

PostScript

LETTERS

Runner's stitch and the thoracic spine

The cause of the abdominal pain commonly referred to as "stitch", but more accurately known as exercise related transient abdominal pain (ETAP), had until recently attracted more speculation than research. Explanations for the pain have included diaphragmatic ischaemia,¹ stress on peritoneal ligaments,² and irritation of the parietal peritoneum.³ Although the exact cause of the pain remains to be elucidated, a neurogenic explanation has not appeared in the literature.

We present the case of an elite runner who, after a thoracic spine trauma, developed severe and recurrent episodes of ETAP which were relieved by localised treatment. As a follow up study, we made observations on 17 other runners who often experience episodes of ETAP and found that palpation of specific facet joints could reproduce symptoms of ETAP shortly after an episode of the pain had been relieved.

Case study

The patient was a 25 year old man who had been actively involved in competitive running for about 16 years. During this time, he claimed that he occasionally experienced mild symptoms of ETAP during training and competition, although they were not disruptive to performance. After trauma to the thoracic spine in a trampoline accident, he began to experience ETAP with greater frequency and severity. Subsequently he would develop ETAP often when walking and consistently when running. The presentation of the pain essentially forced him from competition. He had received advice from several medical practitioners before being referred to our clinic about two years after the trauma. Symptoms had been present and persistent when running was attempted for this time period.

On questioning, it became apparent that several conditions that stress the spine exacerbated the experience of ETAP. For example, torso hyperextension coupled with the jolting movement of downhill running provoked the pain. In addition, tight erector spinae after a surfing session seemed to increase the likelihood of experiencing ETAP.

On assessment, vertebral hypomobility was observed at the T7-T8 level, presumably as a result of the trampoline accident. Accompanying this was hypermobility of the T8-T9 joint. Palpation of the left T8-T9 facet joint resulted in reproduction of sub-costal pain consistent with the patient's exercise induced experience of ETAP. Further, the site of the pain reproduction was consistent with the dermatome distribution arising from the T8-T9 level. Palpation of the joints above and below the joint caused no pain referral.

Localised treatment aimed at mobilising the T7-T8 joint was administered in the clinic, and the patient was given mobilising exercises to perform. After about four weeks

of treatment, he reported a reduction in the symptoms of ETAP. He has since resumed competitive running.

Further observations

In response to the presentation of this case, we recruited 17 other runners who spasmodically experienced ETAP during training and competition. Ethical clearance was obtained from the Avondale College human research ethics committee. The subjects exercised on a treadmill after a meal until ETAP developed, at which time the exercise session was terminated. After the pain had completely disappeared, the subjects lay in a prone position and were submitted to a spinal assessment, in which facet palpation was performed along the length of the thoracic region. The subjects were not informed of the intent of the study but were asked to volunteer any symptoms of pain or discomfort that arose.

In eight cases (47%), the exact previous experience of ETAP was reproduced through palpation of the thoracic spine at various levels from T8 to T12. In another six cases (35%), the subjects reported lateral and anterior pain referral towards the site of ETAP as a result of the palpation. The longitudinal site of ETAP along the abdomen correlated significantly with the level of the thoracic spine producing symptoms on palpation. This result was observed when only the subjects reporting exact reproduction were analysed ($r = 0.78, p < 0.05$) and also when all subjects reporting any form of pain referral were included in the analysis ($r = 0.63, p < 0.05$). Further, the level of the spine producing symptoms on palpation and the site of ETAP were consistent with dermatome distribution.

Discussion

There has been little or no mention in the literature of the possible role of the spine in the cause of ETAP, although there are several observations that indicate that its contribution may be noteworthy. Firstly, in 1941 Capps¹ anecdotally observed that people with an increased kypholordotic postural alignment seemed most susceptible to the pain. To investigate this further, we recently confirmed in a study involving a comprehensive postural assessment of over 150 subjects that increased kyphosis is provocative of ETAP.⁴ This observation is interesting given that intercostal nerves arising from the thoracic region supply the anterior abdomen. In addition, it has been suggested that "jolting" of the torso is primarily responsible for evoking the pain,² and this action is known to result in intervertebral compression.⁵ As a side issue, we documented that horse riding is one of the sporting activities most likely to induce ETAP.³ It has also been asserted that an episode of ETAP can be relieved through body inversion.² Finally, we have argued that the characteristics of the pain, being well localised, lateral, and mostly sharp, are consistent with somatic rather than visceral abdominal pain.⁶ An explanation for the pain involving the intercostal nerve would account for these pain characteristics.

The extent to which the thoracic intercostal nerves may contribute to the experience of ETAP is worthy of further investigation. It seems plausible that, in some cases mechanical compression of the nerve root may refer pain distally, resulting in abdominal pain. Alternatively, irritation of the nerve may sensitise it to stimuli such that the threshold required for activation is lessened. Hence, in this study, palpation after the pain had been relieved may have allowed tissues innervated by the intercostal nerves, such as the abdominal musculature or parietal peritoneum, to recreate sensations of pain.

Although the significance of the observations documented in this report remains to be fully elucidated, they do indicate that the spine may play a role in the genesis of ETAP. Further studies are required to determine the extent to which the spinal nerves are involved and the potential benefits of improving spinal integrity in ETAP sufferers. Certainly, the spine should be considered when treating patients with recurrent symptoms of ETAP.

D P Morton

Avondale College, Cooranbong, NSW, Australia

T Aune

Total Physio Centres, Cooranbong, NSW 2265, Australia

Correspondence to: Dr Morton, Avondale College, Lifestyle Education, PO Box 19, Cooranbong, NSW 2265, Australia; darren.morton@avondale.edu.au

doi: 10.1136/bjism.2003.009308

References

- 1 Capps RB. Causes of the so-called side ache in normal persons. *Arch Intern Med* 1941;68:94-101.
- 2 Sinclair JD. Stitch: the side pain of athletes. *N Z Med J* 1951;50:607-12.
- 3 Morton DP, Callister R. Characteristics and aetiology of exercise-related transient abdominal pain. *Med Sci Sport Exerc* 2000;32:432-8.
- 4 Morton DP, Aune T. Role of the thoracic spine in the experience of exercise-related transient abdominal pain. *Proceedings of the Australian Conference of Science and Medicine in Sport*. Sports Medicine Australia, 2001.
- 5 Garbutt G, Boocock MG, Reilly T, et al. Running speed and spinal shrinkage in runners with and without low back pain. *Med Sci Sport Exerc* 1990;22:769-72.
- 6 Bynum TE. Abdominal pain. In: Stein JH, ed. *Internal medicine*. St Louis: Mosby, 1994:402-8.

Science versus opinion

Dr Berger recently responded¹ to my article in the *British Journal of Sports Medicine* entitled: Berger in retrospect: effect of varied weight training programmes on strength.² Dr Berger presented no scientific evidence to support his opinion on single versus multiple sets, cited references that were irrelevant to the topic, and challenged the reported statistical analyses.

Dr Berger claimed that most athletic and therapeutic professionals have added credence and support to the belief that multiple sets are required for optimal gains in strength because they use multiple sets in their practice and research (p 372).¹ However, just

using multiple sets in practice is not evidence that multiple sets are superior to a single set; it merely perpetuates an unsubstantiated belief. Table 4 (p 322) in my retrospect² lists 57 studies that reported no significant difference in strength gains as a result of performing a greater number of sets. Dr Berger³ failed to cite a single study to support his opinion on the superiority of multiple sets. In fact, Dr Berger's own follow up studies,^{4,5} which are described in my retrospect,² failed to support his opinion—that is, a greater number of sets did not result in a significantly greater strength gain in either of his follow up studies.^{4,5}

Dr Berger claimed that some early studies compared different weight training programmes (p 372),¹ and he cited five studies.^{6–10} However, these studies (described below) are irrelevant to the issue of single versus multiple set resistance training.

Delorme *et al*⁶ treated eight men and 11 women with poliomyelitis-weakened and atrophied knee extensor muscles. Their goal was to increase muscular strength and hypertrophy. The patients performed three sets of 10 repetitions (intensity not reported) of knee extension and leg press exercises four times a week for one to four months. Isometric force increased 1–200%. No statistical pre to post training analysis was reported. All the patients performed three sets of each exercise; consequently, there was no comparison of single versus multiple sets.

Krusen⁷ randomly assigned military aged male (n = 23) and female (n = 1) patients with poliomyelitis to one of two knee extensor strength training protocols (sessions per week not reported). One group performed five repetitions with 25% 5RM, five repetitions with 50% 5RM, and five repetitions with 75% 5RM twice a day for 3–23 weeks. The other group performed five repetitions with 100% 5RM, five repetitions with 125% 5RM, and five repetitions with 150% 5RM, with limited range of motion on the last two sets twice a day for 3–19 weeks. Krusen claimed that, to rule out the difference in initial strength, 11 subjects in each group were matched for strength and compared after five weeks of training. Knee extension 1RM increased in both respective groups (74.5% and 60.0%), with no significant difference between groups in strength gains. A comparison of single versus multiple sets was not possible because both groups performed three sets of exercise. The actual comparison was between a low intensity protocol (five repetitions with 75% 5RM) and high intensity protocol (five repetitions with 100% 5RM), which resulted in similar strength gains.

McMorris and Elkins⁸ trained the right triceps in nine healthy men and three women (20–29 years of age) with four sets of exercise five times a week for 12 weeks. One group performed four sets of 10 repetitions with 25%, 50%, 75%, and 100% 10RM in ascending order, while the other group performed four sets of 10 repetitions with 100%, 75%, 50%, and 25% 10RM in descending order. McMorris and Elkins⁸ reported a 5.5% greater gain in strength for the group that performed the ascending protocol. There was no statistical analysis reported. Because all the patients performed four sets of each exercise, no comparison of single versus multiple sets was possible.

Hellebrandt and Houtz⁹ reported their observations of what they described as 620 experiments in 17 male and female

competitive athletes. Wrist flexion and extension exercise varied from 3 to 40 sets of 25 repetitions with 25–100% 25RM three to five times a week for three to seven weeks. There was no statistical analysis reported, and more importantly, no comparison of single versus multiple sets.

McGovern and Luscombe¹⁰ reported on a two part investigation. In part 1, 10 healthy men (20–30 years of age) were divided into two groups (A and B). Group A performed one warm up set of unilateral quadriceps exercise for five repetitions using 50% 10RM, followed by one set of 10 repetitions with the 10RM. Group B performed one range of motion warm up set (no resistance), followed by three sets of 10 repetitions with 100%, 75%, and 50% 10RM, respectively, and one minute rest between sets. A third group (C) of five male orthopaedic patients (20–30 years of age) performed three sets of 10 repetitions with 50%, 75%, and 100% 10RM respectively. The groups trained five times a week for three weeks. No pre to post training or between group statistical analysis was reported. However, the authors stated that the increase in 10RM was similar for groups A, B, and C (7.7, 8.7, and 7.7 kg respectively) in part 1. In part 2, 28 male and female orthopaedic patients (20–30 years of age) trained five times a week for four weeks. Half the patients performed one set of 10RM and the other half performed three sets of 10 repetitions with 50%, 75%, and 100% 10RM. The two groups of patients showed similar gains in 10RM (5.8 and 6.0 kg). McGovern and Luscombe concluded that the two training protocols produced similar strength gains, with a time saving advantage to the single set group.

The study by McGovern and Luscombe¹⁰ was the only one of the five studies^{6–10} cited by Dr Berger¹ that compared single versus multiple sets, and they reported similar strength gains as a result of one set or three sets of exercise.

Dr Berger claimed that the medical applications of strength training for physical therapy have historically involved multiple sets, and that one set was the exception (p 372).¹ In 1951, Delorme and Watkins¹¹ specifically stated that increasing the resistance after each of three sets provided an advantage for warming up the muscles in patients suffering from poliomyelitis, but probably did not contribute to the increase in muscular strength. They concluded that performing one set or three sets of 10RM would produce a similar increase in strength (pp 27–8).¹¹ Contrary to Dr Berger's claim that one set was an exception, and contrary to Dr Berger's study³ where his nine groups performed one, two, or three maximal sets, Delorme *et al*,⁶ Krusen,⁷ McMorris and Elkins,⁸ and McGovern and Luscombe¹⁰ actually trained all their subjects with only one maximal set.

Dr Berger claimed that he was not the genesis of an unsubstantiated belief (p 372).¹ In fact, I noted in my retrospect² that Dr Berger's study³—not he personally—was perhaps the genesis of the unsubstantiated belief that multiple sets are required for optimal strength gains (p 319).² The flow chart (fig 1, p 320) in my retrospect² clearly shows that Dr Berger's study³ was the only strength training study cited.

Dr Berger stated that therapists and coaches are flinging down the gauntlet in support of multiple sets (p 372).¹ Apparently, Dr Berger is claiming that therapists and

coaches are challenging others to show that multiple sets are not superior. However, a primary tenet of science is that the burden of proof rests entirely on those making the claim. If Dr Berger and the therapists and coaches he refers to claim that multiple sets are superior, then the entire burden of proof is on them to support that claim with peer reviewed strength training studies.

Dr Berger claimed that the 0.05 level of probability does not always supersede common sense when attempting to control extraneous factors in strength research (p 372).¹ In fact, it was Dr Berger who chose the 0.05 level of probability for his study.³ In my retrospect,² I simply reported the level of probability that he used.

Dr Berger claimed that if he had concluded that one set was as good as multiple sets in 1962, practitioners in the field would have confronted him to express their disagreement (p 372).¹ It is not my fault that the reviewers and the editor of *Research Quarterly* initially failed as gatekeepers and that in the last 40 years other scientists in the field have not challenged Dr Berger's methodology, statistical analysis, and conflicting conclusions.

Dr Berger noted that Arthur Jones, who developed and sold Nautilus machines, was a proponent of single sets and that he has never presented any acceptable scientific evidence to support his belief (p 373).¹ Mr Jones was never mentioned or cited in my retrospect² because Mr. Jones' beliefs, or anyone who agrees or disagrees with those beliefs, are completely irrelevant to Dr Berger's study,³ my retrospect,² and Dr Berger's response¹ to my retrospect.

Dr Berger claimed that the data in tables 1, 2, and 3 of my retrospect² were inappropriately used according to acceptable statistical protocol (p 373).¹ In fact, it was Dr Berger who used the statistical analyses that I reported in my retrospect.² The data in table 1 (p 320) of my retrospect² which show the increase in 1RM bench press for each of Dr Berger's nine groups, were gleaned directly from Dr Berger's table 1 (p 172).³ The data in table 2 (p 320) of my retrospect² which show the groups that differed significantly at the 0.05 level of probability, were from Dr Berger's table 4 (p 176).³ When I questioned Dr Berger about the three typographical errors in his table 4, he said that he was not aware of these errors and claimed that no one else had ever questioned them before our telephone conversation in 1998. The correct data are shown in table 2 of my retrospect (p 320).² Table 3 (p 321) in the retrospect² shows that for the groups who performed the same number of repetitions (two, six, or 10), seven out of nine of Dr Berger's comparisons showed no significant difference in the magnitude of strength gain. For example, the strength gain in the 3×6 group was significantly greater than the 2×6 group, but not greater than the 1×6 group. Likewise, 3×2 was significantly greater than 2×2, but not significantly greater than 1×2. The data are all from Dr Berger's table 4 (p 176).³

Dr Berger claimed that comparisons between his nine groups were not valid for critiquing his study (p 373).¹ I did not make any comparisons between Dr Berger's groups. However, I did accurately note in my retrospect² exactly what Dr Berger reported in his study.³ Dr Berger's subsequent admission that he erroneously made comparisons among the nine groups in table 4 of his study³ (p 373)¹ nullifies his previous claim.

Dr Berger claimed that when he compared the main effects among one, two, or three sets, and among two, six, or 10 repetitions, three sets and six repetitions resulted in the greatest improvement in strength (p 373).¹ In his Results section¹ Dr Berger stated that analysis of covariance was used to test for a significant interaction between sets and repetitions—that is, whether one, two, or three sets might be better in combination with two, six, or 10 repetitions. The *F* ratio was not significant at any period of training (p 178).³ Dr Berger also reported in Conclusion 6 that training with one, two, or three sets in discrete combination with two, six, or 10 repetitions per set (interaction) was not more effective for improving strength than other combinations of sets and repetitions (p 181).³ However, in Conclusion 7 Dr Berger claimed that a combination of 6RM performed for three sets was more effective in improving strength than any other combination of sets and repetitions (p 181).³ These antithetical statements were reported in my retrospect.² Dr Berger failed to address them in his response¹ or in our personal correspondence (1998).

Dr Berger noted that he spoke with me about my misuse of statistics and that he suggested that I consult a statistician (p 373).¹ I followed his advice and consulted two statisticians before submitting my retrospect.² Their opinion was that Dr Berger should not have made the 36 individual comparisons that he reported because the multiple comparisons increased the chance of a type I error, which could have led to rejection of the null hypothesis when the null hypothesis was actually true.

Dr Berger stated that he still unequivocally supports multiple sets over a single set for optimising strength (p 373).¹ Dr Berger is perfectly entitled to his opinion, but it has nothing to do with supporting his belief with scientific evidence. As noted in my retrospect,² Dr Berger expressed his opinion about the superiority of multiple sets in our personal correspondence (1998), and in at least five publications.^{12–16} The only study he cited to substantiate his opinion was his 1962 study.³ Dr Berger neglected to cite his own follow up studies^{4–7} that failed to support his opinion.

Dr Berger suggested that I conduct research in the hope of gaining support for my position, although he does not define my position (p 373).¹ In fact, my position is simply that if someone in the scientific community makes a claim about anything, they should be held accountable to support that claim with peer reviewed scientific evidence. As noted in the flow chart (p 320) of my retrospect,² most of the physiologists who recommend multiple sets of each exercise cite each other's opinion, reviews and books, cite nothing at all, or they cite Dr Berger's study.³ Because I am not making a claim about the superiority of one strength training protocol over another, I am under no obligation to conduct research. I am simply challenging the widely held belief that multiple sets are superior to a single set, and in turn, challenging the apparent genesis of that belief—Dr Berger's study.³

R N Carpinelli

Human Performance Laboratory, Adelphi University,
Garden City, NY 11530, USA;
q-nautilus@worldnet.att.net

References

- 1 **Berger RA.** Response to "Berger in retrospect: effect of varied weight training programmes on strength." *Br J Sports Med* 2003;**37**:372–3.
- 2 **Carpinelli RN.** Berger in retrospect: effect of varied weight training programmes on strength. *Br J Sports Med* 2002;**36**:319–24.
- 3 **Berger RA.** Effect of varied weight training programs on strength. *Res Q* 1962;**33**:168–81.
- 4 **Berger RA.** Comparative effects of three weight training programs. *Res Q* 1963;**34**:396–8.
- 5 **Berger RA.** Effect of varied sets of static training on dynamic strength. *Am Correct Ther J* 1972;**26**:52–4.
- 6 **Delorme TL, Schwab RS, Watkins AL.** The response of the quadriceps femoris to progressive resistance exercises in poliomyelitic patients. *J Bone Joint Surg* 1948;**30**:834–47.
- 7 **Krusen EM Jr.** Functional improvement produced by resistive exercise of quadriceps muscles affected by poliomyelitis. *Arch Phys Med* 1949;**30**:271–7.
- 8 **McMorris RO, Elkins EC.** A study of production and evaluation of muscular hypertrophy. *Arch Phys Med Rehabil* 1954;**35**:420–6.
- 9 **Hellebrant FA, Houtz SJ.** Mechanisms of muscle training in man. *Phys Ther Rev* 1956;**36**:371–83.
- 10 **McGovern RE, Luscombe HB.** Useful modifications of progressive resistance exercise technique. *Arch Phys Med Rehabil* 1953;**34**:475–7.
- 11 **Delorme TL, Watkins AL.** *Progressive resistance exercise. Technic and medical application.* New York: Appleton-Century-Crofts, Inc, 1951:27–8.
- 12 **Berger RA.** Application of research findings in progressive resistance exercise to physical therapy. *J Assoc Phys Ment Rehabil* 1965;**19**:200–3.
- 13 **Berger RA.** Strength improvement. *Strength and Health* 1972 Aug:44–5.
- 14 **Berger RA.** *Conditioning for men.* Boston, MA: Allyn and Bacon, 1973.
- 15 **Berger RA.** *Introduction to weight training.* Englewood Cliffs, NJ: Prentice-Hall, 1984.
- 16 **Berger RA.** *Applied exercise physiology.* Philadelphia, PA: Lea & Febiger, 1982.

Science versus opinion

Dr Berger recently responded¹ to my article in the *British Journal of Sports Medicine* entitled: Berger in retrospect: effect of varied weight training programs on strength,² which is a critical analysis of one of Dr Berger's early studies.³ In his response,² Dr Berger presented no scientific evidence to support his opinion on single versus multiple sets. He cited references that were irrelevant to the topic, and challenged the reported statistical analyses. I have responded to each of Dr Berger's claims in a brief review⁴ which differentiates the scientific evidence from Dr Berger's opinion.

R N Carpinelli

Human Performance Laboratory, Adelphi University,
Garden City, NY 11530, USA;
q-nautilus@worldnet.att.net

doi: 10.1136/bjism.2003.011163

References

- 1 **Berger RA.** Response to "Berger in retrospect: effect of varied weight training programmes on strength." *Br J Sports Med* 2003;**37**:372–3.
- 2 **Carpinelli RN.** Berger in retrospect: effect of varied weight training programmes on strength. *Br J Sports Med* 2002;**36**:319–24.
- 3 **Berger RA.** Effect of varied weight training programs on strength. *Res Q* 1962;**33**:168–81.
- 4 **Carpinelli RN.** Science versus opinion. www.bjism.bmjournals.com/cgi/letters.

CALENDAR OF EVENTS

Medicare India

6–8 April 2004, New Delhi, India

This exhibition and conference will be held for the first time, following India's ambitious "health for all" programme launched in 2002.

Further details: Rob Grant, Kinex Log, 5 New Quebec Street, London W1H 7DD, UK; tel: +44 (0)207 723 8020; fax: +44 (0)207 723 8060; email: rob.grant@kinexlog.com; websites: www.medicare-expo.com and www.kinexlog.com

12th International Congress on Physical Education and Sport

21–23 May 2004, Kornatini, Greece

Round tables, seminars, oral communications, and posters will take place within the frame of the congress. The congress will include an international audience of doctors, physicians, therapists, scientists and coaches.

Further details: Secretariat of the congress, email: icpes@phyed.duth.gr; website: www.phyed.duth.gr/icpes2004

The 6th STMS World Congress on Medicine and Science in Tennis in conjunction with the LTA 2004 Sports Science, Sports Medicine and Performance Coaching Conference

19–20 June 2004, London, UK

Keynote speakers include Professor Per Renstrom (SWE), Professor Peter Jokl (USA), Professor Savio Woo (USA), Dr Carol Otis (USA), Dr Mark Safran (USA), Dr Ben Kibler (USA), Prof Bruce Elliott (AUS), and Professor Ron Maughan (UK).

Further details: Dr Michael Turner, The Lawn Tennis Association, The Queen's Club, London W14 9EG, UK; email: michael.turner@LTA.org.uk

9th European College of Sports Science Conference

3–6 July 2004, Clermont-Ferrand, France

More than 1500 participants from 70 countries are due to attend.

Further details: website: www.ecss2004.com

The Leeds Sports Imaging Course

6–7 September 2004, Leeds, UK.

This two day course is aimed at both radiologists and clinicians who are involved in sports imaging. The course will comprise an imaging and clinical overview of all relevant joint, bone and soft tissue sporting injuries.

The faculty will comprise internationally recognised skeletal imaging and clinical experts from the UK, Europe and North America who will deliver state of the art lectures and lead sessional discussions. Each session will cover the spectrum of injury for a specific anatomical area beginning with clinical lectures that will allow the subsequent imaging lectures to be placed in context. All aspects of imaging will be discussed but will concentrate on the use of ultrasound, conventional MR imaging and MR arthrography for the diagnosis, staging

and prognosis of sporting injuries. Therapeutic image guided intervention using fluoroscopy, CT and ultrasound will also be demonstrated.

13 RCR category one CME credits have been awarded.

Further details: Carol Bailey, Course co-ordinator, MRI Department, B Floor, Clarendon Wing, Leeds General Infirmary, Leeds LS1 3EX; tel: +44 (0)113 3922826; fax: +44 (0)113 3928241; email: Carol.Bailey@leedsth.nhs.uk

BASEM Conference 2004

14–17 October 2004, Belfast, UK

Main themes: Overuse Sports Injuries and Muscle Injuries. Keynote speakers include: Chris Bradshaw, Medical Director, Olympic Park Medical Centre, Melbourne and Kim Bennell, Assistant Professor, School of Physiotherapy, Melbourne University.

Further details: Email: fionnuala.sayers@greenpark.n-i.nhs.uk

2nd International Ankle Symposium

15–16 October 2004, Newark, DE, USA

The meeting will examine ankle instability and other related ankle pathologies from a multidisciplinary perspective. Attendees will include clinicians and scholars from the disciplines of orthopedics, podiatry, physical therapy, athletic training, biomechanics, and sports medicine. This conference aims to build on the success of the inaugural symposium held in Ulm, Germany in December 2000. Emphasis will be on oral and poster presentations of original research along with selected plenary presentations.

The deadline for abstract submissions is July 1 2004.

Further details: Jay Hertel, PhD, ATC, FACSM, Conference Co-Chair, Penn State University, email: jnh3@psu.edu, or Thomas Kaminski, PhD, ATC, FACSM, Conference Co-Chair and Host, University of Delaware, email: kaminski@udel.edu. Website: <http://www.udel.edu/ias/>

1st World Congress on Sports Injury Prevention

23–25 June 2005, Oslo, Norway

This congress will provide the world's leading sports medicine experts with an opportunity to present their work to an international audience made up of physicians, therapists, scientists, and coaches. The congress will present scientific information on sports injury epidemiology, risk factors, injury mechanisms and injury prevention methods with a multidisciplinary perspective. Panel discussions will conclude symposia in key areas providing recommendations to address the prevention issue in relation to particular injuries and sports.

Further details: Oslo Sports Trauma Research Centre and Department of Sports Medicine, University of Sport and Physical Education, Sognsveien 220, 0806 Oslo, Norway. Email: 2005congress@nih.no; website: www.ostrc.no