New Zealand Journal of
SPORTS MEDICINE

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Included in this Issue

Interview with
David Gerrard

Invited Commentary
The Central Governor of Exercise Performance: Fact or Fiction?

Cardiology Conundrums
T-Wave Inversion in Athletes

NZ JOURNAL OF SPORTS MEDICINE

Instructions to Authors

GENERAL

The New Zealand Journal of Sports Medicine is the official journal of Sports Medicine New Zealand, publishing material relevant to sports medicine and related disciplines. The New Zealand Journal of Sports Medicine welcomes submissions of original manuscripts from both members and non-members of Sports Medicine New Zealand in the following areas: *sports medicine, sports physiotherapy, clinically relevant sports science, rehabilitation, coaching issues as they relate to sports medicine, exercise prescription and training, sports chiropractic, sports podiatry, and sports psychology.*

Manuscripts must not have been published elsewhere except in abstract form. Manuscripts will be reviewed by the editorial board and/or experts in the field of interest. Submissions are in the following categories:

a	Original Research	b	Case Reports	c	Review Articles	d	Editorials
e	Letters to the Editor	f	Sports Medicine Pearls	g	Policy Statements		

SUBMISSION DETAILS

Manuscripts are to be sent to The Editor, New Zealand Journal of Sports Medicine, Sports Medicine New Zealand, PO Box 6398, Dunedin, New Zealand. Telephone +64 3 477 7887, facsimile +64 3 477 7882, email smnznat@xtra.co.nz. The manuscript text should be submitted in MS Word format (if using Vista please save the file as Office 2003 compatible, ie, .doc. Please do not use styles for headings, etc, ensure your manuscript is in the style 'Normal'. (For submission of graphics, please see Tables, Illustrations, Figures, Photos below). The manuscript may be sent via email however one hard copy must also be submitted, including any tables, figures, and photographs. Manuscripts should be double-spaced with wide margins. Each page should be numbered. The manuscript should include the following: *title page; structured abstract (followed by five key words); introduction; methods; results; discussions; conclusion; acknowledgements; references; tables; figures.*

Case reports are to have no more than two figures and are not to include an abstract. There should be no more than 12 references for a case report. The structure of a case report is as follows: *introduction; case report; discussion.*

Structured abstracts are to be no longer than 300 words and should use the following subheadings: *aim; study design; setting; participants/subjects; interventions; outcome measures; results; conclusions.*

Abstracts for review articles should use the following headings: *aim; data sources; study selection; data extraction; data symphysis; conclusions.*

The title page should include the title of the article and a running title not exceeding 45 letters and spaces, authors' names (first name, middle initials, last name), degrees, affiliations with institutes, contact details for the corresponding author (to include name, address, telephone, fax, and email).

The standard for spelling is to be in accordance with the Oxford Dictionary.

TABLES, ILLUSTRATIONS, FIGURES, PHOTOS

One hard copy of tables, illustrations, figures, photos, etc, must be submitted. Tables should be included on a separate sheet rather than in the body of the text. Identify all illustrations with the manuscript title, name of author, figure number, and. if necessary, identification of the top of the image, on the back of the illustration in pencil. All markings should be removed from x-rays before photographing. Please do not produce graphics in Microsoft Word. Graphics should be supplied in TIF, EPS or PDF format, preferably at a resolution of no less than 300 dpi.

STYLE

Drug names: generic only are to be used. Abbreviations: the *American Medical Association Manual of Style* (9th edition 1998) (published by the American Medical Association, 535 North Dearborn St, Chicago, IL 60610, USA) is to be used for abbreviation style. The *List of Journals Indexed in Index Medicus* (Superintendent of Documents, US Government Printing Office, Washington, DC 20402, USA, DHEW Publication No. (NIH) 83-267;ISSN0093-3821) is to be used for abbreviations for journal titles.

REFERENCES

References are to be numbered in alphabetical order. Names of journals should be abbreviated according to the format approved by Index Medicus. All references listed must be sited in the text. Journal titles that are single words only should be spelt out in full. All authors must be listed. Pagination should be inclusive. Examples of the appropriate formatting of references are given below:

Journal Article:	Speedy D B, Kelly M, O'Brien M. The effect of pre-exercise feeding on endurance exercise performance. *NZ J Sports Med* 1998; **26**:34-37.
Book:	McRae R. *Practical fracture treatment.* Edinburgh; Churchill Livingstone, 1989.
Chapter in Book:	Figoni S F. Spinal cord injury. In, Wikgren S (ed.): *ACSM's exercise management for persons with chronic diseases and disabilities.* Champaign: Human Kinetics, 1997; 175:179.

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SPORTS MEDICINE
NEW ZEALAND

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EDITORIAL

When Chris Milne took the Editorial role in 2009, he paid tribute to the Editors before him, comprising the surprisingly short list of Noel Roydhouse, Dale Speedy and Peter Milburn. I would like to add my tributes, but add recognition of the huge influence that Chris has had on the journal, extending well beyond his recent stint as Editor. It doesn't take much looking through back issues of the NZJSM to see that Chris has been a constant source of information, inspiration and intellect. Chris has provided vast amounts of material for the journal, and I would like to voice my appreciation for his efforts over many years. I look forward to his ongoing insight and involvement as the journal moves forward.

On taking on the role of Editor of the NZJSM, I recognise that one of the major differences between myself and my predecessors in this role is that most Sports Medicine NZ members probably have no idea who I am. This may reflect my working career, which to date has predominantly been offshore; what started as a windsurfing sojourn to Perth, Western Australia after completing physical education and medicine at Otago University and never wanting to open a book again, turned into an extended adventure through sports medicine in Australia, the United Kingdom and now Qatar. I hope that I can add value to the journal over the next phase of its development.

The developmental direction of the journal has clearly been a longstanding and hot topic. Previous editorial titles including "Whither the Journal" (Chris Milne 1999), "The Way Forward" (Dale Speedy 2002) and "A Call to Arms" (Peter Milburn 2006) all addressed this issue. At its outset, the journal had clinical sports medicine as its core (Milburn 2006), with the goal of providing SMNZ members with a high quality, useful and relevant resource. These goals remain unchanged, but with the passage of time the number of challenges that the journal has faced in meeting those goals has increased. Information availability via electronic resources was recognised as a threat by Chris Milne as long ago as 1999 ("Whither the Journal" – and addressed by Dr Iain Bell "Sports Medicine and the Internet 2000") and this remains a challenge for the NZJSM, to which we must continue to adapt. In 2002, an editorial debate presented, essentially as diametrically opposing alternatives, the development of a high quality medline listed academic journal ("The Way Forward" 2002 Dale Speedy) and the maintenance of a clinically useful journal for the membership ("Medline Listing" 2002 Peter Sperryn). This debate was rejuvenated in 2007 ("Who do we Serve?" Peter Milburn 2007). My tendency is to side with Gregory Kolt (Summer 2002) when he suggests that a medline listed journal can still have relevance for the membership, but I recognise that the key determinant of both of these objectives is the consistent inclusion of quality content in the journal. Hence our goal for the journal is to encourage quality content, and that will, as always, depend on you the membership. We have a number of initiatives that we would like to establish, re-establish and re-create, in order to facilitate a regular flow of high quality manuscripts. I hope that you will see the evolution of this over the next few editions.

In this edition, you will see the continuation of the "Best of British" series, which first appeared in 2005. We have also consolidated student and post-graduate student manuscripts into a "Post-grad Ponderings" reflecting the importance of encouraging early publishing careers. I hope that as we move forward you will take time from your clinical practices to both read the journal and consider submitting a manuscript.

Evidence Based Medicine

In this journal's "Best of British", Chris Milne chose the article by Paul McCrory[1] and colleagues as his pick of articles. This BJSM editorial, entitled "Sports and exercise medicine – specialists or snake oil salesmen?" takes a big swing with a long handled bat at sports medicine practitioners who reportedly utilise non-evidence based techniques in their practice. I entirely agree with Chris that this article is worth reading, pondering, and pondering some more. Being one of the co-authors on the manuscript that this article criticises[2], this choice by Chris has given me a great opportunity for further rebuttal[3] at the size of the swing that was taken.

Evidence based medicine is frequently associated with level one or two studies, providing "definitive" evidence or otherwise for a treatment, technique or intervention. However, it is important to remember that while the least substantiated form of evidence, expert opinion forms a recognised part of the clinical evidence base. For proponents of evidence based medicine (of which I am one), it is easy to fall back on the lack of evidence from high quality, double blind randomised controlled trials, and exclude a treatment option on this basis. This appears to have been the approach that McCrory and colleagues have taken, claiming the scientific and moral high ground on this issue. However, in 2004, even Chris Milne himself decried the increasing reliance on evidence based medicine and highlighted the need to "keep honing your critical faculties and have faith in your own gut instincts based on experience".

I agree with McCrory and colleagues that sports medicine practitioners whom suggest that the onus is on science to disprove any practice (which experts may suggest work), rather than the burden of proof being on those who use it to substantiate its use, are working outside the acceptable norm. However, I moderate this by recognising that without practitioners who are prepared to think outside the square, go back to basic principles and explore novel treatment options (within the legal limitations we have) based on those principles, it is unlikely that we would ever

move forward in any field of medicine. Remarkably, as you all know, most of what we do in sports medicine lacks a substantial evidence base and the onus is on us all to constantly be challenging this status quo.

I believe that to ignore the evidence of experienced practitioners and years of accumulated experience would potentially be to our (and our patients') great cost. That experience should be harvested, evaluated and scientifically challenged by those with the skills and abilities to do so. That is the benefit of enquiry, interdisciplinary shared approaches and supportive communication; this is what we should be promoting within sports medicine.

1 Franklyn-Millller A, Etherington J, McCrory P. Sports and exercise medicine—specialists or snake oil salesmen? *British Journal of Sports Medicine* 2009; **45**(2):83-84.
2 Orchard J, Best TM, Mueller-Wohlfart H-W, *et al*. The early management of muscle strains in the elite athlete: best practice in a world with a limited evidence basis. *British Journal of Sports Medicine* 2008; **42**:158-159.
3 Orchard J, Best T, Hunter G, Hamilton B. Response to McCrory et al. "Sports and exercise medicine - new specialists or snake oill salesmen?" *British Journal of Sports Medicine* 2009;bjsm.2009.068999Published Online First: 29 November 2009 doi:10.1136/bjsm.2009.068999.

Dr Bruce Hamilton
Editor

A colleague recently suggested that all research is important. In the discussion that followed I proposed there was increasing amounts of research that may have little clinical relevance, to which he replied that without each grain of sand you would not have a beach! A nice analogy which undoubtedly has merit, but I still get the impression there are large parts of the beach that many clinicians involved in sports medicine would never bother visiting. Perhaps the beach has just got too big, built up by forces other than those ensuring high quality, clinically relevant development.

A common theme in all the discussions I've heard about this journal over the years has been the wish to keep it relevant to the clinicians – especially local clinicians. Having been a musculoskeletal/sports physiotherapist for over 10 years I offered to assist with the journal in the hope of helping to continue that theme – easy to say, harder to do. As a full time academic I now have an obligation to undertake research - or should I say publish! Hopefully my time as a clinician and ongoing contact with clinically active colleagues will help maintain the clinical relevance of this research. It's always frustrating to attend conferences where the discussion between the researchers and clinicians is all too often a confused conversation. Clinicians want the take home message for clients on Monday – often they don't get it. This process could be assisted by more collaboration between clinicians and researchers and/or contributions to the journal from clinicians (such as case studies). You may all feel that you are too busy but if you have an idea or you want help to get something done then feel free to let me know. For most there is the added bonus of professional development points to be gained.

Research in sports medicine is important, especially as the call for evidence based practice continues to grow in all related disciplines. However in these times of access to limitless sources off online information the poor old clinician may struggle to find the 'golden sand'. To my way of thinking there is a major onus on the researchers, reviewers and editors to publish quality findings in a way that means something to clinicians. Clinicians should be able to read information in well established journals expecting a high quality of methodology and results that translate into practical applications.

At the risk of getting a little technical one example where researchers could assist is by avoiding reliance on $p < 0.05$ when deciding if outcomes are useful or not. Many have suggested it is better to consider the size of the effect (and its confidence interval) relative to a justified smallest clinically important effect.[1] This allows inferences to be made as to the likelihood the outcome of a study is clinically/practically useful. Recently we made the following statement in a review on the effect of kinesio tape on pain – "the effect of kinesio tape is trivial when compared to established thresholds for meaningful change",[2] this has meaning to a clinician. Greater use of this style of reporting results would help clinicians to find the sunny patch on the beach – or at least give then confidence they are swimming between the flags!

1 Hopkins W G, Marshall S W, Batterham A M, & Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports Exercise*, 2009; **41**(1), 3-13.
2 William S, Whatman, C, Hume P A, & Sheerin K. Kinesio taping in treatment and prevention of sports injuries: A meta-analysis of the evidence for its effectiveness. *Sports Medicine*, 2011, In Review.

Chris Whatman
Assistant Editor

Associate Professor David Gerrard

OBE CNZM MBChB(Otago) FACSP

David Gerrard, academic, Olympian and Commonwealth swimming gold medallist, is an Associate Professor at the University of Otago where he is Director of Development and Alumni Relations in the Office of the Vice Chancellor.

David is Chair Emeritus of Drug Free Sport New Zealand, the New Zealand Drowning Prevention Council, and an immediate past Member of the Medical Committee of the World Anti-Doping Agency (WADA) where he chaired the WADA Expert Group on Therapeutic Use Exemption.

His publications and areas of medical research include paediatric sports medicine, injury prevention, bioethics and anti-doping strategies in sport.

He was an Olympic Team Physician in 1984 and 1988, Chef de Mission of the New Zealand Olympic Team to Atlanta (1996) and Medical Commissioner in Sydney (2000), Athens (2004) and Beijing (2008).

David remains an avid surfer, fancier of Central Otago wines, student of calligraphy, reader of historic novels and devotee of the Highlanders Rugby Team.

Since 1999 when WADA took over responsibility for anti-doping policy and implementation we have seen many changes; how do you feel that WADA has done over this time?

In 1999, WADA set itself the task of bringing consistency to the world of anti-doping. To be truthful I believe they have made huge strides and although the war is still being waged, several critical battles have been won.

Notable successes include consistency in laboratory accreditation, the prohibited list, application of an informed TUE process and the enforcement of harmonised sanctions across International Federations. These areas are under consistent review and with a few notable exceptions, IFs, NOCs and Governments are all fully compliant with the WADA Code and the UNESCO Convention against the misuse of drugs in sport.

Over and above catching athletes who may be deliberately cheating, do you believe that WADA, sports medicine, sport science and the legal field have been able impact on the desire of athletes to cheat in order to win?

I sense that these organisations and professional disciplines have been successful in engendering a common ethical approach to the misuse of drugs in sport. This is evident when athletes provide feedback on how cheating should be dealt with and how regulations such as the "whereabouts" requirements should be implemented. It is right and proper for peer review and opinion to be sought on all issues of anti-doping reform. The athletes themselves deliver a consistent, strong message condemning all forms of cheating - sadly this is offset by a small minority of underachieving competitors who are seduced by financial return or malpractising health professionals or coaches.

Where do you believe the future challenges lie for WADA – is gene doping the future threat to sport?

Clearly gene doping represents unchartered waters. However I am informed that the potential exploitation of this technology is not only possible in humans but is, in fact, already demonstrated in the laboratory animal model. Using viral vectors, genes expressing traits desirable for elite athleticism have already been introduced and shown to benefit their recipients. Bigger muscles, enhanced EPO production and vascular proliferation are among the advantages endowed by gene transfer technology.

WADA has recognised this potential and has enlisted the advice of some of the world's most celebrated genetic researchers, to anticipate how this form of performance enhancement may present, and just as important, how it may be detected.

Even post the 1998 Tour de France, Cycling as a sport has been rocked by numerous drug "scandals". Do you believe that international cycling has finally cleaned up its act?

The reputation of cycling has definitely taken a hammering over the past couple of decades with numerous examples of drug misuse. To be fair, I believe they did themselves no good by engaging in a number of attempts at "cover-up". In the top professional ranks, Landis, Cantador and of course Armstrong have either received sanctions or are linked with doping scandals. There has been clear evidence that the drugs issue in cycling has involved a sophisticated network of collaboration implicating support staff, medical advisors and coaches. However there are signs that the tide is changing and the ICU is now sitting at the WADA table and making every attempt to improve the reputation of their sport. The imminent US Federal case against Lance Armstrong will unfortunately air some dirty laundry, but the public deserves to know the truth in this longstanding debate. So watch this space.

There have been repeated changes to the WADA prohibited list when it comes to the prohibition of asthma medications. Currently, all B2-Agonists except for salbutamol require a full TUE. Is this just based on a lack of evidence for the other generic B-agnoists, or is there something specifically different about salbutamol that warrants this specific treatment?

The whole debate over the place of beta-2 agonists on the prohibited list has consumed a disproportinate amount of time and energy. There are many who contend that the ergogenic effect of these agents is highly questionable when taken in appropriate therapeutic doses. Clenbuterol used systemically is an obvious exception. However the nub of the matter to my mind relates to two issues. First the recognised high prevalence of clinically proven, exercise-induced bronchospasm in athletes and second, the fact that we appear to be focussing on the wrong group of "potential" sports drug misusers. Does this group represent the real drug "cheats"? I suggest we should be investing our energy in monitoring the misuse of peptide hormones and other heavy duty agents with far greater potential for ergogenesis.

However, I qualify these comments by saying that there is a significant duty of care and professional responsibility to be borne by all physicians treating athletes with asthma in any one of its variant forms. An accurate diagnosis based on history, clinical findings and confirmatory laboratory investigations must at all times underpin the diagnosis. Critical to the successful management of asthma is patient education and athletes are no different to any other patients in this regard.

Cardiac screening of athletes is gaining increasing profile and academic attention around the world, with great debate surrounding the use of ECG and Echocardiography. Part of this argument becomes a cost – benefit argument, which is particularly relevant in a small country like New Zealand. In Italy, all participants in sport are required to have a cardiac clearance. Do you feel in New Zealand that we are doing enough to protect our elite and non-elite athletes from potential underlying cardiac anomalies? What would you like to see us doing?

Compulsory pre-participation screening of athletes remains a contentious and highly expensive exercise, cost-prohibitive in the New Zealand setting. I personally favour the system whereby athletes who are identified in our elite "carded" pool become the clinical responsibility of the Medical Director for that sport. At that point it ought to be decided on the basis of history and clinical examination whether "cardiac screening" is an appropriate or necessary option. To undertake extensive screening prior to all or any sports participation is beyond our resources and is of dubious benefit in terms of detecting any undeclared cardiac pathology. I am aware of the Italian mortality data that relate to the implementation of pre-participation screening and find the cost-benefit argument unconvincing.

Platelet Rich Plasma (PRP) appears to be increasing in popularity, despite limited clinical evidence to support its use. Over the years sports medicine has had many "fads"; do you think this is another, or do you think that this may eventually turn out to be a useful tool for the musculoskeletal specialist?

It would appear that the jury is still out on the real therapeutic benefits of PRP. The WADA List Committee was of the mind to withdraw this intervention from the list of prohibited practises and I supported this move. There was no convincing scientific evidence that PRP met any of the criteria for inclusion on the Prohibited List and at the end of the day it continues to be applied by many physicians in the management of a range of musculoskeletal disorders with varying rates of success.

Many years ago, in this journal (I think it was a letter to the editor!) you wrote about the use of non-steroidal anti-inflammatory drugs in sports medicine. Over the last 10 years there has been increasing evidence as to

the impact that NSAIDs may have on athletes bodies. What is your feeling about the use of NSAIDs in elite and recreational athletes and your impression of the current utilization patterns of clinicians?

Yes I did comment on the cavalier use of NSAIDs, and still contend that the potential of these agents is not well understood by many clinicians. And when I hear anecdotal reports of team physiotherapists and other support staff handing out NSAIDs to athletes on scant evidence I am particularly concerned. NSAIDs have a potent anti-prostaglandin effect that has the potential, amongst other things to reduce renal vascular perfusion and the rate of glomerular filtration. In a stressed, dehydrated athlete already imposing insult upon his kidneys, the added effect of NSAID administration is a potential for life-threatening consequences. There have been several documented cases of acute renal failure in athletes mediated through the diminished vasodilatory effect of prostaglandins. This potential also exists with the use of selective COX-2 inhibitors and over-the-counter agents heralded as "safe" alternatives. The bottom line is that while NSAIDs have a place in the acute management of musculoskeletal injury, they should be considered along with primary anti-inflammatory measures and physical therapy. Their use ought to be under the direction of a physician and avoided where there may be added renal stress accentuated by physical insult through sport.

Sports medicine has many elements (elite athlete care, public health, research, recreational athlete, musculoskeletal, medial elements), as well as being truly multi-disciplinary, which is what attracts many practitioners to the field. You have witnessed a massive development of sports medicine over the last 20 years, but where do you think we will be in 20 years' time?

I have often reminded students that sports medicine should not be linked only with the popular image of doctors tending the needs of elite, highly-paid professional athletes whose demands are often inconsistent with "normal" clinical practice. Sports medicine is a team game that applies equally to issues of public health and generic exercise prescription for the wider population. Sport and exercise medicine is an appropriate reflection of the scope of our work that has developed over the last 20 years. What the future holds is anyone's guess but I would favour a stronger relationship with public health as it specifically relates to children and pre-adolescence. We need to establish early patterns of physical activity and healthy eating and address public health issues such as smoking and alcohol misuse in children. Obviously the clinical implications of physical activity for an aging population is becoming another issue of increasing economic significance. I am sure that these will be important challenges for the sport and exercise physicians of the future.

Achieving success on the international stage has always been difficult, but seems to be increasingly challenging with vast resources being mobilised behind athletes in some countries. What do you see as the challenges to NZ's success internationally in the future, and how can sports medicine contribute?

It is an overused expression that Kiwi athletes "punch well above their weight" on the international scene. However hackneyed, I hold with this statement and consider New Zealand – a tiny country with a population of less than that of the state of NSW to continue to produce some of the World's most outstanding athletes.

I am confident that this trend will continue, with the proviso that we aren't seduced into spreading ourselves too thinly. At the risk of offending supporters of certain sporting codes, we are unlikely to ever produce an Olympic gold medallist in gymnastics or platform diving or a World Championship team in ice hockey or handball. But we continue to excell in rowing, equestrian, cycling, athletics and yachting and team sports such as rugby, netball and possibly cricket.

I strongly advocate the focussed use of our limited financial and human resources to the events and sports in which we traditionally excel. Our teams to Olympic and Commonwealth Games should be restricted to those with a proven track record and not as the opportunity to provide "experience" for a future star or simply to make up the numbers. Potential may be tested at regional events, World Cups and other international events that have become so common-place in almost every sport. As a fledgling swimmer in the 1960s I competed in one Commonwealth Games before heading to the Olympics two years later without any intervening overseas experience. It was only at my final Commonwealth Games that I felt remotely familiar with the international competitive arena. Today, swimmers have a schedule of international events placed before them each year and ample opportunity to gain experience and to demonstrate their capabilities.

Finally we must recognise that sport is part of our social fabric in this country and we are obligated to provide talented youngsters with the opportunity to develop as people as well as athletes. We have been blessed with a recent crop of outstanding sports ambassadors in this country – athletes of the ilk of Sarah Ulmer, Hamish

Carter, Georgina and Caroline Evers-Swindells, Valerie Adams, Daniel Carter and Richie McCaw. Add those to outstanding names of the past like Sir Brian Lochore, Sir Murray Halberg and Yvette Williams and we quickly realise that the best athletes are also amongst the best people.

Undergraduate training in sports medicine used to be non-existent. Has this changed now, and what would you like to see in the future?

Undergraduate sports medicine training still remains essentially non-existant. Medical school curricula give little lip service to the topic as such but ironically use elements of sport and exercise medicine to teach subjects such as orthopaedics, musculoskeletal medicine, epidemiology and public health issues including asthma, diabetes, coronary heart disease and obesity. I would like to see more sport and exercise-related cases being introduced to undergraduate students in medicine to reflect the broad implications of our specialist discipline.

In New Zealand, post-graduate education in sports medicine was pioneered in Auckland and Otago Universities through diploma studies that continue to provide general medical practitioners with the basis for their interest in the subject and a qualification required by almost every sport seeking to appoint a medical director. Specialisation in sport and exercise medicine and vocational registration in New Zealand is obtained through Fellowship of the Australasian College, still regarded enviously by many other countries.

Increasing numbers of physiotherapists are completing PhD's in clinical research, adding greatly to the evidence base in sports medicine practice. Unfortunately, limited numbers of physicians are involved in research on a regular basis, which is required if we are to move the profession forward. Do you have any observations or thoughts on how to increase our physician involvement in research?

There is a College requirement for research necessary to obtain Fellowship and this is consistent with other medical colleges. However very few clinicians find time to continue their research interests. There is a wealth of research opportunity and physicians so inclined are encouraged to collaborate with groups existing at recognised tertiary institutions.

Dunedin is a great place to train in medicine and sports medicine (Editor's parochialism!) - what are some of the new developments going on down there to keep it that way?!

Naturally I am about to declare a marked bias for the University of Otago and this will come as little surprise to those who know me.

Otago still boasts a unique collaboration of kindred academic schools and departments that provide the scope of teaching in sport and exercise medicine. These include schools of medicine, physiotherapy, physical education and pharmacy as well as departments of human nutrition and clinical psychology and an injury prevention research unit. The University is complemented by sports facilities including the Moana Pool complex, the Caledonian athletics track, field hockey turf and the new Dunedin Stadium all located within 10 minutes of the main campus. The Dunedin Stadium, to be opened for Rugby World Cup, boasts the only fully covered, natural grass stadium in the world.

Many thanks.

Dr Bruce Hamilton
Editor

Welcome back to this regular column. Although I have passed on the editorship of the Journal to my colleague Bruce Hamilton, I will continue to contribute this column. It seems to have captured the interest of readers and I believe it is a worthwhile service to our members, so here goes.

February

The February issue of BJSM contained a number of useful articles. One of the editorials related to revalidation of sport and exercise medicine from a UK perspective. This is worth consulting for those of us who have concerns regarding audit and reaccreditation to get an external perspective on the issue.

The second editorial was entitled 'High performance sports medicine: an ancient but evolving field'. Two of the leading UK-based protagonists, Cathy Speed and Rod Jaques, explain that high performance sports medicine started in the era of Hippocrates, who described the use of a medical approach is to optimise the effects of and recovery from training, but then lay dormant until the 19th Century, re-emerging when a new era of formal competitive sport began. They define high performance sports medicine as the provision of an integrated model of medical care of the high performance athlete. It focuses on the maintenance and optimisation of health, wellbeing and competition sporting performance under circumstances of high physiological and psychological stress. They champion the interdisciplinary model and this is labelled the Integrative Medicine and Science Support Team (IMAST). They describe the role of the physician as one of medical guardianship and go on to explain that the working environments can vary from the conventional clinical setting to the side of a pitch or training venue, a hotel room, or indeed by distances through telemedicine approaches. As well as caring for the athletes, the practitioner will have to provide care for the support staff; this leads to an occupational health role. They recommend further research to document responses to training programmes that accurately replicate the demands of high performance sport so that these can be integrated into routine clinical practice. This article should be compulsory reading for all of our practitioners who have significant contact with elite athletes, especially those allied to the New Zealand Academy of Sport.

My most recommended article in this issue is entitled 'Sports and exercise medicine – specialists or snake oil salesmen?' Written by leading practitioners including Paul McCrory, the article concentrates on the promotion of Actovegin, a deproteinised hemodialysate manufactured from calf blood plus local anaesthetic, and Traumeel, a compound containing 12 botanical substances. Comments from UK-based physicians suggest that they are under pressure by the athlete to use Traumeel S and that the onus was on "science" to disprove this therapy rather than the burden of proof being on those who seek to use it. As the authors point out, this is wholly at odds with the practice of medicine and other medical specialties. They go on to say that we must resist any pressures which may compromise our standing as rational, safe evidence-based clinicians worthy of specialist status. To do any less compromises our standing as a specialty. To all this I would say "hear hear". There are far too many substances being consumed by suggestible athletes with little or no evidence base for their use and potentially harmful side effects. It is our role as responsible clinicians to be true guardians of and advocates for athlete health and wellbeing and we surrender this role at our peril.

The third article worthy of comment is entitled 'Contribution of free play towards physical activity guidelines for New Zealand primary school children aged 7-9 years'. The authors found that no children met the recommended 60 minutes of moderate to vigorous physical activity each day. Not surprisingly, they found children were more active during school playtime compared to after school and weekends. These are characteristics of the screen generation, be it PlayStation, television, Facebook or a mobile phone. Even the most coordinated of us would find it difficult to run at four minute kilometre pace while texting or looking at a screen without colliding with others.

Another article of New Zealand authorship in the same issue was entitled 'Spreading the word on sports concussion: citation analysis of summary and agreement, position and consensus statements on sports concussion'. Arguably one of the major achievements of sports medicine in the last 20 years has been the publication of international consensus guidelines following the meetings held in Vienna in 2000, Prague in 2005 and Zurich in 2008. These plus other consensus guidelines from ACSM published in 2005 have been widely distributed, with co-publications in many journals. This has led to increasing penetration of their contents throughout the academic and clinical community as evidenced by the rising number of citations. Sridhar Alla, John Sullivan et al from the University of Otago School of Physiotherapy were responsible for collating this paper.

Finally in the February issue, there was an excellent pictorial essay on the spectrum of knee extensor mechanism injuries. This was collated by Bruce Forster and colleagues from the Department of Radiology in the University of British Columbia, Vancouver. The authors beautifully outline the spectrum of knee extensor mechanism injuries with well

labelled diagrams to emphasise key points. They use x-ray and MRI imaging extensively and this article is highly commended for all clinicians who deal with knee injuries, which is most of us.

March

The March issue contained a useful editorial on physical activity as medicine, with the second half of the title being 'Time to translate evidence into clinical practice'. The article uses New Zealand's green prescription as one of the exemplars of this process. This is basically a call to arms by senior Swedish researchers from the world-renowned Karolinska Institute to incorporate exercise into our daily routine of prescribing. The definitive reference work is 'Physical activity in the prevention and treatment of disease' and was originally published in Swedish. However, a recently translated English version is now available and is the most comprehensive guide published to date. It consists of 47 chapters including 33 on specific aspects of different organ system disease and how treatment can be helped with the aid of physical activity.

Later in the same issue is an article on atrial fibrillation in endurance trained athletes. This is written by Paul Thompson, one of the world experts in the area, plus co-authors, and summarises the best available current knowledge. In essence, management of atrial fibrillation in athletes follows similar principles to those used to manage atrial fibrillation in the general population. The authors recommend determining the frequency of symptomatic AF episodes via an event recorder. Their first-line treatment would include pharmacological suppression with Flecainide 50mg twice or three times daily. With persistent atrial fibrillation anticoagulation may be required. They mention radiofrequency ablation as an additional attractive treatment approach and our experience with this intervention is growing all the time; I suspect that within a few years it will assume a significantly greater place in management. The main thing to be aware of with highly competitive athletes is that, if at all possible, they should be maintained in sinus rhythm. The mere presence of atrial fibrillation causes a drop off in maximal cardiac output and this is clearly unacceptable to athletes in sports which require a high level of aerobic fitness.

Later in the same issue there is a continuation of the excellent series on supplements. This particular article discusses glutathione, glutamate and glutamine. Its authors include the renowned Eric Newsholme of Merton College, Oxford, UK, who sadly died recently. He worked with Hans Krebs in his early days and was among the first to suggest that glutamine deficiency may be a limiting factor in the ability to train hard. Glutamine supplementation has been studied by several authors with mixed results. Some have found an ergogenic effect but most have found no benefit. The current consensus would suggest that glutamine is effective in decreasing the self-reported incidence of upper respiratory tract illness but no specific evidence of an effect on any particular aspect of the immune system has been proven thus far.

April Supplement

The April supplement was entitled 'Tackling osteoarthritis in sport'. Timed to coincide with the IOC World Conference on Prevention of Injury and Illness in Sport held in Monaco recently, the issue provides an excellent update on the current state of play with regard to osteoarthritis and sport. Marc Philippon in Vail, Colorado, reported on innovations in hip arthroscopy and surmised that if FAI and the associated chondrolabral lesions can be identified early and treated appropriately, athletes could get back to their same level of activity with good outcomes. They make a strong case for active intervention to resolve bony impingement in an effort to halt the progression of osteoarthritis.

Later in the same issue there was an article entitled 'Contact sport and osteoarthritis'. As the great coach Vince Lombardi pointed out, dancing is a contact sport whereas football is a collision sport. Bearing this in mind, it is no surprise that collision sports are associated with increased development of degenerative arthritis in later life. Some of these cases result from traumatic injury to the ACL or other structures, whereas others have more of a genetic basis. The authors conclude that injury resulting in joint damage is the greatest single risk factor for later arthritis. This is best termed posttraumatic arthritis rather than osteoarthritis.

Meniscal tears have long been implicated in the development of later degenerative change and an article by Ian McDermott entitled 'Meniscal tears, repairs and replacement: their relevance to osteoarthritis of the knee' explores the issue in depth. Most experienced surgeons will tell you that the previously recommended treatment of complete meniscectomy led to dramatic degenerative change in the ensuing couple of decades. More recent treatment via arthroscopic partial meniscectomy has lessened that risk but not eliminated it entirely. Current research focus is on meniscal repair and the use of meniscal scaffolds or the replacement of the entire meniscus by meniscal allograft transplantation. Results of these interventions are under intense scrutiny and the jury is still out – watch this space.

Imaging studies are not always perfectly correlated with clinical symptoms and David Felson writes in the same issue regarding imaging abnormalities that correlate with joint pain. Writing from the North

American perspective, where MRI is used routinely, he concludes that joint pain often originates in the bone or synovium so, logically, these are the places we should look for abnormalities.

David Hunter from the Rheumatology Department of Royal North Shore Hospital in Sydney believes that we need a paradigm shift in our management of lower extremity osteoarthritis in an excellent article in the April issue. He believes we need to focus on people at high risk of developing or with early stage disease in which structural changes may be preventable or reversible. Currently the major focus is on late stage disease. He believes we should focus on the modifiable risk factors, e.g. altered alignment, obesity and muscle weakness, and target each of these specifically.

Finally in the same issue, there is a position statement from the International Paralympic Committee on the scientific principles of classification in paralympic sport. This should be required reading for all clinicians who have a significant involvement in paralympic sport.

April

The routine April issue of BJSM concentrated on tendinopathy. It included leading articles from such luminaries as Hakan Alfredson, who wrote an excellent one page editorial entitled 'Where to now with achilles tendon treatment?' 'Where to' includes mini surgical scraping for patellar tendinopathy.

Later in the same issue there is a comparative study between sclerosing Polidocanol injections or arthroscopic shaving; 52 patients were studied and both techniques showed good clinical results but patients treated with arthroscopic shaving had less pain and returned to sport more rapidly. This is a procedure which may gain more widespread acceptance as a result of these findings.

The technique of Doppler guided mini surgery to treat midportion achilles tendinosis is described by Alfredson in the same issue, and he reported good short term results in 103 patients with midportion achilles tendinosis. The mean score on a visual analogue scale decreased from 77 pre-operatively to 2 post-operatively.

Topical glyceryl trinitrate has been used for a variety of tendon problems; George Murrell and colleagues report on the effects of topical GTN for treatment of lateral epicondylosis at the elbow. They had previously reported short term benefits for up to six months, but on analysing later data at five years there did not seem to be significant clinical benefits compared with a standard tendon rehabilitation programme alone. By contrast, patients with achilles tendinopathy were found to derive sustained benefit from use of topical GTN.

So what do we take out of all of this? Probably the best summation is by Jill Cook in her editorial entitled 'Tendinopathy: no longer a one size fits all diagnosis'. In other words, like many other areas in medicine, once we know more about the issues we can tailor treatment to individual patient requirements.

My pick for most value article in this series of journals surveyed would be that by Paul McCrory and colleagues in the February issue entitled 'Sports and exercise medicine: specialists or snake oil salesmen?' It gets to the heart of the issues confronting modern-day clinicians. We must remain true to our scientific principles whatever peripheral issues are going on. It is only by doing this that we will retain the professional credibility that has been so long in coming to our specialty

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The central governor of exercise performance: Fact or fiction?

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For many years, scientists, coaches and clinicians have been interested in establishing just what limits performance during various types of exercise. Now, in the early new millennium, a group of South African exercise scientists led by Professor Tim Noakes believe they have solved this issue with a postulate they call the *Central Governor*. This model, which they think includes the critical regulator of exercise performance, has provoked considerable thought and has led to several emotionally charged articles with fiery exchanges within the recent scientific literature. For example, articles have appeared entitled "Is it time to retire the "central governor"?,[21] "The improbable central governor of maximal endurance performance"[6] and "Do we really need a central governor to explain brain regulation of exercise performance?".[10] In a feisty rebuttal, Noakes responded with a publication called "Is it time to retire the A.V. Hill model?"[14] - an exercise model that apparently opposes his ideas. Never-the-less Noakes is not alone as other scientists have given support to the central governor hypothesis.[12,13] The present commentary is not meant to be an extensive evaluation of the central governor model, but rather it is my attempt to clarify some of the ideas underpinning the model and to provide a more balanced view on some of the recent spirited discussions, especially in relation to its role in fatigue of sport performance.

What is the central governor and what does it do?

The model was initiated by Ulmer but first laid out in detail over several papers in 2004-2005, when it was proposed that the central governor is a specific subconscious centre in the central nervous system (CNS) that acts in an anticipatory (feed-forward) manner to regulate exercise performance through de-recruitment of skeletal muscle/motor units.[9,17,18,24] It was argued from evolutionary perspectives that such a governor would control motor drive to working skeletal muscles in order to conserve homeostasis and ultimately prevent catastrophe, ie, damage to the heart, skeletal muscles, lungs, or brain itself. Several features of this model are still undergoing development. First, although it includes a subconscious brain centre there is now mounting importance placed on the rating of perceived exertion (RPE) which is a conscious aspect of the sensation of fatigue.[9,15,22] Second, teleoanticipation, a central feed-forward mechanism, is regarded as a key feature but there is also acceptance that feedback from the periphery interacts with the central governor to help set the intensity of exercise.[7,9,15,25] Third, Noakes and associates argue that the central governor is a regulator of exercise behavior, rather than being an enforcer to limit exercise performance. However, this distinction is speculative and cannot be made with certainty simply by measuring the fatigue symptoms. Many studies on the central governor focus on exercise termination during either prolonged or incremental maximal exercise.[4,9,17,18,24] However, several other impairments to performance appear as fatigue symptoms prior to this extreme point.[8] The South African group has also investigated pacing (ie, regulation of exercise intensity) which they regard to be another feature of the central governor,[23] rather than it being just an exercise stopper. Finally, Noakes and coworkers propose that the central governor indirectly controls maximum cardiac output and peak oxygen consumption[16] but that will not be considered further here.

What is fatigue?

There is ongoing debate as to what is the best measure of fatigue. Traditionally, fatigue is defined as any exercise-induced reduction in the capacity of muscles to generate force or power.[1,8] However, we recently

emphasised that fatigue during sport competition can manifest as any one of a number of symptoms relating to impaired performance.[8] This includes a drop-off of pace, loss of maximum speed during a sprint, fewer high-intensity exercise bursts, changes of technique leading to reduced skill outcomes, subjective sensations of fatigue, or impaired decision-making, but their specific occurrence depends very much on the sporting event or task of interest. In most cases the complete cessation of exercise is termed exhaustion.[5,8] When fatigue is quantified as a decline in maximal muscle force production it is usually subdivided into peripheral and central components.

What is peripheral fatigue?

This component involves a direct reduction in the ability of muscle fibres to generate force, and occurs gradually by mechanisms which are not just an on and off switch mediated via the CNS. In consequence, some exercise scientists including myself have taken a reductionist approach to study fatigue induced by electrical stimulation of isolated muscle preparations.[1,3,8] Indeed, the long standing AV Hill model for exercise performance, which is strongly challenged by Noakes,[14] postulates that factors changing in muscle reduce its function and ultimately impair whole-body exercise performance. Hill was one of the early researchers to drive the notion that lactic acid is the culprit which impairs muscle function.[14,17] Despite this hypothesis now losing favor,[1,2] there are still several other peripheral fatigue factors that can impair muscle function such at muscle phosphagen changes, glycogen depletion, electrolyte changes and production of reactive oxygen species.[1,3] In contrast, Noakes appears to totally refute the occurrence of peripheral fatigue[15] despite what many regard as convincing evidence that it occurs.[1,3,5,8] Moreover, we have shown emphatically with subjects who underwent prolonged cycling to exhaustion that peripheral fatigue contributes about two thirds of the 30% decline of maximal isometric voluntary contractile (MVC) force.[20]

What is central fatigue?

This component (ie, the inability of the CNS to maximally activate muscle during fatiguing voluntary contractions) is usually tested with electrical stimulation superimposed on an isometric MVC.[5,19,20] This method indeed tests for any decline of maximal motor drive (ie, de-recruitment or severely lowered motor neuronal firing frequency) due to the preceding exercise, but it does not confirm that impaired motor drive has functional consequences during the sub-maximal dynamic contractions which normally occur during exercise.[8,17] We show that central fatigue assessed in this manner accounts for about one third of the decline of peak MVC force with prolonged cycling.[20] Such effects could, in principle, involve altered command by a central governor. This could occur either centrally or via feedback from the periphery, and possibly includes signals originating from working muscles.[5,8,9,15,18-20,25] Such peripheral signals include hypoglycaemia, hypoxia, acidosis, elevated reactive oxygen species and potentially raised plasma potassium.[5,8,9,19,25] Interestingly, a long held hypothesis for exhaustion during prolonged exercise, that of muscle glycogen depletion, may involve release of the cytokine, interleukin-6, from fatigued muscles to exert detrimental effects via the CNS.[5,8]

What is the sensation of fatigue?

This subjective aspect involves the conscious sensation associated with increasing effort during sub-maximal work (ie, RPE), together with muscle weakness and other sensations that remain after exercise.[8,22] As a result there has been much interest in RPE,[4,8-11,18-20] which is now postulated to be the conscious interpretation made by the homeostatic central governor.[9,15] Its importance becomes apparent since maximal levels of RPE (ie, 18-20 on the Borg scale) are commonly observed at exhaustion when exercise becomes unbearable.[4,7,8,19,20] Moreover, the time-to-exhaustion is intimately related to the rate of rise of RPE early in exercise.[4,20] Clearly RPE is a feature of the CNS but just how it is linked to central fatigue, the loss of muscle force/power and other fatigue symptoms, and the central governor, remains unclear.

It is also recognised that RPE is linked to increasing central motor drive through both central and peripheral factors (eg, hypoglycaemia, hypoxia, acidosis, serotonin, hyperthermia, dehydration), some of which also contribute to central fatigue.[8] In a well designed study, Marcora and associates show that a cognitive challenge leading to mental fatigue also causes a higher RPE in subsequent exercise and a shorter time-to-exhaustion.[11] Clearly, such influences occur through the CNS, as shown with other interventions such as hypnosis.[26] Certainly, more research is needed to understand the factors which underpin and manipulate RPE, and presumably with it an altered exercise performance.

Does the brain regulate exercise performance?

It is established that the brain contains cardiac, vasomotor, respiratory, thermoregulatory, and various other control centres which are able to influence body functions during exercise, in part via feedback loops.[14] So the idea of central regulation is not new. Moreover, higher cortical centres can provide input to these control centres to enhance pre-exercise function in an anticipatory manner as observed with elevated pre-race heart rates. We should also be aware of beneficial motor control adjustments which allow compensatory effects as muscle fibres/motor units gradually fatigue.[1,5,8] A slightly lowered motor neuron firing frequency can convey benefits known as muscle wisdom,[1] a rotation of motor unit recruitment may let fatigued motor units undergo rest and recovery,[5] and motor drive can alternate between muscle groups to permit a sharing of the work load at a more gross level.[8] Motivation and other psychological constructs that act via the CNS can also modify motor output and performance.[5,7,8,10,16,17,19] Yet another CNS feature which can influence performance is that of decision-making, and this aspect often

improves with exercise.[8] However, it appears that it is only with conditions such as severe hypoglycaemia, hyperthermia, or dehydration that decision-making can worsen.[8]

Is there evidence supporting the central governor model?

The initial reasoning involved evolutionary ideas and a need to avoid catastrophe,[9,17,18,24] but further arguments appear to involve more objective evidence. First, a decline of electromyographic activity in working muscles is interpreted as the central governor switching off some motor units.[24] However, this interpretation is not exclusive to a reduced motor drive as it may also relate to electrolyte effects which diminish muscle excitability.[1,3] Second, there is a need to localise the specific central governor area within the cerebral hemispheres, and areas in the frontal lobe and thalamus have been implicated especially in relation to RPE and the perception of fatigue[14,22,26] However, this work is in its infancy. An ultimate experiment here would be the selective ablation of identified central governor neurons and then test for changes to exercise performance but such experiments clearly cannot be done on humans. Third, the presence of an end-spurt, ie, the increased power output at the end of a race,[14,18] is argued that it cannot be explained by anything other than a central controller.[14,15,23] However, this should be addressed correctly by comparing the peak power output at the end of a race with that achieved when the subjects are fresh at the start, since this truly accounts for a contribution from peripheral fatigue. Fourth, the muscles of exhausted individuals usually show less than 30% decline of peak MVC force,[5,8,14-17,20] suggesting a peripheral reserve with motor units still being available to contract.[14,15] In contrast, electrically stimulated isolated muscles, which bypass CNS regulation, sometimes involves a much greater loss of force.[1,8] Thus, arguments have been put forward in support of the central governor hypothesis although none are absolutely conclusive.

Personal viewpoint on the central governor.

The appearance of the central governor model has been valuable to provoke extensive discussion and more critically appraise our understanding of exercise performance. However, it is difficult to obtain good experimental evidence to confirm a role for a central governor, and hence it is difficult to either prove or disprove the model. Never-the-less the model has several appealing aspects and as such should not be retired as suggested.[21] Similarly, being adamant that the central governor is the universal exercise regulating mechanism is most surely flawed. Notably, many researchers who question the importance or need for a central governor still give considerable support to a role of the brain in the regulation of exercise performance,[10,21,25] and are not endorsers of a brainless model as suggested.[15] To that end the brain works in an integrated manner with muscles, heart and lungs during exercise. Although the brain may have the ability to restrict performance all by itself, it is usually influenced by changes external to the CNS such as lowered glucose levels, hypoxia, hyperthermia, dehydration, raised potassium, acidosis etc. So could the central governor really just be the brain itself?

A notable restriction with the central governor model is that it has been applied mainly to exercise cessation during prolonged or incremental maximal exercise. Although pacing has been introduced[14,15,23] other impairments occur before disaster that also need to be considered for a fuller evaluation of exercise performance. Moreover the central governor model has not yet been applied to clinical exercise situations, such as occur with effort syndromes, and this needs development. A major problem arising from the ideas of Noakes and colleagues, in my view, stems from rejection of any contribution from peripheral fatigue.[14,15] It appears that in most forms of sport or exercise that there are fatigue contributions from several sources including peripheral and central fatigue along with fatigue sensations,[8] and the key question really involves establishing the relative contribution of the different aspects. We also recently proposed that with peripheral fatigue there are considerable safety margins so that quite large perturbations of homeostasis can occur before impairment of muscle function.[3] Peripheral fatigue is clearly more complex than simply being a linear model.

The real issue is probably not about establishing which model best explains exercise performance, as they may all contribute to some extent, but instead sorting out just what are the main fatigue factors and what can be done about them. An understanding of central and peripheral processes gives insight as to how best to intervene with the likes of diet, training and psychological tricks. Most importantly we have recently indicated that several fatigue factors can have a protective role prior to being detrimental,[8] although larger changes interactively contribute to peripheral and central fatigue, RPE and the sensation of fatigue, impaired motor skills, and occasionally to decision-making.[8] Hence it may be possible to manipulate some factors and influence performance via many sites which potentially includes actions via a putative central governor.

Summary

It is difficult to comment with certainty about whether the central governor hypothesis is right or wrong but the following comments seem to be acceptable.

1 There is indeed brain regulation of exercise performance. This potentially includes a central governor area although such an entity may be the entire brain.
2 Many fatigue symptoms are mediated via the CNS and not just exercise termination.
3 Central fatigue is involved in most forms of exercise that last for some duration.
4 The occurrence of peripheral fatigue to various extents is a real phenomenon.
5 The conscious sensation of fatigue plays a big part in

exercise performance.

6 Exceedingly high RPE (intimately related to the sensation of fatigue) coincides with impaired exercise performance. Manipulation of the factors which contribute to RPE (eg, lowered blood glucose, hypoxia, acidosis) can alter the time-to-exhaustion and most likely influence several other fatigue symptoms at the same time.

Regardless of whether the central governor model is ultimately confirmed as fact or fiction it is an extremely valuable concept to investigate further and apply to all forms of sporting event along with clinical exercise situations.

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T-wave Inversion in Athletes

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Case Study

A 22 year old Caucasian male international footballer attended our facility for pre-participation screening (1st attendance) before a major competition.

History

For the past six years, he has trained six days per week, two hours per day. No family history of cardiovascular disease or sudden cardiac death in any 1st degree relatives, but he had a post-training syncopal event after training associated with palpitations two years previous. Despite documented T-wave inversion on resting ECG, a full medical clearance was provided at the time following a normal cardiovascular evaluation (echocardiogram, stress ECG and 24 hr Holter).

Examination

Auscultation and physical examination were unremarkable.

ECG (Figure 1)

Marked sinus bradycardia (39bpm) with normal PR, QRS and QT/QTc durations. Right atrial enlargement, incomplete right bundle branch block, ST segment elevation ≥ 3mm in leads V2-V3, deep T-wave inversion in Leads II, III, V4-V6 together with ST segment depression in Leads II, III and aVF.

Echocardiogram

Considered appropriately adapted to his physical training level with normal left ventricular and atrial dimensions and function. There were no regional wall motion abnormalities. Left atrium and valves were morphologically normal and without pulmonary hypertension. Right ventricle and atrium looked normal. His intra-atrial and intra-ventricular septum was intact. Inferior vena cava and hepatic veins were normal and the origins of the coronary arteries were anatomically correct.

Exercise Stress Test

Negative for inducible ischemia and arrhythmias. Baseline T wave inversions normalised immediately with the onset of exercise, but returned to negativity during recovery. There were no abnormal blood pressure responses to exercise or post-exercise. Twenty-four hour ambulatory ECG failed to identify any complex ventricular arrhythmia.

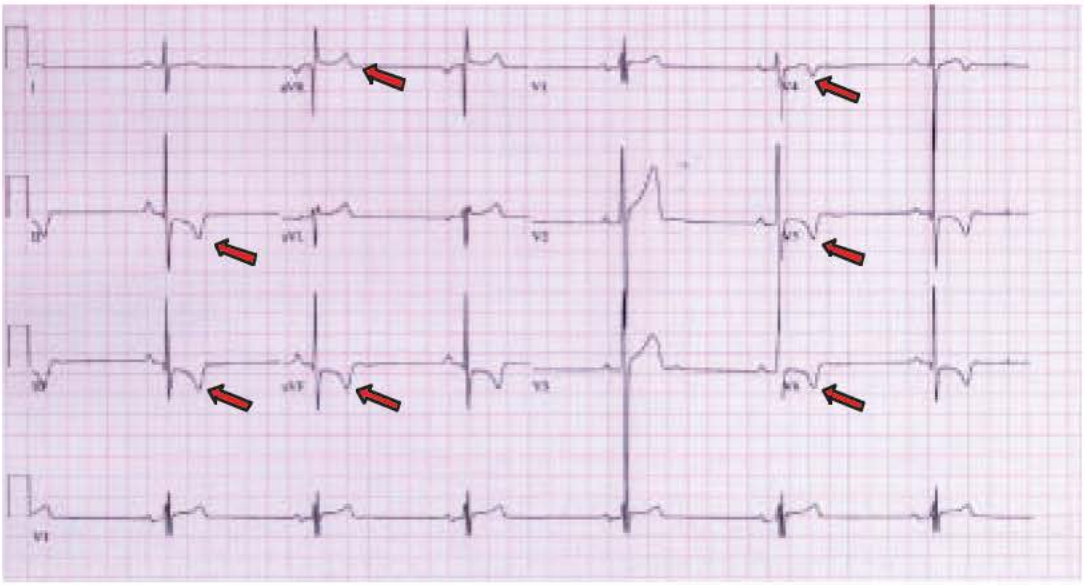
cMRI

Cardiac magnetic resonance imaging (cMRI) incorporating late gadolinium enhancement (LGE) documented the left ventricle to be normal in volume, with good systolic function. Left and right ventricular wall thicknesses were within normal limits, without marked hypertrophy. There was no evidence of abnormal wall motion. Mitral, aortic and tricuspid valves were structurally normal without regurgitation. Both atria were normal in size. There was no evidence of acute myocardial insult or edema. Early and late gadolinium enhancement showed no evidence of myocardial fibrosis or scarring.

Summary

Even following established risk stratification criteria, this player produced a screening conundrum as despite a distinctly abnormal ECG and an undiagnosed syncopal episode, the player presented with normal secondary investigations.

Figure 1: A 22 year old professional football player (Caucasian) without a FH of SCD. Normal echocardiogram, late gadolinium enhanced CMR, cardiopulmonary exercise stress test, 24h Holter.



Management Recommendations

1) authorise full competitive activity; but offer the option of genetic testing, educate the athlete for personal symptoms, and place under annual cardiac examination including history physical examination, resting and stress ECG, echocardiogram and cMRI. 2) propose a systematic cardiac examination (non-invasive examination with ECG and Echocardiography) in first degree-relatives (>10 years old).

Current Status

The player returned to full competition, and at present (8 months post evaluation) has remained asymptomatic (self reported) with no adverse cardiac events.

T-wave inversions in athletes:

Electrocardiographic changes in highly trained athletes are common and usually reflect benign structural and electrical remodelling of the heart as a consequence of regular and intensive physical activity. This remodeling is commonly known as 'athlete's heart'. However, repolarisation abnormalities may be an expression of an inherited cardiac disease, placing the athlete at significant risk of sudden cardiac death, either during or following sport.

Deep T wave inversions in the lateral leads are a major concern for screening physicians, as these ECG alterations are a recognised manifestation of Hypertrophic Cardiomyopathy and Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC).[2] Inverted T-waves may represent the only sign of an underlying inherited heart muscle disease even in the absence of any other features and before structural changes in the heart can be detected.

The clinical significance of deep T wave inversions in athletes is yet to be fully understood, with this player's ECG currently classed as neither physiological nor pathological. Recently, Pelliccia et al (2008) reported on 81 athletes (from a database of 12,550) with diffusely distributed and deeply inverted T waves, with no apparent cardiac disease, who had undergone serial clinical, ECG, and echocardiographic studies for 9 ±7 yrs (range, 1 to 27 yrs). From the 81 identified, five (6%) ultimately proved to have cardiomyopathies, including one who died suddenly from undetected ARVC. The authors suggest that these abnormal ECG's may represent the initial expression of an underlying genetic cardiac disease that may not be phenotypically evident for many years. Pelliccia et al's (2008) investigation underscores the importance of continued clinical surveillance and clinical follow-up for this football player; with serial ECG, echocardiographic and cMRI even in the absence of cardiac symptoms and/or clinically demonstrable heart disease.

cMRI and Genetics

There has been considerable progress in recent years in the development of imaging technologies capable of characterising a much wider number of cardiomyopathic processes.

In particular, the advent of gadolinium enhanced cMRI has dramatically changed the non-invasive work-up of athletes with a suspected cardiomyopathy. cMRI provides a comprehensive assessment of both ischemic and non-ischemic cardiomyopathies providing detailed information on cardiac anatomy, function, tissue characterisation, epicardial and microvascular perfusion, valvular flows, and coronary and peripheral angiography. The presence of LGE is indicative of abnormal myocardial interstitium typically due to the presence of myocardial fibrosis or infarction.

A high proportion of cardiomyopathies are of genetic origin. There is probably a continuum between the initial stage of a fully healthy condition and the stage of obvious cardiac expression with an intermediate stage of mild borderline abnormalities. The earliest phase of a cardiac expression may be characterised by ECG abnormalities such as T-wave inversion, preceding the later development of other identifiable structural abnormalities.

Genetic testing was recently developed for routine clinical use in order to improve both genetic counselling and medical management of patients and relatives.[1] In a patient with an overt cardiomyopathy, the yield or rate of mutation identification is variable according to the disease: 50-70% in HCM and around 40% in ARVD.[3,5] Failure to identify recognised mutation must not lead to the clinical exclusion of a cardiomyopathy or ion channelopathy for three important reasons; 1) not all genetic regions are assessed, 2) current technology is not able to detect some forms of mutation, and 3) a similar phenotype may possibly develop without a specific genetic constitution. In other words, only positive genetic tests have a diagnostic value, but negative tests do not exclude the disease.

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Acute cervical myelopathy in a patient presenting with knee instability – a case report

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Introduction

Obtaining a specific patho-anatomic source for neck and spinal pain can be difficult for the vast majority of cases.[5] An alternate option is to classify mechanical neck or spinal pain into groups, or syndromes, based on homogenous signs and symptoms.[3,7,8] The Canadian Back Institute[2] (CBI) has developed a syndrome based classification system consisting of four distinctive categories or 'patterns' of neck pain (see Table 1). Pattern One represents neck pain that is primarily aggravated with cervical flexion postures and movements. Pattern Two is neck pain that is only aggravated with cervical extension positions. Pattern Three is arm pain associated with cervical radiculopathy and Pattern Four represents symptoms of neck or arm pain and upper motor neuron dysfunction associated with cervical myelopathy. The CBI methodology offers clinicians a carefully structured assessment that places particular emphasis on the importance of a detailed symptomatic history to determine the location and constant or intermittent nature of the pain, and to identify the neck movement and postures that aggravate and relieve symptoms. Another essential component of the CBI classification process is the routine assessment of upper and lower motor neuron function for all patients that present with neck pain. This standardised assessment and classification approach was an essential factor in this case in which a relatively inexperienced clinician (four month post graduate physiotherapist) diagnosed an acute cervical myelopathy in a patient presenting with symptoms of knee instability, and referred the patient appropriately to a specialist for further investigation.

TABLE 1: Diagnostic criteria for patterns of neck pain.
***neck dominant indicates worst pain is proximal to the deltoid insertion and inferior angle of the scapula; arm dominant pain indicates worst pain is distal to the deltoid insertion.**

Pattern of Neck Pain	Location*	Nature	Aggravating and Relieving Postures or Movements
One	Neck dominant pain.	Intermittent or constant pain.	Cervical flexion increases pain.
Two	Neck dominant pain.	Intermittent pain.	Cervical extension increases pain.
Three	Arm dominant pain.	Constant or previously constant pain.	Neck movement and postures affect arm pain.
Four	Neck or arm dominant pain.	Balance or gait disturbance and/or loss of fine motor skills.	Varied - but upper motor neuron signs such as hyperreflexia, spacticity, clonus and positive babinski may be present (clonus and babinski testing is standard with all neck pain patients).

Case Report

A 41 year old male recreational football player attended a routine follow up appointment three months following a right knee arthroscopic meniscectomy and subsequent rehabilitation. He complained of a one week history of intermittent right knee instability that was causing difficulty when walking downhill or descending stairs. He also noticed some weakness in his left knee and was complaining that both lower limbs felt cold and "jittery", especially at night and when cold. He reported no other problems on level surfaces with walking or running. He did not complain of knee pain and could not recall a recent injury to the knee. He intended to play football the following day for his club team.

Because the patient was presenting with new symptoms of bilateral leg weakness and sensation changes without any history of a recent local injury to the knee, the spine was assessed as a possible cause of the symptoms. On further questioning, the patient denied any low back discomfort but when asked about neck pain he recalled being hit from behind in a mild traffic accident two weeks earlier. He had been experiencing mild intermittent left sided neck pain since the accident but these symptoms had been slowly resolving. He had no history of any previous significant neck pain, and at no stage did he ever have any arm pain or paraesthesia distal to the acromion.

Physical examination revealed full cervical range of motion with minor left-sided neck discomfort in all directions. There was grade 5 strength bilaterally in both upper and lower limbs. Sensation testing was normal in his lower limbs. Pulses were present equally in both feet. There was bilateral hyperreflexia in the tendons of his biceps and triceps brachii, brachioradialis, patella and Achilles tendons. There was a positive clonus test with at least four beats

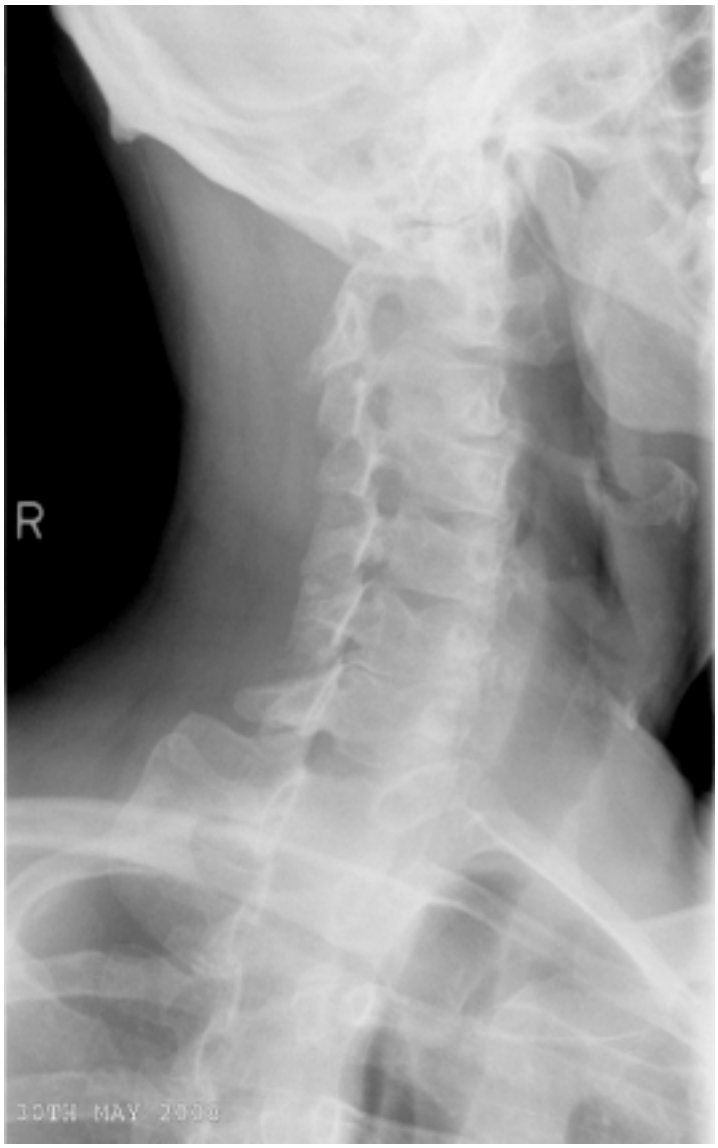

FIGURE 1

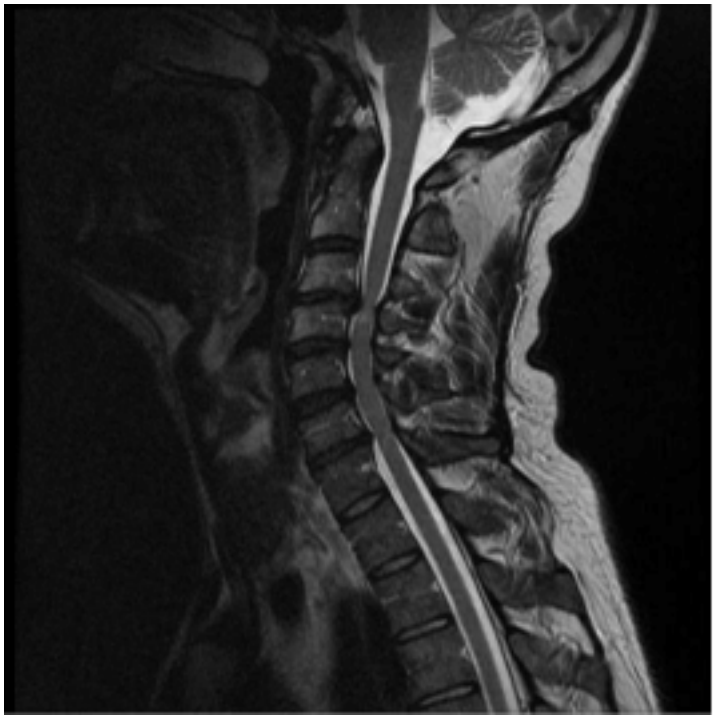

FIGURE 2

of clonus bilaterally. Babinski reponse was negative.

According to the CBI system, these symptoms of neck pain and upper motor neuron disturbance may indicate a cervical myelopathy (Pattern Four). An urgent x-ray was arranged which showed a posterior subluxation of C3 on C4 facets (figure 1). The subsequent MRI scan showed a large disc bulge at C3/4 compromising the spinal canal, where increased signal suggested acute cord oedema (Figure 2).

The patient was referred urgently to the neurosurgical registrar at the Wellington Hospital Emergency Department. An anterior cervical discectomy and fusion (ACDF) at C3/4 was performed two days following admission. When the patient was reviewed several weeks following surgery, he still had a positive clonus test but noticed that the episodes of knee instability had decreased significantly. On following up this patient 3 years after his surgery he states that he now only has occasional 'jumping' sensations in his legs when descending stairs. He describes these symptoms are only present in cold weather. He has no neck pain and has resumed playing football at his previous level. His symptoms have been slowly improving over the last 3 years.

Discussion

Because complaints of neck pain and discomfort are so common, it is easy to become dismissive, especially when the pain is not the significant problem. In this case, a patient presenting with relatively innocuous symptoms of knee instability and mild neck pain had a very serious pathological cause for his symptoms.

In addition to the findings for this patient, there are many other signs and symptoms that could suggest presence of cervical myelopathy. Radiculopathy in the upper limb, bowel and bladder dysfunction and lower limb stiffness or weakness can all commonly be associated with cervical myelopathy.[1,4] Kim et al[6] found the most common complaints from patients experiencing myelopathy from soft disc herniation were gait disturbance and spasticity.
One of the most interesting features of this case was the relative lack of local neck pain and discomfort in someone who had such a potentially serious injury. This finding is however not altogether uncommon as Bednarik et al found that cervical myelopathy may often present without neck pain as spinal cord compression itself is not necessarily symptomatic, especially in the early stages of development.[1]

This patient's positive upper motor neuron lesion signs could have been benign or from an unrelated, upper motor lesion such as multiple sclerosis or a localised cerebral lesion but because the onset of both neck pain and neurological symptoms seemed to relate to a specific recent event (motor vehicle accident), it was necessary to investigate the cervical spine urgently. Since this patient's ACDF surgery he has noticed a slow but steady improvement in both his left and right knee strength and stability. This in itself supports the theory that the neck pathology was most likely the cause of his lower limb symptoms, and that his symptoms were not related to an alternate upper motor neuron pathology or previous right knee meniscetomy surgery.

This case reinforces the need to consider cervical myelopathy in the presence of symptoms associated with lower limb weakness or gait disturbance. It also emphasises the importance of routine upper and lower motor function screening as an essential component of a cervical spine assessment.

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Blast from the Past

Recently I had cause to go and look at Don O'Donoghue's classic text Treatment of Injuries to Athletes. I have the third edition published in 1976. The first edition was published in 1962. For those of you who are unaware, this is widely regarded as the first true sports medicine textbook. Don O'Donoghue was Professor of Orthopaedic Surgery at the University of Oklahoma Medical School and team physician to the University of Oklahoma football team.

The book is a classic and reading it shows how far we've come in the 35 years since this edition was published. He prefaces the book with the comment, "Everything I know I learnt at my mother's knee or some other joint." In the first edition he goes on to say, "I owe a debt of gratitude to the American athlete. That which began as an interesting sideline to a life devoted largely to paediatric orthopaedics has burgeoned to become perhaps my major interest." By the time of the third edition he still obviously loves his work. He comments in there, "I still greatly appreciate the American athlete."

The book starts off with a Bill of Rights for the School and College Athlete which was drawn up by the American Medical Association Committee on the medical aspects of sports. It emphasises good coaching, good officiating, good equipment and facilities and good health supervision. This is certainly a great place to begin.

The book itself commences with a chapter on prevention of injuries and he states, "The physical conditioning of a player should be a continuous thing." So often this is forgotten. He outlines a recommended summer high school conditioning programme and it is fascinating to note under Item 11 the comment "Temperate living". This involves four aspects:

1 Sleep, which he defines as the great restorer and recommends a minimum of nine hours.

2 No intoxicants - he goes on to say it is not a food it is a poison.

3 No smoking.

4 No drugs.

This would make good reading for today's athletes. He goes on to describe the playing arena and athletic equipment plus protective strapping for the ankle.

Part 2 consists of general principles of treatment. He lists five precepts for care of the athlete, which he terms the Five A's:

1 Accept athletics.

2 Avoid expediency.

3 Adopt the best method.

4 Act promptly.

5 Achieve perfection.

He then goes on to champion the role of the physical examination. He comments that it may not be possible to examine the player at once but the goal should be to examine him as soon as it can be done properly. The book also mentions x-ray examination and laboratory testing, but was published before the explosion in medical imaging which has occurred in the past few decades. Following that explosion the role of physical examination has been downgraded in some centres, to the detriment of the player, in my opinion. Then follows a chapter on the principles and the management of specific injuries, both soft tissue and bony injuries.

The majority of the book is taken up with recommendations for diagnosis and treatment of injuries in specific areas. Starting from the head down, it shows examples of subdural and extradural haematomas; Frank Netter's pictures are not for the squeamish. Interestingly, there are no concussion guidelines listed, this book predating their development. Moving down the body to the shoulder, sternoclavicular disorders are well-covered. AC joint injuries are discussed and he is clearly a fan of surgical fixation of third degree AC joint injuries, as indeed are most American authors. There is relatively less information on glenohumeral joint problems or rotator cuff injuries, which consume a large part of our daily work in this millennium.

The chapter on spinal injuries has a good deal of information on the hyperextension injuries of the neck which are so common in American football. There is some mention of low back problems but nothing like in the depth that one would expect given the frequency of these conditions in most people's practice.

The chapter on hip and groin rightly mentions the importance of conditions such as slipped upper femoral epiphysis. Our knowledge of groin injuries is far from complete but this book shows how far we have come. It has no mention of entities such as sports hernia or labral injury, and only scant mention of osteitis pubis - in the old days this was thought to be an infective condition.

Moving down to the knee, he devotes a good deal of discussion

to what was later termed O'Donoghue's unhappy triad. This is a severe knee injury involving the medial collateral ligament, medial meniscus and anterior cruciate ligament. He was one of the first to recognise the significance of cruciate ligament injuries and describes repair of the ACL using mattress sutures. He points out that an avulsion injury involving a bony plug provides a good opportunity for early surgical intervention with good results. Anterior knee pain was not well understood in the early days and, as befits a surgeon, there is plenty of discussion on techniques such as chondral shaving, trephine and drilling, facetectomy and even patellectomy. Fortunately, our knowledge of non-operative treatment has improved exponentially and so the need for surgery for anterior knee pain has dramatically reduced in the last few decades.

Moving down the leg there is discussion of shin splints but, as he points out, there is "considerable and often raucous argument as to what is actually meant by the term". He goes on to say, "the title shin splints is 'a waste bucket' including many different conditions." He correctly identifies that the tibialis posterior origin is most commonly involved. There is even mention of elevation of the heel by a half inch felt pad and/or the insertion of a cork and leather arch support flexible enough to permit the foot to flex. My how things have changed - we now have a whole industry built around orthotics. In America they tend to be overly in love with rigid devices and one wonders how many athletes would be better off using the flexible materials advocated by O'Donoghue.

Finishing off with the ankle and foot, he emphasises early weight-bearing and active rehabilitation following ankle injuries.
The final chapter concerns rehabilitation following athletic injuries and is written by Fred Allman, one of the other early giants of our discipline from Georgia Tech in Atlanta. Once again the exercises include the simple things such as neck strengthening, which we have built on in subsequent decades. A lot of these have stood the test of time but a few have been discarded in the light of subsequent evidence.
All in all, it is salutary to pick up a volume such as this. In 1976 a single author with a few fellow contributors was able to encapsulate the entire field pretty well in just over 800 pages. Such is not the case today and it is a measure of how far we have come. However, those of us of a certain age will fondly remember being taught about O'Donoghue's unhappy triad and it is interesting to read the description from the guru himself.

For anyone with an interest in how our discipline began, I would highly recommend this book.

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American College of Sports Medicine Annual Conference, Denver 2011

The American College of Sports Medicine Annual Conference was held in Denver Colorado over the week of 31 May 31 – 4 June. This is a huge meeting (everything is big in the US), with over 6,000 participants (more than 1,200 international), and 3,200 presentations in multiple concurrent streams. Typically there is a heavy focus on physiology, and this year was no exception. From my perspective this makes it interesting, for as a clinician, it drags you out of overtly clinical presentations, and enduces consideration of a more 'basic' scientific and multi-disciplinary approach to thinking.

This year there were a couple of trends that were worth noting. Firstly, there were many clinical ultrasound (US) sessions. I have not noticed this on the programme before, but illustrates that even in the USA the use of US by sports medicine practitioners is expanding. My own feeling is that this is an immensely useful extension of the clinical examination, and in my experience is a useful tool in the clinic (for education, monitoring, guided injections). Paradoxically, I firmly believe that if a diagnosis is required, and other differentials need exclusion, the opinion of a skilled radiologist remains paramount.

Secondly, there continues to be a trend to increasing study of genetics in sports medicine. The impression I received overall from the large number of posters and presentations, was that the evidence for most genetic influences on performance remain contradictory, limited in study power and plagued by limitations in study design. This does however remain one of the really interesting areas to watch in both exercise performance and health.

Finally, if the number of presentations are anything to go by, there appeared to be very little interest in the clinical care of the elite athlete and the specific challenges that this imposes. Rather, there was a predominance of presentations considering the health of the population and exercise prescription for health. This is a very important element of the sports health industry, and may reflect the direction sports medicine is heading.

ACSM is worthwhile attending, but choosing which presentation to attend can be challenging and frustrating – when you realise you are trapped in a lecture which is perhaps not what was expected, and there may be two or three other lectures you could be at. I met a number of kiwis (not all working in NZ!) and it was great to see that individual sports in NZ were recognising the importance of having their sports medicine staff (physiotherapists and nutritionists) attend this large international conference, and to support it financially. Denver is a beautiful place, and well worth a visit! The following summarises a few of the topics and lectures that I managed to get to and maintain an attention span in the face of dire jet lag and lecture fatigue. I hope that it may encourage you to look at some of the work produced by the excellent presenters, on really interesting and important topics – such that you can check the accuracy of my recollections!

Exercise and Health

Karim Khan, a Fellow of the Australasian College of Sports Physicians but well known around the world, gave a typically colloquial, entertaining and thought provoking talk regarding the importance of physical activity (PA) in health ("Supersize my Exercise! Learning from Mad Men, the Malboro Man and Freakonomics to Promote Physical Activity"). Highlighting the increasing evidence that lack of fitness results in more deaths than smoking, diabetes and obesity ("smokadiabesity") combined, as well as the positive impact that exercise can have on killers such as cancer and cognitive function, Karim presented a strong case for the prescription of exercise. The paradox being that despite the increased evidence for the benefits of exercise in recent years, exercise levels of populations internationally continue to decrease. Prescription of exercise at an individual level needs to be personalised to optimise health, which may differ from the global population based recommendations – with evidence presented for a hyperbolic relationship between exercise volume and healthy outcomes. Another interesting point for me, was the use of "exercise as the 5th vital sign" (see Sallis BJSM (45) 6; p 473 – 474), recording current exercise patterns as a marker of risk – an easy and effective tool in clinical practice. Importantly, Karim highlighted the increasingly recognised need for research on implementation of practices, rather than further evidence that exercise is a useful tool – that is, how can we best apply what we now know, for better clinical outcomes.

Karim highlighted the movement around the world for exercise as medicine, but to my mind, the rest of the world is just catching up with NZ's Green Prescription programme.

Professor Juleen Zierath ("Promoting Effects of Exercise in Diabetes and Obesity; Translating Basic Science into Preventive Action and Treatment", Joseph B Wolffe Memorial Lecture: Karolinska Institute Stockholm, Sweden) gave an insightful lecture into the complex but progressive unraveling of the mechanisms behind the benefits of exercise in type II diabetes. We are all well aware of this benefit, but the science underlying this is fascinating. One point of note was the loss of "metabolic flexibility" in diabetes, whereby there is an increased preference for fat storage over fat metabolism, and the effect that muscle fibre type may have on this. Exercise, amongst other things, will increase Glut 4 receptor expression, thereby enhancing glucose uptake.

Advancing and Sustaining Evidence-based practice in Clinical Settings

For a clinician, this lecture (Bernadette Melnyk, Exercise is Medicine Clinical Lecture) was a bit of hard work, but a couple of elements rang true with my way of thinking. The first was the highlighted point that evidence based practice is not all about level one, double blind randomised controlled trials. By contrast, while it does involve that assessment, it also incorporates consideration of both patient preferences and values, and the individual clinician expertise. As per my editorial in this edition, I believe strongly that the opinion of experts and experience needs to be considered in any evidence based practice. Secondly, as with Karim in his lecture, Dr Melnyk highlighted the need for translational approaches, to ensure that evidence is adequately incorporated into our clinical practice.

Sudden Cardiac Death (SCD) in the Fitness Setting

Both speakers (Kyle McInnis / Barry Franklin) highlighted the unpredictability of SCD in the over 35 population, and the fallibility of exercise stress testing in this group. Clearly illustrated was the surprising frequency of SCD or near death in fitness centres, with unpublished data from the USA suggesting up to 50% of fitness facilities may have experienced this. Rapid intervention and application of shock through the Automated External Defibrillater (AED) remains the best management, but this requires training and confidence from the fitness centre staff. With the demographics of fitness centre utilisation moving towards an older, middle-aged (not sure that I'm comfortable with that term) population, there is an increasing need for awareness of this issue in fitness centres.

Nutritional Support for the Injured Athlete. A Focus on Muscle and Tendon Injury

The most striking impression I received from this session (Kevin Tipton (UK),Stuart Phillips (Canada), Bronwen Lundy (UK) – introduced by NZ's own Jeni (I-really-do-speak-like-this) Pearce was the lack of specific data available to support this topic (bearing in mind I left before the end, so probably missed the best part!). This probably reflects the challenges imposed by research in this area, rather than either the scientific reality, or a lack of interest. Important in rehabilitation, particularly from surgery, is the maintenance of an adequate energy intake, and the avoidance of unnecessary prolonged immobilisation. Immobilisation will result in disuse atrophy (affecting the muscle, tendons, ligaments and bones), predominantly as a result of decreased protein synthesis – however and importantly, in short durations of immobilisation, amino acid (AA) supplementation may offset that disuse atrophy (the numbers discussed were: 16g essential AA, with 30 g CHO, 3 x daily between meals). In the long term immobilisation however, AA supplementation has not been shown to be useful. While protein breakdown is fairly stable during the day, protein synthesis fluctuates constantly with a reduction in synthesis between meals. Other than nutrition however, it is important to note that only a very low number of isometric contractions are required to maintain muscle strength, and offset disuse atrophy. Unfortunately, as most of us may already be aware, for some reason, the calf muscles are more difficult to preserve – they didn't give me an answer to this perplexing question.

In a separate session, Professor Marco Narici (Manchester Metropolitan University) discussed "Muscle and Tendon Remodelling in Use and Disuse" and presented a really interesting story. He reminded me that Myogenic Regulatory Factors (responsible for or reflecting muscle adaptation) are increased within 12 hours of strength training – while measured muscle hypertrophy may not adequately reflect these early changes. Furthermore, more subtle architectural changes in muscle, including "pennate insertion angle" (the angle the fascicles insert into the tendon/fascia) may be better measures of muscle adaptation. Muscle bulk may increase by adding sarcomeres both in parallel and series, the latter obviously increasing the length of the fascicles. Interestingly, it is proposed that concentric training may result in increases in the pennate angle, while eccentric training will increase fascicle length. Bed rest will both decrease the muscle fascicle length and the pennate angle and result in a loss of muscle at a rate of 0.4% per day

(supposedly 2700 sarcomeres per day!) – but again, each muscle may respond differently to both training and bedrest. Similarly, tendon responds to training with increased tendon stiffness and increased tendon cross sectional area, while bed rest will reduce the stiffness of the tendon.

Rugby Injuries

"Improving Safety in the Rugby Tackle" seemed an unlikely topic for ACSM (despite that fact that the USA won the first Olympic Rugby medal). Unfortunately, other than an entertaining insight into the range of injuries sustained while on tour in New Zealand, this session failed to deliver anything novel.

It was always going to be tough convincing a non-surgeon, who has spent most of his career managing chronic overuse injuries, that ACL was really an interesting injury (Per Renstrom "ACL – The Most Intriguing Injury in Sport"). However, Per Renstrom, bringing a generation of experience and perspective, gave a very interesting talk on this, probably the most talked about sporting injury (at least at orthopaedic oriented sports medicine conferences). He highlighted (despite the progression in clinical surgical techniques) the lack of level one research in ACL (highlighting the recent and well documented randomised trial in NEJM showing no medium term differences between surgical and non surgical management of young non professional athletes as an exception). He spent some time discussing the importance of anatomical reconstruction of the ACL, but remained unconvinced as to the benefits of a double bundle reconstruction (as compared to a well performed single bundle), particularly when one considers the technical challenges, and the issues with re-do of double bundle surgery (a challenge our clinic has experienced). His conclusion that the choice of surgeon was perhaps more important than the choice of technique may well be widely shared! The challenges of return to play decisions remain, and again the lack of evidence was highlighted, but his feeling was that the function and "movement quality" needs to be a key consideration. Finally, he highlighted the risk of osteoarthritis post ACL reconstruction, which, given our failure to reduce the rates of primary ACL injury, continues to be a serious long term consequence of sports participation.

Genetics

Further developing the concept of the genetic associations of tendinopathy, this group (Malcolm Collins / Martin Schwellnus, Cape Town, South Africa) have extended their fascinating work into performance in endurance sport – using the high participation events in SA as their population (namely the two Oceans Ultramarathon and the SA Ironman). Continuing to focus on the COL5A1 gene they presented two posters suggesting that a TT genotype may increase mRNA stability, leading to increased Type V collagen production with subsequent increased fibril assembly and density. This may then result in altered mechanical properties of the musculo-tendinous unit with three outcomes:

1 Reduced flexibility,

2 Increased Achilles tendinopathy risk, and

3 Increased running economy and secondary performance.

A nice concept!

By contrast, in a session entitled "Ethiopian and Kenya Distance Runners: What makes them so good?", Prof Yannis Pitsilades from Glasgow highlighted how despite almost 10 years' work on the population genetics of these athletes, they are really no closer to identifying any population based differences in performance ability. They continue to pursue this tantalising question, but increasingly recognise the immensely important environmental factors such as socio-economic drive, diet, altitude and lifestyle.

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Not all wrist lumps are ganglia

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Introduction

Wrist lumps are commonly encountered in both general and sports medicine practice. Whilst the vast majority are benign and many are transient, there is a danger in viewing them all as insignificant, or quickly jumping to pre-formed conclusions about their nature. Practitioners should have a framework for evaluating these lesions such that an atypical presentation of a wrist lump will trigger appropriate investigations. This case illustrates a wrist lump initially misdiagnosed as a simple ganglion with important implications for the patient.

Case Report

A 37 year-old male university lecturer presented with a lump over his right wrist. Present for at least two years, it had been slowly enlarging over this time but was not painful unless knocked. He denied any altered sensation in his forearm, wrist and hand and had no restriction or pain with any hand or wrist movements. On examination he had a 3.5 cm x 2 cm mass situated over the dorsal ulnar border of his right wrist at the level of the ulnar styloid. It was relatively mobile and firm but not tethered, fluctuant or pulsatile. It was mildly tender on firm palpation but without radiation or paraesthesia elicited by this pressure. Power and sensation was normal in the hand and wrist. A further lump, 1.5 cm by 1.5 cm with a similar consistency to the first, was found on the medial aspect of his left upper arm 10 cm distal to the axilla and just medial to the body of biceps brachii, but he was less concerned about this one as it was not causing him any discomfort.

A diagnosis of ganglion was made for the wrist lump whilst the left arm lesion was suspected to be a fibroma. Given the stability of these lesions it was decided to monitor and to review if there was any change in them.

The patient re-presented three months later. Whilst there had been little or no change in the lesions, he was keen to proceed with definitive management as the wrist lump was more frequently being knocked and causing discomfort. The examination was unchanged. After discussing the procedure with him, aspiration was attempted under local anaesthetic in sterile conditions but despite several attempts no fluid of note was aspirated. The texture of the lump felt through the needle was different to what would be expected from a simple ganglion and in fact felt solid right through. The diagnosis was revised to unspecified soft tissue tumour and a referral was made to an orthopaedic surgeon for consideration of excision.
An MRI scan was subsequently performed clearly showing that the right wrist mass arose from the ulnar nerve and the left arm mass arose from the median nerve. They were reported as likely neurofibromas. The patient proceeded to uncomplicated surgical excision as a day case with histological analysis of both lesions being benign schwannoma.

Post-operatively, he had some mild tingling in the distribution of the dorsal branch of the ulnar nerve on the right and in a median nerve distribution on the left but otherwise had no change in his upper limb sensation or function. He was advised to monitor for further soft tissue masses as well as any new neurological symptoms.

Discussion

Whilst ganglia are the most common wrist lumps seen in general practice making up 60% of masses found around the wrist and hand,[9] there are certain characteristics that aid their diagnosis. Location is a primary consideration with 65% of ganglia situated in the dorsal wrist position overlying and arising from the scapholunate joint. A further 20-25% of ganglia are volar overlying the distal radius, with the other 10-

15% of ganglia arising from flexor tendon sheaths at other locations throughout the wrist.[7] An dorsal ulnar border wrist ganglion would be extremely rare other than if there was involvement of the extensor carpi ulnaris tendon sheath. Ganglia also have a characteristic texture being filled with mucoid fluid and are thus "ballotable", not a feature present in the case. A typical ganglion has a volume of around 0.95ml,[10] translating to a diameter of around 1.2 cm, and it is only rarely that they are larger than 2cm.[8]

The differential diagnoses for wrist lumps is expansive, but each has a characteristic history and examination. Giant cell tumours of the tendon sheath are next most common, being firm and always arising in the vicinity of tendons, usually flexor.[1] Aneurysms, more often pseudoaneurysms, are classically pulsatile, arise from arteries and are usually associated with trauma to the vessel. The common neural tumours of the peripheries are neurofibromas and schwannomas, the former incorporating many different types of nerve sheath cells and structural elements. Schwannomas are a homogenous benign tumour of Schwann cells, the myelin secreting cells of the nerve sheath.[5] Schwannomas, being rare, are frequently (as in this case) misdiagnosed, with up to 38% being misdiagnosed as ganglia.[6] Other less common masses include sarcomas, haemangiomas and granulomas.

Schwannomatosis is a recently described variant of neurofibromatosis characterised by having only peripheral schwannomas. Diagnosis of schwannomatosis requires two or more histologically confirmed non-intradermal schwannomas in a patient over 30 yrs old.[3] Multiple neurofibromas are the hallmark of type 1 neurofibromatosis and bilateral acoustic neuromas are typical of type 2 neurofibromatosis.[4] Schwannomatosis is thought to be due to a mutation in SMARCB1, a tumour suppressor gene, and is felt to be autosomal dominant. Familial cases are however relatively rare due to incomplete penetrance.[2] Upon diagnosing schwannomatosis it is important that patients are aware that while they may form schwannomas anywhere in the central nervous system, peripheral tumours are more common. Thus, any unexplained neurological symptoms should prompt early referral for appropriate investigation.

In this case, a soft tissue tumour should have been much higher on the initial differential. The clue to an underlying neural tumour was the additional mass also arising in the vicinity of a peripheral nerve. MRI is the diagnostic tool of choice for differentiating these masses,[11] particularly for the nerve sheath tumours. Ultrasound is a clinically reasonable convenient alternative. Attempted aspiration of non-pulsatile fluid filled lumps or fine needle aspiration of more solid lumps has the added benefit of potentially yielding a histological diagnosis.

This case illustrates the importance of keeping an open mind to the full differential diagnosis of lumps about the wrist. An appropriate evaluation may minimise the chance of misdiagnosing something rare, that we may encounter only once in our medical career.

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