NEW ZEALAND

Journal of Sports Medicine

Vol 39 • No 2 • 2012

Official Journal of Sports Medicine New Zealand Inc.

CONTENTS

EDITORIAL:
Mesmerising: The Art of Modern Sports Medicine 31
Bruce Hamilton

BEST OF BRITISH: 33
Chris Milne

ORIGINAL RESEARCH:
Site of acute hamstring strains and activities associated with injury: A systematic review 36
Richard N Storey, Mark D Stringer and Stephanie J Woodley

Can 2D video be used to screen dynamic lower extremity alignment in young athletes? 41
Chris Whatman, Patria Hume, Wayne Hing

Injuries in amateur representative rugby league over three years 48
Doug King and Trevor Clark

An investigation of surf injury prevalence in Australian surfers: A self-reported retrospective analysis 52
Rudi A Meir, Shi Zhou, Margaret I Rolfe, Wendy L Gilleard, Rosanne A Coutts

CASE REPORT:
Occult peripheral neuropathy in an elite hockey player 59
Jake Pearson , Stuart Mossman

HOW I TREAT:
Lateral Epicondylitis (Tennis Elbow) 61
Bruce Hamilton and Chris Milne
Comment 1 - *Jordan Salesa and Louise Johnson*
Comment 2 - *Paul Wharam*

CARDIOLOGY CONUNDRUMS:
The Black African Athlete's Heart 64
Michael Papadakis, Sanjay Sharma, Nathan Riding, Othman Salah, Bruce Hamilton, Hakim Chalabi and Mathew G Wilson

REPORTS:
International Society of Biomechanics in Sports (ISBS) 67
Chris Whatman

The Medical Road to the London Olympics 69
Lynne Coleman

ABSTRACTS:
Selected Abstracts from the 2012 NZ Sports Medicine Conference Handbook 71

PUBLISHER
SPORTS MEDICINE NEW ZEALAND
PO Box 6398, Dunedin
NEW ZEALAND
Tel: +64-3-477-7887
Fax: +64-3-477-4459
Email: smnznat@xtra.co.nz
Web: www.sportsmedicine.co.nz

ISSN 0110-6384 (Print)
ISSN 1175-6063 (On-Line)

EDITOR
Dr Bruce Hamilton

ASSISTANT EDITOR
Mr Chris Whatman

Contributors

Site of acute hamstring strains and activities associated with injury: A systematic review

Richard Storey

Richard Storey is in his fourth year of studying medicine at the Otago Medical School (Dunedin). He completed his research on the hamstring muscles in 2012 as a part of his honours year, and has a keen interest in anatomy particularly aspects which are clinically-relevant. His focus on sporting injuries stems from being an established hockey player, representing Southern (Otago and Southland combined) who play in the yearly National Hockey League.

Mark Stringer

Mark Stringer is Professor of Anatomy at the Otago School of Medical Sciences, University of Otago, Dunedin, New Zealand. He previously practiced as a paediatric surgeon in the UK, becoming Professor of Paediatric Surgery, before resuming a passion for clinical anatomy. Since coming to New Zealand he has maintained broad research interests in all aspects of clinical anatomy, including surface anatomy, anatomical errors in clinical practice, surgical anatomy, and anatomy education. He is Chairman of the Anatomy Committee at the Royal Australasian College of Surgeons and an anatomy examiner for the Royal Australian and New Zealand College of Radiologists.

Stephanie Woodley

Stephanie Woodley is a lecturer in the Department of Anatomy, University of Otago. Stephanie worked some years as a physiotherapist, which stimulated her research interest in musculoskeletal anatomy and its application to clinical practice. Her focus is on the hip and pelvic regions, including work related to hamstring strains, gluteal tendinopathy, trochanteric bursitis, and pregnancy-related symphyseal pain.

Can 2D video be used to screen dynamic lower extremity alignment in young athletes?

Chris Whatman

Chris Whatman is a senior lecturer in the School of Sport and Recreation at AUT University. He holds a Masters in Sports Physiotherapy from the University of South Australia and recently completed his PhD. Chris's research interests include the prevention of lower extremity overuse injuries, specifically looking at the assessment of lower extremity movement patterns. He maintains his links to industry as a consultant to the AUT Running Mechanics Clinic and is also on the national executive of Sports Medicine New Zealand.

Injuries in amateur representative rugby league over three years

Doug King

Doug King is the Lead Clinical Nurse Specialist in Minor Injuries at the Hutt Valley District Health Board Emergency Department. Doug has a Bachelor of Nursing and has completed post graduate studies in sports medicine. Completing his PhD in rugby league injury epidemiology in 2010 Doug is currently undertaking a second PhD in sport-related concussion specifically looking at concussion epidemiology, health professional knowledge, severity of impacts from match participation resulting in concussive incidents and sideline assessment tools for concussion in rugby union while working with amateur teams. Doug was a member of the NZRL medical panel focusing on team medic training and rugby league concussion. His research interests included sports injury epidemiology, tackle related injuries, amateur team medic skills and sport-related concussion.

Trevor Clark

Trevor Clark is a lecturer at the Australian College of Physical Education having completed his MSc while a professional rugby league player in the United Kingdom. Returning to New Zealand Trevor has worked with the NZ Warriors, North Harbour rugby, and as a sports science lecture at Wintec and Massey University. Trevor is currently completing his PhD at Massey University researching the effects of rugby league participation on the health and wellbeing of Māori males in New Zealand. Trevor has a background of coaching amateur rugby league teams at the local domestic, representative and International levels of participation. Trevor's research interests are sports psychology, injury prevention and performance enhancement in rugby league players.

An investigation of surf injury prevalence in Australian Surfers: A self-reported retrospective analysis

Rudi Meir

Rudi Meir is a senior lecturer at Southern Cross University and has extensive experience working as a specialist skills and conditioning coach in professional rugby league and rugby union. He has published widely on a range of topics related to sport science including the long-term consequences of sports injuries. His current research interests relate to enhancing sports performance and the development of reactive agility.

Shi Zhou

Professor Shi Zhou is an exercise physiologist at Southern Cross University. He was originally educated in China and obtained his PhD at The University of Melbourne. He has broad research interests in exercise science and currently focusing on neuromuscular and metabolic responses and adaptations to exercise and training.

Margret Rolfe

Margaret Rolfe is a Biostatistician based in Lismore (Northern NSW) working in the School of Health and Human Sciences at Southern Cross University, the University Centre for Rural Health- North Coast and is an Adjunct research Fellow at the University of Sydney. She has recently completed a PhD in Bayesian Statistics at QUT. Her research interests include chronic disease, Bayesian network models, application of multilevel models in sports science, geographic information system, longitudinal modelling, and health epidemiology.

Wendy Gilleard

Wendy Gilleard has focused her research interests in the field of biomechanics for 22 years and is currently based at Southern Cross University as an Associate Professor. She holds a PhD from the University of Sydney. Her primary research approach is the use of three dimensional motion analyses. She has supervised projects on a broad range of rehabilitation and sporting applications and reviews for a number of rehabilitation focused journals.

Rosanne Coutts

Dr Rosanne Coutts is an accredited exercise physiologist at Southern Cross University. She is a practitioner member on the committee of the National University Accreditation Program which is responsible for the accreditation of Sport and Exercise Science programs across Australia and Asia. Her research interests are broad within the Exercise Science arena; with a particular focus on the behavioural aspects.

Occult peripheral neuropathy in an elite hockey player

Jake Pearson

Jake Pearson is a sports physician practicing in Wellington. His scope of clinical practice remains broad, with a particular interest in tendon disorders. He also enjoys teaching and gets a lot of satisfaction from assisting people return to physical activity and function following injury or illness.

Stuart Mossman

Stuart Mossman is a Clinical Neurologist with a research position at Capital and Coast District Health Board with an interest in vestibular balance disorders. He has a private practice in Wellington.

Mesmerising: The Art of Modern Sports Medicine

One of the many things that first attracted me to work in Sports Medicine was the interesting and complex people we get to both work with and treat. As a result, practicing good Sports Medicine, like many of the specialties, requires that the practitioner hone the art of both verbal and non-verbal communication in order to optimise the consultation process.[1]

I have been fortunate enough over the last few years to travel and meet with many high profile Sports Medicine Practitioners that sociologist and outspoken critic of Sports Physicians John Hoberman, cynically refers to as the "celebrity sports physicians".[2] Celebrity Sports Physicians often utilise treatment modalities that are unique (and therefore lacking good scientific evidence base) which separates them from mainstream medicine practitioners - and they are often as famous as the athletes they treat. Word of mouth among athletes (and then via the media) is the preferred advertising modality, supported by strong links with coaches and administrators in specific sports. They are, by (often their own) definition, the source of the definitive medical opinion, and (I'm told) the opinion which the most elite of athletes will want to access. Typically, in our ultra-conservative medical establishment, our approach is to frown upon these "tall poppies" and to criticise from the distance. However, having spent time with a number of European practitioners that I would consider to fit the bill as "celebrity practitioners", there are some consistent and desirable traits to be observed (over and above the novel treatment modalities utilised, which is another discussion). While I am in no way a fan of the self promotion (and "to be sure" many other aspects) of this type of practitioner, I believe that consideration of some of the consistent elements of their clinical style is worthwhile, as from it we may pick up some positives for our own clinics. I write this as a dedicated cynic, but one who has returned from each of these clinical sojourns motivated to provide a better, not worse, service to athletes and patients.

Firstly, each practitioner works predominantly within only one or two sports, with great knowledge, enthusiasm and understanding of the demands of those sports. The passion for their sport is not derived from a calculated business model, but rather the business is derived from the passion and knowledge which was, in each case, a lifelong experience. With that lifelong involvement in the sport, came the gradual integration into the subculture of the sport, through constant involvement with athletes, who subsequently become coaches and administrators. While often outside the organised mainstream medical support for the sport, this long term involvement empowers the celebrity sports physician with immense credibility, within that sporting culture.

Secondly, "the Athlete comes first", is a total and fundamental commitment. This focus, combined with the personable, charismatic, self confident, energetic and sports specific knowledge exuded, convinces the patient that they are what is important. Not imaging. Not referral letters. Not record keeping. Not the waiting room. Not lunch, dinner or sleep. What the athlete needed was the focus, frequently at the exclusion and often to the detriment of all else. Compare this situation, with my last consultation where I was typing in the notes, looking at the imaging and rushing to see the next patient, and it is easy to understand who the patient would rather see. The unfortunate side effect of this focus is consistently long hours of work, with clinics just expanding to take the time required. The European version of the celebrity sports doctor would allegedly have no problem working until 2 or 3 in the morning when required.

Given my cynicism (based on the lack of evidence) of many of the treatment modalities employed, the positive clinical results that I have observed from these clinics places me in an academic conundrum. To escape this, it seems rational to consider that if it's not the prescribed treatment that's doing the job, then perhaps it's the "sell" which is the critical element. The consistent elements described above form what I believe to be a critical part of the sell, which we can incorporate into our own practice with both athletes and non-athletes. Indeed, while we were all taught this at our respective clinical schools, there is increasing evidence that the quality of the consultation process, can be a key determinant in the patient outcome.[3, 4] Moving away from the "doctor centred" biomedical model of consultation and towards a more holistic, patient centred model, may be all that these "celebrity physicians" are doing best. This then is me - someone with no artistic ability - trying to reinforce the importance of understanding the "art" of sports medicine.

There is of course the risk of placing too much emphasis on the art of the consultation, and using this as a rationale for neglecting a strong scientific patho-physiological approach to our management of injuries and illnesses. I don't want to be seen as the latest sucker in a long line of people to be "taken in" by what was first popularised by the German Physician Franz Mesmer (1734 – 1815). As a young man, Mesmer completed his doctorate (which must have made a fascinating read) entitled "on the influence of the planets on the human body". He then began to treat patients using techniques based upon his belief in a natural magnetism between both animated and inanimate objects, by gestures such as holding hands which (he alleged) allowed the transfer of energy and illness. This healing technique became known as "mesmerism". "Healing" a range of conditions reportedly involved grand, powerful convulsive cathartic experiences, and as his reputation developed so too did his healing power. Those poor unfortunates who could not afford his treatment were taught to hug trees (the original "tree huggers") in order to gain the same magnetic benefit. Mesmer, who in his time would almost certainly have classified as a celebrity practitioner, had a huge following. Reflecting the profile (or notoriety) that he gained, in the late 18th century the French government appointed a high level investigation into the scientific credibility of "mesmerisation". This investigation team included such identifiable names as Antoine Lavoisier and Benjamin Franklin, and unfortunately for Mesmer, they firmly and definitively refuted both the underlying science and the reported clinical benefits of his "healing" methodology. Despite this, over the last 200 years, the concepts of Mesmer have morphed in various different ways, surviving today as both the lay concept of being "mesmerised", and in numerous forms of alternative medicine. How much of our holistic approach to the consultation then, is rooted in the same factors that enabled Mesmer, with such a seemingly implausible treatment approach, to be so popular?

Lest I be considered otherwise, I need to state that I am not (routinely anyway) an advocate of unsubstantiated therapies, working one's self to an early grave, working in clinical isolation with poor communication between practitioners, or paying lip-service to scientific methodology. I am however an advocate of good clinical practice, and the patient centred, energetic, confident and knowledgeable approach of the celebrity sports practitioner is engaging, and I'm convinced, beneficial. Whilst it may be a hangover from a long discredited medical practice, elements of the "mesmerising" impact of a good consultation may ultimately be what we can learn from our much maligned celebrity colleagues.

References

1. Heath C. Participation in the medical consultation: the co-ordination of verbal and nonverbal behaviour between the doctor and patient. *Sociology of Health and Illness*. 1984; **6**: 311-338.
2. Hoberman J, Mortal Engines: The Science of Performance and the Dehumanization of Sport. First ed. 1992, New York: The Free Press. 374.
3. Little P, Everitt H, Williamson I, Warner G, Moore M, Gould C, Ferrier K,Payne S. Observational study of effect of patient centredness and positive approach on outcomes of general practice consultations. *British Medical Journal* 2001; **323**: 908-911.
4. Thomas K. General practice consultations: is there any point in being positive? *British Medical Journal*. 1987; **294**: 1200-1202.

LONDON 2012

What a fantastic Olympic Games. Having had the privilege of being in London as a tourist for the Olympics, I can say first hand that it was a supreme effort from the Brits in hosting the 24th Olympics. Not only did they put on an amazing spectacle, but as the home nation they performed above expectations. As of course, did the kiwis, in both the Olympics and Paralympics. My congratulations go out to all members of the team, and in particular to the medical support team who played such an important role in the success. This is no easy task.

There were obviously many highlights, and we will all have our own special moments. My favourite moment was watching the mens 800m final (live), won in a world record by David Radishe (1.40.91)– but not only that, there were six other PB's and one seasons best in the field of eight – what an amazing spectacle. It would perhaps be wrong to call it a race, as it really was a procession, but the quality was fantastic. Out of interest (there's always a kiwi angle), its 50 years since New Zealands Peter Snell claimed the world record with a time of 1.44.3 in Christchurch in 1962, and Radishe is the 7th individual to hold the record since Snell!

It's now less than four years to the next Olympics, and planning will have already begun. Bring on Rio.

CHRIS MILNE

APRIL

THE APRIL 2012 EDITION concentrated largely on cardiac issues which have been very much to the fore in the last year or two. Jonathan Drezner and colleagues looked at the accuracy of ECG interpretation in competitive athletes and studied the impact of using standardised ECG criteria. They found that providing standardised criteria to assist with ECG interpretation significantly increased the ability to distinguish normal from abnormal findings. Prior to training accuracy was 73% in GP registrars and GPs, and 78% for sports physicians, compared with 85% for cardiologists. Following relatively brief training and application of a two-page ECG criteria tool, these figures improved to 92% for the GP registrars, 90% for GPs, 91% for sports physicians and 96% for cardiologists. These figures are impressive and doubtless we will hear more of this as Dan Exeter, one of the ACSP registrars, is conducting a study in ACSP Fellows and registrars.

In the same issue is an article entitled 'Sudden cardiac death: Mandatory exclusion of athletes at risk is a step too far'. Co-written by Lynley Anderson from the University of Otago, Dan Exeter and Lynne Bowyer, this article points out that while screening can provide useful information to at-risk athletes, the authors argue that making decisions about their future athletic career, in particular mandatory exclusion, is paternalistic and such decisions are not rightfully within the domain of medicine. A very useful, thought-provoking article for all clinicians.

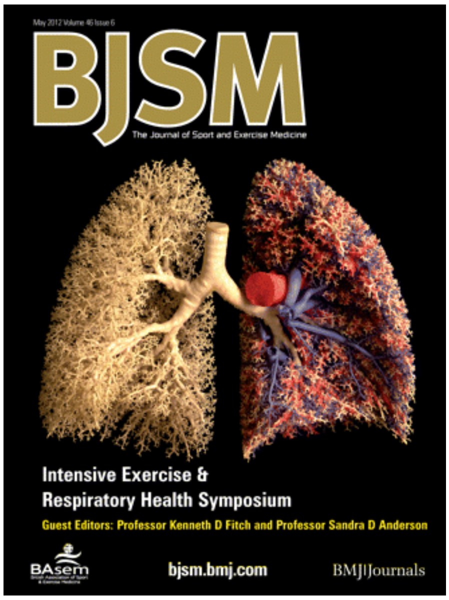
Again in the same issue there is an article looking at the prevalence of ECG abnormalities in West Asian and African male athletes, performed by a combined group from Qatar, the UK and France. Your editor, Bruce Hamilton, was one of the co-authors. They found that West Asian and Caucasian athletes demonstrated low rates of abnormal ECG findings compared with black African athletes. Future work should look at the genetic mechanisms behind these ECG and myocardial adaptations and, in particular, in New Zealand we need some data from our Polynesian athletes.

The editorial in this issue was entitled 'Repudiation of the magic bullet approach to health improvement: A call to empower people to get moving and take charge'. Everyone is looking for the magic bullet, and the magic bullet happens to be exercise. The challenge now is to keep spreading the message and motivate people to become more active. More of this in the June IOC issue (see below).

There was a useful review article by King and colleagues from Brisbane regarding exercise, appetite and weight management. It concentrated on the variable compensatory responses and eating behaviour and how these contribute to variability in exercise-induced weight loss. Gastric emptying, appetite peptides, substrate oxidation variation and behavioural traits all interact to influence how exercise affects appetite and weight loss.

MAY

THE MAY ISSUE could well be termed the asthma issue; it contained eight papers from a Symposium on Respiratory Health at the IOC Conference held in Monaco in 2011. Starting with physiology, Don McKenzie, who will be well-known to New Zealanders, comments that whilst the lungs have adequate reserve capacity in the average individual, in elite endurance and trained athletes a lack of adaptation can be a limiting factor in maximal performance. Limitations can include exercise induced arterial hypoxaemia, vocal cord dysfunction, expiratory flow limitation and respiratory muscle fatigue.



In the next article Sandra Anderson, another person well-known to New Zealanders, and her colleague Pascale Kippelen discuss airway injury during high level exercise. This is usually because of dehydration, stress and mechanical stress during the high level breathing required for intense exercise.

Later in the same issue Ken Fitch, another leader in the field, gives a useful overview of asthma and airway hyperresponsiveness in Olympic athletes. During his long career he notes that management of asthma has improved with use of inhaled corticosteroids in addition to beta agonists, and athletes with asthma have now been able to consistently outperform their peers. Inspiring polluted or cold air is a significant factor in some, but not all, sports and is highly dependent on where the athlete trains.

Boulet examines cough and upper airways disorders in elite athletes. He covers the main area of upper respiratory tract infections, rhinitis (both allergic and non-allergic) and vocal cord dysfunction. This is a very useful article for clinicians managing athletes with respiratory disorders.

As part of the A to Z of Nutritional Supplements, Castel and colleagues look at protein in this issue. They propose the following strategies to allow repair, remodelling, adaptation and gains in lean mass in athletes:

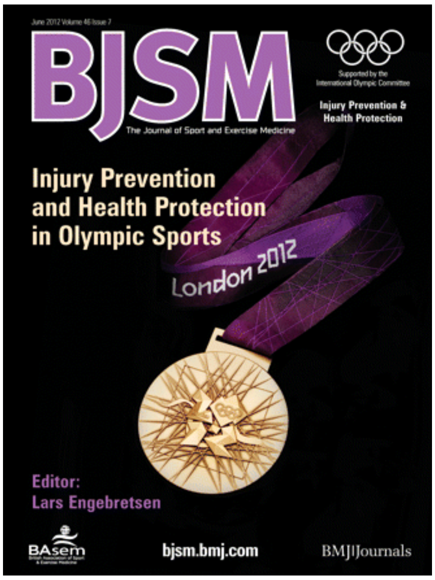
1. Daily intakes higher than the RDA (1.2-1.6g/kg/day)
2. Emphasis on dietary source proteins enriched in leucine
3. Consume protein in doses of 20-25g per serving to maximise adaptive responses
4. Equally spaced protein meals throughout the day
5. Consumption of protein immediately after exercise

JUNE

THE JUNE IOC ISSUE OF BJSM continued the food theme with an article by Roy Shephard on physical performance and training response during Ramadan. This is relevant as Ramadan occurred during the 2012 London Olympics, however it didn't stop Mo Farah, a Muslim, winning two gold medals in endurance events. There was also a summary statement of an IOC workshop on the issue as part of the editorial at the start of the issue.

Michael Evans, a Canadian, gives his top 10 learnings about making a health message that people give to one another. He prepared a video entitled '23 and 1/2 hours' and posted it on YouTube in December 2011; by February 2012 it had over 2 million hits. Amongst his 10 points is the observation that stories trump data and to think in a collaborative fashion when you make health media. He also explains that we need to tell people why rather than just what so the message has more focus. The video link is http://www.youtube.com/watch?v=aUaInS6HIGo. Watch it yourself. It only takes a couple of minutes and then you will know what your patients are commenting on. It is a superb example of how we can engage and support people where they are.

Later in the same issue is an article by Kristian Thorborg, who spoke at the SMNZ Conference last year, entitled 'Why hamstring eccentrics are hamstring essentials'. He points out that Nordic hamstring prevents injuries and reinjuries. The programme should be titrated during the mid-season break, starting with one session in Week 1, two sessions in Week 2 and three sessions from Weeks 3-10 before continuing with a maintenance programme of one session per week after Week 10. Well worth a read if you are dealing with teams of athletes who have hamstring injuries.



In this IOC issue an excellent summary of the health issues faced by Olympic athletes at the Beijing Summer Games in 2008 and the Vancouver Winter Games in 2010. Analysis of the comprehensive injury and illness data revealed that between 7 and 11% of all athletes incurred an injury or suffered from at least one illness during the Games. The surveillance system was continued during the London 2012 Olympics.

This issue also contained an excellent summary article on optimising the respiratory health of elite athletes. Drawing on the contributors to the Respiratory Symposium mentioned in the May issue, the authors proposed the following strategies to reduce the impact of environmental factors on airway injury:

1. Limiting exposure to cold air
2. Increasing the water content of the inspired air
3. Avoiding byproducts of chlorination, especially nitrogen trichloride
4. Avoiding other airborne pollutants by exercising away from major highways or city centres, and planting trees adjacent to high traffic areas

They also mention pharmacological management where the above measures fail.

Of particular interest to New Zealand readers is a study of illness during the 2010 Super 14 rugby tournament. This was a prospective study involving nearly 23,000 player days and involved all teams. The common illnesses were the expected respiratory and GI tract infections plus skin conditions. The incidence of illness was 20.7/1000 players days. The authors suggest that dermatology be included in the team physician training programmes offered by organisations and international federations.

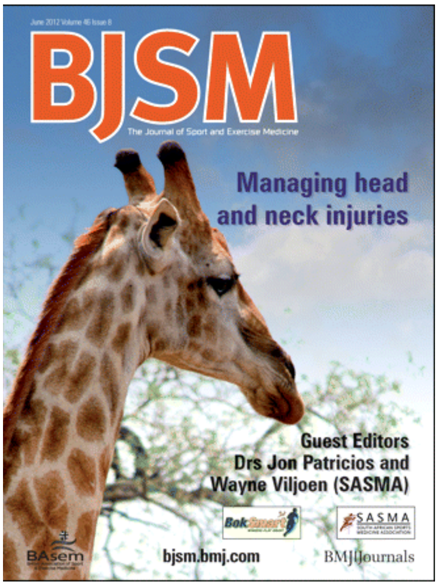
Ankle injuries are an ongoing concern. A systemic meta analysis by Australian authors including Peter Blanch, who will be familiar to New Zealand physiotherapists, found that higher postural sway, lower postural stability, lower inversion proprioception, lower eccentric eversion strength at slower speeds and higher concentric plantarflexion strength at higher speeds were all associated with significantly increased risk of ankle injuries. These then are the things we should work on in the off season to minimise time out of competition.

Finally in this issue, there was an excellent article on the mechanisms of traumatic shoulder injury in elite rugby players. A UK group identified three common mechanisms:

1. The try-scorer mechanism - where the flexed arm of a player driving forward is driven over the head by contract with the ground
2. The tackler mechanism - where the abducted arm of the tackling player is forcibly extended behind the player
3. The direct hit mechanism - where medially directed compressive force to the adducted arm by contact with another player or the ground results in injury

There are excellent diagrams of each of these three mechanisms and the challenge now is to come up with preventive strategies to minimise episodes of shoulder instability in rugby players.

Between 7 and 11% of all athletes incurred an injury or suffered from at least one occurrence of illness during the Games ... risk factor and injury mechanism analyses in high risk Olympic sports are essential to better direct injury prevention strategies.



JULY

IN THE JULY ISSUE there was an emphasis on head and neck injury. Kuster and colleagues from Scotland challenged the established dogma that hyperflexion is the dominant mechanism of cervical spine injury in rugby. Instead they propose that buckling is the primary mechanism of cervical spine injury, and this in accord with the observation that facet dislocations have been reported to occur most often within the lower cervical spine, primarily C4/5 and C5/6. Kuster and colleagues used a lot of video analysis and also cadaveric studies.

Cripton and colleagues from the Department of Mechanical Engineering at the University of British Columbia challenged this analysis and comment that ex vivo cadaveric studies have significant limitations. They offer an alternative biomechanical engineering based perspective. They comment that "what exactly is happening in the in vivo spine during cervical injury remains unclear". In summary, they believe it is premature to abandon the hyperflexion concept of injury as a major contributor to cervical spine injuries in rugby.

We are all interested in what makes champions, and a South African group headed by Ross Tucker from Cape Town conducted a review of the relative contribution of genes and training to sporting success. They comment that elite performance is a polygenic trait. Sex, height and VO2max are three characteristics that are genetically determined. They challenge the dogma that 10,000 hours is required for expert performance, and the concept of talent transfer suggests that a more broad view of training and practice may be required for certain sports. This seems to be the way the world is moving and many countries, including our own, have tried to identify potential elite competitors, particularly in emerging sports. The inclusion of Women's Sevens rugby in the 2016 Rio Olympics has been a spur to identify talented female athletes (in particular in New Zealand) who could be part of that team.

Asymptomatic status following sports concussion can be a misnomer, as exercise can provoke symptoms in non-concussed individuals. Paul McCrory plus two colleagues from the School of Physiotherapy at the University of Otago examine this in depth and conclude that there is a need to operationally define the term asymptomatic, particularly when making return to play decisions following a sports concussion. Watch this space for further discussion of this area at international concussion meetings.

Patellofemoral pain is the single most common problem in sports medicine, and knee braces and foot orthotics have been proposed as part of the solution. Swart and colleagues form the Netherlands found moderate evidence for no effect from knee braces and conflicting evidence for tape and foot orthotics. By contrast, exercise, e.g. the McConnell regime, is a well-proven, long-term solution for patellofemoral pain. My own experience has been that orthotics have the most to offer in those athletes with excess subtalar joint pronation, as one might expect.

Plenty of reading in these four issues, and it is good to see our New Zealand researchers getting their material published in this major journal.

Site of acute hamstring strains and activities associated with injury: A systematic review

Richard N Storey

Mark D Stringer, MS, FRCP(UK), FRCS(Engl)

Stephanie J Woodley, PhD, MSc, BPhty

Department of Anatomy
Otago School of Medical Sciences
University of Otago
PO Box 913
Dunedin 9054
New Zealand

Correspondence
Stephanie Woodley
Department of Anatomy
Otago School of Medical Sciences
University of Otago
PO Box 913
Dunedin 9054
Email:
stephanie.woodley@anatomy.otago.ac.nz
Tel: +64 3 4697353
Fax: +64 3 479 7254

ABSTRACT

Aim: To systematically review the literature and provide an overview of the precise location of acute hamstring muscle strains and the activities associated with injury.

Data sources:

Medline and EMBASE (1947 to August 2012) databases and reference lists of relevant studies were searched.

Study selection:

Peer-reviewed articles documenting acute hamstring strain injuries verified by cross-sectional imaging (computed tomography, magnetic resonance imaging, ultrasound).

Data extraction:

One reviewer initially screened each title and abstract and two reviewers independently extracted the data.

Data synthesis:

Of the 1069 screened articles, 15 studies fulfilled our inclusion criteria. These studies reported a total of 732 imaging-confirmed hamstring strains in 632 individuals, predominantly young adult males. Two-thirds of strains (67%) affected a single muscle; 27% of participants had injuries that involved more than one muscle. In nine studies that provided sufficient anatomical detail, the musculotendinous junction (MTJ) was the site of injury in 72% of strains, most often the proximal MTJ. The site and activity associated with hamstring strains appear to be related: sprinting is commonly associated with strains of the long head of biceps femoris with or without additional pathology in semitendinosus. Slow-speed stretching injuries predominantly affect semimembranosus but can also involve adjacent muscles such as quadratus femoris or less commonly, adductor magnus.

Conclusions:

A relationship between the anatomical site and activity associated with hamstring injury is emerging as more studies report detailed findings using modern imaging techniques. Together with biomechanical studies, these data are improving our understanding of acute hamstring strains which should allow better clinical evaluation, and help with the development of more targeted therapy and prevention strategies.

Key words:

Athletes, magnetic resonance imaging, muscle, tendon, ultrasound

INTRODUCTION

Acute hamstring strains are frequent sporting injuries.[22,26,41] Epidemiological studies show that hamstring strains affect between 3.4% and 19% of athletes each season, with the highest rates recorded in sports involving sprinting, kicking and jumping.[1,22,29,41] These injuries impose a major financial burden on professional sporting organisations; Australian football, English football and English rugby union clubs report between 5 and 7.5 hamstring strains per club, per season.[8,27,41] Each season this amounts to an average of 123 missed days per club in professional English rugby union,[8] 21 missed matches per club in the Australian Football League,[27] and 90 days or 15 matches per club in English football.[41] Despite rehabilitative efforts, repeat strains are common, with a recurrence incidence of between 13.9% and 63.3%.[18,21,23,26,31,39]

Although diagnosis of a hamstring strain is usually based on findings from clinical examination,[13] additional investigations such as magnetic resonance imaging (MRI) (Figure 1) and ultrasound enable identification of the precise location, severity and extent of the injury. Several morphological factors have been identified that influence the time taken to regain pre-injury level of function following a hamstring strain. These include the specific muscle that has been injured,[4-6,10] the site of injury within the muscle-tendon complex[2-4,6] and the extent of injury.[10,18,30,32] Therefore, an understanding of the exact location of hamstring strain and pattern of injury is essential to inform clinical diagnosis and rehabilitation strategies. The aim of this systematic review is to provide a description of our current understanding of the precise anatomical sites of hamstring strains, and document the activities associated with injury.

Figure 1. *Axial (A) and coronal (B) T2-weighted fat saturated MR images of a 10-day old strain to the proximal MTJ of biceps femoris long head in a professional rugby player; note the feathered hyperintensity (arrows) extending from a tear in the proximal MTJ. (C) Axial proton density weighted image showing complete disruption of the MTJ with hyperintensity progressing into semitendinosus (3 day-old injury). (D) Sagittal STIR image also showing extensive muscle tearing, haemorrhage, and associated perimyseal oedema. The arrows show tearing along the common biceps femoris/ semitendinosus tendon at the MTJ. (BFlh) Biceps femoris long head. (ST) Semitendinosus. (SMt) Semimembranosus tendon. Images courtesy of Dr Grant Meikle, Dunedin Hospital.*

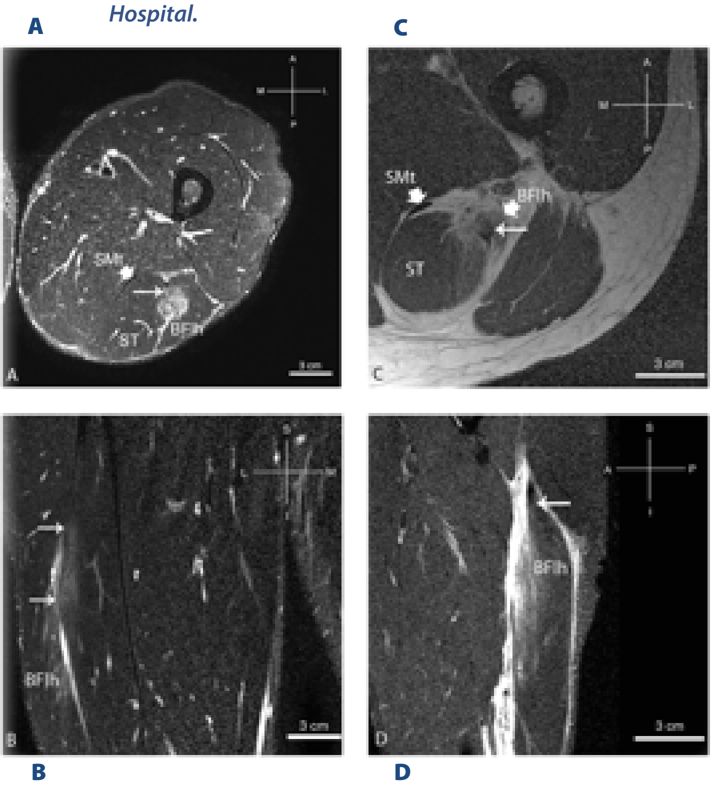
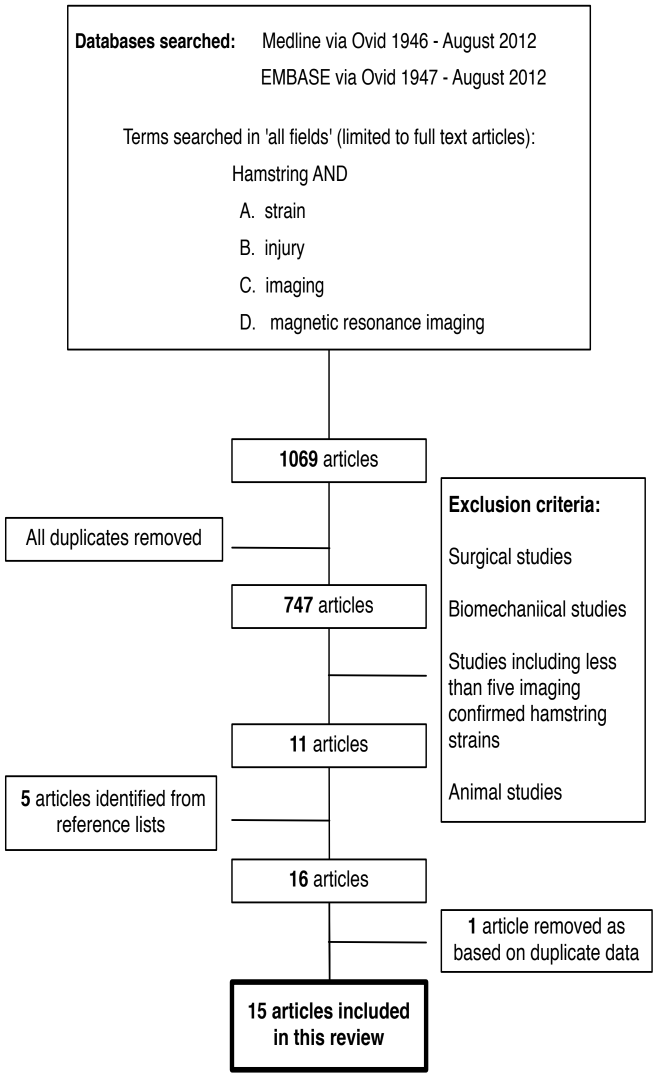


Figure 2. *Summary of search method*



METHODS

Searches of Medline (1948-August 2012) and EMBASE (1947-August 2012) electronic databases were undertaken using Ovid. The following terms were searched in 'all fields': "hamstring strain", "hamstring injury", "hamstring and imaging", and "hamstring and magnetic resonance imaging". Peer-reviewed papers were considered eligible for inclusion if they (1) documented the location of acute hamstring strain injuries confirmed by computed tomography, MRI or ultrasound and (2) were written in English. Avulsions, subacute injuries, chronic hamstring tendinopathies and recurrent strains were excluded, as were small studies that included less than five hamstring strains verified by imaging, or whose focus was surgical, biomechanical or animal-based research. Reference lists from retrieved articles were also searched to optimise coverage. An overview of the search strategy is shown in Figure 2.

One reviewer (RS) screened all titles and abstracts for eligibility. Papers meeting the inclusion criteria were analysed independently by two reviewers (RS and SW), with any disagreement resolved by consensus. The following data were extracted: the total number of hamstring strains confirmed by imaging; which particular hamstring muscle or muscles were strained; the specific site in the muscle-tendon complex that was affected; the activity associated with the injury; and various demographic details such as age and gender.

RESULTS

A total of 1069 articles were identified; after removing duplicates and applying exclusion criteria, 11 remained. Five additional articles were identified by a hand search of reference lists, giving a total of 16 papers. However, one of these studies[21] was excluded as it was based on duplicate data[10] (Figure 2). The 15 studies that satisfied the inclusion criteria are summarised in Appendix 1. Collectively, 1579 subjects were included within the 15 studies, 1052 of whom had a clinically diagnosed acute hamstring strain(s). Of these, 632 individuals had a total of at least 732 discrete hamstring injuries confirmed by imaging. The latter is an underestimation as, in the event of injury to more than one muscle, some authors did not specify the exact number of injuries or their distribution across muscles, only recording the principal muscle injured (that which displayed the

TABLE 1. ***Muscles affected in acute hamstring strain***

AUTHOR	Biceps femoris		Semitendinosus		Semimembranosus		Unspecified	TOTAL
	Isolated	Combined	Isolated	Combined	Isolated	Combined		
Garrett et al.[17]	5	2		2	1			10
Pomeranz and Heidt[30]	6		1		5			12
Speer et al.[33]	11		2		4			17
Brandser et al.[7]							5	5
De Smet and Best[12]	6	5	3	5	1			20
Slavotinek et al.[32,a]	14	12	3	12		2		43
Koulouris and Connell[20]	118	6	8	1	16	5		154[b]
Verrall et al.[38,a]	32	23	4	26	5	5		95
Connell et al.[10,a]	35		2		3		2[c]	42[d]
Gibbs et al.[18,a]	8	5	2	5	2			22
Verrall et al.[39,a]							26[e]	26
Askling et al.[4]	10	8		7			1	26
Askling et al.[6]					2	11		13[f]
Askling et al.[5]		4		6	16	9		35
Ekstrand et al.[13]							180[g]	180
Subtotals	245	65	25	64	55	32	214	
TOTALS	310		89		87		214	700

a Studies that examined injuries in Australian football players;
b The location of 15 additional strains was not reported;
c Two participants had injury to >1 muscle, these muscles were not described;
d As reported at baseline MRI scan;
e Authors state that biceps femoris was the 'principal muscle' injured in 26 participants. From the data presented, the number of isolated and combined injuries can not be determined: 17 participants sustained injury to a single muscle and 13 participants had combined injuries (other muscle involvement has not been described) giving a total of at least 43 strains;
f Data were taken from the first (n=11) or final (n=2) scans;
g Number of combined injuries not known: if more than one muscle was injured, the muscle with the most extensive pathology was deemed the primary muscle.

TABLE 2. ***Structures affected in hamstring strains***

AUTHOR, DATE	Biceps femoris			Tendon		Muscle Belly	TOTAL
	Proximal	Distal	Unspecified	Proximal	Distal		
Pomeranz and Heidt[30]	2	2		1		7	12
Brandser et al.[7]	5						5
De Smet and Best[12]	9	6	5				20
Slavotinek et al.[32,a,b]	1	3	24				28
Koulouris and Connell[20,a,c]			98			21	119
Connell et al.[10,a,b]			22				22
Askling et al.[4,c,d]	12	6		6	2	2	28
Askling et al.[6,c,e]	2			2			15
Askling et al.[5,e,f]	1			16			26
TOTALS	32	17	149	45	2	30	275

a Studies that included Australian football players;
b Data relate to number of participants rather than number of strains;
c Some participants presented with injury to more than one structure;
d Studies that included sprinters or edocumented slow-speed stretching injuries;
f Unable to fully interpret data.
MTJ: musculotendinous junction.

most extensive pathology).[10,13,39] With regard to demographics, males comprised at least 74% of the sample in 12 of the studies, with seven studies exclusively recruiting men. Variation was evident in the reported age ranges of participants, with two studies documenting mean values of 28.2[20] and 23.6 (standard deviation 3.2) years,[39] respectively (Appendix 1).

Muscle(s) injured

After excluding 214 cases where the affected hamstring muscle was unspecified or it was not clear which muscle(s) was implicated in isolated or combined injuries, the majority of acute hamstring strains (63.8%) involved biceps femoris (Table 1). Thirteen of the 15 studies reported that biceps femoris was the most likely muscle injured in a hamstring strain, as confirmed by imaging.[4,7,10,12,13,17,18,20,30,32,33,38,39] A distinction between the two heads of the biceps femoris muscle was made in fewer than half of the studies. In these six studies, the long head of biceps femoris was affected in 93% of 71 strains.[4,5,10,12,17,32] Indeed, only six injuries to the short head of biceps femoris were reported.[32] In two studies, both published by Askling et al,[5,6] semimembranosus rather than biceps femoris was most frequently injured but this was in the context of slow-speed stretching activities mostly in female dancers, rather than in relation to rapid powerful movements, which is the predominant mechanism in most other studies.

There was less consistency about the muscle that was next most likely to be injured in an acute hamstring strain. Collectively, semitendinosus was affected in 18.3% of cases while semimembranosus represented 17.9% of the total injuries (Table 1). In the 15 studies, 38 other pathologies were found within muscles not classified within the hamstring group. These were mostly quadratus femoris strains (61%) occurring during extreme hip movement in slow-speed stretching, and non-specific strains to adductor magnus, gluteus maximus and vastus lateralis.[5,6,30,32,38,39]

In participants with an isolated hamstring strain (n ≈ 325, 67%), the frequency of muscle involvement was as follows: biceps femoris 75.4%, semimembranosus 16.9%, semitendinosus 7.7% (Table 1).[4-7,10,12,13,17,18,20,30,32,38,39] In addition to acknowledging that strains affect more than one hamstring muscle[13] twelve other studies addressed combined injuries. Two of these did not state which muscles were involved when more than one muscle was injured.[10,39] Considering the remaining ten studies, multiple strains occurred in 97 (27%) individuals. However, there were marked differences between individual studies: seven of the ten studies reported participants with two or more hamstring strains in 25-50% of cases[4,5,12,17,18,32,38] in contrast to one study where this figure was less than 5%;[20] and in another (retrospective study) was 100%.[7] Askling et al.[6] reported strains affecting multiple muscles in 85% of patients with slow speed stretching injuries. In six of the ten studies, three involving Australian Football league players, two in mostly track athletes, and which only included sprinters the long head of biceps femoris was most frequently injured in combined pathologies, with semitendinosus the next most likely muscle to be injured.[4,12,18,32,38] One study, reported injury to the conjoined tendon of biceps femoris and semitendinosus;[7] another, which included cricketers and waterskiers, found semimembranosus to be next in frequency after biceps femoris.[20] In two reports, where semimembranosus was most commonly injured (83% and 87% of cases), additional muscle strains usually affected quadratus femoris (27% and 87% of cases) and/or adductor magnus (10% and 33% of cases);[5,6] both studies focused on slow stretching type strains and found that three separate muscles were injured. There was no report of acute synchronous injury to all three hamstring muscles.

Structure and site affected

Nine imaging studies described which region of the muscle-tendon complex was affected by injury (Table 2).[4-7,10,12,20,30,32] The musculotendinous junction (MTJ) was involved in 198 (72%) of 275 strains confirmed by imaging[4-7,10,12,20,30,32] but in three studies more precise details were not reported.[10,20,32] In the two studies focusing on slow-speed stretching injuries the proximal tendon was affected in 38 (93%) cases.[5,6] Other sites of tears are shown in Table 2. With respect to the general location of all strains, nine studies categorised injuries as occurring in proximal, middle or distal parts of the muscle, but these regions were poorly defined if at all. Of the 334 strains classified in this way 151 were proximal, 105 middle, and 78 distal.[4-7,12,20,30,32,38]

Activities associated with injuries

With regard to sports-specific injuries, Australian football is the code that has been most extensively investigated to date (seven studies), with other studies focusing on track athletes, dancers and European footballers (Appendix 1). In the five studies that exclusively recruited Australian football players, isolated hamstring strains mostly affected biceps femoris (89/110, 80.9%) whilst semitendinosus and semimembranosus had similar rates of injury (11/110, 10.0% and 10/110, 9.1%, respectively).[10,18,32,38,39] With regard to synchronous injuries in this group of athletes, there are insufficient data to determine the muscle (semitendinosus or semimembranosus) most likely to be injured in association with biceps femoris long head.[10,32,38] The proximal or distal MTJ is the site of injury in as many as 93% of cases.[32]

Both Garrett et al.[17] and Askling et al.[4] investigated sprinters and showed that acute hamstring strains in these athletes tended to affect the long head of biceps femoris with or without the involvement of semitendinosus. There are limited data on hamstring strains in track and field jumpers. De Smet and Best[12] noted that all three athletes with an isolated injury to semitendinosus were track and field jumpers; injuries were located at either the distal MTJ (two injuries) or the proximal intramuscular MTJ (one injury). The specific site of injury for the other 12 athletes involved in this study was not stated.

Hamstring strains in slow-speed stretching activities such as dancing have been investigated by one group of researchers. In a cohort of dancers performing modern and classic styles, the most commonly injured muscle was semimembranosus (13 of 15 cases).[6] In a subsequent study of athletes incurring an acute hamstring strain during movements involving extreme joint angles (sagittal split, side split, stretching and high kick) semimembranosus was again the most commonly injured hamstring muscle (25 of 30 cases).[5]

DISCUSSION

The aim of this review was to provide a summary of the current evidence relating to the anatomical sites of hamstring strain, and to comment on the activities associated with injury. Our search strategy identified fifteen relevant studies, which investigated predominantly male athletes.

Muscle(s) injured

Reporting of muscle involvement was usually consistent, particularly for an isolated hamstring strain. The long head of biceps femoris is the most frequently affected hamstring muscle in acute strains, mainly in association with powerful movements such as sprinting. Semimembranosus appears to be more at risk of injury in slow-speed stretching type strains. De Smet and Best[12] were the first to report acute hamstring strains affecting more than one muscle. Since all hamstring muscles are subject to overstretching or excessively forceful contractions, it is not surprising that more than one muscle may be injured synchronously. With one exception,[17] studies published before 2000 only reported strains to a single muscle,[7,12,30,33] but this may reflect the limitations of imaging techniques that were available a decade or more ago. The pattern and frequency of combined acute hamstring strains appears to be related to the mechanism of injury.

Structure and site affected

The MTJ acts as an interface between two diverse, yet synergistic tissues. Biomechanically, this area involves a concentration of tensile forces, marking an abrupt change in the elastic modulus of the tissues and representing a point of maximum stress in the skeletal muscle-tendon unit.[28,37] Studies by Garrett et al.[16] and Noonan and Garrett[25] show that strains produced by excessive active or passive stretch are most likely to occur near the MTJ i.e. where skeletal muscle fibres insert into the collagen of a tendon.[35] The findings of this review concur that this is the case for active strains in the long head of biceps femoris. However, the outcomes are not so certain for semimembranosus. Semimembranosus, more often injured in extreme passive stretching movements, seems more likely to strain at its proximal free tendon than its proximal MTJ. There may be specific anatomical differences between the architecture of the MTJ in these two muscles, which could account for these distinct sites and mechanisms of injury.

There is a direct relationship between the length, cross-sectional area, and volume of the strain injury, as determined by MRI, and the duration of the recovery period.[10,32] It is also well known that different tissues heal at different rates.16 It is therefore surprising that many studies fail to report which specific structures within the hamstring muscle-tendon complex are involved in the acute injury. Pomeranz and Heidt[30] suggested several physical factors associated with a prolonged recovery including complete tendon or MTJ rupture and distal tendon or MTJ lesions. In contrast, in sprinters at least, the closer the lesion to the ischial tuberosity (determined by palpation or MRI), the longer the recovery time.[4] In this same study it was also noted that strains involving the free proximal tendon were associated with longer recovery times to pre-injury status compared to strains involving the MTJ or muscle alone (35 vs 14 weeks, respectively).[4] Injuries to free tendons at other sites such as the Achilles tendon, patella tendon and supraspinatus are also often associated with prolonged periods of rehabilitation.[4]

The anatomy of the muscle and mechanism of injury also influence healing and recovery. A comparison between sprinting strains (affecting the long head of biceps femoris) and slow-speed stretching injuries (affecting semimembranosus) show a median return to pre-injury status at 16 and 50 weeks, respectively.[4,6] There may be additional anatomical factors such as blood supply of the tendon or MTJ that influence the speed of recovery.

Activities associated with injury

Results from this systematic review suggest that different sports predispose to different patterns of acute hamstring injury, although more data are required to confirm these correlations. Australian football involves sustained periods of sprinting, kicking and jumping. Its injury profile is comparable to that of soccer and rugby union[27] although the mechanisms for hamstring injuries may differ between these sports.[13] Like sprinters, biceps femoris is most often injured in Australian footballers. One investigation of the mechanism of hamstring strains in elite Australian footballers found that 73% occurred during running or sprinting whilst the player was accelerating rapidly.[14] Biomechanical analyses of running and sprinting have estimated that the biceps femoris muscle-tendon unit sustains the greatest stretch of all three hamstring muscles. During treadmill sprinting, the biceps femoris muscle-tendon unit stretches by an average of between 9.5 and 12% of its length as compared to 8.1% for semitendinosus and 7.4% for semimembranosus.[19,36] Thelen et al.[36] noted that peak muscle-tendon lengths were significantly greater in biceps femoris than in semitendinosus and semimembranosus although these did not increase linearly with faster running speeds. These maximal measurements occurred just before heel strike, when the hamstrings are strongly contracting eccentrically, a period frequently identified as the likely moment of injury during running.[9,19,36] Eccentric contraction is well established as a cause of muscle strain,[15,25,36] related to the magnitude of force, rate of force development, and/or change in length of the muscle-tendon unit rather than total range of motion.[19] A high percentage of fast twitch fibres,[25] and dual innervation of biceps femoris by the common peroneal and tibial nerves[40] have also been suggested as factors contributing to the risk of injury to the hamstring muscles.[11,16,34]

Semimembranosus is predominantly implicated in slow-speed stretching activities such as dancing,[5,6] but further studies with larger numbers are required to confirm these findings. Askling et al.[5,6] suggest that a combination of extreme hip flexion and knee extension such as in the sagittal splits position can cause an acute strain in the proximal free tendon of semimembranosus. Reports of combined pathology involving quadratus femoris and adductor magnus, suggests that these muscles are also susceptible to strain with this type of mechanism. The purported association between slow-speed stretching and semimembranosus strains may relate to specific anatomical features of the muscle. Semimembranosus has the longest proximal tendon and MTJ of all the hamstring muscles[40] and more distally it is mostly composed of short unipennate and multipennate fibres.[24] This architecture means that when the muscle is stressed across the knee and hip joint, greater strain may be generated in the muscle-tendon unit of semimembranosus compared to other hamstring muscles.

CONCLUSIONS

In conclusion, this systematic review emphasises the emerging relationship between the site and mechanism of acute hamstring strains. In sprinting, the long head of biceps femoris is most likely to be injured, either in isolation or in combination with semitendinosus. Jumping and kicking may also have a specific spectrum of injury but further data are required to establish this. Semimembranosus appears to be more at risk from slow-speed stretching, often accompanied by strains to quadratus femoris. In general, the MTJ is involved most often and proximal structures are more frequently affected than their distal counterparts. However, published reports of hamstring strains need to be more precise both in terms of the affected muscle(s) and site-specific details. These factors are not simply of academic interest since they also impact on prognosis. More detailed studies on the anatomy of acute hamstring strains have the potential to advance our understanding and improve both diagnosis and treatment of these debilitating injuries.

ACKNOWLEDGEMENTS

We thank Dr Grant Meikle, Dunedin Hospital, for providing the MR images. No financial assistance was provided for this project.

References

1. Askling CM, Lund H, Saartok T, et al. Self-reported hamstring injuries in student-dancers. *Scand J Med Sci Sports*. 2002; **12**:230-5.
2. Askling CM, Saartok T, Thorstensson A. Type of acute hamstring strain affects flexibility, strength, and time to return to pre-injury level. *Br J Sports Med*. 2006; **40**:40-4.
3. Askling CM, Tengvar M, Saart T, et al. Sports related hamstring strains - two cases with different etiologies and injury sites. *Scand J Med Sci Sports.* 2000; **10**:304-7.
4. Askling CM, Tengvar M, Saartock T, et al. Acute first-time hamstring strains during high-speed running : A longitudinal study including clinical and magnetic resonance imaging findings. *Am J Sports Med*. 2007; **35**:197-205.
5. Askling CM, Tengvar M, Saartock T, et al. Proximal hamstring strains of stretching type in different sports. *Am J Sports Med*. 2008; **36**:1799-904.
6. Askling CM, Tengvar M, Saartok T, et al. Acute first-time hamstring strains during slow-speed stretching: Clinical, magnetic resonance imaging, and recovery characteristics. *Am J Sports Med*. 2007; **35**:1716-24.
7. Brandser EA, el-Khoury GY, Kathol MH, et al. Hamstring injuries: radiographic, conventional tomographic, CT, and MR imaging characteristics. *Radiology*. 1995; **197**:257-62.
8. Brooks JHM, Fuller CW, Kemp SPT, et al. Incidence, risk and prevention of hamstring muscle injuries in professional rugby union. *Am J Sports Med*. 2006; **34**:1297-305.
9. Chumanov ES, Heiderscheit BC, Thelen BJ. The effect of speed and influence of individual muscles on hamstring mechanics during the swing phase of sprinting. *J Biomech*. 2007; **40**:3555-62.
10. Connell D, Schneider-Kolsky ME, Hoving JL, et al. Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. *Am J Roentgenol*. 2004; **183**:975-84.
11. Croisier J. Factors associated with recurrent hamstring injuries. *Sport Med*. 2004; **34**:681-95.
12. De Smet AA, Best TM. MR imaging of the distribution and location of acute hamstring injuries in athletes. *Am J Roentgenol*. 2000; **174**:393-9.
13. Ekstrand J, Healy JC, Walden M, et al. Hamstring muscle injuries in professional football: the correlation of MRI findings with return to play. *Br J Sports Med*. 2012; **46**:112-7.
14. Gabbe B, Bennell K, Finch C, et al. Predictors of hamstring injury at the elite level of Australian football. *Scand J Med Sci Sports*. 2006; **16**:7-13.
15. Garrett WE. Muscle strain injuries: clinical and basic aspects. *Med Sci Sports Exerc*. 1990; **22**:436-43.
16. Garrett WE, Califf JC, Bassett FH. Histochemical correlates of hamstring injuries. *Am J Sports Med*. 1984; **12**:98-103.
17. Garrett WE, Rich FR, Nikolaou PK, et al. Computed tomography of hamstring muscle strains. *Med Sci Sports Exerc*. 1989; **21**:506-14.
18. Gibbs NJ, Cross TM, Cameron M, et al. The accuracy of MRI in predicting recovery and recurrence of acute grade one hamstring muscle strains within the same season in Australian Rules football players. *J Sci Med Sport*. 2004; **7**:248-58.
19. Heiderscheit BC, Hoerth DM, Chumanov ES, et al. Identifying the time of occurrence of a hamstring strain injury during treadmill running: A case study. *Clin Biomech*. 2005; **20**:1072-8.
20. Koulouris G, Connell D. Evaluation of the hamstring muscle complex following acute injury. *Skeletal Radiol*. 2003; **32**:582-9.
21. Koulouris G, Connell D, Brukner P, et al. Magnetic resonance imaging for assessing risk of recurrent hamstring injuries in elite athletes. *Am J Sports Med*. 2007; **35**:1500-6.
22. Lysholm J, Wicklander J. Injuries in runners. *Am J Sports Med*. 1987; **15**:168-71.
23. Malliaropoulos N, Isinkaye T, Tsitas K, et al. Reinjury after acute posterior thigh muscle injuries in elite track and field athletes. *Am J Sports Med*. 2011; **39**:304-10.
24. Markee JE, Logue JT, Williams M, et al. Two-joint muscles of the thigh. *J Bone Joint Surg Am*. 1955; **37**:125-42.
25. Noonan TJ, Garrett WE. Injuries at the myotendinous junction. *Clin Sport Med*. 1992; **11**:783-806.
26. Orchard J, Best TM. The management of muscle strain injuries: An early return versus the risk of recurrence. *Clin J Sport Med*. 2002; **12**:3-5.
27. Orchard J, Seward H. Epidemiology of injuries in the Australian Football League, seasons 1997 - 2000. *Br J Sports Med*. 2002; **36**:39-44.
28. Ovalle WK, Nahirney PC. Netter's Essential Histology. USA: Saunders Elsevier; 2008.
29. Petersen J, Thorborg K, Nielsen MB, et al. Preventive effect of eccentric training on acute hamstring injuries in men's soccer. *Am J Sports Med*. 2011; **20**:1-8.
30. Pomeranz SJ, Hedit RS. MR imaging in the prognostication of hamstring injury. Work in progress. *Radiology*. 1993; **189**:897-900.31.
31 Sherry MA, Best TM. A comparison of 2 rehabilitation programs in the treatment of acute hamstring strains. *J Orthop Sports Phys Ther*. 2004; **34**:116-25.
32. Slavotinek JP, Verrall GM, Fon GT. Hamstring injury in athletes: Using MR imaging measurements to compare extent of muscle injury with amount of time lost from competition. *Am J Roentgenol*. 2002; **179**:1621-8.
33. Speer KP, Lohnes J, Garrett WE. Radiographic imaging of muscle strain injury. *Am J Sports Med*. 1993; **21**:89-96.
34. Standring S, Borley NR, Collins P, et al. Gray's Anatomy: The Anatomical Basis of Clinical Practice. 40th ed. Edinburgh: Churchill Livingstone; 2008.
35. Thelen DG, Chumanov ES, Best TM, et al. Simulation of biceps femoris musculotendon mechanics during the swing phase of sprinting. *Med Sci Sports Exerc*. 2005; **37**:1931-8.
36. Thelen DG, Chumanov ES, Hoerth DM, et al. Hamstring muscle kinematics during treadmill sprinting. Med Sci Sports Exerc. 2005; 37:108-14.
37. Tuite DJ, Finegan PJ, Saliaris AP, et al. Anatomy of the proximal musculotendinous junction of the addutor longus muscle. Knee Surg Sports Traumatol Arthrosc. 1998; 6:134 - 7.
38. Verrall GM, Slavotinek JP, Barnes PG, et al. Diagnostic and prognostic value of clinical findings in 83 athletes with posterior thigh injury. Am J Sports Med. 2003; 31:969-73.
39. Verrall GM, Slavotinek JP, Barnes PG, et al. Assessment of physical examination and magnetic resonance imaging findings of hamstring injury as predictors for recurrent injury. J Orthop Sports Phys Ther. 2006; 36:215-24.
40. Woodley SJ, Mercer SR. Hamsting muscles: Architecture and innervation. Cells Tissues Organs. 2005; 179:125-41.
41. Woods C, Hawking RD, Maltby S, et al. The Football Association Medical Research Programmes: an audit of injuries in professional football - analysis of hamstring injuries. Br J Sports Med. 2004; 38:36-41.

Appendix 1. *Summary of studies included in the systematic review*

Author	Sample characteristics (no. of individuals with imaging confirmed acute hamstring strains/total no. included in study, sporting discipline, % male, age range[a])	Probable mechanism of injury	Imaging (technique, and interval between injury and imaging)
Garrett et al.[17]	8/10, college athletes “Mostly male”; 18–24 years	Sprinting (7), kicking a high ball (1)	CT Usually < 2 days
Pomeranz and Heidt[30]	12/14, range of professional athletes Male: 100%; 20–29 years	Not known	MRI < 8 days
Speer et al.[33]	17/50, professional and recreational athletes Male: 74%; 17–42 years	Not known	CT (n=10), MRI (n=7) Usually 2 to 4 day
Brandser et al.[7]	5/22, mostly sports-related trauma Male: 82%; 13–61 years	Not known	MRI 8 < 1 week
De Smet and Best[12]	15/15, university athletes Male: 93%; 18–22 years	Sprinting (3), jumping (7), team sports (5)[b]	MRI 1-5 days. Mean: 3 days
Slavotinek et al.[32]	30/37, Australian football Male: 100%; 17–32 years	Sprinting, kicking, jumping	MRI 2 to 6 days. Median: 3 days
Koulouris and Connell[20]	154/170, mostly Australian football 91% male; Mean age 28.2 years	Not stated	MRI and/or US 1 – 10 days. Mean: 4.7 days
Verrall et al.[38]	68/83, Australian football Male: 100%; age not stated	Sprinting, kicking, jumping	MRI 2 – 6 days. Median: 3 days
Connell et al.[10]	42/60[c], Australian football Male: 100%; 17–33 years	Sprinting, kicking, jumping	MRI and US
Gibbs et al.[18]	12/77, Australian football Male: 100%; 18–33 years	Sprinting, kicking, jumping	MRI 1 – 3 days
Verrall et al.[39]	30/162, Australian football Male: 100%; Mean (SD) age 23.6 ± 3.2 years[d]	Sprinting, kicking, jumping	MRI 2 – 6 days. Median: 3 days
Askling et al.[4]	18/18, sprinters Male: 57%; 15–28 years	Sprinting	MRI 4 – 42 days, four scans at different time points
Askling et al.[6]	13/15, dancers Male: 7%; 16–24 years	Slow-speed stretch	MRI 4 – 42 days, four scans at different time points
Askling et al.[5]	28/30, range of sports (24 elite, 6 recreational) Male: 27%; 16-53 years	Splits, high kick, stretching	MRI 1 – 52 weeks. Median: 13 weeks
Ekstrand et al. [13]	180/816, European professional football Male: 100%, age not stated	Sprinting or high-speed running (70%), overuse (5%), stretching/sliding (5%), shooting (4%), twisting/turning (4%), passing (2%), jumping (2%)	MRI 24 – 48 hours.

[a]CT: computed tomography MRI: magnetic resonance imaging; US: ultrasound
Reported age of all participants unless stated;
[b]Group includes strains affecting more than one site;
[c]As reported at baseline MRI ;
[d] Age reported for only those participants with acute first-time hamstring injury.

Can 2D video be used to screen dynamic lower extremity alignment in young athletes?

Chris Whatman,[1]
Patria Hume,[2]
Wayne Hing[3]

[1]Senior lecturer, Sports Performance Research Institute New Zealand and Health and Rehabilitation Research Centre, AUT University, Auckland, New Zealand

[2]Professor, Sports Performance Research Institute New Zealand, AUT University, Auckland, New Zealand

[3]Professor, Bond University, Queensland, Australia

Correspondence:
Chris Whatman
email: chris.whatman@aut.ac.nz

ABSTRACT

Purpose: To investigate the associations between simple two-dimensional (2D) video measures of alignment [knee frontal plane projection angle (FPPA) and lateral pelvic tilt] and three-dimensional (3D) frontal and transverse plane pelvis, hip and knee kinematics in young athletes during lower extremity functional tests. Reliability of the 2D measures was also investigated.

Methods: Pelvis and lower extremity 3D kinematics at peak knee flexion were quantified in 23 uninjured young athletes (11 ±1 years) during three lower extremity functional tests (Small Knee Bend [SKB], Single Leg SKB and Drop Jump). A nine camera motion analysis system captured all tests from retro-reflective body markers and Visual3D and Labview were used to process all 3D data. Lateral pelvic tilt and knee FPPA from 2D video were calculated using SiliconCoach7. Magnitudes of Pearson correlation coefficients were used to describe associations between the 3D kinematics and the 2D measures of alignment. Intra-class correlation was used to quantify the reliability of the 2D measures.

Results: Reliability for all 2D measures was excellent (ICC ≥ 0.98). There were moderate (r = 0.33 to 0.47) associations between knee FPPA and hip joint kinematics and small to moderate (r = 0.21 to 0.40) associations with knee joint kinematics. There were large to very large (r = 0.71 to 0.88) associations between knee FPPA and femoral and tibial segment frontal plane positions. There was a very large association between lateral pelvic tilt measured in 2D and 3D (r = 0.71, 90% CL, 0.56 to 0.81).

Conclusions: Simple 2D measures of lower extremity alignment may be useful for identifying kinematics that should be suspected of placing young athletes at risk of injury; however they should not be used to quantify 3D joint angles.

INTRODUCTION

Assessment of lower extremity dynamic alignment is recommended when screening young athletes for injury risk, during rehabilitation or for performance enhancement.[1] Due to the cost and time involved in a full biomechanical analysis, assessment in the clinic is usually performed via visual observation of functional tests, although simple two-dimensional (2D) video analysis is also being used. There is evidence visual assessment of pelvic position and knee position relative to the foot during slow controlled movements is a useful clinical tool.[2,3] However the reliability and validity of visual ratings of faster movements has been reported as worse than for slower movements and simple 2D video analysis may enhance reliability and validity, during these faster movements.[3] A limitation of 2D video analysis is the uncertainty in the link between these 2D measures and three-dimensional (3D) kinematic measures that have been linked to injury. Excessive lower extremity valgus (based on 3D kinematics) is thought to place athletes at increased risk of injury such as noncontact anterior cruciate ligament tear and patellofemoral dysfunction.[4,5] A lack of pelvis and hip control is considered a key factor in the development of this valgus alignment.[6] As 3D motion analysis is not practical in most clinical settings 2D measurement techniques using simple, portable, inexpensive equipment could be a useful clinical alternative and/or complement to visual assessment in some situations. Further studies are required to clarify the association between 2D measures of pelvic and lower extremity alignment and 3D kinematics in young athletes and the reliability of these 2D measures.

Poor dynamic alignment is a combination of poor frontal and/or transverse plane control of the pelvis, hip, knee and foot.[5,7-9] The most commonly reported measure of this alignment in 2D appears to be the knee frontal plane projection angle (FPPA).[10-12] It is also common clinically to assess the lateral tilt of the pelvis. The drop jump is the most commonly reported lower extremity functional test for assessing lower limb dynamic alignment in young athletes.[13] However the use of other clinical hip and knee flexion tests in this age group is common including the small knee bend (otherwise known as the partial squat). Single leg tests are also used and the single-leg mini squat has been recommended for use in young athletes.[1] The reliability of 3D kinematics during the small knee bend test and the drop jump in this group (ICC=0.62 to 0.98) has been reported previously.[14]

The 2D method of measuring knee FPPA has been reported as associated (r = 0.50-0.60) with 3D calculated knee valgus during side jump, side step and shuttle run tasks in adult male college basketball players.[15] Willson and Davis[10] also reported a moderate association (r = 0.32-0.48) between 2D knee FPPA and 3D hip adduction and knee external rotation, and a moderate association (r = 0.49-0.61) between knee FPPA and 3D femoral and tibial rotations, during a single leg squat in adult females with and without patellofemoral pain. In contrast Ekegren et al.,[16] reported 3D joint angles in a drop jump were not associated with corresponding 2D angles measured from video. Olson et al.,[12] in another study of adult females concluded that exercises to improve lower extremity alignment may improve knee FPPA but this may not be related to specific changes in 3D joint kinematics. There have not been any studies of the associations between 2D measures of alignment (knee FPPA and lateral pelvic tilt) and 3D frontal and transverse plane pelvis, hip and knee kinematics in young athletes during common lower extremity functional tests.

PURPOSE

To investigate the associations between 2D measures of alignment (knee FPPA and lateral pelvic tilt) and 3D frontal and transverse plane pelvis, hip and knee kinematics in young athletes during three common lower extremity functional tests (Small Knee Bend, Single Leg SKB and Drop Jump). Reliability of the 2D measures was also investigated.

METHODS

Participants

Twenty three young athletes (mean ±SD: 11 ±1 y, 153 ±10 cm, 44 ±8 kg) with no musculoskeletal problems volunteered for this study. All athletes were part of a structured long term athlete development programme and competed in a variety of sports. The study was approved by the university ethics committee. All participants/ parents received verbal and written information regarding the study and all athletes gave assent prior to participation. All parents also provided written informed consent prior to testing.

Instrumentation

A nine camera motion analysis system (Qualysis Medical AB, Sweden) sampling at 240 Hz collected kinematic data. Athletes' pelvis and both legs had retro-reflective markers (19 mm diameter) secured to anatomical locations (sacrum, bilateral ASIS's, iliac crests, greater trochanters, medial and lateral femoral epicondyles, mid-patella, medial and lateral malleoli) by an experienced musculoskeletal physiotherapist. Four cluster marker sets were attached to the thigh and shank of each leg. The anatomical markers were used for construction of a skeletal model using Visual 3D (C-Motion Inc, USA).

Testing protocol

All twenty three athletes each attended the motion analysis laboratory on one occasion. Following instrumentation of the retro-reflective markers a static standing trial was collected. The order of the three functional tests was randomized among athletes and all athletes performed three trials of each test. For all tests athletes were given standardized verbal instructions prior to each test (Table 7.1) and the researcher demonstrated each test. Further details on these particular tests and their reliability have been published previously.[17] The Small Knee Bend (SKB), and the Single Leg SKB on each leg, were performed by all athletes with the range of motion determined by their maximum ankle dorsiflexion range of motion. For the Drop Jump athletes started on a 25 cm high box and dropped directly down off the box onto the force plate and immediately jumped vertically as high as possible. All three functional tests were simultaneously recorded from an anterior view on digital video (Panasonic, USA) sampling at a rate of 60 Hz and simultaneously by the 3D system. The video camera was positioned on a tripod in front of athletes and perpendicular to the frontal plane, level with the floor based on the tripod spirit level, at a height of 0.86 m and a distance of 3.7 m. All 3D kinematics and 2D measures of alignment were calculated from the same trial of each test.

Data processing

All static and motion (functional test) trials were tracked using the Qualysis motion capture software and exported to Visual 3D (C-Motion Inc, USA). In Visual 3D the rigid link model (pelvis, thigh, shank and foot) created from the static file was assigned to all imported motion files to calculate kinematic data (joint angles).

All pelvis and lower limb kinematic data were exported to a customised Labview programme and processed to provide joint and segment angles at maximum knee flexion. The two-dimensional (2D) video footage was imported into SiliconCoach7 (New Zealand) for calculation of the knee FPPA and 2D lateral pelvic tilt also at maximum knee flexion. The knee FPPA was calculated as the angle formed by a line connecting the ASIS to the centre of the patella and a line connecting the patella to the midpoint of the ankle malleoli in a manner similar to that reported previously[10,12] (see Figure 7.1). The angle was negative if the patella was medial to a line from the ASIS to the midpoint of the ankle malleoli. The 2D pelvic angle was calculated as the angle formed by a line connecting the ASIS's and a horizontal laboratory aligned line (see Figure 7.1). All 2D angles were measured by a single, experienced physiotherapist on two occasions, separated by one week, using the zoom tool in SiliconCoach7 to aid in marker identification.

Table 7.1: *Description of the small knee bend functional tests used in the study.*

Functional test	Test description
Small Knee Bend (SKB)	Starting from a standing position, athletes performed a partial squat (hip and knee flexion) with the trunk maintained in an upright position while looking straight ahead. Athletes were instructed to continue the SKB until they reached maximum dorsiflexion without lifting their heels and then return to upright standing.
Single Leg SKB	Standing on one leg, with the contralateral hip in neutral and knee flexed to approximately 80°, athletes performed a SKB as described above.

Figure 7.1: *Example of (A) Knee frontal plane projection angle and (B) 2D lateral pelvic tilt, during a Single Leg SKB.*

Statistical analyses

Pearson correlation coefficients were calculated to assess the magnitudes of the associations between the 2D measures of alignment and 3D pelvis, hip and knee kinematics. Correlations were based on 46 kinematic measures (right and left legs from all 23 athletes). The magnitudes of these correlations were described as trivial (0.0-0.1), small (0.1-0.3), moderate (0.3-0.5), large (0.5-0.7), very large (0.7-0.9), or extremely large (0.9-1.0) [18]. An intra-class correlation coefficient (ICC) was calculated to investigate reliability of the 2D measures. The ICC classifications of Fleiss[19] were used to describe the magnitude of ICC values (<0.4 = poor, 0.4 to 0.75 = fair to good, >0.75 = excellent).

RESULTS

The mean 2D and 3D kinematics recorded at peak knee flexion during each of the functional tests are described in Table 7.2. The 2D measures of alignment (knee FPPA and lateral pelvic tilt) demonstrated excellent reliability (ICC ≥0.98). There were moderate (r = 0.33 to 0.47) associations between knee FPPA and hip joint kinematics and small to moderate (r = 0.21 to 0.45) associations with knee joint kinematics (see Table 7.3). There were large to very large (r = 0.71 to 0.88) associations between knee FPPA and femoral and tibial segment frontal plane positions. Knee FPPA was also moderately associated with pelvic segment frontal plane position (r = 0.31), but it was not associated with pelvic transverse plane position (r = 0.06). There was a very large association between lateral pelvic tilt measured in 2D and 3D pelvic lateral tilt during the single leg SKB (r = 0.71, 90% CL, 0.56 to 0.81).

Table 7.2: *Angle and segment positions (°) at maximum knee flexion (mean both legs ± SD) for 23 healthy young athletes.*

		Small Knee Bend	Drop Jump	Single Leg SKB
2D	Knee FPPA	3.4 ±10.1	-3.9 ±10.1	-8.4 ±10.6
	Lateral pelvic tilt	-	-	4.2 ±3.7
3D	Hip Frontal*	-0.9 ±5.9	2.2 ±5.3	14 ±6.0
	Hip Transverse†	1.7 ±6.0	-0.2 ±6.3	2.5 ±6.4
	Knee Frontal‡	3.4 ±5.8	-0.9 ±5.9	6.5 ±5.2
	Knee Transverse†	-1.7 ±5.2	-0.5 ±7.3	-4.2 ±6.6
	Femur Frontal*	-1.1 ±4.7	1.9 ±4.8	11.2 ±4.9
	Femur Transverse†	1.7 ±7.1	-0.3 ±8.3	4.9 ±8.4
	Tibia Frontal‡	2.5 ±3.9	0.9 ±5.1	3.9 ±4.2
	Tibia Transverse†	-3.3 ±6.0	1.6 ±8.1	9.9 ±13.8
	Pelvis Frontal	-	-	-7.7 ±9.0
	Pelvis Transverse	-	-	-1.9 ±7.1

* +ve = adduction,
† +ve= internal rotation,
‡ +ve = abduction, - pelvic angle not measured in the small knee bend or drop jump,
FPPA = Frontal plane projection angle,
2D = two-dimensional,
3D = three-dimensional.

DISCUSSION

The purpose of this study was to investigate the associations between 2D measures of alignment (knee FPPA and lateral pelvic tilt) and 3D frontal and transverse plane pelvis, hip and knee kinematics in young athletes during three common lower extremity functional tests (Small Knee Bend, Single Leg SKB and Drop Jump). The reliability of the 2D measures was also investigated. Young athletes with poor dynamic alignment that increases their risk of injury should be identified in order to facilitate appropriate intervention. Simple, portable, inexpensive video measures of 2D alignment and/or visual assessment may be the most appropriate for clinical use.

Reliability of the knee FPPA and lateral pelvic tilt was excellent for all functional tests. However the knee FPPA was not strongly correlated to the 3D hip and knee joint angles. There were moderate associations of the knee FPPA with hip frontal (adduction) and hip and knee transverse plane angles but only small correlations with knee frontal plane (abduction/valgus) angles. These findings are in keeping with those of Willson and Davis[10] who also found the weakest correlation was with knee joint valgus (r = 0.21)in a group of adult females with and without patellofemoral pain during a single leg squat. Willson and Davis[10] suggested this may be because there are multiple combinations of pelvic, hip and knee rotations that might contribute to a larger negative knee FPPA and this also appears to be the case in our young athlete population. As such the magnitude of 3D lower extremity transverse and frontal plane joint rotations in young athletes cannot be accurately determined by the magnitude of the 2D knee FPPA.

Table 7.3: *Associations between knee frontal plane projection angle and 3D joint/segment transverse and frontal plane kinematics expressed as Pearson correlation coefficients (90% CL).*

		Small Knee Bend	Drop Jump	Single Leg SKB
Joint rotations	Hip Frontal	0.37 (0.14 to 0.56)	0.47 (0.25 to 0.64)	0.43 (0.21 to 0.61)
	Hip Transverse	0.46 (0.24 to 0.63)	0.35 (0.11 to 0.55)	0.33 (0.09 to 0.53)
	Knee Frontal	0.21 (-0.04 to 0.43)	0.29 (0.05 to 0.50)	0.23 (-0.02 to 0.45)
	Knee Transverse	0.45 (0.23 to 0.63)	0.33 (0.09 to 0.53)	0.40 (0.17 to 0.59)
Segment rotations	Femoral Frontal	0.79 (0.68 to 0.87)	0.71 (0.56 to 0.81)	0.77 (0.65 to 0.85)
	Femoral Transverse	0.42 (0.19 to 0.60)	0.51 (0.03 to 0.67)	0.42 (0.19 to 0.60)
	Tibial Frontal	0.88 (0.81 to 0.93)	0.81 (0.70 to 0.88)	0.85 (0.76 to 0.91)
	Tibial Transverse	0.07 (-0.18 to 0.31)	0.08 (-0.17 to 0.32)	0.13 (-0.12 to 0.36)
	Pelvic Frontal	-	-	0.31 (0.07 to 0.52)
	Pelvic Transverse	-	-	0.06 (-0.19 to 0.30)

- pelvic angle not measured in the small knee bend or drop jump.

Of the 3D joint rotations, the hip joint showed the strongest association with knee FPPA. This may be important to clinicians given the recent focus on hip function and lower extremity injury. A review by Reiman et al.,[20] stated there was mounting evidence that hip weakness contributes to knee injuries across all ages and Powers[6] also made a strong case for the link between abnormal hip kinematics and injury. Simple clinical assessment of the knee FPPA visually or assisted by video may help identify those young athletes in need of interventions designed to improve hip kinematics in weight bearing. Females with patellofemoral pain have been shown to have larger femoral frontal plane angles (2D measure) during a single leg squat than a control group[21] and a trend towards larger knee frontal plane projection angles has also been reported.[10] Additionally 2D analysis of the single leg squat has been proposed as a useful screening tool for low back injury risk in adolescent cricket fast bowlers.[22] Further prospective studies investigating the use of simple 2D analysis as a screening tool for risk of injury are needed.

The 2D knee FPPA was most strongly associated with 3D femoral and tibial segment frontal plane position. Larger negative knee FPPA's (patella positioned more medially) were associated with greater femoral adduction and tibial abduction. This is again in keeping with the findings of Willson and Davis[10] who also reported these as the strongest associations (r = 0.49 to 0.61). In the current study knee FPPA also had a moderate to large association with femoral transverse plane segment position (larger negative knee FPPA's were associated with increased femoral internal rotation). The smaller associations between the FPPA and 3D joint rotations (compared to segment rotations) may be due to relative rotations of two segments in the laboratory coordinate system. For example simultaneous frontal plane motion of the femur and pelvis may result in little change in the hip joint frontal plane angle. Excessive adduction/internal rotation of the femur and/or abduction of the tibia are suggested to contribute to overuse injuries such as patellofemoral pain.[6] These suggestions however appear predominantly based on theory considering the relative position of two segments at a joint (joint rotation) rather than the individual segment positions. Nevertheless the knee FPPA may be useful in identifying young athletes with femoral and tibial alignment that places them at increased risk of injury. Given the practical and time benefits for clinical use it may be sufficient to assess this knee FPPA using visual rating and/or 2D video analysis of functional tests such as the small knee bend or mini squat. Visual rating of alignment in young athletes during these functional tests has been shown to differentiate athletes based on knee FPPA.[3] Assessment of faster tests such as drop landings may necessitate video analysis due to limitations noted with visual assessment.[3]

There was a strong association between the 2D measure of lateral pelvic tilt and the magnitude of 3D pelvic rotation in the frontal plane during the single leg SKB. Assessment of weight bearing pelvic alignment is common clinically as it is thought to give a further indication of hip muscle function as outlined by Crossley et al.,[23] Again this assessment of pelvis alignment is commonly made via visual observation in the clinic and this has been shown to be reliable in young athletes during slow functional tests.[3] Visual assessment and/or 2D measures of pelvic alignment should therefore be useful in identifying young athletes with a 3D lateral pelvic tilt that is suggestive or poor hip muscle function.

A limitation of this study was the lack of exact synchronisation between the 2D and 3D measures. The 3D data were collected at a much higher sampling rate than the 2D and thus the 2D image used to measure the knee FPPA and pelvic tilt may not have occurred at the exact time point of the 3D data. We have also only taken measures at the single time point of maximum knee flexion and as such our findings do not indicate how 2D and 3D measures compare throughout the tests (e.g. at different degrees of knee flexion or for joint excursions). Maximum knee flexion (mean ±SD: SKB = 90 ±17º, Single SKB = 84 ±10º, Drop Jump = 90 ±11º) was chosen as this is where maximum frontal plane deviation is likely to be seen clinically and this was also the easiest point to identify visually to synchronise the 2D and 3D angles. Additionally, although the total number of limbs we reported on is in keeping with similar previous studies,[10] we had a relatively small sample size of only 23 young athletes. This could lead to greater uncertainty in the true associations in the population. Precision for a correlation is thought to be adequate when the uncertainty in the estimate (represented by its confidence interval) does not span more than two qualitative magnitude thresholds.[24] Our confidence intervals show that we appear to have adequate precision for the majority of correlations. The lower correlations still provide useful information but they must be interpreted with more caution.

CONCLUSION

Two-dimensional measures of alignment during lower extremity functional tests demonstrated excellent reliability and small to large associations with 3D pelvic, hip and knee kinematics. The 2D video measures may assist visual assessment and provide a useful clinical indication of young athletes needing appropriate intervention, however they should not be used to quantify 3D joint angles.

Practical implication

Simple 2D video measures may prove a useful tool for clinicians wanting to assess dynamic pelvis and lower extremity alignment in young athletes.

References

1. Örtqvist, M., et al., Reliability and reference values of two clinical measurements of dynamic and static knee position in healthy children. *Knee Surgery, Sports Traumatology, Arthroscopy*, 2011: p. 1-7.
2. Ageberg, E., et al., Validity and inter-rater reliability of medio-lateral knee motion observed during a single-limb mini squat. *BMC Musculoskeletal Disorders*, 2010. **11**: p. 265.
3. Whatman, C., P.A. Hume, and W. Hing, The reliability and validity of physiotherapist visual rating of dynamic pelvis and knee alignment in young athletes. *Physical Therapy in Sport*, 2012. In Press.
4. Hewett, T.E., et al., Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. American Journal of Sports Medicine, 2005. **33**(4): p. 492-501.
5. Powers, C.M., The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: A theoretical perspective. *Journal of Orthopaedic and Sports Physical Therapy*, 2003. **33**(11): p. 639-46.
6. Powers, C.M., The influence of abnormal hip mechanics on knee injury: a biomechanical perspective. *Journal of Orthopaedic & Sports Physical Therapy*, 2010. **40**(2): p. 42-51.
7. Sahrmann, S.A., Diagnosis and Treatment of Movement Impairment Syndromes2002, London: Mosby.
8. Earl, J.E., J. Hertel, and C.R. Denegar, Patterns of dynamic malalignment, muscle activation, joint motion, and patellofemoral-pain syndrome. *Journal of Sport Rehabilitation*, 2005. **14**: p. 215-233
9. Willson, J.D. and I.S. Davis, Lower extremity strength and mechanics during jumping in women with patellofemoral pain. *Journal of Sport Rehabilitation*, 2009. **18**(1): p. 76-90.
10. Willson, J.D. and I.S. Davis, Utility of the Frontal Plane Projection Angle in Females with Patellofemoral Pain. *Journal of Orthopaedic & Sports Physical Therapy*, 2008. **38**(10): p. 606-616.
11. Herrington, L., Knee valgus angle during landing tasks in female volleyball and basketball players. *Journal of Strength and Conditioning Research*, 2009. **25**(1): p. 262-6.
12. Olson, T.J., et al., Comparison of 2D and 3D kinematic changes during a single leg step down following neuromuscular training. *Physical Therapy in Sport*, 2011. **12**(2): p. 93-99.
13. Ford, K.R., G.D. Myer, and T.E. Hewett, Reliability of landing 3D motion analysis: implications for longitudinal analyses. *Medicine and Science in Sports and Exercise*, 2007. **39**(11): p. 2021-8.
14. Whatman, C., P.A. Hume, and W. Hing, Kinematics during lower extremity functional screening tests in young athletes – are they reliable and valid? *Physical Therapy in Sport*, 2012. In Press.
15. McLean, S.G., et al., Evaluation of a two dimensional analysis method as a screening and evaluation tool for anterior cruciate ligament injury. *British Journal of Sports Medicine*, 2005. **39**(6): p. 355-62.
16. Ekegren, C.L., et al., Reliability and validity of observational risk screening in evaluating dynamic knee valgus. *Journal of Orthopaedic and Sports Physical Therapy*, 2009. **39**(9): p. 665-74.
17. Whatman, C., W. Hing, and P. Hume, Kinematics during lower extremity functional screening tests-Are they reliable and related to jogging? *Physical Therapy in Sport*, 2011. **12**(1): p. 22-9.
18. Hopkins, W.G., et al., Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise*, 2009. **41**(1): p. 3-13.
19. Fleiss, J.L., The Design and Analysis of Clinical Experiments Wiley library classics1999, New York: Wiley.
20. Reiman, M.P., L.A. Bolgla, and D. Lorenz, Hip Function's Influence on Knee Dysfunction: A Proximal Link to a Distal Problem. *Journal of Sport Rehabilitation*, 2009. **18**(1): p. 33-46.
21. Levinger, P., W. Gilleard, and C. Coleman, Femoral medial deviation angle during a one-leg squat test in individuals with patellofemoral pain syndrome. *Physical Therapy in Sport*, 2007. **8**(4): p. 163-168.
22. Crewe, H., et al., Functional screening test associated with altered trunk and pelvis kinematics and low back injury incidence in adolescent fast bowlers, in 30th Annual Conference of Biomechanics in Sports2012: Melbourne. p. 123-126.
23. Crossley, K.M., et al., Performance on the single-leg squat task indicates hip abductor muscle function. *American Journal of Sports Medicine*, 2011. **39**(4): p. 866-73.
24. Hopkins, W.G. and B.F. Manly, Errors in assigning grades based on tests of finite validity. Research quarterly for exercise and sport, 1989. 60(2): p. 180-2.

Injuries in amateur representative rugby league over three years

Doug King, PhD[1]
Trevor Clark, MSc,[2]

1. Sports Performance Research Institute New Zealand
School of Sport and Recreation
Faculty of Health and Environmental Science
Auckland University of Technology, New Zealand
2. Research Centre for Maori Health and Development
Massey University Wellington
Wellington, New Zealand

Correspondence
Doug King
Emergency Department
Hutt Valley District Health Board
Private Bag 31-907
Lower Hutt
New Zealand
Email: dking@aut.ac.nz

ABSTRACT

Aim: To determine the incidence, nature and causes of match injuries sustained during amateur representative rugby league.

Methods:
A prospective cohort study was undertaken on the 2008, 2009 and 2010 amateur representative rugby league competition in New Zealand using definitions and procedures as suggested for epidemiological studies in rugby league.

Results:
Incidence of injuries was 355 per 1,000 match hours (forwards 435 per 1,000 match hours, backs 236 per 1,000 match hours). A total of 30 missed match injuries were recorded (83 per 1,000 match hours). The upper limb (124 per 1,000 match hours) and haematomas (135 per 1,000 match hours) were the most common injury site and type. The majority of injuries were sustained in the tackle (339 per 1,000 match hours) with the tackler recording significantly more than the ball carrier (RR: 1.6; 1.1 to 2.2; p=0.015).

Conclusions:
This study showed that the overall risk of injury for amateur representative rugby league players in New Zealand is similar to that previously reported; the nature, site and cause of injuries is also similar to previously reported studies on amateur rugby league.

Key Words:
rugby league, injury causation, incidence of injury, nature of injury, risk of injury

INTRODUCTION

Played internationally,[1, 2] rugby league is competed at all levels of participation from junior to senior professional levels. Studies reporting on rugby league match injuries have shown the incidence varies at the different participation levels. In a study on professional rugby league participants it was reported that this varied from 114[3] to 243[4] injuries per 1,000 match hours while semi-professional participants recorded an injury rate of 115[5] to 825[6] per 1,000 match hours. At the amateur level for club based competitions it has been reported that the incidence of injuries also varies from 10[7] to 70[8] per 1,000 match hours. Although the number of published studies reporting on rugby league injuries is increasing,[8-10] there still remains a paucity of published studies. As well there have been no recently published injury studies at representative level of rugby league.

In reporting on injuries at the semi-professional[6] level of participation it was identified that the higher incidence may be expected given the superior physiological capacities of semi-professional participants when compared with amateur level participants.[6, 11] Conceivably, the increased physiological capacities of semi-professional participants may produce a higher playing intensity resulting in the higher injury rate[6,11] but is this similar for players selected into representative competition teams? While it has been reported that there are variances in the incidence of injuries between amateur, semi-professional and professional there has been no reports on whether participating in a representative competition at these levels differs. Does an amateur representative rugby league competiton have a higher incidence than a domestic amateur competition; is it similar to other levels of competition and what are these differences? Therefore, the aims of this study were to record and analyse match injuries sustained by amateur rugby league players competing in a representative competition over three consecutive competition seasons and to compare this with other studies reporting on amateur rugby league match injuries to see if the injury patterns differed.

METHODS

A prospective cohort study was undertaken on an amateur representative rugby league team from a district in New Zealand over the 2008, 2009 and 2010 competition seasons. The competition was held at the end of the domestic rugby league competition and comprised of seven teams over the August to October period. The definitions and procedures used were in agreement with the definitions, collection and reporting methods suggested for rugby league.[12] The study was approved by the Auckland University of Technology Ethics Committee.

Players' baseline information, age (years), stature (metres) and body mass (kilograms), and their informed consent was obtained: baseline data were summarised as mean (±SD). Match exposure were calculated on the basis of 13 players (7 backs and 6 forwards) per team exposed for 80 minutes per team-match. The medic in attendance at matches recorded and reported all injuries throughout the study period on a standardised injury reporting form regardless of severity.[12] The incidence of injury was reported as injuries per 1,000 match hours and 95% confidence intervals (CI). Independent ***t***-tests were used to assess differences in baseline data and a one-sample chi-squared (χ^2) test was used to determine whether the observed injury frequency was significantly different from the expected injury frequency. To compare between injury rates risk ratios (RR's) were used. Results were considered significant at p<0.05.

RESULTS

A total of 90 players competed in the representative teams during the 2008 to 2010 competition period. The age, stature and mass of the players are shown in Table 1. Forwards were significantly older (t=4.25; p<0.001), taller (t=7.94; p<0.001) and heavier (t=10.64; p<0.001) than backs. The teams competed in 21 matches over the duration of the study resulting in 363 match exposure hours. There were 129 injuries recorded resulting in a match injury rate of 355 (299 to 422) injuries per 1,000 match hours. The overall incidence of injury as a function of grouped injury severity, site, type, nature, match period and playing position are presented in Table 2. Forwards recorded significantly more total injuries (RR: 1.5; 1.1 to 2.0; p=0.017); concussions (RR: 0.2; 0.0 to 1.4; p=0.058); injuries to the head-neck (RR: 2.3; 1.0 to 5.0; p=0.045); upper limbs (RR: 2.7; 1.5 to 4.8; p=0.001); as the tackler (RR: 2.1; 1.2 to 3.7; p=0.010); and in the fourth quarter of matches (RR: 2.2; 1.2 to 3.3; p=0.001) than backs. A cross tabulation of the main sites and types of injuries recorded is shown in Table 3.

DISCUSSION

This study reports for the first time on amateur representative rugby league injuries in New Zealand. Similar to other studies[13] forwards were older, taller and heavier than backs for the current cohort of players. The finding of an injury incidence of 355 injuries per 1,000 match hours is higher than some,[9,14] but not all,[8,15] studies reporting on amateur participation injuries but lower than for semi-professional[6] participation match injuries. The ball carrier recorded more injuries than the tackler which is similar to some,[8] but not all,[6,16,17] rugby league studies. Collisions sustained by the ball carrier may be associated with a greater risk of injury than the tackling players. The impact forces that are generated from the ball carrier as they attack the defensive line of players may be distributed among the defenders, but the combined forces of the tacklers onto the ball carrier is absorbed by the single ball carrier.[18] Repeated collisions may have a cumulative effect and may result in the musculoskeletal structures of players being exceeded and an injury occurring from the resultant stress.[19] Further studies are warranted to explore the effect of repeated collisions and the incidence of injuries in rugby league players.

The tackle situation has been reported to be the most common cause of all concussions in rugby league.[2] The tackle is reported to be the most important skill in rugby league[20] Success in the tackle is dependent on the individual players tackling ability, the ability to tolerate the associated physical collisions and the capacity to 'win' the tackle contest.[20] With the emphasis on increased play-the-ball speed and dominance of the ruck the tackle is an important area for injury prevention to focus on. In a recent study[21] reporting on tackles in professional rugby league, the most common tackle-related injuries occurred when the tackle involved contact with the ball carrier at shoulder height or mid torso region, in the blind vision area, and with two or more tacklers in total being involved. Injury prevention programs designed to highlight these areas may be useful to assist in reducing the incidence of injuries, especially concussions, in the tackle situation. In addition the development of correct tackling technique (defense) and falling in the tackle (attack) training programs may assist in reducing the incidence of concussions, and other injuries, in the tackle.[22]

The nature of injuries for the current cohort of players is similar to other studies reporting rugby league injuries.[1, 2] Contusions and strains were the most commonly reported injury type which is similar to other studies reporting rugby league injuries.[2] Given the nature of rugby league as a collision sport this finding is not unexpected. Previous studies reporting on amateur and professional rugby league match injuries also reported that the most common injury types were contusions and strains.[2] Given that rugby league requires players to undergo rapid acceleration, deceleration and changes of direction, the high incidence strains is not unexpected.[9] Similarly, the number of physical collisions encountered by players during matches would explain the high incidence of contusions.

The finding that more concussions were recorded by backs than forwards (31 vs. 6 per 1,000 match hours) was unexpected. Unfortunately there are no other studies specifically reporting on concussion at any level of rugby league limiting the exploration of this further. Backs are typically employed in a more open running environment and may encounter the bigger forwards at speed while forwards are employed in a more closed short distance environment. These differences may account for the differences in the incidence of concussions to back when compared with forwards but other factors such as fitness level, tackle technique, fatigue may also be involved.[18, 23] All identified concussions were removed from the field of play and underwent a return to play protocol requiring a full medical assessment prior to them being able to return to contact activities and match participation. Further studies reporting on the incidence of concussions in amateur rugby league are warranted to enable comparisons.

The finding that more injuries happened in the second than the first half of matches is similar to other studies reporting on amateur[8] but differs from semi-professional[6] levels of participation. This may be due to the higher aerobic fitness of semi-professional players when compared with amateur players.[6, 11] Unfortunately no aerobic levels of fitness were recorded in the study and further studies reporting on injuries at representative levels of participation should consider including this to enable comparisons. Another consideration for the increased injury incidence in the second half of matches is whether the team is winning or losing the match. All teams play aggressively with the purpose of dominating the opposition in order to win the match.[9] This over-aggressive playing style may result in an increased injury rate in the second half of matches.[24]

The finding that forwards recorded more injuries when compared with backs (436 vs. 286 per 1,000 match hours) is consistent with other studies reporting on amateur[9] and professional[25] levels of participation. The finding that there was a lower incidence of injuries in backs is not unexpected given forwards spend a higher percentage of match activities in physical collisions and tackles.

[3] What was unexpected was the higher incidence of injuries for backs in the second quarter (123 vs. 96 per 1,000 match hours) and first half (143 vs. 125 per 1,000 match hours) when compared with forwards. This difference may be reflective of the match tactics employed over the competition seasons. Further research is warranted to see if this finding was specific to this cohort of players or whether this is reflective of this level of participation.

In comparing our study with other amateur rugby league injury studies[8, 9] it can be seen that the incidence of injuries at the representative level of participation was higher (355 vs. 160[9] per 1,000 match hours) over a similar time period but less than a division two competition (701[8] per 1000 match hours). These differences may be related to the location of the different competitions (Queensland, Australia;[9] Otago, New Zealand[8]) while the current study required travel around New Zealand as part of the competition requirements. Other considerations are the fitness levels, atmospheric conditions and skill qualities of the different groups under study. Further research is warranted to further explore similar participation levels of competition over more than one competition season to identify if the identified consideration does impact on the incidence of injury in amateur rugby league.

The conducting of this study does have some limitations. Although the study encompassed three years of consecutive representative rugby league the competition period was limited. In addition to this the players involved entered into the competition having completed a domestic competition of up to 20 weeks. No injury history was recorded when the players were selected into the representative teams. Further studies reporting on representative rugby league competitions should consider including this as part of the study.

In summary, this study reported for the first time on amateur representative rugby league players. Although the findings were similar to other studies in regards to the injury types and sites there were observable differences to other studies in the incidence of injuries when comparing match quarters. As well there is a need for future studies reporting specifically o the incidence of concussion in rugby league.

Table 1: ***Baseline data for the amateur representative rugby league players.***

Playing Group (No. Players)	Mean (±SD)		
	Age, Yrs.	Stature, m	Mass, kg
All Players (n=90)	23.0 (4.1)	1.81 (0.05)	95.3 (14.8)
Backs (n=40)	21.9 (3.7)	1.77 (0.05)	85.5 (10.7)
Forwards (n=50)	24.1[a] (4.0)	1.82[a] (0.05)	101.4[a] (14.1)

SD = Standard deviation;
a = Significantly different (p>0.05) from Backs

Table 2: ***Incidence of injuries with 95% confidence intervals grouped by injury severity, site, type, nature, match period and playing position***

	Injury incidence per 1,000 match hours (95% CI)		
	Forwards (n=73)	Backs (n=56)	All Players (n=129)
Injury Severity			
Transient	340.1a (262.4 to 441.0)	214.8 (158.8 to 290.7)	272.7 (223.9 to 332.0)
Mild	53.7 (27.9 to 103.2)	20.5 (7.7 to 54.5)	35.8 (20.8 to 61.7)
Moderate	17.9 (5.8 to 55.5)	10.2 (2.6 to 40.9)	13.8 (5.7 to 33.1)
Major	23.9 (9.0 to 63.6)	40.9 (20.5 to 81.8)	33.0 (18.8 to 58.2)
Total	435.6a (346.3 to 547.9)	286.4 (220.4 to 372.2)	355.3 (299.0 to 422.2)
Injury Site			
Head/Neck	71.6a (40.7 to 126.1)	107.4 (70.0 to 164.7)	90.9 (64.6 to 127.8)
Upper Limbs	185.0a (130.1 to 263.0)	76.7 (46.3 to 127.3)	126.7 (94.9 to 169.1)
Lower Limbs	125.3 (81.7 to 192.2)	71.6 (42.4 to 120.9)	96.4 (69.2 to 134.3)
Chest/Back	53.7 (27.9 to 103.2)	30.7 (13.8 to 68.3)	41.3 (24.9 to 68.5)
Injury Type			
Haematoma	185.0a (130.1 to 263.0)	92.1 (58.0 to 146.1)	135.0 (102.0 to 178.6)
Strain	113.4a (72.3 to 177.8)	46.0 (24.0 to 88.5)	74.4 (51.0 to 108.4)
Sprain	17.9a (5.8 to 55.5)	61.4 (34.9 to 108.1)	44.1 (27.0 to 71.9)
Fracture	41.8 (19.9 to 87.6)	30.7 (13.8 to 68.3)	35.8 (20.8 to 61.7)
Other	35.8 (16.1 to 79.7)	20.5 (7.7 to 54.5)	27.5 (14.8 to 51.2)
Dislocation	35.8a (16.1 to 79.7)	5.1 (0.7 to 36.3)	19.3 (9.2 to 40.4)
Concussion	6.0 (0.8 to 42.4)	30.7 (13.8 to 68.3)	19.3 (9.2 to 40.4)
Nature of Injury			
Ball Carrier	226.8 (165.0 to 311.6)	189.2 (137.1 to 261.2)	206.6 (164.7 to 259.0)
Tackler	185.0a (130.1 to 263.0)	87.0 (54.1 to 139.9)	132.2 (99.6 to 175.4)
Collision	6.0 (0.8 to 42.4)	5.1 (0.7 to 36.3)	5.5 (1.4 to 22.0)
Other	17.9 (5.8 to 55.5)	5.1 (0.7 to 36.3)	11.0 (4.1 to 29.4)
Match Period			
0-20 min	29.8 (12.4 to 71.7)	20.5 (7.7 to 54.5)	24.8 (12.9 to 47.6)
21-40 min	95.5 (58.5 to 155.8)	122.8 (82.3 to 183.1)	110.2 (80.8 to 150.2)
41-60 min	107.4 (67.7 to 170.5)	81.8 (50.1 to 133.6)	93.6 (66.9 to 131.1)
61-80 min	202.9a (145.0 to 283.9)	61.4 (34.9 to 108.1)	126.7 (94.9 to 169.1)
1st Half	125.3 (81.7 to 192.2)	143.2 (98.9 to 207.4)	135.0 (102.0 to 178.6)
2nd Half	310.3b (236.4 to 407.2)	143.2 (98.9 to 207.4)	220.3b (177.0 to 274.3)

CI: Confidence Interval. Significantly different (p<0.05) from (a) Backs; (b) 1st half

Table 3: *Cross tabulation of the site and type of injuries for all players (n=129).*

Injury Type[12]	Injury incidence per 1,000 match hours (95% CI)				
	Head/Neck	Upper Limb	Lower Limb	Chest/back	Total
Haematoma	33.0 (18.8 to 58.2)	44.1 (27.0 to 71.9)	38.6 (22.8 to 65.1)	19.3 (9.2 to 40.4)	135.0 (102.0 to 178.6)
Strain	11.0 (4.1 to 29.4)	27.5 (14.8 to 51.2)	19.3 (9.2 to 40.4)	16.5 (7.4 to 36.8)	74.4 (51.0 to 108.4)
Sprain	2.8 (0.4 to 19.6)	16.5 (7.4 to 36.8)	24.8 (12.9 to 47.6)	0.0 -	35.8 (20.8 to 61.7)
Fracture	11.0 (4.1 to 29.4)	19.3 (9.2 to 40.4)	2.8 (0.4 to 19.6)	2.8 (0.4 to 19.6)	35.8 (20.8 to 61.7)
Other	14.5 (6.5 to 32.2)	0.0 -	8.3 (2.7 to 25.6)	2.8 (0.4 to 19.6)	27.5 (14.8 to 51.2)
Dislocation	0 -	19.3 (9.2 to 40.4)	0.0 -	0.0 -	19.3 (9.2 to 40.4)
Concussion	19.3 (9.2 to 40.4)	0.0 -	0.0 -	0.0 -	19.3 (9.2 to 40.4)
All Types	90.9 (64.6 to 127.8)	126.7 (94.9 to 169.1)	96.4 (69.2 to 134.3)	41.3 (24.9 to 68.5)	355.3 (299.0 to 422.2)

CI: Confidence Interval

References

1. Gabbett T, King T, Jenkins D. Applied physiology of rugby league. *Sports Med* 2008; **38**(2):119-38.
2. King D, Hume P, Milburn P, Guttenbeil D. Match and training injuries in rugby league: A review of published studies. *Sports Med.* 2010; **40**(2):163-78.
3. Stephenson S, Gissane C, Jennings D. Injury in rugby league: a four year prospective survey. *Br J Sports Med* 1996; **30**(4):331-4.
4. Estell J, Shenstone B, Barnsley L. Frequency of Injuries in different age-groups in an elite rugby league club. *Aust J Sci Med Sport* 1995; **27**(4):95-7.
5. King D, Gabbett T. Injuries in the New Zealand semi-professional rugby league competition. *NZ J Sports Med* 2009; **36**(1):6-15.
6. Gabbett T. Incidence of injury in semi professional rugby league players. *Br J Sports Med* 2003; **37**(1):36-44.
7. Raftery M, Parker R, Stacey E, Peat J, Wang H. Incidence of injury in junior rugby league in the Penrith and district rugby league area. Children's Hospital Institute of Sports Medicine; Research and Development Office, The New Children's Hospital, Westmead. 1999.
8. King D, Gabbett T. Amateur rugby league match injuries in New Zealand. *NZ J Sports Med* 2009; **36**(1):16-21.
9. Gabbett T. Incidence, site, and nature of injuries in amateur rugby league over three consecutive seasons. *Br J Sports Med* 2000; **34**(2):98-103.
10. King D. Injuries in the New Zealand Bartercard cup competition. Dunedin: University of Otago; 2007.
11. Gabbett T. Influence of physiological characteristics on selection in a semi-professional first grade rugby league team: A case study. *J Sports Sci* 2002; **20**(5):399-406.
12. King D, Gabbett T, Gissane C, Hodgson L. Epidemiological studies of injuries in rugby league: Suggestions for definitions, data collection and reporting methods. *J Sci Med Sport* 2009; **12**(1):12-9.
13. King D, Hume P, Milburn P, Guttenbeil D. A review of the physiological and anthropometrical characteristics of rugby league players. *Sth Afr J Res Sport, Phys Ed Rec* 2009; **31**(2):49-67.
14. Pringle R, McNair P, Stanley S. Incidence of sporting injury in New Zealand youths aged 6 - 15. *Br J Sports Med* 1998; **32**(1):49-52.
15. King D, Gabbett T. Injuries in a national women's rugby league tournament: An initial investigation. *NZ J Sports Med* 2007; **34**(2):18-22.
16. Gabbett T. Science of rugby league football: A review. *J Sports Sci* 2005; **23**(9):961-76.
17. Gissane C, Jennings D, White J, Cumine A. Injury in summer rugby league football: the experiences of one club. *Br J Sports Med* 1998; **32**(2):149-52.
18. Gabbett T, Jenkins D, Abernethy B. Physical collisions and injury in professional rugby league match-play. *J Sci Med Sport.* 2011; **14**(3):210-5.
19. Takarada Y. Evaluation of muscle damage after a rugby match with special reference to tackle plays. *Br J Sports Med* 2003; **37**(5):416-9.
20. Gabbett T. Physiological and anthropometric correlates of tackling ability in rugby league players. *J Strength Cond Res.* 2009; **23**(2):540-8.
21. King D, Hume PA, Clark T. Nature of tackles that result in injury in professional rugby league. *Res Sports Med.* 2011; in print.
22. Gabbett T. Influence of playing position on the site, nature and cause of rugby league injuries. *J Strength Cond Res* 2005; **19**(4):749-55.
23. Gabbett T, Ryan P. Tackling technique, injury risk, and playing performance in high performance collision sport athletes. *Int J Sports Sci Coach.* 2009; **4**(4):521-33.
24. King D, Gissane C. Injuries in amateur rugby league matches in New Zealand: A comparison between a division one and a division two premier grade teams. *Clin J Sports Med.* 2009; **19**(4):277-81.
25. Gissane C, Jennings D, Cumine A, Stephenson S, White J. Differences in the incidence of injury between rugby league forwards and backs. *Aust J Sci Med Sport* 1997; **29**(4):91-4.

An investigation of surf injury prevalence in Australian surfers: A self-reported retrospective analysis

Rudi A Meir PhD, CSCS,
Shi Zhou PhD,
Margaret I Rolfe PhD,
Wendy L Gilleard PhD and
Rosanne A Coutts PhD
School of Health and Human Sciences
Southern Cross University
Lismore, New South Wales
AUSTRALIA

Correspondence
Dr Rudi Meir
School of Health and Human Sciences
Southern Cross University
P.O. Box 157, Lismore 2480
AUSTRALIA
Tel: +2-66203767
Fax: +2-66203880
e-mail: rudi.meir@scu.edu.au

ABSTRACT

Aim:
To establish the prevalence and type of injuries sustained by Australian surfers in a 12 month period.

Study design:
Injury history was collected via a self-reported online survey instrument.

Participants:
685 self-selected surfers living within Australia with a mean (±) age of 31.7 ± 12.9 (range = 12-67) years participated in this research.

Methods:
This research involved a retrospective survey of surfers who completed an online survey that comprised of 44 major questions.

Results:
Respondents to this survey reported spending on average of 8.3 (in winter) to 10.9 (in summer) hours per week in the water. 38.4% (272 of 685) of the respondents indicated that they had sustained an injury in the 12 months prior to completing the survey, which was severe enough to keep them out of the water while it healed. A number of these had sustained more than one injury with the total number of reported injuries being 389. The three most common locations of injury were the knee (n = 62, 15.9%); the ankle and foot (n = 58, 14.9%); and the torso (n = 54, 13.9%). 19.3% (118 of 685) of respondents sustained an injury that required them to attend hospital for assessment.

Conclusion:
Participation in surfing may lead to injury with one in three surfers possibly experiencing an injury severe enough to keep them out of the water for varying periods of time in any 12 month period. It is speculated that some of these injuries are likely a product of the total time spent surfing, which may increase the stress on key anatomical structures.

Keywords:
Water sports, sporting injuries

INTRODUCTION

Surfing is an iconic Australian pastime enjoyed by 2.5 million participants in this country[1.] With its origins in Polynesian culture, evidence suggests that Hawaiians were making primitive surfboards and using them to ride waves around 1,000 years ago.[2] The first World Championship in the sport was held in 1964 in Australia and there is now a World Tour that has both male and female events with competitors vying for significant prize money and the right to be crowned World Champion. Yet, in spite of its obvious appeal and popularity, research into the safety of surfing is sparse.

Surfers vary in their level of involvement within the sport, from those who only get to the water occasionally to those who consider it an essential aspect of their daily lives. A typical surfing session is approximately two hours long[3] but it is not unusual for surfers to spend longer than this in the water and to surf more than once a day. While surfing is seen as a healthy outdoor activity, safety is an important issue. The nature of the environment means that it is inherently dangerous and deaths have been reported in the wider media from drowning and shark attacks directly linked to participation in the sport. A recent study on adventure sports in New Zealand reported surfing as the fourth highest for injury claims, with an injury rate of 11.1 per 1000 participants.[4] However, published research of surf injuries in Australia is limited with some dating back more than 2 decades.[5,6] Investigation into aspects of surf safety have included marine hazards,[7] otological issues,[3] sun exposure,[8] types of injuries or mechanisms[3,7,9] and injury rates.[3,4,10] There are also important physiological aspects related to participation that have not previously been considered. For example, much of the time in the water is spent on paddling,[11,12] which is a highly demanding activity[11] with consequent potential impacts on hydration.

In their research utilising an online survey of American surfers, conducted over an 18 month period, Nathanson et al.[13] reported an injury rate of 0.9 injuries/surfer/year with lacerations being the most common injury type (42% - sample size of 1,348). This was followed by contusions (13%), sprains/strains (12%) and fractures (8%). Thirty-four precent of reported injuries were to the head and 34% to the lower extremities. Research conducted in Australia in 2003 reported a lower injury rate among surfers of 0.26 injuries/surfer/year and 2.2 injuries/1000 surfing days or 1.1 injuries/1000 hours.[3] This study involved survey interviews of 646 surfers over a 4 month period (February to May, 2003) with injury to the lower limb accounting for 45.8%, and head/face accounting for 26.2% of all injuries recorded. It was reported that approximately 50% of all injuries (n = 168) were lacerations and approximately 25% were sprains. Dislocations and fractures combined accounted for approximately 20% of all injuries. In a small (N = 83) retrospective study utilising a combination of hospital emergency department admission surveys and local general practice medical centre records, Roger[14] reported that 80% (n = 66) of all reported surf injuries were lacerations with almost 56% (n = 37) of these being to the head.

Research in 1983 using a postal survey recorded an injury rate of 3.5 injuries per 1000 surfing days.[6] These authors at the time predicted that the frequency of injuries to the shoulder would increase as a result of the paddling stress associated with the development of shorter surfboards. They also speculated that the increased time spent in the water, which was a result of improved wet suit design, would also add to these numbers. However, using the results from Nathanson et al.[13] and Taylor McDonald et al.[3] it appears that these predictions have not been borne out.

Given the sparse level of research information currently available on injuries related to this sport, combined with its high number of participants, it seems appropriate to conduct further analysis into the nature of injuries and their prevalence associated with participation. The results of such research could then be used to implement educational and training based programmes designed to reduce the injury prevalence among the sport's participants.

This study investigated specifically the current rates of participation and time spent by surfers in this pastime along with their pre-activity preparation strategies and hydration practices. This research also investigated the prevalence of surf related injuries in the twelve months immediately preceding this study and the nature of these reported injuries.

METHODS

A self-selected sample of surfers residing in Australia was invited to participate in this research. The survey instrument for this research was a contextually modified (i.e. designed for the sport of surfing) from a previously used version applied to the sports of baseball,[15] cricket[16] and rugby league[17], and adapted for on-line distribution – copies of the survey are available from the lead author upon request. Potential respondents to this survey were advised of its availability via a range of online surf industry sources. However, the principal source of these was the project's industry partner, Surfing Australia, who, in addition to distributing notification directly to its membership by email, also provided a "news" story and link on the homepage of its corporate web site. Additional media releases were distributed to general electronic and print media outlets and surf specific publications promoting this research and inviting interested surfers to consider participating.

Mailed questionnaires are typically accompanied by a cover letter explaining the purpose of the survey and encourage potential respondents to participate in the associated research. However, given that this questionnaire was administered online a cover letter was not warranted. Notwithstanding this, the web site housing the survey had an introductory page that provided a brief overview of the survey, how to complete it and asked for a response within a specified time frame – the survey was made available online for approximately 4 weeks.

The survey tool used a combination of "yes/no", checklist and simple response questions. The first 4 pages presented information related to the rationale for the research, ethics approval and consent, and tips on how to complete and submit the survey successfully. The remaining 10 pages of the survey contained a number of categories of questions but the ones directly related to this current analysis asked questions related to:

i Time spent on surfing and associated information, e.g. hours surfed per week, types of surf, location, equipment used.

ii Personal information e.g. age, gender, whether a recreational or competitive surfer, travel time to surf.

ii Surf safety and injuries, e.g. pre-participation practices, types of injury sustained in past 12 months and their perceived consequences.

Institutional ethics approval was granted for the project (ECN-09-148). After the initial development of the survey form, it was distributed to relevant professionals in the sport/sports injuries field for their input and feedback regarding face validity and relevance of questions. This group was comprised of three sports scientists, a research methodologist, a sport psychologist and the Sport Development Manager for Surfing Australia. This process resulted in a number of small changes being made to the survey tool. This was followed by a pilot study being completed by a total of 27 respondents representative of the target population (i.e. surfers). This process allowed for further refinement and modifications of the survey instrument.

Predominantly a descriptive analysis was employed to summarise and present the results of each question utilising SPSS (IBM SPSS Statistics, ver. 18.0). Cross tabs and chi-squared tests of independence were also utilised to cross-tabulate categorical variables and to thus display their bivariate relationships (association). Where appropriate t-tests were also utilised to test for differences between identified groups of results for continuous variables (e.g. age, years surfed, etc.) and chi-squared test for independence for categorical variables (e.g. type of surf craft, wave size, etc.). An alpha level of $p \leq 0.05$ (95% test of significance) was used for all statistical comparisons. All data were summarised using descriptive statistics (mean [SD]), frequencies and percentages.

RESULTS

A total of 772 persons participated in the survey. However, some respondents did not complete all questions. For a total of 87 participants, the amount of missing data was significant and therefore not included. On this basis descriptive analyses were based on 685 respondents who completed the survey fully.

A total of 585 males, 96 females and 4 transgender persons responded to the survey. The overall mean (SD) age was 31.7 ± 12.9 (range = 12-67) years, with a median of 30 years. Males and females, with a mean age of 31.9 ± 12.9 and 29.7 ± 12.2 years respectively, were very similar in age. The majority of respondents indicated that their place of residence was New South Wales (53.9%), followed by Queensland (17.4%), the Australian Capital Territory (8.3%), Western Australia (7.5%) and Victoria (5.3%). South Australia (3.4%), Tasmanian (2.3%) and the Northern Territory (1.5%). Three (0.4%) respondents indicated their place of residence as "other".

The majority of respondents (71.5%) typically described themselves as "recreational" surfers with 62.4% surfing a short board. The next most popular category of surf craft being used by respondents was a bodyboard (20.3%). A small number (3.2%) of respondents described themselves as being professional surfers who derived their income from the sport. More than half of all respondents (52.4%) had never been a member of a boardriders club, while 23.1% were lapsed members.

The average number of hours per week surfed by respondents in summer was 10.9 ± 8.50 and in winter was 8.3 ± 6.98. Respondents indicated that typically they surfed with a friend (32.9%) or friends (22.9%), or alone (32.1%) and with the majority (78.7%) surfing between 1-2.5 hours on each occasion. However, a number of respondents (19.6%) indicated that they generally surfed for longer than this with sessions lasting a minimum of 2.5 hours and exceeding 3 hours. The majority of respondents (63.2%) also indicated that they typically surfed at beach breaks with a small number (4.1%) indicating that they surf off-shore reefs. The vast majority of respondents (80.7%) most liked surfing waves between 0.9-1.8 metres.

When asked about their pre- and post-surfing preparation only 21.4% (n = 132) of respondents (n = 617) "always" conducted some form of warm-up routine prior to going in the water, while 17.2% (n = 106) indicated that they "never" conduct a warm-up routine. Further, just 1.9% (n = 12) conduct some form of cool-down routine after their session. Similarly, just 21.4% (n = 132) indicated that they "always" drank additional fluids, such as water, prior to going in the water as a means of promoting adequate hydration, while 24.8% (n = 153) indicated that they "never" drank additional fluids before surfing. In contrast 68.4% (n = 422) of respondents "mostly" or "always" drank fluids after their session.

Two hundred and seventy-two respondents (44.5% of 611) indicated that they had sustained a total of 389 injuries related to their surfing in the 12 months prior to the survey that was severe enough to keep them out of the water while the injury healed/recovered. The general location of these injuries (where specified), injury type, and frequency based on these responses are presented in Table 1. The top three most frequent (anatomical) locations of these injuries were to the knee (15.9%), the ankle/foot (14.9%), and torso (13.9%). Of these (n = 611), 19.3% (n = 118) were required to attend hospital for various forms of assessment/treatment. These included setting of fractures, sutures, tests, observation, x-ray, and medical advice. Twenty-five respondents were admitted into hospital requiring a stay of between 1-9 nights in duration.

Table 1: *Rank order of injury according to frequency rate reported by respondents (n = 272).*

Location of injury (category total and % of all incidences n = 389)	Injury description	Total reported injuries (n = 389) f	%	% of reported cases* (n = 272)
Head (f = 50; 12.8%)	Head laceration	15	3.9	5.5
	Face laceration	10	2.6	3.7
	Eye - injury	6	1.5	2.2
	Ear drum	5	1.3	1.8
	Ear injury	4	1.0	1.5
	Nose - fracture	4	1.0	1.5
	Head - concussion	3	0.8	1.1
	Face - nerve damage	1	0.3	0.4
	Jaw - dislocation	1	0.3	0.4
	Eye socket - fracture	1	0.3	0.4
Neck and spine (f = 31; 7.9%)	Neck-injury unspecified	13	3.3	4.8
	Spinal - disc injury	6	1.5	2.2
	Spine - fracture	5	1.3	1.8
	Neck - nerve pain	2	0.5	0.7
	Neck - vertebrae injury	2	0.5	0.7
	Neck - spine vertebrae injury	1	0.3	0.4
	Neck - fracture	1	0.3	0.4
	Spine - fracture	1	0.3	0.4
Shoulder (f = 51; 13.1%)	Shoulder - injury unspecified	24	6.2	8.8
	Shoulder - dislocation	14	3.6	5.1
	Rotator cuff injury	6	1.5	2.2
	Shoulder - laceration	3	0.8	1.1
	Shoulder AC joint	2	0.5	0.7
	Collarbone - fracture	2	0.5	0.7

Table continued over page…

… Table 1 continued: *Rank order of injury according to frequency rate reported by respondents (n = 272)*

Torso (f = 54; 13.9%)	Lower back-injury	30	7.7	11.0
	Ribs - fracture	7	1.8	2.6
	Ribs - cartilage damage	5	1.3	1.8
	Ribs - soft tissue injury	4	1.0	1.5
	Back - laceration	3	0.8	1.1
	Back - soft tissue injury	2	0.5	0.7
	Thorax - soft tissue injury	1	0.3	0.4
	Lung-collapsed	1	0.3	0.4
	Pancreatitis (blunt trauma)	1	0.3	0.4
Arm including hand (f = 27; 6.9%)	Arm/Elbow unspecified	4	1.0	1.5
	Hand/wrist/finger - injury	4	1.0	1.5
	Arm/Elbow - laceration	3	0.8	1.1
	Hand/Finger - laceration	3	0.8	1.1
	Finger - fracture	3	0.8	1.1
	Elbow - dislocation	2	0.5	0.7
	Hand - fracture	2	0.5	0.7
	Elbow - fracture	1	0.3	0.4
	Wrist - fracture	1	0.3	0.4
Hip/groin/leg general (f = 35; 9.0%)	Leg - soft tissue	8	2.1	2.9
	Groin region - soft tissue injury	6	1.5	2.2
	Leg - laceration	6	1.5	2.2
	Hip - injury	5	1.3	1.8
	Hamstring - soft tissue injury	5	1.3	1.8
	Hip - laceration	2	0.5	0.7
	Leg - injury	2	0.5	0.7
	Leg - fracture	1	0.3	0.4

Table continued over page…

This equates to a mean rate of occurrence for all respondents indicating that they had sustained a severe injury (i.e. one that kept them out of the water while it healed) of 3.5 ± 3.4 (median = 2.6) injuries per 1000 hours surfed in the 12 month reporting period. There was no statistically significant ($p < 0.05$) differences in the rate of injury by gender ($p = 0.08$), age ($p = 0.31$), type of board used ($p = 0.14$) or size of waves surfed ($p = 0.21$) with chi-squared tests. However, there was a significant difference between the levels of competition ($p < 0.001$) and surf locations ($p < 0.03$), with those competing at a national level (e.g. Australian Titles) or surfing off-shore reefs respectively being more likely to injure themselves. Injury occurrence was also positively ($p < 0.001$) linked to the total hours surfed, irrespective of time of year, and age.

Respondents reported a total of 195 separate lacerations, which indicates that some respondents had multiple lacerations in this period. Of these 39.5% (n = 77) were identified as requiring stitches. Table 2 provides a summary of the anatomical location where a respondent had a laceration sustained from surfing in the 12 months prior to completing the survey.

Respondents were asked to indicate if, in their opinion, the injuries sustained while surfing in the 12 month period immediately before participating in the survey had impacted negatively on them in some way. They were asked to select from a list of 5 "consequences" (Table 3). 17.6% (n = 91) of respondents indicated that they believed they had incurred additional medical cost associated with their injuries that were not covered by their private health insurance/cover. A total of 1.9% (n = 10) retired early from surfing as a result of their injuries and 34.6% (n = 178) felt that they were currently experiencing limitations in their ability to carry out normal recreational activities due to their surf related injuries. Finally, 6.6% (n = 34) perceived that they had significant loss of income due to extended periods of recovery and/or rehabilitation from their injuries and 7.8% (n = 40) perceived that their injuries had impacted on their ability to perform work for which they had previously been trained.

… Table 1 continued: *Rank order of injury according to frequency rate reported by respondents (n = 272)*

Knee (f = 62; 15.9%)	Knee injury unspecified	34	8.7	12.5
	Knee - laceration	8	2.1	2.9
	Knee - dislocation	8	2.1	2.9
	Knee - cartilage injury	7	1.8	2.6
	Knee - fracture	3	0.8	1.1
	Knee-surgery unspecified	2	0.5	0.7
Ankle and foot (f = 58; 14.9%)	Ankle - soft tissue injury	18	4.6	6.6
	Foot - laceration	18	4.6	6.6
	Foot/Toe injury	9	2.3	3.3
	Foot - fracture	5	1.3	1.8
	Ankle - fracture	4	1.0	1.5
	Ankle - laceration	2	0.5	0.7
	Toe - fracture	2	0.5	0.7
Miscellaneous (f = 25; 6.4%)	unspecified	11	2.8	4.0
	Skin cancer†	3	0.8	1.1
	Rash	2	0.5	0.7
	Stitches	2	0.5	0.7
	Surgery	2	0.5	0.7
	Cuts	2	0.5	0.7
	Nearly drowned†	1	0.3	0.4
	Sea ulcers†	1	0.3	0.4
	Infection	1	0.3	0.4
	TOTAL	389	100.0	143.0

f = frequency of injury reported in each location.
**Respondents may have reported multiple injuries.*
†Reported by respondent as an injury and included in results.

Table 2: *Anatomical site of lacerations as a result of surfing (n = 117).*

Location on body	Responses f	Responses %	% of reported cases* (n = 117)
Head	34	17.4	29.1
Face	28	14.4	23.9
Neck/back	9	4.6	7.7
Chest/abdomen	1	0.5	0.9
Arm	9	4.6	7.7
Hand/finger	10	5.1	8.5
Upper leg	2	1.0	1.7
Knee	10	5.1	8.5
Lower leg/ankle	22	11.3	18.8
Foot/toe	65	33.3	55.6
Unspecified	5	2.6	4.3
Total	195	100.0	166.7

f = frequency of injury reported in each location.
**Respondents may have reported multiple lacerations.*

Table 3: *Perceived consequences of injuries sustained by respondents during surfing or after retirement from surfing.*

Perceived consequence	No	Unsure	Yes	n
Significant loss of income due to extended periods of recovery and/or rehabilitation	462 (89.7)	19 (3.7)	34 (6.6)	515
Limitations with respect to job opportunities that you were previously trained for	454 (88.3)	20 (3.9)	40 (7.8)	514
Medical costs incurred by you that were not covered either by a health fund or club insurance	405 (78.3)	21 (4.1)	91 (17.6)	517
Limitations on current ability to carry out normal recreational activities e.g. walking, gardening, etc.	309 (60.0)	28 (5.4)	178 (34.6)	515
Early retirement from surfing due to injury	483 (93.6)	23 (4.5)	10 (1.9)	516

N.B.: Not all respondents answered this question. Figures in () are percentages.

DISCUSSION

By its very nature the surf can be a hostile and dangerous environment. This research has provided a valuable insight into injury prevalence and injury consequences over a 12 month period within the sport of surfing. One in three surfers is likely to experience one or more surf related injuries as a result of their participation in the sport in any given 12 month period. The mean injury rate of 3.5/1000 hours surfed is higher than that reported by Taylor McDonald et al.[3] of 1.1/1000 hours surfed but is still relatively low when compared with other forms of recreational sport. [3]

The most common form of consequence associated with surfing was a laceration, which agrees with previous research[3,13,14] reporting the most common injury among surfers as being a laceration of some kind. Notwithstanding this, the four most common areas of the body where an injury was reported related to the knee, ankle/foot, torso (principally the lower back) and shoulder. It is speculated that some of these injuries are a result of the mechanical stress and repetitive nature of common movements associated with this sport. This may be particularly the case for competitive surfers, who in this research were statistically more likely to injury themselves. This is also possibly because they can compete in more extreme wave conditions, produce more dynamic and powerful surfing manoeuvres, and have a much higher volume of exposure by virtue of the amount of surfing they do. Combined these factors are likely to increase the stress on major joints of the lower body and back. However, research is needed into the mechanical loads that surfers are exposed to in order to establish a more definitive link with injury prevalence. The authors note that many professional surfers undertake systematic supplemental training that often involves core and proprioceptive work and so the effectiveness of this type of training as an injury reduction strategy needs further examination. Nevertheless, it seems logical that an injury prevention programme that focuses on increasing the stability and mobility around these joints may have an important injury reduction role. Such a programme should aim to address any imbalances that may exist and that typically occur as a result of repetitive movements, and aim to achieve muscular balance to aid in protection of joints and connective tissue. For example, functional balance training on unstable surfaces may not only improve proprioception but also reduce the likelihood of injury to the ankle, knee and lower back.[18-20]

A total of 81 (29.7%) injuries were sustained to the head (f = 50; 12.8%), neck and spine (f = 31; 7.9% combined). This is similar to the rates reported elsewhere[3,13]. In this study injury to the head included lacerations, eye and ear injury, nose and eye socket fracture, jaw dislocation, nerve damage and concussion. Intuitively it would seem likely that the wearing of some form of protective helmet might play a part in reducing these types of injuries. While protective head gear is available and readily accepted in a range of sports (e.g. boxing, cycling, football, cricket) its use by surfers is very low ranging between approximately 2-10%.[13,21] It has been reported[21] that significant numbers of surfers (n = 144 of 395; 36.5%) who did not currently wear any form of protective headgear indicated that they did not recognise a need. This suggests that a significant number of surfers do not perceive a strong enough risk of head injury. Our study did not ask respondents if they wore protective headgear while surfing.

Some injuries were severe enough to stop a surfer from participating in the sport while the injury healed and also required a small number of participants to attend and/or be admitted to a hospital. In a relatively small number of cases some of these injuries appear to have impacted negatively on a participant's ability to generate income, ability to work in their chosen vocation, participate in other forms of recreation and resulted in additional medical costs that were not covered by some form of health insurance.

A total of 17.2% respondents indicated that they "never" conduct any form of warm-up activity prior to surfing and only 21.4% indicated that they "always" conducted some form of warm-up routine prior to going in the water. Our analysis did not establish any link between the lack of warm-up prior to entering the water and an increased potential for injury. Yet the role and benefits of an appropriately applied warm-up has been clearly articulated in the literature,[22-24] although the role of static stretching in this process as an injury prevention strategy is no longer supported.[25] It is speculated that surfers may achieve the benefits of a warm-up simply via the act of paddling out to the surf break. While this research found that the majority of respondents do not undertake a specific warm-up prior to entering the water, it is possible that the low to moderate intensity[11] of paddling out to the surf break is sufficient to adequately prepare the surfer for the more intense and explosive paddling and manoeuvres associated with catching and riding waves.

With respect to pre-surf hydration, 24.8% of respondents indicated that they "never" drank additional fluids before surfing. Given that most surfers will spend 1-3 hours in the water the need for adequate levels of hydration prior to entering the water cannot be over stated. Recreational surfing requires participants to perform numerous bouts of high intensity work interspersed with intervals of lower intensity activities.[11] This type of exercise can result in significant loss of body water characterised by a reduction in body mass.[26] This can lead to dehydration and inadequate temperature regulation, which has a detrimental effect on performance and places increased stresses on the cardiovascular and thermoregulatory systems.[27,28] Even mild fluid loss (2% of body mass) can have an impact on performance, with severe dehydration being lethal.[25,29,30]

This study has several limitations that should be acknowledged and considered when interpreting the results. Not everyone has access to the Internet and identifying lists of users for distribution management is clearly problematic. It may also be that those who respond via this medium may or may not differ from those who did not have access to the survey via the Internet. Further, while the survey instrument was adapted from one used previously in similar retrospective injury research[15-17] it was not validated. In addition, as with all retrospective research involving the self-reporting of information, the authors were relying on the honesty and ability of respondents to recollect their personal injury record accurately in the preceding 12 month period. It also assumed that all respondents had the requisite literacy skills to understand amd comprehend what was being asked in the survey.

CONCLUSION

Surfing is a popular and growing recreational and competitive sport. However, as with all sports it has inherent risks and this is reflected in the results of the current study, which shows that 1 in 3 surfers are likely to sustain at least one injury in any 12 month period severe enough to keep them out of the water. This is not unexpected given the dynamic nature of the sport and its environment. While the focus on this current research has been predominantly on recreational surfers there is evidence that competitive surfers, who execute more complex (e.g. aerial surfing) manoeuvres, may be at even greater risk of more severe injuries. To this end research into injury prevention strategies should be implemented and targeted generally across all participants.

What this study adds?

This research indicates that 1 in 3 surfers are likely to experience one or more surf related injuries as a result of their participation in the sport in any given 12 month period.

Common anatomical sites of injury are the knee, ankle/foot, and the torso.

The most common form of consequence associated with surfing appears to be a laceration.

ACKNOWLEDGEMENT

The authors wish to acknowledge the financial assistance of the New South Wales Sporting Injuries Committee who provided funding for this project through the New South Wales Sporting Injuries Insurance Scheme. Industry partner, Surfing Australia, actively supported this project by promoting participation to its members and assisting with the distribution of the online survey via its corporate web site.

References

1 Surfing-Australia. *Surfing Australia Fast Facts*. http://www.surfingaustralia.com/statistics.php. Accessed 17th November, 2011.

2 Warshaw M. *The Encyclopedia of surfing*. San Diego, CA: Harcourt, 2003: xiii.

3 Taylor McDonald D, **Bennett D, Carter M, Garewal D, Finch CF.** Acute injury and chronic disability resulting from surfboard riding. *J Sci Med Sport* 2004; **7**(4):429-437.

4 Bentley T, Macky K, Edwards J. Injuries to New Zealanders participating in adventure tourism and adventure sports: an analysis of Accident Compensation Corporation (ACC) claims. *N Z Med J* 2006; **119**(1247):U2359.

5 Lowden BJ, Pitman AJ, Pateman NA, Ross K. Injuries in international surfboard riders. *J Sports Med Phys Fitness* 1987; **27**(1):57-63.

6 Lowden BJ, Pateman NA, Pitman AJ. Surfboard-riding injuries. *Med J Aust* 1983; **2**(12):613-616.

7 Nathanson A, Bird S, Dao L, Tam-Sing K. Competitive surfing injuries: A prospective study of surfing-related injuries among contest surfers. *Am J Sports Med* 2007; **35**(1):113-117.

8 Dozier S, Wagner R Jr., Black SA, Terracina J. Beachfront screening for skin cancer in Texas Gulf coast surfers. *Southern Med J* 1997; **90**(1):55-58.

9 Avilés-Hernández I, Garcia-Zozaya I, DeVillasante J. Nontraumatic myelopathy associated with surfing. *J Spinal Cord Med* 2007; **30**(3):288-93.

10 Roger P, Llyod B. Surfboard injuries: A study over time. *Sport Health* 2006; **24**(3):21-23.

11 Meir RA, Lowdon BJ, Davie AJ. Heart rates and estimated energy expenditure during recreational surfing. *Aust J Sci Med Sport* 1991; **23**(3):70-74.

12 Mendez-Villanueva A, Bishop D. Physiological aspects of surfboard riding performance. *Sports Med* 2005; **35**(1):55-70.

13 Nathanson A, Haynes P, Galanis D. Surfing injuries. *The Amer J of Emerg Med* 2002; **20**(1):155-160.

14 Roger P. Injury in the surf. *Sport Health* 2002; **20**(2):23-28.

15 Meir RA. Weatherby RP, Rolfe MI. A retrospective analysis of major and significant injuries and their consequences reported by retired Australian baseball players. *Open Sports Med J* 2010; **4**:119-126.

16 Noble-Jerks J, Weatherby RP, Meir R. Self-reported skin cancer protection strategies and location of skin cancer in retired cricketers: A case study from the membership of the Emu Cricket Club. *J Sci Med Sport* 2006; **9**(6):441-445.

17 Weatherby RP, Meir RA, McDonald KN, Russell R. Consequences of participation in professional rugby league: A question of workers compensation? Football Australasia: Reducing the Injury List. *J Sci Med Sport* 1999; **2**[supplement 1]:10.

18 Gillett J, O'Brien L, Ryan M, Rogowski J. Strategic exercise prescription for baseball: Bridging the gap between injury prevention and power production. *Strength Con J* 2009; **31**(5):81-88.

19 Ruiz R. Functional balance training using a domed device. *Strength Cond J* 2005;**27**(1):50-55.

20 Verhagen E, Van Der Beek A, Twisk J, Bouter, L, Bahr, R, van Mechelen W. The effect of a proprioceptive balance board training program for the prevention of ankle sprains: A prospective controlled trial. *Amer J Sport Med* 2004; **32**(6):1385-1393.

21 Taylor McDonald D, Bennett D, Carter M, Garewal D, Finch C. Perceptions of surfboard riders regarding the need for protective headgear. *Wilderness Environ Med* 2005; **16**(2):75-80.

22 Safran MR, Seaber AV, Garrett WE. Warm-up and muscular injury prevention. An update. *Sports Med* 1989; **8**(4):239-249.

23 Shellock FG, Prentice WE. Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Med* 1985; **2**(4):267-278.

24 Woods K, Bishop P, Jones E. Warm-up and stretching in the prevention of muscular injury. *Sports Med* 2007; **37**(12):1089-1099.

25 Shrier I. Stretching before exercise does not reduce the risk of local muscle injury: A critical review of the clinical and basic science literature. *Clin J Sport Med* 1999; **9**(4):221-227.

26 Burke LM, Hawley JA. Fluid balance in team sports: Guidelines for optimal practices. *Sports Med* 1997; **24**(1):38-54.

27 Cochrane DJ, Sleivert GG. Do changing patterns of heat and humidity influence thermoregulation and endurance performance? *J Sci Med Sport* 1999; **2**(4):322-332.

28 Francesconi RP, Hubbard RW, Szlyk PC,*et al*. Urinary and hematologic indexes of hypo hydration. *J Appl Physiol* 1987; **62**(3):1271-1276.

29 Jennings S, Robertson S, Jennings D, *White J, Gissane C.* Body mass loss as an indicator of dehydration in summer rugby league. *Coach Sports Sci J* 1998; **3**(3):31-33.

30 Oppliger RA, Bartok C. Hydration testing of athletes. *Sports Med* 2002; **32**(15):959-971.

Occult peripheral neuropathy in an elite hockey player

Jake N Pearson, MBChB, BPhed (Hons), FASCP[1]
Stuart Mossman, MBChB, FRACP[2]
1 Capital Sports Medicine, Wellington
2 Neurology Department, Wellington Public Hospital
Correspondence
Jake Pearson
Capital Sports Medicine
131-135 Lambton Quay
Wellington
Phone: +64 4 499 5732
Fax: +64 4 499 5735
Email jakep@capitalsportsmed.co.nz

INTRODUCTION

Charcot-Marie-Tooth (CMT also known as hereditary motor and sensory neuropathy) disease is a group of hereditary progressive peripheral neuropathies named after the 3 physicians who first described this condition in the late 1800s. There are 2 main sub-types, one that affects the myelin sheath (CMT-1), and one that affects the nerve axon (CMT-2).[1] There is a typical pattern of involvement of primarily lower limb neuromuscular dysfunction, classically resulting in pes cavus, foot-drop, reduced proprioception, and reduced or absent reflexes. Although sensory loss is present on examination, it is not a major complaint. As with other neuropathies the longer nerves of the lower limbs are affected prior, and more severely, than those of the upper limbs, but with particular selection of the anterior and peroneal compartments of the leg.[2] It is the most common inherited neuromuscular condition, with an estimated incidence of at least 1 in 2500 1. Autosomal dominant is the most common form of inheritance, however there are also autosomal recessive and X-linked forms. The severity and progression is highly variable, even within an affected family, due to the large number of genetic variations and incomplete penetrance.

CASE REPORT

An 18 year old male elite age-group hockey player was referred by his national team physiotherapist after it was noted that he was unable to walk on his heels during a training exercise. On further questioning, he expressed frustration at not being able to improve his speed despite a concerted effort, and a feeling of poor balance. He had since discovered that his mother was likewise unable to walk on her heels. He was otherwise healthy and on no regular medications. On examination, there was wasting of the anterior compartments of both lower legs with prominence of the anterior tibial spine and marked wasting of Extensor Digitorum Brevis (EDB). Gait was flat-footed without a gross foot drop, but he could not walk on his heels. Resistance testing of the lower limb revealed 4-/5 weakness of ankle dorsi-flexion and toe extension, and 5-/5 of both eversion and inversion. Ankle reflexes were absent. No plantar response could be elicited. There was reduced perception of vibration at each hallux to 6 s (compared with 30 s at the thumb), but otherwise sensation testing was unremarkable. Romberg's test was negative. In the upper limb, the only abnormal finding was 5-/5 power of Abductor Pollicus Brevis. Following specialist neurologist review, bilateral nerve conduction studies revealed markedly reduced amplitude of the sensory action potential of both sural nerves and absent compound muscle action potentials of EDB. Nerve conduction velocities were near-normal, the slight slowing being attributable to dropout of fast conducting fibres. This confirmed a pattern of primary axonal loss i.e. CMT-2.

DISCUSSION

CMT is the most common inherited neuropathy for which patients may not be symptomatic often until later years with its slow progression over time. Athletes, with their increased physical demands and body awareness, may be more likely to notice mild deficits and hence present earlier than the general population. A common presentation of CMT is with ankle instability,[1] a common presenting complaint in sports medicine. Clinicians are encouraged to screen for CMT in these patients with, for example, a routine heel walking challenge (although formal manual testing is more sensitive – all subjects with normal motor strength can resist the examiner's efforts to overcome ankle dorsiflexion) and assessment of ankle reflexes. Taking a family history is important, although approximately 50% of cases are due to new gene mutations. While a pes cavus foot-type can indicate an underlying neurological condition, more commonly it relates to bone formation without an underlying neuromuscular problem.

Those diagnosed with CMT are advised to remain fit and active for the same reasons as the general population, however the implications for an elite sportsperson are less clear. For this athlete, options to improve speed are limited to concentrating on proximal muscles as attempted strengthening of his ankle dorsiflexors are ultimately likely to be unrewarding in the context of neurogenic weakness. Concentrating on optimising skill and tactical abilities through appropriate training is recommended.
For his sporting ability it is important to provide a splint to support ankle dorsiflexion and to assist in foot clearance, with the additional benefit of providing improved ankle stability.[2] He did not require this for day to day ambulation which was without any significant functional impairment. As with any peripheral neuropathy, foot care is crucial in limiting injury from impaired sensation, and podiatry involvement to optimise footwear and monitor foot condition is appropriate, particularly given the additional demands of his weightbearing sport.

A genetics referral is appropriate for advice, support and counselling regarding the risk for future offspring. All patients with CMT are advised to perform regular calf stretches to delay the development of contractures that inevitably result secondary to the progressive weakness of their antagonist muscles.[2]
The prognosis is extremely variable, but ultimately this is a progressive condition. CMT-2 tends to be more indolent than CMT-1;[1] nevertheless the effect on this patient's sporting aspirations are likely to be significant.

Conclusion

CMT is a relatively common condition that clinicians should be aware of, and consider screening for in patients presenting with cavovarus foot and ankle instability. While there is no treatment aimed at cure, there are a number of very worthwhile supportive measures that can be instigated.

References

1. Reilly MM, Murphy SM, Laura M. Charcot-Marie-Tooth disease. J Peripher Nerv Syst 2011; 16;1-16.
2. Guyton GP. Current concepts review: Orthopaedic aspects of Charcot-Marie-Tooth disease. Foot Ankle Int 2006; 27:1003-1010
3. Mallik A, Weir A. Nerve conduction studies: essentials and pitfalls in practice. J Neurol Neurosurg Psychiatry 2005; 76:ii23-ii31.

Nerve Conduction Study Interpretation Basics

(adapted and summarised from Mallik & Weir [2005][3])

Nerve conduction studies (NCSs) involve the initial electrical stimulation of a peripheral nerve, usually via a surface electrode. The time taken for the impulse to travel a known distance further along the nerve can be used to calculate nerve conduction velocity (NCV). The electrical reaction of a muscle supplied by that nerve (compound muscle action potential [CMAP]), or of a sensory nerve either proximally or distally (sensory nerve action potential [SNAP]) are typically also measured.

Peripheral nerve pathology primarily affects axons or myelin, whereas in reality it is usually a mixed picture with one predominating. In axonal loss the most striking finding is typically a reduction in the CMAP amplitude as fewer motor axons are connected to the muscle fibres. If there has been time for collateral reinnervation then the immature nerve fibres will conduct slower may result in a more spread out CMAP, a finding termed "dispersion". Particularly in conduction block there will often be a reduction in the CMAP amplitude and some dispersion due to axons' conduction velocities being variably affected. With increasing severity of axonal loss there may also be slight slowing of the NCV. With a demyelinating process the classic finding is a marked slowing of the NCV, or if severe a conduction block. The SNAP amplitude is reduced in both axonal and demyelinating pathologies, however can be very useful for locating sensory neuropathies.

The most common peripheral nerve abnormality encountered in sports medicine is a focal nerve entrapment, which will typically produce an area of focal demyelination and if severe and/or prolonged then a degree of axonal loss as well. The above is a gross oversimplification, and there are vastly more complicated aspects to NCSs, and other variations of testing including F-waves and repetitive nerve stimulation, however these are beyond the scope of this brief review. Electromyography (EMG) is a separate test that is often performed in conjunction with NCSs, and measures the electrical responses of muscle. This information can help differentiate myopathic from neurogenic muscle wasting, determine the distribution of neurogenic pathology, and provide supportive evidence for the pathophysiology of peripheral neuropathy.

Lateral Epicondylitis (Tennis Elbow)

Bruce Hamilton

Qatar Orthopaedic and Sports Medicine Hospital

Doha, Qatar

Chris Milne

Anglesea Sports Medicine

Hamilton, New Zealand

Lateral epicondilitis or “tennis elbow” is a common cause of lateral elbow pain, with a prevalence of 1.3% in 30-64 year olds in Finland.[6] Tennis elbow underplays how commonly this condition presents to the sports medicine clinic, as it is not only tennis players, but golfers, paddlers, freezing workers, typists and others who may present. Similarly, the term epicondylitis may no longer be appropriate, particularly in chronic cases, due to the pathophysiology which is now better understood as a tendinopathy of the extensor carpi radialis brevis (ECRB), and extensor tendinopathy may therefore be a more appropriate term. The exact patho-physiological process of tendinopathy remains to be determined, but at least after the acute phase, there appears to be no significant inflammation.

Patients typically present with focal symptoms just distal to the lateral epicondyle, with or without more distal radiation. Pain is exacerbated by wrist extension and grip. The differential to consider is highlighted in table one. Key features to consider are the nature of work, exacerbating factors and any increase in participation in aggravating activities. A change in racquet head or handle size, or a particular provoking event, usually in backhand should be considered. Similarly, in golf playing with the dominant hand in to a pronated a position (the slice) may cause increase stress on the extensor origin. Computer and keyboard position may be additional provocative factors to consider.

Examination of the patient includes a careful assessment of the range of motion and stability, which is typically normal, excluding significant intra-articular pathology. Tenderness seems to be on and just distal to the lateral epicondyle, but there may also be tight bands or “trigger points” distal to this. If the tenderness is predominantly distal, then a posterior interosseus nerve entrapment should be considered. Pain localising to the lateral epicondyle will be marked with resisted wrist and middle finger extension. The shoulder and neck are worth looking at, to exclude additional factors, and in a sportsperson, assessing the kinematic chain is worthwhile.

Investigations may depend on your clinical setting. In my current workplace, with no access limitations, I often perform an x-ray and ultrasound to assess the state of the joint and the tendon. MRI may be useful if there is diagnostic uncertainty, or signs of intra-articular pathology. In reality these investigatons have rarely changed the initial management in the presence of no clinical uncertainty, but may provide security prior to any intervention.

Treatment

Depending on the situation I typically try to have patients avoid the aggravating activity for a variable period of time – usually weeks. This may mean avoiding tennis, golf or paddling, if it has not been stopped already. If activity must continue, I do encourage the use of an elbow brace. My thinking is that this alters the force translation, potentially offloading the origin – they do seem to provide symptomatic relief. I tend to avoid NSAIDs when possible due to their negative impact on both muscle and tendon regeneration, but encourage movement and stretching with ice, for pain relief. My mainstay of initial treatment is to substitute the aggravating activity with a controlled aggravating activity. That is controlled isometric, concentric and eccentric wrist extension, depending on the severity of symptoms. Rather than use weights, tins of cat food or elasticated bands, when possible, I instruct the patient to perform repetitions of wrist extension (straight arm) against their contralateral hand – three sets of 10, at least twice a day. As with the “Alfredson” achilles programmes, this may provoke symptoms for a period of time. Reducing pain means time to increase the resistance and progress from concentric to eccentric in the usual way. Not all patients seem to manage this and so using a weight over the edge of a table is a fall back for me! This is combined with a stretching programme for the lateral elbow with nerve stretches thrown in for good measure, although I acknowledge the limited evidence for this approach.[5, 7, 8] I would typically trial this for a minimum of 4- 8 weeks depending on the clinical situation. Assessment of grip position for golf and racquet sports is worthwhile, prior to a graduated return if symptoms are resolving.

In the recalcitrant epicondylitis, more invasive options are available, including GTN patches, corticosteroid injections, platelet rich plasma (PRP), autologous blood[1] and in Europe traumeel and activegan. My preference these days is to avoid corticosteroid injections unless there are no other options, but there is evidence that they are effective in the early phases of lateral epicondylitis to enhance symptom relief (although this flies in the face of the described pathology).[2, 8] Unfortunately, they do not have any sustained benefit over physiotherapy, and they do carry additional risk. My preference when able is to trial PRP, although the evidence for this is again limited.[4] My approach to rehabilitation after any injection is as outlined above - after a couple of days recovery. If a patient won’t buy in to the rehabilitation programme, I am reluctant to inject. I do like to follow up all injections at one week to assess, and a flare of pain for a couple of days is not unusual after any injection technique.

I have no experience with referring patients for surgical treatment, usually release of the ECRB origin. It is clear that the proceedure can be effective, when reserved for those patients in which the conservative means outlined above have failed and it has the additional benefit of identifying concurrent pathology which may contribute to the resistance to treatment.[3]

Table 1: *Pragmatic Differential Diagnosis of Lateral Elbow Pain*

Posterior interosseus Nerve Entrapment
Referred – Neck / Shoulder / trigger points
Regional Pain Syndrome
Impingement
Seronegative arthropathy
Osteochondritis Dessicans Capitellum

References

1. Edwards SG,Calandruccio JH. Autologous blood injections for refractory lateral epicondylitis. *J Hand Surg* [Am]. 2003; **28**: 272-8.
2. Hay E. Pragmatic randomised controlled trial of local corticosteroid injection and naproxen for treatment of lateral epicondylitis of elbow in primary care. *British Medical Journal*. 1999; **319**: 964-968.
3. Owens B, Murphy K,Kuklo T. Arthroscopic release for lateral epicondylitis. Arthroscopy: *The Journal of Arthroscopic and Related Surgery.* 2001; **17**: 582-587.
4. Peerbooms JC, Sluimer J, Bruijn DJ,Gosens T. Positive Effect of an Autologous Platelet Concentrate in Lateral Epicondylitis in a Double-Blind Randomized Controlled Trial: Platelet-Rich Plasma Versus Corticosteroid Injection With a 1-Year Follow-up. *American Journal of Sports Medicine*. 2010; **38**: 255-262.
5. Pienimaki T, Tarvainen T, Siira P,Vanharanta H. Progressive Strengthening and Streching Exercises and Ultrasound for Chronic Lateral Epicondylitis. *Physiotherapy*. 1996; **82**: 522-530.
6. Shiri R, Viikari-Juntura E, Varonen H,Heliovaara M. Prevalence and Determinants of lateral and Medial Epicondylitis: A Population Study. *American Journal of Epidemiology*. 2006; **164**: 1065-1074.
7. Smidt N, Assendelft W, Arola H, Malmivaara A, Green S, Buchbinder R, van der Windt D,Bouter L. Effectiveness of physiotherapy for lateral epicondylitis: a systematic review. *Annals of Medicine*. 2003; **35**: 51-62.
8. Smidt N, van der Windt D, Assendelft W, Deville W, Korthals-de Bos I,Bouter L. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. *The Lancet*. 2002; **359**: 657-662.

Comment 1

Jordan Salesa and Louise Johnson
Physiotherapists
High Performance Sport New Zealand

This is a reasonably common condition in athletes and the general public and is one frequently treated by physiotherapists - some key points in our approach are outlined below.

Examination

ECRB inserts into the base of the middle finger metacarpal and hence we agree resisted testing of the middle finger is important along with wrist extension. We would emphasise the need to test with the elbow flexed and extended – extension being more sensitive as it increases the compression on the tendon. Assessing grip strength (again in elbow flexion and extension) is also a valuable reassessment sign and has been included as a predictor of outcome alongside age.[3] We have used a simple, inexpensive dynamometer for measuring grip strength - alternatively it can be rated against pain. Additionally we would add the resisted assessment of supinator which is often involved in this condition – commonly this is done with the handshake hold, ramping up the resistance until you can break the hold.

Lastly we would place greater emphasis on assessment of the kinetic chain, especially the shoulder external rotators, posterior chain, thoracic spine and even further afield! Optimal function of the kinetic chain is crucial to unloading the ECRB tendon and providing a long-term solution.

Treatment

As with Bruce and Chris key for us initially is to unload the tendon and ensure pain levels can be kept in an acceptable range so you can then specifically start an uploading/ strengthening programme on the tendon. Several clinical tools are useful to modulate and modify the tendon pain e.g. soft tissue massage, trigger point release, acupuncture – especially accessing the posterior interosseus nerve (POI) and C7 spinal level, braces, Ktape, mobilisation with movement (MWMs) - especially lateral glides, nerve flossing, high frequency TENS and using ice massage before and after loading. Activity can also be modified e.g. palm up lifting to ensure biceps flexes the elbow, avoiding reaching ADL's like excessive mouse work, advice on gripping pattern and keeping weighted objects close to the body.

Subsequently we're keen to monitor the pain closely. Jill Cook speaks of "Keeping the tendon cool" and avoiding compressive loads (for a great podcast from Jill on tendinopathy go to http://itunes.apple.com/au/podcast/physioedge/id454714085). We set an acceptable pain level e.g. 2/10 and monitor this during exercise, after and the next morning to ensure you are not overcooking the tendon.

Kinetic chain enhancement is mandatory. Focus on addressing scapula kinematics and posterior chain strength e.g. if external rotation of the shoulder is not ideal then the wrist extensors will want to help thus overloading the tendon compression. Try strengthening the shoulder external rotators with a theraband on a door handle ensuring it is performed thumbs up – win win. While you are relatively unloading the tendon also look at the lower quarter - go mad on the Kinetic chain work!

A comment on the use of corticosteroid injections - we're not so keen on their use in athletes as the long term outcome is reportedly worse.[2]

Exercise Principles

No two tendons are alike and an understanding of the state of the tendon will help in your exercise prescription. A healthy young tendon that has had a sudden load and is therefore in a reactive state may just need two weeks rest. In contrast a tendon with lots of miles on the clock, in a state of disrepair and/or degeneration may take months to decrease symptoms. It is these tendons that are a challenge. We work more closely within set pain levels – avoiding spikes in pain.

This is based on the thinking of Jill Cook and Craig Purdam when working with a tendon problem that is compression based.[1]

We suggest a base line daily dose of exercise e.g. 50 reps of broom handle rotations or eccentric wrist curls. We then use cyclic high, low, medium tendon load days – knowing it takes three days for the cellular recovery post the high load day and ensure these days have a combination of concentric and eccentric exercises. What constitutes these loads will be determined by the irritability of the tendon – hence the grip strength test is a good indicator. Try really hard never to prescribe more than 3 exercises for any given load day. A useful trick is to use a dose of isometrics daily (high intensity, 40s hold, 4-6 reps) pre-training as anecdotally this is great for pain modulation in the subsequent training session (like a dose of panadol!).

We are also mindful that it will take 100 days for the tendon matrix to remodel and have useful collagen. A trick is to think of that as 12 weeks – then cut that in half to 6 weeks, then 3 weeks and 10 days. Have a plan for these 3 months in your head but only prescribe 10 days at a time. Expect a slow consistent change in critical signs every 10 days or so. Usually if the load levels are correct then a nice pain reduction is achieved in the first 6 weeks however all rehab strengthening must be continued for the 12 weeks to ensure longevity of tendon health. Often a maintenance programme has to be set for the length of an athletic career.

REFERENCES

1 Cook J L, & Purdam C R. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *British Journal of Sports Medicine* 2009; **43**(6), 409-416. doi:10.1136/bjsm.2008.051193

2 Coombes B K, Bisset L, & Vicenzino B. Efficacy and safety of corticosteroid injections and other injections for management of tendinopathy: a systematic review of randomised controlled trials. *Lancet*, 2010; **376**(9754), 1751-1767. doi:10.1016/s0140-6736(10)61160-9

3 Vicenzino B, Smith D, Cleland J & Bisset L. Development of a clinical prediction rule to identify initial responders to mobilisation with movement and exercise for lateral epicondylalgia. *Manual Therapy* 2009; **14**(5), 550-554.

Comment 2

Paul Wharam

Sports Physician

Auckland, New Zealand

Bruce and Chris have covered the aetiology, investigations and treatment for lateral epicondylitis (tendinosis of the common extensor tendon). I would like to stress that the basic investigations should include both an X-ray and an ultrasound scan. MRI scanning is occasionally useful if intra-articular or ligamentous pathology is suspected.

My own treatment of lateral elbow epicondylitis has changed over the last 20 years. Working in the UK with a two month wait for physiotherapy I initially advised a local corticosteroid injection which I would repeat if symptoms returned. I then studied acupuncture and practiced periosteal pecking of the common extensor tendon origin (multiple stabbing of the tendon origin with an acupuncture needle as tolerated by the patient). Since working in a referral based sports medicine practice I have advocated the use of eccentric strengthening and occasionally added a local corticosteroid injection in patients compliant with strengthening but limited by discomfort.

More recently I have trialled autologous blood injections and have been surprised by the results. I now suggest that corticosteroids injections be avoided as lateral epicondylitis is not an inflammatory condition despite the "itis". Working in a referral based practice most patients have recalcitrant epicondylitis. I initially advise them to continue with a physiotherapy monitored strengthening programme for three months but check their exercise techniques and loads being applied to the tendon. I avoid NSAIDs but allow patients to use a forearm brace and encourage stretching of the forearm extensor muscles on a regular basis. I stress that avoidance of excessive loads will help the tendon heal.

Patients that have failed this regime I consider autologous blood injections which I perform under ultrasound guidance, with local anaesthetic. I inform patients that the evidence for this procedure is low and encourage them to do there own internet research. I explain that the tendon may be sore once the anaesthetic effect has reduced. Following the injection, I stop all loading activities and eccentric strengthening (I initially continued these but found better results in those who ignored this advice!) and review on a six weekly basis - as collagen formation is then maximal.

Once lateral elbow pain has resolved I get patients to return to strengthening and subsequently loading activities. In my experience the hypoechoic areas in the tendon reduce but do not normalise as pain resolves.1 I have not collated all treatment results but feel that my results are similar to those reported by Edwards with 70-80% experiencing improvement,[2] but not the 100% reported by Connell.[1]

Patients that don't improve with autologous blood injections are given alternative treatment options including a return to physiotherapy, acupuncture, shock wave therapy and surgery.

At the recent Wimbledon tennis conference (June 2012) Karim Khan and Michael Kjaer presented on mechanotherapy and mechanotransduction in tendinosis. Both talks concentrated on the recent popularity of platelet rich plasma (PRP) and the lack of evidence for its use in tendinosis.[3] One of the delegates present did mention his own published case studies and when I asked if they felt that all their comments were equally applicable to autologous blood injections (given that it is far simpler and less expensive to perform than PRP), the answer was yes. In the recent literature regarding lateral epicondylitis, autologous blood injections have been compared to PRP injections with similar (approximately 70%) improvement in both groups.[4] Furthermore, both autologous blood injection and Shockwave therapy have been shown to be superior to corticosteroid injections in the management of lateral elbow pain.[5,6] By contrast, a study comparing autologous blood, saline and corticosteroid injections found similar improvement in all groups.[7] As a result of the ambiguity in the literature, further well designed studies are required, and optimal management is yet to be detemined.

REFERENCES

1. Connell D.A, et al. Ultrasound-guided autologous blood injection for tennis elbow. *Skeletal Radiology* 2006; **35**(6):371-7).

2. Edwards S.G, et al. Autologous blood injections for refractory lateral epicondylitis. *J Hand Surg* 2003; **28A**:272–278.

3. de Vos R.J, et al. Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. *JAMA* 2010 ; **303** : 144 – 9.

4. Creaney L, et al. Growth factor-based therapies provide additional benefit beyond physical therapy in resistant elbow tendinopathy: a prospective, single-blind, randomised trial of autologous blood injections versus platelet-rich plasma injections *Br J Sports Med* 2011; **45**:966–971.

5. Kazemi M, et al. Autologous blood versus corticosteroid local injection in the short-term treatment of lateral elbow tendinopathy: A randomized clinical trial of efficacy. *Am J Phys Med Rehabil* 2010; **89**:660–667.

6. Ozturan K.E, et al. Autologous Blood and Corticosteroid Injection and Extracoporeal Shock Wave Therapy in the Treatment of Lateral Epicondylitis. *Orthopedics* 2011; **33**(2):84-91).

7. Wolf J.M, et al. Comparison of Autologous Blood, Corticosteroid, and Saline Injection in the Treatment of Lateral Epicondylitis: A Prospective, Randomized, Controlled Multicenter Study. *J Hand Surg* 2011; **36A**:1269–127).

Paul Wharam

Paul Wharam is a sports physician working in Auckland (Millennium Institute of Sport and Queen Street, CBD). Paul qualified in the UK and has subsequently worked both in Australia and New Zealand. His current interest is the use of ultrasound guided injections as will become evident if you continue reading the journal. His latest foolish challenge is to compete in the 2013 Maratona cycle event in the Italian Dolomite mountains.

MICHAEL PAPADAKIS, SANJAY SHARMA, NATHAN RIDING, OTHMAN SALAH, BRUCE HAMILTON, HAKIM CHALABI, MATHEW G WILSON

The Black African's Heart

Michael Papadakis,[1] Sanjay Sharma,[1]
Nathan Riding,[2] Othman Salah,[2]
Bruce Hamilton,[2] Hakim Chalabi[3]
Mathew G Wilson[2]

1. Department of Cardiovascular Sciences, St Georges University of London, UK
2. Department of Sports Medicine, ASPETAR, Qatar Orthopaedic and Sports Medicine Hospital, Doha, Qatar
3. CMO's Office, ASPETAR, Qatar Orthopaedic and Sports Medicine Hospital, Doha, Qatar

INTRODUCTION

Regular participation in intensive physical exercise is associated with several structural and electrophysiological cardiac adaptations that enhance diastolic filling and facilitate a sustained increase in the cardiac output that is fundamental to athletic excellence. Such cardiac adaptations are collectively referred as the "Athlete's Heart" and are frequently reflected on the 12-lead ECG and imaging studies. Thorough knowledge relating to exercise associated cardiovascular adaptation is imperative for the purposes of differentiating physiological adaptation from cardiac pathology, since an erroneous diagnosis of cardiac disease has potentially serious consequences for the athlete's physical, psychological, social and financial wellbeing.

Accurate interpretation of the athlete's ECG and echocardiogram is crucial, particularly when one considers the continuous expansion of pre-participation screening programmes. This short review attempts to highlight the impact of African/Afro-Caribbean ethnicityally upon the cardiovascular adaptations to exercise and provides a practical guide for the interpretation of baseline investigations in this cohort.

Cardiovascular Adaptation in African/Afro-Caribbean Athletes

Structural Adaptations

Studies by Papadakis et al.[1] and Basavarajaiah et al.[2] in highly trained, male British and French male athletes revealed that black male athletes exhibit an LV cavity size of similar magnitude to white caucasian athletes (52.6 ± 4.4 vs. 52.6 ± 4.3 mm, p>0.05) but demonstrate slightly larger LA (35.4 ± 4.5 vs. 34.7 ± 4.7 mm, p=0.002) and aortic root (30.2 ± 3.3 vs. 29.5 ± 3.3 mm, p<0.001) diameters.

The most striking difference between the two ethnic groups however, was the greater LV wall thickness measurement in black athletes, with 12% of the black athletes exhibiting LVH (defined as a LV wall thickness > 12 mm). In agreement with studies in Italian athletes, only 1.6% of white athletes exceeded a LV wall thickness > 12 mm. None of the athletes exhibited a wall thickness > 16 mm (Figure 1). All athletes with LVH revealed a normal or increased LV cavity size and normal diastolic indices, indicating that the LVH was reflective of intense training rather than a mild form of HCM. Finally, Rawlins et al.[3] observed similar results in a large cohort of nationally ranked female athletes. Just over 3% of the black females demonstrated a maximal LVWT of > 11 mm as opposed to none of the caucasian white athletes.

12-lead ECG

According to a study of American football players,[4] black athletes exhibit a greater prevalence of repolarisation changes compared to caucasian white counterparts. However, it was not until recently that a study by Papadakis et al.[1] attempted to quantify the prevalence, distribution, pattern and most importantly, the significance of repolarisation changes in black athletes. The authors compared ECGs of 904 elite male black athletes with those of 1819 caucasian white athletes, 119 black sedentary individuals and 52 black patients with HCM. Patients with HCM were chosen for comparison since in clinical practice the presence of LVH and co-existent repolarisation anomalies frequently raises the diagnostic dilemma of physiological LVH versus HCM.

T-wave inversions were present in 23% of the authors black athletes compared to only 3.7% of white athletes. The Aspetar experience of T-wave inversion in black athletes competing in Qatar is comparable (Figure 2). T-wave inversions in black athletes were predominantly (12.7%) confined to contiguous anterior leads (V1-V4) and only 4.1% of black athletes T-wave inversions in the lateral leads (Case examples from Aspetar; Figure 3-5). In contrast, both black controls and black HCM patients exhibited lower prevalence of T-wave inversions in leads V1-V4 (4.2% and 3.8%, respectively) with most T-wave inversions in HCM patients (76.9%) involving the lateral leads. During subsequent comprehensive evaluation and follow-up (69.7 ± 29.6 months) of a significant proportion of athletes, 1 black athlete survived a cardiac arrest and 2 athletes (1 black athlete, 1 caucasian white athlete) were diagnosed with HCM. All three athletes exhibited T-wave inversions in the lateral leads. Based on these results the authors concluded that T-wave inversions in leads V1-V4, commonly associated with convex ST-segment elevation in black athletes, are likely to represent an ethnic variant of "athlete's heart". Conversely, T-wave inversions in the lateral leads may represent the initial expression of HCM and merit further cardiovascular evaluation and regular follow-up. Almost 6% of black athletes exhibited T-wave inversion in at least 2 inferior leads. The prevalence of T-wave inversion in the inferior leads was not dissimilar to sedentary black individuals and black patients with HCM but the precise significance of this pattern of repolarisation has not been clarified.

Although ST-segment elevation was more prevalent in black athletes compared to caucasian White athletes (63.2% vs. 26.5%, p<0.001), it was also highly prevalent (65.5%) in sedentary individuals, suggesting an ethnicity-related effect. More detailed inspection of the morphology of the ST-segments revealed that although the concave/

saddle-shaped patterns simulating acute pericarditis were common in both groups, convex ST-segment elevation in leads V1-V4 simulating acute anterior myocardial infarction or the Brugada phenotype, were 6-fold more common in the athletes, indicating a physiological response to training.

Clinical Implications of Ethnically Determined Cardiovascular Adaptation to Exercise

Pre-participation screening is becoming a global phenomenon, involving athletes of diverse ethnic groups. Main sporting bodies including FIFA, UEFA and the International Olympic Committee have endorsed the European recommendations, despite limited understanding of the impact of ethnicity upon the 12-Lead ECG and cardiac structure.[5] Emerging studies suggest that ethnicity is an important determinant of cardiovascular adaptation to exercise, which should always be considered during assessment of an athlete. We offer a practical approach to screening athletes of African/Afro-Caribbean ethnicity based on adaptation of the current criteria.

Screening of African/Afro-Caribbean Athletes

Anterior T-wave inversions confined to leads V1–V4, especially when preceded by convex ST-segment elevation, are likely to represent an ethnically determined, physiological response to exercise. In the absence of cardiac symptoms or a family history of SCD or cardiomyopathy the athlete may continue to compete without the need for further investigation. The authors concede however, that the presence of physiological anterior T-wave inversions in African/Afro-Caribbean athletes may pose a significant challenge in identifying a small number of individuals who exhibit the ARVC phenotype, particularly in cases where T-wave inversions are not preceded by marked ST-segment elevation. In the absence of long-term follow-up data and lack of studies delineating the ECG morphology of African/Afro-Caribbean athletes with cardiomyopathy, individual physicians may opt to perform imaging studies.

The transthoracic echocardiogram of African/Afro-Caribbean athletes should be interpreted with caution, particularly in the context of co-existent repolarisation anomalies. Athletes of African/Afro-Caribbean origin are more likely to exhibit LVH compared to Caucasian counterparts with wall thicknesses of up to 16 mm, which pose significant challenges in differentiating physiological LVH from HCM. This conundrum is of particular significance since exercise-related SCD secondary to HCM in the USA is reportedly higher in athletes of African/Afro-Caribbean descent.[6] A hypertrophic response to exercise may also be evident in athletes as young as 14 years old. With regards to gender differences African/Afro-Caribbean female athletes do not appear to exceed a LV wall thickness of more than 13 mm. Based on available studies, it would be reasonable to infer that an absolute maximal LVWT of 16 mm and an absolute maximal LVWT of 13 mm probably represent the physiological upper limit of LVH in an asymptomatic male and female African/Afro-Caribbean athletes, respectively, outside the context of a family history of HCM.

CONCLUSION

The paradigm of African/Afro-Caribbean athletes illustrates the effect of ethnicity on cardiovascular adaptation to exercise and the importance of adopting ethnicity specific criteria when evaluating elite athletes. Review of available data indicate that although ethnicity specific guidelines should be adopted for the interpretation of the 12-lead ECG and echocardiogram of athletes of African/Afro-Caribbean descent, further research is necessary to delineate the limits of normality in a number of other ethnicities.

Learning Points

- Ethnicity is an important determinant of cardiovascular adaptation to exercise and its impact should be considered when evaluating an athlete.
- Black athlete's exhibit marked repolarisation anomalies and significant LVH; extrapolation of ECG and echocardiographic criteria used to diagnose potentially serious disease processes in Caucasian athletes would result in an unacceptable number of unnecessary investigations.
- In black athletes anterior T-wave inversions confined to leads V1–V4, preceded by convex ST-segment elevation and an absolute maximal LVWT of 16 mm (13 mm for females) are likely to represent an ethnically determined, physiological response to exercise. In the absence of cardiac symptoms or a family history of sudden cardiac death or cardiomyopathy the athlete should be allowed to compete.
- With recent evidence demonstrating pronounced cardiovascular adaptations occur more frequently in black athletes than their Caucasian counterparts, it is worth noting that not all ethnicities have been formally evaluated in this context. For example, we have much to learn about athletes of Arabic, east-Asian, and Maori / Pacific Islander descent.

REFERENCES

1. Papadakis M, Carre F, Kervio G, et al. The prevalence, distribution, and clinical outcomes of electrocardiographic repolarization patterns in male athletes of African/Afro-Caribbean origin. *Eur Heart J*. 2011; **32**(18):2304-13.
2. Basavarajaiah S, Boraita A, Whyte G, et al. Ethnic differences in left ventricular remodeling in highly-trained athletes relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 2008; **51**(23):2256-62.
3. Rawlins J, Carre F, Kervio G, et al. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation*. 2010; **121**(9):1078-85.
4. Magalski A, Maron BJ, Main ML, et al. Relation of race to electrocardiographic patterns in elite American football players. *J Am Coll Cardiol*. 2008; **51**(23):2250-5.
5. Bille K, Figueiras D, Schamasch P, et al. Sudden cardiac death in athletes: the Lausanne Recommendations. *Eur J Cardiovasc Prev Rehabil*. 2006; **13**(6):859-75.
6. Maron BJ, Doerer JJ, Haas TS, et al. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. *Circulation*. 2009; **119**(8):1085-92.

Figure Legends

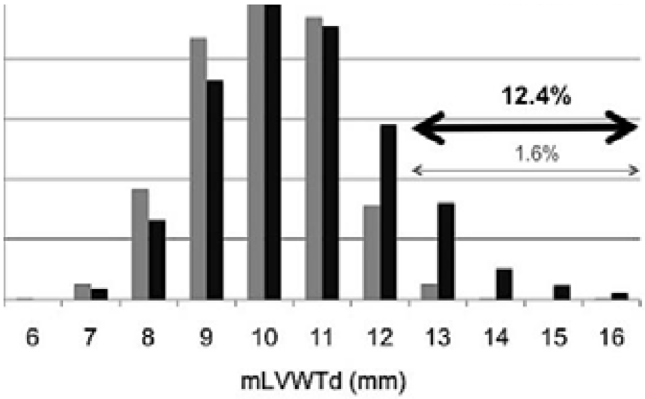


Figure 1: *Histogram demonstrating the distribution of maximal left ventricular wall thickness at end-diastole (mLVWTd) as percentage (%) of the total black athlete (black bars) and Caucasian athlete (grey bars) cohort, respectively.*

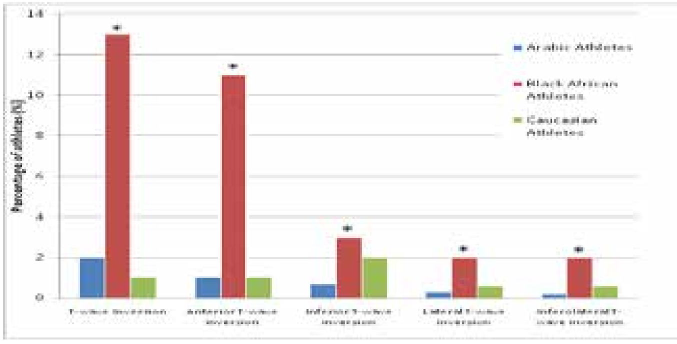


Figure 2: *Data from our facility demonstrating the prevalence and distribution of T-wave inversion amongst Arabic, Black African and Caucasian athletes. * denotes significant difference (p<0.001) between Black African vs. Arabic and Caucasian athletes.*

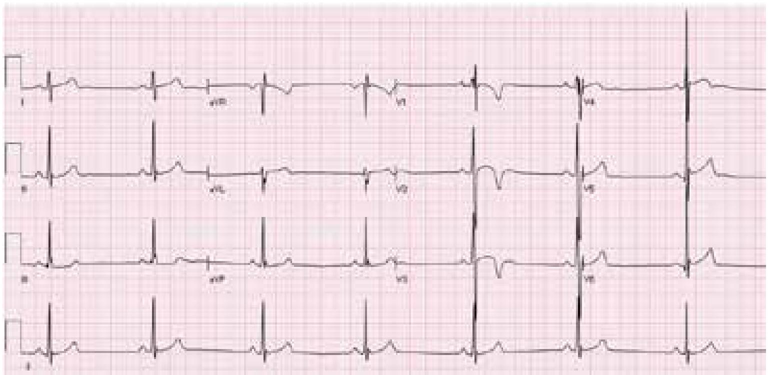


Figure 3: *A 19-year old asymptomatic Black African soccer player with marked bradycardia (42bpm), IRBBB, T-wave inversion in Leads V1-V3, Early Repolarization in Leads V2-V6 and voltage criteria for LVH.*

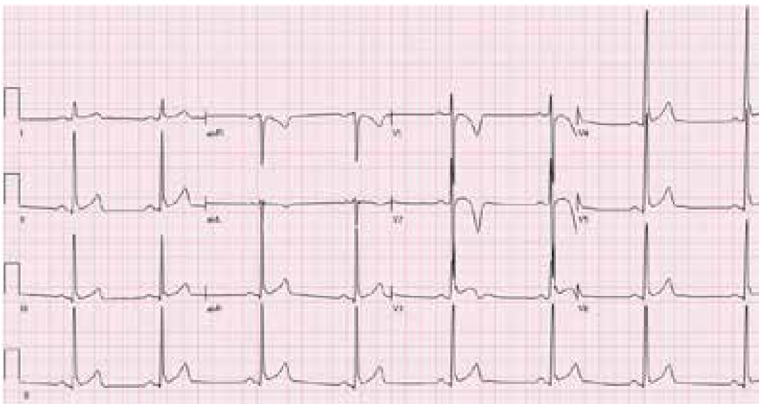


Figure 4: *A 23-year old asymptomatic Black African basketball player demonstrating sinus bradycardia (46bpm), T-wave inversion in V1-V2, Early Repolarization in Leads II, aVF, V3-V6 and voltage criteria for LVH.*

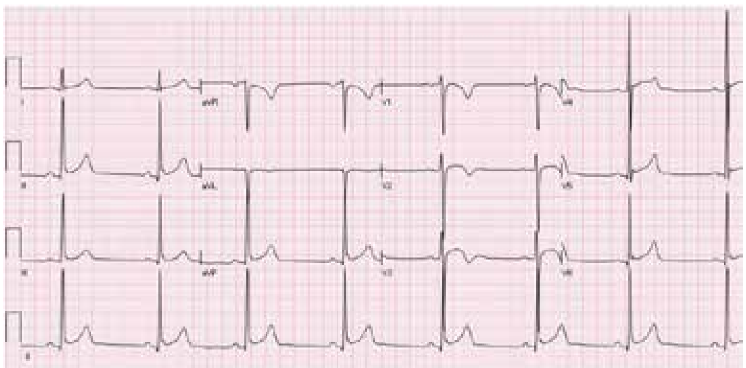


Figure 5: *A 24-year old asymptomatic Black African soccer player demonstrating sinus bradycardia (46bpm), T-wave inversion in Leads V1-V2, Early Repolarization in Leads II, III, aVF, V3-V6 and voltage criteria for LVH. Note: T-wave in V3 is not >-2mm and thus not considered inverted.*

CHRIS WHATMAN

International Society of Biomechanics in Sports (ISBS) Conference

Topic: Sports performance biomechanics, Injury prevention, Sports equipment/technology biomechanics

Location: Melbourne, July 2012

The 30th conference of the International Society of Biomechanics in Sports (ISBS) was held in Melbourne in July this year. The primary purposes of ISBS are:

- To provide a forum for the exchange of ideas for sports biomechanists, researchers, coaches, andteachers.
- To bridge the gap between researchers and practitioners.
- To gather and disseminate information and materials on biomechanics in sports

This was truly an international conference with papers presented from 35 countries. Outside of the official conference programme Melbourne is undoubtedly a world leader for the sports enthusiast. Delegates enjoyed trips to AMMI Park (although the Storm lost!), tours of the MCG and Rod Laver arena and loads of AFL in the local pubs! Although the conference had a major performance focus there is definitely a secondary focus on injury with one keynote on sports injury prevention, an applied sports medicine session and five oral presentation sessions dedicated to injury prevention. I will focus this report on highlights from the injury prevention/sports medicine sessions.

Running Injuries: Forefoot versus rearfoot and barefoot versus shod: A Biomechanist's Perspective

Joseph Hamill (University of Massachusetts Amherst)

For me this was one of the highlights of the conference. Having been in the UK last year to hear presentations promoting the rationale for barefoot running it was interesting to hear an alternate view from well-known biomechanist, Professor Joseph Hamill. Hamill's key point was that the focus should be on the footfall pattern [rearfoot (RF), midfoot (MF) or forefoot (FF)] rather than barefoot versus shod. He highlighted there is little evidence to support the current push by some to alter footfall patterns from RF to FF in an effort to reduce injuries. It has been reported that loading rate and peak vertical impact forces are reduced with FF/barefoot running. Hamill argued that with alternative analysis impact peaks were present in both RF and FF running and presented evidence that natural FF runners had a higher loading rate (and higher tibial stress) than natural RF runners. It was also noted that the link between impact and injury is far from clear. Additionally Hamill argued FF running increased the eccentric load on the Achilles complex, placed additional strain on the plantar fascia and required a stiffer knee (increased lower extremity joint stiffness has been implicated in running injuries). Hamill also argued that RF running is more economical (while acknowledging a lack of studies over durations longer than seven minutes) and referred to a study reporting that runners who started a race as FF runners changed to RF midway through the race. Hamill further proposed that humans have likely always been RF runners (80% of runners run RF) and this was not due to the advent of running shoes. Additionally he cited evidence that footfall pattern was well learned and doesn't change easily. The conclusion was that footfall pattern is probably task and surface dependent (and not a question of barefoot or shod) and that there is minimal data and/or rationale to show that changing from RF to FF running will reduce injury or alter performance.

Movement screening tool identifying athletes at risk of developing patellar tendinopathy

Kerry Mann (Charles Sturt University)

This paper presented a promising looking screening tool for identifying junior basketball players with asymptomatic patellar tendon abnormality (PTA), apparently a risk factor for developing symptomatic tendinopathy. Hip joint flexion/extension range of motion and knee flexion angle at initial contact during a stop-jump landing and quadriceps flexibility were significant predictors of PTA (95% accuracy). Given the recent focus on frontal and transverse plane control of the hip and knee as key risk factors for overuse related knee injury it's interesting to see motion in the sagittal plane highlighted in this study. The use of a stop-jump task rather than the more common drop-jump is also of interest – the authors suggest this task better reflects the combined vertical and horizontal components of many jump tasks in sports and thus may be a more appropriate screening tool. Unfortunately for the clinician the study used three-dimensional motion analysis and thus it is unclear whether or not these hip and knee kinematics could be assessed in the clinic. The task is probably too quick to rate visually but given the sagittal plane nature of the movement standard video might be an alternative for clinicians and/or coaches. This study provides evidence screening for risk of injury can be successful and suggests it may need to include a reasonably sports specific task.

Functional screening test associated with altered trunk and pelvis kinematics and low back injury incidence in adolescent fast bowlers

Helen Crewe (Curtin University of Technology)

This was another interesting screening study investigating the use of the single leg decline squat (SLDS) test as a screening test of pelvic/hip control in adolescent male fast bowlers. Twenty five bowlers were prospectively screened (including a lumbar MRI scan, three-dimensional (3D) motion analysis of the SLDS test and bowling action) and tracked during the subsequent cricket season for low back injury. Additionally all bowlers who did not experience back pain were repeat scanned at the end of the season to check for asymptomatic pars abnormalities. If detected these bowlers were included in the injured group. Results showed injured bowlers had significantly increased knee valgus angle bilaterally during the SLDS test. There was also a moderate association between the pelvis and hip control during the SLDS test and that during the delivery phase of the bowling action on the non-dominant leg. Again the 3D motion analysis methods limit the clinical application of these findings however visual rating and/or 2D video analysis of the SLDS may be a useful screening test in cricket – although studies have shown that 2D measurement of a frontal plane knee angle can't be used to quantify 3D knee valgus. Unfortunately the authors presented limited detail on how the SLDS was performed with no mention of how the depth of the squat was standardised or the reliability of the test. Several authors have suggested the need to control sagittal plane range of hip and/or knee motion during squat tests as this has an effect of the amount of transverse and frontal plane pelvic, hip and knee motion and thus may lead to different test results. Why the authors choose a squat on a decline board rather than the more common single leg squat is also not mentioned. Nevertheless this is one of the few prospective studies linking screening of a functional movement test with increased risk of injury.

Lower limb musculoskeletal stiffness can predict overuse injuries in high level adolescent female athletes

Mark Moresi (Australian Catholic University)

Continuing the screening theme this study investigated whether measures of lower limb musculoskeletal stiffness (MSS) can prospectively predict overuse injury in adolescent female athletes (national level gymnasts, track and field athletes). Conclusions from the study were that stiffness was a predictor of overuse injury over the subsequent 12 months (77% of athlete outcomes correctly predicted). Interestingly the training hours in this study did not predict injury prompting the authors to suggest that in this high level adolescent group the magnitude of loading is more important than the volume. A limitation noted by the authors was that injury risk was obviously based on baseline MSS and this may have changed over the course of the study. Again the challenge for those without access to force plates (and the time to use them) is how to transfer these findings into the clinical assessment/screening.

Kinematic analysis of five ankle inversion ligamentous sprain injury cases in tennis

Daniel Tik-Pui Fong (The Chinese University of Hong Kong)

While case studies don't provide the highest quality of evidence this was a presentation that adds to what we know about the mechanism involved in the lateral ankle sprain. It involved some clever analysis of injuries captured on television including the Michael Stich injury which never looks any better no matter how many times you see it! If you haven't seen it head to Youtube and take a look. The five injury incidents captured in televised tennis competitions were analysed by a model based image-matching motion analysis technique. Interestingly results suggested a large and sudden inversion and internal rotation at the ankle, but not plantarflexion, was the cause of injury. This suggests that supination (widely believed clinically to be the mechanism in many ankle sprains) may not always be the case. These are however similar mechanisms and thus these findings may not change prevention or management strategies.

Biomechanical services and research for athletes and coaches to enhance performance and prevent injury

Nick Brown (Australian Institute of Sport)

This was an invited presentation in which Dr Nick Brown from the Australian Institute of Sport gave his thoughts on how biomechanics research integrated into a high performance centre can improve performance. He emphasised how sport biomechanists assist by identifying and advising on movement technique with an understanding of the mechanical basis of sport specific performances. A key point for me was his view that kinematic analysis is often insufficient and that kinetic analysis is often needed to provide a deeper understanding of potential mechanisms underlying injury and athletic performance. The example of cycling was discussed where kinematic variability is low, but where underlying force application on the bicycle's pedals and joint-specific powers during pedalling can vary. This type of analysis can obviously only be achieved with appropriate equipment and the integration of biomechanical services and research into an elite training environment. We are fortunate to now have this type of integration at the AUT Millennium campus on Auckland's Northshore – home to High Performance Sport NZ.

The relationship between shoulder pain and scapular mobility in teenage baseball players

Chung-Nien Chen (Chung Shan Medical University)

This poster presentation investigated the difference in static scapular position (as assessed by the lateral scapula slide test) between teenage baseball players with and without shoulder pain. Those of you who know more about shoulders than me will be able to judge the value of such a test clinically. Nevertheless the authors reported a significant difference in scapula position with the arm abducted to 90 degrees – scapula of the painful group closer to the spine. The conclusion suggested that rhomboid and trapezium muscle tightness may be a contributing factor and this may result in abnormal scapulo-humeral rhythm and subsequent shoulder pain.

Comparison of kinesio taping and sports taping in functional activities for collegiate basketball players: A pilot study

Chia-Hsin Tsai (Chung Shan Medical University)

Interest in Kinesio tape has spiked again with the extensive coverage of the recent London Olympics. A recent review colleagues and I wrote on the topic for the journal Sports Medicine has promoted numerous comments/enquiries from all over the world. This study by Tsai and colleagues is another suggesting the effects of Kinesio are possibly a little over hyped as I'm sure many of you suspect. The study showed little effect of kinesio tape (applied to the lower leg) on ankle range of motion, plantarflexor muscle strength and endurance, vertical jump, or dynamic balance. The authors did include a control group and a rigid taping group for comparison to the kinesio group but the numbers in each group were small (n=6) and there were possible flaws in the analysis. More quality studies of the effects in sports injuries and performance are needed – the common cry!

The Medical Road to the London Olympics

The London Olympics 2012 must undoubtedly be recorded in Olympic history as one of the most successful to date. The organisation of the event was thorough, the facilities impeccable and the audiences educated and articulate. Couple all these aspects with the outstanding performances from the worlds' most elite athletes and the result was truly top class.

Team Selection

The NZ team's medical road to London began April 2011 when I was appointed as Health Team Leader by the New Zealand Olympic Committee (NZOC) and ratified by High Performance Sport New Zealand (HPSNZ). Once appointed I set about preparing to bring together the health pool team for London. Over the games I have led previously – Melbourne and Delhi Commonwealth Games and Beijing Olympics – I was able to analyze consultation rates and nature and with the assistance of HPSNZ I was able to understand the desire for individual sports to include health professionals in their own team accreditations. The accreditation system is a complex calculation dependent on qualified athlete numbers. As can be appreciated these numbers need to be estimated in the initial phase but can only be confirmed as individual athletes and teams qualify for the forthcoming Olympics. Fortunately, the NZOC have had years of experience in estimating low, medium and high athlete numbers and accordingly the accreditations overall for the team. Consequent consultations between NZOC with National Sporting Organisations (NSOs) determined which numbers were to be allotted to individual sports for officials (non-athletes) were conducted. We were then able to determine numbers of accreditations remained for performance support staff including the health pool team.

With the NZOC I negotiated the number of positions across several health disciplines required to provide safe expert service to our elite athletes. As the games approached, we firmed up the actual numbers and advertising for positions and short-listing commences. For short-listing I consult with the lead discipline practitioners working within HPSNZ for their recommendations. For London the NZOC, HPSNZ and I were committed to having health providers who were experienced in pinnacle events and were embedded in the day to day delivery of services to elite athletes. That said, I am always looking for good 'team fit'. Working every day together in confined settings at a time when emotions are highly charged – sometimes for the worse rather than better – is a unique challenge and we require highly skilled people to fill these positions. The interview panel is composed of the Chef de Mission, Health Team Leader and a member of the Athletes Commission and every appointment for London was outstanding, fully supported by HPSNZ. Given the popularity of the roles, numerous excellent candidates could not be selected. Eventual team members were: Doctors: Graham Paterson (Sports Physician), Chris Milne (Sports Physician) and Chan Dassanayake (General Practitioner/Sports Doctor); Physiotherapists: Jordan Salesa, Helen Littleworth and Louise Johnson; Massage Therapists: Clint Knox and Yvette Latta.

Health appointments within the various sports were made entirely by the sport, and comprised health professionals well known to their athletes and with sports specific knowledge, together with experience at world championships and other international competition.

Logistics

As Health Team Leader I attended several NZOC management meetings, to ensure the integration of Health Services at every level of the Olympic campaign and liaised with the medical members of the London organising committee, disseminating information amongst the wider health team.

By utilising information on medical supply consumption gathered from previous events, I was able to estimate volumes and detail items of medical supplies that we would require. Those items that could safely be sent on sea freight were collated and sent early, with the remaining items accompanying health team members. The logistical work involved in this is not small, and I am very grateful for the help and support given by my wider health team pool.

In 2012, we made a collective decision with HPSNZ to have a 'paperless' Olympic health service and with the assistance of Jordan Salesa – lead physiotherapist – we were able to achieve this. As a result, medical information of athletes participating at the games time is readily available to their health support team following the games.

Prior to leaving for London the full health team attended the Sports Performance Conference. This enabled pool team members to interact with sport support staff, to familiarise themselves with the sports needs, and most importantly to establish connections with people with whom they would be working with in London. Those sports that would require assistance from our health team at games time was detailed and these needs formed the basis of the rostering of health team members once we were in London.

With the entire health team present, we held a trauma/ACLS type session to ensure all health team members were confident in dealing with both basic first aid and resuscitation. Overall, this was an invaluable opportunity to network and understand requirements from the sports specific health professionals.

Jordan and I left NZ five days ahead of the other health poolteam members to begin the job of creating our clinic within the NZ residence space in the athlete's village. I already knew those items that would be supplied by the London Organising Committee (LOCOG) prior to arrival – various pieces of equipment and some consumables. We located the sea freight and began the unpacking and setting up rooms, in readiness for a safe working health environment.

Our full health team arrived a day prior to athletes and then - well, what can I say other than - 'let the games begin'!!

The clinic was open for service from 0770-2200 every day and a doctor slept overnight in the clinic to answer emergency queries. At least one doctor, a physiotherapist and a massage therapist were available all day at the clinic and we also attended those events that required specific professional attendance (and where there was no sport specific medical accreditation) and all this was done through careful rostering and a lot of common sense.

At games time the role of the Health Team Leader is to be the link between overall team management and the health pool team. I support and liaise with the wider health team, facilitate treatment and investigations and liaise with the IOC medical commission, polyclinic doctors and services. The position needs to be across all things medical,each day meeting with the Chef de Mission and other key management personnel to review the day, upcoming few days and key events, as well as contributing to the overall operations management during games time.

At the end of the competition time we have the 'not so wonderful' task of packing up and taking apart our home of five weeks. We take detailed inventory and determine product to return to NZ. We debrief and expand this to a celebration of the whole campaign with athletes and team members who we came to know incredibly well. It is a wonderful celebration of sport but also tinged with some sadness as we know we will be saying goodbye to these incredible people and this incredible event for another four years.

Summary

This was our medical road to London. Along the way there were many fun times, and much camaraderie between all team members, be they athletes or officials. Supporting athletes at a pinnacle event requires all those individuals that athletes come in contact with to be 'aware' in every sense of the word. Aware of all the preparation that has gone into their respective journeys to this event; aware of their highly geared physical and mental health; aware of being responsive and accessible, and most importantly not getting 'in the way'. We are facilitators - we diagnose, intervene judiciously, treat promptly and most importantly strive to keep all the hopes and dreams of athletes and the nation on track.

At times there is a need for honesty, which can be devastating if it means ruling an athlete out of competition. The NZOC believes most definitely in the principle that it is not merely enough to line up at the start of competition, an athlete needs to be able to do justice to themselves, their sport and their country. The athlete deserves to be able to execute their performance to the very best of their known ability.....to do justice to all the hard work they have put in over a very long time. It was with great sadness that I had to announce the withdrawal of Adrian Blincoe from his 5000 km race in London. The decision was made mutually between the athlete and the medical team. As disappointed as we all were for Adrian he made that decision for himself and was in the end reconciled to the decision. His professionalism was inspiring.

The whole games journey is a magical mix of physical challenge, mental sharpness and mind-blowing experiences. The friendships generated are made all the more special by being part of the wider NZ team. Our NZ heritage is celebrated and is so incredibly unique, and magnetic, for both ourselves and other countries around us. They literally swarm our village home when we welcome athletes and celebrate success. It is humbling and exalting all at the same time. I am incredibly proud of all the athletes I have been fortunate to work with, and I am especially proud to be a Kiwi representing my country and profession overseas.

It is simply the best of times ... and I feel incredibly honoured and privileged to have led our health team over the past 7 years. I truly look forward to working with athletes and high performance sport for many years to come.

Lynne Coleman

Leader, NZ Medical Team,

2012 London Olympics

Appendix 1: ***London 2012 consultation statistics***

Doctor	
Chest/Respiratory	10
Dental	3
ENT	25
GI	17
Gynae	5
Misc	8
Musculoskeletal	60
Neurological	6
Occular	3
Psychological	1
Skin	21
Wound	6
Total	165
Massage	
Post Event/Training	179
Pre Event/Training	38
Total	217
Physiothrapy	
Accupuncture	9
Massage	14
Multimodal	158
Strapping	5
Total	186
TOTAL	568

MUSCLE-TENDON INJURIES IN THE LOWER LIMB TARGETING SPECIFIC PATHOLOGIES

Kristian Thorborg

Specialist in Musculoskeletal and Sportsphysiotherapy, PhD, Associate Professor Arthroscopic Centre Amager, Copenhagen University Hospital, Hvidovre, University of Copenhagen, Denmark

Muscle-tendon injuries in the lower limb are common in high-performance sports. Muscle-tendon injuries related to the hip and knee, however, have not been scientifically investigated to the same extent as e.g. injuries to the achilles muscle-tendinous complex. Complex clinical scenarios and multiple diagnoses in the hip- and knee-region make it challenging for researchers and clinicians to include effective diagnostic tools, and relevant functional assessment procedures, for muscle-tendon pathology in these regions.

During the last decade the development of reliable and valid assessment procedures, including clinical entities, patient-reported outcome measures and specific structural assessments, such as strength, pain-provocation and flexibility measures, have improved our understanding of existing functional deficits related to muscle-tendon injuries in the hip and knee. This, now provides clinicians with simple tools to address functional deficits related to specific anatomical structures in the individual athlete, introducing a more targeted approach. Furthermore, improved understanding of relevant functional deficits related to muscle-tendon injuries in the hip and knee, has also initiated the development of new exercise-based scientific interventions targeting specific problems.

In several RCT's on prevention and treatment from our center, specific exercises have been shown to be effective in reducing or recovering troublesome injuries, such as acute and overuse muscle-tendinous problems, in the hip- and knee-region. In addition, several new and simple exercise-based approaches have recently been investigated for their physiological effects, showing great promise. This new research highlights specific exercises that induce relevant contraction improvements, such as eccentric strength-improvements, and optimal neuromuscular activation patterns, making these exercises relevant for future prevention and treatment of specific muscle-tendon problems, in the hip and knee. Furthermore, as adherence to exercise is a vital factor for obtaining the desired physiological and clinical effect, novel methods for monitoring and ensuring adherence in research and clinical practice, is also addressed.

All aspects will be covered in the key-note lecture (Dr. Matt Marshall lecture), presented in an exercise-overview lecture, and introduced in three practical workshops, concerning clinical examination, physical assessment and exercise prescription, including hands-on practice.

References

Hölmich P, Uhrskou P, Ulnits L, Kanstrup I-L, Nielsen MB, Bjerg AM, Krogsgaard K.Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. *The Lancet* 1999; **353**:439-443.

Hölmich P, Hölmich LR, Bjerg AM. Clinical examination of athletes with groin pain: an intraobserver and interobserver reliability study. *Br J Sports Med* 2004; **38**:446-451.

Hölmich P. Long-standing groin pain in sportspeople falls into three primary patterns, a "clinical entity" approach: a prospective study of 207 patients. *Br J Sports Med* 2007; **41**:247-252.

Hölmich P, Larsen K, Krogsgaard K, Gluud C. Exercise program for prevention of groin pain in football players: a cluster-randomized trial. *Scand J Med Sci Sports* 2010; **20**:814-21.

Hölmich P, Nyvold P, Larsen K. Continued Significant Effect of Physical Training as Treatment for Overuse Injury: 8- to 12-Year Outcome of a Randomized Clinical Trial. *Am J Sports Med* 2011

Jacobsen JS, Thorborg K, Søballe K, Ulrich-Vinther M. Eccentric hip abductor weakness in patients with symptomatic external snapping hip. *Scand J Med Sci Sports* 2012 Sep 12. [Epub ahead of print].

Jensen J, Hölmich P, Bandholm T, Zebis MK, Andersen LL, Thorborg K. Eccentric strengthening effect of hip-adductor training with elastic bands in soccer players: a randomised controlled trial. *Br J Sports Med* 2012 Jul 4. [Epub ahead of print].

Petersen J, Thorborg K, Nielsen MB, Hölmich P. Acute hamstring injuries in Danish elite football: A 12-month prospective registration study among 374 players. *Scand J Med Sci Sports* 2010; **20**:588-92.

Petersen J, Thorborg K, Nielsen MB, Budtz-Jørgensen E, Hölmich P. Preventive Effect of Eccentric Training on Acute Hamstring Injuries in Men's Soccer: A Cluster-Randomized Controlled Trial. *Am J Sports Med* 2011; **39**:2296-303.

Thorborg K, Petersen J, Magnusson SP, Hölmich P. Clinical assessment of hip strength using a hand-held dynamometer is reliable. *Scand J Med Sci Sports* 2010; **20**:493-501.

Thorborg K, Bandholm T, Petersen J, Weeke K.M.Ø, Weinold C, Andersen B, Serner A, Magnusson S.P, Hölmich P. Hip-abduction strength training in the clinical setting: Heavy versus moderate load. *Scand J Med Sci Sports* 2010; **20** Suppl 2:70-7.

Thorborg K, Roos EM, Bartels EM, Petersen J, Hölmich P. Validity, Reliability And Responsiveness Of Patient-Reported Outcome Questionnaires When Assessing Hip And Groin Disability: A Systematic Review. *Br J Sports Med* 2010; **44**(16):1186-96.

Thorborg K, Couppé C, Petersen J, Magnusson S.P, Hölmich P. Eccentric hip adduction and hip abduction strength in elite soccer players and matched controls. A cross sectional study. *Br J Sports Med* 2011; **45**:10-3.

Thorborg K, Serner A, Petersen J, Madsen T.M., Magnusson S.P., Hölmich P. Hip adduction and abduction strength profiles in elite soccer players: Implications for clinical evaluation of hip adductor muscle recovery following injury. *Am J Sports Med* 2011; **39**:121-6.

Thorborg K, Hölmich P, Christensen R, Petersen J, Roos EM. The Copenhagen Hip and Groin OutcomeScore (HAGOS): development and validation according to the COSMIN checklist. *Br J Sports Med* 2011; **45**:478-91.

Thorborg K, Roos EM, Christensen R, Petersen J, Hölmich P. The iHOT-33: How Valid Is It? *Arthroscopy* 2012; **28**:1194-5.

Thorborg K, Bandholm T, Hölmich P. Hip- and knee-strength assessments using a hand-held dynamometer with external belt-fixation are inter-tester reliable. *Knee Surg Sports Traumatol Arthrosc*. 2012 Jul 7. [Epub ahead of print]

Thorborg K. Why hamstring eccentrics are hamstring essentials. *Br J Sports Med* 2012; **46**:463-5.

IMPACT OF AGE, SEX AND ETHNICITY UPON THE CARDIOVASCULAR ADAPTATIONS ASSOCIATED WITH EXERCISE

Mathew Wilson
Cardiovascular Physiologist
ASPETAR, Qatar Orthopaedic and Sports Medicine Hospital

Regular exercise reduces the risk of cardiovascular disease and subsequent sudden cardiac death (SCD). However, a small, but notable proportion of athletes die suddenly due to a number of inherited and congenital disorders of the heart, which may predispose to malignant ventricular arrhythmias. Such tragedies are highly publicised, particularly when high-profile athletes are involved.

Due to the steady trickle of SCD's in young athletes, several major sport governing bodies, including the International Olympic Committee (IOC) and Fédération Internationale de Football Association (FIFA) have 'recommended' the implementation of systematic cardiac screening programmes – a trend increasingly being adopted by national and international sport governing bodies worldwide. Whilst the prevalence of inherited or congenital conditions are rare, modern cardiovascular diagnostic technology can identify potential SCD conditions with a high degree of accuracy and reliability, lending support to the establishment of preparticipation screening programmes to ascertain pathology.

Yet there is mounting evidence that an athlete's age, sex and ethnicity have an important determination on the objective manifestations of cardiovascular adaptations to exercise. Accurate interpretation of the athlete's ECG and echocardiogram is crucial, as inappropriate interpretations would result in an unacceptable number of unnecessary investigations and worryingly, potential false disqualification from competitive sport. This presentation attempts to highlight known age, sex and ethnically determined differences in cardiovascular adaptation to exercise and provides a practical guide for the interpretation of baseline investigations in athletes of diverse ethnic backgrounds.

INJURY SURVEILLANCE AND INJURY PREVENTION IN THE AUSTRALIAN FOOTBALL LEAGUE

Hugh Seward
Executive Officer, AFL Medical Officers Association

The AFL Medical Officers Association have monitored AFL player injuries consistently for 20 years and the results have provided a useful tool to facilitate injury prevention and improve management in the context of a changing and evolving football code.

Patterns of specific injuries over this 20 year period demonstrate the variety of influences on injury patterns.

- Increasing professionalism leading to improved fitness and conditioning has contributed to increased work rate and impacted on soft tissue injuries.
- Game characteristics, such as increased speed of players, has led to increased hamstring injuries, but has been counteracted by coaching strategies through interchange.
- Hamstring recurrence rates have reduced through better medical management.
- Advances in medical knowledge and practices have led to more games missed for hip injuries through early intervention. This may, in the future, show a decline in the lasting impact and recurrence of hip and groin injuries.
- Targeted rule changes have been shown to be successful in reducing PCL Injuries.
- The recent focus on concussion management has altered the pattern of games missed for this condition.

Successful injury prevention requires a comprehensive understanding and monitoring of injuries. The development of evidence based programs must be combined with an understanding of implementation strategies to translate the theory into a long term behavioral change. The presentation will also discuss some of these concepts.

MOTOR PATTERN STABILITY DURING THE TRIATHLON CYCLE RUN TRANSITION IMPLICATIONS FOR PERFORMANCE AND INJURY

Jason Bonacci
Physiotherapist and Biomechanist
Geelong, Australia

Triathlon is a multi-discipline sport that involves sequential swimming, cycling and running. Success in the sport is determined by the ability to excel at and link the three separate disciplines. In preparation for competition triathletes often practice two or more of these disciplines in the one training session or practice each discipline separately with only short recovery periods. There is evidence to suggest that this type of multi-discipline training interferes with neuromuscular adaptation in these athletes. Specifically, patterns of leg muscle activation in trained triathletes are less developed than cyclists matched for training loads and are more similar to that of novice cyclists. A proportion of triathletes also show interference in neuromuscular execution when performing running after cycling. This interference is present as soon as running commences and persists for the duration of the run leg. Those that demonstrate this interference have a 2.5 times greater likelihood of having a history of lower extremity overuse injury. A recent pilot study has shown that plyometric training can be used as an effective intervention to correct this aberrant neuromuscular activation, without any risk of a decrement in run performance. Changes in running kinematics after cycling are more closely related to alterations in running economy, with changes in ankle kinematics being the most predominant factor. In contrast to the findings of moderately trained triathletes, elite international triathletes demonstrate no interference in kinematics and muscle recruitment when running after cycling, and this may be a factor that contributes to the success of these athletes.

BREATHING PATTERN DISORDERS AND THE ATHLETE

Tania Clifton-Smith
MNZSP, DipPHTY, DipMT

'If breathing is not normalised no other movement pattern can be'[5]

Little attention has been paid to the breathing pattern of the athlete that is until recently. Historically this area of research has been dominated by sports physiologists who have focused on ventilation and the delivery of oxygen. Research is now beyond the capacity of ventilation and starting to look at the muscles of respiration and even breathing patterns. [2,3,4] Prolonged alterations of function as seen in chronic breathing pattern disorders inevitably leads to structural changes. These commonly involve accessory breathing muscles as well as cervical, thoracic and lumbar muscles and articulations, neural tensions and fasciae. Structural adaptations can prevent normal breathing function, and abnormal breathing function ensures continued structural adaptation. These biomechanical malfunctions trigger physiological and potentially psychological imbalances as well. [1]

This presentation aims to highlight the importance of incorporating breathing pattern awareness into assessment and treatment programmes for the athlete. A case history of a National Swimmer presenting with bilateral rotator cuff tendinitis will demonstrate how the awareness of breathing patterns and the use of inspiratory muscle training assisted in changing motor patterns, improved performance and reducing the tendonitis.

Breathing and the muscles of respiration can be a major factor in the ultimate training and outcomes of anyone participating in sport. Athletes and in particular the elite athlete needs to be assessed and treated with all three categories in mind: biomechanics, physiology and psychology. Biomechanics needs to consider postural and respiratory demands.

References

1 Bradley D. Physiotherapy breathing rehabilitation strategies. In: L Chaitow, D Bradley & C Gilbert (Eds.). Multidisciplinary Approaches to Breathing Pattern Disorders. Edinburgh: Churchill Livingstone, 2002.p.173-195

2 Lewit K. Relation of faulty respiration to posture with clinical implication. *Journal of the American Osteopathic Association* 1980; **79**:525–529

3 McConnell A, Sharpe G. The effect of inspiratory muscle training upon maximum lactate steady-state and blood lactate concentration. *Eur J Appl Physiol* 2005, **94**, 277–284

4 Volianitis S, McConnell AK, Koutedakis Y, McNaughton L, Backx K & Jones DA . Inspiratory muscle training improves rowing performance. *Med Sci Sports Exerc* 2001. **33**, 803–809

5 Vickery R. The effect of breathing pattern retraining on performance in competitive cyclists, 2007 http://repositoryaut.lconz.ac.nz/handle/10292/83

VISUAL RATING OF DYNAMIC PELVIS AND KNEE ALIGNMENT IN YOUNG ATHLETES: CAN YOU BELIEVE WHAT YOU SEE?

C Whatman,[1,2] P A Hume,[1] W Hing[2,3]
[1]*Sport Performance Research Institute New Zealand, AUT University, New Zealand.*
[2]*Health and Rehabilitation Research Institute, AUT University, New Zealand.*
[3]*Faculty of Health Sciences and Medicine, Bond University, Australia*

Background: Dynamic alignment of the lower extremity is considered a key risk factor for several overuse injuries.[1] Common measures of dynamic alignment include frontal plane pelvis position and the medio-lateral knee position relative to the foot during functional tasks involving hip and knee flexion. Techniques for assessing the dynamic position of the knee relative to the foot have been specifically noted as important in screening young athletes.[2]

Purpose: To investigate the ability of physiotherapists to visually rate dynamic pelvis and knee position in young athletes during three lower extremity functional tests [Small Knee Bend (SKB), Single Leg SKB and Drop Jump].

Methods: The pelvis and knee alignment of 23 young athletes, during three lower extremity functional tests, was visually rated by 66 physiotherapists. Peak two-dimensional (2D) and three-dimensional (3D) lower extremity kinematics were also quantified. Physiotherapist ratings were compared to the consensus visual ratings of an expert panel of three physiotherapists. Consensus ratings were also compared to the peak 2D and 3D kinematics. Sensitivity, specificity, the diagnostic odds ratio (DOR) and differences in kinematics between groups based on the expert visual ratings were calculated to assess the validity of ratings. Physiotherapist experience and functional test velocity were further assessed as factors affecting rating accuracy.

Results: Sensitivity (≥80%) and specificity (≥50%) were acceptable for all tests except the Drop Jump. Experience (DOR 1.6 to 2.8 times better) and slower movement (4.9 times better) were factors in better rating accuracy. Peak 2D and 3D kinematics were different between groups rated as having good versus poor alignment by the experts (likely to almost certainly), except for knee abduction angle.

Conclusions: Visual rating by physiotherapists is a valid tool for identifying young athletes with poor frontal plane dynamic pelvis and knee alignment. Ratings are better with slower movements and possibly with increased clinical experience.

References

1 Boling, M. C., Padua, D. A., Marshall, S. W., Guskiewicz, K., Pyne, S., & Beutler, A. A Prospective Investigation of Biomechanical Risk Factors for Patellofemoral Pain Syndrome. *The American Journal of Sports Medicine*, 2009; **37**(11), 2108-2116.

2 Örtqvist, M., Moström, E., Roos, E., Lundell, P., Janarv, P.-M., Werner, S., & Broström, E. (2011). Reliability and reference values of two clinical measurements of dynamic and static knee position in healthy children. *Knee Surgery, Sports Traumatology, Arthroscopy,* 2011; 1-7.

THE EFFECT OF INSTRUCTION ON LOWER LIMB KINETICS AND KINEMATICS DURING LANDING IN NOVICE NETBALL PLAYERS

C A Cross,[1] M Boocock[2] and D Reid[2]
[1]*Master of Health Science Candidate AUT University*
[2]*Health and Rehabilitation Research Institute AUT University*

Introduction: Netball is the largest female participation sport in New Zealand. Lower limb injuries, in particular to the ankle and knee, are common during landing in netball. Instruction on landing technique is often implemented in lower limb injury prevention programmes; however, these programmes commonly involve multiple neuromuscular training techniques such as proprioception, strength, balance, speed and plyometric training. The effect of instruction alone has not been widely researched. Although guidelines for landing are in use within New Zealand netball, these guidelines have not been specifically evaluated. The aim of this study was to determine the effects of instruction (based on Netball New Zealand guidelines) on ankle and knee joint kinematics and kinetics in a group of novice female netball players.

Methods: Thirty-nine young female netball players were allocated by school to an instruction or control group. All participants attended the Health and Rehabilitation Research Institute's movement laboratory, AUT. Participants performed both double and one-to-two foot landings after running forwards to catch a netball. After baseline testing, the instruction group received visual, verbal and feedback instruction on their landing technique for 20 minutes while the control group performed repeated landings without any instruction during the same time period. Ankle and knee kinematics and kinetics were assessed using three dimensional (3D) motion analysis (Qualysis Medical, Sweden) and 3D force platforms both prior to and following instruction or practice. Data was analysed with a mixed model ANOVA using SPSS (SPSS Inc. Chicago) version 19 statistical software.

Results: Eighteen participants in the instruction group (mean age 11.9 years +/- 0.55/ height 1.57m +/- 0.06/ mass 49.1kg +/- 7.31) and 18 in the control group (mean age 11.9 years +/- 0.59/height 1.54m +/- 0.1/ mass 45.1kg +/- 10.79) successfully completed the session. The instruction group demonstrated significantly greater ankle dorsiflexion and knee flexion following instruction ($p < 0.05$). They also significantly reduced vertical joint reaction forces at the ankle and knee, as well as anterior joint reaction forces at the ankle ($p < 0.05$). The control group did not significantly alter their landing mechanics following landing practice.

Discussion: Following a 20 minute period of landing instruction based on Netball New Zealand guidelines, a group of novice netball players were able to alter their landing biomechanics in a way that has potential implications for injury prevention in the lower limb. Reduced knee flexion during landing has been shown to correlate with increased injury risk to the anterior cruciate ligament (ACL) and this study shows that instruction alone can increase knee joint flexion. Further support for the benefits of instruction using Netball New Zealand landing guidelines is evident by the significant reduction in joint reaction forces at the ankle and knee. This study did not assess the long term benefits of instruction on landing in netball; however it provides the basis for further research.

SOUTHERN MEN AND VITAMIN D: AN RCT ON SUPPLEMENTATION, VITAMIN D STATUS, BONE DENSITY & PERFORMANCE

K A Fairbairn,[1] T L Perry,[1] IJM Ceelen,[2] and CMS Skeaff,[1]
[1]*Department of Human Nutrition, University of Otago;*
[2]*Institute of Sport and Exercise Studies, HAN University of Applied Sciences.*

Introduction: Observational evidence indicates that severe vitamin D deficiency is associated with poor bone health, decreased muscle strength and physical performance; however, little is known about the effects of suboptimal vitamin D status on these outcomes, particularly in athletes. A high prevalence of suboptimal vitamin D status has been observed in athletes overseas. Thirty-two percent of New Zealanders have suboptimal vitamin D status1, thus it is possible that New Zealand athletes have suboptimal vitamin D status, perhaps compromising their bone health, muscle strength, and performance.

Research Questions:

1) What is the prevalence of suboptimal vitamin D status (defined by serum 25-hydroxyvitamin D (25(OH)D) concentrations below 50 nmol/L) amongst elite rugby union players living in southern New Zealand?
2) What is the effect of vitamin D supplementation on serum 25(OH) D concentrations, bone mineral density, muscle strength and physical performance in these athletes?

Methods: Fifty-eight elite rugby union players were randomised to receive either 3,570 IU/day vitamin D or placebo for 11 to 12 weeks. Serum 25(OH)D concentrations were measured in serum samples collected at baseline, 5 to 6 weeks and 11 to 12 weeks. Physical performance was measured at the same time points using standardised New Zealand Rugby Union rugby performance tests. Bone mineral density (BMD) and body composition were measured at baseline and weeks 11 to 12 using dual X-ray Absorptiometry.

Results: Participants had a mean ± SD age of 21 ± 3 years, were 185 ± 7 cm tall, weighed 96.6 ± 11 kg, and with BMI values of 28.2 ± 2.1 kg/m2. Percent body fat was (mean ± SD) 14.6 ± 4.6 %, BMD was 1.47 ± 0.08 g/cm2 and their mean z-score for BMD was 2.79 ± 1.0 standard deviations above their age-adjusted mean.

Table 1: Serum 25(OH)D concentrations (nmol/L) by treatment group (n=58)

Time-point	Vitamin D group	Placebo group
Baseline	100.8 ± 29.0	92.6 ± 18.7
Weeks 5 to 6	103.5 ± 21.4	91.2 ± 20.2
Weeks 11 to 12	104.5 ± 25.7	88.3 ± 24.6

There were no significant differences in any of the baseline characteristics by treatment group. No participants had suboptimal vitamin D status at baseline. At Weeks 11-12, four participants had suboptimal vitamin D status. Vitamin D supplementation had no significant effect on changes in body composition or performance tests over the study period.

Conclusions: In our study population of elite rugby union players residing in southern New Zealand, 93% have optimal vitamin D status, including through the winter months. There is also no evidence of Vitamin D supplementation affecting body composition or rugby union performance over 12 weeks in this vitamin D replete population.

References

1 Ministry of Health. 2012. Vitamin D Status of New Zealand Adults: Findings from the 2008/2009 New Zealand Adult Nutrition Survey. Wellington: Ministry of Health.

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 Reports
b Case
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.

Copyright

Sports Medicine New Zealand Inc,
PO Box 6398,
Dunedin, New Zealand,
tel +64-3-477-7887, fax +64-3-477-7882,
email smnznat@xtra.co.nz.

Published by: Sports Medicine New Zealand, Po Box 6398, Dunedin, New Zealand