Head injury

The role of helmets in skiing and snowboarding

P McCrory

Helmets need to be developed using injury data and suitable standards

Haid was the first to raise the issue of helmet use in skiing when, in 1955, he published a study of fatal ski injuries treated in Innsbruck in Austria and suggested that head injury might be less severe if a helmet was worn.[1] Since then the main proponent of helmet use in all aspects of skiing has been the Swiss neurosurgeon, Sooyoung Oh, although the evidence presented for his recommendations is anecdotal.[2 3]

The issue of sport specific helmet design becomes more important. Because at the present point in time we have only limited data on precise injury mechanisms in skiing and snowboarding, which suggests that there is no common mechanism to the occurrence of concussive injuries or skull fractures in these sports.[4 5] This would mean that any helmet would have to be designed differently for these two sports. Injuries occurring in ski racing occur at far higher impact velocities than recreational skiing and furthermore usually occur on ice rather than snow. Once again, any potential helmet would have to be designed with these factors in mind. The ability of a conventional ski helmet to protect the brain from the velocities achieved in elite World Cup downhill racing is unachievable at present. Attention to other safety factors such as slope maintenance, flexible race gates, and crash barriers at races therefore becomes a more effective safety factor than helmet use.

Without valid helmet material and manufacturing standards, there is no guarantee that an off the shelf helmet has any protective capacity whatsoever. Furthermore depending on the nature and quality of the materials used, there is a potential for increasing cerebral injury rate significantly. The only international standard applicable for skiing helmets is the British standard BS EN 1077-1996, which sets out minimum standards requirements and helmets testing protocols for alpine skiers (including children) in competitions.

Issues about helmet maintenance and fit become important as well. This is especially true in children who may potentially be a group with the most to gain by wearing a suitable protective helmet. If the helmet does not fit firmly, then the protective capability is reduced. If the helmet is oversized and loose, as many parents may purchase a helmet with "room to grow" for their child, the chances of this being effective are small and potential problems in reducing vision and hearing may increase collision rates which is already the major injury mechanism in the paediatric age group.

Because of cost, helmets are usually made in two or three shell sizes with variations in liner thickness to accommodate a range of head sizes in each shell size. This means that the largest size in each shell is critical since its liner is the thinnest. The requirements of a size 8 helmet which must decelerate a 6.0 kg head are different to a size 6¾ helmet which must protect a 4.2 kg head. The outer shell size remains the same in both cases but the liner thickness is the critical factor. Simple changes such as shifting head sizes into a larger shell may therefore protect greater numbers of participants. The problem is all too obvious in skiing where participants vary from small children through to adults and any proposed helmet must cope with this basic fact.

The possibility of a large helmet having a guillotining effect on the cervical spine needs to be explored. Until appropriate design standards exist, this risk must be viewed with some caution. There is evidence from the literature that children have a higher rate of neck and back injuries and the effect of helmets in contributing to this problem has not been adequately excluded.[4 5]

If a suitable helmet was available, then who would benefit? The highest risk groups would be ski racers, ski jumpers, freestyle aerialists, and children in all categories of skiing. The evidence that adult recreational skiers would benefit by helmets is not proven. Fortunately catastrophic ski injury, although tragic, is rare.[5–7] The risk of injury is reducing at all ski resorts over time and this may have more to do with slope maintenance and grooming, improved ski equipment, improved skill development through ski school instruction, skier fitness, and education. There is probably still much to do in terms of slope control—for example, restricting or removing ski passes from skiers who violate the skiers responsibility code by skiing too fast, out of control, or dangerously placing themselves as well as other slope users at risk.

At the present time no suitable helmet exists which is likely to protect all skiers and snowboarders. Although theoretically attractive in reducing head injury rates, the idea of skiers obtaining inferior and potentially dangerous helmets should not be encouraged. Better injury data analysis and suitable helmet standards are required to initiate the process of developing an appropriate protective helmet. Significant head injuries are rare in skiing and snowboarding and even if all participants wore helmets, it would not eliminate fatal injuries and may potentially increase the rate of other neurological injuries.

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Tendons

Tendon healing: can it be optimised?

N Maffulli, H D Moller, C H Evans

Growth factors may be useful in tendon healing, possibly introduced using gene therapy

Tendon disorders are a major problem in sports and occupational medicine. Tendons have the highest tensile strength of all connective tissue because of a high proportion of collagen in the fibres and their closely packed parallel arrangement in the direction of force. The individual collagen fibrils are arranged into fascicles which contain blood vessels and nerve fibres. Specialised fibroblasts, tenocytes, lie within these fascicles and exhibit high structural organisation.[1] Histologically, they appear as star shaped cells in cross sections. In longitudinal sections, they are arranged in rows following the direction of the tendon fibres. This specialised arrangement is related to their function, as tenocytes synthesise both fibrillar and non-fibrillar components of the extracellular matrix, and are able to reabsorb collagen fibrils.[2] The fascicles themselves are enclosed by epitenon. This is surrounded by the paratenon, and the potential space between them is filled by a thin, lubricating film of fluid which allows gliding of the tendon during motion.

BIOLOGY OF TENDON HEALING

Tendon healing is classically considered to occur through extrinsic and intrinsic healing. The intrinsic model produces obliteration of the tendon and its tendon sheath. Healing of the defect involves an exudative and a formative phase which, on the whole, are very similar to those associated with wound healing.[3] Extrinsic healing occurs through the chemotaxis of the specialised fibroblasts into the defect from the ends of the tendon sheath.[4] The process can be divided into three phases: inflammation, repair, and organisation or remodelling. In the inflammatory phase, occurring three to seven days after the injury, cells migrate from the extrinsic peritendinous tissue such as the tendon sheath, periosteum, subcutaneous tissue, and fascicles, as well as from the epitenon and endotenon.[5] Initially, the extrinsic response far outweighs the intrinsic one. This results in the rapid filling of the defect with granulation tissue, tissue debris, and haematoma. The migrating fibroblasts still play a phagocytic role, and are arranged in a radial fashion in relation to the direction of the fibres of the tendon.[1] Biomechanical stability is given by fibrin.

The migrated fibroblasts begin to synthesise collagen around day 5. Initially, these collagen fibres are randomly orientated. Tenocytes become the main cell type, and over the next five weeks collagen is continuously synthesised. During the 4th week, a noticeable increase in proliferation of fibroblasts of intrinsic origin, mainly from the endotenon, takes place. These cells take over the main role in the healing process and both synthesise and reabsorb collagen. The newly formed tissue starts to mature, and the collagen fibres are increasingly orientated along the direction of force through the tendon. This phase of repair continues for two months after the initial injury. Final stability is acquired during the remodelling induced by the normal physiological use of the tendon. This further orientates the fibres into the direction of force. In addition, cross linking between the collagen fibrils increases the tendon tensile strength. During the repair phase, the mechanically stronger type 1 collagen is produced in preference to type 3 collagen, thus slightly altering the initial ratio of these fibres to increase the strength of the repair.[6]

Despite intensive remodelling over the following months, complete regeneration of the tendon is never achieved. The tissue replacing the defect remains hypercellular. The diameter of the collagen fibrils is altered, favouring thinner fibrils with reduction in the biomechanical strength of the tendon.

In tendinopathic and ruptured Achilles tendons, there is a reduction in the proportion of type 1 collagen, and a significant increase in the amount of type 3 collagen,[7] responsible for the reduced tensile strength of the new tissue which is due to a reduced number of cross links compared with type 1 collagen.[8] Recurring microinjuries lead to the development of hypertrophied biologically inferior tissue replacing the intact tendon.

CYTOKINETIC MODULATION OF TENDON HEALING

Growth factors and other cytokines play a key role in the embryonic differentiation of tissue and in the healing of tissues.[9] Growth factors stimulate cell proliferation and chemotaxis, and aid angiogenesis, influencing cell differentiation. They regulate cellular synthetic and secretory activity of components of extracellular matrix. Finally, they influence the process of wound healing.

In the normal flexor tendon of the dog, the levels of basic fibroblast growth factor are higher than the levels of platelet derived growth factor (PDGF). In injured tendons, the converse is true.[10] Under the influence of PDGF, chemotaxis and the rate of proliferation of fibroblasts and collagen synthesis are increased.[11] Fibroblasts of the patellar tendon show increased proliferation in vitro after the administration of basic fibroblast growth factor.[12] In addition, an angiogenic effect is evident.[13] During the embryogenesis of tendon, bone morphogenic proteins (BMP), especially BMP 12 and 13, cause increased expression of elastin and collagen type 1. Also, animal studies have shown that BMP 12 exerts a positive effect on the healing processes of the patellar tendon.[14]

The growth factors of the transforming growth factor β superfamily induce an increase in mRNA expression of type 1 collagen and fibronectin in cell culture experiments.[15]

High expression of collagen type 1 seems to be essential to achieve faster healing of tendons. Consequently, there should be a shift from the initial production of collagen type 3 to type 1 early in the healing process. The afore mentioned growth factors could potentially be used to influence the processes of regeneration of tendons therapeutically. However, it is unlikely that a single growth factor will give a positive result. The interaction of many factors present in the right concentration at the right time will be necessary.

GENE THERAPY TO PROVIDE GROWTH FACTORS

Growth factors have a limited biological half life. Given the complexity of the healing process of tendons, a single application of growth factors is unlikely to be successful. As there is no bioavailability of oral proteins, repeated local injections would be necessary to maintain levels in the therapeutic range. This can be technically difficult in operatively treated tendons. The transfer of genes for the relevant growth factors seems an elegant alternative.[16] After cellular uptake and expression of genes, a high level of the mediators can be locally produced and secreted.

To achieve this goal, vectors are used enabling the uptake and expression of genes into target cells. Vectors can be broadly grouped into viral and non-viral. Viral vectors are viruses deprived of their ability to replicate, into which the required genetic material can be inserted. They are effective, as the introduction of their genetic material into host cells forms part of their normal life cycle. Non-viral vectors have specific characteristics that enable penetration of the nucleus—for example, liposomal transport. The genes are released in the vicinity of the target cells without systemic dilution.

There are two main strategies for transfer using vectors. In in vivo transfer, the vectors are applied directly to the relevant tissue. In vitro transfer involves removal of cells from the body, the gene transfer in vitro, and subsequent culture of these cells before they are reintroduced into the target site. Direct transfer is less invasive and technically easier, and can be started during treatment of the acute phase of the injury. A disadvantage is the non-specific infection of cells during the injection process. In addition, owing to the amount of extracellular matrix present, a vector with high transgenic activity is necessary to be able to transfer the gene to enough cells.

Indirect transfer of genes is safer. The relevant cell type is isolated and genetically modified. Before reintroduction into the body, cells can be selected and tested for quality. Owing to the work involved in this technique, it would be more suitable for the treatment of degenerative processes rather than acute injuries. The first studies on the feasibility of this procedure have been conducted using marker genes.[17] The main gene used, lacZ, codes for the bacterial β galactosidase which is not present in eukaryotic cells.

The addition of a suitable substrate changes the staining properties of the cells that express the new gene, enabling the effectiveness of transmission and the duration of expression of the foreign gene to be ascertained. With the vectors currently available, the gene is expressed for six to eight weeks in tendon tissue.[18] Using this strategy, the transfer and expression of the PDGF gene into the patellar tendon of rats lead to an increase in angiogenesis and collagen synthesis in the tendon over four weeks. Gene expression of this duration could influence the whole healing process of tendons and could be the start of an optimised healing process.

In summary, tendon healing, even when successful, does not result in normal tendon. Mostly, the result is functionally satisfactory despite morphological differences and biomechanical weakness compared with a normal tendon. The therapeutic use of growth factors by gene transfer seems promising in the quest to produce a new tendon that is biologically, biomechanically, biochemically, and physiologically "normal".

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Concussion

What advice should we give to athletes postconcussion?

P McCrory

Good early management of concussion in athletes is important for recovery outcomes and return to play

The practical management of concussion can be divided into three broad areas where the issues and treatment priorities differ considerably. These areas are immediate, early, and late management. The first of these relates to on field first aid, the second to the early management on the day of the injury, and the third to the issue of return to play at a later date. It is the second of these areas that this paper will focus on.

IMMEDIATE MANAGEMENT

This is where the clinician is in attendance at a sporting event and is called on to manage the acute brain injury. The major priorities at this early stage are the basic principles of first aid. Once these basic aspects of care have been achieved and the patient stabilised, then consideration of removal of the patient from the field to an appropriate facility is necessary. At this time, careful assessment for the presence of a cervical spine or other injury is necessary. The clinical

management may involve the treatment of a disorientated, confused, unconscious, uncooperative, or convulsing patient.[1] The immediate treatment priorities remain the basic first aid principles of "ABC—airway, breathing, and circulation". Once this has been established and the patient stabilised, a full medical and neurological assessment exam should follow.

EARLY MANAGEMENT

This refers to the situation where an athlete has been brought to the medical room for assessment or alternatively to an emergency department or medical facility following a concussive injury. Assessment of injury severity is generally best performed in a quiet of a medical room rather than in the middle of a football field in front of 100 000 screaming fans with television cameras following every move of the doctor.

When assessing the acutely concussed player, various aspects of the history and examination are important.[2] These are summarised in box 1.

Box 1 Early assessment of concussion—history

- History: time and place of injury
- Mechanism of injury (eyewitness account or video)
- Presence or duration of loss of consciousness (LOC)
- Immediate postinjury behaviour
- Acute concussive symptoms/signs
- Presence of convulsions postinjury
- Past medical history (including history of previous brain trauma)
- Medication use

The common symptoms of concussion have been examined in prospective studies and include headache, dizziness, blurred vision, and nausea.[2] It is worth noting that the presence of headache is not confined to concussion with up to 20% of sporting athletes reporting exercise related headache.[1] Given that much emphasis is placed upon headache as an important symptom of concussion, medical assessment needs to be accurate in ascertaining the nature and cause of the players' symptoms.

When examining a concussed athlete, a full neurological examination is important. Because the major management priorities at this stage are to establish an accurate diagnosis and exclude a catastrophic intracranial injury, this part of the examination should be particularly thorough. Although the Glasgow Coma Scale is often used in the setting of head injury to provide a baseline assessment of conscious state, this scale is insufficiently sensitive or specific for the assessment of the majority of sporting concussions.[3]

In recent times the application of simple neuropsychological tests have created considerable interest as a means to objectively diagnose concussed athletes. The standard approach of asking the orientation items—for example, day, date, year, time, date of birth, etc—has been shown to be unreliable in following concussive injury. This aspect of memory remains relatively intact in the face of concussive injury and should not be used.[4] More useful, as demonstrated in prospective studies, are questions of recent memory. These have been shown to be more sensitive in discriminating between concussed and non-concussed individuals. A typical question battery is that proposed by Maddocks *et al* (see box 2).[4] The standardised assessment of concussion (SAC) is a less practical but valid alternative.[5]

Box 2 Post concussion memory assessment ("Maddocks questions"[4])

- Which ground are we at?
- Which team are we playing today?
- Who is your opponent at present?
- Which quarter is it?
- How far into the quarter is it?
- Which side scored the last goal?
- Which team did we play last week?
- Did we win last week?

Some clinicians utilise other tests of new learning or immediate and recent memory function, such as three item recall or digit span in order to determine whether post traumatic amnesia (PTA) has resolved.[6]

Having determined the presence of a concussive injury, the patient needs to be serially monitored until full recovery ensues. Consideration of return to play and the role of neuropsychological testing is not relevant at this stage until full clinical and cognitive recovery has occurred. If the concussed player is discharged home after recovery, then he should be in the care of a responsible adult.

The use of computerised tomography or magnetic resonance scanning to ascertain the presence or absence of cerebral pathology is necessary in certain situations. The geographical availability of the various imaging techniques may influence imaging strategies. It is the author's practice not to routinely scan patients with uncomplicated mild concussion—for example, Cantu grade 1 or Colorado grades 1 and 2.

The treating clinician also must face the decision of who should be referred on to a hospital emergency facility or neurosurgical centre. There are a number of urgent indications that are listed in box 3. Apart from these "cookbook" type approaches, referral to such a centre depends on the experience, ability, and competency of the physician at hand. If the team physician happens to be a neurologist or neurosurgeon experienced in concussion management, then the clinical referral pathways will be different to a family practitioner called to assist at a football match after an injury has occurred. The overall approach should be "when in doubt, refer".

Box 3 Indications for urgent referral

Any player who has or develops the following:

- Fractured skull
- Penetrating skull trauma
- Deterioration in conscious state following injury
- Focal neurological signs
- Confusion or impairment of consciousness >30 minutes
- Loss of consciousness >5 minutes
- Persistent vomiting or increasing headache postinjury
- Any convulsive movements postinjury
- More than one episode of concussive injury in a match or training session
- Where there is assessment difficulty, for example, an intoxicated patient
- Children with head injuries
- High risk patients, for example haemophilia, anticoagulant use
- Inadequate post injury supervision
- High risk injury mechanism, for example, high velocity impact, missile injury

There is published evidence that the postconcussion recovery rates vary between individuals. Some patients may take days, whereas others may take weeks to recover fully from a concussive injury.[4 7–11] The demands and inherent risks of a given sport, as well as differences in individual recovery rates, must be taken into account when the medical decision of returning to play is taken. The main areas of cognitive impairment postconcussion relate to the domains of speed of information processing, reaction times, planning, switching mental "set", and disturbances of new learning and memory.[2] It is the first two of these domains that provides the greatest concern. An athlete in the stages of recovery postinjury will experience several days (or weeks in some cases) of slowed thinking and an inability to process information quickly. This is the principal reason that delays return to sport with the fear that an athlete performing suboptimally will experience further injuries simply because he or she is unable to meet the cognitive demands required for that sport. In the case of motor racing, premature return to sport whilst still

symptomatic may be fatal. To some degree this is the rationale behind the graded return to sport programmes as recommended for concussion.[12] The often quoted concern regarding the so called second impact syndrome in this setting has been demonstrated to be unfounded.[13 14]

When we step outside the world of sport it is clear that athletes in this postconcussive state will be at risk in the real world as well. Driving motor vehicles then becomes a significant concern. No concussed athlete should drive a motor vehicle until they are fully recovered both from a clinical and cognitive standpoint. Dangers exist in the workplace as well and these need to be factored in to the management equation. It seems somewhat bizarre that we devote enormous resources to computerised neuropsychological testing postinjury to accurately predict return to play yet when a footballer leaves our medical room or hospital emergency department, specific management advice is often lacking. The "head injury card" given out by emergency departments is typically designed for more severe brain trauma and to detect possible intracranial complications rather than provide the patient with a useful plan of management. Clearly this is a significant source of potential medicolegal claims against the doctor concerned if such advice is not given.

Does this matter? Recent work from both American and Australian emergency departments has suggested that the provision of detailed information to concussed subjects reduces their period of disability following the injury. As is the case with psychological interventions following all kinds of trauma it seems that if a patient knows what to expect and is reassured that the symptoms experienced are both real and entirely appropriate during the recovery process then the outcome is better.[15] To some degree this is easier in sport given that most experienced sports physicians have seen many cases of concussion and can provide a more comprehensive explanation than that by a junior casualty doctor and the athlete usually has peer support from other teammates who have been through similar injuries. The combination of these factors means that psychological disability is often minimised in the sporting setting.

In summary, the steps to good early management of concussion are as follows:

1. An accurate diagnosis of concussion and exclusion of intracranial pathology by a thorough history and exam with consideration of neuroimaging if appropriate.

2. A detailed (and preferably written) explanation of concussion and the normal symptoms experienced following injury. Such an explanation needs to be written in plain English rather than "medical English". Such an explanation should encompass more than the standard "head injury advice card" as used in emergency departments.

3. A clear management plan given to the concussed athletes setting out the period of time off work (or school), driving restrictions where relevant, and any other restrictions to their activity. The period of restriction is should be until full clinical and cognitive recovery occurs and then the recovery should be medically documented for medicolegal reasons. In general terms, a concussed athlete should not play or train until all postconcussive symptoms have fully resolved.

4. A medical follow up—for example, an outpatient appointment—where an clinician experienced in concussion and mild brain trauma can assess the recovery of the patient and provide support or guidance where indicated.

5. Thorough and contemporaneous documentation of each of the above steps in the patient's medical record.

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