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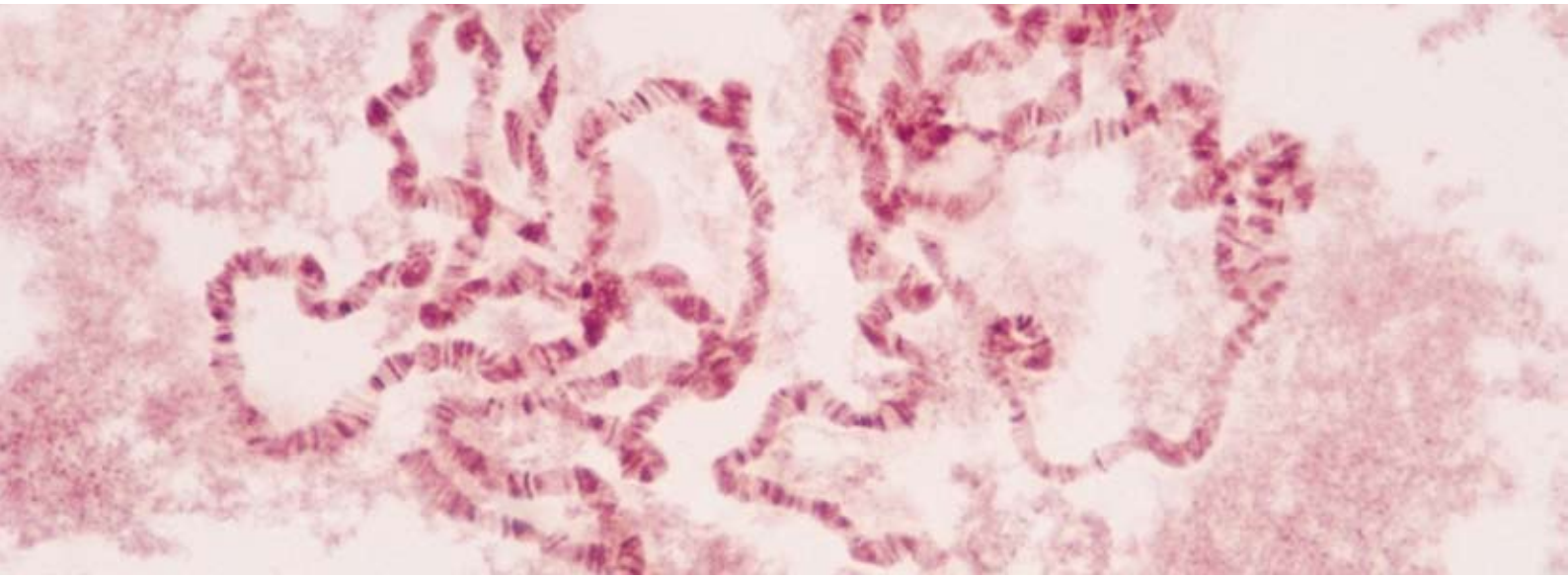
Developing an SMA Position on Genetic Testing



In September 2008, the Queensland Branch of Sports Medicine Australia (SMA) requested that the SMA National Board facilitate the production of an SMA "Position Statement" in relation to genetic testing of children, with particular regard to its potential to unfairly bias against individual participants. SMA has had a long history in the production of position statements and other forms of medical and safety advice for people and organisations involved in sport and physical activity. As a first stage in the process, a briefing paper containing much of the information below was prepared for the Board. As always, feedback or opinions from members is welcomed.

Background

In regard to genetic testing, in June 2004, following consultation with a wide number of groups in sport (including SMA), the Australian Sports Commission (ASC) published a *"Policy on the Conduct of Genetics Research and the Use of Genetic Information in Sport"*. The Policy was strongly informed by a Report titled *"The Protection of Human Genetic Information"* produced by the Australian Law Reform Commission (ALRC) and the Australian Health Ethics Committee (AHEC).[1] (SMA and the Australasian College of Sports Physicians were also consulted in the preparation of this Report.) This Report looked in part at "inappropriate discriminatory use of human genetic samples and information" – the major issue of concern to the SMA Queensland Branch – but in the context of anticipated benefits such information could bring to the Australian community.

The ASC Report

The ASC Report makes the point that the ASC is charged under the ASC Act (1989) with developing programs to find the best sporting talent in Australia. The ASC says that in discharging this role it has a responsibility different to a "generic health promotion agency" and it argues that genetic testing "conducted along appropriate guidelines" is both essential to this role and not discriminatory – an approach in relation to elite sport that is supported by the ALRC and the AHEC.[2]

The ASC Report also lays stress on the equally import role across all levels of sport of genetic testing for injury prevention. (see further discussion below).

The Report also stresses that the use of genetic information in athlete selection must conform to the Disability Discrimination Act 1992. To this end, the Report says, "Good Science is needed"… because the genetic information must be "reasonably reliable and relevant".[3]

Developments Post 2004

1. Genetic Doping

Since 2004, the issue of genetic doping has received a great deal of additional publicity and has led to heightened general suspicion about any genetic research related to sport. This is a major concern to genetic researchers and there has been much discussion about re-writing position statements to try and make a clear distinction between the ethical dilemmas

involved in genetic research and genetic testing and the straight out illegality of genetic doping. This is particularly important, because it seems inevitable that gene doping will happen in sport. We already have the technology to do it successfully in animals. The ASC through the Australian Institute of Sport (AIS) sees its role as the gathering of normative data that can be used for future comparison testing of elite athletes – the most effective means of discovering gene doping cheats.[4]

2. Politicisation of the Issue

Under the previous Federal Government, Ministers for Sport (responsible for the ASC and research in this area) actively discouraged ASC initiatives in genetic and doping research, apparently because the issue was considered a political "hot potato" with the potential to cause the Government embarrassment.

Since the change of Government, a number of members of Federal Parliament, possibly for personal/religious/political reasons, have sought to have the issue of genetic testing and research raised as part of the Senate Estimates process.[5] This latter development caused the ASC to re-canvas the Steering Group responsible for the development of the ASC Policy for advice on an appropriate response. The Steering Committee advised the ASC that in its view, the linking of gene doping with genetic testing for other reasons was unfortunate, as they are two separate issues. The Committee's advice was to produce a new policy which clearly separated genetic testing as dealt with under the current policy from gene doping, which should be simply condemned as cheating.[6]

3. Commercialisation of the Issue

In recent times there have been two broad developments in this area:

a. Gene Patenting.

b. Fee for service genetic testing to profile individuals for athletic talent or disease risk.

The issue of gene patenting is a very controversial area in medical research and probably of no relevance to SMA – except that SMA would probably be supportive of calls, such of those of Professor Ian Frazer, for revisions to patent laws to prevent the locking up of information that could lead to the development of new treatments.[7]

Many current fee for service "genetic testing" for athletic or disease profiles are almost certainly "dodgy" and fail the ASC Policy's test of being "good science"; however, there is no doubt that many such schemes will profit their promoters. Of greater relevance is testing for genes known to be markers for certain injury types and developing a position on these is a major concern of the AIS/ASC (see below).

Current ASC/Australian Institute of Sport (AIS) Position

1. The AIS is currently developing a more detailed position on genetic testing for talent identification with the Hastings Centre in the USA.
2. Genetic markers for certain injury types currently exist (e.g. tendon rupture; concussion/brain damage), but until we know the degree of importance these markers play in risk, there is little point advocating or undertaking widespread screening – of populations or individuals.
3. Gene Doping has been successful in animals and will inevitably move into sport for humans. The ASC wishes to establish a "normative data bank" that can be used in testing to detect gene doping in elite athletes.[8]

The SMA national Board is currently considering the following recommendations:

1. SMA should re-endorse the ASC Policy on the conduct of genetics research, while emphasising the importance of the principal that access to sport should be freely available to all Australians.
2. SMA should support the ASC in its efforts to conduct genetic research AND to establish a normative genetic databank to counteract gene doping.
3. SMA should caution the general public against the use of current commercial genetic profiling schemes for athletic talent and disease susceptibility.

This article is being written at a time when the Crawford Inquiry into sport is touring the country holding public forums where people involved or interested in sport are encouraged to make their views known. A constant theme at these forums is the appropriate divide between funding for elite sport and funding for community sport. It is probable that decisions made in relation to this will be much more relevant to the average person's access to sport in Australia than policies around genetic testing.

Position Statement on Exercise and Hypertension

Sports Medicine Australia (SMA) and the Australian Association of Exercise and Sports Science (AAESS) have jointly released a new position statement on exercise and hypertension. The research for the position statement was undertaken by AAESS members with the aim of updating existing position statements, such as that of the American College of Sports Medicine, to take into account new research findings. The new position statement was published in the most recent (March 2009) edition of the Journal of Science and Medicine in Sport (JSAMS).[9]

AAESS and SMA promoted the position statement through a joint media release which pointed out that hypertension (high blood pressure) is a leading contributor to premature death and disability from cardiovascular disease. Currently afflicting 29% of the Australian population, it is more likely to develop in people who are physically inactive, overweight or who consume excess dietary sodium or alcohol. The cost of antihypertensive therapy is a major impact on community resources, with approximately $300 million spent annually under the Australian Pharmaceutical Benefits Scheme on anti-hypertension drugs. These costs will only rise as the population ages.[10]

The position statement is the first of a planned on-going collaboration between SMA and AAESS to produce new or revised statements on a range of sports medicine related areas. The statements will be fast-tracked for publication through the Journal of Science and Medicine in Sport.

Do they or Don't they…

The pursuit of excellence, or simply the effort to gain an edge over the opposition, have thrown up many challenges to sports medicine and science – especially with the increasing involvement of technology in sport.

Recent editions of the Journal of Science and Medicine in Sport (JSAMS) have provided contrasting viewpoints on the efficacy of one such innovation – compression garments.

The first edition of JSAMS for 2009 had an article looking at the effects of compression garments in netball which concluded "repeated performances at high speed were improved…"[11] The second edition of JSAMS in 2009 contained an article looking at the effects of wearing compression garments on thermoregulation which concluded that "Compression garments do not affect physiological performance during team sport activity in temperate conditions".[12]

Alright – I confess to quoting VERY selectively and both articles found both negative and positive things to say about compression garments. What I hope I have done is whetted your appetites to read the complete articles. The exact references for each are below, or go to www.sma.org.au/publications/JSMS/

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1 *ALRC/AHEC* Report No 96, March 2003, ALRC, Canberra.

2 *Policy on the Conduct of Genetics Research and the Use of Genetic Information in Sport* (ASC, June 2004, page 4).

3 Ibid, page 4.

4 Professor Peter Fricker, Head, AIS, 19.12.08.

5 Senator Cory Bernardi, *Senate Estimates*, February 2009.

6 Professor Ronald Trent, University of Sydney, *email to ASC advisory panel* 3/12/08.

7 Professor Ian Frazer, *Sydney Morning Herald*, 21.11.08.

8 Professor Peter Fricker, Head, AIS, 19.12.08.

9 *JSAMS* 12:2 March 2009, page 252.

10 *AAESS/SMA Media Release* 21.4.09.

11 Higgins et al, *JSAMS* 12.1.2009, page 223.

12 Houghton et al. *JSAMS* 12.2.2009, page 303.

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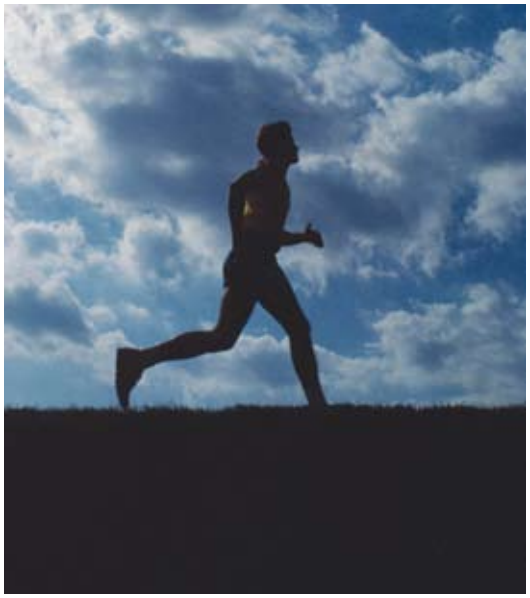

It is always important to be aware of, and take heed of, consensus expert opinion in medicine, but I feel it is equally important to not routinely accept it as gospel. Therefore I tend to love medical anecdotes where a doctor with a radical but intelligent idea which dissented from consensus opinion has later been proven to be correct. The most famous of these in recent Australian medical history was the story of *Helicobacter pylori*, which was proven by two Australian doctors to be a common cause of stomach ulcers.[1] Their story starts with a hypothesis that Robin Warren came up with and Barry Marshall tested. When they initially tried to publish the idea, it was considered so radical that they couldn't even get it accepted as a paper at the Gastroenterological Society of Australia annual conference.[1] Twenty years later they won the Nobel Prize for medicine.

The popular TV medical show *House* is based around a physician called Gregory House who routinely shows up his more conservative medical colleagues by thinking outside the square and coming up with radical but generally correct diagnosis. On the theme I am writing it is a refreshing show in that it paints the medical establishment as often being too conservative. Where *House* is a frustrating show is that it sets an unrealistic ideal that there is such a fantasy expert who is so clever that with enough tests and clever history taking that he can diagnose absolutely everyone. In the show of course, virtually everyone turns out to have a curable disease. If only the real world were so obliging.

I also like to ponder real world counter-arguments that sometimes it is better to just treat a problem functionally rather than insisting on making an anatomical diagnosis in every case. Low back pain is probably a classic example where a large number of patients can be most effectively managed by simply treating their condition (with, for example, mobilisation, core strengthening, NSAIDs, moderate exercise) rather than worrying about whether the pain is coming from their facet joints or discs.[2] I certainly wouldn't say that this is the case for every low back pain patient, but if you had to decide whether to spend someone's last $300 on a lumbar MRI scan or four sessions of functional treatment, in many cases you should probably opt for the latter.

However, in this article I want to write about a diagnosis that as a profession we haven't yet asked enough questions about, which is a sub-type of osteoarthritis. We should all be thinking more about its underlying cause and what we can do to perhaps treat it far more effectively than we do. This diagnosis I will call "Rapidly progressive osteoarthritis". This reminds me of a great line from the movie "A Few Good Men" where Lt Kaffee (Tom Cruise) asks Col Jessep (Jack Nicholson) if the danger in a certain situation was "grave danger"? The answer which Colonel Jessep gave was "Is there another kind?" Joints with osteoarthritis are obviously in danger of further degeneration, but are they are they all in "grave" danger? Is there another kind of osteoarthritis than rapidly progressive osteoarthritis?

These questions have a double answer in that all joints with osteoarthritis are in danger of deteriorating but you'd only call it "grave" danger once you have the benefit of hindsight and have seen the deterioration occur. It seems to be the current status quo in medicine that we tend to treat all cases of osteoarthritis as being the same animal. We genuinely can't give a straight answer to someone who asks "how many years does my knee (or hip or other joint) have left in it?". I can't understand why our profession isn't trying harder to work out what causes some joints to pack it in over a matter of months whereas others can go for years without any change for the worse.

With our regular private patients, who pay to see us each time, we tend to get disproportionate follow-up from the patients who are doing OK but aren't perfect. The complete cures generally don't return to tell us face to face that they are asymptomatic, whereas the complete failures probably seek alternate advice. This discrepancy is doubtless greatest of all for orthopaedic surgeons, who regularly hear vicious attacks on their colleagues by the patients in their offices. They make lots of money but are prone to becoming grumpy as sub-consciously they probably realise what some of their patients say about them in the offices of other surgeons.

Although elite athletes are different to regular patients in many ways, one of the biggest differences is that we actually get better follow-up than we do with most of our normal patients, even if it is just watching them on TV. To illustrate the lack of predictability about joint degeneration I'd like to draw on an example that is already in the public sphere.[3] Joel Selwood, now one of the gun midfielders with the Geelong Cats in the AFL, was a particularly outstanding junior, but had two knee operations for significant chondral damage in the year before he was drafted. A number of clubs apparently were put off drafting him because of his medical history. His story is now a happy one for the player and for Geelong, who took the risk with him. However, I don't believe that this warning, presuming it was given, should be seen as a black mark against any of the medical teams who made it to their recruiting staff. Just as it is thorough to know whether a prospective recruit is a bad kick, it is thorough to know whether he has a good or a bad injury history. There is even a recent paper from the NFL showing how much greater the risk is (of not playing games) for a significant orthopaedic injuries such as knee chondral damage.[4] Sometimes it might be worth recruiting someone in spite of a major injury query – Chris Judd and his shoulders pre-drafting by West Coast and then his groin injury pre-Carlton would be a good example of a risk that has twice paid off, with the benefit of hindsight. It is probably not in as good taste to discuss the many examples of poor recruits who have had ongoing problems with pre-existing injuries and have therefore turned out to be risks which didn't pay off.

As hard as it is for recruiting staff to pick which of the top eighteen year old players will be the best players three years later, it seems to be equally hard for us as doctors to tell which grade III chondral lesions will be asymptomatic in three years and which ones will have progressed on to frank osteoarthritis. I have had two patients this year who were extremes in lack of progression. One was told he needed a knee replacement in 1970 and still hadn't needed it yet and another was told in the same decade he should stop running because of knee osteoarthritis yet has run a dozen marathons since. Both of these patients were seeing me because of Achilles tendon pain. Yet all of us have seen patients who have had a knee which had rapidly progressed from being OK for running to being in knee replacement territory within a few years or even months.

A similar dilemma relates to the value of knee chondroplasty as a procedure itself.[5–7] There are multiple major published papers showing that knee chondroplasty, *on average*, is no better – or may even be worse than – conservative management. Our medical system, which won't fund hyaluranon injections, shoe wedging or physiotherapy interventions that have been shown to be helpful in osteoarthritis, continues to fund a procedure that a Cochrane review claims has "gold" evidence of not being helpful.[8] The problem for the government is that there would be widespread outrage (particularly from surgeons, but also from some patients) if arthroscopic chondroplasty was no longer funded by Medicare. I feel this is because there *are* a significant number of patients who get substantial improvement from a chondroplasty and these patients would be furious to have to pay for the procedure fully themselves. The problem is that – if you believe the RCTs which we know should be more reliable than our clinical observations – there must be an equal body of patients who are made substantially worse by arthroscopic chondroplasty which balance out the ones who get better. Clinical optimism leads us to hope that every arthroscopic chondroplasty will turn out like Joel Selwood, but if we are honest we admit that we see some dismal failures.

So having led in with all of these questions, I want to re-toss up a hypothesis[9] which, if correct, could explain all of these paradoxical findings. The hypothesis is that "Rapidly progressive osteoarthritis" is caused by subtle intra-articular (or subchondral bone) infection and that the progress of osteoarthritis, in the absence of infection, is relatively slow and

benign. When I use the terms infection, I mean possibly any infection (bacterial, viral, fungal or other microorganis) – but what I don't mean is "in-your-face" *Staph. aureus* septic arthritis. Classic septic arthritis certainly causes extremely rapid joint degeneration but it also generally gets rapidly diagnosed and (usually successfully) treated with joint lavage plus intravenous antibiotics.

Current dogma is that bacteria which cause septic arthritis can be routinely grown in pathology laboratories and that the main defence that bacteria has against modern medicine is to develop antibiotic resistance. The medical profession hasn't taken seriously a very plausible defence mechanism that microbes may develop against modern medicine – the inability to grow on an agar plate in a pathology laboratory.[9] The average doctor has the view that if a biopsy or fluid tap is taken from a knee and doesn't grow microbes then there are no microbes present. In other words: the same intellectual error that stopped doctors before Marshall and Warren from thinking of a microbial cause of stomach ulcers.

The infective theory of osteoarthritis progression would explainwhy surgery is curative for some patients with chondral damage but actually worsens the condition in others. If a chondroplasty manages to drill away the focus of infection it might fix the problem, but surgery also runs the risk of introducing further organisms through the surgical portals. But we shouldn't expect a revolution any time soon in surgeons diagnosing a whole lot more post-operative infections in order to better treat those patients who are made worse by their surgical procedure. Since infection is seen as a black mark against the treating surgeon, the dogma that osteoarthritis "is sometimes rapidly progressive for unexplained reasons" is one that sits far more comfortably with surgeons that the thought that post-operative infections are quite common.

So how does one treat an infection in a joint when the causative organism is unknown? Perhaps it has unwittingly already been done – a RCT shows improvement in osteoarthritis with the antibiotic doxycycline,[10] although most experts regard that doxcycline in this trial was working as an MMP-inhibitor. My instinct would be to treat with one or a combination of broad-spectrum antibiotics actually injected intra-articularly. This would mean attacking another one of medicine's sacred cows – that you shouldn't inject antibiotics directly into joints! Of interest is that some vets are happy to inject antibiotics into horse joints[11–12] and it seems to be safe in rabbits.[13] I've tried to work out why and how it became medical dogma that antibiotics shouldn't be injected intra-articularly in humans. The logic is certainly at least thirty years old, but it seems that the argument against intra-articular injection of antibiotics is twofold: (1) that it can lead to "synovitis" and (2) that intravenous antibiotics enter the synovium efficiently and are therefore effective at treating septic arthritis, so that intra-articular antibiotics are not necessary.[14] These two arguments together seem somewhat illogical – if antibiotics can successfully enter the synovium and, antibiotics can cause synovitis, then we should see synovitis caused by intravenous antibiotics as well. Antibiotics in the joint may have benefits and risks and these should be equally pertinent whether or not the antibiotic got to the joint via the bloodstream or via an intraarticular injection. One confounder with observation of "synovitis" in a joint after an intra-articular injection is that it could have been caused by an infection itself. It probably comes down to a question of dose, in that the local dose from an intra-articular injection would probably be higher and be more likely to be closer to a toxic dose. There is only one antibiotic on the market –

flucloxacillin – approved for intra-articular injection (at a dose of 250–500mg/day) and so with the other major antibiotics there is very little data available about toxicity in local tissues like joint. It may simply be defensive medicine to avoid intra-articular injections given that these are not approved by the manufacturers of most antibiotics. Of course the big advantage of intra-articular antibiotics would be that it could be done as an outpatient procedure in minutes, as opposed to intravenous antibiotics which require an expensive hospital stay. There may be some patients, particularly those awaiting joint replacement who have already given up their joint as being beyond the point of no return. The practical advantage of a single rather than ongoing injection(s) is obviously of great benefit in animals[11] who might be less obliging if asked to sit still with an IV drip in situ for three days.

The problem with my hypothesis is that it is very difficult to prove, although you can manage osteoarthritis according to Table 1 and admit you don't fully understand the pathogenesis. Marshall and Warren took over a decade to convince the medical establishment of the common existence of one organism in the stomach. If there were regularly more than a dozen fastidious organisms that can cause hard-to-prove infections in joints, then exhibiting instances of these is only proof for that individual case rather than for the disease in general. The only answer to the solution I can see is a much better medical records database. If there was a country-wide database of all cases of osteoarthritis and, over a large number of patients, perhaps it could be shown that certain patients could have joint replacements successfully deferred with the use of intra-articular antibiotics.

The current status quo possibly suits some of the bacteria which don't get seen and possibly suits the surgeons who don't see them. Remember that there are over 50,000 joint replacements getting performed per year in Australia under a health system which handsomely rewards procedural treatment and pays next to nothing towards surveillance and prevention. So whether we have a problem depends on whether you are a doctor, bureaucrat or an unlucky patient.

	Septic arthritis	Rapidly progressive osteoarthritis	Relatively benign osteoarthritis
Joint swelling	Substantial, red, hot	Effusion mild-moderate	Effusion mild or not present
Lab results	High ESR, CRP, WCC, high joint aspirate white cell	?moderately high ESR, CRP, white cells present in aspirate	Normal
Joint fluid culture	Positive	Negative	Negative
Cause	Pathogenic bacteria (e.g. *Staphylococcus aureus*)	?fastidious bacteria (e.g. *Kingella Kingae*9),?rheumatological condition, ?localised osteoporosis	Trauma
Treatment	Rest & intravenous antibiotics, ?joint lavage	Hyaluranon injections, ?doxycycline, ?other antibiotics (including perhaps intra-articular), ?surgery	Physiotherapy, moderate loading, orthotics, glucosamine

Table 1 – Possible management protocol for osteoarthritis

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The 2009 World Anti-Doping Code and Medical Support Personnel

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In January 2009 the first revision of the World Anti-Doping Code (the Code) came into effect with surprisingly little discussion. At the risk of being branded a "chicken little" and scaring readers, there are very real and serious challenges posed by the new code for the conduct of SMA members. For example, you, the support person, could face serious penalties for having a life saving, but prohibited, drug on your person or for expressing a critical view of the Code while in the presence of athletes under the age of eighteen. Both the World Anti-Doping Agency (WADA) and Australian Sports Anti-Doping Authority (ASADA) have been silent on the interpretation and application of several new and dubious aspects of the Code. We offer this commentary as both a warning and a call to action.

Of concern for SMA members are changes that specify the responsibilities of what the Code calls "athlete support personnel". Page 128 of the Code defines athlete support personnel as "any coach, trainer, manager, agent, team staff, official, medical, paramedical personnel, parent or any other person working with, treating or assisting an athlete participating in or preparing for sports competition". This basically means anyone with any connection to sport, which is probably most of Australia. Under the new Code, athlete support personnel can be sanctioned for a number of reasons. This means SMA members must be aware of their responsibilities and the implications and consequences arising from those responsibilities, or face the prospect of being *banned from sport.*

Violations and Sanctions

There are two Articles of the Code that support personnel need to be aware of. The first is Article 2, which describes anti-doping rule violations. The second is Article 10, which describes the sanctions that can be imposed upon athlete support personnel. In combination, these tell you what you are responsible for and the consequences of non-compliance.

As most SMA members are aware, there a number of "anti-doping rule violations". These are set out under Article 2 of the Code. There are eight possible violations:

- **Article 2.1** covers violations arising from a positive drug test
- **Article 2.2** relates to use or attempted use by an athlete (and strict liability)
- **Article 2.3** is about athletes refusing, failing to evading sample collection
- **Article 2.4** gives effect to out-of-competition testing
- **Article 2.5** protects doping control from tampering or attempted tampering
- **Article 2.6** is about the possession of prohibited substances or methods
- **Article 2.7** relates to trafficking of prohibited substances or methods
- **Article 2.8** sanctions administration, attempted administration, assisting, encouraging, aiding, abetting, covering up or any other type of complicity involving an attempted or actual anti-doping rule violation.

So what is considered an anti-doping rule violation for an athlete support person? Articles 2.1–2.3 make the athlete culpable for drug use. That is, it appears that the Code is not interested in whether athlete support personnel are using prohibited substances or methods. However, Articles 2.4–2.8 do have implications for support personnel.

Article 2.4 includes the rules about correct filing of information on athlete whereabouts. It is unclear from the Code whether an athlete support person with administrative responsibility for filing documents on behalf of an athlete may be liable for sanction if the requirements are not met. The Australian Athlete Whereabouts system makes it the responsibility of the athlete, although this may vary by country. Article 10.3.3 puts the penalty for infraction as a one – two year ban.

Article 2.5 is a fairly obvious violation. If an athlete support person is found to have tampered with or attempted to tamper with any part of the doping control process, he or she could be sanctioned. This could include giving athletes incorrect advice about the testing procedures, selecting the testing vessel for the athlete, dividing the samples for the athlete, or preventing a Doping Control Officer from witnessing sample production. It is incumbent upon athlete support personnel who are acting as chaperones or advocates on behalf of athletes to have clear and accurate knowledge of the system to ensure they make no silly mistakes. The sanction under Article 10.3.1 is two years.

Article 2.6 creates risks for athlete support personnel. Article 2.6.2 states the following:

> "Possession by an athlete support personnel in competition of any prohibited method or any prohibited substance, or possession by an athlete support personnel out of competition of any prohibited method or any prohibited substance which is prohibited out of competition in connection with an athlete, competition or training, unless the athlete support personnel establishes that the possession is pursuant to a therapeutic use exemption granted to an athlete in accordance Article 4.4 (Therapeutic Use) or other acceptable justification".

In a nutshell, Article 2.6 implies that if an athlete support person is carrying a prohibited substance or method, they can be sanctioned for two years under Article 10.2. The Code gives an out of "other acceptable justification". The comment to Article 2.6.2 uses the example of a team doctor carrying substances for dealing with acute, emergency situations. The problem here is that the acceptable justification is ambiguous. For example,

Article 2.6.2 makes it possible for a sports psychologist carrying beta blockers for a heart condition to be banned from sport for two years. So, regardless of intent, support personnel will be expected to demonstrate the same degree of vigilance as athletes about determining the legality of what substances they carry with them or use.

The mechanism given to athletes as a method for avoiding possession sanctions for required medication is to apply for a Therapeutic Use Exemption (TUE). [There is no mention if support personnel should also apply for a TUE as well. Article 2.6 does suggest that support personnel would need to establish a TUE.] This could see a flood of TUE applications clogging the system, which could well

overwhelm places like ASADA trying to manage them. Either that, or athlete support personnel are going to have start avoiding the use of acceptable therapeutic medication for their condition (e.g. pseudoephedrine for a cold) to meet their obligations under the Code. This represents an obvious threat to the health of athlete support personnel, but this is what WADA and ASADA see as appropriate to protect the integrity of sport.

Article 2.7 is another obvious violation. It is clear that if an athlete support person carries trafficable quantities of a prohibited substance or method, they could be sanctioned. Of course, if a support person uses international travel to purchase bulk supplies of a prohibited substance or method that are otherwise legal to bring into Australia (e.g. buying up cheap prohibited pain killers for a chronic injury), they could be sanctioned. The minimum sanction under Article 10.3.2 is four years.

Article 2.8 can produce an ethical dilemma for sport support personnel. The focus of Article 2.8 would appear to be on preventing support personnel from the direct encouragement, aiding and abetting of an anti-doping code violation by an athlete, which is fair enough given the aims of the Code. However, are there indirect behaviours that would be subject to scrutiny and possible sanction? For example, just how much, if at all, can sport support personnel, including parents, openly discuss and possibly criticise anti-doping codes and procedures without it being considered an act of "aiding and abetting" the commission of a Code violation? In other words, the mere possession of a point of view that runs contrary to the anti-doping code may be construed as an offence.

The consequences of a Code violation are severe. Under Article 10.3.2, it specifically prescribes that an incident involving a minor and athlete support personnel mandates "lifetime ineligibility". Under Article 14.2, the identity of an athlete support person found to have committed an anti-doping rule violation could be publicly disclosed. The impact of the disclosure could be far more wide reaching for the athlete support person than any actual sanction.

Education of Athletes and Athlete Support Personnel

Article 18.2 prescribes that athlete support personnel should educate and counsel athletes regarding anti-doping policies and rules. This presumes that athlete support personnel are appropriately educated. In the Australian experience, there has been little in the way of education for the broad church of athlete support personnel. Ignorance of the law is not considered a plausible defence, so it is expected that there will need to be a wholesale commitment by SMA support personnel to professional development in this area. This will likely involve significant economic costs, but it may be a small price to pay in order to prevent inadvertent anti-doping rule violations. The cost of educating such a large body of people – potentially any person even tangentially connected to sport – may be prohibitive, meaning ignorance could become the main cause of anti-doping rule violations among athlete support personnel. The consequence may well be that athlete support personnel are denied access to sport, and therefore jeopardising athletes' training and performances. For example, if parents are banned, who is going to drive future Olympians to the pool every morning?

Roles and Responsibilities of Athlete Support Personnel

Article 21.2 defines the roles and responsibilities of athlete support personnel under the Code. Personnel are supposed to be knowledgeable about and comply with every aspect of anti-doping, co-operate with the athlete testing program, and use their influence on athlete values and behaviour to foster anti-doping attitudes. The latter point has some dubious consequences. It is not enough that sport support personnel, including parents, might have to stifle any critical comments about doping and doping control in sport. It would seem that they cannot even remain neutral on the issue. Article 21.2 seems to go further. It appears that there is an obligation for support personnel to actively promote anti-doping, or else.

Why we are worried

We are concerned that the broadening of policing powers can increase professional risks for sport support personnel and stifle constructive public debate. Forewarned is forearmed. SMA members need to be aware of the implications of the new Code for professional practice. At the same time, SMA also needs to exercise some political muscle to ensure the interests of athletes and their support personnel are not jeopardised by unreasonable or dubious means to purge doping from sport.

Sports Medicine Australia and "Smartplay"



Sport and recreation injury – and fear of becoming injured – is a known barrier to participation in sport and physical activity and is a significant public health issue. By helping facilitate the delivery of safe sport and active recreation, Sports Medicine Australia (SMA), SMA members and accredited Sports Trainers help facilitate lifelong physical activity.

Main aim

Smartplay is Sports Medicine Australia's sports injury prevention program. Its main aim is to help reduce the frequency and severity of sport and recreational injuries, as a means of encouraging and maintaining greater participation in physical activity.

Smartplay achieves this by promoting safer sport and recreation messages through the development, distribution and delivery of effective training, education and health promotion resources focused on injury prevention and management. These include:

- **General injury prevention resources –** warm up, drink up, gear up, fix up, footwear for safety and mouthguards.
- **Sport specific injury prevention fact sheets –** some sports include Australian football, baseball, basketball, cricket, gymnastics, hockey, netball, rugby union, running, soccer, softball, tennis and volleyball (there are twenty-seven sports represented – for the entire list visit www.smartplay.com.au).
- **Injury specific fact sheets –** AC joint, achilles tendon, ACL, ankle sprain, hamstring, quadriceps contusion, gastrocnemius and meniscus.
- **Policies and guidelines –** some include blood rules, emergency planning, hot weather guidelines, safe landing, safety personnel, safety guidelines for children and young people in sport and recreation (for the entire list visit www.smartplay.com.au).

Support

Smartplay also aims to provide easy to use support in many ways in the pursuit of sport safety, including:

Resource development and distribution

- Working in partnerships to develop sports injury prevention resources.
- Providing expertise on resource development.
- Providing access to resources via the Smartplay website, mail outs and ordering.
- Developing sports injury articles for publications (sports newsletters and magazines) and websites.
- Distributing monthly sports safety e-newsletters.

Training

- Building the skills and knowledge of personnel to deal with injuries through injury prevention and awareness training.
- Facilitating links to other Sports Medicine Australia programs such as the Safer Sport Program, Sports Injury Tracker, CleanEdge and Get SMART on line course.
- Providing access to Train the Trainer courses on injury prevention and safety.

Raising awareness

- Raising awareness of sports injury issues by disseminating key messages at grassroots level, through training, resources and media.
- Increasing the profile of sports injury prevention with industry, government and the public.

Supporting and contributing to research

- Delivering cutting edge research at forums and workshops to highlight the importance of sports injury research initiatives.
- Communicating emerging research through training, resources, the Smartplay website and media.
- Providing access to research (Resources, Your Sport section) via the Smartplay website.
- Working with research partners to support and contribute to the ongoing evidence base for sports injury prevention.

Advocacy

- Influencing key stakeholders to place injury prevention on the agenda.
- Supporting and contributing to the ongoing evidence base for sports injury prevention.
- Supporting partnerships to reduce the incidence of sports injury.
- Delivering ongoing expertise and advice on sports injury prevention issues.

Over the years the Smartplay program has grown into a vital part of the sports injury prevention landscape, offering sports participants crucial information to reduce the risk of sports injuries. It has developed partnerships with researchers, state sporting associations, local government, regional sporting assemblies, schools, media and many other community stakeholders. It has become a hub for injury prevention advice and information for stakeholders to build safer sporting environments.

Smartplay currently operates in each state of Australia. Resources are available from Sports Medicine Australia state branches, with samples available for free to SMA members, SMA Sports Trainers and schools and further copies available at a small fee (please note: the number of resources available will depend on state branch capacity).

Smartplay encourages all those working within the sport sector to utilise the resources and support offered by this program to help in reducing the frequency and severity of sport and recreational injuries to encourage and maintain greater participation in physical activity.

For more information

Visit www.smartplay.com.au or contact your local Sports Medicine Australia state branch.

Smartplay is funded by the Australian Government Department of Health and Ageing.

Smartplay Victoria is funded by VicHealth and the Department of Planning and Community Development (Sport and Recreation Victoria).

Smartplay Western Australia is funded by Healthway and the Government of WA Department of Sports and Recreation.

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Surgical procedures performed at an NRL club over 10 years

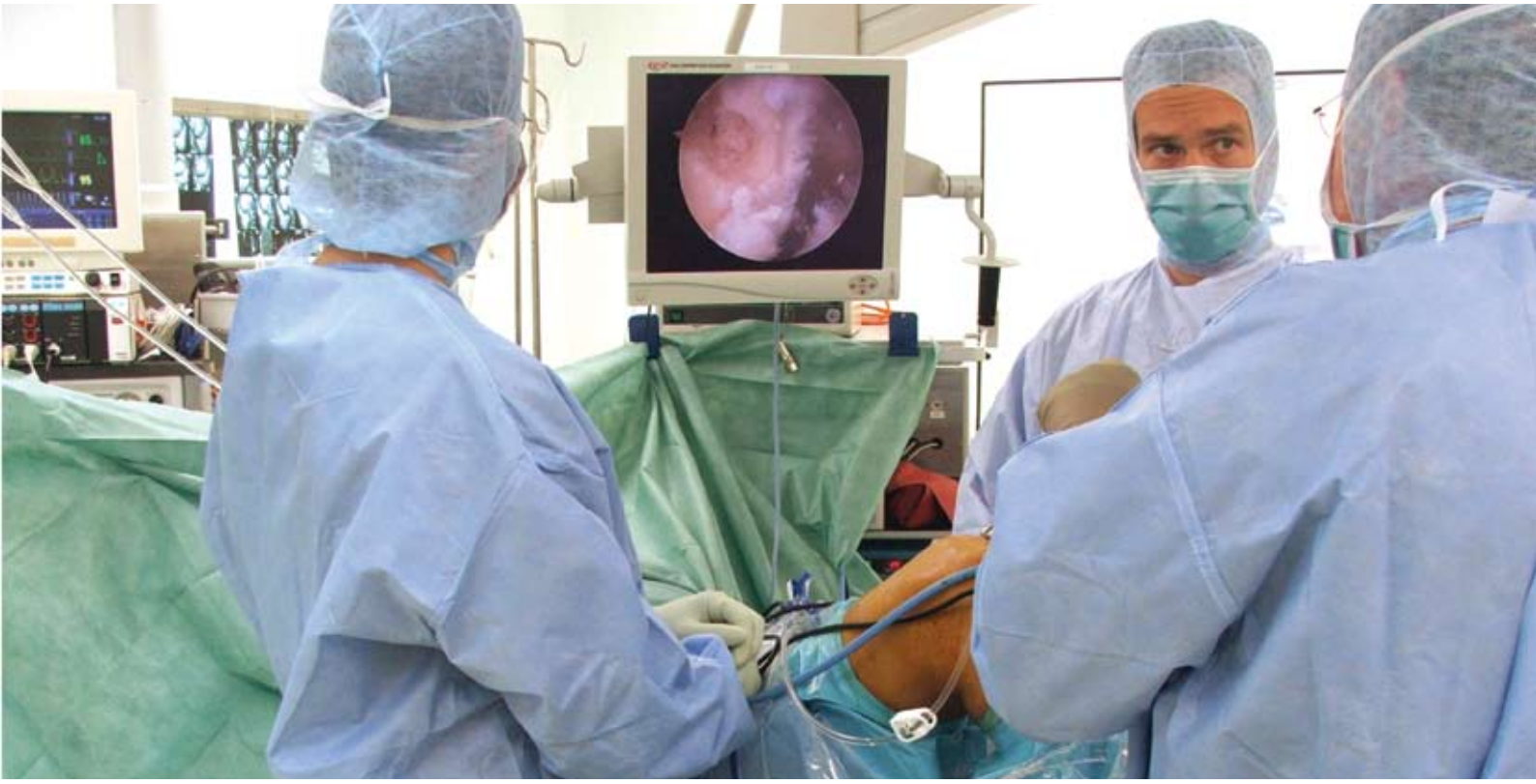

Figure 1 – knee arthroscopy

John Orchard, Sommit Dan, Elizabeth Steet, Merv Cross, Des Bokor, Leon Neve and Jim Carmichael

Abstract

Introduction & Aim

To document the incidence of surgery at a professional rugby league team.

Methods

A prospective database of injuries treated, including operations performed, was kept at the Sydney Roosters NRL team (two senior grades and one junior grade) over the period January 1998 – December 2007. Outcomes, including return to play times, revision surgery required and retirements due to injury were analysed.

Results

A total of 693 (426 senior and 267 junior) player seasons were studied. There were 247 operations performed, representing a rate of surgery of 35.6 operations per 100 player seasons. A slight majority (55%) of these procedures were done in the off-season. The five most common procedures were 4.9 knee arthroscopies per 100 player seasons (25 primary procedures + 9 revision procedures), 4.2 adductor tenotomies per 100 player seasons (25 primary procedures + 4 revisions), 3.0 shoulder reconstructions per 100 players seasons (19 primary procedures + 2 revisions), 3.2 knee reconstructions per 100 player seasons (20 primary operations + 2 revisions) and 2.9 ankle arthroscopies per 100 player seasons (19 primary operations + 1 revision). For both knee and shoulder reconstructions there appeared to be no net change in level of play between pre- and post-operative games. The failure rate of all surgery was 5%.

Conclusions

There is a high rate of surgery utilised for professional rugby league players. This series of moderate size shows good results (in terms of successful return to the same level of professional sport) for those operations that we chose to regularly use, including patellar tendon ACL grafts, open Bankart repair shoulder reconstructions and adductor tenotomies.

Introduction

It is considered that professional football players require surgery more frequently than amateur footballers or, particularly, non-athletes, and that rugby league is a sport leading to a high rate of surgery. However, there has been very little published data regarding the incidence of surgery.

The aim of the study was a descriptive paper to document the incidence of major surgical procedures at a professional rugby league club over ten years. The rates of various procedures will be discussed from a perspective of how much the surgical profile would differ from other professional sports (e.g. Australian football, soccer) and how it may differ from other professional rugby league clubs, reflecting the medical bias of this team in particular.

Methods

A prospective database of injuries treated, including operations performed, was kept at the Sydney Roosters NRL team (two senior grades and one junior grade) over the ten-year period January 2008 – December 2007.[1] Operative reports and games played were cross checked to ensure accuracy of database details. Outcomes, including some return to play times, revision surgery required and retirements due to injury were analysed.

All procedures that required hospital admission for surgery and were related to playing football were included. Stitching/stapling of wounds and reduction of dislocated joints which did not require hospital admission were not included. Joints which required reduction in theatre under general anaesthetic were included. Guided injections of cortisone, even if performed in a hospital facility, were not included. Appendicectomy, tonsillectomy and removal of wisdom teeth were not included as these operations were not considered to be related to football. Reduction of nasal fractures performed as a sideline or dressing room procedure were not included, but were included if they required hospital admission/general anaesthetic.

Results

A total of 693 (426 senior and 267 junior) player seasons were studied. There were 247 operations performed, representing a rate of surgery of 0.356 operations per player per season, detailed in Table 1. A slight majority (55%) of procedures were done in the off-season.

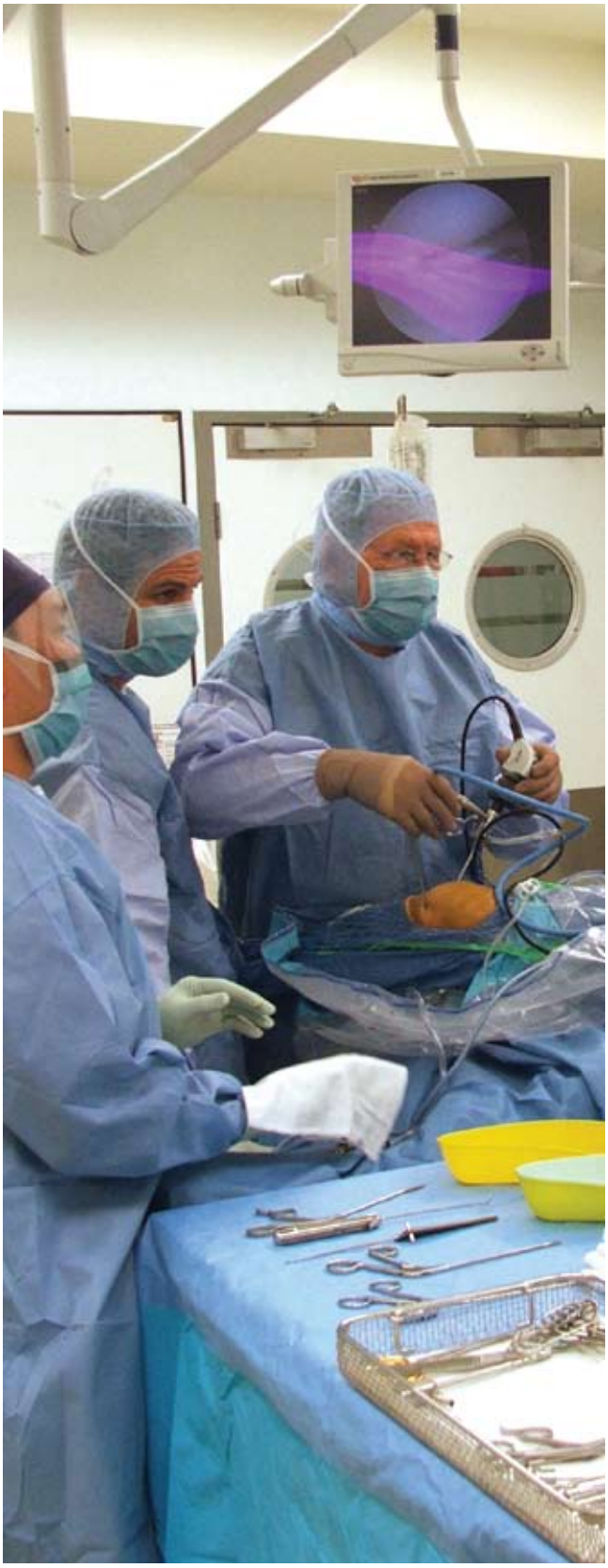

Table 1 – Operations performed over ten seasons at the Sydney Roosters

Body region	Surgery type	In-season	Off-season	Total (revisions from this series, revisions from elsewhere)	Rate (total) per 100 player seasons
Head/neck	Facial fractures – open reductions	13	0	13 (1,0)	1.9
	Sinus/nasal procedures including closed reductions under anaesthetic	1	2	3 (0,1)	0.4
Shoulder	Fractured clavicle fixation	1	0	1 (0,0)	0.1
	Shoulder reconstructions incl. Bankart repairs	4	17	21 (0,2)	3.0
	Shoulder arthroscopes incl. SLAP procedures	3	13	16 (0,1)	2.3
	A/C joint procedures	0	9	9 (1,2)	1.3
Elbow & Arm	Elbow surgery	2	3	5 (0,0)	0.7
	Forearm fracture fixation	3	0	3 (0,0)	0.4
Hand	Fracture fixation – hand	12	1	13 (0,1)	1.9
	Wrist/finger reconstruction	2	5	7 (0,0)	1.0
	Wrist arthroscope	0	3	3 (0,0)	0.4
	Hand tendon repair	1	2	3 (0,0)	0.4
Lumbar surgery	Discectomy	3	0	3 (0,0)	0.4
Hip/Groin/Thigh	Adductor tenotomy	5	24	29 (3,1)	4.2
	Hernia repair	0	4	4 (0,0)	0.6
	Other hip/groin/thigh surgery	5	1	6 (0,0)	0.9
Knee	Knee reconstructions	22	0	22 (0,2)	3.2
	Knee arthroscopes	17	17	34 (6,3)	4.9
	Other knee surgery	5	6	11 (3,0)	1.6
Shin/Foot/Ankle	Shin compartment release	2	1	3 (0,0)	0.4
	Ankle arthroscopy	2	18	20 (1,0)	2.9
	Fractured shin bone	7	0	7 (0,1)	1.0
	Other foot & ankle surgery	2	2	4 (1,0)	0.6
General	Removal of metal procedures	0	7	7 (7,0)	1.0
Total		112	135	247 (24,13)	35.6

Knee arthroscopies

The most commonly required procedure was the knee arthroscopy (approximately 15% of all operations). Half of these were done in the off-season and half in-season.

There were twenty-five primary surgeries performed and nine revisions. Of the revision procedures, three were revisions of previous operations performed outside this series (i.e. players recruited from other clubs), four were arthroscopies done on players who had previously had knee reconstructions in this series and two were revisions of arthroscopies in this series. Therefore of the twenty-five primary arthroscopies in this series, 8% (two procedures) required revision. Of the twenty-two knee reconstructions, 18% (four procedures) required minor revision in the form of subsequent arthroscopy.

The primary pathology noted was six medial meniscal tears, thirteen lateral meniscal tears, three loose bodies and thirteen chondral lesions. One lateral meniscal tear was able to be treated with a repair (meniscal arrow), whereas the other meniscal tears were treated with partial menisectomy.

Of those done mid-season, there was a median four games missed before return to play. The quickest mid-season return was in just under three weeks from surgery (two players missed only two matches). The slowest return was one player who took ten months to return from a significant chondral lesion of the lateral femoral condyle.

Knee reconstructions

Patella tendon autografts (nineteen cases, figure 2), fixed with interference screws, were generally used for ACL (anterior cruciate ligament) reconstructions.[2] In two cases, hamstring tendon autografts were used, fixed with interference screws. In one case the ACL was directly repaired as it avulsed from its femoral attachment. Two cases were revisions of ACL reconstructions performed elsewhere and in both of these, patellar tendon autograft was used (one ipsilateral as the previous reconstruction had used hamstring tendons and one contralateral as the previous reconstruction had used the patellar tendon, fixed with a transfix screw).

None of the knee reconstructions required revision whilst at our club, although one of the hamstring autografts required a revision two years later when the player had moved to a different club. However, as mentioned, 18% of these players later underwent knee arthroscopy. There was no apparent net change in level of play between pre- and post-operative games (see Table 2). In general, one would expect about 30% of lower grade players to go on to play first grade football and about 15% of first grade players go on to play representative football. This series showed a much higher level than expected return or progress of players on to representative rugby league, with an average progress of lower grade players on to being first grade players. This finding is impressive given that ACL reconstructions in the NFL[3] and AFL[4] have been associated with poor career progression. One NRL player who had a revision procedure was not able to re-sign a first grade contract although this was not considered to be due to problems with the knee surgery. A second player was injured playing in his first grade debut – he actually underwent bilateral ACL reconstructions, having an acute injury from the game and an old partial tear of the ACL on the other knee. This player to date has continued his career in the lower grades but has not returned to first grade.

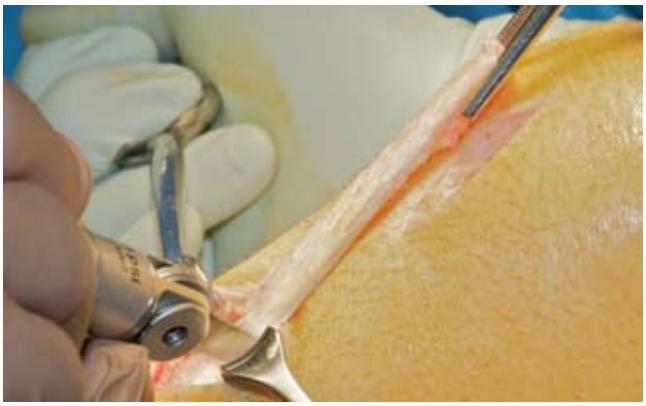

Figure 2 – patellar tendon graft harvesting

Three of the players with patellar autografts (and also the player who had the direct repair) managed to achieve State of Origin selection after successfully returning from ACL procedures, two of these for the first time subsequent to their injury. One additional player was selected as a country representative player and one player, who had transferred to rugby union, was selected for the Wallabies, both of these with patellar tendon autografts. One player who had a hamstring autograft subsequently played Test football for Samoa. Of the seven players who played representative football after an ACL reconstruction, three were outside backs (who require the most agility), three were forwards and one was a utility player.

The median time taken for return to play was eight months, with the lowest successful return to play time being 4.5 months and the highest twelve months. The player who returned 4.5 months after injury was the only one who played in the same season as the original injury, with all other players tending to resume playing early in the season after their injury, which ranged from six to twelve months.

Table 2 – Fate of Sydney Roosters players who underwent ACL reconstruction

	Later representative player	Later first grade player	Later lower grade player	Total
Previous representative player	2	0	0	2
Previous first grade player	4	4	2 (3*)	10
Previous lower grade player	1	1	7	9
Total	7	5	9	21

* Two players one of whom had a bilateral ACL reconstruction

Table 3 – Fate of Sydney Roosters players who underwent shoulder reconstruction

	Later representative player	Later first grade player	Later lower grade player	Retired	Total
Previous representative player	3	0	0	0	3
Previous first grade player	1	4	0	1	6
Previous lower grade player	1	2	9	0	12
Total	5	6	9	1	21

Shoulder reconstructions and other surgery

Open Bankart repairs (eighteen cases) were generally used for shoulder reconstructions. In two cases arthroscopic Bankart repairs were performed.[5] In one case there was an open direct capsular repair (i.e. neither a Bankart nor HAGL lesion). No revisions of these procedures were required (i.e. no dislocations or subluxations occurred subsequent to shoulder stabilisation procedures in this series). There was a median return to play after shoulder reconstruction of 5.5 months. There was no difference in level of play pre- and post- shoulder reconstruction (see Table 3). Only one player did not play football after his shoulder reconstruction and this was after having made a previous decision to retire at the end of the season in question, after which he had the procedure. He successfully had a career in manual work and felt his shoulder was strong enough to play again and was certainly not the cause of his retirement.

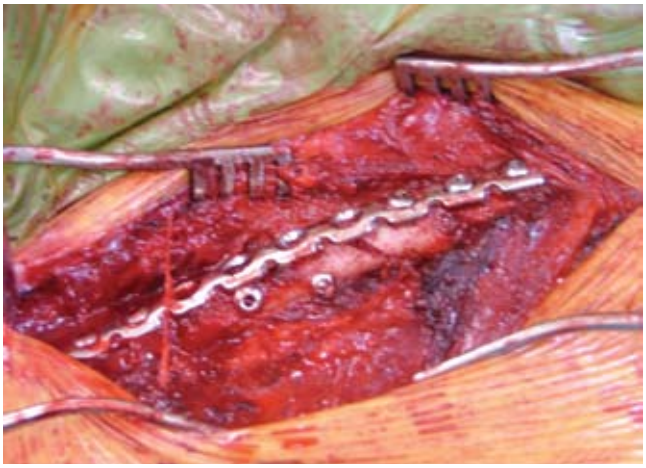

Figure 3 – plated clavicle fracture

Other than the twenty one reconstructions for glenohumeral instability, the following other shoulder surgeries were performed: nine primary acromioclavicular procedures (one reconstruction and eight debridements/resections), sixteen shoulder arthroscopies (including three SLAP procedures in combination with A/C procedures, seven other SLAP procedures and one mini-open rotator cuff tendon repair) and one internal fixation of clavicle fracture (Figure 3). All eighteen shoulder dislocations that occurred at the club over the ten year period were able to be reduced without general anaesthetic.

As can be seen from Table 1, the general approach to shoulder instability at the club, which was highly successful, was that players suffering significant shoulder instability (dislocation or recurrent subluxation) could play the season out (after missing a few games) and then were strongly encouraged to have a shoulder reconstruction at the end of the season.

Groin and hip surgery

There were four hernia repairs performed and twenty nine adductor tenotomies (Figures 4 and 5). The other six hip/groin/region region procedures included two testicular repairs, two reductions of dislocated hips under general anaesthetic, one debridement of deep scar tissue in the thigh and one acute thigh compartment release. Although there were five MRI-documented cases of hip labral tear, no hip arthroscopies were performed. Groin and hip injuries are treated by a variety of different surgeries.[6] At our club there was a particular bias towards the primary use of adductor tenotomy for chronic groin pain. This perhaps can be explained by the fact that Dr Orchard's predecessor as club doctor at the Roosters, Dr Neil Halpin, was a specialist in performing adductor tenotomy procedures in rugby league players. There were many players in the squad in 1998 who had previously undergone a successful adductor tenotomy, particularly using Dr Halpin. When future players suffered from chronic groin pain, they spoke to players who had suffered the injury in the past and, having heard of generally successful results of their procedures, opted to choose the same operation.

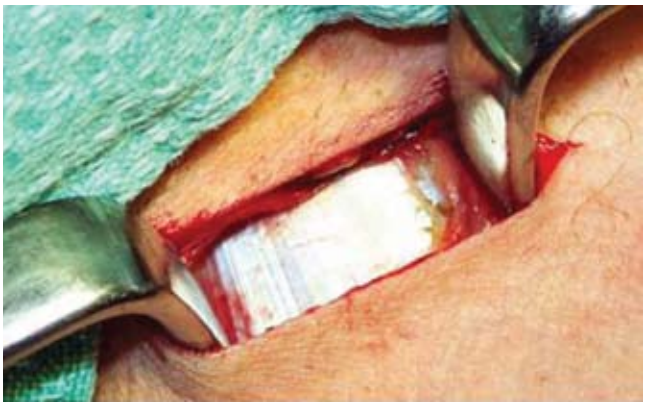

Figure 4 – adductor tendon pre-tenotomy

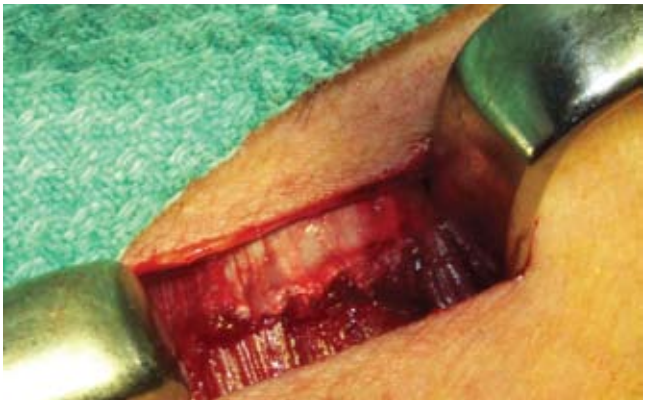

Figure 5 – adductor tendon post-tenotomy

Ankle arthroscopy & foot surgery

There were twenty ankle arthroscopy procedures, the vast majority of which were able to be done at the end of the season. Four procedures included removal of posterior impingement lesions (os trigonum or enlarged posterior process) – one of these was required in-season as an acute procedure for an acute fracture of the os trignonum.

There were four fibula fractures that involved the ankle joint and required surgery. Two were lateral malleolus fractures fixed (and sufficiently stabilised) with plate and screws. Two were low fibula fractures that required plate and screws plus a syndesmosis screw in addition. Therefore these subsequently required a further operation to remove the syndesmosis screw. Two players required sydnesmosis stabilisation in the absence of fibula fracture. Both of these players had run on the injury fairly soon after sustaining it and therefore may have been partial syndesmosis tears which extended upon further weightbearing. A syndesmosis screw was used in both these cases, which was subsequently removed two months later.

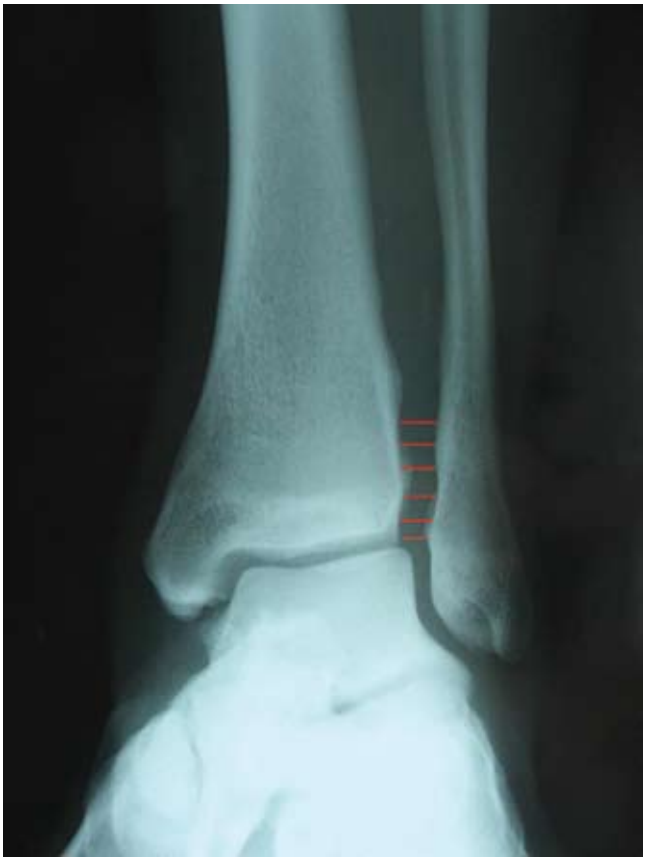

Figure 6 – syndesmosis separation seen on weightbearing X-ray

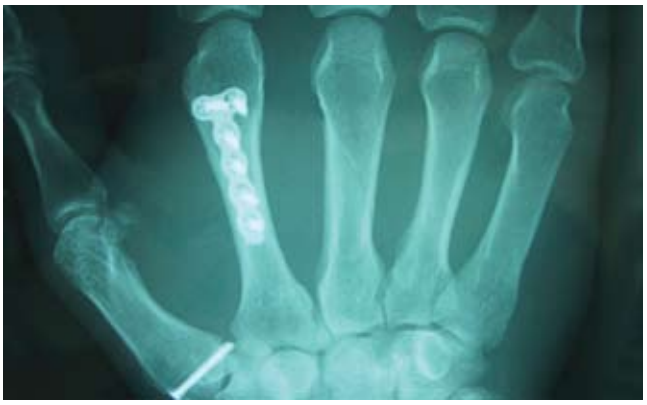

Figure 7 – fixation of Bennett's fracture and second metacarpal fracture

Hand surgery

There were seven wrist/hand ligament reconstruction procedures, the majority of which were performed at the end of the season. There was one thumb carpometacarpal stabilisation, one thumb metacarpophalangeal volar plate acute repair and one delayed reconstruction of a fourth proximal interphalangeal joint. There were four scapholunate reconstructions performed – none of these were done acutely although one was performed in-season towards the end of the season.

Of the hand tendon lesions, there were two mallet finger repairs performed and one acute fourth finger flexor tendon repair performed. There were three cases of complete flexor tendon ruptures which occurred mid-season to players who had a strong desire the play out the season and who opted not to undergo acute repair. None of these players decided to have a delayed reconstruction of the tendon and all lived with the slight permanent disability associated with the flexor contraction and poor strength in the one finger. In all three cases the players involved played in finals series in successful teams in that season and this likelihood appeared to be the major determining factor in opting against surgery.

Of the thirteen hand and finger fractures that required surgery, four fractures affected first metacarpal, three affected second metacarpal and one each affected third and fifth metacarpals. There were three scaphoid fractures that required Herbert screw fixation, one of which was a revision from a player who has sustained the injury at another club and who required a bone graft for non-union. There was one trapezium acute fracture which required internal fixation.

Other fracture fixations

Of the twelve facial fractures which required thirteen surgeries, three involved the orbital floor. One of these which also required an acute revision as the diplopia did not resolve after the first surgery. One of the other players suffered an orbital floor fracture in a Preliminary Final win and elected to play the Grand Final with diplopia prior to having his fractured fixed in the week after the Grand Final. The third orbital floor fracture was fixed acutely.

There were six unilateral zygomatic-maxillary complex fractures which required elevation and two unilateral mandibular fractures which required plating. There was one extremely severe La Fort type bilateral maxillary fracture with posterior displacement that required emergency hospital admission for intubation, as the fracture threatened the airway. This occurred during a Test match rather than a Roosters NRL match.

There were three radius fractures which required plate and screw fixation. None of these players underwent removal of metal in this series.

There were three tibial shaft fractures which required fixation with a tibial rod. One of these was a revision procedure to a player who had previously had a fractured tibia managed with plate and screws and who re-fractured through a screw hole subsequent to plate removal. The two other tibial fractures required acute surgery (tibial rod fixations). One of these players (an outside back) subsequently underwent removal of the rod whereas the other player (a forward) has kept the rod in place. Both of these players required additional associated minor surgery – one was an ankle procedure for spur associated with distal pin and one was a knee procedure for debridement of scar associated with proximal pin.

Surgical failures and operations which required revision

The three operations which were considered, in this series, to be surgical failures which required fairly acute revision prior to return to play were:

1. One orbital floor plate which failed to cure diplopia and was revised within weeks.
2. One hernia repair for chronic groin pain which failed to cure the pain and which was revised with a successful adductor tenotomy.
3. One knee arthroscopy for lateral meniscal tear which had ongoing swelling after partial lateral menisectomy and which required revision partial lateral menisectomy and chondroplasty.

There were a further six revision operations that involved the player returning to play for the following season but complaining of ongoing symptoms and requiring a revision procedure at the end of the next season including:

1. One revision A/C joint debridement.
2. Two further revision adductor tenotomies.
3. One medial ligament reconstruction, which was performed initially with a ligament tightening (delayed repair) and then on revision reconstructed with a hamstring graft.
4. One revision prepatellar bursectomy.
5. One further revision knee arthroscopy.

There were thirteen procedures which were revisions of operations performed elsewhere (outside this series).

There were fifteen operations in this series that were technically considered to be revision procedures but which were not considered surgical failures, including seven removal of metal procedures, two debridement of scar procedures after screw removal, five knee arthroscopies subsequent to otherwise successful ACL reconstruction and one syndesmosis screw insertion after diagnostic ankle arthroscopy.

There was one surgical procedure considered to be a failure in a player who subsequently retired, and for which no revision procedure was attempted. This was a sesamoidectomy for chronic sesamoid pain. The player was able to move onto a career in manual work but complained of ongoing ball of foot pain and felt that he would not have been able to continue to play football if he had desired to.

Overall there were ten operations from this series that were considered surgical failures (four acute and six after one season). There were another fifteen operations that were revision procedures where the initial surgery achieved its primary aim (i.e. fracture fixation, ACL reconstruction). There were thirteen revisions from procedures performed outside this series.

Therefore the surgical failure rate for the 210 primary operations was 5%.

Causes of players leaving the club

Table 4 shows a record of status of players upon leaving the club, indicating that only two players seemed to be physically unable to play football after leaving the club, one of which was due to a medical condition seemingly unrelated to football which did not involve surgery. The majority of players leaving the club either had no outstanding injury problems or moved directly to another club in the NRL or English Super League.

Table 4 – Role of injury in Sydney Roosters players leaving the club

Injury status at time of leaving club	Junior players	Senior players
A – player was physically no longer able to play rugby league	0	2
B – injury was a major contributing cause to the player leaving the club	1	7
C – injury was a minor contributing cause to the player leaving the club	16	16
D – injury had no apparent bearing on the player leaving the club	61	41
Player moved directly to another NRL or UK Super League team	34	102

Injuries which were treated conservatively where surgery could have been used

Some injuries which we generally treated conservatively where surgery could have been used included medial ligament tears, hip labral tears, knee posterior cruciate ligament tears, ankle syndesmosis sprains and some hand fractures.

Table 5 – Matches missed from 64 medial ligament injuries

Matches missed	Number of injuries
0	21
1	10
2	8
3	13
4	6
5	4
7	1
8	1

Table 5 shows that the vast majority of medial ligament injuries returned to play in under six weeks. Generally our management of medial ligament injuries included a quick return to play with strapping for grade 0–1 injuries. For grade 2–3 injuries we treated with a valgus-limiting brace for an unspecified number of weeks, checking the laxity of the medial ligament every few days and removing the brace as soon as the ligament regained tightness. There were only two players in which this management appeared to be less than optimal. One player had six weeks in a brace and did not tighten up and required an end-of-season repair, which was not successful, and then a reconstruction the following season. He is still playing rugby league successfully but felt as though his knee function was limited for a long time by ongoing medial laxity. A second player had a previous history of surgically-repaired medial ligament on the other knee at a different club. This was successful but with a return to play of approximately twelve weeks. We were able to get him back to play with conservative management after seven weeks, which was somewhat of a success. However, he did remark that despite returning to play he ultimately felt more confident with the surgically-repaired knee. Both of these players who had less than optimal conservative outcome were grade three medial ligament tears at the tibial end of the ligament. Based on our experience, we believe that femoral end grade three medial ligament tears can generally be managed conservatively in professional footballers, enabling quick and successful return to play. This advice probably applies to many tibial end medial ligament tears and also to posterior cruciate ligament tears.

Generally we successfully managed acute isolated ankle syndesmosis tears conservatively, with an extended (4–6 weeks) period of non-weightbearing in a brace. Those players who required syndesmosis stabilisation in our series either had an associated fibula fracture (which itself required surgery) or did not undergo an initial period of immobilisation.

Despite at least five cases of hip labral tear, we were able to manage all players without needing to use hip arthroscopy. We are aware that hip arthroscopy is a much more commonly-utilised procedure in AFL players in Victoria in particular. This probably reflects a combination of the following factors: (1) That AFL players have higher demands on the hip joint due to regular punt kicking (2) That surgeons in Melbourne have more experience with hip arthroscopy and are more likely to recommend it as treatment.

The majority of hand fractures which required surgical fixation were to first and second metacarpals, as these bones are more likely to displace if they are not surgically fixed. In general, players with isolated third and fourth metacarpal fractures and many phalangeal fractures were able to be managed conservatively, including some who were able to continue playing without missing matches with the assistance of local anaesthetic injections.

Discussion

This case series of moderate size shows good results in terms of successful return to the same level of professional rugby league for the surgeries chosen. At this team, generally patellar tendon ACL grafts were used for knee reconstructions,[7] open Bankart repairs were used for shoulder reconstructions[5] and adductor tenotomies were used for groin surgery.[8] Other types of surgery are available. Typically hamstring autografts are the major alternate surgical option for knee reconstructions, arthroscopic Bankart repairs are the major alternate surgical option for shoulder reconstructions and hernia repairs and hip arthroscopies the major surgical options for hip & groin pain. There are certainly biases and preferences which underpin choices of surgical technique. In a professional rugby league team, the most pertinent factor in the choice is the (previously successful) results of past cases. We would not necessarily submit that patellar tendon ACL grafts, open shoulder stabilisations and adductor tenotomies necessarily always lead to better results than the alternate operations that are available – simply that in our club over a prolonged period with twenty or more of each surgery performed

that the results have been very good in terms of return to successful play. The gold standard level of evidence is the randomised control trial, yet it is doubtful if an RCT could ever be performed in the setting of professional athletes. It is quite conceivable that the slightly reduced morbidity of hamstring tendon (or for that matter non-autologous graft) knee reconstructions and arthroscopic shoulder reconstructions may make these operations more favourable choices in lower demand athletes. It is also possible that other aspects of surgical technique or post-operative rehabilitation have led to our good observed results rather than simply broad choice of operation type. That the current literature does not conclusively show proven differences between most surgical techniques means that we believe that "past results" will continue to dictate the choice of surgery for professional athletes. We would encourage professional athletes to choose surgery type and also surgeon based on who and what their peers have had successful outcomes with. We accept that this is somewhat of a subjective choice. Hopefully this large case series can give a realistic appraisal of the likely success rate of surgery in professional athletes with good quality surgical technique and post-operative rehabilitation.

Comparison with AFL professional teams

Surgery in AFL players

An informal survey was undertaken in season 2004 of the AFL injury survey in which teams could voluntarily hand over data of players who undergo surgery during, or after, the season.[9] Eight teams (half the competition) provided data of surgical operations. If it is presumed that these eight were representative of the other eight teams, then it is likely that:

1. Slightly over 200 operations were performed on AFL players in 2004 (approximately three operations for every ten listed players).
2. Approximately half of the conditions that required surgery did not result in any missed playing time (i.e. the operations were performed in the off-season). Shoulder reconstructions, knee and ankle arthroscopes and various hand procedures were the surgeries that were most likely to be done in the off-season and not impact on playing time.

Table 6 – Operations performed at 8 out of 16 AFL teams in season 2004

Body region	Surgery type	On injuries causing missed games	On injuries where no game was missed	Rate (operations per 100 player season)	Rate higher in AFL or NRL
Head/neck	Facial fractures	5	2	2.0	
	ENT, Dental procedures	0	1	0.3	
Shoulder	Fractured clavicle fixation	2	1	0.9	
	Shoulder reconstructions/repairs	4	6	2.9	
	Shoulder arthroscopes	1	3	1.2	NRL
	A/C joint procedures	1	0	0.3	NRL
Elbow & Arm	Elbow surgery	1	1	0.6	
	Forearm fractures	2	1	0.9	
Hand	Hand surgery	10	10	5.8	AFL
Hip/Groin/Thigh	Groin surgery	1	1	0.6	NRL
	Hamstring surgery (incl. neurolysis)	3	2	1.5	AFL
	Hip arthroscopy	2	1	0.9	AFL

Body region	Surgery type	On injuries causing missed games	On injuries where no game was missed	Rate (operations per 100 player season)	Rate higher in AFL or NRL
Knee	Knee reconstructions	7	0	2.0	
	Knee arthroscopes	7	10	5.0	
	Other knee surgery	0	1	0.3	
Shin/Foot/Ankle	Shin surgery	1	1	0.6	
	Ankle arthroscopy	3	10	3.8	AFL
	Other foot & ankle surgery	4	2	1.8	
Medical illness	Other surgery	2	1	0.9	
Total		56	54	32.1	Similar

Rates of many surgical procedures in this NRL series are quite similar to the small number of reports of surgical procedures at AFL clubs. Perhaps NRL players may undergo slightly more surgery as the injury profile of the competition is more contact-orientated (compared to the AFL with has a more non-contact profile). However, the overall rates of surgery in these two competitions appear to be quite similar. NRL players probably undergo more shoulder surgeries than AFL players, but fewer hip and ankle arthroscopies. The higher rate of groin surgery in this NRL series may reflect team bias of the Sydney Roosters and the preference to use adductor tenotomy early in chronic groin pain, rather than a reflection that chronic groin pain requires more surgery in NRL players than AFL players.

The structure of the medical team is fairly similar in the NRL compared to the AFL, with the majority of team doctors either sports physicians or GPs with a strong sports medicine experience. Most players have their surgery performed in the city in which their team is based at the recommendation of the team doctor. Occasionally players will be sent interstate for either an operation in the home state of the player or to use a specific surgeon proficient in a particular procedure.

Surgical management at AFL and NRL teams may diverge slightly in the near future due to the relative financial strengths of the two competitions and perhaps due to the existence of free agency in the NRL. AFL clubs have tended to substantially increase their spending on medical staff and medical expenses over the past decade. This is partially because of the perception that medical outcomes strongly affect team performance in the AFL and partially because of the increased profitability of AFL teams. Salary cap laws have limited highly profitable teams from spending this profit on playing staff, so a trend has been to spend on football support staff like coaching, recruiting and medical staff to gain an advantage. By comparison, in recent years NRL teams have faced reduced profitability because of the lesser TV rights deal and over-reliance of NSW teams on leagues club grants, which have been substantially reduced due to recent taxation laws. In general, many NRL teams have tried to limit or even reduce their spending on all football (non-player-payment) expenses out of apparent financial necessity.

A further difference for the increased prominence of medical team performance in the AFL is the lack of free agency. Because established players can only move from club to club through trading and the draft, there is relatively little movement of players between teams. An AFL team that achieves poor results from surgery is possibly stuck with an injury-prone list that will lead to ongoing high rates of missed games and, potentially, lower trade-value. By comparison, there is enormous player movement between clubs in the NRL due to the presence of free agency. A player who has a poor result from surgery is individually affected due to his reduced market value. However, if he is off contract, as many players are from year to year, his club has no particular hold over him to consider him an “asset”. Poor surgical results can often become someone else’s problem if a club simply makes a player a poor offer of retention and he is signed by someone else. In this series, good surgical results with respect to ACL reconstructions have not necessarily been to the benefit of this club! There are five current representative players (as of

2009) who have had successful ACL reconstructions with the Sydney Roosters who are now playing at other clubs. This may be somewhat of an inevitable outcome with free-agency. A club who has a highly-paid player who misses a season with an ACL injury may be more reluctant to offer him top market value, but when there are many other clubs bidding it may be in the player's interest to move clubs and retain a high salary. In the NRL, a club does not receive any compensation if a player leaves, nor does the recruiting club pay compensation. In the AFL, a player who is coming back from an ACL injury may be less likely to move clubs, as a second club may not want to part with a draft pick for a player who is coming back from a major injury.

Comparison with European professional soccer teams

Rates of surgery at NRL clubs are almost certainly higher than in professional soccer, although there is very little information about the latter. The most common injuries in soccer players are muscle strains, which are typically managed conservatively. Tibial fracture fixation procedures and groin surgeries (particularly hernia repairs) are probably more common in soccer players but surgery to the upper body is probably far less common in soccer players.

There is a very different culture in European soccer with respect to surgery in that many players prefer to undergo surgery in their home country rather than in the country of their team. Choice of surgeon is therefore sometimes made directly by the player and his management staff than by the team medical staff.

Club doctors in Europe, relative to Australia, are more likely to be GPs than sports physicians and therefore probably have less of an opinion on choice of surgical technique, leaving the decision entirely to the surgeon.

Despite the huge player salaries and transfer fees in European soccer and therefore the high importance of surgical outcomes, there is a distinct void in the literature regarding surgery in professional soccer players. This may reflect a poor sports medicine culture with respect to soccer at the highest level in Europe. Many of the medical systems in Europe are highly public in nature and therefore probably do not optimally cater for professional athletes. The solution to surgical failures for the top clubs may be to "buy another player" rather than to assess whether surgical outcomes have been optimal!

Comparison with NFL professional teams

Rates of surgery in professional American football appear to be very high and perhaps may even exceed that presented here for an NRL team. Collision injuries are extremely common in the NFL although overuse injuries are probably far less common. Case series from the NFL have often been presented in the *American Journal of Sports Medicine*.[3, 10–11] Generally the US surgical literature from professional football is the world's most comprehensive.

The make-up of the medical team in the USA is typically quite different to Australia. The majority of team doctors are surgeons themselves, which possibly adds a bias towards the recommendation of surgery by the team doctor. The quality of surgery in the USA at the top end may be amongst the highest in the world because of the for-profit mentality of the health care system. However, conflicts of interest may mean that the best surgeon is not always chosen for each procedure. Because of the enormous profits in private surgery in the USA and the open-slather approach to medical advertising, some teams actually appoint their medical staff based on sponsorship arrangements with the team rather than medical merit. There is a greater upside for a surgical group to be associated with a team in the USA as they can advertise this association to the general public and possibly make a healthy profit out of it. These arrangements tend to also breed a degree of scepticism in the players and their managers, who sometimes realise that team medical appointments are not necessarily made on merit. As a result, there is often a player or manager-based demand for second opinions and the use of independent consultant surgeons. The risk of lawsuit is also substantially higher in the USA and hence recommended management of players may be far more defensive. Ironically it may lead to surgery being offered more regularly (with the risks and complications detailed) so that there can be no accusation of failing to definitively treat a condition.

Conclusions

There is a high rate of surgery utilised for professional rugby league players. This series of moderate size shows good results (in terms of successful return to the same level of professional sport) for patellar tendon ACL grafts, open Bankart repair shoulder reconstructions and adductor tenotomies.

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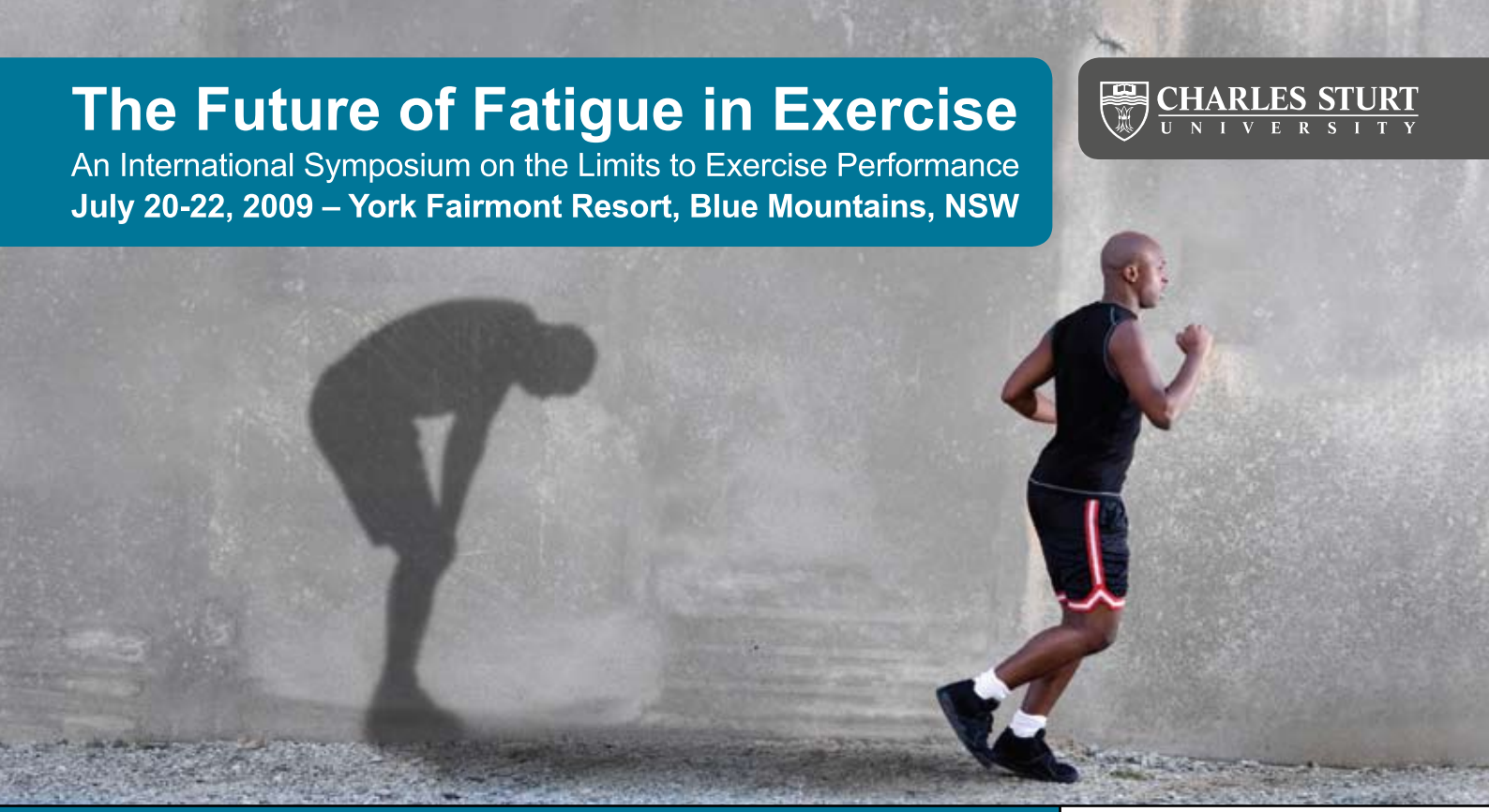

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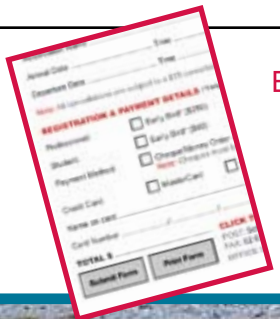

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Ultra-endurance Nutrition: Does the type of carbohydrate matter?

Alan McCubbin

Carbohydrate – a limiting factor in ultra-endurance performance

The long and continuous nature of ultra-endurance sports like ironman triathlon, ultra-marathon running and stage race road cycling places unique physiological demands on athletes. The sheer length of these sports mean that the body's ideal carbohydrate needs over the duration of the event will exceed the body's capacity to store it. Carbohydrate loading will maximise glycogen storage (endogenous carbohydrate), but providing carbohydrate during exercise through food and fluid (exogenous carbohydrate) is vital to maximise the amount of carbohydrate available for oxidation, and to at least some degree preserve glycogen stores for when they may be most needed – in a sprint finish.

Traditional recommendations have been to ingest carbohydrate at a maximum rate of sixty grams per hour, since it was thought that further increases in carbohydrate ingestion rate could not further increase its uptake and oxidation, and would increase the risk of gastrointestinal disturbance.[1] However carbohydrate oxidation during cycling at 60% of VO_2 max can be as high as one hundred and twenty grams per hour.[2] Ingesting sixty grams per hour of carbohydrate spares glycogen, because it provides an alternate source of carbohydrate to be oxidised (compared to no carbohydrate ingestion).[2] However, over an extended period of exercise there comes a point where glycogen storage will diminish and no longer make up this shortfall, since total carbohydrate oxidation exceeds the sixty grams per hour ingested. The result will therefore be a decline in carbohydrate as a proportion of the fuel oxidised, and a decline in performance, commonly known by athletes as "bonking" or "hitting the wall".

In examining reasons that ingested carbohydrate uptake and oxidation plateaus at a certain point, several possibilities emerge. Hawley et al showed that intravenous infusion of glucose could dramatically increase the absolute amount of exogenous carbohydrate oxidised, suggesting that the limiting factor was gastric emptying and/or intestinal absorption of ingested carbohydrate.[3]

Multiple types of carbohydrate can be beneficial

In 2000, a research group at the University of Birmingham began to look at the absorption of different types of carbohydrate.[4] They noted different transport pathways in the gut for glucose and fructose; SGLT1 for glucose and GLUT5 for fructose. Subsequent work investigated the absorption and oxidation of carbohydrate with varying amounts of glucose and fructose (the simple sugar found naturally in honey and some fruits).

What resulted was a series of publications from 2004–2006 demonstrating that ingestion of glucose as a sole source of carbohydrate resulted in endogenous oxidation rates of around sixty grams per hour, however addition of fructose could result in an increase of a further thirty grams per hour of carbohydrate oxidation.[5-13] The source of glucose and fructose was not important.[11] Combinations of glucose in the form of free glucose, sucrose (disaccharide of glucose and fructose), maltose (disaccharide of glucose and glucose) and varying molecular weights of glucose based polymers (maltodextrin, a low molecular weight polymer and amylopectin maize starch, a high molecular weight polymer) produced similar findings. Only trehalose, a disaccharide of glucose and glucose that does not have the same effect on tooth decay as other carbohydrates, was not successful.[14]

It seemed that the observed increase in exogenous carbohydrate oxidation and subsequent sparing of glycogen stores from ingestion of glucose and fructose at 60 and 30g/hr respectively might be of benefit to endurance athletes, however these studies did not measure performance outcomes per se. One of the studies measured Rating of Perceived Exertion (RPE) and cycling cadence over five hours of cycling, with an observed trend for reduction in RPE, and better maintenance of cycling cadence in the final hour for the group receiving a combination of glucose and fructose as opposed to a group receiving the same amount of glucose alone.[13] The glucose + fructose group also reported less feeling of "stomach fullness" compared to the glucose only group.

To date there has been only one published study looking directly at the performance benefits of ingestion of a combination of glucose and fructose on endurance exercise performance, published in early 2008.[15] Participants completed two hours of cycling at 55% Wmax, followed by a time trial to complete a set amount of work (roughly equal to about one hour of cycling at 75% Wmax). Subjects consumed either 90g/hr of glucose, 90g/hr of glucose and fructose in a 2:1 ratio (60g/hr glucose plus 30g/hr fructose), or water. The group consuming glucose showed a 10% improvement in time trial performance compared to the group ingesting water. However the glucose and fructose group showed an additional 8% performance improvement above that of the glucose only group.[15]

This result unsurprisingly had flow-on effects in the supplement market. A few companies have already changed their product formulations to achieve a 2:1 ratio of glucose:fructose, claiming this to be the ideal ratio. However it should be noted that it is the total amount of glucose and fructose ingested per hour, not the ratio in individual foods that would allow for increased intestinal absorption and oxidation of carbohydrate. The same ratio when only 45g/hr of carbohydrate is ingested found no difference to glucose alone.[16] This is likely to be due to saturation of the glucose intestinal transporters (SGLT1) at 60g/hr of glucose; additional fructose only being of benefit where total carbohydrate intakes exceed 60g/hr.

The practical implications

The first question raised by many would be the capacity of ultra-endurance athletes to consume greater than 60grams per hour of carbohydrate. A 6% carbohydrate-electrolyte drink consumed at around 800ml per hr would provide 48g of carbohydrate, and many athletes would struggle to consume greater volumes of fluid than this. Gastric emptying of water appears to be maximal at around 1000ml/hr,[17] and for those with lower rates of sweat loss than 1000ml/hr, fluid consumption would not be recommended to these levels. Thus achieving an intake of 90g/hr of carbohydrate in this example would require 42g from food sources, such as one sports bar or 1.6 gels an hour.

Another potential strategy to achieve 90g/hr of carbohydrate ingestion would be to increase the carbohydrate concentration of the sports drink consumed, however more highly concentrated solutions are known to reduce the rate of gastric emptying and fluid delivery.[18] Recently, the same research group have published data suggesting that addition of fructose can actually increase gastric emptying above that of glucose alone.[17] In this study an 8.6% solution of glucose and fructose in a 2:1 ratio achieved a similar rate of gastric emptying and "fluid delivery" to water, reducing RPE compared to glucose or water alone when cycling for two hours at 61% VO_2max.[17] At this concentration, 90g/hr of carbohydrate could be achieved with 800ml of sports drink and just one gel, or half a sports bar.

It should also be noted that it is the total amount of each type of carbohydrate, not the ratio in particular foods or fluids ingested that is important. Athletes consume a wide variety of foods and fluids during ultra-endurance exercise, with varying amounts of glucose, fructose, disaccharides or their polymers. With current food labelling laws there is no guarantee the exact amounts of glucose and fructose in products can be determined, although the ingredients list may give some indication of the presence or absence of them.

What about athletes with fructose malabsorption?

Fructose malabsorption is a relatively common disorder in people with Irritable Bowel Syndrome, whereby fructose or fructans (a polymer of fructose molecules in some vegetables and wheat) are not completely absorbed by the small bowel. Excess fructose then enters the large bowel, creating an osmotic effect that draws water with it. The fructose is then fermented by colonic bacteria, resulting in production of gas, which together with the osmotic effect can cause bloating, abdominal discomfort and motility changes (diarrhoea and/or constipation). At least a third of people with Irritable Bowel Syndrome (IBS) are thought to be fructose malabsorbers. Fructose malabsorption can be diagnosed effectively and treated on a day to day basis with a great improvement in gastrointestinal symptoms.

Recommendations for management of fructose malabsorption state that it is only when the amount of fructose exceeds that of glucose, or when total fructose intake exceeds the amount the is able to be absorbed that gastrointestinal symptoms occur. Given that this concept fro endurance exercise aims to maximise both glucose and fructose absorption (but not exceed their capacity) and that twice the amount of glucose to fructose can be absorbed, this is unlikely to present a major issue. Some individuals with severe fructose malabsorption may have reduced capacity to absorb fructose, and the level ingested may need to be lowered from 30g/hr to a level that eliminates gastrointestinal symptoms. Excessive quantities of fructans (over 0.5 grams per serve) may cause gastrointestinal symptoms in susceptible individuals. For athletes during competition fructans would likely only come from white bread (0.6g per slice), other products made with wheat flour (4g per 100g), or bananas (0.6g per 90g banana).

Summary

Carbohydrate is a limiting factor in performance in ultra-endurance sports, with glycogen depletion a major cause of fatigue. Ingesting carbohydrate will help to spare glycogen, however the current recommended rate of ingestion (60g/hr) will not completely spare glycogen because total carbohydrate oxidation will exceed ingestion at a competition pace. Maximising the absorption and hence oxidation of ingested carbohydrate by providing 60g/hr of glucose plus 30g/hr fructose during exercise may be a way to improve glycogen sparing, and has been shown to increase cycling time trial performance over three hours of exercise compared to ingestion of glucose alone.[15] The addition of fructose also appears to improve gastric emptying and fluid delivery compared to glucose alone, allowing ingestion of carbohydrate solutions of at least 8.6% without detrimental effects on gastrointestinal comfort.[17]

Blanket recommendation of the 2:1 ratio of glucose:fructose should be interpreted with caution. If athletes are not ingesting greater than 60g/hr of carbohydrate, then there is no benefit of specifying the type or ratio to consume. Caution may also be needed with individuals that have severe fructose and fructan malabsorption, but because the maximal amount of glucose absorbed is twice that of fructose, severe gastrointestinal problems are unlikely.

It will be fascinating to see where this research leads in future. Obvious avenues include the performance outcomes of athletes from a variety of different endurance sports, and ingesting carbohydrate in solid or liquid form. Regardless, the recommendation of specific carbohydrate types and quantities may become commonplace in endurance sport in the years to come. For specific information on achieving adequate carbohydrate intake during sport or about diagnosing or managing fructose malabsorption, speak to a sports dietitian (www.sportsdietitians.com.au).

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Summer football – a toxic mix? Journalism, predictions and reality

John Brotherhood[a] and Phil Chapman[b]

They say that it is the game played in heaven. In reality its origins are in the mists and mud of the English vales. And in the stygian gloom of Welsh valleys, where smoke from winter chimneys falls down the hillsides. And in pierless Wigan where another version is played in the bone-aching damp of west Lancashire. What then is fast and furious professional rugby doing in the muggy weather of late Brisbane summer?

Problems with heat stress

The Brisbane Times recently reported that players in a Super 14 rugby match played in Brisbane in early March lost as much as seven kilograms of sweat. This is almost certainly an exaggeration. Such a sweat loss suggests a prodigious sweat rate exceeding four kilograms per hour for the eighty minute game. According to team medical and coaching staff actual sweat losses did not exceed five kilograms. But even this indicates sweat rates exceeding three kilograms per hour and provides clear evidence of severe heat stress.

The heat stress for this game can be estimated.[1] The dominant drive for sweating arose from exercise heat production. The rate of heat production by a rugby forward with body mass 110kg, maximum aerobic power 55mlO2/kg/min and playing at an overall intensity of 75% VO2max is about 1590 Watts. In the reported conditions of the Super 14 game -32°C and 53 % relative humidity – convective heat loss would have been negligible and temperature regulation dependent almost entirely on evaporative cooling. To balance the player's heat load of 1590 Watts a sweat evaporation rate of about 2.4kg per hour would be required.

So why the very high sweat rates?

The explanation is likely to be that there was environmental limitation to sweat evaporation. Sweat evaporation depends on absolute humidity and air movement over the skin and is impeded by clothing. Estimation of the evaporative capacity of the environment for the conditions of the game suggests that the maximum rate of evaporation that could be achieved was about 1.6kg/h providing evaporative cooling of about 1100 Watts. Thus only 70% of the required evaporative cooling could be achieved resulting in body heat storage at 490 watts and a progressive rise in body temperature. In warm conditions, in the face of restricted evaporation, the increasing core temperature together with high skin temperature increases the drive for sweat production in a physiological attempt to regain control of body temperature. This, at least in part, explains the players' high sweat production.

For the rugby forward heat stress must be exacerbated by the heat produced by "static" muscle activity in scrums and mauls, activities in which air movement over the skin, so important for evaporation, is minimal. As one player remarked "It was pretty tough, especially forwards coming out of scrums. It probably goes up another 5–6 degrees in a scrum when you've got that many bodies there".

Sport in the heat

Heat stress in sport arises from exercise and the environment. Body core temperature, of most concern for health and safety, depends on exercise intensity and is independent of a wide range of environmental conditions. Skin temperature, on the other hand, varies with environmental conditions. The stress and discomfort of warm conditions is due to high skin temperature and is exacerbated in high humidity by wet skin. Thus the main difference in stress between summer and winter football lies in the skin temperature. Football can be played in some conditions in which body temperature control is not possible, albeit at considerable discomfort to players, because core temperatures may not reach intolerable levels in the duration of play. The risk of over-heating would be reduced further by "drink breaks". Such breaks may be less important for water replacement than for allowing core temperature to fall. Although sweat rates can be high in summer football dehydration is progressive and becomes significant only late in the game. Dehydration is often blamed for poor performance but highly trained players tolerate dehydration well and poor performance is just as likely to be due to some other cause of heat intolerance. Skill may also suffer in the heat for other reasons – hands wet from running sweat make ball handling as difficult as playing in rain.

In warm conditions the immediate post-game recovery period may present as much difficulty for players as the game itself. In these conditions the high skin temperature leads to increased skin blood flow. On cessation of exercise the skin full of blood combined with reduced leg muscle pumping may result in an excessive drop in blood pressure accompanied by light headedness or fainting.[2] Clearly there is risk of injury if a player falls in a faint. Thus players should be encouraged to keep moving immediately after exercise or be assisted to lie down with raised legs if they feel faint.

Heat stress and professional sport

The costs of heat stress in sport are distress and discomfort, impaired performance and compromised safety. Some of these effects were graphically described in the Brisbane

Times. A player who fumbled the ball a number of times was reported to have said "I felt a bit off on the field, the heat really got to me". After the game some players were said to have felt light-headed and appeared to be disoriented, one apparently fainted and struck his head when he fell, and other players were said to have vomited. These dramatic events seem to have been overstated. None the less they serve to illustrate the physiological if not medical consequences that can arise from strenuous exercise in the heat. Medical and coaching staff agreed that the players were "spent" after the game, but they did not have any significant concerns for their health.

Warm and humid conditions pose a difficult problem for professional sport. Commercial obligations – "the play must go on" – and duty of care to players must be balanced. Players may to some extent protect themselves from excessive heat stress by slowing down, but that in turn may be undesirable, causing performance to fall below players' and spectators' expectations.

In determining appropriate strategies for managing competition in warm and humid conditions event organisers and athletes must decide what level of heat stress is tolerable based on acceptable decrements in performance, levels of discomfort and risk to health and safety. Obviously the risk of having to play in excessive heat is minimized by scheduling competitions in the late autumn and winter. But if competition in warm conditions is unavoidable there should be guidelines which include predictions of environmental conditions that are likely to impose significant stress on players and strategies for reducing the stress of competing in hot and humid conditions. Individual players must also always have the right to take a break or cease play if they feel over-stressed.

For example, the ARU heat stress policy allows "drink breaks" in addition to the half time break and the AFL allows unlimited player interchanges. While monitoring and managing player performance levels has become part of the art of coaching in Australian Football and players have the protection of highly qualified and knowledgeable medical staff this is not available to community sport.

Heat stress and community sport

Unlike professional sport community sport can exercise more choice and control over the conduct of competition in warm weather. Players must judge the impact of heat stress for themselves, modify their exercise intensity and request breaks in play if they feel overstressed. It is also very important to recognize that individual heat tolerance varies widely.

Even amongst well trained athletes some individuals are more susceptible to heat stress than others. Heat tolerance can also vary from day to day in the individual, perhaps related to subclinical illness. Observers must be alert for poor performance, loss of skill and coordination and other abnormal responses, and instruct affected players to cease play.

Research needs

Estimates of heat stress in sport similar to those used here were used to inform the guidelines in SMA's Beat the Heat fact sheet (www.sma.org.au/information/launch.asp). But with present knowledge estimation of heat stress in football is inevitably crude because it is based on many assumptions. Information is not available about fundamental matters such as exercise intensity and energy expenditure (exercise heat production), skin temperatures, core temperatures, air movement over players (critical to convective and evaporative heat exchange) and sweat rates. Furthermore, the validity of prediction of heat stress has not been tested by comparison with direct observation of its impact on players. Data with which to examine statistical associations between environmental conditions and effects on players are required to provide evidence-based policies and guidelines for managing heat stress in sport. Sweat loss provides the comprehensive indication of heat stress since it accounts for exercise heat production, heat exchanges with the environment, and the effects of humidity. Routine measurements of players' sweat losses, and their subjective assessments of thermal sensation, stress and impact on performance, along with record of air temperature and humidity, could reveal the limits of tolerable environmental heat stress for football.

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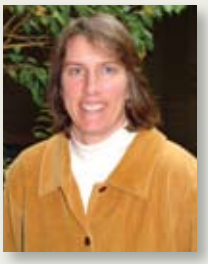
"be active '09" keynote and invited speaker profiles

Dr Carolyn Broderick • University of New South Wales

Carolyn Broderick is a sports physician and specialist in Paediatric Sports Medicine. Carolyn's current research interests include sports injury surveillance in children and exercise prescription for children with chronic disease.

Dr Carolyn Emery • University of Calgary, Canada



Carolyn Emery works in paediatric orthopaedics and sport medicine. Her research focuses primarily on injury prevention in sport, with a particular interest in the child and adolescent population. The primary focus of Carolyn's research program is the identification of risk factors for injury and evaluation of prevention strategies to reduce the risk of injury in youth sport.

Plenary Session NPAC Keynote Speaker

Mr Mark Fenton • *Asics Sponsored Speaker*
National Center for Bicycling and Walking & the University of North Carolina's Pedestrian and Bicycle Information Center

Mark Fenton is a contributing editor for Health magazine, was host of the America's Walking series on PBS television, and is author of numerous books including Pedometer Walking and The Complete Guide to Walking for Health, Weight Loss, and Fitness. Mark has numerous research publications related to exercise science and activity promotion, as well as on community planning and design to promote physical activity and health. He is a vocal pedestrian and bicycle advocate and recognised authority on public health issues and the need for community, environmental, and public-policy initiatives to encourage more walking and bicycling. His expertise includes planning policies and engineering approaches to create active living environments.

Refshauge Lecturer

Professor Caroline Finch • *Australian Sports Medicine Federation Fellows Sponsored Speaker*
University of Ballarat

"Sports injury prevention – no longer lost in translation"

Caroline Finch is Australia's leading sports/physical activity injury epidemiologist. Her research has been published in over 250 peer review journals, research reports and other publications. Her injury research focuses on methodological advances in sports injury surveillance and injury data coding; evaluations of the effectiveness of injury prevention measures; assessing attitudinal and behavioural barriers towards the uptake of safety measures; and the translation of research evidence into policy and practice.

Dr Paul Fleming • Loughborough University, United Kingdom

Paul Fleming has a research portfolio primarily in the areas of geomaterials in transport infrastructure & built environment and sports surfaces & interactions. Current and recent work on sport surface research includes: measuring player perceptions; contrasting player experience to mechanical test methods; simplified assessment test methods; modeling the behaviour of shockpads in surface systems; mechanisms of traction resistance; sport movement player surface loading; in-shoe pressure measurement systems; sustainable drainage systems; effects of maintenance on carpet life; and many more.

Mr Simon Gianotti • Accident Compensation Corporation (ACC), New Zealand

Simon Gianotti is a sport injury prevention practitioner from New Zealand who works with sports organisations to develop and implement community prevention programmes on a national scale. These sports include rugby union, rugby league, soccer (football) and netball. In addition he has developed successful prevention initiatives to reduce specific injuries such as concussion, strains and sprains. Simon's recent work has been developing links between the promotion of physical activity (particularly walking, running and cycling) and sports injury prevention within the same programme.

Dr Marc Hamilton • ***Queensland Government Sponsored Speaker*** • University of Missouri, USA

"Inactivity Physiology: a new paradigm for exercise science"

Marc Hamilton performs translational research integrating molecular mechanisms with pragmatic lifestyle issues, especially studies seeking to understand the causal mechanisms linking sitting too much with vascular and metabolic disease risk factors – the metabolic effects of physical inactivity. The interest in "inactivity physiology" is bringing a new appreciation of the role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, some cancers, and cardiovascular disease. He has argued that sitting too much is not the same as exercising too little. He will summarise the evidence leading to the emerging new discipline of "inactivity physiology".

Professor Steve Harridge • King's College London, UK

"Blood sweat and satellite cells. Adaptability of the aged muscle to overload"

Steve Harridge is a physiologist with a wide ranging (genes to function) research interest in human skeletal muscle function and plasticity, with a particular focus on ageing muscle. Steve is Convenor of the Human Physiology Special Interest Group of The Physiological Society.

Dr Karl Landorf • ***Asics Sponsored Speaker***
La Trobe University, Australia

"Do foot orthoses prevent injury? A systematic review"

"What do we really know about plantar heel pain/plantar fasciitis?"

Karl's main research focus is the evaluation of the effectiveness of musculoskeletal interventions and his primary teaching is in the area of diagnostic imaging of the foot and ankle. His current research projects include evaluations of interventions for plantar heel pain, osteoarthritis in the foot, and painful plantar callus. In addition, he is involved in investigations relating to health outcome assessment instruments used in podiatry, muscle activity in the lower extremity, and the use of X-ray measurement for foot abnormalities. Karl is also actively involved in compiling systematic reviews that summarise the evidence relating to interventions used in podiatry.

Dr Michael Lloyd • Queensland Academy of Sport & Australian Institute of Sport, Australia

"The knee bone's connected to the head bone – the role of sport psychology within a multidisciplinary sports medicine practice."

Michael Lloyd's work encompasses a wide variety of issues including, performance enhancement, pain management, treatment adherence, and other related personal counseling. Michael has done extensive research into areas such as performance routines, motivation, pre-competition emotions and arousal, and has presented his research findings both nationally and internationally.

Professor Tom Marwick • Princess Alexandra Hospital & University of Queensland, Australia

Tom Marwick is trained in Medicine and Cardiology and has authored about four hundred papers, thirty-six book chapters and six books in the field of cardiac imaging and particularly echocardiography.

Dr Lyle Micheli • Children's Hospital Boston & Harvard Medical School, Boston, Massachusetts, USA

"Back Pain in Young Athletes"

"ACL Complex Injuries in Young Athletes"

Lyle Micheli, is director of the Division of Sports Medicine at Children's Hospital Boston and Clinical Professor of Orthopaedic Surgery at Harvard Medical School. In November of 2005, Dr. Micheli assisted in the writing of an IOC Consensus Statement on the Training of the Elite Child Athlete. Other interests include ballet and rugby. His present research activities are focused on the prevention of sports injuries in children, including assessment of risk factors and injury occurrence, as well as assessment of dysfunctions of the shoulder in children and young adults.

Plenary Session ACSMS Keynote Speaker

Dr Lorimer Moseley • ***Queensland Government Sponsored Speaker*** • Prince of Wales Medical Research Institute, Australia

"Pain. Do you get it?"

Lorimer Moseley is a scientist and a clinician. His work in understanding complex pain disorders, and in developing and testing novel strategies to manage them, has received world-wide recognition. He has written two books, several book chapters and over sixty papers in top-flight journals. His work has been the focus of numerous popular press articles in fifteen countries. In 2007, he was judged to be the outstanding mid-career clinical scientist working in a pain-related field by the International Association for the Study of Pain

Dr Chris Rissel • University of Sydney, Australia

Chris Rissel, Director of the Health Promotion Service, Sydney South West Area Health Service, and for the last six years has a strong record of published research with over two hundred peer-reviewed publications and dozens of other reports and papers. He has worked in the areas of migrant health, tobacco control, sexual health, and is currently active in physical activity and active travel research, with a number of large intervention studies focused on cycling and walking.

Dr Mark Tarnopolsky • McMaster University Medical Center, Hamilton, Ontario, Canada

"Sex differences in exercise metabolism and nutritional implications."

"Exercise and ageing – benefits of different types of exercise"

Mark Tarnopolsky's research focuses on nutritional, exercise and pharmacological therapies for neurometabolic (primarily mitochondrial) and neuromuscular disorders, and aging. In addition, he studies the physiological and molecular aspects of mitochondrial adaptation to exercise, aging and the metabolic syndrome. His laboratory has been also been very interested in understanding sex differences in muscle damage and in response to endurance exercise and the role of 17-B-estradiol.

Plenary Session NSIPC Keynote Speaker

Professor Willem van Mechelen • ***Brisbane City Council Sponsored Speaker*** • VU University Medical Center, Amsterdam, Netherlands

"Sports injury prevention: the proof of the pudding is in the eating."

Willem van Mechelen, MD, PhD, FACSM, FECSS is employed by the VU University Medical Centre in Amsterdam as a full professor of Occupational and Sports Medicine. He leads a group of about forty persons who conduct primary care research in the area of work, physical activity, sport and health. Willem is a board certified occupational physician, epidemiologist and human movement scientist.

Dr Evert Verhagen • VU University Medical Center, Amsterdam, Netherlands

Evert Verhagen, PhD is an occupational epidemiologist and human movement scientist and his work is on the prevention of ankle sprains, but also has a strong focus on safety and injury prevention in youth sports and physical activity, as well as the uptake of the interventions within a broad sporting population.

Calendar of upcoming events

Sports Physiotherapy Australia (SPA)

Upcoming events

In the past twelve months, seventeen APA Sports Physiotherapists have now passed rigorous exams with the Australian College of Physiotherapists to become APA Specialist Sports Physiotherapists and Fellows of the College.

SPA are excited by the development of the new spinal course by SMA in conjunction with the existing Australian Surf Lifesavers course which will provide our members with a great opportunity to upskill in this critical emergency care area.

For more visit www.physiotherapy.asn.au

Australian College of Sports Physicians (ACSP)

The Australian College of Sports Physicians is pleased to announce its new President – Dr Andrew Garnham.

In other news, ACSP recently held its ACSP Clinical Sports Medicine Conference with the annual ACSP conference scheduled after SMA's "be active '09" conference. ACSP members are also looking at conducting activities at "be active '09" in conjunction with Sports Physiotherapy Australia. Watch this space for more information!

For more visit www.acsp.org.au

Sports Dietitians Australia (SDA)

Upcoming events

(International Society for the Advancement of Kinanthropometry)
Exercise Research Australia (ERA) will be facilitating a series of ISAK Level 1 courses in 2009.

Queensland
Level 2 course: Saturday 10 – Tuesday 13 October 2009

Nutrition for Exercise and Sport Course

Victoria
Friday 13 June, Saturday 5 September

Queensland
Saturday 25 July, Saturday 31 October

New South Wales
Saturday 20 June

South Australia
Saturday 22 August, Saturday 14 November

For more visit www.sportsdietitians.com.au

Australian Association for Exercise and Sports Science (AAESS)

The AAESS title, logo and "Accredited Exercise Physiologist" title and logo were officially trademarked earlier this year. Exercise Physiologists are university trained allied health professionals who specialise in the delivery of exercise, lifestyle modification and behavioural modification for the prevention and management of injuries and chronic diseases. Exercise Physiology is now the sixth most utilised allied health service under Medicare in Australia and AAESS represents over 2000 members.

AAESS National hosts ongoing workshops in all capital cities to support the continued enhancement of professional qualifications, knowledge and competency. For information about the workshops, membership or to find an Exercise Physiologist visit www.aaess.com.au

The AAESS Business Forum: Sydney 23 – 24 May
Is your business on track?

In May 2009, AAESS will launch the first inaugural AAESS Business Forum. The key objective of this forum is to support businesses in the sport and exercise industry to develop, grow, and become profitable and successful operators.

Run over one weekend this forum will highlight everything you need to know about running a business, including:

- Developing business partnerships
- Achieving growth and success
- Working effectively
- Marketing for success
- Standing out from the crowd
- Website needs of a small business
- Your insurance and legal obligations
- Financing and leasing
- Top 10 tax tips.

AAESS continuing education opportunities

May

Musculoskeletal Case Studies of the Spine and Lower Extremity
31 May, Brisbane 8:30am – 5:30pm (8 CEP's)

June

Improving Communication: Skills for Clinicians and Clients
20 June, Adelaide 9:00am – 5:00pm (8 CEP's)

TAS Seminar Series – Strategies to Enhance Adherence to Exercise + Motivational Interviewing
24 June, Launceston 6:00pm (2 CEP's)

For more visit www.aaess.com.au

Sports injury prevention; the proof of the pudding is in the eating?



Professor Willem van Mechelen, MD, PhD, FACSM, FECSS

Department of Public and Occupational Health
VU University Medical Center, Amsterdam, the Netherlands.

What have I been doing all these years?

Well that's fairly simple. I have been trying to lead an active life, but moreover I have been studying both the benefits and the threats of a physically active and sporty lifestyle.

When I first started doing this seriously, some twenty years ago, there was still a big "disbelieve" amongst the medical profession about the positive relationship between a physically active lifestyle and health. The professor of cardiology, who taught me cardiology basics at medical school in the early eighties of the previous century, actually ridiculed physical activity and exercise. He bluntly stated during classes that *"it is better to sit still, because exercise might drop you dead".*

I had a hard time buying that statement, as a trained PE-teacher. At the same time in European politics and in the media there was a great emphasis on the negative side of participation in sports; i.e. *"it might get you injured"*. As a function of the latter, around 1986 a European Union program was launched; *"Sports for all; the prevention of sports injuries"*. That program marked the beginning of my scientific career. Because there was money available, I started to study the primary prevention of sports injuries, later followed by other pragmatic topics in the broad area of physical activity, sports and exercise, work and health.

What have we been doing all these years?

Well that's also fairly simple; we have been endlessly counting sports injuries.

Most papers on sports injury epidemiology deal with the incidence and sometimes with the severity of sports injuries to assess the magnitude of the sports injury problem. Target groups for such studies vary from the entire population, to specific subgroups according to gender, sports, age, level of play, and so on. Usually such studies reveal that there is a substantial problem, although even to date many papers still don't adequately account for exposure (game and training time), thereby providing inadequate information on actual risk.

What we have been doing next is assessing factors and mechanisms that play a vital role in increasing sports injury risk, but to a lesser extent. If the aim is to prevent sports injuries, these two steps are logical of course; if there is no problem no prevention is needed and if there is a problem, one needs to know about causes and mechanisms in order to prevent effectively. Risk factors that pop-up consistently in the literature are previous injury, age, gender, level of play and exposure. Not so strange, if this is what one studies.

Next step would be to conduct studies that attempt to lower injury risk. The design for such studies is that of a Randomized Clinical Trial (RCT's). In performing such RCT's it helps to work from a conceptual model that describes the sequence of affairs that eventually lead to sports injury. In the early days of sports injury epidemiology a number of fairly simplistic conceptual models were put forward, such as the model described by myself in 1992 and that described by Winne Meeuwisse in 1994. In 2001 Parkkari and colleagues

published a review in Sports Medicine in which up to that time point only sixteen RCT's had been described in the literature: a fairly meager result, I would dare to state.

What have we been doing in recent years?

In recent years we have continued to count injuries and we have continued to assess risk factors and mechanisms, because that's fairly easy to do. Since 2001 obviously some additional RCT's on the effectiveness of preventive interventions have been carried out also. In addition, adaptations of the conceptual models have been put forward and new conceptual insights were formulated and described. Examples of such insights refer to the implementation phase of interventions and to the fact that sports injury prevention requires above all, behavioral change.

However, from what is reported in the literature in recent years, it clearly seems difficult to push the boundaries of sports injury prevention research beyond the counting stage.

Often it is said as an excuse that it is difficult to conduct RCT's in this arena. I can't deny that this is true. Yes, conducting RCT's is difficult, but not impossible. After all, if we can put astronauts and cosmonauts on the moon, what kind of argument are we dealing with? Reported difficulties are associated for instance with a lack of collaboration from the side of sports authorities, difficulties in recruiting subjects and loss to follow-up of participating sports persons. And of course, we will have to deal with complex issues related to the measurement of what we "need-to-know". But maybe there is a need for alternative approaches when designing and conducting sports injury prevention RCT's.

What should we be doing in the next few years?

If we have a closer look at the major difficulties associated with sports injury prevention RCT's, it appears that many of these problems relate to implementation issues and to the fact that participation in sports is a form of human behavior. And so is implementing preventive measures.

Although these behavioral aspects of preventive trials have been recognized in recent years, behavioral aspects have received little systematic attention when designing preventive sports injury prevention interventions. Perhaps we should learn therefore from RCT's conducted to change human behavior related to detrimental lifestyles, such as physical inactivity.

In the past decade health promotion researchers, also in my group, have applied successfully the Intervention Mapping (IM) protocol when designing behavioral change interventions aimed at increasing levels of physical activity in the population. Basically the IM protocol consists of five steps:

1. Definition of program objectives, based on a thorough analyses of the health problem;
2. Selection of adequate theories and methods to realize behavioural change;
3. Design of the intervention program, as well as the selection, pre-testing, and production of the intervention materials;
4. The development of a plan for the implementation; and
5. Evaluation.

An important feature of the IM protocol is a continuous and consistent dialogue with all stakeholders involved to make sure that the proposed intervention is acceptable and feasible from a practical, implementation standpoint. It guarantees that intervention materials and activities are tailored to relevant characteristics of the target population, as well as to the abilities and opportunities or the program implementers and intermediaries. We have tried to learn from the IM protocol and our experiences in physical (in-)activity RCT's in designing two of our recent sports injury prevention interventions. One RCT concerned the primary prevention of sports injuries in school children aged ten to twelve years and another RCT concerned the secondary prevention of recurrent lateral ankle ligament sprain in adult sports persons.

Both trials proved highly effective. Because the results of these RCT's are either under review or in press, I cannot reveal the results here. I suggest however that you check future issues of the BMJ for the ankle trial results.

So what should we be doing in the next few years?

Well, I believe that we should take into account thoroughly the behavioral aspects of sports injury prevention interventions by applying the IM protocol, or comparable approaches to the design of interventions.

What else should we be doing in the next few years?

Not much has changed since the beginning of my career; a still often heard argument is that participation in sports is a cost driver to society, because sports persons get injured. It is hard to deny that sports persons get injured. However, the cost argument should be countered by conducting integral cost-effectiveness studies comparing active and inactive persons, in which both the direct (medical) costs, as well the

indirect (work absenteeism, as well as work presenteeism) costs are being accounted for. Another type of analysis to do in the years to come is cost-effectiveness analysis alongside preventive RCT's.

This type of analysis will show that well-designed sports injury prevention interventions are highly cost-effective, as compared to care as usual, or to "doing nothing".

Was I eating pudding last Christmas?

Well, to be honest I was not eating pudding. I went skating instead. It had not been freezing hard enough in the Netherlands to skate on natural ice for a long time. Last time was some ten years ago. However, this Christmas it froze for a relatively long time. So we had a time window between December 29 and January 12 to skate on natural ice. This change in weather occurred rather unexpectedly. Because I was spending my time at our summer house on the isle of Texel, I had no skates there and no skating clothes. Consequently I went to buy skates, put on my new skates and my old blue jeans and off I went. A bit rusty, but who cares if there is ice.

I was not the only one who did so. Literally millions of my fellow countrymen did the same. All rusty and most of them on at least ten year old skates, probably with some sort of clothing and protective gear not entirely fit for the purpose.

During this period an estimated 13,000 skaters were seen at an emergency department of a hospital for an ice-skating related injury (8% of them was hospitalised), whereas in other years during the same time period "only" 680 of such injuries were seen, related to skating on artificial ice. 98% of the injuries were due to a fall. The direct medical cost of these fifteen days of hilarious fun were estimated at a total of £15 million, whereas the indirect cost was estimated at £67 million. I have no time to go into other details of the statistics.

You'll need to wait for my presentation in Brisbane in October for the details.

However, the question to ask is: do we need an elaborate IM protocol to set up an effective natural ice skating injury prevention intervention? Or if we can just do with some common sense as well?

I leave it up to you to decide. After all, I assume that you don't have irregular spells of nature-dictated sports madness Down Under, so you will have an unbiased opinion on this matter. However, before you come up with your judgment, may I kindly remind you of Dr. John Snow, M.D., who put an end to the 1854 London cholera outbreak which he had traced to water from a pump in Broad Street. He terminated the epidemic by just simply using his common sense – he took the handle off the pump.

Sometime we only need to act upon what we see and neither theory nor elaborate scientific research are needed to put effective sports injury prevention interventions in place; "the proof of the pudding lies then in the eating".

Professor van Mechelen will be a keynote Speaker at Sports Medicine Australia's annual conference this year "be active '09", to be held at the Brisbane Convention Centre 14–17 October.

Professor van Mechelen will be part of a group of outstanding speakers in the National Sports Injury Prevention component of the conference. Other speakers include:

- Professor Caroline Finch (University of Ballarat – the Refshauge Lecturer – one of the world's leading sports injury epidemiologists)
- Dr Carolyn Emery (University of Calgary, Canada – sports injury prevention in children and adolescents)
- Dr Paul Fleming (Loughborough University, UK – artificial sports surfaces)
- Dr Evert Verhagen (VU University, Holland – sports injury prevention programs).

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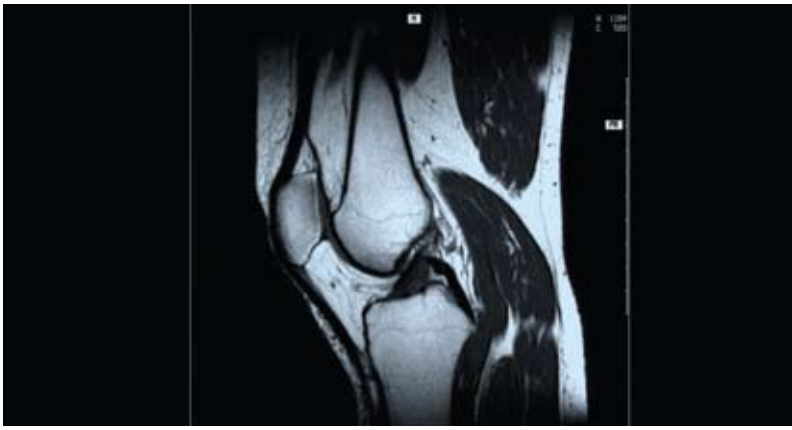
ACL injuries in adolescent athletes



Loretta O'Sullivan

Adolescence is a specific and unique developmental phase which is quite different from either childhood or adulthood. The development, transitions and transformations that occur biologically, cognitively, psychologically and socially, are more rapid and far greater than any other developmental phase, with the exception of infancy. Sports physiotherapists working with adolescent athletes should not only consider the development of the musculoskeletal system and how that impacts on pathology and injury and thus the physiotherapy management of these, but should consider all aspects of development, along with the various environs and contexts of the adolescent athletes. This will enable sports physiotherapists working with active adolescents to deliver the most effective and appropriate treatment plans for adolescent athletes, make a positive contribution to the education and development of the adolescent athlete as a whole, and responsibly and effectively "market" intervention programs for adolescent athletes.

There is a lack of data-driven research specifically addressing the unique needs of the adolescent, including those of the adolescent athlete. There is also a lack of community and healthcare services specifically for adolescents, with most facilities and treatments being extensions of either paediatric or adult services and not specifically designed for adolescents. Most of the good quality, data-driven research relating to adolescent athletes relates to the knee, particularly ACL injuries, thus, this article will focus on ACL injuries in adolescent athletes. It is beyond the scope of this article to discuss all the development, transitions and transformations that are occurring during adolescence, except to say that sports physiotherapists must always be aware of these and the influence they have on the adolescents they treat.

ACL injuries

Neuromuscular aspects

Landing from a jump and the cutting manoeuvre have been identified as the most common mechanisms of injury for ACL ruptures. ACL ruptures occur at four to six times higher rates in girls than boys after puberty; there is no difference in rates pre-puberty. Two high-quality studies investigated these skills in male and female basketballers: one, landing from a jump; and the other, the cutting manoeuvre. The first study compared aspects of the vertical jump in pre-adolescent and adolescent athletes. In this study, boys demonstrated a significant increase in vertical jump height, a significant decrease in landing ground reaction forces, a maintenance of take off ground reaction forces, and a significant decrease in loading rate, post-puberty. The authors concluded that boys appeared to undergo a neuromuscular spurt at puberty. Girls, in contrast, demonstrated no change in vertical jump height, no change in landing ground reaction forces, a significant decrease in take off ground reaction forces on the dominate side only, and a significant decrease in loading rate. Girls ground reaction forces in both landing and take off and the rate of loading was significantly higher than boys. Thus, it was concluded that girls did not undergo a neuromuscular spurt at puberty. Girls demonstrated increased relative landing ground reaction forces and a decreased ability to attenuate landing forces compared with boys, post-puberty.

In the study investigating the cutting manoeuvre, it was found that females had increased knee valgus and ankle eversion angles compared with males. The increased knee valgus angle was significant at initial contact and there was a trend in this direction at maximum. The authors suggested that females may rely more on ligaments, rather than musculature,

to absorb a significant portion of ground reaction forces in cutting than males, and that ligament dominance may be a risk factor in ACL injuries in adolescent female athletes. The authors also concluded that knee valgus angle and ankle eversion angle may be a factor in the gender-related differences in ACL injury rates in adolescent athletes. The findings of this study seem to support those of the first study, in that adolescent girls appear not to have the same neuromuscular control as boys of this age group.

The authors of both these studies suggested that, although anatomical and hormonal factors may contribute to the increased rate of ACL injuries in adolescent female athletes, they are difficult and controversial to modify, and that the greatest opportunity for risk identification, modification and intervention may therefore lie with biomechanical and neuromuscular factors. Both studies advocated the introduction of neuromuscular training in female adolescent athletes, Quatman et al. advocating this training take place at or near the onset of puberty. It would appear that neuromuscular training should involve both jumping/landing skills and the cutting manoeuvre. Sports physiotherapists are in an ideal situation to be involved in the screening and implementation of such programs in adolescent athletes.

Ligament laxity

In two more recent studies, ligament laxity was examined in relation to ACL injury in adolescent athletes. One study investigated the effects of gender and pubertal maturational status on ligament laxity, and the other investigated the effects of generalised joint laxity on ACL injury risk in female adolescent athletes. The first study found that prior to puberty there was no gender difference in respect to ligament laxity, but at the onset of puberty, girls developed greater generalised ligament laxity which was maintained post-puberty, whilst boys did not develop ligament laxity. The authors suggested that this increase in joint ligament laxity in females at puberty may contribute to the increased incidence of knee and ACL injuries in female adolescent athletes. The findings of the second study indicated that increased knee laxity measures may contribute to an increased risk of ACL injuries and found that in female adolescent athletes, a positive measure of knee hyperextension increased the risk of ACL injury fivefold. * suggested that measures of knee joint laxity may be used in conjunction with measures of neuromuscular control of the knee joint to more accurately identify female adolescent athletes at high risk of ACL injury, allowing for targeted intervention programs.

Avulsion fractures

Avulsion fractures are common in adolescence and avulsion fractures about the knee are particularly common. It has been reported that up to 80% of ACL injuries in skeletally immature patients are avulsions of the tibial spine; this is due to the distal fibres of the ACL being stronger than the adjacent bone at this stage of development. Plain radiographs are taken at a variety of angles including tunnel and oblique projections in order to fully appreciate the separation. MRI is used following plain radiographs as it depicts the avulsed bone—presentations are often subtle on plain radiographs, the multiplanar capabilities of the MRI are useful in revealing the exact location and extend of the lesion. MRI can also distinguish bone oedema patterning in impaction from avulsion; the state of the ACL, which is generally intact but displaced superiorly; and other soft tissue and bony injury such as meniscus, other ligament injury or bone marrow oedema. MRI is the gold standard in imaging these injuries, allowing the orthopaedic surgeon to better plan the surgery. ACL avulsion injuries need to be surgical reduced within one week of injury, so it is crucial that all adolescents who sustain an ACL injury have plain radiographs ASAP to establish if an avulsion fracture has occurred, and then to arrange referral for MRI and to an orthopaedic surgeon.

Outcome measures

To date there have been no knee scoring scales specifically designed for adolescents with ACL injury or other knee pathology. In a recent article, two knee scoring scales—Lysholm knee scoring scale and the WOMAC osteoarthritis index—were tested for construct validity and responsiveness in adolescents (12–17 years) and young adults (18–35 years). The Lysholm knee score was designed to determine the functional status of adults following ACL injury and is used extensively in the literature. It has also been found to be highly responsive to patellofemoral pain syndrome and meniscal tears in adults. The WOMAC osteoarthritis index was developed for patients with hip and knee osteoarthritis. The authors found that in adolescents, the Lysholm knee scoring scale demonstrated adequate construct validity and high responsiveness for traumatic and atraumatic knee pain/injury, whereas the WOMAC osteoarthritis index did not show as high responsiveness in adolescents. Thus, while not specifically designed for adolescents, it would appear that the Lysholm knee scoring scale would be highly appropriate for use by sports physiotherapists working with adolescent athletes who have sustained a knee injury, specifically an ACL injury.

Pain/catastrophising

In a study examining differences in pain, catastrophising and effective distress (depression and anxiety) twenty-four hours post-ACL reconstructive surgery between adolescent and adult athletes, it was found that there was a significant difference between the groups for pain, catastrophising and anxiety. Adolescent athletes reported greater pain and catastrophising than adults; however, if controlled for catastrophising, there was no difference between the groups. The aspects of catastrophising that were significant were pain-related helplessness and rumination (dwelling/mulling over). The authors suggested that the relative lack of understanding of injury, lack of experience with recovery and potential threat to competition in adolescent athletes may be a factor in the differences demonstrated in this study. Identifying differences in pain experiences between adolescents and adults may assist sports physiotherapists to better deliver rehabilitation to adolescent athletes post-ACL surgery. Clinically, the results of this study would suggest that "pre-hab" is very important in the adolescent athlete undergoing ACL reconstruction, and that sports physiotherapists should devote a significant portion of this "pre-hab" to education and discussion regarding the injury, rehabilitation, recovery and return to sport.

Furthermore, the ratio of complex regional pain syndrome in children and adolescents in the lower and upper limbs is 8:1, which is in contrast to adults, where CRPS is much more prevalent in the upper limb. There is also a much higher ratio of females with CRPS in children and adolescents than in males (7:1); again this is in contrast to the adult population where the female:male ratio is 2–4:1. Adolescents and children have a very good prognosis compared to adults, who generally have a poor prognosis and it has been shown that early weightbearing seems to be very important in preventing and treating CRPS 2 in children and adolescents. Thus, it is very important, particularly in females, to encourage early weightbearing and functional activity in adolescent athletes who sustain any lower limb injury, including ACL injury—this would form part of the "pre-hab".

Conclusion

ACL injuries are common in adolescent athletes and female adolescent athletes are at a higher risk.
Sports physiotherapists are well placed to identify at risk athletes, implement injury prevention programs, assist with the immediate management of these injuries to help prevent complications of CRPS, implement "pre-hab" programs to prevent post-op complications and enhance recovery, and to implement post-op and long-term rehabilitation programs. Sports physiotherapists working with adolescent athletes need to be aware of the unique and specific injury profile of adolescent athletes, which relates to the musculoskeletal development occurring at this phase. They also need to be aware of the other developments, transitions and transformations that occur biologically, cognitively, psychologically and socially, and how this will impact on the delivery of sports physiotherapy treatments.

Loretta O'Sullivan, BPhty, Post Grad Dip Sports Physio, FACP, is a Specialist Sports Physiotherapist with a sub-specialty in adolescents in sport. She is currently a doctoral candidate in clinical physiotherapy at the University of Melbourne—the focus of her doctorate is adolescents in sport. Her research is on knee cap in teenage girls who play sport. Loretta owns and operates twelve9teen sports physiotherapy, a sports physiotherapy clinic specifically dedicated to active teenagers. Loretta also works in the orthopaedic out-patients clinics at the Mater Children's, Mater Adults and Royal Children's Hospital as a Specialist Sports Physiotherapist for the new adolescent orthopaedic physiotherapy screening clinics. She is also the team sports physiotherapist for Terrace 1st XV rugby team.

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REPAIR, RECOVER & REFUEL.

The Melbourne Vixens netball team represent their home city in the elite Australia and NZ Championship competition. The Melbourne Vixens includes Australia's best female athletes and a new generation of netball stars, with seven Australian squad members in the team, including recent World Champions Julie Prendergast, Bianca Chatfield and two-time Commonwealth Games gold medallist Sharelle McMahon.

Sports Dietitian Kerry Leech speaks with Sharelle McMahon, captain of the Melbourne Vixens Netball team.

Q. What is your favourite food?
I'm a little partial to chocolate but my favourite meal is chicken and vegetable risotto.

Q. Cereal or toast for breakfast?
Definitely a cereal girl, eating muesli, yogurt and milk helps me to keep going through the morning.

Q. Sharelle, you are working with Netball Victoria as well as playing and training with the Vixens - how do you fit it all in?
I'm very busy. I manage it with a very up to date diary!

Q. So how do you manage healthy meals on the run?
I need to be organised and pack food each morning. It makes drinks like Sustagen important as I can have them in the car on the way to or after training.

Q. What flavour Sustagen is your favourite?
That's easy, Chocolate - I told you I am a chocolate girl!

Q. How do you feel Sustagen helps your recovery?
Netball is a hard game, I tend to come out of each game with a few bumps and bruises. Sustagen after each game helps to get the recovery process started and provides a great source of protein and carbohydrate.

Q. So what now for Sharelle McMahon?
The Vixens are finished for the season but the Australian team has international matches over the next few months against New Zealand and England. So plenty of training camps, travel and tough matches. No slowing down for me!

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Lateral Elbow Pain: A Sport and Exercise Physician's Perspective

"Tennis elbow" is a common injury related problem. However, not all lateral elbow pain is caused by playing tennis. Any repetitious stereotyped activity can cause this problem, and indeed the most recalcitrant elbow pain I have seen is in workers boning meat in a freezer works.

Our understanding of the pathophysiology of tennis elbow has changed dramatically in the last twenty years. Previously, it was thought to be an inflammatory condition, hence the term lateral epicondylitis was the preferred term when I graduated from Medical School in 1980. However, in recent years detailed histopathology studies have confirmed that in most cases the condition arises as a result of tendinopathy of the wrist extensors, principally extensor carpi radialis brevis (ECRB). Tendinopathy is best described to patients as a failed healing response.

The causation of tennis elbow problems can be split into intrinsic factors such as muscle weakness, muscle imbalance and lack of flexibility, plus mal-alignment. By contrast, the extrinsic factors include the volume and intensity of any activity plus any change in these, or indeed in the type of activity. Poor technique and inadequate recovery can also play a part. It is also pertinent to remember the kinetic chain and bear in mind that in hitting a tennis backhand shot, for example, the majority of the force generated comes from movement of the legs and trunk and only a small minority from movement of the arm. Poor technique, e.g. hitting a shot late, and equipment issues such as the size of the racquet handle and stringing of the racquet can also play a part.

Less commonly, lateral elbow pain can arise from a direct blow to the area, e.g. on banging the elbow against the side of a door or some other object. A direct blow to the area can cause atypical pathology, e.g. a partial tear or, on occasions, even a fracture of the radial head.

Most patients will report focal symptoms around the lateral epicondyle and just distal to this. The pain is worse with any activity involving wrist extension and occasionally they may report dropping things. This phenomenon occurs because of reflex inhibition of the ECRB and related muscles. In simple terms, the only way the muscle can acutely unload itself is to relax and the hand lets go of whatever it is holding. Bilateral symptoms are atypical and should lead to the clinician to suspect an associated seronegative arthropathy or some other contributor to enthesopathy.

Examination of the lateral elbow may reveal bruising in the acute phase. More commonly, the patient is seen a few weeks down the track and this bruising will have subsided. At that stage there is usually tenderness over the lateral epicondyle and for a few centimetres distal to that site. Usually there is a full range of motion of the elbow, and the elbow will be stable to varus and valgus stress.

The hallmark of ECRB overload is pain on resisted extension of the wrist and middle finger. There may be associated weakness secondary to pain inhibition. If the tenderness is slightly more distal, i.e. about three centimetres down from the lateral epicondyle, then one should be suspicious

of posterior interosseous nerve entrapment. This is less common than ECRB tendinopathy, but should be considered in anybody with lateral elbow pain that is not resolving with standard treatment. An additional clinical clue is pain on resisted supination. Occasionally there will be referred pain from the neck, so an examination of the neck plus assessment of any associated neural tension signs is worthwhile. Finally, one should not overlook the possibility of secondary regional pain syndrome. Look in particular for vasomotor changes in the hand on the affected side.

In terms of investigations, usually none are required. If there has been a direct blow to the area then I would recommend an X-ray with particular emphasis on obtaining additional views of the radial head to exclude a fracture at that site. Ultrasound scan is not necessary to make a diagnosis of tendinopathy of the ECRB. If, however, there has been a direct blow then the chance of an associated partial tear of the ECRB tendon is increased.

Over the years I have been operating as a referral only practitioner, I have performed a significant number of injections and my routine is to organise an ultrasound scan in those people I inject. This at least gives me an idea of the size of any tear they may have prior to considering an injection. A small tear is not a contraindication to injection but needs to be considered as part of the overall clinical picture when deciding on the optimal treatment.

Management of any overuse tendinopathy is along standard lines. Firstly, one should use ice massage and rest the area for a few days to allow the effects of the acute injury to settle. Then a progressive stretching and strengthening programme can be undertaken. Ideally, this should be a progressive concentric then eccentric strengthening regime. Experience from the achilles tendon, when extrapolated to the elbow, would suggest that the emphasis should be on eccentric loading and the aim is to build up to around ninety repetitions per day. I advise patients to get a 500g tin of canned food and put this in a sock and rest their forearm across a table. They can then perform repeated wrist extensions using gravity as resistance. The weight can be varied up or down depending on their degree of symptoms. A tennis elbow band is useful to reduce load through the injured area.

The above plan works well for about 90% of those affected by lateral elbow pain. It can be expected to provide substantial resolution of symptoms and functional improvement over a period of eight to twelve weeks.

What to do for the remaining 10% of sufferers? These people deserve consideration of adjunctive therapy. Traditionally this has involved an injection of corticosteroid. However, more recent evidence based treatment has included autologous blood injection or the use of GTN patches or iontophoresis of cortisone.

All of these adjunctive treatments have their advocates. The most important thing to stress is that they are not a substitute for the concentric then eccentric regime outlined above. When injections and other treatments become known to those in the community, there are always a few who will present to their doctor and demand such a treatment without going through the necessary concentric then eccentric rehabilitation. To inject such people without reminding them that they need to go through with a programme of progressive strengthening is to potentially court disaster. Such patients, I believe, need to experience the pain that often accompanies the first couple of weeks of an eccentric programme and work their way through that so they can see that the benefits will ultimately accrue. If they do not even embark on such a programme then one can pretty well guarantee that once the acute effect of their injection wears off, they will be back demanding another one and not be committed to any reasonable strengthening regime. Rather, I would suggest that they embark on the rehabilitation and be warned of the fact that symptoms can be temporarily exacerbated for the first couple of weeks. The aim in such people is to progressively build them up to the ninety repetitions per day.

There are still a few who struggle and those people could be offered an injection on the understanding that it is merely a means of trying to settle their acute symptoms to enable them to then pursue a full concentric then eccentric programme in earnest. It is not an excuse to cut loose and be lazy.

Surgery is rarely required for this condition. In the rare cases where the above treatments have been tried and been unsuccessful then release of the ECRB tendon can be performed. The presence of secondary complex regional pain syndrome is a strong relative contraindication to surgical treatment.

Finally, if things are not improving in the expected timeframe one should question the diagnosis. Most people have uncomplicated tendinopathy but the differential diagnoses include the following:

1. A partial tear of the ECRB tendon which is more likely if there has been a direct blow to the area or a high energy injury.

2. Entrapment of the posterior interosseous nerve which is usually manifest by pain and tenderness a few centimetres distal to the typical site. Classically these patients are worse when they apply a tennis elbow band, as this band is applying extra pressure over the area of entrapment of the nerve.
3. Referred pain from the cervical spine should not be overlooked. If there is an associated neck injury with radiculopathy then typically C7 will be the nerve root most commonly associated with lateral elbow pain. One should check for reduced power of elbow extension and subtle differences can be picked up once experience is gained in performing this evaluation.
4. A fracture of the radial head should not be missed. This is frequently only a subtle finding on routine X-rays, and views to profile the radial head increase the fracture detection rate. Management should be in association with an experienced orthopaedic surgeon. Resolution of the block to extension can take many months.
5. In a javelin thrower one should have a high index of suspicion for osteochondral injury and early onset posttraumatic arthritis. This is virtually inevitable in all high level javelin throwers who have been training and competing for over ten years. Put simply, the elbow was not designed to tolerate such load on a repeated basis. Rather, it is designed for fine movement and to place the hand in as many positions as possible.

The above article is by no means comprehensive but should give clinicians a few pointers as to what to look for if you see a person with lateral elbow pain that is not responding as one would ordinarily expect. If in doubt, refer to an experienced colleague rather than reaching for the referral pad and simply ordering more expensive imaging.

In summary

1. Most lateral elbow pain relates to tendinopathy of the ECRB tendon.
2. The vast majority of cases will respond to a concentric then eccentric strengthening programme.
3. A few will require adjunctive therapy.
4. Imaging should be used selectively.

Chris Milne
Sport and Exercise Physician, Hamilton, New Zealand
Immediate Past Present, Australasian College of Sports Physicians

Letter to the Editor

3 March 2009

Thank you for your informative journal.

I write in response to the article by Dr Paoloni concerning the appalling state of affairs with respect the MBS cover of injection therapies.

Further to the concerns raised by the fact that if there is not a joint or bursa at the end of the needle, there is no medicare rebate available for this procedure. There is a much broader issue with respect to sports medicine and item numbers. Currently, almost no procedural work that is done as part of normal sports medicine practice is rebateable. This adds a considerable cost to the sporting community or erodes the profitability of the sports doctor.

It is difficult to work out why there is no item number for measuring muscle compartment pressures whilst there are item numbers for measuring pressures in arteries, central veins and intracranially.

There is no item number for the use of Dexamethasone iontophoresis.

In a more general sense, there is no item number for ultrasound guidance of a joint or bursal injection other than the injection fee. Paradoxically, there is an item number to do the same injection using a CT scan – a far more expensive item.

Whilst there are many paradoxes in the MBS, this will not change unless there is lobbying to have this changed. The difference between surgical item numbers and non-surgical item numbers is appallingly large. It appears that physicianly activity is brought down to the lowest common denominator and surgical activity is infinitely broad and seemingly growing with every new procedure.

A last word of caution. It would be very dangerous to think that the introduction of procedural item numbers, as recommended by Dr Paoloni, should be restricted to sports physicians. It is this very elitist concept that already makes the MBS a dogs breakfast. To consider that a doctor other than a sports physician is in anyway less skilled in performing the type of procedures that he outlines is dangerously incorrect. Joint and soft tissue injections should be rebated through the MBS at the same rate for all doctors who are trained and skilled in doing them.

Yours sincerely
A/Prof Gavan White

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