIR (Resident Medical Intern) system," although President Zapatero resigned four weeks later leaving no time to pass this through the Council of Ministers.

In 2014, President Rajoy's government approved the core subject decree for specialised training[4] which would have brought back training in our specialisation, although this was repealed a few weeks later and withdrawn by accepting a contentious-administrative appeal brought by the Spanish Immunology Society, according to the ruling of 12 December 2016, by the Supreme Court[5].

In this term of office, the Ministry of Health executive board run by the director general of Professional Regulation set up a technical operating group to determine access to the qualification for a specialist doctor in Sports Medicine. It comprised representatives from the Ministry of Health, AEPSAD, CSD, Autonomous Communities who promised to cover the costs of training Sports Medicine specialists (Catalonia, Andalusia, Castillas-León, Asturias and the Balearic Islands) and the Spanish Society of Sports Medicine that coordinated the group, with the mandate of drafting a training programme adapted to the European directive, a draft of the specific Royal Decree for Sports Medicine as for Legal and Forensic Medicine[6] and the corresponding budgeting.

In October 2020, with these tasks practically finalised, Dr Rodrigo Gutierrez, general director of Professional Regulation at the Ministry of Health, resigned and was replaced by Dr Viçenc Martínez. Several meetings were held with the latter and he seemed to want to reach a satisfactory solution, but then he also resigned.

Contact was renewed with the new general director of Professional Regulation at the Ministry of Health, Dr Celia Gómez, and it became clear that she did not value the work carried out so far. It was decided that the specialised training would be carried out via the Royal Decree that had been approved that year[1].

The Royal Decree determines a few directives that we would like to mention.

Application for the specialisation. The procedure to request a new Health Sciences specialist qualification could be initiated by one or several national scientific societies validly established in relation to the corresponding area of Health Sciences specialisation, that confirms the representation of at least sixty percent of professionals in this area or by the Human Resources Commission for the National Health System.

It is highly improbable that the Human Resources Commission of the National Health System would request the Sports Medicine specialisation so, in all likelihood, the application should be made by

the Spanish Society of Sports Medicine, the only entity that meets the requirements in the Royal Decree.

Representation. The standard determines that the scientific society must confirm representation of at least sixty percent of the professionals in that area. With a view to having to present nominal representations, a census of specialists has been drawn up and they were all sent a representation document so that the SEMED might apply for the speciality.

Specific nature. The R.D. determines that the specialisation must represent an extensive and differentiated field of practice in Health Sciences and develop an extensive set of contents and skills that are significantly different to those already included in other specialisations or qualifications in Health Sciences.

In this respect, the following content is considered to be specific and exclusive to the specialisation: competitive sport care (musculoskeletal system pathology related to the sport, prevention of injuries, strength tests and medical suitability check-ups) and doping, with significant exercise prescription content.

Financial viability. The specialisations must be financially feasible in the long term and guarantee practice by specialists.

Finalising the procedure. If the ruling goes against it, a new application cannot be presented for the same specialisation for five years following the date of this ruling.

The Spanish Society of Sports Medicine and the Sports Medicine Task Force are working together to present an application that meets all the requirements determined by the standard in force and they fully expect for this specialisation to be restored.

It is expected that these wishes will be echoed by the entire collective of specialist doctors and any that work in Sports Medicine who wish to take the specialisation, and it is understood that if the application is rejected, it would mean the de facto disappearance of the specialisation as a new application could not be presented for five years following this ministerial decision.

We are convinced that the doctors involved are going to pull together and work towards this common goal so that this opportunity is not lost.

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Traumatic head injuries. Introduction
Traumatismos craneoencefálicos. Introducción

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Traumatic brain injuries (TBI) in sports represent a severe health problem that affects athletes and everyone around them, including sports medicine specialists as TBIs represent the main causes of death and disability among players in some sporting disciplines (boxing, American football, rugby, ice hockey, lacrosse, wrestling, etc.). A traumatic brain injury affects the brain tissue by temporarily or permanently altering brain function. The patient's life depends on its diagnosis, evolution and outcome[1,2].

Sports-related cerebral concussion (CC) is defined as a mild traumatic brain injury that leads to complex deterioration of neurological function. It mainly happens as the consequence of direct or indirect impact to the head or neck when playing contact sports and, in general, it is usually transitory[3].

It often happens in contact sports although athletes' reporting of the injury is problematic because of severe repercussions from the health and safety protocol which clashes with the athletes' wish to carry on playing. As a result, many athletes do not report this type of injury when there is no clear clinical evidence. After suffering concussion, the chances of getting it again increase 2 to 4 times and it can occur with less impact[4].

CC is influenced by the athletes' kinanthropometry characteristics, age, having suffered previous concussions and it is also known that accumulative impacts on the head reduce the tolerance to this injury.

Sports-related concussion is characterised by presenting a transitory deterioration of brain function that includes losing consciousness in 8-20% of cases[5]. The short and long-term consequences of CC in sport vary according to the severity of the injury; evolution is usually favourable in most cases in the first 24-72 hours.

After a TBI, initial evaluation and triage must be based on clear guidelines (losing consciousness, neurological deficit, Glasgow Coma Scale and other signs of mental impairment) identifying patients that require immediately resuscitation, admission and observation, imaging diagnosis, emergency neurosurgery, etc. The sports concussion assessment tool (SCAT5) is very effective and one of the most widely-used to date; it can assess symptoms, level of consciousness, function, balance and detect possible signs of a severe brain injury[6,7]. On the other hand, cognitive evaluations based on digital approaches are objective tools with a great future in analysis of these injuries[3,8].

Clinical practice for CC is heterogeneous with a wide variety of symptoms and signs that can present motor, sensory and emotional deficits that might go unnoticed in an initial analysis. The assessment includes various tools that analyse cognition, vision, balance...such as questionnaires. Classification schemes for concussion are based on the presence and duration of the symptoms and signs[9].

Correct care by a sports doctor is very important after an athlete receives a blow to the head with a certain intensity. The patient requires immediate attention and although they might have been confused for just a few seconds, they must leave the sports field to avoid the second impact syndrome given that a second blow can be much more dangerous than the first[10].

TBI requires an urgent aggressive treatment, to prevent or reduce an irreversible injury to the nervous system. Regarding CC treatment, studies are not conclusive on the use of certain drugs (AINE, paracetamol, tricyclic antidepressants, amantadine, melatonin, etc.) when treating the acute phase of the concussion, or the post-traumatic cerebral oedema and, although symptomatic treatment is recommended, it is very important that the doctor is up to date on this matter and understands when surgery is required and what type of operation is most recommended[11,12].

In terms of deciding when to return to training and competition after suffering a CC episode, the central nervous system (CNS) must

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have clearly returned to normal, thinking only of the athlete's health. After concussion, it is recommended to rest, both physically and intellectually, and abstain from sporting activities until the patient is entirely asymptomatic, although there are no clear protocols on how long is most appropriate. Some research even questions rest after the 2 first weeks or before and suggests light or moderate physical activity to start[13]. Classically, compulsory rest is recommended, but when the light concussion symptoms have been resolved (in 24 to 48 hours in most cases), according to some criteria, non-contact activity can be allowed (running, cycling, lifting objects) and if the symptoms do not return, the athlete could be authorised to train and compete[14].

In any case, CC recovery times and prognosis vary widely, and it is very important to ascertain the ideal and safe moment to return to competition depending on the evolution[15].

The risk should also be assessed for consequences or mental damage as a result of the chronic traumatic brain injury (CTBI), related to repetitive blows to the head, initially described in boxers and now extended to other sports. It is characterised by presenting neurodegenerative changes that are reasonably similar to Alzheimer's disease. Therefore, return to competition is not the only factor to consider, but also long-term mental health[16].

It might be supposed that after recovering from a TBI, the rate of musculoskeletal injuries should be the same as if this injury had not taken place, and if the risk increases, this might be because the NS does not respond correctly in terms of reflexes and coordination[17].

Some data indicate that the % of musculoskeletal injuries in athletes' post-concussion are greater after returning to play[18] and lead us to think that a post-TBI after-effect had not been detected among these athletes.

Further studies are required to find out more about recurring concussions. Recurring injuries (second, third or fourth cerebral concussion) require a longer period of asymptomatic rest, although there are no scientific data to corroborate this. Tests to analyse cognitive and neurophysiological dysfunction are required that provide objective information on the cognitive deficits[19].

The world of sport should be constantly aware of preventing TBI and particularly CC. We must focus on individual risk factors and the specifics of each sport using strategies that modify the rules to limit contact, the equipment and the playing technique. Primary CC prevention is vitally important to protect athletes' health and safety. In contact sports, training on specific collision techniques for each sport is very important. The American Academy of Neurology mentions the importance of baseline neuropsychological assessments to improve interpretation of concussions[20].

This is not easy as in many sports, the game depends on physical contact and it is unlikely that contact sports will ever be entirely safe, despite improvements to protection systems, fundamentally protective helmets with on-going design improvements[14,21,22].

Rehabilitation of patients who have suffered a TBI is fundamental to reduce long term functional disability and achieve a proper recovery.[12,23]

Although TBI and CC care has changed greatly over the last 20 years, and there are consensus documents in this respect, we should look in greater depth at knowledge of the physiological changes that take place in CC. We should also make progress in determining markers that can identify the persons with risk of mental deterioration and death so that they can be caught in good time. Improvements to diagnosis techniques should continue. For all these reasons, this focus study is very important as it analyses the latest progress in TBI and cerebral concussion in sport and figures out the direction of the research related to fundamental aspects of the topic.

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Concussion and traumatic brain injury

Conmoción cerebral y traumatismo cráneoencefálico

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A concussion is a mild form of traumatic brain injury which affects brain functions, generally temporarily, with symptomatology of slight loss of consciousness, concentration and memory, headache, confusion, affecting balance and coordination[1-5].

A traumatic brain injury (TBI) is known as a blow or impact, a jolt in the cranium or facial region producing an injury on the scalp or face, affecting the level of consciousness to a certain extent which might injure neurones and cause biochemical changes that must be assessed, diagnosed and treated. Its severity is conditioned by the cause (traffic accident, workplace accident, impact from a fall, sport, attack, others), its location and the appearance of subsequent complications (cognitive, behavioural, physical, balance, coordination and/or emotional)[4,5].

TBIs can be mild (brief alteration of mental state or consciousness, headache, dizziness, vomiting, blurred vision, behavioural changes) or evolve progressively to become severe (persistent loss of consciousness or reasoning/behavioural problems after suffering an injury)[5,6]. Long term effects depend on severity, location of the injury, the patient's age and prior state of health.

Although most medical consultations for TBI are mild, traumatic head injuries are the top cause of death and disability in the population aged under 45 years old in developed countries. In 2007, the WHO estimated that it would be the top cause of mortality in 2020[7]. In Spain, three out of four patients with TBI are men aged between 15 and 30 years old; the main cause is traffic accidents (decreasing), falls among people aged over 65 years old (increasing), childhood accidents during leisure activities and during sport[8,9]. Neurological after-effects involving disability stand at 15% (Spanish Neurology Society, 2018)[8].

Sports-Related Concussion (SRC)

Concussion is considered to be one of the most complex injuries in sport because it is difficult to assess, diagnose and provide care for it[3]. Sports doctors are uniquely skilled to give the required care throughout an SRC from acute assessment to return to playing and care for SRC complications and coexisting medical issues thanks to their knowledge, experience and daily proximity to the athlete[10]. Most SRC are resolved within 1 to 4 weeks, although there are cases with complicated and/or lengthy recovery stages that might require a multidisciplinary team with experience in checking on and caring for SRCs.

SRC characteristics include[2,3,10]:

- Aetiology, due to acute trauma from a blow, collision or repetitive exposure to impacts (opponent, ground or ball) and/or jolts to the head, face or neck (whiplash), and in any other part of the body with a driving force to the head (attack, defence, tackle).
- The urgent care, assessment and diagnosis that take place on the playing field.
- Immediately prohibiting sport (training, competition) for any affected athlete, irrespective of the type of activity and age, given that this is an acute injury where the diagnosis can change quickly, and which requires caution and fast action.
- Care and treatment must be swift and managed properly in the short and long term, including the return to sport, to guarantee the athlete's health and avoid consequences.

Sports with the greatest risk of suffering a concussion, at any age, include any which involve physical contact with drive, crashes, blows, falls, sporadic or repetitive impact and others with a risk of high speed

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collision, above all without the right protection or safety gear, such as American football, hockey, rugby, boxing and football[11-18]. Statistically, a greater proportion of women than men are assisted for SRC.[2] Two recent systematic reviews show a greater statistically-significant incidence rate for concussion among women than men, in football and basketball[19] and a greater prevalence of concussion and symptom-reporting among women. The role of biological gender should be investigated to evaluate its importance in SPC assessment, care and subsequent treatment[20].

Clinical diagnosis

Most SRCs present without loss of consciousness or clear neurological signs that might be transitory and appear in the first 24-48 hours. The inflammatory cell activation, axon degeneration and altered plasticity can occur in the sub-acute and chronic stages of concussion[10]. Even without clear symptomatology in the acute phase, it can thereby appear a posteriori, masked by behaviour that is considered normal in the athlete's surroundings (family, social, sporting).

Risk factors to develop a subsequent disorder include more symptoms early on (such as headaches: and fatigue), previous multiple concussions, psychiatric disorders (anxiety, depression), being unconscious for longer, or amnesia and an earlier age[5]. Consequently, patients should be monitored after the acute phase of the concussion[3].

Clinical diagnosis can be made with the following symptoms which are reasonably unspecific to be able to make a diagnosis themselves[2,3,10]:

- Physical: headache, dizziness, affected vision, balance and sensitivity to light and noise.
- Cognitive: confusion, difficulty to concentrate with expression and memory problems.
- Emotional: irritability, exacerbation of feelings (sadness, lack of energy, effusiveness, rage, fear), the jitters.
- Sleep disorders: changes to sleep patterns with an increase or decrease in the usual number of hours, drowsiness affecting the period of wakefulness or activity.

At the time of a possible injury, the medical professional on the ground assesses the behaviour of the injured person in terms of orientation, memory, concentration, movement and balance, speech and reasoning, plus a cervical exploration to evaluate other injuries[3,10]. If an SRC is suspected, the examination should be exhaustive and specific. The injured person should not be left alone, and their condition should be supervised for the first few hours. Sports organisations must allow enough time to perform this assessment. OPne aspect that could be improved is determining a given time in sports rules that still do not include it.

Any athlete that suffers increased symptomatology after the suspicion of a concussion must withdraw from the activity until an additional assessment can confirm or rule out SRC.

The justification for immediate withdrawal from the sports field and consequent evaluation would be the presence of warning signs or visible indicators such as loss of consciousness, convulsions due to the impact, difficulty remaining upright, empty gaze, lack of motor coordination or balance[3,10]. Any of these observed or reported symptoms must imply stopping any sporting activity for at least the rest of the day.

Presenting more severe head injuries with lengthy loss of consciousness, intense or worsening headaches, vomiting that does not stop, declining mental state, focal neurological shortfall or suspicion of a significant head injury (skull fracture, intracranial haemorrhage) must trigger the emergency plan.

Diagnosis

A medical examination is the first step to diagnose a possible head injury.[2,3,5,10] If there are signs or symptoms from one or more of the examination areas (neurological, motor, sensory, cognitive), an SRC must be suspected, a differential diagnosis determined, and the appropriate care strategy followed. The medical professional who knows the athlete is the most appropriate person to detect subtle changes in their personality and performance. The concussion assessment must be performed in a distraction-free environment with plenty of time for the examination and to complete the concussion tests.

In terms of differential diagnosis, it must be distinguished from symptoms due to drugs, alcohol, use of medicine or other injuries (vertebra, peripheral vestibular dysfunction) or other co-morbidities (psychological or medical afflictions)[3,5,10].

An SRC can cause neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional alteration instead of a structural injury and, as such, no abnormality is seen in the standard structural neuroimaging studies[3].

Underlying factors prior to an SRC have been demonstrated[21,22], such as the type of sport, a prior history of concussion, age, suffering migraines, fatigue and sleep alterations, that can be predicted from subsequent symptomatology with a longer recovery and the appearance of symptoms related to the cognitive-somatic-sensory axis.

Consequently, the concussion diagnosis is a clinical diagnosis[2,3,10,21-23] working from the clinical history and the secondary assessment in the first instance and then according to how the injury evolves:

The clinical history included in the medical check-up prior to taking part in the sport, plus the usual examinations regarding balance, reflexes, hearing and sight, an assessment of previous concussions or other traumatic brain injuries (number, recovery evolution and time between injuries), presence of other premorbid/comorbid conditions, or other factors, that can make diagnosis and/or care for the concussion difficult, including a background of learning disorders, mood swings, attention deficit, motion sickness or sensitivity to movement, personal or family history of migraines and information on current medicine consumption. These data will be used, in the event of an SRC, to provide assistance, improve care and draw up an emergency plan.

The secondary assessment with a physical and neurocognitive examination, using a fast, reliable and standardised assessment system. This should be performed immediately and/or in a distraction-free

environment with the concussed person at rest, depending on how badly hurt they are.

Currently, the best developed tool with the widest field of implementation available for secondary assessment is the SCAT Sport Concussion Assessment - which is updated periodically, according to scientific evidence, by the Concussion in Sport Group (CISG) from the second International Consensus Conference on Concussion in Sport held in Prague in 2004[2,3,10,24,25]. The 5th version, SCAT5, is currently in use, as the latest version agreed on by the panel of experts from the 5th International Consensus Conference on Concussion in Sport held in Berlin in 2016[3,26]. Its official supports are SCAT5 for athletes aged 13 or over[27,28] and paediatric SCAT5 for children from 5 to 12 years old, due to their differences from adults[29-32].

SCAT is useful immediately post-injury to differentiate between concussed and not concussed athletes. It takes 10 minutes, but it seems to be significantly less useful 3 to 5 days after injury[4,10,25]. The symptom verification list, however, is clinically useful when monitoring recovery. It is recommended to use SCAT post-injury in situ with part of the questionnaire and finish it subsequently, in a quiet place, away from where the injury took place to make the data more objective (changing rooms, consulting room).

SCAT5 contains indications, instructions, questions to verify the symptoms and clinical assessments that are performed immediately and a few hours after the sports trauma with a concussion or suspicion of concussion. It includes an evaluation of the motor function (movement), the sensory function, coordination of reasoning and reflexes. It encompasses the system to assess memory with the Maddocks questions, the Glasgow Coma Scale (GCS), the Standardised Assessment of Concussion (SAC), the Modified Balance Error Scoring System (mBESS), the ruling based on the questionnaire scoring and advice for whoever is going to monitor the convalescent athlete. It is recommended to question the athlete on symptom assessment for an acute/post-acute phase, when taking the test. In the stable or baseline situation, it will be filled in by the affected person, and will be a self-assessment[26-28].

The paediatric SCAT5 includes tests and assessments scored according to paediatric age[29,31].

Other tools and tests that can be used in the secondary assessment, along with the SCAT5 or alone, according to presented symptomatology, are the CogSport (cognitive function tests), Automated Neuropsychological Assessment Metrics (ANAM), system of Central or Immediate Nervous System Vital Signs[3,10,25].

Vestibular/Ocular Motor Screening (VOMS) is useful to assess the vestibulo-ocular reflex in people aged over 8 years old[33,34] . The sensitivity (probability of correctly identifying the concussion) and the specificity (probability of correctly identifying the absence of concussion) of the sport concussion diagnosis can increase when combining multiple assessment tools.

Regarding the test-retest reliability (sensitivity and specificity), as a novel concept, the SCAT5 includes optional lists of 10 words and longer sequenced lists of digits counting backwards[27], to minimise the ceiling effect (obtaining the maximum score or close to it in most people), which was a bias in the SCAT3 memory and concentration assessment score. There are currently no applied sensitivity and specificity studies for SCAT5 and children's SCAT5 and it remains to be demonstrated whether these changes have made them more reliable than previous versions.

All the tests most widely used in the secondary evaluation, including any from SCAT5, can be applied as pre-season assessment (verification of symptoms, assessment of balance and cognitive function), above all in the case of post-concussion[10,21-24]. However, the whole battery of assessment tool tests is not essential or required to care for SRC properly.

It should be highlighted that, to optimise diagnosis from the result or score obtained from secondary evaluation tools, it is necessary to know about the limitations both in the case of the assessor and the subject[10]. The assessor can make a methodological mistake in the scoring if they are not familiar with the psychometric properties of the tools that they are using in relation to the symptoms presented by the concussed person to be assessed. Likewise, the subject can change the result of the self-assessment tests by performing them repetitively and thereby memorising them.

To reach an accurate diagnosis, all tests must be interpreted in combination with a decisive presentation of concussion.

The diagnostic imaging tests, including CT and MRI scans do not diagnose SRC[3,10]. However, in the case of suspecting an intracranial haemorrhage, they can help to rule out a potentially deadly brain injury, that might require immediate surgical attention.

Fluid biomarkers and genetic tests are important research tools, but they require additional validation to determine their whether they are clinically useful in SRC assessment[35]. More research is required on current limitations as a diagnostic resource for concussion.

Treatment and return to sporting practice

After a concussion, the symptomatology is generally resolved sequentially until the previously normal situation is restored, taking 1 to 4 weeks (10-14 days for adults, over 1 month for children) with cognitive and physical rest that reduces the post-concussion symptomatology as it demands less brain energy[3,6,10,30-32,36]. 80%-90% of teenagers and adults improve in 2 weeks, recovering their previous balance and cognitive function. Each person's recovery time is different and treatment must be individualised with gradual progression, limited by the symptoms[3]. It is currently not possible to quantitatively standardise the recovery period as the physiological aspect might take longer than the clinical symptomatology and there are no diagnostic tools to measure physiological changes (magnetic resonance, blood flow to the brain, electro-physiology, heart rate, fluid biomarkers, among others) which have been validated by the clinical interpretation[3]. More research is required in this field.

Should the recovery time top one month, this is usually related to a history of prior concussions, suffering migraines, sleep and psychiatric variations (anxiety, depression)[5,21,22], more evident in children, teenagers

and young adults[32,36]. The recovery delay is also influenced by a greater degree of symptom presentation and the severity, loss of consciousness, retrograde amnesia or post-trauma amnesia[3], and the early appearance of headaches and depression[36]. More research studies are required to set clinical profiles with the duration of the recovery period, the age and the gender.

The athlete must rest physically and cognitively to a certain extent to ease remission of symptoms, starting with 24-48 hours and spanning several days. Their symptomatology should be monitored by a person nearby, who is aware of the possible variations (worsening headaches, drowsiness, vomiting, inability to recognise persons/places, variations in behaviour and speech).

If behaviour changes are observed, or vomiting, worsening headaches, double vision or excessive drowsiness, this would constitute an emergency and you would have to get in urgent contact with your doctor or with the emergency services[3,10,26].

In most cases, after getting through the first 24-48 hours and after a few days' rest, the athlete will be able to gradually increase their level of daily physical activity and non-contact aerobic exercise, as long as the symptoms do not get worse or become exacerbated[37]. Several studies refer to a short recovery with sub-symptomatic threshold aerobic exercise between 3 and 7 days after the injury, in adolescent males and young people[37,38]. There are currently insufficient consensus tests to prescribe complete rest for over 24-48 hours to improve recovery[3,10].

When the athlete is capable of performing all usual daily activities asymptomatically, they can begin the phase to progressively return to sports practice[3,10]. This should involve a gradual exercise programme following stages (type of exercise, intensity, duration, with/without contact), run by a doctor. The duration time for gradual incorporation into sport is multi-factor (age, history, type of sport and level, etc.) and must be managed individually. Different stages must be defined, conditioned by the clinical evolution, as a strategy for gradual return to sport[27], such as setting aside more than 24 hours for each one, not introducing endurance training until the 3rd or 4th week or the last stages and going back to the previous stage if the athlete's condition gets worse with a frequency of at least 24 hours. When to introduce early training, and in which cases, is still under investigation[10]. Early activity and exercise do not replace the gradual return to sport.

Among children and young people, it is important to consider that their learning ability can be affected by concussion. Coordination is therefore vitally important between the doctor, parents, carers, when appropriate, and teachers to manage the plan to return to school[6,26,27]. In children, non-sporting games should also be considered, particularly in unpredictable environments (school playground), to avoid a relapse or a further injury[6,29-32]. The return to playing sport is conditioned by an absence of symptomatology in daily physical-cognitive activities and academic learning activities.

If clinical symptoms persist during the sequential evolution of the recovery, this may be associated with recurring injuries of the cervical column and the peripheral vestibular system that might require a multidisciplinary professional group for well-managed therapy including psychological, cervical and vestibular rehabilitation[3,10].

Symptomatic pharmacological treatment has not been properly corroborated[3]. If it can be justified, the athlete should not play again until this treatment is complete as it might be masking or modifying the SRC symptomatology. The doctor should assess when this return is appropriate.

Regarding treating concussion with dietetic supplements such as group B vitamins, vitamins C, D, E, magnesium, branched-chain amino acids, N-acetyl cysteine, N-methyl-D-aspartate, Nicotinamide riboside, fatty acids ω-3, creatinine, curcumin, resveratrol, caffeine and melatonin, with evidence of improving symptomatology in animal models, protecting or accelerating recovery, it cannot be extrapolated and lacks sufficient research in humans, and would require more research to be used rigorously, reliably and safely[36,37].

In summary, it can be concluded that post-concussion treatment must be individualised by introducing physical activity and gradual cognitive activity, limited by symptoms, with sub-symptomatic aerobic exercise and therapy programmes according to variations in the vestibular and cognitive-behavioural function. Return to sporting practice can be determined with gradual programming of physical exercise monitored by a doctor, conditioned by lack of symptomatology in daily activities and school or academic learning, in students.

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Post-concussion syndrome in sport

Síndrome postconmoción en el deporte

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Introduction

Post-concussion syndrome is a common consequence of a traumatic brain injury, and it is defined as a set of symptoms including headaches, dizziness, neuro-psychiatric symptoms and cognitive deterioration[1,2]. Post-concussion syndrome is most frequently described as the scenario of a mild trauma, although it can also occur after a moderate or severe TBI. Similar symptoms are also described after whiplash injuries. The underlying physiopathology is not defined. Test results can be abnormal, or not; when they are present, test anomalies do not consistently follow a defined pattern.

A mild traumatic brain injury comes after a non-penetrating head trauma, and it is usually defined as mild due to a Glasgow Coma Scale (GCS) score of 13 to 15, 30 minutes after the trauma[3]. Concussion is an alteration in the mental state caused by a trauma that might imply loss of consciousness.

Epidemiology

Between 30 and 80 percent of patients with mild to moderate head injuries experience some post-concussion syndrome symptoms. This apparently wide incidence range demonstrates variability among the studied patient population and the criteria used to diagnose post-concussion syndrome, either using individual symptoms or defined clinical criteria. Two clinical criteria are commonly used: the International Disease Classification, 10th edition (ICD-10) and the Diagnostic and Statistics Manual of Mental Disorders, 4th edition (DSM-IV)[4]. They give very different results, even within the same patient population.

Several studies have attempted to associate head injury severity with post-concussion syndrome among patients with a mild traumatic brain injury (TBI) using a variety of measurements including the Glasgow Coma Scale (GCS), how long consciousness was lost or post-traumatic amnesia lasted, and the presence or extension of abnormalities seen in the CT or MRI scan. In general, the severity of the injury does not correlate clearly with the risk of post-concussion syndrome. However, at least one study suggests that prior history of concussion, particularly if recent or multiple, is a risk factor for prolonged symptoms post-concussion[5-9].

Studies of patient cohorts with mild and moderate TBI have consistently found that being female and older are risk factors for PCS. While the nature of the head injury has not been systematically studied as a risk factor, some studies suggest that patients with sports-related concussion have a better natural history than any with mild TBI resulting from a car accident, a fall or an attack. This can reflect different severity for the physical and/or psychosocial impact, and/or a different inclination for PCS. This can also contribute to gender differences, as the relative weighting of accident vs sports injuries as a cause for the TBI may be greater in women than in men.

Physiopathology

There are various theories for the pathogenesis of PCS. Some maintain that the disorder is structural and biochemical and that it is the direct result of the head injury; others state it has a psychogenic origin. It is possible, even probable, that both contribute, and can have a different impact on different symptoms at various points in the syndrome.

Neuro-biological factors

A series of structural and biochemical changes have been documented in animal models of head injuries and in neuropathological studies on humans. One study compared regional brain volumes in magnetic resonance images (MRI)[10-11] in 19 patients one year after a mild

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traumatic brain injury (TBI) with 22 paired control subjects. The patients had measurable global atrophy compared to the controls. Certain areas of regional volume loss (such as the cingulate cortex) were correlated with lower neurocognitive measurements, clinical scores for anxiety and post-concussive symptoms.

Physiological and functional neuroimaging (computerised tomography due to emission of a single photon [SPECT], tomography due to emission of positrons [PET][12-13] and functional magnetic resonance) also documents more extensive areas of abnormality than observed in the computerised tomography (CT scan), supporting the idea that a structural or physiological head injury plays a role in causing PCS. However, many of these neuroimaging findings are not specific to head injuries and can also be observed in patients with migraines or depression. Furthermore, the studies do not consistently show a relationship between the extent of the abnormalities observed in these studies and the degree of deterioration or the severity of the symptoms experienced by the patient. One exception is a study that correlated acute findings in the CT perfusion examination at the time of the TBI with disability at six months. The role of these factors in producing the clinical PCS symptomatology remains unclear.

Psychogenic factors

The psychogenic contribution to PCS is suggested by several empirical and clinical observations. The PCS symptom complex (headaches, dizziness and sleeping issues) is similar to the somatization that is observed in psychiatric disorders such as depression, anxiety and post-traumatic stress disorder (PTSD). Furthermore, anxiety and depression can cause subjective and objective cognitive deficits similar to those observed in PCS, that improve with antidepressant treatment.

Several studies suggest that both psychiatric inclination (low capacity for confrontation, limited social support and negative perceptions) such as psychiatric comorbidity (depression, anxiety and panic, acute stress and PTSD) are more prevalent in patients with PCS compared to the general population controls and/or to patients with head injuries who do not develop persistent PCS.

However, studies on the interaction of depression, anxiety and cognitive performance in other populations with mild TBI are limited. Some researchers do not find a substantial correlation between the level of the depressive symptoms and the cognitive deficits in patients with mild TBI, while others have found a correlation in the response to antidepressant treatment in a subset of patients.

The association between psychiatric disease and PCS has not been established. Limitations in the methodology, including cross-discipline design and the selection bias for patients and control groups make it impossible to draw solid conclusions. Furthermore, this association may have several explanations. Patients with prior psychiatric diseases may be more likely to suffer head injuries as a result of more prevalent alcoholism, motor or physical impediments derived from their illness or medicines, and for other reasons. Alternately, patients with psychiatric diseases may be more likely to develop PCS after a head injury. Finally, the head injury may cause or precipitate the psychiatric disease in susceptible individuals.

Other factors

The lowest rates or even absence of post-concussion brain symptomatology, occasionally reported in some countries and among children, suggest a prominent role of sociocultural factors in the PCS pathogenesis.

The idea that pending compensation claims contribute to the presence and duration of PCS symptomatology go back to the original reports at the end of the 19th century. The studies show a relationship between persistent PCS and potential financial compensation. However, association does not clearly imply causality. Some patients with pending lawsuits improve with or without treatment and PCS occurs in the absence of lawsuits. On the other hand, the fact that patients do not recover after the claims have been resolved does not necessarily invalidate this theory, as a financial agreement can, in fact, reinforce the disease.

Clinical characteristics

The most common complaints in PCS are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and sensitivity to noise. The relative preponderance of these symptoms varies from one study to another depending on the clinical environment, the time passed since the injury and other variables. For example, among 118 patients who volunteered for a mild traumatic brain injury (TBI) treatment study, one month after the injury, there were reports of headaches in 78%, dizziness in 59%, fatigue in 91%, irritability in 62%, anxiety in 63%, sleep disorders in 70%, memory lapses in 73% and noise sensitivity in 46%. Among the patients sent to a headache clinic, approximately half had cognitive complaints and a quarter had psychological complaints; 17 percent had an isolated headache complaint.

Headaches

Headaches are estimated to occur in 25 to 78 percent of persons after a mild TBI. Paradoxically, the prevalence, duration and severity of the headaches is greater among people with mild head injuries compared to any with more severe trauma[15-16]. A significant number of patients have pre-existing headaches, but studies contradict themselves regarding whether this is a risk factor for post-traumatic headaches.

According to criteria from the International Headache Society (IHS), the headache should start within seven days of the injury. The seven-day

start is arbitrary, particularly because the aetiology of the post-traumatic migraine is not understood. Three months seems a more reasonable latency for the start than seven days.

Most post-traumatic headaches can be classified by the IHS type in a similar way to non-traumatic headaches. Migraines and tension headaches predominate. In most series, tension headaches are the most frequent (75 to 77 percent). Many patients (27 to 75 percent) have more than one type of headache.

Tension headaches can occur daily, either as a constant pain or intermittently for a variable duration. Distribution can be generalised, nuchal-occipital, bifrontal, bitemporal, like a head band or a cap, and they are characteristically described as pressure, oppressive or deaf pain. Excessive use of analgesics complicated 42 percent of post-traumatic headaches in a series.

The migraine headache is typically lateralized, pulsing or stabbing, with associated photophobia and nausea. This occurs with and without a visual aura[17].

Recurring migraine attacks, with and without an aura, can be the result of a mild head injury. The impact can also cause acute migraine episodes, often in adolescents with a family history of migraines. Originally known as a "footballer's migraine" to describe young men who played football and had multiple migraine attacks with aura, triggered only by impacts, similar attacks can be triggered by a mild head injury in any sport.

In the same way, there are many post-traumatic cranial pains, such as post-traumatic temporomandibular pain where patients might complain of pain in their jaw or hemicrania or ipsilateral frontotemporal pain or post-traumatic neural pain. Headache syndromes attributed to the trauma in the case reports include cluster headaches: continuous hemicrania; short unilateral neuralgiform headache attacks with conjunctival injection, tears, sweats and nasal discharge (SUNCT), short unilateral headaches with cranial autonomic symptoms; and paroxysmal hemicrania.

Dizziness

Approximately half the patients report dizziness after a light head injury. While some patients with PCS have unspecific dizziness (vertigo), others report real vertigo that can be due to a benign paroxysmal positional vertigo or a labyrinthine concussion. Several studies suggest that dizziness complaints at the time of the injury, and afterwards, identify patients who run the risk of a lengthy recovery.

Sleep disorders

Sleep disorders are also reported, generally insomnia, in approximately one third of patients in the acute phase after a mild injury and in approximately half of patients in the chronic phase.

The most common manifestations of sleep disorders and wakefulness after a TBI are excessive daytime drowsiness, an increased need to sleep and insomnia. Less commonly, patients experience variations in their circadian rhythms; abnormal movements or behaviour during sleep, such as talking in their sleep, grinding their teeth and representation of dreams; and sleep-disordered breathing.

Psychological and cognitive symptoms

Over 50 percent of patients report personality changes, irritability, anxiety and depression after a mild TBI. They might find that they are intolerant to noise, emotional excitation and large crowds, and more susceptible to the effects of alcohol. Patients also report memory and concentration impairment[18-20]; this can be corroborated by the objective deficits of the neuropsychological tests. In a typical case, these are more prominent immediately after the injury and they are resolved in the following weeks and months.

A significant number of patients (15 to 20 percent) will develop symptoms that meet the criteria of a psychiatric disease. These include acute stress and post-traumatic stress disorders (PTSD) plus anxiety, panic disorder and depression.

Diagnostic tests

Wise use of tests should be individualised for each patient. Patients with persistent complaints of visual or vertigo symptoms should be referred to an ophthalmologist or an ear, nose and throat specialist. A psychiatric assessment must be considered for patients with prominent psychiatric symptoms.

Neuropsychological tests: these tests are not useful in most patients with symptoms following a concussion. However, when they are performed by an expert and experienced psychologist, the neuropsychological evaluation can be useful to assess selected patients with prominent cognitive or psychological complaints, ensuring their mild nature and their limited reach. The monitoring studies for non-selected patients after a mild traumatic brain injury (TBI) demonstrate small measurable deficits in the neuropsychological tests. Cognitive domains that seem particularly vulnerable to the effects of the head injury include attention, working memory, processing speed and reaction time. Deficits are generally light; severe intelligence and memory deficits are not associated with a mild TBI. Abnormalities are most prominent in the first week after the TBI and disappear over time. After three months, mild TBI patients have a similar performance as a group to the control subjects. In one study, approximately 15 percent of patients have persistent cognitive deficits.

Neuroimaging

Many patients assessed for a mild TBI will have had a CT scan or an MRI scan, as part of their acute assessment. Approximately 10 percent of CT scans in mild TBI are abnormal, demonstrating a mild subarachnoid haemorrhage, subdural haemorrhage or contusions. The MRI scan is

more sensitive than the CT scan, demonstrating abnormalities in approximately 30 percent of patients with normal CT scans.

Patients with PCS who have not had an MRI scan and have incapacitating complaints should have an MRI on their brain to exclude a more serious pathology that would identify either a worse prognosis or an alternative cause for their symptoms.

Other advanced neuroimaging techniques, including functional magnetic resonance, magnetic resonance spectroscope and diffusion tensing image (DTI), are being investigated in TBI patient assessment. In one study, the patients with evidence of a traumatic axon injury in DTI were more likely to demonstrate objective evidence of cognitive impairment compared to patients with normal studies. A meta-analysis concluded that although DTI is sensitive to a wide range of group differences in the diffusion metrics, DTI currently lacks the specificity required for significant clinical application in individuals.

Treatment

PCS treatment is individualised according to the patient's particular complaints. Peace and quiet is often the main treatment, as most patients will improve in three months. In the absence of specific treatments to prevent or treat PCS, most doctors take a symptomatic approach[20].

Cognitive or physical rest

Cognitive or physical rest after a concussion has shown no convincing evidence of improvement in terms of faster recovery or in the long-term clinical results. Patients must avoid activities that might lead to a second concussion while they still have symptoms from the initial event. We do not formally recommend any other type of rest period. Patients must limit activities that worsen their symptoms in the first few days after the injury and then gradually return to their previous level of activity according to what they can tolerate.

Headache treatment

Information on the treatment of headache syndromes, specifically in the post-traumatic environment, is limited to series of cases:

- Amitriptyline has been widely used for post-traumatic tension headaches, and for unspecific symptoms such as irritability, dizziness, depression, fatigue and insomnia[21].
- Occipital neuralgia frequently responds to blocking the major occipital nerve with local anaesthetic, and it can also be combined with an injectable corticosteroid.
- Propranolol or Amitriptyline alone or in combination produce a response rate of 70 percent in 21 of a series of 30 patients properly treated with post-traumatic migraines.
- Excessive use of analgesics was a common contributing factor to the post-traumatic headache in 19 to 42 percent of patients. These patients respond to withdrawing analgesics as favourably as patients whose headaches were not post-traumatic.
- Patients with post-traumatic paroxysmal hemicrania and hemicrania continua have responded to treatment with indomethacin.

Donepezil has had positive results in preliminary studies featuring patients with more severe TBI, but it has not been extensively studied in PCS. Six patients with chronic symptoms after a mild head injury reported a subjective cognitive improvement in an open study on Donepezil[22].

In the absence of specific controlled studies for PCS, this data suggests that post-traumatic headaches probably respond to treatments used for migraines and tension headaches in other environments. Doctors caring for these patients state that the recovery delay from post-traumatic headaches might be due to an inappropriately aggressive treatment, excessive use of analgesics or co-morbidity.

Treatment of sleep disorders and wakefulness: there are behavioural and pharmacological treatments for most sleep disorders and wakefulness in patients with traumatic brain injuries (TBI). Treatment varies according to the dominant symptom or the specific sleep disorder, and the relevant comorbidities. Beyond improving symptoms, potential benefits of the successful treatment for sleep disorders and wakefulness in the population with TBI include improving functional results and quality of life.

Psychological and cognitive complaints

Current evidence does not provide information to treat these complaints that are specific to the post-trauma environment.

Use of cognitive rehabilitation for cognitive difficulties after a mild head injury is controversial. Although there was good support for using a systematic review in military/veteran populations, studies are lacking on other populations. Given that cognitive rehabilitation can be expensive, prospective studies are required to demonstrate their efficacy before being able to recommend generalised application. When psychological symptoms are particularly prominent, support psychotherapy and use of antidepressants and tranquillisers can be useful. Once again, there is only limited data to support a specific treatment approach for the PCS environment. In one study, 15 patients with mild TBI who also meet the criteria for major depression were treated with Sertraline for eight weeks, achieving substantial remission of depressive symptoms, and an improvement to cognitive measurements. An open study among 20 patients with depression after a TBI demonstrated symptomatic improvements with treatment using Citalopram and Carbamazepine. Small random trials have found that cognitive-behavioural therapy improved anxiety and/or depression symptoms in patients who had had a mild TBI[23-24].

Random and control simulated hyperbaric oxygen studies on treating persistent PCS have not systematically demonstrated a benefit in the symptoms or in the cognitive tests.

Education

One of the most important roles for the doctor is patient education. Many patients find peace of mind knowing that their symptoms form part of a well-described syndrome.

Early education and support can also affect the PCS progress.

This was illustrated in a monitoring study on 73 patients with a mild TBI. Those who report a belief at the time of the injury that they were likely to have lasting negative effects had a greater chance of lasting symptoms after three months than anyone who did not back this belief.

Most of the studies, although not all, suggest that early intervention with information and peace and quiet can benefit patients with mild TBI by reducing the PCS severity.

Prognosis

Natural history

The symptoms and disability attributed to PCS are greater in the first 7 to 10 days for most patients. After one month, symptoms improve and in many cases are resolved. A greater symptom load in the initial presentation seems to be associated with a greater risk that symptoms will last for over one month. The vast majority of patients have recovered to a large extent after three months[25-26].

A minority (10 to 15%) have symptoms that last for one year or more. Due to biased information, it is possible that this number is inflated, and the general prevalence is much lower[27-28].

Persistent post-concussion syndrome

Patients with incapacitating symptoms lasting for several months or one year can become more disabled than immediately after the injury. Although the whole complex of symptoms persists in most cases, emotional symptoms seem to be particularly prominent. In general, the studies could not define the risk factors for this sub-set; it has not been systematically demonstrated that the premorbid psychosocial factors or psychiatric disease define which patients run the risk of a lengthy issue.

An exhaustive review of the studies that examine the recovery prognosis after a mild traumatic brain injury (TBI) made the following points:

- Medical-legal problems are a consistent, strong risk factor for persistent symptoms and disability after a mild TBI.
- Repeated concussions can lead to more severe, longer cognitive deficits, but the cross-discipline design of the studies excludes causal inference.
- Gender is an inconsistent risk factor for persistent symptoms.
- Patients with a score of 13 on the Glasgow Coma Scale (GCS) have higher disability rates than those with a GCS of 15, although this can be attributed to other injuries. Patients with complicated TBI (intracranial hematoma or depressed skull fracture) can also be at risk of more persistent symptoms.

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Concussion and traumatic brain injury in sport

Conmoción cerebral y traumatismo craneoencefálico en el deporte

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Epidemiology and sports have the greatest incidence

Sport and physical exercise have become increasingly popular over the last few years and so injuries are also on the rise. Although traumatic head injuries (TBI) represent a small proportion of all sporting injuries, their short- or long-term consequences mean that they are an important cause for concern, with possible implications for sports. The main focus within TBI is Cerebral Concussions (CC) that are thought to represent 80% of visits to the emergency room related to traumatic head injuries. There is a suspicion that, in many cases, the CC is not diagnosed or not assessed by medical professions[1], although as evaluation tools have developed that include neuropsychological tests, neuroimaging and analysis of balance and gait, there has been an improvement in the diagnosis and care for patients with this pathology[2].

Traumatic head injuries

Traumatic brain injury (TBI) is an important health issue as it affects millions of people all over the world every year with high mortality and morbidity incidence. 75% of TBI are due to traffic accidents, affecting young people under 25 more. According to some research, TBI are among the three main causes of death due to traumatic injuries and a high percentage of survivors of these injuries end up with disabling after-effects[3].

Recent estimations indicate that around 69 million people experience a TBI every year[4]. A review a few years ago on incidence in various European countries found 235 cases (adding together hospital admissions, people going to A&E and deaths) for every 100,000 inhabitants and year[5] and a more recent study found an incidence rate between 47 and 850 cases per 100,000 inhabitants per year and a mortality rate between 3 and 28 cases per 100,000 inhabitants and year[6]. The worldwide incidence rate varies from one set of statistics to another depending on age, geographic location and exposure to other risk factors.

In Spain, the overall incidence is 47.3 per 100,000 inhabitants per year (one of the lowest in Europe),[7] although other studies talk about 150-250 cases/100,000 inhabitants per year[8] and, although it is the cause of 1% of all deaths, 80% of cases are mild and recovery is good.

TBI are much less frequent in sport than other types of injuries, although their repercussions and consequences can be more severe and their incidence is rising worldwide, because participation in sport is increasing and there is better knowledge and medical control of it.[9]

The annual hospital incidence of sport-related TBI lies between 3.5 and 31.5/100,000 athletes per year, although other sources present much higher incidence figures. This represents 20% of all TBI and more than half occur in children and teenagers[10]. Between 60 and 80% of hospitalisations related to sport are due to TBI[11].

Sports that imply the greatest risk of TBI are rugby, American football, ice skating, football, horse-riding, cycling, some water sports, etc. On most occasions, the mechanism is usually contact between players or due to the sports equipment used[12]. Incidence is greater in sports which require the use of a helmet, possibly because the risk is higher.

In terms of gender, they affect men more (66-75%) although there is a greater risk for women, at least in some sports, fundamentally due to body constitution[13].

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TBI are more frequent during competition than in training and both incidence and severity are fundamentally influenced by the type of sport, the position of the player and the age[9].

Concussion

Cerebral concussion (CC) or mild traumatic brain injury (mild TBI) is the most common type of TBI, representing 70-90% of all of them. According to some statistics, this affects over 6/1,000 persons a year, of which between 1 and 3/1,000 require medical attention[14].

Sporting injuries are among the most frequent causes of concussion; in fact, CC accounts for between 5 and 9% of sports injuries[15]. In a wide-ranging survey in Canada, it was found that over 54% of all CC were sport-related[16] and it is estimated that there are between 1.6 and 3.8 million secondary sport-related cases in the USA every year[15,17].

In a meta-analysis carried out 10 years ago, it was found that the CC incidence rate related to 12 high-risk sports among children and teenagers (≤ 18 years old) was 0.23/1,000 exposures, and the sports with the greatest incidence were rugby, ice hockey and American football (with rates of 4.18, 1,2 and 0.53/1,000 exposures respectively)[18], and another analysis carried out on 25 high-risk sports found an incidence of 0.45 CC/1,000 exposures[19].

The risk of suffering a CC in sport is going to depend on the type of sport and almost all studies agree that it is greater in contact sports, including ice hockey, American football, rugby, football, lacrosse, and boxing, horse riding, wrestling, skiing, martial arts and cycling[15,20].

A recent study among young athletes (11-17 years old) assessing 27 sports revealed an average CC rate of 0.39/1,000 exposures. The highest rate was in American football (0.92)[21] and in a systematic review focused on sports with the highest risks of CC, higher incidence was found in rugby (3-3.9 CC/1,000 hours of exposure). The study looked at American football, rugby, ice hockey and football (with the lowest incidence: under 2.5 CC/1,000 hours)[13].

CC are more frequent during competition than in training. Consequently, when analysing various sports, Zuckerman *et al*[19]. find a rate of 1.28 cases/1,000 exposures during competitions compared to 0.26 during training. On most occasions (over 70%), the mechanism is usually contact between players, or the equipment used (15%)[12].

In relation to age, the majority of sport-related CC affect young people (school children and university students) due to less experience and because participation is higher in this age range; every year there are between 1.1 and 1.9 million sport-related CC among under 18s in the USA[17] which represents 8.9% of sports injuries in secondary school and 5.8% of university injuries[20].

Some studies observe a greater incidence of concussion in university-aged athletes, affecting more than 12% during the school year[9,21], while for others, the incidence is greater among younger athletes (school age)[22]. The difference probably lies in differing study protocols, varying the exposure time, the sports, etc.

In terms of gender, results are conflicting though most studies demonstrate that women are at greater risk of concussion[12,21,19]; the most frequently repeated opinion is that in sports governed by the same rules, women are usually at greater risk, and it has been found in some sports, such as football or basketball, women have a relative CC risk 1.5-2 times higher than men[9].

This greater incidence among women is due, among other reasons, to the fact that women have a weaker cervical musculature than men, which reduces the stability and rigidity of their neck[12]. In women's sport, CC seem to be more frequent in football (due to elbowing, cracking heads or ball) followed by ice hockey, lacrosse and basketball[13].

Evidence indicates that athletes with a history of CC are at greater risk (three to five times higher) of suffering additional concussion in the future. Recurring cases are estimated to be 9%[19].

A study carried out among 2,552 NFL players found that more than 60% had presented one or more concussions in their playing career and 24% reported that they had suffered three or more CC[23].

However, it should be considered that mild TBI in sport is more usual than the statistics might lead us to believe as in many cases they do not report it to medical services and so it goes undiagnosed (up to 50% according to some statistics) either due to lack of knowledge (only 10% incur loss of consciousness) or due to the belief that the injury is not serious or to avoid being taken out of play. On the other hand, the US Emergency Department recognises that less than 13% of sport-related TBI are assessed in the emergency room[24].

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Consequences of heading the ball in football

Consecuencias del golpeo de balón con la cabeza en el 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

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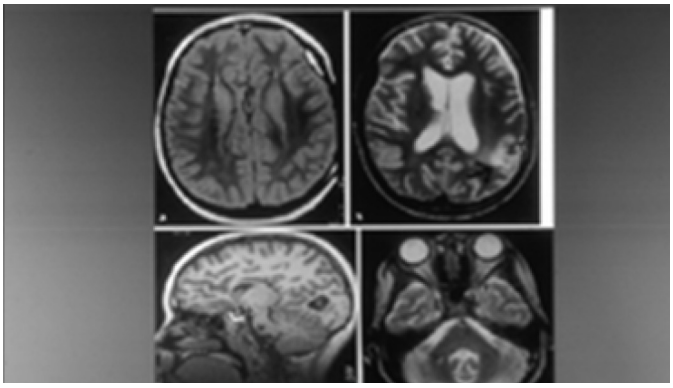
Figure 1. Diffuse axonal injury.



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].

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The use of the helmet in the prevention of brain damage (acute and chronic)

El uso del casco en la prevención del daño cerebral (agudo y crónico)

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Introduction

Between 10 and 36% of traumatic pathologies are related to physical activity. Of these, up to 12% affect the craniofacial (CF) region and their incidence has increased worryingly from year to year. To understand the magnitude of the problem, nearly 4 million CF injuries require medical attention in the US alone. American football and ice hockey are the most injury-prone sports[1].

Research on the mechanisms of CF injuries in recent years has focused on their etiopathogenesis and strategies to minimise their severity. Changes have been made to the rules and regulations, and protective equipment has been developed which has evolved and improved over the years.

With the measures taken, the severity of CF injuries has decreased, but the effects on concussion are not so positive. Furthermore, many concussions are not reported by the athlete for fear of being taken off.

Helmets were one of the first personal protection methods and still are. They are the best way to prevent moderate or severe TBIs. However, there are no positive data on their efficacy against mild TBIs (Traumatic Brain Injuries).

Each helmet is designed to protect against the potential impacts to the head in each sport.

Helmets reduce the chance of a severe traumatic brain injury after collision by reducing head acceleration on impact, reducing both brain-skull collision and the sudden deceleration which causes axonal injuries[2].

Linear and rotational acceleration movements are the mechanisms responsible for concussions and more serious CF injuries.

Linear acceleration describes the translation of the head. It is currently the variable most used to certify helmets in the sports industry, and has been since the linear acceleration peak was associated with harmful pressure waves within the skull[3].

Linear acceleration has also been used as a measurement variable to predict the risk of skull fracture. To certify a helmet, it must be able to withstand forces of approximately 250-300 G. The use of this variable in manufacturing has reduced the incidence of severe brain injury and skull fracture but has had a limited effect in decreasing the incidence of concussion.

All impacts to the head cause translation and rotation. Assessing rotational acceleration is imperative. It has been shown that the type of diffuse brain tissue shearing associated with concussion is related to the severity of head rotation during impact. Brain tissue has a high resistance to translation but a very low resistance to rotation[3].

In the manufacture of helmets, only parameters which measure translation are taken into account. Rotational acceleration needs to be analysed when designing helmets. The exact determination of rotational acceleration is difficult because the movement of the head caused by an impact is measured rather than that of the brain, which floats freely in the cerebrospinal fluid and moves at a different speed to that of the skull in response to a collision. This inability to correctly measure rotational acceleration may be one of the reasons why the incidence of concussion has not decreased despite changes in helmet design.

When an impact occurs in the CF region, it can cause collision between the brain and the skull, either on the impact side (coup) or the opposite side (contrecoup). The high-speed deceleration associated

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with these impacts can also result in diffuse axonal injury. Depending on the extent of these circumstances, a neurological pathology may occur, although the threshold necessary to provoke these is still unknown[2].

Football, ice hockey and inline hockey helmets are effective against linear acceleration impacts but not against rotational acceleration impacts [4].

The degree of protection a helmet affords against TBIs depends, among other factors, on: design, thickness, dimension and type of materials. The design of a helmet involves finding a balance between comfort and optimal safety.

Helmets are basically made up of four fundamental parts: the outer shell, an impact-absorbing liner, padding and a retaining system. The outer shell is made of a hard material designed to disperse impact energy and thus decrease the force before it reaches the head. It also needs to be able to prevent objects reaching the head from penetrating. Generally, it is made with materials such as: plastics (polycarbonates, thermoplastics, Dyneema) and fibres (carbon, glass, Kevlar, tricomposite). The lining, located inside the outer shell, serves to absorb the impact, acting as a "mattress". It is important for the helmet to fit tightly so that it can fulfil its function properly.

The absorption of energy caused by rotational and translational forces and by impact speed is different, depending on the shell design. Currently, there is not a single design which offers protection against all types of impact.

The thickness of the foam and the dimensions of the helmet are important in order to absorb impact energy.

It is more difficult to design helmets which can safely reduce low-energy accelerations and pressures which are distributed all over the brain than it is to manufacture helmets to absorb focal impacts [5].

A primary goal is to decrease maximum deceleration and increase the time over which deceleration occurs; this can be achieved by a thicker, more flexible layer of material which improves energy absorption at the time of impact. Size should not favour any increase in rotational acceleration generated by impact.

When interpreting data on the safety benefits of helmets, it is important to know that a reduction in linear or rotational acceleration is not necessarily associated with a similar reduction in concussion risk. The injury risk curve describing the likelihood of concussion for a specific rotational acceleration is not linear but sigmoidal (S-shaped).

For it to be used for a certain activity, a helmet must meet certain criteria in order to be approved and certified, and must include the corresponding certifications in its specifications:

- Baseball: NOCSAE ND022 for the batter, NOCSAE ND0204 for the catcher.
- American football: NOCSAE ND022, NDOO6.
- Ice hockey: NOCSAE NDO30, ASTM F1045.
- Studies evaluating the effectiveness of helmets in preventing injuries have a number of limitations:
- No standardised definition of concussion.
- No standardised way to examine the effectiveness of a helmet.
- Small samples which decrease statistical power.
- No control groups without helmets.

Although studies have been conducted to see if using other protective measures, such as faceguards (visors or full face shields) and mouthguards, together with helmets, reduces the incidence and severity of concussion, there are no conclusive studies which demonstrate any benefit.

In the 1960s and 1970s, the use of mouthguards was made mandatory in many sports: football, ice hockey, lacrosse, field hockey and boxing. Mouthguards are effective in preventing dental alveolar injuries but not the severity of concussion.

Use of helmets in different sports

Football

Concussion related to collision sports such as American football and hockey has been extensively studied. Lately, lower-risk contact sports, such as football, have also attracted attention. Football is the most widely played sport in the world, with more than 265 million players.

One of the most frequent actions when playing is voluntarily striking the ball with the head and although it may be a mechanism capable of causing injuries and neurological sequelae, we know that most injuries are caused by head-to-head and elbow-to-head impact.

As long ago as 1999, Delaney[6] recommended both the mandatory use of a soft protective helmet in those populations most susceptible to TBIs: goalkeepers, players aged between 6 and 15, and players with a history of head injuries, and prohibiting under-15s from heading the ball in the rules and regulations; a recommendation which was not accepted by the associations which govern the game because it would completely change youth football and, these claimed, the brains of young people, even though they may seem more vulnerable, have greater plasticity and may be able to compensate for impacts.

Helmets would be useful to reduce the impact of head-to-head and elbow-to-head blows, but are not effective when it comes to reducing the impact of the ball when heading[6] and players wearing helmets may behave more aggressively on the pitch.

Where emphasis should be placed is in youth football because of the possible consequences of repeated contact between the ball and the head. Children are maturing. The size of their skulls and brains is disproportionate (at the age of 5, the skull is 90% adult size) with respect to the rest of the body and their necks are not prepared to withstand blows like those of adults.

Consideration should be given to the need for independent doctors not conditioned by either the player or the coach to evaluate concussions produced during matches.

Baseball

Baseball is not considered a dangerous sport. However, there is a real risk of injury as a result of uncontrolled pitches, batted balls and collision on the field.

Most CF injuries are caused by the ball, which can travel at speeds of up to 145 km/h, directly hitting the batter.

Studies recommend: improving the safety of helmets, wearing face shields and cages, using softer balls and reducing the linear speed of metal bats (rotating speed).

CF injuries account for about a quarter of all baseball injuries: 10% are concussions and more than 30% fractures. Even in children from 5 to 14 years old, direct impact causes four deaths every year, which are totally avoidable[7].

The use of helmets has been mandatory since 1971. In professional leagues, these must completely cover the top and back of the head and the two ears on the sides. They must also fit snuggly. The helmets are made of very resistant thermoplastic (acrylonitrile butadiene styrene or ABS), which lends them 300% more rigidity. In 2018, C-shaped flaps began to be used to cover the jaw. The protection they afford is good against low-speed impacts but not so good against high-speed impacts.

It is mandatory for children up to 15 of age to wear helmets and polycarbonate eye protection. Also recommended are the use of low-impact balls and the prohibition of metal bats, as is technically correcting batting to avoid being exposed during pitches.

The use of a helmet is recommended for players when batting, waiting to bat and running the bases, and for base coaches.

Catchers should also always wear a helmet, mask and throat protection.

Ice hockey

Ice hockey is an aggressive sport involving a high risk of injury, due to contusions from impact with hard surfaces: wood, glass, ice, goalposts, sticks, pucks and between players.

It is estimated that up to 25% of professional players have had concussion at least once. The main mechanism leading to injury is impact to the head from the opponent's shoulders, elbows and hands[8]. The use of helmets became mandatory in Europe in 1963, in college hockey in Canada in 1965 and in the U.S. professional league (NHL) in 1979.

To reduce the risk of injury, numerous changes have been made over the years, not only in the protective equipment (helmets and face protection) but also in the regulations, and protocols have been developed to prevent, recognise and treat concussion.

The new technologies used in the manufacture of helmets allow improved protection against linear acceleration, but they do not completely protect against the rotational acceleration which has been linked to concussion, nor are they completely effective against impacts from the puck at a speed of over 90 km/h[9]. The puck can move at a speed of 145-180 km/h when shot and 50-100 km/h when being passed.

The helmets consist of a hard outer shell made of vinyl nitrile, which helps to disperse the force from the point of contact and does not deform but compresses and returns to its original state. The lining may be made of vinyl nitrile foam, expanded polypropylene foam or another material which absorbs energy and reduces the chances of concussion.

A study conducted in Canada in 1978 reported 43 eye injuries which led to blindness and pre-college and college players were obliged to wear face visors together with helmets[10].

Although face protection was first used in 1972, it wasn't until 1996 that the NHL made it mandatory.

There are three types of face protection: visor, full face shield and cage.

Visor: made of transparent waterproof plastic which gives the player unhindered vision, designed to protect against harm to the eyes from sticks and pucks. The teeth and jaw, however, remain exposed. 94% of NHL players wear visors.

Cage: made of aluminium, steel or titanium. Although cages provide the maximum possible protection, they can hinder vision.

Full face shield: offers the same protection as a cage but does not impede vision. Made of sturdy plastic, full face shields offer a large field of vision and have openings at the bottom for the mouth. Obligatory for under-18s.

In a study which assessed which type of face protection better served its function when it came to concussion following an impact, no differences between the different options were obtained, although those who used only a visor were slower to recover when concussed. This may be due to the fact that players wearing visors tilt their helmets back in order to have better vision, thereby decreasing protection of the forehead, which is where most impacts occur, and increasing the risk of serious concussions.

Skaters must wear at least a visor and the goaltender must wear a full face shield or cage (which neither sticks nor the puck can enter).

Compulsory additional protection: mouthguard for under-20s and neck guard for those under 18. Mouthguards are not mandatory in the NHL despite the fact that, in a study of 1,033 players, the concussion rate was 1.42 times higher in those who did not use them.

The game is played with a wooden or synthetic stick with a length of 163 centimetres (cm), a width of 3 cm and a thickness of 2.54 cm. The puck has a diameter of 7.62 cm and a thickness of 2.54 cm, and weighs 156-170 grams. Due to its size, it is capable of injuring the eye because it can enter the socket.

American football

Some two million people play American football in the US. It is the sport which causes the greatest number of concussions.

In 1905, 18 players died and 159 suffered serious skull injuries, and the US president Theodore Roosevelt threatened to ban the game unless urgent action was taken.

The main injury-causing mechanism, accounting for 61% of cases, is helmet-to-helmet impact.

In 1939, helmets were made mandatory in college football and in 1943 in the National Football League (NFL). Facemasks have been required since 1962.

With helmet use, the severity of CF trauma has decreased; the incidence of brain injury-related deaths dropped from 150 deaths in 1974 to 25 in 1994. The risk of skull fracture has also fallen by 60%-70% and the risk of focal brain contusion by 70%-80%, but the risk of concussion has dropped by only 20%.

Most impacts are received on the front of the helmet, meaning there is more linear and rotational acceleration and, consequently, more concussion. Impacts on the top of the helmet are less harmful. These data need to be taken into account in the design of helmets.

A prospective cohort study analysing injury rates among players wearing different types of helmets found that those wearing Riddell's Revolution helmet had significantly lower concussion rates (31% reduction in relative risk and 2.3% reduction in absolute risk) compared to those wearing other helmets, because it reduces g-force by 50%[11].

To date, no helmet is effective for impact speeds of 42 km/h or higher. However, most players do not reach these speeds but ones which are within the range of protection offered by the current helmet design.

More studies are needed to evaluate the energy-absorbing characteristics of the different materials used in the manufacture of helmets: vinyl, nitrile and expanded polypropylene work well for multiple low-energy impacts and polystyrene (PS) for high-energy impacts[11].

The biggest mistake is to think that the helmet alone prevents concussions, and that is not true, because these occur when the brain moves inside the skull and the helmets currently available do not prevent this from happening, although they do protect from lacerations, fractures and eye injuries.

The history of concussions and sub-seizure impacts received must also be added.

Rugby

Rugby is a contact sport which, despite having an incidence of CF injuries similar to that of American football and ice hockey, does not require the use of protection as these do.

The use of a helmet decreases the scalp lacerations and "cauliflower ears" typical of rugby[12] and even significantly decreases the incidence and severity of TBI according to Brooks[13], though not so according to McIntosh[14].

The only protective helmets allowed in rugby consist of polyethylene foam skull caps with a recommended thickness of 10 mm and a density of up to 45 kg/m^3.

Critics of their use argue that they are not advisable for all players because there is some evidence that they can encourage more aggressive behaviour and that the main preventive measure against TBIs should be that players use correct tackling techniques[15].

Wearing mouthguards with helmets does not decrease the incidence of concussion in those players who use them.

Field hockey

A study by Gil Rodas[16] which recorded the incidence of injuries in an elite Spanish club over three seasons highlights the low incidence of injuries to the face and skull: only 1-3% were reported in both sexes. The results are similar to those of Barboza[17].

The only mandatory protections are: shinpads, gloves and mouthguards.

Most CF injuries are caused by ball impact, collision with other players and falling to the ground. CF injuries caused by stick impact are very rare.

Defenders wear face masks at penalty corners to avoid eye injuries and incised wounds with contusion in the facial region by direct impact of the ball. When shot, the ball can reach speeds of up to 180 km/h.

In the United States, it has been mandatory for pre-college players to wear goggles since 2011.

The ball weighs 156-163 grams and has a diameter of 22.4-23.5 cm. It is made of plastic and is hollow inside with a 1-cm wall. It is similar in size to a tennis ball or baseball. The stick has a maximum length of 105 cm and must weigh no more than 750 grams.

Inline Hockey

The protective equipment is similar to that used for ice hockey.

Following the studies by Hutchinson[18] and Varlotta[19], which showed most injuries were to the head and face, 38% and 21%, respectively, under-19s were required to wear full facial protection and over-19s had to wear visors.

With the measures taken, Moreno Alcaraz[20] reported that the incidence of CF injuries decreased to 7.5%.

Quad hockey

Due to the high speed at which the game is played, the continuous contact between players, the weight and speed of the ball, and the use of sticks to strike it, there is a considerable risk of CF injuries in quad hockey.

The stick can be made of wood, plastic or other non-metallic material, has a curved end (blade) which is used to propel the ball, measures 110 cm in length and weighs approximately 500 grams. The ball, made of cork with 2-cm thick rubber coating, measures 23 cm in circumference and weighs 160 grams. When struck, it can travel at 70-125 km/h, with peaks of up to 160 km/h. Rebounds off other players and rink structures can cause the ball to move at a speed between 108 km/h if it is at ground level and 36 km/h if it is at a height of one metre. The size of the ball prevents direct impact to the eye. Players can reach speeds of up to 70 km/h on their skates.

The obligatory protection for players includes gloves, kneepads, shinpads and groin guard, and goalies must also wear a throat protector, breastplate and guards to protect the legs.

Team sports with similar cases of CF injuries took the decision to modify the regulations and require the use of preventive methods

years ago, and these have been effective in decreasing the incidence and severity of CF injuries.

Statistical records have been created to determine the incidence and assess the mechanisms, severity and most frequent type of CF injuries, and thus be able to implement modifications in the rules of the game and suggest new safety measures.

Last season in Spain, serious injuries with functional and aesthetic consequences occurred, including:

- Double lower jaw fracture with instability which required treatment with osteosynthesis and subsequent orthodontics. Mechanism: long shot.
- Aggression with the stick to the skull of an opponent, resulting in suture and TBI.
- Direct contusion with the stick in the face which required 40 stitches in the cheekbone area.
- Impact with the stick in the eye with permanent visual impairment.
- In two cases analysed, the scalp was only very mildly affected and this should be taken into account in the design of specific helmets for roller hockey[21].

In Catalonia, a geographical area where there are more players, monitoring injuries sustained in the last three seasons showed that 25% of the total were CF. Injury mechanisms: stick (39%), an opponent's elbow (24%) and the ball (21%).

The data confirm and ratify the need to review the rules and regulations of the game, bringing in new safety measures and requiring the use of helmets and face protectors.

Despite the evidence, in a survey of players in Catalonia, only 61% are in favour of using additional protection measures; of these 8% would wear a helmet and 4% faceguards, and what they would value most would be panoramic vision (19.7%), protection (18.58%), comfort (16.62%) and a light weight (11.73%).

Currently there are two helmet prototypes with built-in faceguards, one made by a federative body and another by a private company, which differ in design and are pending approval and certification for use. To prevent the helmet from causing injuries voluntarily or involuntarily, it should not be optional as it has been until now but obligatory for all players so they are all equally protected.

Conclusions

A balance between safety and comfort must be achieved in the design of helmets, which must protect wearers from injuries, be low weight with a perfectly adjustable fit, and permit a wide range of vision and peripheral protection.

Helmet designs usually use linear accelerations as injury criteria without evaluating rotational acceleration, which causes most concussions. However, until now the only type of test performed for the certification of helmets has been to use a drop platform representing a fall to the ground and linear acceleration.

Concussion has been shown to be complex in nature and is not easily described using engineering parameters. There are many types of injury mechanisms associated with it, meaning that a single mechanism cannot effectively describe the risk of injury for all concussions. Impacts to the head cause a variety of dynamic responses and it is an illusion to think we can predict the risk of all kinds of concussion.

At present, helmets in most sports are effective at cushioning the impacts responsible for serious brain injury but not for concussion-causing impacts.

There is no epidemiological evidence that the use of faceguards or mouthguards in conjunction with helmets reduces concussions.

In those sports in which the use of helmets and faceguards is not possible, changes in the rules and regulations, improving technique and strengthening the neck muscles should assume their function.

What we do not know is what happens to these players later or the risk to which they are exposed if they continue on the playing field after concussion. The mass media record the moment of the trauma, but not what happens afterwards because it is no longer news.

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Assessment and immediate management of traumatic brain injury

Valoración y manejo inmediato del traumatismo craneoencefálico

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Introduction

With a significant morbimortality rate in people under 45 years of age, in Europe it is estimated that traumatic brain injuries (TBIs) have an incidence of 235 cases per 100,000 inhabitants/year and an approximate mortality rate of 11%[1]. TBI is the cause of neurodegenerative sequelae such as Alzheimer's, Parkinson's and chronic traumatic encephalopathy (CTE), the latter being particularly relevant in athletes who repeatedly sustain mild TBIs[2].

That is why the initial assessment of all TBIs in athletes, the measures taken and the decision to return to competition are decisive for their health.

Initial assessment of traumatic brain injury

The severity of a TBI is defined by two concepts: the primary injury, caused by the traumatic agent which directly affects the brain, and the secondary injury, which causes ischaemia and hypoxia, produces an inflammatory response with the release of neurotransmitters and metabolic deregulation, and leads to raised intracranial pressure (ICP), all of which mark the outcome and prognosis of the injured person[3].

After assessing and stabilising the airway and respiratory and cardiovascular functions, and correctly managing the cervical spine manually or with a spinal motion restriction (SMR) device, attention must be paid to the central nervous system (CNS).

The data from the initial examination must be recorded in writing so that any deterioration in the clinical evolution of the patient can be detected. The neurological examination should be as complete as possible.

If the injury is isolated or located on the skull or face, examination and assessment focussing on detecting signs which may alert us to an alteration in neurological function should be carried out in order to establish its severity and start to take appropriate therapeutic measures.

The neurological assessment should be as thorough as possible and begin with anamnesis.

Anamnesis

The usual medical details with history, allergies and treatments must be taken, together with a detailed medical record of the cause and mechanisms of the trauma, and the characteristics of where the head was struck.

Information should be collected from the patient if he/she can collaborate or from witnesses about:

Initial loss of consciousness, its duration or presence of seizure.

- Initial loss of consciousness, its duration or presence of seizure.
- Signs of temporo-spatial disorientation or amnesia
- Signs of problems paying attention or with language.
- Assessment of deep tendon, plantar and pupillary reflexes.
- Changes in strength, sensitivity, coordination and/or gait.
- Other signs to watch out for are dizziness, sustained or progressive headache, and vomiting.

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Initial clinical examination

The skull and face are inspected for wounds, contusions, scalp laceration, depression as a sign of skull fracture, or signs of skull base fracture such as otorrhagia, epistaxis, panda/raccoon eyes (periorbital ecchymosis) or the appearance of Battle's sign, characterised by ecchymosis over the mastoid process.

The deep tendon, plantar and pupillary reflexes are assessed, as is any change in strength, sensitivity, coordination and gait.

The Glasgow Scale is a good assessment and prognosis evaluation tool which varies depending on whether the patient is able to collaborate or not. Its main limitation is in the assessment of children, facial trauma, people with language disorders and those who do not know the language.

Pupillary assessment

The pupillary examination (Table 1) can provide relevant information because it tests the functioning of the third cranial nerve. The size, shape, symmetry and light response of the pupils should be assessed. One dilated pupil, anisocoria, with poor responsiveness usually indicates uncal herniation. Two small or medium-sized but responsive pupils indicate metabolic encephalopathy or diencephalic transtentorial haemorrhaging. Unresponsive miotic pupils indicate pons damage or opioid use. Hippus (rhythmic dilation and contraction of the pupil) has no meaning.

The absence of pupillary light reflex and anisocoria suggest a structural injury [4].

Level of consciousness

The Glasgow Coma Scale (GCS) assesses the level of consciousness according to three types of response which have a direct relationship with the neurological function: eye response, verbal response and motor response.

It is currently the starting point for TBI assessment.

This type of assessment designed for adults needs to be adapted for children who are unable to communicate orally due to their age.

Neurological assessment scales

The neurological assessment of TBIs is carried out using assessment scales. The most widely used is the Glasgow scale.

Glasgow Scale

The Glasgow Scale (Figure 1) was created by Graham Teasdale and Bryan Jennett, members of the Institute of Neurological Sciences at the University of Glasgow, in 1974. Their goal was to provide a simple and reliable method of recording and monitoring the level of consciousness of patients with traumatic brain injury.

The Glasgow Coma Scale (GCS) (Table 2) assesses eye, verbal and motor responses by scoring each separately and adding them up to arrive at a score that quantifies the neurological situation of the patient and their prognosis.

It should be performed in all cases of TBI as soon as possible and periodic assessments should be carried out to detect improvement or worsening of the clinical picture.

Eye-opening assessment

If after a TBI, the patient keeps their eyes open spontaneously, a score of 4 is given.

If they keep them closed and have to be asked to open them, a score of 3 is given.

If only a painful stimulus (pressure on a finger lasting 10") makes them open their eyes, 2 points are given.

Finally, if no stimulus is able to make them open their eyes, a score of 1 is given.

This assessment is difficult in situations such as facial trauma with orbital hematomas or eyelid oedema.

Assessment of the verbal response

If after a TBI, the patient is able to maintain a guided conversation and answer the simple questions: What's your name? Where are you? What day of the week is it?, a score of 5 is obtained.

If they are confused or disoriented by the same simple questions, they score 4.

If their conversation is incoherent or meaningless, they are given a score of 3.

If the patient only emits incomprehensible sounds or babbles, they score 2.

If no verbal response is noted, 1 point.

Assessment of motor response

The assessment of the movement of a patient who has suffered a TBI is carried out by ordering them to do simple actions such as lifting their

Table 1. Pupillary examination.

Size	Symmetry	Responsiveness
Miotic: < 2mm diameter Mydriatic: > 5mm diameter Normal: 2-5 mm diameter	Isocoria: equal Anisocoria: unequal Dyscoria: irregular	Responsive: contract with light Unresponsive: do not contract with light

Figure 1. Updated Glasgow Coma Scale (GSC).

GLASGOW COMA SCALE : Do it this way

GCS EYES VERBAL MOTOR

Institute of Neurological Sciences NHS Greater Glasgow and Clyde

CHECK	OBSERVE	STIMULATE	RATE
For factors Interfering with communication, ability to respond and other injuries	Eye opening , content of speech and movements of right and left sides	Sound: spoken or shouted request Physical: Pressure on finger tip, trapezius or supraorbital notch	Assign according to highest response observed

Eye opening

Criterion	Observed	Rating	Score
Open before stimulus	✔	Spontaneous	4
After spoken or shouted request	✔	To sound	3
After finger tip stimulus	✔	To pressure	2
No opening at any time, no interfering factor	✔	None	1
Closed by local factor	✔	Non testable	NT

Verbal response

Criterion	Observed	Rating	Score
Correctly gives name, place and date	✔	Orientated	5
Not orientated but communication coherently	✔	Confused	4
Intelligible single words	✔	Words	3
Only moans / groans	✔	Sounds	2
No audible response, no interfering factor	✔	None	1
Factor interferring with communication	✔	Non testable	NT

Best motor response

Criterion	Observed	Rating	Score
Obey 2-part request	✔	Obeys commands	6
Brings hand above clavicle to stimulus on head neck	✔	Localising	5
Bends arm at elbow rapidly but features not predominantly abnormal	✔	Normal flexion	4
Bends arm at elbow, features clearly predominantly abnormal	✔	Abnormal flexion	3
Extends arm at elbow	✔	Extension	2
No movement in arms / legs, no interfering factor	✔	None	1
Paralysed or other limiting factor	✔	Non testable	NT

Sites For Physical Stimulation

Finger tip pressure | Trapezius Pinch | Supraorbital notch

Features of Flexion Responses

Modified with permission from Van Der Naalt 2004
Ned Tijdschr Geneeskd

Abnormal Flexion
Slow Sterotyped
Arm across chest
Forearm rotates
Thumb clenched
Leg extends

Normal flexion
Rapid
Variable
Arm away from body

For further information and video demonstration visit www.glasgowcomascale.org

Table 2. FOUR *(Full Outline of UnResponsiveness)* scale.

Eye Response	
E4:	Eyes tracking horizontally or vertically, or blinks twice to command
E3:	Eyelids open but not tracking
E2:	Eyelids open to loud or painful stimuli
E1:	Eyelids open to pain
E0:	Eyelids remain closed with pain
Motor Response	
M4:	Gives thumbs-up, clenches fist or makes peace sign to command
M3:	Localises pain (applying a supraorbital or temporomandibular stimulus)
M2:	Flexion response to pain (includes decorticate posturing and withdrawal responses) in upper extremities
M1:	Extensive response to pain
M0:	No response to pain or generalised myoclonus status
Brain stem reflexes	
B4:	Pupil and corneal reflexes present
B3:	Pupil reflex absent on one side
B2:	Pupil or corneal reflexes absent
B1:	Pupil and corneal reflexes absent
B0:	Absent pupil, corneal and cough reflexes
Respiration	
R4:	Not intubated, regular breathing pattern
R3:	Not intubated, Cheyne-Stokes breathing pattern
R2:	Not intubated, irregular breathing
R1:	Intubated, breathes above ventilator rate
R0:	Intubated, breathes at ventilator rate or apnoea

arms or legs and moving their fingers, or with more elaborate actions such as touching the tip of their nose with their right index finger [5].

If they obey the order given, they get a score of 6.

If they do not obey, a painful stimulus is applied (pressure to the trapezius or supraorbital notch for 10"). If they try to alleviate the pain by locating the point that hurts, they are given 5 points.

If the pain causes them to move and try to withdraw from the stimulus, they score 4.

If the pain causes an abnormal flexion of the arms, the score is 3. This may indicate decorticate posturing, which can manifest itself as stiffness with bent arms.

If the pain causes them to stretch out their arms and hands, it is indicative of decerebrate posturing and the score given is 2.

If the painful stimulus does not cause any movement: 1 point.

The sum of each of these points gives a score between a maximum of 15 points and a minimum of 3 points. The following classification is made according to this scale:

- Mild TBI. GCS score of 15-13 points.
- Moderate TBI. GCS score of 12-9 points.
- Severe TBI. GCS score of < 9 points.

It is important to carry out this assessment before sedating or paralysing the patient[6,7].

Interpretation of the Glasgow Coma Scale

According to the score obtained with the Glasgow Coma Scale, three levels of TBI severity are established: mild, moderate and severe.

Mild head trauma: Score of 13-15. The patient has experienced an alteration in their level of consciousness or unconsciousness lasting less than 30 minutes. The symptoms or signs they show are headache, confusion and amnesia. There is complete neurological recovery even though some of these patients have transient concentration or memory difficulties.

Moderate head trauma: Score of 9-12. The patient is clinically lethargic or stuporous. Patients with moderate TBI require hospitalisation and can develop post-concussion syndrome, manifesting a state of neurological instability after a mild or moderate TBI. The symptoms may include dizziness, headache, fatigue and difficulty concentrating.

Severe brain trauma: Score of 3-8. The patient is in a comatose state, is unable to open their eyes or follow orders and is suffering from significant neurological injuries. Structural injuries such as skull fracture or intracranial haemorrhage are usually seen in computed tomography (CT) scans. These patients require admission to the Intensive Care Unit (ICU) or neurosurgical treatment. Recovery is lengthy and usually incomplete. A significant percentage of patients with severe TBI do not survive more than one year.

Other neurological assessment scales

The Brussels Coma Score, Grady Coma Scale and Innsbruck Coma Scale are not widely used because they do not have good inter-observer reliability and have lower predictive capacity than the Glasgow Coma Scale.

One scale to consider, as an alternative in intubated patients, is the FOUR scale (Full Outline of UnResponsiveness) developed by Wijdcks *et al*[8], which could prove very useful in coma assessment because it covers other brain stem functions. This scale evaluates four aspects: eye opening, motor response, pupillary and corneal reflexes and respiratory pattern (Table 2).

There is another simpler assessment scale which is in disuse as far as health professionals are concerned but which could be of interest to those who are not (athletes, coaches, physical trainers, etc.) thanks to its simplicity: the AVPU scale.

An estimation of a patient's level of consciousness is arrived at by observing their behavioural response to different stimuli.

- A: Alert.
- V: Responds to verbal stimulus.
- P: Responds to painful stimulus.
- U: Unresponsive to stimuli.
- The assessment procedure is simple after a TBI:
- If the patient is awake and talking to the assessor, they are categorised as A (alert), even if they are disoriented.

- If the patient is not fully awake, it is necessary to see if they respond to the assessor's voice. For example, by opening their eyes, talking or moving. If they do, they are V (responding to verbal stimuli).
- If the patient does not respond to the voice, apply a painful stimulus, such as pressing or pinching the trapezius and checking for a response (opening of eyes, moaning or movement). If there is a response, the patient is classified as P (responsive to pain).
- Those who do not respond are U (unresponsive).

Therapeutic strategy for different situations

At the scene

The initial management of a TBI seeks to identify patients at vital risk and provide adequate assessment and diagnosis in order to avoid the development of possible complications[9].

TBI is often part of the context of a polytrauma patient, and any patient who has sustained a TBI should be evaluated for trauma with indication of surgical intervention. The ITLS *(International Trauma Life Support)* assessment in the extra-hospital environment and the ATLS *(Advanced Trauma Life Support)* assessment at hospital level set the standard for action in the event of polytrauma.

This procedure includes the stabilisation and control of the cervical spine as soon as possible either manually or with a cervical collar.

Once the possible injuries which could put the life of the injured person at risk are under control, the neurological assessment should include the patient's level of consciousness based on the Glasgow Coma Scale (GCS) and the presence of anterograde or retrograde amnesia. Any signs of focal neurological deficit should also be detected, including: pupillary size and responsiveness, limb paresis and cranial nerve deficit.

It is also important to perform a detailed inspection of the skull and face, looking for contusions, wounds, embedded objects and deformities which may suggest fractures or cranial collapse.

Otorrhagia, orbital haematomas, mastoid ecchymosis (Battle's sign) and cerebrospinal fluid otorrhea and rhinorrhoea are indicative of skull base fracture.

It is also very useful to know, if possible, the injury mechanism involved because it can provide information on the energy generated in the trauma and the damage we should expect, which may not be evident at the time.

Finally, it is advisable to know some aspects of the patient's previous medical history, such as allergies, current treatments (anticoagulants, etc.) and toxic habits.

We know that the symptoms which may arise as a result of a TBI are varied, but it is important to be aware of the signs which should make us suspect intracranial structural injury, whether they manifest themselves from the start or later on.

Warning signs:

- Progressive decline in or loss of consciousness, modification of GCS score.
- Alteration in behaviour.
- Focal neurological deficit.
- Constant amnesia over time.
- Presence of seizure.

Complications should also be suspected when associated with:

- High-energy accidents.
- Persons over 65 or under 2 years of age.
- Alterations in coagulation.
- Consumption of alcohol or narcotics.

Referral and transfer

Most head injuries do not present any neurological symptoms. In these cases and after a thorough study and assessment, GCS 15, it is not necessary to refer them to a health centre, although supervision is recommended for 24/48 hours in case warning signs appear.

Regardless of their GCS, patients with TBI should be transferred to a hospital which has permanent comprehensive care with 24-hour Computed Tomography (CT) scanner, ICU and 24-hour neurosurgical service (level 3 hospital) if the following circumstances arise:

- Presence of neurological focality.
- Presence or suspicion of cranial fracture-collapse.
- Non-immediate post-traumatic seizure.
- TBI as a result of high-energy accidents.
- Cranial CT scan identifying the appearance of a recent traumatic injury.
- Whenever there is any doubt regarding the diagnosis or it is thought that evaluation, monitoring or neurosurgical treatment may be required.
- Over-65s.
- Anticoagulant treatment or coagulation disorders.
- In paediatric ages which present altered behaviour or irritability.

All TBIs with a GCS under 13 should be transferred by Advanced Life Support (ALS) ambulance with specific monitoring and treatment capacity, as should all patients presenting:

- Warning signs.
- Cranial wound with embedded object, cranial collapse.
- Suspected skull base fracture.
- Polytrauma.
- High-energy accident.

TBIs with GCS over 13 can be transferred in a Basic Life Support (BLS) Ambulance.

Hospital level

In the hospital emergency department, the care provided previously should be continued and the diagnostic and therapeutic measures unavailable in a non-hospital environment should be taken.

Computed tomography (CT) scanning is the test of choice for the

diagnosis of acute intracranial injury and is sometimes complemented with cervical spine radiology, especially when there are signs of suspected cervical spine involvement. Indications of when CT scans should be performed are described in Table 3.

The relevant therapeutic measures should be taken according to the level of consciousness indicated by GCS, which will again be reassessed at this stage.

Table 3. Computed tomography (CT) indication assessment.

Category	Characteristics	CT (yes/no)
Mild		
1	GCS: 15	NO
2	GCS: 15 + warning signs	YES
3	GCS: 13-14 with/without warning signs	YES
Moderate	GCS: 9-12	YES
Severe	GCS: ≤ 8	YES

Mild TBI with GCS of 15

The patient should be kept under observation for 4 hours and can be discharged for 24 hours if no neurological deterioration is appreciated. If any warning sign appears or the patient's level of consciousness declines during that time, a CT scan should be performed with evaluation by neurologists.

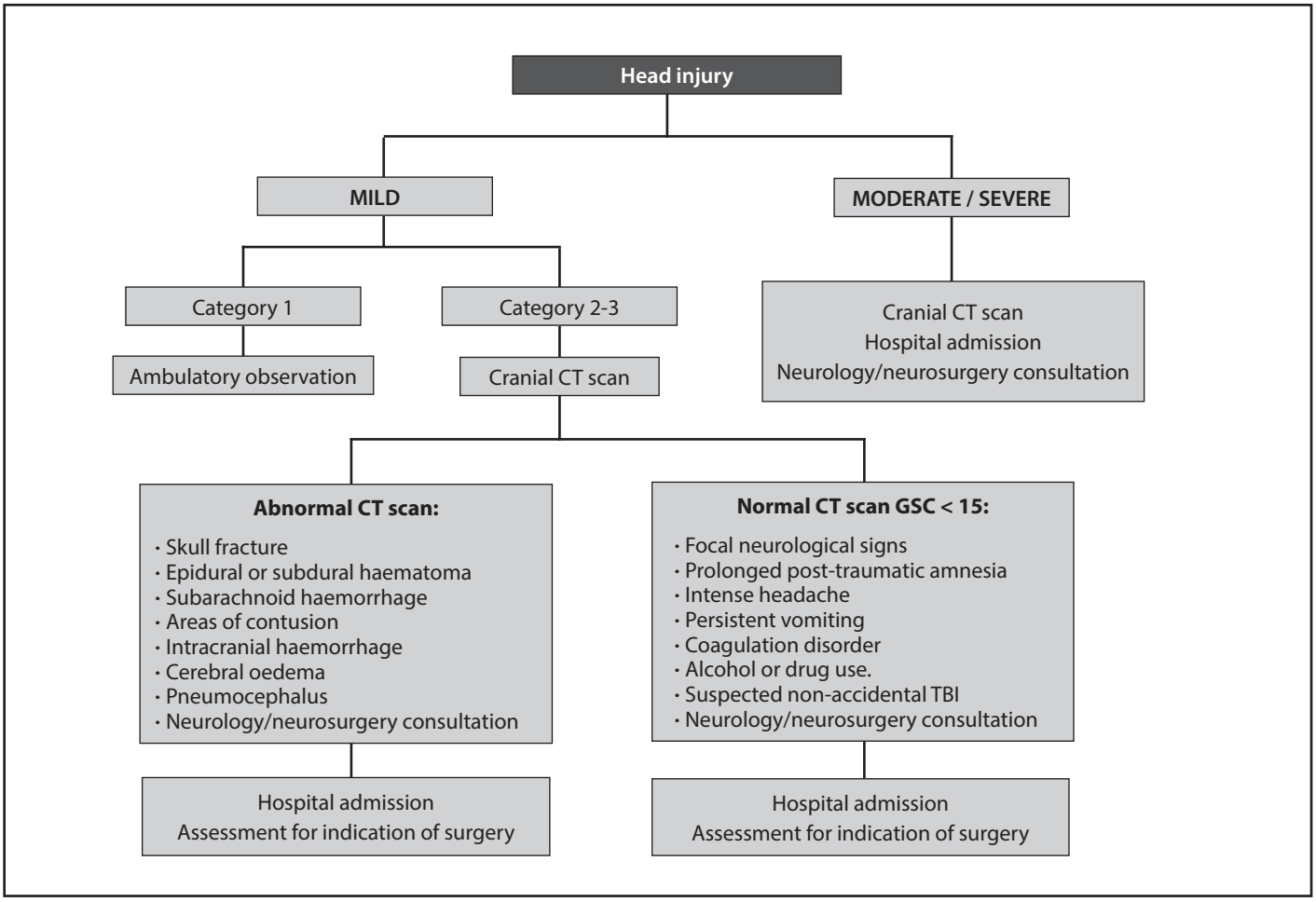
Mild TBI with GCS of 15 presenting a warning sign over time

A CT scan should be performed, the neurology service should be consulted and the patient should be kept under observation for 24 hours, monitoring vital signs and periodically re-evaluating level of consciousness (GCS) or the appearance of neurological focality.

Some type of antiseizure treatment may also be given and a neurosurgical evaluation should be carried out.

If the warning signs appear in patients over 65 years of age, there is neck pain or pain on examination, the patient has suffered a high-energy accident or fall from height, a three-projection radiological study of the cervical spine should also be requested.

Figure 2. Strategy of action when faced with a traumatic brain injury (TBI).



Mild TBI with GCS of 13-14

A cranial CT scan and evaluation by the neurology service is always required.

In these cases, the presence in the CT scans of signs of structural involvement such as cranial fracture, epidural or subdural haematoma, subarachnoid haemorrhage, intraparenchymal haemorrhage with signs of contusive foci or pneumocephalus requires an urgent neurosurgical evaluation in order to establish the therapeutic measures to deal with the injuries.

In the case of a normal CT scan in which warning signs persist, an examination by the neurology service will also be necessary in order to determine the action to be taken, including hospital admission.

In the presence of moderate or severe TBI, coordination of the emergency, radiodiagnosis, neurology, neurosurgery and intensive care departments is required to provide the best therapeutic response and ensure the best possible neurological outcome.

Figure 2 describes the algorithm of action for a traumatic brain injury (TBI)[9,10].

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Indications for surgery

Indicaciones quirúrgicas

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Introduction

The therapeutic management of traumatic brain injury (TBI) will depend on the clinical situation of the patient, the severity of the TBI (measured by the Glasgow Coma Scale – GCS) as well as the presence of structural lesions or physiopathological changes in the brain that could have a significant impact on intracranial content.

Most TBIs are mild and are treated by neurological monitoring and/or medical treatment. However, severe TBIs with structural intracranial lesions or that involve a solution of continuity between the central nervous system and the external environment may require surgery.

To better understand the indications for surgery in TBIs, the subject will be addressed according to the different lesions or complications that may occur following TBI.

Cranial fractures

Cranial vault fractures

Most cranial vault fractures are linear and have no repercussion on the intracranial structures. However, some fractures do require surgery, such as open fractures in which there is a solution of continuity between the intracranial content and the external environment and, therefore, an important risk of infection; or depressed fractures that could cause injury to the underlying brain parenchyma, meninges or intracranial blood vessels, as well as major cosmetic defects[1]. In these cases, it is important to remove bone splinters and foreign bodies, repair the damaged structures and reconstruct the morphology of the skull, either by joining the bone fragments or, if this is not possible, through the use of titanium mesh or the application of bone cement.

Cranial base fractures

Most cranial base fractures are linear and extend through a fracture of the calvarium. Although they do not generally require surgery, the treatment of some conditions associated with fractures of this type may be necessary:

- *Cerebrospinal fluid (CSF) fistulas:* CSF fistulas increase the risk of infectious meningitis and intracranial hypotension syndrome. CSF fistulas are more common in the anterior cranial fossa (at the level of the lamina cribrosa) and through the sphenoid sinus, with rhinoliquorrhea being a characteristic sign. Fractures of the petrous bone may cause fistulas with otoliquorrhea, the leakage of CSF towards the ear. CSF fistulas can be treated conservatively through the placement of a lumbar drain or through the surgical sealing of the defect.
- *Post-traumatic facial nerve lesion:* This occurs as a result of a fracture of the petrous bone. Petrous bone fractures can be longitudinal (70-80%) or transverse (20%), the latter being the cause of most traumatic lesions of cranial paired nerves VII and VIII. Surgical treatment of the facial nerve lesion consists in nerve decompression and is performed in the event of progressive post-traumatic facial palsy or when conservative therapies have not been successful.
- *Vascular disorders:* Post-traumatic aneurysms, carotid-cavernous fistulas and vascular sections are prominent. Vascular lesions are serious complications and are generally associated with clivus fractures.

Fractures of the anterior cranial base affecting the frontal sinus are relatively frequent in contact sports. Fractures of this nature are generally accompanied by CSF fistulas and pneumocephalus. Furthermore, the presence of a connection between the upper respiratory tract and the intracranial cavity increases the risk of infection by germs from the nasal

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microbiome. Fractures affecting the frontal sinus anterior wall are treated conservatively, unless there is a major anatomical defect; while posterior wall fractures carry a greater risk of CSF fistulas and, consequently, of infection (subdural empyemas, brain or mucocele abscesses) In these cases, surgery is recommended. The operating procedure consists in the removal of the posterior wall of the frontal sinus, a process termed cranialization; subsequently mucus marsupialisation is performed towards the nasofrontal duct and, finally, the duct is plugged with sealants, muscle flaps or even fat.

Tension pneumocephalus

Pneumocephalus, also known as pneumotocele, consists in the presence of air in the intracranial cavity and generally appears in open cranial fractures or when a CSF fistula occurs. In general, there are no symptoms. Most cases are either resolved spontaneously or following oxygen therapy with FiO_2 at 100%[2]. In some cases (cranial fractures or after a craniotomy) a valve effect occurs, permitting air to enter but not to exit the intracranial compartment, leading to a sharp increase in intracranial pressure (ICP). This phenomenon is termed pneumocephalus and must be treated by surgery whenever it causes severe neurological symptoms. Surgery consists in the release of the pressurised air either through burr hole trephination or by puncturing a pre-existing closed burr hole.

Epidural haematoma

Epidural haematoma occurs in approximately 14-35% of patients with severe TBI[3]. It generally affects young patients, either because it is the age group that is most prone to suffer TBIs with a moderate-high impact (for example, sports accidents) or because the dura is more attached to the inner table of the skull than in older patients.

In most cases, the source of the haemorrhage is the lesion of the middle meningeal artery as it exits the pterion groove, as a result of a temporoparietal fracture. In this context, arterial bleeding occurs, which strips the dura away from the inner table of the skull causing an intracranial mass effect. Less than 10% of cases of epidural haematoma are located in the frontal region, occipital region or in the posterior fossa. In such cases, the source is either the lesion of the meningeal veins or the dural sinuses.

An epidural haematoma represents a surgical emergency in most cases. Epidural haematomas in children are more dangerous than in adults, given that children have a smaller intracranial volume. For this reason, it is recommended to establish a lower surgical treatment threshold for patients in the paediatric age group[4].

Following the practical recommendations with evidence level III , surgery must be performed on epidural haematomas with a volume of more than 30 cubic centimetres regardless of the patient's clinical condition, measured by the GCS, clots with a maximum thickness of more than 15 millimetres, a haematoma that causes a midline shift of more than 5 millimetres, patients with epidural haematoma evidenced by CT imaging and a score of less than 9 on the GCS, and patients with an epidural haematoma and presence of neurological focal deficits[5]. Likewise, with evidence level III, immediate surgical evacuation is strongly recommended in those cases of acute epidural haematoma with a GCS score of less than 9 and anisocoria.

The standard operating procedure for epidural haematomas consists in the performance of a craniotomy, clot evacuation, achieve appropriate haemostasis (in most cases it is necessary to clot the bleeding vessel) and prevent haemorrhagic recurrence through the placement of stay sutures on the dura mater.

For asymptomatic patients and for small epidural haematomas, conservative therapeutic management is possible[6]. In these cases, it is important to ensure neurological monitoring and the performance of serial CT scans of the brain. The use of corticosteroid drugs in a descending dose, is controversial.

Acute subdural haematoma

An acute subdural haematoma is a surgical emergency. These haematomas are frequently associated with injury in the underlying brain parenchyma and can cause symptoms, either due to the mass effect exerted by the haematoma or to the direct injury to the bran parenchyma after impact. The source of the subdural haematoma could be due to the accumulation of blood around the parenchymal laceration site or due to the rupturing of a superficial vessel as a result of the rapid acceleration-deceleration forces suffered by the brain during the movement of the head. As a result, acute subdural haematomas have a high morbidity/mortality rate, and even more so when surgery is not performed right away. In fact, it is estimated that mortality for acute subdural haematomas treated within the first four hours since the traumatic event is 30% while, after this time, mortality increases up to 90%. It should be mentioned that secondary cranioencephalic trauma caused by a motorbike accident has a worse prognosis, particularly for patients not wearing a helmet.

Chronic treatment with antithrombotic drugs, and anticoagulants in particular, considerably increases the risk of acute subdural haematoma. This is important in older athletes.

Following the practical recommendations with level of evidence III, surgery must be performed on acute subdural haematomas with a thickness of more than 10 millimetres or that cause a midline shift of more than 5 millimetres regardless of the patient's clinical condition, measured by the GCS, as well as acute subdural haematomas that have a thickness of less than 10 millimetres or a midline shift of less than 5 millimetres if the patient drops two or more points on the GCS since the time of the trauma up to the time of the medical care at the hospital, or if there are alterations in the pupil reflex (anisocoria, fixed mid pupil or bilateral mydriasis) or if the ICP is greater than 20 millimetres of mercury[7]. With level III evidence, it is recommended to monitor the ICP in all patients with acute subdural haematoma and a GCS score of less than 9[8].

Surgical procedure consists in performing a craniotomy, opening the dura mater, evacuation of the subdural clot and blood vessel haemostasis. In the event of the laceration of the cerebral cortex, it is possible to remove the contused brain parenchyma, considering the risk/benefit and possible neurological sequelae if it is an eloquent area. The dura mater can be sealed by direct suture, with flaps/plasties or, if there is considerable brain swelling, there is the possibility of not performing duroplasty or even craneoplasty. In the same way as for epidural haematomas, the placement of stay sutures on the dura mater prevents the appearance of epidural collections.

Chronic subdural haematoma

Chronic subdural haematomas have a pathophysiological mechanism that is different to that of acute subdural haematomas. Although it is likely that chronic subdural haematomas were initially acute subdural haematomas (in most cases there is a prior TBI), chronic subdural haematomas exhibit an inflammatory clotting response that leads to the formation of membranes and to the proliferation of neovessels. In most cases, when blood becomes chronic, it undergoes an enzymatic degradation process and liquefies, losing volume. The shrinkage of the clot volume leads to the tearing of small subdural neovessels, giving rise to fresh bleeding (re-bleeding) and to the development of a new subdural haematoma. This process may be repeated a number of times, creating very large subdural haematomas.

Chronic subdural haematomas are frequent in older adults and with factors predisposing to bleeding (antithrombotic drugs, chronic alcoholism, coagulopathies, etc.)[9], and are therefore infrequent in athletes. Likewise, very large chronic subdural haematomas are common in older patients and/or with brain cortical atrophy, given that there is a larger subdural space for the haematoma and they better tolerate re-bleeding until the onset of symptoms.

Surgical treatment is required for chronic subdural haematomas with a maximum thickness of more than 1 centimetre or in cases of focal neurological deficits or altered mental condition.

Post-traumatic brain haemorrhage

Post-traumatic brain haemorrhages or cerebral haemorrhagic contusion result from acute bleeding in the brain parenchyma. Post-traumatic brain haemorrhages are more frequent at locations in which the sharp deceleration forces of the head cause the brain to impact against bony protuberances (for example the frontal lobes, temporal poles and occipital lobes)[4]. A delayed post-traumatic brain haemorrhage can occasionally occur, in other words outside the first 72 hours; this is due either to blood extravasation towards the necrotic tissue, the convergence of various microhaemorrhages or to coagulopathies. It is important to perform serial imaging studies in order to detect bleeding of this type.

Following the practical recommendations with level of evidence III, post-traumatic parenchymal haemorrhages should be surgically treated when a patient has a score of 6-8 on the GCS or exhibits progressive neurological deterioration and has a volume of more than 20 cubic centimetres in the temporal lobes or greater than 50 cubic centimetres in any location, causing a midline shift of more than 5 millimetres and/or showing imaging signs of brain herniation. Surgery must also be performed on those post-traumatic brain haemorrhages causing neurological deterioration and exhibiting intracranial hypertension that is resistant to pharmacotherapy[10].

Surgical procedure consists in performing a craniotomy and the evacuation of the cerebral parenchymal haematoma. It is important to ensure good haemostasis and the application of anti-oedema measures, given that in many cases oedema is associated with the cerebral parenchymal contusion. In the event of a cerebral oedema or external cerebral herniation (through the craniotomy) decompressive craniectomy should be performed[11]. Likewise, the risk/benefit of a surgical operation should be assessed with regard to the patient's vital/functional condition, all the more so when the haemorrhage is located in eloquent areas. In these cases, it is important to assess the prior functional condition, potential comorbidities and the psychosocial situation of the patient.

Cerebral oedema (swelling)

Cerebral oedema or *swelling* appears as a result of structural damage to the nerve tissue, not only in the neuronal cells themselves but also at the hematoencephalic barrier, producing a mixed vasogenic-cytotoxic oedema. In most cases, cerebral oedema is associated with a primary cerebral lesion, and haemorrhagic contusions in particular. In other cases, a delayed oedema appears as a result of secondary lesions such as microhaemorrhages, cerebral infarctions, or due to changes in the metabolic mechanisms for the self-regulation of the brain.

The surgical indications are a point of contention. According to the practical recommendations with level of evidence III, the performance of decompressive craniectomy is recommended for patients with post-traumatic diffuse cerebral oedema and a structural lesion in the cerebral parenchyma or exhibiting intracranial hypertension that is resistant to pharmacotherapy. The procedure must be performed within the first 72 hours after the traumatic event, being more effective when performed earlier on or whenever the failure of the cerebral autoregulation mechanisms is observed through an increase in the ICP[12]. The DECRA study reported an increase in the survival of patients with severe TBI and refractory intracranial hypertension, treated through decompressive craniectomy[13]. However, the increased survival rate is closely related to an increase in neurological disability[14]. In these cases, the risk/benefit of the surgical operation must be assessed, and particularly in older patients or those with a poor baseline condition.

Post-traumatic hydrocephalus

Post-traumatic hydrocephalus consists in the excessive pathological accumulation of CSF in the cerebral ventricles following TBI. It generally appears weeks of months after TBI, particularly in the case of a severe

TBI. When the hydrocephalus appears more than 6 months, or even years, after the traumatic event, it is difficult to differentiate it from the ex-vacuo hydrocephalus secondary to a diffuse axonal injury.

Standard surgical procedure to treat post-traumatic hydrocephalus consists in a ventricular shunt placement[15]. Post-traumatic hydrocephalus must be treated when there are signs of intracranial hypertension, papilledema, the presence of ventriculomegaly and transependymal oedema in the imaging tests, high values of ICP in one or more lumbar punctures, or positive scores in the provocation test[16].

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The prevention of traumatic brain injury

Prevención del traumatismo craneoencefálico

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Introduction

Although in sports in general, and in contact sports and speed sports in particular, there is a risk of sustaining traumatic brain injuries either due to sport-specific mechanisms or to other accidental mechanisms, prevention strategies can considerably reduce their occurrence, intensity and structural and functional injury. These measures span a broad spectrum, ranging from an education in values directed at athletes up to changes in the sports regulations, the use of protections and the materials used in sports[1,2].

Use of protections

The concept of sports protection includes the use by athletes of protective items such as a helmet or mouthguard, as well as safety guards, padding and coverings for static equipment on the playing field, such as the basketball base and the goalpost bases on the rugby pitch.

Sports regulations for numerous contact and speed sports require the use of specific helmets for each type of sport (boxing, martial arts, American football, ice hockey, Alpine skiing, cycling, etc.) given the evidence on their preventive and protective effect against traumatic brain injury, although not all studies have demonstrated their protective effect against structural brain damage[3,4]. Over the last decade, the use of new sports helmet designs and materials has successfully increased protective performance against different impact intensities and linear acceleration (establishing for example the risk of fracture at impacts greater than 200-300 g.). However, there is a need to consider other factors such as rotational acceleration and the Gadd Severity Index (which relates the duration of the impact producing cerebral motion with the magnitude of the cerebral translation) in order to achieve more specific protection with regard to isolated intense trauma cases and to repetitive cases of a lower magnitude[3,4].

Changes in the regulations

Another important prevention strategy is the adaptation of the sports rules and regulations in order to reduce trauma injuries by direct contact between players, as well as to optimise medical assistance in those cases in which an accident occurs.

In some sports, such as American football, the change in the regulations prohibiting direct head contact among players is a good example of the repercussion that regulation changes can have on a reduction in injuries and repercussions on the health of the athletes[5].

In this same regard, it would also be of interest to introduce regulatory changes that would permit a speedier medical intervention in traumatic events in which treating the athlete is a race against time.

On the other hand, in the case of football, the regulatory change in the size and weight of the ball could be a good strategy in reducing the repercussions of impact during ball-heading, particularly for junior players and women's football[6].

Preventing violence in sport

Competitiveness and aggressiveness are two positive qualities for athletic performance, providing that they do not lead to non-sporting or violent attitudes against the adversary. Violence in sport is frequently manifested in aggressive behaviour towards the opponent, thereby increasing the risk of sports injuries and, in particular, the risk of more serious injuries such as the case of traumatic brain injury.

While the promotion of fair play is important as a positive and educational strategy, of equal importance is the adoption of strict sanctioning measures against violent attitudes of this nature, including such measures in the rules and regulations of the sports in which actions of this type may occur. These measures should not only be directed at athletes but

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also at any team members who encourage this type of violent attitudes and actions[7].

The transfer of knowledge

The implementation of educational schemes to educate athletes, trainers, fitness coaches and healthcare person in the basic concepts and coordination in emergency situations in the event of a traumatic brain injury is of the utmost importance for the correct care of injured athletes[1].

Likewise, the conveyance of ethical values, values of respect for opponents and fair play, is a key factor in the prevention of sports injuries in general, and for traumatic brain injuries in particular[1,8]. Furthermore, this educational and formative strategy should not only be directed at athletes but also at their family and social environments. Along these lines, informative videos, supportive websites, dissemination strategies through the social media, can all be very useful and effective tools to achieve this purpose.

Visual-motor-sensory training

One of the most innovative preventive strategies is virtual visual-motor-sensory training. These techniques, which were initially designed for the rehabilitation of patients with brain injuries, are now being implemented for the improvement of athletic performance, having been proven to be effective in traumatic brain injury prevention.

Visual-motor-sensory training is performed through virtual exercises that positively stimulate the neuroplasticity of the brain through vestibular exercises, ocular motor activities, cervical control, and coordination and postural balance movements. The results have shown improvements in movement and balance control, with an effect on an improvement in performance and on traumatic brain injury prevention[9,10].

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Return to training and play following concussion or traumatic brain injury

La vuelta al entrenamiento y la competición tras una conmoción y traumatismo craneoencefálico

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Return to play following Brain concussion or Traumatic Brain Injury (TBI) is a constantly evolving subject. 20 years ago, return to play was a dichotomous decision, where it was left to the athlete to decide whether or not to return to unrestricted sports practice once the symptoms had disappeared[1]. This type of management of the return to sports practice was associated with further concussions in the same season, most of which occurred in the first 10 days after the injury[2]. In year 2000, a document put forward the idea of a gradual return to sports practice (RTP or Return to Play)[3], which was adopted by the Concussion in Sport Group at the first world conference in 2001[4]. This RTP protocol was revised and modified at the Concussion in Sport world conference held in 2016[5].

RTP is a protocol to be implemented following concussion in which there is a gradual increase in the sport-specific physical demands. In this respect, athletes are required to be symptom-free before beginning the first stage of the RTP protocol[5,6]. This RTP protocol has 6 stages (Table 1) and permits a symptom-free athlete to progressively advance stage by stage. Each stage must have a 24- to 48-hour duration. Should a symptom occur, then the athlete must return to the previous stage and remain symptom-free for at least 24 hours before advancing to the following stage. Although the RTP protocol is widely accepted, it was empirically prepared and, therefore, studies need to be made in order to assess the effectiveness of the advancement in stages, as well as the duration of the same[6].

The consensus statements on managing sport-related concussion recommend rest as part of RTP[6,7]. Rest is justified for at least three reasons. Firstly, rest probably reduces post-concussion symptoms, easing discomfort during the acute recovery period by mitigating post-concussion symptoms. Secondly, after concussion, rest may promote recovery by minimizing brain energy demands during metabolic and haemodynamic recovery at a neuronal level[8]. Thirdly, in the first 7-10 days following concussion, athletes have an increased risk of sustaining another concussion[9]. However, studies have not clearly demonstrated the benefit of strict rest in recovery after concussion[7,10]. Although there are currently no studies to clarify the optimal rest time[11], there is consensus on recommending a 24- to 48-hour period of relative physical and mental rest[5,6,12].

After a brief period of rest, activity should be resumed below cognitive and physical symptom-exacerbation thresholds. Exercise has an effect on the nervous system, improving: the balance of the autonomic nervous system, CO_2 sensitivity, increasing the expression of brain-derived neurotrophic factor genes, the state of mind and sleep[13,14]. Due to changes in the functioning of the autonomic nervous system and the control of cerebral blood flow in brain concussion[15,16], exercise may help in recovery. Studies have shown that symptom sub-threshold exercise improves recovery after brain concussion[17-20], this exercise intensity threshold can be determined through a test performed on a treadmill or exercise bike[21,22]. Due to this evidence, aerobic exercise and activity in the early stages of recovery following concussion do not replace RTP and must be integrated into the latter.

There must be no contact in stages 1-4 of RTP, and an athlete must be symptom-free in order to advance to stage 5 where contact exercises are introduced. As a result, medical assessment is required to give authorisation to advance to contact exercises. In this regard, neurocognitive tools such as SCAT[5] or similar are useful for this assessment[5,12]. This RTP

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Table 1. Strategy for Gradual Return to Play (RTP).

Stage	Rehabilitation stage	Exercises to be performed	Objective of the stage
1	Symptom-limited activity	Daily activities that do not provoke symptoms	Gradual reintroduction of work/school activities
2	Light aerobic exercise	Light running, swimming, static cycling, at a slow to medium pace	Increase heart rate
3	Sport-specific exercise	Running drills, no head impact activities	Add movement
4	Non-contact training drills	Progression to more complex training drills (e.g. passing drills). May start progressive resistance training	Exercise, coordination and cognitive load
5	Full contact practice	Normal training activities	Restore the athlete's confidence and allow trainers to monitor skills
6	Return to play	Rehabilitated player	Complete physical and cognitive recovery

Table 2. Consensus Recommendations.

Recommendations	Class of evidence	Level of evidence
6-stage RTP protocol.	I	C
Physical and mental rest for 24-48 hours following brain concussion.	I	C
Start aerobic exercise after a 24- to 48-hour period of rest, following brain concussion at an intensity below the symptom threshold, as part of the RTP.	IIa	B
Perform RTP under supervision (rehabilitator or physiotherapist).	IIa	C
The athlete must be assessed and authorised by a specialist doctor with experience in brain concussion before moving on to stage 5 or starting contact exercises.	I	C
The minimum time required to advance to stage 5 or to the start of contact exercises must be 12 days for adult athletes.	IIb	C
For athletes under 19 years of age, the minimum time required to advance to stage 5 or to the start of contact exercises must be 28 days.	IIb	C
Athletes under 19 years of age must have resumed normal educational activities before advancing to stage 5.	IIa	C

RTP: gradual return to play.

must be performed under supervision (rehabilitator or physiotherapist) and must never be done alone[20]. The time taken for the RTP varies, depending on the athlete's age and medical history, and must be managed on an individual basis. The time described for elite athletes to return to play following brain concussion ranges from 7 to 10 days[23-25]. However, recent studies on American football players reported a time of 12 days[26] and close to 30 days for military athletes[27]. In a review conducted, it was reported that the physiological recovery of the central nervous system takes from 15 to 30 days, while symptom resolution took less time[28]. Furthermore, it has been reported that there is a greater probability of sustaining further concussion in the first 10 days of recovery[2].

On the other hand, the reported recovery time for adolescents and children is greater, being approximately 4 weeks[29]. Furthermore, students should not return to normal sports practice until their school attendance is back to normal[5]. However, due to a scarcity of studies, it is not possible to determine the age at which recovery as an adult can be considered. Based on this evidence, it is reasonable to consider that there must be a minimum period of 28 days and students must have resumed normal school life before starting contact exercises. Considering current evidence, as a consensus we would recommend (Table 2):

- Perform a 6-stage RTP protocol.
- Ensure physical and mental rest for 24-48 hours following brain concussion.
- Start aerobic exercise after a 24- to 48-hour period of rest, following brain concussion, at an intensity below the symptom threshold, as part of the RTP.
- Perform the RTP under the supervision of a rehabilitator or physiotherapist.
- The athlete must be assessed and authorised by a specialist physician with experience in brain concussion before moving on to stage 5 of the RTP protocol or starting contact exercises.
- The minimum time required to advance to stage 5 or to the start of contact exercises must be 12 days for adult athletes.
- For athletes under 19 years of age, the minimum time required to advance to stage 5 or to the start of contact exercises must be 28

days. Moreover, athletes must have resumed normal educational activities before advancing to stage 5 or to the start of contact exercises.

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Conclusions of the "Avilés" Working Group on Sports Medicine

XI Conference. Barcelona, October 2022

The conference was organised by the Department of Sport and Health of the Spanish Commission for Anti-doping in Sport (CELAD) and by the *Secretaria General de l'Esport i l'Activitat Física* (SGEAF - Catalan General Secretariat of Sport and Physical Activity) through the CAR (High Performance Centre) of Sant Cugat and the *Consell Català de l'Esport* (Catalan Council of Sport). The conference was held at the High Performance Centre of Sant Cugat and at the Colet Museum of Barcelona.

CONCLUSIONS:

- Insistence on the importance of a medical examination prior to sports participation, directed at preventing Sudden Death. A sport-medical check-up that includes anamnesis, physical exam and ECG is essential as a starting point. The performance of more specific tests such as an echocardiogram or effort tests would be recommended as complementary for athletes aged over 14 or 15 years or for a suspected cardiac pathology.
- "Extreme" endurance exercise, continued for many years, can cause changes in the cardiac structure and function and create an arrhythmic substrate. For this reason, and particularly for athletes practising sports of this type, it is important and necessary to carry out regular heart examinations.
- The myocarditis rate due to SARS Cov-2 is low and, in general, mild. Following vaccination, the rate is even lower, affecting the 18 to 25- year age band. The practice of sport is not recommended during infection. However, the prescription and practice of sport is in fact a great help in the post-COVID patient recovery process.
- With regard to infection from SARS Cov-2, no increase in sudden death in sport has been observed.
- The performance of effort tests must be standardised, according to the criterion of the sports medicine specialist, although it is important to maintain the use of face masks by healthcare personnel in all cases and to conduct the tests in places that are well-ventilated and with good air renewal. In the case of a symptomatic athlete, a prior diagnostic test must be made.
- Attention is drawn to the importance of prevention in sport in general and in high-level sport in particular. It is worthy of note that athletes cannot achieve a high performance if they are not healthy.
- There is a need to advance in the prescription of physical exercise for mental health. Given that physical activity modulates neuroplasticity and stimulates therapeutic adherence, its use is recommended in the treatment of mental health disorders, with an individualised prescription for each patient.
- It is important to take account of the studies and contributions being made in our society on sexual and gender diversity, sport and its implications.
- Sports medicine units should be linked to the hospital departments together with the rest of the medical specialties due to the added value they bring to the healthcare of our athletes, whatever their level, and any doing physical exercise in general, as well as their usefulness as a preventive and therapeutic tool for the different pathologies.
- In Spain, the current lack of training of sports medicine physicians has resulted in a serious shortage of these specialists and this will continue to worsen in the forthcoming years if a MIR (Resident Medical Intern) training system in this specialty is not approved in the near future.
- This shortage is having a considerable impact on the entire sports system, on the health of the athletes in general, and on high-level athletes in particular. A country with important international sports results cannot afford to part with these professionals who are making a substantial contribution to the healthcare of our athletes and to the improvement of their performance. Likewise, the training of Sports Physicians is the specialist benchmark for an active and healthy society with safe sports practices.
- Yet again the "Aviles" Working Group would urge the Ministry of Health to approve, as a matter of priority and urgency, the training of Sports Medicine Physicians.

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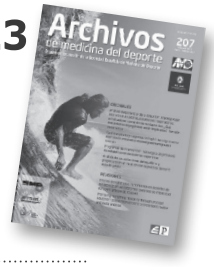

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