

Leaders

Snowboarding injuries

Since the inception of the idea of riding a board on the snow in the 1970s, the popularity of the winter sport of snowboarding has burgeoned. Snowboarding is the only area of the winter sports market that has continued to grow. The 1994–1995 NSAA Kottke National Business Survey indicated that 14% of the 54 million area visits in the United States were generated by snowboarders.¹ It has been reported that 80% of children who participate in snow sports have ridden snowboards by their 12th birthday.² Industry analysts project that by the early 2000s more than 40% of those on the slopes will be snowboarders.

With the rise in popularity of snowboarding there has been a change in the injury pattern of these winter sports participants as compared with skiing. There has also been the recognition of an ankle injury that is specific to and only occurs in snowboarding. Along with a number of other medical facilities in Colorado, our clinic participated in a 10 year survey of snowboarding injuries (1988–1999). A total of 7430 snowboarding related injuries were seen in 7051 patients; 74.1% of those injured were male and 25.9% were female. Of the injured snowboarders, 45.2% were beginners, 31.4% intermediate, and 23.4% expert. There were significantly more upper extremity injuries than with skiing, which accounted for 49.1% of all injuries.

Ankle injuries accounted for 12% of all injuries, and fractures of the lateral process of the talus fractures accounted for 3%. Lateral process fractures, or snowboard-

er's talus fractures, are problematic and continue to be underdiagnosed and under-reported. Any acute and/or persistent anterolateral ankle pain in a snowboarder should be considered a talus fracture until proven otherwise. Most of these fractures are not able to be diagnosed by plain radiographs and require computed tomography imaging for definitive diagnosis. Most snowboarder's talus fractures need operative treatment with excision of fracture fragments or internal fixation of the fractures.

With the continued growth of snowboarding it will be increasingly more important for practitioners to be familiar with the diagnosis and treatment of snowboarding injuries. The studies have resulted in identifying and defining of a spectrum of injuries different from those of alpine skiing. Now that the spectrum of snowboarding injuries has been identified, the challenge will not only be the appropriate treatment of such injuries but also education about, research into, and prevention of such injuries. This will not only be the responsibility of the health care provider but also that of manufacturers, ski area owners and developers, snowboard shops, as well as snowboarders themselves.

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- 1 SnowSports Industries America National Snowboarder Survey. McLean, Virginia: SnowSports Industries America, 1995 and 1996.
- 2 Meyers C. On the edge: new riders on the Olympic stage. *Ski Magazine* 1996;25:25.

“You don't have to . . .”: walking to a healthier nation

One of the best pieces of public health news in recent years has been that you do not have to be a marathon runner, sports champion, or even regular jogger to derive substantial health benefits through exercise: regular moderate physical activity has cardioprotective and other health benefits.¹ From this and our low levels of exercise as a population, it can reasonably be concluded that promoting regular moderate physical activity—active living—is not only the most feasible route for exercise promotion but also the one that will yield the largest population health gain.²

HEBS (the Health Education Board for Scotland) has been something of a pioneer of the active living approach in the United Kingdom.^{2–4} We place a strong emphasis on walking because of its accessibility. Walking is easy for most people to contemplate and do, regardless of age or fitness level. It does not require special skills, expensive equipment, or facilities. It can be built into everyday life—for example, in commuting, shopping, and leisure. And the risk of injury is generally low.⁵ HEBS commissioned qualitative research conducted in 1995 supported this notion of accessibility, with preference being shown for walking over swimming or dancing. The same research, however, suggested that walking was not generally viewed

as “real” exercise, there being a belief that to be beneficial exercise has to make people sweaty and out of breath. Also, there were negative “non-aspirational” perceptions of walking, including (older) age profile, low status as a form of transport, and a boring image. Further developmental research pointed to the value of giving people “surprising” information about the value of walking.

A few years on, if you ask people what they think of when they hear of “HEBS”, their answer will probably include the name “Gavin”. They are referring to the TV advertising campaign that arose from the developmental research. Paradoxically using a sporting hero to promote regular moderate activity, the advertisement features Gavin Hastings comparing walking a mile with energy equivalent amounts of vigorous exercise. He points out that “you don't have to” take part in sweaty, frenetic, or very demanding forms of exercise to gain health and fitness benefits; in essence you can walk to good health. In the first phase of running the advert on TV, it was backed up by a special telephone helpline offering a pack containing the HEBS self help guide *Hassle free exercise* and information on local level physical activity facilities and contacts.

Formal evaluation of the first phase of the campaign is reported in detail elsewhere.⁶ Campaign awareness and walking related knowledge and beliefs were monitored through adult population surveys. Self reported changes in physical activity levels were assessed through a panel study involving a sample (initially 700) drawn from the 4036 people who had called the helpline during its first six weeks. A composite measure of “stage of change” (precontemplation, contemplation, preparation, action, maintenance) was derived from information provided by helpline callers at baseline and follow up.⁷ The rate of successful follow up at one year in the panel study was 58%. The sociodemographic profile of respondents at one year was similar to that at baseline, except for a slightly higher attrition rate for younger people.

Campaign awareness was highest in the primary target group (socioeconomic groups C2DE). There was before/after evidence of an impact on the general adult population’s knowledge and beliefs about walking as a form of exercise, the biggest increase being in knowledge of exercise equivalence information specific to the campaign. This is evidence of success of the major campaign objective of “repositioning” walking in the minds of the public. Also, in the panel study there was a discernible shift in stage of change (in the right direction) between baseline and follow up. Furthermore, 48% of the helpline callers successfully contacted at one year reported being more active.

The panel study of helpline callers was of course potentially open to initial self selection bias, and to subsequent drop out and “desire to please” bias. Suppose for the sake of argument that almost 2000 people (48% of 4036) were motivated and helped to become more active through the advertisement and helpline. Even in the absence of any such effect on people who viewed the advert but did not call the helpline—and disregarding the important informing and agenda setting roles of the campaign—this would be a worthwhile outcome and indeed would represent good value for money. However, this amount of behavioural change would not be detectable even in a fairly substantial survey of the general population.

In evaluation we therefore need to tap into “captive populations” (such as helpline callers) where they exist, and to manage potential bias through study design and analysis.

In any case, *Gavin*, with repeated showings, has undoubtedly caught the attention of the people of Scotland. Awareness of the advertisement in the adult general population runs consistently at around 90%, and I have referred to its centrality to people’s awareness of HEBS. In 1997 *Gavin* was voted favourite advertisement in a readers’ poll conducted by *The Scottish Sun* as part of the Scottish Advertising Awards. This is no mean feat, and its significance in evaluation terms should not be underestimated. It is evidence that health education advertising can have a wide appeal and become part of the fabric of the nation, more than holding its own with more expensive and less socially useful advertising.

The campaign and other health education efforts—in schools, through the workplace and health service, and in other settings—are of course but pieces in a jigsaw of factors affecting the nation’s levels of activity. Policies and action in areas such as community safety, transport, pollution control, urban and rural planning, and access to facilities are needed to make it more appealing and more feasible for people to build physical activity into their everyday lives at all stages and ages.

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- 1 Pate RR, Pratt M, Blair SN, *et al*. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402–7.
- 2 Wimbush E. A moderate approach to promoting physical activity: the evidence and implications. *Health Education Journal* 1994;53:322–36.
- 3 HEBS. *Promoting physical activity in Scotland: a policy statement*. Edinburgh: Health Education Board for Scotland, 1995.
- 4 HEBS. *The promotion of physical activity in Scotland: a strategic statement*. Edinburgh: Health Education Board for Scotland, 1997.
- 5 Davison RCR, Grant S. Is walking sufficient exercise for health? *Sports Med* 1993;16:369–73.
- 6 Wimbush E, MacGregor A, Fraser E. The impacts of a national mass media campaign on walking. *Health Promotion International* 1998;13:45–53.
- 7 Buxton K, Wyse J, Mercer T. How applicable is the stage of change model to exercise behaviour? A review. *Health Education Journal* 1996;55:239–57.

Role of exercise counselling in health promotion

Despite the clear health benefits that can be attained through adopting a more active lifestyle, most adults in the United Kingdom as well as other industrial nations remain underactive. Faced with this epidemic, there is a growing need for physical activity interventions that can be widely disseminated to all segments of the population across the lifespan.

One promising avenue for physical activity counselling and support lies with the primary care doctor and other health care professionals. The strengths of incorporating physical activity advice and support as part of routine health care include the ability to reach a substantial portion of the population repeatedly over time, the consistency and continuity of message content and delivery, and the willingness among patients to act on their doctor’s advice.^{1,2} Despite these strengths, however, a number of barriers to physical activity counselling in primary care have been documented, including lack of time, reimbursement, and training in physical activity or behaviour change counselling.³ Although such barriers present continuing challenges to the health promotion and

health care fields, the potential public health impact that primary care settings can have on health behaviour change, including physical activity, merits continued investigation.

Although a relatively large body of research exists on advice and counselling by doctors for other health behaviours, such as smoking, relatively little systematic research has been conducted to date on physical activity promotion in primary care. The studies that have been undertaken have taken advantage of a growing body of knowledge, underscoring the utility of applying empirically supported behavioural strategies in facilitating physical activity change. Such behavioural strategies, derived primarily from social cognitive theory and its derivatives, include: identifying specific practical physical activity goals tailored to the patient’s needs and circumstances; structuring initial patient expectations so that they are realistic; identifying those benefits related to becoming more physically active that are most germane to the patient’s own health status; encouraging the patient to keep track of his or her own physical activity patterns through simple self

monitoring tools; and providing continual interest, encouragement, and support for physical activity. Some of these behavioural strategies have been used in studies in which primary care doctors have been trained to deliver brief advice and counselling on physical activity, with encouraging results in the short term.⁴⁻⁵ In one study, for example, a written goal oriented exercise prescription from general practitioners, in addition to verbal advice, was particularly effective in promoting increased physical activity over a six week period.⁶ More discrepant results obtained from longer term multiple risk factor programmes, however, suggest that more intensive interventions may be needed to obtain longer term effects in at least some segments of the population. Such interventions could include the use of health educators and professionals in addition to the doctor. Health educators and other allied health professionals can provide a level of advice and counselling beyond that which doctors, constrained by time and similar barriers, are typically able to deliver.⁷ One promising approach awaiting more extensive investigation involves using brief advice from the doctor as a means of setting the stage for physical activity change in conjunction with specific referral to other health care based or community based health educators or providers. In this way, the perceived credibility and authority of the doctor can be harnessed as a catalyst for change, while the very real time constraints facing many doctors are recognised.⁸ The challenge remains to structure the referral network effectively such that patients will successfully follow through with the referral. To maximise the potential benefits of this type of referral network, continuing communication between the doctor and referral source is essential.

In addition, the studies targeting primary care providers have focused almost exclusively on doctors involved in family practice and internal medicine. Yet, other primary care specialties, such as paediatrics and obstetrics-gynaecology, reach important segments of the population for whom physical activity information and messages are particularly relevant. Future research should target the full range of primary care practice.

While face to face instruction and counselling for physical activity have traditionally been the norm in most countries, a growing scientific literature has underscored the utility of mediated channels for delivering physical activity advice and information in an efficient, effective, and potentially less costly fashion. For instance, in the United States, at least 13 randomised controlled investigations have systematically evaluated the use of telephone based physical activity advice and support, either in conjunction with or independent of advice from the doctor.⁷⁻⁹⁻¹⁰ The telephone supervised physical activity approach has been

shown to be effective in both older and younger adult populations, women as well as men, cardiac patients, older family carers of relatives with dementia, and overweight patients. It has been found to be effective in promoting physical activity of various types—for example, endurance, strength, flexibility, general conditioning—intensities—for example, moderate intensity exercise, more vigorous exercise—and formats—for example, home based, group based, combinations of home based and group based exercise. Telephone and similar mediated approaches allow both the health professional and the patient a level of convenience and flexibility that is often diminished or lacking in group based physical activity regimens.

In summary, to reach the public health goals on physical activity in the United Kingdom, United States, Australia, and other countries continued efforts to involve primary care providers and other health professionals as active facilitators of the physical activity message are strongly indicated. Primary care advice in conjunction with referral to appropriate community organisations may help to facilitate the long term increases in physical activity participation that are critical for health promotion and disease prevention. Telephone and other mediated approaches to physical activity promotion provide a promising avenue for programme delivery, in primary care as well as other community settings.

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- 1 Williford HN, Barfield BR, Lazenby RB, *et al.* A survey of physicians' attitudes and practices related to exercise promotion. *Prev Med* 1992;21:630-6.
- 2 Goldstein MG, DePue J, Kazura A, *et al.* Models for provider-patient interaction: applications to health behavior. In: Shumaker SA, Schron SB, Ockene JK, *et al.*, eds. *The handbook of health behavior change*, 2nd ed. New York: Springer, 1998:85-113.
- 3 Pender NH, Sallis JF, Long BJ, *et al.* Health care provider counseling to promote physical activity. In: Dishman RK, ed. *Exercise adherence*, 2nd ed. Champaign, IL: Human Kinetics, 1994:213-35.
- 4 Calfas KJ, Long BJ, Sallis JF, *et al.* A controlled trial of physician counseling to promote the adoption of physical activity. *Prev Med* 1996;25:225-33.
- 5 Marcus BH, Goldstein MG, Jette A, *et al.* Training physicians to conduct physical activity counseling. *Prev Med* 1997;26:382-8.
- 6 Swinburn BA, Walter LG, Arroll B, *et al.* The green prescription study: a randomized controlled trial of written exercise advice provided by general practitioners. *Am J Public Health* 1998;88:288-91.
- 7 King AC, Sallis JF, Dunn AL, *et al.* Overview of the activity counseling trial (ACT) intervention for promoting physical activity in primary care settings. *Med Sci Sports Exerc* 1998;30:1086-96.
- 8 Stevens W, Hillsdon M, Thorogood M, *et al.* The cost effectiveness of a primary care-based physical activity intervention in 45-74 year old men and women: a randomised controlled trial. *Br J Sports Med* 1998;32:236-41.
- 9 DeBusk RF, Haskell WL, Miller NH, *et al.* Medically directed at-home rehabilitation soon after clinically uncomplicated acute myocardial infarction: a new model for patient care. *Am J Cardiol* 1985;55:251-7.
- 10 King AC, Rejeski WJ, Buchner DM. Physical activity interventions targeting older adults: a critical review and recommendations. *Am J Prev Med* 1998; 15:316-33.

Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin

Traditional dogma would have it that pain in tendinopathy arises through one of two mechanisms. Firstly, it may result from inflammation in "tendinitis". Secondly, it may be due to separation of collagen fibres in more severe forms of tendinopathy. The latter situation parallels the mechanism of pain with collagen separation after an acute grade I or II ligament injury (fig 1).

Despite the wide acceptance of these two classical models of pain production, a number of studies provide data

inconsistent with either theory. Consider first the inflammation mechanism. Histopathological examination of surgical specimens from patients with chronic tendon pain are devoid of inflammatory cells.¹ This applies to tissue from the Achilles, patellar, lateral elbow, medial elbow, and rotator cuff tendons. Furthermore, prostaglandin E2 (a marker of the inflammatory process) is no more abundant in patients with Achilles tendon pain than in normal controls.²

Unfortunately, the collagen separation theory does not hold up under scrutiny either. The following five observations about pain and collagen in the patellar tendon are inexplicable. (a) Patients who have patellar tendon allograft anterior cruciate ligament reconstruction have minimal donor site knee pain, yet collagen has been excised. (b) Such patients are generally pain-free (and back at sport) despite the persistence of abnormal collagen for two or more years.^{3,4} (c) Similarly, after open surgery for jumper's knee, the imaging appearance of the tendon—that is, collagen status—does not correlate consistently with knee pain.⁵ (d) Patients with jumper's knee can also be treated by an arthroscopic debridement of the infrapatellar fat pad and the posterior border of the patellar tendon without operation on the collagen defect in the tendon itself.⁶ (e) Large asymptomatic ultrasonographic hypoechoic regions (abnormal collagen) can be found in patellar tendons of some athletes who have never had a history of jumper's knee.^{7,8}

Such discrepancy between collagen structure and pain is not confined to the patellar tendon. Patients with partial (non-perforated) rotator cuff tears were found to have more pain than those with complete perforations⁹ despite the former having less collagen damage. Clearly there is more to tendon pain than discontinuity of collagen per se.

Nociceptors provide significant afferent pain pathways. In the knee, they are located in the retinaculum, fat pad, synovium, and periosteum,¹⁰ and all these structures may play a role in the tendon pain pathway. Biochemical irritants may include extravasation of glycosamines, especially chondroitin sulphate,^{11,12} from damaged tendon.

The five observations listed above can be explained with what we term a “biochemical” hypothesis (fig 2). We speculate that the pain of patellar tendinopathy is largely due to biochemical agents irritating nociceptors located in the fat pad immediately posterior to the patellar tendon. In 39 cadaver dissections of the proximal patellar tendon,¹³ we consistently identified a thin layer of fat adherent to the posterior portion of the patellar tendon. In the corresponding tissue specimens from patients operated on for chronic

jumper's knee, this fat tissue contained increased Alcian blue stain (and thus glycosaminoglycans), presumably leaked from the adjacent region of tendinosis.

To our knowledge, the key irritant biochemical agent has not yet been identified, and this presents a challenge for tendon biochemists. Using microdialysis, Alfredson recently identified an abnormal amount of the excitatory neurotransmitter, glutamate, in subjects with painful Achilles tendinopathy.² Until these histopathological and biochemical findings are correlated with some measure of pain, we can only speculate as to whether they are causative, or merely byproducts of nearby tendinosis.

Of interest, in the rotator cuff pain and pathology study quoted above,⁹ collagen damage was inversely related to pain, but the presence of substance P (a nociceptive neurotransmitter) was significantly associated with pain. Nerve fibres immunoreactive to substance P were localised around vessels in the subacromial bursa and in the non-perforated rotator cuff.⁹

Although the data presented may suggest a biochemical cause of pain, other workers consider mechanical impingement of the fat pad as a cause of anterior knee pain. The Australian physiotherapist, Jenny McConnell, recognised fat pad impingement as a cause of anterior knee pain (not necessarily tendon pain) over 10 years ago. Johnson proposed that impingement caused the pain of patellar tendinopathy.¹⁴ The infrapatellar fat pad is an extremely sensitive region¹⁵ and contains a large number of nociceptors, but as tendon pain occurs at many anatomical sites, it does not appear logical that a structure related to only one tendon—that is, the patellar fat pad—would necessarily play a unique role in a problem as widespread as tendinopathy. Further, the clinical observation that the pain of jumper's knee does not disappear and may actually increase when palpation is performed with the knee in full extension would appear to argue more for a biochemical than a mechanical cause of pain in tendinopathy. Nevertheless, the jury requires more evidence.

If our biochemical hypothesis proves to have some validity, it would have significant clinical and research implications. In clinical management, the aim of treatment would be to modify the biochemical milieu, rather than to focus on reducing inflammation or necessarily augmenting collagen repair. Collagen repair may, of course, improve the biochemical milieu and thus explain why eccentric strengthening programmes can help.¹⁶ Researchers would be encouraged to pursue a pharmaceutical approach focused on reducing the irritant (but not necessarily inflammatory) biochemical compounds around the tendon. Surgery may play a role through denervation. Thus, if sports medicine researchers collaborate with basic scientists who understand pain physiology, knowledge will be

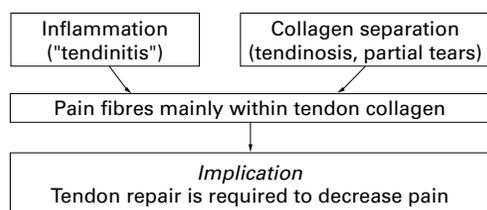


Figure 1 The classical “inflammatory” and “structural” tendon pain models.

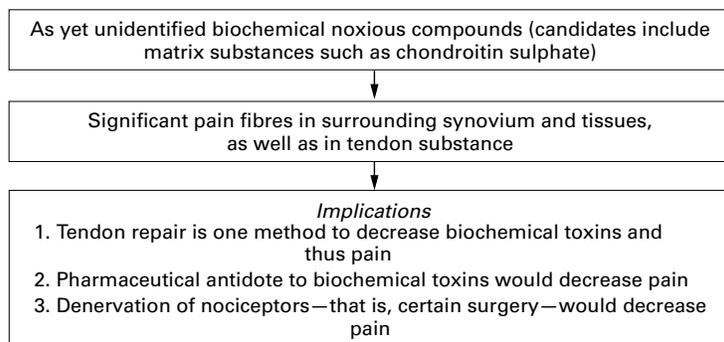


Figure 2 Contemporary “biochemical” tendon pain model.

advanced in both fields, and we will progress toward the goal of alleviating the pain of what is often structurally rather a trivial problem.

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- 1 Khan KM, Cook JL, Bonar F, *et al.* Histopathology of common overuse tendon conditions: update and implications for clinical management. *Sports Med* 1999;27:393–408.
- 2 Alfredson H. In situ microdialysis in tendon tissue: high levels of glutamate, but not prostaglandin E₂ in chronic achilles tendon pain. *Knee Surg Sports Traumatol Arthrosc* 1999;7:378–81.
- 3 Adriani E, Mariani PP, Maresca G, *et al.* Healing of the patellar tendon after harvesting of its mid-third for anterior cruciate ligament reconstruction and evolution of the unclosed donor site defect. *Knee Surg Sports Traumatol Arthrosc* 1995;3:138–43.
- 4 Kiss ZS, Kellaway D, Cook J, *et al.* Postoperative patellar tendon healing: an ultrasound study. *Australas Radiol* 1998;42:28–32.

- 5 Khan KM, Visentini PJ, Kiss ZS, *et al.* Correlation of US and MR imaging with clinical outcome after open patellar tenotomy: prospective and retrospective studies. *Clin J Sport Med* 1999;9:129–37.
- 6 Coleman BD, Khan KM, Kiss ZS, *et al.* Outcomes of open and arthroscopic patellar tenotomy for chronic patellar tendinopathy: a retrospective study. *Am J Sports Med* 2000;28:183–90.
- 7 Cook JL, Khan KM, Harcourt PR, *et al.* Patellar tendon ultrasonography in asymptomatic active athletes reveals hypochoic regions: a study of 320 tendons. *Clin J Sport Med* 1998;8:73–7.
- 8 Lian O, Holen KJ, Engebretson L, *et al.* Relationship between symptoms of jumper's knee and the ultrasound characteristics of the patellar tendon among high level male volleyball players. *Scand J Med Sci Sports* 1996;6:291–6.
- 9 Gotoh M, Hamada K, Yamakawa H, *et al.* Increased substance P in subacromial bursa and shoulder pain in rotator cuff diseases. *J Orthop Res* 1998;16:618–21.
- 10 Witonski D, Wagrowska-Danielewicz M. Distribution of substance-P nerve fibres in the knee joint in patients with anterior knee pain syndrome. A preliminary report. *Knee Surg Sports Traumatol Arthrosc* 1999;7:177–83.
- 11 Benazzo F, Stenardo G, Valli M. Achilles and patellar tendinopathies in athletes: pathogenesis and surgical treatment. *Bull Hosp Jt Dis* 1996;54:236–40.
- 12 Józsa L, Kannus P. *Human tendons*. Champaign, IL: Human Kinetics, 1997: 576.
- 13 Khan KM, Bonar F, Desmond PM, *et al.* Patellar tendinosis (jumper's knee): findings at histopathologic examination, US and MR imaging. *Radiology* 1996;200:821–7.
- 14 Johnson DP. Magnetic resonance imaging of patellar tendonitis. *J Bone Joint Surg [Br]* 1996;78:452–7.
- 15 Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intra-articular anesthesia. *Am J Sports Med* 1998;26:773–7.
- 16 Alfredson H, Pietila T, Jonsson P, *et al.* Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med* 1998;26:360–6.

$\dot{V}O_2$ slow component and performance in endurance sports

For almost 80 years, physiological studies have attempted to explain endurance performance and to develop ways of improving it by training. Performance for a runner can be represented by the relation of his/her personal power (velocity) to time to exhaustion (time limit).¹

There are particular velocities that delineate intensity domains which are determined by oxygen uptake ($\dot{V}O_2$) and blood lactate response versus time.^{2,3} We are going to use them to define the slow phase of $\dot{V}O_2$ kinetics $\dot{V}O_2$ slow component) which only appears during intense exercise.

A high range of work can be identified at which there is a sustained increase in blood lactate and a decrease in arterial pH with time. These responses decline back towards a baseline value. Oxygen uptake increases in a monoexponential way and stabilises at about 80% in high level marathon runners for at least an hour and a half of continuous exercise. After that time, it is possible for oxygen consumption to increase because of thermoregulatory constraints, and this increase is called the “ $\dot{V}O_2$ drift”. This intensity of exercise corresponds to the velocity that can be sustained during a marathon and is equal to about 80% of the velocity associated with $\dot{V}O_{2MAX}$ determined in an incremental test—that is, $v\dot{V}O_{2MAX}$.⁴ During this type of exercise both lipids and carbohydrate are used as fuel.

At a higher intensity, the maximal lactate steady state occurs⁵ when the rate of appearance of blood lactate equals the rate of its disappearance. $\dot{V}O_2$ stabilises after three minutes at about 85% $\dot{V}O_{2MAX}$. This corresponds to the highest velocity that an athlete can sustain for an hour (85% $v\dot{V}O_{2MAX}$ for a well trained endurance athlete); carbohydrate (and lactate even) is the main substrate for this exercise.

At a higher intensity, at about 90% $v\dot{V}O_{2MAX}$, the rate of appearance of blood lactate exceeds the rate of disappearance and therefore blood lactate increases. After the first

monoexponential increase in $\dot{V}O_2$, there is a second increase after about three minutes which is defined as the $\dot{V}O_2$ slow component. $\dot{V}O_2$ reaches a delayed steady state which is higher than the $\dot{V}O_2$ requirement estimated from the relation between $\dot{V}O_2$ and moderate work rate. For instance, in this case the athlete can run at 90% $v\dot{V}O_{2MAX}$ and reaches and stabilises at 95% $\dot{V}O_{2MAX}$ at the sixth minute of exercise (time to exhaustion at this velocity being about 10–15 minutes). This corresponds to the so called “critical power” which is the vertical asymptote of the hyperbolic relation between power (velocity) and time.⁶ Time limit at the critical velocity is reduced to less than 30 minutes because of rapid glycogen depletion.^{7,8} The critical velocity is the highest velocity below its maximal level ($\dot{V}O_{2MAX}$) at which oxygen consumption can reach a steady state.

Above this critical velocity, during high intensity exercise, neither $\dot{V}O_2$ nor blood lactate can be stabilised, and both rise inexorably until fatigue ensues, at which point $\dot{V}O_2$ reaches its maximum value.⁹

The initial very small component (phase 1), resulting from a sudden change in the venous return in combination with a small change in the mixed venous gas tension, is not fitted into the following equation. In fact, the parameters for the oxygen uptake kinetics were obtained from a two component exponential model in which the first component accounted for the fast component (phase 2) and the second component accounted for the slow component (phase 3). The oxygen uptake kinetics are described as a function of time by the following equation¹⁰: $\dot{V}O_2(t) = A_0$ (baseline) + $A_1(1 - e^{-(t-TD_1)/\tau_1})$ (fast component) + $A_2(1 - e^{-(t-TD_2)/\tau_2})$ (slow component) where A_0 is the resting baseline value, A_1 and A_2 are the amplitudes for the two components, τ_1 and τ_2 are the time constants for the two components, and TD_1 and TD_2 are the time delays from the onset of exercise for the two components.

Hence, the so called $\dot{V}O_2$ slow component is the second amplitude (A_2) of the increase in $\dot{V}O_2$ that appears at TD_2 . This second amplitude represents about 10% of the first (A_1) and depends on the absolute intensity of exercise because $\dot{V}O_2$ is regulated by the split of ATP and phosphocreatine.¹¹ The value of the $\dot{V}O_2$ slow component can reach 500 ml/min and is generally considered to be significant when the value is above 200 ml/min. To avoid the use of this complicated equation which necessitates the use of software such as Sigma plot (SPSS), the $\dot{V}O_2$ slow component can be identified as described initially by Whipp and Wasserman¹² by calculating the difference in $\dot{V}O_2$ measurement between the 6th and 3rd minute or, if the exercise is performed until exhaustion, between the third and last minute.¹³

The appearance of this slow $\dot{V}O_2$ component is mainly due to the recruitment of fast fibre type II fibres with fatigue.¹⁴ It has been shown that type II fibres have a phosphate to oxygen ratio that is 18% lower than in type I fibres, probably because of a greater reliance on the α -glycerophosphate shuttle than the malate-aspartate shuttle.¹⁵ Therefore more oxygen is required to produce the same level of ATP turnover and sustain a given power output. The other 15% is due to an increase in cardiac and ventilation work. Training decreases the $\dot{V}O_2$ slow component at the same absolute velocity, mainly because of an increase in the distribution of type I fibres and an increase in mitochondrial and capillary density.^{16, 17} A decrease in the $\dot{V}O_2$ slow component can also appear for the same relative velocity (in % $v\dot{V}O_{2MAX}$) because of an increase in the maximal lactate steady state.¹⁸ However, during intense exercise, the amplitude of the $\dot{V}O_2$ slow component is not linked to endurance at all. Moreover, it has been reported that triathletes that had no $\dot{V}O_2$ slow component in running compared with cycling had the same endurance time in these two types of exercise (at 90% of the power or velocity associated with $\dot{V}O_{2MAX}$). These triathletes also had the same maximal lactate steady state at 82% of velocity or power associated with $\dot{V}O_{2MAX}$ in running and cycling.

Endurance training decreases the $\dot{V}O_2$ slow component at the same velocity.¹⁹⁻²² Personal data on high intensity training have shown that the decrease in the $\dot{V}O_2$ slow component at the same absolute intensity (90% $v\dot{V}O_{2MAX}$) is not correlated with an improvement in performance (endurance time) at this velocity (+ 40% of time limit).

A more interesting fact about this $\dot{V}O_2$ slow component phenomenon is for training at $\dot{V}O_{2MAX}$ as it creates a broad range of exercise intensities for which $\dot{V}O_{2MAX}$ will occur, provided that the exercise is continued to the point of exhaustion.⁹

Hence, it may be possible to describe a new relation between time spent at $\dot{V}O_{2MAX}$ ($t_{lim}\dot{V}O_{2MAX}$) and velocity as a percentage of the velocity associated with $\dot{V}O_{2MAX}$ determined in an incremental test ($v\dot{V}O_{2MAX}$). The relation between time to exhaustion at $\dot{V}O_{2MAX}$ and velocity follows a function that has a peak around 100% $v\dot{V}O_{2MAX}$ in well trained runners who have no, or only a low value for, the $\dot{V}O_2$ slow component (<200 ml/min). In less well trained subjects, the $\dot{V}O_2$ slow component means that they spend longer sustaining $\dot{V}O_{2MAX}$ at 90% $v\dot{V}O_{2MAX}$ than at 100% $v\dot{V}O_{2MAX}$.^{23 25 26} However, fit endurance athletes have to run at close to 100% of $v\dot{V}O_{2MAX}$ to elicit $\dot{V}O_{2MAX}$ because they have no $\dot{V}O_2$ slow component.^{23 24}

Therefore, in training, if the aim is to elicit $\dot{V}O_{2MAX}$, it may be useful to determine the velocity for which time spent at $\dot{V}O_{2MAX}$ is maximal.²⁵ To determine at which velocity the longest time at $\dot{V}O_{2MAX}$ is obtained during con-

tinuous exercise, the critical velocity at $\dot{V}O_{2MAX}$ can be determined using the critical power model. Instead of total time limit run, only the time run at $\dot{V}O_{2MAX}$ is plotted against the distance run at $\dot{V}O_{2MAX}$. The slope of this plot is the critical velocity at $\dot{V}O_{2MAX}$. This relation between $t_{lim}\dot{V}O_{2MAX}$ and velocity can be used to determine the velocity that elicits the longest time to exhaustion at $\dot{V}O_{2MAX}$.^{26 27} This velocity is not significantly different from $v\dot{V}O_{2MAX}$ determined from an incremental protocol, but is significantly higher than the critical velocity classically determined using a two parameter critical power model and the total distance-time.²⁶

The existence of this $\dot{V}O_2$ slow component phenomenon raises the question of how athletes can adapt their training to improve performance. In fit runners, who are not at a high level ($v\dot{V}O_{2MAX} = 19$ km/h), eight weeks of training at high intensity was shown to remove the $\dot{V}O_2$ slow component at the same absolute velocity (V Billat, A Demarle, J Slawinski and JP Koralsztejn, unpublished work). This was because $v\dot{V}O_{2MAX}$ increased, and at the same velocity was at a lower percentage of $v\dot{V}O_{2MAX}$ than before training. The time limit at this previously high intensity training was doubled (20 v 10 minutes). At the same relative velocity to $v\dot{V}O_{2MAX}$, the $\dot{V}O_2$ slow component was comparable with that before training, which means that this high intensity training (twice a week) has to be calibrated at least every two months in this case.

In conclusion, the $\dot{V}O_2$ slow component phenomenon, which was first described by Margaria *et al* in the sixties²⁸ and then by Whipp and Wasserman in the seventies,¹² has been widely focused on in the nineties. In the light of this, it should be possible in the next five years to use the knowledge to diversify training and to explore endurance training effects and fitness.

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- 1 Billat V, Koralsztejn JP, Morton H. Time in human endurance models. From empirical to physiological models. *Sports Med* 1999;27:359-79.
- 2 Whipp, BJ. The slow component of O_2 uptake kinetics during heavy exercise. *Med Sci Sports Exerc* 1994;26:1319-26.
- 3 Whipp BJ, Ozyener F. The kinetics of exertional oxygen uptake: assumptions and inferences. *Med Sport* 1998;51:139-49.
- 4 Billat V, Koralsztejn JP. Significance of the velocity at $\dot{V}O_{2MAX}$ and its time to exhaustion at this velocity. *Sports Med* 1996;22:90-108.
- 5 Brooks GA. Anaerobic threshold: review of the concept and direction for future research. *Med Sci Sports Exerc* 1985;17:31-5.
- 6 Monod H, Scherrer J. The work capacity of synergy muscular groups. *Ergonomics* 1965;8:329-38.
- 7 Housh DJ, Housh TJ, Bauge SM. The accuracy of critical power test for predicting time to exhaustion during cycle ergometry. *Ergonomics* 1989;32:997-1004.
- 8 Jenkins DG, Quigley BM. The Y-intercept of the critical power function as a measure of anaerobic work capacity. *Ergonomics* 1991;34:13-22.
- 9 Gaesser GA, Poole DC. The slow component of oxygen uptake kinetics in humans. In: Holloszy JO, ed. *Exercise sport science review*. Baltimore: Williams & Wilkins, 1996:35-70.
- 10 Barstow TJ, Molé PA. Linear and nonlinear characteristics of oxygen uptake kinetics during heavy exercise. *J Appl Physiol* 1991;71:2099-106.
- 11 Walsh ML. Possible mechanisms of oxygen uptake kinetics. *Annals of Physiological Anthropology* 1992;11:215-23.
- 12 Whipp BJ, Wasserman K. Oxygen uptake kinetics for various intensities of constant-load work. *J Appl Physiol* 1972;33:351-6.
- 13 Billat V, Richard R, Binsse VM, *et al*. $\dot{V}O_2$ slow component for a severe exercise depends on type of exercise and is not correlated with time to fatigue. *J Appl Physiol* 1998;85:2118-24.
- 14 Poole DC, Schaffartzik W, Knight DR, *et al*. Contribution of exercising legs to the slow component of oxygen uptake kinetics in humans. *J Appl Physiol* 1991;71:1245-53.
- 15 Willis WT, Jackman MR. Mitochondrial function during heavy exercise. *Med Sci Sports Exerc* 1994;26:1347-54.
- 16 Holloszy JO, Coyle EF. Adaptation of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol* 1984;56:831-8.
- 17 Hoppeler H. Exercise-induced ultrastructural changes in skeletal muscle. *Int J Sports Med* 1986;7:187-204.
- 18 Yoshida T, Udo M, Ohmori T, *et al*. Day-to-day changes in oxygen uptake kinetics at the onset of exercise during strenuous endurance training. *Eur J Appl Physiol* 1992;64:78-83.
- 19 Berry M, Moritani T. The effects of various training intensities on the kinetics of oxygen consumption. *J Sports Med Phys Fitness* 1996;22:90-108.
- 20 Casaburi R, Storer TW, Ben-Dov I, *et al*. Effect of endurance training on possible determinants of $\dot{V}O_2$ during heavy exercise. *J Appl Physiol* 1987;62:1533-8.

- 21 Gaesser GA. Influence of endurance training and catecholamines on exercise $\dot{V}O_2$ response. *Med Sci Sports Exerc* 1994;26:1341–6.
- 22 Womack CJ, Davis SE, Blumer JL, et al. Slow component of O_2 uptake during heavy exercise: adaptation to endurance training. *J Appl Physiol* 1995;79:838–45.
- 23 Billat V, Binsse V, Petit B, et al. High level runners are able to maintain a $\dot{V}O_2$ steady state below $\dot{V}O_{2max}$ in an all-out run over their critical velocity. *Arch Physiol Biochem* 1998;106:38–45.
- 24 Jacobsen DJ, Coast R, Donnelly JE. The effect of exercise intensity on the slow component of $\dot{V}O_2$ in persons of different fitness levels. *J Sports Med Phys Fitness* 1998;38:124–31.
- 25 Billat V, Slawinski J, Bocquet V, et al. Intermittent runs at $\dot{V}O_{2max}$ enables subjects to remain at $\dot{V}O_{2max}$ for a longer time than severe submaximal run. *Eur J Appl Physiol* 2000;81:188–96.
- 26 Billat V, Blondel N, Berthoin S. Determination of the velocity associated with the longest time to exhaustion at maximal oxygen uptake. *Eur J Appl Physiol* 1999;80:159–61.
- 27 di Prampero PE. The concept of critical velocity: a brief analysis. *Eur J Appl Physiol* 1999;80:162–4.
- 28 Margaria R, Mangili F, Cuttica F, et al. The kinetics of the oxygen consumption at the onset of muscular exercise in man. *Ergonomics* 1965;8:49–54.

Should nasal fractures be treated on the spot?

We all know the scenario at the side of the rugby pitch, as the team attendant or doctor yanks a nose straight before any pain is perceived by a front row “beauty” who then returns to play. But should nasal fractures be treated in this way, on the spot?

Would sports medicine doctors let their own broken noses be treated on the spot?

“Yes, I’d have it put back pitch side—once I’ve retaliated and before the pain starts.”

“Depends who by.”

“Yes I’d like it put back immediately . . . but I’ve had my nose put back on the field several times and then had to have a surgeon sort it all out later, but that’s OK.” (Personal discussions at the BASM 1999 conference)

What are the dangers of this procedure?

If looks are spoilt, an athlete may be persuaded to sue who ever tampered with their nose and make the culprit pay for their private plastic surgery and loss of modelling career. Worse still would be the scenario of an underlying cribriform plate fracture (and possible other complications) being further disrupted by incompetent attempts at nasal fracture reduction or uncontrollable bleeding of the nose pitch side and miles from help.

Some questions before letting the doc sort the nose

Is the heat and pressure of the sports field the place to be deciding the severity of the fracture even if you are a doctor?

How much ENT training has the average sports doctor had?

Would an ENT surgeon reduce a fracture pitch side?

Would an ENT surgeon be happy for doctors, physiotherapists, or first aiders to reduce a nasal fracture “on the spot”?

ENT surgeons’ opinions varied among those I contacted.

“Allowing a GP colleague, if experienced, or ENT surgeon to put their nose back pitch

side. But not a ‘bag man’ pitch side or a casualty SHO (even after an *x* ray) to do the same procedure.”

“Bleeding following repositioning is unpredictable and it should be done in a hospital environment by an ENT colleague.”

“Yes I would let an ENT colleague, GP or first aid/bag man put my nose back pitch side as I don’t think *x* rays are necessary for nasal fractures.” (Personal correspondence with ENT surgeons in Scotland)

The Defence Unions referred to the Bolam Defence of “accepted practice” for a sports doctor. “If a member were to treat a displaced nasal fracture on the spot, and there was an unsatisfactory outcome, it may well be alleged that it was negligent to undertake such a procedure. In defending a member we would need to take into account the training and experience of the member and an independent expert opinion from a practitioner in the same speciality.” (Personal correspondence with The St Paul International Insurance Agency).

“Advocates of an on the spot treatment of nasal fractures would have to show good supporting evidence that the outcome is at least as good, if not improved, by undertaking urgent reduction, rather than waiting for ENT specialist care a few days after the injury.” (Personal correspondence with the Medical Defence Union).

My search for what is accepted practice for “on the spot” treatment of nasal fractures proved fruitless!

In conclusion, I think that until “What is accepted practice for sports medicine doctors” is tested in a court of law, we are still left with no straight answer to the question. In simple terms, individual doctors must ask themselves whether they are competent to undertake the procedure.

First aiders are covered by the Good Samaritan Act of 1983¹ to administer first aid according to the accepted practices and manuals of the voluntary first aid societies; this does not include reducing nasal fractures.

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1 *Mod Law Review* 1990:53.

LETTERS TO THE EDITOR

Survey of sports injury prevention programmes in the European Community

EDITOR,—I would like to provide a brief report of a survey of sports injury prevention programmes and related research projects in the European Community between September and December 1998.

With the cooperation of partners in the Netherlands (the Netherlands Olympic Committee and the Netherlands Sports Federation), Austria (the Austrian Institute for Home and Leisure Safety), and Belgium (Flanders Red Cross) a questionnaire on sports injury prevention and related research was devised, piloted, and distributed to a sample of 368 sports, health, or safety organisations in Europe. The objective of the study was to determine the number of institutions involved in injury prevention work, and also to seek information on the nature and quality of the work being undertaken.

A total of 86 questionnaires were returned from 77 different organisations in 28 European countries. The largest number of returns was received from Austria (13 returns), Belgium (9 returns), Ireland and the Netherlands (7 returns), and Finland, Germany, and Norway (6 returns each). 87% of the organisations responding to the survey were involved primarily in either sport, safety, or education and research. The other 13% provided health care services.

Forty two out of 86 respondents (58.8% of the returns and 11.2% of the total questionnaires distributed) reported that they were currently running a programme on sports injury prevention or related research. Less satisfactorily, only 14 of the injury prevention projects (16.3% of those responding and 3.8% of the original sample) were based upon research data or had any kind of inbuilt quality control mechanism (such as an assessment of the effectiveness of the programme). Respondents were also requested to send in examples of their injury prevention materials and to provide comments on the provision of injury prevention programmes in their sport. Many of the programmes were found to consist only of warm up and stretching exercises; these were often poorly described and of doubtful value. Few of the programmes were supported by empirical evidence or addressed risk factors specific to individual sports.

Some of the comments returned with the questionnaires included the following:

- "This questionnaire is not relevant to us. Our members look after their own injuries."
- "There are hardly any injuries in our sport." (A sport known to produce a moderately high incidence of overuse injuries.)
- "Injury prevention measures don't work."
- "Stretching and warm up are a waste of time."

Reviews of the literature on sports injuries^{1,2} show that injury prevention measures are most effective when directed at particular sports and population groups, and

that measures directed towards the extrinsic causes of sports injuries (for example, problems with the rules or with personal and playing area equipment) are often the most effective ways of reducing the incidence of sports injuries. Furthermore, a number of studies have failed to show that warm up and high levels of flexibility are effective in reducing the incidence of sports injuries.

Because of the low response rate the findings of our survey must be regarded as preliminary. However, they do suggest the following:

1. A significant number of sporting organisations are ignoring the problem of sports injuries.
2. Injury prevention programmes are often not based upon good empirical evidence and frequently do not address the risk factors specific to particular sports.
3. Quality control and follow up measures are rare in the context of injury prevention programmes.
4. The lack of appropriateness and effectiveness of some injury prevention programmes is being noticed by athletes and sports administrators.

Notwithstanding the limitations of our study, I find the results a source of concern. I would urge your readers to impress upon sporting organisations the need to take injury prevention seriously, and to do all they can to ensure that the measures adopted are based upon the results of empirical research, and that quality control measures are put in place.

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- 1 Renstrom PAFH, editor. Sports Injuries: basic principles of care and prevention. Oxford: Blackwell, 1993.
- 2 Watson AWS. Sports injuries: incidence, causes, prevention. *Physical Therapy Reviews* 1997;2: 135-51.

Use of imaging data for predicting clinical outcome

EDITOR,—I write with reference to the letter of Khan and Kannus.¹ I concur that Gibbon and colleagues are not in a position to draw a conclusion on the diagnostic ultrasound screening of athletes suggesting that sonographic abnormality will lead to a complete rupture. However, I also do not fully agree with the authors of the letter that tissue based pathologies found by Kannus and Jozsa² may be more subtle than can be detected by sonography.

To explain, I feel that part of the problem, resulting in this divergence of opinion, lies both with the diversity of the diagnostic ultrasound equipment used and the skill of the operator. There is little standardisation of either of the techniques used by operators or, in particular, equipment specification. To this end, articles that report studies correlating diagnostic ultrasound findings with other clinical markers have to be carefully interpreted.

Colleagues and I have been regularly performing musculoskeletal ultrasound examinations, particularly on Achilles and supraspinatus tendons. We regularly visualise degenerative changes in asymptomatic tendons that do not go on to rupture or produce significant problems.

We have conducted a three year prospective controlled study that linked diagnostic ultrasonography data to clinical presentation/symptoms. The results showed a strong correlation between the ultrasound findings and the clinical markers used, such as pain, stiffness, and functional ability.

To conclude, ultrasound is a useful and I believe an effective tool to aid in the diagnostic process to evaluate tendon pathology. However, it is only part of the process and, in isolation, can be as misleading as it is helpful.

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- 1 Khan K, Kannus P. Use of imaging data for predicting clinical outcome. *Br J Sports Med* 2000;34:73.
- 2 Kannus P, Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. *J Bone Joint Surg [Am]* 1991;73:1507-25.

Denial of mental illness in athletes

EDITOR,—Professor Schwenk makes the important point that elite athletes are not immune to serious mental illness and that many of the symptoms of overtraining may, in another context, be considered diagnostic of depression.¹

I have usually considered the following to be a helpful differentiator between the two conditions. Patients with depression will almost always resist any suggestion that they may be more physically active. In contrast, the complaint of the athlete with what has been termed either overtraining or the chronic fatigue syndrome will usually be that they desperately wish to exercise. However, whenever they do exercise, they become profoundly fatigued such that the exercise is not pleasant and further compounds their state of chronic fatigue.

However, after reading Professor Schwenk's article, it struck me that, as fatigue is a symptom that is perceived centrally in the brain, it may be that this distinction is not as clear cut as one may conclude. Could exercise intolerance, as opposed to exercise avoidance, be a symptom of depression in elite athletes?

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- 1 Schwenk TL. The stigmatisation and denial of mental illness in athletes. *Br J Sports Med* 2000;34:4-5.

BOOK REVIEWS

Facilitated stretching. 2nd ed. Robert E McAtee, Jeff Charland. (Pp 143; soft cover; £13.95.) Leeds: Human Kinetics Europe Ltd, 1999. ISBN 0-7360-0066-6.

This is the second edition of a book previously published in 1996 which has been reorganised to make it easier to use and broaden the scope of stretches presented.

Chapter 1 begins with the historical basis of PNF, discussing the work of Kabat and later Knott and Voss. It then goes on to explain the myotactic stretch reflex and the

role of muscle spindles, together with the role of the Golgi tendon organ in the inverse stretch reflex (autogenic inhibition) and its function in Chaitow's muscle energy technique, where muscle elongation takes place during "post-isometric relaxation".

Chapter 2, "Stretching basics", skims over the subject of whether it is necessary to stretch. Although it is admitted that there is no clear agreement of the value of stretching, personally I would have liked to have seen quoted some references both for and against stretching. Again in this chapter the ideal scenario is stated "stretch after warming up, exercise, then stretch again after exercise as part of the cool-down process" but then this is followed by "If time is a factor . . . we recommend skipping the pre-exercise stretching and concentrate on the post-exercise stretching." As a physiotherapist, I would advise the opposite—that is, stretch before exercise—as I feel this helps to reduce injury. This apart, the rest of the chapter is well written and briefly considers several different types of stretching, concluding with a detailed description of how to carry out the techniques for both therapist and subject, emphasising the importance of positioning to minimise the risk of injury to both, and how to isolate individual muscles.

The final section of part I describes patterns of movements for both upper and lower limbs with useful black and white photographs to assist with understanding.

Part II of the book contains the stretches and is divided into chapters on stretches for the lower extremity, upper extremity, torso, and neck. The general layout begins with the anatomy of the muscle group, accompanied by a line drawing, a table that shows origin, insertion and action, and functional assessment, showing normal ranges of movement. The stretches are then described with relevant photographs showing the positions of subject and partner. Finally there is a "self stretch," with description and black and white photograph.

The final chapter in part II is entitled "PNF in physical therapy" and differs from the previous section of the book, as it deals with treatment of injury and the role of PNF in rehabilitation, providing case presentations and treatment programme.

In a literature review in the appendix it states that "eight of the fourteen studies reviewed (57%) found that PNF stretching is significantly more effective for increasing ROM and flexibility than static, ballistic or passive stretching" but does not provide sufficient information for one to read these studies and compare the protocols used. Furthermore the number of references used throughout the book is comparatively small considering the wealth of studies now being published on the subject of flexibility.

Overall I feel the book is well written and informative supported by good drawings and photographs. My only reservation is the cover of the book. Although the second edition has been published this year, the colours, style of presentation, and photographs give the impression that the book belongs in the 1970s! Notwithstanding this, I feel the book will be of great value to everyone working within the field of rehabilitation and sports injury.

Analysis

Presentation	15/20
Comprehensiveness	15/20
Readability	17/20
Relevance	17/20
Evidence basis	13/20
Total	77/100

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HIV/AIDS in sport. Impact, issues and challenges. G Sankaran, K A E Volkwein, D R Bonsall. (Pp 137; soft cover; £21.50.) Leeds: Human Kinetics Europe Ltd, 1999. ISBN 0-88011-749-4.

The complexity of the issues surrounding HIV and AIDS in sport is dealt with in a concise yet comprehensive manner in this book. The issues range from epidemiology and immunology of HIV to ethical and legal matters. The chapter dealing with the basic science of HIV was informative and yet written in such a way as to be within the grasp of someone not in that field. A similar section dealing with exercise and immune function was well covered, and I agree with the conclusion that more work should be carried out in this particular area. Personal accounts of both amateur sports people and international sport stars were insightful, but lacked depth and skimmed the surface of the full implications in this difficult area. However, these may lie beyond the scope of such a broad ranging book. The chapters dealing with legal and ethical issues were, on the whole, difficult to read and perhaps not geared for the layman. The helplines would only really be appropriate to readers living in the United States. This and the high price are the only criticisms I have of a neat and well presented book that is bound to become well thumbed by those in the field.

Analysis

Presentation	15/20
Comprehensiveness	15/20
Readability	12/20
Relevance	18/20
Evidence basis	15/20
Total	75/100

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Boxing and medicine. Ed R C Cantu. (Pp 207; £28.50.) Leeds: Human Kinetics Europe Ltd, 1995. ISBN 0-87322-797-2.

I enjoyed reading this book. A book consisting solely of accounts of boxing related injuries would probably have been a chore to read and reinforced my prejudices about a pastime in which participants have to punch their opponents to score points. However, this is a well balanced book that sets out the medical aspects of boxing in a logical fashion.

The differences between amateur and professional boxing are explained, and there are chapters that address the ethical and social aspects of the sport in America. Robert Cantu, the editor, a past president of the American College of Sports Medicine and medical director of the National Centre for Catastrophic Sport Injury Research, has overseen contributions from a number of eminent people. Experts look at the medical

problems associated with boxing and what evidence there is to confirm how they occurred. It is pointed out that there is a lack of well controlled studies of boxing injuries. The Johns Hopkins Medical Institute study of central nervous system function in amateur boxers is a linear prospective investigation, which was reported by two of the team in the chapter discussing the risk of brain damage. Initial findings (1994) of impaired cognitive function being related to the number of previous bouts but reported as being not clinically significant needs further elucidation, and follow up results should be interesting.

The editor points out that safety changes have been made in American football, professional boxing, and, in particular, amateur boxing through informed medical advice. He would like to see the safety and preventive medicine aspects of the amateurs incorporated into the professional world, and the final chapter of the book is an excellent account of how professional boxing could be made safer. Measures are outlined with reasons why they are necessary.

The IOC medical commission does not now permit theophyllines and systemic steroids, which are suggested as permissible for asthma treatment. Otherwise the role of the ringside doctor is extremely detailed, comprehensive, and includes an excellent account of safety measures that need to be present.

Boxing is an extremely contentious subject, but this book will be of interest to any sports physician involved in boxing or who at least wishes to be able to take part in reasoned debate.

Analysis

Presentation	17/20
Comprehensiveness	17/20
Readability	16/20
Relevance	18/20
Evidence basis	17/20

IAN MCGIBBON

Kircudbright

SYMPOSIUM REPORT

Symposium on football medicine

This one day symposium took place at Liverpool Medical Institution on 16 March 2000. A total of 65 delegates from the whole spectrum of sports physicians, surgeons, and therapists across the United Kingdom attended, in addition to local general practitioners.

Mr Steve Bollen debated the management of ankle ligament injuries; a sound evidence basis substantiated his conclusion that, although surgery for chronic instability and pain does afford good results, operative intervention has little place in the acute management of even a grade three injury.

Professor David Chadwick reported on the latest Australian data from the Victorian State Injury Surveillance System, and the concept of "convulsive convulsions" was discussed. He suggested that the second impact syndrome may be a myth, as it is not reported

in certain sports such as boxing where it might be expected.

Professor Wayne Gibbons demonstrated the use of ultrasound—as scanners become cheaper they could be used for “near-patient testing”. The demonstration on MRI challenged anatomy textbooks, in particular, the existence of the conjoint tendon which may be an embalming artefact.

Dr John Hunter’s presentation on the “Effects of exercise on the gut” included joggers’ diarrhoea, and it seems that it is not a general effect of exercise, but certain people such as the young and poorly trained may be more susceptible.

Mr David Rees from the Elite Sports Assessment Centre showed the facilities and techniques used at their sports injuries laboratory in Oswestry. In particular, anterior cruciate ligament rehabilitation and assessment was discussed.

The symposium concluded with Professor Klenerman discussing foot and ankle injuries. Early controlled mobilisation was preferred to immobilisation in plaster after Achilles tendon repair.

STEVE McNALLY

General practitioner and Medical Officer to
Liverpool FC Academy

NOTES AND NEWS

Annual meeting of the American College of Sports Medicine

A large contingent from the UK travelled to the annual meeting of the American College of Sports Medicine. Nic Mafulli gave the annual BASEM lecture to a select group of tendon experts, and many other prominent BASEM members gave important presentations. The number of UK participants at this meeting has increased greatly and already a number of research groups are planning to contribute to next year’s meeting in Baltimore.

Guidelines for advising on injury treatment and prevention

There is increasing awareness in the sporting and medical community of the need for medical input in injury treatment and prevention. The British Medical Association is currently considering the need to issue guidelines to doctors who may be acting, or interested in acting, as a medical advisor to sports clubs or at other public events in a voluntary, rather than a professional full time capacity. It is envisaged that the issues covered would include reference to the courses run by the Football Association, the National Sports Medicine Institute, and any other relevant organisations. Such issues as insurance, responsibilities of the doctor, relationship with competitors’ GPs, legal and contractual arrangements, responsibility for crowd injuries, and the need for knowledge of injuries specific to the sport/event would be covered. It is likely that any guidelines issued would appear on the BMA website with links to other organisations and would be sent to interested doctors. The British Medical Association has consulted with a number of bodies about the guidelines, including the

RCGP sport and exercise medicine working group, BASEM, and NSMI. Any individual who wishes to make their views known can contact any of these groups or may wish to contact Nick Harrison at the BMA on 020 7383 6225 or nharrison@bma.org.uk.

Stimulated by the articles on education in this issue?

The School of Postgraduate Medical and Dental Education at the University of Wales College of Medicine (UWCM) recently advertised their Diploma/MSc in Sports Medicine. The purpose of this course is to educate doctors and chartered physiotherapists who wish to develop their expertise in sports medicine. It will be organised primarily as an open distance learning programme and is PGEA approved. The cost is £1800 for national students and £3585 for international students. Further details are available from Mr Gareth Irwin, University of Wales Institute, Cardiff, Cynod Road, Cardiff CF23 6XD; tel: 0292 041 6537; email: Girwin@uwic.ac.uk.

BASEM 2000 conference

There is already considerable interest in the BASEM 2000 conference in Tewkesbury on 3–5 November. The congress continues to develop and the combination of outstanding international speakers, the very best of research from the UK, and a vibrant social programme ensures its continued success. The current interest and controversies surrounding the management of head injury in sport will attract considerable academic and media interest when Dr Bob Cantu, one of the leading researchers from the USA, addresses this topic on the afternoon of Saturday 4 November. Our other keynote speaker, Professor Norbert Bachl from Austria, promises a fascinating lecture on the effect of living on Space Station Mir. We also look forward to hearing about European developments in sport and exercise. Further details are available from Mrs Sue Roberts, BASEM Company Office, 12 Greenside Avenue, Frodsham, Cheshire WA6 7SA. Tel: 01928 732 961; email: basemoffice@compuserve.com.

CALENDAR OF EVENTS

British Association of Sport and Exercise Sciences Annual Conference

29 August–1 September 2000; Liverpool, UK
Further details: BASES 2000, Event Management Services, Egerton Court, 2 Rodney St, Liverpool L3 5UX. Tel: 0151 231 3585. Fax: 0151 709 5057. Email: ems@livjm.ac.uk

Diploma in Sport and Exercise Medicine, Great Britain and Ireland

This two part diploma examination will be held twice a year. Part 1 of the examination, consisting of a multiple choice question and

short essay paper will be held in April and September in London, Glasgow, or Dublin. Successful candidates will proceed to part 2 of the examination in either June or November. This consists of an oral and a clinical, based on two OSCEs, and will be held at a single centre which will rotate every six months
Further details: Examinations Department, Royal College of Surgeons in Edinburgh, Nicolson Street, Edinburgh EH8 9DW.
Website: www.rcsed.ac.uk

2000 Pre-olympic scientific congress

7–13 September 2000; Brisbane, Australia
Themes running through the programme include:

- Role of the Olympic Games in promoting health for all
- Impact of elite athlete sports medicine on the general community
- Ethical issues and ergogenic aids
- Sports medicine, sports science, and physical activity in the new millennium
- Funding of elite sports versus physical education
- Regional issues and developing countries
- Sport for whom? Nations, corporations, spectators or athletes?
- Manipulating athletic bodies: science, training, technology, and drugs in the 21st century.

Further details: Congress Manager, Sports Medicine Australia, PO Box 897, Belconnen Act 2616, Australia. Tel: +61 2 6251 6944. Fax: +61 2 6253 1489. Email: smanat@sma.org.au

19th congress of sports medicine

13–14 October 2000; Bruges, Belgium
Topics include:

- Sports physiotherapy
- Children and sports
- Arthroscopy and sports traumatology
- Medical ethics, doping, and sports

Further details: Dr Michel D’Hooghe, President Brusport, Hospital AZ Sint-Jan AV, Ruddershove 10, B-8000 Brugge, Belgium. Tel: +32 50 452230. Fax: +32 50 452231. Email: brucosport@azbrugge.be
Website: <http://user.online.be/brucosport/index.htm>

1st Moscow International Forum: Sport medicine science and practice on the eve of the 21st century

20–25 October 2000; Moscow

Further details: Organising Committee of the Forum, Yachshuk AM, Zemlyanoi Val 53, Moscow. Tel: +7 928 29 92

Symposium: training, overtraining, and regeneration in sport—from the muscle to the brain

26–28 October 2000; University of Ulm, Germany

Topics include:

- Training and regeneration in sports
- Metabolism, training, and monitoring
- Cellular protection and immunological function
- Muscular adaptations and stress proteins and cytokines

- Peripheral mechanisms for adaptation and regeneration
- Hypothalamic hormonal regulation and the central nervous system

Further details: Dr J M Steinacker, Abt. Sport and Rehabilitationsmedizin, Medizinische Klinik und Poliklinik, Universitätsklinikum Ulm, 89070 Ulm, Germany. Tel: +49 731 502 6966; fax: +49 731 502 6686; email: org.sportmed@medizin.uni-ulm.de
Website: www.uni-ulm.de/sportmedizin

British Association of Sport and Medicine congress

3–5 November 2000; Tewkesbury, UK

Lectures include:

- Muscular conditioning during space station MIR flight
- Health enhancing physical activity—an upgrowing challenge for sports medicine

Further details: Mrs Sue Roberts, BASEM Company Office, 12 Greenside Avenue, Frodsham, Cheshire WA6 7SA. Tel/fax: 01928 732 961; email: basemoffice@compuserve.com
Website: www.pmhcs.com/basem

20th national congress of the Société Française de Médecine de Sport: Physical activity, sport and health

6–8 December 2000; Paris, France

Topics include:

- Physical activity and fertility
- Sport and aging
- Rehabilitation

Further details: Pranacom, 40 rue des Blancs Manteaux, 75004 Paris, France. Email: pranacom.ifrance.com
Website: www.sfms.asso.fr

NSMI/BASEM Current concepts meeting on tendinopathies

8–9 December 2000; Cambridge, UK

Subjects covered include:

- Tendon science
- Achilles tendon
- Rotator cuff

Further details: Barry Hill, NSMI Medical Education, Medical College of St Bartholomew's Hospital, Charterhouse Square, London EC1M 6BQ. Tel: 020 7251 0583 x237; fax: 020 7251 0774; email: barry.hill@nsmi.org.uk
Website: www.nsmi.org.uk

CORRECTION

The authors of Khan *et al* (*BJSM* 2000;34:81–83) have conceded an error. They referred to patellar tendon allograft instead of patellar tendon autograft, and regret any confusion they may have caused.

True or false?—answers

(T = true; F = false)

p 246: Pedersen BK, Toft AD. Exercise effects on lymphocytes and cytokines
(1) T; (2) F; (3) T; (4) F; (5) F.

Essay question—answer

p 246: Pedersen BK, Toft AD. Exercise effects on lymphocytes and cytokines
Exercise induces increased levels of cytokines in the blood. The levels of TNF, IL-1, IL-6, IL-1ra, IL-8, IL-10, MIP-1 β and sTNF-R increase. IL-6 increases more than any other cytokine, the increase being up to 100-fold that measured at rest.

Multiple choice—answers

p 252: Rath E, Richmond JC. The menisci: basic science and advances in treatment

1 (e); 2 (c); 3 (a); 4 (c); 5 (b).

Essay questions—answers

p 252: Rath E, Richmond JC. The menisci: basic science and advances in treatment.

- 1 This patient might present with recurrent joint line pain, episodic swelling, clicking. Often there will be no or trivial trauma. Physical examination may disclose an effusion, with joint line tenderness, and pain on forced flexion.
- 2 The meniscus serves several important functions, most notably force distribution and joint surface protection. Preserving healthy meniscal tissue will reduce the long term risk of osteoarthritis.