Concussion in sport
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Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001*

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Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries

In November 2001, the first International Symposium on Concussion in Sport was held in Vienna, Austria. This symposium was organised by the International Ice Hockey Federation (IIHF), the Federation Internationale de Football Association Medical Assessment and Research Centre (FIFA, F-MARC), and the International Olympic Committee Medical Commission (IOC).

The aim of the symposium was to provide recommendations for the improvement of safety and health of athletes who suffer concussive injuries in ice hockey, football (soccer), and other sports. To this end a range of experts were invited to address specific issues of epidemiology, basic and clinical science, grading systems, cognitive assessment, new research methods, protective equipment, management, prevention, and long term outcome, and to discuss a unitary model for understanding concussive injury. At the conclusion of the conference, a small group of experts were given a mandate by the conference delegates and organising bodies to draft a document describing the agreement position reached by those in attendance at that meeting. For the purpose of this paper, this group will be called the Concussion in Sport Group (CISG).

INTRODUCTION

This review seeks to summarise the findings of the Vienna conference and to provide a working document that will be widely applicable to sport related concussion. This document is developed for use by doctors, therapists, health professionals, coaches, and other people involved in the care of injured athletes, whether at the recreational, elite, or professional level.

During the course of the symposium, a persuasive argument was made that a comprehensive systematic approach to concussion would be of potential benefit to aid the injured athlete and direct management decisions.[1] This protocol represents a work in progress, and, as with all other guidelines or proposals, it must undergo revision as new information is added to the current literature and understanding of this injury.

The concussion in sport protocol includes:

(1) Clinical history
(2) Evaluation
(3) Neuropsychological testing
(4) Imaging procedures
(5) Research methods
(6) Management and rehabilitation
(7) Prevention
(8) Education
(9) Future directions
(10) Medicolegal considerations

A REVISED DEFINITION OF CONCUSSION

Over 35 years ago, the committee on head injury nomenclature of the Congress of Neurological Surgeons proposed a "consensus" definition of concussion.[2] The American Medical Association and the International Neurotraumatology Association subsequently endorsed this definition.[3] This definition was recognised as having a number of limitations in accounting for the common symptoms of concussion. In addition, there was an inability to include relatively minor impact injuries that result in persistent physical and/or cognitive symptoms. Seeking to transcend these limitations, the CISG has developed the following definition of concussion.

"Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

(1) Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.

(2) Concussion typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously.

(3) Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.

(4) Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.

(5) Concussion is typically associated with grossly normal structural neuroimaging studies.

THE CISG CONCUSSION PROTOCOL
Clinical history

Recognising the importance of a detailed concussion history and appreciating the fact that many athletes will not recognise all the concussions that they may have suffered in the past, a detailed concussion history is of value. The athlete currently at a high performance level in collision sport has seldom had the first concussion on presentation in the consultant's office. The history should include specific questions as to previous symptoms of a concussion, not just perceived number of past concussions.[4] It is also worth noting that dependence on the recall of concussive injuries by teammates or coaches has been shown to be unreliable.[5] The finding that there is increased risk of subsequent concussive injuries after a first concussion is documented, although the reasons for this remain controversial. The clinical history should also include information about all previous head, face, or neck injuries as these may have clinical relevance to the present injury. It is worth emphasising that, in the setting of faciomaxillary injuries, coexistent concussive injuries may be missed unless specifically assessed.

Specific questions about disproportionate impact and matching of symptom severity may allude to progressively increasing vulnerability to injury—that is, more pronounced persistent symptoms from smaller hits. The pathophysiological nature of this phenomenon remains unclear.

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Table 1 Scale of postconcussion symptoms

	Rating						
	None			Moderate			Severe
Headache	0	1	2	3	4	5	6
Nausea	0	1	2	3	4	5	6
Vomiting	0	1	2	3	4	5	6
Drowsines	0	1	2	3	4	5	6
Numbness or tingling	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sleeping more than usual	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional than usual	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervousness	0	1	2	3	4	5	6
Other	0	1	2	3	4	5	6

Adapted from Lovell and Collins.[13]

One of the issues that was speculated upon at the conference was whether concussion represents a unitary phenomenon with a linear spectrum of injury severity or whether different concussion subtypes exist. These subtypes may represent differences in clinical manifestations (confusion, memory problems, loss of consciousness), anatomical localisation (cerebral *v* brainstem, for example), biomechanical impact (rotational *v* linear force), genetic phenotype (ApoE4 positive *v* ApoE4 negative), neuropathological change (structural injury *v* no structural injury), or an as yet undefined difference. These factors may operate independently or interact with each other. It is clear that the variations in clinical outcome from the same impact force require a more sophisticated approach to the understanding of this phenomenon than is currently available.[6]

The traditional approach to severe traumatic brain injury using loss of consciousness as the primary measure of injury severity has acknowledged limitations in assessing the severity of concussive injury. Findings in this field describe association of loss of consciousness with specific early deficits but does not necessarily imply severity. Further work in this area may help to explain these findings.[7]

There is renewed interest in the role of amnesia (anterograde/retrograde) and its manifestation of injury severity.[8] Published evidence suggests that the nature, burden, and duration of the clinical postconcussive symptoms may be more important than previously recognised.[9–11]

Concussion grading scales
The CISG recognised the strengths and weaknesses of several existing concus-sion grading scales that attempt to characterise injury severity, but no single system was endorsed. It was the recommendation of the CISG that combined measures of recovery (see below) should be used to assess injury severity (and/or prognosis) and hence individually guide decisions on return to play.

In the absence of scientifically validated return to play guidelines, a clinical construct is recommended using an assessment of injury recovery and graded return to play. The protocol outlined below is adapted from the Canadian Academy of Sport Medicine (CASM) guidelines.[12] Sideline evaluation includes clinical evaluation of signs and symptoms, ideally using a standardised scale of postconcussion symptoms (table 1) for comparison purposes, and acute injury testing as described below under neuropsychological testing.

Evaluation
Sideline evaluation including neurological assessment and mental status testing is an essential component in the protocol. These evaluations are ideally developed in language translations for international sporting groups (an example of such a sideline evaluation developed at McGill University is available in English and French; for a copy, contact author KMJ). In the acute assessment of concussive injury—that is, concussion diagnosis—brief neuropsychological test batteries that assess attention and memory function have been shown to be practical and effective. Such tests include the Maddock's questions[14] and the Standardised Assessment of Concussion (SAC).[15] It is worth noting that standard orientation questions—for example, time, place, person—have been shown to

be unreliable in the sporting situation compared with memory assessment.[14 16]

It is recognised, however, that abbreviated testing paradigms are designed for rapid evaluation of concussion on the sidelines and are not meant to replace comprehensive neuropsychological testing, which is sensitive enough to detect subtle deficits that may exist beyond the acute episode.

Signs and symptoms of acute concussion
If any one of the following symptoms or problems is present, a head injury should be suspected, and appropriate management instituted. A player does not need to have lost consciousness to suffer a concussion.

(a) Cognitive features
Unaware of period, opposition, score of game

Confusion

Amnesia

Loss of consciousness

Unaware of time, date, place

(b) Typical symptoms
Headache

Dizziness

Nausea

Unsteadiness/loss of balance

Feeling "dinged" or stunned or "dazed"

"Having my bell rung"

Seeing stars or flashing lights

Ringing in the ears

Double vision

Other symptoms such as sleepiness, sleep disturbance, and a subjective feeling of slowness and fatigue in the setting of an impact may indicate that a concussion has occurred or has not resolved.

(c) Physical signs
Loss of consciousness/impaired conscious state

Poor coordination or balance

Concussive convulsion/impact seizure

Gait unsteadiness/loss of balance

Slow to answer questions or follow directions

Easily distracted, poor concentration

Displaying unusual or inappropriate emotions, such as laughing or crying

Nausea/vomiting

Vacant stare/glassy eyed

Slurred speech

Personality changes

Inappropriate playing behavior—for example, running in the wrong direction

Appreciably decreased playing ability

Neuropsychological assessment after concussion

The application of neuropsychological testing in concussion has been shown to be of value and continues to contribute significant information in concussion evaluation.[17] It has been shown that cognitive recovery may precede or follow resolution of clinical symptoms, suggesting that the assessment of cognitive function should be an important component in any return to play protocol.

In the consideration of injury recovery or return to play, such test strategies must assess the cognitive domains of information processing, planning, memory, and switching mental set. Numerous paradigms are in current use. Examples of these include paper and pencil tests (McGill ACE, SAC), condensed batteries (McGill ACE), comprehensive protocols administered by neuropsychologists (NHL, Australian football), and computerised test platforms—for example, IMPACT, CogSport, ANAM, Headminders.[18]

> The consensus of the CISG was that neuropsychological testing is one of the cornerstones of concussion evaluation and contributes significantly to both understanding of the injury and management of the individual.

Overriding principles common to all neuropsychological test batteries is the need for and benefit of baseline preinjury testing and serial follow up. Recent work with computerised platforms, however, suggests that performance variability may be a key measure for diagnosis of acute concussion even in the absence of a baseline test. This strategy is currently the subject of continuing research. Inherent problems with most neuropsychological tests include the normal ranges, sensitivity and specificity of tests, and practice or learning effect, as well as the observation that players may return to baseline while still symptomatic.[17 19] In part, these may be a problem of the currently available pen and paper tests. Computerised testing using infinitely variable test paradigms may overcome these concerns. Computerised testing also has the logistical advantage that the tests may be administered by the team doctor or be web based rather than having to employ a neuropsychologist for a formal assessment. The strengths and weaknesses of such testing have been recently reviewed.[18]

The consensus of the CISG was that neuropsychological testing is one of the cornerstones of concussion evaluation and contributes significantly to both understanding of the injury and management of the individual. Organised sport federations have access to and should attempt to employ such testing as appropriate. To maximise the clinical utility of such neuropsychological assessment, baseline testing is recommended.

Neuroimaging

It was recognised by the CISG that conventional structural neuroimaging is usually normal in concussive injury. Given that caveat, the following suggestions are made. Brain computed tomography (or where available magnetic resonance imaging (MRI) brain scan) contributes little to concussion evaluation, but should be used whenever suspicion of a structural lesion exists. Examples of such situations may include prolonged disturbance of conscious state, focal neurological deficit, seisure activity, or persistent clinical or cognitive symptoms.

Newer structural MRI modalities, including gradient echo, perfusion, and diffusion weighted imaging, have greater sensitivity for structural abnormalities; however, the lack of published studies as well as the absence of preinjury neuroimaging data limits the usefulness of this approach in clinical studies at the present time. In addition, the predictive value of various MRI abnormalities that may be incidentally discovered is not established at the present time. Promising new functional imaging—for example, PET/SPECT/fMRI—technologies, while producing some compelling findings, are still at the early stages of development.[20]

Although neuroimaging may play a part in postconcussive return to play decisions or for the assessment of moderate to severe brain injury, it is not essential for otherwise uncomplicated concussive injury.

Research methods

A number of research protocols and data evaluating concussion injury assessment, injury susceptibility, and brain function after injury were presented at the Vienna conference. All of these techniques, while offering great potential for injury assessment, must be considered experimental at this time. As much as possible, elite and professional teams are well placed to contribute to these efforts through athlete recruitment for studies showing the scientific value of such approaches.

Electrophysiological recording (ERP, EEG) has shown reproducible abnormalities in the postconcussive state in brain function.[19] Similarly, balance testing has shown impairment after injury, although the mechanism for this is not established. Biochemical serum markers of brain injury (including S-100b, NSE, MBP) were proposed as means of detecting cellular damage if present.

Genetic phenotyping has been shown to be of benefit in traumatic brain injury. Published studies have shown that ApoE4 is a risk factor for adverse outcome following moderate to severe brain injury.[21] Similarly ApoE4 has been shown to be a risk factor for the development of chronic traumatic encephalopathy in boxers.[22] The significance of ApoE4 in concussion risk or injury outcome is unclear. Other published studies have noted the association of a particular calcium subunit gene abnormality with brain swelling after minor head trauma.[23]

Such research is vital in contributing to the science of concussion and will potentially provide valuable information for such important issues as clinical management, return to play guidelines, and long term outcome. Therefore research should be continued and encouraged by sporting organisations.

Management and rehabilitation
Acute response

When a player shows ANY symptoms or signs of a concussion:

(1) The player should not be allowed to return to play in the current game or practice.

(2) The player should not be left alone; and regular monitoring for deterioration is essential.

(3) The player should be medically evaluated after the injury.

(4) Return to play must follow a medically supervised stepwise process.

A player should never return to play while symptomatic. "When in doubt, sit them out!"

Rehabilitation

It was the consensus of the CISG that a structured and supervised concussion rehabilitation protocol is conducive to optimal injury recovery and safe and successful return to play. The rehabilitation principles were common to all identified programmes and are outlined below. Important principles state that the athlete be completely asymptomatic and have normal neurological and cognitive evaluations before the start of the rehabilitation programme. Therefore the more prolonged the symptom duration, the longer the athlete will have sat out. The athlete will then proceed stepwise with gradual incremental increases in exercise duration and intensity, and pause or backtrack with any recurrence of concussive symptoms. It is appreciated that, although each step may take a minimum of one day, depending on the duration of symptoms, proceeding through each step may take longer in individual circumstances.

Return to play protocol

Return to play after a concussion follows a stepwise process:

(1) No activity, complete rest. Once asymptomatic, proceed to level (2).

(2) Light aerobic exercise such as walking or stationary cycling.

(3) Sport specific training—for example, skating in hockey, running in soccer.

(4) Non-contact training drills.

(5) Full contact training after medical clearance.

(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 symptoms occur after concussion, the patient should drop back to the previous asymptomatic level and try to progress again after 24 hours.

Prevention

As part of the clinical history, it is advised that details of the protective equipment used at the time of injury be sought, for both recent and remote injuries. The benefit of this approach allows modification and optimisation of protective behaviour and an opportunity for education. That said, there are relatively few methods by which concussive brain injury may be minimised in sport. The brain is not an organ that can be conditioned to withstand injury. Thus, extrinsic mechanisms of injury prevention must be sought.

> ## Rule changes and rule enforcement play a key role in reducing and preventing concussions.

Helmets have been proposed as a means of protecting the head and theoretically reducing the risk of brain injury. In sports in which high speed collisions can occur or which have the potential for missile injuries—for example, baseball—or for falls on to hard surfaces—for example, gridiron, ice hockey—there is published evidence that use of sport specific helmets reduces head injuries.[3] For other sports such as soccer and rugby, no sport specific helmets have been shown to be of benefit in reducing rates of head injury.[24] Some believe that the use of protective equipment may deleteriously alter playing behaviour so that the athlete actually increases his or her risk of brain injury.[25]

Although the use of correctly fitting mouthguards can reduce the rate of dental, orofacial, and mandibular injuries, the evidence that they reduce cerebral injuries is largely theoretical, and no clinical evidence for a beneficial effect in reducing concussion rates has yet been demonstrated clinically.[26]

Consideration of rule changes, such as no head checking in ice hockey, to reduce the head injury rate may be appropriate where a clear cut mechanism is implicated in a particular sport. Similarly, rule enforcement is a critical aspect of such approaches and referees play an important role.

Conditioning of the neck muscles may be of value in reducing impact forces transmitted to the brain. Biomechanical concepts dictate that the energy from an impacting object is dispersed over the greater mass of an athlete if the head is held rigidly. Although attractive from a theoretical standpoint, there is little scientific evidence for the effectiveness of such measures.

Rule changes and rule enforcement play a key role in reducing and preventing concussions.

Education

As the ability to treat or reduce the effects of concussive injury after the event is minimal, education of athletes, colleagues, and those working with them, as well as the general public is a mainstay of progress in this field. Athletes and their healthcare providers must be taught how to detect concussion, its clinical features, assessment techniques, and principles of safe return to play. Methods to improve education including various web based resources (for example, www.concussionsafety.com), educational videos, outreach programmes, concussion working groups, and the support and endorsement of enlightened sport groups such as FIFA, IOC, and IIHF who initiated this endeavour have enormous value and must be pursued vigorously.

The promotion of fair play and respect for opponents are ethical values that should be encouraged in all sports and sporting associations. Similarly coaches, parents, and managers play an important part in ensuring these values are implemented on the field of play.

Future directions

Efforts to evaluate long term outcome and any association with repeated concussion, molecular markers, imaging, and functional deficits must guide continuing investigation in this work. Efforts to expand knowledge of injury that may or may not be associated with particular manoeuvres inherent to the game, such as heading in soccer, must be elucidated.

A proposal was made that this concussion working group be identified and given a mandate to provide continuing leadership in the continued development and updating of guidelines and maintenance of the pursuit of a high standard of care in concussion.

Medicolegal considerations

Although agreement exists about the principal messages conveyed by this document, the authors acknowledge that the science of concussion is at the early stages and therefore management and return to play decisions remain largely in the realm of clinical judgment on an individual basis. It is the intention of the group to analyse the medicolegal aspect of concussions in sports and to offer here a summary of the state of the art and to direct future efforts.

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Female athlete triad syndrome
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New criteria for female athlete triad syndrome?

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As osteoporosis is rare, should osteopenia be among the criteria for defining the female athlete triad syndrome?

The American College of Sports Medicine (ACSM) has provided a great deal of impetus to educating healthcare providers, athletes, and the general public about the potential harm of a "serious syndrome consisting of disordered eating, amenorrhoea and osteoporosis".[1] We recognise and respect the importance of research and attention to this clinical problem and commend the ACSM on its contribution to date.[2] To their credit, the authors of the most recent position stand acknowledged that there were no data reporting prevalence on this condition,[3] and they encouraged further research. Since then, Mayo Clinic physiatrist Tamara Lauder[4] has published two important papers showing a 0% prevalence of the female athlete triad (as defined by ACSM) despite 34% of this military population being at risk of disordered eating. Therefore we re-examined the prevalence of one component of the female athlete triad, osteoporosis, in studies of athletic women with menstrual disturbance. The syndrome can be no more prevalent than any one of its diagnostic criteria alone. Thus, if osteoporosis is only present in a small proportion of the population, then it follows that the female athlete triad can only be prevalent in an equally small, or smaller, proportion of that population.

DIFFERENTIATING OSTEOPOROSIS FROM OSTEOPENIA

Because of the increasing public awareness of osteoporosis and its complications, medical practitioners must not use the term as a synonym for "low bone mass".[5] The current standard for measuring bone mass (bone mineral density; BMD) is by dual energy x ray absorptiometry, and since 1994 the term osteoporosis has had diagnostic criteria based on this technique.[3 6 7] Osteoporosis is defined as BMD more than 2.5 standard deviations below the mean of young adults. The term osteopenia describes BMD scores between 1 and 2.5 standard deviations below the mean of young adults. Scrutiny of many papers examining BMD data in athletes at risk of the female athlete triad syndrome (table 1) suggests that osteopenia has a significant prevalence but that osteoporosis is relatively uncommon, even in this selected population. In the substantial reviews of Bennell *et al*,[8 9] menstrual disturbance was associated with a mean 10.3% lower lumbar spine BMD, which reflects the lower limit of normal BMD and very early osteopenia (T score about −1.0). Not surprisingly, numerous authors reporting bone health of sportswomen have used osteopenia as the appropriate term.[8 10–13] Interestingly, even in the significant pathology of anorexia nervosa, the mean BMD of patients reflects osteopenia rather than osteoporosis.[11] A crucial point is that significant osteopenia—that is, T-score of −2.0—in a 20 year old may provide a worse prognosis for long term bone health than osteoporosis in a 65 year old with a T-score of −2.6.

Osteoporosis can, and does, occur in athletes[14 15] (table 1), but we argue that requiring this condition to be present in the female athlete triad syndrome relegates the syndrome to relative obscurity. It is unlikely that the prevalence of osteoporosis in athletes with disordered eating could be greater than the prevalence of osteoporosis in anorexia nervosa (table 2). Therefore, the female athlete triad, as currently defined, most likely has a lower prevalence than anorexia nervosa. This is borne out by the data of Lauder *et al*[4] showing that the prevalence of anorexia nervosa was < 8% but the prevalence of the female athlete triad was 0%. Anorexia nervosa has an overall age adjusted incidence per 100 000 person years of 14.6 for females and 1.8 for males.[16] Thus, if osteoporosis is a diagnostic criterion for the female athlete triad, the triad should have an age adjusted incidence of substantially less than 0.015% in the population at large. Note that this calculation is not based on anorexia being an essential component of the triad—it is not. These data merely recognise the fact that osteoporosis, as strictly defined, affects only a proportion