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Instructions to Authors

GENERAL

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

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

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

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

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

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Scurvy, Gissy and the Plague of Tendinopathy

When Englishman Captain James Cook landed in Poverty Bay on October 8 1769 (less than a kilometer from where I grew up in Gissy), little did he know that he would set off a chain of events that would, amongst other (obviously less notable) things, culminate in the publication of this journal. Cook of course was not the first European to discover NZ, that honour belonging to Dutchman Abel Tasman who parked his ship off the Nelson area a few years earlier, many centuries after our Polynesian ancestors first inhabited this fine land. However, Cook's journey was notable for more than just charting NZ, as his was a voyage of scientific investigation (observing the traverse of Venus from Tahiti), as well as one of exploration. It was this combination of tasks that resulted in a prolonged tour but it was the low incidence of scurvy on board Cook's ship that separated his voyage from that of his peers. Life was tough in those days, and Cook must have been concerned about the voyage, as for over 150 years, scurvy had plagued sailors and resulted in thousands of lives lost, ships abandoned and ultimately failed missions. Typically, up to 50% of sailors on board "long haul" voyages may have been expected to suffer a slow and agonising death, and between the years 1500 - 1800 up to 2 million sailors may have perished from scurvy. Cook however was facilitated in his journey by the endeavours (excuse the cheap pun) of his sailing forebears, for without their attempts to find a cure for scurvy, it is unlikely that Gissy, New Zealand and for that matter much of the southern hemisphere would have been known to Europeans until many years later. Unlike many of his peers, Cook instigated all the contemporary theoretical and practical means of preventing scurvy, and while the specifically successful treatment wasn't known, he was the first mariner to successfully combat scurvy, and hence have enough surviving sailors to enable his successful navigation of the globe. It was only in the late 18th century, when it became clear that naval dominance could be achieved through the elimination of scurvy, that any real investment in a cure was made, and shortly after the recognition of citrus fruit as a prevention was identified. Remarkably, the presence and absence of scurvy amongst naval seamen may have played a crucial role in the independence of both America (from Britain) and Britain (from Napoleon) respectively. Scurvy is now known to result from a deficiency of Vitamin C (ascorbic acid), found in fresh fruit and vegetables in abundance, but scarce on long haul shipping due to the inability to store fresh fruit and vegetables and the use of copper pots which reduced Vitamin C availability. Despite this, the management of scurvy continued to fluctuate with its reappearance in 19th and early 20th Century polar exploration, and it continues to appear in nutritionally deficient countries. Remarkably however, 300 years earlier in 1601, Sir James Lancaster stumbled across a genuine cure for scurvy, lemon juice, but due to the lack of scientific evidence of its curative ability, this effective treatment was slowly lost over the years, only to be replaced by multiple unsubstantiated theories and concoctions, resulting in thousands of deaths and vast costs; hence, the treatment for scurvy remained elusive for almost 200 further years. The medical thinking of the time was focused on the balance of the humors, which restricted the ability of clinicians of the day to consider deficiency as a cause of such a profound disease, and technology limited their ability to investigate adequately.¹

Vitamin C is of course involved in the synthesis of collagen, the absence of which results in collagen breakdown and symptoms of scurvy result from this. It would be brave or foolish to try and link the high frequency of tendinopathy that we observe in many sports with a deficiency of vitamin C (although a quick search in "journal google" will reveal a clear and profound relationship), and fortunately, I am not going to try that today. However, such are the vast number of aetiological theories and treatment options currently available for the treatment of tendinopathy,² a comparison with the convoluted history of understanding scurvy seems more than reasonable. In the late 1990's, the term tendinitis was discarded as representative of an age of dogma, and not reflective of the newly identified histo-pathology. Tendinitis was replaced by the catchy terms "tendinopathy", "tendinosis" and to describe the histopathological changes observed, the term "degenerative". In the last 10 years there has been an abundance of research trying to establish the aetiology, pathophysiology and best management of this challenging condition, and it is clear that we have come a long way in our understanding of its complexity.³ However, despite the abundance of both in vivo and in vitro research and the explosion in information availability through the internet, we appear only marginally closer to a definitive understanding of either the aetiology or management of "tendinopathy". Many fundamental questions remain unanswered, and authors are now questioning the use of terms such as degenerative when describing the pathology. Management approaches necessarily remain pragmatic, regional and inconsistent in the absence of a clear aetiology.

Given our necessarily temperocentric views, one wonders if we are making the same mistakes as our 18th century colleagues. Is it that we actually have the answers in front of us, but are unable to put the puzzle together, blinded by

our beliefs or recycled dogma - as was the case in the 17th century management of scurvy? Or is it simply that we don't yet have the tools required to answer the questions we are asking, or the understanding to ask the right questions? Asking the right questions is a good start, but as we are finding out, providing the answers is a lot more challenging. As practitioners in the burgeoning field of Sports and Exercise Medicine it is incumbent on us all to challenge dogma and search for the most appropriate means of managing injuries such as tendinopathy. While not all of us are in the privileged position of being able to perform high level clinical trials, critical self analysis and the accumulation of objective, quantifiable data is achievable and mandated for us all in order to ensure we are offering our patients the highest level of care. In critical reasoning and with the accumulation of such data, perhaps we will avoid the delays that our medical forefathers faced in understanding equally fascinating clinical conditions.

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Dr Bruce Hamilton
Editor

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This issue of the NZJSM is special in that it marks only the second time in over 40 years of publication that it is being produced immediately after winning the Rugby World Cup. I would like to add my congratulations to all those involved, particularly from the Sports Medicine and Science side, who clearly had a significant impact on the result. Of course, rugby was not the only international sporting code to have great success this year, and New Zealand should go into next year's Olympics with confidence that it has both the athletes and the support services needed to perform exceptionally well.

It was great being back in NZ for the Annual Conference held recently in Queenstown. Having not been there for 20 years, it was great revisiting the beautiful area, re-uniting with old friends, making new friends and both watching and engaging in most interesting presentations and discussions. My thanks to Ian Murphy and the Organising Committee for putting on such a great meeting and I look forward to next year's conference in Auckland

In this journal, you will find a selection of the abstracts from the conference as well as a broad range of articles. We are constantly looking for ways of improving the content of the journal and we hope to continue to add features as we move into 2012 - your involvement in your journal is always appreciated. In the New Year, you should once again be receiving a hard copy of the journal in the post. We recognise that while electronic journal access is convenient, the attraction of the paper copy remains, and as such we intend to print an annual edition of the journal, including all of the content published electronically during the year.

I would like to thank all those who have contributed articles to the journal over the last 12 months, those that have acted as reviewers and in particular Brenda Allum for her ongoing hard work in preparing the journal.

Enjoy and see you next year!

Bruce

Reply to review on “The Central Governor of exercise performance: Fact or fiction”

I appreciate Professor Cairn’s carefully balanced review of the Central Governor Model (CGM) – The central governor of exercise performance: Fact or fiction⁵. I am especially grateful for his conclusion that the model “is an extremely valuable concept to investigate further and apply to all forms of sporting events along with clinical exercise situations” (p.50).

Indeed models are developed for exactly that reason. They make predictions which can be tested. By testing those predictions we can determine whether or not the model is helpful. By modifying the model or indeed rejecting it if there is no other option, we advance our understanding especially of complex phenomena like the regulation of human exercise performance.

What many may not remember is that when it was first proposed in 1998¹², the CGM represented a significant mindshift in our discipline. For then everyone “knew” that, according to the traditional A.V. Hill model^{11,14,15}, exercise performance was “limited” solely by the development of a catastrophic peripheral fatigue in the exercising muscles. Indeed this interpretation continues to be argued in some of the world’s leading journals⁷. To have suggested that the brain is the final regulator of exercise performance was, at that time, considered to be too ludicrous to be taken seriously.

Now, of course, the argument has changed. As the evidence of the brain’s importance in the regulation of exercise performance continues daily to accumulate (for example – ^{2,3,9,10,22}), the traditional “brainless” model¹⁷ of human exercise performance becomes increasingly less defensible.

As a result the argument has now become in effect: “Of course we know that the brain is important. That is not new. In fact we have always known it. But that regulation has nothing to do with a mythical central governor”. This is in keeping with the legendary statement of Louis Agassiz who more than 100 years ago described how scientific knowledge advances: “Every great scientific truth goes through three phases. First people say it conflicts with the Bible. Next they say it has been discovered before. Lastly they say they always believed it”.

In this way some of those who only a few years ago were influential backers of the A.V. Hill model of exercise regulation have suddenly undergone a Damascus Road transformation without ever having to acknowledge it. Instead the villain becomes the person who first suggested that there was a need for a new interpretation^{4,7,19}.

It must by now be reasonably obvious^{16,17} to most reasonable and disinterested scientists that the brain is indeed the final regulator of human exercise performance. Whether or not this is due to a so-called “central governor” is largely irrelevant. The achievement of the CGM has been to focus the debate in this field by developing an alternative and easily testable model as Professor Cairn acknowledges. To quote Albert Einstein: “The mere formulation of a problem is often more essential than its solution, which may be merely a matter of mathematical or experimental skill. To raise new questions, new possibilities, to regard old problems from a new angle requires creative imagination and marks real advances in science”.

Elsewhere^{11,16} I have reviewed why this alternative approach conforms with the Popperian model of scientific inquiry but contrasts with the authoritarian Kuhnian model which, in my view, has prevented a reasonable debate of this topic for perhaps the last 50 years or so.

My sole point of contention with Professor Cairns’s article is his claim that “(Noakes) totally refutes the occurrence of peripheral fatigue”. In my article to which Professor Cairns refers, I specifically referenced studies showing the presence of peripheral fatigue and included them in the most recent diagram of the model. My point is simply that for at least three reasons, peripheral fatigue acting perhaps as a peripheral governor as McIntosh and Shahi⁸ suggest, cannot be the factor “limiting” exercise performance.

Firstly, the development of peripheral fatigue or alternatively a peripheral governor cannot explain the anticipatory components of exercise control – especially the choice to begin exercise at different intensities dependant entirely on the anticipated or known duration of the planned exercise bout^{13,23}.

Secondly, these peripheral phenomena cannot explain the exercise “endspurt”^{13,23} since how would a peripheral governor confined to the muscles “know” for certain exactly when the end of any exercise bout is rapidly approaching?

But, most tellingly, neither of these mechanisms can explain why subjects slow down or indeed terminate all forms of dynamic^{1,9,18,20,21} or isometric⁶ exercise without a 100% recruitment of all the available motor units in their exercising limbs.

For, as repeatedly argued¹⁸, peripheral mechanisms can only regulate or limit the force output of muscle fibres that are actively contracting since these peripheral models explicitly require that “fatigue” is caused solely by continuous contractile activity. According to that model, these peripheral mechanisms will cause the termination of exercise only when all the available motor units are contracting and all have fatigued, causing the reduction in the work output that we define as “fatigue”.

Thus the absence of complete recruitment of all available motor units in the exercising limbs at the point of fatigue or exercise termination is the single best evidence that central, and not peripheral mechanisms regulate exercise performance¹⁸.

Whenever exercise terminates with motor unit recruitment reserve, then the sole conclusion must be that the central nervous system is the ultimate regulator of that exercise performance (regardless of the extent to which a peripheral governor or peripheral fatigue will also be active at that moment).

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August

The August issue of BJSM was headed up by a useful editorial entitled “Changing gears: bicycling as the panacea for physical inactivity”. Written by Adrian Bauman and colleagues, the article summarised the health benefits and risks of cycling and noted individual, social and environmental barriers to cycling. It documents the controversy with regard to helmet use and efforts within various communities to get more people cycling. In summary, cycling is not a panacea for physical activity, but it has great potential and this is only partly realised at present.

Later in the same issue was a useful article on Injury-prevention priorities according to playing position and professional rugby union players. This study demonstrated individual position-specific differences in match injury profile; for example forwards sustain injuries to their shoulder, knee and ankle most frequently, whereas for the backs the most frequently injured sites were the shoulder, hamstring and knee. With this data an appropriate injury prevention programme can be tailored for each playing position.

The issue concluded with another in-series of A to Z of Nutritional Supplements. This issue concentrated on leucine, lecithin and gamma-linolenic acid. The expert reviewers commented that although lecithin maintains plasma choline levels after exercise, there does not seem to be a good reason to take supplementary lecithin in the hope of improving performance. Likewise, there is little evidence that supplementary leucine or gamma-linolenic acid is necessary for peak sporting performance.

The September Injury Prevention and Health Protection issue sponsored by the IOC contained a landmark consensus statement on the health and fitness of young people through physical activity and sport. The IOC

assembled an expert group in January 2011 and their findings are detailed over 10 pages. The article concludes with recommendations for sports organisations, governments, non-governmental organisations and researchers. As befitting its comprehensive nature, 168 references are supplied. Sedentary behaviour in youth is a major concern in most developed countries. Increased screen time, either in front of a television or computer monitor, is associated with sedentary behaviour and there is strong evidence that children spend increasing time being sedentary as they grow older. This article by Russell Pate and co-workers dovetails nicely with a second article in the same issue entitled “Sedentary behaviour interventions in young people: a meta analysis”. The authors comment that their literature review indicated the interventions designed to reduce sedentary behaviours in young people tended to have only small effects. They comment that this may be due to strong environmental cues and that time spent away from a screen may be allocated to other sedentary behaviours, e.g. listening to music, talking or sedentary hobbies. Clearly there are major challenges to getting our children on their bikes, or at least out of the house.

In the same vein, Van Sluijs and co-workers examine the effect of community and family interventions on young people’s physical activity levels. Their conclusion is that although the study quality is improving, the effect of family and community based interventions remains uncertain.

Later in the same issue is an article expressing more optimism; Lyle Micheli, who has previously been a speaker at our SMNZ Conference, and fellow experts see great promise in improving the fitness and health of children through sport. Their article starts with the well-known African

proverb “It takes a village to raise a child”. They comment that it is a global village that needs to step into action and sport is the logical vehicle for improving the physical activity profile and fitness of our young people.

September

The routine September issue was chock full of useful articles: I even got one published myself entitled “Practising sports and exercise medicine in an environment of rising medical costs”. This is one of the major challenges facing today’s clinicians. Fifty years ago the limits to medicine were largely around what we could do. Now the limits are increasingly around what we can afford. We clinicians are increasingly facing questions from well informed patients requesting high technology imaging, for example. It has been a significant contributor to rising medical costs. I advocate a collaborative approach where clinicians, administrators, funders and the patient all have to play a role in accepting that there needs to be some rationing of access to expensive diagnostic and therapeutic interventions, and increasingly people who wish to have an extra investigation for their own reassurance should be asked to fund these themselves.

Sudden cardiac death is a hot topic at present. Jonathan Drezner and colleagues ask the pertinent question: Where did the science go? They comment that systematic reporting systems are crucial. ECG screening does not need to be mandatory but it can be recommended for at-risk populations such as competitive athletes. They comment that adding an ECG greatly increases the sensitivity to detect conditions at risk of sudden cardiac death and has been shown to enhance the cost-effectiveness of a screening process. The issue is one of ongoing debate.

Later in the same issue was an article by Lynley Anderson of Otago University entitled Bloodgate: were the punishments fair? This refers to events of April 2009 during a rugby match between Leinster and Harlequins when a Harlequins player faked a blood injury and the physiotherapist and doctor were both closely involved in the scam. The doctor was given a warning by the Medical Council but the physiotherapist was struck off before being reinstated by a High Court decision in January 2011. Anderson comments that the health aims of doctors and physiotherapists may be distorted by the environment of professional sport. Teaching ethics and creating professional guidelines may not be enough; we need to recognise the pressures and provide adequate support and protection to health professionals involved in sport.

Innovation is necessary to remain at the head of the pack. A trans-Atlantic collaboration between Cathy Speed of the UK and Bill Roberts of the USA provides a background for why people innovate in high performance sports medicine and provides advice on how to do this. They argue for a diverse group that is likely to be more creative.

Chronic exertional compartment syndrome can often be difficult to diagnose. There is a very useful head to head series of articles within the September issue. On the one hand, Mark Hutchison of Chicago argues that all four compartments in each leg should be tested as then one has absolute proof of the pressures generated by exercise. By contrast, Matt Hislop and Mark Batt recommend a minimalist approach and conclude that not all compartments need to be measured routinely. They advocate additional time spent in history-taking to try and identify which particular compartments are involved and test only the more symptomatic leg, thus reducing the number of needle insertions. Finally,

they conclude that measuring resting pressures is not necessary in the investigation of chronic exertional compartment syndrome. For any clinicians with an interest in this condition, these articles make very worthwhile reading.

Upper respiratory tract infections are the commonest infection seen in the community. David Nieman, who has published widely on these issues, and colleagues studied a group of 1002 adults aged 18 to 85 years and followed them for 12 weeks during the winter and autumn seasons whilst monitoring respiratory tract symptoms and severity using the Wisconsin Upper Respiratory Symptom Survey. They found that those subjects who performed aerobic exercise for five or more days per week had a 43% reduction in the number of days with URTI compared to those who were largely sedentary, i.e. performing only one day or less of aerobic exercise per week. The message is clear - get out there and get active and you should stay healthier.

Wrapping up this busy September issue was the usual section on nutritional supplements. Leptin is a hormone which plays a crucial role in the regulation of appetite, body fat mass, basal metabolic rate and gonadal function. Theoretically, it could be used as an anabolic agent when combined with strength training. The reviewers conclude that there is currently insufficient knowledge regarding this substance and do not recommend supplementation. Magnesium supplements are frequently used, but unless there is evidence of deficiency there is no evidence for any benefit from magnesium supplementation. Medium chain triglycerides are used commonly by bodybuilders, however they frequently cause gastrointestinal distress and any theoretical metabolic benefits are heavily outweighed by this side effect. Therefore, the authors

do not recommend their use as supplements.

October

The October issue included an article by the guru of achilles tendinopathy treatment, Hakan Alfredson, who presented at our conference some years ago. His concentric then eccentric strengthening regime has been very successful in treating most people. In those cases where improvement did not occur and operation was necessary, he found an enlarged plantaris tendon in 58/73 consecutive tendons. These people were treated with ultrasound and Doppler guided scraping and extirpation of the plantaris tendon, i.e. the plantaris tendon was cut and removed. He often found richly vascularised fat tissue interposed between the achilles and plantaris tendons and hypothesised that the plantaris tendon could be considered a co-factor in treatment resistant midportion achilles tendinopathy. Watch this space.

Could genetics play a role in achilles tendinopathy? A combined group from Australia and South Africa examined this issue via a pathway based approach. They investigated the association of sequence variance. Independently, no associations were observed between any of the polymorphisms tested and the risk of tendinopathy. However, the allele combinations of five polymorphisms were found to have a highly significant relationship with achilles tendinopathy. No doubt this is an area for further research.

Later in the same issue was an intriguing article entitled "Referrals from a primary care-based sports medicine department to an orthopaedic department: a retrospective cohort study". This study examined nearly 5000 patients seen at a sports medicine department in Fort Worth, Texas. Only 2.4% of

those patients required referral to the orthopaedic department, the remainder could be managed non-operatively. Of the referred patients, surgery was offered to 68% of them. The average wait for initial consultation decreased from 97 to 19 days in general orthopaedics, and 199 to 70 days in the orthopaedic spine clinic. The authors comment that expansion of primary care-based sports medicine services could relieve over-burdened orthopaedic departments of patients with conditions not requiring surgery. To that I say “hear, hear” as this is an issue in many countries around the world.

Later in the issue is a review article on injection treatments for patellar tendinopathy. The authors found 11 articles on seven different injection treatments including dry needling, autologous blood, high volume local anaesthetic, platelet rich plasma, sclerosis, corticosteroid injections and aprotinin. The authors comment that all seven different injection treatments seem promising but that steroid treatment often showed a relapse of symptoms in the long term. They comment that results should be interpreted with caution as few high quality studies have been conducted, and recommend further research.

The series on nutritional supplements continues with comments on melatonin and jetlag. It is unclear whether the main action is hypnotic or chronobiotic and they comment that mild heat loss by peripheral vascular dilatation following melatonin secretion may be the mechanism that initiates sleep. Certainly melatonin is widely used and there seem to be few side effects reported. Methylsulphonylmethane is an organic sulphur compound which is claimed to improve osteoarthritic pain. However, there is a lack of high quality evidence for many of the beneficial claims and the authors state that no definite recommendations can be

made on current evidence. Melamine is a substance that was much in the news in late 2008 as it was added to infant milk formulas. It is toxic to humans, even at low quantities, and the authors do not recommend its supplementation. Athletes should be aware of possible melamine contamination in protein-rich foodstuffs.

Finally, there was a systematic review entitled “Is exercise effective in promoting wellbeing in older age?” This was indexed on the Physiotherapy Evidence Database. The meta analysis pooled the results from four trials using the SF-36 mental health measure and a self-esteem measure. The review provided support for the role of exercise in improving the mental wellbeing of older people. The message is as previously - keep on keeping on.

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The Superior Labral Anterior to Posterior (SLAP) Lesion: An elusive pathology

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Abstract

Aim

The SLAP (Superior Labral Anterior to Posterior) lesion has become a popular clinical and radiological diagnosis. The purpose of this article is to review the international literature of the SLAP lesion and describe our experience with the diagnosis and surgical treatment of this lesion in a New Zealand practice.

Data Sources

We reviewed the available literature on SLAP lesions based on an index medicus Pubmed search and proceedings from relevant international meetings attended by the senior author (KDM). We included original articles, review articles and metaanalyses in our search. We also include data from a 2 year period of the senior subspecialist practice.

Data Extraction

The data from these studies was reviewed in combination with that from the senior authors practice and is included for illustration purposes with associated referencing.

Conclusions

The clinical presentation, imaging diagnosis and arthroscopic diagnosis are variable and imprecise. There is debate about the clinical relevance and treatment of SLAP lesions. There is no universally agreed clinical test to diagnose a SLAP lesion. Surgical treatment results range from very good to unsatisfactory. Patient populations differ from predominantly young baseball throwing athletes in USA and some Asian series, to older non throwing populations in European studies. These groups may differ from a New Zealand patient population. With selective indications, we achieved good results in our patients.

Key Words

Shoulder, Arthroscopy, Superior Labrum, Biceps, Repair

Introduction

In 1985² Andrews described the Superior Labral tear in overhead athletes. Snyder⁴⁷ coined the acronym SLAP (Superior Labral Anterior Posterior) lesion in 1990. Snyder's original classification is quite long and descriptive but can be summarised as follows:

Type 1 Marked fraying of the superior labrum with a degenerative appearance but no disruption of either the labral attachment or that of the biceps anchor.

Type 2 The biceps anchor and superior labrum are detached or partially detached from the superior glenoid. The biceps labral anchor is unstable.

Type 3 Disruption of the superior labrum with a "bucket handle" tear through the labrum with the peripheral labrum and biceps anchor remaining firmly attached.

Type 4 Bucket handle labral tear which extends into the biceps tendon. The remainder of the tendon and labrum are intact.

Other subtypes have been described^{33,36} but the Snyder Classification is most commonly used. There is general agreement that a Type 1 may be asymptomatic and not require treatment, with the surgical treatment option being debridement. Type 3 and 4 lesions can be recognised and treated at surgery. For Type 3 and 4 lesions treatments include excision of the bucket handle tear with or without repair of any residual biceps / labrum detachment, or tenodesis. The Type 2 lesion is more frequently reported, but more difficult to diagnose, with more diversity of opinion on treatment.

This article focuses on the Type 2 SLAP lesion. What is the normal anatomy? How do you get a SLAP lesion? How do you diagnose a SLAP lesion? What are the results of treatments internationally and locally? We attempt to answer these questions.

Anatomy

To understand the SLAP lesion, we must understand the normal anatomy. At the 12 o'clock position on the 'glenoid clock', articular cartilage

extends 4 or 5 mm over the superior margin of the glenoid, beneath the biceps and labrum. The superior labrum at the 12 o'clock position has a meniscoid margin which covers the superior extension of articular cartilage.⁶ Forty to sixty percent of the biceps insertion is into the supraglenoid tubercle with the remainder inserting into the labrum. Histologically, usually most of the biceps insertion into the labrum is into the postero-superior labrum.^{49,50} In the postero-superior 10 o'clock position, the labral attachment to the glenoid is meniscal in shape in 50% and rounded in 50%. By comparison the inferior labrum of the shoulder from the 4 o'clock to the 8 o'clock position is a rounded extension of articular cartilage with hyaline cartilage transitioning to fibrous tissue with a firm attachment.

There is a loose connective tissue attachment of the superior labrum to the glenoid which has elasticity. This construction indicates the natural mobility of the superior labrum and its close relationship with the long head of biceps (LHB). There are variations in the attachment of the mobile antero-superior segment of the labrum to the glenoid, including a sublbral foramen (present in 3.3%), middle cord pattern (present in 8.6%) and the Buford complex (present in 1.5%).⁴⁴ It is important to recognise these normal variants as surgical fixation of them may restrict external rotation.

The superior labral and biceps anchor complex function is a topic of debate. The labrum is widely accepted to act to deepen the concavity of the glenoid and widen its diameter improving concavity compression and excursion distance in rotation. In conjunction with the glenohumeral ligaments and bony anatomy it acts as a static constraint to instability. The superior labral complex (SLC) has a role to play in anterior stability and in experimental models SLAP lesions have been shown to increase the load on the inferior glenohumeral ligament (IGHL), reduce ability to resist external rotation and increase anterior and superior translation under load.^{35,40,45} The LHB itself has also

been shown to affect translation⁴¹ especially in mid abduction and in shoulders that are unstable for other reasons.¹⁹ The LHB is also thought to act as a depressor of the humeral head in the presence of rotator cuff tear.²⁶

Pathogenesis

Causes of SLAP tears can largely be divided into traumatic, attritional and degenerative. Traumatic lesions may occur in compression due to falls onto the outstretched limb, more commonly in the forward flexed position⁹ or with direct impact onto the shoulder. They can also occur in tension. Examples include using the limb to break a fall from a height, grasping at a heavy object below waist level or being pulled anteriorly as in waterskiing or drag ski lifts. These events cause sudden tension on the LHB tendon in the superior, inferior or anterior directions. The position of the humeral head in the glenoid at the time of impact or traction plays a role in the type of injury sustained.³

Attritional injuries may occur in overhead and throwing athletes. Considerable investigation of the pathogenesis of SLAP tears in these patients has been performed. One theory suggests that the primary pathology is a tight posterior capsular complex coupled with a relatively loose anterior capsule frequently seen in this group.⁶ This may be associated with increased humeral retroversion and may develop as an adaptive anatomical variation in throwers. The increased external rotation achieved in the late cocking phase of throwing is contributed to by postero-superior humeral migration at this point which allows greater tuberosity clearance. This has been borne out by cadaver studies.¹⁵ Such increased external rotation puts increased tension on the fibres of the LHB anchor resulting in a so called "peel back" lesion as the superior labrum complex displaces medially over the glenoid rim. The posterior vector produced by such motion puts the superior labrum at risk as it is weakest in this direction⁴⁶ and SLAP lesions may result.

Degenerative tears of the superior labrum are commonly seen in elderly and middle aged patients and may be the result of age related degeneration of the labral tissue rather than any specific event or attrition. They are not always symptomatic.

In summary, there is no specific history for a SLAP lesion. They may occur in different patient populations spontaneously, gradually or traumatically from a variety of mechanisms.

Diagnosis

The clinical presentation of SLAP lesions is extremely variable. Coexisting pathology may be present and more symptomatic. It is essential that the clinician determines the relative significance of the clinical presentation, imaging and operative findings.^{25,30} The patient may present with a history of gradual or sudden onset of symptoms, with a wide range of trauma mechanisms possible.

Symptomatic SLAP lesions cause pain, but the pattern is variable.²⁵ Pain, which may be sharp or dull, is typically felt deep inside the shoulder and may radiate either anteriorly or posteriorly mimicking anterior or posterior labral lesions or indeed acromioclavicular pathology. Exacerbation of pain can be expected with heavy lifting, overhead motion and pushing. Throwing athletes frequently report pain in the late cocking and early acceleration phase of throwing.

Weakness in these positions may also be a feature and development of labral tear related cysts and suprascapular nerve compression may cause infraspinatus weakness. Lack of trust in the shoulder and a sensation of giving way are also common especially in the overhead position. Patients may report mechanical symptoms of clicking and locking. Essentially the SLAP lesion may present with the symptoms of any other shoulder pathology and given the propensity for concomitant pathology it is difficult to delineate which is truly the cause of the patient's symptoms.

Clinical examination for SLAP tears is rarely decisive. It is important to examine the shoulder for other pathologies e.g. instability, rotator cuff disorders, capsulitis and pain syndromes. Wasting of infraspinatus may indicate suprascapular nerve compromise which may be secondary to compression by a sublabral cyst. These are frequently associated with SLAP tears. Posterior capsular tightness may be present and may be a contributor to the pathogenesis of SLAP tears. It is important to differentiate apprehension from pain and important also to remember that Bankart lesions in combination with SLAP tears are common, especially in those under 40.³⁰

While many special tests have been described^{27,28} concerns regarding the accuracy of these techniques have been expressed.^{24,34,43} Biceps tests, like Speed's test, may aid in suggesting pain in from the long head of biceps. Several tests to isolate the superior labral complex have been described including the Kim's biceps load tests (I and II), Liu's Crank test, Jobe's relocation test, McFarland's compression rotation test, forced shoulder abduction test and O'Brien's active compression test.^{16,27,28,32,34,37,39} These tests aim to provoke pain by stressing the superior labrum and long head of biceps. Perhaps the most commonly used is O'Brien's active compression test. O'Brien reported a sensitivity of 100% and a specificity of 98.5% but independent assessors have been unable to reproduce these results for this or other described tests.^{24,34,42,43}

Imaging plays a central role in the diagnosis of SLAP lesions. Magnetic resonance imaging (MRI) is the best modality to assess the labrum. Whether MRI arthrography or non contrast MRI is most useful in diagnosing SLAP lesions has yet to be resolved.¹⁸ We prefer MRI arthrography for examination of the labrum. The interpretation of pathology in this region is impeded by the variability of the local anatomy however specific findings suggestive of SLAP lesions include tracking of

contrast under the superior labrum, best seen on coronal cuts, and increased signal intensity in labral tissue and the biceps anchor on T2 axial cuts.^{22,48} It has been suggested that positioning the arm in the abducted and externally rotated position may improve diagnosis by displacing the lesion.^{8,23} Nonetheless even with arthrography one study reported that sensitivity may be as low as 89%, specificity 78% and accuracy 82%.²⁰

Arthroscopy is the gold standard for diagnosis but even at arthroscopy it can be difficult to agree on what is and what is not a type 2 SLAP lesion. Gozebie et al¹² surveyed the opinion of fellowship trained expert shoulder and sports surgeons. Members of the American Shoulder and Elbow Society and the American Orthopaedic Society of Sports Medicine were asked to comment on arthroscopic videos. There was high interobserver and intraobserver variability and difficulty distinguishing a Type 2 SLAP lesion from normal. Jia et al, however, reported good reliability in a small study group of experienced shoulder surgeons.²¹

Treatment

Type 1 SLAP lesions may need no treatment or debridement. Little has been written regarding outcomes of treatment of the less common Type 3 and 4 lesions. Few studies exist documenting the natural history of conservatively treated SLAP lesions.¹⁰ In cases with a concomitant rotator cuff tear, cuff repair with debridement of the SLAP 2 tear¹ or with tenotomy of the LHB¹¹ have better outcomes than repair. These studies involved patients ranging from 45-60 and over 50 years of age respectively. More recently Koh et al have shown no difference except in the appearance of a popeye sign between patients treated with tenotomy rather than tenodesis when treating biceps pathology identified at rotator cuff surgery.³¹

Reported results of surgical treatment of isolated Type 2 SLAP lesions with arthroscopic repair vary considerably. Some North American literature

reports favourable results. Morgan et al³⁶ reported 97% good and excellent results in 102 patients. The mean age of their patients was 33 years. Fifty-three were throwing athletes with an average age of 24 and all had dominant arm injuries. The other 49 were non throwers with a single traumatic event and an average age of 40. Twenty-nine patients had dominant arm injuries and 20 had non dominant pathology. Kim et al,²⁹ reported 94% good and excellent results in 34 patients in their Korean series. The mean age was 26. Eighteen were overhead athletes, 12 were contact athletes and 4 were not involved in organised sport. In a Japanese series of 40 overhead athletes,¹⁷ Ide et al reported 90% good and excellent results in 40 overhead athletes with a mean age of 24. Brockmeier and Altchek⁵ studied outcomes at 2 years of isolated SLAP type 2 lesion repairs in 47 patients with a mean age of 36 years. In total 28 of 47 were overhead athletes of varying levels. Overall 25 patients had a specific traumatic event that triggered their pain and 22 had an insidious onset with 20 of these being involved in overhead sports. Eighty-seven percent of patients in this study reported good or excellent results with 71% returning to pre-morbid level of sports participation. Having an identifiable traumatic event was associated with improved outcome in both subjective assessment and level of competition.

Conversely, poor results of SLAP lesion repair have been reported in Europe. The French Arthroscopy Society presented a multicentre retrospective study comparing repair of Type 2 SLAP tears to tenodesis. The mean age of their patients was 36 years, 52% had a traumatic, 30% had a progressive and 18% had mixed aetiologies. Thirty-three patients had a repair and 20 had tenodesis. In the tenodesis group 90% were satisfied or very satisfied, whereas only 57% of the repair group were satisfied or very satisfied ($p < 0.01$).¹⁴ In their non randomised prospective study, Boileau et al also published superior results for biceps tenodesis compared to SLAP repair.⁴ There were differences in the

ages of each patient group (37 in the SLAP repair group versus 52 in the tenodesis group) and small sample sizes (10 repairs and 15 tenodeses). The authors reported significantly better results in the patients with biceps tenodesis. Eighty percent of patients in the tenodesis group were satisfied compared with 40% in the repair group. Return to sports was also superior in the tenodesis group with 87% of athletes returning to their previous level of competition compared with 20% in the SLAP repair group. Eighty percent of patients in the SLAP repair group and 60% patients in the tenodesis group were overhead athletes. Overall 60% reported a specific traumatic event.

Despite these geographical differences it is generally recognised that overhead throwing athletes, especially baseball players have inferior outcomes after SLAP repair than other patients.^{5,13,17,29,30,51} It is also widely accepted that poorer outcomes are seen with SLAP repairs performed in the presence of other pathologies, such as rotator cuff tears. Debridement or tenotomy/tenodesis rather than repair is preferred in combination with treatment of the major pathology.^{1,11} Further debate exists about the repair of SLAP tears in older patients³⁸ but despite this recommendations lean towards repair in appropriate cases under 40 and tenodesis or tenotomy above this age.⁷

A New Zealand Perspective Patients and Methods

Over a two year period, between 1st November 2007 and 31st October 2009, of new patients attending the senior author (KDM), 457 had an MRI scan of the shoulder. Of these 148 (32.39%) had a report which included a SLAP lesion. During the same period the senior author performed 41 arthroscopic SLAP repairs. In 24 of these cases other significant procedures were performed concomitantly, usually arthroscopic stabilisations for glenohumeral instability, acromioplasties or less commonly rotator cuff surgery. Excluding these 24 patients and 4

other cases requiring treatment of Type 3 SLAP lesions left 17 cases where the repair of the Type 2 SLAP lesion was the isolated therapeutic procedure. In 5 of these 17 cases there was a paralabral cyst. There were 15 males and 2 females. The mean age at surgery was 35 years (range 19-51 years). All were funded by the Accident Compensation Corporation (ACC).

The injury mechanisms are listed in table 1. Most patients had already received physiotherapy and 4 patients had subacromial cortisone injections prior to referral to our practice. The mean time from injury to surgery was 25 months (range 4 months – 8 years). All had an MRI diagnosis of a SLAP lesion. All were repaired with a knotless anchor technique, using 1 postero-superior anchor in 12 cases and 2 anchors (1 antero-superior and 1 postero-superior) in 7 cases. Patients were asked to complete the American Shoulder and Elbow Society Score (ASES) pre operatively and 6 months post operatively. At 6 months patients were asked to report their satisfaction with the options being dissatisfied, not sure, satisfied and very satisfied. Patients were asked at 1 to 3 years follow up (mean 25 months) to once again rate satisfaction and also estimate the “percentage of normal” for their treated shoulder.

Table 1: Mechanism of Injury

Mechanism of Injury	Number
Fall (Soccer, Snowboard, Bike, Stairs)	6
Collision (Rugby, Soccer, Squash)	4
Combat	2
Heavy Lift	2
Road Traffic Accident	1
Repetitive Overhead	1
Repetitive Throwing	1

Results:

Sixteen patients completed the pre operative ASES score and 11 of these (69%) completed their 6 months ASES scores with an increase from 46 to 82 points. The highest score on this scale is 100 points. (Fig 1) Of those that completed their 6 month assessment,

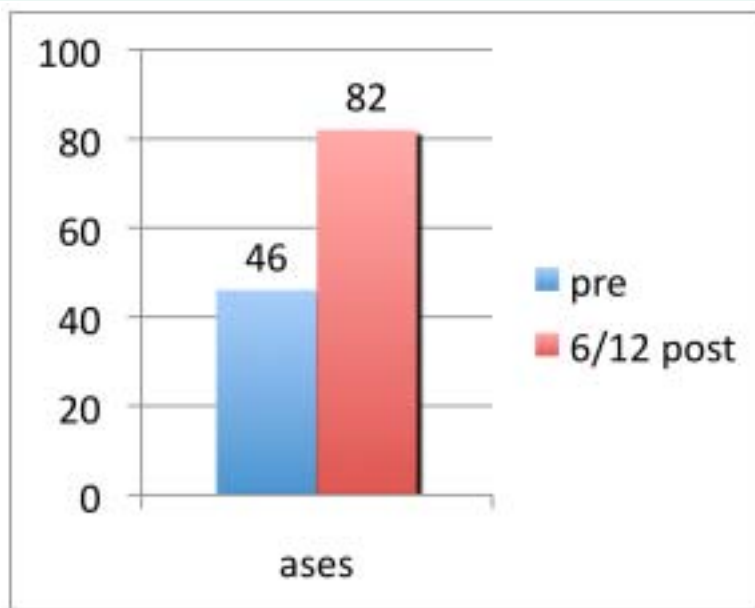


Figure 1: Pre-operative and 6 month Post-operative ASES scores.

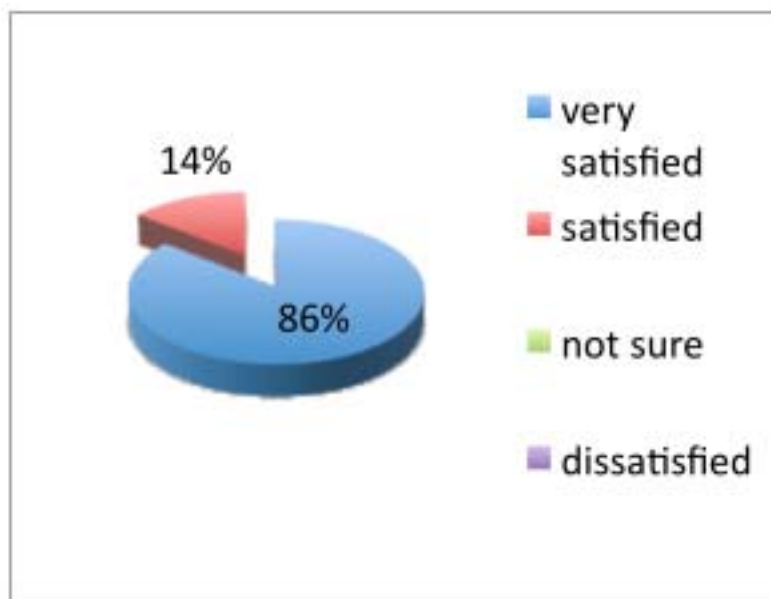


Figure 2: Satisfaction at minimum 1 year follow up.

82% were very satisfied with the remaining 18% satisfied. At a minimum of 1 year follow up, 14 out of 17 (82%) of our patients were contactable. Once again there was a high satisfaction rate. (Fig 2) The mean percentage of normal rating was 94% (85-100%).

One patient, who was not contactable for follow up, was experiencing pain with collisions at rugby. At the index procedure he was noted to have a

posterior labral tear that was debrided. He was offered further surgery with a view to posterior labral repair, but has subsequently left New Zealand. One patient had a reoperation. He was happy with his shoulder but more than a year post surgery he reported a painless graunching sensation in his shoulder. He had an arthroscopy at 22 months following the index procedure to exclude suture anchor prominence. At surgery a labral flap was debrided and the graunching has

not recurred. One patient had mild median nerve symptoms for several months following surgery. Neurophysiological studies suggested mild compromise of the median nerve in the infraclavicular region and the carpal tunnel. His symptoms resolved adequately without intervention. In 2 patients slow progress was reported in their notes. One with aching at 1 year reported he was 'very satisfied' and rated the shoulder as 90% normal at final follow up. Another with slow progress recorded at 3 months post surgery reported he was 'very satisfied' and rated the shoulder as 95% normal at final follow up.

Illustrative Case:

A 20 year old elite swimmer (Olympic trialist) and surf life saver hit a lane rope swimming backstroke and experienced sudden severe pain in his right shoulder. His pain was ongoing despite rest and physiotherapy. He was unable to swim or throw. He had sharp pain reaching out. The shoulder did not feel unstable to him. On examination he had a good range of movement, with pain on internal rotation in abduction and pain on the apprehension Test. O'Brien's compression test was negative and Speed's biceps test was positive. Prior to referral he had cortisone subacromial injection without improvement and had seen a physiotherapist. His MRI reported a SLAP lesion. Figure 3 demonstrates the appearances of the superior labrum and biceps after debridement. Figure 4 demonstrates the postero-superior repair with a single knotless anchor. Post operatively he wore a sling for 4 weeks then started physiotherapy. At 3 months from surgery he was comfortable for activities of daily living and had regained near full range of movement (Figures 5) and rehabilitation included gym strengthening. His ASES score improved from 47/100 preoperatively to 98/100 at 6 months postoperatively. He was able to swim and returned to work as a surf lifeguard. He reported he was 'very satisfied' with the outcome on his 6 month post operative questionnaire.



Figure 3: Arthroscopic photograph of the debrided type 2 SLAP lesion demonstrating abnormal mobility of the superior labral and biceps complex.



Figure 4: Superior labrum and biceps anchor following SLAP repair.

Discussion

There are a wide range of proposed aetiologies for SLAP lesions, with an equally wide range of symptoms described. No clinical presentation or test will allow the clinician to diagnose a SLAP lesion with certainty. The

anatomy of the superior labrum is variable. There is a normal recess beneath the biceps insertion and the postero-superior labrum may have a meniscoid shape. There is some mobility of the normal superior labrum and biceps. It can be difficult to

distinguish a Type 2 SLAP lesion from normal anatomy on MRI scans and even at arthroscopy. In our practice over a 2 year period, it was common for a patient's MRI scan to include a report of a SLAP lesion with approximately one third of MRI scans reporting a SLAP lesion, with or without other pathologies. In this time period we performed just 41 SLAP repairs. SLAP repair was an isolated procedure in only 17 of these.

The results of surgical treatment in the literature are variable. We believe that part of this variation may relate to differing patient populations. We have described good results in a New Zealand practice but wish to stress how selective we are in considering whether an MRI diagnosis of a SLAP lesion is clinically relevant.

If a patient has a traumatic event with onset of mechanical symptoms, then labral abnormalities may be relevant. With increasing age, however, degeneration of the labrum may occur and it is difficult to determine what may be normal for age and whether the labrum is a cause of symptoms. We consider most other pathologies e.g. instability, rotator cuff tears, capsulitis or regional pain syndromes to be dominant in causing symptoms. Not all labral tears remain symptomatic and a period of conservative treatment including posterior capsular stretching and scapular stabilising is worth a trial in most cases. Vigilance for suprascapular nerve compression is however important. This can often occur in patients with a paralabral cyst related to SLAP tears and requires more urgent surgical intervention.

Our guidelines for selection for surgical repair of a SLAP lesion are:

- Traumatic onset with mechanical symptoms
- Symptoms ongoing for more than 6 months
- Patient age usually less than mid 40's and physically active
- No capsulitis, regional pain syndrome or other dominant



Figure 5: Forward elevation 3 months post SLAP repair right shoulder.

pathology such as a rotator cuff tear.

- An MRI arthrography proven SLAP lesion with the presence of a paralabral cyst an even clearer indication.
- Confirmation of the SLAP lesion at arthroscopy.

We prefer repair with 1 postero-superior anchor taking care not to restrict antero-superior labral mobility to avoid stiffness in external rotation.

Conclusion

Diagnosis of SLAP lesions can be difficult. Careful correlation of clinical presentation and diagnostic

tools is essential to avoid poor surgical outcomes. Use of a well devised algorithm for treatment can be of use in identifying which patients will benefit from which intervention, but definitive indications are yet to be determined.

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Nutritional considerations and challenges for flat racing jockeys

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Abstract

Flat race jockeys need to maintain a low body mass that allows them to meet the weight handicaps assigned to the horses they are riding. This review examines the anthropometric characteristics, chronic and acute weight management and dietary practices jockeys employ to maintain or make weight. It was found that most jockeys are small in stature and have low total body mass and body fat levels. Typical means of manipulating body mass include food/fluid restriction, saunas, hot baths, diuretics, laxatives and appetite suppressants. These practices place jockeys at increased risk of heat related illness during race meeting and even increase the likelihood of falls and are therefore discouraged. Reported energy intakes are indicative of restrained eating behaviours and likely increase risk of low energy availability and poor bone health. The reported carbohydrate intake is unlikely to meet the demands of training and competition. It is recommended that jockeys target a hydrated body mass that is within 2% of the minimum weight they perceive necessary to be able to maximise riding opportunities. A long term approach needs to be made to weight loss, with moderate energy restriction and appropriate exercise. Acute weight loss for competition should be achieved via use of a low residue meal plan and moderate fluid and sodium restriction. Replenishment of carbohydrate stores and minimising the fluid deficit should be the focus of dietary food and fluid intake during race meetings.

Key words: Low energy availability, weight making, dehydration, heat stress, jockeys

Introduction

“In horse racing it is widely accepted that the physiological status of the jockey is secondary to that of the equine athlete”¹⁸(p66)

Horse racing is one of the most popular sports in the world, enjoyed by billions of people worldwide. However, as this quote attests, it is the health and conditioning of the horse that is of primary importance.

Unique among weight category sports, the weight a jockey must attain for a given race is set by the weight-handicap allocated to the horse, which is based on ability or age.⁴ For each race in which they ride, jockeys will receive a base monetary amount, plus a percentage of any prize money on offer. The incentive

to achieve a weight that will maximise riding opportunities, coupled with weight handicaps that are often well below the jockeys natural weight, has seen many jockeys adopt “extreme” weight making behaviours that are contrary to both health and performance.²¹

While there have been studies examining the physical characteristics^{7,8} dietary habits^{10,11} and weight making practices^{10,11} of jockeys, there currently exists no published guidelines on the nutrient requirements of jockeys in training and competition. This review examines some of the existing literature on flat racing jockeys, before making dietary recommendations for training and racing. Information presented in this review with regards racing regulations for competition is specific to jockeys competing in

Australian race meetings, unless otherwise stated.

Training

Jockeys typically ride early morning track work 5-6 days a week, lasting between 3-5 hours, which is often followed by trial or official race meetings later in the day. During track work, jockeys will ride multiple horses, each of which they will be required to “work” according to the trainer’s instructions.¹¹

Most studies suggest that jockeys will do some form of training outside of their track and racing commitments. Most of the cross training activities are aerobic in nature, eg, running and riding.^{14,10} Rather than being driven by motivation to improve specific “jockeying performance”, these cross training activities are often used as a means of

aiding in the maintenance of riding weight or as an acute weight making strategy.²¹ Concomitantly, it has been reported that jockeys are reluctant to undertake any resistance training for fear of gaining extra muscle mass.⁹

Competition

Race meetings are held all year round, meaning jockeys have the opportunity to ride as often or as little as they like. By extension, the timing and length of their “season” and “off-season” will vary. While one study reported that jockeys will average of 344 rides a year, the study found this did range from 10-1200.⁷

Jockeys can ride in up to 2-4 race meetings a week, in which they will ride between 1-8 races over a period of 5-6 hours. Race durations range from 1-3 minutes, with the length of time between races typically 30-40 minutes¹¹. Jockeys are required to weigh-in no later than 45 minutes before each race, while those on horses that earn prize money, along with the next best finisher, have to weigh in directly after the race as well⁴. Race meetings are held in a wide range of environmental conditions.

Physiological Requirements of Training and Racing

The work of Trowbridge et al¹⁸ represents the only empirical data on the physical

demands of horse riding. They studied seven jockeys during fifteen race meetings. They found average heart rates ranged from 136 to 178 beats·min⁻¹ (peak - 162-198 beats·min⁻¹) and were always above 80% of the jockeys’ measured maximum during races. Mean lactates, taken soon after races had finished, ranged from 5.1 – 11.7 mmol · l⁻¹. The authors noted that lactates tended to be higher in those races where the horses had a chance of a top placing, suggesting that as the horse works harder, the greater the physiological strain placed on the jockey.

Though only involving a small number of riders, this study suggests jockeys require a high level of aerobic endurance to be able to tolerate long periods in the saddle, while at the same time have the anaerobic capacity to be able to produce high intensity efforts during races and “fast” track sessions. The fact that thoroughbred horses can weigh up to 500 kg and reach speeds up to 60 km · h⁻¹²¹ suggests jockeys also require a degree of upper body functional strength to be able to control the horse.

Optimal Physique

The optimal physique for a jockey is one that simply allows him or her to maximise riding opportunities. While the minimum weight for all races is 43.5 kg⁴, it has been noted that weight handicaps are usually

between 52-58 kg. As this is inclusive of the riding saddle and accessories, this means that in effect jockeys need to be half to one kilogram lighter than this. Therefore jockeys need to maintain a weight below 55 kg to allow them to race regularly.¹⁴

Table 1 summarises studies that have examined the anthropometrical characteristics of race jockeys. It shows that they are typically small in stature, have low body mass index (BMI) and low skinfolds or percentage body fat. Hill and O’Connor⁸ conducted somatotype profiling of jockeys, classifying them as ectomorphic-mesomorphs, indicating that mesomorphy (musculo-skeletal robustness) is dominant while ectomorphy (linearity, slenderness) is more dominant than endomorphy (relative fatness).

Weight Management Practices of Jockeys

The studies that have described the weight making practices of jockeys have found most regularly use one or more chronic or acute strategies to maintain or make weight.^{7,11,14,10} Reported methods include those designed to induce a reduction in body fluid levels (restricted fluid intake, saunas, exercising while wearing sweat suits, hot baths, diuretics), a reduction in fat/muscle mass (energy restriction, appetite suppressants) or a reduction in

Table 1: Anthropometrical data of flat race jockeys (Mean ± SD)

Publication	Study Population	Body Mass (kg)	Height (cm)	BMI (kg.m ²)	% Body Fat	Sum 7 Skinfolds
Hill et al (1997) [^]	65 professional jockeys	52 ± 3.2 (42-60) [^]	161 ± 6.4 [^]	20.1 ± 1.4 (17.7 - 23.6)	Not reported.	Not reported.
Hill and O’Connor (1999) ^{^^}	n = 8 (6 males, 2 females)	52.6 ± 1.9 kg	161 ± 3	20.4 ± 1.0	9.9 ± 3.9	Not reported.
Hill and O’Connor (2000) (M)	31 professional jockeys (28 males, 3 females)	51.8 ± 2.0 kg**	161.1 ± 5.4**	20.0 ± 1.3** (17.5-22.9)	7.5 ± 1.8 (4.6%-10.9% (M)) 15.3 ± 0.6 (14.9-15.9)	53.0 ± 11.3 ^{^^^} 64.0 ± 3.9 ^{^^^} (F)
Leydon and Wall (2002)	6 males 14 females	52.8 ± 2.4 (M) 49.3 ± 3.4 (F)	162.3±4.4 (M) 156.2±4.0 (F)	20.1 ± 1.5 20.2 ± 1.5	11.7 ± 2.9 (M)* 23.6 ± 3.8 (F)*	Not reported.
Moore et al (2002)	116 senior and apprentice jockeys male 91 female 25	53 ± 0.4 (M) 51 ± 0.6 (F)	162 ± 8.0 (M) 157 ± 1.0 (F)	20.3 (M) 20.5 (F)	Not reported.	Not reported.
Pruscino et al (2008)	7 male jockeys	53.9 ± 2.7	164.1 ± 4.0	20.0**	Not reported	32.9 ± 5.6
Warrington (2009)	27 professional jockeys (17 flat and 10 national hunt jockeys)	53.1 ± 4.1	160.0 ± 0.1	19.88 ± 1.26	8.99 ± 2.48 7.88 ± 1.73 (Equation based on sum 7 skinfolds)	44.3 ± 10.2

[^] Self reported values via questionnaire.

^{^^} Did not provide separate figures for males/females

^{^^^} Sum of 9 skinfolds

* Measured via DEXA scan

** Calculated from the mean height and weight provided

gastrointestinal contents (laxatives, vomiting). While similar weight management behaviours have been described in other weight category sports,¹ a major concern is that jockeys have to make weight on a weekly basis, rather than just for a few major competitions during the year. The health and performance implications of some of these practices are described briefly below (See Fogelholm (1994); Walberg-Rankin (2006) and Sawka et al (2007) for more detailed reviews).

Energy Restriction

The findings of the dietary surveys (See Table 2) would suggest many jockeys regularly use chronic or acute energy restriction to manipulate body mass. Leydon and Wall¹¹ suggested the reported values for energy intake in their study were well below the jockeys' estimated requirements, which is consistent with the findings of Hill et al,⁶ who found that the average reported total energy intake was below the average calculated Resting Metabolic Rate (RMR), measured by indirect calorimetry (6190 ± 565 kJ; range 5535 – 7186 kJ). Periods of energy restriction will lower RMR¹, which in effect will lower the energy prescription required to help jockeys maintain their desired weight. Further, chronic energy restriction is also associated with inadequate intake of carbohydrate, protein and a range of micronutrients¹. Two studies^{10,11} also reported that intakes of a number of micronutrients, including calcium and iron, to be below the Recommended Daily Intake (RDI).

When the average energy intakes reported in these studies are expressed kJ/per kilogram body mass, it suggests that many of the jockeys are at risk of periods of low energy availability, which is known to result in a suppression reproductive function and bone formation (See Manore et al (2007) for review). The only study to examine menstrual status in jockeys found that 36% of the female jockeys in their study reported menstrual abnormalities. Interestingly, a recent study of male professional jockeys found no evidence of hypogonadism, but found jockeys had whole-body osteopenia and 12% were osteoporotic. Leydon and Wall¹¹ found that almost half of the jockeys (44%) in their study to be osteopenic. It has been suggested that the high prevalence of bone disorders and lower than expected bone mineral density (BMD) for their age was likely due to the prolonged periods of energy deficit, combined with other poor lifestyle factors, eg, smoking, and the small oestrogenic stimulus provided by horse riding.²¹ This is of special concern for jockeys, given the high risk nature of the sport.

Using the Eating Attitudes Test-26 (EAT-26), both King and Mezey⁹ and Leydon and Wall¹¹ classified 20% of the jockeys as suffering from disordered eating, while another study³ found jockeys scored higher on the EAT-26 when undertaking acute weight loss compared to times when they were not. As the EAT-26 is known to underestimate the true prevalence of disordered eating in athletes,¹ true prevalence of disordered eating amongst jockeys may be much greater. It has also been shown that scores for depression,

anger and fatigue were significantly increased, and scores for vigour significantly reduced, when the jockeys were undergoing rapid weight loss compared to times when they were not.³

Fluid Loss

Any degree of dehydration will reduce the body's ability to produce sweat, which in turn increases the risk of heat injury.²⁰ In addition, dehydration of >3%, or less extreme losses combined with heat stress, will negatively affect cognition, mood and mental status during exercise.¹⁷ Therefore, the high intensity nature of horse racing, coupled with the often severe fluid losses jockeys will incur to make weight, reported to be as high as 11% of total body mass,¹⁰ will not only predispose jockeys to suffering heat illness, especially during warm environments, but may also increase the likelihood of falls.

Two studies which have measured the hydration status of jockeys have both shown jockeys to have greater average upon waking Urine Specific Gravity (USG) values on race days when compared with non-race days.^{15,21} Further, Pruscino¹⁵ noted that despite a relatively modest average acute weight loss (2.1 ± 0.4%) and the cool to temperate (9-20°C) environment in which they were racing, found that mean core temperatures in jockeys still rose 1.2°C above resting values after five races.

Dietary Recommendations for Training

The dietary goals of professionals jockeys may be broadly described as to:

Table 2: Reported dietary intakes of flat race jockeys (Mean Daily Intake ± SD)

Publication	Jockey Pop	Survey Method	Energy		CARBOHYDRATE			PROTEIN			FAT	
			kJ	kJ/kg	g	g/kg	% of Overall Energy Intake	g	g/kg	% of Overall Energy Intake	g	% of Overall Energy Intake
Labadarios et al (1993)	93 senior male jockeys	7 day food diary	8100	153	219	4.1	43.4	77	1.5	15.2	74	33.9
Hill et al (1999)	professional jockeys (6 male, 2 female)	7 day weighed food diary	5781±828 (4734-6964)	112±15.1	180	3.4	50±9	65	1.2	18±3	46.9	30±5
Leydon and Wall (2002)	6 males 14 females	7 day weighed food record	6769±1339(M) 6213±1797(F)	128(M) 126(F)	179±56(M) 174±50(F)	3.4(M) 3.5(F)	45.5±5.5	58±12 47±16	1.1(M) 0.9(F)	15±4.5(M) 13.5±3.0(F)	54±23(M)	34.5±7.2

- 1 Provide sufficient carbohydrate to support the aerobic and anaerobic energy production during track work and races.
- 2 Maintain a weight that maximises riding opportunities and promotes maintenance of RMR and lean body mass.
- 3 Provide an adequate energy intake to ensure normal physiological function and avoid consequences of low energy availability.
- 4 To ensure adequate intake of all micronutrients, especially calcium and iron.

Long Term Weight Management

Jockeys should be encouraged to adopt dietary and exercise habits that allow them to achieve a hydrated body mass that is within 2% of the minimum weight they perceive necessary to be able to maximise riding opportunities. This will help ameliorate the need to adopt chronic energy restriction and “extreme” acute weight making strategies known to have deleterious effects on health and performance. Extremely low levels of body fat levels should be avoided, as this will reduce cushioning of internal organs,¹ a major consideration of jockeys given the risk of falls during racing. When working with jockeys, health professionals, especially dietitians, always need to be sympathetic to the incentive of jockeys to achieve a weight well below that considered healthy to ride a particular horse for a given race.

Assessment of energy availability should be undertaken with jockeys. Table 3 provides an example of a calculation of energy availability for a female jockey. Values 125 kcal/kg FFM day⁻¹ (30 kcal/kg FFM⁻¹ day) are indicative of low energy availability.¹²

Carbohydrate Requirements

Data from Trowbridge et al,¹⁸ coupled with reports of training volumes undertaken by jockeys, would suggest they require at least 5-7 g/CHO/per kg/Body mass to support the aerobic and anaerobic demands of training and racing, although a target of 3-5 g/CHO/per kg/Body mass may be more appropriate on light training/rest days. Both these target ranges provide the jockey with the opportunity to adjust carbohydrate intake to reflect their daily training load. Interestingly, two studies^{6,7} which examined the carbohydrate intakes of jockeys reported low intake values, likely reflecting the jockeys’ desires to restrict overall energy intake (see Table 2).

Protein Requirements

Jockeys should target a protein intake of 1.2-1.6 g·kg⁻¹·day⁻¹. This level of intake will promote better maintenance of lean body mass, especially during periods of energy restriction and/or inadequate intake of carbohydrate. The higher diet induced thermogenesis (DIT) of protein¹³ and increased satiety associated with protein rich food provides additional support for jockeys to aim to achieve the higher end of this intake target.

Calcium and Iron

Athletes following a low energy diet are at risk of suboptimal intake of a range of micronutrients, especially iron and calcium. Adequate intake of calcium is especially important for jockeys given the role it plays in achieving and maintaining optimum bone density.

Dietary Recommendations for Competition

Preparation for Race Day

The dietary goals of jockeys in the lead up to race meetings are to achieve the weight required to ride the horses for which they have been assigned, ensure

adequate carbohydrate stores and be well hydrated. For jockeys that do not need to make weight, they should aim to consume 5-7 g/CHO/per kg/Body mass the day before to promote adequate glycogen stores. Those with a full schedule of rides and that have undertaken early morning track work on the day prior, should aim for the high end of this range, while those with a more limited schedule of rides, the lower end.

If the jockeys need to make weight for rides, they should be encouraged to adopt a low residue meal plan, in combination with a mild fluid and salt restriction. Such a plan, based around low fibre, carbohydrate rich foods, may allow jockeys to achieve a 300-400 g decrease in body mass,⁴ while ensuring the adequate carbohydrate and energy intake to promote optimal performance. The amount of fluid restriction should be guided by the absolute amount of weight the jockey needs to lose. It should be limited to the last 24 hours prior to the race meeting, given the negative impact hydration may have on riding performance.

Day of and During the Race Meeting

The goal of nutrient intake on the day of a race meeting is to ensure adequate carbohydrate stores and minimise fluid deficit.

The requirement to weigh in after races and the often short amount of time between each, act as obvious barriers to the jockey practicing optimal nutrition practices. Therefore, the decision on the type and volume food/fluid consumed in between races should be guided by their post race weight and the weight they need to make for subsequent rides. While needing to be mindful of the overall weight of the foods and fluids, jockeys should aim to choose carbohydrate rich options that will aid in re-fuelling. For those jockeys for whom weight is not a concern, they should consider consuming sodium containing fluids and/or be encouraged to consume fluids with foods. For those jockeys for whom weight is a concern, they should aim to consume low sodium fluids in between meals, and be more aggressive with their fluid intake once their riding commitments have finished.

Table 3: Example of energy availability for a female flat riding jockey.

Energy Availability = Energy Intake - Training Energy Expenditure

*Body mass: 49.3 kg

*% Body fat: 23.6

Lean body mass: 37.7 kg

*Mean energy intake: 6213 kJ

Energy cost of daily training (3 hours track work): 4000 kJ

Energy availability: 6213 - 4000 = 2213

Energy availability: 2213 / 37.7 = 58 kJ per kilogram fat free mass (low energy availability)

*Mean value reported from the study of Leydon and Wall (2002)

Recovery

The goal of recovery after race meetings is to refuel carbohydrate stores, promote repair of damaged muscle tissue and promote rehydration. Intake of 0.5-1 g/CHO/per kg/Body mass and 10-20 g of protein in the form of a meal or snack soon after the race meeting serves as a useful target.¹ Jockeys should aim to consume fluid 125-150% of estimated sweat losses to promote optimal rehydration. In the situation in which jockeys are unaware of their sweat losses, they should be encouraged to have available a range of fluids that will promote voluntary intake and consume these with their recovery meal/snack. These targets are especially important if the jockey has track work commitments the following day.

Conclusion

To optimise riding opportunities many flat race jockeys adopt extreme weight strategies that are contrary to both health and performance. In this sense, the ends, ie, achieving a weight that allows them to ride a horse in a race, justifies the means, ie, extreme weight loss practices such as severe energy restriction and use of diuretics/laxatives. Studies examining the dietary practices of jockeys provide evidence of restrained eating behaviours, compromising overall energy intake and that of key macro and micronutrients.

It is recommended that jockeys take a long term approach towards their weight management, targeting a hydrated body mass that is within 2% of the minimum body mass they perceive necessary to be able to maximise riding opportunities. The target weight must be one that can be maintained without the need for severe energy restriction and/or excessive exercise behaviours. Acute weight loss for competition should be achieved via use of a low residue meal plan and moderate fluid and sodium restriction. Replenishment of carbohydrate stores and minimising the fluid deficit should be the focus of dietary food and fluid intake during race meetings.

Key Areas for Future Research

- Examine the impact of pre-cooling interventions on physiological strain and subjective markers of performance.

- Investigate the energy cost of jockeys during track work and races.
- Examine the effects of the different acute weight making strategies used by jockeys on objective and subjective markers of performance.
- Identify the physiological variables that impact on the performance of the jockey.
- Examine the typical sweat rates of jockeys during training and racing in a range of environmental conditions.
- Investigate the health implications of repeated use of acute weight making practices on long term health.

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Acute ocular trauma: Recognition and management in the sports arena

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Introduction

Ocular trauma contributes significantly to rates of visual impairment and blindness worldwide, and is the leading cause of unilateral vision loss.¹ In developed countries, sport and recreational activities account for up to 25% of all ocular injuries.²⁻⁴ Remarkably, sports related ocular injuries are often more severe than other causes of ocular trauma (such as occupational) and are therefore more likely to require hospitalisation and cause long term visual impairment.^{3,5,6} For children, sport and recreational activities account for more than 50% of all ocular injuries, and thus children are disproportionately affected and at greater risk than the adult population.^{7,8} Consequently, the lifetime impact of ocular trauma is often far greater for children than for adults.

While there is relatively limited global and regional data available on the rates of eye injuries associated with sports, ocular injuries in Australia predominantly occur from badminton, squash, Australian rules football and cricket.³ In contrast, North American sports that account for the most eye injuries include basketball and baseball,⁹ while soccer (football) and squash accounted for the majority of injuries in Scotland.² Interestingly, the majority of injuries requiring admission from racket sports were due to contact with the racket rather than the projectile itself.

The topic of ocular health is often not well taught, and is perceived as a complex and daunting subject. While rare, the appropriate recognition and management of ocular injury is critical; this article aims to provide a practical guide for practitioners working with athletes in assessing, and appropriately managing acute ocular injuries on the sports field.

Rapid Ocular Assessment in the Sports Arena

The goal of the initial ocular assessment is to determine the nature and severity of the injury. This may be assisted by having the appropriate equipment in your kit. (*Table 1*)

In a suspected ocular injury, as with any injury, assessment should begin with a concise history.

- 1 Mechanism of injury; consider the circumstances, severity and likely objects involved. Is this likely to be a blunt injury versus an open globe injury? An open globe eye injury (where an immediate referral to an ophthalmologist or emergency department is warranted) may be suspected by history alone if secondary to flying objects (especially small objects with high speeds) or motor vehicle accidents, and may present with only reduced vision or minor signs on examination.
- 2 Vision is the best measure of ocular health: has vision changed since the injury? Ask if contact

lenses are currently being worn as a lost lens may simply account for the visual impairment.

- 3 Is there pain? What is the nature of the pain and where is its location. A deep aching pain is suggestive of a serious injury, while superficial pain and photophobia are commonly associated with a corneal foreign body or abrasion and is relieved by local anaesthetic drops.
- 4 Previous ocular history such as previous ocular injury or ocular surgery, laser surgery and/or vision loss (slightly decreased vision in a patient's previous lazy eye may not be the result of an injury).

The ocular examination after acute injury is determined by the severity of the injury. A quick glance suggesting an open globe injury (ie, a deflated globe, asymmetrical pupil or signs of herniated tissue) requires no further evaluation but immediate referral.

1 Visual Acuity

If the athlete is unable to open their eyes due to pain, instilling a drop of anesthetic (amethocaine or lignocaine 1%) can assist. If the eye becomes comfortable, the problem is likely originating from the ocular surface. Use a Snellen chart or reading chart to test the vision (alternatively if no eye chart is available, using any print is fine) and record the distance from which it can be read. With the advent of smart phones, various applications

are available that can help in assessing vision that remove the need to carry an eye chart. If nothing can be seen on an eye chart, counting fingers should then be tested. Visual loss may be graded into mild, moderate (visual acuity worse than 6/12) and severe (counting fingers or less). The exact degree of vision is less important than gaining an overall appreciation for the severity and persistence of vision loss.

Any reduced vision, without an obvious cause (ie, a corneal abrasion), that is not resolving should be considered a red flag for serious ocular injury.

2 Examine the Globe and Pupil

(see Figure 1)

When examining the globe, always open the lids from the bony orbital margins above or below the eye to avoid applying pressure to the globe, particularly until an open-globe injury can be excluded (see Figure). Ensure the eye is not deflated, check the pupils for size and shape and compare with the opposite eye. A pupil dragged to one side indicates either a serious contusion or open-globe injury as the tissue in the eye has either been torn or is herniating out of the globe. Compare the pupils for size and reaction to light including direct and consensual responses, as well as

looking for a relative afferent pupillary defect (RAPD), which can be assessed by the swinging light test.

Pupil responses:

- i Direct: Pupil constricts with light stimulation
- ii Consensual: The pupil constricts with light stimulation from the other eye

Swinging light test: In healthy eyes, when the light is moved from one eye to the other eye, both remained constricted due to the direct and consensual responses.

RAPD: Both pupils dilate when the light is moved from the unaffected eye to the affected eye, indicating a problem with the sensory (afferent) component of the light reflex in the affected eye.

Look at the front of the eye for blood in the anterior chamber (hyphaema) located between the cornea and the iris. Note that while textbooks typically show a fluid level in the eye, initially the anterior chamber of the eye may simply appear hazy. Using an ophthalmoscope, quickly check for a red reflex and compare with the other side, absence of which may indicate bleeding deeper within the eye. Assess the surrounding tissue including the

sclera, while maximising the view by examining the eye turned in various directions. Take care in examining for dark masses underlying the conjunctiva which may signify globe rupture and prolapsed intraocular tissue that has herniated through the sclera (white of the eye) (see Figure 1). Look for any lacerations of the conjunctiva or sclera as this will require urgent ophthalmological review. Look for foreign bodies on the conjunctiva or cornea, remembering to always evert the lids when examining for a foreign body. While subconjunctival haemorrhages are usually benign, take care with any that encompass 360 degrees around the cornea or where the posterior aspect cannot be visualised, as this may indicate a more serious injury such as globe rupture or orbital fracture.^{10,11}

3 Cornea

Examine for corneal abrasions with fluorescein and a blue light either on an ophthalmoscope or alternatively a torch with a blue filter. If an abrasion is present, evert the upper lid to check for a foreign body. This can be done by having the athlete look down and applying light pressure with a cotton bud approximately 1 cm above the lash line. The eyelashes can then be gently pulled away from the globe and over the cotton bud allowing the lid to flip on itself. (See Figure 2)

4 The Eyelids

Look at the lids for any contusions or lacerations. There are four main anatomical regions to consider; the skin around the medial aspect of the eyelids, where the tear ducts drain into the nose; the superotemporal area of the lids housing the lacrimal gland; lacerations involving the lid margin; and lastly lacerations with protruding fat or associated ptosis. These areas will require careful examination and surgical repair. Always be cognisant to lid lacerations or superficial injuries that may be associated with an underlying laceration to the globe. Do not remove any herniated fat.

5 Eye Movements

Finally, assess for any restricted eye movements, particularly upgaze which



Figure 1 Safely opening the eyes.



Figure 2 Everting the eyelids.

may signify an orbital floor fracture. Look at the symmetry of the two eyes, a sunken eye or any forward displacement of the eye that may indicate a bleed behind the globe. Check for tenderness around the orbit as well as sensation over the cheeks and forehead (orbital fractures can disrupt the nerves which supply sensation to the forehead and cheeks leading to anaesthesia).

Management of Acute Ocular Trauma in the Sporting Arena

The majority of minor injuries in Australasian sports where vision is minimally impaired or improving, are likely to be abrasions and contusions that can be managed conservatively by a medical practitioner. However, following a careful assessment, referral is indicated with the following indications:

- 1 Lacerations or penetrating injuries of the lids or globe

- 2 Restricted eye movements (ophthalmoplegia) or, a black eye with an associated subconjunctival haemorrhage without a visible posterior limit
- 3 A sunken or protruding eye
- 4 Abnormal pupil size or shape compared with the opposite eye
- 5 Hyphaema
- 6 Absent red reflex/asymmetrical red reflexes
- 7 Prolapsed tissue (appearing as a dark mass under the sclera)
- 8 Photophobia without a corneal abrasion or foreign body
- 9 Any degree of visual loss not improving over 1-2 hours, including mild visual loss without an identifiable cause

If at any stage open-globe injury is suspected, including rupture from a blunt injury/contusion, intra ocular foreign body or penetrating injury, immediately cease the examination, place a non-pressure protective plastic shield over the eye, provide oral analgesia and anti-emetics, keep the

athlete nil by mouth, and refer immediately to an ophthalmologist/emergency department. A complete exam is not required on the sports field and all topical ointment and drops should be avoided. If no ophthalmologist is available, oral antibiotic (we recommend ciprofloxacin 500 mg b.d [note that ciprofloxacin is relatively contraindicated in some elite athletes due to a suspected association with tendinopathy or rupture]) and tetanus prophylaxis should be initiated.¹² A conjunctival laceration may indicate an underlying scleral laceration, therefore the athlete should not return to play but instead be referred for slit lamp evaluation that day.

For corneal abrasions consensus suggests that there is no benefit in patching the eye when considering either healing time or pain. Patching the eye may actually decrease the external oxygen supply to the surface of the eye.^{13,14} Instead, manage the injury with intensive topical antibiotics (eg, Chloramphenicol drops or ointment) and dark glasses to manage the photophobia is sufficient. Isolated corneal abrasions do not require a referral unless visual acuity or pain fail to resolve within 1-2 days. Contact lenses should be avoided until the eye feels normal for one week and the lesion is healed.

Corneal foreign bodies should be removed with a moistened cotton bud and the upper lid everted to exclude a retained foreign body. Note that many symptomatic foreign bodies are microscopic, and running the cotton bud over the everted lid may remove these and improve symptoms. If you are unable to see the foreign body and symptoms persist, or you suspect it has penetrated the eye, do not attempt further removal and refer to an emergency department.

While many uncomplicated blunt trauma injuries will not result in clinically significant damage or require the athlete to stop play, significant ocular contusion can lead to hyphaema, traumatic iritis or damage to the retina and associated

structures.¹⁵⁻¹⁷ Signs of serious contusion requiring immediate referral include photophobia, decreased vision, a deep pain not relieved by local anaesthetic, a hazy appearing anterior chamber or blood aqueous level, and absent red reflex or asymmetrical pupil responses. Appropriate initial management includes oral analgesia (avoid oral NSAIDs due to the risk of re-bleeding) and cycloplegics which dilate the pupil (cyclopentolate 1% one drop 3 times daily). Steroid eye drops may only be commenced after a full ophthalmic assessment. The main complications of hyphaema are increased intra-ocular pressure from obstruction of the drainage angle and re-bleeding. Strict bed rest is unnecessary if hospital admission is not required after an ophthalmological assessment, however maintaining an upright or semi-upright position, even while sleeping, and limiting activity for 5-7 days or until cleared by an ophthalmologist is recommended.¹⁴

Orbital blowout fractures should be suspected when there is asymmetrical extra-ocular muscle movement with associated diplopia. This is typically seen on upgaze with an inferior blow-out fracture. Urgent assessment by an ophthalmologist and/or plastic surgeon is warranted, along with managing the athlete by shielding the eye.⁸ Children can sustain 'white-eyed orbital blow-out fractures' where there is a white 'normal' looking eye without surrounding bruising and the orbital soft tissue becomes trapped in the fracture leading to tissue ischaemia. This condition commonly masquerades as a head injury with headache, nausea and vomiting along with irritability, however there is marked diplopia and pain with eye movements. White-eyed orbital fractures require prompt referral and management.¹⁸ Avoid nose blowing with orbital fractures due to the risk of associated infection from forcing infected material into the orbit.¹¹ Orbital roof fractures may present with a black eye and subconjunctival haemorrhage without a posterior limit visible.¹¹ With regard to flying or travelling, there is generally no

contraindication with closed globe injuries or orbital fractures and players may return home safely with the majority of injuries.¹⁹

Lacerations to the eyelids require careful examination and exploration, including checking for damage to the globe. Lacerations involving the lid margins, the drainage outflow system, the area of the lacrimal gland or that cause ptosis or herniated tissue, should be referred to an ophthalmologist for repair within 24 hours. Cease play if these areas are involved.

Contact lenses present a special scenario. If the sportsperson has any ocular pain or symptoms we recommend removing the contact lens in order to accurately assess. While this can be done with a finger, there are relatively cheap contact lens removers that are available. Examine the eye for any foreign bodies or corneal abrasions and check for any tears in the contact lens.

Prevention

While up to 90% of ocular injuries may be preventable through education, appropriate eyewear and by removing common hazards, the practicality of preventative measures for ocular injuries varies between sports.^{20,21} Despite easily available eye protection for sport, it is probably under utilised, with one Australian series reporting less than 2% of people in the community wearing eye protection at the time of injury.⁶ At all times the practitioner should encourage eye protection when practical. Individuals at high risk, particularly those with functionally one eyed vision (vision less than 6/12 - the level required to drive), should consider avoiding high risk sports or using properly fitting athletic eye protection, designed for that sport, after appropriate counselling on the risks involved.^{5,22} In addition, eye protection should be considered in those athletes with myopia, diabetes mellitus, previous eye injuries or previous eye surgery.⁹

Table 1: Eye Kit; Ocular Sports Equipment

- 1 Ophthalmoscope or torch with a blue light
- 2 Local anaesthetic eyedrops
- 3 Fluorescein strips/eyedrops
- 4 Antibiotic ointment
- 5 Eye pads and shields
- 6 Cotton buds
- 7 Contact lens remover
- 8 Visual acuity chart/electronic application
- 9 Dark glasses

Table 2: Ocular Assessment

- 1 History
- 2 Visual acuity
- 3 Pupils
- 4 Globe / Cornea / anterior chamber and red reflex
- 5 Lids (always evert the upper lid) and face
- 6 Extra ocular movements

Table 3: Signs and symptoms requiring urgent referral.

- 1 Open globe injury or suspicion from history or exam
- 2 Scleral/conjunctival lacerations
- 3 Foreign bodies imbedded in the globe
- 4 Absent red reflex
- 5 Suspected orbital fracture or a protruding eye
- 6 Severe pain
- 7 Complete visual loss

Summary

The most important diagnosis to exclude in suspected ocular trauma is an open globe injury. Once you have ruled this out, you are essentially dealing with either: a contusion to the globe (which should be referred if a hyphaema is present, if vision is affected or if you suspect traumatic iritis), an orbital fracture or a corneal abrasion/foreign body. Minor contusions and corneal abrasions are not uncommon and can generally be managed on the sports field. The red flags outlined above act as a basic guideline for referring, while performing a thorough assessment as outlined will assist in ensuring that the athlete can safely and confidently return to play without a vision threatening injury.

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Dr Albert Vosseler

Albert Vosseler is currently completing his second year as a Medical Officer in Gisborne which has included six months of ophthalmology training. Albert graduated from the University of Otago where he also undertook research on laser eye surgery for the treatment of glaucoma. He has since gained his accreditation in the USA and hopes to enter into ophthalmology training in Australasia. Albert's vocational interests along with his surfing, skiing and tennis hobbies have led to an awareness of ocular trauma in the sports arena.

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Graham is a general ophthalmologist working at Gisborne Hospital and also in private practice. Graham has sub-specialty training in paediatric ophthalmology and strabismus from the UK and Canada. He is a member of American Society of Cataract and Refractive Surgery, the Australian and NZ Paediatric Special Interest Group. He is also an honorary clinical fellow at the University of Otago and his research interests include population eye health, preventative ophthalmology and amblyopia. He is currently participating in two HRC funded research projects including the Dunedin Multidisciplinary Health and Development Study. In 2001 he won the American Academy of Ophthalmology 5 km race making him the "fastest ophthalmologist in the world"!

Role of exercise stress testing in athletes with repolarisation abnormalities

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In the last edition of the NZJSM, we presented a case study of an asymptomatic athlete with deep T-wave inversion in Leads II, III, V4-V6 together with ST segment depression in Leads II, III and aVF. In the clinical work up of this athlete, he underwent maximal cardiopulmonary exercise stress testing and these repolarisation abnormalities transiently improved resulting in the appearance of a near normal ECG. In this cardiology conundrum, we examine the diagnostic role of exercise stress testing (EST) in the evaluation of athletes displaying abnormal ECG repolarisation patterns.

Management of Athletes with Repolarisation Abnormalities

In order to plan optimal management and treatment strategies in athletes with a suspected cardiomyopathy or ion channelopathy, it is critical to establish both an accurate diagnosis and underlying aetiology. This is often difficult given available technological and published evidential limitations. However in our experience, the minimum cardiac work-up required in the presence of T-wave inversion in asymptomatic athletes include; 1) thorough personal symptom and family history questioning, 2) resting 12-Lead ECG, 3) echocardiography, 4) cardiac magnetic resonance, 5) maximal cardiopulmonary exercise testing incorporating blood pressure response, 6) 24h Holter ECG (including 1 exercise training session), and where possible, 7) 12-Lead ECG and echocardiography of first-degree relatives (> 10 years old). Despite being extensive, these evaluations will

minimise the risk of a false positive or false negative result for the athlete.

Question: “Does the normalisation of T wave inversion upon the onset of exercise lessen the likelihood of underlying cardiac disease?”

Answer: The challenge of dealing with athletes who demonstrate repolarisation abnormalities at rest, but often returns to normality during exercise or adrenergic stimulation has received surprisingly little attention among the sports cardiology community. However, in our experience, only very few patients with true hypertrophic cardiomyopathy (HCM) demonstrate normalisation of repolarisation abnormalities during exercise. Thus, whilst the normalisation of repolarisation abnormalities purports a better prognostic outcome than a failure of normalisation or a worsening of abnormalities, the isolated observation of T-wave inversion normalisation cannot exclude an underlying pathology and thus further investigations remain warranted.

Case Based Evidence to Support this Supposition Athlete One

An asymptomatic black male professional football player with deep T-wave inversion in Leads I, II, III, aVF, V5-V6 (Figure 1). Echocardiography and late gadolinium enhanced cardiac magnetic resonance (CMR) demonstrated no evidence of cardiomyopathy *per se*; no ‘classic’ features of dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy or hypertrophic

cardiomyopathy. EST on initial presentation was negative for exercise inducible arrhythmias, and he completed 11 min 02 sec of a ramped cycling protocol with a maximal heart rate of 181 bpm (maximal minute power of 340 watts). Blood pressure response was normal during and post-exercise. Baseline T wave inversions normalised immediately at the onset of exercise, and returned to negativity 3 minutes into recovery. On the basis of the clinical and investigative results, the athlete was provided medical clearance, given appropriate education to seek immediate medical attention if becoming symptomatic (eg, dizziness, palpitations, syncope, etc) and was required to undergo an annual clinical evaluation, including genetic testing. Three months after receiving medical clearance, the athlete suffered a transient loss of consciousness after 45 minutes of intense football training. A new 12-Lead ECG, echocardiogram, CMR scan, cardiopulmonary exercise stress test, and a 24hr Holter ECG were requested and matched previous findings. Repeated EST demonstrated an appropriate blood pressure response to exercise, whilst baseline T wave inversions again normalised with the onset of exercise. However, starting at 170 bpm (91% age predicted maximum), and lasting past volitional exhaustion and into 5 minutes of static recovery, several episodes of supraventricular tachycardia and atrial fibrillation were recorded (Figures 2 a, b), consistent with the presence of an underlying cardiac pathology.

Athlete Two

An asymptomatic black male professional basketball player without



Figure 1: Marked sinus bradycardia with 1st degree AV block, elevated ST-segment with an upward convexity followed by T-wave inversion in leads V2–V4, and T-wave inversion in Leads I, II, III, aVF, V5-V6.

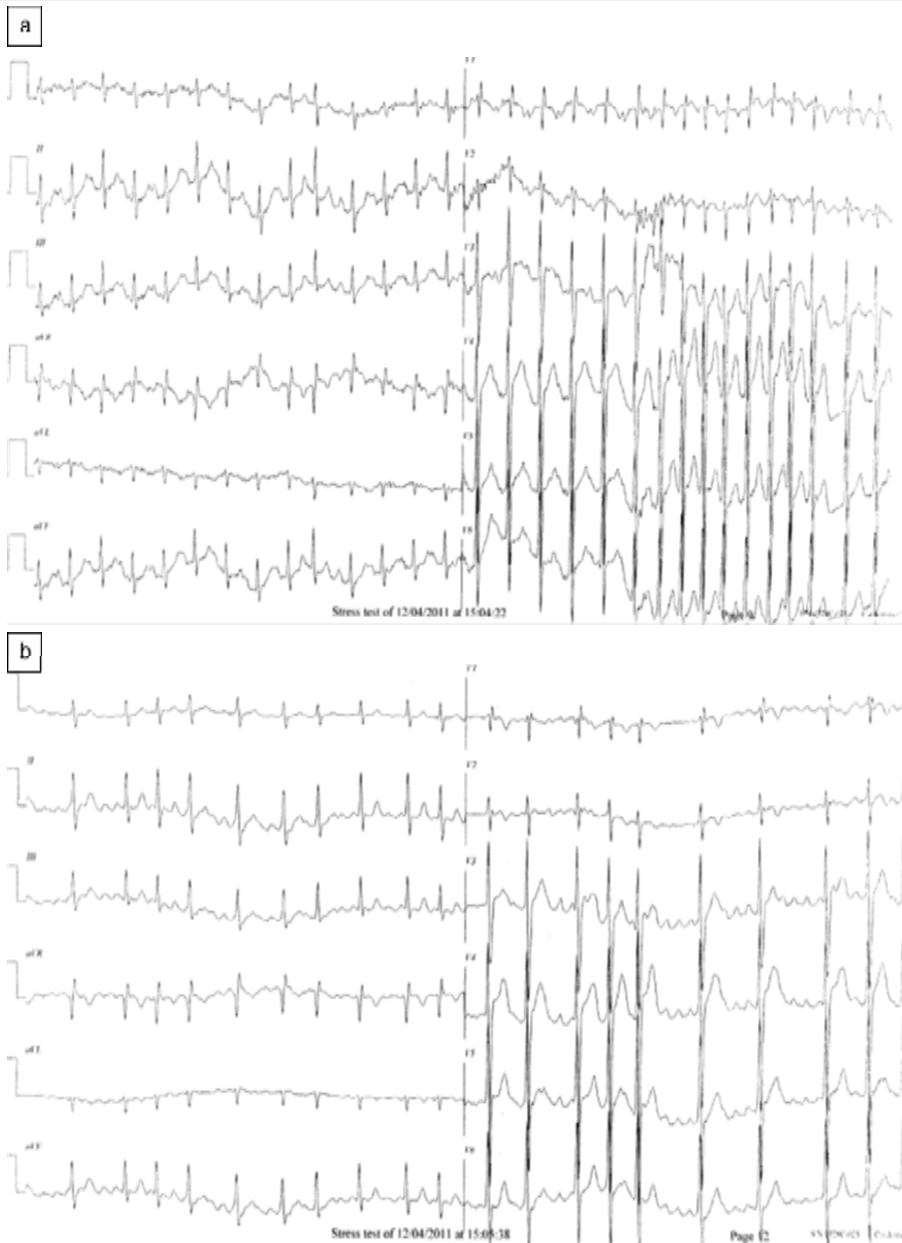


Figure 2: Documented run of supraventricular tachycardia at 19 seconds into recovery (a), and atrial fibrillation at 1 minute 35 seconds into recovery (b). Note: T-wave modification in infero-lateral leads from negativity at rest (Figure 1) to positivity with exercise.



Figure 3: T-wave inversion in V5-V6, with left anterior fascicular block and left axis deviation.

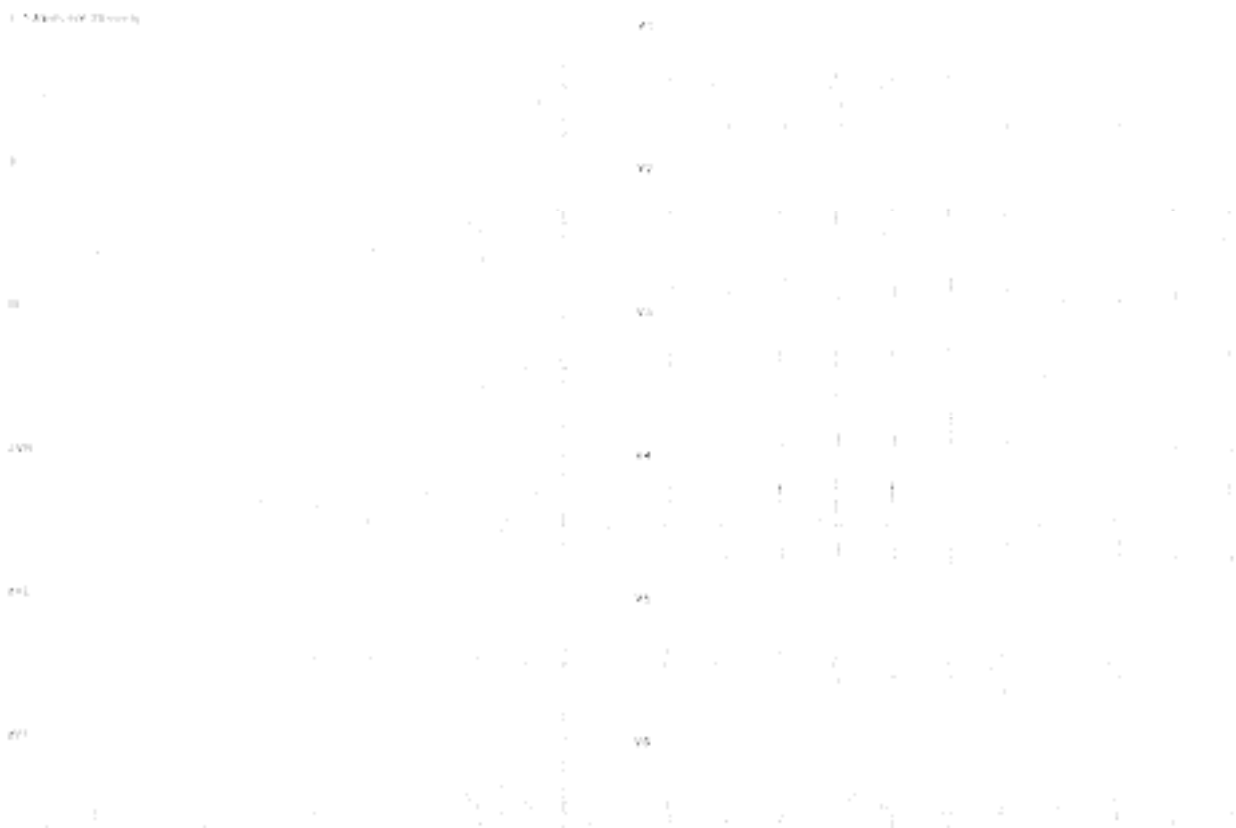


Figure 4: T-wave modification in infero-lateral leads from negativity at rest (Figure 3) to positivity with exercise.

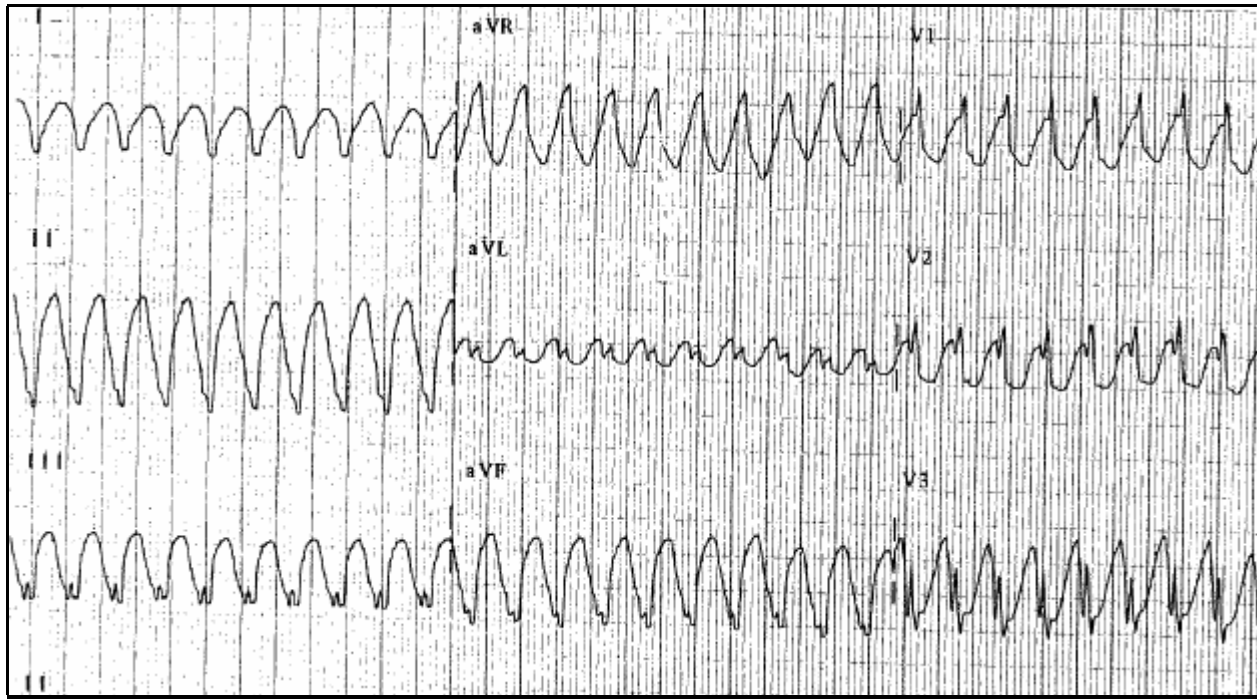


Figure 5: Broad complex ventricular tachycardia

a family history of cardiovascular disease or sudden death presented with T-wave inversion in V5-V6, with left anterior hemiblock and left axis deviation (Figure 3). Echocardiography demonstrated no evidence of cardiomyopathy and EST was negative for exercise inducible arrhythmias. Blood pressure response to exercise was normal, whilst baseline T wave inversions normalised immediately at the onset of exercise (Figure 4). Subsequently, a CMR scan demonstrated no evidence of hypertrophic cardiomyopathy or any fibrotic infiltrate associated with an early cardiomyopathic process. On the basis of the clinical and investigative results, the athlete was provided medical clearance, and given the same follow-up advice as athlete one above. Ten months after attending pre-participation screening, the player collapsed whilst playing basketball. On arrival to the A&E department, he complained of chest pain, was irritable, restless, sweating and had cold extremities. An ECG demonstrated broad complex ventricular tachycardia at a rate of 240 bpm (Figure 5). He underwent a single synchronised DC shock of 360 joules; following which he reverted to sinus rhythm, and became hemodynamically stable.

Specific Importance of Exercise Stress Testing

As these two cases demonstrate, when examining an athlete with T-wave inversion on resting ECG, the resolution of this electrocardiographic abnormality with exercise does not imply that the T-wave inversion is a physiologically benign finding. The role of exhaustive exercise stress testing in these athletes is used principally to assess for the development of ischemic changes, abnormal blood pressure response, and arrhythmias. Thus in our opinion, the improvement of repolarisation abnormalities with exercise is irrelevant and should not be utilised in the diagnostic continuum. It is worth briefly mentioning the beneficial role of integrating metabolic gas analysis with maximal EST. Sharma et al¹ demonstrated that maximal cardiopulmonary exercise testing can be used to differentiate physiologic hypertrophy in elite athletes from asymptomatic but genetically confirmed HCM patients with mild hypertrophy. Typical heart rate and blood pressure responses to maximal exercise failed to distinguish the two entities, but a peak- $\text{VO}_2 \geq 50$ ml/kg/min or $> 20\%$ above the predicted maximum VO_2 differentiated athlete's heart from

HCM. Therefore, in those athletes with T-wave inversion and with a surprisingly low VO_2 maximum, a pathological aetiology should be considered.

Take Home Message

- The normalisation of T-wave inversion upon the onset of exercise does not lessen the likelihood of an underlying cardiac disease.
- Exercise stress testing specifically assesses for the inducement of ischemic changes, abnormal blood pressure response, and arrhythmias with the onset of exercise.
- In our opinion, T-wave normalisation with exercise stress testing is an incidental finding only, and should not be utilised for prognostication.

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UK Sport and Exercise Medicine (UKSEM) Conference 2011

Topic: Sports science, medicine, conditioning, rehabilitation and performance coaching

Location: London

Dates: 23 to 26 November

Overview

This multidisciplinary conference now in its third year has replaced the British Association of Sport and Exercise Medicine conference. It is not only endorsed by this group but many others including FIFA, the FA, the IRB, the RFU, ACSM and BMJ Journals and was opened by Princess Anne – herself a former olympian. The conference was promoted as Europe’s largest interdisciplinary conference on sports science, medicine, conditioning, rehabilitation and performance coaching. The push to maintain a multidisciplinary conference (as is the focus in practice) is something we need to take note of in New Zealand. It appears to me that there are an increasing number of subgroups holding conferences related to sport and exercise medicine in New Zealand and we should try and reverse this trend. Many of the presenters spoke of the importance of all disciplines communicating well in the management of athletes and surely this means we should also be holding joint conferences. I note in the November edition of SportsMedNewZ Stu Thompson’s called for less competition and more cooperation in sports medicine in New Zealand and this is certainly the theme in the UK.

A theme of many of the presentations I attended was the importance of quality coaching in athlete success. A repeated key of quality coaching was a sound knowledge of movement competency from the young to the elite athlete. There is reasonable evidence that achieving (in youth athletes) and maintaining (in elite athletes) optimal movement patterns is important in maximising performance and reducing injury. Practitioners across all disciplines including sports medicine,

sports science, physiotherapy, conditioning, coaching and education spoke of the importance of physical literacy/movement competence.

There were several sessions on barefoot running and on the pros and cons of orthotics. There is good evidence orthotics reduce injuries but comfort is probably key to selection rather than specific design. Barefoot running requires further research but the key is it may teach us about optimal running technique which was emphasised as more important than arguing about whether or not to wear shoes!

Although based on the observations of a journalist the key to producing elite performers (the so called “Talent Code”) is suggested to be maximising reachfulness (practising on the edge of your ability and embracing struggle) and motivation. This obviously requires further research but provides interesting food for thought for coaches and trainers.

FIFA presented a number of studies and there now seems good evidence for their 11+ warm-up programme. They have published results showing a reduction in non contact injuries in football and this programme seems immediately transferable to local sports including soccer and netball.

Selected Presentations

The format of the conference consisted mainly of the keynotes and invited speakers presenting 30-40 min oral presentations with relatively few original research offerings, thus I have focused on these invited presentations.

Keynote presentations:

What we can learn about running from barefoot running

Daniel Lieberman

Given the explosion of publicity on this topic of late hearing Professor Lieberman speak was certainly a highlight of the conference as was his very measured approach to the topic. It is worth noting that Professor Lieberman is not a sports medicine expert but an expert in evolutionary biology. He reviewed his well publicised thoughts on how humans have evolved to be endurance athletes and run considerable distances (10 to 25 km), as until recently (in evolutionary terms!) this was how we caught dinner! The major emphasis of the talk was the need to stop trying to polarise opinion on this topic which should not be a question of shoes or no shoes but a question of how to run well. Professor Lieberman hypothesises that a barefoot running style minimizes impact peaks (due to shorter stride and forefoot/midfoot ground contact), increases proprioception and strengthens the foot and this may decrease injury risk. Thus barefoot running may teach us the key to running well is how you run not what is on your feet. This is a clear message that needs to go out to athletes and support staff so they are educated on this topic and we need to wait for more research in this area as was emphasised by Professor Lieberman. Specific concerns noted by Lieberman were athletes that transitioned too quickly, the unknown effect of minimalist shoes on proprioception and the absence of any data on children.

The impact of physiotherapy on sport and exercise medicine

Kim Bennell

This was an excellent presentation summarising many years of research

in the areas of patellofemoral dysfunction, tendinopathy and lumbar stabilisation. It was pleasing to hear that physiotherapy treatment of these common problems here in New Zealand is well in line with current thinking. We still need to focus on motor control (gluts and quads) and proximal control in the management of patellofemoral issues, eccentrics are still used in tendinopathy but in line with the work of Jill Cook and Craig Purdam we need to recognise there are different stages of this pathology and manage accordingly and while motor retraining for low back pain (multifidus and TVA) is still recommended it may only work for subgroups and overall may be no better than other structured exercise programmes. Professor Bennell also highlighted the need for researchers and clinicians to continue to work closely together to guide the most important research questions – I think this is something we are acutely aware of in NZ sports medicine.

The Blueprint of High Performance
Daniel Coyle

Best selling sports and science journalist Daniel Coyle presented (without any slides!) a very thought provoking and entertaining talk on the keys to producing elite performers. His ideas are based on four years visiting the world’s greatest talent hotbeds in sport, art, business and music including a Russian tennis and Brazilian soccer club. He proposed the keys to producing extraordinary performers were the right kinds of practice, coaching and motivation. Two points emphasised in the talk that made sense to me (although I have no expertise in coaching) were to maximise what Coyle termed ‘reachfullness’ and motivation. Reachfullness is about valuing intense practice on the edge of your ability and embracing the struggle and mistakes that occur – it was suggested this maximises the velocity of learning. Coyle suggested the key to motivation was having someone successful “in your windshield” to follow – he noted how younger siblings were very often faster than their older brothers and sisters. He noted that mixing age

groups, choosing spartan over luxury and praising effort over ability as all being important. Obviously all this is based on observations alone and I’m not familiar with the state of research in this field but it gives local coaches food for thought.

Mindsight: Programmes for Success
Dan Siegel

Dr Siegel is a professor of psychiatry as well as an internationally acclaimed author. Although not an area I know much about this presentation seemed a timely reminder of the importance of mental function in sport performance. It was interesting to hear personality features (resilience, outlook, social intuition, self awareness, sensitivity to context and attention) are trainable and Dr Siegel spoke of the applications of this “brain training” for athlete success. He also mentioned frontal lobe brain functions that are trainable and the best predictors of life success at age three – and perhaps athlete success? – these were bodily regulation, attuned communication, emotional balance, fear extinction, flexibility, insight, empathy, morality and intuition. In terms of a practical tool he presented the “Healthy Mind Platter” a tool that provides a daily diet for a healthy mind – applicable to the athlete and the general public! The platter includes sleep time, physical activity time, focus time, connecting time, play time, and time in and can be downloaded with additional information from; www.drdansiegel.com/resources/healthy_mind_platter.

The quest for physical literacy
Kelvin Giles

This was another interesting presentation and is obviously a hot political topic in UK sport. It did feel like a bit of a sales pitch (somewhat confirmed by going to the website promoted by the presenter www.movementdynamics.com) but the message was still important. Local research suggests many school children don’t have appropriately developed motor skills and it would be interesting to know how kiwi kids compare! Giles proposed high performance sport was built on what

had gone before and that the development of fundamental movement competency was essential. He suggested physical literacy was a basic right of every child alongside the three R’s, being not only crucial to later high performance but also to general health. Giles suggested the solution was to put movement competency into the education curriculum but this was apparently meeting resistance as there was no room for it! He went so far as to recommend physical literacy be tested alongside reading and writing. He also cited the need for physical education specialists in primary school and for teacher and coach education to include more on movement development. I must say this seems very much in keeping with the thoughts of rehabilitation and conditioning practitioners in NZ who consider movement competency/patterns key to performance and injury prevention. If this topic is not on the agenda of Sport NZ then it should be.

Other interesting oral presentations:

ACL rupture, treatment options and return to play

Richard Frobell

This talk was a timely reminder that not everyone who ruptures their ACL needs an immediate reconstruction and that although there are over 11,000 published articles on the topic quality evidence for best practice when dealing with ACL injury is still lacking – interestingly the majority of articles relate to surgery rather than rehabilitation. The question of surgery versus conservative management and all various combinations remains unclear. Frobell made the interesting observation that although the major reason for reconstruction was to be able to return to sport only 60% do so and thus one has to wonder at the appropriateness of the surgery. The emphasis of the talk was on the need for prevention as this is where the best evidence is and the focus on a multidisciplinary approach. Frobell did comment that surgery is obviously a very good option for many high performance athletes but noted that further research is required to match

all individuals to the best management and return to sport criteria still need work.

Hamstring injury risk and return to play

Carl Askling

This talk highlighted that there is still little consensus on the management of hamstring injuries. Re-injury rates remain high and thus questions must be asked about treatment and return to sport criteria. Askling suggested treatment needs to specifically target the involved part of the muscle (medial or lateral – although specific exercises to achieve this were not presented – his publications may provide these) and muscle power was also noted as important. Two main mechanisms of injury were highlighted (i) an acceleration injury typical of sprinters and (ii) a stretching injury typical of dancers. Stretching injuries were suggested to take longer to recover (months) and in contrast the comment was made that if there were no findings on an MRI scan return was not surprisingly much quicker (usually within 10 days). Exercises combining hip and knee motion and lengthening exercises under load were proposed as the best management approach and there may be some evidence this provides quicker return to sport. Askling has also developed an active SLR test suggested as the best indicator of readiness for return to sport and this may be worth considering. The test did however require bracing the knee in extension which may not always be practical clinically however the outcome was based on a simple subjective VAS scale rating.

Update on Plantar Fasciitis

Simon Bartold

Simon is a well known Australian podiatrist who gave a review on the current thinking around plantar fasciitis. He highlighted the pathology is likely to be similar to tendinopathy (degenerative rather than inflammatory and due to a lowered energy dissipation ratio in the fat pad) and risk factors included BMI, reduced passive dorsiflexion, reduced 1st MTP ROM, prolonged standing and a spur

on x-ray (which for years had been considered irrelevant). In terms of treatment he concluded no benefit from shockwave therapy or steroid injection and benefit from taping (short-term), night splints (short-term), stretching the fascia, trigger point dry needling and soft orthotics (although from the talk by Benno Nigg the design of the orthotic does not seem important!).

Orthotics – what do they do – what do we know?

Benno Nigg

Dr Nigg is a very well known biomechanist who has published hundreds of articles on the topic of human locomotion. In his presentation he reviewed a number of his studies and concluded that orthotics work but he was still unsure exactly how! He summarised good evidence to show (as I think we all now know) that orthotics do not re-align the skeleton – in his words “the skeleton doesn’t care what you do to the shoe”. From his studies kinematic changes with orthotics were small, subject specific and not systematic. There however were substantial changes in joint moments and muscle activity – the latter seeming to be his preferred hypothesis as to how orthotics influence performance and injury. There is evidence orthotics reduce injury but this was based on orthotics self selected for maximum comfort – non customised and prefabricated.

11+ nationwide implementation of preventative programme to reduce injuries in football

Mario Bizzini

This was one of several presentations from the FIFA Medical Research and Assessment Centre (F-MARC). The 11+ programme is a series of 11 exercises done as a warm-up – FIFA promote it as the “complete warm-up”. Results of a study looking at its effectiveness (published in the American Journal of Sports Medicine in 2011) have shown promising reductions (around 10%) in non-contact injuries in football. The focus of the programme (as with other similar programmes) is on neuromuscular control and Dr Bizzini

emphasised the importance of coach and player education and compliance. He also highlighted the use of high profile players as ambassadors for the programme. Given our local problem with non-contact knee injuries in netball it would seem that we should be looking at introducing these types of education programmes/warm-ups in New Zealand – especially in youth sport. Obviously FIFA have a massive budget and this made need some consideration for implementation locally.

Sports science in the media in 2011: From 10,000 hours to genes to unfair advantages

Ross Tucker

An interesting presentation on the age old debate of whether elite performance comes from training or genetics or both. Tucker emphasised training was important and his summary statement was that “training is the realization of genetic potential”. He proposed that being dogmatic about a magical number of hours such as 10,000 was inappropriate and that the evidence shows there is a lot of variation between individuals – once again prospective studies are required.

Chris Whatman

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2011 NZ Sports Medicine Conference

Location: Queenstown

Dates: 27 to 29 October

The 2011 Sports Medicine New Zealand Conference was held during October in the picturesque surrounds of Queenstown. It provided an excellent opportunity for sports medicine practitioners from many disciplines to come together for three days to hear a vast array of high quality presentations from a high quality range of speakers from New Zealand and abroad.

We were extremely fortunate to be able to obtain the services of Per Hölmich and Kristian Thorborg on their travels down under to provide a workshop on the assessment of hip and groin pathology. The following morning saw Per Hölmich present the Dr Matt Marshall Lecture. Alongside these two excellent presenters, we were also fortunate to have Stefano Della Villa with us from Northern Italy where he works as a physiatrist. He was able to provide us with an excellent account of his approach to the rehabilitation of the elite athlete following injury. Dr David Hughes from Canberra also accepted our invitation to present with a lecture on non-physical pain generators in whiplash injury providing a valuable insight around aspects of both pain generation and management that are not always considered in our setting.

In conjunction with these international speakers we were delighted with the vast array of presentations from local practitioners. This was led by the keynote presentation provided by Dr Peter McNair on lower limb muscle performance changes with injury. The conference programme followed the format of previous years, with the Saturday programme focused on the individual discipline groups. Physicians and physiotherapists separated off to receive updates in key areas relevant to their respective areas of sports medicine.

Also introduced was the great case presentation competition with a number of practitioners providing case presentations for the education of colleagues, whilst also introducing a competitive slant on things. It is hoped that this competition will obtain a regional and parochial focus in the future.

On the social front, an excellent conference dinner was enjoyed by the majority of delegates with highlights including the dual presentation of Dick Tayler and Sir Colin Meads. We were also lucky to have Dr Deb Robinson, the victorious Rugby World Cup All Black doctor with us who shared some anecdotes relating to her involvement with the team during the tournament.

We are grateful to all those who attended and especially to all those who made an effort to present in some format during the conference. Feedback to date has been very positive and we are grateful to those who have taken the time to complete the feedback survey following the conference. This is already assisting us as we begin planning towards the 2012 conference in Auckland.

On behalf of the Organising and Scientific Committees, I look forward to seeing you there.

Dr Ian Murphy
Chairman
Conference Scientific Committee

High Performance Sport New Zealand changes to enhance New Zealand sportspeople winning on the world stage

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As of 8 August 2011 there has been a change in the support structure and names of organisations supporting elite sport in New Zealand. The New Zealand Academy of Sport (NZAS) has now merged with Sport and Recreation New Zealand (SPARC)'s high performance unit to form one national entity - **High Performance Sport New Zealand** (HPSNZ). This amalgamation has come about with the goal of improving the use of resources supporting elite sports performance in New Zealand. The new organisation is responsible for developing and supporting the country's elite athletes and coaches, and expanding a network of world-class training facilities.

The Government's expectations of the increased high performance sport investment are:

- creating a more focussed high performance system;
- ensuring the system is as efficient as possible;
- effecting a cultural change with an increased focus on excellence;
- implementing a one-stop shop for our athletes;
- recruiting and retaining world class coaches;
- maximising technology and innovation; and
- increasing the number of dedicated HP facilities.

The objectives of the merger of NZAS North Island, NZAS South Island and the SPARC HP Unit to form HPSNZ are:

- simplify and streamline the high performance system to the benefit of athletes, coaches and sports organisations;
- lead to a culture change with an increased focus on excellence;
- improve support for athletes, coaches and NSOs;
- improve collaboration and knowledge sharing; and
- reduce administrative and governance overheads.

As a result of changes over the last couple of years, there are now less contracted providers explicitly dealing with elite athletes across the full range of disciplines, but especially in the rehabilitation area - medicine, physiotherapy, massage therapy, osteopathy and chiropractic. There are a number of drivers for these changes as follows:

- 1 There are less carded athletes in the system than when the academy was first set up
- 2 There has been a centralisation of services to designated service centres in Auckland, Lake Karapiro, Wellington, Christchurch (until the earthquake struck) and Dunedin.
- 3 A desire to have key providers working and being immersed in the sports they service
- 4 The need for consistent quality reporting systems particularly electronic reporting
- 5 Improved inter-professional integration and team working in an athlete centered model

We will now expand on these drivers to provide a rationale for the changes.

1 Less Numbers of Athletes

The previous Academy of Sport high system had reduced the number of carded sports and set very high targets for athletes to be supported. The standard is "world class" (typically top 16 in the world rankings), or on a development pathway to medal in a pinnacle event such as World championships or Olympics. The funding is now targeted to these four year cycles to allow for adequate planning and development. There are now fewer athletes supported, and hence there are fewer providers required. The process of deciding who continued to provide service was based on a range of factors; the number of services provided in the last year, the number of providers already in that area, and the geographical area required to provide services to athletes to name a few. There are still many providers who actually meet the criteria to be a provider but either saw very few if any athletes or their service utilisation had declined. In cases where there is still significant expertise, HPSNZ would like to still use those providers on a case by case basis, and when pre-approved by the sport paying for the services. Where appropriate, HPSNZ has also retained providers with long standing and existing relationship with key athletes. However with newly carded athletes, sports are being encouraged to use the established service centers where possible as a greater range of coordinated service are provided at these centers than individual provider clinics.

2 Service Centralisation

In order to provide a world class service to athletes most countries in the world have developed designated service centers that bring together the desired expertise in order to ensure the athletes achieve their goals of 'winning on the world stage'. In the early models of the academy it was thought that having a wide coverage of providers to allow athletes to stay closer to home would be useful. However there was mixed compliance to the reporting systems required by the academy and also fragmentation of how the various services were delivered. This was not necessarily intentional but more a function of the geographical spread and lack of electronic reporting systems. With the new service centers up and running there have been significant improvements in coordination of services, with teams of providers working with athletes and along with this enhanced quality control via appropriate reporting mechanisms.

3 Immersion of Key Providers

Within the service centers there has been a move to have key providers immersed into the sports. A good example of this in the Rowing NZ model. There now are full time physiotherapists employed to work with the rowers along with a full time strength and conditioning expert and a biomechanist. These providers are supported by sports psychology, sports medicine, massage and nutrition in either full or part time roles. The key rehabilitation providers are also the ones that travel with the teams on their overseas campaigns. It is envisaged that these providers will also be appointed to Olympic Games teams so that there is complete consistency of services. Many others sports now have some of or all of this immersion in place.

4 Quality Reporting Systems

The rehabilitation centre at the AUT Millennium Campus was the first service centre to move to a full electronic client record (ECR), implementing the Gensolve system. It is hoped that as the other service centers are developed that Gensolve will also be used there. This system has improved the communication across providers. It has also now been linked with the new electronic Athlete Management System – ZED which contains all the key information about the athlete. This system is web based and accessible to authorised providers. The key drive in the system is the athlete's individual performance plan (IPP). All services must be coordinated and monitored to ensure that the performance goal is achieved and this is achieved more easily with coordination of

activities in the service centres. Key fields in the Gensolve ECR are uploaded daily into ZED so that providers in other disciplines can see appropriate parts of the rehabilitation plan with less duplication of paper work.

5 Improved Inter-professional Integration

This is a key area of the new environment. The IPP and athlete centered approach is critical to the new environment. Athletes appreciate services that are coordinated, where the goals are clear and when all providers in the team are on the same page and are talking together about the goals. There has been significant movement in this area and there have been a number of professional development days already instigated to ensure this inter-professional approach is enhanced. An example of this is the inter-disciplinary approach is the development of the Movement Competency Screen (MCS) developed by Matt Kritz and Professor John Cronin from AUT University. This PhD project developed and validated a tool based on five key movement patterns which is being used as a communication tool between strength and conditioning providers and rehabilitation providers. The five key movements are used to decide if an athlete moves well or poorly and how their movement patterns might influence training and performance. The MCS helps set appropriate loads for rehabilitation exercises and ensures all parties know what the athlete can do and what needs modification in the gym training programme. The MSC has now been loaded into the Gensolve programme and this has become a standard element of all athlete assessments upon presentation with an injury. It is also now the starting point for the previously applied muscle balance assessments. Once the MCS is completed further sport specific tests can be undertaken by the physiotherapists.

There are two other key areas of development. Firstly the philosophy within the provider's teams is performance enhancement and injury prevention rather than injury treatment. To achieve this time is allocated in the performance centers to allow for integration and time for providers to work together to enhance the training programmes.

Secondly there has been an effort to embed research around the athlete performance development. This has been particularly successful in the strength and conditioning area where service providers are also completing PhD and Masters research projects. This initiative will be extended

in the rehabilitation area to increase the research into injury prevention and the most effective injury rehabilitation protocols. This will be lead by Duncan Reid (Assoc Professor at AUT and Director of the Rehabilitation Centre at the AUT Millennium campus).

Future Challenges

With a smaller number of providers integrating with elite athletes there is a risk that expertise will be lost. There is also a risk that getting to the top of this pyramid and working with truly elite athletes becomes more challenging and as such, succession planning and career pathways need to be considered. As there is no funding for services to athletes that are not carded, sports and HPSNZ need to consider systems that encourage providers to get involved in sports at junior and development levels. This may be the so called 'hard yards' that providers need to do to position themselves for opportunities that may arise in the future. Fortunately, there are also opportunities to travel overseas with teams, with key providers not always available to travel. In this way sports need to develop touring providers and this model has worked well with sports like Hockey where is a team of providers that are available for tours on a rotational basis.

Conclusion

The changes to High Performance sport described in this paper provide a rationale for the future developments in this area. New Zealand is a country that has always 'punched about its weight' in the international sporting arena. Support services to athletes have grown over time and as these grow refinements are required to ensure we offer the most up to date and world leading approaches. These changes will ensure that an athlete has all the required support to meet the overall goal of HSPNZ- "more new Zealanders winning on the world stage."

Duncan Reid
Rehabilitation Director HPSNZ
AUT Millennium Rehabilitation Centre

Tony Edwards
Medical Director HPSNZ

Sharon Kearney
Physiotherapy Director HPSNZ

Sports Medicine New Zealand 2011 Chairman's Report

Presented at the National Council Meeting held in
Queenstown on 27 October

It is great to see that after a year of financial turmoil, earthquakes and ecological disaster, my first year as chairman has concluded with two significant positives: first, Sports Medicine New Zealand has continued to remain a financially sound organisation, and the All Blacks hold the Webb Ellis Trophy. I cannot truly claim to have made any significant contribution to either, but I do want to offer my continuing thanks and appreciation for the fantastic work that Brenda Allum continues to provide well above and beyond what we could ever expect. Thank you, Brenda.

Our esteemed President, Mr Chris McCullough, has nicely summarised the main focal points for SMNZ in 2011 in his report, so I will not repeat them.

Sports Medicine New Zealand has two main faces it shows to the world; our conference and our journal. This year's conference again looks like it will be very successful with a great programme and a fantastic venue.

I encourage you all to support our journal and its new editorial team of Bruce Hamilton and Chris Whatman and I challenge each member of SMNZ to submit one single item for publication in the next 12 months. It doesn't matter what the item is - a research paper, a review article, a brief commentary on a sporting or sports medical happening, a clinical pearl of wisdom (anything you have picked up that has helped you in your practice and could possibly help others), or even a letter to the editor. Bruce is also looking for case studies and journal article reviews and is developing templates and guidelines to assist anyone who wants to do something in this area. The journal is about Sports Medicine in New Zealand, and it is you, our members, who make up this sports medicine community. And it is only you who can write about it - for your colleagues and for the rest of the world. I believe that, with your input, the NZ Journal of Sports Medicine can inform, educate and report and be a valuable resource for us all. Bruce and Chris are working hard to achieve this, and I again thank them for their time, their energy, and their vision for our journal.

And what of Sports Medicine New Zealand in the future? SMNZ is the oldest of the organisations currently representing sports medicine in New Zealand, and it is an umbrella organisation with a wide membership open to all those with an interest in the areas of medicine, sport and exercise in their broadest sense. But there are many other organisations competing for membership dollars and conference attendees. I believe there is a need to compete less and to co-operate more and I hope to pursue this philosophy over the coming years through discussions with the various other groups. This philosophy is also reflected in the direction High Performance Sport New Zealand is moving. I hope we can develop innovative ways to better work together, to share resources, to encourage continued membership and to develop and enhance relationships between the groups. Recent talks with Sport and Exercise Science New Zealand have been a start, as has the sponsorship High Performance Sport New Zealand has provided for our conference this year.

Finally, I wish you all an enjoyable conference, a happy Christmas and a hope that New Zealand will continue to prosper socially, culturally, economically and on the sporting fields of the world.

Dr Stuart Thomson
National Chairman

Selected Abstracts from the 2011 NZ Sports Medicine Conference

held 27-29 October, Queenstown

Reproduced from the Conference Handbook published by Sports Medicine New Zealand.

CLINICAL ENTITIES IN ATHLETES WITH LONGSTANDING HIP AND GROIN PAIN - DIAGNOSIS, TREATMENT AND PREVENTION

Per Hölmich

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Groin and hip pain in association with sports activities is a serious and recurrent problem in sports medicine, in particular in connection with football, rugby and icehockey. The incidence of groin pain is 5 – 18 % per year. The pain may originate from various anatomical structures such as bone, joint, muscle, tendon or ligament but abdominal or gynaecological disorders, referred pain and nerve entrapment can also cause groin pain.

The muscles of the femur, the abdominal muscles and the muscles of the back all work in synergy to balance the pelvis and to control the movements of the trunk in relation to the pelvis and the legs.

Poorly understood terms as adductor tendinitis, osteitis pubis, symphysisitis, sports hernia, pubalgia and likewise are used in the literature. However, these terms do not fulfil the demands for a true “diagnosis” and they are in relation to these patients so far without scientific basis. The term “clinical entities” is suggested relating the origin of the pain to specific anatomic structures. Examples of entities are Adductor related groin pain, Iliopsoas related groin pain and Inguinal related groin pain. (Hölmich BJSM 2007) Pathology in the hip joint is an important cause of groin pain itself and a predisposing factor for primary as well as secondary groin pain as well.

Clinical Examination

The examination techniques used needs to be standardised, reproducible and valid. This is imperative to make reliable conclusions, to report to and cooperate with colleagues and to be able to work scientifically. A number of techniques have been tested and described in the literature, but there is still a need for further testing and validation.

Treatment

Most treatment modalities suggested in the literature are based on the experiences of clinical practice, lacking randomized trials. It is important to be aware of all causes for the groin pain in the planning of a treatment program. The result of treatment should be evaluated using validated patient based outcome scores. (Thorborg et al, BJSM 2011)

Adductor-related Groin Pain

One clinical randomized trial has described a specific exercise program found to be highly effective in the treatment of adductor-related groin pain (Hölmich et al, The Lancet 1999), and to have a long lasting effect (Hölmich et al AJSM 2011).

The literature regarding tenotomy of the adductor tendons is contradictory and not scientifically based. It is the experience of this author that a tenotomy of the adductors is rarely indicated. Most patients can be treated successfully with the above-mentioned training program combined with sufficient treatment of concomitant injuries.

Conclusion

Groin injuries are difficult to treat. In many cases they tend to be ignored by both athletes and trainers thus developing into a chronic stage. The treatment should be based on a systematic examination and an exact identification of the structures causing the pain. Muscle imbalances and biomechanical problems should be identified. The physician, the therapist, the trainer and the athlete need to be aware that the treatment in most cases will be longstanding. Supervision, encouragement and patience are imperative to reach a good result.

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WHAT'S NEW IN SPORTS NUTRITION FROM THE IOC CONSENSUS MEETING 2010

Jeni Pearce

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The first consensus conference to review the evidence for the role and impact of nutrition in sports performance met in 1991 under the Medical Commission of the International Olympic Committee in Switzerland. This was followed up in the 2003 Consensus meeting producing a series of journal articles, a book and videos of the meeting.

The latest developments in scientific evidence in sports nutrition were again reviewed in 2010 by a team of sports nutrition experts. Day one of the two day conference focused on the advances in scientific research in six specific areas (energy balance, carbohydrates for training and competition, fluids and electrolytes, protein and muscle growth and repair, special nutrients including vitamin D and antioxidants, and dietary supplements) in sports nutrition.

The second day examining the practical application of the science across a range of sports (strength sports, power sports, team sports, endurance sports, winter sports, aesthetic and weight class sports). A number of new developments in sports nutrition were highlighted including energy availability, wider ranges in the guidelines for carbohydrate intakes, the amount and composition of carbohydrate, the timing and level of protein

CONFERENCE ABSTRACTS

recommended for recovery from training sessions to maximise protein synthesis and support for a limited number of well researched supplements supported by strong scientific evidence.

This session will review some of the highlights and developments in the science, the changes to the recommendations since 2003 and the final consensus statement produced on the third day of meeting.

PAIN FROM OVERUSE INJURY IN 104 COMPETITIVE AND RECREATIONAL ROAD CYCLISTS, MOUNTAIN BIKERS AND TRIATHLETES

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Background: Occurrence of overuse injuries and pain in cycling may differ among cyclists of different disciplines [1]. The knee and low back have been reported as the main injury sites in elite cyclists, but data have not been provided for other cycling disciplines and experience levels.

Aim: To describe the occurrence of pain in cyclists of different disciplines and experience level.

Methods: A survey of 104 cyclists (49 recreational non-competitive road cyclists, 21 competitive road cyclists, 13 non-competitive mountain bike riders and 21 competitive triathletes) provided information on characteristics of existing pain during cycling, perceived comfort on the bicycle (yes or no) and cycling training volume (hrs/week).

Results and Discussion: Percentage values in Table I are presented within each cycling discipline and for all cyclists. For all cyclists surveyed, 38% had existing pain even though 85% reported they felt comfortable on the bicycle. Fifty six

percent reported both pain and comfort, therefore, comfort on the bicycle is probably not a good indicator for overuse injury risk. The knee joint and low back were frequently reported sites of pain for all the cycling disciplines and levels which supports findings from other studies on road cyclists.¹ A larger percentage of triathletes reported pain compared to the other cycling disciplines. The larger percentage of ankle-foot pain in triathletes during cycling may be a cross-over effect due to additional running training.

Conclusion: Triathletes report more ankle-foot overuse pain than other cycling disciplines. Comfort on the bicycle and cycling training hours per week are probably not good indicators of overuse injury risk. Training intensity and total training volume of triathletes (including running and swimming) should be measured to gain further understanding of risk factors for overuse injuries.

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THE EFFECTS OF COMMUTER CYCLING FOR 10 WEEKS ON FITNESS, HEALTH AND BODY COMPOSITION

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Introduction: Commuter cycling interventions in Northern Europe have resulted in mixed effects to cardio-respiratory fitness,^{1,2,3} body composition,^{1,2} and blood pressure^{1,2} after eight,² ten³ and twenty-six weeks¹. Mixed results between studies may be due to methodological issues in the research. Therefore, the purpose of this study was to examine the effects of commuter cycling for 10 weeks on peak oxygen uptake ($\dot{V}O_{2peak}$), body mass, waist-to-hip ratio, blood pressure

and quality of life among healthy adults using a randomised controlled study design.

Methods: Fourteen men (mean age 37 ± 7 years, body mass 76 ± 9 kg, O_{2peak} 42.5 ± 8.5 ml•kg⁻¹•min⁻¹) and fourteen women (mean age 35 ± 9 years, body mass 70 ± 10 kg, O_{2peak} 31.5 ± 5.4 ml•kg⁻¹•min⁻¹) were recruited. Participants completed two testing sessions before and after the intervention. In the first session, peak oxygen consumption and quality of life (SF-36 survey) were measured. In the second, blood pressure, body mass and waist-to-hip ratio were recorded after a 12 hour fast. Participants were stratified by gender and O_{2peak} and randomised into intervention and control groups. The intervention group (n=14) were given bicycles and instructed to cycle to and from their work place three or more days per week, for 10 consecutive weeks. The control group (n=14) were asked to continue commuting with their current mode of transportation. Data were analysed using a two-factor (group x time) repeated measures ANOVA.

Results: There was 100% adherence in the intervention group, while one control participant withdrew due to illness. A time x group interaction showed that O_{2peak} increased significantly (p = 0.02) by 3.2 ± 4.6 ml•kg⁻¹•min⁻¹ in the intervention group, but did not change in the control group. While, diastolic blood pressure significantly (p = 0.05) increased by 8 ± 10 (mmHg) in the control group, but did not change among intervention participants. There were no significant effects for body mass, waist-to-hip ratio, quality of life parameters, or systolic blood pressure.

Conclusion: Ten weeks of commuter cycling in New Zealand is beneficial for improving cardio-respiratory fitness, but not blood pressure, body composition or quality of life.

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Table I: Cyclists' age and training volume, comfort during cycling, percentage of cyclists with pain, and body sites of pain for recreational cyclists, mountain bikers, competitive cyclists and triathletes.

Cyclist Discipline	Age (years)	Training Volume (hrs/wk)	Comfort (%)	Pain (%)	Upper Body (%)	Low Back (%)	Butt-Hips (%)	Knee (%)	Ankle-Foot (%)
Competitive cyclists (n = 21)	37 ± 11	8 ± 4	86	33	29	43	43	50	7
Recreational cyclists (n = 49)	41 ± 9	7 ± 3	85	41	52	48	43	87	9
Mountain bikers (n = 13)	39 ± 10	5 ± 3	85	38	25	88	0	75	0
Triathletes (n = 21)	38 ± 10	6 ± 2	79	58	13	25	25	75	38
Total (N = 104)	40 ± 10	7 ± 3	85	38	30	41	28	61	9

**CASE REPORT
DIETETIC MANAGEMENT
FOLLOWING MULTIPLE JAW
BREAKS IN AN ELITE RUGBY
UNION PLAYER**

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Introduction: In professional sport, injuries to key players can have crucial implications on team performances. Severe injuries are common in rugby union, often followed by weeks to months of recovery and rehabilitation. Often, body composition suffers during this rehabilitation period, due to reduced energy expenditure via a reduced training load, combined with stable (or increased) energy intake due to boredom, and/or poor food choices. Deficiencies in energy, protein, carbohydrate, fat, vitamins and minerals can negatively affect wound healing¹. The flow or catabolic phase of the body's stress response entails a higher requirement for glucose to heal tissues, and also involves greater protein catabolism². Good dietetic management over the recovery period can not only accelerate tissue repair and recovery, but also ensure optimal body composition when returning to play.

Case Report: This case describes the dietetic intervention strategies employed with an elite rugby union player who sustained consecutive broken jaw injuries. There were three primary goals of dietary management: 1) to provide a nutritional environment that optimised the healing and recovery process; 2) to minimise changes in body composition; and 3) assist this player in achieving his goal of being available for the All Black squad in 2011.

Early education regarding the stress response to injury and surgery were provided, and dietary strategies over different time periods following the subsequent surgery discussed. These included the need for a relatively high carbohydrate, protein and energy intake in the immediate post- surgery period, to avoid substantial losses in body weight and muscle mass, further complicated by the inability to eat solid food. In the absence of established guidelines for athletes undergoing surgery, dietary requirements were extrapolated from clinical guidelines. Estimated energy requirements were 11-12 MJ/day following surgery, protein requirements

were estimated at 1.5g/kg body mass, and carbohydrate requirements were estimated at 3-5g/kg body mass. Practical advice was provided on the best way to achieve nutritional goals via a liquid diet. Liquid supplements including 'Ensure Plus', 'TwoCal HN' and sports nutrition protein-carbohydrate supplements were provided to boost energy, carbohydrate and protein intake. Regular reviews of dietary intake and body composition were undertaken to monitor the player's ability to maintain energy balance.

Discussion: Early dietetic intervention was paramount in correction of misconceptions regarding energy and carbohydrate requirements. Practical advice on liquid food options and supplementation was well received and adhered to. Regular dietetic monitoring ensured maintenance of body weight within 3kg and of Sum of 8 Skinfolds within 14mm for both injuries. Access to dietetic support following major injury/surgery can ensure optimal recovery, particularly with regard maintenance of body composition.

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**MUSCLE INJURY AND
REHABILITATION**

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Muscle Injuries are very common traumas in Sports Medicine. Depending on the trauma mechanism muscle injuries are classified as direct and indirect. The direct form is the contusion, the indirect one is strain. Muscle strains can be divided in three grades according to their severity: mild (first degree), moderate (second degree) and severe (third degree).

An immediate and accurate diagnosis is the key to plan an appropriate rehabilitative treatment. The diagnosis is based on a careful history of the trauma followed by a physical examination and imaging procedures. The initial imaging technique we require is the ultrasonography (US). Magnetic Resonance is preferred if there is any discrepancy between the patient's symptoms and the ultrasonography and in the evaluation of the deeper muscles.

Although the post acute management of muscle injuries known as RICE (rest, ice, compression and elevation) is already accepted there is no consensus about the rehabilitation program. The primary objective of rehabilitation programmes are returning the athlete back to competition with a minimal risk of re-injury. There is evidence that the risk of re-injury can be minimised by utilising core stability training (1) and eccentric strength training (2).

Our protocol consists of 3 phases, each characterised by the achievement of clinical and functional goals.

- Phase 1: Aerobic conditioning and range of movement recovery
- Phase 2: Strength and extensibility recovery
- Phase 3: General and specific conditioning on the field

The transition from one phase to the next is allowed when the different progression criteria are met. Establishing criteria for the return to sport remains a challenge for future research.

Our experience of 10 years consists of 1343 injuries (non contact muscle lesions). The mean age was 30,4 and the 95% were male. 79% injuries occurred in football players and in the 37% were involved the hamstrings. We noticed a reduction of re-injury over the time.

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**TRAUMATIC ANTERIOR
SHOULDER INSTABILITY**

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Aim: To define the patient demographics and pathology in patients in a New Zealand shoulder surgery practice, undergoing shoulder anterior instability surgery and develop an algorithm for

surgical treatment of traumatic shoulder instability.

Method: A 4 year cohort of patient's having shoulder stabilisations from 2005 – 2008, were reviewed. A database was created using information obtained from clinical notes, imaging studies, operative findings and surgical procedures. Known recurrence rates were recorded and checked against the digital imaging patient records.

Results: After excluding revision stabilisations and posterior stabilisations, there were 193 primary anterior shoulder stabilisations. The mean patient age was 24 (range 15 – 49) with the median younger. 80% were males. There was near equal incidence of the dominant and non-dominant arm being affected. 85% reported playing sport, with 50 different sports listed. Many participated in multiple sports. Sports were classified as 'collision', 'contact and falling risk' and 'overhead'. Approximately 50% listed collision sports (rugby, rugby league or combat). Approximately 50% listed contact and falling risk sports (e.g. snowboarding, netball, mountain bike). Approximately 15% listed overhead sports (e.g. throwing, rock climbing). With regard to glenoid bone lesions on preoperative imaging, there were bone changes in the glenoid in 15% of patients. On examination under anaesthetic at the time of surgery 16% were Grade 4 instability, where the shoulder is extremely unstable and locks out of joint. During this study period 64% of procedures were performed arthroscopically and the remainder through an open approach. Surgical decision making was on a case-by-case basis. For the 92 patients who listed rugby as a sport, 60 underwent an arthroscopic stabilisation and 32 underwent an open stabilisation. With regard to recurrences (known dislocations or subluxations) the overall rate was 13%.

Conclusions and Discussion: Known risk factors for recurrent instability after shoulder stabilisation surgery include young age, at risk sports and activities, hyperlaxity and bone lesions. Most New Zealand patients with shoulder instability are young, have an extremely high activity level and frequently play high-risk sports. There is a significant incidence of bone lesions. Each patient needs to be considered carefully with regard to these factors and the pathology of their instability. The surgeon should have a range of procedures in his / her inventory to address each situation. Contemporary options include open and arthroscopic Bankart and capsular shift repairs, Laterjet

and bone block procedures for bone deficiency and arthroscopic Remplissage for Hill Sachs lesions.

UPDATE IN CHONDRAL INJURY REPAIR/ REHABILITATION

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Cartilage knee injuries are frequently observed in athletes and typically caused by high-impact stress and torsional loads. The management of chondral lesions is difficult depending to the limited regenerative capacity of articular cartilage. Several techniques have been applied through the years for the treatment of cartilage lesions including Microfractures, Osteochondral Autograft Transfer (Mosaicplasty), Osteochondral Allograft Transplantation and Autologous Chondrocyte Implantation (ACI). The treatment has been recently improved by the arthroscopic second-generation ACI with three dimensional hyaluronan-based scaffolds that reduces the joint surgery trauma and may accelerate the recovery, when combined with proper rehabilitation protocols.

The rehabilitation program after articular cartilage repair is a critical component of the treatment process and represents the key for an optimal clinical and functional outcome. The post-operative rehabilitation protocol should be progressive, goal-oriented, individualised and should take into consideration the patient's age, the cartilage repair technique and the lesion's size and location. To ensure a successful outcome, rehabilitation should occur in a controlled setting, with skilled therapists and with access to rehabilitation swimming pool, gym, and field, under the supervision of the orthopaedic surgeons.

We recently evaluated if the hyaline-like tissue obtained with the regenerative cell-based approach allows to these highly demanding athletes a better functional recovery with respect to the outcome offered by the bone marrow stimulation approach. We analysed the results obtained in high-level football players affected by knee cartilage lesions and treated with arthroscopic second generation autologous chondrocytes implantation or

microfracture technique, and we compared time taken to recover, return to previous activity level and functional outcome at short and medium follow-up.

CARDIAC SCREENING/ECG INTERPRETATION IN THE ATHLETE

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Pre-participation screening of athletes for identification of cardiovascular diseases that predispose to sudden death during sport is increasingly required for many sporting disciplines. Such screening usually involves a medical history and physical examination and, while the role of a 12-lead electrocardiography (ECG) remains controversial, this is increasingly required by sporting authorities. The purpose of ECG screening of athletes is to improve the detection of underlying cardiac abnormalities that may be associated with the risk of sudden death. Electrocardiographic changes in athletes are common and reflect the physiological adaptation to exercise training – so called "athletes heart". Consequently it is important to correctly distinguish between those ECG changes associated with exercise training and those which may be due to other cardiovascular conditions. Controversy still exists with regard to ECG screening of athletes. However, in the current New Zealand context, ECG screening is required in a number of sporting disciplines and thus a standardised approach to ECG screening is required. Recent recommendations from the Sections of Sports Cardiology and Exercise Physiology of the European Association of Cardiovascular Prevention and Rehabilitation and by the Working Group on Myocardial and Pericardial Diseases of the European Society of Cardiology provides the most contemporary recommendations for ECG screening of athletes.¹

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