

# *Australasian Musculoskeletal Medicine*



- Myths and critical reasoning
- Core stability exercise programs
- Sacroiliac joint pain
- Neutrophins and semaphorins
- Shoulder pain referral zones
- Greater trochanteric pain syndrome
- Prolotherapy

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*Australasian Musculoskeletal Medicine* is published by the Australian Association of Musculoskeletal Medicine for medical practitioners interested in the etiology and management of musculoskeletal disorders.

Opinions expressed are those of the authors and not necessarily those of the editor or the Association.

Editorial comment may reflect the opinions of the editor alone.

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# Editorial: “Nullius in verba”

This issue of *Australasian Musculoskeletal Medicine* (AMM) is the first of which I am editor and it is a privilege to be involved in this manner. Over the years I have gained much professionally from many of my colleagues in the musculoskeletal field, from reading this journal, attending AAMM conferences and courses, and informally from direct discussions. In many ways it is almost a duty to contribute something in return and being the editor of this journal is something that falls within this ambit.

Over many years of being a member with the AAMM and especially from being exposed to the studies of Professor Nik Bogduk and others, I have absorbed something that is characterized by the motto of the Royal Society: “Nullius in verba”. This translates roughly as “on the word of no-one”. The implication is that that we should look at the scientific evidence rather than simply accepting someone’s personal opinions. Indeed, I believe, we must continue this focus – to withstand the domination of “authority” and to verify all statements by facts determined by good research and studies. Professor Bogduk has addressed this theme in this issue of the AMM journal in a clear and incisive manner. Thanks are again expressed to Professor Bogduk for his continued support and direction. I have heard the current AAMM President Dr Geoff Harding also verbalize this theme at times with the statement that we need to simply “quote the research” and he provides a current example in his president’s report that demonstrates how “authority” can be detrimental to patient outcomes. Other articles presented in this edition of the AMM journal are an attempt to condense current scientific studies into a useful clinical context and also hopefully fulfil this basic concept.

Appreciation is extended to the authors who have contributed to the 2012 AMM journal. The effort of researching and writing a paper for publication is considerable but the payoff for the author is a much better understanding and in-depth knowledge of a particular topic. Papers of interest in this edition include an excellent review on the sacroiliac joint by Drs Bruce Mitchell, Paul Verrills, and David Vivian and a thought-provoking review of the lack of evidence for the commonly promoted concept of core strength/stability for the management of back pain by Dr Christine Brooks, with a comment from Dr Phillip Watson. I am sure readers will find these papers of clinical value. Dr Grant Thompson from NZ has submitted a interesting look at steroids for trochanteric bursitis and Dr Steve Jensen has an article of shoulder pain referral that is also of direct clinical value. Dr Peter Jackson has submitted two brief cases that also illustrate the theme of the journal especially with regards to the knee case. A long-term contributor to the journal is Dr Margaret Taylor who has once again compiled the educational options and provided a review of some of the latest studies on her passion of prolotherapy. The efforts of all these authors is much appreciated.

This journal and the AAMM “Backbone” newsletter provide ample opportunity to publish, discuss, and reflect on clinical cases and I would encourage readers to pursue such options. Another venue for publishing case studies, literature reviews or original research that is available to us is the *International Musculoskeletal Medicine Journal*. In this way we can

analyse our work and progress can be made in helping our many patients with chronic musculoskeletal pains. It is always satisfying, as a professional to be able to help relieve pain and suffering and it should be our professional duty to continue to refine and improve our clinical skills and treatments.

It has been most satisfying in the past few years to be able to offer patients reasonably effective treatment for chronic lumbar spinal pain due to active zygoapophysial arthritis. This has arisen from the fundamental research and trials over many years by our colleagues and the advent of interventional radiology. The art and difficulty, however, still arises in identifying this subgroup but at least there is now a treatment we can offer these patients to help relieve their chronic pain.

Areas for which good treatments are desperately needed include an effective nonsurgical option for chronic discogenic pain and development of a protocol that prevents the neo-innervation of intervertebral discs after acute injury. Other areas include the perennial problem of chronic neuropathic pain. Basic research, however, is revealing a complex interaction of neurotransmitters, and signalling molecules that are starting to shed light on the underlying biology. I have provided a review of some of these studies in this issue of the journal. Undoubtedly, this fundamental research will provide avenues for treatment in the future, such as monoclonal antibodies or similar. Hopefully, over the next few years that I am editor, I will be able to report that these and other problems have been solved successfully.

The annual combined AAMM and NZAMM conference is always worthwhile to attend and this year our Kiwi colleagues are hosting it in Wellington. Whilst it may be windy and chilly for Aussies, Wellington is a picturesque place to visit. The program looks interesting and useful clinically and I am looking forward to presentations such as that on “Surgical advances in osteoarthritis, articular cartilage transplantation; meniscal injuries and osteoarthritis”. There is more about the conference later in the journal, so make sure to book flights and accommodation and plan on attending. Thanks are extended to our Kiwi colleagues for organising this conference.

On a different tack and providing free educational material is an Internet resource known as YouTube. I am sure many are already familiar with it. There are some excellent clips available on anatomy, examination techniques, and the like. I could recommend several but a “standout” clip (there are actually two and a further one on the brachial plexus) that I discovered recently is <http://www.youtube.com/watch?v=iDXUwErttJA&feature=relmfu>. This clip is a clear, and entertaining, presentation on the anatomy of the upper limb and it is given in such a way as to make it easy to remember. I think we would all have liked to have had an anatomy professor teach like this one.

Your AAMM committee has been active during the past 12 months promoting the field of musculoskeletal medicine and acting in your professional interests. The President, Dr Geoff Harding, warrants our gratitude for all his hard work and effort, without which the AAMM would not continue to function. Other committee members have also contributed in regular teleconference hookups and should be acknowledged.

Enjoy reading the 2012 AMM journal.

**Dr Tom Baster**  
*Newnham Road Medical Centre, Brisbane*

# From the AAMM President

These days I find myself wondering about the state of medicine in Australia, especially that of general practice. Of course, I am no expert on the subject given that I have been in full-time musculoskeletal medicine since about 1989. However, I do read the press and have discussions with colleagues who are in general practice, and I am also privy to the impressions of those patients who come to see me about their musculoskeletal pain problems. There seems to be a paradoxical change underway where, in spite of care plans and improved technology, and the growth of large multi-doctor clinics, many patients are actually feeling depersonalised by the “system”.

Those of you in general practice and having an interest in musculoskeletal medicine might disagree with me about the following observation and I would be interested to hear from you if you do. The impression I have is that there has been a change in the system where the doctor (GP) is losing influence, where true EBM is not as influential as before, and there is a rise – and rise – of alternative health paradigms which are becoming accepted as mainstream health alternatives.

The paradox surrounding EBM is that although medicine hangs onto “evidence” as being core to better practice, the general public continues to seek out “cures” which have no evidence behind them and where the practitioners are thought to be just as scientific as the medical practitioners. EBM doesn’t seem to have influenced the public much at all. I believe, moreover, that musculoskeletal medicine, as taught in the various Diploma programs, is generally supported by good evidence.

Mostly, as you know, I’m conservative and I’m concerned about where one will be able to find a “good” GP in a few years time. I think a “good” GP is one who is not afraid to bite the bullet and do things rather than plan things, such as investigations, referrals to specialists, and the like. My impression is that many of the younger GPs are not willing to undertake procedures for fear of being sued for making mistakes, or because it takes too long to learn the skills for little direct reward. Our GPs seem to be rewarded more and more for administrative tasks which might – or might not – lead to direct improvements in patient well-being.

At some recent talks which I gave to about 90 GPs, I asked “who performs intra-articular injections of any kind”. There were positive responses from no more than 10. Among the reasons given for not performing injections were: “I don’t want to cause infection”, “I don’t want to get sued”, “There’s no financial incentive”, “It’s easier to refer to radiologist for ultrasound-guided injection”, and “It’s too difficult to learn”. This I find a rather disconcerting state of affairs.

When I decided to start up a musculoskeletal medicine practice, it was at a time when general practice was struggling with almost universal bulk billing, meaning one had to work long hours to pay the bills and make a reasonable living. Musculoskeletal medicine offered a chance to practise new skills and “escape” from the system where one depended on the government to increase the rebate – which it never really did adequately. Now, however, there seems to be more incentive for GPs to remain in general practice and not make the move out.

So what does this mean for musculoskeletal medicine? The two lessons I think we need to learn are that, firstly, we need to continue to educate GPs in and around our local areas in the skills needed to assess and treat musculoskeletal pain. Secondly, we need to increase the awareness of the discipline as much as possible to ensure that there will be newer graduates interested in following in our footsteps. Unfortunately, many of us are in our fifties and sixties (and seventies!) and headed out the door – often with no-one around to take over. We need to raise our profile as much as possible. Soon the AAMM will have a new website which will go some of the way towards doing this, but we also need to promote ourselves and our message more energetically.

In this last week I have written a report for a WorkCover insurer about a patient who had been told by a specialist “the injury was an exacerbation of pre-existing degenerative changes and now that exacerbation has ceased”. Note, not “and I believe that that exacerbation has ceased”. I presented the current evidence in my report with respect to the diagnosis and with respect to the prognosis and added the references at the end of my report. There were 13 in all. The report directly addressed all of the points made by the specialist whom I considered to be simply wrong or not having any evidence to support his findings. My patient had the claim re-instated on the basis of this report. I recall Nik Bogduk saying once that we need to quote the experts in our evidence, and use them to support our experience. Having 13 experts backing up your opinion is powerful. We need to address the sometimes non-evidence-based assertions made by certain experts and not lack confidence in challenging these opinions. Doing this more often will certainly help raise the profile of the discipline.

I thank my colleagues in the AAMM for their continued support and encouragement and look forward to seeing you all at our annual scientific conference in Wellington later this year.

*Geoff Harding*

# From the NZAMSM President

During 2011-2012, NZAMM has been continuing its cycle of educational activities. Central to this are the musculoskeletal workshops presented around the country by musculoskeletal physicians. Last year's workshop theme was "The Shoulder". The workshops were well received by participants in Rotorua, Wellington, and Auckland. The theme for the 2012 workshops is "Low Back Pain". This along with other topics will be presented at the NZMA GP CME meeting in Rotorua on 7 June 2012 and at other meetings during the year.

NZAMM members have been invited to and attended AFMM Retreats. The latest one was in Auckland in March. The variety of case and topic presentations has been interesting and provided an excellent opportunity for CME and for members to share their knowledge and experience. I encourage members to attend these retreats.

The Australasian Musculoskeletal Medicine journal continues to be published, thanks largely to the efforts of our Australian colleagues. I encourage members to support "their Journal" and to contribute articles for publication in the form of original research, reviews, case reports, and letters to the editor. Last year, we trialled a subscription to the journal International Musculoskeletal Medicine. Unfortunately, there was insufficient member support for continuing the subscription for 2012.

The main event for NZAMM for 2012 is the scientific meeting in Wellington on 14-16 September. The title of the meeting

is "Joint Matters: Topical issues on joint function, disease, pain, treatment, and rehabilitation". We are honoured to have Professor Lars Arendt-Nielsen from Denmark and Dr Jeremy Lewis from the UK presenting, along with NZ and Australian speakers. There will be keynote lectures involving basic sciences to clinical application, complemented by workshop sessions.

Clinical presentations will be from musculoskeletal physicians, rheumatologists, orthopaedic surgeons, radiologists, physiotherapists, podiatrists, and occupational therapists. The latest research and developments in a variety of topics relating to the joint will be presented so that there will be much of interest to all practitioners in the field of musculoskeletal medicine.

Wellington, "NZ's Capital of Cool", is an excellent and convenient location, with lots to do and see for delegates and accompanying persons. The notice for the scientific meeting is featured in this journal and on the NZAMM website [www.musculoskeletal.co.nz](http://www.musculoskeletal.co.nz).

The event is a combined meeting of the NZAMM, AAMM, and AFMM. We look forward to renewing acquaintances with members and our Australian colleagues. So mark the date on your calendars and see you there!

*Charlie Ng*

## JOINT MATTERS

### COMBINED ANNUAL SCIENTIFIC CONFERENCE

of the AAMM, AFMM, and NZAMM

Friday 14 September - Sunday 16 September 2012

Amora Hotel, Wellington, NZ

for more details see back page



# Myths and critical reasoning

*Professor Nikolai Bogduk, Newcastle Bone and Joint Institute, Royal Newcastle Centre, Newcastle, Australia*

It must be an anthropologic feature, but a large proportion of humans are kindly, placid, and compliant folk. They are happy to defer to authority. This feature extends to medical practitioners.

Many medical practitioners pursued a career in medicine for noble reasons: they wanted to be able to help patients. So, they were happy to learn from anyone who was prepared to teach them. Their compliant nature, however, rendered them susceptible to authority figures.

Medical education consisted of authorities teaching students what they should know. The trademark of the authority was a set of slides named aetiology, pathogenesis, clinical features, investigations, and treatment. This education served its purpose; it taught would-be practitioners what to do.

The susceptibility to authority, however, carried over into continuing professional development. Anyone with a set of slides looked like an authority, and so was accepted as an authority by practitioners of a polite and compliant nature. In under-developed fields such as musculoskeletal medicine or manual medicine, it was easy for anyone to set themselves up as an authority.

All that they had to do was address a clinical problem that begged a solution, and invent a solution. If they addressed that solution with slides in the appropriate format, compliant practitioners would readily accept the speaker as an authority, and would adopt the solution.

What medical education did not teach is how to be discerning. Medicine never admitted that its professors might be wrong. It did not teach students how to question the professor.

For undergraduate purposes this might be pragmatic. There is so much to learn. We cannot afford the time to question and justify everything, so we have to take what is taught on trust. But in the postgraduate arena these concessions should not apply. Trust can be abused. For new knowledge, serious questions serve to protect kindly but compliant practitioners from gullibility. The fundamental questions are:

Is it true; and

How do we know that it is true?

Musculoskeletal medicine is replete with examples of how previous teaching has proved to be untrue. Few assertions have withstood scientific scrutiny. Indeed, the track record of professed knowledge is such that if someone once said something it is now more likely not to be true.

Many examples might be invoked, but I shall illustrate with two. The first exemplifies simple, forensic, bibliographic enquiry. The second illustrates the application of the rules of biostatistics.

## Back pain can't be diagnosed

A common claim in the literature is that back pain can't be diagnosed. More accurately, the claim is that a cause of back pain cannot be determined in 80% of patients or more.

This claim serves the contemporary psychosocialist revolution. The psychosocialists abjure the biomedical model: that symptoms are caused by injury or disease, and that treating the injury or disease will relieve the symptoms. Rather, patients complain of back pain because of psychological reasons and, therefore, require psychological treatment, not medical treatment. Politically, an apparently well-established fact that back pain cannot be diagnosed serves this psychosocialist revolution well.

A critical consumer, however, might ask:

Is it true; and

How do we know that it is true?

The first forensic step is to find the statement and look for the references that support it. In this exercise, however, it becomes conspicuous that although many authors espouse and endorse the purported fact,<sup>1-4</sup> few provide evidence. Rather, they no more than cross-reference others who have done the same. Such mutual support serves to sustain a purported fact, and if articulated often enough it becomes accepted as a well-known truth. Political or ideological consensus, however, does not make it true.

Upon deeper forensic enquiry, I traced the source to a study published in 1966 by a British GP.<sup>5</sup> That study did, indeed, fail to find a diagnosis in 80% of cases. So, its conclusions do serve the purposes of nihilists and defeatists. However, those who cite its conclusions do not disclose to readers other aspects of the study.

For diagnosis, the study relied on history, conventional examination, and plain radiography. Very few causes of back pain can be diagnosed by these methods either jointly or severally. A history of malaise, fever, and a penetrating injury, strongly hints at sepsis.

The combination of spinal pain, morbid aversion to movement, and a history of cancer is virtually pathognomonic of metastatic disease. However, infection and cancer are rare causes of back pain, accounting for only about 1.5% of presentations to primary care.<sup>6</sup> Nor does plain radiography improve diagnostic yield. Xrays might detect fractures but these are not necessarily symptomatic. They might detect advanced infection or neoplasia, but these are rare. Conversely, spondylosis, spondylolysis, and spondylolisthesis are common features on plain radiographs but they bear no valid relationship to pain, and cannot be invoked as diagnoses.<sup>6</sup>

The urban myth needs adjustment. It should be cast as back pain cannot be diagnosed if one uses methods that are not capable of establishing a diagnosis. Perhaps this is akin to relying on a stethoscope to diagnose headache.

The study in question, and the origin of the urban myth, predate the invention of CT scans and MRI. They predate the development of medial branch blocks and sacroiliac joint blocks. Discography had been invented some 17 years earlier but was not in common use until 20 years after the study.

In 1966, the concepts of zygapophysial joint pain and of discogenic pain were only whispers, and sacroiliac joint pain had been relegated to a memory of the 1940s and 1930s. So, the potential diagnoses and the means for their detection were simply not available when the study was performed and the myth was generated. Therefore, the conclusion can be elaborated in a different way: back pain cannot be diagnosed if you decline to use methods that are capable of making a diagnosis.

In patients with chronic back pain, figures have emerged (since 1966). The prevalence of discogenic pain is around 40%;<sup>7,8</sup> and that of sacroiliac joint pain is around 20%.<sup>9,10</sup> Vexatious is the prevalence of lumbar zygapophysial joint pain. Estimates of its prevalence depend on how stringent the criteria are for a positive response to blocks, and whether or not controlled diagnostic blocks are used.<sup>11,12</sup> Under stringent criteria, the prevalence is probably 5% or less in injured workers, but may be as high as 40% in elderly patients with no history of injury.<sup>11,12</sup>

These figures do not necessarily transpose to patients with acute back pain. These patients might have causes that have yet to be detected, or causes that have yet to be invented. We do not know because invasive investigations have not been applied to patients with acute pain; but nor should they.

Acute back pain has a good prognosis if managed well, and does not need to be investigated.<sup>13</sup> Only if pain persists do any investigations become indicated.<sup>13,14</sup> In that event, MRI can be useful. High-intensity zones and Modic lesions have a prevalence of about 30% and 15% respectively. Upon finding one or other of these signs an investigator can be 60-70% confident that the affected disc is painful.

Ideologically, there is a big difference between “back pain can’t be diagnosed” and “acute back pain does not need to be diagnosed”. Nor does the latter mean that acute back is a condition not deserving of medical attention. Medical care is not limited to drugs and xrays. Good medical care encompasses conscientious, concerted, confident, and convincing, explanation, encouragement, informed reassurance, and concern;<sup>6,13</sup> and is not predicated on a diagnosis having been made. The need for diagnosis arises when pain persists; and to avoid psychological decline the pursuit of diagnosis should be sooner than later.<sup>6,14</sup>

## Sacroiliac pain

When still a young postgraduate student I was made to feel deficient because I had not undergone a course of instruction in manual medicine and so I could not diagnose sacroiliac joint pain by manual examination; nor could I treat it with manipulation. Repeatedly, authority figures would appear

with boxes of slides and assertions. They would refer to various manual manoeuvres, and were adamant that these manoeuvres would diagnosis sacroiliac joint pain. Around me I saw others become convinced and adopt what was taught. I could have waited for the next available course, and paid my fees. Instead, I pursued other lines of inquiry.

I participated in studies of diagnostic blocks of the sacroiliac joint or its ligaments.<sup>15,16,17</sup> I did so because I felt that letting a local anaesthetic do the work was more objective than relying on mystical manual signs. I briefly studied physical examination<sup>18</sup> but others took over. They assessed the reliability and validity of manual examination for the diagnosis of sacroiliac joint pain. Our respective domains met, because the latter investigators used our diagnostic blocks as the criterion standard for diagnosis.

Formal studies have shown that many of the professed signs of sacroiliac joint pain lack reliability.<sup>19</sup> For most tests, two observers testing the same patients fail to agree significantly beyond chance on whether the sign is positive or not (Table 1).

The only tests on which observers agree sufficiently often to make the test reliable are the Gaenslen test and the thigh-thrust test. Yet, although these two tests might be reliable, they are not valid.

Reliability		
Yes	No	Questionable
Gaenslen thigh-thrust	compression Gillett sacral thrust cranial shear Patrick	gapping

**Table 1. The reliability of clinical tests for sacroiliac joint pain, based on van der Wurff, et al.<sup>19</sup>**

Research has shown that no single physical sign is diagnostic of sacroiliac joint pain. Patients who are positive to three or more provocation signs are more likely to have sacroiliac joint pain, but even combinations of signs fall short of being diagnostic. Individual tests have positive likelihood ratios between 2 and 3; combinations of tests have likelihood ratios between 0 and 4 (Table 2). Such likelihood ratios, however, increase the likelihood that the patient actually does have sacroiliac joint pain from a pretest probability of 0.20 (20%) to only between 0% and 50%. Thus, although the likelihood of the patient having sacroiliac joint pain is increased by finding positive signs on examination, that likelihood still falls short of being diagnostic.

It took a professional lifetime for the evidence to emerge, but the bottom line for me is that I did not miss out for not having learned manual medicine. My earlier scepticism – or rather, reluctance to trust authority figures – was eventually vindicated. Neither I nor my patients would have benefited beyond placebo from my having learned practices that were not valid.

Source	Test	Positive Likelihood Ratio	Diagnostic Confidence
Szadek, et al. <sup>20</sup>	compression thigh-thrust three or more	2.0	33%
		2.8	41%
		3.3	45%
van der Wurff, et al. <sup>21</sup>	any one	1.7	30%
	any two	2.2	35%
	any three	4.0	50%
	any four	1.4	26%
	any five	0.0	0%

**Table 2. The validity of individual tests for sacroiliac joint pain, or combinations of tests. Based on Szadek, et al.<sup>20</sup> and van der Wurff.<sup>21</sup> Diagnostic confidence is percentage chance that the patient has sacroiliac joint pain when the test is positive.**

At the time, I had not learned the intellectual skills required. I have learned them since. For assertions about physical examination, the fundamental questions apply:

Is it true; and

How do we know that it is true?

These questions are amplified by the pivotal issues concerning physical examination that rely on a knowledge of biostatistics:

Does the test have reliability; and

Is it valid?

On how to answer these questions, I have published previously.<sup>22,23,24</sup> Perhaps readers might care to adopt asking these questions, and learn how to calculate the answers, in order to avoid being easily impressed by so-called authorities, and only later being embarrassed when science reveals that what they believed has proved false.

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# On rethinking core stability exercise programs

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## Abstract

Core stability training has gained wide acceptance as a treatment for low back pain rehabilitation, maintenance of a healthy back, and improved sport performance. This article examines aspects of core stability/strength exercise program approaches, with a focus on research suggesting that core stability and core strengthening programs are misconceived. It outlines a brief overview of spinal stability research and its application to core stability/strengthening programs within both the rehabilitation and sport performance enhancement sectors and briefly explains why these programs violate essential motor control and training theory principles.

## Introduction

In 1989 the San Francisco Spine Clinic used the term "core stability" in reference to their low back pain patient rehabilitation exercise programs.<sup>1</sup> In the late 1990s "core stability" programs similar to those used by the San Francisco Spine Institute ventured out of physical therapy settings and into the commercial fitness and competitive sport settings. The impetus for this appears due to University of Queensland researchers Hodges and Richardson<sup>2</sup> who, in 1996 reported that subjects with low back pain had a slower acting transverse abdominis than healthy subjects. Unlike the more esoteric language emanating from spinal stabilization research before 1996, Hodges and Richardson were talking anatomical language understandable to fitness practitioners who knew all about "abs". A differentiated "six-pack" rectus abdominis had sex appeal, and made them look fit and healthy. Now, not only could their clients achieve an attractive abdomen by participating in their exercise programs, they could concurrently avoid low back pain! The transversus abdominis took on a "god-like" status alongside the rectus abdominis.

Today, some core exercise programs aim to strengthen weak abdominal and trunk muscles and are better described as "core strengthening" programs. Other programs are designed to improve the recruitment of underactive muscles that are not adequately fulfilling their spinal stability function and are best described as "core stability" programs. "Core strengthening" programs are biased toward the fast twitch fatiguing fibers with the goal of strengthening them. "Core stability" programs aim to improve the recruitment and endurance of the slow twitch endurance fibers with the goal of restimulating them, resetting their onset of firing, and providing them with endurance capacity. The terms "core stability", and "core strengthening" are now firmly entrenched in the vocabulary of Pilates instructors, personal trainers and high performance sport coaches and their meanings

are frequently intermixed.

According to Lederman<sup>3</sup> this entire industry is flourishing from roots planted in the soil of six largely unsupported assumptions. These include:

1. Some muscles are more important than other muscles to spine function and stabilization.
2. Weak abdominals, specifically a poorly functioning transversus abdominis and multifidi, leads to low back pain.
3. Strengthening abdominal and other trunk muscles can reduce back pain.
4. A unique group of "core" muscles work independently of other trunk muscles.
5. By normalizing the timing of the "core muscles" the patient will have relief from back pain.
6. Back pain is related to spine instability.

Reeves<sup>4</sup> likens the entire situation to John Godfrey Saxe's poem about six blind men and the elephant. Similar to the elephant, spine stability data has many potential applications depending on one's point of view. Practitioners have jumped on the core stability exercise program bandwagon with minimal knowledge about the complex nature of the spinal mechanism.

They are currently confusing core strength, core stability, motor control, and force transfer and many programs are violating the well-known principles of motor control principle of learning transfer and training the theory principle of specificity. They also ignore mounting evidence suggesting the current application of spinal stability research to core exercise programs could possibly be incorrect. Within the sport performance sector there is growing awareness about the lack of evidence showing that these programs translate into improved sport performances.<sup>5</sup>

The conceptual problem may be much deeper than a misapplied application issue. It may lie with the research

itself. Gracoveski<sup>6</sup> has illustrated that the mechanical and mathematical theoretical models used in spinal stability research require many assumptions so they can correctly depict human movement. He used the example of spinal stability models predicting that:

1. Muscles both pushed and pulled.
2. Abdominal pressure will be 20 times higher than blood pressure.

In the first instance the models constrain muscles to just pulling, even though they theoretically permit them to also push, while the abdominal pressure issue is not addressed.

## Rationale for spinal stability exercise

The idea that there must be a particular position where the spine is “stable”, and therefore remain pain free, has its roots in mechanics. In his influential 1989 work Bergmark<sup>7</sup> defined mechanical stability as: “the ability of a loaded structure to maintain static equilibrium even at small fluctuations around an equilibrium point. If stability does not prevail, an arbitrarily small change of the position is sufficient to cause collapse”.

To account for the fact that the spine was able to move within a fairly wide range without collapsing, White and Panjabi<sup>8</sup> derived the notion of “clinical stability”. This was defined as: “the ability of the spine under physiologic loads to limit patterns of displacement so as not to damage or irritate the spinal cord or nerve roots and, in addition, to prevent incapacitating deformity or pain due to structural changes”.

Spinal instability occurred when an excessive range of movement was not under protective control from stabilizing muscles. Clinical spinal instability occurred due to muscle imbalance around the spine, degenerative diseases of the spinal column, and/or neural control malfunctions.<sup>9,10,11</sup>

Bergmark<sup>7</sup> used mathematics and mechanics to provide a framework for the complex nature of spinal stability. From this framework two schools of thought subsequently developed – one attributed to Stuart McGill from Canada, and the other to Paul Hodges from Australia.<sup>12</sup> Coming from a biomechanics perspective McGill recognized the relative contribution from every muscle source depending on the dynamics involved. McGill proposed spinal stability exercise programs that focused on controlling spinal posture in “biomechanically sound” positions. The exercises stressed co-contraction and recruitment of the entire trunk musculature in an effort to strengthen the stabilizing muscles surrounding the spine.<sup>13,14</sup>

Hodges,<sup>15,16</sup> on the other hand, took a motor control approach and focused on the timing of individual muscle activation. His research suggested that low back pain related to loss of motor control, specifically over transversus abdominis (TrA) and multifidus (Mf). The exercise programs isolate TrA and Mf and then train them with the emphasis placed on re-establishing the “correct” timing for the onset

of muscle firing.

Both McGill and Hodges defend their respective positions against growing criticism that they place too much emphasis on the abdominal musculature and how to best recruit these to achieve spine stability.<sup>17</sup>

## What is the “core”?

In the absence of its musculature the passive system of the spinal column (i.e., vertebrae, facet articulations, intervertebral discs, spinal ligaments, and joint capsules) will buckle under loads of only 90 newtons (about 9 kg). Routine daily activities demand loads of up to 20 times greater than this.<sup>18,19</sup> The increase in spinal load-carrying capacity is made possible by optimal coordination of two other subsystems.<sup>10,11</sup> The first is an active musculoskeletal subsystem (i.e., muscles and tendons surrounding the spinal column). The second is a neural and feedback subsystem (i.e., force and motion transducers located in ligaments, tendons and muscles, and the CNS control centres).

The first formal description of the “core” came from Bergmark<sup>7</sup> who was, in turn, influenced by Panjabi's<sup>10,11</sup> three subsystems – the passive spinal subsystem, the active musculoskeletal and neural subsystems. Bergmark further subdivided the active musculoskeletal subsystem into a local muscle system (muscles with an origin or insertion at the vertebrae), and a global muscle system (those muscles and intra-abdominal pressure that transferred the load directly between the thoracic cage and the pelvis). The local system included multifidi, interspinals, intertransversarii muscles, medial and lateral divisions of the local erector spinae muscles, and quadratus lumborum. The global system included the global erector spinae muscles, the internal and external obliques, the rectus abdominis, the lateral parts of the quadratus lumborum muscles that were inserted at the twelfth ribs, and psoas). All the thoracic erector spinae muscles were assigned to the global system based on Bogduk's<sup>20</sup> description of the erector spinae. The remainder was assigned to the local system. The global system was conceived as balancing the outer load so that the force transferred to the lumbar spine could be handled by the local system.

In the current literature the term “core” generally refers to the abdominal and trunk musculature as outlined by Richardson and Hodges.<sup>21</sup> They described the “core” as a box with the abdominals in the front, paraspinals and gluteals in the back, the diaphragm as the roof, and the pelvic floor and hip girdle musculature at the bottom. The diaphragm and pelvic floor were important for intra-abdominal pressure that was believed to help stabilize the spine by “pushing” against it.

Comerford<sup>22</sup> is an exception to the general descriptive approach. His description of the “core” is similar to Bergmark's approach. Comerford begins by dividing the core musculature into an inner wall and outer wall. The inner wall is made up of spinal stabilizing muscles. These include the diaphragm, transversus abdominis, segmental multifidus, posterior

psoas, and the pelvic floor.

The outer wall is made up of the global stability muscles and global mobility muscles. The outer wall influences postural alignment, contributes to the production of movement and controls range of motion. In Comerford's model "core" muscles are further categorized as having a local motor control role, a global stabilizing role, or a global mobility role as follows:

1. *Muscles with a local motor control role.* These muscles provide stiffness to the spine by controlling segmental translation. Their activity is continuous and independent of the direction of movement. Muscles in this group include transversus abdominis, segmental lumbar multifidus, and the posterior fasciculus of psoas major.

2. *Muscles with a global stabilizing role.* These muscles generate force to control/limit range of movement. They shorten through the full range of joint motion, can hold a position isometrically, and eccentrically contract to control the return of the spine to its neutral position. They also perform low threshold eccentric deceleration of movement. Their activity is non-continuous and direction dependent. Muscles in this system include the external obliques, and superficial multifidus.

3. *Muscles with global mobility role.* These muscles generate force to produce range of movement. They concentrically contract to produce acceleration, have a high load shock absorption capacity, and their activity is phasic. Muscles in this system include rectus abdominis, iliocostalis, rectus femoris, and the hamstrings.

Comerford contends that the outer wall is the problematic area. It can develop muscle imbalances where some global mobilizer muscles dominate the stabilizer muscles, or create restrictions that cause compensatory movement patterns. He combines strengthening and motor control for efficient transfer of forces in his exercise program design approach and claims he needs to have this detailed breakdown of the "core" to ensure correct therapy.

## Application to the high performance sector

### a) Rehabilitation and spine health maintenance

The promotion of core stabilization training for high performance athletes is based on two rationales. The first relates to rehabilitating athletes who have low back pain, with the goal of hastening their return to competition. Up to 30% of high performance athletes suffer from periodic low back pain.<sup>23,24</sup> In professional sports, low back pain is the most common cause of lost playing time.<sup>23</sup> The second rationale is for prevention purposes.

Both rehabilitation and prevention programs are based either in motor control or muscle strength training, with the goal of alleviating or preventing low back pain in athletes. The cause of the low back pain could be due to a timing

issue, abnormal muscle structure, or low strength. Multifidus atrophy has been seen in low back pain non-athletes<sup>25</sup> and in elite athletes.<sup>26</sup> Multifidus can also suffer from fatigue.<sup>27</sup> If multifidus fatigues then the spine loses some of its stabilizing protection.

Core strength requirements for spine stabilization turn out to be quite low. Rectus abdominis averages 2% of its maximal voluntary contraction during walking.<sup>28</sup> This is a very low strength requirement based on the muscle's reserve strength capacity. In addition, core-strengthening programs are not sufficiently challenging to stimulate improvements in strength. Any improvements in strength are likely due to changes in neural activation rather than muscle hypertrophy. Improved neural activation is a well-known phenomenon in sport training theory and is attributed to more efficient motor unit recruitment. While improved motor unit recruitment may ultimately prove important to correcting atrophy of specific "core" muscles by restimulating them, according to Lederman:<sup>3</sup>

- There is currently no evidence that reduced trunk muscle strength or endurance will predispose the individual to low back pain.
- Findings regarding loss of trunk muscle strength and atrophy in response to chronic low back pain are inconclusive.

There appears no consideration to the possibility that low back pain among healthy, young, fit athletes is a symptom of overtraining. Selye's<sup>29</sup> work describing how the body first adapts to stress, and then eventually maladapt when there is inadequate recovery time for the systems to return to homeostasis, is a well-accepted concept in training theory. Low back pain in athletes may simply be due to their bodies entering the maladaptation phase due to ongoing stressful training without adequate rest for recovery and supercompensation to occur. Improvements attributed to stability training may simply be due to the removal of the athlete from the high stresses of training, and restoration has occurred.

### b) Application to performance enhancement

From a performance enhancement point of view, the focus has been on "core strengthening" in the belief that strong abdominal and trunk musculature will permit a more efficient transfer of forces. While the idea of strengthening a subset of core muscle so they can transfer a higher force load faster makes intuitive sense, the prescribed exercises have no transfer effect because they are extra-functional – i.e., they lie outside the individual's functional movement skill set necessary to perform their sport. In core strengthening programs the body learns a specific motor pattern that has no transfer to specific sport performances because these programs violate the important training theory of "specificity." It is, therefore, not surprising that there is currently no substantial evidence that core stability/strengthening programs enhance sport performance.<sup>5</sup>

There is no debate that the core musculature transfers



forces. The question is: how does this occur? In 1975, when comparing the cross-section of the leg muscles to that of the erector spinae muscles, it became obvious to Farfan<sup>30</sup> that the power produced by the leg muscles could not possibly be channeled up the body by the much smaller erector muscles. Farfan began exploring how the thoracolumbar fascia might be involved in channeling forces from the powerful gluteus maximus through the trunk musculature to the upper extremities. This work stimulated some spinal stability researchers to begin thinking more broadly about how the spine was stabilized as it transferred forces through the body. One puzzle was the reason behind the redundancy of the spinal musculature and it was believed this held a clue.<sup>31</sup>

The detailed anatomical description of the thoracolumbar fascia provided by Bogduk and MacIntosh<sup>32</sup> provided sufficient understanding to propose how the thoracolumbar fascia worked with the abdominals to stabilize the spine.<sup>31</sup> A pull of transversus abdominis on the lateral raphe of the thoracolumbar fascia induces a force that brings the tips of the spinous processes together. In other words, Panjabi's three subsystems cannot work without the thoracolumbar fascia.<sup>31,33</sup> Mathematical analysis indicated that a 250 kg lift required the fascia to support four times what the spine musculature could do. Any damage to the fascia would severely weaken the spinal machinery resulting in an abnormal increase in spine compression and torsion.<sup>34,35</sup> A damaged thoracolumbar fascia may hold an explanation for low back pain in healthy fit athletes.<sup>34-35</sup>

Interesting ideas are also coming from Tom Myers<sup>36</sup> who, over the past 20 years, has been popularizing the notion that the body's network of fascia, of which the thoracolumbar fascia was just one albeit significant part, connects every part of the body from head to toe. Myers proposes that the connective tissue system is the third holistic network (the cardiovascular system and nervous system being the other two).

Influenced by the original writings of South African anthropologist Raymond Dart and his double spiral relationship of muscles of the trunk,<sup>37</sup> Myers developed the concept of the spiral line that he conceived to be a fascial connection between the shoulder and opposite leg, extending through the hip, to the ankle and the plantar fascia. He has also introduced the concept of tensegrity to practitioners – a word based in architecture. Tensegrity describes a closed structural system composed of a set of three or more compression struts within a network of tension tendons. The combined parts are mutually supportive so that the struts do not touch one another, but press outwardly against the tension network to form a firm, triangulated, prestressed, tension and compression unit. Myers uses tensegrity to explain how the fascia at one part of the body affects the action of muscles and joints in another part of the body. It also helps explain why excessive tension in one part of the body can present as pain in another part.

Recently Langevin<sup>38</sup> published a thought-provoking theoretical proposition supporting the potential importance of connective tissue as the body-wide signaling network.

This is how all the cells, tissues, organs and organ systems, including the muscles, “know” how to react to dynamic movement and are always in synch with their specific reaction to the movement. The interplay between cells, connective tissue matrix, and mechanical forces is known to control long-term sculpting of connective tissue. Since connective tissue plays an intimate role in the function of all other tissues, a complex connective tissue network signaling system integrating the body's mechanical forces may influence the function of all physiological systems. This provides further support for functional movement over extra-functional movement. Extra-functional movement will not “train” the correct connective tissue signal mechanisms relevant to functional movement.

## Current state of our knowledge

There is no debating that the passive human spine cannot carry very high loads without the complex integrative actions of other structures including muscles, neural and connective tissue. The notion that there is a distinct core group of muscles, and one or two muscles in particular – i.e., transversus abdominis and multifidus – that performs the spinal stabilizing role, is not supported by the literature.<sup>3</sup> Patients undergoing abdominal surgery, for example, do not appear to have a higher incidence of low back pain.

Using core stability and or strengthening exercises to change the firing pattern of a muscle, and expect a transfer to another dynamic context violates the basic training theory of specificity. The typical core stability exercise program contains movement patterns different from spine stabilization during standing, walking, running, etc. In addition, muscles change their contraction pattern following an injury as a protective strategy. Timing alterations in transversus abdominis seen in low back pain patients may be due to this protective mechanism. Muscle atrophy that is observed in the multifidus muscles of athletes may be due to a long-term maladaptive compensatory effect.<sup>29</sup> Full-blown overtraining takes a long time to manifest – sometimes a year or more. This may be the explanation for multifidus atrophy. The athlete has continued in a training program that is beyond current physiological capacity, has managed reasonably well for a period of time by using compensatory patterns of movements, before eventually experiencing low back pain. In attempting to prevent the athlete from damaging the back the stimulation of some muscles may have been reduced to restrict movement. When the athlete continues to train by using compensatory movements the increasingly weakened multifidus, and the compensatory actions of other muscles, eventually results in muscle imbalances that subsequently lead to low back pain.

In terms of performance enhancement, it is entirely possible that the lack of transfer of core stability/strengthening on sport performance is due to our primitive understanding about how force is transferred throughout the body. Knowledge in this area is still evolving. Langevin's proposal of connective tissue as a signal network, and the known plasticity of



connective tissue, leaves the door wide open for new and exciting ways of thinking about how to improve force transfer throughout the body.<sup>39</sup> This might indeed be the new frontier for high performance sport training. This stream of research potentially provides new insights into designing core stability and strengthening exercise programs for both rehabilitation and performance enhancement that are functionally relevant to the individual's current movement repertoire.

## Conclusion

- The most logical strategy for low back pain patients and athletes with low back pain is to withdraw from the stressors causing an overload on the spine. The non-athlete will benefit from gradual introduction to a sound overall physical fitness program to bring their physiological capacity up to the demands required by their most strenuous life activities.
- During the athlete's recovery from low back pain a focus on relearning correct movement technique, and using functional movements to correct muscular imbalances, will likely be more beneficial in the long term than learning new non-functional motor control patterns by participating in the typical core-stability/strengthening program.
- Athletes are probably wasting their valuable training time by including extra-functional core strengthening/stability training in their training routines. There is no transfer of this training to sport performance.

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## To the editor

The article by Christine Brooks in this edition of *Australasian Musculoskeletal Medicine* titled “On rethinking stability exercise programs” is about a subject that I have been aware of for some time and is of direct clinical interest to those in the musculoskeletal medicine field.

I would recommend the paper by Lederman (reference 3 in Dr Brook’s paper) for further insights into the lack of good evidence on this subject. In that paper Professor Lederman raises six assumptions made by the core stability enthusiasts:

- That certain muscles are more important for stabilization of the spine, in particular transversus abdominis;
- That weak abdominal muscles lead to back pain;
- That strengthening abdominal or trunk muscles can reduce back pain;
- That there is a unique group of “core” muscles working independently of other trunk muscles;
- That a strong core will prevent injury;
- That there is a relationship between stability and back pain.

Professor Lederman addresses them as being unproven and unlikely. Dr Christine Brooks in her paper takes this analysis a step further.

Her final comments are the most important take-home message for clinical use, with the general conclusion that a sound physical fitness program is the best option. I wonder whether “core stability” has really been taken as gospel by the University Queensland physiotherapists, and others, and promulgated almost without question!

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# Sacroiliac joint pain: Diagnosis and treatment

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## Introduction

Over the last two decades, the sacroiliac joint (SIJ) has increasingly been recognized as an anatomical source of pain that figures in the differential diagnosis of a patient presenting with low back pain (LBP) and/or buttock pain with or without more distant referred pain.<sup>1-7</sup> The SIJ is innervated and thus has the potential to be a source of pain.<sup>2,8-13</sup>

As SIJ pain refers into the buttock and iliac crest near the posterior-superior iliac spine, and also into the groin, abdomen, and leg including the foot,<sup>14,15</sup> it can be confused with referred pain from other sources, particularly from the lumbar intervertebral disc, lumbar zygapophysial joint (ZJ), hip joint and radicular pain. It is essential that the clinician recognizes these potentially confounding features and takes adequate steps to differentiate between SIJ pain and other pain presentations.

In the early 1900s, the SIJ was thought to be the principal source of LBP,<sup>16</sup> and an important cause of "sciatica".<sup>17</sup> Subsequently, and particularly after the discovery of the disc prolapse, it was considered that the lumbar spine and in particular the lumbar intervertebral disc was responsible for most back problems.<sup>17</sup>

Acknowledgement of the SIJ as a source of pain in the ensuing decades commenced in rheumatological literature, but this largely related to seronegative arthropathies, and case reports of various rare infections and tumours.<sup>18,19</sup> Subsequently there have been substantial developments in the basic sciences relating to the SIJ, initially and primarily in the osteopathic, physiotherapy, and chiropractic literature,<sup>18,19</sup> and later in biomechanical and radiological literature.<sup>20-26</sup> The advent of imaging-controlled diagnostic interventions has allowed for a more rational approach to diagnosis, and as a result there is again an increasing awareness that the SIJ is an important cause of LBP and referred pain into the pelvis and leg. It is now estimated that the SIJ may be the cause of 15-38% of all cases of LBP.<sup>4,27-30</sup>

While the SIJ can be a source of pain in various disease states, this article is restricted to mechanical disorders.

## Anatomy and biomechanics

The SIJ is the articulation between the triangular sacrum and the two ilia. It is a true diarthrodial synovial joint, and is unlike any other joint in the body. Only the ventral third of the joint is a true synovial joint

with a joint capsule and synovial cells;<sup>31</sup> the remainder is composed of three ligaments,<sup>32</sup> the ventral sacroiliac ligament, the interosseous sacroiliac ligament, and the posterior sacroiliac ligament.<sup>33</sup> From foetal life onward the iliac surface is fibrocartilage and the sacral surface is hyaline cartilage. Subsequent arthrosis of the joint tends to affect the fibrocartilaginous iliac side more than the sacral hyaline cartilage.<sup>34</sup> The dorsal transition between the ligamentous and synovial components shows marked individual variability including osseous clefts, cartilage and subchondral defects, and vascular connective tissue in the bone marrow.<sup>31</sup>

The articular cartilage of the SIJ does not appear to degenerate in a similar manner to other synovial joints in which articular cartilage defects result in bony ankylosis. The SIJ articular cartilage is maintained even in the elderly; it seems that fibrous tissues contribute most to ankylosis so that with aging there is interposition of fibrocartilage-like tissues within the joint (complete fibrous ankylosis).<sup>35</sup> Bony fusion seems to occur only in ankylosing spondylitis.<sup>36</sup> Degenerative changes and intra-articular SIJ ankylosis are substantially more common in men than women.<sup>36-38</sup>

The sacrum contains four foraminal pairs on either side, S1 to S4. Each pair has a ventral and dorsal aperture. On xray, the ventral component of the foramen is the most obvious; the smaller dorsal foramen can be difficult to visualize. It is important to recognize this when performing imaging-guided procedures into a sacral foramen. The dorsal component of each foramen may be difficult to visualize on a static image particularly because of the superimposition of the larger ventral component and from bowel gas. However, with the use of CT scan or more preferably a C-arm image intensifier, the dorsal component can be identified. With the C-arm, this is achieved by varying the amount of obliquity and observing that the more superficial dorsal component moves to a greater extent relative to the deeper ventral component (Figure 1).

The synovial part of the joint is more prominent caudally. At the level of the S1 foramen, the ventral 25% of the joint

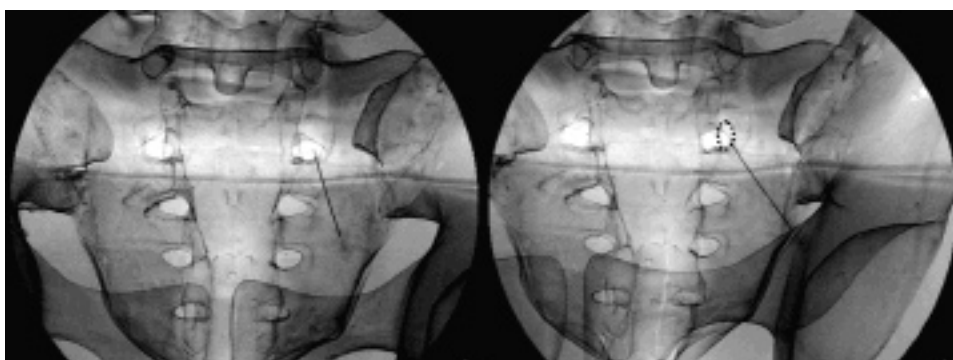
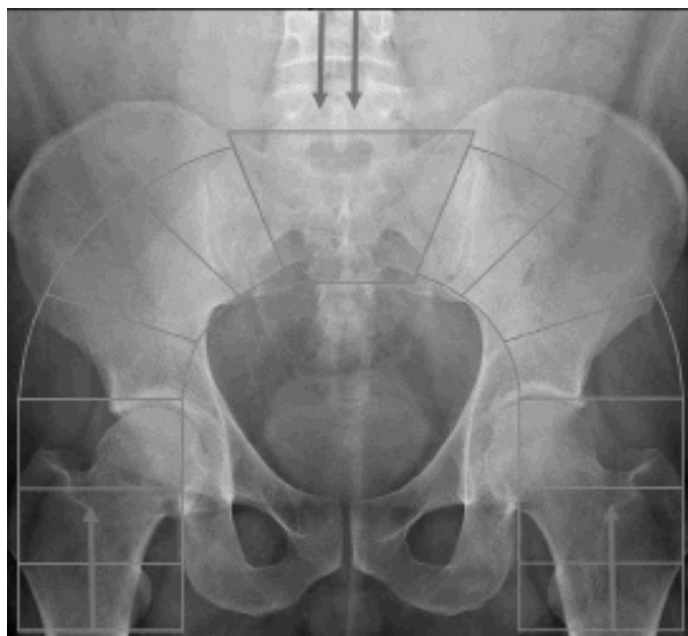


Figure 1





**Figure 2**

is synovial; at S2, the ventral 50% to 75% is synovial and at S3 100% of the SIJ is synovial.<sup>39</sup>

The pelvis is made up of three bones, with the sacrum positioned as the keystone in an arch from femur to femur (Figure 2). The stability of the sacrum within the pelvis is dependent on: (a) the shape and orientation of the sacrum and its articulations with the ilia, (b) the integrity of the ligamentous structure around the joint, and (c) the extent of muscular compression across the joint.

Variations in the shape and orientation of the sacrum and its articulations are rarely a problem except when these aberrations are quite extreme; for example, after pelvic fractures and in some congenital conditions. The ligamentous function of the SIJ is dependent on intact, stable ligaments and the orientation of the sacrum. The sacrum pivots in a sagittal plane around its true articular joint surface by 6–11°.

Tilting of the superior sacrum in an anterior direction is called sacral nutation. This is its normal position, encouraged by the lumbar lordosis. Excess nutation can occur. Superiorly, such excess nutation is limited directly by the deep interosseous and long dorsal ligaments and indirectly by the ilio-lumbar ligament. Inferiorly, it is limited by the sacrococcygeal and sacrotuberous ligaments.

If all of these ligaments are intact, nutation of the sacrum has the benefit of helping to pull the pelvic ring closed, thus compressing the SIJs. This, the so-called “locked” position of the pelvis, has great biomechanical strength with force transfer occurring primarily through a well-supported and mechanically advantaged joint surface and its ligaments. This passive locking mechanism, dependent on the shape of the keystone and the integrity of the articular capsule and surrounding ligaments, is called form closure of the SIJ.

Conversely, tilting of the superior sacrum in a posterior direction is called counter nutation. It has the effect of

“unlocking” the pelvis and creating a loss of passive compression across the SIJs.

The ligaments and capsule of the SIJ cannot, however, provide adequate compression across the joint surface on their own. The primary compressors of the SIJs are muscular; their actions are termed force closure of the SIJ. Three main muscle groups have been identified: (a) muscles of the pelvic floor, (b) transverse abdominus, and (c) a posterior sling consisting of latissimus dorsi through the thoracolumbar fascia to the contralateral gluteals.<sup>20–22,40–49</sup>

## Innervation

Sacroiliac joint innervation is important as the only method for making a diagnosis of SIJ origin pain is an anaesthetic block of the joint or its nerve supply, and one possible<sup>46–49</sup> method of treatment is radiofrequency (RF) treatment directed at target nerves. The SIJ is definitely innervated and it can be a source of pain. The periarticular tissues of the SIJ contain mechanoreceptors and nociceptors.<sup>2</sup>

Nerve fibres varying from 0.2 micron to 2.5 microns in diameter end in five morphologically different terminals and these terminals are present in the SIJ capsule and adjacent ligaments.<sup>9</sup> Substance P and calcitonin gene-related peptide (CGRP) immunoreactive nerve fibres have been found in the anterior SIJ capsule and interosseous ligament,<sup>13</sup> the superficial layer of sacral and iliac cartilage, and the surrounding ligamentous structures.<sup>12</sup>

Nerves supplying the SIJ are distributed not only to the superficial and deep dorsal sacroiliac ligaments, but also to the sacrotuberous and sacrospinous ligaments; the dorsal rami continue their course laterally, sandwiched between superficial and deep portions of sacroiliac ligaments, and pierce the origin of the gluteus maximus muscle.<sup>50</sup>

It is considered that the synovial component of the SIJ has a different innervation to the posterior ligamentous component. The synovial joint is likely to be innervated mainly by ventral sources;<sup>8,9</sup> its upper ventral portion is innervated mainly by the L5 ventral ramus and the lower ventral portion by the S2 ventral ramus or by branches from the sacral plexus.<sup>9</sup> The synovial component has minimal innervation by the sacral dorsal rami.<sup>51</sup>

The dorsal sacroiliac ligaments are innervated by at least the L5 dorsal ramus and lateral branches of the S1–S3 dorsal rami. The L4 medial branch may be involved. The upper dorsal ligamentous structures are innervated by the L5 dorsal ramus; the lower dorsal ligaments by nerves arising from a plexus composed of lateral branches of the dorsal rami of the sacral nerves.<sup>9</sup> These nerves range from 0.292 mm to 0.997 mm in diameter, and the nerves supplying both the synovial and ligamentous components of the SIJ complex have similar diameters.<sup>9</sup>

The lateral branches of the sacral dorsal rami emerge from the sacral foraminae in a varied array, radiating cephalad, transverse or caudad.<sup>51</sup> Once they have emerged they do not run in a constant plane,<sup>11,51,52</sup> but run across the dorsal sacrum either through, superficial to, or deep to the dorsal sacroiliac ligament at a variable depth of up to 1 cm superficial to bone (Figure 3).<sup>51,53</sup>



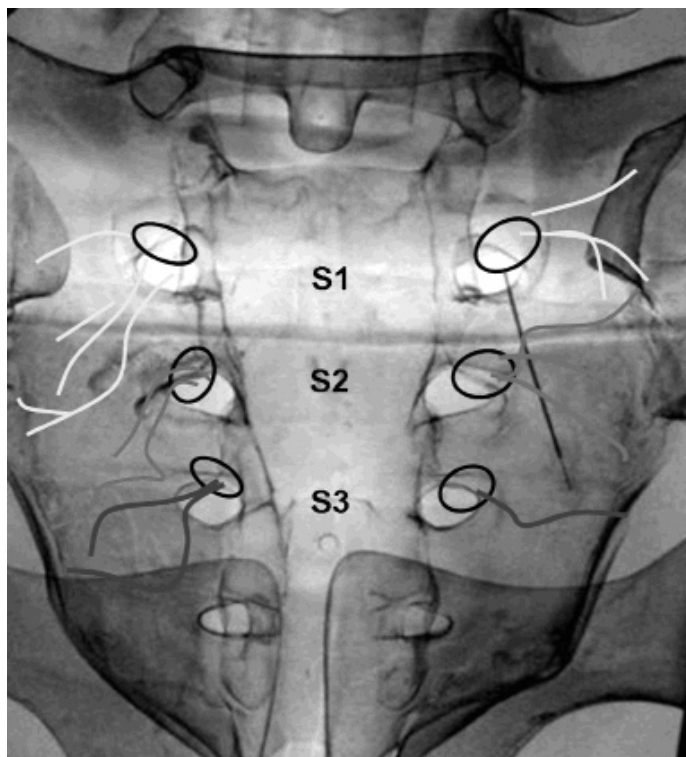


Figure 3

## Pathophysiology

As noted above, the SIJ is innervated and has the potential to be a source of pain. It can become painful as a result of both intrinsic and extrinsic factors.

Intrinsic mechanisms include definitive biomedical processes such as sacroiliitis and tumors. These constitute red flag conditions and are not covered in this article. The other intrinsic mechanism considered to be a risk factor in SIJ origin pain relates to aberration of biomechanical function. The technical terms used to describe these biomechanical features are form closure and force closure. Poor form closure of the SIJ is caused by inefficient bony structure/alignment, absent or stretched SIJ ligaments, or sacral counter nutation. Poor force closure is considered to arise through pain inhibition and poor firing of the compressive muscles (Figure 4).<sup>54-60</sup> It is likely that long-term lack of force closure across the SIJ can lead to increased strain on the ligamentous structures, which, over time may lengthen and

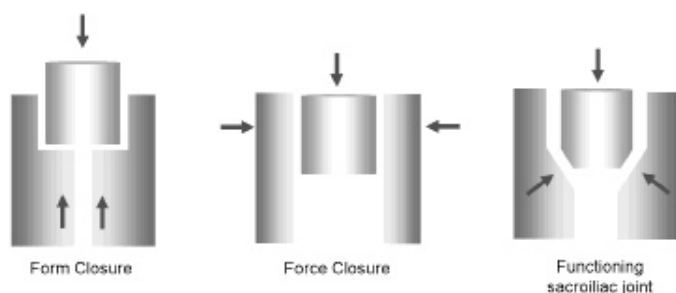


Figure 4

cause further loss of pressure across the joint. Such a joint may then be resistant to muscular retraining.

Extrinsic mechanisms causing loss of form or force closure include trauma (macro or repetitive microtrauma), infection and pregnancy.<sup>20,40,56,61-66</sup>

Pain in the region of the SIJ is not uncommon after posterior iliac graft harvesting.<sup>67</sup> It occurs in 6-39% in patients who have had iliac grafting for spinal fusion.<sup>68,69</sup>

SIJ pain is also not uncommon after spinal fusion. The prevalence of SIJ pain, diagnosed on the basis of 75% or more pain relief with local anaesthetic block, in one group of patients with significant and chronic LBP post-lumbar fusion surgery was found to be 35%,<sup>70</sup> a figure that seems to be similar to that in other reports.<sup>71</sup>

In a study of long-term outcomes from SIJ radiofrequency neurotomy (RFN), SIJ pain was considered to be idiopathic in 30% of cases, and to derive from motor vehicle accidents in 9%, from a fall or slip in 24%, from overload or a work injury in 26% and from other incidents in 11% of cases.<sup>72</sup>

## Clinical presentation and diagnosis

The diagnosis of SIJ pain is predicated on diagnostic anaesthetic injection as clinical and radiological tests have been found to be inaccurate. The most significant feature on clinical assessment of a patient considered to have SIJ origin pain is the site of pain; if the patient points to the posterior superior iliac spine (PSIS) then the pain is more likely to derive from the SIJ.<sup>28</sup> Clinical examination has possible utility when examination findings are considered collectively. Radiological findings are of no particular use other than in the exclusion of some red flag conditions.

The International Association for the Study of Pain (IASP) has proposed three criteria for the diagnosis of SIJ pain.<sup>73</sup> In particular, diagnosis requires that:

- The patient has pain in the region of the SIJ
- The patient's pain is reproduced by clinical tests that selectively stress the joint, and
- The patient's pain is completely relieved by selective delivery of local anaesthetic.

## Site of pain

In asymptomatic subjects, noxious stimulation of the SIJ evokes pain in the low back, buttock, and upper posterior thigh.<sup>4</sup> In patients with SIJ pain established by fluoroscopically guided SIJ injection, 94% described buttock pain, 72% described lower lumbar pain, 14% described groin pain, 50% described associated lower-extremity pain, 28% described leg pain distal to the knee, and 14% reported foot pain.<sup>14</sup> SIJ pain rarely extends above L5.<sup>28</sup> SIJ origin pain has a similar referral pattern to pain derived from the lumbar spine and from the hip joint,<sup>14,28</sup> and thus, analysis of pain patterns is not in itself a reliable diagnostic factor.<sup>74</sup>

## Type of pain

SIJ pain can present with local PSIS pain and/or somatic referred pain. Local SIJ region pain can be deep and aching,

but it can also be sharp and activated by movement. Somatic referred pain is generally described as diffuse, aching and poorly localized. It is different to typical lumbar radicular pain, which is generally described as long, thin sharp lancinating pain that can concentrate distally.<sup>75</sup> Because SIJ referred pain can extend into the leg it can be confused with radicular pain, potentially leading to unnecessary spinal treatment.<sup>17,76</sup> If the predominant pain is radicular, it is most likely to arise from the lumbar spine due to disc prolapse or canal stenosis. However, it is not impossible for lumbosacral radicular pain to be caused by SIJ pathology. In one series, ventral capsule disruption was present in 70% of patients diagnosed with SIJ pain by intra-articular block.<sup>30</sup> Joint injury, if associated with inflammation, can theoretically be associated with extra-sacral peri-neural inflammation and pain.

It is also important to distinguish neuropathic pain from somatic referred pain. Neuropathic pain typically presents with descriptors such as burning, buzzing and tingling, and has clinical features including allodynia. It can occur in association with somatic referred pain.<sup>75,76</sup> Although uncommon, lumbo-sacral plexopathy, which is defined as neurological deficit derived from the lumbo-sacral plexus, is more common after sacral fractures than among the entire population of patients with pelvic and acetabular fractures.<sup>77</sup> Thus, it stands to reason that SIJ trauma and pain can be associated with local neural damage, and hence, neuropathic pain.

## Clinical examination

The validity of physical examination tests is reduced because they tend to stress adjacent soft tissue structures as well as the lumbar spine and hips.<sup>61</sup>

Singular examination tests such as palpation and movement tests are generally considered to be unhelpful in the diagnosis of SIJ pain.<sup>27,61,78,79,80</sup> However, combined tests may be useful. Synovial SIJ pain can be predicted when three of five pain provocation tests are positive with a sensitivity and specificity of 91% and 78%, respectively, and a consequent likelihood ratio of 4.1.<sup>81-86</sup> The specificity improves with the absence of centralization of pain. The tests used are:

- The distraction test
- Posterior pelvic pressure provocation (P4)
- Gaenslen's test
- The compression test, and
- The sacral thrust.

It is unknown whether these tests can predict ligamentous sources of SIJ pain; clinical examination has not been assessed using ligament injection as the criterion standard. These tests individually can be positive in up to 20% of the asymptomatic population.<sup>87</sup>

The SI joint is more likely to be the source of pain if

- a) In identifying the site of pain, the patient points to the posterior superior iliac spine (buttock dimple)
- b) Pain is predominantly below the L5 level<sup>28</sup>
- c) The sacral sulcus is tender.

Tests for SIJ instability have been proved reliable particularly in the post partum population, but there are no data supporting their efficacy in the management of SIJ pain except in the post-partum population.<sup>44,45,66,88-90</sup>

Multiple authors raise the concept of SIJ dysfunction, where the self-locking mechanism of the SIJ complex fails due to a loss of form and/or force closure.<sup>20-22,40-49,54-57,59,62-66,88-92</sup> The tests used to assess SIJ dysfunction are reliable and valid.<sup>56-59</sup> The presence of SIJ dysfunction is proposed by these authors as a putative cause of SIJ pain. However, although outcome studies on treating this SIJ dysfunction show significant improvements in disability, changes in pain are less impressive.<sup>54,57,91,93</sup> A recent study has, however, shown a compelling pain response in 50% of patients.<sup>94</sup>

## Imaging

The diagnosis of SIJ origin pain is difficult because there are no valid or reliable correlations between imaging changes and SIJ pain. Imaging therefore cannot be used as a criterion standard for diagnosis or as a basis upon which to assess the validity of treatment. In one study diagnostic CT-guided intra-synovial SIJ injections had a sensitivity of 57.5 %, a specificity of 69% and a consequent poor likelihood ratio of 1.9,<sup>68</sup> thus negating the use of CT in a presentation of putative SIJ origin pain except to rule out red flag conditions. Bone scan has a very low sensitivity but a high specificity for SIJ pain diagnosed with diagnostic blocks, and is thus not worth performing.<sup>95</sup> Similarly, plain radiography and MRI cannot reliably detect non-red flag SIJ origin pain.<sup>28</sup>

Changes are often noted on imaging but they are not clinically significant. For example, the CT appearance of the SIJ is closely related to the patient's age, gender, BMI, and, in women, parity.<sup>96</sup> The widths of the SIJ space and of the subchondral sclerosis on the iliac and sacral sides narrow over time; they were measured to be 2.3+/-0.4 mm, 2.5+/-1.6 mm and 1.4+/-0.5 mm, respectively, in patients younger than 40 years of age and 1.9+/-0.2 mm, 3.6+/-2.1 mm and 2.3+/-1.1 mm, respectively, in patients older than 40 years of age.<sup>96</sup> SIJ changes include increased joint space narrowing and loss of joint space uniformity. Subchondral sclerosis appears to be wider and less uniform in the elderly.<sup>96</sup> Osteophytes are present even in younger patients and their prevalence increases with advancing age.<sup>96</sup> CT has identified six anatomical variants termed accessory joints (19.1% of assessed SIJs), "iliosacral complex" (5.8%), bipartite iliac bony plate (4.1%), crescent-like iliac bony plate (3.7%), semicircular defects at the sacral or iliac side (3%), and ossification centres (0.6%).<sup>97</sup>

## Diagnostic injections

Properly conducted SIJ injection is considered the criterion standard diagnostic technique.<sup>27</sup> There are a number of intricacies and subtleties in this diagnostic approach to SIJ pain that need to be understood. Diagnostic injections can be performed using various forms of imaging guidance such as C-arm fluoroscopy, ultrasound and CT. More recently, image fusion, in which a software technology matching real-time ultrasonography and a previously obtained CT,

has been tested and found to be accurate, but it is slow, taking on average about 20 minutes.<sup>98</sup> MR-guided sacroiliac and other spinal injections can also be performed in open high-field MRI using fast TSE sequence designs.<sup>99</sup> However, although there was a reported accuracy of drug delivery of 100% for nerve root injections, the accuracy for ZJ and SIJ delivery was only 87%, and the average time taken was 29 minutes (range 19-67 minutes).<sup>99</sup>

### Site of injection

There are two components to the SIJ and thus it appears that SIJ pain can be established as the likely source of pain only if both components are assessed. The need to assess both components, however, should be predicated on the treatment that might ensue from such a diagnostic approach. As discussed earlier, the ligamentous component of the joint is innervated by at least the dorsal ramus of L5, and certainly by the lateral branches of the dorsal rami that emerge from the S1, S2, and S3 foraminae. If the treatment is to be RFN of these nerves, then it stands to reason that the diagnostic injection should be directed at these nerves. As the synovial SIJ is innervated ventrally, nerve blocks cannot be used as a diagnostic test for synovial SIJ pain; lateral branch blocks do not anaesthetize the synovial SIJ.<sup>51</sup> The only method that can be used to diagnose synovial SIJ pain is intra-articular injection. This might be used as a test if steroid or other material is to be injected into the synovial component, or if a surgical procedure such as SIJ is considered relevant.

As the sacral lateral branches run at a variable depth and have a variable course over the sacrum, it is recommended that these nerves be blocked using multi-site, multi-depth sacral lateral branch blocks, as this method renders the interosseous and dorsal sacral ligaments insensate in 70% of subjects.<sup>51,53</sup> Another option to lateral branch blocks is to inject into the ligamentous component of the SIJ itself using contrast to exclude extraneous injection.

### Number of injections

Are controlled blocks required? In high prevalence conditions, such as in the search for ZJ pain in a population of post-whiplash neck pain patients, it has been established that double-blocks are required, one with a short-acting anaesthetic and another with a longer-acting anaesthetic.<sup>100,101</sup> As the prevalence of an index cohort decreases within a tested population, the chances of false-positive findings with single-blocks rises substantially. In such circumstances, triple-blocks may be necessary, with the addition of a placebo arm. The use of a control block is recommended by Hansen, et al., as their review of two studies with 54<sup>102</sup> and 120<sup>103</sup> patients found a false-positive rate for the technique of 20-22% for a single block.<sup>104</sup> On the other hand, Mitchell, et al. assessed 1146 consecutive double-block technique combined intra-articular and ligamentous injections over a 2.5-year period and found that the first block predicted the control block result in 85% of cases for a positive block and in 87% for a negative block.<sup>105</sup>

### The use of contrast

Contrast is required in all instances, as it confirms that the injectate is in the joint or ligament or adjacent to the nerve, and it excludes intra-vascular injection or extravasation into surrounding tissues. Ventral extravasation has been reported in as many as 61% of all SIJ injections;<sup>3</sup> if so, false positive blocks occur due to the close proximity of sheaths of the adjacent nerve trunks or roots, including the lumbosacral trunk and the L5 and S1 nerve roots.<sup>80</sup> Thus, when local anaesthetic is injected into the SIJ, a possible short-term complication is leg weakness for the duration of the local anaesthetic action. Any injection into the SIJ without contrast, even under CT, should be viewed with scepticism at least in respect of its diagnostic utility.

### Volume

In intra-articular SIJ injection the accepted maximum volume is 2.5 ml,<sup>51</sup> but less should be injected if there is increased pain or pressure. Ligamentous injection volume is about 2 ml. The multi-site multi-depth injections onto the lateral branches of the dorsal rami from S1 to S3 require 0.2 ml per infiltration.<sup>51</sup>

### Interpretation

In research, it is generally considered that the criterion for a positive block should be 100% pain relief or very close to it. In clinical practice, reduction of VAS in the order of 80% may be considered a positive finding.<sup>16</sup>

### Summary

Pain from either or both components of the SIJ can be suspected when the presenting pain concentrates over the SIJ. A combination of physical tests may predict a positive diagnostic block regime. Imaging tests are unhelpful in diagnosing such pain, but when indicated may be helpful in excluding red flag conditions. Controlled blocks are used as the criterion standard for diagnosis.

## Treatment

A number of modalities are available for the treatment of putative SIJ pain. At present, however, the literature regarding the efficacy of each is limited.

By the time a target-specific diagnosis is made by injection, it is likely that the patient will have failed numerous trials of conservative management including physical therapy, medications, bracing, kinesiology and exercise, and it is assumed this includes targeting specific retraining of the pelvic floor, transverse abdominus and the posterior sling. The diagnostic injection into the joint or the dorsal interosseous ligament typically includes cortisone and may thus be therapeutic in itself. If it is not, percutaneous radiofrequency neurotomy (RFN) can be considered. Stabilization of the joint through prolotherapy or fusion may also be appropriate.



## Efficacy

### **Corticosteroid injection**

Sacroiliac corticosteroid injections have not been tested with randomized controlled trials (RCTs). Accordingly, a systematic review concluded that the evidence supporting therapeutic injection is limited.<sup>104</sup> However, many case series studies report that intra-synovial SIJ corticosteroid injections provide good to excellent pain relief with a duration of up to 10 months.<sup>32</sup>

A retrospective practice audit of 155 patients who underwent diagnostic and therapeutic fluoroscopically guided contrast confirmed SIJ injections with local anaesthetic and corticosteroid were considered positive if they produced 50% or greater pain relief during the local anaesthetic block phase and if there was two weeks or more subsequent pain relief. Of the 155 patients, 69 (45%) had had previous lumbar surgery and 120 (77%) were positive responders over a mean duration follow-up period of 44 months (range 26-101 months).<sup>106</sup> The positive responders received a mean of 2.7 injections per patient; 40 required one injection only, 29 required two, 22 required three, and 27 required four or more. The mean duration of response for those receiving more than one injection was 9.3 months per injection (range 1-58 months). There were no adverse events.

In practice, corticosteroid and other injections can be inserted into either or both the synovial or ligamentous component of the SIJ. Intra-synovial etanercept is now injected in the treatment of ankylosing spondylitis. A case series reported that it improved both clinical features and morphological parameters significantly, and that it was safe and cost-effective.<sup>107</sup>

### **Radiofrequency neurotomy**

Sacroiliac treatment using cooled RFN has been assessed positively with a RCT.<sup>108,109</sup> In this trial, 28 patients were equally divided into treatment with cooled RFN and sham treatment groups. Patients in the treatment group received denervation of the L4 medial branch, the L5 dorsal ramus and at the S1-S3 lateral branches. The proportion of participants experiencing greater than 50% pain relief at one, three, and six months postoperatively in the treatment cohort was 79%, 64%, and 57%, respectively; in the placebo group, the proportion of participants experiencing greater than 50% pain relief at one and three months postoperatively was 14% and 0%, respectively. Subsequently, 11 patients crossed over to RFN treatment, and of these, at one, three, and six months the proportion of patients reporting improvement was 64%, 55%, and 36%, respectively. The treatment effect was seen to diminish by 12 months; at that time only 14% (two patients) of the treatment group had persistent pain relief. It was suggested that larger studies were needed to further assess the efficacy of SIJ RFN.

The same group reported on 77 patients who underwent lateral branch SIJ RFN treatment using a 50% reduction in pain at six months as a successful outcome. Of these, 40 (52%) obtained a positive outcome. The multivariate analysis found that predictors of an unsuccessful outcome were age older than 65 years and pain radiating below the

knee. They also noted that cooled, rather than conventional RFN, was associated with a higher percentage of positive outcomes and that no single clinical variable reliably predicted treatment results.<sup>110</sup>

Previous case series had indicated that SIJ RFN may have a role to play in the treatment of SIJ pain. The first main paper describing SIJ RFN used a stereotactic technique. After displaying the anatomy of the lateral branches of the sacral dorsal rami, Yin, et al. reported in a retrospective audit that 64% of 14 patients treated with SIJ RFN reported successful outcome for at least six months, with 36% achieving total relief.<sup>111</sup> Kapural, et al. performed a retrospective chart review on their initial cases using the cooled RFN technique; short-term efficacy was apparent as 18 out of 26 cases were good at three months.<sup>112</sup> They also performed a safety audit on the first 100 cases. There were no significant complications other than short term (less than six weeks) pain exacerbation four cases, and in two cases there was an area of cutaneous numbness over the buttocks.<sup>112</sup>

Mitchell, et al. performed a prospective consecutive case series on 82 cases using a traditional RFN method directed at the L4 medial branch, the L5 dorsal ramus and the S1-S3 lateral branches.<sup>11</sup> The cases were divided equally into 5-7 months and 8-13 months follow-up. Greater than 50% pain relief was achieved in 22% of patients in the 5-7 months group and 42% of patients in the 8-13 months group. Patient satisfaction was 58% versus 63% in the respective groups. At eight months, the average reduction in VAS was 33%, with the 42% of patients with greater than 50% relief reporting an average reduction of 74% in VAS.<sup>105</sup>

Outcomes from treating the SIJ complex with traditional RF needles are dependent to some degree on the cause of the original injury; patients able to identify the cause of their injury (whether it be from a motor vehicle accident, fall, overload/work injury, etc.) are more likely to report excellent pain relief following RFN than those unable to pinpoint the mode of injury.<sup>72</sup>

While sacral RFN is a safe procedure, there has been a case report of a permanent L5 sensory radiculopathy following a bilateral L3 to L5 RFN.<sup>113</sup> However, such a complication should arise only from inaccurate positioning of the needle.

### **Prolotherapy**

Prolotherapy for LBP including SIJ region pain and tenderness appears to be no better than placebo. In a RCT, Yelland, et al. tested patients with LBP that might have included putative SIJ pain with injection onto any local tender structure either with a combination of 20% glucose and 0.2% lidocaine or with saline.<sup>114</sup> The outcome was that prolotherapy was equally effective as placebo but that both seemed to be somewhat effective in that in 46% of cases there was a 50% pain reduction and 42% of cases there was a 50% reduction in Roland-Morris disability index.

In a descriptive prospective trial case series study, Cusi, et al. examined functional outcome measures and improvements in load transfer on clinical examination but not pain after CT-guided injections of 50% glucose on



three occasions six weeks apart.<sup>91</sup> Functional improvement occurred in 76% of patients at 3 and 12 months follow up and in 32% at 24 months follow up.

In a small case series of prolotherapy on the ligaments of the SIJ, Mitchell, et al. found 86% patient satisfaction, with 50% of patients having an average reduction of pain of 64%. 64% of patients reported feeling stronger.<sup>94</sup> In a much larger cohort (N=77) with longer-term follow-up, as yet unpublished data showed 71% of patients felt stronger, 38% of patients had less than 75% pain relief with a further 22% having less than 50% pain relief. Interestingly, pain relief was highly correlated with improvements in stability. 76% of patients were satisfied.

### Surgery

Surgery with SIJ arthrodesis (via a modified Smith-Petersen technique) for putative SIJ pain, diagnosed by pain relief with intra-articular joint injections under fluoroscopic guidance, has been studied and found in one series to be reasonably successful in terms of physical functioning, role physical, bodily pain, vitality, social functioning, role emotional, and neurogenic and pain indices.<sup>115</sup>

A case series on 15 consecutive patients treated with percutaneous SIJ fusion using hollow modular anchorage screws filled with demineralized bone matrix after diagnosis with a single SIJ injection of local anaesthetic and steroid under image intensifier control reported that at a mean follow-up of 17 months there was a significant improvement in disability, physical function, and pain; of the 15, 13 reported good to excellent improvement.<sup>116</sup>

However, a more sobering picture emerges from a retrospective study on 17 patients who underwent bilateral SIJ fusion with internal fixation and decortication of the SIJ after diagnosis via local anaesthetic joint infiltration, temporary external fixation or bone scan as at an average follow-up period of 39 months only three patients (18%) reported moderate or absent pain. The rest had either marked or severe pain.<sup>117</sup>

Another much less invasive technique, SIJ debridement, was retrospectively studied on 38 patients with SIJ pain diagnosed with SIJ injection. At a follow-up period of two years 61% had 50% or more reduction in pain and 53% had 75% or more reduction in pain.<sup>118</sup>

Additionally, percutaneous, CT-guided stabilization from S1 to S2 has been reported to be a potentially acceptable treatment for recalcitrant SIJ pain.<sup>119</sup>

### Neuromodulation

Neuromodulation with spinal cord stimulation, peripheral nerve stimulation,<sup>120</sup> or sacral nerve stimulation can potentially relieve persistent pain from the SIJ that is recalcitrant to other therapies.

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# An introduction to neurotrophins and semaphorins with respect to internal disc disruption

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## Abstract

Chronic lumbar spinal pain is a common clinical problem with the underlying pathology being internal disc disruption in many such cases. This is associated with neo-innervation and neo-vascularization of the inner parts of the disc. The biological signals involved in this process belong to a group of proteins called semaphorins and neurotrophins. This paper reviews the literature on these proteins based on a PubMed, Scirus, and Google search.

A neurotrophin called nerve growth factor is present in the intervertebral disc in low levels and is upregulated following injury and by inflammation, with an increase in nociceptive neurons. Brain-derived neurotrophic factor is also upregulated by inflammation and this can lead to neo-vascularization and neo-innervation. Whilst glial-derived neurotrophic factor is increased in the dorsal root ganglion following disc inflammation, the implication of this is unclear. Finally a semaphorin known as Sema3A, which has an inhibitory effect on neuron growth, is reduced in the disc following injury and this may tip the balance towards neo-innervation.

## Introduction

Chronic lumbar spinal pain due to internal disc disruption (IDD) is a common problem in clinical practice, with varying published prevalence figures of between 39-56% using the gold standard of provocative discography.<sup>1,2</sup>

The primary pathological process is considered to involve a disruption of the structure of the annulus fibrosis (AF) and it also may involve the vertebral endplate. The AF disruptions are described as radial or circumferential tears. In patients with chronic discogenic back pain the distinctive histological finding is an ingrowth of vascularized granulation tissue extending from the outer AF into the nucleus pulposus (NP).<sup>3</sup> This can result in the ingrowth of nociceptive nerve fibres, either independently or associated with blood vessels, into the inner parts of the disc.<sup>4</sup>

At least two basic questions arise from consideration of the above:

1. What biological signals are involved in this process of neo-innervation and neo-vascularization?
2. Is there a method of preventing such neo-innervation to prevent the onset of chronic discogenic pain?

This article provides an introduction to these signals which are known as neurotrophins and semaphorins with respect to IDD.

## Methods

A search of PubMed, Scirus, and Google was performed using the keywords "neurotrophins", "semaphorins",

"neurotrophins and back pain", "semaphorins and back pain". Where reference was made to relevant articles within the primary articles located by the search, these articles were also sourced and reviewed.

## Definitions

**Neurotrophin:** A group of proteins that promote neuron growth, development, and function. In generally accepted usage it consists of a family of four structurally related proteins. These are nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), neurotrophin-3 (NT-3), and neurotrophin-4/5 (NT-4). However, a further group can be included in this definition and these are known as the glial cell line derived neurotrophic factors. This group consists of glial cell line-derived neurotrophic factor (GDNF), neurturin (NRTN), artemin (ARTN), and persephin (PSP).

**Semaphorins:** A class of secreted and transmembrane proteins that generally deter neuron growth. They also function in other systems such as the immune system, in bone growth and the cardiovascular system. There are eight major classes, with only four occurring in vertebrates. One of the most studied is Sema3A. This repels axons from the dorsal root ganglia (DRG) and some cranial nerves.

**Tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ):** A cytokine involved in inflammation, released mainly by activated macrophages, but also by mast cells, fibroblasts, and neurons.

**Interleukin 1 beta (IL-1 $\beta$ ):** A cytokine that is an important mediator of the inflammatory response to infection and injury. It is produced by activated macrophages.

## Discovery Neurotrophins

The idea that there was a trophic substance guiding the growth of axons has been around since the early 1900s.<sup>5</sup> In the 1930s Viktor Hamburger demonstrated that a limb bud transplanted into a 2.5-day embryo chicken resulted in an increase in the number of neurons in the DRG, ventral horn and sympathetic system, whereas removal of a limb bud had the opposite effect.<sup>6</sup>

Further progress occurred in 1948 with the observation that mouse sarcoma cells transplanted into chicken embryos induced sensory innervation from the adjacent dorsal root ganglion into the neoplastic tissue.<sup>7</sup>

Other embryonic tissues also developed increased innervation indicating that there was a soluble and diffusible factor present. Changing to tissue culture methods showed this clearly with sensory or sympathetic ganglia developing a halo of nerve fibres growing towards the nearby mouse sarcoma tissue. Even an extract of minute dilution had an effect on the growth of neuron projections.<sup>7</sup> This discovery and further work was rewarded with the Nobel prize in medicine in 1986 for those involved – Rita Levi-Montalcini and Stanley Cohen.

The next step was to identify this “nerve growth factor” as it was called. To determine the nature of this factor in 1953 Stanley Cohen used snake venom to degrade any nucleic acids present, and to his astonishment found that snake venom alone induced an enormous outgrowth of nerve fibres.<sup>8</sup> Using the idea of homologous organs the search returned to the mouse, where the similar organ is the salivary gland, and it too was found to harbour the same compound in large quantities.<sup>8</sup>

This was a milestone in neurobiology research – a compound secreted by tissues that could attract nerve fibres. It was called nerve growth factor (NGF) and proceeding research using knockout studies and anti-NGF antibodies in mice have shown it is the trophic factor for sensory and sympathetic neurons,<sup>9</sup> the implication being that there should be others for motor nerves. NGF is also an important component in the immune system and contributes significantly to the process of inflammation.<sup>10</sup>

Brain-derived neurotrophic factor (BDNF) was the next to be found after about 10 years of searching<sup>11</sup> and more recent additions include neurotrophin-3 (NT-3) in 1990<sup>12</sup> and neurotrophin 4/5 (NT-4/5) one year later.<sup>13</sup> BDNF is involved in the differentiation and survival of sensory nerves, regulation of nociceptive function and modulation of hyperalgesia. It also can induce the formation of blood vessels.<sup>14</sup> NT 4/5 is the trophic factor for motor nerves and NT-3 for proprioceptive fibres.<sup>15</sup>

The structure of the four neurotrophins is very similar between species, and they share a common set of receptors. A receptor called p75 is activated by all four and generally leads to apoptosis (cell death). Trk-A ( tyrosine kinase A – verbalized as “track-A”) is activated by NGF, Trk-B by BDNF, and Trk-C by NT-3.

To complicate matters, however, a further series of

neurotrophic factors called glial cell line-derived neurotrophic factors have also been described. These also have a trophic effect on motor, sympathetic, and sensory neurons. The first found was glial cell-derived neurotrophic factor (GDNF) in 1993,<sup>16</sup> followed by neurturin (1996)<sup>17</sup> and artemin (1998).<sup>18</sup> Persephin was also found in 1998.<sup>19</sup> The receptors for this class are called glial cell line-derived neurotrophic factor family receptors  $\alpha$  (GFR $\alpha$ ) 1-3. GDNF may have a role to play in maintenance of chronic pain relating to IDD but there is little in the literature about the other three with respect to this.

## Semaphorins

The first indications of compounds that could cause collapse of neuron growth cones were reported in 1990. An extract of chick embryo brain was found to cause the collapse of dorsal root ganglion (DRG) growth cones.<sup>20</sup> The grasshopper embryo, however, provided a better model and in 1992 fasciclin 1V was reported. It was found to repel neuronal growth cones in the developing limb buds. It was soon renamed semaphorin 1 and additional semaphorins were found in various species including the fruit fly, humans, and chickens.

Since then about 30 different semaphorins have been described and they have been divided into eight classes. Some are from nonvertebrate or viruses, but the vertebrates seem to have 20 different semaphorins.<sup>21</sup> They can be either secreted or membrane bound, and all have the characteristic of a 500 amino acid block called a sema domain.<sup>22</sup> The cell membrane receptors for semaphorins are called plexins and neuropilins.

Semaphorins not only affect nerve growth but they also have a major role in immune function and bone development. The main semaphorin of interest is called Sema3A and this has been shown to repel axons from the DRG and several cranial nerves. The neuronal growth cone arresting action of semaphorins seems to be related to the opening of Ca channels in the cell membrane.<sup>23</sup>

## Does it all start with a tear?

For the initiating event of structural failure in the annulus fibrosis to become clinically relevant, the tear needs to extend towards the periphery and possibly also involve endplate disruption.<sup>24, 25</sup>

Following this, an inflammatory phase ensues with the entry of various immune system cells including T lymphocytes which release various cytokines and TNF- $\alpha$ . Mast cells and macrophages also increase in the damaged area some time later.<sup>26</sup> Granulation tissue forms with neo-innervation and neo-vascularization.<sup>4</sup>

With regards to this process, what roles do the neurotrophins and semaphorins play? At present there is information for only some of them, these being NGF, BDNF, GDNF, and Sema3A.

## 1. NGF

- This is detectable in both the AF and NP at low levels in normal non-painful discs.<sup>27</sup>
- AF cells in the presence of macrophages secrete TNF- $\alpha$ , other cytokines, and nitric oxide.<sup>28</sup>
- AF injury results in increased release of IL-1 $\beta$  and TNF- $\alpha$ .<sup>29</sup>
- Patients with discogenic lumbar spinal pain have higher levels of NGF in the discs compared to discs that are not painful.<sup>30</sup>
- Discs excised with high intensity zones on MRI have prolific small round cells and fibroblasts positive for TNF- $\alpha$ .<sup>29</sup>
- NGF gene expression in the NP is stimulated by the inflammatory cytokines IL-1 $\beta$  and TNF- $\alpha$ .<sup>31</sup>
- Mast cells and macrophages are abundant in the zone of granulation tissue but not in the aging disc or normal disc<sup>32</sup> and these cells release NGF and cytokines.<sup>33</sup>
- Extracted medium from NP and AF of patients with painful intervertebral disc (IVD) promotes axonal growth and production of substance P. Anti-NGF treatment reduces this axonal growth.<sup>34</sup>
- The neurons innervating the disc are calcitonin gene related peptide-immunoreactive. These are known to be nociceptive. Disc inflammation results in an increase in these NGF-dependent neurons.<sup>35</sup>

Thus, it seems that NGF present in the disc normally at low levels is upregulated with injury and inflammation, especially by TNF- $\alpha$ , and that nociceptive neurons increase in the IVD in response to NGF.

A potential clinical application of this could involve blockade of TNF- $\alpha$  and NGF in the early stages of intervertebral disc disruption in an attempt to prevent neo-innervation. This concept has been assessed experimentally in the laboratory rat with the use of doxycycline – a nonspecific TNF- $\alpha$  inhibitor, and infliximab – a specific TNF- $\alpha$  inhibitor.<sup>36</sup> Both systemic and local application seemed to reduce pain behaviour markedly after a surgically induced annular tear. A clinical trial using epidural etanercept has also been reported as providing significant pain relief, although this trial was more related to patients with radicular pain.<sup>37</sup> Further research is needed into this as a possible route of preventing the development of chronic pain from IDD.

## 2. BDNF

- BDNF is present in all parts of the IVD with increased expression in disc degeneration.<sup>38</sup>
- About 13% of the neurons innervating the IVD are BDNF immunoreactive.<sup>39</sup>
- BDNF expression is highest in the outer AF and least in the nucleus pulposus.<sup>38</sup>
- BDNF induces the recruitment of endothelial cells and the formation of vascular structures.<sup>40</sup>
- IL-1 $\beta$ , but not TNF- $\alpha$ , leads to a significant increase in BDNF.<sup>31</sup>

Thus it seems as though BDNF, upregulated by the inflammatory cytokine IL-1 $\beta$ , can induce the formation of vascular structures in the annular tear. Some nociceptive nerve fibres are also dependent on BDNF.

A potential clinical application of these findings is that blockade of BDNF might prevent neo-vascularization and reduce the number of nociceptive neurons. This has been tried in the laboratory rat where intradiscal anti-BDNF significantly suppressed CGRP and local BDNF after disc puncture.<sup>41</sup> There are no reports of human trials.

## 3. GDNF

- GDNF promotes survival and differentiation of dopaminergic and motor neurons.
- In discs injected with an inflammation-causing substance (Freunds adjuvant) GDNF was significantly increased in the DRG after two weeks. NGF was not increased in the DRG.<sup>42</sup>
- Intrathecal GDNF has a potent analgesic effect in neuropathic pain.<sup>43</sup>

The potential clinical applications here are uncertain as the research seems contradictory at this stage, with GDNF having an analgesic effect when given intrathecally but inducing hyperalgesia when administered into peripheral tissues.<sup>44</sup> Further research is needed.

## 4. SEMA3A

- Normal discs have high levels of SemA3A in the outer AF.<sup>45</sup>
- Degenerate discs have a significant decrease in SemA3A in the outer AF but it is still evident in the cell clusters of the NP.<sup>45</sup>
- Factors that lead to reduction in SemA3A expression are related to the presence of cytokines and macrophages.<sup>45</sup>

The reduction in the blocking signal of SemA3A could tip the balance towards nerve and blood vessel ingrowth. One mechanism that might have clinical application here is that SemA3A stimulates the production of hydrogen peroxide as a step in axonal inhibition.<sup>46</sup> This is possibly of relevance to an existing treatment for discogenic pain which involves injection of ozone and oxygen into the disc. A recent meta-analysis of this treatment indicated it was effective;<sup>47</sup> however, further research is needed, such as directly applying SemA3A to injured discs.

## Conclusion

The ingrowth of nerve and blood vessels into an annular tear involves a complex web of interacting cytokines, neurotrophins, and semaphorins. Clinical applications at this stage are focussed mainly on TNF- $\alpha$  and NGF but other neurotrophins or semaphorins may turn out to be equally important. It is intriguing that doxycycline may have a role to play in the initial treatment of discogenic back pain. Several



puzzling questions remain, however, such as why many annular tears and grossly abnormal discs seen on MRI are asymptomatic. What factor is it that determines chronicity in patients who do report chronic pain of discogenic origin and how can this be prevented? The surveyed current literature is not yet able to answer such clinical questions.

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# Shoulder pain referral zones

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## Abstract

Shoulder pain is very common. It has been estimated that approximately 10% of the adult population will experience an episode of shoulder pain in their lifetime, whilst shoulder pain is the third most common musculoskeletal presentation in general practice, after low back and neck pain. It has also been reported that up to 41% may have persistent symptoms at 12 months.<sup>1</sup>

The astute clinician needs to be able to differentiate between potential sources of “shoulder” pain, including intrinsic conditions of the shoulder joint complex, the neck and, uncommonly, non-musculoskeletal conditions.

To the patient, “shoulder” may mean anywhere around the shoulder girdle, including but not necessarily limited to the region around the shoulder joint complex itself, but also to the region of the scapular, and spreading up towards the neck.

Anatomical pain maps form an integral component of the clinical assessment of all painful conditions. Insofar as using published data, such maps can help with the differentiation of the potential source of presenting clinical problems. This is particularly so in those presenting with “shoulder pain”.

This paper provides a summary of available data pertaining to pain referral zones as a result of musculoskeletal conditions which may present as “shoulder” pain.

## Introduction

As a practitioner working full time in musculoskeletal pain medicine, this author is often the last port of call in those in whom pain is persistent and interfering significantly with their activities of daily living (ADLs), despite an often seemingly exhaustive list of previous investigations and treatments. For conditions presenting as “shoulder” pain, these have often incorporated invasive treatments targeting the components of the shoulder joint complex, including but not limited to the sub-acromial space (SAS), the rotator cuff tendons (RCT), and acromioclavicular joint (ACJ), including injections, often under ultrasound guidance, as well as various surgical procedures, all to no avail. Furthermore, close scrutiny of the outcome of the injections will often reveal that there was no short-term improvement in symptoms as one would expect if local anaesthetic were injected into the source of pain. Despite this, many such patients go on to have surgical procedures aimed at these same structures targeted by injection.

In this author's experience, a substantial proportion of patients who, subjected to a thorough clinical assessment incorporating a thorough history, including the use of anatomical pain maps, and a comprehensive yet not overly time-consuming musculoskeletal examination, can be demonstrated not to have an intrinsic shoulder problem at all. Rather, their predominant index pain is from an alternative source, by far the most common of which is pain referred from the cervical spine.

Such a misdiagnosis probably contributes significantly to the high persistence rate and long-term morbidity associated with shoulder conditions presenting to general practitioners.<sup>1</sup>

The dilemma arises when such “neck” patients also have restricted active shoulder range of motion on clinical assessment, from which it is concluded that there must therefore be intrinsic shoulder pathology to explain this loss of motion. They therefore undergo imaging studies of the shoulder, and these studies demonstrate lesions which, on face value, are amenable to potential interventional procedures.

However, it has been known for many years now that, in terms of shoulder imaging, asymptomatic individuals can commonly have demonstrable shoulder pathology on imaging studies, particularly with advancing age.<sup>2,3</sup> Also, the interobserver reliability of such imaging studies is questionable.<sup>3</sup> Thus, when interpreting the clinical relevance of any such imaging, the clinician must proceed with caution, and match the patient's clinical presentation to such imaging.

It has been recognized that cervical spine dysfunction *per se*<sup>4</sup> can lead to restriction of shoulder range of motion. The mechanisms for the loss of shoulder motion in cervical spine pain syndromes is beyond the scope of this paper, but, put simplistically, may represent some compromise of the normal kinematic chain, as well as reflex muscle “tautness” or “spasm” in shoulder girdle muscles innervated by lower cervical segments.

Pain referral zones (PRZs) for cervical spine zygapophysial joints (ZJs) have been extensively studied and published involving healthy volunteers<sup>5</sup> and various patient populations.<sup>6</sup> PRZs for cervical discs<sup>7</sup> and thoracic ZJs<sup>8</sup> have also previously been published. In particular, lower cervical spine discs and ZJs, as well as upper thoracic ZJs may all refer pain to the shoulder girdle, and should be considered

in the differential diagnosis of “shoulder pain”.

Less well known is that pain maps have also been published for the subacromial space (SAS),<sup>9</sup> acromioclavicular joint (ACJ)<sup>9</sup> and sternoclavicular joint (SCJ).<sup>10</sup> A study is in progress to demonstrate the PRZs of the glenohumeral joint (GHJ).<sup>11</sup>

Such maps of PRZs potentially offer great assistance to the clinician in the differentiation between the shoulder joint complex and alternative sources of the pain in such patients.

The available data will now be summarized.

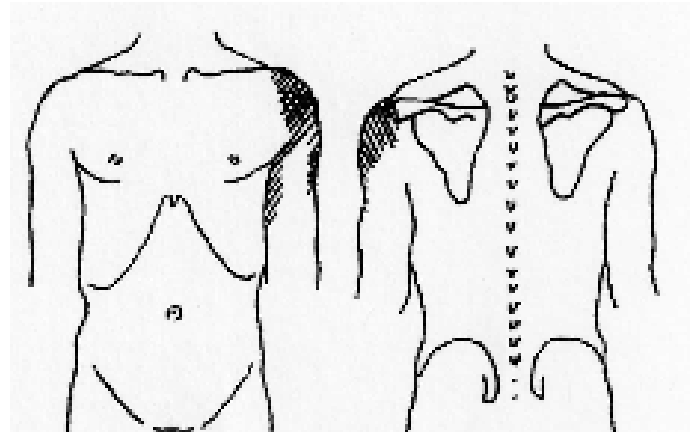
## Subacromial space

Gerber, et al.<sup>9</sup> sought to determine the patterns of pain referral from the subacromial space by selectively injecting hypertonic saline into this region in nine healthy volunteers.

They noted that irritation of the subacromial space produced intense pain that required immobilization of the shoulder, and caused a feeling of pressure within the shoulder. The lateral deltoid was found to be the most painful region, followed by pain over the lateral acromion. Two patients felt pain in the supraspinatus area, but none felt pain over the ACJ, sternocleidomastoid muscle, lateral clavicle, triceps, or supraspinatus fossa. One subject had referred pain distally to the forearm and hand. No subject reported pain in the scapular region (Figure 1, Table 1).

Acromion lateral border	10
Deltoid - lateral	10
Deltoid - anterior	10
Deltoid - posterior	3
Upper arm – lateral aspect	3
Upper arm – medial aspect	3
Acromion – posterior aspect	2
Upper trapezius	2
Thumb	2
Coracoid	1
Radial forearm	1
Ulnar forearm	1
3rd metacarpal and 3rd digit	1
5th digit	1
Acromioclavicular joint	0
Upper arm – posterior aspect	0
Neck	0
Supraspinatus fossa	0
Lateral clavicle	0

**Table 1. Pain described after irritation of the subacromial space (N=10). (From Gerber, et al.<sup>9</sup>)**



**Figure 1. Distribution of pain observed after irritation of the subacromial space. In some experiments, pain was perceived predominantly in anterior, in some predominantly in lateral, and in some predominantly in posterior parts of the hatched area. (From Gerber, et al.<sup>9</sup>)**

## Acromioclavicular joint

Similarly, Gerber, et al., in the same paper,<sup>9</sup> reported the outcome of selectively injecting hypertonic saline into the ACJ, targeted this joint 15 times in 10 healthy volunteers, including both left and right ACJs.

They noted that pain was centred over the ACJ, and was unpleasant and generally of burning quality. Of note was radiation of pain into the trapezius and supraspinatus muscles, as well as to the anterolateral neck. It did not produce pain at the posterior aspect of the shoulder or into the scapular region (Figure 2, Table 2).

Local pain	15
Supraspinatus fossa	12
Upper trapezius muscle	12
Lateral clavicle	12
Anterolateral deltoid region	9
Medial aspect of upper arm	4
Thumb	4
Sternomastoid muscle	3
Posterolateral acromion	2
Radial forearm	1
Infraspinatus fossa	0
Triceps region	0
Posterior deltoid region	0

**Table 2. Localization of pain after irritation of the acromioclavicular joint. (From Gerber, et al.<sup>9</sup>)**

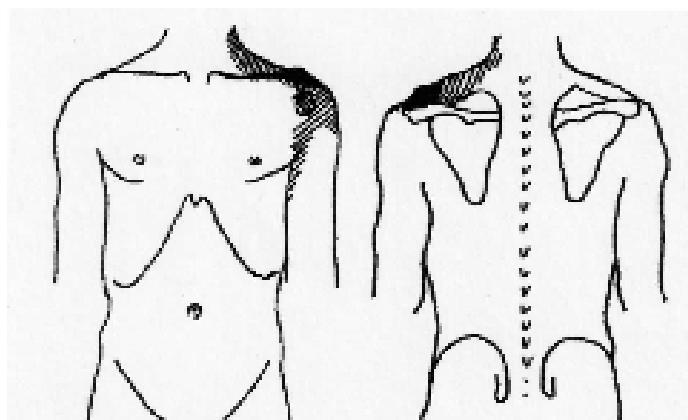


Figure 2. Distribution of pain observed after irritation of ACJ. (From Gerber, et al.<sup>9</sup>)

## Sternoclavicular joint

Hasset, et al.,<sup>10</sup> using similar techniques, demonstrated PRZs from the SCJ in nine asymptomatic healthy subjects. They found that pain started at the medial end of the clavicle and then spread to surrounding regions, including the anterior trapezial fold, out to the lateral clavicle, and over the anterior aspect of the shoulder, to the neck and to the jaw.

In one of their subjects the pain radiated down the arm as far as the elbow as well as deep in the throat, thus mimicking the PRZ of myocardial ischemia.

These authors noted that pain was not referred to the chest in any of their subjects, which had previously been reported.

Nor was pain referred to the posterior neck, occiput, or interscapular region.

The composite pain diagrams pertaining to this study are presented in Figure 3.

## Glenohumeral joint

A search of Medline failed to uncover any studies on PRZs for intrinsic glenohumeral joint (GHJ) pathology.

Vivian and colleagues<sup>11</sup> are currently investigating PRZs of patients referred to a private radiology practice for GHJ hydrodilatation, most commonly because of a clinical diagnosis of adhesive capsulitis, or frozen shoulder. In these patients, local anaesthetic agents are included in the injectate as a matter of routine.

They used aggregate data of only those subjects who achieved greater than 50% pain relief at 30 minutes and two hours post-injection, and thus were deemed to have "concordant pain" consistent with a diagnosis of shoulder (GHJ) joint pathology, to determine the PRZs from the GHJ.

They found that about 37% had pain localized to the shoulder only. Almost two-thirds of their sample (63%) of patients had pain referred to the upper limb, most commonly to the anterior upper arm, and forearm, whilst 21% had pain referred to the hand.

The remaining 37% showing referred pain patterns away

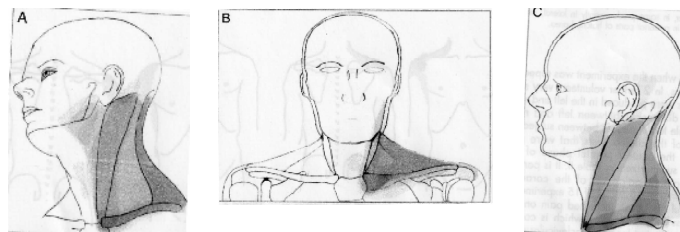


Figure 3. Composite illustration of distribution of pain observed after irritation of the sternoclavicular joint. The density of shading reflects the number of subjects reporting pain in that area. In one subject, pain was referred to the arm which was not depicted in this figure.

A. Anterolateral view B. Anterior view C. Lateral view. (From Hasset, et al.<sup>10</sup>)

from the shoulder had referred pain from the shoulder to above the clavicle, but not to the scapular region.

Number of patients observed	
No pain	0
Shoulder pain only; no referral	5 (37%)
Referral down arm but not below elbow	3 (16%)
Referral below elbow but not past wrist	5 (26%)
Referral into the hand	4 (21%)
Other	2 (10%)

Table 3. Patterns of pain referral in patients with pain originating from the GHJ. (From Vivian, et al.<sup>11</sup>)

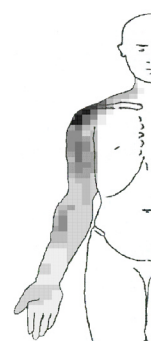


Fig 4: Patterns of pain referral in patients with pain originating from the GHJ. The density of shading reflects the number of subjects reporting pain in that area. (From Vivian, et al.<sup>11</sup>)

## Discussion

These studies pertaining to PRZs for various components of the shoulder joint complex involve small numbers. Those involving the SAS and ACJ were in healthy volunteers, whilst those into the GHJ were mostly presumed to be suffering from adhesive capsulitis.

There can be little question that these studies should be replicated, in varying populations, and in higher numbers, as has been undertaken, for example, for cervical facet joint PRZs.<sup>5,6</sup> This would add to the scientific weight, and therefore clinical usefulness of these pain referral patterns.

Nonetheless, accepting the findings of these studies on face value, certain conclusions relevant to clinical practice



can be drawn from the PRZ studies performed thus far:

- Pain originating from the subacromial space has as its centre pain over the lateral deltoid and lateral acromion.
- Pain centred over and within the ACJ most likely has that joint as its source.
- Pain along the clavicle spreading between the shoulder and the neck may, uncommonly, be referred from the ACJ or GHJ, or, less commonly, SCJ, but will not have as its origin structures within the subacromial space.
- SCJ mediated pain should be easily differentiated by its centre being at the medial end of the clavicle.
- Pain located in the posterior aspect of the shoulder joint is unlikely to have as its source the SAS, ACJ, or SCJ.
- Pain referred to the posterior aspect of the shoulder may originate from the GHJ, but its centroid is most likely located over the lateral acromion or deltoid.
- Pain referred to the scapular region does not have as its source any intrinsic pathology of the shoulder joint complex.
- Pain located in the scapular region, or posterior aspect of the shoulder most likely is referred from the cervical spine, or less commonly thoracic spine.

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Whilst for an individual patient differentiation as to the source of their shoulder can be difficult, the use of known PRZs can be a useful adjunct to the clinical assessment, and guide the practitioner towards the most appropriate diagnostic and therapeutic endeavours.

It goes without saying that further studies, incorporating symptomatic and asymptomatic individuals, as well as larger patient numbers are required to confirm the above data. Such information in turn may lead to improved patient outcomes, as well as a minimization of well-intentioned but ill-directed and inappropriate investigations and treatments for those presenting with “shoulder pain”.

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# Do corticosteroid injections relieve greater trochanteric pain syndrome?

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## Introduction

**G**reater trochanteric pain syndrome (GTPS) is a common and frequently debilitating condition presenting with pain at or around the greater trochanter, sometimes referring to the lateral hip or lateral thigh. The condition is often chronic.<sup>1</sup>

Excluding lumbar or pelvic referred pain, the incidence has been recorded in 1.8 per 1000 adults per year in a primary care study<sup>1</sup> with a prevalence of 10-25%<sup>1,2</sup> but estimated to occur in 20-35% of sufferers of chronic low back pain.<sup>3,4</sup> There is a strong gender bias, with the female to male ratio 2-4:1, and the most frequent age group are those between the fourth and sixth decades. Previous labels for this condition have included calcareous peritrochanteric bursitis,<sup>5</sup> trochanteric syndrome,<sup>6</sup> hip periarthrititis, trochanteric (subgluteal) bursitis,<sup>7,8</sup> trochanteric tendinobursitis, and hip rotator cuff tears, with the condition most commonly known as trochanteric bursitis.

Bursitis has been shown to be an inaccurate label, with an absence of signs associated with bursitis of erythema, oedema, and rubor,<sup>9</sup> no histologic evidence of bursitis,<sup>10</sup> infrequent bursal changes on ultrasonic imaging,<sup>11</sup> and no advantage of fluoroscopic-guided specific intra-bursal injections compared with blind injections.<sup>12</sup> Histologic examination has frequently revealed tendinosis, including collagen degeneration with fibre rupture, eosinophilic exudate, fibroblastic proliferation, prominent small blood vessels, dystrophic calcification, but no acute inflammation.<sup>11</sup>

Diagnosis of GTPS is inexact, relying on clinical history and examination. Whilst imaging, particularly ultrasound and MRI, frequently demonstrate pathology, so did 88% of asymptomatic people in a study<sup>13</sup> of MRI of the pelvis, with sensitivity of MRI being 1.00, specificity 0.12, producing a positive likelihood ratio of only 1.13, and negative likelihood ratio 0. Ege Rasmussen and Fano<sup>14</sup> proposed diagnostic criteria of lateral hip pain and distinct tenderness about the greater trochanter, and one of the following:

- pain at the extreme of rotation, abduction, or adduction
- pain on hip abduction against resistance
- pseudoradiculopathy
- positive FABERE test.

To this, others have added a positive Trendelenburg gait or test, pain after standing on the affected leg for 10-30 seconds, or pain upon resisted realignment of the externally rotated hip.

However, if the gold standard is local pain and point tenderness, any other examination technique is not likely to reach 100% sensitivity and specificity, and therefore weakens the post-test diagnosis, making additional testing

superfluous.

GTPS is often treated with corticosteroid injection. The aim of this literature review was to establish what evidence exists for the efficacy of corticosteroid injections for GTPS in the general population.

## Search and study selection

A search was conducted for relevant studies published in PubMed (including Medline) using the keywords “gluteal tendinosis”, “gluteal tendinopathy”, “gluteal rupture”, “gluteal pain”, “gluteus tendinosis”, “gluteus tendinopathy”, “gluteus rupture”, “gluteus pain”, “hip abductor pain”, “greater trochanter pain”, “greater trochanteric pain”, “rotator cuff hip pain”, “rotator cuff tear”, “lateral hip pain”, “lateral hip tear”, “snapping hip pain”, “trochanteric bursitis pain”, with no limit regarding the year of publication.

From 2235 references were culled any studies not in English, not involving live humans, as well as any studies primarily related to hip joint pathology or surgery, or systemic or referred disease (such as cancer, fractures, infection, neuropathy, vasculopathy, other organ systems).

Reviews of GTPS were gleaned by hand for any studies missed by the PubMed search.

Ten original studies published in journals were identified and the full reprints analysed.

## Results

Ten studies (seven uncontrolled case series, three randomized) were extracted and are summarized in Table 1.

The seven uncontrolled case series from 1958 to 1966<sup>9,14-19</sup> contain a total of 267 subjects. None of the studies reports whether they obtained ethics committee approvals beforehand. All are performed in specialist rheumatology or orthopaedic medical outpatient clinics, and numbers of subjects entered into each study range from 3 to 75. Two out of the seven studies don't specify their criteria for diagnosis,<sup>14-19</sup> whilst three studies use pain, and two specify both pain and local tenderness.

Two studies don't profile the age distribution of their subjects, whilst the average age of the total 180 patients in the other five studies is 56.6 years old. One study doesn't give an analysis of gender<sup>15</sup> but analysis of the total 216 patients in the other six studies where a gender breakdown is given provide a total of 166 females (77% of total), 50 males (23% of total) for a female to male ratio of 3.3:1.

Three studies didn't specify the duration of symptoms; one reported only a few days,<sup>14</sup> whilst the three remaining studies gave ranges of under 24 hours to 2.5 years,<sup>15</sup> 1-5

Study	Ref	Population	Ethics Approval	Study Size	Criteria	Age	Gender	Duration	Study Type	Treatment	Site of Injection	Measures	Follow-up	Findings
Barker 1958	14	Specialist practice	Not stated	3 injected	"Bursitis"	42, 51, 59 yrs	2 F: 1 M	"Few days", 3 weeks in two cases, not specified in 3rd	Non-randomized open case series	Procaine & prednisolone, hydrocortisone, procaine & hydrocortisone		Pain free	1.5 - 2 yrs	All well at duration
Gordon 1961	15	Orthopaedic Clinic	Not stated	51	Lateral hip/thigh pain	Ave 46 yr (23-67 yr)	Not specified	Ave 64.5d (<24h-2.5yrs)	Non-randomized open case series	24 inj'd with 10-15 ml equal parts 2% procaine & 0.15% pontocaine. 27 inj'd with procaine-pontocaine mixture followed by 50-100 mg hydrocortisone. Repeated 5-7 days later if required.		Excellent, Good, Fair, or Poor	5-7days	22/23 excellent or good after procaine-pontocaine injections, 27/28 excellent or good after procaine-pontocaine plus hydrocortisone
Raman & Haslock 1982	16	100 consecutive Rheumatoid patients	Not stated	15	Pain over trochanteric bursa	Not stated	14 F: 1 M	1-5 years	Non-randomized open case series	10 mg triamcinolone hexacetanide with 2% lignocaine to 10 ml		Pain free	8 weeks + 8 weeks	All pain free at 8 weeks, or 8 weeks after 2nd injection
Ege Ras-mussen & Fano 1985	17	Inpatient rheumatology & physical medicine patients	Not stated	62 identified, 58 invited, 36 entered study	Little & Cyriax criteria including Lateral hip pain, local tenderness (see text)	Ave 57.9 yr (16-82 yrs)	29 F: 7 M	19.5 mnths (0-276 months)	Non-randomized open case series	40-80 mg methyl-prednisolone or 20-40 mg triamcinolone		Excellent/ Improved/ Unchanged	Ave 23.2 mnths (6-73M)	27/36 pain free, 9 relapses
Karpinski & Piggott 1985	9	Orthopaedic Clinic (Population 3000 patients)	Not stated	15 entered, 12 given steroid injections	Trochanteric pain	Ave 43yrs (12-59 yrs)	11F: 4M	Not specified	Non-randomized open case series	Steroid	Tip of greater trochanter	Relief or not	9-79 months	8 relief, 1 non-responder repeated
Schapira et al. 1986	18	Rheumatology Clinic	Not stated	72 entered, 59 injected	Little's criteria	Not stated	48 F: 24M	Not specified	Non-randomized open case series	40 mg methyl-prednisolone & 2 ml xylocaine 2%	"Local", not specified	Disappearance of pain and dysfunction	Not recorded	71% recovered following 1 injection 22% recovered following 2nd injection, 7% recovered following 3rd injection
Shbeeb, et al. 1996	19	Rheumatology Clinic	Not stated	94 identified, 75 injected	Criteria used but not specified	Ave 66.2 yrs	62 F: 13M	Not specified	Non-randomized open case series	Gp 1 N = 20 6 mg beta-methasone; Gp 2 N -32 12 mg betamethasone; Gp 3 24 mg betamethasone. All with 4 ml 1% xylocaine	Bony prominence (as per Little), and around it	Improvement Yes/ No/Unsure; VAS; Lower extremity questions; Analgesia use	Mail follow up 1w, 8w, 26w	77.1% reported improved at 1w; 68.8% reported improved at 6w; 61.3% reported improved at 26w; 24 mg betamethasone more likely to have sustained improvement at week 26 (p < 0.0123)
Sayegh, et al. 1999	21	Orthopaedic Department	Not stated	300 - 1/2 injection group, 1/2 conservative treatment group	Not stated	Not stated	Not stated	Not specified	Method randomization not specified, Comparison study	Injectate not specified	Local injection tender point peri-trochanteric area	Oswestry Disability Index	day 1, 1m, 6m, 24m, 36m, 48m	During all periods, injection group had better outcome than conservative group, p < 0.0004
Cohen, et al. 2009	23	Ambulatory care pain centres	Registered study, Ethics approval, Patient consent	79 identified. 65 entered - 33 sham fluoroscopy, 32 fluoroscopy	Pain lateral hip over 3 mnths duration	55.2 yrs (21.0-85.0 yrs)	56 F: 9 M	3.9 yrs (0.1 -16 yrs)	Multicentre, randomized, double blinded	60 mg depo-methylprednisone & 2.5 ml 0.5% bupivacaine intrabursal by fluoroscopy, or sham	Bursa	NRS, SF-36, Oswestry, Medication use, Global perceived effect, composite "successful outcome"	3 months	12/32 bursa on 1st attempt at fluoroscopy, 12/33 in sham procedure; 39/64 (61%) positive outcome (over 50% pain relief & satisfied) at 1 mnth; 28/64 (44%) positive global perceived effect at 3 mnths - no significant diff btwn fluoroscopy or sham group; little difference btwn intra- or extra-bursal gps
Brinks, et al. 2011	22	Primary care	Pre-registered, Ethics Approval, Informed Consent	159 recruited. 120 randomly assigned - 60 usual care incl physio, 60 injection	1w duration pain	56.3 yrs (18-80 yrs)	92 F: 28 M	28 (23.5%) over 6 mnths; 34 (28.6%) 2-6 mnths; 57 (47.9%) 1-2 mnths	Multiple practices, computer randomized, open label. Intention to treat	40 mg triamcinolone & 1-2% xylocaine to 5 ml	Most tender point on greater trochanter	Likert Scale, NRS, QOL, WOMAC Pain & Function Domains, Adverse Effects (at 1/52)	6w, 3m, 6m, 9m 12m.	At 3 m, 55% injection gp totally or strongly recovered cf 34% usual care, OR 2.38 (much stronger effect on WOMAC OR 12.40 for pain, OR 11.36 for fn); at 12 m 61% inject gp recovered cf 60% usual care, OR 1.05; hip & low back pain subgroup mirrored overall benefits at 3 m

**Table 1. Summary of original studies of corticosteroid efficacy for greater trochanteric pain syndrome.**

years,<sup>16</sup> and 0-276 months.<sup>17</sup>

Reflecting the availability of corticosteroids at the time, the 1958<sup>14</sup> and 1961<sup>15</sup> studies used prednisolone or hydrocortisone along with local anaesthetics (procaine or pontocaine). The subsequent open case series used triamcinolone, methylprednisolone, or betamethasone,<sup>20</sup> with one study not specifying which steroid was used.<sup>9</sup>

Five studies<sup>14-18</sup> don't specify the point of injection whilst the other two specified the tip of the greater trochanter<sup>9</sup> or bony prominence.<sup>20</sup> Outcome measures were either vague ("pain free") or appear simply a verbal report of excellent, good, fair, or poor at most. One study went a little further with assessment<sup>20</sup> and assessed pain on a 100 mm Visual Analogue Scale (VAS), a standardized set of questions about lower extremity function and analgesia use, and asked whether the patients had improved or not or were unsure. Follow-up of the various studies ranged from 5-7 days<sup>15</sup> to 2-6 years or wasn't reported.<sup>18</sup>

Given that these were case series, without comparison with a control group, or the natural history of the condition, the best outcomes for the longest duration appeared to be the study by Shbeeb, et al.<sup>20</sup> which gave a time scale of 77.1% improved at one week post-injection, 68.8% improved at six weeks, and 61.3% improved at 26 weeks. Their study compared different dosages of betamethasone (6 mg, 12 mg, and 24 mg) and found a significant advantage for the 24 mg dose at 26 weeks.

In a published proceeding, with minimal information provided Sayegh, et al.<sup>21</sup> split a group of 300 patients with greater trochanteric bursitis pain syndrome (GTBPS) into two. There are no details provided on the process of randomization, demographics, duration of symptoms, or even the nature of the injectate. During all periods of follow-up up to 48 months, the injected group had a significantly improved outcome as measured by the Oswestry Disability Index.

Cohen et al. (2009)<sup>12</sup> published the first double blind study of corticosteroid injection for greater trochanter pain but this study was primarily of the use of fluoroscopy for guiding greater trochanter injections. Across three pain clinics, they did fluoroscopic-guided bursal injections (60 mg depo-methylprednisone, 2.5ml 0.5% bupivacaine) for 32 patients, with a sham procedure for the control group of 33. Twelve out of the 32 fluoroscopy-guided injections were in the bursa at first attempt, whereas 12 out of the 33 sham procedures ended up being in the bursa. Outcomes were assessed using a Numeric Rating Scale (NRS), SF-36, Oswestry Disability Index, medication use, global perceived effect, and a composite "successful outcome". At one month, 39 out of 64 patients (61%) had a positive outcome (greater than 50% pain relief and satisfied). Those patients not relieved at one month were then excluded from the study to receive alternative medical care. At three months, 28 of the original injection group of 64 (44%) (one drop-out) had a positive outcome (same criteria as at one month). There was no significant difference between the fluoroscopy group and the sham fluoroscopy group in overall outcome.

In the first true randomized test of the use of injectable corticosteroids for greater trochanteric pain syndrome,

Brinks, et al.<sup>22</sup> included 159 patients recruited by 81 Dutch primary care physicians. After 39 exclusions (7 did not meet the inclusion criteria, 32 refused to participate), the remaining 120 patients were remotely and randomly assigned into a control group of usual therapy (including physiotherapy and analgesia) or an injection group (40 mg triamcinolone with 1-2% xylocaine in a 5 ml syringe into the most tender point on the greater trochanter). The design was based on intention to treat. Outcome measures included the Likert Scale (from 1 meaning fully recovered down to -7, worse than ever), a 0-10 Numeric Rating Scale (NRS), EuroQoL Quality of Life (QoL) scale, and Western Ontario and McMaster University OA Index (WOMAC) Pain and Function domains administered at 6 weeks, 3 months, 6 months, 9 months, and 12 months. Adverse effects of injections were solicited at 6 weeks. At 3 months, 55% of the injection group had totally or strongly recovered compared with 34% of the usual care group (Odds Ratio (OR) 2.38 (95th CI 1.14-5.00)). At 12 months, 61% of the injection group had recovered, compared with 60% of the usual care group (OR 1.05 (95% CI, 0.50-2.27)). A hip and low back pain subgroup at 3 months revealed 58% of improvement in the injection group, 32% in the usual care group, which is little different from the overall groups as a whole.

## Discussion

Of the 10 original research papers gleaned from the literature, seven are non-randomized uncontrolled open case series with no mention of ethics approval or patient consent, and frequently done in specialist rheumatology or orthopaedic clinics. The biased practice population could well be a select group of patients different to those found in the community or primary care. With no control group it is simply not possible to tell whether the treated group has done any better than the natural history of this condition. Most of these studies have used minimal outcome measures such as simply whether the patient or treating physician has deemed the patient has improved or not. Follow-up appears to be ad hoc, with no firm intervals, and in three of these studies is very short, or not specified.<sup>18</sup>

Cohen, et al.'s study,<sup>23</sup> whilst randomized and double blinded, appears to be fundamentally flawed in that injections were directed at the trochanteric bursa even though bursitis had generally been discredited by this time as the potential source of lateral hip pain in favour of tendinosis. Possibly this explains the poorer outcome for those patients receiving injections compared with the earlier case series.

The Dutch study<sup>22</sup> appears well designed from conception to conclusion. Whilst the injection group was compared with a usual care group, the effect of corticosteroid and local anaesthetic is studied, but a confounder not excluded is the placebo value of having an injection with no comparison sham group and the non-blinding of the treatment provider and patient.

As there is a small risk of bodily penetration by needles, a future study could compare the effect of injection with



active ingredients versus a non-steroid injection (such as local anaesthetic alone) with the treatment provider also blinded.

## Conclusion

The current review uncovered seven case series which appear to demonstrate relief of GTPS symptoms. The best quality randomized open label study<sup>22</sup> confirmed a significant improvement for the injection group over usual care at 3 months but this benefit appeared extinguished by 12 months.

**Conflict of Interest:** None reported.

## Acknowledgement

The author wishes to acknowledge Drs Peter Catt and Steven Jensen for the inspiration to explore this topic. This review was conducted as part of the University of Otago Aviation Medicine AVMX785 Research Methods paper and the advice and guidance of Ms Julie Meyers (lecturer) and Ms Karen Johnson (student learning advisor) is also acknowledged.

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# Review of recent reports on prolotherapy

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**R**esearch into prolotherapy continues in several centres and some recent published papers are presented here with comments.

In an editorial in *American Family Physician* of December 2010, Rabago, Yelland et.al.<sup>1</sup> reported that "The strongest data supporting the effectiveness of prolotherapy for any musculoskeletal condition, compared with saline control injections, is for severe refractory lateral epicondylitis".

One such trial involving lateral elbow pain was the Scarpone trial (2008).<sup>2</sup> Participants treated with prolotherapy (n=10) reported approximately a 90% reduction in resting elbow pain on a 10-point visual analog pain scale compared to a 22% reduction in the control group (n=10) at baseline and at 16 weeks (absolute effect size of 68%;  $P < .01$ ). The prolotherapy group also demonstrated improved isometric strength ( $P < .05$ ) at 16 and 52 weeks compared to the control group.

In other limited studies, other overuse injuries have responded well to prolotherapy. These include Achilles, adductor tendinopathies, and plantar fasciitis. They are tabulated in Table 1 below. Comment is made on only some of these studies in this review. Abstracts for those not presented are readily available from PubMed.

**Table 1. Chronic musculoskeletal conditions treated with prolotherapy**

Condition	Ref.	Type of study
Achilles tendinopathy	3	High-quality small RCT
Coccygodynia	4	Prospective single arm study
Knee osteoarthritis	5,6	Moderately strong-quality RCT and prospective single arm study
Lateral epicondylitis	7	Small RCT
Degenerative disk disease	8	Prospective single arm study
Nonspecific low back pain	9,10,11	RCTs
Plantar fasciitis	12	Prospective single arm study
Sacroiliac joint dysfunction	13,18	Prospective single arm study

RCT=randomized controlled trial.

Participants in the Achilles tendinopathy study by Coombes<sup>3</sup> responded earlier, with less money spent on treatment, when physical therapy and prolotherapy were combined, compared with either treatment alone.

The double blind randomized control study of steroid versus glucose prolotherapy for chronic lateral epicondylitis by Carayannopoulos et al.<sup>7</sup> was a comparison of two injections of either steroid, or prolotherapy, given one month apart. Both groups (N=24) improved significantly from baseline, but there were no real differences between the groups with follow-up over six months.

**Comment:** We know from other studies that steroid-treated elbows do not always maintain their improvement at 12 months, so the follow-up was too short for comparison.

In 2011 Hart (2011)<sup>14</sup> published a review of studies of steroid or other injections for tendinopathy, including local anaesthetics, sclerosants, platelet rich plasma (PRP), aprotinin, botulinum toxin, and glycosaminoglycans. There were 41 articles that met the inclusion criteria. For painful lateral epicondyle conditions, steroid was found to be the most effective in the short term with improvement in pain and function, but intermediate and long-term outcomes were inferior to orthotic devices, physiotherapy, and PRP. The results were similar for rotator cuff tendinopathy.

**Comment:** This was very similar to a 2010 review by Coombes and Bisset from Queensland<sup>15</sup> who concluded that "Despite the effectiveness of corticosteroid injections in the short term, non-corticosteroid injections might be of benefit for the long-term treatment of lateral epicondylalgia. However, response to injection should not be generalized because of variation in effect between sites of tendinopathy."

Martins et al. (2011)<sup>16</sup> assessed the mechanical behaviour and the histology of collagen fibers after prolotherapy with 12.5% dextrose injected into the rat Achilles tendon compared with saline, steroid, and no treatment. There was no difference in maximum load at failure and no difference histologically in mature and immature collagen fibres. The authors concluded that dextrose is not deleterious to tendons.

**Comment:** This raises the question as to what prolotherapy is actually doing. Clinically, prolotherapy is used in two ways: firstly on strained ligaments, usually at the enthesis where the tendon and fascia join the periosteum. This is where strain has changed the physical properties of the collagen, making it more extensible.<sup>17</sup> Secondly, it is used for tendinopathy, which is a different pathology. In this clinical setting the solution is not injected into the tendon, but into adjacent tissues. The Martins paper did not study Achilles tendinopathy but the effect of prolotherapy on healthy collagen. However, as strain injury usually occurs at the enthesis, not in healthy collagen in the middle of the tendon, (apart from occasional exceptions) one would wonder whether normal parallel bundles of collagen would be significantly strengthened by the injection of anything. However, at a strained enthesis, even dry needling might stimulate an inflammatory and healing response with the formation of new collagen with possible clinical benefit. It is unfortunate that in this paper injections were done into tissue that was not clinically abnormal.

Kim et al. (2010)<sup>18</sup> published a prospective, randomized, controlled trial comparing prolotherapy with intra-articular steroid injection in relieving sacroiliac joint pain. The patients had at least three months sacroiliac joint pain, confirmed by  $\geq 50\%$  improvement in response to local anaesthetic block. The treatment involved intra-articular dextrose prolotherapy or triamcinolone under fluoroscopic guidance, with three injection given every second week. Pain relief at 15 months of greater than 50% was 58.7% (95% confidence interval [CI] 37.9%-79.5%) in the prolotherapy group and 10.2% (95% CI 6.7%-27.1%) in the steroid group ( $p < 0.005$ ). This was a significant difference.

The conclusion was that intra-articular prolotherapy provided significant relief of sacroiliac joint pain, and the

effects lasted longer than those of steroid injections.

**Comment:** Sacroiliac joint prolotherapy is usually not just into the cartilaginous part of the joint but “peppered” over a large area from the interosseous ligament in the upper part of the joint, all over the posterior sacroiliac ligaments and around the lower cartilaginous joint. The study is unclear whether the injections were only intra-articular or if they were the usual ligamentous injections. Also the patients received only three injections, whereas in clinical practice, four to six treatments are often used, so this was a good result for prolotherapy treatment.

Refai H et al. (2011)<sup>19</sup> compared 10% glucose with saline in a randomized double blind trial for temporomandibular joint (TMJ) hypermobility. Both groups were given mepivacaine 2% and were given four injections six weeks apart. At the end of the study, both groups showed significant improvement in TMJ pain on palpation, and in number of locking episodes, but reported an insignificant improvement in joint clicking. Maximal mouth opening had improved significantly more in the prolotherapy group at the final assessment at three months compared to the control group.

**Comment:** Using 10% glucose, a non-inflammatory strength, is useful in a trial as it doesn't unblind the treatment, but in practice 20% is usually used and comparison with local anaesthetic control may be more significant. This is the common dilemma in musculoskeletal research: what to use as the control. The study was also of small size – only 12 patients.

Finally, Rabago et al.<sup>6</sup> published preliminary data from a RCT comparing prolotherapy with saline and exercise in knee osteoarthritis. The full results have not yet been published but the figures for the prolotherapy group, which compare baseline to result at one year, show that the prolotherapy group has decreased pain and improvement in stiffness and function, as measured by the WOMAC score. Intra-articular and extra-articular injections at 1, 5, and 9 weeks (and if desired at 13 and 17 weeks) progressively improved WOMAC score. At 4 weeks there was 17.2% improvement (WOMAC score of 7.6±2.4 points) and at the 52-week follow-up the improvement was 36.1% (WOMAC score of 15.9±2.5 points,  $p<0.001$ ).

**Comment:** Using prolotherapy for knee pain has been considered clinically useful for some time and it is reassuring to have this study to apparently confirm this. The full report is eagerly awaited.

**Conclusion:** Ongoing research into prolotherapy continues to face the same challenges as with all musculoskeletal medical research, that blinding of an active treatment is more difficult to achieve than trials involving, for example, a new drug. The complex nature of most musculoskeletal injuries or pain also requires practitioners to use more than one modality. Mobilization, manipulation, dry needling and other injections, exercise therapy, massage and others are just a few of the options musculoskeletal therapists can use if appropriate. It is satisfying, however, to see some university departments starting to report studies on prolotherapy. Funding for comprehensive research into this area is generally less available compared to some of the more “glamorous” areas of medicine.

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# Brief Case Presentations

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## 1. Knee Pain

A woman in her late twenties, whose hobby was long-distance running, presented with persistent right knee pain that had developed one year previously. She was referred to an orthopaedic surgeon who, without examination and investigation, performed an arthroscopy on her knee following which the pain became persistent and severe.

She was treated with regular physiotherapy for the next year without benefit.

A third party referred her to the clinic where the appearance and function of the knee were normal.

Careful palpation revealed tender induration of the tendons of the pes anserinus.

Using diluted 1% xylocaine to reduce her pain experience the bevel of a 30-gauge 1-inch needle was passed through the tenderest loci in the musculo-tendinous junctions and teno-periosteal attachments.

At follow-up she claimed 98% improvement and the treatment was repeated "just in case".

**Comment 1:** Not all knee region pain is intra articular.

**Comment 2:** What is going on here? Some practitioners claim that it is the special injectate that is providing the ongoing pain relief but people who dry needle these areas also claim success.

**Comment 3:** Could this be an example of Karel Lewits' "needle effect"? In other words, could the bevel of the needle be acting as a scalpel or chisel, mechanically deactivating degraded collagen or destroying dysfunctional C fibre terminals?

**Comment 4:** Cost of treatment was that of two consultations. No imaging, pathology, or referral requests were made and there is no procedural fee.

## 2. Vulvodynia

A 44-year-old woman presented with a 24-year history of widespread musculoskeletal aching pain in all four quadrants comorbid with visceral pain. She also had persistent fatigue. She was on multiple medications including Norspan patches, Xanax, and Prothiaden, without much benefit. Embedded in this pain experience was vulvodynia which was interfering with her marriage.

Self-palpation had led her to identify algogenic loci in the body of each pube.

When these loci were palpated they were identified them as being the attachment areas of the abductor longus and brevis and on examination of these muscles, hypertonic tender bands were also identified.

Using a 25-gauge needle tip as a mini scalpel/chisel, under the cover of diluted xylocaine as a mercy procedure these were mechanically deactivated the musculo-tendo-periosteal junction as well as the central trigger points in the main body of the adductors and followed this with post isometric relaxation technique to restore increased resting length of the muscle fibres.

This treatment obtained a 50% relief of pain and it was repeated on two more occasions until the problem became a non-issue.

**Comment:** Enigmatic pain such as vulvodynia may not be totally psychogenic and requires a precise local palpatory examination to exclude peripheral causes of somatic pain. Should the examination prove positive very simple bedside, non-pharmaceutical treatment can be used to help the sufferer. At the last visit, the patient informed me that her marriage had improved.



# Journal abstracts

**The following is a selection of abstracts which you might find relevant to your practice. The opinions of the reviewers are their own.**

**Matsumoto M, Okada E, et.al. Modic changes in the cervical spine: Prospective 10-year follow-up study in asymptomatic subjects. *J Bone Joint Surg Br* 2012 May;94(5):678-83.**

We conducted a prospective follow-up MRI study of originally asymptomatic healthy subjects to clarify the development of Modic changes in the cervical spine over a ten-year period and to identify related factors. Previously, 497 asymptomatic healthy volunteers with no history of cervical trauma or surgery underwent MRI. Of these, 223 underwent a second MRI at a mean follow-up of 11.6 years (10 to 12.7). These 223 subjects comprised 133 men and 100 women with a mean age at second MRI of 50.5 years (23 to 83). Modic changes were classified as not present and types 1 to 3. Changes in Modic types over time and relationships between Modic changes and progression of degeneration of the disc or clinical symptoms were evaluated. A total of 31 subjects (13.9%) showed Modic changes at follow-up: type 1 in nine, type 2 in 18, type 3 in two, and types 1 and 2 in two. Modic changes at follow-up were significantly associated with numbness or pain in the arm, but not with neck pain or shoulder stiffness. Age ( $\geq 40$  years), gender (male), and pre-existing disc degeneration were significantly associated with newly developed Modic changes. In the cervical spine over a ten-year period, type 2 Modic changes developed most frequently. Newly developed Modic changes were significantly associated with age, gender, and pre-existing disc degeneration.

**Comment:** Chronic neck pain is a common presentation in musculoskeletal practices and is difficult to treat with any degree of consistent results. This study in asymptomatic volunteers with an average age of 40 years initially and 50 years at the 10-year follow-up is an interesting study in the development of neck "pathology" over time. An earlier Norwegian study reported in *Spine Journal* (1994;19(12):1307-9) by Bovim reported a prevalence of neck pain of 34.4% in the previous 12 months, with a 13.8% prevalence of pain lasting more than 6 months. This figure of 13.9% of subjects developing modic changes almost matches the figure in the Norwegian study of the prevalence of chronic neck pain – is it due to disc inflammation or degeneration? This Japanese study, however, mentions that modic changes were significantly associated with arm symptoms rather than neck pain. Thus it seems to confirm that the cervical disc changes that occur as we age are not solely responsible for the relatively high prevalence of the chronic neck pain and that we need to look elsewhere (i.e., the ZAJ). – *Dr Tom Baster*

**De Carvalho BR, Puri A, Calder JA. Open rotator cuff repairs in patients 70 years and older. *ANZ J Surg* 2012 Apr 23. doi: 10.1111/j.1445-2197.2012.06034.x. [Epub**

**ahead of print].**

**Background.** Symptomatic rotator cuff tear is a commonly diagnosed problem in patients over the age of 70; however, there is controversy regarding the management of this condition. We set out to investigate whether this group has satisfactory results with operative management of their rotator cuff tears.

**Methods.** Retrospective review of one surgeon's patients who have undergone an open rotator cuff repair at age 70 or older. Outcome assessment included history of work and recreational activities, review of medical records, clinical examination, the Simple Shoulder Test (SST) and the Constant Shoulder Score (CSS).

**Results.** A total of 96 patients (104 shoulders) underwent open rotator cuff repair during the study period. Sixteen patients (16 shoulders) were lost to follow-up leaving 80 patients (88 shoulders) for review. Mean duration of symptoms was 18.3 months, mean age at surgery was 74.2 years and mean time to follow-up was 40.8 months. The mean SST and CSS scores were 9.8 and 80.1, respectively. In both tests, patients scored best in the pain relief categories and worst in strength-measuring areas. A total of 73 patients (92.7%) reported satisfaction with their surgery. None of these were limited by their shoulders in returning to pre-injury independence, work or recreations. They were either completely pain free or had only mild symptoms.

**Conclusion.** Patients in our study reflected a high satisfaction rate of 92.7% as well as excellent pain relief and a high level of function when related to their daily activities, independence and recreations or work. Level of evidence: Level IV (observational study without control - retrospective study).

**Comment:** It is always difficult to decide what to do about rotator cuff tears in elderly patients.

As a general rule I have usually declined to refer them on for surgical treatment and have opted for conservative methods. Overall my "impression" is that this has been the best option especially in the context of patient comorbidities. However for the well and active elderly patient who has sustained a rotator cuff tear after a fall, or other trauma, maybe a surgical repair is warranted. There are a few elderly patients in my practice whom I will reassess in the light of this study, which is, admittedly, only by one orthopaedic specialist from Tauranga in New Zealand. – *Dr Tom Baster*

**Littlewood C, Ashton J, Chance-Larsen K, et al. Exercise for rotator cuff tendinopathy: a systematic review. *Physiotherapy* 2012;98(2):101-9. Epub 2011 Oct 5.**

**Background.** Shoulder pain due to rotator cuff tendinopathy is a common problem. Exercise is one intervention used to address this problem but conclusions from previous reviews have been mixed.

**Objective.** To systematically review the effectiveness of exercise, incorporating loaded exercise (against gravity or resistance), for rotator cuff tendinopathy.

**Data sources.** An electronic search of AMED, CINAHL, Cochrane Central Register of Controlled Trials (CENTRAL), MEDLINE, PEDro and SPORTDiscus was undertaken from their inception to November 2010 and supplemented by hand searching related articles and contact with topic experts.

**Study eligibility criteria.** Randomised controlled trials evaluating the effectiveness of exercise, incorporating loaded exercise, in participants with rotator cuff tendinopathy.

**Study appraisal and synthesis methods.** Included studies were appraised for risk of bias using the tool developed by the Cochrane Back review Group. Due to heterogeneity of studies, a narrative synthesis was undertaken based upon levels of evidence.

**Results.** Five articles detailing four studies were included, all of which were regarded as presenting a low risk of bias. Overall, the literature was supportive of the use of exercise in terms of pain and functional disability.

**Limitations.** The results should be regarded with some degree of caution due to limitations associated with the studies including lack of blinding, no intervention control groups and limitations of the outcome measures used.

**Conclusion and implications of key findings.** The available literature is supportive of the use of exercise but due to the paucity of research and associated limitations further study is indicated.

**Comment:** Shoulder pain is the third most common musculoskeletal presentation and the diagnosis and treatment of the various conditions that affect this joint often involve a recommendation for exercises. This properly conducted review of the effectiveness of exercises for rotator cuff tendonopathy supports the view that, although the evidence is only weakly in favour, it is still a worthwhile recommendation. They report on several categories such as supervised exercises versus no intervention and home exercises versus multimodal physiotherapy. The latter option of home exercises versus physiotherapy suggests that there is no difference in outcome for patients. Part of my treatment of supraspinatus tendonopathy and impingement is demonstration and recommendation to perform home exercises for the other three rotator cuff muscles, and for latissimus dorsi and pectoralis major. I will be continuing this for now. – *Dr Tom Baster*

**Nakamae T, Ochi M, Olmarker K. Pharmacological inhibition of tumor necrosis factor may reduce pain behavior changes induced by experimental disc puncture in the rat: an experimental study in rats. *Spine* (Phila Pa 1976) 2011;36(4):232-6.**

**Study design.** Pain behavior assessment in rats following disc puncture (DP) and simultaneous tumor necrosis factor (TNF) inhibition.

**Objective.** To assess if treatment with TNF inhibition could reduce the pain behavior changes induced by DP

in the rat.

**Summary of background data.** Annular tears with leakage of nucleus pulposus have been suggested to be one possible cause of low back pain (LBP). In an experimental model, it was recently shown that DP might induce specific pain behavior changes. The aim of the present study was to a study if inhibition of TNF might reduce such pain behavior changes.

**Methods.** Sixty rats underwent facetectomy and puncture of the fourth lumbar disc. The rats were simultaneously treated with doxycycline locally at 0.3 and 3.0 mg/kg and systemically at 3.0 mg/kg, or infliximab locally at 0.5 and 5.0 mg/kg, and systemically at 5.0 mg/kg, (n=10 for each subseries). The rats were videotaped at 1, 3, 7, 14, and 21 days after surgery. The videos were analyzed regarding presence of wet-dog shakes (WDS). Data from a previous study with sham surgery and DP without treatment were included for comparison.

**Results.** All groups treated with doxycycline resulted in a statistically significant reduction of WDS compared to the group without treatment (DP). In infliximab treated animals, WDS decreased with statistical significance compared to the nontreated DP group at all analyzed days except for the group with high dose local treatment where a statistically significant reduction was obtained only at days 14 and 21.

**Conclusion.** The present study showed that TNF inhibition induced a marked reduction of wet dog shakes. It is not fully understood if wet-dog shakes may relate to LBP, but in view of recent clinical findings one may consider clinical studies of TNF inhibition for the treatment of LBP.

**Comment:** This is a fascinating report and it is starting to become clear how important some of these inflammatory chemicals, such as TNF- $\alpha$ , are in initiating and, more importantly, in maintaining pain. Doxycycline is a readily available and very cheap drug and one would hope that further studies reveal it to be a useful agent in blocking the development of chronic back pain secondary to Internal disc disruption. – *Dr Tom Baster*

**Bosscher HA, Heavner JE. Diagnosis of the Vertebral Level from Which Low Back or Leg Pain Originates. A Comparison of Clinical Evaluation, MRI and Epiduroscopy. *Pain Pract* 2012 Mar 19. doi: 10.1111/j.1533-2500.2012.00549.x.**

**Background.** The precise localization of painful structures in the spine of patients with low back pain and/or pain radiating (LBP/RP) to the lower extremities is important for targeted therapeutic intervention. The aim of the study reported here was to determine and compare the spinal segment(s) where pain was elicited via endoscopic evaluation vs. the vertebral level from where the pain was thought to originate as determined by clinical evaluation and by MRI.

**Method.** Observational cohort study of 143 patients 19 to 88 years of age undergoing spinal canal endoscopy (epiduroscopy) in a combined academic and private practice setting January 2008 to December 2008. Patients were asked

whether pain generated by pressure upon epidural structures with the tip of an endoscope was similar in character and distribution (concordant) to the pain for which patients sought treatment. Notes from clinical evaluation and MRI reports were reviewed, and segmental level determined to be the locus of pathology was tabulated.

**Results.** One hundred twenty-five (87%) patients reported maximal reproducible pain at a specific level during epiduroscopy. The most common level was at L4 to L5 (87 patients). The least common level was L5 to S1 (2 patients). In only 40 patients did the level determined by clinical evaluation correlate with the level at which pain could be reproduced during epiduroscopy. MRI indicated a specific vertebral level that corresponded to the level at which pain could be reproduced during epiduroscopy in 28 of 143 (20%) patients. The results of the 3 diagnostic methods were significantly different ( $P < 0.01$ ).

**Conclusion.** Results of this study indicate that epiduroscopy is more reliable than is either clinical evaluation or MRI for determining the vertebral level where clinically significant spinal pathology occurs in patients with LBP/RP.

**Comment:** Epiduroscopy has been around for a while now and seems to be used mainly for releasing adhesions and injecting local anaesthetic/steroid around nerve roots. Another use is for "probing" suspect discs to elicit concordant pain akin to provocative discography. Yet again, clinical evaluation and MRI comes up poorly in general terms in identifying accurately the most likely level. The L4-5 disc also keeps coming up as the usual "culprit" in discogenic pain rather than the L5-S1 level. – *Dr Tom Baster*

**Amr YM. Effect of addition of epidural ketamine to steroid in lumbar radiculitis: one-year follow-up. *Pain Physician* 2011;14(5):475-81.**

**Background.** Treating sciatica with epidural steroid injection has been a common practice worldwide. N-methyl-D-aspartate (NMDA) receptors are an important component of pain pathways.

**Objectives.** The aim of this study was to evaluate the safety and efficacy of epidurally administered NMDA receptor antagonists (ketamine) for the treatment of chronic low back pain secondary to radiculopathy and its effect on patients' quality of life.

**Study design.** Randomized, double blind controlled trial.

**Setting.** Hospital outpatient setting.

**Methods.** Two hundred participants aged 25 to 50 years with a diagnosis of lumbar radiculopathic pain secondary to disc herniation were randomized into 2 equal groups. Group I received 80 mg of triamcinolone (2 mL) and 0.25% bupivacaine (3 mL) plus 30 mg (3 mL) of preservative free ketamine. Group II received 80 mg of triamcinolone (2 mL) and 0.25% bupivacaine (3 mL) plus 3 mL of 0.9% saline. Pain scores were obtained before injection, immediately after injection, one week, one month, 3 months, 6 months, 9 months, and one year post injection. The Oswestry Low Back Pain Disability Questionnaire was used at baseline

and at one month, 3, 6, 9, and 12 months after injection for assessment of quality of life. Patients were asked to report any side effects, particularly those related to ketamine, including nausea, vomiting, visual or auditory hallucinations, and delirium.

**Results.** Immediately after injection there was no statistically significant difference between Group I and II regarding pain scale scores. After one week of injection, pain relief was significantly better in Group I compared to Group II and then at all evaluation times. The Oswestry Low Back Pain Disability Questionnaire score decreased significantly ( $P < 0.05$ ) from 72 (range 62- 83) and 70 (range 57- 82) to 8 (range 2 - 12) and 17 (range 9 - 27) at one month; 6 (range 4 - 12) and 18 (range 14 - 22) at 3 months; 12 (range 9 - 16) and 28 (range 22 - 34) at 6 months; 17 (range 9 - 24) and 31 (range 21 - 35) at 9 months; and 17 (range 8 - 22) and 33 (range 20 - 37) at 12 months in the groups, respectively. Six patients in the ketamine group showed short-lasting delusions lasting for  $45 \pm 12$  minutes after injection.

**Limitations.** The limitations include a lack of placebo control.

**Conclusion.** Epidurally administered ketamine seems to be a safe and useful adjunct to epidural corticosteroid therapy in chronic lumbar radicular pain.

**Comment:** Ketamine has been around for many years and is in use in several centres for chronic pain by infusion. This study confirms another use for this drug in improving the effectiveness of epidurals and it is not really a surprising outcome. Ketamine has, however, been misused as a recreational drug and there are reports of adverse effects from chronic use such as ulcerative cystitis and neurocognitive impairment. Whilst questions remain on the long-term toxicity, it seems that ketamine will not become a frontline musculoskeletal drug but it certainly is one to continue to keep tabs on. I have used it successfully in the past for a patient with severe pain from arachnoiditis. – *Dr Tom Baster*

**Gofeld M, Bristow SJ, Chiu S. Ultrasound-guided injection of lumbar zygapophyseal joints: an anatomic study with fluoroscopy validation. *Reg Anesth Pain Med* 2012;37(2):228-31.**

**Background.** Diagnostic and therapeutic injections of the zygapophyseal joint (z-joint) are routinely performed under radiologic guidance (e.g., fluoroscopy, computed tomography). Technically, these procedures could also be completed using ultrasound guidance, but existing evidence insufficiently supports this alternative imaging method, and it cannot therefore be recommended as a standard practice. There has also been no published proof-of-concept study using a routine fluoroscopy control for ultrasound-guided z-joint injections.

**Methods.** A cadaver study was performed to validate ultrasound as an imaging modality for z-joint injections. Fifty z-joint injections were performed on 5 nonembalmed specimens. In-plane ultrasound approach was implemented. Zygapophyseal joints were accessed through a needle



placement under the joint capsule into the posterior synovial recess. Iohexol was thereby injected, and fluoroscopy was subsequently performed.

**Results.** In 44 (88%) of 50 performed injections, the intra-articular spread of the contrast agent was clearly observed on the fluoroscopy image. In 6 (12%) of 50 cases, the contrast flow appeared in the soft tissues. In 4 of the 6 failed injections, the z-joint gap was not evident on an ultrasound image. No intravascular, nerve root, or epidural injections were observed.

**Conclusions.** Ultrasound may be a viable alternative to fluoroscopy or computed tomography as a guidance method for lumbar z-joint injections.

**Comment:** This article is more about ZAJ intra-articular injections than medial branch blocks but it shows the use of this technology is quite anatomically reliable. The more one reads about it the more it seems as if it is almost a necessity to have in the modern practice of musculoskeletal medicine. Unfortunately the machines are quite expensive and there is no rebate available for non-radiologists to help defray the cost of the equipment. If there were a rebate available to interested GPs to perform ultrasound-guided subacromial or indeed ZAJ blocks, it would actually cost the Medicare purse less than what is reimbursed to radiologists. Such logic, however, is beyond our bureaucrats but maybe it is something the AAMM should lobby for. – *Dr Tom Baster*

**Mehling WE, Gopisetty V, et.al. The prognosis of acute low back pain in primary care in the United States: a 2-year prospective cohort study. *Spine* (Phila Pa 1976) 2012;37(8):678-84.**

**Study design.** Prospective cohort study.

**Objective.** To assess the prognosis of patients presenting with acute low back pain (LBP) in a primary care setting in the United States.

**Summary of background data.** Practice guidelines for acute LBP based on return-to-work outcomes underestimate the development of chronic pain in the primary care setting. Because of differences in inclusion criteria, chronic pain definitions, and national health systems, prognostic cohort studies have reported a wide range of results limiting interpretation and generalization. Current data from carefully designed prognostic studies of acute LBP are lacking for the US primary care system.

**Methods.** Members of a large health service organization were enrolled after seeking medical care for acute LBP, with or without sciatica, of up to 30 days duration, with no episode in the past 12 months and no history of spine surgery. We conducted phone interviews at baseline, 6 months, and 2 years. Based on receiver operating characteristic analyses, a combination of global perceived recovery with pain intensity was used as primary outcome for chronic pain. Recurrence and multiple secondary outcomes were assessed to allow for comparison with other studies.

**Results.** Six hundred five patients had an average pain intensity of 5.6 (numeric rating scale = 0-10) and disability

of 15.8 (Roland-Morris scale = 0-24). Eight percent had declared sick leave between pain onset and baseline interview. Thirteen percent of 521 patients (86% follow-up) experienced chronic pain at 6 months and 19% of 443 patients at 2 years. At 6 months, 54% had experienced at least 1 LBP recurrence, and 47% in the subsequent 18 months.

**Conclusion.** The prognosis of strictly defined acute LBP, with or without sciatica, is less favorable than commonly stated in practice guidelines based on failure to return to work. Broad initiatives to develop new means for the primary and secondary prevention of recurrent and chronic LBP are urgently needed.

**Comment:** This study is really a rehash of several that have shown that a significant percentage of patients with acute back pain progress to chronic back pain or have regular recurrence of their pain. Whilst most patients do indeed recover over several weeks, we can expect a few, about 13% in this study, not to do so. This figure is similar to the 2004 electoral roll study of 3000 Australians by Walker (*J Manip Physiol Ther* 2004; 27(4):238-44) which found a 10% prevalence of significant back pain-related disability in a six-month period, although this was not necessarily chronic pain. It seems as if 80% of us will have back pain at some stage in our lives – we are indeed fortunate that most of us recover! – *Dr Tom Baster*

**Butterworth PA, Landorf KB, Smith SE, Menz HB. The association between body mass index and musculoskeletal foot disorders: a systematic review. *Obes Rev* 2012 Apr 13. doi: 10.1111/j.1467-789X.2012.00996.x. [Epub ahead of print].**

The primary aim of this systematic review was to investigate the relationship between body mass index (BMI) and foot disorders. The secondary aim was to investigate whether weight loss is effective for reducing foot pain. Five electronic databases (Ovid MEDLINE, Ovid EMBASE, Ovid AMED, CINAHL and The Cochrane Library) and reference lists from relevant papers were searched in April 2011. Twenty-five papers that reported on the association between BMI and musculoskeletal foot disorders met our inclusion criteria and were reviewed. The evidence indicates: (i) a strong association between increased BMI and non-specific foot pain; and (ii) a strong association between increased BMI and chronic plantar heel pain in a non-athletic population. The evidence is inconclusive regarding the relationship between BMI and the following specific disorders of the foot; hallux valgus, tendonitis, osteoarthritis and flat foot. With respect to our second aim, there were only two prospective cohort studies that reported a reduction in foot symptoms following weight loss surgery. In summary, increased BMI is strongly associated with non-specific foot pain in the general population and chronic plantar heel pain in a non-athletic population. However, there is currently limited evidence to support weight loss to reduce foot pain.

**Comment:** Obese patients often complain of lower limb pain, hips and knees mainly, but many also have various



foot problems. These often seem to be “plantar fasciitis” or osteoarthritis in the tibio-talar joint.

This study doesn’t really help us much as it reports only a strong association with “non-specific” foot pain. The chronic heel pain category in this review could probably be attributed to what is commonly diagnosed as plantar fasciitis in Australia but what might be called something else overseas. The conclusion that there is limited evidence to support weight loss to reduce foot pain is a bit hard to accept intuitively and may simply be a reflection of some inconclusive studies weighting this review. – *Dr Tom Baster*

**Magalhaes FN, Dotta L, Sasse A, et al. Ozone therapy as a treatment for low back pain secondary to herniated disc: a systematic review and meta-analysis of randomized controlled trials. *Pain Physician* 2012 Mar-Apr;15(2):E115-29.**

**Background.** Low back pain (LBP) is one of the most common and important health problems affecting the population worldwide and remains mostly unsolved. Ozone therapy has emerged as an additional treatment method. Questions persist concerning its clinical efficacy.

**Objective.** The purpose of our study was to evaluate the therapeutic results of percutaneous injection of ozone for low back pain secondary to disc herniation.

**Study design.** A systematic review and meta-analysis of randomized controlled trials.

**Methods.** A comprehensive literature search was conducted using all electronic databases from 1966 through September 2011. The quality of individual articles was assessed based

on the modified Cochrane review criteria for randomized trials and criteria from the Agency for Healthcare Research and Quality.

**Outcome parameters.** The outcome measure was short-term pain relief of at least 6 months or long-term pain relief of more than 6 months.

**Results.** Eight observational studies were included in the systematic review and 4 randomized trials in the meta-analysis. The indicated level of evidence for long-term pain relief was II-3 for ozone therapy applied intradiscally and II-1 for ozone therapy applied paravertebrally. The grading of recommendation was 1C for intradiscal ozone therapy and 1B for paravertebral ozone therapy.

**Limitations.** The main limitations of this review are the lack of precise diagnosis and the frequent use of mixed therapeutic agents. The meta-analysis included mainly active-control trials. No placebo-controlled trial was found.

**Conclusions.** Ozone therapy appears to yield positive results and low morbidity rates when applied percutaneously for the treatment of chronic low back pain.

**Comment:** This is another treatment that has been around for a while and seems to be mainly a European modality. The grade of recommendation of 1B for paravertebral injection seems quite attractive. Such a level is a recommendation to use in most patients without reservation, with benefits outweighing risk. I am not aware of this treatment being used in Australia and it is something that would be worth discussing with our interventional radiologist colleagues. – *Dr Tom Baster*

# Educational Activities

## Masters, Diploma, and Certificate Courses in Musculoskeletal Medicine

### Flinders University Diploma/Certificate in Musculoskeletal Medicine

Date	Title/Key Resource Person	Venue	Provider	Contact	CME Points
2012	Graduate Diploma in Musculoskeletal Medicine	Flinders Medical Centre	School of Health Sciences, Bedford Park SA 5042	Mr Don Bramwell, Ph +61 8 8204 4673; <a href="mailto:donald.bramwell@flinders.edu.au">donald.bramwell@flinders.edu.au</a>	TBA

### Australian School of Advanced Medicine, Macquarie University - Masters Degree in Musculoskeletal Medicine

Date	Title/Key Resource Person	Venue	Provider	Contact	CME Points
17-23 Feb 2013	Master of Advanced Medicine in Musculoskeletal Medicine (2-year part-time Distance Learning with 2 on campus Intensives of 1 week each in each year)	Sydney	Macquarie University, Sydney	A/Prof Rod Ayscough or A/Prof Michael Creswick via Scholar Administrator Julie Stone Ph +61 2 9812 3512 Fax +61 2 9812 3600 Email: <a href="mailto:julie.stone@mq.edu.au">julie.stone@mq.edu.au</a> or visit website <a href="http://www.medicine.mq.edu.au">www.medicine.mq.edu.au</a>	

### Australian College of Physical Medicine Fellowship Program

Date	Title/Key Resource Person	Venue	Provider	Contact	CME Points
2013	Fellowship Australian College of Physical Medicine, Part II (Part 1 is Masters in Physical Med or Musculoskeletal Med from Sydney or Macquarie Unis)	Sydney	Australian College of Physical Medicine	Michael Creswick Ph +61 2 9481 9585 <a href="mailto:michael.creswick@mq.edu.au">michael.creswick@mq.edu.au</a> or visit website <a href="http://www.physicalmedicineaustralia.com.au">www.physicalmedicineaustralia.com.au</a>	TBA

**University of Otago Diploma/Certificate in Musculoskeletal Medicine, plus new qualification:  
Masters/Diploma/Certificate in Health Sciences (Pain and Pain Management)**

Date	Title/Key Resource Person	Venue	Provider	Contact	CME Points
Feb-June 2012	<i>Distance taught papers</i> MSMX 704: Pain  MSMX 708: Pain management  MSMX 705: Regional Disorders (Spine)  MSMX 706: Regional Disorders (Limbs)	Distance taught papers - fortnightly audioconferences ex University of Otago, Christchurch	University of Otago	Enrolments: Veronica McGroggan Ph +64 3 364 1086 Fax +64 3 364 0909 <a href="mailto:veronica.mcgroggan@otago.ac.nz">veronica.mcgroggan@otago.ac.nz</a> or Geoff Harding Ph +61 7 3269 5522 Fax +61 7 3269 6407 <a href="mailto:drgeoffh@bigpond.net.au">drgeoffh@bigpond.net.au</a> website <a href="http://www.uoc.otago.ac/departments/msm">www.uoc.otago.ac/departments/msm</a>	Mixture of points, including small group points
June-Dec 2012	MSMX 707: MSM Rehabilitation  MSMX 710: Recreational and Sports Injuries  MSMX 711: Pain Assessment  PAIX 701: Neurobiology of Pain  PAIX 702: Biomedical aspects of pain  PAIX 703: Psychosocial and cultural aspects of pain				
27-31 Aug 2012	MSMX 701: Clinical Diagnosis - on campus paper, Part 2				

**Other Musculoskeletal Medicine Educational Activities**

Date	Title/Key Resource Person	Venue	Provider	Contact	CME Points
14-16 Sept 2012	Australasian Musculoskeletal Medicine conference Theme: "Joint matters"	Wellington	AAMM AFMM NZAMM	<a href="http://www.musculoskeletal.co.nz/content/conferences">www.musculoskeletal.co.nz/content/conferences</a>	
20-22 July 2012 and November 2012	Prolotherapy and neural prolotherapy - injections for pain	Physiotherapy Association, Adelaide	Dr Margaret Taylor	Email: <a href="mailto:taylorme@internode.on.net">taylorme@internode.on.net</a> Ph: 08 8379 1254	40 RACGP Category 1 & ACRRM 6 + 20

# JOINT MATTERS

*Topical issues on joint function, disease, pain, treatment, rehabilitation*Friday 14<sup>th</sup> to Sunday 16<sup>th</sup> September 2012

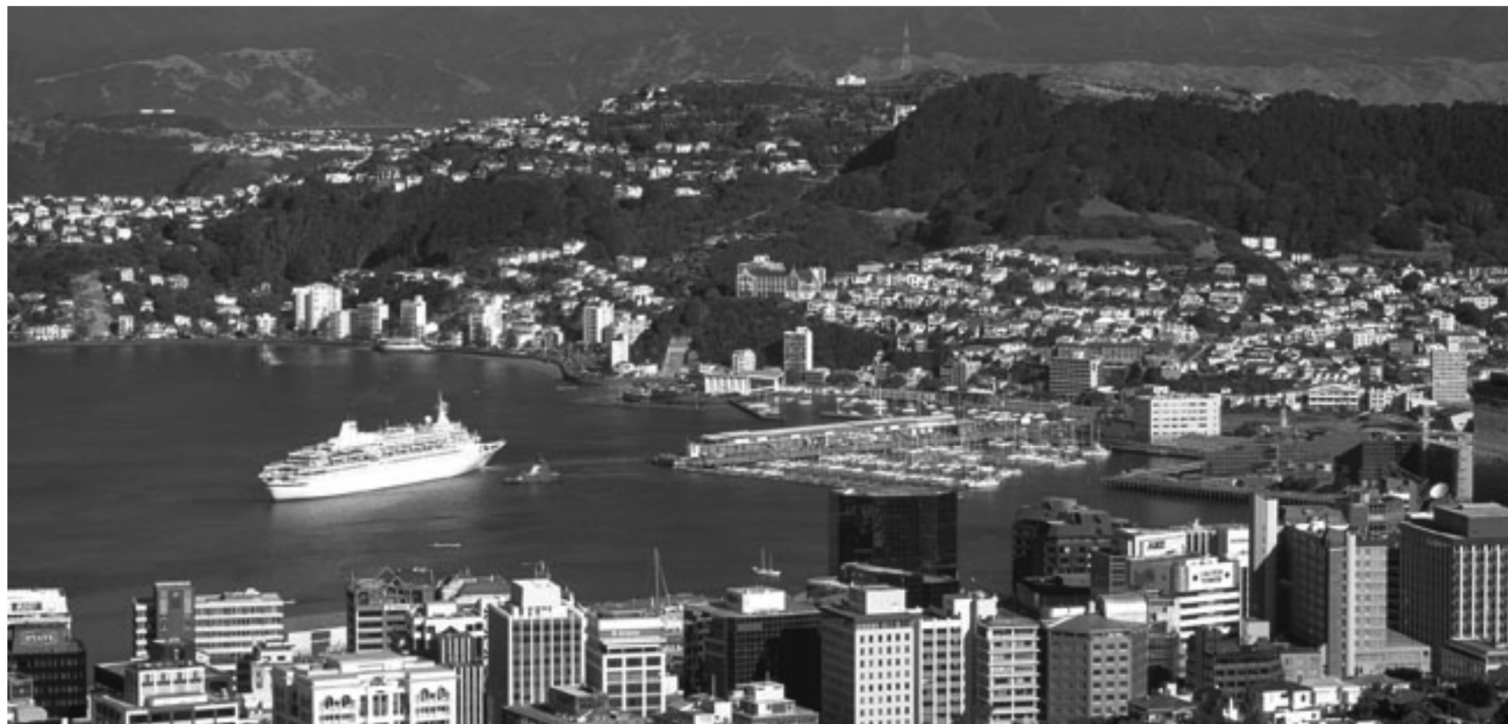
Amora Hotel, Wakefield Street, Wellington, New Zealand

Combined meeting hosted by: NEW ZEALAND ASSOCIATION MUSCULOSKELETAL MEDICINE  
AUSTRALIAN ASSOCIATION MUSCULOSKELETAL MEDICINE – AUSTRALASIAN FACULTY MUSCULOSKELETAL MEDICINE

Prof Lars Arendt-Nielsen	Dr Jeremy Lewis	Assoc Prof Gary Hooper	Dr John MacVicar Dr Jim Borowczyk	Other Speakers
Alborg University Denmark Center for Sensory-Motor Interaction  Joint & musculoskeletal pain – mechanisms & therapy	Consultant Physiotherapist & Visiting Professor  Research Lead, Therapy Department, Chelsea and Westminster Hospital  Shoulder pain diagnosis, impingement, tendinopathy, injection & rehabilitation	University of Otago Christchurch, NZ  Associate Professor & Head of Department of Orthopaedic Surgery & Musculoskeletal Medicine  Surgical advances in osteoarthritis, articular cartilage transplantation; meniscal injuries and osteoarthritis	Musculoskeletal Pain Physicians  Christchurch, NZ  Chronic cervical and lumbar zygapophysial joint pain: diagnosis and treatment	Dr Terence Macedo, Rheumatologist  Spondyloarthropathies ..... Greg Lynch, Physiotherapist  Knee joint rehabilitation: postoperative, ACL, patellofemoral pain ..... Orthopaedic Surgeons  Hip pain, Femoroacetabular impingement, Ankle & foot ..... Podiatrist  Gait & foot biomechanics ..... Musculoskeletal Physicians  Occupational Therapist  Radiologist

The 2012 Conference program combines keynote lectures involving basic sciences to clinical application, complemented by workshop sessions. Clinical presentations will be from musculoskeletal physicians, rheumatologists, orthopaedic surgeons, physiotherapists, podiatrists and occupational therapists. Presentations will highlight modern advances and research with an emphasis on both evidence based and practical medicine.

The audience will be a combination of medical practitioners, both specialists (musculoskeletal physicians, pain physicians, occupational physicians, rehabilitation physicians, sports physicians, rheumatologists, orthopaedic surgeons) and general practitioners, and allied health personnel from the nursing, occupational therapy, podiatry, manual therapy and physiotherapy professions.



THIS IS AN ADVANCE MEETING NOTICE. A FULL PROGRAM AND REGISTRATION PACKAGE WILL BE AVAILABLE LATER IN THE YEAR. PLEASE MARK YOUR DIARY FOR 14-16 SEPTEMBER 2012