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Return-to-Play Criteria After Athletic Concussion

A Need for Revision

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Management of a sport-related concussion, especially involving return-to-play decisions, is one of the most important challenges confronting sports medicine professionals. Current guidelines result from thoughtful consensus recommendations by expert committees but are chiefly based on the resolution of symptoms and the results of neuropsychological testing, if available. Adherence to this paradigm results in most injured athletes resuming competition in 1 to 2 weeks.

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A minireview of recently published studies using a variety of techniques suggests that persistent cerebral dysfunction occurs for at least 1 month in many concussed athletes. In view of the frequency (>300 000 sport-related concussions estimated per year in the United States), the young age of the injured individuals, and the lack of procedures to document the complex postinjury healing process in the brain, a postconcussion return-to-play (RTP) interval of at least 4 weeks is imperative.

The management of cerebral concussion occurring in athletes might, in its current state, be characterized as an "anxiety disorder" typically occurring among sports medicine physicians and athletic trainers. Diagnosis, although defined in consensus statements by expert committees,[1-4] cannot be precisely confirmed by currently available imaging or laboratory procedures, and no specific therapy has been proven effective. Although RTP criteria exist, they are evidence based at level 4 (supported by expert opinion) but not as defined in the following:

> Evidence-based medicine is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence-based medicine means integrating individual clinical expertise with *the best available external clinical evidence from systematic research* [italics added].[5(p71)]

Lacking reliable and specific measures of brain damage and/or dysfunction, the resolution of symptoms (at rest and with exertion) and, where available, the use of neuropsychological tests have been relied on by physicians and trainers to provide *estimates* of the appropriate time for athletes to resume practice and play.[4] Recent guidelines suggest that return to practice may occur when all symptoms have cleared and results of neurologic examination and cognitive evaluation of the athlete are normal. If aerobic, strength, and specific sport performance challenges are successfully accomplished, RTP may be recommended.[4]

During recovery in the first few days after concussion, it is important to emphasize to the athlete that physical and cognitive rest are required. Activities that require concentration and attention may exacerbate symptoms and delay recovery. Return to play after a concussion follows a stepwise process as outlined by the Canadian Academy of Sports Medicine[6]: (1) no activity, complete rest until asymptomatic with normal neurologic examination results; (2) light aerobic exercise, such as walking or stationary cycling, and no resistance training; (3) sport-specific exercise with progressive addition of resistance training; (4) noncontact training drills; (5) full-contact training after medical clearance; and (6) game play. With this stepwise progression, the athlete should continue to proceed to the next level if asymptomatic at the current level. If any postconcussion symptoms occur, the patient should drop back to the previous

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asymptomatic level and try to progress again after 24 hours.

Athletes who minimize and/or deny symptoms to promote their quicker RTP complicate the utility of these recommendations. In addition, neuropsychological test results may lack a baseline for comparison, may vary in reliability on serial testing, and are confounded by known practice effects.[7] Furthermore, numbers of contact sport athletes diagnosed as having their first concussion report that they have experienced similar past episodes that were not diagnosed.

A follow-up database for concussed athletes was compiled between October 1998 and October 2007 at Pace University, covering 9 years to date. For 52 concussions occurring in 49 different athletes (2 events in 3 athletes), the mean (SD) symptom duration was 6.3 (4.7) days, excluding 3 outliers with symptoms lasting more than 30 days. Twenty-five athletes (51%) had experienced prior medically diagnosed concussions (range, 1-3). Return to practice and/or RTP occurred a mean (SD) of 13.8 (5.2) days after injury, excluding 7 athletes either "sitting out" the remainder of the sport season or retiring secondary to recurring concussions. Evidence suggests that, for younger athletes and for those with a learning disability or prior concussions, RTP time should be extended.[3] An excellent brief overview of the most recent consensus statements has been published.[8]

The summary and agreement statement of the 2nd International Conference on Concussion in Sport[4(p196)] defined concussion as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces." Most important, the consensus statement notes that "there is little published evidence that concussion injury severity correlates with the number and duration of acute concussive signs and symptoms and/or degree of impairment on neuropsychological testing,"[4(p197)] concluding that "the science of concussion is at early stages, and therefore, *management and return to play decisions remain largely in the realm of clinical judgment on an individualized basis* [italics added]."[4(p202)] The definition by the Second International Conference, although true, is less succinct than one offered a decade ago by the American Academy of Neurology: "a trauma-induced alteration in mental status."[9] If one accepts alteration of mental status (cerebral function) as the operant factor determining the presence or absence of postconcussion effects, then the results of several recently published studies raise questions of concern relating to current RTP criteria.

Using different methods to assess cerebral function, these reports reveal altered brain function for 1 month in most concussed athletes tested. Because few of the researchers assessed their athletes for longer periods, the total duration of the measured abnormal responses remains unknown. These studies are briefly summarized herein according to the method used.

ELECTRICAL RECORDINGS OF BRAIN WAVE ACTIVITY

Several studies have reported electroencephalographic recordings of event-related potentials in concussed athletes. These methods involve presenting complex visual or auditory signals that require an individual to react by tapping the correct key on a computer. This stimulus-response paradigm detects attention and information processing deficits. Dupuis et al[10] found decreased amplitude in a positive wave (reflecting attentional-cognitive response) occurring 300 milliseconds after a specific visual stimulus (P300 wave). This finding was noted for 1 week to 6 months after concussion and was most marked in the group of persistently symptomatic athletes. Neuropsychological testing of these affected athletes did not reveal attentional deficits. Because a correlation was found between persistent symptoms and decreased P300 measurements in this cohort, the study did not add to usual clinical assessment of asymptomatic athletes. However, the time delay between concussion and event-related potential recording was significantly longer in the asymptomatic group than in the symptomatic group, possibly explaining the fewer electroencephalographic abnormalities measured in the asymptomatic athletes.

The same group restudied another 20 athletes, controlling for the time since last concussion, and found that both asymptomatic and symptomatic athletes showed reduced P300 amplitudes in response to an auditory stimulus at 5 weeks after concussion.[11] Because only a single measurement was performed, it remains unknown whether the abnormal finding is reversible. In addition, the number of athletes studied was small. However, the researchers conclude that after concussion "the fact that asymptomatic athletes have an electrophysiological profile similar to that of symptomatic athletes challenges the validity of RTP guidelines for which the absence of symptoms is a major issue."[11(p1159)]

In their most recent study[12] of multiconcussed athletes (≥2 concussions) who were challenged with a visual event-related potential, researchers from the same laboratory reported long-lasting P300 amplitude suppression compared with nonconcussed or singly concussed athletes despite having equivalent symptom and neuropsychological test scores. These findings were manifested for up to 2½ years, suggesting permanent deficit.

TRANSCRANIAL MAGNETIC STIMULATION PARADIGMS

An electromagnetic coil applied to the head that emits pulses of skull-penetrating, neuron-activating magnetic energy generates transcranial magnetic stimulation. De Beaumont et al[13] used transcranial magnetic stimulation with motor-evoked responses detected by an electromyogram recorder attached to an index finger muscle. With different protocols, they measured resting motor threshold, intracortical inhibition and facilitation, excitability of the corticospinal system, and the duration of a cortical silent period. Control, singly concussed, and multiply concussed groups were studied, with all concussion events having occurred more than 9 months before study. Abnormality of the intracortical inhibitory system, manifested by increased cortical silent period response, was seen in the multiply concussed athletes (who had also sustained the most severe concussions). The best correlation for increased silent period response proved

to be the severity of prior concussion. These results show that multiple sports concussions may result in functional brain abnormality not detected by symptom scores or neuropsychological measurements persisting for more than 9 months.

MULTITASK EFFECTS ON GAIT STABILITY

In a series of experiments using gait stability measurements during simultaneous performance of cognitive tasks, van Donkelaar et al[14] and Halterman et al[15] demonstrated that attentional and biomechanical performance deficits increase as task complexity increases and that, even with "mild concussions" (no loss of consciousness and symptomatic recovery in 1-2 weeks), these deficits persist for a full month after injury. In these tests, a concussed athlete's attentional ability appears limited so that the performance of combined tasks exceeds the athlete's limits of attention. Executive function is, therefore, impaired, resulting in difficulty for athletes to switch between 2 simultaneous tasks. Therefore, an individual with a "resolved concussion" may appear normal when performing isolated tasks but display noticeable deficits when attempting multiple tasks simultaneously. This finding has significant implications concerning performance capability for athletes returning to play under current guidelines.

The same group studied concussed and nonconcussed collegiate athletes with a gait stability protocol first performed with undivided attention and then while simultaneously completing mental tasks (serial 7's, months backward, and 5-letter words spelled backward). Testing was performed 2, 5, 14, and 28 days after concussion. Again, concussion impaired gait stability during dual tasks for up to 28 days after injury.[16]

BALANCE TESTING

Slobounov et al[17] tested 38 athletes with grade 1 concussions 10, 15, and 30 days after injury. A virtual-reality, 3-dimensional, stereovisual field system was used to destabilize posture, which was recorded using a force plate and motion analysis system. All athletes were asymptomatic by day 10. All concussed athletes demonstrated impaired visual-kinesthetic integration at 2 weeks, but the multiply concussed athletes had not recovered after 30 days. Studies were not performed beyond this point.

IMAGING STUDIES

Although reports[18-24] did not specifically study athletes and many of the patients had brain injury more severe than usually seen in sports injury, they included patients with cerebral concussions classified as mild traumatic brain injury (simple concussion). In addition, a meta-analysis[25] has concluded that, for non–sport-related concussions and sport-related concussions, the overall effects on neuropsychological outcomes are comparable.

POSITRON EMISSION TOMOGRAPHY

Bergsneider et al[18] studied patients with mild to severe head injuries using positron emission tomography to measure cerebral glucose metabolic rate. Reduction to approximately half-normal values occurred in many of their patients with mild injuries and persisted for 2 to 4 weeks. In another study, the same group observed that the decreased glucose metabolic rate "begins to resolve approximately one month following injury" and concluded that it "is seemingly stereotypic across a broad range and severity of injury types."[19(p135)]

DIFFUSION TENSOR MAGNETIC RESONANCE IMAGING

In 1997, Geddes[20] reviewed the results of histologic and immunohistochemical studies in animals and humans, showing that head injury produced diffuse axonal damage. Axonal swelling that formed a "bulb" occurred with blockage of fluid and β-amyloid precursor protein transport between neurons and their synapses. Specific staining identified diffuse axonal damage within a few hours of injury. Blumbergs et al[21] had previously studied 5 patients (aged 59-89 years) with mild concussions who died 2 to 99 days after injury of non–concussion-related causes. Immunostains with antibody to β-amyloid precursor protein showed multifocal axonal injury in all patients, especially involving cerebral areas important for memory function.

Recent studies[22,23] indicate that lesions of axonal injury may be identified with diffusion tensor magnetic resonance imaging specifically measuring water diffusion along cerebral neuronal axons. Kraus et al[24] used this noninvasive technique to assess in vivo axonal integrity. Among patients with mild traumatic brain injury, axonal damage correlated negatively with cognitive measures of attention, memory, and executive function, and all severities of brain injury resulted in axonal damage. In a few serial studies, including the study by Arfanakis et al,[22] some brain regions showing reduced diffusion after sudden injury were still abnormal 30 days later. Although, to our knowledge, no serial studies of diffusion tensor magnetic resonance imaging in concussed athletes are yet available, the histologic study[21] cited herein suggests the possibility of persisting change for more than 30 days.

A summary of the recent studies indicating cerebral dysfunction for longer than 2 weeks in concussed athletes and nonathletes with traumatic brain injury is provided in the **Table**.

CONCLUSIONS

No definitive conclusions can result from this brief review. The techniques cited herein are research tools not amenable to routine study of many patients because they require complex equipment, skilled procedure performance, and sophisticated interpretation of results. In addition, there are assumptions implicit in electrophysiologic, biomechanical, and imaging procedures that must be accepted with caution. These tools are currently unavailable to clinical professionals for guiding RTP decisions for their athletes.

Despite these limitations, it appears that simple concussion results in cerebral dysfunction, reflecting brain cellular damage. A variety of assessment techniques applied

Table. Summary of Recent Studies Indicating Cerebral Dysfunction of Longer Than 2 Weeks in CA and in Nonathletes With TBI

Measurement	Abnormal Response Duration	Patient Group	Reference No.
EEG ERP response	5 wk	CA, S and A	11
	30 mo	CA, multiconcussions	12
TMS	9 mo	CA, multiconcussions	13
Multitask-challenged GS	28-30 d	CA	14-16
Destabilized posture BT	14 d	CA, first concussion	17
	28 d	CA, multiconcussions	17
PET scan glucose uptake	2-4 wk	Nonathletes with TBI	18-19
DTI MRI	30 d	Nonathletes with TBI	22-24

Abbreviations: A, asymptomatic; BT, balance testing; CA, concussed athletes; DTI MRI, diffusion tensor imaging magnetic resonance imaging; EEG, electroencephalogram; ERP, event-related potential; GS, gait stability; PET, positron emission tomography; S, symptomatic; TBI, traumatic brain injury; TMS, transcranial magnetic stimulation.

to athletes with concussion have measured significant degrees of functional abnormality persisting beyond the usual RTP intervals (1-2 weeks) supported by current guidelines. In addition, the complexity of the neurometabolic cascade occurring after concussion outlined by Giza and Hovda[26] casts doubt as to whether concussed athletes can truly resolve their injury during this brief interval. Laskowitz and Vitek[27] have emphasized that apolipoprotein E genetic polymorphism is an important modifier that affects functional outcome after brain injury and may partially explain varying consequences of concussion. If one accepts the American Academy of Neurology definition that concussion is a trauma-induced alteration of mental status, the cited studies indicate that cerebral dysfunction persists for at least 1 month after injury. This indicates that safe RTP might require at least 4 to 6 weeks to facilitate more complete recovery and to protect from reinjury, reported by Guskiewicz et al[28] to occur much more frequently in the immediate period after a concussion. Consequently, for athletes competing in many sports, concussion would be a season-ending injury.

This idea is a significant departure from current practice and will probably provoke concern and resistance at all levels of sport. Nevertheless, given the prevalence of sports head injury and the numbers of young brains at risk, a postconcussion RTP interval of at least 4 weeks is imperative. Future studies that use longer follow-up periods may conclude that even this time requires extension to permit complete healing.

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