Concussion in children

Can we manage sport related concussion in children the same as in adults?

P McCrory, A Collie, V Anderson, G Davis

Evidence based guidelines are required for the management of concussive injury in children

Consensus guidelines for managing sport related concussion in adults have been increasingly widely implemented. So far, there are no guidelines that enable clinicians to manage similar sporting concussive injuries in children. Furthermore, there are a number of important anatomical, physiological, and behavioural differences between adults and children that suggest that adult guidelines will need to be either modified or rewritten to manage injuries in this age group.

**EPIDEMIOLOGY OF CONCUSSION IN CHILDREN**

The annual incidence of traumatic brain injury (TBI) in adults is remarkably constant worldwide and has been estimated at between 180 and 300 cases per 100,000 population. This is believed to be an underestimate of the true incidence as an equivalent number of mild injuries are treated by general practitioners and do not result in hospital admission. Direct sport participation accounts for approximately 15–20% of all such TBI and in children a further smaller percentage of TBI is associated with play activities.

In children aged 15 years and under, the estimated incidence rate of TBI is 180 per 100,000 children per year of which approximately 85% are categorised as mild injuries. In the US, it has been estimated that more than 1 million children sustain a TBI annually and that TBI accounts for more than 250,000 paediatric hospital admissions as well as more than 10% of all visits to emergency service settings.

In child and adolescent populations, few well controlled studies exist to identify the age specific frequency and outcome of sport related concussive injuries.

**INTRINSIC DIFFERENCES BETWEEN CHILD AND ADULT CONCUSSION**

The most common cognitive sequelae of concussive injuries in children are the same as for adults, namely reduced speed of information processing, poor attention, and impaired executive function. Concussion may also have a significant negative secondary impact upon educational and social attainment, as these processes are critical for performing common day to day activities in childhood and adolescence, such as acquiring new knowledge and attending to school work. The nature, magnitude, and duration of these post-concussion cognitive impairments are yet to be determined, and the academic and social consequence for the child remains unknown at this time. In addition, it has been reported that children may suffer from a variety of post-concussion behavioural sequelae despite normal neuropsychological testing.

It is also known that brain tolerance to biomechanical forces differs between adults and children. In broad terms, a two to three fold greater impact force is required to produce clinical symptoms in children compared to adults. This is due to a combination of factors, including an age dependent physiological response to mechanical stress, the differing geometry of the skull and brain, and the constitutive structural properties of the head. This means that if a child exhibits clinical symptoms after head injury, then it is reasonable to assume that they have sustained a far greater impact force compared to an adult with the same post-concussive symptoms.

There are rare but well recognised post-traumatic clinical sequelae that occur predominantly in children and teenagers, such as diffuse cerebral swelling. The concussive impact, however trivial, sets in train the rapid development of cerebral swelling that usually results in brainstem herniation and death. Its cause is unknown but is thought to involve disordered cerebral vascular autoregulation. Although repeated concussive injuries have been proposed as the basis for this syndrome (the so-called second impact syndrome), the evidence for repeated concussion as a specific risk factor is not compelling. It is more likely that a single impact of any severity may result in this rare complication; however, participation in sport simply draws attention to incidental concussive injuries in this setting. There is also limited published evidence that a specific genetic abnormality predisposes to brain swelling following mild head injury in children.9

**NEUROPSYCHOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS**

Over and above any cognitive effects of concussion, there is an additional issue that makes assessment difficult, namely the fact that the brain is cognitively maturing during this period. This has two major implications. Firstly, the child’s brain potentially may be more vulnerable to the impact of head injury than the more mature adult brain due to the disturbances of neuronal maturation caused by brain trauma. Secondly, unlike adults where cognitive function is relatively stable over time, children’s cognition continues to develop. Thus any assessment of baseline or post-injury cognitive function needs to factor in the normal maturation in cognition that is occurring over this period. Pilot unpublished cross sectional data collected in 180 healthy children using a computerised cognitive test paradigm (CogSport) indicates substantial improvement in performance between the ages of 9 and 18 years on tests of simple and choice reaction time, working memory, and new learning. The largest improvements in test performance occur between the ages of 9 and 15, with minimal changes after this age paralleling adult performance (fig 1).

These developmental changes are of comparable magnitude to post-concussive impairments observed on computerised cognitive post-injury assessment in adults. This has the potential for confusion in assessment given that maturational improvements occurring between baseline and post-concussion testing may offset any injury related cognitive impairment in concussed children and adolescents.

**RISK FACTORS FOR SEQUELAE AND POOR RECOVERY AFTER CONCUSSION IN CHILDREN**

It has been argued that individuals are not at equal risk for symptoms and cognitive dysfunction following concussion, and also that some individuals may be at higher risk of sustaining concussion than others. There is circumstantial evidence that a prior history of brain injury is a risk factor for subsequent concussive injury in children.

Other potential risk factors that may predict more severe neurobehavioral
sequelea following injury, especially in younger children where the brain is less mature, include the presence of pre-morbid cognitive, attention, and behavioural impairments.15–26

It has also become a widely held belief that having sustained a sport related concussive injury, an athlete is then more prone to future concussive injury. The evidence for this in sport is limited with most studies being methodologically flawed.15–19 It has been suggested that individuals with more aggressive playing styles may be at the greatest risk of concussion.

Although helmet use may be effective in preventing superficial head injury in children, its role in preventing concussive injury and other forms of traumatic brain injury remains unproven. Furthermore, recent studies have shown a differential behavioural response of children to protective equipment with some adopting increased risk taking behaviour and hence paradoxically increasing their risk of sustaining a concussive injury by wearing a “protective” helmet.30–31

There is also evidence that an individual’s genetic make up may predict outcome from head injury. In particular, an association has been identified between the apolipoprotein E ɛ4 allele and poor clinical outcome in adult patients with mild head injury.15–18 Although only in the early stages of understanding, the interaction between genetic and environmental factors may be critical in the development of post-concussive phenomena. These data suggest that it may be possible to identify individuals who are at greater risk for poor outcomes from concussion and in the future management practices may need to be tailored to incorporate such information.

CAN WE USE ADULT CONCUSSION RETURN TO PLAY STRATEGIES IN CHILDREN?

There have been numerous attempts in the past to formulate evidence based concussion management guidelines,35 with that developed at the recent Vienna Consensus Conference1 being most widely accepted today. In broad terms, this approach recommends baseline cognitive testing to enable accurate individual assessment of recovery, in order to guide return to play following concussion. There are no current guidelines for diagnosis and management of concussion in children beyond generic recommendations for observation and neuroimaging following childhood mild head injury. This is also reflected in the variable specialist clinical management that may be offered in this situation. In a recent pilot study of paediatric neurosurgical management of sport related concussive injury in children, there was no consensus between the surgeons being studied as to the significance of specific clinical symptoms or on recommendations regarding hospitalisation, time off school and sport, or the use of protective equipment following injury.38

The “comparison to own baseline” model of assessment remains a powerful method of assessing change in cognitive function after concussion, and in the absence of conflicting evidence, should be adopted as a conservative approach to identifying post-concussion cognitive deficits in children as it is in adults. The central issue is how often baseline testing should be conducted. During the period of rapid cognitive maturation (8–15 years of age), baseline testing would have to be performed at least 6 monthly to enable accurate comparison for serial testing. Apart from elite junior athletes, such regular testing would be beyond the resources of most sports and individuals. For any child or adolescent athlete participating in collision sport or where there is a significant risk of concussion, annual cognitive testing should be considered. Any statistical decision about whether cognition has changed from baseline following concussion must also include an adjustment for developmental changes in cognition. This in turn requires knowledge of how performance changes on specific tests over time. Figure 1 demonstrates the cognitive maturation process that is maximal between 8 and 15 years of age and gives an estimate of the degree of this change. Beyond 15 years of age an annual baseline test would be suitable, as for adult athletes, and be applied in the same fashion as for adults and without any developmental increment.

THE CLINICAL MANAGEMENT OF CONCUSSION IN CHILDREN

Current adult management of concussion involves an initial diagnosis using a validated assessment tool such as the Maddocks questions36 or the Standardized Assessment of Concussion.37 Neither tool has been specifically tested or validated in children with concussion.

One preliminary study suggests that high school aged children (14–18 years) may also have prolonged cognitive recovery when compared with young adults (18–25 years), but that symptom recovery is equivalent between these groups.34 This finding raises the possibility that symptom ratings and cognitive testing may be differentially sensitive to concussion in minors and adults; however, there is no such information available for younger children. This in turn raises concerns as to how sport related concussion assessment may be performed in these age groups and the validity of existing assessment tools.

Return to play concussion guidelines recommend baseline cognitive evaluation of all individuals participating in contact and collision sport.7 The heterogeneity of concussion ensures that individual comparison to baseline allows more sensitive identification of post-concussion symptom elevation and cognitive dysfunction than arbitrary classification according to a retrospective grading scale.39–40

Statistical models used to determine the significance of any observed post-concussion cognitive change require knowledge of how cognitive test performance and symptom ratings change in
healthy, uninjured individuals. In adult populations (and potentially children aged 16 years and over) available data suggest that cognitive performance remains relatively stable over time on tests commonly used in concussion management. These findings support the need for prospective serial investigation of cognitive and behavioural function in healthy and concussed children and adolescents, including specific assessment of how cognitive processes (including response variability and change within individuals) may be affected. Such data will be invaluable in informing the development of concussion management guidelines in this population, and the interpretation of post-concussion cognitive test data.

The issue of neuroimaging is often raised for children following mild head injury. With young patients, this issue is often problematic given that they may require a general anaesthetic in order to obtain adequate images, although new generation spiral CT scanners are able to perform extremely rapid imaging sequences. If imaging is desired by the health practitioner in this setting or by the subsequent development of symptoms of intracranial pathology, then CT scanning is the imaging modality of choice in the emergency setting. The American Academy of Paediatrics guidelines state that there is no indication for routine use of skull x-ray in paediatric concussion and “no data are available that demonstrate that children who undergo CT scanning early after minor closed head injury with loss of consciousness have different outcomes compared with children who receive observation alone after injury.”

In broad terms, a previously neurologically healthy child with a concussive injury who has normal mental status, no abnormal or focal abnormalities on neurological exam, and no physical evidence of skull fracture simply requires observation by a competent caregiver. The risk of clinically significant intracranial pathology in this setting is less than 0.02% although earlier studies had suggested higher figures. It is likely that many of these early studies suffered from selection bias and over estimated this risk in this situation.

SUMMARY
At the present time, there are no evidence based guidelines using which sport related concussive injury in childhood and adolescence can be scientifically managed. There are significant differences between adults and children in this regard and a child who is symptomatic following head injury is likely to have sustained a far greater impact force as compared to an adult with the same post-concussive symptoms.

The extent and duration of the cognitive effect on children with acute concussive injuries is variable and there may be persistent effects on scholastic performance and behaviour long after the clinical concussive symptoms and measurable neuropsychological impairment have resolved. Even subtle and transient impairments in attention and information processing skills can have a dramatic effect on the young child’s capacity to cope with school demands, with these issues being particularly critical for those at later secondary school levels.

Based on pilot data, cognitive maturational growth is greatest in those under 15 years of age and beyond this time plateaus to an adult level of performance. Although comparison to baseline cognitive performance remains a powerful method of assessing function following a concussion injury, its application in children under 15 years of age is problematic given the rapid cognitive maturation that is occurring in this period. With regular baseline testing, an “adult” management strategy could be adopted in this age group, whereas in its absence only an estimate of normal age related cognitive function can be made. Beyond 15 years of age, it would be reasonable to follow the adult concussion management consensus guidelines utilising a “return to baseline” approach.

It is suggested that concussive symptoms take longer to resolve than in adults although this may be a surrogate marker of the biomechanical differences between children and adult concussion as outlined above. It is critical therefore that concussed children and adolescents not resume sport, school, or training until all the physical symptoms fully resolve. This is important also because of the risk of diffuse cerebral swelling that may occur in children after a single head injury no matter how trivial the impact may be. The adoption of a conservative adult management strategy with a thorough assessment of symptom resolution followed by “return to baseline” cognitive function remains the most appropriate management strategy in this age group.

Further research is required to characterise the duration and nature of the subclinical cognitive impairment that may exist during this recovery period in children. Increased awareness of these issues by those involved in the management of a child with concussion may assist in avoiding problems caused by this putative impairment.


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REFERENCES
Sport psychology and concussion

I

n recent years, there has been great interest in examining the psychological effects of athletic injuries. This has also extended to interventions in which coping strategies have been suggested to enhance recovery. Concussive injuries, which are common to many sports, hold particular problems in this regard. For example, a concussed athlete may be prone to experience isolation, pain, anxiety, and disruption of daily life as a result of the injury. This may be a problem for individual sport athletes—for example, professional skiers—who do not have the support of team mates to help them through their rehabilitation and recovery, as well as team sport athletes whose team mates may inadvertently pressure them to return to play.

Besides the physical loss resulting from an injury, there may also be psychological distress. Commonly reported emotion responses resulting from athletic injury have included anger, denial, depression, distress, bargaining, shock, and guilt.1-3 These are particularly seen in career ending injuries. Such emotional distress can negatively affect the athletes’ recovery process.

“...concussed athletes in team sports seem to have fewer long term problems”

Injured athletes have also reported feelings of isolation and loneliness. Researchers found that athletes prevented from participating in their activity have lost contact with their team, coach, and friends.1,3 For example, Gould et al4 examined the emotional reactions of US national team skiers to season ending injuries and found that 66.6% cited lack of attention and isolation as a source of stress during their injury. In another study of injured athletes, Brewer et al5 surveyed 43 sports medicine practitioners to discover side effects of psychological distress. These side effects included exercise addiction, weight control problems, family adjustment, and substance abuse. These problems have been reported individually as well as being associated with depression and anxiety and have been shown to cause severe health complications.7

Injured athletes have reported different levels of satisfaction with the social support they have received after injury. In particular, team mates have been shown to have a greater affect on the emotional state of injured athletes than coaches or medical professionals.8 This leads one to speculate that individual sport athletes may experience different adjustment difficulties while recovering from a concussion. This may also suggest why concussed athletes in team sports seem to have fewer long term problems than non-contact athletes.6 This is an environment in which team mates are likely to have experienced similar injuries, there is a greater corporate memory of such injuries and hence more reassurance as to the likely recovery time frame and validation of subjective symptoms experienced by the injured athlete.

UNIQUENESS OF CONCUSSION INJURIES

A number of unique characteristics of concussion injuries exist. Firstly, a concussion is an “invisible injury”. This

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means there are no crutches, swelling, stitches, or other visual signs of the injury. This makes it very difficult for a casual observer to identify the athlete as injured. A second unique characteristic of concussion injuries is the overlap of post-concussive symptoms with psychological responses to injury. A third problem may be a loss of fitness (through loss of both aerobic and resistance training activity), particularly if the post-concussive symptoms persist. A fourth problem, unique to professional sport, is the relentless media commentary that typically occurs after injury. This increases pressure on injured athletes to regain their sporting place.

...it is often not considered acceptable to spend prolonged time rehabilitating a concussion injury.

Another issue surrounding concussion injuries is the lack of acceptance or understanding of long term rehabilitation. Whereas it is often considered necessary to rehabilitate an orthopaedic injury for a number of months, it is not considered acceptable to spend prolonged time rehabilitating a concussion injury. This may result in more anxiety and frustration for the athlete and coach. Finally, concussion injuries are unique because there is at present no standard intervention technique. For most other injuries, rehabilitative treatments are available—for example, physiotherapy, medication, exercise, surgery, etc. However, this is not the case for concussion injuries; the athlete may leave the doctor with either no specific management or at best a minimally structured treatment plan.

SOCIAL SUPPORT GROUPS

It is not uncommon for people who have experienced life events such as addiction, illness of a family member, injury, or a significant loss to have difficulty dealing with the stress and anxiety of their situation. As a result of the distress encountered, different methods of coping and psychological support have been developed. Throughout the last 20 years, the role of social support in dealing with disease has increased significantly. Support groups have been developed to educate, prevent isolation, and help in coping. Positive outcomes from support groups have been proven effective for sufferers of AIDS, cancer, obsessive compulsive disorder, and pregnancy loss, and families of critically ill patients. It has also been shown that general social support is beneficial for athletes suffering from injuries. A support group for injured athletes may also provide the same benefits as it has for non-athletic populations.

Intervention strategies and psychological rehabilitation techniques have included imagery, relaxation, modelling, goal setting, positive self talk, social support and support groups, pain management, simulation training, education, stress management, and cognitive reconstruction. Social support and communication have been two most commonly suggested rehabilitation strategies. Lynch stressed the importance of the sport psychologist in encouraging athletes to discuss their experiences with others. These interactions were designed to help reduce the injured athlete’s feelings of isolation and loneliness.

SOCIAL SUPPORT IN ATHLETIC INJURY REHABILITATION

Two distinct types of social support have been used during injury rehabilitation in an athletic setting: support groups and peer modelling. Support groups for injured athletes have allowed injured athletes to come together to voice their concerns, share ideas about coping, learn vital performance enhancement strategies, and realise that they are not alone. The goal is that athletes will support one another both mentally and physically by helping deal with the demands of rehabilitation and not participating in their sport.

Numerous studies on non-athletic populations have been published showing the benefits of support groups in reducing anxiety, depression, and isolation and enhancing coping strategies. To date there have been very few academic sources that have addressed the use of support groups for athletic injury and only one for concussion. Granito et al. offered anecdotal support for an injured athlete support group programme, and Horton et al. attempted to determine if participation in social support groups could reduce negative psychological side effects in concussion. It was shown that participants in the experimental concussed group improved their mood state, reducing effects such as anger, confusion, frustration, anxiety, depression, and isolation.

Social support has also been proven to have a significant effect on rehabilitation adherence. Udry reported that the most discriminating factor for rehabilitation adherence was level of social support. Athletes who perceived social support for their rehabilitation had better rehabilitation adherence, higher levels of motivation, and adopted a goal mastery orientation towards their rehabilitation.

SUMMARY

The use of sport psychology techniques in the management of concussion may assist in solving some of the real and very practical problems facing clinicians, namely certain aspects of the post-concussive syndrome and the influence of anxiety and other adverse psychological states, which in turn may impact on injury outcome. Techniques derived from other areas of psychology such as peer modelling may be successfully used in this setting.

References


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Manual therapy

To treat or not to treat: new evidence for the effectiveness of manual therapy

M M Sran

Manual therapy has been shown to be effective for certain conditions but more research is needed to identify other suitable patients.

Recent randomised clinical trials found manual therapy to be more effective than other methods of conservative management for low back and neck pain. On the other hand, some randomised clinical trials, systematic reviews, and meta-analyses concluded that there was no evidence that spinal manipulative therapy is superior to other standard treatments for patients with low back or neck pain. This provides the clinician with a Shakespearean quandary—to treat or not to treat using manual therapies? Therefore, this leader addresses the question: what explains these apparently inconsistent data?

DEFINITIONS AND SEARCH STRATEGY

The term manual therapy has many connotations, but for this leader it includes manually performed assessment and treatment methods (which can include joint, neural tissue, and/or muscle techniques). The term manipulation is typically used to describe small amplitude thrust techniques performed with speed.

I searched Medline, Cinahl, and Embase databases for randomised clinical trials comparing spinal manual joint techniques (mobilisation with or without manipulation) or manipulation only with other conservative treatments for back or neck pain. Only studies published as full papers, in English, between 1 January 1998 and 31 December 2003 were included. Pilot studies were not included. Table 1 outlines search strategies for each database.

<table>
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<th>Table 1</th>
<th>Search strategy</th>
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<tr>
<td>Database</td>
<td>MeSH headings</td>
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<tr>
<td>Medline</td>
<td>Manipulation, orthopaedic</td>
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<td></td>
<td>Manipulation, chiropractic</td>
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<td>Manipulation, osteopathic</td>
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<td>Physical therapy techniques</td>
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<td></td>
<td>Musculoskeletal manipulations</td>
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<td>Comparative study</td>
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<td>(Back or neck) and pain</td>
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<td>Chiropractic</td>
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<td>Chiropractic manipulation</td>
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<td>Manipulation, orthopaedic</td>
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<td>Osteopathy</td>
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<td>(Back or neck) and pain</td>
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<tr>
<td>Embase</td>
<td>Manipulative medicine</td>
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<td>(Back or neck) and pain</td>
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DIFFERENCES IN MANUAL THERAPY THAT MAY EXPLAIN STUDY FINDINGS

Whether or not the study used manual therapy or manipulation only

Four of the 13 studies reported better results in the manual therapy group than the other group(s). Five of the remaining nine studies used manipulation only, and all but one reported no significant difference or a poorer response than the other group(s).

Use of a variety of manual therapy techniques, rather than joint manipulation alone, appears to yield better results. For example, Jull et al studied the effectiveness of manual therapy delivered by physical therapists, specific exercise therapy delivered by physical therapists, combined manual and specific exercise therapy, and a control group, for treatment of cervicogenic headache. At the 12 month follow up, both manual therapy and specific exercise groups had significantly reduced headache frequency and intensity, neck pain, and disability. In this study, manual therapy included both low velocity cervical joint mobilisation techniques and high velocity manipulation techniques. These results are relevant to physical therapists with postgraduate certification in manual therapy, as they are well trained in both of these techniques. Similarly, Hoving et al...
<table>
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<tr>
<th>Reference</th>
<th>Population characteristics (n)</th>
<th>MT limited to manipulation only</th>
<th>MT delivered by:</th>
<th>Clinically relevant, guideline based MT</th>
<th>Interventions, groups</th>
<th>Control group</th>
<th>Dose (MT or manipulation)</th>
<th>Results</th>
<th>Effect size for positive studies</th>
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<tr>
<td>Aure et al⁷</td>
<td>20–60 years; chronic LBP &gt; 8 weeks, less than 6 months (49)</td>
<td>No</td>
<td>Physical therapists</td>
<td>Yes</td>
<td>1. MT plus ET. 2. ET alone</td>
<td>No</td>
<td>45 min (15 min MT); 2 sessions/week, 8 weeks</td>
<td>Significantly larger improvements in MT group (maintained at 1 year follow up)</td>
<td>0.78</td>
</tr>
<tr>
<td>Hoving et al²</td>
<td>18–70 years; pain or stiffness in the neck for at least 2 weeks (183)</td>
<td>No</td>
<td>Physical therapists</td>
<td>Yes</td>
<td>1. MT plus specific exercise training. 2. Active exercise focused physical therapy. 3. Continued care by GP</td>
<td>No</td>
<td>45 min; 1 session/week for up to 6 weeks</td>
<td>Physical therapy including MT more effective than physical therapy without MT or continued care by a GP</td>
<td>Not given</td>
</tr>
<tr>
<td>Jull et al⁶</td>
<td>18–60 years; cervicogenic headache at least 1 week for 2 months–10 years (200)</td>
<td>No</td>
<td>Physical therapists</td>
<td>Yes</td>
<td>1. MT. 2. ET (low load endurance training). 3. Combined MT and ET. 4. Control</td>
<td>Yes</td>
<td>30 min, 8–12 sessions, 6 weeks</td>
<td>MT as effective as ET and both significantly better than control</td>
<td>0.80</td>
</tr>
<tr>
<td>Moseley⁷</td>
<td>Chronic LBP &gt; 2 months (57)</td>
<td>No</td>
<td>Physical therapists</td>
<td>Yes with respect to clinical relevance (individualised and variety of techniques allowed but no references cited for MT techniques)</td>
<td>1. MT, specific exercise training, and neurophysiology education. 2. Medical management by GP</td>
<td>No</td>
<td>2 ×/week, 4 weeks</td>
<td>Combined physiotherapy treatment including MT, specific exercise training, and neurophysiology education resulted in improved function and pain at 1 and 12 months.</td>
<td>Not given</td>
</tr>
<tr>
<td>Giles &amp; Muller³</td>
<td>17 years or older; mechanical back or neck pain for a minimum of 13 weeks (115)</td>
<td>Yes</td>
<td>Chiropractors</td>
<td>No for LBP. No for neck pain</td>
<td>1. Spinal manipulation. 2. Sports physician follow up (limited) and medication. 3. Acupuncture (needle)</td>
<td>No</td>
<td>20 min, 2 ×/week, maximum 9 weeks</td>
<td>Greater short term benefit for manipulation, but not for neck pain. Acupuncture more effective for neck pain.</td>
<td>Not given</td>
</tr>
<tr>
<td>Andersson et al</td>
<td>20–59 years; LBP lasting at least 3 weeks but less than 6 months (178)</td>
<td>No</td>
<td>Osteopaths</td>
<td>Yes</td>
<td>1. Osteopathic treatment. 2. “Standard care” by physicians</td>
<td>No</td>
<td>1 ×/week for 4 weeks</td>
<td>No significant differences between groups. Both groups improved</td>
<td>Not given</td>
</tr>
<tr>
<td>Bronfort G et al⁷</td>
<td>20–65 years; mechanical neck pain for at least 12 weeks (191)</td>
<td>Yes (but this group also received 45 min of sham microcurrent therapy)</td>
<td>Chiropractors</td>
<td>No. A reference for the use of spinal manipulation for LBP is cited, but only cervical and thoracic spine techniques were used.</td>
<td>1. Spinal manipulation plus upper body and neck strengthening exercise. 2. Aerobic exercise plus MedX cervical extension and rotation machine. 3. Spinal manipulation</td>
<td>No</td>
<td>20 × 1 hour sessions over 11 weeks</td>
<td>No significant difference between groups with respect to pain, neck disability, medication use</td>
<td>Not given</td>
</tr>
<tr>
<td>Cherkin et al⁷</td>
<td>20–64 years; LBP minimum 7 days after seeing physician (321)</td>
<td>Yes</td>
<td>Chiropractors</td>
<td>No, side lying only</td>
<td>1. Chiropractic manipulation. 2. Education booklet. 3. McKenzie exercises</td>
<td>No</td>
<td>Up to 9 × over 1 month</td>
<td>No significant difference between groups</td>
<td>Not given</td>
</tr>
<tr>
<td>Curtis et al⁷</td>
<td>21–65 years; acute LBP of less than 2 months (295)</td>
<td>No (manipulation plus muscle energy techniques)</td>
<td>Physicians with limited training (18 h) in manual therapy</td>
<td>No</td>
<td>1. Manipulation and muscle energy techniques plus enhanced care 2. Enhanced care alone</td>
<td>No</td>
<td>Initial plus 4 follow ups; 2 ×/wk for 2 weeks</td>
<td>Only 43% of patients in the MT group actually received the planned treatment; no significant difference between groups</td>
<td>Not given</td>
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compared physical therapy including manual therapy with physical therapy without manual therapy for patients with chronic neck pain. Of note, they allowed the use of low velocity joint mobilisations but no high velocity low amplitude thrust techniques (synonymous with “manipulation”).

Was the choice of intervention based on clinically relevant treatment guidelines (“best practice”) of the discipline?

Assessment and treatment protocols used in randomised controlled trials (RCTs) are not always similar to clinical practice guidelines, which are typically textbooks or guidelines written by experts in the field/discipline and based on current available evidence. Treatment protocols that do not mirror clinical practice have been examined in some studies. For example, Andersson et al compared osteopathic treatment (including manual therapy) with “standard care” by doctors. However, the reported standard care included medication, active physical therapy, ultrasonography, diathermy, hot or cold packs (or both), use of a corset, or transcutaneous electrical nerve stimulation (TENS). Clearly health maintenance organisation doctors do not have the time (45 minutes), equipment, or skills—that is, active physical therapy—to provide this treatment. Further, two of the groups studying manipulation by chiropractors included participants with back or neck pain, yet they only cited references for low back pain management. Three studies used very restricted manual assessment and/or treatment techniques which do not reflect best practice. Three of the five studies with positive results used manual treatment (by physical therapists) based on published guidelines or clinical texts written by experts in the field.3–5

The dose of manual therapy or manipulation (minutes, sessions, weeks)

The optimal dose is also a consideration. Time per session, number of sessions, and number of weeks are all important factors for therapists, patients, and payors.

Knowing the optimal treatment duration has obvious implications on cost effectiveness, but probably also has an impact on the effectiveness of manual therapy. Despite the importance of these variables, there is great variability between the protocols used in these 13 studies. One study compared chiropractic care only, medical care only, medical care with limited physical therapy, and chiropractic care with modalities but did not prescribe a treatment dose.
However, they did monitor use of the various treatment modes and time per session and found that one third of patients randomly assigned to medical care with physical therapy had no physical therapy visits, and 20% of patients in the chiropractic groups received concurrent medical care, whereas only 7% of patients in the medical care groups received concurrent chiropractic care. They also report that chiropractors and medical providers in their study spent an average of 15 minutes with patients at each visit, and physical therapists averaged 31 minutes per patient visit.

Only six studies reported the time per session. Time varied from 20 to 60 minutes per treatment. Of interest, three of the five studies with positive results allowed between 30 and 45 minutes per treatment. One (of the studies with positive results) did not report treatment time, and the other had mixed results (positive for back pain but not for neck pain) and allowed 20 minutes per treatment.

The total number of sessions varied from 5 to 20, with a frequency of between once a week and three times a week. Some studies did not prescribe a maximum or minimum number of sessions and a week (table 2).

The number of weeks of treatment varied from 3 to 12. Of note, the five studies with positive results used between four and nine weeks of treatment.

Combination therapies
A number of studies have investigated a combination of therapies such as two healthcare professionals or a combination of manual therapy or manipulation with another mode of treatment.

Of note, four of the five studies with positive results used manual therapy in combination with another aspect of physical therapy (exercise therapy, specific exercise training, and neuro-physiology education). Similar positive results were not seen in chiropractic, physical therapy visits, and 20% of patients randomly assigned to medical care with chronic neck pain. Further investigations of the effectiveness of manual therapy in special populations are needed. Pilot studies have been conducted in patients with thoracic pain, cervicobrachial pain syndrome, and we have conducted studies on the safety of manual therapy in the osteoporotic spine. The next step is for researchers to conduct well designed RCTs to determine the effectiveness of manual therapy for pain and disability in these populations.

ACKNOWLEDGEMENTS
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SUMMARY AND CONCLUSIONS
In summary, I return to the question that was the genesis of this leader, what explains the apparently inconsistent data in the field of manual therapy outcomes? Critical appraisal suggests that more precise interventions are successful in treating low back pain, chronic neck pain, and cervicogenic headache. There are clinically relevant differences between studies reporting positive results of manual therapy and those reporting no significant difference over other conservative treatments. Specifically, the treatment protocol needs to reflect what therapists are actually doing in clinical practice—that is, using manual therapy technique or combining manual therapy with other modes of treatment such as specific exercise training. Interventions based on best practice guidelines/texts appear to be more successful, and physical therapy including manual therapy at a dose of 30–45 minutes per session, for four to eight weeks has been shown to be effective.

Further research is needed to identify populations who are most likely to improve with manual therapy. For example, Flynn et al identified five variables to form a clinical prediction rule for patients with low back pain who are likely to respond favourably to a specific manipulative technique. In that study decisions on the side to be manipulated were not based on clinical best practice guidelines and only one manipulation technique was used (thus not representative of clinical practice), yet this approach to refine clinically relevant procedures may prove very useful.

Finally, manual therapy is not only used in the treatment of low back and neck pain. Further investigations of the effectiveness of manual therapy in special populations are needed. Pilot studies have been conducted in patients with thoracic pain, cervicobrachial pain syndrome, and we have conducted studies on the safety of manual therapy in the osteoporotic spine.

RETROSPECTIVE POWER
Retrospective power has limitations as described in detail elsewhere, thus all RCTs should calculate power a priori.

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ELECTRONIC PAGES

BJSM Online case reports: http://bjsm.bmjjournals.com/

Aseptic bone necrosis in an amateur scuba diver
G D M Laden, P Grout
A case is reported that provides further evidence of an old occupational hazard, dysbaric osteonecrosis, presenting in a new population (sports scuba divers) who also appear to be at risk. It highlights the need for an accurate diagnosis of diving related illness.
(Br J Sports Med 2004;38:e19) http://bjsm.bmjjournals.com/cgi/content/full/38/5/e19

Abdominal coarctation in a hypertensive female collegiate basketball player
B Sloan, S Simons, A Stromwall
The purpose of the preparticipation examination is to identify health conditions that might adversely affect an athlete while participating in sport. Hypertension is the most common. This case report details a female basketball player found to be hypertensive, and complaining of fatigue, at her preparticipation physical examination. Presentation, diagnostics, treatment, and final outcome of coarctation involving the abdominal aorta are summarised.
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Clinical and magnetic resonance imaging features of cricket bowler’s side strain
D Humphries, M Jamison
The clinical features of 10 cases of lateral trunk muscle injury in first class cricket pace bowlers are described. Typically the injury occurs during a single delivery, is associated with considerable pain, and prevents the bowler from continuing.

The clinical picture is typical of a muscular or musculo-tendinous injury. The most consistent clinical tests were focal tenderness on palpation and pain with resisted side flexion towards the painful side. The magnetic resonance image in 70% of cases was consistent with an injury to the internal oblique, the external oblique, or the transversalis muscles at or near their attachments to one or more of the lowest four ribs. The injury occurs on the non-bowling arm side. Recovery can be prolonged. The injury was a recurrence in six of the 10 cases. The biomechanics of the injury are not yet understood.
(Br J Sports Med 2004;38:e21) http://bjsm.bmjjournals.com/cgi/content/full/38/5/e21
To treat or not to treat: new evidence for the effectiveness of manual therapy

M M Sran

doi: 10.1136/bjsm.2003.010876

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Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twerpendal et al. is important for a number of reasons, not all of which may have been emphasised sufficiently by the authors.

Firstly, it confirms that a rate of fluid intake of 1000 ml/h is too high for a group of female runners running at ~10 km/h and who would therefore complete a 42 km marathon in about 4.25 hours. As the athletes drank 4 litres and gained 2 kg during the trial, their average rate of weight loss (as opposed to sweat rate) was about 500 ml/h. As not all of the weight loss during exercise is sweat and as much as 1–3 kg of this weight loss may result from fuel and water losses that do not contribute to dehydration, the absolute maximum rate at which these athletes should have ingested fluid during exercise was probably even less than 500 ml/h. This is substantially less than the drinking guidelines of the American College of Sports Medicine and the Gatorade Sports Science Institute, which have promoted rates of fluid ingestion of up to 1200–1800 ml/h. As there is no evidence that gaining weight during exercise improves performance, there is good evidence that athletes who either lose no weight or who gain weight during exercise are increasingly likely to (a) have an impaired performance, (b) develop troubling gastrointestinal symptoms, or (c) finish the race with serum sodium concentrations below those of the athletes who ingested hyponatraemic encephalopathy. It is not immediately clear why the authors chose such high rates of fluid intake in these athletes. Except, perhaps, if they wished to "prove" the value of sodium ingestion during exercise. I note, for example, that the study was funded by a commercial company that, I am informed, markets a sports drink containing sodium chloride.

For it seems highly probable that if athletes overdrink so that they retain fluid and gain weight, then the extent to which their serum sodium concentration falls will be influenced, albeit to a quite limited extent, by the sodium content of the ingested fluids. This indeed was shown by the results of this study. But whether that finding has relevance to the sodium requirements of athletes who are specifically advised not to overdrink during exercise to ensure that they do not develop hyponatraemic encephalopathy is an entirely different question, which cannot be answered with the study design chosen by these authors.

For example, the presence of a control group who drank according to the dictates of thirst ("ad libitum") and not according to the guidelines of influential sports medical and commercial organisations, so that they may be less prone to overdrink and so to gain weight during exercise, would have established that athletes who lose more than 1–3 kg during exercise do not develop symptomatic hyponatraemic encephalopathy even though they are both dehydrated and sodium deficient. Rather, they are more likely to finish such races with raised serum sodium concentrations.

I would rather argue that a fundamental feature of all prospective trials that aim to evaluate a novel intervention such as the role of sodium ingestion in the prevention of hyponatraemia during exercise should be to compare the new intervention with the currently established best practice.

As the currently established best practice is not to ingest fluid at such high rates that weight is gained during exercise, because this practice can produce a fatal outcome, so this study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defendable evidence base are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest Group.

Fortunately the data of Twerpendal et al. do allow some calculations to estimate the likely value of the extra sodium that was ingested by two of their groups. Thus, the athletes in their study had an average body mass of 54 kg. According to the formula of Montain et al., their predicted extracellular fluid (ECF) volume would be about 14.5 litres (25% of body weight). As the starting serum (and ECF) sodium concentration ([Na+]i) in the three groups of runners was ~137 mmol/l (table 3 of their article), the average total ECF Na+ content of the three experimental groups was 1899–1993 mmol at the start of the race. As weights increased by 1.8–2.1 kg in the three groups during exercise (table 3 of their article), the increases in ECF volume would have been 450–525 ml in the respective groups, assuming that the ECF increased in proportion to the increase in total body water (TBW). Multiplying this new ECF volume by the serum [Na+] after the race gives the new total ECF Na+ content after the race. As shown in table 1, the total ECF Na+ content increased by 34 mmol in the group that ingested the high salt drink (H) during the race, but fell by 23 mmol in the group drinking water (W). As all groups ran for about four hours, according to these calculations based on these assumptions, the hourly rates of Na+ loss would have varied from 6 to 21 mmol/h, giving a sweat [Na+] of 12–42 mmol/l in the W and H groups respectively (as their total sweat losses were ~2 litres in each group).

The clear paradox identified by the calculations in table 1 is that (a) the total Na+ loss apparently increases with increased Na+ intake and (b) the estimated Na+ loss in the group who ingested only water during the race (W) is less than one third of that in the group who ingested the most Na+ (H) during the race.

As these calculations are based on two real measurements (body weight changes and changes in plasma [Na+]), this apparently ludicrous conclusion can only be explained if the Na+ ingestion during exercise increases whole body Na+ losses in sweat and urine or (b) the estimated ECF volume in the W group after exercise is less than the value calculated. That is, specifically in the W group, the ECF volume contracted despite an increase in TBW of 1.9 litres. Indeed, this response is to be expected. There is consistent evidence that the response of the ECF and the intracellular fluid (ICF) volumes to fluid ingestion during prolonged exercise are influenced by the Na+ content of the ingested fluid so that the ECF volume is likely to fall less if either water or a dilute Na+ drink is ingested, or to expand if a concentrated (50–100 mmol/l) Na+ drink is ingested at the same rate that body weight is lost during exercise. In the latter case, any reduction in the TBW appears to come from a reduction in the ICF.

For example, if each group did indeed lose 84 mmol Na+ as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat [Na+] of ~40 mmol/l, then the true ECF volume in the W group after the race would have been 14.5 litres—that is, it is unchanged from the starting value. This value (equivalent to 1993 mmol) is calculated as: (pre-race ECF Na+ content – 84) in mmol divided by post-race serum [Na+] in mmol.

Indeed, if subjects in the W group did lose 84 mmol Na+ during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race [Na+] would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is therefore that intra-group comparisons (ie, comparing the groups who ingested Na+ with those who ingested only water during exercise) are inappropriate analyses. It is more reasonable to compare the new intervention with the currently established best practice. The recent recommendations by the International Marathon Medical Interest Group is one of the stable guidelines for athletes who are exercising to ensure that they do not develop hyponatraemic encephalopathy.
encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na⁺ deficit either during exercise¹¹−¹³,¹⁷−¹⁸ or at rest.¹⁹ A recent paper²⁰ confirms this prediction by showing that mathematical modelling supports the argument that changes in TBW exert a much greater effect on serum [Na⁺] than does whole body Na⁺ content in those who over-drink and hence gain weight during exercise. Perhaps the point of these calculations is to show that it is not possible to calculate the state of Na⁺ balance in athletes during exercise and so to determine whether or not athletes have developed a Na⁺ “deficit”²¹,²²−²⁵ simply by measuring serum [Na⁺]. This is because the ECF volume will not be the same before, during, and after exercise and will change depending on the nature of the fluid ingested and the extent of any fluid deficit or excess that develops during exercise.²⁶,²⁷ But more importantly, these calculations clearly show why the regulation of the TBW and the ECF volume will have a much greater influence on serum [Na⁺] than will either the expected Na⁺ losses in sweat or the amount of Na⁺ ingested from sodium-containing sports drinks.²⁸

For example, a 1 litre (7%) reduction in the ECF volume would “release” 140 mmol Na⁺ into the contracted ECF volume. This means that it is possible to lose 140 mmol Na⁺ in sweat and urine without any change in serum [Na⁺] provided that the ECF volume were to contract by only 7%. If sweat [Na⁺] is about 40 mmol/l, as appears to have been the case in this study of Twerenbold et al.¹⁹ (table 1), then this 140 mmol is the equivalent of the Na⁺ content of about 3.5 litres of sweat.

As athletes in this study sweated at a maximum rate of only 500 ml/h when running at 10 km/h, this means that simply by reducing their ECF volume by 1 litre, those athletes could have maintained their pre-race serum [Na⁺] while running for seven hours and drinking just sufficient water to allow for a 1 litre reduction in ECF volume and without requiring any Na⁺ replacement whatever. This simple calculation explains why those endurance athletes who, before about 1969, were advised either not to drink at all, or only sparingly during exercise,²⁹ always finished the races with raised serum [Na⁺]³⁰−³³ despite having incurred what might have been quite sizeable Na⁺ deficits.

In contrast, athletes in this study who believed the incorrect advice that ingesting Na⁺ at high rates is essential to maintain a normal serum [Na⁺] during exercise,²⁹−³⁰,³²−³⁵ so they overdrank sufficiently to increase their ECF volume by 1 litre, would need to ingest and retain at least an additional 140 mmol Na⁺ in addition to the ~80 mmol lost in sweat (table 1). This is equivalent to the Na⁺ content of 1.24 litres of the low and 7.5 litres of the high sodium drinks respectively in this trial. To maintain fluid balance in this four hour trial when drinking at those high rates and sweating at about 500 ml/h, they would then need to urinate at rates of 1375–2600 ml/h. Both of these rates exceed the maximum at which human kidneys are able to produce urine at rest,³⁵,³⁶ let alone during and after prolonged exercise.³⁵ Drunking at such rates would therefore only lead to progressive fluid accumulation and ultimately death from hyponatraemic encephalopathy.³⁷

In summary, these calculations explain (a) why contraction of the ECF in athletes who lose body weight during exercise will maintain the serum [Na⁺] even in the face of quite large and unreplaced Na⁺ loss in sweat, and (b) why the ingestion of sodium-containing sports drinks in the vain hope of matching the rates of Na⁺ loss in sweat can only lead to fluid retention and progressive hyponatraemia, as elegantly shown by this study.¹⁹ Indeed if this inappropriate behaviour is approached with sufficient vigour, ultimately the result will be death from hyponatraemic encephalopathy,³⁷ which, as these calculations and this study again show, cannot occur without the presence of distinct fluid overload.³⁸

Finally, it is important to note that, even though Na⁺ ingestion marginally increased serum [Na⁺] in the group that ingested the most concentrated Na⁺ drink, this practice was without benefit as running performances were unaltered by Na⁺ ingestion, and the incidence of symptoms was no different between the groups as no athletes reportedly developed symptoms. However, the symptoms of mild hyponatraemic encephalopathy are mild and may not have been sought with sufficient diligence. For example, all subjects, myself included, in our study in which mild hyponatraemia was induced by fluid overload at rest,³⁵ developed quite disabling symptoms at serum [Na⁺] of ~136 mmol/l or lower. Indeed it would have been most interesting to determine whether the presence of subtle mental symptoms was different in the three groups in this study, as all had similar degrees of fluid overload despite different serum [Na⁺]. If the symptoms in this condition are due purely to fluid overload, then the incidence of symptoms should have been the same in all groups despite different serum [Na⁺]. Alternatively, if the symptoms are related to the degree of hyponatraemia, then they should have been most obvious in the W group, who finished with the lowest post-race serum [Na⁺]. My bias would be to expect that the extent of any symptoms are more likely related to the degree of fluid overload, and hence the increase in the ICF, than to the level to which the serum [Na⁺] has been reduced.

Table 1  Sodium balance calculations for three groups of runners running at ~10 km/h for four hours while ingesting solutions with different [Na⁺]

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<tbody>
<tr>
<td>Pre-race</td>
<td>(mmol/l) (A)</td>
<td>(litres) (B)</td>
<td>(mmol) (A-B)</td>
<td>(mmol/l) (C)</td>
<td>(litres) (D)</td>
<td>(mmol) (C-D)</td>
<td>(mmol/l) (E-A-B)</td>
<td>(mmol)</td>
<td>(mmol/l)</td>
</tr>
<tr>
<td>H</td>
<td>137.3</td>
<td>14.50</td>
<td>1991</td>
<td>134.8</td>
<td>15.02</td>
<td>2025</td>
<td>-34</td>
<td>118</td>
<td>84/21</td>
</tr>
<tr>
<td>L</td>
<td>137.2</td>
<td>14.50</td>
<td>1989</td>
<td>132.8</td>
<td>14.95</td>
<td>1985</td>
<td>-4</td>
<td>7</td>
<td>76/19</td>
</tr>
<tr>
<td>W</td>
<td>137.5</td>
<td>14.50</td>
<td>1993</td>
<td>131.8</td>
<td>14.95</td>
<td>1970</td>
<td>-23</td>
<td>0</td>
<td>24/6</td>
</tr>
</tbody>
</table>

H, High sodium intake; L, low sodium intake; W, water during exercise.

*Based on 25% of mean body weight of 57.7 kg for the total group of runners. Weights for different groups were not reported.

†From table 2 of Twerenbold et al. to convert mg sodium (table 2) into mmol sodium, divide by the molecular weight of sodium (22.99).

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BOOK REVIEWS

Tennis

It is widely recognised that each sport has its own unique demands and injuries. Therefore the IOC, IFF, ATP, WTA, and Society for Tennis Medicine and Science should be congratulated on producing, in this publication, a comprehensive overview of tennis sports medicine. Together they have assembled an impressive array of experts in this field to write succinct and relevant chapters.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their view of recovery time from surgery—for example, three weeks for arthroscopic debri- dement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographs and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!

Dying to win

Dying to win gives an eye opening account of the extent to which drugs play a major role in sport. Doping is not new and has been used in sport since ancient Olympic times; it is just that drug use in modern times is at such a level of sophistication, it is now an industry in its own right. The book describes the privileged position sport holds in society, having appeal for both the participant and the spectator. This has led to the massive media interest, commercialism, professional- ism, and governmental regulation and manipulation. Economic pressure in the industrialised world and governmental propaganda in the former East Germany, and more recently China, pave the way for increasing pharmaceutical intervention in sport. With the fall of the GDR, the world saw for the first time what it had long suspected, the extent of systematic doping on a State run basis, and the most interesting fact is that the East German sports records? Further, the book takes a look at the next big issue surrounding drugs in sport—
genetic engineering.

Dying to win does not just describe the evolution of doping. It explains the complex relation between anti-doping policy, implementa- tion of those policies, and the role of governments, the IOC, and international and national sporting organisations. With the ever increasing involvement of the legal profession, a vicious circle occurs: it becomes too costly for sporting organisations to fight court battles, with their reliance on Government funding depending on results and sanctions set in accordance with what will stand up in courts. This all leads to the relative inertia of the governing bodies to be pro-active in the anti-drugs campaign.

The inception of the World Anti-Doping Agency (WADA) after the 1998 Winter Olympics in France has fiasco provided a way forward to standardise and implement anti-doping policy across the world by an independent body.

Problems and solutions to anti-doping policy are addressed. The major problem is inadequate definition of doping—to quote Arthur Gold “The definition lies not in words but in integrity of character.” It is interesting to note that those behind the athlete, namely coach, administrators, medical profession, and scientists, all seem to lose perspective along with their ethics and “integrity of character” when the race for “gold” is on. Dying to win suggests that these people should be held just as accountable to themselves. Another unfortunate aspect of anti-doping policy is the difficulty in detecting some abused drugs and the fact that these strategies often lie behind the ability of the pharmaceutical industry to develop new drugs, often for genuine medical reasons but with the unfor- tunate ability to enhance performance. Education is proposed as a key aspect to anti-doping policy, and parallels with its success in the use of recreational drugs are made. Governments also play a role in limiting supply, decreasing demand for drugs, and the implementation of independ- ent bodies to carry out testing. The success of anti-doping policy is also hard to measure. Fewer positive tests may simply reflect a move to less detectable methods rather than a decrease in use, and success may be better measured in terms of fewer world records.

Dying to win gives an accurate account of the problem of doping in sport and the difficulties and complexities in finding solutions to the problems. It makes interesting and provocative reading for anyone interested in sport, from the athlete and coach to the sport administrator, the medical profession, and governments.
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