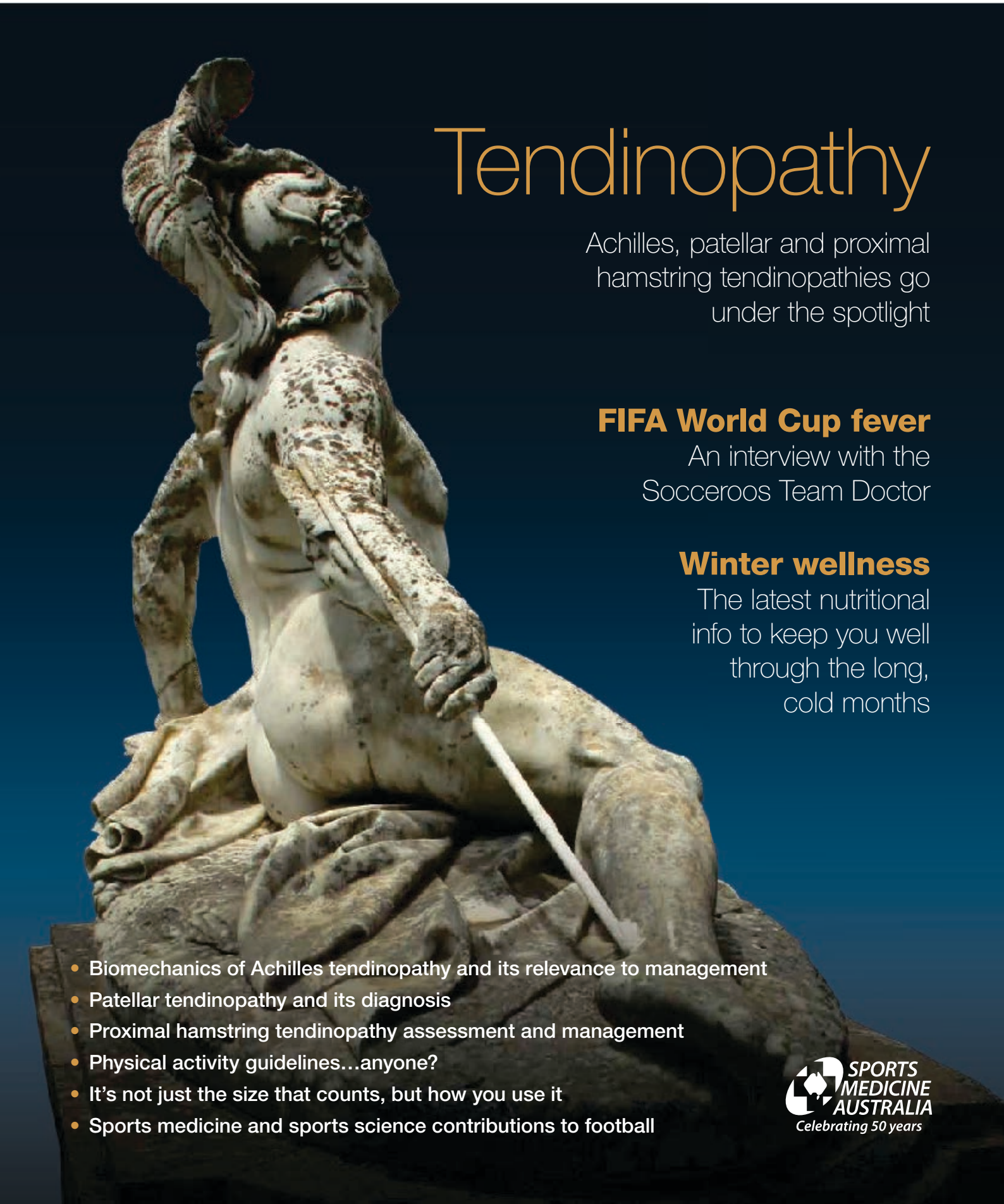
sport health

VOLUME 32 • ISSUE 1, 2014



Tendinopathy

Achilles, patellar and proximal hamstring tendinopathies go under the spotlight

FIFA World Cup fever

An interview with the Socceroos Team Doctor

Winter wellness

The latest nutritional info to keep you well through the long, cold months

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- Patellar tendinopathy and its diagnosis
- Proximal hamstring tendinopathy assessment and management
- Physical activity guidelines…anyone?
- It's not just the size that counts, but how you use it
- Sports medicine and sports science contributions to football

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Celebrating 50 years

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Physical Activity Guidelines… Anyone?

SMA CEO Nello Marino visited The Athlete's Foot recently to test out their brand new and exclusive fitting technology FITZI. FITZI gives an assessment of a person's foot type and shoe requirements to make sure their shoe fit is spot on for their foot type, sport and position, meaning greater performance, support, comfort and reduced risk of injury. If you have a worthy cause or issue related to sports medicine or physical activity that you would like promoted in Sport Health via a promotional item email nello.marino@sma.org.au

SMA CEO Nello Marino discusses the implications of the recently released national Physical Activity Guidelines and highlights SMA's first "Twitter chat".

According to the World Health Organisation, physical activity levels of many people, both in Australia and around the world, are less than the optimal level recommended to gain a health benefit. Without going over a very well-trodden path, the health benefits of physical activity are very clear and the evidence continues to mount.

What does baffle me is why the Australian Physical Activity Guidelines which were recently released, hardly caused a blimp on the Australian news radar.

These guidelines were released in February following a lengthy review of existing evidence and a number of comparative international guidelines. The process of review brought together numerous eminent physical activity and sedentary behaviour researchers and practitioners from Australia and abroad. Physical activity researchers such as Wendy Brown, Fiona Bull, Nicola Burton, Tony Okely, Jo Salmon and Adrian Bauman, not to mention the many others involved, did an exceptional job in leading the development of the numerous sets of guidelines.

However, what sort of splash have the guidelines made since their announcement after several years of development? Arguably not much.

This is concerning on a number of fronts, particularly given the guidelines are such great pieces of work and from a health practitioner perspective, there are some very simple and pertinent messages that we need to be conveying to all of our clients.

The new guidelines most definitely build on previous physical activity guidelines, and align Australia's recommendations to numerous similar statements internationally. Some general highlights of the new guidelines include:

- A change in the weekly recommended aggregate of physical activity required to maintain health from a flat 150 minutes per week to between 150 to 300 minutes per week. Clearly an endeavour to encourage us to strive for more than just the minimum and an acknowledgment of "a little more is better still". Mind you I still don't believe this is too arduous when you consider the recommendation is one hour, including incidental activity, at least 5 times per week.
- The recommendation to limit sitting time and to limit screen time, particularly for children and adolescents. Again supported by the evidence that the amount of sitting time has a negative effect on health and has the potential to undo the efforts acquired through being active.
- The inclusion of muscle strengthening exercise in every week.

I'm greatly concerned that many practitioners aren't even aware that the guidelines were released which would suggest one of a number of factors including poor promotion, lack of interest and receptivity at a community level or

most concerning, a diminished commitment to the promotion of physical activity by our federal government. I dearly hope it isn't the latter.

Ironically for SMA it has proven to be one of the more popular social media stories we've experienced to date, suggesting that those with enough interest to follow an organisation such as SMA in social media are committed to ensuring the guidelines are promoted.

Given our current diminishing levels of physical activity and our rising levels of obesity and other lifestyle diseases, I sincerely hope our community starts to take up the valuable messages contained in these guidelines and that a little more effort is made in the future to ensure there is awareness of the guidelines in order to give them some chance to be adopted effectively.

Welcome SPNZ

I'd like to extend a warm welcome to members of Sports Physiotherapy New Zealand (SPNZ) who have recently taken up subscription of *Sport Health* and will provide regular content to *Sport Health*. *Sport Health* now forms part of the great range of member benefits available to SPNZ members. We look forward to many continued contributions from the SPNZ executive and members and we look forward to greater clinical exchange between SMA and SPNZ members. More information on SPNZ can be found at sportsphysiotherapy.org.nz.

Twitter Chat

On the suggestion of some of my great (Gen Y) staff SMA hosted its first "Twitter Chat". This comes as part of our strategy to keep trialling new platforms on which to spread the word about sports medicine, science and injury prevention. For those unfamiliar with "Twitter Chats", it's effectively a staged forum of tweeters responding to a series of questions and comments. Not dissimilar to conducting a face to face forum, except for the fact that you are responding online and within the confines of 140 characters. The topic of sports injury prevention was the theme of the first chat which was conducted was over a strict one hour timeslot. Participants from around the globe got involved and for a first up effort it generated over 150 exchanges in response to five posed questions posed by the moderator (@SMACEO).

Thanks to Sheree Bekker, one of the "Twitter" chat participants from Federation University, some of the tweets from the "Twitter" chat (those that used #sportsinjprev) have been recorded via Storify. This can be viewed at http://sfy.co/hdq4.

Nello Marino

Chief Executive Officer
Sports Medicine Australia
nello.marino@sma.org.au

Follow SMA CEO Nello Marino on Twitter @SMACEO

Is this the highest concentration of injury prevention researchers and practitioners ever seen in one place? #IOCprev2014 – April 10, 2014

Sports Medicine Australia calls for ban on lift tackle @SMACEO @doclarkins http://sma.org.au/2014/04/call-to-ban-lift-tackle/ – April 1, 2014

Get in on the AFL action via this exclusive Sport Health extract & go behind the scenes w a @WestCoastEagles physio http://bit.ly/1fxccBf – March 13, 2014

SMA now has a dedicated media twitter account! Jump on @sma_news for the latest media releases, research news and updates – March 7, 2014

The national sports injury prevention conference is part of BeActive October, Canberra. An opportunity for more F2F chatting #sportsinjprev – March 6, 2014

And that's a wrap for our first ever twitter chat based on Sports Injury Prevention! It's been a great success!! #sportsinjprev – March 6, 2014

Member news

Sport Health correction

Within the contents section of the previous edition (Volume 31, Issue 4, 2013/14), *Sport Health* incorrectly listed the author of "Behind the scenes with a sports physiotherapist" as "Kate Spagnolo" as a result of an administrative error. The correct spelling of the author's name is Kane Spagnolo. *Sport Health* would like to apologise to Kane for the error and any confusion that may have occurred as a result of the error.

Social media channels

Stay engaged with the latest happenings of Sports Medicine Australia via the following social media channels.

Twitter

Follow:

- @SMACEO for news, events and member updates from SMA CEO Nello Marino
- @sma_news for SMA media updates, the latest industry news and research developments
- @beactive2014 for all your conference info and news
- @_JSAMS to keep up to date with the latest from the Journal of Science and Medicine in Sport
- @SMAChairman for updates from SMA Chairman Peter Nathan

Facebook

Search: Sports Medicine Australia or go to www.facebook.com/sportsmedicineaustralia

LinkedIn

Search: Sports Medicine Australia

Make the most of your membership

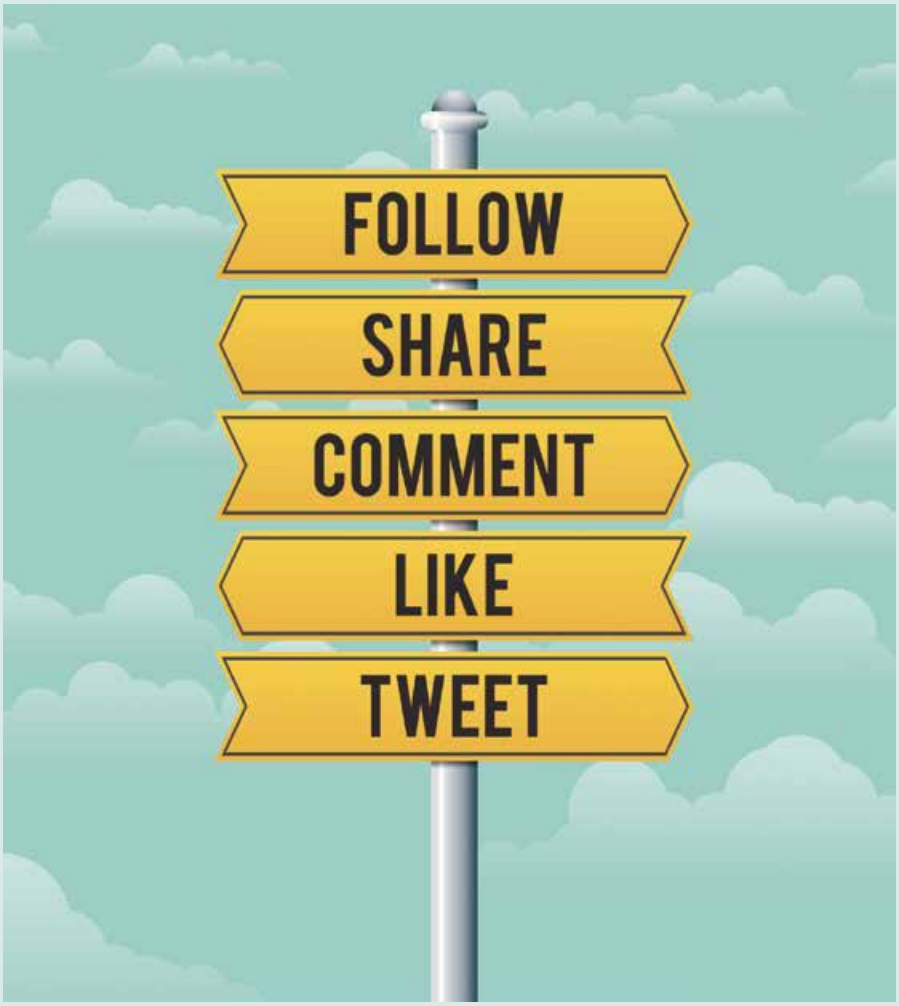
If you haven't checked out the SMA Member Portal for a while you're not taking full advantage of all your SMA membership has to offer. With webinars and the latest issues of the *British Journal of Sports Medicine* and the *Journal of Sports Medicine and Science* just one click away, the Member Portal is also a great way to network and locate an expert or specialist. Jump on the Member Portal via sma.org.au and make the most of your SMA membership.

Journal of Science and Medicine in Sport

The May edition of JSAMS has a number of fascinating studies – make sure you check out the following free access articles by visiting Editor's Choice at http://jsams.org

Bock et al. Community-based efforts to promote physical activity: A systematic review of interventions considering mode of delivery, study quality and population subgroups
May 2014 (Volume 17 | Issue 3)

Silva NL, et al. Influence of strength training variables on strength gains in adults over 55 years-old: A meta-analysis of dose-response relationships
May 2014 (Volume 17 | Issue 3)



be active 2014

Canberra, October 15–18 2014

Conference Registration for be active 2014 is now open

The conference, incorporating the Australian Conference of Science and Medicine in Sport (ACSMS), the National Physical Activity Conference (NPAC), and the National Sports Injury Prevention Conference (NSIPC), will focus on current research and practice in areas relating to the promotion of, and safe participation in, all kinds of sport, exercise and physical activity. be active 2014 will provide an interactive educational forum of the highest standard, promoting the latest physical activity and health developments amongst key and influential industry professionals.

Currently, our keynote speakers for be active 2014 include:

- Professor Danny Green (Refshauge Lecturer)
- Professor Stuart Biddle
- Dr Darren Burgess
- Dr Tim Gabbett
- Professor Paul Hodges
- Dr Jill McNitt-Gray
- Dr Andrew McIntosh
- Professor Neville Owen

be active 2014 will also offer a jam packed social program to maximise your networking opportunities all whilst enjoying what our nation's capital has to offer. Events include:

- **Welcome Reception** (one ticket is included with each full registration).
- **ASMF Fellows Dinner.**
- **Scientific Poster Session** where senior members of SMA will provide feedback to individual researchers and discuss their research (one ticket is included with each full registration).
- **Discipline Group Dinners.**
- **Conference Dinner** which is the premier social event of be active 2014 and includes local entertainment, dancing and fine food (one ticket is included with each full registration).

Stay tuned for the preliminary program which is due out in June.

Check out what else is on offer at sma.org.au/conferences-events/conference. Remember early bird conference registration closes 31st July, 2014.

Alice Springs Masters Games: Volunteer and receive a FREE SMA Sports Trainer Membership

11–18 October 2014: SMA-NT will be providing the medical clinic to support the athletes from across Australia and overseas during the Masters Games. Sports Trainers are encouraged to be part of the SMA team, with all volunteers receiving a FREE SMA Sports Trainer Membership. Email neilia.ginnane@sma.org.au to register.

Mentor Program

The SMA Mentorship Program has expanded nationally in 2014, after the success of the pilot program run in Victoria last year. We have a total of 36 mentors and mentees from across the country (even one based in China!) catching up regularly with the mentees gaining the benefits of the wealth of knowledge and expertise provided by their mentors. They will continue the program until October/November this year.

5 minutes with… Kane Spagnolo

Melbourne based West Coast Eagles Physiotherapist and Lakeside Sports Medicine Centre Physiotherapist

What originally attracted you to physiotherapy?

Being the younger athlete I more often than not found myself in and out of the physio throughout the season and more often than not I always came away feeling much better for the visit. Over time I was under the illusion that being a physio seemed to be such a relaxed and stress free job (swing and a miss on that one!) but it also prompted a fascination in me as to understand how the body adapts and responds to various treatment and rehab prescriptions and I guess this is where my interest peaked in physiotherapy.

Learning more about the profession from my physio at the time and the challenge with keeping up and pushing the barriers with learning and injury management particularly from a sports perspective definitely appealed to me and I've thoroughly enjoyed the learning since.

Sport Health readers have already being treated to an insight into what a typical day as a West Coast Eagles physio consists of in our last edition, what does a typical day as a Lakeside Sports Medicine Centre physio entail?

It's a great little clinic located inside Melbourne Sports and Aquatic Centre (MSAC) and as such we see a lot of sporting injuries. Due to the different sports that are performed in and around the Centre we tend to get many different athletes from various backgrounds and this keeps the caseload quite dynamic. Our doctor, Dan Bates, is also doing a lot of really interesting work with management of OA pain so I feel that gives a great variety to my day which I think has really enhanced my learning and experience.

What is the favourite aspect of your job?

Undoubtedly the people whom I've been lucky enough to have studied with and worked with in private practice and some great people whom I highly respect out at West Coast. I still keep in contact with those friends who I studied with in Perth and have been lucky enough to work with some really great people here in Melbourne and formed some lifelong friendships and for that I feel really lucky.

What has been the highlight of your career so far?

I don't know if there has been any clear standout highlight per se. I really feel fortunate to have worked with some great people here in Melbourne as well as experienced some amazing moments as part of the support staff at West Coast over the years. Hopefully the rest of my career is just as satisfying as it has been thus far.

Where do you see yourself in 5 years?

I hope to be still involved in sports physiotherapy as I really enjoy that aspect of my work but privately hopefully I'll be settled into a nice family lifestyle and managed to do a bit more travelling in the next few years as well.

Outside of your role as a physiotherapist, what else are you passionate about?

I like to get out and try different restaurants and with Melbourne we're definitely spoiled for choice. I never drank coffee before moving this way but I like to go check out new places all the time so that has definitely become a new passion of mine in recent years.

When I get home back to Perth I like to get away to the beach and soak up all things coastal. If I get a chance to get down south and have a bit of a fish with some mates that usually involves a bit of banter which is always great for a laugh. I live close to the bay here so I'm usually in and around the "beach" a lot as well, whether it be on the paddle board, running or trying to tire out my overly energetic dog.

What's the best piece of advice you've ever received?

Probably from my Dad, which was to do what you are passionate about and it will feel like you've never worked a day in your life.

Name four people, living or not, you would invite to a dinner party and why?

- Michael Jordan – a bit of a no brainer but for me he's the greatest athlete I've ever seen and I imagine he would have a few interesting stories to tell from both on and off the court.
- Dave Chapelle – I was lucky enough to see him perform earlier this year in Melbourne. He's a controversial American comedian and if you haven't seen any of his work then you must check it out, hilarious on a different level.
- Ellen Hoog – she's a Dutch international hockey player and apart from being a gun at her sport I think she'd bring a certain level of beauty to otherwise what might be considered a pretty ordinary table. Also guaranteed to have the ability to silence a few big names there when she speaks.
- Mark Finicuane – Fini is one of the physios out at West Coast and apart from being a ripping bloke, who would bring a level of intelligence to the table second to none, he's the only guy I know who has featured on the back of a cereal box from his Iron Man days – an honour that only he and the illustrious Michael Jordan can claim to share – and I think that would make a great icebreaker at the table.

Favourites

Travel destination: I have a trip booked to Peru and Bolivia later this year so I'm pre-empting that might crawl up my favourite list pretty quickly but Cable Beach in Broome, WA would be up there.

Sport to play/watch: Love to still play basketball and get out and have a bit of a run, but when it comes to watching sport I still think a high quality AFL game is hard to beat.

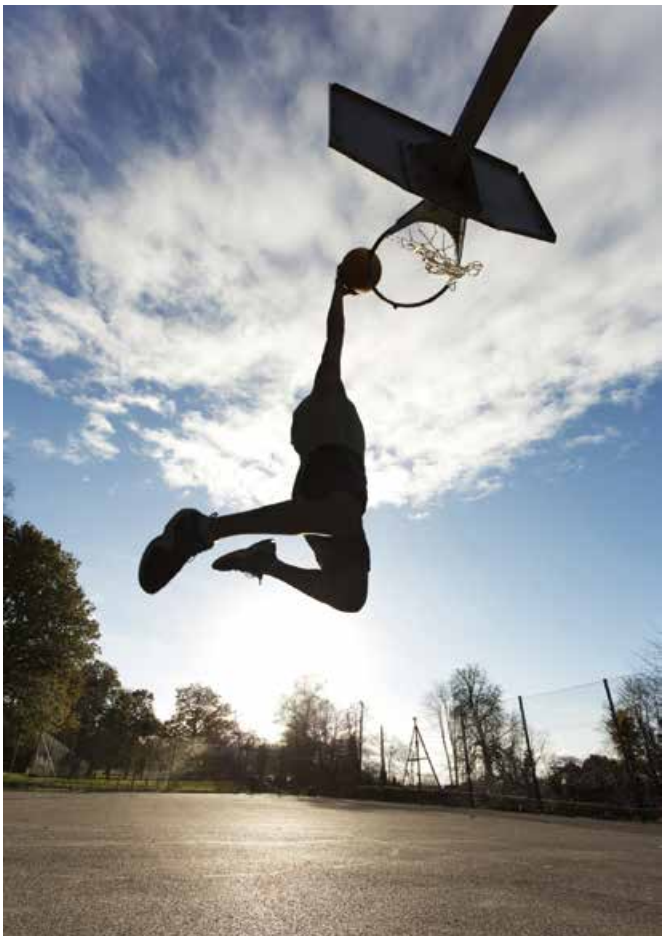
Cuisine: I tend to eat out a lot, but my favourite meal is all things breakfast and Melbourne has a lot of great choices. My favourite though is Dukes in Windsor. When it comes to dinner there is a ripping restaurant on Flinders Lane in the city called Chin Chin that does some pretty amazing food and is always a must for when I have interstate visitors.

Movie: I'm drawing a bit of a blank at the moment but I'll say anything sport/war related which is probably a bit clichéd but hey.

Band/song: Pretty hard to choose as I tend to go through phases with this but both British India and Boy and Bear are getting a bit of play at the moment.

Book: I'm not a big reader as study tends to chew up a lot of this time but I found the Ben Cousins autobiography intriguing, especially since we grew up in the same part of Perth.

Gadget: My MacBook Air is pretty much my most used electronic device. I'm currently studying two post graduate Masters courses (Sports Physio and Strength & Conditioning) so I rely on it pretty heavily and knock on wood it hasn't given me any grief.



Biomechanics of Achilles tendinopathy and its relevance to management

Figure 1: The statue of the warrior Achilles in Hyde Park, London, seen from behind to visualise the tendon or "Achilles' heel".

Dr John Orchard examines the biomechanics of Achilles tendinopathy in relation to management.

The Achilles tendon is named after the Greek legend of the warrior Achilles (Figure 1) who was famed to have only one weak spot in his entire body, after his mother tried to make him immortal by dipping him into the river Styx shortly after birth.

"…the therapist and patient need to work together towards the Blackjack hand of 21 without busting, or to cook the soup that is 'not too hot, not too cold but just right'."

By legend, the only vulnerable part of his body remained the Achilles tendon by which she held him, and where he was later killed after being struck there by an arrow. Ironically direct contact injuries to the Achilles tendon in humans are quite rare. Most Achilles injuries – whether ruptures or tendinopathies – occur due to the intrinsic load of accelerating the weight of the body. And, when comparing real-world patients to the warrior Achilles (who was immune from injury elsewhere), clinicians are painfully aware that whatever pathologies cause Achilles pain are also able to affect tendons throughout the body.

"So just when we thought that diagnosis of Achilles pain was easy (and it was only management which was complicated) we may be about to enter a new era where Achilles pain needs to be subcategorised into different types, which may all respond to different management."

In clinical physician practice – and this article is written from a clinical rather than scientific perspective – I like to differentiate between diagnostic problems and management problems. In fact, the ability to work-up diagnostic challenges is a hallmark of physician ("medical") practice versus surgical practice (where the focus tends to be "can I help this patient with an operation (management)"?) and para-medical practice ("can I help this patient with conservative management, e.g. exercises/stretching/massage/manipulation/orthotics etc.?"). As a general rule for the musculoskeletal world, we more often encounter diagnostic challenges in the central parts of the body (e.g. back pain/groin pain/shoulder pain) and more often encounter pure management challenges with peripheral parts of the body (e.g. plantar fasciitis, tennis elbow, knee medial ligament tear). For the areas where diagnosis is difficult, such as back pain, it actually makes sense to most-often start management with non-specific non-invasive management and advice, rather than obsess about diagnosis[1]. But when pain has persisted and failed first line conservative management, diagnostic workup becomes justified.

Achilles tendinopathy is traditionally meant to be completely a "management" problem, which is another way of saying that diagnosis is straightforward. Pain arising from the Achilles tendon usually localises very well and the patient can point to the painful part of the heel with a finger, differentiating plantar fascia heel pain from Achilles tendon heel pain (Figure 2). A rupture can be distinguished from a tendinopathy on history and with a prone calf squeeze test. The nearby diagnosis of ankle posterior impingement can also be distinguished by less localised pain and with a forced plantar flexion test.

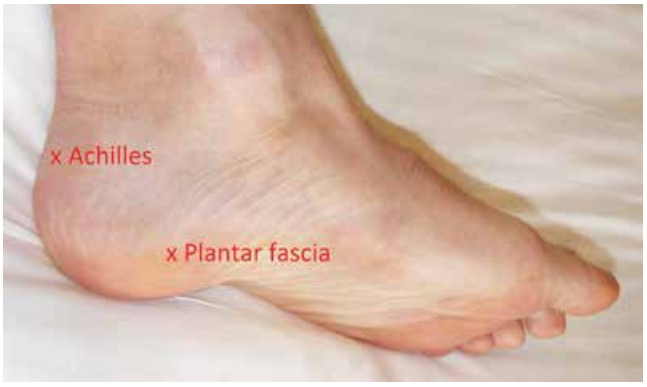


Figure 2: Location of Achilles pain usually makes the superficial diagnosis easy.

So Achilles diagnosis is easy and management is difficult: can we move on to management now? Not so fast, as there is one elephant in the room left to deal with. If, Mr Einstein, Achilles diagnosis is so straightforward, why all the fuss about the change in terminology from Achilles tendinitis to Achilles tendinopathy? Good question, concerning a small matter of all of us clever PhD and Professor geeks not actually being able to tell you with any certainty why Achilles tendons get painful! Except that we're pretty certain it isn't just inflammation.

Or that your mother dipped you into the River Styx at birth holding you by the heel. So if you could work out why Achilles tendons get painful, you might have a better chance of working out how to successfully manage them.

"So what lessons are there for treating a painful Achilles tendon from all of this discussion about mechanics? The number one lesson is to understand that the Achilles (like all other tendons in the body) is in a constant struggle to work out whether to become thicker and stronger or not."

If we want to try to understand how the Achilles tendon stuffs up, it helps to start with understanding how it works when it isn't stuffing up. We know that Achilles tendons make the calf muscle-tendon unit biomechanically much more efficient, particularly when bounding at constant speed, like the way kangaroos and wallabies move[2] (Figure 3). So, Achilles tendon, you have one job, which is to be a useful spring! We'll keep things simple, when the calf muscle stretches you out, recoil back to your resting length for us. One job, one instruction. OK, two more instructions, don't snap when

Figure 3: A Wallaby with view of the right Achilles tendon.

you're getting stretched and don't complain about it hurting. There are a few hundred tendons in the body and we can't work as a team if every one of you complains every time you get stretched. So please Achilles tendon, just recoil as quickly as you possibly can without snapping or complaining (if possible), as you don't want to leave all of the work accelerating the body to the poor old calf muscle.

So Achilles mechanics is pretty simple if you're the body, but it's quite difficult if you're the tendon. Imagine being a rubber band and being told to recoil as quickly as possible but without snapping. That's like telling a Blackjack player to get as close to 21 as possible without going over it. Which is why playing Blackjack is easy and difficult at the same time. It is easy describing the rules and strategy of Blackjack, but hard knowing whether to sit or draw. The Achilles (and all tendons of the body) spend their entire lives going through this same dilemma. The closer you get to failure, the more spring/recoil you give. Add a few more collagen fibres and you'll insure against rupture, but you won't recoil quite as quickly. So the Achilles tendon keeps looking around for signs of damage and if there are signs the tendon is under stress, lay down a few more fibres to protect against failure. However, if there isn't much evidence of tendon stress, then don't worry about replacing fibres, as it appears they aren't currently needed (and would possibly just make the tendon less elastic).

"The Achilles tendon is named after the Greek legend of the warrior Achilles who was famed to have only one weak spot in his entire body, after his mother tried to make him immortal by dipping him into the river Styx shortly after birth."

There are a few good anecdotes that, in combination, illustrate the dilemmas associated with being a good Achilles tendon. The most famous of these in sports medicine circles is the Hakan Alfredson (personal) Achilles story. Hakan Alfredson is the famous Swedish Achilles surgeon and we'll call him Alfredson when talking about him professionally and Hakan for this story, when we are referring to Hakan the recreational jogger in the early 1990s. Hakan hasn't been able to run without Achilles pain for ages and nothing he has tried to relieve the pain has worked. As Alfredson the surgeon, he has cured many cases of chronic Achilles pain with surgery and he's also noted that most patients with chronic Achilles

pain who happen to rupture their tendon seem to be cured of the chronic tendon pain once they have (eventually) recovered from a surgical repair. So Hakan the runner wants Achilles surgery, but he lives in Sweden and must go on a waiting list to have it, as all the hospitals are public and not even surgeons themselves are allowed to jump the queue. The only exception is if you have a ruptured tendon, this qualifies as an emergency case and you get the surgery straight away. So Hakan tries to jump the waiting list by rupturing his own Achilles tendon and he tries to do this by doing repeated heel drops over the edge of a step on the injured side. The exercises cause him a lot of pain, but don't rupture the tendon. In this case what doesn't kill you makes you stronger, and within a few weeks Hakan's Achilles pain is gone. Not content with curing himself, Alfredson tries to cure the world and publishes a moderate quality study (Fifteen recreational joggers in their early 40s compared to similar historical control group with no randomisation or blinding) which becomes of major historical importance[3], crediting previous authors who described successful treatment of tendinopathy with eccentric exercise[4].

The next story is not nearly as interesting, being about me as a runner, but it helps complete the jigsaw puzzle of what the Achilles actually does. For consistency with the Hakan references when talking about myself as a runner I'll call myself John. John has only run one marathon, 23 years ago in the tough days before they had invented gel shots, and is not interested in ever doing another. He can remember being in lots of pain, everywhere in the legs during the event, and everywhere in the lower limb muscles for days after the run. However John distinctly remembers both Achilles tendons swelling up, developing crepitus on movement and getting very sore starting from about a week after the race and lasting about a week. It is an n=1 study but a good learning tool. The overload (running a Marathon), followed by everyday living activities only, led to transient Achilles tendon pathology after a delay in time (1 week), rather than immediately.

"Is there a role for interventional management in Achilles tendinopathy? Of course there is, but the lesson that the patient must 'get' is that interventional management cannot be used as a substitute to getting the underlying load management decisions correct."

What else does a propelling a body as far as a Marathon distance do to an Achilles tendon? Well, extrapolating from Tim Noakes Lore of Running[5], it probably makes Achilles tendons reform themselves so they are more resistant to stretch. This makes Achilles tendons stronger (more likely to withstand future injury) but slower (giving slightly less recoil on each landing). Noakes has sustained for many years that the best way to win a major Marathon event is to be an excellent distance runner who hasn't done many Marathons[5]. Obviously you need to have excellent aerobic capacity and biomechanical efficiency of your running to finish a marathon in world class time, which is stating the obvious, so an elite marathon runner should have elite 5 km and 10 km times. But the surprising observation from Noakes – which is borne out by the history of the Marathon event – is that if you keep running these events you start to slightly slow down in them. Therefore the Marathon is an event where the world King and Queen are usually rapidly changing. As soon as you run an event fast enough to win an Olympic Marathon Gold or set a new World record, for example, you soon find that no matter how hard you train you have probably lost 1–2% of your pace. This probably relates to changes in all of the major tendons of the lower limb, but particularly the biggest of them all, which is the Achilles.

"Achilles tendinopathy is traditionally meant to be completely a "management" problem, which is another way of saying that diagnosis is straightforward."

The stress of breaking the world record or winning an Olympic Gold in the 100 m doesn't have the same impact on the Achilles tendon, allowing someone like Usain Bolt to stay as the top ranked sprinter for longer. However if you

Figure 4: An Achilles surgeonfish (Tang), not to be confused with Achilles surgeons like Hakan Alfredson and Nic Maffulli.

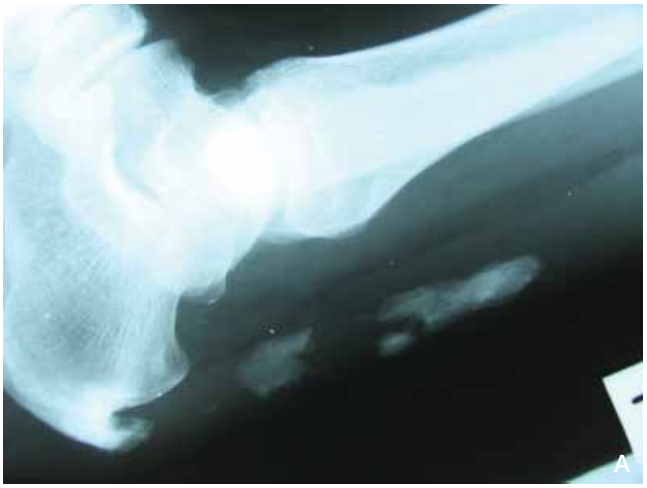


Figure 5A: Achilles ossification (an extreme case both affecting insertion and mid-tendon).

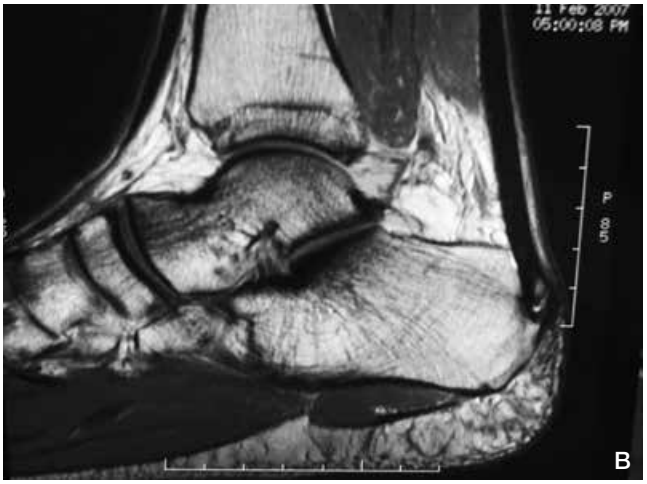


Figure 5B: Insertional calcification/ossification can be demonstrated on MRI as well, but no better than X-ray or US.

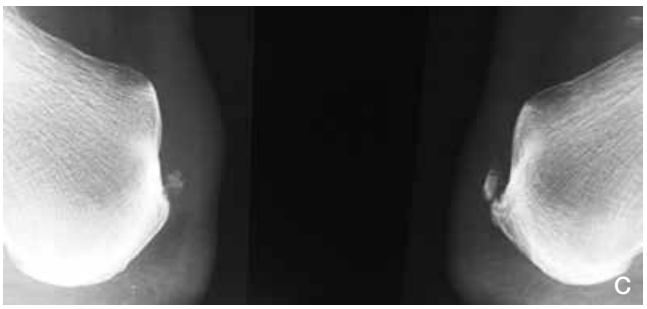


Figure 5C: This X-ray pair shows a more established (attached) spur on one side (probably ossification) and possibly more recent calcification within the tendon on the other.

rupture the Achilles tendon and recover from the surgical repair, as a 100 m sprinter you never regain the same pace (it stays approximately 2–3% down for good). Donovan Bailey encountered this after winning the 100 m Gold in Atlanta then rupturing his Achilles (playing social basketball) prior to the next Olympics. In Sydney he was a beaten semi-finalist. Football players can recover from Achilles ruptures and still be very good players, because of the skill element involved, but they almost never can regain blistering pace if they once had it. The surgically-repaired Achilles tendon is actually stronger in one sense than it was before (which is one of the few times that surgeons say this accurately) but it is thicker and stiffer, so it gives less recoil and spring, and contributes less to propulsion. Hakan Alfredson didn't care about this when he was trying to rupture his own tendon – he just wanted the end of his own Achilles pain. But for the elite athlete with Achilles tendinopathy it is a matter of wanting to have your cake and eat it as well. The elite athlete wants a thicker stronger tendon to get over the pain associated with Achilles insufficiency, but not one that is so much stronger that it means the Achilles does not recoil as much, such as a surgically-repaired one.

"The overload (running a Marathon), followed by everyday living activities only, led to transient Achilles tendon pathology after a delay in time (1 week), rather than immediately."

My final case vignette is about a professional footballer I looked after early in my career but who I won't name as his story is not in the public domain. He had chronic Achilles pain and hadn't been able to play for a month despite treatment, and with his team about to embark on a finals campaign he looked devastated to be about to miss the finals. He asked whether it would be possible to have the Achilles injected with local anaesthetic to play for the finals and after weighing up how important a finals series is to a professional player, I agreed to do it (after warning him of the risk of rupture). We decided to try it out at a training session beforehand and at this session he actually managed to rupture the tendon (whilst under local anaesthetic). Although this is another n=1 case study, it goes to show that whatever was making his Achilles tendon painful, it was doing so in a protective fashion because the tendon really was insufficient and prone to rupture if it was fully loaded.

So what lessons are there for treating a painful Achilles tendon from all of this discussion about mechanics? The number one lesson is to understand that the Achilles (like all other tendons in the body) is in a constant struggle to work out whether to become thicker and stronger or not. A painful tendon basically is one that needs to get stronger but is sending out painful signals to protect itself against rupture. We know from clinical experience – and study – that eccentric strengthening exercises tend to be superior to rest at curing tendinopathy pain. But we also know that ongoing heavy overload can make tendinopathy worse (and highly aggressive strengthening exercises can be in this category). So the therapist and patient need to work together towards the Blackjack hand of 21 without busting, or to cook the soup that is "not too hot, not too cold but just right". Therapists can classify patients into reckless individuals that are pushing through the pain barrier too much and need to back off, those who have become "pain-averse" who need to strengthen more, or those who oscillate between the two extremes and never hit the magic window of sufficient strengthening without overload. Most of the treatment of the therapist is advice on tactics, since the patient is the one living with the painful tendon 24 hours a day and has to make the right decisions on an ongoing basis. The patient can be warned that he or she might only pay the price for an overload days or even weeks down the track. But on the upside, any load upgrade that can be tolerated can usually be banked and maintained with the expectation of further strengthening. If the patient is a team athlete with a competitive season, he or she can be reassured that continuing a season with an ongoing overload is not going to be catastrophic, but that eventually the proper rehab will be needed in the off-season (and total rest is unlikely to constitute the "proper" rehab).

"There are a few hundred tendons in the body and we can't work as a team if every one of you complains every time you get stretched. So please Achilles tendon, just recoil as quickly as you possibly can without snapping or complaining (if possible), as you don't want to leave all of the work accelerating the body to the poor old calf muscle."

Is there a role for interventional management in Achilles tendinopathy? Of course there is, but the lesson that the patient must "get" is that interventional management cannot be used as a substitute to getting the underlying load management decisions correct. In a generic sense though, it may be that Achilles injections, so long as they don't harm the tendon (like cortisone might) could all work equally well. That is, the beneficial effect of an injection may be due to the insult around the tendon which could have the effect of stimulating the laying down of better organisation of more collagen fibres.

Despite my introduction of Achilles tendinopathy as a "management" problem and not a diagnostic one, it is becoming more apparent that there are subtypes of Achilles tendinopathy that may need to be managed differently. The most specific of these is calcific (or more correctly ossific) insertional Achilles tendinopathy. This can be painless in which case it doesn't need treatment. If the calcification is associated with pain, then shock wave has been shown to be helpful[6] and surgery may be required for high demand patients.

"Not content with curing himself, Alfredson tries to cure the world and publishes a moderate quality study (Fifteen recreational joggers in their early 40s compared to similar historical control group with no randomisation or blinding) which becomes of major historical importance, crediting previous authors who described successful treatment of tendinopathy with eccentric exercise."

Another specific subtype is the hypervascular appearance on Colour Doppler ultrasound. This again can be non-painful and doesn't require treatment. If painful, it may respond to ultrasound-guided polidocanol injection to the anterior (ventral/deep) surface of the tendon. It has been recognised that hypervascularity can be a normal finding in athletes and it also can be associated with rheumatological disease, in which case it is unclear whether it would respond to management of the inflammatory condition.

We are starting to recognise (with more extensive use of routine ultrasound examination in the clinic) that true partial tears of the Achilles tendon do exist and we need to manage these more conservatively (i.e. perhaps these cases need more rest/immobilisation than others).

Alfredson is a big believer that plantaris hypertrophy and tendinopathy can cause the medial Achilles to become painful, possibly through direct impingement, and that an extra-tendinous minor surgical procedure can be used to relieve this source of pain.

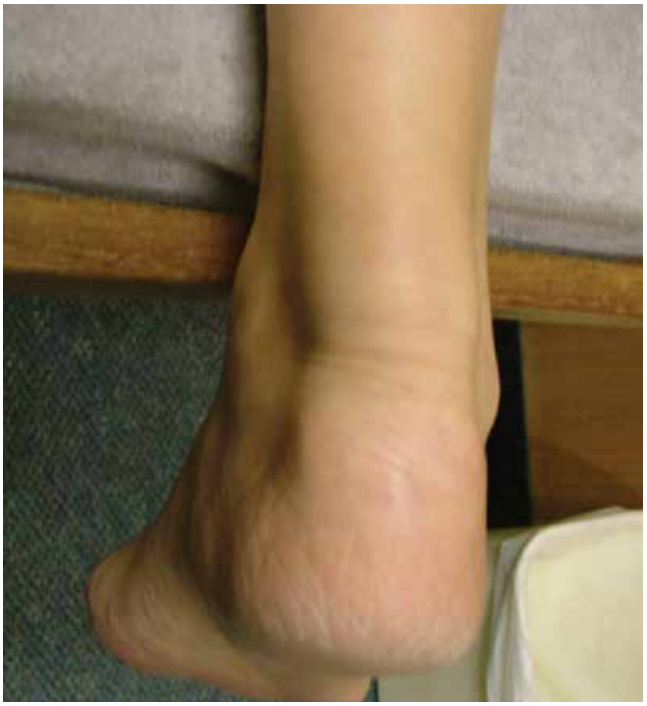


Figure 6: There is some evidence that the "average" mid-Achilles tendon with a lump may be associated with elevated cholesterol, but when it is a gross lump and there are cholesterol deposits elsewhere (e.g. around eyelids) is can be labelled Xanthomatous disease.

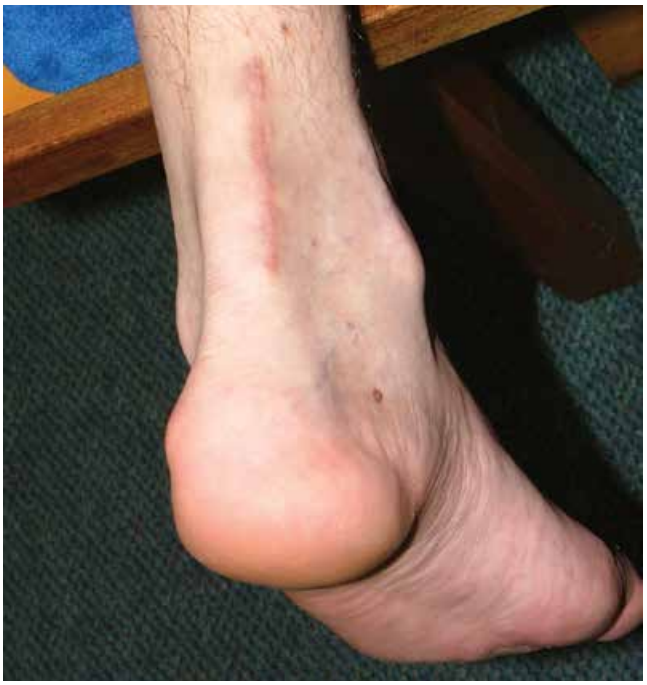


Figure 7: The average Achilles after surgical repair will be wider/thicker than normal, but across the length of the tendon rather than as a discrete lump in the mid-part of the tendon. Often the tendon will (eventually) become pain free.

The painful Achilles as a by product of systemic disease is also recognised, particularly in the case of hypercholesterolaemia. This is called xanthoma in the extreme cases, but again it is unclear in both these cases and in the milder cases whether lowering of cholesterol with diet or statin drugs is helpful.

The Achilles and calf can also be painful as a side effect of chronic nerve impingement in the low lumbar spine, a condition which is extremely common in middle to old age.

"As soon as you run an event fast enough to win an Olympic Marathon Gold or set a new World record, for example, you soon find that no matter how hard you train you have probably lost 1–2% of your pace. This probably relates to changes in all of the major tendons of the lower limb, but particularly the biggest of them all, which is the Achilles."

So just when we thought that diagnosis of Achilles pain was easy (and it was only management which was complicated) we may be about to enter a new era where Achilles pain needs to be subcategorised into different types, which may all respond to different management. This justifies practitioners who wish to subspecialise in tendinopathy needing to profess specialist understanding of the tendon. But it is also worth keeping the perspective that most people with Achilles tendon pain get better. The body – eventually – does a good job of curing the pain of Achilles tendinopathy itself in the vast majority of patients, probably with the help of the patient being advised or stumbling upon the formula of moderately loading the tendon just enough to strength it but not enough to overload it.

Further reading:

Peter Malliaras highlights signs and symptoms of common differential diagnoses in Achilles
http://www.completesportscare.com.au/2014/04/achilles-tendon-differential-diagnosis-peter-malliaras-carly-johnson/?doing_wp_cron=1398127110.6306259632110595703125

References, as indicated within the article, are available at sma.org.au/publications/sport-health

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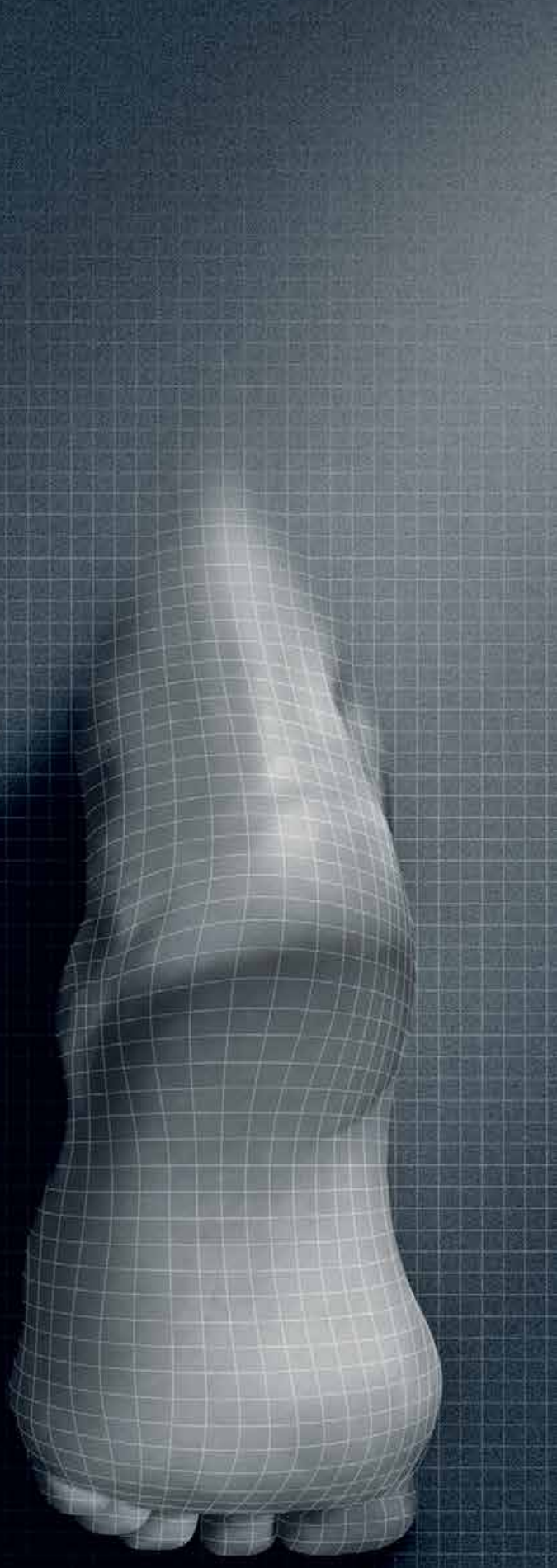

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Patellar tendinopathy and its diagnosis

Professor Jill Cook and PhD candidates Ebonie Rio and Sean Docking of Monash University's Monash Tendon Research group examine the diagnosis of patellar tendinopathy.

Anterior knee pain in the jumping athlete requires careful examination.

Despite the high prevalence of tendon pathology and tendon pain in these athletes that is reported in the literature, the diagnosis of patellar tendinopathy should not be assumed. During recruitment for a recent randomised trial in a volleyball cohort, we had to exclude approximately 50% of the athletes who presented with anterior knee pain. Many of these were being treated at that time for patellar tendinopathy; however clinical questioning and testing revealed that the tendon was not the source of symptoms in that athlete. The differential diagnosis for anterior knee pain is extensive so the purpose of this article is to help clinicians recognise if the tendon is the source of symptoms in the athlete. We will also illustrate how imaging may assist diagnosis, but it can also be misleading, especially in an athletic population. Importantly, tendinopathy remains a clinical diagnosis.

"The differential diagnosis for anterior knee pain is extensive so the purpose of this article is to help clinicians recognise if the tendon is the source of symptoms in the athlete."

Figure 1: Patellar tendon pain is most commonly at the inferior pole of the patella (A), it is also localised to this area when doing a decline squat (B).

"The single leg decline squat is the best clinical assessment tool."

Diagnosis

The key clinical features of patellar tendinopathy are localised pain most commonly at the inferior pole that occurs with patellar tendon energy storage and release (e.g jumping and change of direction), and a single leg decline squat test that reproduces pain at that site (Figure 1). Patellar tendon pain does not refer or spread over time or with additional load and people point to the area of pain with one finger. It is so specific that they don't move their finger and this subtlety is very important. Furthermore, loading causes a dose dependent increase in pain in patellar tendinopathy presentations, so pain will increase as the demand on the patellar tendon is increased. Another important and often distinguishing feature is the fact that they are usually worse the day after excessive loading, whereas this is variable in PFJ, fat pad or plica presentations.

In addition to localised pain, it is important to remember that tendinopathy reduces power and therefore clinical examination is likely to reveal a poorly functioning and powerless kinetic chain. Athletes will land with limited knee flexion in tasks such as hopping or dynamic change of direction tasks.

Differential diagnosis

Patellar tendinopathy is not usually associated with running as this is not a high load activity for the patellar tendon. Extra care with diagnosis needs to be given to the running athlete with anterior knee pain who are frequently treated for their tendon, however it may not be the source of their pain. Furthermore, volleyball and basketball athletes spend long periods of time in knee flexion and may also land on their knees leaving their patellofemoral joint (PFJ) vulnerable to compression that can present as anterior knee pain (the pain is less localised but can mimic aspects of patellar tendinopathy thus careful examination is vital). Both diagnoses usually have pain with stairs and prolonged sitting therefore these features do not distinguish between conditions. Another common clinical challenge is the athlete that complains of pain with cycling. Cycling requires no elastic energy storage of the patellar tendon so clinicians should look for a differential diagnosis or other activities in their training that may be causing tendinopathy.

Clearly there are a number of features that distinguish tendon from other sources of pain. Another difference is that taping to unload the PFJ will fail to change the SLDS pain in people with patellar tendon pain. PFJ taping that changes their squat pain almost certainly indicates the tendon is not involved as the pain source as these techniques offer little in the way of immediate relief for tendons. The best taping we have found to do this provides a diamond around the patella that aims to change sensory input and reduce retropatellar compression – taping with medial glides etc rarely help as it is direct compression in knee flexion that we are trying to unload.

Many clinicians wonder if PFJ and patellar tendinopathy can co-exist. They usually do not. Someone may have tendon pathology on ultrasound and PFJ symptoms – they should be managed as a PFJ presentation as loading for their tendon may exacerbate the PFJ compression and cause ongoing pain. An example of this is the athlete given Spanish squat isometric holds or wall sits that makes their anterior knee pain worse. This position will irritate the PFJ as it is in compression however someone with tendon pain is likely to get relief with this isometric loading. A more extreme example of this is the person given multiple injections into their tendon but who never had tendon symptoms. Tendon pathology on imaging should not change the clinical diagnosis.

"Many clinicians wonder if PFJ and patellar tendinopathy can co-exist. They usually do not. Someone may have tendon pathology on ultrasound and PFJ symptoms – they should be managed as a PFJ presentation as loading for their tendon may exacerbate the PFJ compression and cause ongoing pain."

Imaging

Use of conventional imaging modalities such as ultrasound (US) and magnetic resonance (MR) imaging have been shown to have good-to-excellent accuracy in detecting structural abnormalities[2–4]. Changes within the tendon are described as thickening of the tendon, focal areas of hypoechogenicity on US or increased signal on MR, and infiltration of blood vessels. However, caution is advised in basing a diagnosis of patellar tendinopathy solely on imaging. Like other soft tissue structures (ie intervertebral discs and cartilage), there is a disconnect between structural abnormalities and clinical symptoms. This is demonstrated by previous studies showing that 22% of volleyball players have pathological patellar tendons despite a lack of anterior knee pain[5]. Imaging should not be utilised as a test to exclude the tendon as the source of symptoms. Clinically, we frequently have seen structural changes on imaging in patients with patellofemoral joint pain, which can be misleading. New imaging modalities, such as ultrasound tissue characterisation (UTC), may be useful in assessing and monitoring the extent of structural pathology once a clinical diagnosis of tendinopathy is ascertained[6].

"The key clinical features of patellar tendinopathy are localised pain most commonly at the inferior pole that occurs with patellar tendon energy storage and release (e.g jumping and change of direction), and a single leg decline squat test that reproduces pain at that site."

A number of authors have frequently described the limitation of conventional imaging modalities in differentiating areas of pathology and partial tears[7]. There is no general consensus on the features and parameters in differentiating between tendinosis and partial tears with the differential often relying on clinical history (sudden vs. insidious onset of pain). Partial tears in the normal part of the patellar tendon are exceedingly rare, yet microtears can occur within the degenerative lesion, which are of little clinical importance and should be managed as a tendinopathy.

Categorising patellar tendinopathy in the continuum of tendinopathy[8], one of the most common presentations in the patellar tendon is reactive-on-degenerative (Figure 2). A focal area of disorganisation is observed within the central

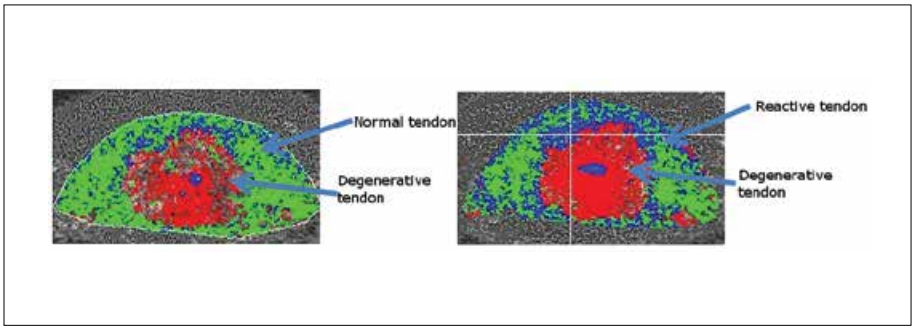

Figure 2: Example degenerative and reactive-on-degenerative patellar tendon over time. The degenerative lesion of the tendon has not altered, with diffuse reactive changes observed in the normal part of the tendon coinciding with an flare in symptoms.

portion of the tendon at the patellar insertion. It had been theorised that an acute onset of pain coincides with reactive changes in the surrounding normal tendon. Conventional imaging modalities have been unable to detect these reactive changes in the normal tendon. As UTC is sensitive to subtle changes in tendon structure, these reactive changes in the surrounding tissue can be seen. Identifying these changes has a substantial role in athlete management, as treatment can be directed at settling down the reactive portion.

"Whilst the diagnosis remains clinical, new imaging modalities such as UTC offer the clinician greater insight into common tendon presentations such as the reactive on degenerative tendon."

Conclusion and clinical message

Patellar tendinopathy is a clinical diagnosis of localised tendon pain that is exacerbated by activities that load the patellar tendon such as jumping and change of direction. However, even in jumping athletes anterior knee pain may be caused by a number of other structures. The single leg decline squat is the best clinical assessment tool. People that have changes on ultrasound but no clinical tendon symptoms should avoid interventions for their tendon and be managed according to the clinical diagnosis. Whilst the diagnosis remains clinical, new imaging modalities such as UTC offer the clinician greater insight into common tendon presentations such as the reactive on degenerative tendon.

References, as indicated within the article, are available at sma.org.au/publications/sport-health

Proximal hamstring tendinopathy assessment and management

Dr Peter Malliaras, Clinical Director at Complete Sports Care and Adj. Professor Craig Purdam, University of Canberra and Head of Physical Therapies at the Australian Institute of Sport, look at assessment and management of proximal hamstring tendinopathy.

Introduction

Proximal hamstring tendinopathy (PHT) is common among distance runners and athletes performing either primarily sagittal plane (e.g. sprinting, hurdling) or change of direction activities (e.g. various football codes and hockey)[1,2]. It can affect people who do not participate in sport, commonly peri-menopausal females and often in this demographic it is bilateral. PHT is characterised by deep localised ischial tuberosity region pain that is often worse during or after running, lunging, squatting and sitting. To date there is very limited evidence to guide management, often it can be longstanding and limit the ability to play sport and basic functions such as walking and sitting.

The pathological features in PHT are similar to those seen in common tendinopathies such as the Achilles and patellar[3].

Tissue samples from pathological proximal hamstring tendons show increased cellularity, an accumulation of ground substance, collagen disorganisation, and neurovascular ingrowth[4]. Cook and Purdam[5] have recently proposed the continuum model of tendon pathology, where diffuse increased cellularity and ground substance (reactive tendinopathy) precedes focal areas of collagen disorganisation and neurovascular ingrowth with progression over time to a morphology with discrete islands of degenerative tendinopathy. Malliaras et al.[6] demonstrated a progression from diffuse reactive pathology to localised degenerative pathology on ultrasound in the patellar tendon. There is no evidence of similar structural groups on imaging in PHT. Most studies report localised tendon pathology at the enthesis, bone oedema and insertional tendon clefts[2,7,8]. As with the Achilles, patellar and other tendinopathies, asymptomatic tendon pathology is not uncommon, alternatively a clinical diagnosis of PHT without significant demonstrable pathology is also seen[8]. This means the treatment focus should be on modifying cell signaling and pain.

Tenocyte signaling is thought to drive tissue changes and pain in tendinopathy. Some tenocytes fundamentally change their morphology, developing a more chondroid appearance and this seems to correlate with increased ground substance[9]. There are multiple signaling substances, such as cytokines and growth factors (e.g. IL6, TNFα TGFβ1), neuronal factors (e.g. acetylcholine, substance P), tissue regulators (e.g. matrix metalloproteinases, heat shock proteins, nitric oxide) that have a role in tendon adaptation but are also up-or down-regulated in pathology[10].

"From a clinical viewpoint, failure of conservative treatment is often related to inaccurate diagnosis or failure to recognise a comorbidity (e.g. sciatic neuritis), loading into hip flexion too early, not identifying and addressing hamstring atrophy/weakness (i.e. only performing compound and functional loading such as lunges and squats), and not gradual enough progression into functional and energy storage loading."

The exact nature of local pain generation has yet to be identified, but many of the signaling substances have been variously implicated, as they can be produced by both the local nerves and non-neuronal sources such as tenocytes, including production of substance P, calcitonin-gene related peptide and glutamate[11]. Attia et al.[12] identified a correlation between aggrecan expression in tendinopathy and pain. The continuum model proposes that very irritable or "reactive" pain may be related to tenocyte overstimulation and signaling, which is an appealing theory because it can explain pain in the absence of demonstrable imaging pathology[5]. Bilateral sensory changes that may reflect medium term spinal modulation have been reported in tendinopathy potentially indicating central sensitisation[13], although this has not been investigated in PHT.

"The key to management of all tendinopathies is progressive loading to reduce pain and restore function, performed within a pain monitoring framework. Some pain is acceptable during and after exercise, but symptoms should settle reasonably after exercise (within a few hours) and should not progressively worsen over the course of the loading program."

Aetiology of tendinopathy is multifactorial involving load related extrinsic factors, and intrinsic factors. Extrinsic factors may include training errors, poor running or even walking gait mechanics (PHT is common in race walkers)[1, 2]. Common training errors include increasing volume or intensity during energy storage (i.e. impact/lunging) loads too quickly, particularly the sudden introduction of sprint work or hills. Running can be an issue, even if not involving hills or sprint work, but this is usually modulated through kinetic chain intrinsic factors, commonly musculoskeletal alignment, motor patterning and or flexibility issues. Common kinetic chain deficits will be described in the following section.

Tendon compression at the ischial tuberosity or immediately distal is commonly a factor in PHT as it is in many insertional tendinopathies and has implications for management[14]. This may simply be a case of compression in association with energy storage load, as in the common example of a runner with increased anterior pelvic tilt statically or dynamically (which would affect the biceps origin primarily) or a lack of lumbo-pelvic control in the frontal plane dynamically which has the potential to affect the semimembranosus origin more specifically due to the lateral placement of its origin[2, 15]. Otherwise, compression may be the primary load insult, for example in yoga and pilates involving sustained end range hip flexion postures for extended periods. Deep loaded deadlifts and squats, as is common in "cross fit", can incite compressive symptoms. In some patients compressive load from sitting is the main load inciting factor[2]. Sitting alone is more common as an inciting factor in the older female perimenopausal demographic. Regardless of means of onset, not uncommonly it can be stretching, sitting or squats/deadlifts that perpetuate the patient's ongoing PHT.

The risk of PHT from an individual's compressive and energy storage load profile is further moderated by their intrinsic risk profile. Aside from musculoskeletal intrinsic factors these include genetic polymorphysms (e.g. COL5A1 that encodes for collagen type V), age, BMI, metabolic issues (e.g. lipid level imbalance, glucose intolerance, insulin resistance), hormones and medication (e.g. fluoroquinolone antibiotics)[10]. It is important to determine individual patients' intrinsic risk profile as this will impact tissue capacity and the threshold for tendon overuse injury and may be partially reversible in management.

"Proximal hamstring tendinopathy (PHT) is common among distance runners and athletes performing either primarily sagittal plane (e.g. sprinting, hurdling) or change of direction activities (e.g. various football codes and hockey)."

Diagnosis and assessment

Diagnosis hinges on typical tendon pain behavior, which includes well localised ischial tuberosity pain, that "warms up" with impact activity but is worse after. "Reactive" or irritable tendons may flare for days after provocative activity, whereas "stable" tendon pain settles within 24 hours. After initial "warm up" pain may return towards the end of a long run, which may be indicative of poor muscle endurance. There may be stiffness in the morning or after prolonged rest. Sitting for long periods, especially on harder surfaces, is often cited to aggravate symptoms. More diffuse symptoms may indicate lumbar or sacroiliac joint (SIJ) somatic referral, radiculopathy or sciatic nerve compromise in the buttock which is a common co-morbidity given its proximity to the hamstring origin[16–18].

"…compression may be the primary load insult, for example in yoga and pilates involving sustained end range hip flexion postures for extended periods. Deep loaded deadlifts and squats, as is common in "cross fit", can incite compressive symptoms."

Multiple pathologies that may also give buttock symptoms need to be considered in differential diagnosis (Table 1)[1, 19]. Thorough examination of the lumbar spine, SIJ and hip is essential, including pain provocation tests (e.g. Laslett's SIJ provocation tests, lumbar palpation, FADDIR)[19]. As mentioned above sciatic nerve irritation or neuritis in the buttock interfaces is a common co-morbidity that should be considered. Sciatic nerve compression or adhesions from a pathological hamstring origin have been reported[18]. Sciatic nerve may also be irritated at the piriformis interface[16]. Slump and straight leg raise assessment will identify sciatic or radicular involvement and "hamstring slump" (Figure 1) in anterior pelvic tilt can sensitise sciatic nerve involvement around the buttock, but these neuromeningeal and stretch tests may also be painful with an irritable hamstring origin tendon or ischiogluteal bursa and lack specificity. The sciatic nerve is more likely to produce diffuse pain and paraesthesia, but not always, so sometimes thorough palpation of the buttock neural interfaces is a key differential sign between tendon and sciatic nerve, as is the addition of significant hip adduction and internal rotation with slump or straight leg raise, or in combination with localised piriformis contraction[20] to provoke neural symptoms. Changes in presenting signs, symptoms or pain sites are common features of sciatic involvement, which is a frequent frustration for the athlete, clinician and other invested parties.

Figure 1: Hamstring slump in lumbar extension.

"The pathological features in PHT are similar to those seen in common tendinopathies such as the Achilles and patellar."

Lack of palpation tenderness around the ischial tuberosity is not uncommon and does not rule out a diagnosis of PHT in isolation. For this reason functional challenges (squat, lunge, arabesque) should also be included within the routine assessment of this region. Cacchio et al.[21] investigated the diagnostic performance of three passive tests in posterior hamstring tendinopathy, all involving stretching maneuvers. In the authors' experience, these tests may be negative in less symptomatic cases of PHT, for example, among patients who have load managed and have stable symptoms. This reinforces the importance of a detailed history to determine tendon pain behavior in diagnosing PHT. In many patients palpation is very tender on both sides, possibly indicating bilateral hyperalgesia, or normal sensitivity to palpation. Overall, palpation is a poor indicator of severity and a poor diagnostic sign in isolation.

Table 1: differential diagnosis of PHT

Other diagnoses	Key features
Sciatic nerve at the ischial tuberosity	Sciatic tenderness at QF May be provoked by hamstring slump, worse with hip adduction/IR Diffuse thigh radiation of symptoms
Piriformis syndrome	Sciatic tenderness at piriformis May be provoked with piriformis stretch/contraction or slump with adduction/IR Diffuse thigh symptoms
Ischiogluteal bursitis	Pain on stretch or localised palpation Irritable symptoms with sitting MRI or US confirmation
Lumbar facet arthorpathy, disc degeneration, radiculopathy	Diffuse leg referral Inconclusive hamstring loading tests
Ischiofemoral impingement	Palpation tenderness over QF – variable Possible reproduction with femoral external rotation in hip neutral MRI diagnosis if not responding
Unfused ischial growth plate (rare)	Avulsion history in teenage years MRI diagnosis if not responding
Apophysitis or avulsion	In younger athletes Sudden incident, e.g. kicking
Chronic compartment syndrome of the posterior thigh (rare)	Pain that does not warm up Diffuse whole of hamstring discomfort
Posterior pubic or ischial ramus stress fracture	Female athletes, overtraining Tenderness over posterior pubic or ischial ramus

Single leg squatting is assessed in regards to pain and function, including coronal/frontal and sagittal plane movement patterns. In the sagittal plane, loss of lumbo-pelvic extension control (anterior tilt) in squatting or lunging will increase hamstring origin compression. The long lever bridge (Figure 2) often reproduces pain in PHT, as does the arabesque. This can be performed slowly initially, and then with progressive speed if asymptomatic. Other functional tests, including short lever bridge and single leg squatting may not consistently reproduce symptoms; unless the tendon is reactive.
Running or walking gait analysis, or sports specific movement pattern assessment is critical in managing PHT. Patients often display over-striding and/or a heavy heel strike in walking quickly or running, poor anterior pelvic tilt control, and/or lack of terminal hip extension.

Kinetic chain deficits may increase hamstring origin stress concentration. Common kinetic chain deficits may include "hamstring dominance" where there is poor gluteus maximus synergic contribution and dominant hamstring activation when examined in standing or prone hip extension. This may be associated with restriction or dominance in hip flexor muscles, particularly tensore fascia lata, iliopsoas and rectus femoris. Some patients present with hamstring weakness and atrophy, often in relation to long-standing symptoms. Changes in activation of the hamstring component of adductor magnus may also be an issue, often becoming hypertonic secondary to hamstring weakness.

Hamstring flexibility can be variable. Greater hamstring tensile stress and absorbed energy has been reported at end range among flexible compared with less flexible people[3], which may increase enthesis compression and injury risk in populations that are flexible and stretch, e.g. yoga. Alternatively some athletes present with straight leg raise of no more than 20 or 30 degrees. Distal kinetic chain weakness or restriction or quadriceps dysfunction are less common but possible, hence thorough individual assessment related to the sporting demands of the individual is important. Increased anterior pelvic tilt in standing and sitting or increased hip flexion in sleeping postures may be a feature. In sharing these clinical presentations it is recognised that our knowledge of muscle actions and synergies in this region requires significant further study, and current assessments may be limited[22].

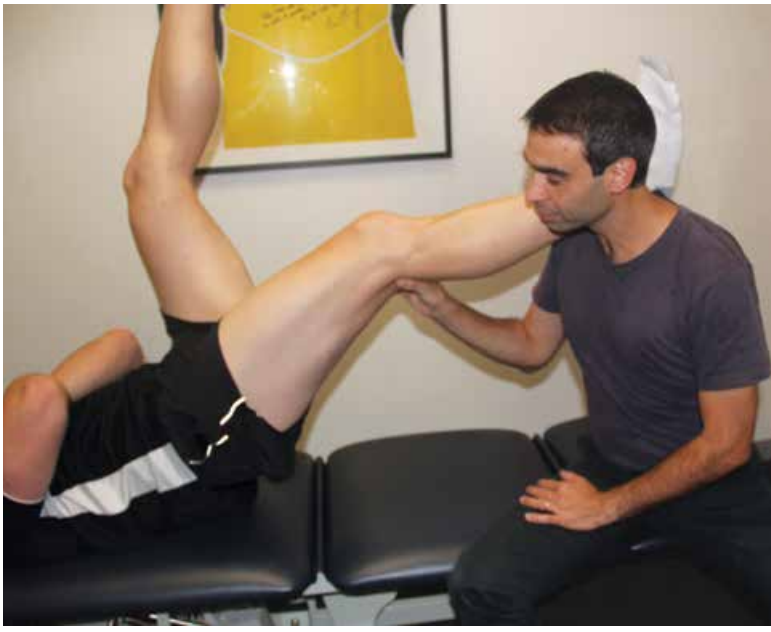

Figure 2: Long lever bridge assessment.

Recently the Victorian Institute of Sport Assessment (VISA), originally developed as a pain and function outcome questionnaire, has been adapted for the hamstring origin[23]. This is an appropriate outcome measure but is limited in demonstrating short-term change in symptoms. Visual analogue scale rating (/10) of painful functional tests (e.g. long or short lever bridge or later the arabesque) is preferable for short-term within session clinical assessment, and patient specific functional scale rating of key functional deficits can be used for within and between session assessment.

Management

The key to management of all tendinopathies is progressive loading to reduce pain and restore function, performed within a pain monitoring framework. Some pain is acceptable during and after exercise, but symptoms should settle reasonably after exercise (within a few hours) and should not progressively worsen over the course of the loading program. There are limited narrative reviews[1,2] and case series[24] outlining management of PHT, primarily based on clinical experience.

"For recalcitrant lesions, consideration needs to be around whether the presenting features are solely that of the tendinopathy or if involvement of other structures, most commonly the sciatic nerve, are a feature."

The primary goal among patients with irritable symptoms is to settle their pain. This can be achieved via multiple first line interventions including isometric exercises, ice, load management, manual therapy (e.g. soft tissue work, trigger point therapy, dry needling). Load management is critical. In practice, abusive compressive (loaded hip flexion) and energy storage loads are limited until pain settles to a stable level. Not uncommonly the patient may be able to continue some steady state running, within pain/aggravation guidelines, but hills, starts and hurdles are best avoided until later stages. Cycling on a stationery bike in standing may also be tolerated, as will swimming and normally water running. There should be less pain with these low load activities and ideally pain should settle within 24 hours of a high loading bout (e.g. a lunging or a run). Posture modification should involve reducing hamstring origin compression from anterior pelvic tilt and hip flexion in standing, sleeping and sitting (through unloading cushions). In truly reactive or irritable patients, all energy storage activity will need to be ceased for a period of time, usually 2–8 weeks. Anti-inflammatory medication (e.g. ibuprofen) is useful for settling "reactive" tendon pain and may also influence tenocyte overstimulation and signaling[25].

Figure 3: Prone hip extension – can be isometric or isotonic.

Figure 4: Leg curl – can be isometric or isotonic.

Specific tendon loading

Stage 1 rehabilitation – Isometric load: Utilisation of isometric holds of the hamstrings whilst avoiding compression of the origin. It appears holds of 30–60 seconds repeated 3–4 times and repeated around 4 times per day are effective (Rio 2013) in reducing pain and commencing load of the myotendinous unit (examples include leg curl, bridge holds, straight leg pull down, trunk extensions. See Figures 3 and 4). Importantly resistance should be added fairly quickly and progressed for optimum effect. The hip should be near neutral hip flexion-extension position or in minimal hip flexion (e.g. 20–30 degrees for straight leg pull downs). A good prognostic sign for isometrics is an immediate reduction in pain with hamstring loading tests post exercise.

"Common training errors include increasing volume or intensity during energy storage (i.e. impact/lunging) loads too quickly, particularly the sudden introduction of sprint work or hills."

Stage 2 rehabilitation – hip neutral, hamstring load: As symptoms become more "stable" (e.g. minimal pain with lower load tests such as short lever bridge, less reactive to provocative load) exercise to restore hamstring bulk and function is introduced. Loaded hip flexion is minimised to protect the enthesis against compressive stimulus. Suitable exercises include prone hip extension, leg curl, bridging, and Nordic hamstring lowers (e.g. Figures 3, 4). The focus is slow isotonic concentric-eccentric loading performed every other day. Stage 1 exercises can be continued on the "off" days, particularly if symptoms are still present and isometrics have an immediate positive effect on symptoms.

"The continuum model proposes that very irritable or 'reactive' pain may be related to tenocyte overstimulation and signaling, which is an appealing theory because it can explain pain in the absence of demonstrable imaging pathology."

Stage 3 rehabilitation – hip flexion, hamstring load: Gradual re-introduction of hip flexion range into isotonic hypertrophy and functional exercises. Commenced when there is minimal pain with higher loading hip flexion tests, e.g. long lever bridge, arabesque. Exercises may include slow step ups, deadlifts, hip thrusts, walking lunges, sled pull/push (e.g. Figures 5–7). Generally performed every second day in place of or mixed with stage 2 exercises, guided by 24 hour pain response to provocative tests e.g. bridge. Addition of loaded hip flexion may lead to provocation, hence constant monitoring and "tuning" of load is generally necessary through this phase. Muscle bulk and capacity should be fairly equivalent at the completion of this stage.

Figure 5: Split squat (static) or walking lunge.

"From a clinical standpoint peritendinous corticosteroid injection would appear to provide good assistance when rehabilitation plateaus, or to gain early symptomatic relief in the reactive earlier stages of PHT."

Stage 4 principles: Energy storage loading. Reintroduction of power/elastic stimulus for the myotendinous unit. Can be commenced when there is minimal pain during and after high load tests (e.g. arabesque) and equivalent strength in single leg stage two and three exercises. Early in this phase, hip flexion range during exercise may be limited to limit

Figure 6: Sled Push.

Figure 7: Hip biased deadlift – limited knee flexion.

compression as the higher elastic loading is added. Weights are generally reduced as the speed of movement is increased. As this is the most provocative stage, this would generally be instituted every third day, followed by a stage 1 day to settle the tendon then a strengthening (stage 2–3) day to form 3 day high-low-medium tendon load cycles twice a week, with a rest day allowed.

Figure 8: A-skips.

Examples of early to later stage 4 exercises include fast sled push/drag; 15–20 steps, repeated for 3–4 sets using the affected leg, fast split squats, alternating forward leg, graduating range over time, bounding, A-skips, stair bounding, hill bounding (if required), scooter on grass and gradual reintroduction of sport-specific squat and lunge activities including off-axis work (e.g. Figures 6 and 8). Again, sessions would include perhaps only 3–4 of these activities graduating to higher tendon load over weeks. Care should be taken with the introduction of hill running sessions as these can be quite provocative. The consideration should be around introducing these activities as stepping stones back to return to sport, yet these activities should not be added to the normal training volume as one resumes sport, to avoid overload.

Kinetic chain interventions

In brief, these should include consideration of three-dimensional lumbopelvic control in functional single leg stance tasks, including reducing lordotic postures in functional movements. Reduction of hamstring dominance (if it is a feature) via increased gluteus maximus contribution (activation and capacity) is critical. Greater gluteus maximus capacity and bulk reduces stress concentration at the hamstring origin as well as providing some cushioning in sitting. Gluteus medius and minimus strength and re-education may be necessary if frontal plane hip and pelvis control is compromised. Hip flexor dominance can be associated with poor gluteal function and also often needs to be addressed to enable lateral hip stability function. This may include improving hip extension or rotation flexibility,

and/or retraining balance between TFL and gluteal lateral stabilises by avoiding exercises that bias TFL (e.g. clams). Restricted hip flexion often may require attention, particularly in sports that require this range in lunging with a flexed trunk or high kicking. Soft tissue work, seatbelt distraction/mobilisation into flexion and home exercises are all useful interventions. In severe restriction surgical opinion may be sought. The distal kinetic chain is generally less of an influence in PHT, although knee joint dysfunction (extension deficit) may result in increased semimembranosus activation. Similarly, biceps femoris activation may increase with superior tibiofibular joint hyper or hypomobility. Clinically, ankle dorsifexion restriction has the potential to alter kinetic chain load sharing on take-off and landing from a jump, likewise hallux limitus would appear to affect gluteus maximus activation on push off.

"Diagnosis hinges on typical tendon pain behavior, which includes well localised ischial tuberosity pain, that 'warms up' with impact activity but is worse after. 'Reactive' or irritable tendons may flare for days after provocative activity, whereas 'stable' tendon pain settles within 24 hours."

Other treatment options

For recalcitrant lesions, consideration needs to be around whether the presenting features are solely that of the tendinopathy or if involvement of other structures, most commonly the sciatic nerve, are a feature.

In terms of graduating interventions for a "pure" tendinopathy, some evidence exists for Extracorporeal Shockwave Therapy (ESWT) and in the authors' experience it can be a powerful pain modulator but is less effective or can even flare "reactive" symptoms. Cacchio[26] utilised a radial ESWT unit over a 4 week period, followed up at 3 and 12 months in a cohort of 20 PHT patients aged 18–25. A superior result to traditional conservative treatment was reported. The mechanism of effect remains unclear, with some reports of therapy induced neuropraxia explaining the loss in pain.

Injections

In meta-anlyses of injection therapies in tendinopathy there has been no clear recommendation formed for any injectable substance with the main finding being a similar outcome in chronic patellar tendinopathy for PRP, whole blood, dextrose, polidocanyl and dry needling[27, 28]. The exception was corticosteroid injection which provided a better short term outcome but poorer long term outcome. No such meta-analysis has been undertaken for PHT as only 3 articles describe injection interventions to date, two utilised PRP[29,30] and one corticosteroid[31]. The use of corticosteroid in a mixed demographic (age range 10–65 yrs) and mixed proximal hamstring pathology provided relief in 50% at 1 month and 24% at greater than 6 months. The PRP studies utilised similar cohorts in terms of age (17–73) and gender distribution for a total of 27 PHT injuries. Moderate to complete relief could be interpreted across the 2 proximal hamstring cohorts at better than 80%. There are methodological and reporting differences that preclude deeper analysis.

From a clinical standpoint peritendinous corticosteroid injection would appear to provide good assistance when rehabilitation plateaus, or to gain early symptomatic relief in the reactive earlier stages of PHT. It is also of value when neural symptoms predominate, however it is less useful for the "pure" tendinopathy in more advanced cases. PRP appears to be utilised frequently in PHT domestically. It may provide some benefit in more advanced/degenerative type tendinopathies, although in the authors' experience (which is skewed to more recalcitrant cases) there may be prolonged exacerbation, especially when administered intra-tendinously when symptoms are reactive or irritable.

Conclusion

This review has highlighted key differential diagnoses, kinetic chain assessment findings and management for patients presenting with reactive to degenerative PHT symptoms. From a clinical viewpoint, failure of conservative treatment is often related to inaccurate diagnosis or failure to recognise a comorbidity (e.g. sciatic neuritis), loading into hip flexion too early, not identifying and addressing hamstring atrophy/weakness (i.e. only performing compound and functional loading such as lunges and squats), and not gradual enough progression into functional and energy storage loading. It needs to be stressed that every patient presents individually and this may necessitate a focus on particular phases of rehabilitation or kinetic chain factors. For example, some patients do not have hamstring dysfunction whereas others have marked dysfunction so clearly rehabilitation focus will diverge in these groups.

References, as indicated within the article, are available at sma.org.au/publications/sport-health

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FIFA World Cup Fever

With the FIFA World Cup about to kick off, *Sport Health* talks to Dr Mark Jones, Sport and Exercise Medicine Physician and team doctor for the Socceroos.

What is your professional background? Where and what have you studied?

I am a Sport and Exercise Medicine Physician working in private practice in a multi-disciplined Sports Medicine clinic and multi-disciplined Occupational medicine clinic in Wollongong. I perform a consultancy in work-related injuries in Sydney and Wollongong also. I have been club doctor for the Western Sydney Wanderers football club in their initial two A-league seasons. I am also team physician for the Socceroos, having commenced with the team in a shared capacity in 2006 and being the sole team doctor since 2010.

I graduated from med school in 1984, achieved my Diploma in Sports Medicine from London Hospital (UK) in 1987–1988, and gained my Fellowship from the Australasian College of Sports Physicians in 1994.

I spent more than 15 years working part-time in Aboriginal health and 16 consecutive years as club doctor for Illawarra Steelers Rugby League team and subsequently the merged entity St George-Illawarra Dragons NRL team.

"It is a challenging World Cup group, but life is about meeting challenges. Challenges create opportunities to grow in your knowledge, skills, and abilities, as a person and as a team."

How did you get involved in working with elite sport?

My interest and desire was sparked as a med student by seeing one of my surgical tutors on TV on the sideline as team doctor for the Australian rugby team.

I enjoyed a range of sports and competed through med school and adulthood as a distance runner to international level. Athletics administrators asked me to be the doctor for NSW teams at the Nationals, and eventually I became team doctor for some National team T & F overseas trips, including an Athletics World Cup.

During my intern year, I rang the Balmain Rugby league club doctor, who kindly allowed me to spend that season on the bench with him, observing.

I also enjoyed travel, wanted to live overseas for a while, and therefore enrolled in the 9 month full-time London Hospital Sports Med Diploma course. Upon returning from London in 1989, I moved to Wollongong, and received a phone call out of the blue asking if I wanted to take over as club doctor for Illawarra Steelers Rugby league team.

Sport as a hobby had developed into my career.

"…be mindful of maintaining a healthy balance with your family life, because international or elite sport does place extra demands and sacrifices on your family. The support of your family allows you to achieve your goals and enjoy the experience so much more."

You have worked as a club doctor for two different football codes – rugby league and soccer – what is different in a sports medicine sense between the two codes?

Football (soccer) injuries can be acute, though overuse injuries seem to be more common by virtue of the running, twisting, kicking involved in a game and at training. Addressing predisposing factors to overuse injury becomes very important in football. Obviously, lower limb, truncal/pelvic injuries predominate. Sideline medical decision-making is dictated by football allowing only three replacements per match and no interchange. If the acute trauma occurring in a match is significant, you do not have the luxury of time to allow a player to recuperate on the sideline to allow possible return to play. Decisions and treatment have to be quick and accurate.

The regular travel involved in international football and regular mid-week scheduling of international matches requires careful planning regards recovery, nutritional, jet lag, travel fatigue, overload strategies etc. At the Wanderers, we have just recently completed a stint of five games in 15 days, two of them Asian Champions league matches and involving overnight flights to and from China two days before ensuing fixtures. The international footballer tends to be extremely dedicated and personally responsible for their own recovery arising from travel and match demands.

"Input is also given to planning strategies related to travel, jet lag, recovery, climactic issues etc that arise from playing in such a diversity of countries and conditions…"

Tell us about your main duties as team doctor for the Australian Socceroos World Cup team.

I provide medical coverage for the team at all matches and training, in Australia and overseas. I stay at the team hotel and am available for general medical or musculoskeletal conditions suffered by players and staff, screening of players upon arrival into camp, emergency care, sideline treatment, nutritional advice, and am involved in monitoring of player hydration and weight. On one occasion I had to arrange to have a staff member suffering renal vein thrombosis airlifted out of a Slovenian ICU to the UK. During the Asian cup finals in Qatar, one whole day was spent in a Qatari hospital arranging investigations and Urological opinion arising from a player with testicular trauma suffered in a match.

When the Socceroos are not together as a group, our head physiotherapist and I are available for players to seek advice regards injuries, treatment or investigations occurring whilst playing for their overseas clubs. Liaising with their club's medical staff can achieve a satisfactory rehabilitation outcome for the player, and ultimately their club and the National team. On occasions, and in agreement with their club, surgery and postoperative rehabilitation has been arranged in Australia.

Input is also given to planning strategies related to travel, jet lag, recovery, climactic issues etc that arise from playing in such a diversity of countries and conditions; for example, in the winter of 2012 we played a World Cup qualifier in 45° heat in Oman and backed up three days later to play a qualifier against Japan in Brisbane.

For the upcoming World Cup in Brazil, our medical team has provided medical input to planning meetings involving management and coaching staff aimed at strategies for optimum player health and performance, Medical and sports science staff have visited and liaised with experts in the area of recovery, travel medicine, sleep, nutrition, performance etc, reviewed protocols with respect to these areas, and set up a network of medical care requirements in Brazil and Australia. Potential squad members in Australia and overseas are currently having blood screening performed and emailing results, as well as fulfilling recommended vaccination requirements. They are currently undergoing nutrition review and have a phone app regarding training loads, recovery, health and general welfare parameters provided and screened by our sports scientist. I am made aware of any alerts he receives.

Who else makes up the Australian World Cup sports medicine team? Tell us a little about these working relationships in terms of looking after the players.

We will have three physiotherapists who provide day to day treatment and monitoring of player injuries, musculoskeletal screening of every player as they arrive into camp, and prescription of prehab, core, recovery and rehabilitation exercise programs. Strapping, massage, prehab are match day physiotherapy duties. The head physio and I sit on the sideline bench to provide acute care during a match.

There is a team massage therapist for day to day treatment and prevention. Our physiotherapists are manual practitioners so there is significant overlap to assist the massage therapist. This is evident in the post-match recovery massage "flush" and combined multi-tasking, which occurs in the post-match recovery protocols of ice baths, rehydration, nutritional recovery etc.

Our sports scientist monitors training loads via GPS, daily player well-being, sleep, hydration, weight, RPEs etc. He works with our team chef to oversee quality and content of our meals. He allies with the physios for prehab and warm-up requirements at training.

As you can see, there is a collaborative and multi-tasking approach within our medical/science team. Daily meetings are necessary to facilitate this. We also meet daily with the coach.

The Socceroos have a consultant nutritionist and we have arranged podiatry, dental, pathology and cardiac screenings to be performed prior to leaving for Brazil.

FFA's chief medical officer offers advice and back-up as required.

What are some of the more common medical and fitness injuries in soccer players?

Lower limb muscular strains, contusions, and joint sprains. Lower back, pelvic/groin or hip pain are common also.

"On one occasion I had to arrange to have a staff member suffering renal vein thrombosis airlifted out of a Slovenian ICU to the UK."

How do these injuries typically occur?

Acute injuries can occur due to the contact nature of the sport. However, overuse factors play a significant role in football injuries and emphasise the need to monitor training loads, strength, flexibility and proprioceptive deficits among other things. Injuries therefore are just as likely to manifest at or arise from training. The kinetic chain effect arising from repeated twisting, kicking, change of direction contributes to the lumbar, hip and groin injuries.

Your role requires you to travel with the team – what is a typical day like when you're on the go?

The first day or two of a trip are dominated by setting up the medical room and player screenings as players fly in from their various clubs around the world.

Monitoring of player hydration, weight, sleep and well-being occurs prior to breakfast. There is a medical staff meeting and then meeting with the coach prior to breakfast also. All staff prepare for and attend the field training session, commonly held in the morning. The remainder of the day and evening is available for player treatment. Team core, stretching, gym or recovery sessions are interspersed among the training schedule. Any requirement for investigations or specialist/paramedical treatment is organised and attended with the player on an as needs basis.

What are the highlights and challenges of working with elite sportspersons?

For me, highlights include access to the best specialists, knowledge, and sports medicine practices, which is a continued and fantastic learning opportunity. It encourages continued self-evaluation and improvement to enable the provision of up to date evidenced based care to the players. Elite football allows exposure to some amazing countries, cultures, and experiences, both sporting and non-sporting. Standing on the sideline whilst singing the National Anthem is always a special moment.

The Socceroos face a unique challenge due to the vast travel that can be involved for our team, having players competing for clubs all around the world. Some of these challenges can include jet lag, travel fatigue, climactic challenges, cultural differences, variability in standard of medical facilities we may need to access when overseas. Importantly, as a medical/sports science staff, we have to pay careful consideration to any overload issue arising from the elite footballer sandwiching international matches between club commitments that often are two or three days on either side of a qualifier or "Friendly international". However, those challenges are what inspire us as sports med professionals.

What advice would you give other sports medicine professionals looking to work within elite sport?

Be a team player without losing your independent decision making. Everyone within the team (players and staff) is important and contributes to the collective success. Develop rapport with the players but maintain your professionalism at all times. Maintain clarity regards your role within the team at all times. It can be easy to get carried away in the excitement or tenseness of a match. Remain humble.

By all means though, be enthusiastic, and enjoy the privilege and challenges. There is much to be enjoyed, so soak it in.

Finally, be mindful of maintaining a healthy balance with your family life, because international or elite sport does place extra demands and sacrifices on your family. The support of your family allows you to achieve your goals and enjoy the experience so much more.

"My interest and desire was sparked as a med student by seeing one of my surgical tutors on TV on the sideline as team doctor for the Australian rugby team."

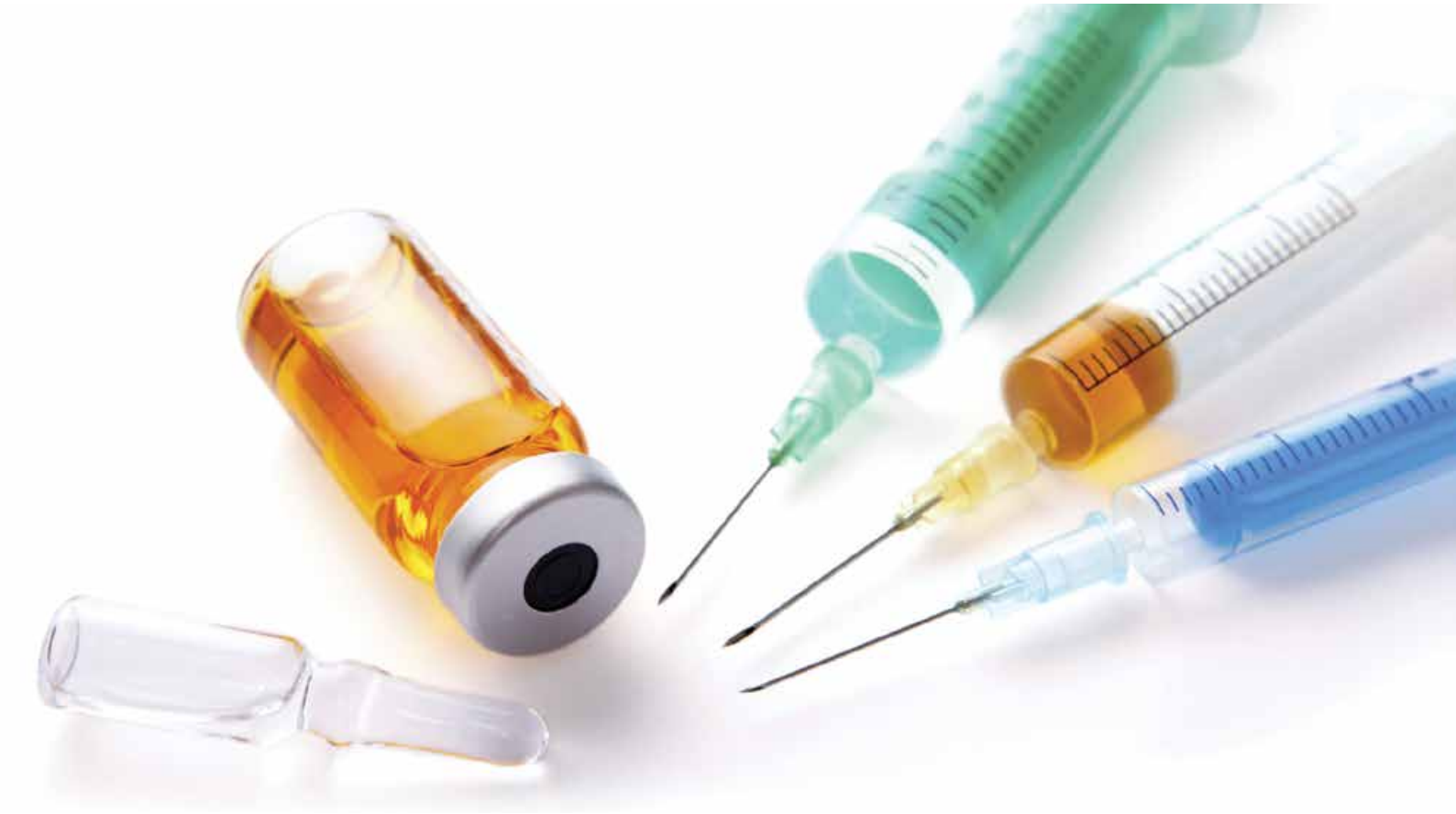
Finally…Australia has drawn a tough World Cup group. What do you think of our chances?

The sports medicine staff supporting the Socceroos endeavour to approach every match or tournament proactively and with a view to giving our best to allow the players and coaching staff to perform at their best.

It is a challenging World Cup group, but life is about meeting challenges. Challenges create opportunities to grow in your knowledge, skills, and abilities, as a person and as a team.

Achilles tendinopathy management

High volume injections, surgery and differential diagnosis



Professor Nicola Maffuli, Dr Alessio Giai Via and Dr Francesco Oliva examine the management of Achilles tendinopathy including high volume injections and surgery. The article also looks at true partial tears, plantaris pathology and MRI and differential diagnosis.

High volume injections for the management of Achilles tendinopathy

The source of pain associated with mid-portion Achilles tendinopathy has not been clarified yet. Some authors hypothesised that the main cause of the pain arises from the surrounding tissues.

Patients with chronic painful tendinopathy of the main body of Achilles tendon (AT) often present neovascularisation outside and inside the ventral part of the tendinopathic area[1]. However, neovascularity in absence of pain is not necessarily pathological, and, in athletes, it can just indicate a physiological response to physical training[2]. The ingrowth of sensory and sympathetic nerves from the paratenon accompanies the neovessel in chronic painful Achilles tendinopathy. These sensory and sympathetic nerves can release nociceptive substances, and may be the primary source of pain[3]. High volume image guided injections (HVIGIs) target the neurovascular bundles growing from the paratenon into the AT. HVIGIs would produce local mechanical effects, causing the neovascularity to stretch, break or occlude, and pain relief could be explained by the destruction of these sensory nerves[3]. Denervation of the AT by releasing the paratenon may be the most important part of this procedure.

"Controversy exists regarding the existence of plantaris tears. Early descriptions of plantaris tears were later disputed, with several investigators questioning their existence. The advent of modern imaging technology along with surgically documented lesions confirmed these entities."

"We reported excellent results in 17 elite athletes after percutaneous surgical repair of Achilles tendon rupture. All patients came back to competing at high level and the average time to return to full sport participation was 4.8 ± 0.9 months (range 3.2 to 6.5)."

Several substances have been injected in and around tendons including normal saline, corticosteroids, local anaesthetic[4,5]. Platelet rich plasma application did not show the expected benefits in Achilles tendon[6]. In preliminary studies in patients with recalcitrant tendinopathy of the main body of the Achilles tendon, a HVIGI decreased the amount of pain perceived by patients, and improved functional activities in the short- and long-term[5,7]. A mixture of 10 mL 0.5% bupivacaine hydrochloride 25 mg of hydrocortisone acetate can be used for HVIGI in chronic Achilles tendinopathy, immediately followed by 4×10 mL of injectable normal saline. Hydrocortisone acetate is used to prevent the inevitable acute mechanical inflammatory reaction produced by the large amount of fluid injected in the proximity the tendon. The injection is performed under ultrasound guidance to avoid intratendinous corticosteroid injections. Patients are allowed to walk on the injected leg immediately, but they are strictly advised to refrain from high impact activity for 72 h. After this period, they are instructed to re-start eccentric loading physiotherapy regime twice daily until they stopped their sporting career. Good results have been reported with this technique at short term follow-up[7]. In a more recent study[8], we used Aprotinin, an 85 amino-acid 65 kD basic polypeptide extracted from bovine lungs. It is a broad spectrum serine protease inhibitor, with inhibition of plasmin, trypsin and kallikrein, forming reversible competitive bonds with these enzymes, inhibiting their proteolytic action and their vasoactive effect in the first stages of inflammation. It may block matrix metalloproteinases (MMPs), including MMP-1, MMP-8 and MMP-13 (collagenases) and MMP-2 and MMP-9 (gelatinases), either directly or via inhibition of plasminogen and plasmin. At one year from the index injection, HVIGI produces statistically significant and clinically relevant improvement in VISA-A score, and is associated with a relatively high rate of return to sport in athletic patients. There were no serious adverse events related to the injections. Aprotinin has been recently withdrawn worldwide following major concerns about postoperative thrombosis and organ dysfunctions in patients who had received this metalloprotease inhibitor intraoperatively in doses several orders of magnitude greater than the ones used in the present study[9]. In our setting, we now use only hydrocortisone acetate for HVIGI.

In conclusion, HVIGI is effective to improve the symptoms of resistant Achilles tendinopathy. It is safe and relatively inexpensive, with the potential to offer an alternative management option before surgery, aiding a quicker return to sport[7]. HVIGIs warrant further investigation to try and understand the bases of its effects, and to better study its role in the management of Achilles tendinopathy.

Surgery for mid-portion Achilles tendinopathy

Tendinopathy of the main body of AT affects both athletic and sedentary patients. The incidence in top-level runners has been estimated between 7 and 9%[10], while 30% of patients have a sedentary lifestyle[11]. The etiopathogenesis of Achilles tendinopathy remains unclear. It has probably a multifactorial origin and it has been attributed to a variety of intrinsic and extrinsic factors[12].

Pain is the cardinal symptom of Achilles tendinopathy. It occurs at the beginning and a short while after the end of a training session. As the pathological process progresses, pain may occur during the entire exercise session, and, in severe cases, it may interfere with activities of daily living. Clinical examination is the best diagnostic tool. Location of pain 2–6 cm above the insertion into the calcaneum and pain on palpation are reliable and accurate tests for diagnosis[13] (Figure 1).

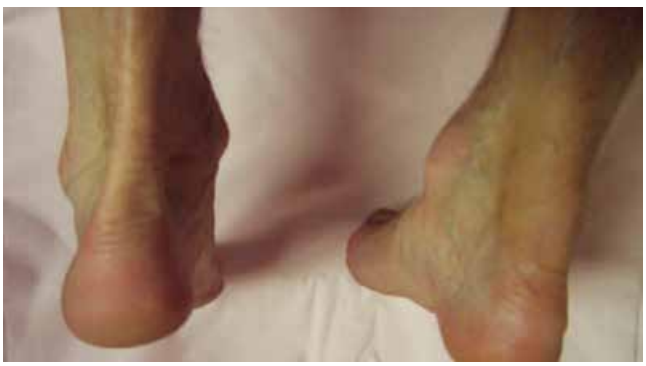


Figure 1: Achilles tendinopathy of the right Achilles tendon.

The management is primarily conservative. Eccentric exercise and shock waves have been proved to be effective for the treatment of Achilles tendinopathy[4,14]. However, conservative management is unsuccessful in 24% to 45.5% of patients, and surgery is recommended after at least 6 months of

conservative management[15]. For AT, frequency of surgery has been shown to increase with patient age, duration of symptoms, and occurrence of tendinopathic changes[16,17]. Both open and mini invasive surgical techniques have been described for treatment of tendinopathy of the mid-portion of Achilles tendon.

"Acute AT rupture is a serious injury. Most (75%) acute ruptures occur during recreational activities in men between 30–40 year old, but 25% of ruptures take place in sedentary patients."

Open surgery

Under locoregional or general anesthesia, the patient is placed prone with the ankles clear of the operating table. A tourniquet is applied to the limb to be operated on. The limb is exsanguinated, and the tourniquet is inflated to 250 mm Hg. The incision is made on the medial side of the tendon to avoid injury to the sural nerve and short saphenous vein (Figure 2). The skin edge of the incision should be handled with extreme care as wound healing problems

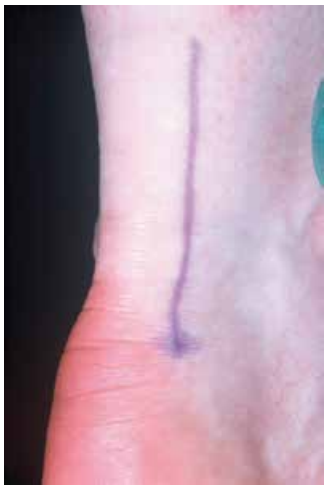


Figure 2: Incision used for open surgery: it lies just posterior to the medial border of the Achilles tendon. It avoids the sural nerve and the short saphenous vein, and the scar is away from the shoe counter.

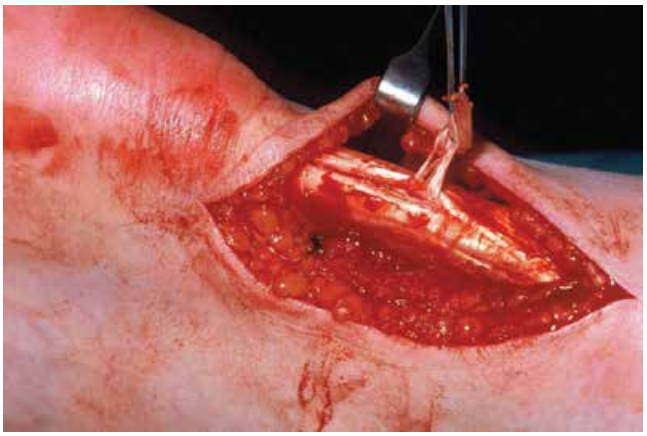


Figure 3: The tendinopathic tissue is excised.

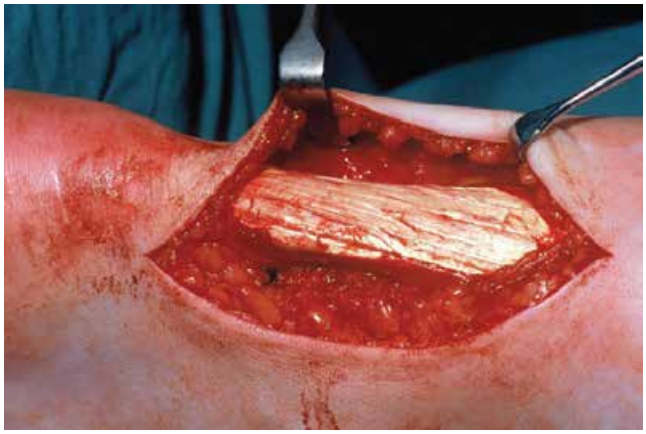


Figure 4: Appearance at the end of the procedure.

are serious problems. The paratenon is identified and incised. In patients with evidence of coexisting paratendinopathy, the scarred and thickened tissue is generally excised. Based on preoperative imaging studies, the tendon is incised sharply in line with the tendon fiber bundles. The tendinopathic tissue can be identified as it generally has lost its shiny appearance and it frequently contains disorganised fiber bundles that have more of a "crabmeat" appearance. This tissue is sharply excised (Figure 3). The remaining gap can be repaired using a side-to-side repair, but we leave it unsutured (Figure 4). If significant loss of tendon tissue occurs during the débridement, a tendon augmentation or transfer could be consider, even if we rarely undertake this additional procedure. Than the subcutaneous tissues is sutured with absorbable material, the skin edges are juxtaposed with Steri-Strips and a routine compressive bandage. The limb is immobilised in a below-knee synthetic weight-bearing cast with the foot plantigrade.

A period of initial splinting and crutch walking is generally used to allow pain and swelling to subside after surgery. After 14 days, the wound is inspected and motion exercises are initiated. The patient is encouraged to start daily active and passive ankle range-of-motion exercises. The use of a removable walker boot can be helpful during this phase. Weight bearing is not limited according to the degree of débridement needed at surgery, and early weight bearing is encouraged. However, extensive débridements and tendon transfers may require protected weight bearing for 4 to 6 weeks postoperatively. After 6 to 8 weeks more intensive strengthening exercises are started, gradually progressing to plyometrics and eventually running and jumping.

"The source of pain associated with mid-portion Achilles tendinopathy has not yet been clarified. The rationale behind the present management modality is that the sliding of the suture breaks the neo-vessels and the accompanying nerve supply decreasing pain."

Successful results have been reported with this surgical procedure. A systematic review of literature showed successful results in over 70% of cases[18], but these relatively high success rate are not always observed in clinical practice, probably because of the poor methods scores of many articles. Patients should be informed of the potential failure of the procedure, risk of wound complications, and at times prolonged recovery time[12]. Possible complications of this surgical procedure are wound healing problems, infection, sural nerve injury, rupture of AT and deep vein thrombosis.

Percutaneous Longitudinal tenotomies

Percutaneous longitudinal tenotomy can be used when there is no paratenon involvement and when the intratendinous lesion is less than 2.5 cm long. The procedure can be performed under ultrasound guide that is able to confirm the precise location of intratendinous lesions and produced similar results to open procedures[19].

Patients are operated as day cases. The patient lies prone on the operating table with the feet protruding beyond the edge, and the ankles resting on a sandbag. A bloodless field is not necessary. The tendon is accurately palpated, and the area of maximum swelling and/or tenderness marked,

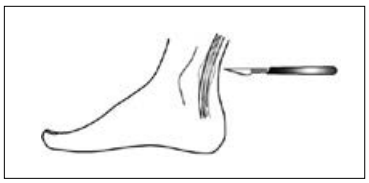


Figure 5: 11-scalpel blade inserted into the predetermined area with sharp edge pointing cranially.

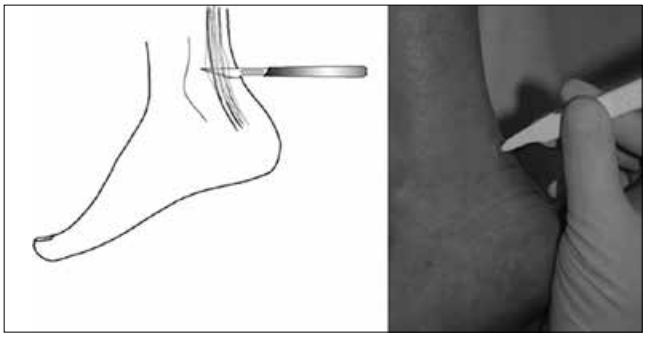


Figure 6: The blade penetrating the whole thickness of the Achilles tendon and a full passive ankle dorsi-flexion movement is produced.

and checked again by US scanning. The skin and the subcutaneous tissues over the Achilles tendon are infiltrated with 10–15 mL of plain 1% lignocaine (Lignocaine Hydrochloride, Evans Medical Ltd, Leatherhead, England). A number 11 surgical scalpel blade is inserted parallel to the long axis of the tendon fibres in the marked area in the center of the area of tendinopathy. The cutting edge of the blade points caudally and penetrates the whole thickness of the tendon (Figure 5). Keeping the blade still, a full passive ankle dorsi-flexion movement is produced (Figure 6). The scalpel blade is then retracted to the surface of the tendon, inclined 45° on the sagittal axis, and the blade is inserted medially through the original tenotomy (Figure 7). Keeping the blade still, a full passive ankle flexion is produced. The whole procedure is repeated inclining the blade 45° laterally to the original tenotomy, inserting it laterally through the original tenotomy. Keeping the blade still, a full passive ankle flexion is produced. The blade is then partially retracted to the posterior surface of the Achilles tendon, reversed 180°, so that its cutting edge now points cranially, and the whole procedure repeated, taking care to dorsiflex the ankle passively. Preliminary cadaveric studies showed that a tenotomy 2.8 cm long on average is thus obtained through a stab wound in the main body of the tendon[20]. A steristrip can be applied on the stab wound. The wound is dressed with cotton swabs, and a few layers of cotton wool and a crepe bandage are applied.

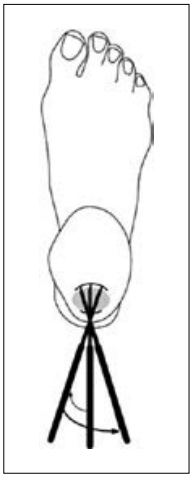


Figure 7: The procedure is repeated with blade inclining the 45° medial and 45° lateral to the original tenotomy.

After surgery early active dorsi- and plantar-flexion of the foot are encouraged. On the second postoperative day, patient are allowed to walk using elbow crutches weight-bearing as able, and weight-bearing are allowed after 2 or 3 days, when the bandage is reduced to a simple adhesive plaster over the wounds. Stationary bicycling and isometric, concentric and eccentric strengthening of the calf muscles are started under physiotherapy guidance after 4 weeks. Swimming and water running are encouraged from the second week. Gentle running is started 4–6 weeks after the procedure, and mileage gradually increased.

We reported excellent and good results in 63% of athletes with unilateral Achilles tendinopathy treated with ultrasound-guided percutaneous longitudinal tenotomy after

failure of conservative management, without any experiencing significant complications[19]. This technique is simple, can be performed on an outpatient basis, requires minimal follow-up care, it does not hinder further surgery if necessary and it should be considered in the management of chronic Achilles tendinopathy after failure of conservative management[20].

"Percutaneous longitudinal tenotomy can be used when there is no paratenon involvement and when the intratendinous lesion is less than 2.5 cm long."

Minimally invasive stripping for chronic Achilles tendinopathy

Four skin incisions are made. The first two incisions are 0.5 cm longitudinal incisions at the proximal origin of the AT, just medial and lateral to the origin of the tendon. The other two incisions are also 0.5 cm long and longitudinal, but 1 cm distal to the distal end of the tendon insertion on the calcaneus. A mosquito is inserted in the proximal incisions (Figure 8), and the Achilles tendon is freed of the peritendinous adhesions. A Number 1 un-mounted Ethibond (Ethicon, Somerville, NJ) suture thread is inserted proximally, passing through the two proximal incision (Figure 9). The Ethibond is retrieved from the distal incisions (Figure 10), over the posterior aspect of the Achilles tendon. Using a gentle see-saw motion the Ethibond suture thread is made to slide posterior to the tendon (Figure 11), which is stripped and freed from the fat of Kager's triangle.

The procedure is repeated for the posterior aspect of the AT. If necessary, using an 11 blade, longitudinal percutaneous tenotomies parallel to the tendon fibres are made. The subcutaneous and subcuticular tissues are closed in a routine fashion, and Mepore (Molnlycke Health Care, Gothenburg, Sweden) dressings are applied to the skin. A removable scotch cast support with Velcro straps can be applied if deemed necessary.

Post-operatively, patients are allowed to mobilise fully weight bearing. After 2 weeks patients start physiotherapy, focusing on proprioception, plantar-flexion of the ankle, inversion and eversion.

The source of pain associated with mid-portion Achilles tendinopathy has not yet been clarified. The rationale behind the present management modality is that the sliding of the suture breaks the neo-vessels and the accompanying nerve supply decreasing pain. Classically, open surgery has provided good results. However, wound complications can occur with these procedures. Minimal invasive technique reduce the risks

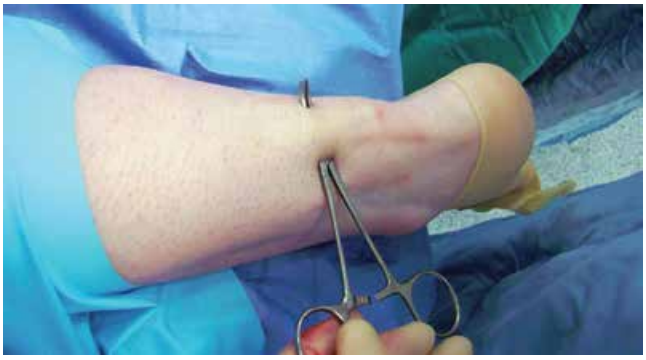


Figure 8: A mosquito is inserted in the proximal incisions.

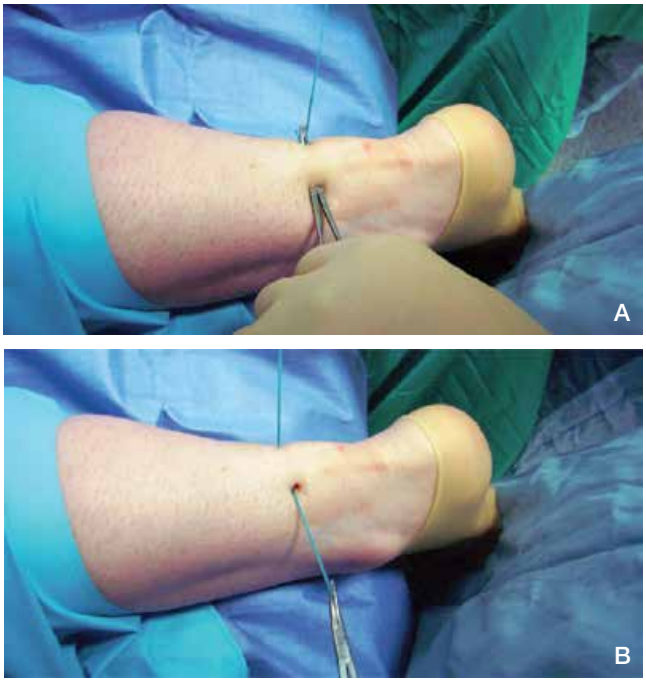


Figure 9: A Number 1 Ethibond (Ethicon, Somerville, NJ) is inserted proximally, passing through the two proximal incision over the anterior aspect of the Achilles tendon.

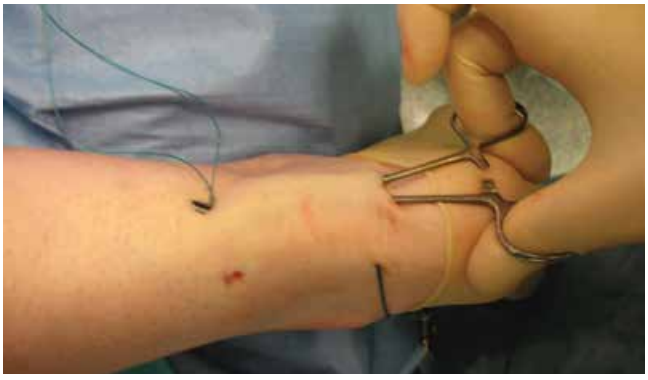


Figure 10: The Ethibond is retrieved from the distal incisions.

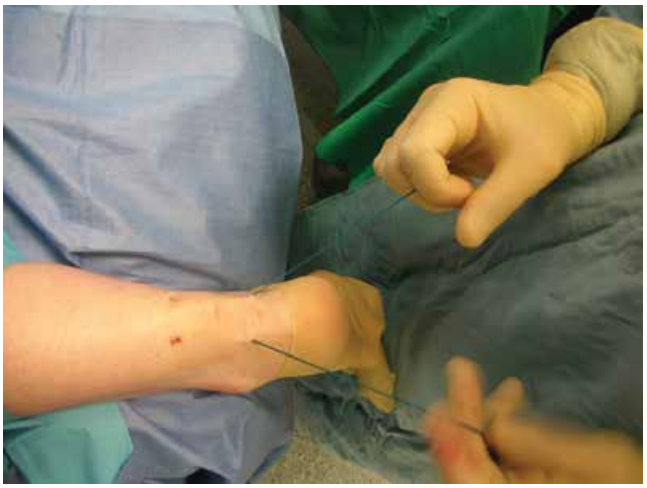


Figure 11: The Ethibond is slid over the anterior aspect of the Achilles tendon with a gentle see-saw motion. The whole process is repeated over the posterior aspect of the tendon.

of infection, is technically easy to master, and inexpensive. It may provide greater potential for the management of recalcitrant Achilles tendinopathy by breaking neo-vessels and the accompanying nerve supply to the tendon[21]. It can be associated with other minimally invasive procedures to optimise results.

"A period of initial splinting and crutch walking is generally used to allow pain and swelling to subside after surgery. After 14 days, the wound is inspected and motion exercises are initiated. The patient is encouraged to start daily active and passive ankle range-of-motion exercises."

Surgical treatment of insertional calcific Achilles tendinopathy

Insertional calcific tendinopathy (ICT) of the AT occurs in different patients populations, including young athletes and older, sedentary and overweight people. Usually, radiographs evidence ossification at the insertion of the Achilles tendon or a spur on the superior portion of the calcaneus. A recent study showed that calcification is present in more than 92% of chronic insertional Achilles tendinopathy[22] (Figure 12), and it is often associated with retrocalcaneal bursitis or Haglund's deformity. The incidence of insertional CT of the AT is not clear. It varies from 5% to the most common presentation in athletes; calcifications of the main body of the tendon are at best uncommon.

The aetiopathogenesis of ICT is still controversial. Rather than formed by precipitation of inorganic ions, it seems to be the results of an active cell-mediated process in which resident progenitor cells with multidifferentiation potential may play a determinant role[23].

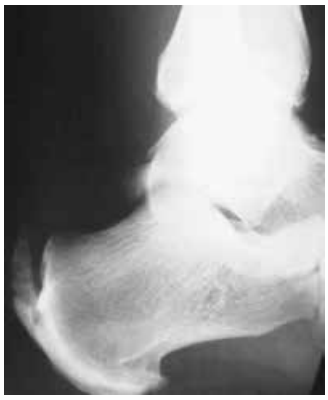


Figure 12: Insertional calcific tendinopathy of Achilles tendon.

The first line of management of ICT of AT is conservative. Eccentric exercises and extracorporeal shock wave therapy provided good results, but symptoms persist in 10% of patients and surgery is needed[24,25,26].

Surgical technique

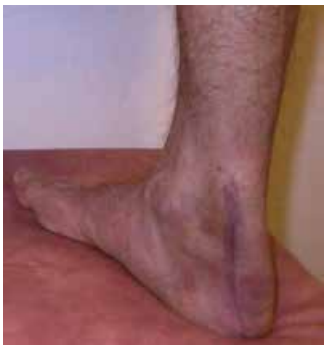


Figure 13: Well-healed incision on the medial side.

The patient is placed prone with the ankles clear of the operating table. A tourniquet is applied to the limb to be operated on. Under locoregional or general anesthesia, the Achilles tendon is exposed through a longitudinal incision that is 1 cm medial to the medial border of the tendon, and is extended from the lower one third of the tendon up to 2 cm distal to its calcaneal insertion (Figure 13). The incision can be extended transversely and laterally in a hockey-stick fashion, if necessary. Sharp dissection is continued to the paratenon, which is dissected from the tendon and excised, taking care to preserve the anterior fat in Kager's triangle and not to injure the mesotenon. The retrocalcaneal bursa is excised, if there is evidence of bursitis. The Achilles tendon is inspected for areas that had lost their normal shining appearance and palpated for areas of softening or thickening. The areas that have lost their normal shining appearance, and the areas that are softer or thicker are explored by way of one to three longitudinal tenotomies; areas of degeneration are excised. The area of calcific tendinopathy is identified, it is exposed starting from its proximal and medial aspect and it is excised from the

calcaneus. The Haglund tubercle may be removed with a saw and a burr or rasp is needed to ensure there are no prominent spicules of bone remaining. The tendon is reinserted in the calcaneus using bone anchors. Two bone anchors are used if 33% to 50% of the AT is disinserted; three bone anchors are used if 50% to 75% of the Achilles tendon is disinserted; four bone anchors are used if 75% or more of the Achilles tendon is disinserted (Figure 14); and five bone anchors are used if the Achilles tendon is disinserted totally. The AT is advanced in a proximal to distal fashion and reinserted in the calcaneum. We normally do not perform a tendon augmentation or a tendon transfer. After release of the tourniquet, hemostasis is achieved by diathermy. The wound is closed in layers using absorbable sutures. The skin wound is dressed with gauze, and sterile plaster wool is applied. A synthetic below-knee cast with the ankle plantigrade is applied.

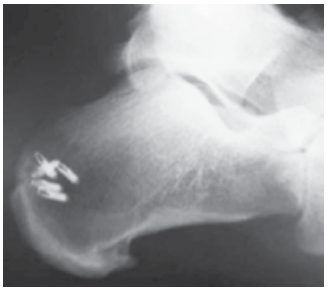


Figure 14: Postoperative result at 24 months from the operation. Five anchors were used.

Patients are discharged the day of surgery within 8 hours of the operation. Patients are mobilised with crutches and immediate weight bearing may be commenced after surgery. After two weeks the wound is inspected, the cast may be removed and patients may commence active plantar flexion, inversion and eversion exercise. A walker boot with the ankle in neutral can be used for 4 weeks. The patients are encouraged to continue to bear weight on the operated limb and to gradually progress to full weight bearing, if they are

not already doing so. Stationary cycling and swimming are recommended from the 2nd week after removal of the boot. We allowed return to gentle training 6 weeks after removal of the boot. Gradual progression to full sports activity at 20 to 24 weeks from the operation is planned according to the patients' progress. Resumption of competition depends on the patients' plans but is not recommended before 6 months after surgery.

We reported excellent and good results in 16 patients of 21 who underwent this surgical procedure[27]. They came back to their pre-injury level of activity at an average of 24.5 weeks following surgery. The remaining 25% patients did not experience problems in the activities of daily living but they could not return to their normal levels of sporting activity. We did not experience any case of traumatic dis-insertion of the reattached tendon. Johansson et al recently reported good outcome in 69% of patients, moderate in 25% and poor 6% of cases at an average follow-up of 35 months[28]. The authors also suggested that good outcome was related concomitant resection of a Haglund's deformity, even if this difference was not statistical significant (p=0.0613), probably due to the small size of the sample.

"Tendinopathy of the main body of AT affects both athletic and sedentary patients. The incidence in top-level runners has been estimated between 7 and 9%, while 30% of patients have a sedentary lifestyle."

Management of rupture in athletic vs non athletic patients

Acute AT rupture is a serious injury. Most (75%) acute ruptures occur during recreational activities in men between 30–40 year old, but 25% of ruptures take place in sedentary patients[29]. The incidence rate ranges from 6 to 18 per 100,000 per year[30]. Management of acute ruptures of the AT is still controversial. In conservatively managed patients, healing in a lengthened position may determine loss of calf muscle strength. In addition, incomplete healing of the gap between the two tendon stumps may contribute to the high re-rupture rate, up to 13%[31].

Operative management provides lower re-rupture rate, early functional treatment, less calf atrophy, and stronger push off than non-surgical treatment, at the expenses of long incisions, wound complications such as infections, and, occasionally, painful scars. But recent well conducted randomised controlled trials showed that conservative and open surgery management produce, in an unselected population, similar functional results[12]. Willits et al[33] showed acceptable and clinically similar outcomes of patients with acute Achilles tendon ruptures, who had been treated with accelerated functional rehabilitation alone compared with those who had received operative repair and accelerated functional rehabilitation, but may result in an higher re-rupture rate. A recent meta-analysis demonstrated that non-operative management using functional bracing with early mobilisation has similar re-rupture rates to open surgical treatment with regard to re-rupture rate, range of motion, calf circumference, and functional outcomes and it has the advantage of a decrease number in other complications[34]. The authors found that the risk of complications for surgically treated patients was 3.9 times that of non-surgically treated patients, which resulted in an absolute risk increase of 15.8%[34].

"In conclusion, HVIGI is effective to improve the symptoms of resistant Achilles tendinopathy. It is safe and relatively inexpensive, with the potential to offer an alternative management option before surgery, aiding a quicker return to sport."

Open, percutaneous, or minimally invasive procedures have been successfully used, especially in young and active subjects. Open surgery provides good strength to the repair, low re-rupture rates, and reliably good endurance and power to the gastrocnemius-Achilles tendon complex. However, open surgical approaches have resulted in high risk of infection and morbidity. Review articles and meta-analysis showed high costs and a 20-fold higher rate of complications than conservative treatment[35]. Therefore, minimally invasive procedures have been successfully used to avoid these complications[36]. Minimally invasive Achilles tendon repair provides many advantages. Major advantages are less iatrogenic damage to normal tissues, lower postoperative pain, accurate opposition of the tendon ends minimising surgical incisions and improved cosmesis. A recent systematic review reported a rate of superficial infections of 0.5% and 4.3% after minimally invasive and open surgery, respectively, and deep infections did not occur in subjects who had undergone minimally invasive repair[37]. The reason for this is that, when performing an open repair, the surgical trauma may add insult to injury. Shorter hospitalisation time and average time to return to working activities was also showed. But the main finding of the study was indications were grossly comparable and functional outcomes were not significantly different between minimally invasive and open surgery. Although sural nerve injury has been reported as a potential complication of this kind of surgery[29], new techniques have minimised the risk of sural nerve damage[38].

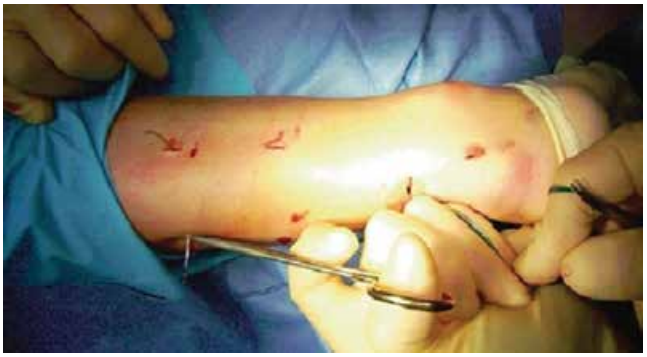


Figure 15: A 9 cm Mayo needle (BL059N, #B00 round-point spring eye, B Braun, Aesculap, Tuttlingen, Germany) is threaded with 2 double loops of number 1 Maxon (Tyco Healthcare, Norwalk, CT), and is passed transversely between the proximal stab incisions through the bulk of the tendon.

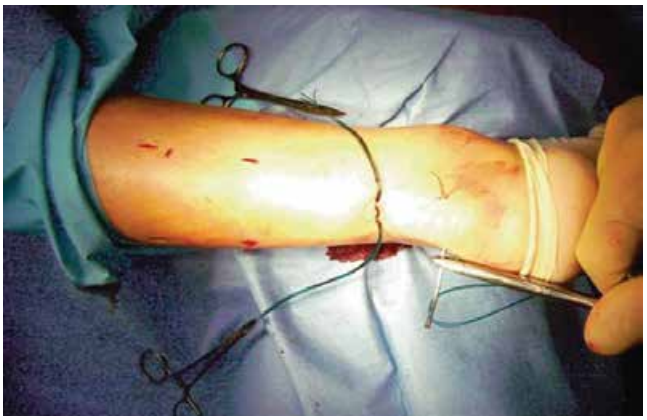


Figure 16: Another double loop of Maxon is passed between the distal stab incisions through the tendon.

Percutaneous Achilles Tendon Repair: surgical technique

A 1 cm transverse incision is made over the defect using a size 11 blade. Four longitudinal stab incisions are made lateral and medial to the tendon 6 cm proximal to the palpable defect. Two further longitudinal incisions on either side of the tendon are made 4–6 cm distal to the palpable defect. Forceps are then used to mobilise the tendon from beneath the subcutaneous tissues. A 9 cm Mayo needle (BL059N, #B00 round point spring eye, Braun, Aesculap, Tuttlingen, Germany) is threaded with two double loops of Number 1 Maxon (Tyco Healthcare, Norwalk, CT, USA), and this is passed transversely between the proximal stab incisions through the bulk of the tendon (Figure 15). The bulk of the tendon is surprisingly superficial. The loose ends are held with a clip. In turn, each of the ends is then passed distally from just proximal to the transverse Maxon passage through the bulk of the tendon to pass out of the diagonally opposing stab incision. A subsequent diagonal pass is then made to the transverse incision over the ruptured tendon. To prevent entanglement, both ends of the Maxon are held in separate clips. This suture is then tested for security by pulling with both ends of the Maxon distally. Another double loop of Maxon is then passed between the distal stab incisions through the tendon (Figure 16), and in turn through the tendon and out of the transverse incision starting distal to the transverse passage (Figure 17). The ankle is held in full plantar flexion, and in turn opposing ends of the Maxon thread are tied together with a double throw knot, and then three further throws before being buried using the forceps. A clip is used to hold the first throw of the lateral side to maintain the tension of the suture. We use 3-0 Vicryl (Ethicon) suture to close the transverse incision and Steri-strips (3M Health Care, St Paul, MN, USA) to close the stab incisions. A non-adherent dressing is applied. Apply a full plaster-of-Paris cast in the operating room with the ankle in physiologic equinus. Split the cast on both medial and lateral sides to allow for swelling. Patients are discharged on the same day of the operation.

We reported excellent results in 17 elite athletes after percutaneous surgical repair of Achilles tendon rupture[39]. All patients came back to competing at high level and the average time to return to full sport participation was 4.8 ± 0.9 months (range 3.2 to 6.5).

The choice of the type of management should take into account the age, occupation, and level of sporting activity of each patient. In elderly or a few selected patients, conservative

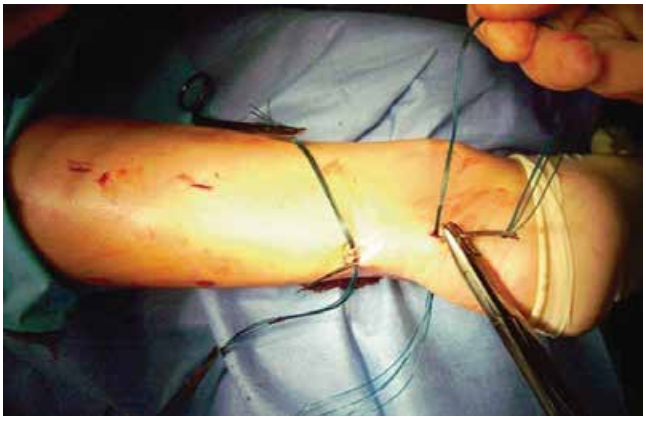


Figure 17: The needle is passed in turn through the tendon and out of the transverse incision starting distal to the transverse passage.

management and early mobilisation achieves excellent results but the associated re-rupture rate is not acceptable in young individuals. Open surgery is frequently associated with higher risk of superficial skin breakdown and wound problems, which can be prevented by performing percutaneous repair. Percutaneous repair followed by early functional rehabilitation, is becoming increasingly common, and may be considered in selected patients.

"Patients with chronic painful tendinopathy of the main body of Achilles tendon (AT) often present neovascularisation outside and inside the ventral part of the tendinopathic area. However, neovascularity in absence of pain is not necessarily pathological, and, in athletes, it can just indicate a physiological response to physical training."

Plantaris tendon injury

Injuries of the plantaris muscle and tendon, also known as "Tennis leg", was first described in 1883 by Powell[40]. The plantaris muscle arises from the supracondylar ridge of the lateral femoral condyle and courses medially as it progresses down the leg to its insertion on the calcaneus, just medial to the Achilles tendon. It runs between the overlying gastrocnemius and the deeper soleus. The plantaris muscle is an accessory plantar flexor of the foot. It has been proposed to assist the larger plantar flexors of the foot with proprioception due to its high density of muscle spindles and it is absent in approximately 7–10% of the population[41].

Controversy exists regarding the existence of plantaris tears. Early descriptions of plantaris tears were later disputed, with several investigators questioning their existence[42]. The advent of modern imaging technology along with surgically documented lesions confirmed these entities[41]. More recently, most investigators have implicated a rupture of the medial head of the gastrocnemius muscle at the musculotendinous junction in the pathogenesis of injury. Actually "Tennis leg" is a clinical entity that is variably attributed to a partial tear or rupture of the gastrocnemius, a rupture of the plantaris tendon or at the musculotendinous junction, or of the soleus muscle, where gastrocnemius muscle is the most frequently involved[43]. A series of 141 patients referred for ultrasound examination after calf strain revealed that 67% had gastrocnemius tears, 21% had hematoma and fluid accumulation but no clear muscle tear, 1.4% had plantaris rupture, and 0.7% had isolated soleus tear[44]. Plantaris injuries may occur after ankle dorsiflexion while the knee is extended[45]. Injuries to the plantaris or soleus have been described during running, tennis and volleyball[46]. Clinically, it may be difficult to distinguish among soleus, plantaris, and gastrocnemius injuries. US and MRI are useful imaging techniques for diagnosis[43]. Even if an isolated tear of the plantaris tendon or muscle is an uncommon condition, its importance lies in the fact that its rupture can mimic different conditions, such as a proximal Achilles tendon tear or deep vein thrombosis (DVT)[47,48]. DVT can occur in 9.9% cases of so called Tennis leg[44]. For this reason, some authors use US and Doppler US to rule out differential symptoms, such as DVT[46]. Furthermore, plantaris tendon can provide plantar flexion of the ankle after AT rupture presenting a confusing picture.

Treatment is mostly conservative. Rest, ice, compression and elevate of the affected leg showed significant reduction in pain and swelling. Unfortunately there is a paucity of good quality data on these uncommon injuries, so firm evidence-based recommendations are not yet available.

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References, as indicated within the article, are available at sma.org.au/publications/sport-health

Sustainability in business

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Does the business you own or work in have an environmental policy? If not, perhaps it's time to take a close look at your daily practice, services and supplier engagement to know if you are acting with environmental sustainability in mind.

An environmental policy represents the general position on environmental issues and the policies and practices you will apply in conducting your business sustainably. It's a commitment to minimising your environmental impacts of your business operations. It's your responsibility to manage the social and environmental impacts of your business.

This responsibility may include:

- Making sure you understand the impact you have on the environment.
- Finding new ways to reduce your impact.
- Considering the environmental implications of any new decisions you make.
- Complying with legislation, local bylaws, and any formal government policies.
- Keeping your staff and contractors up to date with all of your environmental policies.
- Making sure your staff and contractors have the training they need.
- Constantly monitoring, reviewing and reporting on your processes.

Some of the commitments you may adhere to are:

- Driving a culture of continuous environmental improvement with a focus on fuel and energy efficiency.
- Actively pursuing renewable or lower carbon fuels and energy sources.
- Understanding and managing your environmental risks with the goal of minimising or eliminating those risks.
- Ensuring the environment is considered in your investment and corporate strategies, procurement and the products and services you offer.
- Actively working to minimise pollution, manage waste streams and address relevant biodiversity issues.
- Engaging with your customers, employees and shareholder on environmental issues and transparently reporting on your environmental performance.
- Complying with all applicable legal and regulatory requirements.

A sound environmental performance plan contributes to your competitive strength and can only benefit your customers, employees and the wider community.

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Top tips for live-tweeting

With an array of conferences and events on offer this year for sports medicine professionals, live-tweeting is an effective and easy way to promote your research, organisation or institution's online presence. If done well, live-tweeting will also generate meaningful and engaging content for your Twitter account.

Whether you're hosting or just attending a conference or event, master the art of live-tweeting with the following tips:

Find the event hashtag and use it

Using a hashtag allows you to group all of your tweets, enabling people searching for the conference or event hashtag to find them. Dedicated event hashtags also make it clear to your followers that you are tweeting about a particular event, rather than just spouting off random sound bites. If there isn't already an event hashtag in place, make one up. Just keep it short as every character counts!

Tag speakers and companies in your tweets

This will not only make your presence known to those participating, but also increase your chances of being retweeted.

Engage with other live-tweeters

Use the hashtag to search for others who are tweeting and start a conversation. From there, you can always ask to take the conversation offline.

Don't over-tweet

While the conference may be providing an endless supply of content and grabs, over-tweeting can overwhelm your regular followers who may not appreciate a barrage of out of context tweets. While there is no optimal amount of tweets, you should decide where the balance lies between over-dominating the conversation and not contributing enough.

Take pictures and incorporate them into your tweets

Tweets with Twitter pictures are 94% more likely to be retweeted[1] so use that camera.

Finally, remember to bring your smartphone, tablet or laptop to the event and make sure they are fully charged!

A version of this article originally appeared on Sustainability Consult's blog[2], www.sustainabilityconsult.blogspot.co.uk

1 Social News Daily, http://socialnewsdaily.com/17558/tweets-with-twitter-images-are-94-more-likely-to-be-retweeted-study-finds/
2 Sustainability Consult, http://www.sustainabilityconsult.blogspot.co.uk/2014/01/five-tips-for-live-tweeting.html

A Family Superannuation Fund

Brought to you by Steven Skoglund, Director, Davidsons Accountants and Business Consultants

The popularity of Self-managed Superannuation Funds (SMSFs) is continuing to grow with the attraction being a main focus on the ability to have:

- Direct control over investment decisions;
- Alternative investment strategies; and
- Flexible retirement income streams.

SMSFs can provide an effective means for the family (i.e parents, children, grandchildren) to invest together, accumulating family wealth for the whole family, not just for the parents as they approach retirement.

With recent amendments to the Superannuation Regulations it is now a requirement that employers contribute 9.25% of an employee's wages into a complying superannuation fund. The contribution rates are set to increase to 12% by 1 July 2019, meaning funds in superannuation will increase.

However younger family members of generation "X" and "Y" cannot access their superannuation until at least 60 years of age. As this is more than 30 to 40 years away, superannuation is generally of little interest to the younger population.

Consider a SMSF as being a ***Family Superannuation Fund***.

Structured correctly, a Family Superannuation Fund can provide significant benefits to the whole family such as:

- Joint investments: by combining funds and achieving economies of scale;
- Investment Education: Younger family members can learn about equity markets, interest rates, property etc;
- Family asset protection – a cost effective manner of providing insurance for all the members.

With the right investment strategy and a long term investment outlook, the younger generation members could become ***Superannuation Millionaires*** by retirement.

As part of the Family Superannuation Fund strategy, consideration would need to be given to a ***Superannuation Succession Plan***, aligned with the members' Wills and overall estate planning strategy.

Such a plan allows for family wealth to transition between family members in a tax and cost effective manner.

Actions

If the idea of a ***Family Superannuation Fund*** appeals to you, contact Steven Skoglund or any of the Superannuation team at Davidsons on 03 5221 8190.

Ensure your Self-managed Superannuation Fund has a functional ***Superannuation Succession Plan***. Davidsons Superannuation team can advise you on the plan development process.

Davidsons Accountants and Business Consultants specialised service offerings of Tax, Audit, Financial Services, Personal Insurances and Self-Managed Superannuation can provide you with all the information, assistance and advice regarding your superannuation requirements. For more information visit www.davidsons.com.au or follow Davidsons on Facebook and Twitter.

Winter wellness

With the mornings becoming cooler and days shorter, now is a good time to refresh the latest nutrition information to help you keep your athletes fit and healthy over the winter months. Alison Garth, Advanced Sports Dietitian – Sports Dietitian Australia (SDA) gives you the run down.

Vitamin D

The "sunshine vitamin" is most well known for its role in bone health through calcium and phosphorus homeostasis, and it is now increasingly being accepted as having an important role in immune function in the body, particularly T-cell mediated immunity. It is commonly recognised that athletes who predominately train indoors are at high risk of Vitamin D deficiency. As we head in to the winter months, it's a timely reminder that even athletes who train and compete outdoors can be at risk of Vitamin D deficiency (especially those living in the southern states of Australia where the latitude limits UVB exposure and therefore Vitamin D synthesis).

There is still some debate internationally as to the "optimal" level of Vitamin D however, the 2013 Position Statement *Vitamin D and health in adults in Australia and New Zealand* suggests that for optimal musculoskeletal health, a serum 25-OHD level of 50 nmol/L at the end of winter (10–20 nmol/L higher at the end of summer, to allow for seasonal decrease) is required. Recommended sunlight exposure during winter for athletes living in the north of Australia (e.g. Cairns) is ~9–12 minutes/day, in Brisbane or Perth is ~15–19 minutes/day and in the southern states is between 26–47 minutes/day.

"It is commonly recognised that athletes who predominately train indoors are at high risk of Vitamin D deficiency. As we head in to the winter months, it's a timely reminder that even athletes who train and compete outdoors can be at risk of Vitamin D deficiency…"

Dietary sources of Vitamin D are limited yet small amounts can be found in oily fish, egg yolks, mushrooms and fortified milks and margarine. Given the difficulty in obtaining Vitamin D from the sun and diet during the winter months, supplementation with Vitamin D3 may be required for athletes who are Vitamin D deficient. An Accredited Sports Dietitian can work closely with Sports Physicians in identifying and managing athletes with Vitamin D deficiency.

Probiotics

Probiotics are live microorganisms that have been shown to have beneficial effects on intestinal health and immune function. The most common strains of commercially available probiotics are lactobacillus acidophilis and bifidobacterium bifidum – found in foods such as yoghurt and Yakult™ as well as specialised supplements. A recent Cochrane review reports that prophylactic supplementation of probiotics is beneficial in preventing acute upper respiratory tract infections. Similarly, research conducted at the Australian Institute of Sport (AIS) has found favourable reductions in the number of symptom days and symptom severity of respiratory tract infections in athletes. Although there is no definitive recommendation for the dose of probiotic supplementation in athletes, AIS studies show that most athletes will safely tolerate dosages of two billion bacteria per day – although some athletes may benefit from a staggered increase in dosage over one to two weeks to minimise any gastrointestinal side effects. For those athletes looking for a natural source of probiotics, research by Gleeson et al (2011) found favourable benefits, mainly reduced frequency of upper respiratory tract infections, in athletes consuming a daily dose of Yakult™ over a 16 week winter training period. Importantly, prophylactic supplementation must commence at least 14 days prior travel, competition or elevated training loads to allow for colonisation of gut bacteria.

Zinc

Interest in zinc comes from its ability to inhibit replication of rhinovirus, the most frequent cause of the common cold. A 2013 Cochrane review of 18 randomised control trials found that taking zinc lozenges or syrup within 24 hours of the onset of symptoms reduced the duration of the common cold. Furthermore those who took zinc were less likely to have symptoms persist beyond seven days of treatment. However, although there are apparent benefits to zinc supplementation, variability in study designs make it difficult to draw conclusions on the optimal dose, duration and formulation of supplementation to ameliorate symptoms of the common cold. There is some evidence to suggest that five months of prophylactic zinc supplementation produced favourable benefits for children, yet there is currently no research to support this in adults, nor athletes. As a side note, foods high in zinc include seafood (especially oysters), red meat (especially liver), cereal products and baked beans.

"Interest in zinc comes from its ability to inhibit replication of rhinovirus, the most frequent cause of the common cold."

Vitamin C

Despite its popularity, there is little evidence to support the use of high doses of Vitamin C for preventing incidence of the common cold. While some research suggests that Vitamin C (with or without zinc) may have favourable effects on reducing the duration of cold symptoms, there is insufficient evidence to currently make recommendations on the optimal dose and duration of supplementation to benefit recovery from the common cold. There is also a growing body of evidence to indicate that chronic supplementation of antioxidants can actually be detrimental for performance in athletes suggesting that a diet rich in fruits and vegetables, that provides a variety of nutrients, may be more beneficial (and less harmful) than supplementing one nutrient in isolation.

"Despite its popularity, there is little evidence to support the use of high doses of Vitamin C for preventing incidence of the common cold."

Iron

There is research to suggest that low iron levels in athletes are associated with reduced immune function, leaving them more susceptible to colds and flu (particularly during heavy training phases). Athletes, particularly females, should be encouraged to include iron containing foods regularly as part of a balanced diet. Great iron rich options are lean meats, eggs, legumes, almonds, tofu, dried apricots, fortified breakfast cereals and other products with added iron such as Milo™ or Sustagen Sport™. It would also be worthwhile screening "at risk" athletes for iron deficiency and supplementing as appropriate to further support immune function.

"Great iron rich options are lean meats, eggs, legumes, almonds, tofu, dried apricots, fortified breakfast cereals and other products with added iron such as Milo™ or Sustagen Sport™."

Carbohydrates

There is good evidence to support the role of carbohydrates in counteracting the immune suppressive effects of exercise by dampening the rise in cortisol and other stress hormones. It's important that during heavy training phases – particularly during the winter months when colds are more prevalent – athletes are mindful of their carbohydrate intake around exercise.

"It's important that during heavy training phases – particularly during the winter months when colds are more prevalent – athletes are mindful of their carbohydrate intake around exercise."

So there you have it, a quick snapshot of the latest information on the role of nutrition in immune health. Next time you're working with an athlete or team prone to colds and other winter bugs link them in with an Accredited Sports Dietitian nearby to help ensure they stay healthy and on the training track.

Follow this link to find an Accredited Sports Dietitian near you
http://www.sportsdietitians.com.au/findasportsdietitian

Further Reading

Cox A et al. Oral administration of the probiotic Lactobacillus fermentum VRI-003 and mucosal immunity in endurance athletes. Br J Sports Med. 2010; 44:222–226

Gleeson M. Immune function in sport and exercise. J Appl Physiol. 2007; 103:693–699

Hao Q et al. Probiotics for preventing acute upper respiratory tract infections. Cochrane Database of Systematic Reviews 2011, Issue 9. Art. No.: CD006895. DOI: 10.1002/14651858.CD006895.pub2

Hemila H & Chalker E. Vitamin C for preventing and treating the common cold. Cochrane Database of Systematic Reviews 2013. Issue 1. Art. No. CD000983. DOI: 10.1002/14651858.CD000980.pub4

Nowson CA et al. Vitamin D and health in adults in Australia and New Zealand: a position statement. Med J Aust. 2012; 196 (11): 686–687

Singh M & Das RR. Zinc for the common cold. Cochrane Database of Systematic Reviews 2013. Issue 6. Art. No.: CD001364. DOI 10.1002/14651858.CD001364.pub4

West N et al. Lactobacillus fermentum (PCC®) supplementation and gastrointestinal and respiratory-tract illness symptoms: a randomised control trial in athlete. Nutrition Journal. 2011; 10:30

Gleeson M, Bishop NC, Oliveira M & Tauler P. Daily probiotic's (Lactobacillus casei Shirota) reduction of infection incidence in athletes. Int J Sport Nutr Exerc Metab. 2011 Feb; 21(1): 55–64

It's not just the size that counts, but how you use it

Winner of the "ASICS best paper" at last year's Australian Sports Medicine Federation Fellows Awards, Dr Adam Semciw, APAM, provides an article derived from his research into the anterior gluteus minimus.

Gluteus minimus (GMin) is the smallest of the three gluteal muscles. It is a fan-shaped muscle that originates from the ilium, between the anterior and inferior gluteal lines. The distal insertion of GMin attaches by means of a tendon into the apex and anterior aspect of the greater trochanter[1]. Some muscle fibres have also been observed to attach into the anterior superior hip joint capsule[2]. The broad attachments of GMin have led researchers to describe at least two uniquely oriented segments within the muscle: the fascicles of the anterior and posterior segments are oriented vertically and posteriorly respectively [3] (Figure 1A), and arranged parallel to the neck of femur[4] (Figure 1B).

As a whole, GMin is theoretically considered an important femoral head stabiliser[5]. The parallel arrangement of fibres (coronal plane) to the neck of femur seemingly enables them to draw the head of femur into the acetabulum, providing stability for the hip joint (Figure 1B). Yet the unique segmental arrangement of fibres in the sagittal plane (Figure 1A), and the additional capsular attachment of the anterior segment[6] have led researchers and clinicians to attribute independent stabilising roles for each segment[7].

"In summary, current EMG evidence adds to existing biomechanical, cadaveric and radiological data to suggest that 'how we use' anterior GMin is important. By activating in positions of hip joint extension, anterior GMin is proposed to have a vital role in anterior hip joint stability."

Segmental response to pathology

Magnetic resonance imaging (MRI) studies have provided valuable insights into the association between GMin muscle size and joint pathology. Atrophy of GMin has been identified in people with lateral hip pain[8] and advanced hip osteoarthritis[9]. Fatty infiltration of GMin has also been observed one year post-total hip arthroplasty (THA)[10]. Furthermore, specific fatty atrophy of the anterior segment of GMin has been noted following THA[11] and in a sample of elderly cadavers[12]. From these studies, it is clear that GMin muscle size is an important component of joint health. It is also clear that some conditions appear to detrimentally affect anterior GMin muscle size specifically—supporting the notion of functionally independent segments. These imaging and cadaveric studies of muscle size are limited, however, in their ability to identify the function of GMin. For example, MRI studies (including functional MRI) do not provide us with clinically meaningful information regarding real-time temporal activation properties. In other words, they don't tell us "how we use it". How does GMin behave throughout the gait cycle? Is GMin activity delayed in clinical populations? If so, can "normal" temporal function be restored with targeted rehabilitation? These questions cannot be answered with MRI or real-time ultrasound. Electromyography (EMG) remains the best method for investigating muscle function and attempting to answer these clinically meaningful questions.

"Where clinicians suspect anterior hip instability, secondary to anterior GMin dysfunction, there are a number of strengthening techniques that could be prescribed for targeted rehabilitation."

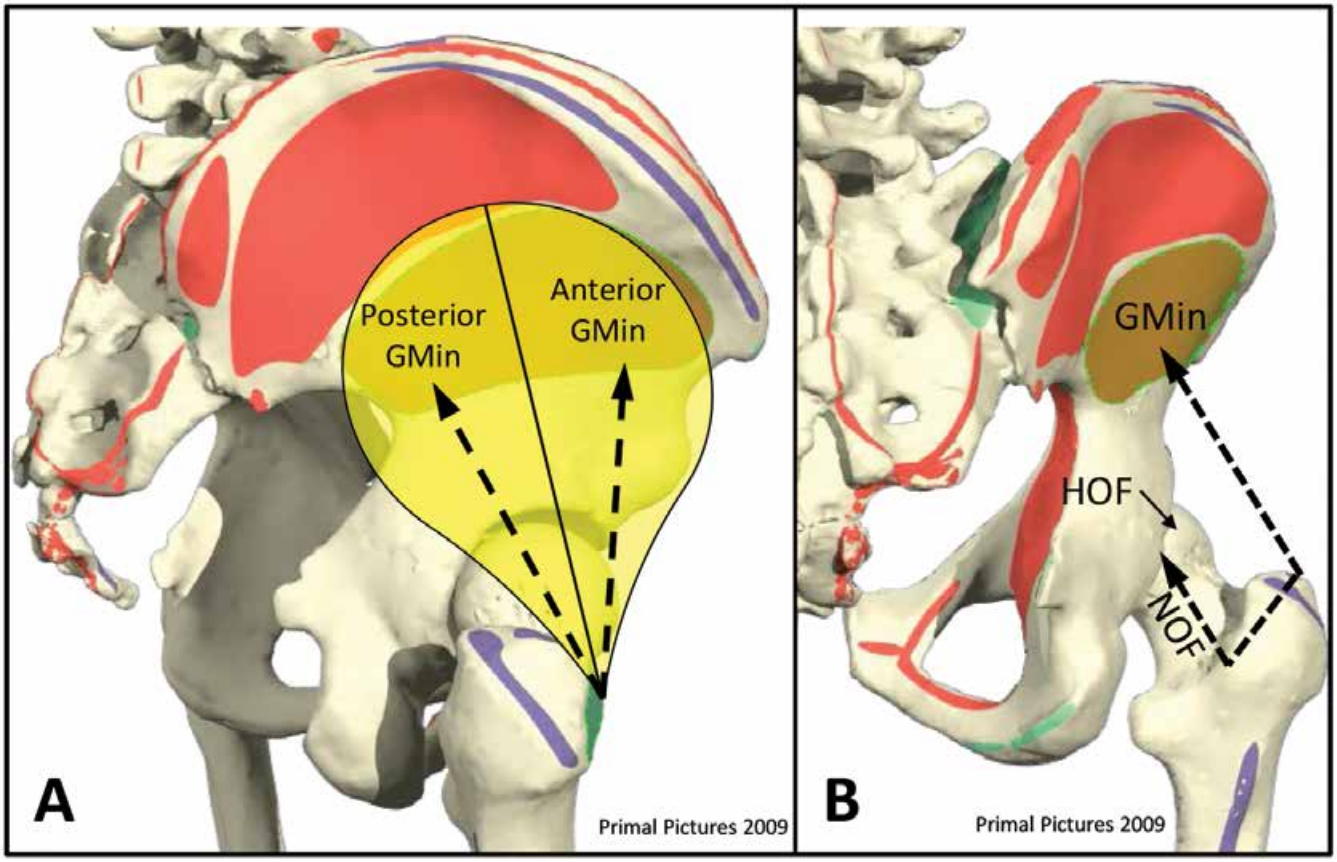


Figure 1: Illustration of gluteus minimus (GMin) fascicle orientation. A: Lateral view of the pelvis, illustrating the anterior and posterior fascicles oriented vertically and posteriorly respectively, in the sagittal plane. B: Posterior view of the pelvis, illustrating the parallel orientation of GMin fascicles to the neck of femur (NOF) in the coronal plane. This orientation would enable GMin to pull the head of femur (HOF) into the acetabulum, facilitating femoral head stability (3D anatomy images. Copyright of Primal Pictures Ltd. primal pictures.com).

Function

Until our recent EMG investigation of GMin[13], there was only one other study conducted, and this was published over 30 years ago using now out-dated processing and analysis techniques. The lack of EMG research into GMin is primarily because it is deeply located, and requires fine-wire EMG investigation—the superior gluteal neurovascular bundle (NVB) provides a further technical challenge in accessing this muscle with EMG electrodes[14]. As a side note, clinicians keen on dry needling GMin should also consider the implications of the NVB. Our results suggest that anterior and posterior GMin are indeed functionally independent[15]. For instance, during a maximally resisted clam manoeuvre, posterior GMin is active at moderate levels (48 per cent of maximum), while anterior GMin is active at low levels (11 per cent of maximum). Therefore, two structurally unique segments are contracting at largely different relative intensities for a given task. We have also shown that this independent function translates into dynamic activities. When walking at comfortable self-selected speed, posterior GMin EMG activity peaks early in stance, while anterior GMin activity peaks significantly later, in mid-to-late stance (Figure 2)[16]. Each segment can therefore be functionally defined by "how we use it".

"Recently, we presented observations from a sample of elite swimmers that revealed additional activity of anterior GMin early in gait when compared with control participants. This was hypothesised to occur because of chronic anterior GMin lengthening, which placed it in a mechanical disadvantage, and thus requiring greater muscle activity to generate tension for a given task."

The later peak of anterior GMin in stance is a unique property not shared by any other gluteal synergist. It is the only gluteal segment that tends to peak in activity as the hip joint extends throughout stance[17]. The functional importance of this role is potentially clarified in light of cadaveric and biomechanical studies that describe large joint forces transmitted by the head of femur onto the anterolateral hip joint capsule and labrum in positions of hip joint extension[18]. Therefore, the later peak of

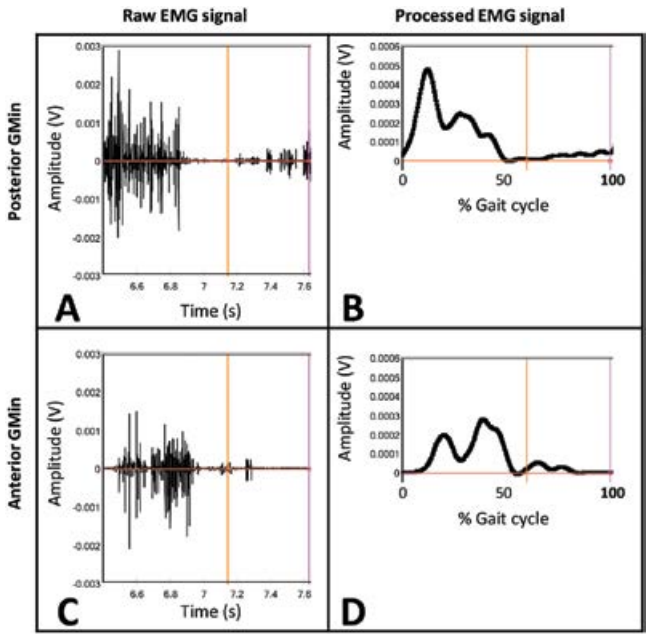


Figure 2: Illustration of gluteus minimus (GMin) EMG activity during one full stride (heel strike to ipsilateral heel strike). Top panels represent raw (A) and processed (B) EMG of posterior GMin. Bottom panels represent raw (C) and processed (D) EMG of anterior GMin. Orange vertical line indicates toe-off. Greater intensity EMG signals are demonstrated early in stance for posterior GMin (panels A and B), and later in stance for anterior GMin (panels C and D).

anterior GMin EMG activity in stance (Figure 2), may reflect a specific role in neutralising these large forces—much like the well-documented role of subscapularis in providing anterior shoulder stability[19]. Consequently, a weak or inefficient anterior GMin could leave the anterior hip joint capsule and labrum vulnerable to shearing and injury. This could manifest in positions of hip extension—for example, when taking large strides, lunging onto a step or running.

"While we have a better understanding of the role of GMin in healthy, pain-free individuals, there remains a paucity of information on how GMin might function in pathological conditions."

What does this mean for clinical practice?

While we have a better understanding of the role of GMin in healthy, pain-free individuals, there remains a paucity of information on how GMin might function in pathological conditions. This void will fill exponentially over the coming years, however, what follows are some potential clinical findings that may lead clinicians to consider dysfunction of anterior GMin.

"As a side note, clinicians keen on dry needling GMin should also consider the implications of the NVB. Our results suggest that anterior and posterior GMin are indeed functionally independent."

Posture

Standing "sway-back" posture may indicate chronically lengthened anterior hip joint stabilisers[20]. Typically, this is associated with a lengthened and inefficient iliopsoas[21]. However, one can speculate that this also reflects a lengthened and inefficient anterior GMin. For example, sway-back is a commonly observed posture in elite swimmers[22]. Recently, we presented observations from a sample of elite swimmers that revealed additional activity of anterior GMin early in gait when compared with control participants[23]. This was hypothesised to occur because of chronic anterior GMin lengthening, which placed it in a mechanical disadvantage, and thus requiring greater muscle activity to generate tension for a given task.

"These imaging and cadaveric studies of muscle size are limited, however, in their ability to identify the function of GMin."

Manual muscle tests

Anterior GMin is highly active in isometric internal rotation, and abduction in the anatomical position[24]. Poor strength in these positions could reflect anterior GMin weakness, although clinicians cannot rule out compensatory changes in synergistic abductor, or internal rotator, muscle activity.

"Magnetic resonance imaging (MRI) studies have provided valuable insights into the association between GMin muscle size and joint pathology. Atrophy of GMin has been identified in people with lateral hip pain and advanced hip osteoarthritis."

Active prone hip extension

Passive anterior hip apprehension testing[25] is typically performed to assess the passive stability of the anterior hip joint. However, an active prone hip extension may test the patient's ability to actively stabilise the anterior hip joint (Wisbey-Roth, personal communication). Pain anteriorly

Figure 3: Photograph of a single-leg bridge. The stance leg (right leg) is required to generate a hip internal rotation torque in order to maintain a neutral pelvis in the transverse plane. This is a moderate to advanced exercise.

during this manoeuvre could indicate an irritable anterolateral labrum—a secondary manifestation of anterior GMin's inability to stabilise the head of femur in the acetabulum.

Where clinicians suspect anterior hip instability, secondary to anterior GMin dysfunction, there are a number of strengthening techniques that could be prescribed for targeted rehabilitation. The ultimate aim of rehabilitation should be to strengthen anterior GMin (a hip internal rotator) in positions of hip joint extension. One suggestion is the single-leg bridge (Figure 3); however, clinicians can be clever with resistance tubing (wrapped around the knee) in order to encourage hip internal rotation in functional, closed-chain tasks. Regardless of which exercise is selected, clinicians should be careful not to reproduce their clients' symptoms during the exercise. It is suggested that clients begin in a position of hip flexion, and progress into greater degrees of hip joint extension.

"Electromyography (EMG) remains the best method for investigating muscle function and attempting to answer these clinically meaningful questions."

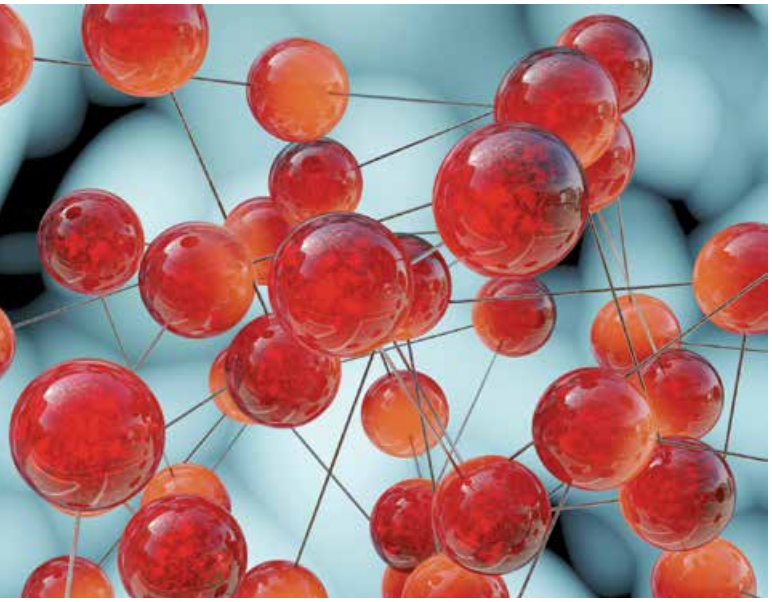
Summary

In summary, current EMG evidence adds to existing biomechanical, cadaveric and radiological data to suggest that "how we use" anterior GMin is important. By activating in positions of hip joint extension, anterior GMin is proposed to have a vital role in anterior hip joint stability. This article has provided some preliminary clinical observations that may lead clinicians to suspect anterior GMin dysfunction, and offered a potential goal for rehabilitation. Further work is currently underway to evaluate anterior GMin dysfunction in pathological conditions, and evaluate the most effective rehabilitation protocols.

Adam Semciw currently works as a physiotherapist in private practice and is a lecturer at La Trobe University, Melbourne. His research and clinical interests revolve around hip and lower limb muscle function in health and disease. If you would like to be involved in a related clinical research topic, contact Adam and his team at a.semciw@latrobe.edu.au.
The author would like to thank Dr Tania Pizzari and Trish Wisbey-Roth for assistance with this article, and acknowledge the support of the Sports Medicine Australia Research Foundation Grant (awarded in 2010) for funding components of this research.

References, as indicated within the article, are available at sma.org.au/publications/sport-health

Profile: SMA Research Foundation Grants



The SMA Research Foundation Grants program was established to provide support to early career postgraduate researchers engaged in sports medicine and disease prevention research. With 2014 applications opening soon, *Sport Health* caught up with Sarah Warby, PhD Researcher at La Trobe University and 2013 SMA Research Foundation Grant recipient, to see how her research project is travelling.

What did you receive the SMA Research Foundation Grant for?

I received the SMA Research Foundation Grant for my PhD research project titled "The effect of exercise based management on multidirectional instability of the shoulder: A pilot randomised controlled trial".

Can you explain your research in practical terms?

In conjunction with Melbourne based shoulder physiotherapist, Lyn Watson and LifeCare clinics of Victoria, we are investigating the effect of two different rehabilitation programs: The Lyn Watson Instability Program and the Rockwood Instability Program, on patients with multidirectional instability (MDI) of the shoulder. Participants will be randomised to one of the two programs. Both programs involve physiotherapy delivery and progression of an exercise program for twelve weeks. Both programs have been shown to be effective for patients with MDI however we aim to establish which one is most effective. Outcome measures will include the Melbourne Instability Shoulder Score (MISS), the Western Ontario Shoulder Index (WOSI), muscle strength scores and patient satisfaction scores. This research will establish evidence-based guidelines for practitioners to use when treating MDI with exercise.

What are you hoping to find?

We hypothesise that the Lyn Watson program will have statistically better outcomes than the Rockwood program due to its focus on scapula control. This hypothesis will be tested in an unbiased manner with assessors and patients both blinded to the treatment they are receiving.

What has been the biggest challenge so far?

The biggest challenge so far is finding participants who have clinically diagnosed MDI and who are willing to participate in the study. Although MDI it is becoming more recognised in the clinical setting, it is not the most common shoulder pathology, therefore finding patients with MDI can be challenging.

How far advanced is the trial?

We currently have seven participants in the trial since it officially commenced in January 2014.

What difference has the SMA Research Foundation Grant made to your research?

The SMA Grant has been imperative to fund the cost of the MRIs that participants must have to exclude a structural lesion of the shoulder and to be included in the trial. This has enabled us to screen and include participants at a faster rate than if we had not had the funding.

What advice would you give to other young and emerging researchers considering applying for an SMA Research Grant?

Have a research question that is of current interest to the sports medicine world, especially in the clinical setting. Try and have clear aims of your research with patient specific outcome measures and preferably an area that needs more research.

Assisting with recruitment

I would like to invite any practitioner who is interested in referring an MDI patient into the trial to please contact me on 0478405258 or sarahannwarby@gmail.com. Patients have the chance to receive free physiotherapy for 12 weeks with some of Melbourne's most established shoulder physiotherapists. The trial will be recruiting patients in 2014 and 2015.

Snapshot: SMA Research Foundation Grants

Applications opening soon

SMA Research Foundation Grants, up to the value of $2,000, are designed to support research conducted by postgraduate students and postgraduate practitioners for the purpose of advancing research in sports medicine and its relationship with disease prevention. Grants are administered by the Sports Medicine Australia Research Foundation Board. A finite number of grants will be issued in a regular allocation period.

Eligibility

Applicants for SMA Research Grants must be a professional or student member of Sports Medicine Australia. Preference will be given to new and emerging researchers* conducting research related to the SMA discipline fields (i.e. Sports Dietitian, Sports Doctor, Sports Physician, Sports Physiotherapist, Sports Podiatrist, Sports Psychologist, Sports Scientist).

* new and emerging researchers with no more than 3 years post-doctoral at the time of submitting their application

Selection criteria

Applications will be judged on:

- The relevance of their research to sports medicine, and disease prevention.
- Benefits gained from the research to the individual, the profession, and Sports Medicine Australia.
- Value for money.

How to apply

Applications will open mid-year so keep an eye on the SMA website sma.org.au and keep reading the monthly Member E-news.

Looking back: 2013 Grants

Katrin Dias

University of Queensland

Effects of exercise intensity on myocardial and arterial function, and intra-adominal fat in obese children and adolescents.

Jackson Fyfe

Victoria University

The effect of prior endurance exercise intensity on the acute molecular responses to subsequent resistance exercise.

Jamie Gaida

Monash University

Achilles tendon and plantar fascia response to load in T1DM with next-generation 3D ultrasound analysis.

Dana Lis

University of Tasmania

The effects of gluten-free diets on performance in non-coeliac athletes.

Sarah Warby

La Trobe University

The effect of exercise based management on multidirectional instability of the glenohumeral joint: A pilot randomised controlled crossover trial.

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SPORTS MEDICINE AUSTRALIA RECOMMENDED

Journal of Science and Medicine in Sport

Sports medicine and sports science contributions to football

Gregory S. Kolt, Editor-in-Chief of the *Journal of Science and Medicine in Sport* looks back at the different football research that has been published by JSAMS in past years.

The 2014 FIFA World Cup in Brazil is now less than a month away. It would be a safe bet that many of our readers are looking forward to that pinnacle event in the football world with great excitement.

"Enjoy the research that has been published in the *Journal of Science and Medicine in Sport* on football, but equally (if not more so) enjoy the achievements and spectacle of the upcoming FIFA World Cup."

Outside of the sheer spectacle of a good game of football, it is apparent to me that it is a game that lends itself to vast input from sports medicine and sports science. A simple database search exposes a vast literature addressing a wide variety of aspects of football performance, injury, and recovery. Of even greater value is that this research is stemming from all corners of the globe, allowing varied approaches to be applied and learnt from. At the *Journal of Science and Medicine in Sport*, we have published extensive football research from regions including Europe, Australasia, the Middle East, North America, and South America.

"In the area of injury Eirale et al (2013) reported on a prospective study of injury in football in first division football clubs in Qatar."

I would like to highlight some of the football research that we have published in recent years and encourage you to refer to these research findings to more fully understand the importance of sports medicine and sports science to football. For example, Akenhead et al[1] examined the distances covered at different accelerations and decelerations during professional competitive football games, and reported that acceleration and deceleration capability are acutely compromised during match play, with implications for transient fatigue. In another recent study Lovell et al[2] examined work-rate of football players immediately after a passive half-time interval. They demonstrated a markedly lower work-rate in the first five minutes after such a passive half-time break. Vescovi[3] focussed on female football players and investigated speed characteristics in those drafted or not drafted in a professional setting. They concluded that sprint speed was able to differentiate those in the two groups, with those in the drafted group able to achieve higher speeds.

"Vescovi (2012) focussed on female football players and investigated speed characteristics in those drafted or not drafted in a professional setting."

Football has also been the focus on studies of carbohydrate supplementation. Russell et al[4] showed that carbohydrate supplementation attenuated decrements in shooting performance by professional academy players during simulated football match-play. In the area of injury Eirale et al[5] reported on a prospective study of injury in football in first division football clubs in Qatar. They found relatively high rates of overuse injury and a high recurrence rate of injury to the thigh. Wilson et al[6] examined the cardiovascular risk factors of professional football players of West-Asian and Black African descent. Despite being matched for activity levels, the players of West-Asian descent had a greater number of cardiovascular risk markers than their Black African counterparts. The last paper I will highlight is that by

Weiler et al[7] who researched changes in markers of bone resorption and bone formation during the off-season of an English Football Association Championship club. It was found that the off-season (8 weeks) resulted in an increase in bone resorption and a decrease in bone formation markers.

"A simple database search exposes a vast literature addressing a wide variety of aspects of football performance, injury, and recovery. Of even greater value is that this research is stemming from all corners of the globe, allowing varied approaches to be applied and learnt from."

Enjoy the research that has been published in the *Journal of Science and Medicine in Sport* on football, but equally (if not more so) enjoy the achievements and spectacle of the upcoming FIFA World Cup.

This is an edited version of Gregory Kolt's "Editorial" published in Volume 17, Number 3, May 2014 *Journal of Science and Medicine in Sport*, http://dx.doi.org/10.1016/j.jsams.2014.03.009; www.jsams.org

Football research in JSAMS

Akenhead R, Hayes PR, Thompson KG, et al. Diminutions of acceleration and deceleration output during professional football match play. *J Sci Med Sport* 2013;16:556–561.

Lovell R, Barrett S, Portas M, et al. Re-examination of the post half-time reduction in soccer work-rate. *J Sci Med Sport* 2013;16:250–254.

Vescovi JD. Sprint speed characteristics of high-level American female soccer players: Female Athletes in Motion (FAiM) Study. *J Sci Med Sport* 2012;15:474–478.

Russell M, Benton D, Kingsley M. Influence of carbohydrate supplementation on skill performance during a soccer match simulation. *J Sci Med Sport* 2012;15:348–354.

Eriale C, Farooq A, Smiley FA, et al. Epidemiology of football injuries in Asia: A prospective study in Qatar. *J Sci Med Sport* 2013;16:113–117.

Wilson MG, Hamilton B, Sandridge AL, et al. Differences in markers of cardiovascular disease between professional football players of West-Asian and Black African descent. *J Sci Med Sport* 2012;15:266–271.

Weiler R, Keen R, Wolman R. Changes in bone turnover markers during the close season in elite football (soccer) players. *J Sci Med Sport* 2012;15:255–258.

Discipline group news and events

The Australasian Academy of Podiatric Sports Medicine (AAPSM)

News:

- AAPSM have recently re-launched our website to provide more services to members – check out www.aapsm.org.au
- If you're not already, get on board with AAPSM's social media networks: follow us on Twitter @_AAPSM_ twitter.com/_AAPSM_ or like us on Facebook www.facebook.com/AAPSM1

Upcoming events:

- **State seminars**
  QLD: Radiology (July)
  SA: Musculoskeletal Screening in Football (July)
  VIC: Screening the Athlete (August)

For more information visit www.aapsm.org.au and keep an eye on our member emails

Exercise & Sports Science Australia (ESSA)

News:

- **Exercise Right Week 26 – 30 May 2014, AEP Awareness**
  ESSA is proud to launch "Exercise Right" week from 26-30 May 2014, the first national awareness week for accredited exercise physiologists in Australia. In an environment cluttered with exercise advice, "Exercise Right" week aims to position accredited exercise physiologists (AEPs) as health professionals qualified to prescribe the right exercise for both healthy people and people with chronic conditions. AEPs are university trained to conduct a full assessment of each individual before prescribing suitable and safe exercise. The right exercise for you. For further information visit www.exerciseright.com.au

For more information visit www.essa.org.au

Australasian College of Sports Physicians (ACSP)

News:

- The ACSP applications for the SEM training program will open on July 1 and close August 8. For more information please visit our website www.acsp.org.au
- There has been a date change for the Part 1 examinations for 2014. They will now be held on August 2 in capital cities around the country. Applications for the August examinations close May 31. For more information, please see our website www.acsp.org.au

For more information visit www.acsp.org.au

Sports Dietitians Australia (SDA)

News:

- As the peak professional body for Accredited Sports Dietitians in Australia, SDA is focused on showcasing their expertise and the importance of a great nutrition plan to achieve performance goals. Our members are appropriately qualified to support teams and athletes to perform at their very best, in a safe and ethical way. And our rigorous accreditation process sets the benchmark for knowledge and practical experience and is recognised nationally and internationally. The SDA website has a range of Fact Sheets and an interactive "Find a Sports Dietitian" map.
- If you're working with a team or group of athletes, are they getting the right nutrition at the right time? Ask an expert – an Accredited Sports Dietitian expert.

Upcoming events:

- **Nutrition for Exercise & Sport Course (1 day)**
  7 June – Adelaide; 14 June – Canberra;
  21 June – Melbourne; 9 August – Hobart;
  30 August – Brisbane & Melbourne

For more information visit www.sportsdietitians.com.au or follow SDA on Twitter @sportsdietaus

Sports Physiotherapy Australia (SPA)

News:

- The SPA recently participated in the "Bone Symposium" at the AIS in Canberra, in conjunction with SMA-ACT, AIS and the ACSP.
- 2014 is a non APA conference year so we look ahead to the SMA "be active" conference in Canberra in October. In addition, we have an upcoming National Tour by shoulder surgeon Ben Kibler. Finally, we have our ongoing suite of PD courses which again this year has grown, with more courses in more locations.

Upcoming events:

- Ongoing Professional Development events – see www.physiotherapy.asn.au for details

For more information visit www.physiotherapy.asn.au

Sports Physiotherapy New Zealand: Introduction

Hamish Ashton, President of Sports Physiotherapy New Zealand (SPNZ) makes his *Sport Health* debut, providing readers with an overview of SPNZ's recent activity.

As the new President of Sports Physiotherapy New Zealand I would like to thank Sports Medicine Australia CEO Nello Marino for allowing us to develop a relationship with SMA and the readers of *Sport Health*. Through this I foresee the development of stronger professional ties which will lead to improvements in sports medicine, both for us as practitioners, as well as outcomes for our athletes. I hope you enjoy reading about SPNZ and what we have been up to lately. We look forward to contributing to the great knowledge base that SMA members have already established.

"Last year we collaborated with one of our top Sports Podiatrists to present a foot mechanics seminar that was held through the regions of NZ."

SPNZ is the Special Interest Group (SIG) of Physiotherapy New Zealand for Sports and Orthopaedic Physiotherapists. Currently SPNZ has approximately 700 members, making it the largest New Zealand SIG. We have a dual mission which involves the promotion of safe participation in physical activity, exercise, recreation and sport for participants of all ages and abilities, as well as supporting our members in the provision of high quality Sports Physiotherapy services to participants of such above mentioned groups.

"One of our big successes to date has been the completion of the NZ Sports Physiotherapy Code of Conduct."

Over the last two years SPNZ has been developing an educational program to help enable its mission and goals. The main program which is still currently under development involves an educational pathway for our future Sports Physiotherapists. This is based on similar principles to that

of the Australian SPA and Chartered Physiotherapists in Sport groups where there are a number of levels of courses leading to an advanced practitioner status. To this a number of stand-alone courses are being run to compliment and add to the main program. These are designed to give further insight into the diversity that is a Sports Physiotherapist. Last year we collaborated with one of our top Sports Podiatrists to present a foot mechanics seminar that was held through the regions of NZ.

"Clinically based Keynote Speakers, which we borrow from across the ditch, are always a draw card for us."

In March we held our biennial Sports Physiotherapy Symposium. This is a key feature of our education program. Unlike many conferences, we have decided to maintain a clinician focus on presentations rather than just pure research. All presentations have a take home message of 'I can use this in my clinic tomorrow'. This focus is appreciated by our attendees. Clinically based Keynote Speakers, which we borrow from across the ditch, are always a draw card for us.

"We have a dual mission which involves the promotion of safe participation in physical activity, exercise, recreation and sport for participants of all ages and abilities, as well as supporting our members in the provision of high quality sports physiotherapy services to participants of such above mentioned groups."

One of our big successes to date has been the completion of the NZ Sports Physiotherapy Code of Conduct. This was launched at our recent Symposium and is a world first for Sports Physiotherapy. Until now there have been codes of conduct for the general physiotherapy population and Sports Doctors/Physicians, but not for the Sports Physiotherapy practitioner. It was acknowledged that those of us working in the field of sport are subjected to different situations than the average physiotherapy population, and having guidelines as to what is good or acceptable practice will benefit our athletes, and us, as well as the profession as a whole. The code can be found on our website and viewed at: http://sportsphysiotherapy.org.nz/sportsphysiotherapy.org.nz/documents/Sports-Physiotherapy-Code-of-Conduct.pdf

Hamish Ashton
President SPNZ

INCORPORATING THE FOLLOWING:

Australian Conference of Science & Medicine in Sport (ACSMS)

National Physical Activity Conference (NPAC)

National Sports Injury Prevention Conference (NSPIC)

Refshauge Lecturer
Professor Danny Green
Winthrop Professor, School of Sport Science, Exercise & Health,
University of Western Australia

Professor Stuart Biddle
School of Sport, Exercise & Health Sciences, Loughborough University, UK

Dr Darren Burgess
High Performance Manger, Port Adelaide Football Club

Dr Tim Gabbett
Sport Scientist

Dr Andrew McIntosh
Biomechanist

Dr Jill McNitt-Gray
Biological Sciences & Biomedical Engineering,
University of Southern California

Professor Neville Owen
Head of Behavioural Epidemiology Laboratory, Baker IDI

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Registration Form

BE ACTIVE 2014

CONTACT DETAILS

Title | First Name | Last Name | DOB | Gender M/F

Profession/Position | SMA Membership No

Organisation/Discipline Group

Postal Address

Suburb/Town | State | Post Code | Country

Phone | Fax | Mobile

Email

Special Requirements - Dietary, Physical etc

REGISTRATION FEES

"be active 2014" includes:

- Australian Conference of Science and Medicine in Sport (15-18 Oct)

- National Physical Activity Conference (15-18 Oct)

- National Sports Injury Prevention Conference (15-18 Oct)

Sports Medicine Australia Membership

Join SMA now to be eligible for one of the ASMF Fellows awards. Conference awards are only available to SMA members. Joining fee of $40 waived for Conference delegates. SMA membership is open to anyone with an interest in or direct involvement with sports medicine, sports science, physical activity promotion or sports injury prevention and a minimum three year full time tertiary degree (or studying full time for a degree for student membership). The FOC student membership is an 'online access' membership only. For more information visit the membership page of the Sports Medicine Australia website.

			Sub Total AUD$
SMA Membership	☐ Full Member - $250	☐ Student Member - $FOC	

Conference Registration	Early Bird Registration On or before 31st July 2014	Late Registration On or after 1 August 2014	
SMA Member Registration - Full	$820	$920	
SMA Member Registration - Student ^	$470	$570	
Non Member Registration - Full	$1050	$1150	
Non Member Registration - Student	$570	$670	
Developing Countries Registration (please refer to website for list)	$500	$600	
One Day Registration * (Please tick which day you would like to attend)			
☐ Wed 15 October ☐ Thurs 16 October ☐ Fri 17 October ☐ Sat 18 Oct			
Registration - Full or Student	$325	$375	

^ Student Registration Student delegates must be full time and must supply a letter from their Head of School verifying full time status.

* One day registration includes entrance to the social program for the day that you are registered only.

Registered delegates receive access to all sessions being offered by the three conferences on their registered days only. Full registered delegates also receive lunch, morning and afternoon teas, tickets to the social program, entrance to the trade exhibition, a detailed Conference Program and a Conference satchel.

Social Program

Costs are included in the registration fee unless otherwise noted above. For catering purposes please Tick if attending.

	Delegate Ticket		Additional Ticket		# Required	
Welcome Reception (Wed 15 October)	$nil	☐	$80	☐		
Conference Dinner (Sat 18 October)	$nil	☐	$135	☐		

☐ Enclosed is my cheque, payable to ASMF LTD

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☐ Please tick if you do NOT wish your contact details to be made available to Conference Trade Exhibitors.

☐ Photographs will be taken during the course of the conference for use in SMA publications and communications. If you do not wish for your photograph to be included as part of these publications or communications please tick the box.

PLEASE FORWARD THIS FORM TO:
ACSMS Conference Secretariat, C/-Sports Medicine Australia, PO Box 78, Mitchell ACT 2911
P: +61 3 9674 8709 F: +61 3 9674 8799 E: acsms@sma.org.au

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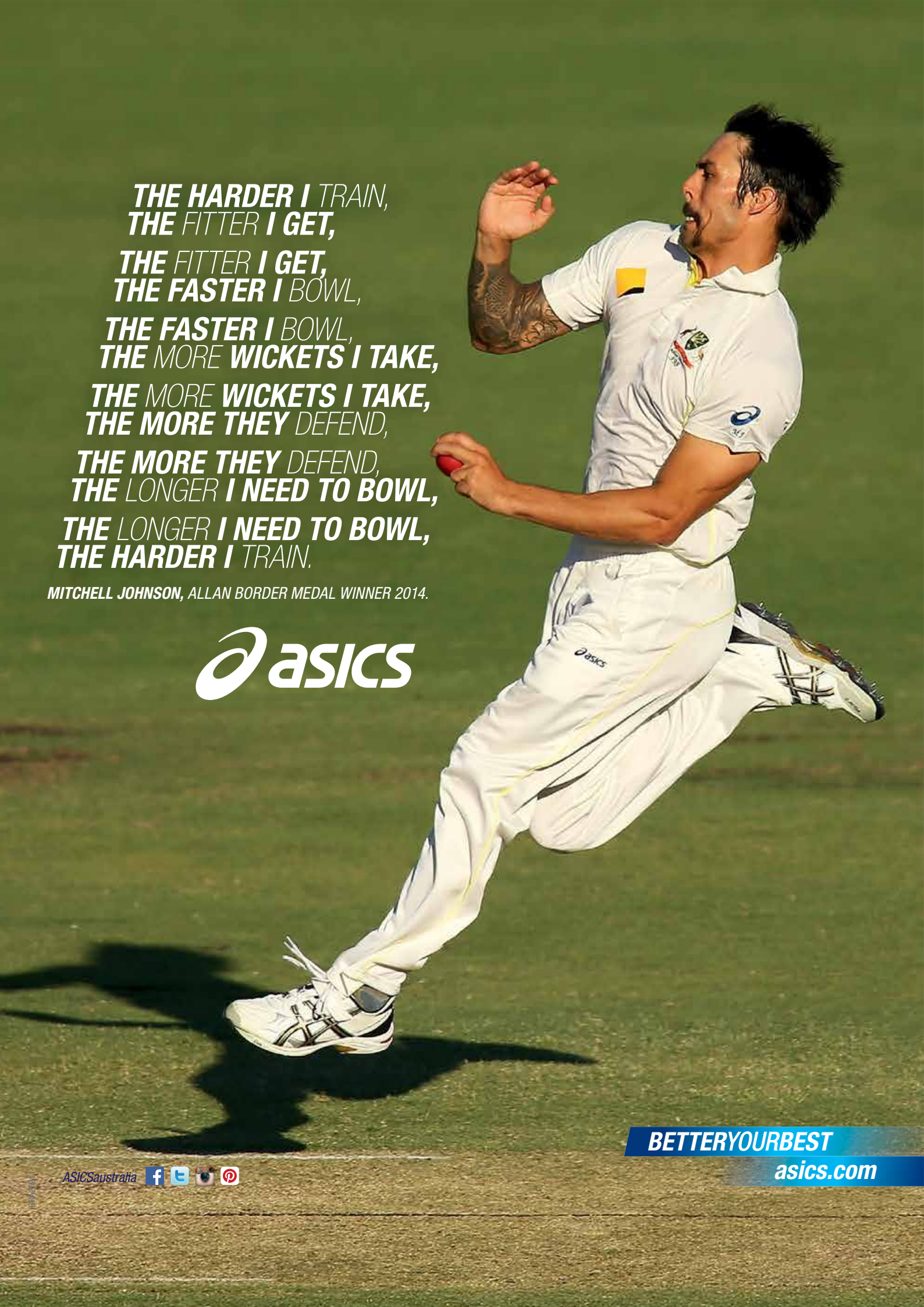
THE HARDER I TRAIN,
THE FITTER I GET,
THE FITTER I GET,
THE FASTER I BOWL,
THE FASTER I BOWL,
THE MORE WICKETS I TAKE,
THE MORE WICKETS I TAKE,
THE MORE THEY DEFEND,
THE MORE THEY DEFEND,
THE LONGER I NEED TO BOWL,
THE LONGER I NEED TO BOWL,
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