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Photo: Doug King

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Concussion in amateur rugby league players in New Zealand: A review of player concussion history

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Conor Gissane completed his PhD in 2003 at Brunel University in the area of epidemiological investigation into rugby league injury. During his time in academia he has worked at Brunel University, along with several visiting positions at Nottingham University, University College Dublin and the University of East London. His main are of research interest is injury in sport. This initially began in rugby league, but has since been extended to several other sports. Conor is also interested in statistics and statistics education and research design. Conor has acted for a reviewer for ten peer reviewed journals and is a member of two editorial boards.

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Reliability of 2D lower extremity alignment measures in elite adolescent ballet dancers

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Erin Bowerman is a strength, conditioning and technique coach for Dance Pilates in Auckland. She runs a High Performance Program for elite adolescent ballet dancers and provides workshops for both teachers and students on both plyometric and pointe training programs currently used and endorsed by the New Zealand School of Dance. With a strong background in ballet she graduated as Top Scholar from the Bachelor of Dance at AUT University before completing her Master of Sport and Exercise (1st Class) in 2013 with a focus on risk factors for overuse injury in elite adolescent ballet dancers. Erin is also currently a 'safe dance practice' lecturer at Unitec.

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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 limb overuse injuries, specifically looking at the screening of 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.

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Exercise-induced bronchoconstriction prevalence in elite Australian Rules footballers

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Karen Holzer is a sports physician based in Melbourne. She has a PhD and post Doctorate in Exercise-induced Asthma and breathing difficulties. She is recognised world wide as a leading researcher and clinician in this area. She played a major role in the preparation and management of the Australian Olympic team for the 2008 Beijing Olympic Games, at which the air pollution and consequent airways complications were the major concern.

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Athletes with limb deficiency

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Holt-Oram syndrome: An incidental diagnosis at athlete screening

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Matt Robinson is a sports medicine registrar currently working at Unisports Sports Medicine, Auckland. He gained a BSc(Hons) in Human Biology from Loughborough University before completing his medical degree at Cambridge University, where he gained a 'blue' for cross-country running. He has previously worked as a registrar in Christchurch and has completed a Fellowship at Aspetar Orthopaedic and Sports Medicine Hospital, Qatar. Matt has worked as the team doctor for both the Canterbury and Counties Manukau rugby teams and was Medical Director for the 2013 BMX World Championships in Auckland. Having represented Wales as a runner, duathlete and triathlete and Great Britain as an Age-Group triathlete, Matt has an interest in running and cycling injuries and biomechanics, as well as exercise for health.

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Movement screening protocols: Rationale versus evidence

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Paul Gamble is an author and strength and conditioning specialist. Paul is originally from the UK where he worked in high performance sports for a decade, beginning his career in professional rugby union with London Irish and latterly serving as National Strength and Conditioning Lead with Scottish Squash where he also oversaw sports medicine and sports science provision for players in the national programme. Since arriving in New Zealand in late 2011 Paul has continued to combine writing and training athletes: in 2012 he founded the Informed Practitioner in Sport website to develop and deliver online continuing education courses for practitioners, and is presently engaged with training track and field athletes.

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Cardiovascular positional anomalies and the competitive athlete

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Maria-Carmen Adamuz is a cardiologist currently working in ASPETAR, Qatar. Originating from Spain, she studied medicine at University of Cordoba before undertaking a PhD and a MBA of Health Institutions. During her career she occupied different managerial and clinical positions becoming the Director of the Cardiology Unit at the Complejo Hospitalario de Jaen (2010) and Director of the Centro Andaluz de Medicina del Deporte (2004). Working both with both elite athletes and diseased individuals for over a decade, Maria-Carmen has considerable experience in dealing with the common inherited and acquired cardiac pathologies that affect young athletes.

Nathan Riding

Nathan Riding is currently undertaking his PhD at ASPETAR in conjunction with Liverpool John Moores University, UK. The focus of his PhD is investigating the impact of ethnicity upon the Arabic athlete's heart. As part of his role within the cardiac screening team, he has tested over 2500 young athletes, publishing 3 papers in internationally recognised peer reviewed journals.

Mathew Wilson

Mathew Wilson is Head of Athlete Health and Performance Research and Lead Performance Physiologist at ASPETAR. Previously, Mathew served as the Cardiac Screening Manager for the CRY Centre for Sports Cardiology within the Olympic Medical Institute, UK. His research focus is on the athlete's heart and mechanisms of sudden cardiac death in young and veteran athletes. Mathew has screened over 20,000 athletes, which has led to the production of over 70 peer-reviewed scientific publications.

Redefining normality in sport and medicine: The power of the hormone

Normality, is of course an arbitrary state. Much as history is determined by the conquerors, it seems our normality is determined by those setting the rules, with one's normality in any given life facet reflected in how far one deviates from a particular population average. However, normality - it is clear - varies between cultures, across genders and over time. A quick reference to the NZ census data provides an easy example of changing norms, with the average life expectancy for a New Zealand woman born in 2010 being 83 years. Fifty years ago, it would have been decidedly abnormal, if not somewhat awesome, to live to a spritely and wise 83 years. While this change may be attributed to improved lifestyles, medicines and healthcare, it seems that there are other norms associated with ageing that are also changing, and the reasons for this may not be so clear. One of those areas under the microscope is hormone profiles and their role in "normal" ageing. There is on-going debate as to whether the observed decline in hormone levels are "normal" or whether they reflect a preventable "disease" state. Aligned with the latter view on ageing being a preventable disease, rather than a normal process, there has allegedly been a rapid increase in the number of practitioners willingly prescribing hormonal "supplementation" to "reverse" the ageing process. What, you may reasonably ask, has a discussion of ageing, the commercialisation of ageing and normality got to do with sports medicine?

Earlier this year, a report appeared in the Wall St Journal regarding an endocrinologist who Nike coach Alberto Salazar claimed to be the "best sports endocrinologist in the world".[1] Ignoring my tendency towards cynically evaluating the scientific validity of this particular statement (such as how many sports endocrinologists has he actually met? is this actually a specialty? and what criteria would be used for such a statement? - all of which may be a bit harsh, after all it is just his opinion), the article generated interesting questions (particularly if the amount of "web chat" that it generated is any guide) – many of which in some way or other come back to what "we" (or perhaps "they", that is those in charge) consider normal. In the article, it is alleged that hypothyroidism is an under-recognised condition in Olympic level athletes (and that 15 athletes Dr Brown has treated have gone on to win Olympic Gold medals) and that the diagnosis of hypothyroidism is missed using "normal" values. Unsurprisingly, hypothyroidism has long been a differential for the tired athlete,[2] and there are multiple high profile examples of athletes in the 80's and 90's with diagnoses of hypothyroidism (although…this was an era rife in doping…), so there is nothing particularly novel in this condition being diagnosed in elite athletes. The controversial element of this report is that Dr. Brown is prepared to treat hypothyroidism, in the presence of what most practitioners would consider to be "normal" hormone profile results – it is because of this "unique" ability that Dr. Brown, is considered to be … the best. Fascinating.

Of course, the loaded implication of this article is that the athletes involved are using thyroid hormone - not as an appropriate replacement for a failing thyroid gland - but as a pharmaceutical tool to enhance their performance. Perhaps not surprisingly, it seems that none of the athletes described in this article claim any performance benefit from being treated with thyroid hormones (despite presumably being able to resume training at levels not previously tolerated). By contrast, the infamous dope(r) Victor Conte, ringleader of the BALCO fiasco allegedly claimed that thyroid hormone "makes all the other drugs work more effectively".[3(p138)]

So, are the athletes normal, hypothyroid or "normal" for athletes? If they are genuinely hypothyroid then what is causing it, and why do we seem to be treating the result and not the cause?

Even a quick perusal of the medical literature will lead one to the conclusion that the medical fraternity appears to be engaged in a debate as to what constitutes a "normal" thyroid state. For example, hypothyroidism is typically determined by symptoms (of which fatigue may be one of many) and an "elevated" serum level of thyroid stimulating hormone (TSH). A normal TSH is typically below the range of 4-5 mIU/L (the lab here in Auckland uses a range of 0.3 – 4.0 mIU/L as "normal"), but some argue that a TSH as low as 2-3 may actually represent an elevated level in some fatigued endurance athletes. Dr. Brown, it would seem, sits firmly in the camp that suggests that the traditional levels of TSH required for a diagnosis of hypothyroidism are outdated and should be "relaxed". It seems that determining what is normal is not actually all that easy and according to the Thyroid Association of NZ Inc. (a patient support group) website, the medical establishment is not keeping up with changing evidence for diagnosis and management of thyroid disorders.[4] Knockers of the traditional medical system (and there are many if you venture on-line) would argue that medicine is too conservative by far, and that practitioners such as Dr. Brown are actually leading the way. However, evidence based officianos (who tend to be the ones making the rules) would point to the lack of scientific evidence to support both this diagnostic and treatment rationale in the normal population. Elite athletes however, are not the "normal" population.

Can we reasonably apply arbitrary population "norms" to elite athletes - individuals who in almost every other physiological, musculoskeletal and psychological index sit well outside the "normal" range? Why should we be content to assume that the local laboratory guidelines apply to an athlete who sits in the .01 percentile of normality in every other way? Despite elite sport having vast amounts of investment, there remains remarkably little data on "normal" ranges for athletes in any area and hence we don't know if the normal ranges for TSH in elite athletes are different to us mere mortals or "norms". Furthermore, while there's no hard evidence to show an impact on the thyroid axis, it is well established that the intense exercise, stress and diet of elite athletes affects other hormone systems in the body.

Hence, on the question of whether this is

actually: 1) a disease, 2) a normal (as opposed to abnormal) human response to heavy training, 3) a treatment looking for a disease or 4) a pharmaceutical company initiative. I have no answer, although in my typically equivocating manner, I would contend that it is not something that I would necessarily close the book on. We should appropriately challenge dogma, and not be resistant to development, but equally, we should not avoid the challenges posed by finding evidence.

Significantly, I have found no evidence, other than the insinuations in this article, that the athletes who were treated with thyroxine (presumably for fatigue) got "better" (whatever that may mean). For arguments sake however, lets assume they do. What then was the cause of the hypothyroid state in these athletes, or can thyroxine just be used arbitrarily for fatigue in athletes in order to enhance recovery (a potential challenge for the WADA list)? Worldwide, the most common cause of hypothyroidism is iodine deficiency. Iodising salt and other foods in western diets had, until recently virtually eliminated this deficiency. However, changing diets and food fashions have resulted in rates of iodine deficiency and subsequently hypothyroidism climbing, in NZ as well as other western countries. Presumably however, iodine deficiency is not the cause of hypothyroidism in athletes being supplemented with thyroxine, as treating the deficiency would negate the need for further and long term treatment with hormones. Is it then an auto-immune disease such as Hashimoto's? This would unlikely generate much diagnostic debate, and the diagnosis should not challenge an endocrinologist. Which brings us back to implication that it is the exercise tolerance or training load that causes hypothyroidism / subclinical hypothyroidism / high normal TSH / fatigue / or whatever term you prefer to apply to it. Current evidence (although somewhat limited) does not seem to support heavy exercise as having a negative or sustained impact on thyroid levels. Yet exercise as the culprit is heavily implied in the Wall St Journal article. Extrapolating this principle, if two individuals perform the same training with Athlete One recovering, whilst Athlete Two does not, then Athlete One will subsequently go on to train better and harder the next day. There is obviously some reason for this differentiation – be it genetic or environmental or a combination of factors. Fortunately, Athlete Two is subsequently found to be hypothyroid (assuming an auto-immune disease and iodine deficiency are excluded and that he/she is actually euthyroid by most "normal" guidelines) by the best endocrine doctor in the world. At this juncture, it would seem that there are two possible courses of action – firstly to reduce, modify or amend the training volume or intensity to a level that is tolerated, thereby accepting the concept that this is our parent-given performance level. Or secondly, supplement with something, anything, in order to facilitate on-going training at a level not tolerated by our body. The latter approach, being applied by some of our colleagues, I believe, is the illogical and tragic outcome of popularist medicine and elite, professional sport colliding. Brutally, sport - as with life it seems - is not fair, but now it seems that we can medicate our way out of anything and level the unfair playing field.

Is it reasonable to treat a condition with medication, when reducing training intensity may have the same effect? If training is believed to be the cause of the "hypothyroid" state, then surely the first line of treatment must be to reduce training and respect his or her physiological limit or maximum response to a given training technique? Surely, this is a treatable condition without medication, or are we just treating elite athletes differently. And if we are, is this ethical? Even though thyroxine may not appear on the WADA prohibited list, it would seem inappropriate and irresponsible to medicate someone when we can treat by withdrawing (or treating) the cause – in this case, excessive training load. Unfortunately, the problem with this argument is that we probably do this all the time. For example, athletes may present with fatigue resulting from subtle iron deficiency, and I will not hesitate to attempt to correct the ferritin levels, while often allowing an athlete to maintain the training load. Is this any different to treating fatigue due to low thyroid function with thyroid hormones? Is it the term hormone that I am scared off by, and should I be more relaxed like our colleague Dr. Brown?

No. I would hope that our norms have not changed so much that it is OK to medicate someone to stay what is currently considered "normal". As with the trend towards medicating for the elixir of youth - pushed by healthy ageing centres (for a great read, see John Hoberman's "Testosterone Dreams") - I am not moved by the push for medicating to enable elite participation. Equally, normality, normal use of medication, and appropriate treatment shouldn't be determined by whether or not something is on the WADA prohibited list.

Normality it seems is not fair, nor is it easy to define. Our perception of what is normal is constantly changing and is influenced by many social and cultural factors, not just medical literature. However, for my money, sport has moved too far from its healthy origins if we are now considering it normal to treat tired athletes with hormone supplements, just in order to keep them "normal". As we enter an era where we can often (and more often than not are expected to) both put a diagnostic label and treat an increasing number of conditions, just "because we can", should not necessarily over-rule "should we" when pontificating over treatment options. Stories such as this should not be dismissed, but should motivate us to question why, how and with what we look after our athletes, and we should consider them an opportunity to reflect on what truly is best practice.

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This column covers the first six months of 2013 with nine issues of BJSM published in that timespan.

JANUARY

The January issue included an interesting article entitled 'Team physician challenges in 2013: dealing with media and travelling across state borders'. Those of us who provide care for our national teams and Super Rugby franchises will be familiar with the issues raised here. It comments on the challenges to confidentiality of athletes' medical information in this era of social media and challenges with providing care overseas such as malpractice coverage, access to care at outside institutions and carrying medicines across international borders. They note that athletes themselves are frequently naïve regarding controlling release of information, but it is up to the sporting league, team, physician and medical staff to protect and educate athletes on their rights.

Later in the same issue there was an interesting article entitled 'The placebo effect: powerful, powerless or redundant'. Placebos have been in use for at least the last three centuries and the authors note that the last half century has seen a shift towards acceptance of the evidence-based medicine paradigm. The authors point out that any placebo must be physically inert. However, there still may be a psychological benefit that is obtained by its use. We all know athletes are suggestible people, and the huge number of athletes taking supplements, many of which would at best have only a placebo effect, means that we need to be aware of these issues. The authors advocate for better control interventions to produce more robust clinical research.

Elizabeth Joy, Steven Blair and co-authors have written a compelling article entitled 'Physical activity counselling in sports medicine: a call to action'. They outline the benefits of physical activity to health in terms of its impact on non-communicable diseases and why sports medicine physicians should lead this movement. They embrace George Sheehan's philosophy that 'every patient is an athlete and every physician is a coach'. They advocate three actions:

1 Giving physicians the methods and support they need to counsel patients; in particular, the use of physical activity or exercise as an additional vital sign

2 Developing programmes to educate physicians; this should start in undergraduate training in medical school and continue through Fellowship training

3 Medical students and physicians need to be active themselves; i.e. to be good role models

A thought-provoking article.

Issue 2 in January included an intervention article considering the question 'Is patellofemoral joint arthritis an under-recognised outcome of anterior cruciate ligament reconstruction?' A group of academic Australian physiotherapists including Jill Cook and Kay Crossley conducted a literature review. They found arthritis affecting the patellofemoral joint in about 50% of post-ACL reconstruction patients, and this occurred both with patella tendon grafts and hamstring grafts. They postulate that prolonged range of motion and strength deficits despite good rehabilitation plus inflammation and concomitant damage to the patellofemoral joint articular cartilage may be the major factors contributing to this unwanted consequence of ACL reconstruction, hence the need for targeted rehabilitation in all our patients.

Exercise is important in falls prevention and an article by Liu-Ambrose and colleagues from Vancouver postulates that this may be via maintaining better cognition as well as its proprioceptive benefits. Still more reasons to remain active into old age.

FEBRUARY

Issue 3 from February 2013 could be entitled the cardiac issue. It presents the results of the Seattle Summit on ECG Interpretation. Drezner and colleagues, including Matthew Wilson who spoke at the SMNZ Conference in 2012, present the modern criteria for ECG interpretation in athletes. These are more sensitive and specific than those used hitherto and are an attempt to reduce the incidence of sudden cardiac death in athletes during sport. They comment on normal ECG findings in athletes including sinus bradycardia, first degree AV block and incomplete right bundle branch block plus isolated QRS voltage criteria for left ventricular hypertrophy. By contrast, any T-wave inversion of >1mm in two or more of leads V2 to V6, 2 and AVF or 1 and ACL or QRS duration >140 milliseconds or complete left bundle branch block should be regarded as pathological.

Later articles in the same issue by the same group of authors include one on normal ECG findings, recognising the physiological adaptation in athletes plus two articles on abnormal ECG findings in athletes. The first of these documents the changes suggestive of cardiomyopathy and the second lists the changes suggestive of primary electrical disease. Collectively, these articles enable the team physician to identify a large proportion of athletes who can be safely reassured that their heart is normal. There will always be a small group who require further evaluation, and in New Zealand we have taken the approach of sending all of our athlete ECGs under the High Performance Sport New Zealand network to Professor Rob Doughty so that there is a consistency of approach.

MARCH

The March issue included an important article on physical inactivity - 'Getting Scotland on the move'. Andrew Murray, its author, pointed out that Scots take pride in their long history of innovation and invention. Scots have contributed the three best friends of the couch potato: the telephone, the refrigerator and the television. He points out that a simple clear message must be communicated effectively, raising an awareness of recommended exercise guidelines. Developing partnerships to influence policy across a range of sectors including education, transport, recreation and health is likely to bring about the best result. Since Scotland has a similar population to that of New Zealand but we have better weather, we have even more reason to take onboard this advice.

Later in the same issue was a detailed article entitled 'Factors associated with patellofemoral pain: a systemic review'. The authors conducted a meta analysis including 47 studies and found that a larger Q angle, sulcus angle and patella tilt angle plus less hip abduction strength, lower knee extension peak torque and less hip external rotation were significant contributory factors to patellofemoral pain syndrome. Foot arch-height index and congruence angle were not associated with patellofemoral pain. Since patellofemoral pain is the single commonest clinical entity we see, this article is worth a further look.

As a companion to the above article, there was a further systematic review in the same issue entitled Gluteal muscle activity and patellofemoral pain syndrome. These authors found 10 case controlled studies with moderate to strong evidence that gluteus medius activity was delayed and of shorter duration during stair negotiation in patellofemoral pain sufferers compared with the normal population. Still more reason to include gluteus medius strengthening in our rehabilitation of this very common condition.

APRIL

Issue 5 in April could well be called the concussion issue. The keynote article was the Consensus Statement on Concussion in Sport produced after the 4th International Conference on Concussion in Sport held in Zurich in November 2012. Written by Paul McCrory and an international team of co-authors, this nine page article provides state of the art advice on concussion management. It should be read by any clinician dealing with concussed athletes. Immediately after the article there are copies of the SCAT3, i.e. the third edition of the Sport Concussion Assessment Tool, and a variation for use in children. A child Maddocks score includes questions such as where are we at now, is it before or after lunch, what did you have for your last lesson or class, and what is your teacher's name.

Later in the same issue there are other concussion related articles including what is the lowest threshold to make a diagnosis of concussion, day of injury assessment of sport related concussion and the effects of rest and treatment following sport related concussion with a systematic review of the literature.

A late article evaluates the evidence for chronic concussion related changes in retired athletes, so-called chronic traumatic encephalopathy (CTE). This has been given a lot of play in the media, but a critical review of the literature has revealed no published epidemiological, cross-section or prospective studies relating to modern CTE. Whilst this no doubt occurs, the extent to which age related changes, psychiatric or mental health illness, alcohol or drug use or coexisting dementing illnesses contribute to the overall picture is largely unaccounted for in the published literature. Therefore, we need to interpret media reports with some caution.

The second April issue included a consensus statement on the terminology and classification of muscle injuries in sport - the so-called Munich Consensus Statement. Previous muscle injury classification systems have been graded 1 to 3 referring to tears of a few, moderate number or complete tearing of fibres without muscle retraction. These authors propose the following:

- **Type 1A**

  Fatigue induced muscle disorder
- **Type 1B**

  Delayed muscle onset soreness
- **Type 2A**

  Spine related neuromuscular muscle disorder
- **Type 2B**

  Muscle related neuromuscular disorder
- **Type 3A**

  Minor partial muscle tear
- **Type 3B**

  Moderate partial muscle tear
- **Type 4**

  Subtotal or total muscle tear or tendon avulsion
- **Contusion**

  Due to direct injury to the muscle by a blunt external force

There is a comprehensive table listing the symptoms, clinical signs, location and ultrasound or MRI imaging findings relating to each type. This is the most comprehensive classification of muscle injuries and provides a good framework for assessing them.

Also in the same issue is a systematic review and meta analysis of the risk factors for hamstring muscle strain injury in sport. These authors identified 34 articles for

analysis. The risk factors most consistently associated with hamstring muscle strain injuries were age, previous hamstring injury and quadriceps peak torque. Surprisingly, things such as the length of the hamstring, dominant limb and slump test appeared to have little correlation with hamstring strain injuries.

Later in the same issue Per Renstrom, one of the giants of Swedish orthopaedics, considers eight clinical conundrums relating to ACL injury in sport. He provides a summary of the recent evidence and a personal reflection on that. He comments that not all patients with an ACL tear need surgery but those with high functional demands have the most to benefit from it. The surgeon should be experienced and use a reconstructive procedure that they know well and are comfortable with. Although there is much debate regarding the technique of ACL surgery and the use of double or single bundle grafts, and the controversy of patella tendon versus hamstring grafts, he believes that it is not the choice of graft or technique that is the key for success, it is often the choice of surgeon. It has been previously noted that a total of 85% of the ACL surgeons do less than 10 ACL reconstructions per year; in other words, send your patients to surgeons who perform this procedure frequently. All in all, an excellent summary of this topic written by a man that I have had the pleasure of working with at several Olympic Games, and arguably the most physicianly orthopaedic surgeon you could hope to meet.

Bonacci and colleagues evaluated the effect of running in a minimalist and lightweight shoe compared with running barefoot. Minimalist shoes have become very popular in recent years but are still not the same as running barefoot. Barefoot running demonstrated less knee flexion at mid-stance and less dorsiflexion at initial contact compared with wearing even a minimalist running shoe.

The regular column on A to Z of Nutritional Supplements included a section on whey protein. It is purported to enhance weight loss or support gains in skeletal muscle mass in association with weight training. The greater anabolic potential of whey is probably attributed to its faster protein digestion and amino acid absorption rates and a higher leucine content. They comment that whey protein supplements appear to be safe to consume, with no direct evidence to suggest that excessive protein intake is harmful to healthy kidneys.

MAY

Issue 7 in May included a summary of the injuries and illnesses seen during the London Olympic Games in 2012. At least 11% of the athletes incurred an injury during the Games and 7% suffered an illness. The incidence of injuries and illnesses varied substantially between different sports, with the highest injury risk in tae kwon do, football, BMX, mountain biking, athletics, weightlifting, hockey, badminton and handball.

A later article analysed usage of the polyclinic at the Olympic Village, with 3220 encounters of which 52% were musculoskeletal, 30% dental and 9% ophthalmic. It should be noted that large Olympic teams like New Zealand provide most of the care to their athletes within the team setting but occasionally athletes will be taken across to the polyclinic for a specialist consultation or imaging, e.g. MRI scans.

Issue 8 in May could be called the injury prevention issue. It included articles on applying implementation science to injury prevention plus an evaluation of the FIFA 11+ programme in Canadian female and youth football players.

In addition, there was an analysis of the effect of the Norwegian ACL prevention study in female handball players 10 years after the study was implemented. Prevention strategies included knee control exercises focusing on hip/knee/toe alignment plus proprioception drills and jumping exercises with two foot landings and technique training with the same focus. The researchers found that the coach was a key partner in implementing an injury prevention strategy, which is hardly surprising. They make reference to the New Zealand Rugby Smart programme in reducing spinal injuries in our country. In summary, they found that the ACL injury rate can be kept low through nationwide preventive initiatives and by focusing on the coach as a key partner in injury prevention.

At the end of this issue was an article entitled 'Ankle taping and bracing for proprioception'. The authors of a systematic review and meta analysis found that joint position sense and sense of movement at the ankle is not increased with the use of ankle tape or ankle braces in those who suffer repeated ankle sprains; in other words, it remains unclear as to how taping and bracing actually work. They do acknowledge, however, that there is evidence that supports taping and bracing for preventing injury so it seems reasonable to keep on doing it for our at-risk players. However, this should not be used as an excuse for them to stop doing proprioception drills.

JUNE

Issue 9 in June included a comprehensive article on the effect of airline travel on performance. It discusses jetlag, sleep deprivation, travel at altitude and nutritional considerations that could negatively affect performance. They listed nine recommendations to decrease the effect of

air travel on performance. These included exposure to natural daylight where possible and exposing travellers to social contact at times appropriate for local time at the destination. In addition, they recommended avoiding caffeine during travel, consuming extra fluids, and eating small meals before and during the flight. They found that short naps of 20-30 minutes could be helpful in recovering from sleep deprivation and restoring a normal state of arousal. All good advice for those of us who need to travel from New Zealand to competitions that may be many time zones away.

Changing topics, genetic biomarkers are much in the news. Martin Schwellnus, who spoke at the SMNZ Conference in Rotorua a few years ago, has written an excellent summary of genetic biomarkers and exercise related injuries with their current clinical application. At this stage there is only weak evidence that genetic biomarkers related to injuries such as achilles tendinopathy can predict outcome. Therefore, there is no indication for routinely testing for them. They may have some application in a research setting.

Finally in this issue there was an excellent review article of sport and exercise related tendinopathies. This followed a symposium held in Vancouver in 2012. The authors comment that tendinopathy is primarily a clinical diagnosis. Imaging can add useful information if a partial tear or new vessel formation is seen, but other imaging abnormalities do not necessarily signal the presence of clinically significant issues. Severity of tendinopathy is assessed along a continuum and many tendinopathies have concomitant muscle atrophy. Therefore, the prescription of a progressive exercise regime, whether concentric, eccentric or both, is logical treatment. The commonest cause of failure is lack of compliance with a progressive regime up to 90 or more repetitions per day. Assuming good compliance has been maintained, then adjunctive treatments including injection of whole blood, platelet rich plasma or high volume local anaesthetic are options to consider. The authors also discussed surgery for the achilles, patella and rotator cuff tendon and the outcome measures including VISA scores. Given the chronic nature of tendinopathies, prevention is an important strategy and the article concludes with comments on knowledge translation, i.e. uptake of research in the clinical setting. Well worth the read.

My pick for the most valuable article in this six months worth of issues would be the Zurich Consensus Statement on Concussion in the April issue. To get consensus amongst doctors from several continents and many sporting codes is a difficult undertaking and the work of this group has greatly enhanced the consistent management of concussion in athletes throughout the world.

Chris Milne

Concussion in amateur rugby league players in New Zealand: A review of player concussion history

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ABSTRACT

Aim

To review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events outside of this time frame and medical review and clearances for return-to-play.

Method

A retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players.

Results

A total of 213 (mean ± SD: age, 19.2 ± 4.4) amateur rugby league player concussion questionnaires were reviewed. There was an average of 4.0 ± 2.6 concussive injuries per participant in the previous two years and an average of 5.0 ± 4.6 concussive injuries per participant the period preceding this. A total of 7.5% participants saw a medical doctor for their concussion; 5.2% completed the required three week return-to-play; and 2.8% reported seeing a medical doctor for medical clearance. No Under 15 yr . old player reported seeing a medical doctor or having a stand down period for return to play.

Discussion

Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings. An unexpected finding was that 7% of all players had active concussive symptoms and was a cause for concern. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.

INTRODUCTION

Traditionally viewed as a transient injury void of long term consequences,[2] sport-related concussions were once trivialised by coaches and "playing through the pain" was regarded as a sign of the individual's toughness and commitment to the team.[2] Even clashes to the head that were often thought of as "just a ding" are now receiving more attention as a potentially serious injury.[28] More recently there has been an increased awareness of the long-term effects of sport-related concussion and the possible link with the tau? pathology termed chronic traumatic encephalopathy (CTE).[2] This link with CTE as well as other neurological conditions such as depression and mild cognitive impairment have been reportedly associated with players who have a history of more than one concussive event.[2, 23]

Players undertaking any sporting activity are at risk of receiving a concussive event.[24] The risk of receiving a concussive event is reported to be increased in players with a history of a previous or multiple concussive events.[11] A way to assist the team medical personnel to manage this risk is to record the players individual concussion history. Often this requires self-disclosure by the player or the management of a concussion history by the team medical person through sideline and post-match examinations. Recently there has been an increase in published research on sport-related concussion relating to the identification, assessment and management in all sporting environments.[5,20] Concussion has become one of the most troublesome injuries facing the sports medicine professional,[15] especially with regard to the early identification of concussive signs and symptoms and appropriate concussive management facilitation.[9,22] Amateur sports such as rugby league in New Zealand do not have the resources available for qualified medical personnel to be at every match.[14] As such it is often the coach or team manager, if the team has one, to make decisions with regard to player welfare and this includes concussion identification and management. The awareness of team management and concussion has been previously reported[14,25,29] and highlights the need for concussion education for all people involved in the management level of sports participation.

Players with a concussive injury returning to their sport are at a greater risk of complications.[11,19] These complications are related to subsequent concussive events and may result in prolonged concussive symptoms and cumulative cognitive deterioration.[20] Often the identification of a sport-related concussion is left to the team coach as there are seldom any medical personnel available at the games and training sessions.[15] As a result concussive signs may be missed, as the coach may be unable to take the required time to fully assess the

player and may miss signs of a player with a concussion. The aim of this study was therefore to review self-reported concussive history of amateur rugby league players to identify concussions within a two year period, concussive events prior to this time frame and medical review and clearances for return-to-play.

METHODS

A retrospective analysis was undertaken on all medical pre-competition questionnaires completed during the 2010 to 2012 competition periods by amateur rugby league players in Wellington, New Zealand. The pre-competition questionnaires were requested as part of the assessment process for the provision of medical coverage to the players by one of the authors (DK). The questionnaires were reviewed for the concussion history of players for the previous two years, prior history of concussion and the Post-Concussion Symptom Scale that was used for the establishment of baseline data for players competing in various rugby league competitions.

SUBJECTS

Over the study period a total of 213 male participants (mean ± SD: age, 19.2 ± 4.4 yr) from domestic and representative amateur rugby league teams agreed to take part in recording their concussion history. All participants were provided with an information sheet and gave either personal informed consent to participate or had parental informed consent before completing the history questionnaire.

Concussion History Questionnaire

All players were asked about their concussion history. This included history of concussions in the current and previous playing seasons, number of concussions, residual symptoms from any concussions reported and medical clearance to return-to-play. Participants were also asked to list the total number of years they had played rugby league. Participants were also asked to identify whether they considered a mild Traumatic Brain Injury or a Concussion to be more serious, or whether they were the same.

Post-Concussion Symptom Scale (PCSS)
The PCSS is a 22-item neuropsychological assessment scale that is a part of the larger Sport Concussion Assessment Tool (SCAT). Developed in the late 1980's,[18] the PCSS was designed to measure the severity of symptoms in the acute phase of recovery from a concussion. Using a 7-point Likert-type scale anchored at 0 (complete absence) to 6 (most severe) players rank each symptom according to the severity that they have experienced. The symptoms are not specific to concussion and even non-injured participants have recorded symptoms on the PCSS.[18] A combined total of 7 symptoms was set as the threshold for the duration of the study.[17] Any player reporting more than 7 symptoms on any assessment were referred for further medical evaluation. This threshold was adopted as an indicator of a head injury having occurred based on prior studies.[17] The internal consistency reliability (Cronbach's α) for the PCSS has been reported to range from 0.88 to 0.94.[18]

Statistical Analysis

All the data collected were entered into a Microsoft Excel spreadsheet and analysed with Statistical Package for Social Sciences (SPSS v.19.0; IBM SPSS Inc, Chicago, IL, USA for windows). Data are reported as means and standard deviations (SD) where appropriate. Correlations between concussion history and symptoms reported were analysed using a Spearman's Correlation Coefficient (rs). To ascertain reliability of the questionnaire and player recall of their concussion history, players who were selected in more than one group (n=20) had their concussion history computed with a weighted Cohen κ coefficient.[3,30] A one-way analysis of variance (ANOVA) was used to investigate differences between the reported concussions, previous history of concussions and the symptoms identified. Statistical significance was set at $p<0.05$. All data are reported as mean ± SD.

RESULTS

Participants reported an average of 4.3 ±2.7 yr. playing experience in rugby league. There was an average of 4.0 ±2.6 concussive injuries per participant in the previous two years (see Table 1). A total of 7.5% of all participants reported seeing a medical doctor for their reported concussion while only 5.2% completed the required three week return-to-play process. Even fewer players (2.8%) reported seeing a medical doctor for a clearance for return-to-play. No Under 15 yr old player reported seeing a medical doctor or having a stand down period for return-to-play. It was identified that 7.0% (n=27) of the participants had high PCSS scores and were assessed to have an active concussion at the time the assessment was being conducted.

Despite 39.0% of players reporting that they had not had a concussion in the previous two years they did report that they had loss of consciousness and "bell ringer" / "dings" from match participation (see Table 2). Players reporting three or more concussions (n=31) also reported the highest mean number of headaches (2.0 ± 1.0), dizziness (2.9 ± 1.9) and previous concussions (6.5 ± 4.1) than players reporting fewer concussions in the previous two years.

There was a correlation observed between concussions reported in the last two years and previous concussions ($rs=0.597$; $p<0.001$); loss of consciousness and previous loss of consciousness ($rs=0.681$; $p<0.001$) and "bell-ringer" / "ding" and dizziness ($rs=0.622$; $p<0.001$) and these were significant (see Table 3). This was similar for previous loss of consciousness and headaches ($rs=0.671$; $p<0.001$); previous concussions and headaches ($rs=0.627$; $p<0.001$) and PCSS score and PCSS severity ($rs=0.916$; $p<0.001$).

The reliability of the self-reported concussion questionnaire yielded a weightedκ coefficient with substantial reliability for concussions ($\kappa=0.797$; $p<0.001$), loss of consciousness ($\kappa=0.816$; $p<0.001$) and bell ringer / ding ($\kappa=0.777$; $p<0.001$) in the previous two years. When compared with the players history of previous concussions ($\kappa=0.864$; $p<0.001$) and loss of consciousness ($\kappa=0.914$; $p<0.001$) the results were similar.

DISCUSSION

An average of four concussive events per player for all age groups is concerning. Even more concerning is the finding that despite players reporting having had no concussions in the previous two year period they reported an average of nearly four concussive events. Although the history of concussions reported in this study was undertaken by the use of a self-reporting questionnaire, there is some support that the concussion history reported may be reliable.[12] The questionnaire utilised in this study was identical for all groups as part of their medical assessment in the

Table 1: Demographics of amateur rugby league participants for age, playing experience, concussive history and associated symptoms for previous two years, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

	Total	U15	U17	Premier
DEMOGRAPHICS				
Age, average SD [range]	19.2 ±4.4 [13-34]	14.5 ±0.6 [13-15]	16.5 ±0.5 [16-17]	21.9 ±3.9 [18-34]
Forwards	119	24	26	69
Backs	94	18	21	55
Experience, average SD [range]	4.3 ±2.7 [1-12]	4.7 ±2.8 [1-9]	3.4 ±2.5 [1-10]	4.4 ±2.7 [1-12]
CONCUSSIVE HISTORY IN PREVIOUS TWO YEARS				
Concussion, average SD [range]	1.9 ±1.2 [0-5]	1.8 ±1.2 [0-5]	2.0 ±1.3 [0-5]	1.9 ±1.3 [0-5]
Loss of Consciousness, average SD [range]	1.2 ±0.2 [0-2]	1.3 ±0.5 [0-2]	1.1 ±0.3 [0-2]	1.2 ±0.4 [0-2]
"Bell Ringer" / "Ding", average SD [range]	2.2 ±2.0 [0-10]	2.3 ±2.4 [0-10]	2.3 ±1.9 [0-10]	2.2 ±1.9 [0-10]
Total, average SD [range]	4.0 ±2.6 [0-11]	4.1 ±2.8 [0-11]	3.8 ±2.5 [0-11]	4.0 ±2.5 [0-11]
SYMPTOMS PREVIOUS TWO YEARS				
Headaches, average SD [range]	1.9 ±1.4 [0-6]	2.4 ±1.8 [0-6]	1.6 ±0.6 [0-3]	1.9 ±1.4 [0-6]
Dizziness, average SD [range]	2.0 ±1.5 [0-6]	1.5 ±0.7 [0-3]	2.2 ±1.6 [1-6]	2.0 ±1.5 [0-6]
Memory Difficulties, average SD [range]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]
Visual Disturbances, average SD [range]	1.3 ±0.4 [0-2]	1.0 ±0.0 [0-1]	1.3 ±0.5 [0-3]	1.3 ±0.4 [0-2]
Hearing Problems, average SD [range]	1.4 ±0.8 [0-3]	1.7 ±1.0 [0-3]	1.0 ±0.0 [0-1]	1.4 ±0.8 [0-3]
Other Symptoms, average SD [range]	1.8 ±1.2 [0-4]	1.8 ±1.2 [0-4]	1.8 ±1.5 [0-4]	1.8 ±1.2 [0-4]
CONCUSSIVE HISTORY IN PREVIOUS YEARS				
Concussion, average SD [range]	2.8 ±2.6 [0-10]	3.0 ±2.8 [0-10]	2.5 ±2.6 [0-10]	2.9 ±2.6 [0-10]
Loss of Consciousness	1.8 ±1.5 [0-6]	1.8 ±1.4 [0-6]	1.8 ±1.7 [0-6]	1.7 ±1.5 [0-6]
"Bell Ringer" / "Ding", average SD [range]	2.4 ±2.3 [0-10]	1.9 ±1.1 [0-4]	2.2 ±2.1 [0-10]	2.5 ±2.4 [0-10]
Total, average SD [range]	5.0 ±4.6 [0-19]	4.5 ±4.3 [0-19]	4.5 ±4.7 [0-19]	5.2 ±4.6 [0-19]
RETURN TO PLAY				
Seen by Dr, n (% of total)	16 (7.5)	0 -	3 (1.4)	12 (5.6)
Stand down (3 weeks) completed, n (% of total)	11 (5.2)	0 -	2 (0.9)	8 (3.8)
Medical clearance to return to play, n (% of total)	6 (2.8)	0 -	2 (0.9)	4 (1.9)
POST CONCUSSIONS SYMPTOMS SCALE				
Score, average SD [range]	7.7 ±6.1 [0-22]	6.5 ±4.8 [0-22]	8.4 ±6.9 [0-22]	7.7 ±6.1 [0-22]
Severity, average SD [range]	10.9 ±17.1 [0-61]	8.6 ±12.1 [0-57]	12.0 ±19.5 [0-58]	10.9 ±17.1 [0-61]

pre-competition period and the use of a self-reported history of concussion measure was of moderate reliability.[12]

Players across all age groups were administered the same questionnaire in different settings where they were gathered for the respective age group competition meetings. Players who were involved in more than one assessment process (n=20) were removed from subsequent analysis to ensure there were no duplication of the results. These players did provide the source for the reliability analysis of the self-reported concussion history indicating the weighted κ statistics were in the "substantial" agreement range according to the guidelines of Landis and Loch.[16]

By utilising the PCSS for a baseline assessment it was planned to compare any PCSS assessment for any player that was suspected of having a concussion. An unexpected finding upon completing the baseline assessment was that 7% of players were identified as having active concussive symptoms. None of the players identified had seen a medical professional for their symptoms nor reported these symptoms to anyone as they felt this was all part of "playing the game". All of these players were advised to seek further medical assistance and required to provide a full medical clearance before they would be allowed to return to match activities. One player was subsequently medically cleared two days later by his own medical practitioner as he "had no signs of a concussion". This was despite the player initially reporting he had received a head clash in a match 10 days previously and had symptoms when the baseline assessment was conducted. Unfortunately this player recorded a concussive incident in the following match.[13] Consequently he was advised not to return to any match activities for the rest of the competition, to seek a full medical review and to refrain from training until medically cleared to do so.

A previous study[22] identified that players will not report concussive symptoms because they do not think it is sufficiently serious enough to necessitate reporting. A lack of knowledge of the risks and potential consequences of concussion was reported to be the most common reason for the non-reporting of a possible concussion.[22] It was also identified that more than a third

Table 2: Demographics of amateur rugby league participants reporting none, one, two, three or more concussions in the previous two years by age, playing experience, concussive history and associated symptoms, concussive history for prior years, return to play requirements and current post-concussion symptoms scores and severity by means, standard deviations and ranges reported.

	Previous 2 Year History			
	No Concussions (n=83)	**1 Concussion** (n=66)	**2 Concussions** (n=33)	**3+ Concussions** (n=31)
DEMOGRAPHICS				
Age, average SD [range]	18.7 ±4.5 [13-34]	19.7 ±4.6 [14-32]	19.2 ±4.5 [14-32]	19.5 ±3.8 [14-28]
Experience, average SD [range]	4.6 ±2.8 [1-10]	4.2 ±2.8 [1-12]	4.0 ±2.9 [1-10]	3.7 ±2.2 [1-8]
HISTORY IN PREVIOUS TWO YEARS				
Concussion, average SD [range]	0.0 -	1.0 ±0.0	2.0 ±0.0	3.8 ±1.5 [3-5]
Loss of Consciousness, average SD [range]	1.0 ±0.0 [0-1]	1.1 ±0.4 [0-2]	1.0 ±0.0 [0-1]	1.5 ±0.5 [0-2]
"Bell Ringer" / "Ding", average SD [range]	3.6 ±2.9 [0-10]	1.2 ±0.4 [0-2]	1.5 ±0.5 [0-2]	2.6 ±1.4 [0-5]
Total, average SD [range]	3.7 ±3.2 [0-11]	2.8 ±0.9 [1-4]	3.3 ±0.9 [2-4]	7.1 ±2.5 [5-10]
SYMPTOMS PREVIOUS TWO YEARS				
Headaches, average SD [range]	0.0 -	2.1 ±1.7 [0-6]	1.3 ±0.5 [0-3]	2.0 ±1.0 [0-3]
Dizziness, average SD [range]	3.3 ±2.1 [0-6]	1.3 ±0.4 [0-2]	1.5 ±0.5 [0-2]	2.9 ±1.7 [0-5]
Memory Difficulties, average SD [range]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]	1.0 ±0.0 [0-1]
Visual Disturbances, average SD [range]	1.5 ±0.5 [0-2]	1.0 ±0.0 [0-1]	2.0 ±0.0 [0-2]	1.0 ±0.0 [0-1]
Hearing Problems, average SD [range]	0.0 -	1.4 ±0.8 [0-3]	1.0 ±0.0 [0-1]	0.0 -
Other Symptoms, average SD [range]	0.0 -	1.6 ±0.4 [0-2]	4.0 ±0.0 [0-4]	0.0 -
PREVIOUS HISTORY OF CONCUSSIVE EVENTS				
Concussion, average SD [range]	1.4 ±0.5 [0-2]	2.1 ±1.2 [0-5]	2.5 ±1.4 [0-4]	6.5 ±4.1 [0-10]
Loss of Consciousness	1.0 ±0.0 [0-1]	1.4 ±1.0 [0-4]	2.0 ±0.0 [0-2]	4.0 ±2.1 [0-6]
Bell Ringer / Ding, average SD [range]	4.0 ±3.8 [0-10]	1.3 ±0.4 [0-2]	2.6 ±1.9 [0-6]	2.5 ±0.5 [0-3]
Total, average SD [range]	3.2 ±3.8 [0-13]	4.0 ±2.0 [0-9]	5.5 ±2.9 [2-10]	10.1 ±7.6 [2-19]
RETURN TO PLAY				
Seen by Dr, n (% of total)	5 (2.3)	7 (3.3)	1 (0.5)	3 (1.4)
Stand down (3 weeks) completed, n (% of total)	3 (1.4)	5 (2.3)	1 (0.5)	2 (2.9)
Medical clearance to return to play, n (% of total)	3 (1.4)	2 (0.9)	0 -	1 (0.5)
POST-CONCUSSION SYMPTOMS SCALE				
Score, average SD [range]	6.5 ±5.5 [0-20]	6.1 ±3.0 [0-12]	7.7 ±7.1 [2-20]	18.5 ±3.6 [0-22]
Severity, average SD [range]	14.4 ±16.7 [0-57]	7.4 ±5.4 [0-18]	14.2 ±22.6 [0-61]	21.4 ±24.7 [0-57]

Table 3: Spearman's correlations between reported history of concussion, loss of consciousness and bell ringers; symptoms experienced post event in the past two seasons; previous history of concussion, loss of consciousness and bell ringers; post-concussion score and severity of amateur rugby league players in New Zealand.

	Concussion	LOC	"Bell Ringer"	Headaches	Dizziness	Memory	Visual	Hearing	Other	Previous Concussion	Previous LOC	Previous "Bell Ringer"	PCSS Score	PCSS Severity
Concussion	-													
LOC	0.30a	-												
"Bell Ringer"	0.18a	0.17b	-											
Headaches	0.45a	0.51a	0.06	-										
Dizziness	0.30a	0.32a	0.62a	0.42a	-									
Memory	0.28a	0.34a	0.20a	0.26a	0.18a	-								
Visual	0.07	0.16b	0.20a	0.35a	0.22a	0.36a	-							
Hearing	0.13	0.20a	0.11	0.29a	0.35a	0.12	0.30a	-						
Other	0.14b	0.32a	-0.11	0.48a	0.16b	0.14b	0.10	0.47a	-					
Previous Concussion	0.60a	0.45a	-0.05	0.63a	0.20a	0.23a	0.40a	0.29a	0.33a	-				
Previous LOC	0.23a	0.68a	0.18a	0.67a	0.40a	0.48a	0.42a	0.12	0.51a	0.50a	-			
Previous "Bell Ringer"	0.31a	0.25a	0.04	0.31a	0.31a	0.13	0.41a	0.24a	0.15b	0.49a	0.22a	-		
PCS	0.14b	0.34a	-0.02	0.21a	0.24a	0.30a	0.14b	0.08	-0.07	0.16b	0.21a	0.30a	-	
PCS Severity	0.07	0.22a	-0.03	0.06	0.12	0.31a	0.15b	-0.12	-0.26	0.01	0.07	0.29a	0.92a	-

LOC: Loss of Consciousness; PCS: Post-Concussion Symptoms Scale; Correlation is significant at (a) p< 0.01; (b) p<0.05

of players failed to report a concussive injury as they did not recognise that they had sustained a concussive injury based on their symptoms.[22] This may have been the case in the current study with 7% of players showing signs of a concussive event. In a recent study[14] involving amateur rugby league team management's knowledge, recognition and management of concussion it was reported that there was an average of 42% of concussion specific questions being answered correctly. Furthermore 39% of team management reported that concussion occurred only when there was loss of consciousness, 26% of team management would not remove a concussed player from the field of play and 20% would let a symptomatic player continue on playing. These findings may be reflective of the concussive history reported with some players reported loss of consciousness but not as concussion. Further research is warranted to explore the concussion awareness of amateur rugby league players.

It is a major concern that only 7.5% of players reported their concussive history identified that they sought medical assistance after having a concussive event. Similarly the finding that only 5.2% of players with concussive events completed a 3 week stand down and even less (2.8%) sought a medical clearance is also concerning. Although the New Zealand Rugby League has a concussion policy, based on an international consensus agreement,[23] in place for the management of concussive events and provides an annual update for the identification and management of concussion it appears this information is not reaching all members of this sporting environment. Additionally, there are no readily available tools for the assessment of concussion on the sideline for the non-medical support person with amateur rugby league teams.

A possible tool that may assist the team medic / trainer is the King-Devick test. Originally designed as a saccadic reading test,[26] the King-Devick has been utilised with amateur rugby league,[13] martial arts fighters, boxing, basketball and American collegiate football players[7,8] and has been shown to readily identify concussive events.[7,8,13] Additionally the King-Devick does not require the operator to be a medical professional and requires less than a minute to administer,[7] unlike the Sports Concussion Assessment Tool which can take approximately 20 minutes to complete away from the sideline environment.[6]

Non-compliance with return-to-play protocols is not unique to amateur rugby league players and has been reported in studies on rugby union,[10,27] ice hockey[1] and US high school athletes.[21] In a study[10] reporting on compliance with return-to-play regulations in schoolboy and community rugby union it was reported that schoolboy rugby had 100% non-compliance with player return-to-play after a concussive event. The current study is similar to suburban rugby non-compliance (95% vs. 97%) but greater than other studies reporting on high school rugby[27] (77.1%), ice hockey (33%)and US high school sports (16.7%) non-compliance and indicates that there are differences in how return-to-play regulations are completed in sporting environments despite international agreement with management of sport-related concussions. Further research is warranted to explore the return to play practices of amateur sports such as rugby union and rugby league.

Players reporting three or more concussions in the previous two years also reported more prior concussions than those players reporting none, one or two concussions. This finding was not unexpected as a prior history of concussion places the brain at risk to repeat injury and symptom exacerbation.[24] As well sports participants with two or more concussions have also been reported to have more neurobehavioural symptoms and impairment on selected neuropsychological tests than players with a history of a single concussion.[4] Recently it has been identified that in some individuals with a history of concussion, there were electroencephalographic and motor changes observed in otherwise healthy individuals with no clinically apparent deficits after their concussion injuries.[2] Unfortunately there are no longitudinal studies completed on the effects of these changes but it was hypothesised that as the individuals age, the changes identified may manifest into clinically significant functional impairments.[2] Of concern is if the players recorded in this study will have these long term changes and, if those with the higher number of reported concussions have a greater electroencephalographic and motor decline than other players with fewer reported concussions. Further longitudinal research is warranted to monitor players with a history of sport-related concussion.

It is acknowledged that this study does have limitations. Primarily the study is retrospective in nature by getting the players to document their concussion history. As such the data obtained is purely reliant upon: (a) The player being honest in recording their individual concussion history; and (b) The player being able to remember the total number of concussive events, associated symptoms and when these occurred. By incorporating the terminology "Ding" and "Bell ringer" into the questionnaire it was hoped that players would be more honest in their replies and this would give a more accurate indication of the actual number of concussive events that occur from participation in rugby league injuries. Despite this the study was viewed as a beginning in the path towards identification of the number of concussive incidents that occur in amateur rugby league in New Zealand.

CONCLUSION

In getting players to record their concussion history as part of a pre-competition medical history it was identified that although some players reported as never had a concussion; they did report that they had previous concussive events. As well only 7.5% of players sought medical assistance after their concussive event and even less reported completing a stand down period or a medical clearance for the concussion. What this may indicate is that there is a lack of knowledge pertaining to the risks and potential consequences of concussions by players and team management. These findings suggest that further educational efforts are warranted targeting both players and team management on the risks associated with, and management of sport-related concussion.

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Reliability of 2D lower extremity alignment measures in elite adolescent ballet dancers

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ABSTRACT

This study investigated the reliability of two-dimensional (2D) analysis of knee and pelvic angles during functional screening tests in elite adolescent ballet dancers. A modified knee valgus angle, lateral pelvic tilt, and depth of movement were measured using 2D video analysis during two dance movements. Between-trial reliability was assessed using intraclass correlation coefficients (ICC) and the typical error (TE) of measurement. The difference in angles between legs was assessed using paired sample t-tests, while the relationship between the movements was measured using Pearson correlation coefficients. Reliability of the modified knee valgus angle in the fondu (ICC = 0.88 to 0.89; TE = 3°) and temps levé (ICC = 0.80 to 0.87; TE = 6 to 8°) was high. There was a significant difference in knee angle between the left and right legs ($p = 0.001$). A strong correlation for the knee angle between the movements on both the left and right legs ($r = 0.93$ to 0.94) was observed. Moderate to high reliability was demonstrated in 2D analysis of knee and pelvic angles during dance movement. This simple technique may be useful to screen movement in dancers.

INTRODUCTION

Injuries to the lower extremities are very common in dancers, with 16-17% of all injuries occurring at the knee joint.[2,3,8] The cause of these injuries is thought to be multifactorial, with poor alignment suggested as one factor contributing to injury risk at the knee.[12] In athletes, poor frontal and/or transverse plane control of the pelvis, hip, knee and foot is considered less than ideal, and it is thought that identifying this may help detect those athletes most at risk for injury.[13,16] This pattern of lower extremity dynamic alignment is also considered important in dancers,[1] and there has been much recent interest in the development of simple, reliable screening methods. A number of studies have been conducted in athlete populations to assess for poor lower extremity alignment with several studies demonstrating a link between poor dynamic alignment and injury.[9,17] Greater frontal plane projection angle (FPPA) of the knee during a single-leg squat in subjects with patellofemoral pain was reported by both Levinger et al,[9] and Willson et al.[17] Currently however no research has assessed elite ballet dancers for lower extremity alignment during functional dance movement.

While three dimensional (3D) motion analysis is recognised as the gold standard for assessing lower extremity movement, due to time and monetary costs it is unsuitable for large populations of athletes and small independent clinics. Therefore clinical assessment is usually performed via visual observation of functional tests, with increasing use of two-dimensional (2D) video analysis both in athlete and dancer populations.[14,16] The majority of previous studies investigating the reliability of 2D video measures in athletic populations have used the single leg squat and drop jump.[13,16] All measurements were recorded at maximum knee flexion, using the FPPA of the knee as the key measure. Intra-rater reliability was reported as excellent for the vertical drop jump (ICC ≥ 0.95),[13,16] and the single leg squat (ICC = 0.92).[13] Within-day test-retest reliability has also been classified as very good for the drop jump (ICC = 0.83 – 0.88)[11,13] and fair to moderate in the single leg squat (right leg ICC = 0.57, left leg ICC = 0.84).[13] Two functional dance movements closely linked to a single leg squat and/or drop jump, and thus with potential to be used as screening tests in a dance population, are the fondu and temps levé. Dance teachers routinely rate the position of the pelvis and lower extremity during these movements, with ideal dance technique requiring maintenance of the knee over the foot and a level pelvis.[1]

The potential for a 2D video analysis approach to be used as a screening tool should be considered, as this measure may assist visual assessment as well as provide an indication of young dancers requiring intervention to reduce the risk of injury.[10,16] There is potential for this 2D method of analysis to be used when screening elite ballet dancers during specific dance movement tasks.[10] However the reliability of 2D video in the assessment of lower extremity alignment during functional dance movement, (fondu and temps levé movements) requires investigation. If the reliability of this screening method in elite ballet dancers can be established, practitioners will be able to use the tests with confidence while also allowing the testing method to be used in longitudinal studies investigating injury risk and the effects of training and/or rehabilitation interventions in dancers. Therefore, the aim of this study was to investigate the reliability of two-dimensional (2D) analysis of knee

and pelvic angles during lower extremity functional screening tests in elite adolescent ballet dancers. In addition, comparisons between the right and left leg, as well as the association between the tests were investigated.

METHODS

Forty five (F=29, M=16) young ballet dancers (mean ±SD: 16.1 ±1.6 years, 1.68 ±0.89 m, 53.4 ±9.5 kg) were recruited from the Australian Ballet School (ABS) in Melbourne. All participants were full-time ballet students at the school. In accordance with institutional requirements ethics was granted for this study. The dancers and their guardians provided written assent/consent prior to participation.

Each of the dancers attended a dance studio on one occasion. Participants wore standard dance practice clothes for testing and were asked to remove all footwear. Tape was used to mark the anterior superior iliac spine (ASIS), centre of the patella and centre of the ankle joint bilaterally. All dancers performed two movements, a fondu (single knee bend in external rotation) and temps levé (single leg vertical jump in turnout) based on standardised verbal instructions. Participants performed three repetitions of each movement on both legs with the arms held in a classical position. As stipulated by ballet technique the fondu and temps levé movements require execution in external rotation (turnout). As a result, two digital cameras (Panasonic, Japan) were used, one parallel to the frontal plane of the body and a second perpendicular to the line of the foot. The second camera position was adjusted to ensure the camera was always perpendicular to the line of the dancer's foot irrespective of the dancer's turnout range (see Figure 1). Based on post-collection visual inspection, any temps levé trials where the dancers foot moved substantially out of line with the camera were omitted from the analysis of the modified knee valgus angle (12 trials, 6 left and 6 right). All cameras were positioned on tripods, level with the floor at a distance of 1-2 metres, and a height of 0.86 metres. The fondu movement was captured at a rate of 30 frames per second (fps) and the temps levé at a rate of 250fps.

Using Kinovea video analysis software (v 8.15) a modified knee valgus angle, (camera 2), depth of movement (camera 1), and lateral tilt of the pelvis (camera 1) were measured for all three trials performed by each dancer (Figure 2). These are the most commonly reported 2D frontal plane alignment measures in athletes.[16] The modified knee valgus angles were measured at the point when the patella was observed to be most medially positioned relative to the second toe. The pelvic angle was measured at maximal knee flexion which was visually estimated. Depth of movement was measured by assessing the distance of the ASIS relative to the floor at the lowest point of the movement in centimetres.

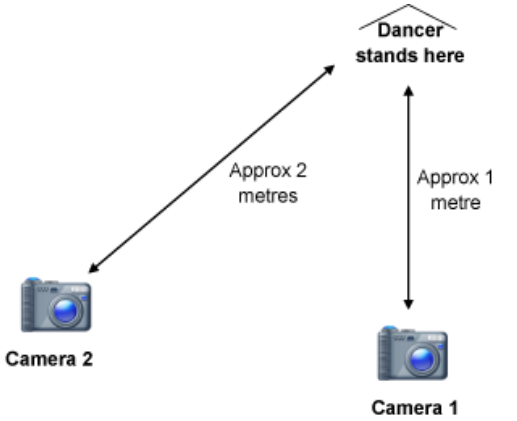

Figure 1: Camera set-up – camera 1 positioned parallel to the frontal plane of the dancer and camera 2 positioned perpendicular to the line of the foot of each dancer in their functional turnout position.

Pilot testing assessing intra-rater reliability for the 2D angle measures was conducted using nine semi-elite female ballet dancers (age 13.3 ±2.1) from Auckland, New Zealand. Two dimensional angles from the same video were measured by a single, experienced dance teacher on two occasions, separated by a period of seven days. Reliability was very high to extremely high for both knee and pelvic measures (ICC = 0.96 – 0.99, TE = 1-2 degrees).

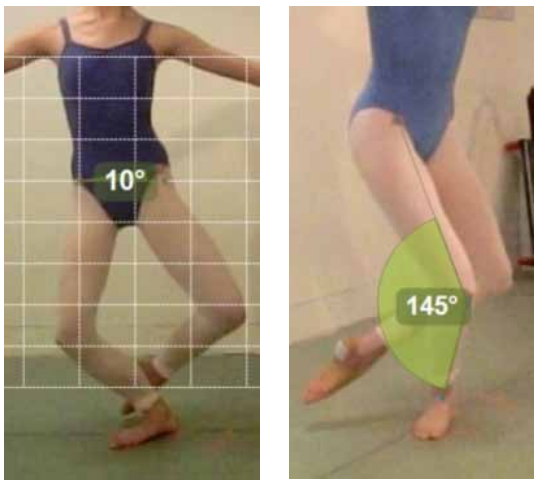

Figure 2: 2D Knee and pelvic measures.
A *(left)* The pelvic angle was measured by assessing the line of the ASIS's relative to the horizontal.
B *(right)* The modified knee valgus angle was calculated by connecting the bony landmarks of the ASIS, centre of patella and centre of the ankle joint.

Descriptive statistics including overall group mean and standard deviations were calculated for all angles. Using a Microsoft Excel spreadsheet by Hopkins[6] for analysis of between-trial reliability, intraclass correlation coefficients (ICC) and the typical error of the measurement (TE) were calculated using the original (raw) data. The magnitude of the ICC values were described as ≥0.99 = extremely high, 0.90-0.99 = very high, 0.75-0.90 = high, 0.50-0.75 = moderate, 0.20-0.50 = low, ≤0.20 = very low[4] (unpublished observations). Using IBM SPSS Statistics for Windows, version 21 (IBM Corp, Armonk, NY) the difference in means between the right and left limbs were compared using a paired sample t-test and qualitative magnitude based inferences were added using an excel spreadsheet.[5] Pearson correlation coefficients were also calculated to assess the magnitude of the associations between knee valgus angle for both movements on both the right and left legs. An alpha level of 0.05 was set as the significance level for all statistical tests.

RESULTS

Peak mean angles and standard deviations for the knee and pelvis during both the fondu and temps levé are presented in Table 1. Between-trial reliability for knee and pelvic angles during three repetitions of the fondu

and temps levé movements are presented in Table 2.

The between-trial reliability of the modified knee valgus angle in both the fondu (ICC = 0.88 to 0.89) and temps levé (ICC = 0.80 to 0.87) movements were high. Between-trial reliability of the depth of fondu and temps levé was high to extremely high (ICC = 0.85 to 0.99). Reliability of the pelvic angle in the fondu ranged from moderate to high (ICC = 0.67 – 0.79) while the temps levé showed only moderate reliability (ICC = 0.68 to 0.71). There was little difference in reliability between limbs.

The TE for the modified knee valgus angle in the fondu was 3°, but was higher in the temps levé ranging from 7–8°. Pelvic angle TE ranged from 1°-2° for both movements.

There was a significant difference in the knee valgus angle between the right and left legs for both the fondu (mean = 10°; p = 0.001) and temps levé (mean = 14.7°; p = 0.001) movements (Table 3). There was also a significant difference in pelvic angle between sides for both the fondu (mean = 1°; p = 0.005) and temps levé (mean = 1°; p = 0.03). For the knee valgus angle there was a strong, statistically significant correlation between the fondu and temps levé tests on both the left (r = 0.93, p=0.01) and right (r = 0.94, p=0.01) limbs.

Table 1. Between-trial reliability during the fondu and temps levé

		TE (°) (90% CL)	ICC (90% CL)	RELIABILITY*
FONDU	**Knee (R)**	3 (3-4)	0.88 (0.81-0.92)	High
	Knee (L)	3 (3-4)	0.89 (0.82-0.93)	High
	Pelvis (R)	1 (1-1)	0.79 (0.67-0.87)	High
	Pelvis (L)	1 (1-1)	0.67 (0.50-0.78)	Moderate
TEMPS LEVÉ	**Knee (R)**	6 (5-8)	0.87 (0.78-0.92)	High
	Knee (L)	8 (6-10)	0.80 (0.67-0.88)	High
	Pelvis (R)	1 (1-1)	0.71 (0.55-0.82)	Moderate
	Pelvis (L)	1 (1-2)	0.68 (0.44-0.76)	Moderate

*TE = typical error, ICC = intraclass correlation coefficient, R=right leg, L=left leg, *Terms for ICC magnitudes: 0.75-0.90 = high, 0.50-0.75 = moderate.*

Table 2. Peak mean angle (°) ±SD, mean difference in angle between right and left leg (90% CL), p value, inference

		PEAK ANGLE ±SD	Difference between Right and Left Sides		INFERENCE*
			MEAN (90% CL)	P-value	
FONDU	Knee (R)	175 ±3			
			10 (6-14)	0.001**	96%, very likely
	Knee (L)	186 ±3			
	Pelvis (R)	3 ±1			
			1 (1-2)	0.005**	0%, most unlikely
	Pelvis (L)	2 ±1			
TEMPS LEVÉ	Knee (R)	161 ±7			
			15 (9-20)	0.001**	99%, very likely
	Knee (L)	176 ±6			
	Pelvis (R)	3 ±1			
			1 (0-2)	0.03**	0%, most unlikely
	Pelvis (L)	2 ±1			

DISCUSSION

Poor lower extremity alignment has been proposed as an injury risk factor for ballet dancers, in particular for knee injury.[12] Dynamic lower extremity alignment has been assessed in athletic populations for injury risk, using a number of different screening tests.[9-11,13,16,17] No research has examined the dynamic lower extremity alignment of dancers using dance-specific movement.

The primary aim of this study was to assess between-trial reliability of simple 2D alignment measures in elite adolescent ballet dancers performing a fondu and temps levé. The excellent ICC values indicate that 2D video analysis of the modified knee valgus angle for both the fondu and temps levé movements is reliable between-trials on the same day. The ICC values for the knee were higher than the ICC values for the pelvic angles, however a contributing factor to the lower pelvic ICC's is the lack of variability between participants (range = 0 to10° for both fondu and temps levé). The low TE values for the pelvis still suggest this is a reliable measurement. The excellent ICC values for the depth of movement for both the fondu (ICC = 0.98-0.99) and temps levé (ICC = 0.85-0.88) indicate that elite adolescent dancers execute both movements with high repeatability. This is important for comparing movements on two separate occasions. Previous studies of similar lower extremity functional tests in athletes have suggested the maximum frontal and transverse plane deviations need to be assessed across the same range of sagittal plane motion for the test to be valid.[15] This idea is supported by studies reporting increased frontal plane motion with increases in sagittal plane motion during lower extremity functional tests.[7,18] Thus, the standardisation of depth is not required for screening within dancer populations, resulting in a simple screening test without the requirement for additional monitoring that adds to time and equipment needs.

The low TE values for both the knee and pelvic angles (TE = 1° – 4°) measured during the fondu further support the reliability of the measure between-trials. The TE values for the knee angles during the temps levés are higher (TE = 6° – 8°) suggesting greater variability with this movement. Based on these error values, practitioners should be able to establish if changes observed between-trials are true changes in individual

performance or a result of measurement error.[11] They also provide an indication to practitioners as to the uncertainty in a single screening measure if using this test. The TE values for the temps levés are higher than those reported in similar studies for the drop jump and single-leg landing.[11] This is most likely due to differences in the camera position, due to the dancers' external hip rotation, which resulted in greater variability in the dancer's orientation relative to the camera. The higher values could also be explained by the increased pace and load of the jump in comparison to the fondu. Practitioners need to be aware if using the temps levé that there is increased variability with this movement in comparison to other tests. No feedback was provided to the dancers in regard to their turnout or foot position relative to the camera during testing and this could be provided to reduce the variability observed during testing.
The significant correlation between the fondu and temps levé movements on both the right (r = 0.94) and left (r = 0.93) legs indicates that good execution of the fondu transfers successfully to a higher load temps levé movement. This may be useful for practitioners due to the simpler and slower nature of the fondu, making it easier to visualize, while also providing a reliable option for screening injured dancers who are unable to perform higher load movements. The significant difference in knee angle between the left and right legs for both the fondu and temps levé tests indicates that both legs should be tested during the screening of dancers for lower extremity dynamic alignment. While there was also a significant difference in pelvic angle between sides, the difference was small and we suggest it is of little practical importance.

There are several limitations in this study readers should be aware of. Firstly, within-day reliability only was assessed thus future studies should evaluate between-day reliability of the fondu and temps levé tests, while inter-rater reliability of the measures should also be considered. Secondly, while some practitioners may use 2D video to screen lower extremity alignment in dancers, many teachers visually rate movement without video assistance. Thus visual rating requires investigation, as well as the use of visual rating in comparison to 2D video analysis. Furthermore, feedback could be provided on the maintenance of turnout and the dancer's foot position perpendicular to the camera during the temps levé, with repeat tests if required, to maximise the reliability of the measurement technique. The use of these measures in comparative investigations between injured and uninjured ballet dancers and dancers of other ages warrants investigation. Finally, using cameras in only two positions does not replicate clinical or teaching strategies in which practitioners or teachers would view a dancer or movement from multiple angles.

CONCLUSION

The reliability of 2D knee and pelvic angles during the fondu and temps levé movements in elite adolescent ballet dancers is moderate to high. Knee angles between the two movements are strongly associated and there is very likely to be a difference in knee angle between legs. This simple screening technique may be useful to dance practitioners and in longitudinal studies examining injury risk.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest and no involvement with any commercial organisation that has a direct financial interest in relation to this manuscript.

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Exercise-induced bronchoconstriction prevalence in elite Australian Rules footballers

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ABSTRACT

Objectives

The primary objective of the study was to determine the prevalence of EIB in a cohort of elite Australian Football League (AFL) players. The secondary objective was to investigate whether clinical features or past history of asthma/EIB were associated with EVH results.

Design

Cross-sectional study

Methods

Players were recruited from two elite AFL clubs. All players were considered eligible for enrolment. Players completed a modified European Community Respiratory Health Survey, providing information regarding respiratory symptoms at rest and related to exercise, risk factors, past and family history and treatment of asthma/EIB. Players also underwent baseline spirometry and EVH testing.

Results

Sixty-five players were recruited. Fourteen out of 65 (22%) players tested EVH positive. Past history of asthma/EIB and low baseline spirometry were associated with an increased risk of EIB as designated by a positive EVH test, but both symptoms and atopy were not. Four out of 65 players tested positive despite having no past history of asthma/EIB.

Conclusions

The rates of EIB in elite AFL players was similar to that reported in other endurance-based team ball sports. A past history of asthma/EIB, or lower than expected baseline FEV1 on spirometry, should alert the physician to the increased likelihood of EIB, however, clinicians should not rely solely on clinical features to diagnose the condition. Consideration should be given to testing all elite AFL players for EIB/asthma with bronchial provocation testing.

Keywords

Asthma; exercise-induced asthma; bronchial provocation tests; Australian Rules; football

INTRODUCTION

Exercise-induced bronchoconstriction (EIB), defined as the transient increase in airways resistance that occurs following vigorous exercise,[1] is a common condition in the athletic population. Numerous studies have highlighted the prevalence of this condition in a number of sports, particularly cold weather sports (ice hockey, figure skating and cross country skiing[2]), endurance-based sports (triathlons,[3] cycling[4] and distance running[2]) and aquatic sports (swimming,[5,6] and water polo[7]). Furthermore, studies have suggested that EIB can be detrimental to sporting performance, both in endurance and power-based sports.[8-10] No studies have assessed the prevalence of EIB in elite Australian Football League (AFL) footballers.

EIB is present in patients with and without asthma. In those with asthma, up to 90% have been reported to develop airway narrowing in response to exercise.[11-13] It has been hypothesised that EIB develops in response to the cumulative damage to the airways that occurs due to the inhalation of large volumes of dry and often cold air, allergens and pollutants, combined with the mechanical trauma that accompanies the hyperpnoea of exercise.[2] Evidence points to hyperpnoea of dry air as the culprit for causing airway epithelial injury rather than exercise per se.[14]

Data from the Beijing Olympics highlights the role of accumulative damage and years of intense aerobic training. Of those approved to use inhaled Beta-2 Agonists, according to athlete completed questionnaire, almost 37% of athletes diagnosed with asthma developed the condition aged 25 years old or older.[15] Contrary to this, numerous studies have failed to reveal a difference in age between positive and negative groups regarding diagnosis of EIB.[16,17,18]

The clinical diagnosis of EIB may be difficult and unreliable,[19] with the presentation often quite vague, bereft of the classical symptoms of cough, chest tightness, shortness of breath and wheeze.[20] Numerous studies have shown poor correlation between a clinical diagnosis of EIB and that of the current World Anti Doping Agency (WADA) and International Olympic Committee – Medical Commission (IOC–MC) recognised diagnostic tests. Both WADA and the IOC-MC currently recommend the Eucapnic Voluntary Hyperpnoea (EVH) challenge test as the gold standard diagnostic test for EIB. The Mannitol Inhalation Test and Exercise Challenge tests are also considered acceptable by WADA and the IOC-MC. Importantly, a recent meta-analysis revealed neither EVH or Mannitol were sensitive enough to identify EIB in all athletes.[21]

Professional ARF (AFL) requires athletes

with elite endurance capabilities, with players often running in excess of 12-13 km per game.[22] Numerous studies have shown the prevalence rates in similar predominantly winter, elite, team based ball sports to range between 18-35.5% in soccer,[17,18] 19-38%[16, 17] in field hockey and 32% in rugby.[17] Given that AFL players run equivalent or greater distances during games and training than the above mentioned sports[22,23] resulting in both higher and more prolonged rates of hyperpnoea, AFL players would be expected to have higher EIB prevalence rates.

The primary objective of this study was to determine the prevalence of EIB in a cohort of elite AFL players. The secondary objective of the study was to investigate whether age and/or a past history of asthma or EIB correlated with EVH testing.

METHODS

Players were recruited from two professional AFL teams. All senior listed and rookie listed players were considered eligible for enrolment. Subjects were excluded if they had a current upper respiratory tract illness, if they had used short acting or long acting Beta 2 Agonist medications within eight and 48 hours of testing respectively, or if they had used an inhaled corticosteroid within the previous five days. Subjects with a past history of asthma or EIB were not excluded from the study. Subjects were asked to refrain from aerobic exercise and caffeine on the morning of the testing.

Ethics approval for the study was obtained from the Royal Melbourne Hospital Ethics Committee.

Once accepted into the study and having given informed consent, subjects were required to complete a standardised respiratory questionnaire adapted from the European Community Respiratory Health Survey.[24] The survey provided detailed information regarding respiratory symptoms at rest and related to exercise, risk factors, past and family history of asthma/EIB, history of atopy, smoking and asthma/EIB medication use.

Subjects were assessed for EIB using the EVH Bronchial Provocation testing according to the guidelines of Argyros' and co workers.[25] Baseline spirometry was performed prior to the challenge, with the greatest FEV1 of three attempts being recorded and used as a baseline for subsequent calculations. Subjects then completed a six-minute single-stepped EVH protocol,[26] breathing a mixture of gases containing 5% CO2, 21% O2 and balance N2 during the test. FEV1 was measured at 1, 3, 5, 7 and 10 minutes after bronchial provocation. The higher of two readings measured at each time point was utilised for analysis. At the completion of the test, 200 mcg salbutamol was administered via a spacer, and spirometry was repeated five minutes later.

A positive test for EIB was defined as a decrease in FEV1 of greater than or equal to 10% from baseline at one or more time points after bronchial provocation.[27] A threshold of 60% of the predicted maximum voluntary ventilation (MVV) per minute was used as a criterion for an adequate EVH test as per Brummel et al.[28]

All testing was performed during the morning hours between the months of February and early May 2008 (late summer and autumn). Testing was performed at the clubrooms of each club.

Statistical analysis was performed utilising SPSS. Athletes were grouped according to whether they demonstrated a positive or negative EVH challenge. One-Way Analysis of Variance (ANOVA) was used to compare morphological data and baseline spirometry between positive and negative EVH athletes. X2 Analysis was used to evaluate symptoms or history of eczema and atopy and history of EIB/Asthma between EVH positive and negative athletes.

A p value of 0.05 was chosen a priori to reflect statistical significance. Values are presented as mean (SD).

RESULTS

A total of 88 players were eligible to participate in the study. Sixty-five subjects completed all testing. Eighteen players from one team (the reserve grade players) were unable to participate due to training commitments and one subject was excluded due to a respiratory tract infection at the time of testing. Four subjects were excluded as they failed to complete the questionnaire. No subject had used asthma medications within the time frame for exclusion.

Fourteen out of 65 (22%) subjects tested positive for EIB. There was no significant difference between the EVH positive and negative groups for morphological data (Table 1). The EVH positive group was not significantly older than the EVH negative group.

Baseline spirometry results revealed a significantly ($p<0.01$) lower baseline FEV1 in the EVH positive group compared to the EVH negative group (Table 2).
EVH testing data demonstrate a mean

Table 1. Summary of morphological data.

	EVH positive (N=14)	EVH negative (N=51)	Total (N=65)	P value
Height (cm)	185.5 (7.3)	187.9 (7.6)	187.4 (7.5)	0.288
Weight (kg)	85.1 (8.4)	88.5 (9.1)	87.8 (9.0)	0.217
Age (years)	21.9 (3.0)	21.9 (3.4)	21.9 (3.3)	0.994

Table 2. Baseline FEV1 between groups.

	EVH Positive (N=14)	EVH Negative (N=51)	Total (N=65)	P Value
FEV1 (Litres)	4.74 (0.57))	5.38 (0.66))	5.24 (0.69))	0.002

Table 3. Symptoms reported per group.

	No of Athletes Recording at Least 1 Symptom	Cough	Wheeze	Chest Tightness	Shortness of Breath
EVH Positive (N=14)	12 (86%)	12 (86%)	5 (36%)	3 (21%)	8 (57%)
EVH Negative (N=51)	43 (84%)	37 (73%)	26 (51%)	8 (16%)	23 (45%)
Total (N=65)	55 (85%)	49 (75%)	31 (48%)	11 (17%)	31 (48%)

maximum fall in FEV1 of 16(+/- 6)% in the positive group and 5(+/- 2)% in the negative group ($p < 0.001$). The largest decrease occurred at three minutes post cessation of bronchial provocation in both groups.

No subjects in this cohort had a positive reversibility test following bronchodilator inhalation suggestive of underlying asthma.

Ten of 14 (71%) EVH positive subjects recorded a past history of asthma based on clinical diagnosis. In comparison, 16 of 51 (31%) EVH negative subjects recorded a past history of asthma ($\chi 2$ 0.007).

Two of 14 (14%) EVH positive subjects recorded eczema and 9/14 (64%) EVH positive subjects recorded allergies. This compared to 6/51 (12%) for eczema and 20/51 (39%) for allergies in EVH negative subjects (X2 0.095). There was no difference between groups in terms of nasal symptoms or hayfever.

Of those in the positive EVH group, 9/14 were already being treated for asthma/EIB compared with 6/51 in the negative group. Three EVH positive subjects used relievers only (Short Acting Beta Agonists or SABAs), two used a preventer (inhaled corticosteroid or mast cell stabilisers such as sodium cromoglycate) only, and four used both preventer and reliever medication. One subject reported using nebulised SABAs when needed and prior to exercise on a regular basis. In the EVH negative group, two subjects used a reliever only, one used a preventer only and three used both. No subjects in either group reported use of controller medications.

DISCUSSION

This study assessed the prevalence of EIB in a cohort of 65 elite ARF players from two professional ARF teams. Using the current WADA and IOC-MC recognised gold standard bronchial provocation challenge test, the EVH test, the prevalence of EIB in this population was found to be 22% (14/65). This prevalence, although greater than that estimated in the general non-athletic population of 6-15%,[12, 29-31] was similar to other studies of EIB in winter ball sports. Furthermore, although the study was performed in athletes involved in a predominantly winter-based sport, the assessment phase of the study was performed late Summer and early Autumn, at a time when the ambient air temperature is warm, average 21.8 degrees celcius, hence negating any potential effect of the cold dry air on the airway mucosa.

However, during the late summer/early autumn months we were assessing the players at a time of and immediately following peak endurance training during AFL "preseason". The preseason phase of AFL training commences in early November and is the period of the largest cardiovascular and heaviest training loads for the players. Traditionally it involves large volumes of running, both aerobic and anaerobic, combined with match simulation, far more so than during peak playing season, which begins late March/early April. This is a prolonged period of repetitive mechanical trauma to the airways from training related hyperpnoea. It is also at a time following which the athlete's airways have been exposed to high allergen loads, combined with dust during the spring and summer months.[32]

Only one study has assessed the prevalence of EIB in an amateur ARF team; unfortunately while not all of the team was screened, 50% of 32 subjects were shown to have a positive EVH test.[33] However, subjects were recruited on a volunteer basis, possibly resulting in a higher number of subjects with underlying subacute respiratory difficulties partaking in the study. Therefore, the prevalence may have been falsely elevated. Players with a past history of EIB or asthma were significantly more likely to record a positive EVH test than those without.

Fourteen out of 65 subjects in this study had a past history of asthma or EIB; however only 71% (10/14) of this group had a positive EVH challenge test. This highlights that approximately a third of the athletes with a previous clinical diagnosis of asthma/EIB tested EVH negative. EVH testing has been shown in a recent meta analysis to have variable sensitivity[21] and a proportion of these results may have been false negatives. Alternatively, a proportion of subjects may have been misdiagnosed with asthma/EIB based on clinical features alone. Finally, given EIB is a fluctuating condition, a number of subjects may have been in a state of remission at the time of testing.

The EVH positive group had significantly lower baseline FEV1 measurements than those in the negative group, despite not having a positive bronchodilator response. This suggests an underlying irritability of the airways. Dickinson et al in a study of elite British athletes,[17] also found a lower baseline FEV1 in those elite British athletes with a positive EVH challenge, regardless of whether the athlete had a past history of asthma/EIB.

Sixty-four per cent (9/14) of the participants in this study with a positive EVH challenge test were regularly using medications for asthma/EIB. Therefore, 36% of EVH positive athletes were not using medications of any kind, suggesting that they may have been regularly training and competing with airways obstruction, and consequently performing at a suboptimal level.

In the EVH positive group, only 67% (6/9) of athletes taking regular medications were taking daily prophylactic medications, the remainder using inhaled beta-2 agonists on an as required basis, depending on a combination of symptoms and/or perceived possible need.

Of the EVH negative group, 12% (6/51) were taking regular medications for asthma/EIB,

67% of whom were taking daily prophylactic medications. This finding is consistent with previous studies performed in sporting populations, demonstrating incongruity between EIB testing results and medication use.[5,17] The impact of under-treatment on individual performance has been suggested in a study performed by Holzer et al.[23] This study revealed an increased VO2 max following a six week regime of inhaled corticosteroid and pre-exercise mast cell stabiliser use in those testing EVH positive compared to controls. The treatment group also had significant improvements in FEV1 at follow up EVH testing.

The current study highlights the requirement for formal screening and diagnosis of EIB in a professional AFL team. This study revealed four asymptomatic players with no history of asthma/EIB on an AFL team list who tested EVH positive. Treatment was initiated to limit the airways narrowing secondary to exercise. Consideration should be given to biannual assessment both during preseason when both the training and allergenic loads are highest, and during the winter season, when the impact of the cold air is at its peak. This may lead to increased positive EVH test results and prompt treatment of EIB in those subjects. That would ideally improve player performance. Granted, further studies are still needed to assess the impact of EIB treatment on sporting performance.

An accurate diagnosis of EIB and assessment of lung function is important to ensure that the athlete maintains optimum lung function through appropriate use of asthma medications. In this study some EVH positive subjects were using only SABAs to control their EIB. Long term daily use of SABAs have been associated with beta receptor down regulation, tolerance and a decrease in efficacy.[34,35] Consideration should be given to regular use of an inhaled corticosteroid for those with EIB.[36] A mast cell stabiliser could be used as an adjunctive preventer. This decreases reliance on SABAs, increasing their efficacy as a reliever if they are used less frequently.

The limitations to this study include;

- Limited sample size.
- Omission of 23 of a potential 88 subjects. As this was mainly due to one team's unavailability due to training commitments we believe this was unlikely to bias our results significantly.
- Timing of testing during warmer seasons may have decreased prevalence rates.

CONCLUSIONS

This study demonstrated that 14 out of 65 (22%) elite ARF players tested positive for EIB on EVH testing. This result is similar to other elite winter team-based endurance sports.

Importantly, either a past history of EIB/asthma, or lower than expected baseline FEV1 on spirometry should alert the physician to the increased likelihood of EIB. Consideration should be given to testing all elite AFL players for EIB/asthma with bronchial provocation testing. Clinicians should recognise that bronchial provocation testing is not without its limitations, and occasionally different bronchial provocation tests may need to be used, or different diagnoses considered.

PRACTICAL IMPLICATIONS

- Clinicians should not rely on clinical features of Exercise-Induced Bronchoconstriction/Asthma to diagnose the condition.
- Consider Exercise-Induced Bronchoconstriction in elite Australian Rules Footballers with a past history of Exercise-Induced Bronchoconstriction/Asthma or a lower than expected baseline FEV1 on spirometry.
- Eucapnic Voluntary Hyperpnoea bronchial provocation testing is the gold standard for diagnosing Exercise-Induced Bronchoconstriction.
- Consideration should be given to testing all elite Australian Rules Footballers for Exercise-Induced Bronchoconstriction using Eucapnic Voluntary Hyperpnoea testing.

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Athletes with limb deficiency

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INTRODUCTION

Disabled sport continues to grow in popularity and profile, with the Paralympic Games now attracting major media coverage and sponsorship, and grassroots participation increasing. Paralympic athletes experience many of the same injuries and illnesses as able-bodied athletes, however they are potentially vulnerable to additional and unique problems. It is recognised that many sports medicine providers will encounter athletes with disabilities relatively infrequently, and thus the aim of this and subsequent articles is to summarise key injury and illness considerations when consulting athletes with a disability, in order to facilitate successful and rewarding assessment and treatment. The first of these articles considers medical issues for athletes with limb deficiency.

True or False?

1 The most common cause of limb deficiency in athletes is amputation due a metabolic condition such as diabetes or peripheral vascular disease.

2 Verrucus hyperplasia (Figure 1) occurring in the stump of an amputee is thought to be related to an underlying viral infection.

3 Athletes with lower limb deficiency have an increased incidence of osteoarthritis in the knee of the contralateral limb.

MEDICAL CONSIDERATIONS FOR ATHLETES WITH LIMB DEFICIENCY

Limb deficiency in athletes is most commonly either congenital or post-traumatic, and more occasionally secondary to a metabolic condition (e.g. diabetes or peripheral vascular disease) or amputation related to a neoplasm or severe infectious disease. The level of the amputation tends to correlate closely with the degree of subsequent dysfunction and disability, and the prosthesis that will be most suitable.

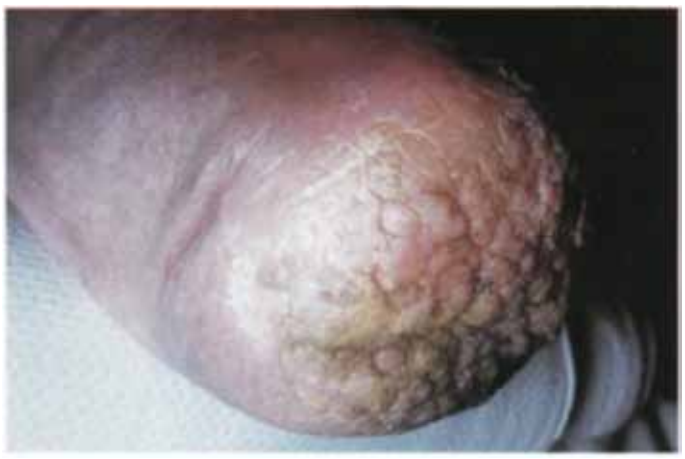

Figure 1. Verrus hyperplasia occurring at the end of a stump. (from Levy SW. Skin problems of the amputee. In, Bowker HK, Michael JW (eds): Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1992.)

Athletes with limb deficiency are classified depending on the functional impact of their disability on the particular sport and event.

A specific and important medical issue for the athlete with limb deficiency is the condition of the skin overlying the stump(s). Proper prosthetic fit is crucial to good stump skin care and involvement of a skilled prosthetist, preferably well versed in the physical demands of the athlete, is important. In New Zealand there are a few prosthetists with some interest and experience in dealing with competitive athletes, and each case requires a unique assessment and prescription. In sports where a prosthesis is worn while training and competing, this will usually be different to what they wear during activities of daily living. Athletes should perform regular skin checks of their stump, and seek early medical assessment if there are any concerns, such as irritation, early maceration or frank skin breakdown. The stump skin should be cleaned regularly and kept dry to avoid problems related to excessive and/or prolonged perspiration. Contact dermatitis is common on or around the stump, and as in other situations the key is identification and avoidance of the irritant (including potentially a period of time not wearing their prosthesis) along with usually brief application of a topical steroid cream. In addition, any mechanical factors that may be contributing should also be addressed. Epidermoid cysts (fluid-filled sacs in the skin) may form surrounding the edge of the

prosthesis, and are caused by a blockage of a hair follicle by sloughed skin cells. They may become secondarily infected. Treatment is based on stabilising any infection with topical or systemic antibiotic therapy and addressing the prosthetic fit issue. Even in the absence of epidermoid cysts the base or anywhere on the stump is prone to bacterial or fungal infections, and treatment (usually topical) should be initiated early and relatively aggressively. Verrucus hyperplasia (Figure 1) is thought to be caused by a prosthesis that is too tight (the 'choke syndrome'). This then causes a suction effect in the distal stump, pooling of blood and subsequent over distension and injury to the blood vessels, and associated overlying skin changes. There may be secondary infection (usually bacterial) but despite the name this is primarily a mechanical and vascular issue rather than viral in aetiology.

There are a number of other non-dermatologic and less common medical considerations in the athlete with limb deficiency. Thermoregulation is potentially impaired, and related to the loss of body surface area.[3] Almost all individuals who have undergone an amputation experience residual sensations from their missing ("phantom") limb/s, and in some this can unfortunately include significant pain. This has both peripheral and central contributors, and while neuropathic pain medication is the most effective of the recognised treatment options, this is renowned as a difficult condition to treat.[2] A clearer local cause of post-amputation neuralgic pain is a neuroma, where the severed nerve ending balls up and in some cases continues to send pain signals. Treatment options include biomechanical adjustments, pharmacologic treatment, and various surgical measures, but early involvement of a neurosurgeon is appropriate. There is a documented increased risk of overuse injury in the contralateral lower limb including knee osteoarthritis,[1] and clearly participation in formal weightbearing athletic competition may increase this risk. A final consideration is the potential for recurrence of any neoplastic disease that precipitated an amputation, either locally or elsewhere, and treatment providers should maintain an appropriate index of suspicion for this.

Thus while athletes with limb deficiency have for the most part the same issues as their able-bodied counterparts, there are a few specific considerations that those involved in their care should be aware of. The key to maintaining good stump health is a well-fitting prosthesis, regular skin care and monitoring, and early treatment of any potential issues. Neurogenic stump pain can have local source and/or be referred. There is an alteration in the distribution of load that may predispose to early osteoarthritis, and potential recurrence of any disease that precipitated an amputation should be kept in mind. Being aware of the considerations above will assist the maintenance of optimal health of our athletes with disabilities and contribute to medal-winning performances on the world stage.

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Holt-Oram syndrome: An incidental diagnosis at athlete screening

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ABSTRACT

The pre-participation screening of athletes plays an important role, not only in the identification of health problems that may potentially compromise sporting performance, but the identification of general health issues which may impact the individual and possibly their kin. A case report of a sportsman presenting with abnormal hand morphology and a history of atrial septal defect repair is presented. A diagnosis of Holt-Oram syndrome was made and further genetic counselling offered. Holt-Oram syndrome is a rare autosomal dominant condition causing variable osseous malformation of the upper limb and structural and/or electrical cardiac abnormalities. This case describes the interesting pathology often associated with triphalangeal thumbs and highlights the need for every medical anomaly to be taken into consideration while performing a pre-participation screen, in order that underlying conditions may possibly be uncovered and managed accordingly.

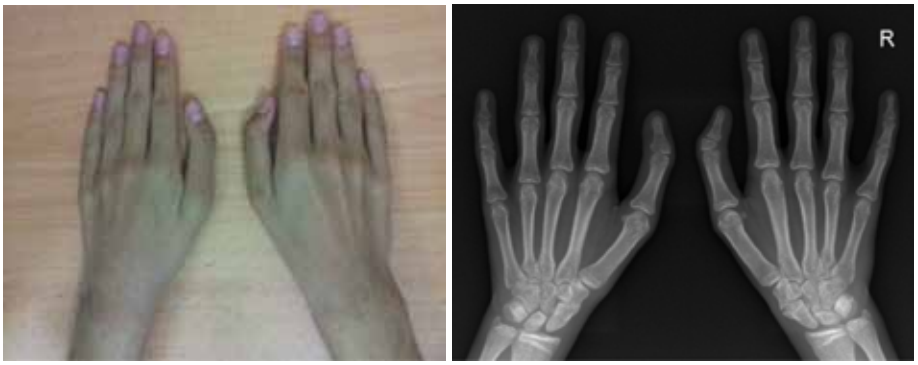
INTRODUCTION

Pre-participation athlete screening is an important tool in ensuring the optimal health of the athlete. While variations of normal exist in all parts of the body and are commonly observed, knowing when a finding is clinically relevant, or when to pursue it further, is often challenging. This case highlights a rare but important association between osseous abnormalities of the upper limb and congenital heart defects.

CASE REPORT

A 16 year-old right-handed Qatari table tennis player presented with pain on the radial aspect of his right wrist following minor trauma sustained in a recent road traffic accident. There was minimal swelling but he continued to experience pain with movement, lifting and when playing both forehand and backhand shots.

On examination, there was an obvious "finger-like thumb" on both hands (Figure 1a). The hands appeared elongated and slim but no other obvious structural abnormality was noted. There was mild pain with both wrist extension and flexion and tenderness to palpation in the anatomical snuffbox, in the region of the base of the first metacarpal and also with palpation of the scaphoid tubercle.



Figures 1a and 1b:
The finger-like appearance of a triphalangeal thumb

The patient had a previous history of a large atrio-septal defect (ASD), which was discovered at a routine pre-participation screening at the age of 12. This was surgically repaired with a patch shortly afterwards and he had an uncomplicated outcome. Over the 2 years following his cardiac surgery, resting and exercise ECG revealed no abnormality and serial echocardiography showed good cardiac function, with an intact septal patch. He had no other medical issues, specifically no renal problems or anaemia. On further questioning, his brother had also undergone a septal defect repair at the age of 4 and had a unilateral "finger-like thumb". Despite specific questioning of the patient and his father, there was no other reported family history of either cardiac or upper limb abnormality. He had 5 unaffected siblings and the aforementioned brother had 2 unaffected young children.

Plain radiographs revealed an incomplete synostosis between the scaphoid and trapezium, as well as an accessory phalanx in the thumb and an elongated first metacarpal (Figure 1b). It was suspected that the patient had suffered a traumatic sprain of this synostosis and he was placed in a supportive brace and reviewed 2 weeks later. At his return visit, his painful symptoms had settled somewhat and additional radiographs of the unaffected left hand were taken, revealing a complete synostosis of the scaphoid and trapezium but no additional phalangeal bones.

The appearance of a triphalangeal thumb, bilateral carpal bone abnormalities and septal

defects in both the patient and his sibling raised the possibility of a range of underlying disorders (Table 1).

Spontaneous and hereditary cases of isolated triphalangeal thumb may occur but there are also many conditions associated with its presentation:

- Syndactyly
- Radial polydactyly
- Lobsterclaw hand
- Triphalangeal thumb-polysyndactyly syndrome
- Klinefelter's (one case described in the literature)[1]
- Fanconi's pancytopaenia

The patient was given a wrist brace and his symptoms gradually resolved over 10 weeks with relative rest. A clinical diagnosis of Holt-Oram syndrome was made, and its possible implications were discussed with the patient and his father. Further genetic investigation and formal counselling was advised but declined.

DISCUSSION

Holt-Oram syndrome is a condition involving osseous malformation of the upper limb and cardiac abnormalities, first described in 1960.[2] Occurring in 1:100,000 live births, it exhibits autosomal dominant inheritance, with complete penetrance but variable expressivity. Spontaneous mutations account for about 40% of cases. Gene carriers have been described lacking either cardiovascular or skeletal findings but not without both.[3] There may, however, also be transmission through a phenotypically normal parent, thought to be due to parental mutation resulting in gonadal mosaicism.[3,4]

Proposed and validated diagnostic criteria are:[5,6]

- An upper-limb malformation involving the carpal bones and, variably, the radial and/or thenar bones
- A personal and/or family history of congenital heart malformation – atrial or ventricular septal defects, anomalous pulmonary venous drainage, persistent left superior vena cava, coronary artery abnormalities
- Possible cardiac conduction disease - increased P-R interval, sinus bradycardia, nodal escape and atrial fibrillation.[2]

Table 1: Differential diagnosis of combined osseous hand malformation and cardiac defects.

Condition	Characteristics
Holt-Oram syndrome	Variable osseous malformation of the upper limb and structural and/or electrical cardiac abnormalities
VACTERL association	A variable combination of vertebral, anal, cardiac, trachea-oesophageal fistula, renal and limb defects.
Aase syndrome	Anemia, decreased leucocytes, bone and joint deformities (including triphalangeal thumbs), narrow shoulders, a cleft palate and deformed ears
Diamond-Blackfan anaemia	Anaemia and low reticulocytes; variable craniofacial malformations, thumb or upper limb abnormalities, cardiac defects, urogenital malformations and a cleft palate.
Townes-Brocks syndrome	Variable external ear abnormalities, anorectal malformations, renal, cardiac, hand and foot abnormalities

More than 70% of individuals who meet these criteria have an identifiable mutation in the "T box" transcription factor gene (TBX5) at chromosome locus 12q24.[5] This gene is responsible for activating several genes, who's proteins are involved with embryonic formation of both the upper limbs and the cardiac septum and electrical conducting system.

Skeletal involvement is typically bilateral, with the left side more severely affected. Familial studies demonstrate that carpal abnormalities, frequently involving the scaphoid, are more common than triphalangeal thumbs. There is a wide variation in the severity of both skeletal and cardiac malformations and no association between the relative severity of their manifestations within any individual.[3] Muscular weakness and hypoplasia of the muscles inserting onto the affected upper limb bones has also been described.[7] In one large study, 38% of individuals required corrective cardiac surgery and 3 out of 55 (5%) had symptomatic complete heart block requiring a pacemaker.[5,6,8]

The mode of inheritance in this case is intriguing, given the apparent lack of affected individuals in the previous two generations. Spontaneous genetic mutation, leading to gonadal mosaicism in one of his parents may explain the fact that only 1 of his 6 siblings was also affected. As a predominantly autosomal dominant condition with a significant cardiac outcome, a formal diagnosis and further information regarding the mode of inheritance of his condition would be important for this athlete's future family. Likewise, a positive diagnosis, and its implications for the possible future development of cardiac arrhythmias, would reinforce the need to continue to attend for regular ECG monitoring.

This athlete underwent pre-participation screening, which facilitated the repair of his ASD. Unfortunately, failure to carefully evaluate his musculoskeletal system resulted in the incomplete elucidation of his true condition. Careful and comprehensive examination, with appropriate follow up of variance from normal, ensures the best outcome for athletes. The authors recommend clinicians having a high index of suspicion for structural or electrical cardiac abnormalities following a finding of thumb or carpal bone abnormalities.

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Movement screening protocols: Rationale versus evidence

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INTRODUCTION

Over recent years movement screening protocols have become popular and their use is widespread in the realms of sports medicine and athletic performance. The rationale for this movement-based approach to screening is intuitively appealing and certain movement screening test batteries, most notably the Functional Movement Screen (FMS) ™, have been highly commercially successful. Closer to home, the movement competency screen (MCS) has achieved prominent use in elite sport, particularly within high performance sport organisations in New Zealand. Indeed, the MCS has been adopted as a critical part of the rehabilitation model within the national organisation High Performance Sport New Zealand.[22] An illustration of the credence placed upon the MCS protocol is that the rehabilitation model as originally presented described a process whereby if an athlete achieves a good pass mark on their MCS evaluation no further investigation or other musculoskeletal assessment was deemed to warranted.[21] Moreover, the results from the MCS are also employed as the basis for programming and progression of training prescribed to athletes.

The Rationale for Movement Screening Assessment Batteries

The great appeal of movement screening batteries is that they feature fundamental movements that are characteristic of the majority of sports and athletic disciplines. Assessing the athlete in this way is postulated to give an indication of function and motor control during 'athletic movements'. Indeed it is inferred that an athlete's motor performance as demonstrated during these composite movement tasks will also provide a representation of their function during more complex athletic activities and sports-related movements – such as running and changing direction.[14]

By extension, the athlete's overall score on the particular movement screening battery (e.g. FMS™ or MCS) is purported to relate to not only their overall risk of injury but also their performance capabilities. In addition to the composite score, the athlete's score on a particular item in the screening battery is also deemed to provide an indication of their intrinsic risk for injury to specific regions of the body – for example, the assessments of shoulder mobility are taken to indicate relative risk for shoulder injury. Essentially, any deficit in mobility or movement 'dysfunction' evident during a particular part of the evaluation process is interpreted as evidence of a musculoskeletal or motor control issue. It is argued that any aberrant movement patterns identified during the particular task are likely to be a consequence of previous injury or otherwise indicative of an underlying fault that will predispose them to injury.[3,4]

The athlete's overall score and their score on individual items in the screening battery are in turn used to guide training prescription, in an effort to address the particular faults indicated by the athlete's screening profile. For example, within New Zealand High Performance Sport systems athletes' MCS scores are used as the basis for determining their initial training and deciding progression.[22] For example, the Movement Competency 100 Protocol © that was presented in the original PhD thesis[14] is employed as a starting point for carded athletes' physical preparation. Similarly, the 'movement competency paradigm' that has been derived from the MCS protocol that describes a step-wise progression from assisted to various conditions is employed as a template for training progression.[14]

Is there evidence to support the theorised link with injury risk and performance?

Due to the intuitive appeal of these methods, and the theoretical rationale presented, the uptake of movement screening protocols has been rapid and widespread. To some extent this is also testimony to the commercial value of such approaches and the success of marketing and promotion – for instance, the FMS™ now includes commercially available official test apparatus kits, training courses and tester certifications. There has been somewhat of a lag with regards to scientific studies to assess the reliability of these assessment procedures and investigate the postulated relationships with injury and performance.[10] However, recently there have been a number of studies and the emerging data has prompted increasing questions about the reliability, validity and sensitivity of these procedures. This is clearly a concern given the widespread use of these protocols in elite sport.

As the best known and most commercially successful, the FMS™ has been the focus for many of these investigations. One early investigation reported that individuals' scores on each the FMS™ component tasks showed only weak statistical relationships with selected measures of athletic performance investigated – the highest of the r values that reached significance was only 0.46.[19] Moreover, there was no significant correlation between FMS™ scores and the measures of core stability employed in this study. Another study investigated the relationship between FMS™ measures, a variety of standard athletic performance assessments (vertical jump, 20m sprint and T-test for change of direction) and a sports skill task in collegiate athletes (golfers).[20] This study failed to find any significant statistical relationships with any of the performance measures examined for either the subjects' combined FMS™ scores or their scores on the individual tests in the assess battery. Furthermore, the preliminary evidence suggests that FMS™ scores are also

not able to discriminate elite versus sub-elite athletes in field sports.[9]

To date there are only two studies that indicate any association between scores on the FMS™ and injury incidence. One study featured a squad of male professional American football players,[13] whereas the other comprised a mixed group of female student athletes representing the same college in soccer, volleyball and basketball.[2] Both of these studies employed a somewhat arbitrary cut-off score of 14 for their statistical analyses – so that a composite FMS™ score ≤14 indicated an apparent significant increase in the relative number of injuries suffered in the season that followed. To put this in context, a score of 14 out of 21 indicates the athlete scored on average 2 out of 3 for each test. When we consider normative values a score in this range would be common for many participants – for example, average composite FMS™ score reported for a young active population is 15.7[23] and similarly average values reported for a sample of team sports players was 15.56.[9]

In contrast to the somewhat positive associations reported in the previous two studies, preliminary data presented at this year's ACSM conference reported no statistical relationship between preseason FMS™ scores and injury during the following season in a cohort of 170 collegiate athletes.[25] There was no association between athletes' composite FMS™ scores and injury, even when the same cut-off score of 14 as used in the previously cited studies was employed. Of the individual components of the FMS™, only the in-line lunge task showed any association with injury incidence.[25]

Based upon the very limited data available, the relationship between scores on popular movement screen batteries such as the FMS™ and injury risk remains highly equivocal. Furthermore, to date there is no relationship established with athletic performance. It should be noted that there are no such studies in the peer-reviewed sports medicine or sports science literature investigating the relationship between scores on the MCS and performance or injury .

Issues of Implementation

A recent study highlighted a major issue with respect to how screening protocols are conducted in the field[11] – specifically, how much information is provided to the athlete with respect to what constitutes 'correct' form for the particular movement task. One school of thought is that the participant should be given minimal direction; the rationale being that they are more likely to demonstrate their natural movement patterns during the screening task. Whilst this seems sound in theory, in reality the screening process is by its nature quite unnatural. Moreover, conducting screening in this way so that very little direction or feedback is provided is likely to increase instances of false negatives. If the athlete is able to immediately correct their movement technique once they are provided with information on what the tester is looking for then it seems very unlikely that the movement flaws registered in fact represent real dysfunction. In this way, the screening results essentially just reflect how the athlete chose to perform the given movement task on that particular occasion, rather than representing a true indication of their ability to execute the movement properly.[10]

Indeed this was the major finding of the recent investigation by Frost and colleagues. The study participants, a group of firefighters, first undertook the FMS™ protocol with minimal instruction. The participants were then verbally provided with information on the scoring criteria. Immediately afterwards the participants performed each of the screening tasks a second time, and their scores were significantly improved compared to their initial attempt undertaken only minutes before.[11]

Reliability and Sensitivity of Movement Screening Protocols

The reliability scores reported in the various investigations employing the FMS protocol to date has varied quite widely, both within (intra-rater test-retest reliability) and between raters. Furthermore, intra- and inter-rater reliability values also differ according to the particular screening task in the protocol. For instance, the shoulder mobility assessment task generally produces highly repeatable scores, whereas the reliability of the hurdle test component of the FMS protocol reported very poor reliability.[24] Participants' scores over time may also not be consistent. One study reported that the scores of 85% of the control group participants who performed no training during the 12-week study period changed from pre- to post-testing; many participants showed 'spontaneous' improvements, whereas for other participants their post-testing scores were in fact worse.[10]

Equivalent reliability data for the MCS are currently lacking , despite the widespread use of this protocol in high performance sport within New Zealand. Essentially, the only values available are those reported in an unpublished doctoral thesis and conference presentations. The original reliability pilot study included only 3 study participants, who were categorised as 'good, 'moderate' or 'poor', respectively, based upon a preliminary assessment of their MCS performance.[14] Even so the average kappa values indicating inter-rater reliability for the tasks in the MCS battery was 0.79. In addition, the agreement between raters was substantially worse for the two subjects who were rated 'good' and 'moderate'.[14] Other data presented as part of a wider netball screening investigation reported inter-rater reliability (2 independent raters and a sample of 20 participants) for the composite MCS score was 'fair' (intra-class correlation coefficient, ICC = 0.77).[22] However, the values for individual movements tasks within the MCS battery varied between 'fair' for the lunge and twist, and poor (ICC < 0.69) for the remaining elements.

Issues of Over-Interpretation

One of the most questionable assumptions regarding movement screening protocols is that the fundamental movement tasks involved are indicative of athletic activities, such as jumping, running and changing direction. Neuromuscular control is highly specific to the nature and constraints of the task. For instance, it has been identified that 'proximal' neuromuscular control of the lower limb not only depends on the gender of the athlete, but also varies in task-dependent manner.[17] Furthermore, movement patterns can also differ for the same task according to the environment and conditions involved. For example, it has been observed that the kinematics of a vertical jump task are altered with the addition of a target to aim for.[8] Similarly, when a stationary dummy defender is added to the movement task environment this is shown to alter the movement mechanics employed

when performing the same cutting change of direction task.[16]

Based upon this information, clearly the movement screen tasks employed in screening batteries such as the FMS or MCS cannot be used as an analogue for other athletic activities. Simply, if one wishes to assess an athlete's movement capabilities during an athletic activity then this requires the screening must involve observing the athlete performing that specific activity. Furthermore, it is unsafe to assume that improvements in neuromuscular control exhibited during a stationary movement task following standard corrective neuromuscular training will transfer to a reactive athletic task, as the data presented in the literature to date indicate this is not the case.[7]

Practical Recommendations for the Use of Movement Screens in the Pre-participation Examination

As described, there are major concerns with many of the movement screening batteries and associated practice currently employed. . However, the movement screening approach does have utility as part of a wider pre-participation examination. Whilst popular movement screening batteries such as the FMS and MCS clearly have major issues, there are nonetheless individual movement screens that have been validated in the literature. One such example is the single-leg squat described by Crossley and colleagues,[5] which was shown to be indicative of measured differences in hip muscle activation and sensitive to differences in selected measures of hip and trunk muscle function between subjects. Tests of dynamic balance, notably the star excursion balance test, have also been shown to have predictive value with regards to lower limb injury risk.[6]

Rather than abandoning movement screening entirely, the practitioner might therefore employ a broadly similar approach, but carefully select each individual movement screens based on their merit, as well as what is appropriate for the sport. In order to help discriminate real movement dysfunction the athlete should also be provided with full instruction on what constitutes proper form for the movement task. Appropriate rating schemes and employing clearly defined assessment criteria can also improve consistency and reliability. For instance, a 'dichotomous' rating system whereby the screening score sheet includes clearly delineated criteria for each segment of the movement for each screening task may assist intra-rater reliability.[26] In general, the screening should also be performed by the same tester(s) on each test occasion to negate any effect of inter-rater reliability. Finally, the movement screen should ideally be jointly scored by both a physiotherapist and the strength and conditioning specialist working with the athlete,[12] and practitioners might also consider video recording to capture all relevant information.

The drop jump test originally employed in a research setting has likewise recently been adapted for use in a clinical and applied setting, requiring only a digital camera and a standard laptop or desktop personal computer.[18] There are a number of software tools that are available to download free of charge for capturing and analysing the digital video footage from movement-based tests of this type. Alternatively, a more task-relevant approach would be to simply employ video footage recorded during performance testing (vertical and horizontal jump tests, speed testing, change of direction assessments etc.) for the purposes of movement screening. In this way, the athlete's movement patterns can be directly captured and assessed as they perform a true athletic movement task, as opposed to the rather artificial process of evaluating movement patterns during a screening task such as a drop jump.

In additional to assessing movement quality during performance testing, when unilateral versions of jump assessments are employed the test scores themselves (e.g. jump height or distance) can also provide relevant information. For instance, there are preliminary data to suggest that side to side differences on a unilateral performance test (single-leg hop for distance) may be indicative of increased risk of lower limb injury.[1]

CONCLUSION/SUMMARY

In their clinical commentary published in 2006 Lorrie Maffey and Carolyn Emery made the prophetic statement that "an ideal preparticipation examination system applicable to all sports does not exist".[15] It is therefore perhaps unreasonable to expect that even the most carefully devised movement screening battery will be applicable or useful for all athletes.

As presented in this article, there are methodological issues and questions of validity and sensitivity with respect to screening batteries such the FMS and MCS.

Clearly it is very important that practitioners who work with athletes are aware of these issues.

There are a number of methodological considerations when undertaking movement screening and the information yielded from these assessments should be interpreted with care and in context. It is also evident that movement based screening should not be viewed as a replacement for a thorough clinical musculoskeletal assessment. Similarly, in order to get a true picture of the movement characteristics and capabilities of the athlete, the profiling process should ideally also include a specific biomechanical assessment appropriate to the sport.

As a general approach the concept of undertaking qualitative assessment to evaluate mobility, stability and movement based upon specified criteria as part of the athletes' pre-participation examination clearly has merit. If the practitioner is willing to invest the time to carefully select movement screens from the literature that are appropriate to the sport and reported to be reliable and valid in terms of predicting injury risk then this can yield potentially useful information. Likewise it is important to be mindful that the manner in which screening is conducted can influence results, so this should be considered and appropriate measures taken. Finally, the results of movement screening should be interpreted in the context of the wider information provided by the musculoskeletal examination and other aspects of the pre-participation assessment.

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Comment

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The author of this paper has raised some fundamental issues and challenges regarding the ability of movement screening as a concept to predict or prevent injury. We would agree that this is a challenging area but one that should not be shied away from because the evidence might not yet be available. There are a number of comments we would like to make to clarify our position on some the key issues raised.

Screening is not a new concept and the medical profession has been screening patients for many years and then making further management decisions based on the screening outcome. Within High Performance Sport New Zealand we screen to gain base line data regarding each athlete, utilise this information to identify potential injury risk and guide a proactive approach. Our aim is to minimise injury risk where we can and keep the athletes training and competing on the world stage. A key concept is that a screen does not provide the underlying reason for the finding; it just starts the process of identification of a potential problem or underlying risk factor. The taking of blood pressure is an example where an abnormal finding does not provide the diagnosis for the altered values, just the start of further evaluation. Movement screening may be viewed in the same way; essentially an inverted pyramid with some key simple yet reproducible tests at the bottom of the pyramid and then proceeding to more complex tests as required at the top. As highlighted, in order for this to be effective the screening tests must be consistent in their application (reliable) and valid, as well as preferably able to be performed with little or minimal

(reliable) and valid, as well as preferably able to be performed with little or minimal training or equipment. Both the Functional Movement Screen (FMS)[1] and the Movement Competency Screen (MCS)[2] have achieved this. Both Vanweerd[3] and Schneiders et a[l4] have demonstrated that novice and experienced raters can perform the tests consistently and reliably. In our experience, the simplicity of the tests has facilitated communication between rehabilitation providers and strength and conditioning team members at a high performance sport level without the need for professional jargon. This has also created a mutual and common understanding about movement patterning, loading of the pattern in the gym and appropriate progressions.

Importantly, the current physiotherapy screening process at HPSNZ involves not just the MCS. HPSNZ uses a Dynamic MCS (DMCS) which takes simple MCS movement patterns and adds a dynamic component (for example: double foot jump and land, broad jump and land, single leg land). All elements of the DMCS are videoed and shared with the athlete and key support staff, assessed for issues or concerns relevant to performance or potential injury risk. The information is then used to guide future proactive therapy focus, stored in an accessible format, and referred back to as necessary (subsequent DMCS, post treatment, or post injury). In addition, the HPSNZ rehabilitation team has incorporated 16 other screening evaluation tools (the Musculo Skeletal screen – MSK) that the athlete progresses to once the initial DMCS screen findings are known. These 16 evaluation tools are a compilation of reliable and validated musculoskeletal tests of movement impairment, control, muscle endurance and range of motion. Examples of reliable tests are the active knee extension test,[5] trunk endurance tests of McGill et al[6] and the ankle dorsiflexion test[7] that has been shown to be a valid predictor of lower limb injury.[8] Considerable debate and trialling of these 16 tools was undertaken by the HPSNZ rehabilitation team before the agreed battery was implemented. It is acknowledged that movement control is complex so it is important to start with some simple measures of key movement patterns that are easily assessed. Further refinement per individual athlete beyond the next 16 test MSK is also undertaken as required and we acknowledge the importance of detailed and individualised musculoskeletal screening – the benefits of which must be balanced against the realities of time and cost.

At HPSNZ we are aiming to identify and minimise injury risk as well as maximising the response to training. We are acutely aware we are not going to prevent every injury of a high performing athlete. However, we are also aware that if we have some key base line information about an athlete via the DMCS and MSK, combined with an intimate knowledge of the demands of the sport, then we have a foundation for proactive facilitation of musculoskeletal function. In addition, the ability to compare pre and post injury function will assist our injury management and rehabilitation.

Subsequently, an individualised programme for minimising injury risk (MIR) plan can be implemented. The process generates informed discussion and is a scaffold for the rehabilitation and strength and conditioning teams to build and optimise athlete training programmes. Ideally we would like to think that this approach may also have a benefit on actual performance

In HPSNZ, this data is also being coupled with collecting injury incidence and training exposure data. In this sense the overall methodology is to collect the information per athlete and sport and then explore the relationship between injuries and the screening. Whilst there are still mixed results from studies investigating movement screening, does this mean we should abandon this methodology? It is intuitively not good injury prevention policy to just let injuries occur in high performance sport without seeking a greater understanding of why. There is no perfect or gold standard test, we agree, but the current staged approach is pragmatic, scientifically orientated and ethically appropriate.

It is well recognised that there is much to do in the field of musculoskeletal screening of elite athletes. While we must be vigilant in our approach we acknowledge that with a small pool of world class athletes in New Zealand we must aim to minimise injury risk and keep our athletes training and competing. We need to be proactive and innovative in our approach but also constantly reviewing our processes. It is a pretty exciting space to be working in!!

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Cardiovascular positional anomalies and the competitive athlete

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BACKGROUND

A 16-year old Arabic male trainee football referee presented at our institution for pre-participation cardiovascular screening. The athlete did not have a family history of cardiovascular disease or sudden cardiac death in any first degree relative younger than 35-years of age. However, the athlete stated his heart was 'abnormally located'. Auscultation confirmed the athlete's vocal description of heart location, with a heart apex on the lower right of the chest wall (i.e. dextrocardia). No murmur was present with normal supine resting blood pressure and peripheral pulses. Hepatic dullness was located at the left hypochondria. Physical examination demonstrated some marfanoid features. These included wrist and thumb signs, plain pes planus, skin striae and scoliosis (global score < 7).[1]

12-Lead Electrocardiography (ECG): A resting 12-Lead ECG using standard leads positions (Figure 1) demonstrated sinus bradycardia (48 bpm), together with right axis deviation (RAD), negative P waves in I, aVL and inferior leads (II, III and aVF), with a positive P wave in aVR. There was reversal of the QRS complex in precordial leads: R tallest in lead V1, progressively diminishing to V6 without transition. Finally, T wave inversion was noted in 1, aVL and all precordials.

A second ECG was performed (Figure 2) with precordial leads positions transferred to the right chest wall, with limb leads remaining in the traditional location. Whilst

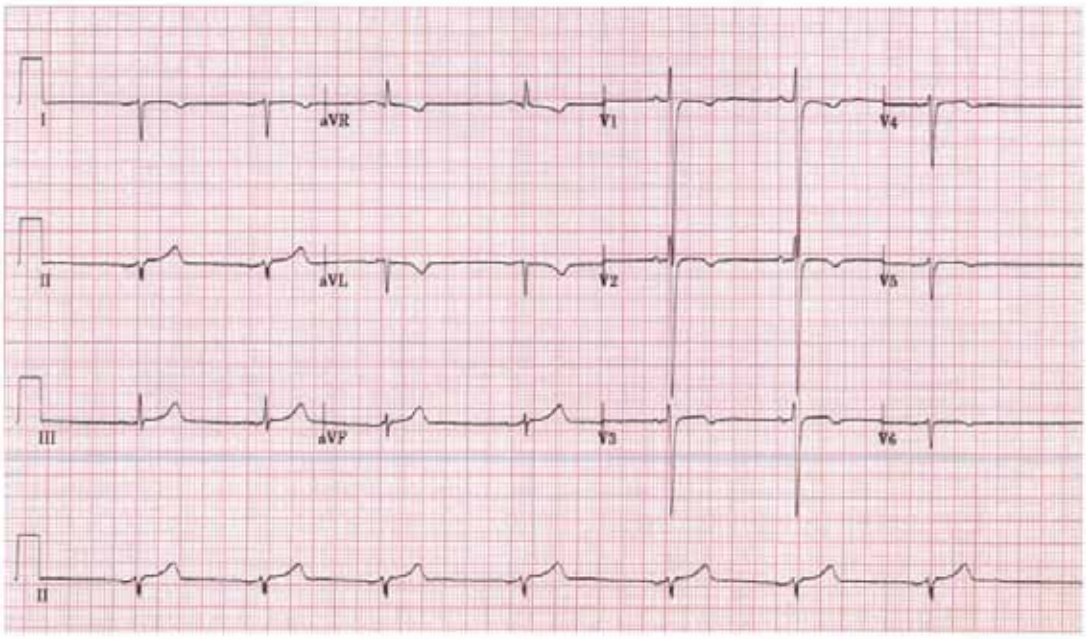

Figure 1

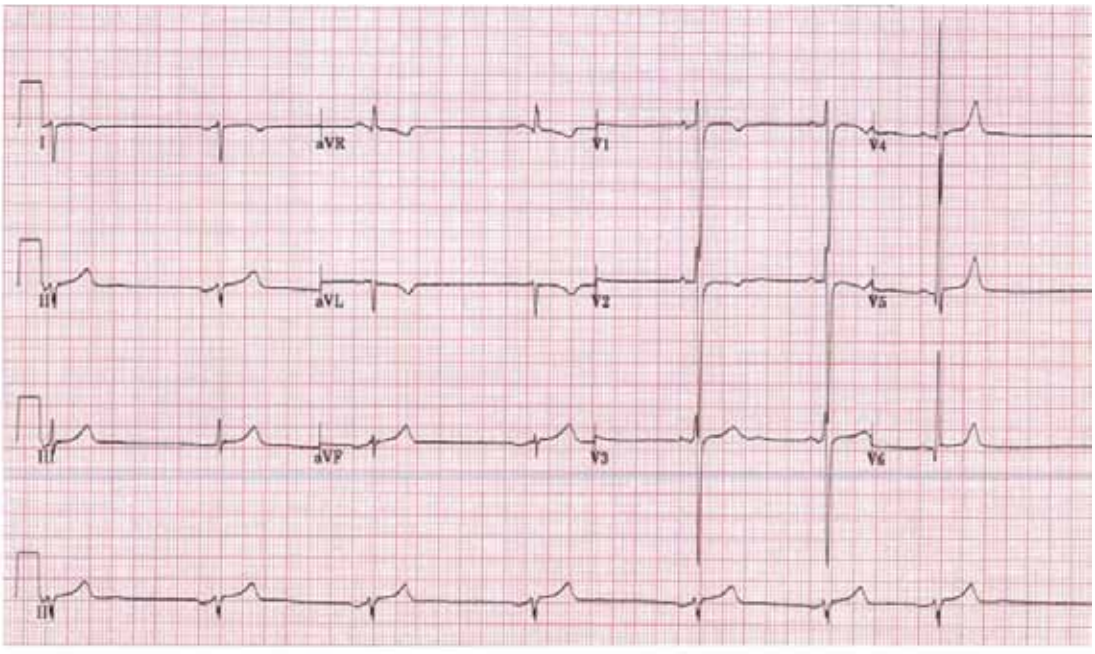

Figure 2

the limb-lead abnormalities observed in ECG 1 remained, all precordial leads presented normally. A clear voltage transition was present, with Sokolow-Lyon voltage criteria for left ventricular hypertrophy noted.

A third ECG was undertaken (Figure 3) using right sided precordial placement together with the reversal of the limb leads. The global appearance of the ECG was within normal limits for a young athletic individual of Arabic ethnicity.

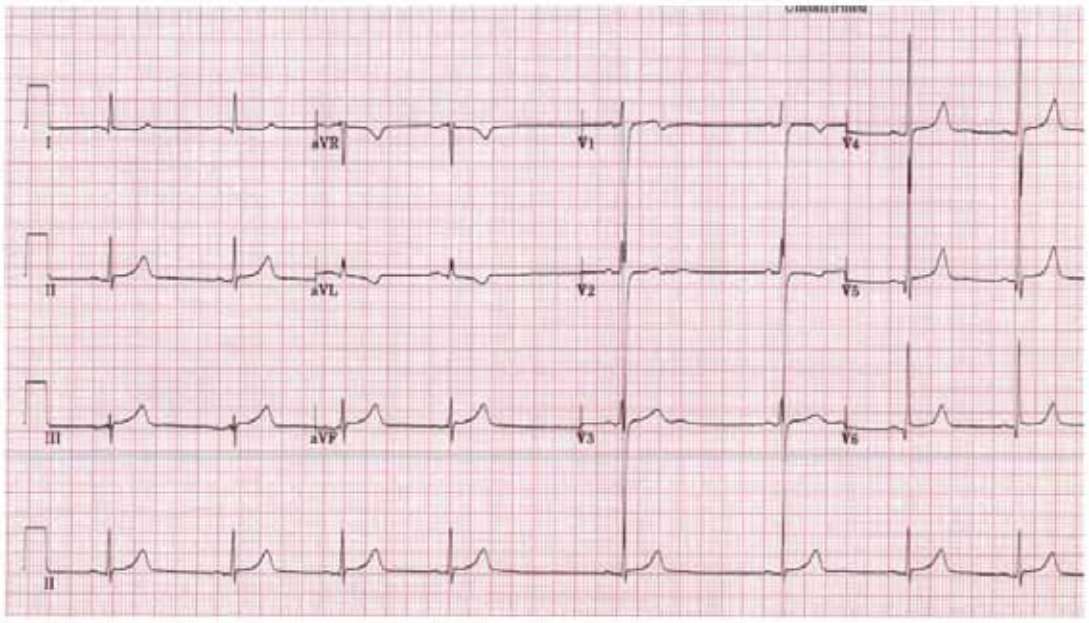

Figure 3

Echocardiography: Echocardiography confirmed dextrocardia with situs inversus atria (i.e. the left atria was positioned on the right whilst the right atria was positioned on the left) together with concordance atrioventricular and ventriculoarterial (i.e. the aorta originated from the left ventricle, although this ventricle was right-sided and the pulmonary artery originated from the right ventricle although again, this ventricle was left-sided). No further congenital abnormality were observed, with the origins of both coronary arteries visible. The inferior cava vein was positioned in the left, draining correctly to the right atrium (located on the left), with normal diameters and flow.

Prominent hypertrabeculation was however observed in both ventricles, primarily located in lateral, anterior and apical position of the left ventricle, but did not meet Chin[2] nor Jenni[3] criteria for left ventricle non-compaction. The aortic arch, descending and abdominal aorta were positioned in the right, with normal diameters and flows in all sections.

Abdominal Ultrasound: An abdominal ultrasound was undertaken which demonstrated a complete situs inversus with multisplenia without further abnormalities.

Cardiac Magnetic Resonance (CMR): Due to the prominent hypertrabeculation, a cardiac magnetic resonance (CMR) scan incorporating late gadolinium enhancement (LGE) was undertaken. Cardiac structure and function was normal and confirmed echocardiographic measurements, with the hypertrabeculation confirmed as not being diagnostic for left ventricular non-compaction. There was no evidence of acute myocardial insult, edema and no evidence of myocardial fibrosis or scarring on LGE.

Diagnosis and Outcome: An asymptomatic 16-year old Arabic trainee referee demonstrating dextrocardia with complete situs inversus and multisplenia. With a confirmed structurally normal cardiovascular system, the athlete was provided with full medical clearance to participate in competitive sport. The athlete was released from care, not requiring annual review.

DISCUSSION

Positional anomalies of the heart are extremely rare and are seldom found during routine physical examination. Cardiac malpositions are defined as an abnormal intrathoracic location of the heart or a location that is inappropriate relative to the position of the abdominal viscera (visceroatrial arrangement). For the anatomically correct heart, it is located in the left hemithorax with its axis (base to apex) pointing towards the left hip (levocardia). When the heart is positioned on the right-side of the chest there are important distinctions to be observed; specifically, when the ECG demonstrates left axis deviation, the finding is termed dextroposition and not dextrocardia, which presents with a right axis deviation (Figure 1).[4] Furthermore, a heart located on the median line of the thorax with the apex pointing either to the right or to the left is termed mesocardia.

Whilst extremely rare, cardiac malpositions often require an extensive work up to differentiate between situs solitus (normal visceroatrial arrangement), situs inversus (mirror image of normal visceroatrial arrangement), and situs ambiguous or isomerism (visceroatrial isomerism).

Situs solitus is defined as a right inferior and superior cava vein connecting to the systemic right atrium on the right side of the heart, with a right-sided liver and a left-sided stomach. Situs inversus is defined as a left inferior and superior cava vein connecting to the systemic right atrium on the left side of the heart, with a left-sided liver and a right-sided stomach; whilst isomerism was defined as an abnormal inferior cava vein connection and/or abnormal relations with the descending aorta, a liver being equally distributed to the right and left sides of the abdomen, and indeterminate stomach situs.[6]

Dextrocardia and Congenital Heart Disease

Dextrocardia occurs in approximately 0.01% of live births.[5] However, 3% of these patients also present with additional congenital heart disease. Consequently, work up often includes a thorough physical examination,

a resting ECG, an echocardiogram and an abdominal ultrasound and/or CT scan.

Isolated dextrocardia with situs solitus or situs ambiguous is extremely rare, but in these cases the incidence of congenital heart disease is considerable.

The most common abnormalities associated to dextrocardia are:

1 the transposition of great arteries and
2 left to right shunts,[6] mainly ventricular septal defects.

Rhythm disturbances are also frequently found in these patients.

Alongside congenital heart abnormalities, the most common non-cardiac malformations are located in the gastrointestinal system, with gastrointestinal malrotation being the most common across all types of dextrocardia.[6] Furthermore, Kartagener's syndrome (a genetic defect in the structure and function of sensory and motile cilia, resulting in multiple ciliopathies) occurs in 25% of individuals who have mirror-image dextrocardia.[7]

When the situs inversus is accompanied by levocardia, the incidence of cyanotic congenital heart disease is 100%.

Sporting Eligibility

Dextrocardia is not listed per se as a disqualifying condition in the 36th Bethesda guidelines for eligibility for competitive sport with cardiovascular disorders.[8] Therefore, after demonstration of a structurally normal and well-functioning heart, the athlete was provided with full medical clearance to participate in competitive sport. It is important to note that the echocardiographic assessment of dextrocardia should establish situs, evaluate atrioventricular and ventricular relationships, distinguish between two ventricles and the functional ventricles, appraise systemic and pulmonary outflows, and determine additional cardiac and extra-cardiac abnormalities.

ECG Differential Diagnosis

When the situs inversus is accompanied by dextrocardia, the QRS complex is negative in I and has a reversal QRS pattern in aVR and aVL, whilst the QRS is reversed in the corresponding right and left precordial leads. Accidental reversal of the left and right arm electrodes (aVR and aVL) produces an ECG pattern similar to dextrocardia, but essentially the precordial leads will be completely normal.[9] Care is warranted to notice this mistake as this QS pattern together with T-wave inversion leads I and aVL usually suggests lateral wall myocardial infarction. **As general rule, a negative P wave in lead I should make one suspect limb lead reversal.** One way to check for this simple error is to compare the QRS vectors of leads I and V6. Both are normally directed in approximately the same direction, because both reflect vector activity toward the left-side of the heart. Disparity between these two leads should prompt correct lead placement confirmation.[10]

CONCLUSION

Positional anomalies of the heart are extremely rare, and yet are often associated with congenital heart disease. The most common abnormalities are transposition of great arteries, left to right shunts and ventricular septal defects. Exclusion of rhythm disturbances and Kartagener's syndrome are also of important in the decision for medical clearance for competitive sport. Dextrocardia is not listed as a disqualifying condition in the 36th Bethesda guidelines for competitive sport eligibility in individuals with cardiovascular disorders. Thus, with the demonstration of structurally normal heart, athletes presenting with dextrocardia with complete situs inversus and multisplenia may be provided with full medical clearance to participate in competitive sport.

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Addendum: Does the normalisation of T-wave inversion upon the onset of exercise lessen the likelihood of underlying cardiac disease?

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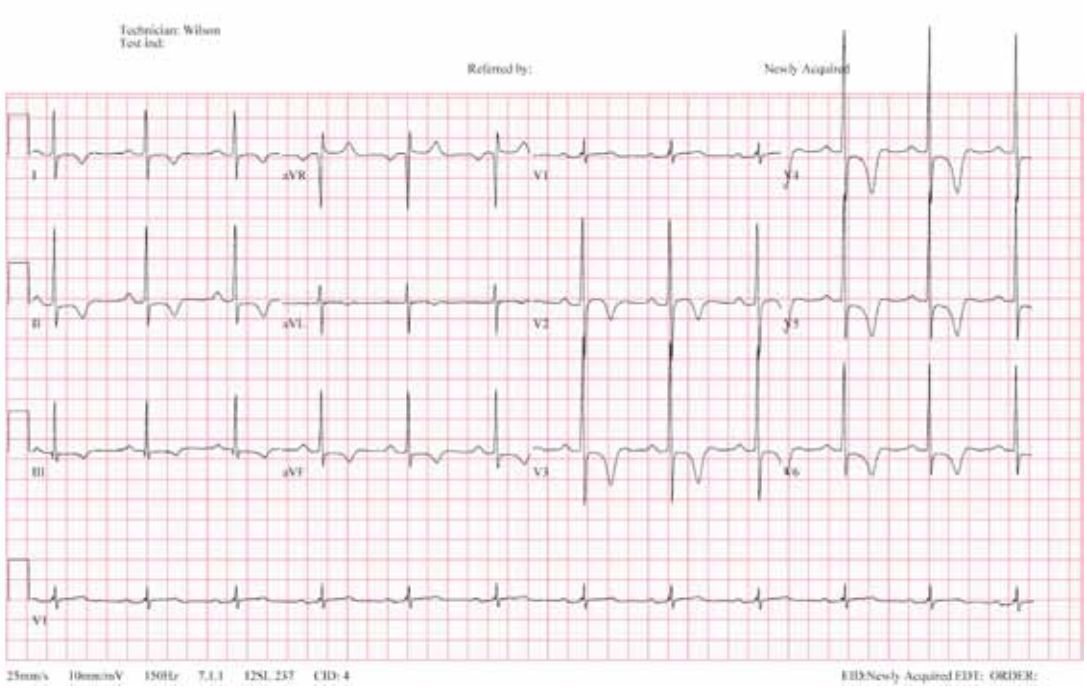

Figure 1

In 2011 in this journal (Vol 38; Issue 3) we posed the question, "Does the normalisation of T-wave inversion with exercise lessen the likelihood of underlying cardiac disease?" The question is important as deep T-wave inversions in ≥ 2 contiguous leads (but not aVR, and III) are a recognised manifestation of hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC). Subsequently, inverted T-waves may represent the first and only sign of an inherited heart muscle disease, in the absence of any other features and before structural changes in the heart can be detected.

As a result of limited evidence base, there remains a significant academic challenge when charged with providing a medical clearance for competitive sport in athletes who demonstrate significant repolarisation abnormalities at rest (such as inferolateral T-wave inversion – Figure 1), which then become normal or near-normal during exercise or adrenergic stimulation. Anecdotal evidence from our laboratory suggests that only very few patients with true cardiomyopathy demonstrate T-wave inversion normalisation during exercise. However, whilst the normalisation of repolarization abnormalities purports a better prognostic outcome than a failure of normalise (or indeed a worsening of abnormalities), the isolated observation of T-wave inversion normalisation cannot exclude an underlying pathology. Accordingly, we concluded that the resolution of this electrocardiographic abnormality with exercise does not imply that the T-wave inversion is a physiologically benign finding and thus should not be utilised as a diagnostic absolute.

Despite our recommendation above, little scientific evidence is available to support our assumption. However, a recent study by Zozri et al.[1] assessed the impact of exercise upon the prevalence of the clinical phenotype of right precordial T-wave inversion in 35 patients with ARVC (19 men; mean age 22.2 ± 6.2 years) against 41 healthy participants with right precordial T-wave inversion free from cardiovascular disease.

A maximal exercise stress test was conducted on all participants which demonstrated that at the peak of exercise, T-wave inversion persisted in 3 patients with ARVC (9%), partially reverted in 20 (57%) or completely normalised in 12 (34%). Indeed, patients with ARVC with or without T-wave inversion normalisation demonstrated a similar clinical phenotype of disease. The overall prevalence of right precordial T-wave changes during exercise (normalisation in addition to partial reversal) did not differ between patients with ARVC and healthy participants (92% vs. 88%, NS), whereas there was a trend toward a greater prevalence of complete normalisation in healthy participants (56% vs. 34%, p = 0.06).

In conclusion, Zozri et al.[1] study supports our previous recommendations in this journal. The normalisation of T-wave inversion upon the onset of exercise does not lessen the likelihood of an underlying cardiac disease. Furthermore, T-wave normalisation with exercise stress testing appears to be simply an incidental finding, and should not be utilised for prognostication.

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World University Games

Kazan, Russia, July 2013

Steve Williams

GP/Sports Doctor

Dunedin

In July 2013 I had the pleasure to travel as team doctor with the New Zealand team to the World University games in Kazan Russia. Slipping below the radar of the average kiwi sports fan the games are 2nd only to the Summer Olympics in size. Twelve thousand athletes made there way to Kazan which is situated 800 km east of Moscow.

With 1.2 million people Kazan has a rich culture and history, the population is a mix of the local Tartar people and Russians. Promoting itself as the "Sports Capital of Russia" Kazan had built some beautiful new venues for the 2013 games.

New Zealand sent a team of 38 athletes and 13 support staff competing in 9 different sports. As well as the support staff for each sport six general support staff were appointed. At the head Pete Wardell was a fantastic Chef de Mission and was well supported by Evelyn Williamson (operations) and Dave Schaper (athlete support). The medical team comprised of myself and physiotherapists Vanessa Trent and Jessica Meyer. As a group we knitted together very well. Working hard when called upon but able to share a joke with each other, often it seemed at my expense!

Jess Meyer at the Aquatic Pool

A steady number of athletes received physiotherapy and medical treatment through the games but happily no significant injuries or illness was encountered during our stay. One of the highlights of the games was the collegial approach amongst the medical staff from the different countries. I even found myself helping out Australian colleagues on more than one occasion!

By in large the athletes were happy with there results. The games providing them with exposure to high class competition, in a games environment which I'm sure will stand them in good stead for the future. As just one example it was fantastic to watch Angie Smit run a superb 800m, clocking a new PB and in doing so qualifying for the world athletics championships in Moscow.

The next summer University games will be held in Guangju, South Korea in 2015.

If the opportunity arises to take part in a World University games I would highly recommend the experience.

New Zealand Team Flag Raising, Kazan, Russia

The Third Annual Medicine of Cycling Conference 2013

Colorado Springs, 21 to 23 September

Judith May

Sports Physician
Tauranga

I attended the third annual Medicine of Cycling conference in Colorado Springs from September 21 to 23. It is a niche conference that would interest any sports medicine clinician who has a professional or personal interest in cycling. Colorado Springs is located 100 km south of Denver and sits at 1839 m above sea level. It is home to the United States Olympic Committee, US Olympic Training Centre, USADA and the USA headquarters of many Olympic sports. The conference was held at the headquarters of USA Cycling, and was attended by 200 doctors and allied health professionals from a variety of specialities.

Medicine of Cycling was formed in 2010 by a group of doctors who saw a need for a forum for physicians involved in cycling to share experiences and develop expertise. As well as organising the annual conference they have developed cycling specific consensus guidelines and provide an online forum.

The first day was dedicated to a medical emergencies in cycling course. The morning session included information on race event planning and equipment preparation. The afternoon was a practical session involving rotating stations in groups of 4 to 5 people. There were stations on airway management, wound management, chest/thoracic trauma, orthopaedic injury and cervical and head trauma. Medicine in Cycling have established specific guidelines for the management of traumatic brain injury and concussion. This is an area that has historically been managed poorly by cycling medical professionals, and is an attempt to get more standardised care as now occurs in other sports with high rates of concussion. You can view the full consensus statement at www.medicineofcycling.com.

The highlight of the second day included a presentation on dermatology in cycling by Dr Timothy Berger. He emphasised that cyclists have high levels of sun exposure and therefore a significant risk of skin cancer. Triathletes have the highest risk with exposure to nine times the safe amount of sun. The head and back of the neck gets the most exposure and therefore has the highest rates of skin cancer in cyclists. Most people only apply one quarter the amount of sun screen that is used in laboratory test conditions. The relationship between the amount applied and SPF achieved is exponential, so most are only achieving a fraction of the true SPF. Dr Berger recommended a double application of broad spectrum SPF 30+20 minutes apart to achieve the true SPF.

Throughout the conference there was much debate on the management of gravel rash. The general consensus was that the most important step is cleaning the wound thoroughly with soap and water. Topical solutions such as betadine and antibiotics should be avoided, and the wound should then be covered with an occlusive dressing such as duoderm or hypafix.

There were various panels and presentations on bike fit. The general message was the need to accommodate to the condition and anatomical restraint of the individual cyclist. A consensus statement has been developed which can be found at http://www.medicineofcycling.com/assets/Medicine_of_Cycling_Bike_Fit_Consensus_Statement.pdf

The third day included presentations on overtraining by Inigo San Millan, the exercise physiologist for professional cycling team Garmin, and nutrition from sports nutritionist Monique Ryan. Both highlighted the recent fad for cyclists to adopt low carbohydrate diets, which is putting the athlete at high risk for overtraining. Other presentations included caring for the female cyclist, facial and dental injuries and cardiovascular screening.

The last session was a Performance Enhancement Drugs in Cycling Round Table including Matthew Fedoruk (Science Director at USADA), Onye Ikwuakor (Director Legal Affairs at USADA), Sean Petty (Chief Operating Officer at USA Cycling), Dr Alan Farrell (Team Physician Team Sky) and Scott Mercier who rode for US Postal in 1997. It made for a fascinating discussion on the past doping culture in cycling, the Lance Armstrong affair and the current challenges faced by those working with pro cycling teams.

Ironman Sports Medicine Conference

Kona, Hawaii, 6 to 10 October 2013

Bruce Hamilton

Sports Physician
Auckland

If you have any interest in Ironman triathlons, medical care in endurance sports, licra, bike kit, or the latest (legal) performance technology aids, then this is the conference for you. This was the first time I have attended this conference, which is run annually by the organisers of the Ironman Medical tent, and runs during the week leading up to Ironman World Championship in Kona. As a result of its timing, you get to watch Kona evolve from a beautiful seaside town into a sprawling triathlon megastore! Despite the numerous (and there are plenty) distractions the conference itself regularly attracts an incredibly high calibre of presenters, and the programme is of a remarkably high quality.

Unsurprisingly, sports cardiology was a hot topic, with names such as Ben Levine, Bert Fields and Greg Whyte (who tragically, while playing golf with yours truly, shot a hole in one on a 184 yard par three – couldn't have happened to a less deserving character…) debating both the pro's and con's of cardiac screening, and the content of any screening if you do perform it. The result: more evidence is needed.

My next pick of interesting topics was listening to Irene Davis. Dr Davis is one of the worlds most published reseachers in the field of gait analysis, and spoke on both gait re-training (or just training) and barefoot running. A take home message for me was the point that often we can "strengthen" and improve "function" of gluteals, core and other muscles involved in gait, but without technical re-training of gait patterns, the likelihood of a positive outcome (that being injury prevention) is low. She highlighted a great process which she has evolved through, from high end digital analysis for research through to the translation of research techniques into simple, practical and achievable methods available in most of our practices. Well worth a listen too.

Another session worthy of highlighting, were the nutrition presentations by the incredible Dr Mark Tarnopolsky. It would be fair to say that I understood about 20%, tried to grasp another 20%, and for the remainder sat stupefied by the incredible output of the man. I found it inspirational, motivational, and if this is possible, at the same time depressing…

The final session is dedicated to discussion surrounding the medical care of Ironman triathletes during the race in Kona. This has been going on now for over 25 years, and the clinical experience of those presenting is impressive. I was particularly taken by the necessarily pragmatic approach required to be taken in many complex situations, and the seriousness with which the care of the athletes is taken.

This conference is worth attending. It was great to see a number of kiwis there, including long time triathlon stalwart Dr John Hellemans. If the content doesn't grab you, the setting, open air and looking directly out over the Pacific Ocean, certainly will. Combine that with an opportunity to work in the Ironman medical tent and the opportunity to win a hard to get entry into the Ironman for the following year, and it all adds up to a great experience.

Robin McKenzie

20.4.1921 - 13.5.2013

Duncan Reid

Vice President International Federation of Orthopaedic Manipulative Physiotherapist (IFOMPT), former president NZ Manipulative Physiotherapists Association (NZMPA)

Robin McKenzie, world renowned New Zealand physiotherapist, died peacefully on 13 May 2013, after a long battle with cancer. Robin was an outstanding physiotherapist and has left the physiotherapy world with a significant legacy in advancing the management of musculoskeletal conditions in particular the areas of neck and low back pain.

Robin completed his physiotherapy training at the New Zealand School in Otago in 1952 and soon after set up his private practice developing a special interest in treating spinal disorders in Wellington. His pathway to international recognition began with a chance observation in his clinic, where a patient presenting with low back pain and buttock pain was asked to go into the treatment and lie down on the bed in preparation for the treatment. Robin had not put the bed end down flat but the patients followed the instructions and lay down prone on the bed in an extreme hyper extended lumbar position. When Robin came in to treat the patient he was aghast, but quickly asked the patient how he was feeling. The patient happily reported that his back pain was the best it had been for days and that the pain in the buttock had gone and moved to his back. So began the development of the McKenzie method of diagnosis and treatment now known as the McKenzie Method of Mechanical Diagnosis and Therapy (MDT). This system has achieved worldwide recognition and is now regarded as part of "normal" management for low back pain. His vision was that all patients with musculo-skeletal pain be taught how to manage their own pain. The two books he wrote specifically for patients, *Treat Your Own Back* and *Treat Your Own Neck* have sold over 6 million copies and are available in 17 languages.

The McKenzie Institute International was established in 1982 and has grown into a worldwide educational organisation. To date, physiotherapists, doctors, chiropractors and other allied health professionals in 37 different countries have been educated in the McKenzie Method.

Robin was a founding member of the New Zealand Manipulative Therapists Association (NZMTA) (now called NZ Manipulative Physiotherapists Association (NZMPA)) in association with other key physiotherapists at the time, Brian Mulligan, Ian Searle and Craig Cameron. The early years of the NZMTA were driven by the need to establish a formal qualification in manipulation. Under the guidance of Brian Mulligan, a two year manipulative therapy course was developed. Robin was one of the first graduates of this course in 1973. This qualification was the first post graduate course of study in manipulative therapy in NZ, and became known as the Diploma of Manipulative Therapy (Dip MT).

In 1980, NZMTA hosted the International Federation of Orthopaedic Manipulative Therapists (IFOMT) conference, with Robin being one of the key note speakers along with other famous manual therapist of the time, Geoffrey Maitland, Alan Stoddart and Professor Vladimir Janda. Robin was also instrumental in bringing James Cyriax to NZ in the years that followed.

Arguably, no other physiotherapist has progressed the knowledge of assessment and treatment of lumbar disc injuries more than Robin McKenzie. His original text written in 1981, *Mechanical Diagnosis of Low Back Pain*,[3] not only became the basis of teaching of the management of discal pain but has been the springboard of a significant body of research validating McKenzie's key clinical observations and management practices. The ability of McKenzie to define three broad classifications of spinal pain, namely posture, dysfunction and derangement are now the corner stone of low back classification systems. Another key phenomenon that McKenzie observed and developed was the concept of peripheralisation and centralisation of pain in response to repeated movements to the affected spinal area. The ability to have the source of pain move more peripherally with a provoking movement and then use movement (often opposite to the provoking movement), to encourage the pain to centralise back to the source, (in the case of low back pain, most often the disc), is now well established and predictive of a good response to treatment.[2] The McKenzie concept of management of Low Back Pain (LBP) has been extensively researched and compared to a range of other therapeutic approaches and found to be highly effective.[1]

Robin McKenzie was an Honorary Life Member of the American Physical Therapy Association, a member of the International Society for the study of the Lumbar Spine, a Fellow of the American Back Society, an Honorary Fellow of the New Zealand Society of Physiotherapists, an Honorary Life member of the New Zealand Manipulative Therapists Association and an Honorary Fellow of the Chartered Society of Physiotherapists in the United Kingdom. In 1990, he was made an Officer of the Most Excellent Order of the British Empire, and in 1993, received an Honorary Doctorate from the Russian Academy of Medical Science. In the 2000 New Year's Honour's List, Her Majesty the Queen appointed Robin McKenzie as a Companion of the New Zealand Order of Merit.

Vale Robin McKenzie

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