Concussion:

Acute Trauma

By: Rebecca Lane
Prepared for: Edith Kunszt, CNP, RNCP, DiHom
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Acute: Concussion (Mild Traumatic Brain Injury (MTBI))

Introduction

A concussion (also called a mild traumatic brain injury (MTBI)) is a transient disorder of the nervous system resulting from a violent blow to the head or a fall. The person typically loses consciousness and cannot remember the events of the occurrence. Several common features of a concussive head injury include:

1. Concussion may be caused either 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 neurologic 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. (McCrory, 48-49)

Symptoms: Three categories of signs and symptoms of concussion include:

- ***Somatic***: loss of consciousness, headaches, fatigue, sleep disturbance, nausea, visual changes, tinnitus, dizziness, balance problems, light/noise sensitivity.
- ***Emotional/Behavioral***: lowered frustration tolerance, irritability, increased emotional irritability, depression, anxiety, clinginess, personality changes.
- ***Cognitive*****:** slowed thinking or response speed, mental fogginess, poor concentration, distractibility, memory/learning difficulty, disorganization, problem-solving difficulties, post-traumatic amnesia. (Aloi, 3)

Diagnosis: Diagnosis of concussion is based on four criteria:

1. **physical and neurological exams** – the SCAT (see Appendix 1) has been developed for sports related injuries and is completed following any head impact injuries during play. Assessment areas include: signs (loss of consciousness, seizure, balance issues); memory; symptom score; cognitive score, neurological signs (speech, pupil, gait assessment).
2. **duration of unconsciousness** (usually less than 30 minutes)
3. **duration of post-traumatic amnesia** (usually less than 24 hours)
4. **Glasgow Coma Scale score** (MTBI have scores of 13 to 15 on the GCS).

Concussions are often under-diagnosed, especially in sport where players know that they will be removed from the game with a head injury and are less likely to mention headache, or other symptoms to remain in competition.

Glasgow Coma Scale

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

Treatment: Usually the signs and symptoms of concussion go away within 7 to 10 days, however the neurological changes take a minimum of 30 days to return to normal (see **Biochemic Markers** section). Concussion sufferers are prescribed rest, both sleep at night and rest during the day. Once symptoms have cleared completely, the client may gradually return to work or play using a phased-in approach.

From a sport perspective, specific rules for return to play have been developed.

1. No activity, complete rest. Once asymptomatic, proceed to level 2.
2. Light aerobic exercise such as walking or stationary cycling, no resistance training.
3. Sport specific exercise (eg, skating in hockey, running in soccer), progressive addition of resistance training at steps 3 or 4.
4. Non-contact training drills.
5. Full contact training after medical clearance.
6. Game play. (McCrory, 52)

Non-players are usually sent home from the hospital and told to return after 24 to 72 hours for further monitoring and to return immediately should symptoms worsen or change to include unconsciousness, convulsions, worsening headaches, vomiting, new bleeding or deafness in either or both ears, and/or extremity weakness.

Prognosis: MTBI has a mortality rate of almost zero. The symptoms of most concussions resolve within a week, but problems may persist.

Recent literature suggests that recurrent concussions have some cumulative effects. In one study of high school athletes, children who had suffered more than two concussions but were currently asymptomatic had neuropsychological test scores that were indistinguishable from those of children with recent concussions. Academic grade

point average was also lower in those with multiple concussions, although it is unclear whether this was a result of injury or if it indicates a predisposition of some children to concussive injury. (Aloi, 8)

Repeated concussions can cause cumulative brain damage such as dementia pugilistica or chronic traumatic encephalopathy or severe complications such as second-impact syndrome. **Second-impact syndrome** (**SIS**) is a condition in which rapid cerebral swelling follows relatively minor head trauma that occurs while a patient is recovering from a prior concussion and is still symptomatic (often within the same game). The second blow to the head can be minor, with the patient becoming mentally dulled within a few seconds or minutes. The syndrome is thought to relate to a disruption of the autoregulation of cerebral blood flow and is characterized by the abrupt onset of cerebral edema. (Aloi, 8) The condition is often fatal, and almost everyone who is not killed is severely disabled.

Modern Classification of Disease

Constitutional:

While a concussion is the result of accidental trauma to the brain, not everyone has the same threshold for concussion. There are several factors that make some more vulnerable to concussion, post-concussive syndrome and severe complications than others:

1. **Genetic protein marker Apolipoprotein E:** ApoE is a fat-binding protein that is essential for a multitude of biological functions. It is involved with neuronal repair and antioxidant activity. Three isoforms of the protein have been described, epsilon 2, 3, and 4. The epsilon 4 isoform (ApoE-E4) has heen shown to be a risk factor for developing Alzheimer's disease. Recent efforts have focused on the possible roles of ApoE-E4 and the ApoE promoter gene in concussion risk. Published studies have demonstrated that ApoE4 is a risk factor for adverse outcome following all levels of brain injury. (Kutcher, 18)

2. **Gender:** Gender is emerging as an important consideration in concussion management. In a study of soccer players, women were found to have a higher incidence of concussion and women also were shown to report more postconcussive symptoms. They found that women were at higher risk of experiencing postconcussive symptoms 3 months after injury. In the paediatric population, one recent study of children (mean age, 14.1 yr) showed that concussed girls had a significantly higher mean symptom score at the time of their initial presentation than boys. (Kutcher, 17)

3. **Age:** Biologically, the developing brain has a unique set of physiologic variables that are changing continuously as children grow. It has been shown that the pediatric brain is more vulnerable to injury than originally expected. (Aloi, 1)
    - The developing brain is 60 times more sensitive to glutaminemediated N-methyl-D aspartate (NDMA) excitotoxic brain injury

- This NDMA-hypersensitivity may make the young brain more susceptible to injury from excitatory amino acids (EAAs), which are present after brain trauma
- The pediatric brain may be more prone to diffuse and prolonged cerebral edema after injury than the adult brain
- MTBI in preschool children may affect their ability to learn to read
- The significance of head injury also may be greater in children because their young brains are still developing skills. Impact to areas of the brain important for skill acquisition is more likely to affect developing skills than well-established ones (Aloi, 2)

4. **Family History:** The real clinical value of knowing a family history of concussion relies on the amount of detail given. There is sufficient evidence to suggest that if parents and relatives have a history of multiple concussions with decreasing force requirements, escalating symptoms, changing personality, retirement from contact sports, and prolonged cognitive and mood effects, the risk to the offspring is greater that they will be predisposed. (Kutcher, 19)

5. **Fatigue:** Athletes are more likely to be concussed after significant physical exertion, especially in the setting of inadequate hydration or caloric intake. A second, more speculative, role for physical fatigue in increasing concussion risk is based on the observation that physical exertion to the point of fatigue can lead to slowed reaction time. An athlete with slowed reaction time will, naturally, have a more difficult time avoiding contact and may be at a higher risk of concussion as a result. It also is important to note that physical exertion alone has been shown to produce concussive symptoms. (Kutcher, 19)

6. **Past and/or Recent Concussions:** There is a significantly increased risk of sustaining a concussion when an athlete is still recovering from a previous concussive injury. There is some published evidence that a recent concussion predisposes an athlete to a second injury. Guskiewicz et al. showed that high school football players who suffered a concussion were three times more likely to sustain a second concussion during the same season, as compared with their nonconcussed teammates. (Kutcher, 19) Previous injury has been shown to impart a 3- to 6-times higher risk of sustaining a subsequent concussion. (Aloi, 1)

Systemic:

Trauma to the head generates immediate impact forces of high magnitude and relatively short duration (5–200 ms) which translate into acceleration forces on the head and brain. These forces include translational (that is, the head's centre moves along a straight line) and rotational (that is, the head moves around its centre of gravity) components, the largest injury capacity being assigned to the rotational component.

Nervous system **-** MTBI can alter the brain's physiology for several hours to several weeks. It sets into motion a variety of pathological events which are usually reversed in a large majority of affected brain cells, but can cause cerebral injury mostly in the form of microscopic diffuse axonal injury.

- *Macroscopic injuries* such as
  - fractures of the skull and face,
  - edema with subsequent brain swelling,
  - significant intracranial haemorrhage and haematoma, or
  - cerebral ischaemia/hypoxaemia can occur, but occur infrequently following mild head injury. (Anderson, 344-345)
- *Microscopic damage*:
  - shearing of the septum pellucidum which may develop into an enlarged cavum septum pellucidum and if severe or repeated may produce fenestrations (McKee (2009), 731)
  - transient elongation or stretch of axons,
  - dissolution of microtubules and neurofilaments and pathological reorganization of neurofilament proteins,
  - neuronal depolarization,
  - widespread neurotransmitter release and postsynaptic receptor activation,
  - Ca^2 influx and activated second messenger systems,
  - initiation of uncontrolled metabolic cascades,
  - cell damage via lipases, proteases, endonucleases,
  - mitochondrial failure and possible mitochondrial necrosis,
  - reactive oxidative species formation and free radical accumulation,
  - altered genetic expression patterns and induction of inflammatory mediators, cell death and transneural degeneration. (Anderson, 344-345)

Digestive system – Liver converts glycogen to glucose and releases glucose to blood. Concussion has an initial period of hyperglycolysis, followed by a phase of metabolic depression, and a third phase of recovery.

Cardiovascular system – Reduction in cerebral blood flow caused by release of excitatory amino acids (EAAs) – glutaminemediated N-methyl-D aspartate (NDMA). The injury induced by these EAAs results in post-traumatic dysautoregulation and a subsequent decrease in cerebral blood flow. These effects may not be seen until 2-3 days after injury and may persist for up to 1 week. (Aloi, 2)

Immune/Lymphatic system – Intracranial haemorrhage, haematoma and/or edema would initiate an inflammatory response. As well, the damage caused by the neurochemical cascade would cause free radical accumulation.This damage severely impairs immune function and if there is a deficiency in antioxidants could leave the brain open to attack from neurotoxins.

Musculoskeletal system – Neuroimaging normally shows no gross structural changes to the brain as a result of concussion, however fractures of the skull or face can occur.

Histopathic:

The primary elements of the neurochemical and neurometabolic cascade following concussive brain injury include:

- ***dissolution of microtubules and neurofilaments and pathological reorganization of neurofilament proteins,***
- ***abrupt neuronal depolarization*** – Shock induced, causing the cascade.
- ***release of excitatory neurotransmitters*** - Excitatory neurotransmitters, chemicals such as glutamate that serve to stimulate nerve cells, are released in excessive amounts as the result of the injury. The resulting cellular excitation causes neurons to fire excessively (and may be responsible for the altered cerebral blood flow).
- ***ionic shifts*** – Excessive neuronal activity creates an imbalance of ions such as potassium and calcium across the cell membranes of neurons. Since the neuron firing involves a net influx of positively charged ions into the cell, the ionic imbalance causes cells to have a more positive membrane potential. This depolarization in turn causes ion pumps that serve to restore resting potential within cells to work more than they normally do.
- ***changes in glucose metabolism*** - This increased need for energy leads cells to require greater-than-usual amounts of glucose. The postinjury events involve three phases: an initial period of hyperglycolysis, followed by a phase of metabolic depression, and a third phase of recovery. (DiFori, 35)

  So, what happens to the *extra glucose* that is not accounted for by oxidative metabolism or anaerobic glycolysis? It is thought to be used to correct ionic fluxes and glutamate depletion which both require large amounts of ATP, and thus ultimately glucose and other substrates, to restore electrical and physiological equilibrium. (Dusick, 1601)
- ***altered cerebral blood flow*** - At the same time, cerebral blood flow is relatively reduced. Thus cells get less glucose than they normally do, which causes an "energy crisis". Lactate may be used as an alternative brain fuel.
- ***impaired axonal function*** – Changes in intracranial pressure and altered cerebral blood flow cause transient elongation or stretching of axons leading to impaired axonal function. Large numbers of neurons may die during this period in response to slight changes in blood flow. Axonal damage may continue for weeks after the MTBI. (McKee (2009), 731)
- ***functional changes to white matter of brain*** – Diffusor Tensor Imaging (DTI) allows us to detect the diffusion of water molecules in white matter tracts. Studies show that 24 hours after injury, the fractional anisotropy (FA) (directional organization of diffusion of water molecules in white matter tracts) was reduced in white matter that otherwise showed a normal MRI. Further, the extent of white matter injury correlated with impaired reaction time. (DiFori, 35-36)
- ***reduced mitochondrial activity*** – This reduced activity is due to swelling of the mitochondria and causes cells to rely on anaerobic metabolism to produce energy, which increases levels of the byproduct lactate.
- ***Disruption of microtubules and neurofilaments.*** (McKee (2009), 731)

Biochemic Markers:

Biochemical serum and cerebrospinal fluid markers of brain injury typically measured after MTBI:

- ***myoinositol***, a glial marker;
- ***Astroglial protein (S100B)*** - S100B may indicate brain injury, but diagnostic tests for it are still experimental. S100 protein is released from astrocytes. However, this isoform is not isolated to brain tissue (Aloi, 7);
- ***choline***, a membrane marker;
- neuron specific ***enolase*** - Neuron-specific enolase (NSE) is an enzyme important for glycolysis in neurons. (Aloi, 7);
- ***myelin basic protein (MBP)***;
- ***glial fibrillary acidic protein (GFAP)*** - measured in serum can be used as a biochemical brain damage marker;
- ***cleaved tau protein (CTP)*** - Cleaved tau protein (CTP), found in axons, is presumed to be released after MTBI when diffuse axonal injury has occurred. This marker may be useful in combination with S100B protein. (Aloi, 7);
- ***lactate***, an indirect marker for ischemia and hypoxia;
- ***N-methyl-D aspartate (NDMA)*** - Excitatory amino acids (EAAs) are present after brain trauma – glutamine mediated N-methyl-D aspartate (NDMA) (Aloi, 2);
- ***N-acetylaspartate (NAA)***, a neuronal marker; and
- energy metabolites ***creatine*** and ***phospbocreatine*** - Magnetic Resonance Spectroscopy (MRS) was performed on athletes at 3, 15, and 30 days after injury. Although the subjects stated they were symptom free by day 3, the *NAA:creatinine* ratio was significantly diminished compared with controls at day 3, with minimal recovery at day 15. By day 30, however, the *NAA:creatinine* ratios had normalized. In three subjects who experienced a second concussion within 15 days, the symptoms were reported to resolve by day 30, and the *NAA:creatinine* ratio normalized by day 45. (DiFori, 37)

Deficiency:

- Proper hydration – dehydration decreases amount of cerebral spinal fluid (CSF), which absorbs shock and trauma to the brain, and also increases potential for fatigue which has an impact on the incidence of concussion.
- Caloric intake – insufficient caloric intake increases potential for fatigue which has an impact on the incidence of concussion. Also, after a concussion, the brain is in a state of hypermetabolism and requires lots of glucose for repair.
- Antioxidants – insufficient antioxidant concentrations would not support the immune response to inflammation and trauma to the brain which would increase the amount of free radicals and therefore the amount of residual damage.
- Levels of dopamine and serotonin are reduced in the cerebrospinal fluid of MTBI victims.

Occupational:
Although concussion is the result of an accident, some occupations have a higher incidence of concussion than others:

- Professional sport ,
- Construction industry,
- Mining industry, and
- Active Military personnel.

Accidental:
The most common causes of concussion are:

- Motor vehicle accidents (Vehicular accident-related concussions can be minimized through proper use of seatbelts and child car seats.),
- Falls (Highest incidence of concussion from falls with people over the age of 65),
- Sports and recreational activities (To some extent, sports- related accidents can be minimized through proper use of helmets and protective equipment.), and
- Assaults.

Conclusion

While there is plenty of talk of the seriousness of concussion, the reality is that most people who experience concussion do not report it and do not take the proper amount of time to recover from this potentially serious brain trauma. In sport, even though there is lots of talk about MTBI, players are told to “brush it off” and get back into play.

During the period when the brain is hypersensitive and autoregulation has not normalized it is most vulnerable to another concussive injury. This can take from 3 to 6 weeks. Further, after recovering from the first concussion, players are more likely to have a second concussion, to have more post-concussive syndrome symptoms and are more likely to develop lifelong complications from the injury.

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Sport Concussion Assessment Tool (SCAT)

This tool represents a standardized method of evaluating people after concussion in sport. This Tool has been produced as part of the Summary and Agreement Statement of the Second International Symposium on Concussion in Sport, Prague 2004

Sports 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 utilized in defining the nature of a concussive head injury include:

1. Concussion may be caused either 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.

Post Concussion Symptoms

Ask the athlete to score themselves based on how they feel now. It is recognized that a low score may be normal for some athletes, but clinical judgment should be exercised to determine if a change in symptoms has occurred following the suspected concussion event.

It should be recognized that the reporting of symptoms may not be entirely reliable. This may be due to the effects of a concussion or because the athlete's passionate desire to return to competition outweighs their natural inclination to give an honest response.

If possible, ask someone who knows the athlete well about changes in affect, personality, behavior, etc.

Remember, concussion should be suspected in the presence of ANY ONE or more of the following:

- Symptoms (such as headache), or
- Signs (such as loss of consciousness), or
- Memory problems

Any athlete with a suspected concussion should be monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle.

For more information see the "Summary and Agreement Statement of the Second International Symposium on Concussion in Sport" in the April, 2005 edition of the Clinical Journal of Sport Medicine (vol 15), British Journal of Sports Medicine (vol 39), Neurosurgery (vol 59) and the Physician and Sportsmedicine (vol 33). This tool may be copied for distribution to teams, groups and organizations.

The SCAT Card

(Sport Concussion Assessment Tool)

Athlete Information

What is a concussion? A concussion is a disturbance in the function of the brain caused by a direct or indirect force to the head. It results in a variety of symptoms (like those listed below) and may, or may not, involve memory problems or loss of consciousness.

How do you feel? You should score yourself on the following symptoms, based on how you feel now.

Post Concussion Symptom Scale

	None			Moderate			Severe
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Balance problems or dizzy	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Vision problems	0	1	2	3	4	5	6
Hearing problems / ringing	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Feeling "dinged" or "dazed"	0	1	2	3	4	5	6
Confusion	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
Drowsiness	0	1	2	3	4	5	6
Fatigue or low energy	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
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6

(follow up symptoms only)

Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6
Trouble falling asleep	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
Other: _____	0	1	2	3	4	5	6

What should I do?

Any athlete suspected of having a concussion should be removed from play, and then seek medical evaluation.

Signs to watch for:

Problems could arise over the first 24-48 hours. You should not be left alone and must go to a hospital at once if you:

- Have a headache that gets worse
- Are very drowsy or can't be awakened (woken up)
- Can't recognize people or places
- Have repeated vomiting
- Behave unusually or seem confused; are very irritable
- Have seizures (arms and legs jerk uncontrollably)
- Have weak or numb arms or legs
- Are unsteady on your feet; have slurred speech

Remember, it is better to be safe. **Consult your doctor after a suspected concussion.**

What can I expect?

Concussion typically results in the rapid onset of short-lived impairment that resolves spontaneously over time. You can expect that you will be told to rest until you are fully recovered (that means resting your body and your mind). Then, your doctor will likely advise that you go through a gradual increase in exercise over several days (or longer) before returning to sport.

Sport Concussion Assessment Tool (SCAT)

The SCAT Card

(Sport Concussion Assessment Tool)

Medical Evaluation

Name: _____ Date _____

Sport/Team: _____ Mouth guard? Y N

1) SIGNS

Was there loss of consciousness or unresponsiveness?	Y	N
Was there seizure or convulsive activity?	Y	N
Was there a balance problem / unsteadiness?	Y	N

2) MEMORY

Modified Maddocks questions (check correct)

At what venue are we? __; Which half is it? __; Who scored last?__

What team did we play last? __; Did we win last game? __?

3) SYMPTOM SCORE

Total number of positive symptoms (from reverse side of the card) = _____

4) COGNITIVE ASSESSMENT

5 word recall	(Examples)	Immediate	Delayed (after concentration tasks)
Word 1 _____	cat	___	___
Word 2_____	pen	___	___
Word 3 _____	shoe	___	___
Word 4 _____	book	___	___
Word 5 _____	car	___	___

Months in reverse order:
Jun-May-Apr-Mar-Feb-Jan-Dec-Nov-Oct-Sep-Aug-Jul (circle incorrect)

or

Digits backwards (check correct)

5-2-8	3-9-1	_____
6-2-9-4	4-3-7-1	_____
8-3-2-7-9	1-4-9-3-6	_____
7-3-9-1-4-2	5-1-8-4-6-8	_____

Ask delayed 5-word recall now

5) NEUROLOGIC SCREENING

	Pass	Fail
Speech	___	___
Eye Motion and Pupils	___	___
Pronator Drift	___	___
Gait Assessment	___	___

Any neurologic screening abnormality necessitates formal neurologic or hospital assessment

6) RETURN TO PLAY

Athletes should not be returned to play the same day of injury.
When returning athletes to play, they should follow a stepwise symptom-limited program, with stages of progression. For example:

1. rest until asymptomatic (physical and mental rest)
2. light aerobic exercise (e.g. stationary cycle)
3. sport-specific exercise
4. non-contact training drills (start light resistance training)
5. full contact training after medical clearance
6. return to competition (game play)

There should be approximately 24 hours (or longer) for each stage and the athlete should return to stage 1 if symptoms recur. Resistance training should only be added in the later stages.
Medical clearance should be given before return to play.

Instructions:
This side of the card is for the use of medical doctors, physiotherapists or athletic therapists. In order to maximize the information gathered from the card, it is strongly suggested that all athletes participating in contact sports complete a baseline evaluation prior to the beginning of their competitive season. This card is a suggested guide only for sports concussion and is not meant to assess more severe forms of brain injury. **Please give a COPY of this card to the athlete for their information and to guide follow-up assessment.**

Signs:
Assess for each of these items and circle Y (yes) or N (no).

Memory: If needed, questions can be modified to make them specific to the sport (e.g. "period" versus "half")

Cognitive Assessment:
Select any 5 words (an example is given). Avoid choosing related words such as "dark" and "moon" which can be recalled by means of word association. Read each word at a rate of one word per second. The athlete should not be informed of the delayed testing of memory (to be done after the reverse months and/or digits). Choose a different set of words each time you perform a follow-up exam with the same candidate.

Ask the athlete to recite the months of the year in reverse order, starting with a random month. Do not start with December or January. Circle any months not recited in the correct sequence.

For digits backwards, if correct, go to the next string length. If incorrect, read trial 2. Stop after incorrect on both trials.

Neurologic Screening:
Trained medical personnel must administer this examination. These individuals might include medical doctors, physiotherapists or athletic therapists. Speech should be assessed for fluency and lack of slurring. Eye motion should reveal no diplopia in any of the 4 planes of movement (vertical, horizontal and both diagonal planes). The pronator drift is performed by asking the patient to hold both arms in front of them, palms up, with eyes closed. A positive test is pronating the forearm, dropping the arm, or drift away from midline. For gait assessment, ask the patient to walk away from you, turn and walk back.

Return to Play:
A structured, graded exertion protocol should be developed; individualized on the basis of sport, age and the concussion history of the athlete. Exercise or training should be commenced only after the athlete is clearly asymptomatic with physical and cognitive rest. Final decision for clearance to return to competition should ideally be made by a medical doctor.

For more information see the "Summary and Agreement Statement of the Second International Symposium on Concussion in Sport" in the April, 2005 Clinical Journal of Sport Medicine (vol 15), British Journal of Sports Medicine (vol 39), Neurosurgery (vol 59) and the Physician and Sportsmedicine (vol 33).