PostScript

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RESEARCH LETTERS

The forgotten Barcroft/Edholm reflex: potential role in exercise associated collapse

Approximately 80% of the athletes who collapse in marathon and other long distance sporting events do so only after they have crossed the finish line.[1] Although it is usually taught that "dehydration" explains this phenomenon, logic suggests this to be unlikely because any dehydration induced hypovolaemia should cause cardiovascular failure when cardiac stress is highest—that is, during rather than after exercise when cardiovascular function is returning to the resting state. Instead it is clearly the act of stopping exercise that is the consequent, albeit paradoxical, cause of post-exercise collapse.

We have previously proposed that this form of exercise associated collapse is caused by the persistence into recovery of a state of low peripheral vascular resistance, compounded by removal of the skeletal muscle pump that maintains the right atrial filling pressure during exercise.[1] According to this theory, the combination of a low peripheral vascular resistance and a sudden reduction in venous return would reduce stroke volume and cardiac output acutely, causing hypotension. Indeed this mechanism was used to explain the development of postural hypotension due to "dehydration exhaustion" in military personnel exposed to eight or more hours of exercise in desert heat without fluid replacement, in the classic studies of Adolph.[2]

Thus Adolph[2] wrote that "the ultimate failure of the circulation (in dehydration exhaustion) is of a peripheral type . . . Dilated blood vessels of the skin require additional blood to fill them; correspondingly, the available compensation for vertical posture are diminished, for the blood vessels of the legs compromise between dilating to carry heated blood and constricting to preserve a greater flow to the head" (p 235). He also reported that: "Lying down promptly relieves the circulation and the symptoms" (p 236). Similarly, Lee[3] defined the physiological abnormality causing heat exhaustion as: "A low arterial blood pressure is the critical event, resulting partly from inadequate output by the heart and partly from the widespread vasodilation". He also noted that: "Treatment is simple – recumbency, fluid and salt administration, gentle cooling, and rectification of any predisposing cause" (p 571).

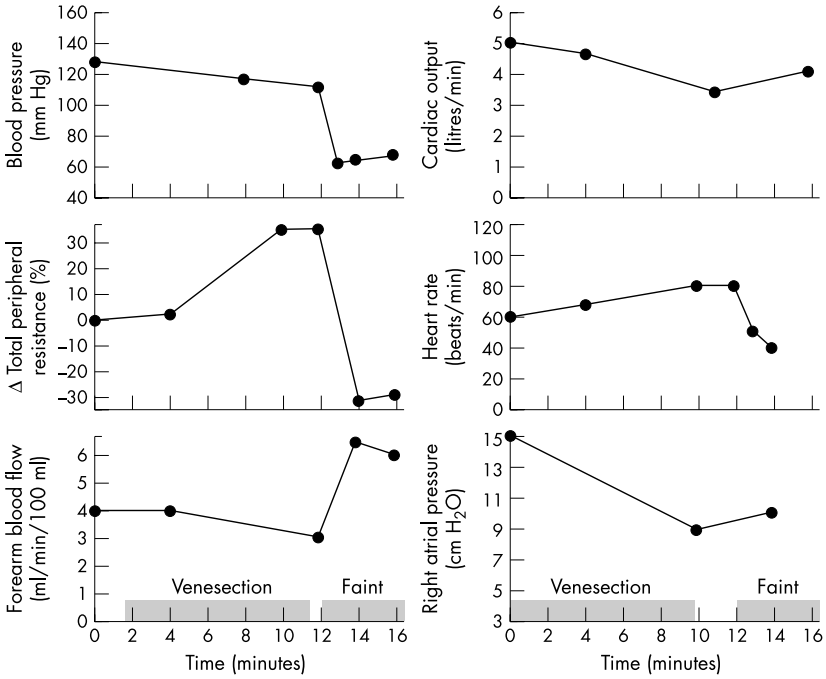


Figure 1 Changes in blood pressure, total peripheral resistance, forearm blood flow, cardiac output, heart rate, and right atrial pressure in subjects who underwent rapid venesection of about 1 litre of blood in 12 minutes. Note that fainting is caused by a sudden reduction in peripheral vascular resistance resulting from an increase in forearm blood flow as right atrial pressure falls, the Barcroft/Edholm reflex. Redrawn from data in Barcroft *et al*.[5]

However, these explanations fail to explain why a compensatory tachycardia is not a usual feature of this form of hypotension,[4] or why the onset of symptoms occurs so rapidly and why they are reversed so rapidly when subjects lie supine in the head down position.[1]

An historic study identifying a potentially forgotten reflex and published in *The Lancet* in 1944 suggests an alternative explanation. Barcroft *et al*[5] studied the effects of rapid venesection of about 1 litre on cardiovascular function measured with right heart cannulation and forearm plethysmography. Studies continued until subjects fainted from the sudden onset of hypotension. Figure 1, redrawn from those historic data, shows the salient findings. Initially, cardiovascular function adapted to progressive blood loss by increasing peripheral vascular resistance and heart rate, each by about 30%. Despite this, average cardiac output, right atrial pressure, and systolic blood pressure all fell by about 40%.

However, the crucial finding was that fainting occurred only when there was a sudden fall in the peripheral vascular resistance associated with an unexpected increase in forearm blood flow (fig 1).

A second study evaluated the authors' hypothesis that it was the fall in right atrial pressure induced by blood loss that activated the reflex reduction in peripheral vascular resistance. A tourniquet was applied to the lower limb and inflated before the beginning of the venesection. Hypotension again developed but after a smaller blood loss of only about 550 ml. Release of the tourniquet immediately corrected the hypotension by reducing forearm blood flow (fig 2). The authors concluded that removal of the tourniquet produced a sudden infusion of blood from the lower limb thereby increasing the right atrial pressure, abolishing the reflex and normalising cardiovascular function.

Hence they proposed the existence of a potent skeletal muscle vasodilator reflex that is activated when the right atrial pressure either falls below some critical value or begins to fall at a particular rate. This reflex appears atavistic because it compounds rather than corrects the hypotension associated with blood loss. The similarity in the rapidity with which hypotension occurs with either progressive venesection or after the cessation of exercise, and without an associated tachycardia, suggests that this right atrial reflex may also cause exercise associated collapse.[1 4]

To my knowledge, some contemporary texts of cardiovascular control during exercise do not specifically mention this reflex. For example, Rowell[6] refers to a later 1945 paper in the *Journal of Physiology* by Barcroft and Edholm,[7] in which the physiological basis for this skeletal muscle vasodilation was studied. However, the relevance of this specific reflex to the development of syncope is not directly discussed. Rather, Rowell presents the findings of

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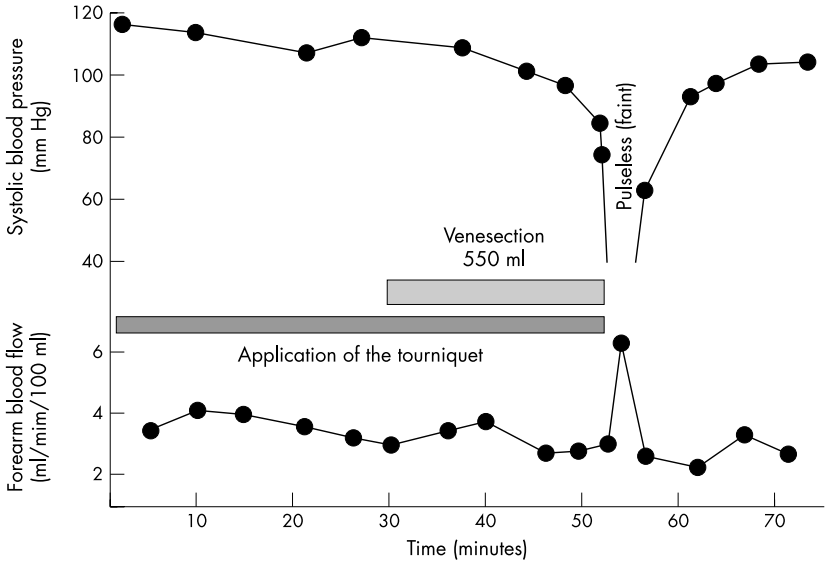


Figure 2 The sudden reduction in systolic blood pressure and the associated increase in forearm blood flow with progressive venesection is immediately reversed by release of a lower limb tourniquet. Redrawn from data in Barcroft *et al*.[5]

Barcroft and Edholm as a possible example of neurogenic sympathetic cholinergic vasodilation in skeletal muscle.

It is of interest that we had empirically discovered some years ago[1] that the optimum management of post-exercise collapse is achieved by elevating the feet and pelvis of collapsed athletes above the level of the right atrium. The results of this manoeuvre are usually dramatic, with rapid reversal of hypotension and the symptoms of dizziness. According to the Barcroft/Edholm reflex, the adoption of this head down position would produce this dramatic response by immediately reversing the low atrial pressure that develops on the cessation of exercise in some susceptible athletes. Hence the effect would not result simply by increasing venous return and hence cardiac output, the more usual explanation,[6] but also by reversing the skeletal muscle vasodilation induced by the Barcroft/Edholm reflex.

Finally, I would like to make the obvious point that the Barcroft/Edholm reflex explains why nursing in the head down (Trendellenberg) position would be the more logical treatment for post-exercise collapse than the provision of intravenous fluids.[1] The reason is that nursing in the Trendellenberg position rapidly increases the right atrial pressure whereas any effect of intravenous fluids on right atrial pressure is likely to be smaller and much delayed.

Furthermore the contribution of the Barcroft/Edholm reflex to exercise associated collapse can be very rapidly evaluated by studying the response of the athlete's blood pressure to changes in posture. Failure of the blood pressure to rise when placed in the head down position must indicate that the Barcroft/Edholm reflex is not active and some other cause for the hypotension, such as myocardial dysfunction or a persistent reduction in peripheral vascular resistance for reasons other than this postulated reflex, must be considered.

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Comparative body fat assessment in elite footballers

Much attention is directed to measuring body composition, particularly fat mass, the aim being to estimate the untoward health consequences of excessive amounts of fat or to assess physical fitness. There are two main types of method of measurement: reference and prediction techniques. The former consist of measuring body density or total body water and dual energy *x* ray absorptiometry, and the latter include measurement of skinfold thickness and bioelectrical impedance analysis.[1] The Tanita body fat analyser is a commercially available foot to foot bioelectrical impedance analysis system.[2] It is a novel method because it measures the weight and percentage of body fat simultaneously while the subject stands barefoot, in contrast with traditional impedance devices which comprise a tetrapolar surface electrode system whereby the weight and height of the subjects are manually entered. In this study, we performed comparative body fat analysis of 29 elite football players using a Tanita analyser and skinfold thickness (SFT) measurements and subsequent predictive equations.

Body fat measurements were carried out using a Tanita TBF-350 (Tanita Corp, Tokyo, Japan). Early in the morning after an overnight fast, subjects stood still on the metal sole plates of the machine wearing only light football shorts. SFTs were measured using Holtain calipers (Crymych, UK) at nine sites on the non-dominant side of the body: biceps, triceps, abdomen, subscapula, suprailiac 1 (midaxillary), suprailiac 2 (anterior axillary), thigh, calf, and chest. All measurements were performed by the same examiner. Predicted body density was calculated as described by Durnin and Womersley[3] using biceps, triceps, subscapula, and suprailiac values. The proportion of body fat was calculated from body density using the equation of Siri,[4] and two equations were used to estimate body fat mass, namely those of Siri[4] and Zorba.[5] Weight was measured using a digital scale and height with a wall mounted stadiometer. Statistical analysis was performed using Pearson correlation coefficients.

Table 1 summarises the basic details and SFT values of the subjects. It seems that the subjects accumulated fat mainly in the abdomen and suprailiac regions and least in the

Table 1 Basic details and skin fold thicknesses (mm) of the subjects

	Mean (SD)	Range
Age (years)	23.62 (3.57)	18–31
Weight (kg)	76.12 (8.34)	64.3–105.4
Height (m)	1.81 (0.05)	1.7–1.92
Body mass index (kg/m^2)	23.20 (1.79)	20.4–28.6
Biceps	4.21 (0.93)	3.2–6.5
Triceps	7.44 (2.46)	4–12.7
Abdomen	10.67 (4.36)	5.3–21.6
Subscapula	9.43 (2.60)	5.3–17.6
Suprailiac 1	7.13 (2.70)	3.4–14.8
Suprailiac 2	10.13 (3.68)	5.3–20.7
Thigh	9.05 (3.24)	5–15.8
Calf	6.96 (2.20)	3.7–12.9
Chest	5.43 (2.14)	3.3–13.1

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Table 2 Correlation coefficients between the measurements

	Body density	Tanita	Zorba	Siri
Body density	–	–0.76	–0.90	–0.91
Tanita	–0.76	–	0.75	0.74
Zorba	–0.90	0.75	–	0.98
Siri	–0.91	0.74	0.98	–

All p values <0.001.

biceps. Table 2 shows correlations between the predictive fat measurements. There were strong correlations between the Tanita measurements and the other estimates. In addition, all the measurements, excluding biceps, triceps, chest, and suprailiac 1, also tended to increase in direct proportion with weight ($p<0.05$ for all values). Body density was found to correlate negatively with all the other measurements ($p<0.001$ for all values).

A similar study to ours was conducted by Utter *et al*[6] in collegiate wrestlers in which three SFT measurements were compared with those obtained with a Tanita analyser (TBF-305). They also showed significant correlations. Cable *et al*[7] studied a group of 192 heterogeneous men, measuring the validity of measurements with a Tanita analyser (TBF-105) compared with underwater weighing and concluded that the bioelectrical impedance system accurately assessed fat free mass.

Although there is some scepticism about using the Tanita body fat analyser and other conventional bioelectrical impedance systems in patients with cardiac disease,[8] we advocate its use in normal subjects, especially in the field of sports medicine, in addition to the reliable conventional SFT measurements,[9] for evaluation of physical fitness.

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LETTERS

Understanding tendinopathies

We commend Professor Murrell for making a major novel contribution to the understanding, and thus potentially the treatment, of chronic tendinopathies.[1] He has shown in patients with supraspinatus tendinopathy that a substantial portion of the cellular compartment of the tendon shows nuclear fragmentation, a key characteristic of apoptosis.[2]

Does Professor Murrell feel that apoptosis would necessarily be a "harmful" pathological event in tendon or might there be an optimal, basal level of apoptosis? We appreciate that excessive apoptosis—programmed cell death—is a feature of degenerative pathologies such as muscular dystrophy and Alzheimer's disease.[3] In these conditions apoptosis may contribute to deterioration of the diseased tissue. On the other hand, it is a characteristic feature in physiological situations such as scar remodelling, in which coordinated death of myofibroblasts is a desirable outcome.[4] A recent study does suggest that proliferation of tenocytes can enhance tendon remodelling, as proliferation of tenocytes after IGF-I injections was accompanied by improved tendon stiffness and reduced lesion in equine flexor tendinopathy.[5] Other studies have shown fibroblast apoptosis from excessive strain[6] or, paradoxically, decreased loading.[7]

We would also appreciate Professor Murrell's opinion on the significance of a third of tendon cells displaying a positive TUNEL assay, months after the onset of symptoms. Could apoptotic cells remain in tendon tissue for months after injury, or might the condition cause continual apoptosis and proliferation concurrently? If the latter were true, would apoptosis be a causal event of tendon degeneration or a secondary effect of scar remodelling? The fact that paratenonitis can directly cause tendinosis and loss of tenocytes suggests a possible causal role of apoptosis.[8]

Does Professor Murrell see growth factors as playing a role in modulating apoptosis in the tendon, and could the effect of growth factors on tenocyte death and survival be tested in vitro or in model systems? Such investigations would be complicated by the many interacting conditions in the tendon which could promote or prevent apoptosis, including integrin mediated signals, soluble factors, and cellular stresses such as hypoxia and reactive oxygen species. The distinct phenotypes of tenocytes within and among tendons would add yet another layer of complexity to the story.

Again, we commend Professor Murrell for this contribution. We will be very curious to know which caspases are activated in the supraspinatus and in other tendinopathies, and whether there would be an inhibitory effect of caspase inhibitors. By showing for the first time that nuclear fragmentation is a significant feature of tendinopathy, Professor Murrell has brought us one step closer to a more complete understanding of this condition.

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Lumbopelvic mechanics

It has annoyed us for a long time when patients present stating that they have been having "core conditioning/core stabilisation/core strengthening/pelvic stabilisation", etc. It is also annoying to find the same terms used in peer reviewed scientific articles with the assumption that they mean something to the readers. Maybe they do to others, and we are missing something! We would like to get some discussion going on this and are happy to open the batting.

We think of lumbopelvic mechanics as three distinct groups:

(1) Intrapelvic stability

(2) Peripelvic stability

(3) Functional stability.

Intrapelvic stability

This is dependent on the transversus abdominis contracting with intact posterior sacroiliac joint ligaments. The anatomy of the transversus abdominis is such that it has a major origin off the iliac crest and inguinal ligament/conjoint tendon to insert into the linea alba. Therefore it and the pelvic floor are the only muscles that give direct closure across the sacroiliac joint. The long lever arm involved gives it great mechanical advantage, as long as the sacroiliac joint ligaments are intact.[1]

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Problems arise from two mechanisms.

(1) Loss of the active structures that provide intrinsic pelvic closure (the transversus abdominis and the pelvic floor muscles) because of

- inhibition of the transversus abdominis caused by first onset low back pain[2];
- pain inhibition through joint inflammation: "osteitis pubis", hip joint pathology, and sacroiliac/lumbar spine joint dysfunction;
- a tear of the conjoint tendon/inguinal ligament disrupting the origin of the transversus abdominis;
- past abdominal surgery inhibiting contraction or affecting the nerve supply to the transversus;
- tearing of the pelvic floor muscles during child birth;
- weakness of the pelvic floor muscles secondary to poor toilet habits.

(2) Loss of passive structures

- pelvic ligamentous laxity due to either body type (hypermobility) or external trauma (either single incident or prolonged postural loading), or hormone induced ligamentous laxity (pregnancy);
- laxity of the sacroiliac joint ligaments will cause loss of the closure moment at the sacroiliac joint. A very small loss of ligament strength here can have a profound effect on the closure moment. Is this how Vleming's posterior sling exercises work on post partum women? Further is this the mode of action of sclerotherapy of the sacroiliac joint ligaments?

Peripelvic stability

Once the pelvis is stable and we have a firm foundation, we can look at the pelvis reacting with the rest of the body.

Pelvis on hip joint (pelvifemoral control)

- Does the knee roll inwards as the subject single leg squats?
- Does the pelvis dip as the patient reaches single leg stance?
- Is the lack of pelvifemoral control a strength or timing problem around the hip joint (gluteus medius and gluteus maximus) or is it an inability to appropriately weight transfer on to that side because of hip, knee, or ankle problems, burnt out nerve root pathology, or just disuse of one side because of chronic injury.

Pelvis on lumbar (and thoracic) spine (lumbopelvic control)

- This is concerned with ability of the deep multifidus to contract to control the lumbar segments and the superficial multifidus to orientate the spine on the pelvis.[3]

Functional stability

Once the muscle strength and activation patterns are in place to allow force transfer through the pelvis, then linking these activities into normal activities and actions and conditioning the lumbopelvic complex can take place. This is really just an "on-field" extension of peripelvic stability. Once the athlete has all the necessary components to hold the pelvis stable on the femur and lumbar spine, can they coordinate that into their particular sporting or every day activity?

This type of stability is more concerned with technique, coaching, and video analysis. Also, as conditioning will no doubt affect the fatigue status of the athlete, high level physical conditioning allows the athlete to maintain a stable pelvis without physical fatigue, hence appropriate strength/endurance and power training is applicable to the type of athletic activity and the stage of the athletic season.

Most gym based strength, conditioning coaching, and fitness programmes fall into this category. However, it is our belief that, if the intrapelvic and peripelvic problems are not addressed initially, that is where these patients break down. Conversely, if inadequate conditioning is performed before return to sport/competition, then the athlete will break down, as there is no transmission of their rehabilitation on to the field of play.

People present at various points along the continuum of disease. Some lack functional stability, and some lack peripelvic strength and coordination, but have a stable pelvis. Others have a pelvis that swings in the breeze.

A recreational athlete with a sedentary occupation does not need the same level of intrapelvic strength and endurance as an Australian Football League onballer or elite soccer player. Yet someone doing a lot of vacuuming and weight bearing with poor ergonomics needs quite good intrapelvic and peripelvic strength without the need for the endurance of an elite athlete in these muscles.

We look forward to hearing the thoughts of others on this topic.

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New Faculty of Sport and Exercise Medicine in Ireland

The launch of the new Faculty of Sport and Exercise Medicine jointly by the Royal College of Physicians of Ireland and the Royal College of Surgeons in Ireland on Tuesday 19 November 2002 should not go unheralded. The first dean of the new faculty is Dr Michael G Molloy of Cork and rugby football fame. This is a highly significant event in the advancement of the discipline of sports medicine in these islands.

The development of any discipline has been shown to have four separate functions defined by McWhinney:[1]

- A unique field of action
- A defined body of knowledge
- An active research programme
- A rigorous training programme

Denis Pereira Gray[2] added to these four functions an important codicil, "In the development of any discipline, the literature is the key".

Because the *British Journal of Sports Medicine* has an international editorial board, which includes Professor Moira O'Brien of Trinity College Dublin amongst its ranks, it would seem appropriate that its editor should open a line of communication with this new faculty to unlock any obstruction in the path of this fledgling.

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Muscle dysmorphia in weightlifters

I would like to raise a concern with regard to the article "Muscle dysmorphia: a new syndrome in weightlifters" by Choi *et al*.[1] Although chronic obsessive behaviour related to fitness and weightlifting is genuinely a concern for both sexes, Choi *et al* fail, in my opinion, to present a valid argument for the construction of "muscle dysmorphia" as being a separate and distinct subcategory of body dysmorphic disorder. Inclusion criteria and procedures in the study by Choi *et al* were identical with those used in the study by Olivardia *et al*[2]—they used identical study populations. Both Choi *et al* and Olivardia *et al* claim that differences exist between their cases (weightlifters preoccupied with their body image, namely insufficient muscularity) and controls (weightlifters not preoccupied with their body image) with respect to body image and a variety of other characteristics. However, in both studies, the aim of which was to distinguish muscle dysmorphia as a distinct clinical entity, individuals were classified as cases or controls on the basis of their body image perception. It is therefore hardly a surprise to find that men who obsess about their body image and who perceive themselves to be insufficiently muscular differ with respect to their body image and their perception of being insufficiently muscular from men who do not have the perception of being insufficiently muscular. Olivardia *et al* discuss this limitation, stating that, although this tautology exists, there are other factors that separate the two groups (higher lifetime prevalence of mood and anxiety disorders, use of steroids and other drugs, and higher scores on many Eating disorders inventory subscales). However, this evidence alone is not sufficient to indicate that these characteristics distinguish a muscle dysmorphia construct, as it is possible that, within this target population (male weightlifters), the proposed muscle dysmorphia characteristics in fact distinguish the other observed disorders. I would advise readers to take the conclusion of Choi *et al* that " . . .muscle dysmorphia may be one negative consequence of physical exercise behaviour . . ." with extreme caution, as its validity as a distinct clinical entity has not yet been proven.

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1–3 October 2003, Leeds, UK
Musculoskeletal sonologists from the UK and mainland Europe will cover all aspects of musculoskeletal ultrasound in lectures and tutorials. The course is open to radiologists, radiographers, and clinicians with a US imaging interest.
Further details: Gill Bliss, MR Department, Clarendon Wing, Leeds General Infirmary, Great George Street, Leeds LS1 3EX. Tel: +44 (0)113 392 3768; fax: +44 (0)113 392 8241; email: gillian.bliss@leedsth.nhs.uk

Back Pain Prevention and Rehabilitation

5 October 2003, Glasgow, UK
A study day with Professor Stuart McGill.
Further details: Yvonne Gilbert, BASEMS Secretary, Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh EH8 9DW; email: y.gilbert@rcsed.ac.uk; tel: +44 (0)131 527 3409. Organised by BASEM Scotland

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, United Kingdom; email: michael.turner@LTA.org.uk

International XVII Puijo Symposium

25–28 June 2003, Kuopio, Finland
"Physical activity and Health—Gender Differences Across the Lifespan.
Further details: Ms Auli Korhonen, Project Secretary, Kuopio Research Institute of Exercise Medicine, Puijo Symposium Secretariat, Haapaniementie 16, 70100 Kuopio, Finland; tel: +358 17 288 4422; fax: +358 17 288 4488; email: puijo.symposium@uku.fi

12th International Biochemistry of Exercise Conference

13–16 July, Maastricht, the Netherlands
Further details: Marllen van Baak; email: m.vanbaak@hb.unimaas.nl; website: www.biochemex.org/IBEC

The Fifth International Conference on Sport, Leisure and Ergonomics

19–21 November 2003, Burton, Cheshire, UK
A three day conference in affiliation with the Ergonomics Society.
Further details: Congress Secretariat, Sport, Leisure and Ergonomics, Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Henry Cotton Campus, 15–21 Webster Street, Liverpool L3 2ET, UK; tel: +44 (0)151 231 4088; email: K.George@livjm.ac.uk

NOTES AND NEWS

Intercollegiate Academic Board of Sport and Exercise Medicine

Professor Donald Macleod has completed his four year term as Chairman of the Intercollegiate Academic Board of Sport and Exercise Medicine. Professor Charles Galasko has been elected by the IABSEM Board to replace him. Professor Macleod has also been replaced as the representative of the Royal College of Surgeons of Edinburgh on IABSEM by Professor Angus Wallace.

Winners of the annual BASEM Prizes

Dr Eileen Mackie (Clopidogrel inhibits platelet activation and exercise induced ischaemia in stable coronary artery disease) and Mrs Eleanor Curry (Role of exercise in multiple sclerosis) (joint winners).
The poster prize was won by Dr Stuart Reid (Injury patterns and injury prevention strategies in the winter sports population attending the English medical centre in Val D'Isere.

Diploma in Sport and Exercise Medicine for Great Britain and Ireland

Details for the above exam can be found on the Royal College of Surgeons of Edinburgh Website at http://www.rcsed.ac.uk alternative applicants can write to: The Royal College of Surgeons of Edinburgh, Eligibilities Section, Careers Information Services, 3 Hill Place, Edinburgh; tel: +44 (0)131 668 9222 *or* Mrs Yvonne Gilbert, Intercollegiate Academic Board for Sport and Exercise Medicine, Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh EH8 9DW; tel: +44 (0)131 527 3409; email: y.gilbert@rcsed.ac.uk

Intercollegiate Academic Board of Sport and Exercise Medicine Diploma Exam

The following were successful diplomates in the Intercollegiate Academic Board of Sport and Exercise Medicine Diploma Exam, the the two exams held in 2001 and 2002:

Dr Andrew I Adair
Dr Abimola Afolabi
Dr Sinead M Armstrong
Dr Terence J R Babwah
Dr Catriona E L Boyle
Dr Susan J Brick
Dr Lawrence J Conway
Dr Alan J Dawson
Mr Patrick D Dissmann
Dr Niall WA Elliott
Dr Christopher J Ellis
Dr Roger K Goulds
Dr Niall A Hogan
Dr James R Hopkinson
Mr Ananta K Jayanti
Dr Michelle Jeffrey
Mr S P Kale
Dr Arun Kumar
Dr Robert M MacFarlane
Dr Kaushal C Malhan
Dr Martin D McConaghy
Dr Lisa A McConnell
Dr Fergal T E McCourt
Dr Ronan M McKeown
Dr Michael G McMullan
Dr Steven R McNally
Dr Paul J Moroney
Dr Leonard D M Nokes
Dr Nanda K G Pillai
Dr Jonathan D Rees
Dr Duncal A Reid
Dr Cristyn C G Rhys-Dillon
Dr Martin O Rochford
Dr Hungerford A T Rowley
Dr Shaun A Sexton
Dr Jason E Smith

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Dr Aravinthan Suppiah
Dr James A Thomas

For further information contact: Mrs Yvonne Gilbert, Administrative Secretary, Intercollegiate Academic Board of Sport and Exercise Medicine, Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh EH8 9DW. Tel: +44 (0)131 527 3409; fax: +44 (0)131 527 3408; email: y.gilbert@rcsed.ac.uk

www.basem.co.uk

The British Association of Soprt and Exercise Medicine has launched its new website—www.basem.co.uk. The site provides information about the educational opportunities in sport and exercise medicine and advice to those wishing to become involved in this area.

Interested in Sports Medicine? Gain a higher degree from Australia's leading University

The Centre for Sports Medicine Research and Education is a multidisciplinary Centre located in the Faculty of Medicine, Dentistry and Health Sciences at the University of Melbourne, Australia. It combines world-class researchers and clinicians working in the area of sports medicine.

Research Higher Degrees

The Centre offers Doctor of Philosophy (PhD), Master of Sports Medicine, Master of Physiotherapy, Master of Science, and Doctor of Medicine degrees. These are available to graduates of health and medical science courses such as physical therapy, medicine and human movement.

Sports medicine at the University of New South Wales

Masters of Sports Medicine

You don't have to leave your practice:

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Web site: www.med.unsw.edu.au/sportsmed

NCPAD NEWS

A monthly publication of the National Center on Physical Activity and Disability. NCPAD is the leading source for information about organisations, programmes, and facilities nationwide providing accessible physical activity and recreation. NCPAD also has a large and growing online library of fact sheets, monographs, and contact information on physical activity and recreation for people with disabilities.
Sign up for this free monthly electronic newsletter by sending an email to: Listserv@listserv.uic.edu, with this message in the body of the e-mail: SUBSCRIBE NCPAD-NEWS yourfirstname yourlastname. If you have any difficulty, you can also sign up for the newsletter by going to http://www.ncpad.org/signup

Study Sports Physiotherapy in Australia's sporting capital at The University of Melbourne

Qualified physiotherapists may now apply for the Master of Physiotherapy by Coursework (Sports Physiotherapy), the Postgraduate Certificate in Physiotherapy (Sports Physiotherapy of the Spine, Pelvis and Lower Limb) or the Postgraduate Certificate in Physiotherapy (Sports Physiotherapy of the Spine, Shoulder and Upper Limb).
The School of Physiotherapy at the University of Melbourne now has approval for these courses and applications are open to international students for full time study.

- Master of Physiotherapy by Coursework (Sports Physiotherapy) NOW CLOSED.
- Postgraduate Certificate in Physiotherapy (Sports Physiotherapy of the Spine, Pelvis and Lower Limb) NOW CLOSED.
- Applications for the Postgraduate Certificate in Physiotherapy (Sports Physiotherapy of the Spine, Shoulder and Upper Limb) close **1 April 2003**.

Please check the website for updates and information about the courses: www.physioth.unimelb.edu.au/postgrad.html

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