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REVIEWS

Sport-related concussion

Comoția cerebrală în sport

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Abstract

Sport-related concussion is a complex brain injury, manifesting through a very large variety of brain dysfunction signs and symptoms (physical and cognitive deficit, abnormal behavior). The symptoms are transient, most often without loss of consciousness and evidence of injury typically absent on standard neuroimaging methods. Some sports and individual playing styles have a greater risk of concussion. The initial assessment of a concussion should include combined visual and postural tests and the clinical follow-up must evaluate the long-term effects (deterioration of the physical or mental status). A rigorous education of coaches and athletes and implementation of certain training strategies can reduce the incidence of sport-related concussion. More studies regarding injury mechanisms will bring valuable information for safety equipment manufacturers and for designing safer competitions.

Keywords: sport-related concussion, signs, symptoms, assessment, neurodegenerative effects.

Rezumat

Comoția cerebrală din disciplinele sportive este o leziune complexă a creierului, care se manifestă printr-o mare varietate de semne și simptome de disfuncție cerebrală (deficit fizic și cognitiv, comportament anormal). Simptomele sunt tranzitorii, cel mai adesea fără pierderea conștienței și dovezile morfologice ale traumatismului sunt de obicei absente la examinarea prin metodele standard neuroimagistice. Unele discipline sportive și stiluri de joc individuale prezintă un risc mai mare de comoție cerebrală. Evaluarea inițială a unei comoții ar trebui să includă teste combinate vizuale și posturale, iar monitorizarea clinică trebuie să evalueze efectele pe termen lung (deteriorarea stării fizice sau mentale). O educație riguroasă a antrenorilor și sportivilor și punerea în aplicare a anumitor strategii de antrenament poate reduce incidența comoției cerebrale din sport. Mai multe studii privind mecanismele lezionale pot aduce informații valoroase producătorilor de echipamente de siguranță și pentru proiectarea unor competiții sportive mai sigure.

Cuvinte cheie: comoția cerebrală din sport, semne, simptome, evaluare, efecte neurodegenerative.

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Definitions

The Sport Concussion Assessment Tool - 3rd and 5th editions (SCAT3 and SCAT5) define sport-related concussion (SRC) as an impairment in brain function caused by a direct or indirect traumatic brain injury (TBI), with immediate and transient symptoms, most often without loss of consciousness (1),(2). SRC represents a frequent injury and a serious concern for a lot of sports worldwide.

The term mild traumatic brain injury (mTBI) is utilized very often as an equivalent term to concussion; practically, SRC represents a subset of mTBI, with particular onset and clinical manifestations in the context of sport (King et al., 2014); (2).

Athletes and trainers should be aware of the possible presence and consequences of a concussion, which must be rapidly identified and correctly managed.

Epidemiology

In the USA, it is estimated that approximately 2% of the population suffered a certain degree of disability as a result of a TBI; and that approximately 80% of TBIs are of mild severity (Ruff et al., 2009). Also in the USA, it is estimated that 5-9 % of all sport-related injuries are SRCs (King et al., 2014).

The majority of adult patients with sport-related concussion are not hospitalized (Vavilala et al., 2017), but alarmingly high percentages (30-70%) of concussions occur in pediatric sports (Hobbs et al., 2016; Yue et al., 2016).

The number of SRCs has increased in the last years. Epidemiological studies largely underestimate the incidence of SRC, because of several factors: athletes do not recognize and do not report a SRC that may stop them from returning to play; clinicians in emergency department units report variable results because of the very complex symptomatology, which can be influenced by other conditions, such as the hydration level, fatigue or depression, or because data are not available (Iverson, 2006; Register-Mihalik & Kay, 2017). It is estimated that 1/3 of concussions remain undiagnosed (Meehan et al., 2013).

Pathophysiology of concussion

SRC may be caused either by a direct mechanism (coup injuries or a direct blow to the moving head) or by an indirect mechanism (contrecoup injuries or an indirect blow elsewhere on the body with an impulsive force transmitted to the head, or a moving head hitting a stationary object) (2); (Register-Mihalik & Kay, 2017). It is reported that indirect concussive injury is more severe (Guskiewicz et al., 2004).

Concussion is a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. What most contributes to its complexity is the fact that there is no known threshold for the hitting force inducing this injury and there is no correlation between the intensity or location of the impact and the severity of the injury (Register-Mihalik & Kay, 2017).

Cellular physiology is affected in the acute phase of concussion: a neuronal depolarization and an abrupt release of excitatory neurotransmitters occur. The depolarization process causes an energy crisis for rebalancing the ionic shift, which leads to a high increase of glucose metabolism and a decrease of cerebral blood flow (Giza & Hovda, 2001; Laskowski et al., 2015; Register-Mihalik & Kay, 2017). The axonal function and structure are also affected: there is an alteration of axolemmal permeability, cytoskeleton and both anterograde and retrograde transport (Laskowski et al., 2015), and axons can undergo progressive degeneration (Morley & Seneff, 2014). The autoregulation of cerebral blood flow (CBF) is impaired or abolished (for example, an abnormal middle cerebral artery flow velocity was reported), which makes the brain vulnerable to ischemia or hyperemia (Vavilala et al., 2017). The expression of dopaminergic receptors is also altered (increased in the prefrontal cortex or initially decreased and then increasing in the striatum, which can explain working memory deficits) (Kobori & Dash, 2006; Wagner et al., 2009).

Cerebral physiology can be impaired for weeks following the acute phase of injury: the brain may be less responsive to physiological neural activation; the ionic imbalance, the energy crisis and the depressed metabolic activity continue, impair cellular function and create a post-concussion vulnerability which may also lead to cell death (Giza & Hovda, 2001; Laskowski et al., 2015; Register-Mihalik & Kay, 2017). Also, the altered cerebral vascular reactivity persist: it was reported that patients with concussion presented reduced CBF at one week, which recovered at one month (Meier et al., 2015).

Clinical diagnosis

SRC is one of the most complex sport-related injuries, and variations in its reporting, diagnosis and management are determined by its extremely varied clinical presentation and by the differences between many available published guidelines for recognition, assessment and management of concussion (King et al., 2014).

SRC causes multiple and non-specific signs and/or symptoms, usually with rapid onset and spontaneous resolution in a sequential course (2). SRC has a wide range of potential effects, manifesting by an impairment of neurological functions, which may consist of:

- symptoms (e.g., headache, dizziness, nausea/emesis, pain localized to eye)
- physical signs (e.g., balance impairment, drowsiness, blurry vision, sensitivity to light or noise, impaired reaction time, fatigue or lethargy, changes in the sleep pattern, or other neurological deficits)
- impaired brain function (e.g., confusion, cognitive deficit, word finding difficulties, memory deficits, difficulty concentrating, post-traumatic amnesia)
- abnormal behavior (e.g., change in personality, depressive symptoms) (1), (Morley & Seneff, 2014; King et al., 2014; Laskowski et al., 2015; Register-Mihalik & Kay, 2017).

Individual cerebral physiological characteristics are responsible for the diversity of the symptoms. An underlying cerebral neurovascular abnormality in SRC (Vavilala et al., 2017) is linked to the intensity of post-concussive headache and to the cognitive symptom burden:

Table I
Concussion grading system

Grade 1	Grade 2	Grade 3
No LOC Transient confusion Symptoms and mental status changes resolve in less than 15 min	No LOC Transient confusion Symptoms or mental status changes last more than 15 min	LOC (seconds or minutes)

a higher vasoreactivity was strongly associated with more severe headaches (Albalawi et al., 2017). Therefore, a detailed history of injury is an important part of evaluation.

Usually, the loss of consciousness (LOC) is not present. The American Academy of Neurology (Ruff et al., 2009; Giza et al., 2013) classified concussions into 3 grades (Table I).

Even if according to the definition of concussion, recovery statistically takes around 15 minutes, there are cases when signs and symptoms may be prolonged and evolve over minutes to hours (2). The majority of concussion symptoms (80-90%) disappear in a short time (7-10 days), but for children and adolescents, recovery may be longer (McCrory et al., 2005; Morley & Seneff, 2014).

For the clinical assessment of concussion, SCAT indicates the Glasgow Coma Scale (GCS) (Table II), a primary, non-invasive clinical tool which calculates a score evaluating the level of consciousness (eye opening, verbal and motor responses) (1); (2). The score indicates a mild (13-15), moderate (9-12), or severe (< 8) brain injury and, through repeated recording for all athletes, may be utilized to determine the course of care and to predict outcome in case of subsequent deterioration (Zollman, 2016).

Table II
The Glasgow coma scale (GCS)

Parameter	Response	Score
Best eye response (E)	no eye opening	1
	eye opening in response to pain	2
	eye opening to speech	3
	eyes opening spontaneously	4
Best verbal response (V)	no verbal response	1
	incomprehensible sounds	2
	inappropriate words	3
	confused	4
	oriented	5
Best motor response (M)	no motor response	1
	extension to pain	2
	abnormal flexion to pain	3
	flexion/withdrawal to pain	4
	localizes to pain	5
	obeys commands	6
	GCS = E + V + M = maximum 15	

Athletes with SRC very frequently report dizziness and visual problems, which requires a more comprehensive assessment of vestibulo-oculo-motor impairments (Mucha et al., 2014). Multidimensional oculomotor and vestibular tests are very sensitive in the detection of subtle neurological signs, and could be used as a combined aid in the detection of deficits following a concussion. These tests can quantify functional impairment, monitor deterioration or recovery, and evaluate treatment efficacy, therefore representing valuable assessment tools in SRC (Mucha et al., 2014; Cheever et al., 2017).

Following a SRC, several oculometric parameters are significantly affected, including pursuit acceleration, latency and gain, saccade amplitude, and speed responsiveness (Liston et al., 2017), and there are several oculomotor tests for assessment. The King-Devick test investigates oculomotor function using a series of charts of numbers that progressively become more difficult to read in a flowing manner (Galetta et al., 2011; King et al., 2014). This test assesses saccadic eye movements and is very useful for a better identification of SRC without reported signs/symptoms, but it does not evaluate other ocular motor functions, which can be important indicators of SRC brain dysfunction, such as pursuit, convergence, or accommodation (Cheever et al., 2017). The King-Devick test was even able to identify unwitnessed SRC on field when SCAT2 components were negative (King et al., 2013).

Additionally, other optometric tests such as near point convergence (NPC) distance, Gaze Stabilization (GST), Rapid Eye Horizontal (REH), Smooth Pursuit - Slow (SPS) and Fast (SPF), Optokinetic Stimulation (OKS) can provide useful information about the evolution and recovery process in SRC and may be useful in deciding to return to sport (Cheever et al., 2017). These tests have different dynamics during the evolution of SRC, they have significantly higher values at the initial assessment of the injury, the King-Devick test improves across time, REH and SPF tests improve after the acute phase, and the OKS test improves even if symptoms are prolonged (Cheever et al., 2017).

The vestibular system is a complex network which shows impairments within the first few days after a concussion. The commonly used tests for the vestibular system are the Balance Error Scoring System (BESS) and the Sensory Organization Test (SOT) (Guskiewicz, 2001; McDevit et al., 2016). Both SOT and BESS tend to be most sensitive in the very acute stage of recovery, but while SOT requires advanced postural analysis equipment, BESS is more subjective, the investigator measures postural stability by counting errors during a series of six balancing stances (Wright et al., 2017; Cheever et al., 2017).

The Vestibular/Ocular Motor Screening (VOMS) is a brief on-field test, which evaluates through symptom provocation and measurement of near-point convergence 5 domains (smooth pursuit, horizontal and vertical saccades, convergence, horizontal vestibular ocular reflex, and visual motion sensitivity), and which also demonstrated high sensitivity in identifying athletes who experienced a SRC (Mucha et al., 2014; Kontos et al., 2017).

Testing the combination of visual and postural tasks is the most sensitive and had the greatest discriminatory outcomes for athletes with SRC (Wright et al., 2017).

From a psychiatric point of view, a concussion can be

immediately followed by depressive symptoms that seem to be associated with the symptoms of concussion itself (Yrondi et al., 2017).

Differential diagnosis is extremely exhaustive, due to the fact that a positive identification of SRC requires the exclusion of other conditions with similar clinical signs and symptoms: the use of toxics (drug, alcohol) or medication; other injuries (cervical) or other comorbidities (vestibular dysfunction, psychological factors) (2).

Neuroimaging evaluation and biomarkers

The diagnosis of SRC is clinical and often very challenging (Wright, 2014). Clinical signs and symptoms are acute, have a rapid resolution and reflect a functional disturbance, not a structural injury.

In emergency departments, a computed tomography (CT) scan is quickly performed, but objective evidence of injury is typically absent on standard neuroimaging investigations (2).

The transcranial Doppler ultrasound (TCD) investigation is a non-invasive bedside tool which can be used for assessing cerebral blood flow and altered neurophysiology, and for monitoring cerebrovascular reactivity and the recovery of SRC (Gardner et al., 2015; Vavilala et al., 2017). Also, at a functional level, in brain research, the effect of injury on brain functions (such as perception and memory) can be investigated by functional magnetic resonance imaging (fMRI) (Toth, 2015).

A recent study (Amyot et al., 2015) compared the effectiveness of seven neuroimaging modalities (CT, MRI, TCD, positron emission tomography (PET), single photon emission computed tomography (SPECT), electrophysiological techniques - magnetoencephalography and electroencephalography, and functional near-infrared spectroscopy). CT, MRI and TCD proved to be the most useful in clinical diagnosis. CT and MRI are not routinely performed and it is a challenge for the physician to decide whether neuroimaging is needed (Table III), but TCD is non-invasive, inexpensive, and can be used in the daily management of TBI. The other modalities have the potential to be diagnostic tools, but are expensive and their clinical utility remains to be confirmed in larger studies; for example, SPECT perfusion imaging has some advantages compared to PET: radiopharmaceuticals are widely available and approved and provide quantitative data both for screening and post-injury monitoring. However, due to the heterogeneity of signs/symptoms in SRC, there is no single imaging modality that is sufficient for all patients (Kutcher et al., 2013).

Currently, serum biomarkers have a limited role in the evaluation and management of SRC (O'Connell et al., 2017). More objective biomarkers of neuronal, axonal and glial injury are clinically needed in order to diagnose, prognosticate the recovery and determine the risk of cumulative impairments after SRC in athletes (2). Recently, for determining potential biomarkers, new perfusion neuroimaging techniques have been explored, which can assess cerebral blood flow dynamics (Douglas et al., 2017).

Table III
Recommendation for CT scan in concussion

Patients with a LOC or posttraumatic amnesia - only if one or more of the following is present	1. Headache 2. Vomiting 3. Age > 60 years 4. Drug or alcohol intoxication 5. Deficits in short-term memory 6. Physical evidence of trauma above the clavicle 7. Post-traumatic seizure 8. GCS score < 15 9. Focal neurologic deficit 10. Coagulopathy
Patients without a LOC or posttraumatic amnesia - if one or more of the following is present	1. Focal neurologic deficit 2. Vomiting 3. Severe headache 4. Age >65 years 5. Physical signs of a basilar skull fracture 6. GCS score < 15 7. Coagulopathy 8. Dangerous mechanism of injury (e.g., ejection from a motor vehicle, a pedestrian struck by a vehicle, or a fall from a height of more than 3 feet or 5 stairs)

Axonal injury in SRC is the primary determinant of outcome following TBI and is reflected in the remodeling of cytoskeletal neurofilament proteins; it was demonstrated that serum axonal protein neurofilament light (NFL) is a highly sensitive biomarker for concussion (Shahim et al., 2017). Another promising diagnostic marker which also has a certain utility in predicting mortality is S100B, a protein mainly found in astroglia and Schwann cells; it increases in cerebrospinal fluid and serum after injury and can distinguish injured from non-injured patients. Other research markers, such as neuron-specific enolase - highly expressed in neuronal cytoplasm, tau proteins - associated with neuronal microtubules, glial fibrillary acidic protein - from astroglial cytoskeleton, and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor - a glutamate receptor involved in fast synaptic transmission from the central nervous system have not yet confirmed their value as serum markers of SRC (O'Connell et al., 2017).

Treatment

There is a lack of consensus regarding the treatment of SRC, as individual variations in signs/symptoms make it challenging for clinicians to approach the management of concussion from all sides, including daily activities and return to school, work, and sport.

Exercise and cognitive activity increase the brain's metabolic activity; therefore the treatment basis for SRC is cognitive and physical rest, allowing for normalization of metabolism and correction of energy deficit (Wright, 2014). This assertion is supported by some studies (Majerske et al., 2008; Brown et al, 2014) in which subjects engaged in the highest amount of activity (cognitive and sportive) scored worse in successive concussion evaluations.

Currently, studies do not support complete rest, but the exact amount and duration of rest is not yet well defined; there is also limited evidence of the use of pharmacotherapy. Individual specific prescriptions for the type and intensity of rest and allowable activity may be more beneficial (2).

As a general principle, drugs that can mask the symptoms of concussion must be avoided. In the acute phase (0-10 h post-injury), acetaminophen can be used for headache (nonsteroidal anti-inflammatory drugs are not recommended), and drugs that alter mental status (benzodiazepines) must be avoided; after this phase, drugs for symptomatic relief may be used, excepting those that affect the central nervous system (stimulants, anti-nausea drugs and antidepressants). Sleep problems in the first few days should be managed conservatively, without medications (Harmon et al., 2013).

Multiple therapeutic interventions, including physical, vestibular, pharmacological and cognitive behavioral therapy, become necessary in case of complications.

Evolution and complications

The evolution of SRC can complicate with prolonged symptom recovery, post-concussion syndrome and second impact syndrome. Repeat concussions may lead to long-term effects, such as depression, mild cognitive impairment and chronic neurodegeneration (Guskiewicz et al., 2005; Guskiewicz et al., 2007; King et al., 2014; Morley & Seneff, 2014). Rarely, SRCs are associated with skull fractures, epidural or subdural hematomas, and edema requiring neurosurgical evaluation (Hobbs et al., 2016).

Several *risk factors* for complications, including young age (Freeman et al., 2008) and gender (female sex can be a predictor of symptom reporting), initial symptom severity, loss of consciousness (Fehr et al., 2017), a history of learning and attention deficit disorders, migraines or mood disorders (Harmon et al., 2013), may influence a prolonged evolution of SRC (King et al., 2014). More probably, certain individual features, a particular natural state, favor an increased susceptibility of the brain to injury: microglia can have a hyperreactive response which can persist for a long time (strongly linked to other neurological disorders), or the normal physiological response to brain injury can be disrupted (a reaction to deficiencies in key micronutrients which leads to impaired antioxidant capacity and associated pathologies), or alterations in cerebral neurovascular response develop (failure of cerebral blood flow autoregulation) (Wright et al., 2017). As a result of the increased metabolic dysfunction, the brain may have impaired ability to recycle cellular debris and, consecutively, increased vulnerability if a subsequent insult (even minor) were to occur (King et al., 2014). Repeated insults can induce a chronic state of slowly progressive secondary neurodegeneration (it can be a tauopathy or a pathological aggregation of tau protein) (Morley & Seneff, 2014; Safinia et al., 2016). Neuroimaging methods showed that the most common abnormalities in chronic traumatic encephalopathy include cerebral volume loss, enlargement of the cavum of the septum pellucidum, cerebral microhemorrhages, and white matter signal abnormalities, all of which have poor sensitivity and specificity (Bonfante et al., 2017). For an accurate correlation between the number of suffered concussions and the prevalence of chronic traumatic encephalopathy, further longitudinal studies are needed (Safinia et al., 2016).

The majority of SRCs recover in one week to 10 days (Len & Neary, 2011). Persistent symptoms and prolonged recovery can be defined as longer than normally expected (>10–14 days in adults and >4 weeks in children) (Makdissi et al., 2017). Persistence for more than 7 days is considered to be post-concussion syndrome (PCS) (Satz et al., 1999; Maroon & Bost, 2011; Morley & Seneff, 2014). Most individuals with PCS report resolution of symptoms by 3 months, but some studies have shown that up to 33% of patients have some persistent symptoms for >6 months, and 15% of patients complain of symptoms >12 months post-injury (Bazarian et al., 1999; Maroon & Bost, 2011; Daroff et al., 2015). PCS is often associated with prior concussion (Guskiewicz et al., 2004).

Multiple therapeutic interventions may be required for SRC complications: an individualized aerobic exercise program in patients with persistent symptoms, targeted physical therapy in patients with cervical spine or vestibular dysfunction, and a collaborative approach including cognitive behavioral therapy to deal with any behavioral issues (2).

Second impact syndrome is a feared complication, a condition in which a second concussion occurs during a "vulnerable period", while an individual is still symptomatic from an earlier concussive event; the second trauma causes a loss of cerebral autoregulation leading to diffuse cerebral edema and increased intracranial pressure within minutes of the impact, and can ultimately cause brain herniation, resulting in coma and death (Laskowski et al., 2015).This syndrome has a 100% morbidity and 50% mortality; it is important to note that all reported cases occurred in athletes younger than 20 years of age (McCrory, 2001).

It is always essential to accurately evaluate concussions; from a psychiatric point of view, the presence of a prior mood disorder can contribute to the later onset of a major depressive disorder. Even practicing a sport can protect against depressive disorders. It was demonstrated that especially after several concussions, a depressive disorder, linked to their frequency and number, can appear (Yrondi et al., 2017). This link will be more clearly explained by further studies regarding the role of gender, preexisting mental health disorders and sport types and competition levels (Hutchison et al., 2017).

Incidence of SRC in various sport disciplines

Athletes practicing several sports risk suffering severe injuries.

Cycling accidents (Amadori et al., 2017) can induce traumatic brain injury or dental and facial lesions; the helmet's design protects against certain head injuries by 65%, but it does not protect the lower third of the face and the teeth, and does not significantly reduce the risk of dental traumas (Amoros et al., 2012). Wearing a helmet was demonstrated to be protective against skull fractures and subdural hematoma, but not against other injuries such as contusions and subarachnoid hemorrhage (Forbes et al., 2017). Against dental injuries, custom made mouthguards offer the greatest comfort and protection (Bemelmanns & Pfeiffer, 2000).

Rugby is one of the sports where concussions are the most common and represent a major threat to players; the use of the headgear is not mandatory and it is not always effective in preventing concussion (Barnes et al.,

2017). Concussion most frequently occurs during the tackle and interventions on the playing manner (reducing speed and acceleration, increasing duration and distance before contact in tackle), and maintaining a correct head position will reduce the risk of concussion (Cross et al., 2017; Sobue et al., 2017). Prevention strategies should be different for each position in the game (Sobue et al., 2017). Also, Attwood et al. (2017) demonstrated that completing a specifically designed exercise program (for example, isometric neck exercises that can increase neck strength) prior to training and match play can reduce concussion incidence by 60%.

The vast majority of confirmed chronic traumatic encephalopathy (CTE) cases have been reported in athletes competing in high-impact sports involving repeated head trauma, such as boxing, American football, ice hockey, and rugby (Safinia et al., 2016; Pearce et al., 2015). Boxing for a long period can cause through a particular kinetic mechanism (angular acceleration/ torsional injury to midbrain and cerebellum) CTE, which was formerly known as dementia pugilistica (DP). This was reported in approximately 17% of professional boxers with long careers and a large number of bouts, who progressively developed after multiple concussions a disease with behavioral, mood and cognitive abnormalities (emotional lability, memory loss), and motor impairments (mild Parkinsonism, dysarthria and mild imbalance) (Tarazi et al., 2016). Olympic-style boxing has a less violent history with serious injuries being rare, and there is no strong evidence supporting a relationship with chronic brain injury (Howell et al., 2017). Nowadays, dementia pugilistica is rare, but even a single bout can produce permanent deficits; therefore boxing is never devoid of risk for permanent brain injury (Castellani & Perry, 2017). As differences between sports are notable (regarding athletes, injury kinematics), CTE in sports should be considered specifically. More research is needed for developing sport-specific exposure animal models to illustrate neurological illness and disease management (Erlanger, 2015). The problem is much broader. Also, the psychological factor cannot be neglected; in football teams, even a correlation between a head coach's leadership style and the incidence of severe injuries was demonstrated (Ekstrand et al., 2017).

Many skiers have experienced two or more head impacts, the magnitude of speed being of particular importance, but concussions are under-reported to a large extent. In all skiing disciplines, backward falls cause impact to the rear of the helmet; facial bone fractures and dental injuries are also reported (Steenstrup et al., 2017).

Other sports that can involve head impact and can be related to TBI's are: ball games - basketball (Covassin et al., 2017), volleyball (Baugh et al., 2017), baseball and softball (Cusimano, Zhu, 2017), floorball (Pasanen et al., 2017), lacrosse (Barber Foss et al., 2017), hurling (O'Sullivan et al., 2017); contact sports - judo (Čierna et al., 2017), wrestling (Kroshus et al., 2017); others - gymnastics (Edouard et al., 2017), horse racing (O'Connor et al., 2017).

Strategies for returning to normal activity

Several guidelines and peer reviewed articles on concussion management recommend a gradual return to usual school or sport activities. However, due to the extremely varied symptoms, there is no guide for the proper management of every concussion. The general principle is that the patient must be free of any symptoms, but individual cases must be treated based on clinical experience.

Regarding the return to school/cognitive activities, it is recommended as a stepwise, gradual process to: 1) start daily activities at home with 5-15 min at a time and gradually build up without having symptoms; 2) training in cognitive activities at home, outside the work environment, for increasing tolerance to cognitive work; 3) return to work activity part-time, with increased breaks during the day; 4) return full time, gradually increasing activities until a full day can be tolerated without symptoms; 5) return to full activities (2); (King et al., 2014). A coordinated effort must be made by all the people involved in recuperation (patient, family, health care providers, teachers) in order to ensure that the patient has the necessary conditions to transit back successfully (rest periods, program flexibility, avoidance of physical exertion) (Wright, 2014).

For sport activities, after the initial 24-48 hours of rest, once an athlete is asymptomatic, the next gradual strategy can be recommended (Wright, 2014; King et al., 2014): 1) gradual reintroduction of activities that do not provoke symptoms; 2) light aerobic exercise, no resistance training, for increasing tolerance without provoking symptoms; 3) specific exercise or running, no head impact activities; 4) non-contact training exercise drills, progressive coordination and resistance; 5) in normal training activities, full contact practice following medical clearance; 6) return to sport, normal game play. There should be at least 1 day or longer for each step. If any symptoms reappear and worsen during exercise, the athlete should go back to the previous step; if asymptomatic, exertion is increased 24 hours later.

Conclusions

1. Concussion is a complex injury that requires a multimodal assessment and treatment process.

2. More studies about injury mechanisms will bring valuable information for safety equipment manufacturers and for designing safer competitions.

3. The initial assessment of a concussion should include combined visual and postural tests.

4. CT, MRI, and TCD are the most useful modalities for the initial evaluation and clinical follow-up of concussion.

5. Sports medicine practitioners should be aware of the extent and nature of concussion and should work with coaches, athletes and trainers to identify and manage concussions.

6. Training strategies for reducing the incidence of head and neck injuries should be included in the education of all coaches and athletes.

7. Rigorous education, continuous modeling of the rules and legislative efforts by coaches and officials reinforce the importance of fair play as the mainstay of sports.

Conflicts of interests

There are no conflicts of interest.

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