

Australasian Musculoskeletal Medicine



- IAP global year against musculoskeletal pain
- Can we be more specific about back and neck pain?
- Use of a “polypill” for acute tendinopathy
- Caudal epidural steroid injections
- Acupuncture in the treatment of osteoarthritis
- Low level laser therapy for neck pain

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Editorial

Use of Pain Diagrams and VAS Charts Post-Procedure

Dr Geoff Harding, Sandgate Spinal Medicine Clinic, Sandgate, Qld

This Journal has an eclectic mix of topics including traditional acupuncture, low level laser acupuncture, epidural injections, a paper by Williams addressing the topic of diagnosis, and Tom Pietzsch's article detailing his experience using caudal epidural injections over a long period of time in practice in North Queensland. The Journal has published a number of papers on this topic in the past, including a contribution by Vic Wilk outlining the protocol recommendations of the International Spinal Intervention Society. Another was a review by Vic Wilk and David Vivian in 1990 on the use of epidurals. Still another was a retrospective study by Chris Jackson and Norm Broadhurst on the use of epidurals in country hospitals in 2000. The Pietzsch article clearly argues that the procedure is safe and effective and worth considering in certain patients who might otherwise be relegated to therapies which might provide no relief at all. Some will argue that this procedure should be done only in a hospital by specialist anaesthetists while others will support the continued use of "caudals" out in the field as part of a relatively low-tech but beneficial add-on to musculoskeletal pain management.

This edition of the Journal contains a number of articles dealing with treatment methods of musculoskeletal pain. In our daily practice, we have to decide whether an intervention has worked in terms of pain relief. Given the widespread nature of pain problems it might be useful to consider the use of pain diagrams not only prior to an intervention, but also AFTER an intervention.

Pain diagrams and pain maps have proved to be very useful in predicting sites of pain pathology in spinal conditions.¹ There is good evidence that they can predict the outcome of various interventions to obtain pain relief. Likewise, visual analogue scales (VAS) are useful in the initial assessment and then the subsequent follow-up of the progress of a pain condition. If, after an intervention, the VAS is reduced by a clinically important factor (usually at least 30% reduction) then perhaps the intervention was of use.

For practitioners who rely on others to perform needle interventions (or any intervention for that matter), it is necessary to ensure that proper use of these pain assessment instruments is appreciated by your interventionalist. In my experience, although an increasing number of radiologists are performing blocks at our behest, they often do not seem to appreciate the value of proper post-procedure follow-up with the patient. Even those who follow the ISIS Guidelines for procedures such as medial branch blocks and nerve root blocks don't always seem to understand the value of

detailed assessment of the outcome. The best way to do that is by interviewing the patient at a suitable time after the procedure, by using a post-procedure pain diagram, and by obtaining an appropriate VAS chart.

A search of the literature shows that most of the reference to use of pain diagrams is in the context of **pre-interventional** measures. Pain diagrams can be used to predict the source (or segment) involved in the pain. However, although visual analogue scales are used routinely to rate pain after an intervention, it is not the case with post-interventional pain diagrams.

Figure 1 is the pain chart of a patient who presents with a pain condition who presents to you for treatment. You might have decided (after appropriate history-taking, examination and trials of treatment) that this is an example of lumbar spine somatic referred pain. You might feel inclined to send this patient for a medial branch block of one or two levels. Since the pain is predominantly left-sided it might be better to perform left-sided blocks first and to address the right-sided component later.

Let's say that you decide to ask for a medial branch block of the left L5/S1 zygapophyseal joint. And let's assume that you know that the radiologist uses (and will record on his report) bupivacaine 0.5% (half-life 6 – 8 hours) as the anaesthetic in this procedure.

In my city, the patient currently is asked by the radiologist to complete a VAS prior to the intervention, then to fill in a VAS at eight-hour intervals for the next 36 hours. No pain diagram is used and no interview takes place between the

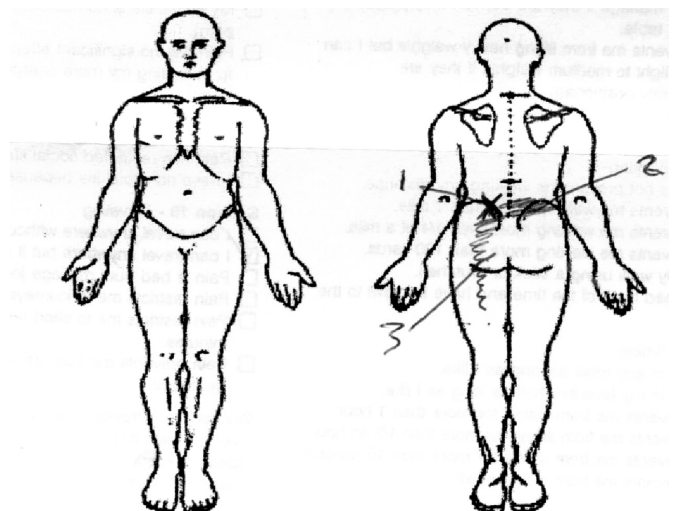


Figure 1.

radiologist (or nurse) and the patient.

Your patient returns to you one week later to discuss the results. The patient tells you that there was no relief from the injection at all. You look at the pain chart and it indeed shows that at the first interval recorded (+8 hours) the pain level on the VAS is “7” – the same as it was prior to the procedure. What are the implications here? Likely that you will decide that the L5S1 zygapophyseal joints is not the source of the index pain.

But wait! Many patients have a “vegetable soup” of pain – some of their pain might indeed be coming from the L5S1 zygapophyseal joint, but some part of their pain might also be coming from some other (secondary) pain generator close by. It is uncommon for a patient to have a pure pain pattern where all of the pain is caused by one site (or indeed one tissue). In fact, the percentage of patients with chronic low back pain having one z joint as the sole source of their pain is now said to be as low as 6%.

What needs to be done is to first get the patient focussed prior to the intervention so as to assess the results properly. Figure 2 is the (hurriedly prepared some years ago and never altered since due to lack of time!) pain assessment tool I use for such patients. I tell them “whatever forms the radiologist gets you to complete, I want you to complete this as well”.

You will see that they have to fill in the total area of their pain just prior to the procedure with horizontal lines and then they cross out (using vertical lines) the area where their pain either disappeared or was substantially reduced. Naturally, for a mixed pattern of pain, only part of the initial area will be crossed out. I then ask them to focus on that area (where the pain has either reduced or gone) and **then** fill in the VAS chart – at, say, two hours post-procedure. “Record the level of pain in this area”. “If you still have some pain elsewhere, fine. This might be coming from some other level – we can deal with that later”.

The VAS chart I use is to be filled in at **hourly** intervals (not eight-hourly intervals which is the norm for my city). This provides a more accurate record of the pain relief because bupivacaine lasts only a short time and it is during that time we need to monitor the pain levels.

It is also important to arrange a follow-up appointment soon after the intervention to discuss the results. The patient needs to have the whole procedure fresh in their mind. This is not possible after a one week (or more) interval. It is ideally done *within* two days of the procedure. It is important to tell the patient prior to the procedure that you want them to recall the details of the procedure at this appointment. “Priming” the patient in this way is more likely to yield useful information.

In my practice I usually find that there are areas of the pain which have totally disappeared with the block whilst there are other nearby areas which are totally unaffected. If not properly assessed, patients will usually “average” their overall pain and give comments like “I think the pain dropped from a 7 to a 4” when in fact the incident pain disappeared and the other pain remained at “7”.

In spite of my providing my patients with this type of pain assessment and telling them to take it with them, I am

surprised to see that many radiologists persist in the 8-hour pain chart and nothing else.

(At another time I might be tempted to discuss the wide variation in the techniques used in performing these blocks – in spite of the existence of the ISIS Guidelines, but that is another matter.)

I would suggest that you make a copy of my pain diagram/VAS combo (Fig. 3) and give that to each patient you send for any diagnostic block. We might be able to influence the radiologists yet!

1. Bogduk, N. The physiology of deep somatic pain. *Australas Musculoskeletal Med* 2002; 7(1).

Surname: _____ First Name: _____
 Date of Injection: _____
 Injection Type & Level's: _____

1. Prior to injection Colour in with horizontal lines where the pain is.

2. At 1 or 2 hours after the injection cross out with vertical lines where the pain has gone.

Figure 2.

Surname: _____ First Name: _____
 Date of Injection: _____
 Injection Type & Level's: _____

1. Prior to injection Colour in with horizontal lines where the pain is.

2. At 1 or 2 hours after the injection cross out with vertical lines where the pain has gone.

Figure 3.

The International Association of Pain (IASP) announces the Global Year against Musculoskeletal Pain - October 2009-October 2010

When moving hurts: Assess, Understand, Take Action

Dr Philip Watson, Musculoskeletal Medicine, Sunnybank Qld

The IASP is focusing on musculoskeletal pain this year. See www.iasp-pain.org/GlobalYear/MSP. I encourage you to look at their website for information, including:

- Campaign details and update
- Events and media coverage
- Musculoskeletal pain fact sheets, and
- Publications and other resources.

Following is an abstract from their website.

Why Musculoskeletal Pain?

IASP decided to focus on musculoskeletal pain because it is an enormous problem that affects millions of people worldwide. According to leading pain experts—including IASP Global Year Co-Chairs Dr. Lars Arendt-Nielsen of the University of Aalborg, Denmark, and Dr. Kathleen A. Sluka of the University of Iowa, USA—more people around the world experience musculoskeletal pain than any other type of pain.

The problem of musculoskeletal pain is complex and far-reaching, encompassing many different types of pain, such as neck pain, limb pain, low back pain, joint pain, bone pain, and chronic widespread pain, just to name a few. Yet, despite the wide-ranging conditions and symptoms, all types of musculoskeletal pain share similar underlying mechanisms, manifestations, and potential treatments.

Challenges and Issues

IASP has identified several major challenges surrounding musculoskeletal pain that the Global Year campaign must address, including the following:

- *Treatment for musculoskeletal pain is not adequate.*
- *At the chronic level, musculoskeletal pain is typically managed, but not cured.*
- *It is often difficult to relate pathophysiological changes to the patient's actual pain, which makes musculoskeletal pain especially challenging to diagnose.*

Moreover, even when the source of the musculoskeletal pain is identifiable, it can still be difficult to link the source and the severity of the pain, as they do not always match. Although the patient's pain is real, you cannot always see it. He or she is in pain, but the clinician cannot determine why or pinpoint the source.

Beyond the suffering and discomfort associated with musculoskeletal pain, there are huge financial and other costs, including medical care expenses, lost work days, and diminished quality and productivity in patients' work and personal lives—all of which are fuelled by worldwide trends, including:

- *Aging populations*
- *Sedentary lifestyles*
- *Increasing incidence of obesity*

New Features and Support Materials

IASP is pleased to offer several new features and support materials for the 2009–2010 Global Year campaign:

- *Online discussion forum on musculoskeletal pain studies*
- *Global Year Event Checklist for chapters and members planning local events*
- *Global Year poster in three sizes, available to download for free from the IASP website, and to print and display at your local chapter meetings and Global Year events (also provided in the centre of the September IASP Newsletter)*
- *Video interviews (to be posted online) discussing several topics related to musculoskeletal pain.*

I encourage you to look at the 23 fact sheets. These 1-2 page notes highlight the important issues, with references, pertaining to each topic

In a multicultural society in which many of us practise, I have often searched the web for information to give patients where English is their second language.

The fact sheets are available in English, Spanish, French, Arabic, and Chinese. Eight fact sheets are also available in German.

From the AAMM President

It has been almost 12 months since the AGM on the Gold Coast and my election as president of the AAMM. I am sorry to say that I had planned to ensure that we had had an issue of the Journal to you long before this. No excuses can be offered except that time seems to be in great demand these days.

The job of editing a Journal is time-consuming. It requires a large commitment of effort and, in the case of our Journal, no financial reward. There have been a number of very committed editors of our Journal over the years and they deserve our highest regard for having done a fantastic job of editing *Australasian Musculoskeletal Medicine*. Unfortunately we can't rely on committed people like them anymore. None of us has the time to take from our families to work for the benefit of others.

Also, the cost of editing a journal is high. Each issue including editing and printing costs about \$10,000. So the committee, having tried to organize the editing of the Journal using an editorial board without success, is considering limiting our Journal to only one issue per year but also subscribing to *International Musculoskeletal Medicine*. The cost of supplying a subscription to all of our members in Australia and New Zealand is actually less than what it costs to publish one issue of the Journal. In return, there would be four journals per year instead of our average of two issues. The subscription would allow members to access the website as well. Hopefully the Newsletter will keep us up-to-date on all of the issues affecting the discipline in this part of the world.

Sadly, last year we lost Jay Govind. Jay's sudden death was a shock to us all, though his life and friendship was an inspiration to us all. This Journal carries a eulogy written by his close friend Professor Nik Bogduk. The signature tune played at Jay's funeral was the jazz classic "Take Five". So, take five to read and reflect on Jay and his influence on musculoskeletal medicine and his friends.

I have been thinking about the theme for this president's address and the word which keeps coming into my head is "change". The significant change at the time of writing this is that the country has a new prime minister. A Gillard government is unlikely to change significantly the direction of health policy from what has been initiated so far.

The planned changes in the delivery of medical care in this country are going to make "doctoring" interesting, to say the least. For those who are in general practice, the changes are fairly well defined (though lots more detail to come).

For those of us who practise musculoskeletal medicine full-time, there is less certainty about the future. My feeling is that there will be more involvement of allied health practitioners in the Medicare system. It is in this environment that I feel that GPs should re-think the way they practise.

In the management of musculoskeletal pain, there is a trend away from "hands-on" manual therapy and more concentration on posture, core strength, and cognitive therapies. I believe that this is leading to a situation where the number of doctors having manual skills will start to decline and that is not necessarily a good thing for the patient who presents with a musculoskeletal pain problem.

We need to maintain our manual medicine skills because our approach is not the same as the allied health practitioners and therefore offers a different perspective for our patients. That means we need to promote our organization as a valuable player in the management of musculoskeletal pain. This was brought out to me at the recent launch of the National Pain Strategy in Canberra where it is apparent that there is little recognition of somatic referred pain as a contributor to chronic pain problems. It seems that most of the pain physicians believe that all the pain is central (neuropathic) pain.

Another realization is that our discipline seems to be equated with physiotherapy or chiropractic. We need to highlight our unique perspective – potentially a "one-stop-pain shop" where the practitioner has skills in psychiatry/psychology, anatomy, pathology, pharmacology, surgery, medicine as well as manual skills.

GPs should reclaim the "hands-on" approach to the extent that others have resigned from it. Hands-on techniques can include injections/blocks and does not mean manipulation alone. We know that a good hands-on examination can, if nothing else, establish or enhance a therapeutic bond between the patient and the treating practitioner. Having established that bond, it facilitates treatment by whatever methods are used – because the patient believes in the process and becomes a willing partner in the rehabilitation journey.

The AAMM annual conference on the Gold Coast featured workshops which were well-attended and the comments from those attending expressed an enthusiasm for learning "hands-on" techniques.

All of the foregoing does not imply that we should abandon our science or delude ourselves into thinking that all musculoskeletal pain problems can be "cured" by manual techniques, but I do think that there is a valid place for maintaining the art as well as the science.

The other change that seems to be on the way is the increasing role that government and bureaucracy seems to be claiming in the practice of medicine – and this will affect musculoskeletal medicine as well.

Some recent cases involving members of the Association and the PSR raise the issue of what is "acceptable practice" for GPs. It seems that some members of PSR panels have the view that any treatment which does not fit the usual "orthopaedic/rheumatological" approach to musculoskeletal pain management is not acceptable practice. This is in spite of the wealth of literature in pain medicine which supports most of what we do.

We need to continue to educate GPs in the art and science of musculoskeletal medicine and show how they can utilize skills which were not taught in medical school yet are evidence based and ethical and effective in managing musculoskeletal pain problems.

Finally, for those who were at the AGM on the Gold Coast where there was discussion around the topic of future amalgamation of the Association with the College of Physical Medicine and the Faculty, I have to report that that has not

continued next page

From the NZAMSM President

The last six months has been busy for the officers and executive of the Association. Late in March many months of organization finally came to fruition with the conference "Spine in Action: Low Back Pain – Can Chronicity be Prevented?" This was held at the Rendezvous Hotel in Auckland.

Invited keynote speakers were Professor Lars Arendt-Nielsen from Denmark, Professor Johan Vlaeyen from Belgium, Professor Jacob Patijn from The Netherlands, and Professor Paul Watson from the UK. They were ably supported by Dr Duncan Reid, Dr Wade King, Professor Nikolai Bogduk, Dr Wolfgang von Heymann, Dr Peter Robertson, Dr Alastair Wilson, Ms Kirsty Powell, and Mr Chris Polaczuk.

Workshops were run by a variety of New Zealand & Australian Association members. The conference was judged a major success for the quality of the program, speakers, and overall relevance.

On behalf of the organizing committee I thank all those who contributed. I would also like to acknowledge the support I received from Charles Ng and Peter McKenzie. The Association is indebted to the sponsorship of the Rose Hellaby Medical Trust, MundiPharma, APT Pharmaceuticals, Douglas, ACC and, lastly, the conference organiser, Sue Peck.

More recently the executive's attention has focused on preparing a submission at ACC's request on the future role of General Practitioners with Special Interest for the triage of patients with musculoskeletal/orthopaedic problems in those regions where access to orthopaedic services are limited. Dr Michael Hewitt is kindly leading this.

It has been a long-held contention of the executive that the Association should have been involved with the initial GPSI pilot introduced in 2005. The executive welcomes this opportunity and hopes it affords diplomates with an additional opportunity to utilize their skills.

The Association strongly supports and endorses the Department of Orthopaedics and Musculoskeletal Medicine proposal of offering a Masters of Health Sciences endorsed in Pain and Pain Management by the University of Otago.

Dr James Watt continues to represent the Association at FIMM meetings; FIMM is pursuing specialist recognition both in Europe and with WHO. The executive sees this as an important and exciting development which, if successful, would give greater recognition for all medical practitioners practising musculoskeletal medicine/ manual medicine.

The Association is sponsoring Dr Wolf Schamberger to NZ in October. It is proposed the meeting will be held in Christchurch. He will give his highly respected workshop on sacroiliac joint dysfunction. Tentatively the workshop will be held Saturday 30 October in conjunction with a Faculty retreat. The Association will hold its annual general meeting at that time. Notices confirming the meeting, retreat, and AGM will be sent shortly.

This is the last president's report for the "Green Journal" I will be writing as my term comes to an end later this year. It has been a privilege and an honour to have served as the Association's president the past two years.

Dr Charles Ng is our president-elect and I know the Association will benefit from his leadership.

Gary Collinson

From the AAMM President, continued

been progressed any further. However, I have made two submissions in that time – one to the Senate Community Affairs Committee Inquiry into the National Registration and Accreditation Scheme for Doctors and other Health Workers on behalf of the three groups and the other submission to the National Pain Strategy on behalf of the College of Physical Medicine as well as the Association. This, I think is evidence that we can begin to cooperate more when promoting the discipline in this country. I have also proposed that the three organizations (as well as the New Zealand Association) engage a single Secretariat to promote the cause as well as to present a common voice to the outside world. This plan is already starting to be trialled in New Zealand now and when the teething problems are sorted, we hope to get agreement for the various players in Australia to join under a common Australasian secretariat. What a nice change that would make!

Geoff Harding

Vale Jay Govind: A Thorn in Your Side But My Close Friend

I knew little of Jay Govind's early life. Whatever he shared with me about those years came in snippets – but informative and revealing ones, each of which was the foundation of one feature or another of the values and attitudes that he expressed in later life.

He remembered with joyful relish his childhood in South Africa. He worked in his father's corner shop, which was located in a suburb for Indians. The community comprised Hindus, Muslims, Seikhs, and others. Yet this community was galvanized by its ostracization in common, under Apartheid. Jay often remarked how not just the children, but the families, shared what became a hybrid culture. They did not discriminate by race or religion; they shared language; they shared cuisine. For Jay, the character in Yann Martel's novel *Life of Pi* was not fiction. Tigers notwithstanding, that novel encapsulated Jay's childhood life. His multicultural origins created Jay the Renaissance Man – albeit of a Hindu flavour.

His remarks about his time at university were more somber. He, himself, did not convey bitterness, but bitterness was the emotion that his stories evoked in me. He recalled how the coloured students were treated in medical school. They had to sit at the back of the lecture theatre. They were permitted last turn at the anatomy specimens and pathology specimens, and were allowed access only to the poorer specimens.

During his student years, and as a resident, Jay was exposed to an amazing spectrum of medicine. In South Africa he saw and treated conditions that, in the first world, are only words in textbooks. He saw aspergillosis. He saw asbestos – not covered by workers' compensation. He treated parasitic diseases that those in Australia learn only as answers for the American visa-qualifying exams.

As a resident he explored anaesthesia, and had training in radiology. These themes were to be resumed later in his career when he embraced interventional pain medicine. Jay had the aptitude, knowledge, and intelligence to become someone important. I believe that he had the desire to become someone important. All he needed was the formality of a career path, training, qualifications, and patronage. But this formality was denied to him. He recognized that doors were closed to him for a career path in South Africa. So, he emigrated to Australia where he trusted that he might succeed.

In Australia, Jay became the clinical superintendent at Gosford Hospital. In that position he expressed his values. He believed in – and he represented – quality, dedication, and accountability. He urged these values in his hospital. They were not welcome. So, neither was Jay. This dissonance with the establishment was a feature that, ironically, later made him welcome with his final set of professional colleagues.

Leaving the hospital system, Jay established a general practice in Terrigal, a small coastal village about 100 km north of Sydney. He also undertook training in occupational medicine. He was a member of the second class of students to undertake this emerging specialty at the University of

Sydney. He graduated with a Diploma of Public Health, and was amongst the founding members of the Australian College of Occupational Medicine. Later, Jay served on the executive of that College. He strove to convince his colleagues of the importance of studying musculoskeletal medicine properly. In this he was thwarted, within that organization, but he prospered elsewhere.

I first encountered Jay on paper. In 1991 the Cervical Spine Research Unit, at the University of Newcastle and the Mater Hospital Newcastle, started studying patients with chronic neck pain after whiplash. At that time, Jay was the medical officer for GIO Insurance in Newcastle. The role expected of him was to undertake assessments of patients with compensation claims, and to compose skeptical reports about them. We started presenting papers at conferences, announcing the results of our research. Jay attended those conferences. Indeed, I remember clearly that there was this entity: a face that appeared at every meeting – of the Pain Society, or of the Association for Musculoskeletal Medicine, or at one-off conferences on whiplash or back pain. What's more, Jay paid attention. He listened. He learned. He thought.

Previously, whenever we received patients, they typically arrived with three reports from insurance doctors, each of which said the same thing: there was nothing wrong with the patient. We were able to prove otherwise, with the tests and treatments that Les Barnsely, Susan Lord, and Barbara Wallis developed. They were PhD students at the time. Then gradually we noticed a change. No longer were there three opposing reports. These became two reports hostile, but one that explained that, in fact, there was something wrong with the patient, that they genuinely did have pain, and that it could be diagnosed. Jay had not converted because of fashion. Rather, he saw the results of new evidence, and acted accordingly.

He became interested in this evidence and its discovery. Our tests involved injecting local anaesthetic under x-ray guidance, in order to find where the pain came from. This resonated with Jay's earlier training in anaesthesia and radiology. He also preferred truth to convention. Writing reports that patients did not have pain was incompatible with the evidence.

He resigned his well-paid job as an insurance doctor to take a lowly paid position with us at the university as the first Fellow of the Cervical Spine Research Unit. In that position, he learned and practised our techniques. As the PhD students graduated and pursued their own careers, Jay became the principal instructor in the Unit. Over many years he taught, mentored, corrected, and directed several doctors in various ways. He looked after Greg MacDonald who wanted to learn how to bring relief to patients in pain. Greg would travel each week from Sydney to Newcastle to spend two days with us.

Later, Geoff Speldewinde did the same, travelling each week from Canberra to learn from Greg.

As well, to various extents, Jay supervised or assisted general practitioners – Wade King and Phil Giles who wanted to learn the skills, and later Ian Painter, an anaesthetist who returned from Holland to take up pain medicine.

From time to time various New Zealanders came to visit and be inspired. They watched Jay perform in the procedure room. In the laboratory, Jay contributed to cadaver courses on how to perform the procedures properly. But in particular, Jay strived to get inside the minds of these doctors, to get them to think, and to see, and to realize what was happening. His particular passion was to have trainees realize not just what they were seeing on an x-ray but what that x-ray was showing yet still not being seen. This subtlety and inscrutability was a hallmark of Jay's teaching. He was not didactic. He wanted to leach realization out of his students.

In this way, Jay expressed the skill and passion of the academic and teacher that Jay would have been had not his career in South Africa precluded. He should have been the professor, but he had been denied the critical early steps through which to achieve this rank. Instead, he gave to his colleagues the virtues of an expert and teacher without ever receiving, yet never requiring, the privileges and social status of academic rank. Jay's academic thirst was expressed in several ways.

He was highly respectful of the work conducted by the young PhD students who preceded him in our Unit. I think he harboured an envy. He would like to have been them, had he been younger and earlier in his career. He certainly became the most vociferous champion of their work in later years.

Becoming, as it were, the second wave of research students in the Unit, he urged us to do more studies, to which he could contribute. He progressively undertook several projects. His first and foremost study established the effectiveness of a neurosurgical operation for the treatment of headache caused by injuries to the neck. His first publication has become the benchmark for this procedure, called radiofrequency third occipital neurotomy. The treatment of headache after whiplash became one of his crusades.

Subsequently, he urged us to perform a cadaver study to show how to perform lumbar radiofrequency neurotomy correctly. More than any other study, this latter study represented Jay's urge to have doctors do things the right way. Later, he participated in a study that described how to perform intradiscal electrothermal therapy for back pain. In the meantime, Jay joined the first class of students who undertook the Diploma in Pain Medicine at the University of Sydney. He continued by converting the diploma into a Masters degree. His thesis has remained a seminal work. He described the differences between neuralgia, neuropathy, and radicular pain, and how these conditions were frequently confused and mistreated. His thesis became the basis of the book that he wrote on evidence-based treatment of lumbar radicular pain.

Jay became president of the Australian Association of Musculoskeletal Medicine when "muscle" was "flavour of the month". In this position he urged members to pursue the truth, rather than the polemic. He was hostile to preaching

about trigger points in the absence of evidence. He opposed the unbridled use of botulinum toxin for conditions in which it patently did not work.

Quietly, behind the scenes, but at the forefront of debate in private, Jay supported the formation of the Australasian Faculty of Musculoskeletal Medicine, and became one of the founding Fellows – by examination – for he opposed privilege by grandfather clauses. As an examiner for subsequent Fellows he promoted questions that others could not understand, but which I could see were clever and cunning.

When the Australasian Faculty of Musculoskeletal Medicine succeeded in convincing the government to fund the National Musculoskeletal Medicine Initiative, Jay joined the executive headquarters of this project. In that role he participated in the analysis of data, and was one of the authors who published the first study on the effectiveness of evidence-based treatment for low back pain.

When the Australasian Faculty of Musculoskeletal Medicine was invited to contribute to the Encyclopaedia of Pain, Jay was a prolific contributor. Persistently, his attitude was for doctors to get it right: to acknowledge the truth, and not simply to comply with fashion and popular hearsay.

It was a characteristic of the Cervical Spine Research Unit, and what became the Newcastle Pain Management and Research Unit at the Royal Newcastle Hospital, that it attracted the rejects and foundlings of society. These were people who disagreed with society or whom society had rejected or never accepted. This became Jay's intellectual environment. Yet it produced work that embarrassed society and its beliefs. Jay seemed to be at home with this. One of his remarks was that the insurance industry could not beat the science of seven doctoral theses on whiplash, so they had to pass an act of parliament to avoid the evidence.

Retiring both from Newcastle and from occupational medicine, Jay became a staff specialist in pain medicine, firstly at Liverpool Hospital in Sydney and eventually at Canberra Hospital. It was in Canberra that he blossomed. His family affirms that the Canberra appointment was the best job he ever had. As head of the Pain Management Unit in Canberra he rejoiced in the staff that he had, and reciprocally his staff admired him for how he looked after his patients and how he supported the department.

Joining the Spine Society is not a great social achievement, but the occasion provided a vignette that encapsulated so many features of Jay and his struggle. Jay applied as an occupational physician and insurance doctor. But he was not an orthopaedic surgeon. Therefore, he was not of worthy status. The proposal was that he be offered associate membership, for he was not well enough known to warrant full membership. It was with controlled anger that I spoke: that as an insurance assessor, Jay had seen countless patients with spinal pain, and had attended more continuing education meetings on the topic than the entire gathered membership combined. The Society conceded. Jay was admitted as a full member, and remained so. In later years he was awarded the prize for best presentation at the annual meeting of the Spine Society of Australia for his work on

neck pain and headaches. In his own right, he achieved the same award as had previously the young PhD students whom he followed, and by whose work he had been inspired. Jay joined the International Spine Intervention Society, and became a regular delegate to its meetings, flying across the Pacific Ocean to attend. In due course he was invited onto the Faculty for these meetings.

With his early lectures, the Americans could not cope. The Indian humour, the satire, subtlety and irony were too much, as were the persistent mentions of 200 Hindu gods. But a climax occurred in 2008. Cervical radiofrequency neurotomy had come under attack by academic pundits. Their assault was vile and founded in falsehood and sophistry. At the ISIS meeting, Jay gave a lecture. He harvested every review that had ever been written. He harvested everything that insurance companies around the world had written. He harvested what judges and lawyers had written. He rose to the defence of the procedure that the PhD students had validated. His lecture was the only lecture ever to receive a spontaneous standing ovation at an ISIS meeting. Previously unintelligible to the American palate, Jay now became the hero of ISIS.

In recognition of his endeavours to promote research and scholarship, and to pursue truth in medicine, Jay was appointed to the editorial board of the journal *Pain Medicine*, as a representative of ISIS. Repeatedly at meetings, he urged members of ISIS to understand how important it was to support and to use this journal. Not rhetoric or politics, but peer review and esteemed scholarship were the means that he urged by which to achieve recognition, and to overcome opposition and rejection – acts and objective achievements, not words and desires.

In 2008, Jay was invited to join the board of directors of the International Spine Intervention Society. He served as chairman of the Standards Committee. In this role he strove to complement the technical guidelines for how to perform procedures correctly with peer-reviewed evidence on the validity and efficacy of these procedures. This work remains unfinished, but his colleagues in New Zealand and Australia have vowed to see his legacy come to fruition.

The esteem with which Jay was held by his colleagues in ISIS was revealed when they learned of his passing. Within minutes of hearing the news, the members of the Executive, independently but uniformly, proposed a memorial to Jay, and offered a scholarship endowment to look after any young children whom he may have still been educating.

At the time of his passing, Jay had three publications about to appear. One of the causes that he adopted and fostered was cervicogenic headache. His own research contributed to the scientific basis of this condition, but resistance to the concept persisted despite the evidence. One of his two last papers was a review article – on cervicogenic headache – invited by the journal *Lancet Neurology*, in which he got to put his case.

His second final paper was on lumbar radiofrequency neurotomy for low back pain. In June 2009, this procedure came under attack – from guidelines committees in the UK, and from insurance companies across the USA. Jay joined

members of ISIS to produce rapidly a review of the evidence for this procedure, pointing out how the procedure had been misrepresented, and how the procedure actually does work if only you did it the way that Jay urged that it should be done – properly. This paper will appear posthumously, too late for Jay to see it.

The third publication yet to appear was an epic chapter that Jay composed on neurolytic blocks and neurotomy in the treatment of pain. This chapter will soon appear in the fourth edition of Bonica's textbook of pain. Although he never became one, Jay finally had achieved one of the hallmarks of a professor – writing the book that students worldwide would study.

Aside from his professional and academic activities, Jay offered something else unique to his colleagues and to those who became his friends. He was inscrutable and offered insights into what others could not see. This virtue stemmed not only from his intellect but from what he experienced in his childhood and young adulthood in a strife-torn South Africa, and Africa at large.

Those who would listen would be enthralled by his stories and explanations of what really happened, and why, in various world events; and why the expected or preferred propaganda was false.

Perhaps this was best evident in his relationship with Peter Lau. To Peter, Jay taught to look for the hidden agenda: to perceive the ulterior motives for what was going on. Jay shared his insights on all manner of national and international politics. No one knew as much about what had really happened, and what was still happening throughout Africa, India, or anywhere in the world that ended with “-stan”. Jay had political x-ray vision that saw immediately through the emperor's new clothes, and he could help anyone who wanted to learn, to see through the veneer of propaganda and spin. He could see the truth because he grew up in the reality of social oppression and moral corruption, whereas those in the first world were brought up as victims of successful political lies.

I close with a selection of remarks that others have written in recent days.

I am deeply saddened to hear this terrible news. I had the chance to meet with Jay last year at ISIS meeting, and I had been in touch with him through emails since then. He was a great person to work with and had been a great mentor for me. I was looking forward to see him again this July. It is hard to believe that he will not be among us. Please accept my deepest condolences during this difficult time. My thoughts and prayers will be with Jay and his family and friends. If there is anything I can do to help, please let me know. – *Aysel Attli, the Turkish lady anaesthetist in Washington*

I have had two, memorable, in-depth conversations with Jay where he was mentoring me. Those conversations were powerful and full of his logic and gift for the language. He was quietly persuasive, signs of both passion for his cause (pain medicine) and respect for others. This is a great loss. The AAMM meeting is in July. We will find a suitable way to

honour him. – *Geoff Harding, Queensland*

It saddens me to inform you that Jayantilal Govind, ISIS Board Member and Chair of the Standards Committee, passed away this last Saturday morning. Jay was only 64 years old. Jay was a prolific writer, editor, clinician, researcher, and an incredibly kind man. This is a massive loss to his family, our society and the spine pain medicine community at large. We will all adapt to this loss in our own personal way. The standards committee will obviously need to be restructured. We need to honor Jay's name by moving forward with what he believed in, delivering the truth about our interventions. – *Paul Dreyfuss, President of ISIS*

I feel deeply sad. I had great respect for his gentle assertiveness and his wisdom. He was unfailingly warm and welcoming and his friendship was strong and true. I will miss him very much. – *James Watt, New Zealand*

Jay was a unique individual. His humility, honesty, intelligence, humor, and quiet demeanor are traits I truly admired. – *Milton Landers, past president of ISIS*

My final words are those of his pupil, *Peter Lau*:

Jay has taught me always to look for the motive not only behind other people's action but behind mine as well. I will always remember this as Jay Govind's rule for honesty.

Nikolai Bogduk
26 June 2009

Positions available

Positions are now available in all states. For information, check the relevant websites.

Registrar Applications for 2011 Victoria

Applications for Rehabilitation Registrar Positions in 2011 in Victoria are now open. Most applications to employing institutions close on Monday 16 August 2010 but please check with the institutions you are applying to. Applicants must apply directly to employing institutions and then submit a Registrar Priority List.

Details about available positions, position descriptions and relevant contact persons are published on the Victorian Branch Page on AFRM website. Details about the selection process are also outlined.

For any further information about Victorian Registrar Applications please contact Rachael Nunan on 03 8804 2735 or rachael.nunan@easternhealth.org.au.

Rehabilitation Registrars for 2011 Qld

Applications for registrar positions in Rehabilitation Medicine in Queensland opened on Tuesday 22 June, 2010 and close on 19 July, 2010.

The Queensland Branch of the AFRM is a rapidly growing and dynamic Branch. There will be approximately 23 registrar positions in Queensland in 2011.

Registrar in Rehabilitation Medicine positions are advertised by individual Queensland Hospitals as part of the standard Queensland Health recruitment process. Please see <http://www.health.qld.gov.au/medical/rmoinfo.asp>.

For further information regarding the Application and Selection Process please see the Queensland Branch page of the Faculty Website <http://afrm.racp.edu.au/> or contact Tim Geraghty on 07 3176 2928 or timothy_geraghty@health.qld.gov.au

Dr Tim Geraghty

Can we be more specific about back and neck pain?*

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The initial clinical assessment of spinal pain consists of diagnostic triage into (i) serious spinal pathology, (ii) other pathological entities, and (iii) non-specific symptoms. The non-specific group comprises the major burden of spinal illness. There are two broad approaches to the diagnostic challenge of non-specific spinal pain. One approach is to split the group into sub-groups explained by separate pathophysiological abnormalities. The focus of this work is to describe these proposed abnormalities in greater detail and to discover which clinical features distinguish them. The other approach is to lump all of those with non-specific features into one group, and consider the common psychosocial factors that are relevant to the whole group. A variety of pathological and non-pathological models have been proposed. Pathological models include ligament laxity, facet syndrome, discogenic pain, spondylosis, and instability. Non-pathological models include a pain-spasm-pain cycle, muscle inhibition and deconditioning and somatic dysfunction. All of these models are problematic and do not fully account for all of non-specific spinal pain. Another approach is to consider these two different approaches together and to consider this non-specific category as orthogonal dimensions of pathology and dysfunction.

Physicians think they do a lot for a patient when they give his disease a name. – Immanuel Kant, German philosopher (1724–1784)

Introduction

Problems of definition

Until the report of the Quebec Taskforce on Spinal Disorders in 1987, the textbook classification of pain in the lumbar region was usually in terms of pathological labels, such as “slipped disc”, or “spinal osteoarthritis.”¹ Since then, national consensus, guideline or evidence-review bodies^{2–5} have acknowledged that there is a very low frequency of serious or meaningful underlying pathological disease. They have combined most pain from the lumbar region together, without a pathological label as low back pain.

This symptom description is often given deliberately vague adjectives such as “simple”, “mechanical”, or “non-specific”. The beneficial consequence of this classification has been that it has allowed the epidemiology of this non-specific symptom to be described more clearly, and has made the strategy of diagnostic triage explicit. However, it has not clarified the underlying aetiology, and it has separated low back pain from symptoms in the rest of the spine and other regions of the musculoskeletal system. There has been less written about other regions of the spine, but there is

growing consensus that a similar strategy can be used to classify neck pain.⁶

Diagnostic triage

A basic distinction in the classification of low back pain has been made by a number of writers between specific and non-specific back pain.^{7,8} Specific back pain can be attributed to a particular cause; non-specific pain (also called “mechanical” or “idiopathic”) cannot. Since the Quebec Task Force report in 1987, the classification of low back pain has embraced the concept of diagnostic triage,² which has been incorporated into low back pain management guidelines throughout the world.^{3–5,9,10} Diagnostic triage has also been extended to the diagnosis of other musculoskeletal complaints,¹¹ including neck pain.⁶

Mechanical or non-specific pain

It has been estimated that definite pathology cannot be diagnosed in 85% of patients with low back pain.^{12,13} However, this non-specific group should not be just a diagnosis of exclusion. The essential feature of this condition is “mechanical” back pain, that is, a pain that varies according to physical activity and posture. Patients tend to have an onset of symptoms at 20–55 years of age. The pain is usually in the lumbosacral region and is often difficult to delineate accurately. Pain radiation may be non-segmental to one or both buttocks or thighs.^{4,9} The concept of “non-specific” or “mechanical” pain in the neck and upper back has not been clearly defined, but there is no reason to believe that its essential features would differ from that in the lower back. The aetiology of all non-specific spinal pain remains controversial. Many authors argue that it comprises a collection of different pathological entities that have not yet been adequately described.¹⁴ What are the possible underlying pathological mechanisms?

Pain syndromes arising from specific spinal structures

For any structure to cause pain, three criteria should be satisfied:¹⁵ (i) it should be innervated; (ii) it should be capable of causing pain similar to that seen clinically; and (iii) it should be susceptible to diseases or injuries known to be painful. Many structures in and around the spine can give rise to pain, but which structures are implicated in non-specific pain? Several different spinal pain syndromes arising from separate spinal structures have been proposed. Some of these rely purely on clinical criteria for diagnosis, whilst others

* First published in *International Musculoskeletal Medicine* 2009; 31(1): 5-14). Reproduced here with kind permission. See www.maney.co.uk/journals/imm and www.ingentaconnect.com/content/maney/imm.

can only be reliably diagnosed using invasive tests.

Trigger points and myofascial pain

Asyndrome of myofascial pain has been described, where regional pain is associated with the presence of one or more "trigger points" in affected muscle.^{16,17} The identification of trigger points is a subjective clinical judgement based on the following criteria:¹⁸ (i) a trigger point must be present in a muscle, consisting of a palpable, tender, firm, fusiform nodule orientated in the direction of the affected muscle's fibres; (ii) the muscle must be specified; (iii) palpation of the trigger point reproduces the patient's pain or referred pain; and (iv) elimination of the trigger point relieves the patient's pain. Elimination may be achieved by stretching the affected muscle, dry needling the trigger point, or infiltrating it with local anaesthetic. However, the aetiology of trigger points is unknown. More importantly, their relevance to non-specific spinal pain is uncertain. Their sensitivity, specificity and predictive value has not been determined.

Ligament laxity

It has been suggested that if ligaments are rendered stretchable under load, they can become painful. This pain is aggravated by prolonged static posture, in standing or sitting, and improved by activity, and has been dubbed "theatre and cocktail party back". A treatment has been proposed to provoke a sterile inflammatory reaction, by injection of a "sclerosant" (or proliferant) solution, usually consisting of dextrose, glycerine and phenol, leading to fibroblast proliferation and new collagen production.¹⁹ This sclerosant injection treatment, or "prolotherapy" has been subjected to a randomized, controlled trial.²⁰ However, the treatment package also included spinal manipulation and exercises, so it is not possible to determine the effectiveness of the injections alone.

Facet syndrome

Sprains of the zygoapophysial or facet joints have been postulated as a cause of spinal pain. Facet joint capsule tears, capsular avulsion, subchondral fractures, and intra-articular haemorrhages have been found in biomechanical and post-mortem studies.²¹ Minor trauma may cause facet joint capsule sprains and effusions; however, such changes have not been demonstrated in studies using diagnostic ultrasonography of the spine.²² Intra-articular injection of facet joints can reproduce or relieve spinal and referred pain,^{23,24} and it has been postulated that a sub-set of patients with non-specific spinal pain have facet joint pain. Ghormley²⁵ first suggested the existence of a "facet syndrome" as a cause of low back pain. Eisenstein and Parry²⁶ suggested that the clinical features of this syndrome consisted of pain at rest, relieved by motion, with painful restriction of trunk extension. However, response to facet joint injection is not associated with any set of clinical features.^{24,27} Radiologically controlled diagnostic blocks remain the only way of diagnosing facet joint pain, but are associated with a false-positive rate of 38%, and a placebo rate of 32%.²⁸ The International Society for the Study of Pain recommends that the following criteria should

be satisfied. The blocks must be radiologically controlled; arthrography should confirm that injection has been made selectively into the target joint without any injected material spilling into adjacent structures. The patient's pain should be totally relieved by local anaesthetic injection, and should be validated by an appropriate control test, such as no pain relief after injecting a non-active agent, or no pain relief after injecting local anaesthetic into a control site.¹⁸ It has been suggested that osteoarthritis is the pathological cause of facet joint pain. However, radiological changes in the facet joints cannot be firmly linked to spinal pain.²⁹ The prevalence of facet joint pain in the population with non-specific pain is not known, as most studies have been carried out on highly selected populations of patients in secondary or tertiary care. Finally, a systematic review of randomized, controlled trials found no evidence that facet joint injections improve pain or function in patients with chronic low back pain.³⁰

Discogenic pain

Lindblom³¹ first noted that injection of contrast material into lumbar intervertebral discs, (discography) could produce low back and referred pain in patients with no evidence of disc prolapse or nerve root compression.

Although the internal architecture of the disc can be seen with this technique, the key feature of discography is the patient's response to disc provocation, and not the disc's appearance. It is claimed that discography is rarely painful in asymptomatic patients, even in those with abnormal discs,³² but is frequently painful in those with low back pain. For discography to be positive, it must reproduce the patients' pain. It is claimed that discography determines when a degenerate disc has become symptomatic. Some lumbar discs that are painful during discography show evidence of external annular disruption; others are intact externally, but show evidence of "internal disc disruption" on computerized tomographic (CT) discography. The characteristic features of internal disc disruption are radial fissures through the annulus fibrosis, which reach its outer innervated one-third.³³⁻³⁵ Most research has been carried out on lumbar discs, but discography has also been used on cervical discs.³⁶

The International Society for the Study of Pain states that provocation discography alone is insufficient for the diagnosis of discogenic pain, because of the possibility of false-positive responses. Either a local anaesthetic should be used to relieve the patient of their pain, or provocation of two adjacent intervertebral discs should not reproduce the patients' pain.¹⁸ Despite these strict criteria, false-positive discography can be produced in subjects with chronic pain or abnormal psychometric testing according to depression and somatization scales.³⁷ Also, some patients who had no history of low back pain but had undergone posterior iliac bone graft harvesting for non-lumbar procedures, experienced their usual buttock pain during lumbar discography.³⁸

So, the ability of patients to separate spinal from non-spinal pain during discography is questionable. The prevalence of discogenic pain is uncertain as most studies have been carried out on highly selected populations of patients in secondary or tertiary care. No conventional clinical

finding in the history or examination has been shown to be associated with a positive discogram.³⁵ In conclusion, the relevance of discogenic pain in patients with non-specific pain is uncertain.

Degenerative change

One pathological condition affecting many of these spinal structures that might be responsible for non-specific pain is spinal degeneration or spondylosis, which comprises thinning of the intervertebral disc and osteoarthritis of the facet joints. The sequence of events leading to spondylosis can be summarized as follows. Structural derangement of the disc leads to prolapse or degeneration, resulting in thinning of the disc with forward tilting about the axes of the facet joints. This, in turn, leads to anterolateral bulging of the annulus resulting in osteophyte formation. Stresses on facet joints result in remodelling and the development of osteoarthritis. All of these produce changes in the mechanics of the spine.

Possible sources of spinal pain include: (i) within the disc following in-growth of nerves accompanying vascularization of clefts and prolapses; (ii) pressure on pain sensitive structures, such as the outer annulus, ligaments, dura mater, or nerve roots by osteophytes of vertebral bodies; (iii) posterolateral disc prolapses or osteophytes stabilising such prolapses impinging on nerve roots in intervertebral foramina; (iv) osteoarthritis of facet joints; (v) pseudoarthroses formed on neural arches due to facet joint osteophyte formation following disc degeneration; (vi) fracture of facet joint end plates; (vii) narrowing of spinal canal by posterior disc prolapses and facet joint osteophytes; and (viii) trabecular microfractures in vertebral bodies and around Schmorl's nodes.³⁹

In clinical practice, spondylosis is commonly diagnosed with plain radiographs of the spine. If non-specific spinal pain is caused by spondylosis, then it should be associated with radiological signs of degenerative change, such as disc space narrowing, osteophytes, or sclerosis.

A systematic review of observational studies comparing radiographic changes with low back pain found that there was only a small association, with odds ratios ranging from 1.2 to 3.3.²⁹ Most studies have been cross-sectional in design and have not examined the temporal relationship between pain and degenerative change, which is an important criterion for causality.

More recent studies have found a weak correlation with radiological degenerative change in the lumbar spine and low back pain severity, but not disability scores.⁴⁰ In the cervical spine, increasing levels of spinal degeneration have been correlated with chronicity of symptoms and higher disability ratings.⁴¹

Perhaps the most striking finding in all of these studies is the lack of specificity, with large numbers of abnormal radiographs showing signs of spinal degeneration in asymptomatic subjects.

Similarly, in magnetic resonance imaging (MRI), degenerative changes are associated with spinal pain,⁴² but are common in asymptomatic subjects as well.^{43–45} The

relevance of radiographic degenerative change in patients with non-specific spinal pain is questionable. Some authors argue that as these changes are so common they should be considered a "normal age-related process like grey hair,"⁴⁶ and should not be considered as disease diagnoses.⁴⁷

Instability

Spinal fusion operations are used for treating "spinal instability" secondary to various pathological processes such as trauma, neoplasia and infection, to correct or control deformity, pain, and loss of function. Instability can also be caused iatrogenically following spinal surgery. Extrapolating from biomechanical studies of the functional spinal unit,⁴⁸ and from these observations of clinical instability following surgery, it has been proposed that degenerative change leads to spinal instability, so that physiological loads induce abnormally large deformations in the spine. This has been defined clinically as "the loss of the ability of the spine under physiologic loads to maintain relationships between vertebrae in such a way that there is neither damage nor subsequent irritation to the spinal cord or nerve roots, and in addition there is no development of incapacitating deformity or pain from structural changes."⁴⁹ Where instability is restricted to a single segment of the spine, it may be amenable to spinal fusion.⁵⁰ There is no objective clinical test, but suggested symptoms include: (i) pain on prolonged standing, slow walking or straightening from a stooped position; (ii) the back "giving way" causing the patient to fall to the ground without warning; or (iii) having to twist a contorted spine back into position.

Suggested examination findings include the extension "catch," a sudden jerky movement in the mid-range during active extension, and a ratchet-like motion of the spine when straightening up from a flexed forward position.⁵¹ The predictive value of these symptoms and examination findings is unknown. A number of radiological findings have been suggested including the presence of traction spurs⁵² and excessive intervertebral displacement on dynamic flexion–extension views.⁵³ However, others have found that such radiological change could not predict the presence of the symptoms of instability, even in patients with spondylolisthesis.⁵⁴

There is a lack of evidence for the effectiveness of spinal fusion for degenerative spondylolysis compared with natural history, placebo, or conservative management. Fourteen trials have been published, but they have compared only two or more surgical techniques, and most reported short-term, technical, or surgical outcomes rather than patients' self-reported health status.^{55,56}

Because of this, and the lack of evidence for predictive value of any symptom, sign, or radiological finding the usefulness of the clinical syndrome of instability is uncertain. Its relevance in the population with non-specific spinal pain is unknown.

Non-pathological models for non-specific spinal pain

In addition to the difficulty linking pathological changes to non-specific spinal pain, pathological abnormalities such as herniated discs, bulging discs, or annular tears are commonly found in asymptomatic subjects.^{44,45,57–59} Although the non-specific category is an amorphous group, it has arisen by default as a consequence of the inability to explain symptoms adequately, by means of pathological change.⁶⁰ In common with other chronic pain states, it has been shown that mood disorders, perceptual styles, cognitive and social factors are important in determining who develops back pain and who becomes disabled by it.⁶⁰ Once pain becomes chronic there is a high probability that other regions will be painful, and will be accompanied by associated problems such as psychological distress.^{61,62} There is an increasing emphasis on general pain management for these chronic pain syndromes, regardless of where the regional focus happens to be.⁶³

If pathological models are insufficient to explain non-specific spinal pain, what physiological mechanisms are associated with these painful states? Pain is a complex phenomenon with important psychological and social components. The integration of these elements with the neurobiology of pain is increasingly being realised by neurophysiologists. An important concept in this understanding is the plasticity of the nervous system.

Neural plasticity

The nervous system is not “hard-wired” for pain perception, but exists in a variety of distinct states or modes. This property is referred to as “neuronal plasticity.” The simplest description involves three different states. The first is the “normal” or “physiological” state, where non-injurious stimuli applied to healthy tissue is perceived as non-painful sensation. The second is the “sensitized” state, in which similar non-noxious stimuli are applied to inflamed or damaged tissue and are perceived to be painful. The third is the “suppressed” state, where noxious stimuli are perceived as non-painful. These different states depend upon changes in the function, chemistry, and structure of the primary sensory neurones, the dorsal column of the spinal cord, and higher regions of the brain.⁶⁴

Influence of emotion

The central role of emotional distress in the experience of pain was implicit in the International Association for the Study of Pain’s definition as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”¹⁸ Of course, emotional distress is not only a component of pain, but may be present in anticipation of pain, as a consequence of pain, contributing to the cause of pain, or a concurrent problem present for a different reason. The neurophysiological basis of this close association is that nociceptive signals affect multiple pathways projecting from the thalamus to the cortex, in particular the limbic system,

which has an important role in many emotional states.⁶⁵ But a source of nociception is still needed, what is its source in non-specific spinal pain? Various disturbances of physiology have been proposed.

Pain–spasm–pain cycle

It has been postulated that a pain–spasm–pain cycle can be the cause of spinal pain, rather than any “structural” pathology. Spasm can be defined as involuntary electrogenic muscle contraction. Electrogenic refers to the presence of alpha motor neuron and neuromuscular end-plate activity mediating the contraction, observed by electromyographic (EMG) activity.

Spasm can be contrasted with electrogenic stiffness, which is muscle tension arising from electrogenic muscle contraction in normal subjects who are incompletely relaxed, and with contracture arising endogenously within muscle fibres independent of EMG activity.⁶⁶ It is known that muscle spasm is present in patients with spinal pain, that pain can cause muscle spasm, and that muscle spasm can be painful.⁶⁷ However, the existence of a positive feedback loop has been dismissed by others,⁶⁶ because there is often no EMG activity in the hypertonic muscles of spinal pain patients, and where there is, its timing and intensity does not correlate with the pain. Indeed, muscle pain tends to inhibit, rather than facilitate, voluntary and reflex activity of the same muscle.

Muscle inhibition and wasting

There is a large body of evidence that lumbar paraspinal muscles of patients with low back pain operate sub-optimally, with reduced activity in free dynamic movements, reduced muscle strength, and increased muscular fatigability.²² Much of this change can be attributed to deconditioning due to reduced activity, but wasting of the multifidus, measured using ultrasound imaging, occurs selectively and rapidly after a first episode of low back pain. The changes are segmental and unilateral, corresponding to the level and side of symptoms, possibly due to segmental inhibition rather than a general effect of disuse.⁶⁸ Wasting persists even after remission of the painful symptoms, and it has been postulated that this may predispose to recurrence,⁶⁸ and that specific exercises for the multifidus may help to reduce recurrence.⁶⁹ Trunk muscles, in particular the transversus abdominus and the diaphragm, may play a key role in stabilizing the spine.⁷⁰ It is not just wasting and deconditioning that are important, but features of a clearly disordered neuromuscular control of movement have been discovered. Patients with back pain had delayed contraction of these muscles in anticipation of arm movements compared with normal controls.⁷¹ Similar changes in neck muscles have also been found in subjects with neck pain. These have included altered co-ordination between the deep and superficial muscles, greater neck muscle fatigue under sustained low loads, and deficits in kinaesthetic sense.⁷²

Dysfunction of the musculoskeletal system

There is clear evidence that loss of function may occur in the absence of pathological disease. The concept of dysfunction involves abnormal functioning of the neurological and muscular components of the spine, sufficient to cause symptoms and disability independent of any structural pathology. The presumed sources of nociception are mechanical in stressed tissues, and chemical in overactive muscles. In non-specific spinal pain, it has been proposed that this “somatic dysfunction” is diagnosed by finding a combination of asymmetry of anatomical landmarks, asymmetry of joint movement (usually restriction), tissue texture changes in the soft tissues, and tenderness.⁷³

The “facilitated segment”

Several neurological mechanisms have been proposed to explain these clinical findings in somatic dysfunction.²² In the 1940s and 1950s, American osteopaths performed simple experiments on the paravertebral muscles and electrical skin resistance of subjects’ backs, and reported evidence of spontaneous EMG activity and increased sympathetic activity at segmental levels associated with signs of somatic dysfunction.^{74–78} Korr and colleagues^{77,78} proposed the concept of the “facilitated segment” to explain these findings as follows. Minor trauma was proposed to cause increased afferent input from muscle spindles to the dorsal horn of the spinal cord. This increased the sensitivity of interconnecting neurones, which in turn increased nociception, and resulted in increased tenderness found at these levels. It also increased motoneurone and sympathetic output, which produced range-of-motion restriction due to shortened hyperactive muscles, and tissue texture changes due to sustained muscle contraction, and sympathetic induced circulatory changes. So, tissue injury from trauma, inflammation or postural stress markedly altered the sensory input from articular and peri-articular structures, which initiated aberrant motor and sympathetic responses, causing the segmental facilitation seen in somatic dysfunction.^{77,78} Denslow and Korr’s^{74–78} original experiments have been criticized by modern standards, because of their lack of control subjects, insufficient presentation of data, and absence of statistical analysis.⁷⁹ In the “facilitated segment” model, the tissue texture changes and restricted movement are caused by sustained paravertebral muscle contraction, which would produce increased EMG activity at these levels; however, others have failed to confirm this.⁷⁹ In fact, there is evidence of decreased EMG activity in the deep paravertebral muscles in back pain patients.^{80–83} Although palpable muscle hypertonicity has frequently been blamed on increased muscle activity, or “muscle spasm,” resting muscle has no electrical motor activity, and other changes in muscles that do not require electrical activity have been postulated to explain these changes.⁶⁶

Altered proprioception

There is increasing evidence that pain interferes with normal proprioception. Decreased proprioception has been demonstrated in experimental muscle pain,^{84,85} lumbar

muscle fatigue,⁸⁶ and in patients with back pain,^{87–89} in terms of decreased awareness of lumbar position and direction of motion, or decreased cutaneous touch perception. Increased lumbar paraspinal activity has been demonstrated in low back pain subjects that is not due to reflex contraction, but to a combination of voluntary “guarding” behaviour and a complex change in motor control strategy.^{90–93} Spinal pain and a regional deficit in proprioception may result in the inability to execute co-ordinated contractions of deep segmental muscles, which may further adversely affect control and proprioception.²² As discussed above, low back pain results in reflex inhibition and atrophy of the deep paraspinal muscles,^{68,80–83,93} the muscles that contribute most to intervertebral stability.⁶⁹ Lack of control from these deep stabilizing muscles, combined with impaired proprioception and non-specific guarding activity, may leave the affected segment vulnerable to the effects of further and repeated trauma.²²

Reliability and diagnostic accuracy of examination findings

Somatic dysfunction can be diagnosed only with subjective clinical findings, and requires a high degree of palpatory skill, but how reliable are such palpatory findings? Most studies of reliability and validity have been performed by chiropractors and physiotherapists rather than osteopaths.

Systematic reviews of tests for the lumbar spine and pelvis concluded that the reliability and validity of these tests had not been clearly established.^{94–97} Only tests that elicited tenderness had consistently acceptable results. Studies testing motion palpation for the lumbar spine and the sacroiliac joints had mixed findings, whereas visual inspection of positional asymmetry showed consistently unacceptable agreement. There are several possible explanations why interobserver reliability has been hard to demonstrate. Most studies used asymptomatic subjects, and most examiners found it easier to agree on normal rather than abnormal findings. Higher values for the kappa statistic are obtained for agreement on positive rather than negative findings, so the use of asymptomatic subjects may make it difficult to demonstrate agreement beyond chance.⁹⁴

When examining active movements, the patient may not perform the movements consistently from one examiner to the next. The soft tissues of the subject may be such that the manual contacts are inconsistently placed. Idiosyncrasies among examiners may produce dissimilar palpatory findings. The testing procedure when performed repetitively may alter joint mobility. Validity studies are rare and most have lacked comparison with a gold standard. Some studies used a manual model to test motion palpation. Others have tested manual examination of the cervical spine against segmental diagnosis made by anaesthetic blocks and found good agreement for eliciting tenderness,⁹⁸ or a combination of positional asymmetry, tissue texture changes and tenderness.⁹⁹ A systematic review of sacro-iliac joint tests concluded that neither mobility nor pain provocation tests were of diagnostic value.⁹⁵ In particular, the specificity of these findings is uncertain, as it is common to find areas of restricted

intervertebral movement in asymptomatic subjects.¹⁰⁰ These might represent areas of sub-clinical dysfunction, or may represent false positive findings suggesting a low level of specificity. Further studies of the reliability and diagnostic accuracy of the somatic dysfunction diagnosis need to be performed, involving all regions of the spine. It is important to recognise that lack of evidence for reliability and validity of physical signs is common in other areas of the orthopaedic examination.^{101–103}

Lack of evidence for the somatic dysfunction model

In conclusion, there is a “somatic dysfunction” model that attempts to explain the features of non-specific pain by mechanisms that are independent of structural spinal pathology, which can occur in all regions of the spine.

Somatic dysfunction can be diagnosed only by clinical findings, as there are no objective diagnostic tests. These clinical findings have uncertain reliability and unknown validity. Although the concept of somatic dysfunction is intuitively appealing, more research is needed regarding which clinical features are reliable, have predictive value, and can be linked to the physiological and psychological disturbance seen in non-specific spinal pain.

Relationship between pathology and dysfunction

All of these pathological and non-pathological models are problematic, and might be criticized as providing convenient labels to justify favoured treatments by clinicians, for example, facet syndrome for facet joint injection, instability for spinal fusion, and somatic dysfunction for spinal manipulation. None of these models can fully explain the aetiology of non-specific spinal pain; neither does splitting into separate pathological sub-categories nor lumping together into a biopsychosocial whole.¹⁰⁴ We have previously proposed a classification of illness that includes orthogonal dimensions of pathology and dysfunction.¹⁰⁵ For this purpose, pathology is narrowly defined as pathological processes that cause gross or microscopic structural change that may, but not necessarily, result in disturbance of function. Dysfunction, by contrast, is abnormal functioning of the body, caused by, or manifested as, disturbed physiological or psychological processes independent of known structural pathology (Fig. 1). The challenge for researchers is to describe the clinical epidemiology of these different pathological and dysfunction syndromes. The challenge for clinical practice is to determine to what extent each is contributing to an individual patient's illness.

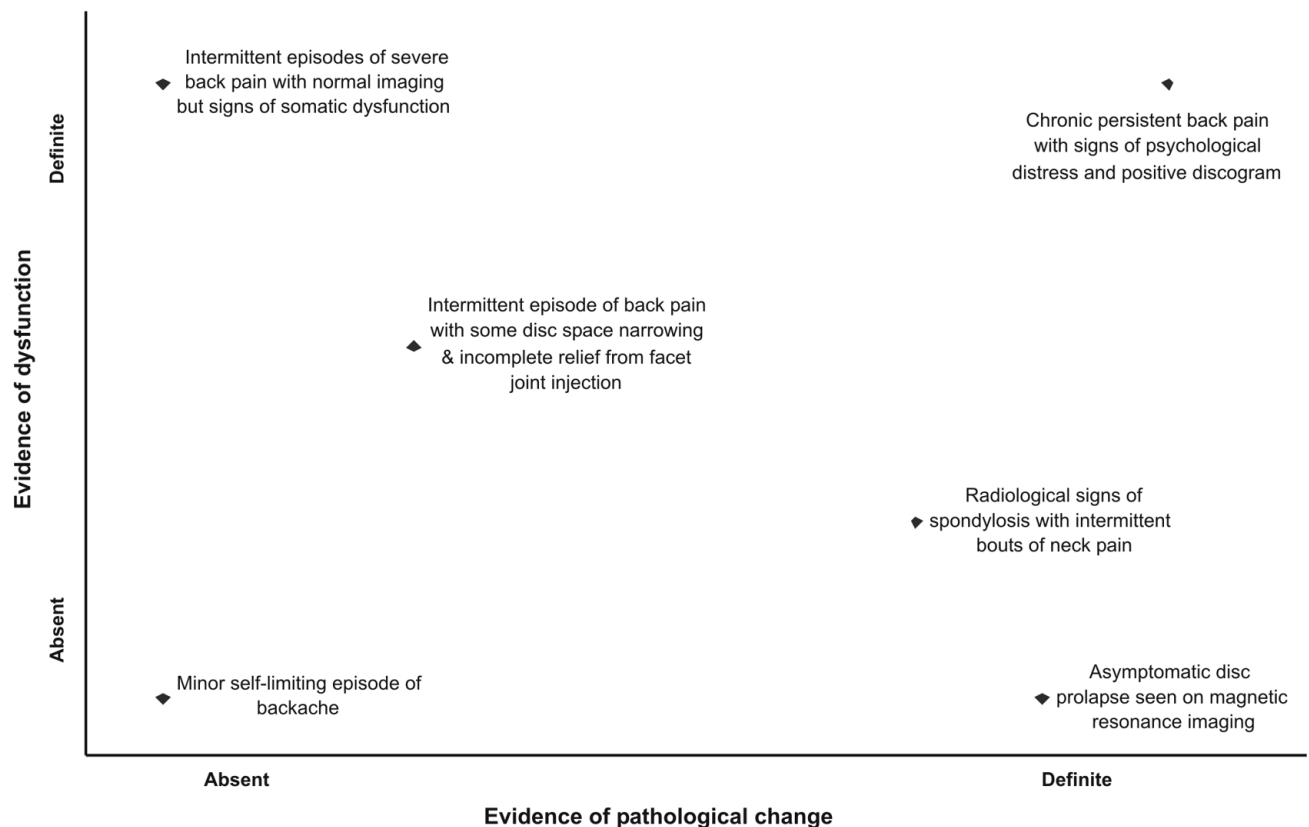


Figure 1. Hypothetical scatter plot of dysfunction versus pathology in non-specific back and neck pain. **Dysfunction:** abnormal functioning, either physiological or psychological, which is reversible and not dependent on pathological processes. **Pathology:** abnormal functioning caused by structural pathological change, either gross or microscopic.

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Use of a “polypill” for acute tendinopathy – case series of 20 patients

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Abstract

Objective. Acute tendinopathy is an injury that is commonly seen in general practice and sports medicine clinics. Management of the condition can be difficult, and has traditionally been limited to rest, NSAIDs, and adjuncts such as ice and physiotherapy. The aim of this study was to determine the efficacy and tolerability of a “polypill” comprising ibuprofen and doxycycline, with adjunctive use of omega-3 fatty acids (“fish oil”) and green tea.

Method. Patients with symptoms determined to be due to an acute tendinopathy were identified. After informed verbal consent, they were offered treatment with the polypill.

Results. Initial review occurred 1-3 weeks after commencement of treatment – 19/20 patients reported an improvement in symptoms at this review. Half of the patients reported resolution of symptoms at five weeks or less. 15 patients (75%) were able to complete their prescribed polypill course. Only two patients (10%) ceased polypill treatment as a result of adverse effects. One patient failed to report any change in symptoms. The median and mode duration of treatment with the polypill was four weeks.

Conclusion. The resolution (or improvement) of symptoms in most patients in four weeks or less suggests that the observed effect of the polypill therapy for most patients may be due to the combined anti-inflammatory and analgesic effects of ibuprofen. There may be only a subgroup of patients suffering from an acute tendinopathy for whom the polypill is appropriate treatment.

Suggestions are made for areas of further polypill research.

Introduction

Tendinopathy is a broad term encompassing painful conditions occurring in and around tendons, in response to injury and overuse.¹ Acute tendinopathy is a condition commonly seen in general and sports medicine practice, and occurs in a broad cross-section of the community. It is not uncommon to see the condition even in the elderly after an episode of over-exertion, such as gardening. The condition can be difficult to manage, and treatment is, to a large extent, empirical. The usual treatment prescribed for these conditions is rest and ice, paracetamol for pain, and a non-steroidal anti-inflammatory drug (NSAID). NSAIDs are frequently used in the treatment of acute athletic injuries despite there being little evidence to support their ability to enhance healing.²

A recent paper³ theoretically investigated a “polypill” approach to the management of athletes with acute tendinopathy, utilising a selective NSAID prescription as well as the use of inhibitors of tumour necrosis factor- α (TNF- α). The authors concluded that ibuprofen should be the NSAID of choice as it has been demonstrated to be the only NSAID (of six NSAIDs studied) not to have a detrimental effect upon tendon repair after experimental transection.⁴

Previous work has determined that tendon cells are known to have selective binding sites for TNF- α on their surfaces, and that TNF- α has been implicated in enthesopathy associated with spondyloarthropathy.⁵ Fallon et al.³ suggested that TNF- α may affect both structural change and pain in activity-induced tendinopathy. In spondyloarthropathy, TNF- α can be inhibited by TNF- α blockers such as etanercept, or

by monoclonal antibodies (adalimumab and infliximab), but these agents have serious adverse effects, and are expensive. The authors suggest that a less expensive and less potent effect can be obtained by the use of doxycycline, which is known to block the action of TNF- α ;⁶ or by using a macrolide antibiotic, which is known to inhibit the production of pro-inflammatory cytokines including IL-1, IL-6, and IL-8, as well as TNF- α .⁷ Doxycycline inhibits the breakdown of connective tissue, and inhibits mediators of inflammation in animal models,⁸ and has been shown to increase collagen turnover in human cases of tendon pathology.⁹

It is suggested that ibuprofen may work synergistically with doxycycline to dampen inflammatory responses.³ Interestingly, some NSAIDs (naproxen, diclofenac) as well as celecoxib, have been demonstrated to increase TNF- α levels.^{10,11}

Some practitioners are known to favour the use of selective cyclo-oxygenase Type II (COX II) inhibitors such as celecoxib in the acute phase of tendinopathy. Celecoxib has been shown to *inhibit* tendon cell migration and proliferation in rat Achilles tendon tissue in vivo without affecting the expression of collagen,¹² and in head-to-head studies with naproxen in cases of acute shoulder tendinitis (and bursitis) shows equal efficacy,¹³ suggesting that celecoxib may have an effect only upon the pain associated with the injury without promoting the healing of the injury. Finally, Fallon et al.³ suggested incorporating adjunctive non-pharmacological substances reported to have an inhibitory effect on TNF- α such as the omega-3 fatty acids (“fish oil”),¹⁴ and the polyphenols and catechins contained in green tea.¹⁵

This paper examines the efficacy of a polypill approach to the treatment of acute tendinopathy in 20 patients, and of the ability of patients to adhere to the suggested polypill regimen.

Method

Sequential patients with acute symptoms suggestive of tendon pain were identified. Those patients with a diagnosis (or suspected diagnosis) of acute tendinopathy were, after informed verbal consent, offered a polypill medication regimen of ibuprofen 400 mg tds, doxycycline 100 mg die, omega-3 fatty acids (fish oil) (to maximum dose allowed on product label), and ad lib green tea. Patients were also advised to use paracetamol 1g qid prn, as an analgesic. Patients were permitted to continue adjunctive treatment (for example, ice application, physiotherapy, gentle exercise) at their own discretion.

Patients were not offered polypill treatment if their symptoms were of greater than two weeks' duration, or if they had a contraindication to the use of any component of the polypill. Patients were eliminated from the study if subsequent imaging (if undertaken) determined a pathological process other than an acute tendinopathy.

Patients were first reviewed between one and three weeks after commencement of treatment, and again a week or two after initial follow-up. Attempts were made to follow patients to the completion of their course of treatment. Treatment with ibuprofen and doxycycline was ceased when symptoms had either resolved, or greatly settled (after discussion with the patient). Treatment was also ceased if the patient was intolerant of any of the medications contained in the polypill.

Follow-up of patients beyond cessation of polypill therapy was not formally arranged, and occurred on an opportunistic ad hoc basis.

Results

Twenty-three patients were identified as suffering from an acute tendinopathy and were suitable for commencement of polypill therapy. Three patients were eliminated when subsequent imaging revealed a pathological process other than an acute tendinopathy. These diagnoses are outlined in Table 1.

Age	Sex	Initial Diagnosis	Imaging modality	Final Diagnosis
49	M	supraspinatus tendinopathy	ultrasound	subacromial bursitis
64	M	subscapularis tendinopathy	ultrasound	subacromial bursitis
17	M	(L) adductor magnus tendinopathy	ultrasound	iliopsoas bursitis

Table 1. Characteristics, and initial and final diagnoses for patients eliminated from further participation in “polypill” study.

Twenty patients were enrolled into the study. The study group comprised 13 men and seven women. They ranged in age from 15 to 83 years, with a mean of 37 years. Nine of the patients had sustained their injury as a result of playing sport (Australian Rules football eight, basketball one), and one patient had sustained her injury while ballroom dancing. Other patients had sustained their injury outside of a sporting environment.

Diagnosis was determined clinically in 13 cases. Five cases were diagnosed by ultrasonography (U/S) and one case by magnetic resonance imaging (MRI). One clinically diagnosed injury was confirmed by U/S. Characteristics of the patients in the study as well as an outline of their polypill treatment are in Table 2.

All 20 patients underwent review at 1-3 weeks after commencement of polypill therapy. At time of first review all but one of the patients (Case No.12) reported an improvement, or significant improvement, in their symptoms. polypill therapy was ceased at initial review for four patients – two patients were ceased as their symptoms had resolved

No	Age	Sex	Sport	Tendinopathy	Diagnosis Modality	Polypill Duration	Comments
1	79	F	-	@ supraspinatus	U/S	4/52	Improved @ 9/7 Resolved @ 4/52
2	48	M	-	(L) peroneus longus	Clin	4/52	Improved @ 3/52 Full exercise @ 8/52
3	83	M	-	@ supraspinatus and short head biceps	Clin	4/52	Significantly improved @ 1/52 Pain-free @ 5/52
4	61	F	Ballroom dancing	(L) supraspinatus	U/S	2/52?	Improved @ 2/52 FTA further appts.
5	77	F	-	@ biceps brachii	Clin	3/52	Significantly improved @ 10/7. Pain-free @ 3/52
6	47	F	-	@ golfer's and tennis elbows	Clin	2/52	Ceased doxycycline @ 2/52 and ibuprofen @ 3/52 - unable to tolerate GIT AEs Golfer's resolved @ 2/52, with sig. improvement in tennis elbow
7	25	F	Basketball	@ supraspinatus	U/S	4/52	Significantly improved @ 4/52. Accidental reinjury @ work

Table 2. Demographics, site of tendinopathy, duration of treatment with “polypill”, and treatment commentaries for 20 studied patients.

(Case Nos.11 and 20); one patient (Case No.6) ceased treatment as he was unable to tolerate the gastrointestinal adverse effects (GITAEs) of polypill therapy; and one patient (Case No.15) elected to cease doxycycline but continue with the other components of the polypill.

At subsequent review, 12 of the 16 continuing treatment patients reported resolution of their symptoms. One patient (Case No.15) had resolution of his symptoms after using a modified polypill prescription. Of the remaining four patients, one patient (Case No.12) again reported no change in symptoms. One further patient (Case No.16) had to prematurely cease polypill therapy due to GIT AEs. He reported that his symptoms had significantly improved despite the limited time of polypill therapy. Two patients failed to attend a follow-up appointment. A summary of the final outcome for the patients enrolled in the study appears in Table 3.

Outcome	Number of Px
Resolution of symptoms	14
Partial treatment	1#
No effect	1
Lost to follow-up	2*
Ceased due to GIT AEs	2*
Total	20

Table 3. Final outcomes of 20 patients enrolled into “polypill” study.

Symptoms resolved with 2/52 polypill and further 2/52 of NSAID and non-pharmacological agents.

*Both patients reported improvement in symptoms at initial review.

For patients who completed the prescribed course of polypill treatment, total duration of treatment ranged from two to eight weeks, with both median and mode duration of therapy being four weeks.

The ability to adhere to the polypill prescription appeared to be good. Fifteen out of 20 patients (75%) were known to complete the prescribed course of medications and non-pharmacological adjuncts. Two patients (10%) had to withdraw from, or modify, therapy as a result of GIT AEs. There were no reports of other AEs, and in particular, there was no report of doxycycline-induced photosensitivity.

Follow-up of patients beyond completion of treatment occurred only on an ad hoc basis, and at time of writing there was only one episode of (accidental) injury recurrence (time from completion of study. The range was seven weeks to 10 months).

Discussion

This is the first published study to investigate the use of a polypill in the treatment of acute tendinopathy. The study demonstrated that the treatment of acute tendinopathy via the use of a polypill results in a rapid improvement in the

symptoms experienced by almost all patients.

Knobloch's speculation¹⁶ that the use of the polypill for only four weeks may have no effect at all upon tendon metabolism, coupled with the observation that both the median and mode duration of treatment was only four weeks, suggests that the major observed effect of the polypill therapy for most patients may be due to the combined anti-inflammatory and analgesic effects of ibuprofen. That only four patients required six or more weeks of polypill treatment to achieve symptom resolution suggests that there may be only a subgroup of patients for whom longer-term suppression of TNF- α and/or other factors such as cytokines and matrix metalloproteinases is required to facilitate a more rapid recovery.

The polypill was well tolerated. Eighteen of 20 patients appeared to suffer no AE to the components of the polypill. The observed rate of GIT AEs (10%) compared favourably to the 10% rate of GIT AE expected from ibuprofen alone.¹⁷ Patient age, level of outdoor activity, and seasonal factors may have determined the non-observance of doxycycline-mediated photosensitivity. Study numbers may have been too low to elicit less common AEs to the components of the polypill.

The influence of adjunctive therapies upon the success of the polypill is not known. In the treatment of chronic tendinopathy, adjunctive physiotherapy such as the “eccentric loading” training programs used in Achilles tendinopathy has been demonstrated to facilitate healing and recovery.¹⁸ Further research on the concurrent use of the polypill and adjunctive therapies is required.

There were several limitations to the study. Firstly, the study was an unblinded, non-randomized sequential case series. All patients who were suitable for inclusion in the study were offered polypill treatment. The study was not able to determine the degree of adherence to the polypill regimen by each patient, nor could the study calculate the total dose of fish oil or green tea. The study was unable to take into account the possible increased motivation towards recovery of some of the participants. Study numbers were too small to determine whether age or gender affected the treatment outcome. Finally, the study followed patients beyond resolution of their initial injury on an ad hoc basis, so while at time of writing there was only one report of injury recurrence, the real injury recurrence rate is unknown.

The results of this study suggest that the treatment of acute tendinopathy via a polypill approach shows promise, but only in some patient groups.

Multi-arm trials should be undertaken to investigate the efficacy of the polypill in the treatment of acute tendinopathy. Trials should also be undertaken to determine the efficacy of the polypill in the treatment of chronic tendinopathy.

Key messages

Acute tendinopathy is a disorder for which the exact aetiology is not fully understood;

Ibuprofen 400 mg tds for two weeks appears to settle

the symptoms of most patients suffering from an acute tendinopathy.

There may be a subgroup of patients suffering acute tendinopathy for whom suppression of TNF- α and factors such as cytokines by a polypill may result in a more rapid recovery.

The polypill regimen as suggested in the original paper is safe, and appears to have no unexpected adverse effects.

The author declares that this project was entirely self-managed and self-funded.

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The author declares no conflict of interest.

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Retrospective Study of 157 Caudal Epidural Steroid Injections in 92 Patients Over an 8-Year Period

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Introduction

The first record of epidural injections is from France. In 1901 Sicard & Catherine and in 1909 Coussade & Chauffard used epidural injection to relieve back pain. I could find no actual record of what substance they injected, but there is a suggestion, that it was either cocaine or a narcotic.¹

James Cyriax, the doyen of orthopaedic medicine, first used epidural injections with local anaesthetics, as a means of diagnosing low back pain, in 1937. When patients returned with their back pain alleviated, he realized that he stumbled on the therapeutic application of epidural local anaesthetic injections.

In his *Textbook of Orthopaedic Medicine*, Cyriax states that the most effective treatment for low lumbar pain is manipulation, epidural injection, and bed rest. Since then, of course, it has been proven, that bed rest has little if any value in the treatment of low back pain.²

Overall review of the literature on epidural steroid injections indicates that there has been a widespread endorsement of the procedure over the past 30+ years. The therapeutic uses of epidural steroid injections with local anaesthetic are many: from intractable, chronic back pain to referred sciatic pain. It is, however, very important that the patient understands that relief of their pain may be only temporary.

In his book *Spinal Manipulation*, JF Bourdillon, a Canadian orthopaedic surgeon turned orthopaedic physician, describes the uses of caudal epidural injections with a local anaesthetic and hydrocortisone, giving lasting relief from back pain in some patients. Unfortunately, a number of physicians using his methods did not have any consistent results with the use of hydrocortisone.³

More than 40 published papers (which I will not list) have described experience with over 4,000 patients having caudal epidural steroid injections. Only four of these papers have reported unfavourably on the results of the procedure. The greater part of the literature describes the use of caudal and lumbar epidural steroid injections, which by and large have been used only for patients with radicular pain or pain referred to as "sciatica".⁴

Mount et al. listed 287 patients and following the injection 140 had complete relief with 46 better than before. This represents an improvement for 85% of the patients treated.⁵

Goebert et al. had 137 patients of whom 57% were better for a minimum of three months. They had a complication rate of 0.9%.⁶

In Poland, Czarski et al. recorded a result following the

injection in 123 patients. The result was 70% better, 12% marginally better, and 18% no improvement at all.⁷

Methods and results

My experience is over a period of about 30 years with about 700 epidural caudal injections. Unfortunately, until my results were entered into a computer system, I found that extracting the results manually proved a very laborious undertaking.

In this present study it was surprising that there were just fractionally more female than male patients, considering the fact that males are more likely to be involved in heavy physical labour.

Most of the patients were self-referred; however, there were eight referrals by chiropractors and six referrals by other medical practitioners. Seven patients were from out of town and one even from Victoria.

The oldest patient, female nursing home patient, is 92 years old and demands "her injection" every three to four months. According to the nursing staff she must obviously be getting pain relief, as her request for analgesia is reduced after her caudal epidural steroid injection. This patient was actually referred to me by one of our musculoskeletal colleges, Dr Roger Watson, as the patient found it difficult to travel to his rooms and get up the stairs at his surgery.

The youngest patient was one of the reception staff in our surgery. She was 24 at the time and, in the last three years since her caudal injection, she has not had any appreciable back pain problems.

I find selecting patients sometimes quite difficult. After a thorough history and spinal examination, including SLR and the slump test, a certain number of patients do have either a CT scan or an MRI (some arrive with their scans already) to help with the assessment process. My impression is that patients with spinal stenosis and arthritic changes and little if any sciatica seem to do better than patients with a large bulging disc compressing exit foramina of the lumbar nerves. I well remember a 40-ish male patient with severe sciatic pain, who heard of the caudal injection and insisted that he wanted to try it. It was quite obvious that with the large disc protrusion pressing on his L5 nerve he was not a candidate but he absolutely insisted, having travelled from out of town "just to have the needle". Against my better judgment I relented. The injection did not help and he had some relief after spinal surgery.

The injection

I felt it was important to standardize the procedure **as much as possible!** At the initial consultation, patients are told that they will most likely experience temporary postural hypotension and some weakness in the legs. I insist that somebody drives them home and that they lie down for at least a couple of hours until their legs feel normal. They are told that their pain will most likely get worse for the first 4-5 days and they are not likely to experience any improvement in the pain until the sixth or seventh day.

Patients are always asked to come back for a review in seven days, or at least phone back in a week or so, if unable to return.

I am asked by patients all the time: "How long does the relief from a caudal epidural injection last"? I have had some patients coming back (for unrelated consultations) months and years later, and they insist their pain is either gone or so minimal, that it needs no or minimal analgesia. On the other hand, there are patients who come back at irregular intervals, to have another injection.

I am certain that patients are not stupid or masochistic and if they return voluntarily and request a caudal epidural steroid injection, it must be because, previously, it had some beneficial effect. There is also no question that the procedure does not work in some patients.

Contraindications

If the patient has a bleeding disorder or is on an anticoagulant, it is preferable not to inject. If the patient is on warfarin, stop the warfarin (if clinically not contraindicated), before undertaking the procedure.

Whilst not a total contraindication, patients with severe fluid retention/CCF are not really suitable for this procedure, as a substantial amount of fluid is injected.

Any infection in the area is a total contraindication!

In the case of recent major surgical procedure, it is advisable to wait until the patient has recovered before proceeding to a caudal injection. However, previous spinal surgery or possibly altered anatomy may not necessarily be a contraindication, but could make the procedure more difficult.

Hypersensitivity or allergy to any of the components of the injection is also a contraindication.

Any patient with high fever, especially one with a suspected diagnosis of meningitis, **MUST NOT** have the injection.

Poor outcome to the injection will most likely result in severe radiculopathy, due to a large disc protrusion.

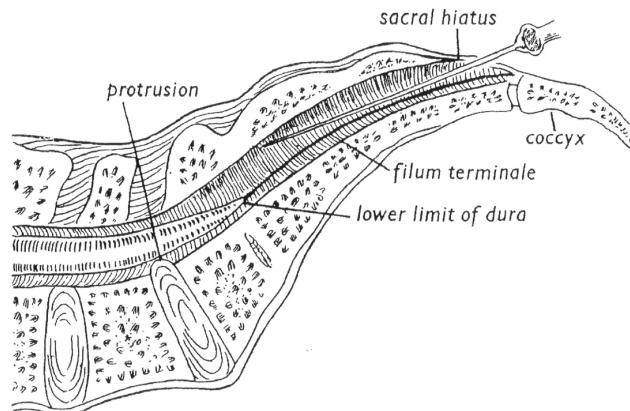
Be careful of suspect Workers' Compensation cases, because they may not have genuine pain. Also beware of the patient with normal SLR and slump test, whose pain does not decrease with medication.

The injection

I usually use 40 ml of 0.5% xylocaine in two syringes, (one can always use less, but never more). The xylocaine in the first syringe (20 ml) is also used to infiltrate the site of the injection. The second syringe contains 20 ml of xylocaine together with 80 mg (two single dose vials) of Depo Medrol (methylprednisoloneacetate). These vials do not contain any preservatives.

The patient is lying supine, with a couple of pillows under the stomach. This gives good exposure and access to the caudal hiatus. The gluteal muscles must be relaxed; otherwise the procedure becomes very difficult. I sometimes have a problem with some muscular male patients who involuntarily pull their buttock cheeks together.

The area of the injection is liberally sterilized with Betadine and, unless the patient is very obese, the caudal hiatus is relatively easily palpated. The area under the skin is then infiltrated with some of the local anaesthetic from the first syringe. A spinal needle is then introduced through the sacral hiatus. After making certain that there is no "bloody tap" coming back into the syringe, the remainder of the local anaesthetic in the first syringe is slowly injected. This is followed by the second syringe with the local anaesthetic and the Depo Medrol.



Epidural injection. The needle in position.

During the injection I continually talk to the patient, and ask whether they have any strange sensation, especially in their head or legs, or feel nauseated. Also our nurse keeps an eye on the patient's pulse for rate changes and irregularities. If the patient's speech starts to slur, the injection is stopped immediately.

The patient then remains on the table for about 10-15 minutes. After that time they are taken by wheelchair to a waiting car and driven home by a friend or relative.

To prevent any postural hypotension effect, the carer is told to open the door of the home and make sure there is a clear passage way for the patient to get out of the car and walk straight into a bed. The patient is asked to remain resting until all the numbness disappears from their legs and they do not feel light-headed when they stand up.

As mentioned before, all patients are requested to return

for a reassessment in about a week's time, or if they are from out of town, to phone the surgery informing me how they feel.

Results

As a great number of patients had more than one caudal epidural steroid injection, I assessed the results of this retrospective study, stating how each individual patient responded to each individual injection.

Some patients had only one injection. However, quite a few had more than one. One patient over the surveillance period of eight years had a record of 13 injections. As you can see from the attached chart, she reported her pain to be much better or even totally relieved each time.

Patient responses were categorized as follows:

Total pain relief	22
Feeling much better but still some residual ache	67
Better with about 50% pain relief	33
Only slight improvement in pain	13
NO Pain relief at all (or worse)	14
Total injections followed up	149
No follow up	8
Total number of injections	157

Discussion

So the final result of this retrospective study is:

- That 59.6% or nearly 60% of patient responses to the epidural caudal steroid injection were reported as total or near total pain relief.
- 30.8% of injections resulted in patients getting some relief, but still having some residual pain and discomfort.
- Only 9.3% of injections resulted in no pain relief.

The procedure appears to be safe, provided the operator fully understands the contraindications and most importantly, does not proceed with the injections if there is a "bloody tap" in the syringe. I would therefore recommend this procedure, in carefully selected patients as an alternative means of giving patients with severe back pain and/or sciatic pain at least temporary pain relief.

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Acupuncture in the Treatment of Osteoarthritis of the Knee: Evidence and Consensus

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Osteoarthritis is the most common form of arthritis. It affects millions of people in Australia and many more worldwide. Osteoarthritis, at this point in time, has no cure. The significance of this disease is illustrated not only by its prevalence in the community, but also by the impact it has on that community. It affects patients in terms of pain and disability, financial costs, and quality of life. It also imposes a financial burden on all levels of society – the family, local community, business, and government. And its impact is increasing with time in line with the phenomenon of the ageing population.

In 1990, WHO estimated osteoarthritis was the 10th leading cause of “non-fatal burden” in the world; it accounted for 2.8% of total YLD (years of healthy life lost due to disability). By 2000 it had become the 4th leading cause of YLD and accounted for 3.0% of YLD globally.¹

A 2003 WHO Bulletin stated:

As the population of the world grows older and medical advances lengthen average life expectancy, osteoarthritis will become a larger public health problem — not because it is a manifestation of ageing but because it usually takes years to reach clinical relevance. An older population lives on through those years, so physicians, surgeons, architects, and city planners, as well as designers of furniture and cars will have to take notice.²

Prevalence and impact of osteoarthritis in Australia

In 2007, Access Economics³ estimated that 3.85 million Australians or 18.5% of the population have some form of arthritis. The most prevalent is osteoarthritis, which affects 7.8% of the population, or 1.62 million people. This represented an increase from 1995, when 14.7% and 6.4% of the population was estimated to have arthritis and osteoarthritis, respectively.

The health cost of osteoarthritis is formidable and increasing. Access Economics estimated that allocated health costs for osteoarthritis were \$837.9m in 2000, and it increased to \$1,948m in 2007. Osteoarthritis was estimated to have accounted for 63% of hospital inpatient expenditure, 30% of hospital outpatient expenditure and 75% of aged care expenditure. In 2004–2005, more than 41,000 total hip and knee replacement were performed for osteoarthritis.² For the same period of time, \$145 million and \$186 million were spent in Australian public hospitals on hip and knee replacements, respectively. Additionally, more hip and knee replacements were performed in private hospitals than in public hospitals.

The economic impact of arthritis extends beyond direct health system costs. Access Economics estimated that non-

financial costs of arthritis (which osteoarthritis is the largest component) to be \$7.37 billion in 2007. Non-financial costs included productivity costs (such as employment impacts, absenteeism and premature death), carer costs, aids and modifications costs, travel costs, program costs (such as community care and welfare) and “deadweight loss” (impact of loss of taxation revenue).

Furthermore, there are intangible costs of arthritis such as loss of quality of life, loss of leisure, physical pain and disability attributed to arthritis. When converted into dollar terms, Access Economics estimated the loss of well-being cost in 2007 amounted to \$11.7 billion.

Management of osteoarthritis

There is no cure for osteoarthritis. Extensive guidelines are available for the management of osteoarthritis from organizations such as the American College of Rheumatology (ACR), the European League Against Rheumatism (EULAR) and the Osteoarthritis Research Society International (OARSI). All guidelines included recommendations relating to general treatments, pharmacological, non-pharmacological, and surgical modalities.

Joint replacement therapy is accepted as the most definitive treatment for osteoarthritis of the hip and knee. Its effectiveness has largely been established. But it is costly and associated with significant risks and complications. It is generally agreed that such a procedure should not be carried out until the disease has reached a certain stage. There are also many situations when surgery is not suitable or contraindicated.

Otherwise, treatment of osteoarthritis is directed at treating the main symptoms – pain and restriction in movement. Medications provide only pain relief temporarily and may not be sufficiently effective. Furthermore, medications such as NSAID and COX-II inhibitors should not be used long term, because of the potential risk of side effects.

Much attention, and therefore research, has been directed towards the use of non-medicinal modalities and complementary medicine. In the AIHW analysis of ABS 2004–05 National Health Survey, 12% of people with osteoarthritis reported that they visited a GP or specialist for their condition in the two weeks before the survey.⁴ In the same period of time 6% of females and 4% of males reported visiting other health professionals (non-medical).⁵

Surveys showed that approximately 50% of the Australian population used at least one form of complementary medicine per year and that 20% actually consulted at least one practitioner of complementary medicine per year. Research indicates that many GPs in Australia have accepted therapies such as acupuncture, chiropractic, hypnosis and meditation as potentially beneficial. Over 80% of the GPs surveyed had

referred patients for a complementary therapy at least a few times a year. Nearly 20% practised one complementary therapy. Acupuncture appears the most popular, with at least 15% of Australian GPs practising this treatment.⁵

However, traditional Western medicine literature does not recommend acupuncture as frontline therapy for the treatment of osteoarthritis. There are also no direct data on the current use of acupuncture for treatment of knee osteoarthritis in Australia.

With the increasing volume of research data now available on the topic of acupuncture and knee osteoarthritis it is conceivable that there may be evidence to support the use of acupuncture as a frontline tool in the management of knee osteoarthritis.

Recent systematic review/meta-analyses on acupuncture for osteoarthritis of the knee

A PubMed search on “acupuncture” and “knee osteoarthritis” yielded a number of systematic reviews and meta-analyses. The two most recent systematic review/meta-analyses were published in 2007. They were selected for review to evaluate the value of acupuncture in the management of knee osteoarthritis.

Manheimer E et al. Meta-analysis: Acupuncture for osteoarthritis of the knee. *Annals of Internal Medicine*, 19 June 2007¹⁰

Institutions involved

- University of Maryland School of Medicine, Baltimore, Maryland, USA
- Technische Universität, München, Germany
- VU University Medical Centre, Amsterdam, the Netherlands

Inclusion criteria

- Trials comparing acupuncture with sham acupuncture, usual care or waiting list control group for patients with knee osteoarthritis
- Randomized trials longer than 6 weeks

Not included

- Dry needling or trigger-point therapy
- Trials comparing only 2 different types of acupuncture

Study characteristics

- Mean duration of knee pain = 5 years or more
- All patients had to have a diagnosis of osteoarthritis
- All but one trial required radiological evidence
- 11 trials (2821 patients) accepted for systemic analysis and 9 trials (unspecified number of patients) accepted for meta-analysis
- The largest trial recruited 330 patients in the active

treatment group; 4 larger size trials had 150 or more patients in the active treatment group

- Point selection – 8 RCTs used a set formula; 2 RCTs used a flexible formula and 1 RCT used a pragmatic approach at the discretion of the treating physician
- Superficial needling – 1 trial; deep needling to elicit de qi – 9 trials; 1 trial – not mentioned
- Electrical stimulation was used in 4 trials
- Adequacy of acupuncture and sham acupuncture was assessed by 2 acupuncturists independently – based on 4 aspects; choice of acupuncture points, number of sessions, needling technique and experience of the acupuncturist. Details of the criteria were not provided.
- Internal validity of the trials was assessed using the 11 item scale developed by the Cochrane Collaboration Back Review Group (2003).

Results

- Compared with sham acupuncture, acupuncture provided clinically irrelevant improvement in pain and function in the short term and at 6 months.
- Compared with waiting list and usual care control group, acupuncture provided clinically relevant short-term improvement and those improvements were largely maintained at 6 months. However, the sham group also showed greater improvement compared with usual care control group.
- 2 sham controlled trials showed clinically relevant benefit of acupuncture compared with sham. Both utilized non-penetrating methods (non-penetrating needles and patch electrode with mock electrical stimulation). Credibility of the sham was not tested in both trials.
- 2 other sham controlled trials which utilized superficial penetrating needling sham found no or minimal clinical relevance between true and sham acupuncture, and it provided clinically relevant improvement similar to that of true acupuncture.

White A et al. Acupuncture treatment for chronic knee pain: a systematic review. *Rheumatology*, 10 January 2007¹¹

Institutions involved

- Peninsula Medical School, Universities of Exeter and Plymouth, United Kingdom
- Primary Care Musculoskeletal Research Centre, Keele University, Keele, Staffordshire, United Kingdom
- British Medical Acupuncture Society, Royal London Homeopathic Hospital, London, United Kingdom

Inclusion criteria

- Randomized trials
- Trials including:
 - * Adults who had either chronic knee pain on most days for at least 3 months, or a diagnosis of osteoarthritis or osteoarthritis of the knee

- with radiological confirmation.
- * Comparing acupuncture with sham acupuncture, other sham treatment, no additional intervention (usual care) or an active intervention.
- * Outcome measures which included pain or function, measured with any instruments.

Not included

- Post-operative knee pain
- Studies comparing different forms of active acupuncture
- Forms of acupuncture without needles

Study characteristics

- Mean baseline WOMAC pain score in majority of studies was 9/20 or more.
- Knee pain rather than diagnosis of osteoarthritis used as inclusion criteria.
- All but one trial required radiological diagnosis; study that did not require radiological diagnosis was included only in the systematic review but not in the meta-analysis.
- 13 trials (2362 patients) were accepted and included in the systemic review and 8 trials (unspecified number of patients) accepted for meta-analysis.
- The largest trial recruited 330 patients in the active treatment group; there were 3 larger size trials which had 150 or more patients in the active treatment group.
- Treatments were standardized to some extent in all studies and, in all but one study, the treatment was described sufficiently well to be replicable.
- Adequacy of acupuncture was defined as “adequate” based on the following criteria:
 - * Consisted of at least 6 treatments.
 - * At least one per week.
 - * At least four points needled for each painful knee for at least 20 minutes.
 - * Either needle sensation (de qi) achieved in manual acupuncture, or electrical stimulation of sufficient intensity to produce more than minimal sensation.
- A control was defined as a “true sham” only when it avoided stimulating nerves in the same neurological segments as the knee joint. Even superficial penetration with needles is regarded as unacceptable because it has the potential to be physiologically active.
 - * Two studies used true (that is, virtually inactive and involved no skin penetration on or near the knee) sham acupuncture as control. One included penetrating needles in sham points in the abdomen.
 - * Five studies were excluded because they used superficial acupuncture at non-points on or near the knee.
- Internal validity of the trials was assessed using a 9-item scale developed by the Cochrane Collaboration Back Review Group (1997).

- For cross-over studies, only the first arm was included to avoid the effects of carry-over treatment.

Results

- For “sham acupuncture control” studies:
 - * For pain reduction both in the short and long term, true acupuncture was superior to sham acupuncture.
 - * For improvement of function both in the short term and long term, true acupuncture was superior to sham acupuncture.
 - * It was noted that there was a strongly positive study which resulted in the heterogeneity of the result. When this study was omitted from the calculation, the results remained positive.
- For “no additional treatment control” studies:
 - * For pain reduction, acupuncture was significantly superior with no significant heterogeneity.
 - * For improvement in function, acupuncture was significantly superior, but with significant heterogeneity because of a study with included intensity physiotherapy to all groups.
- For “education as control” study (one study only):
 - * Acupuncture was superior for both pain and function, both in the short and long term.
- For “TENS-like acupuncture as control” study:
 - * Acupuncture was not shown to be significantly better.
- The authors also noticed that:
 - * For the one study that provided strongly positive results, strong treatment using 4 pairs of electrical stimulation was used. The placebo was a non-penetrating blunt needle sham acupuncture; and both groups were given diclofenac.
 - * A study that used “inadequate” acupuncture with only 2 needles showed no significant effect.

Comparison of the two systematic reviews/meta-analyses

White et al. included 13 studies⁸⁻²⁰ in their systematic review and 8 studies^{8-12, 17-19} in their meta-analysis. Manheimer et al. included 11 studies^{8-14, 17-19, 21} in their systematic review and nine studies^{8, 9, 12-14, 17-19, 21} in their meta-analysis. There were 10 studies^{8-14, 17-19} selected by both groups for systematic review and six studies^{8, 9, 12, 17-19} selected by both groups for metaanalysis.

White et al. concluded that there is evidence to suggest that acupuncture is superior to placebo for the treatment of chronic knee pain. It therefore can be considered an evidence-based option in its treatment. However, the results are not strong enough to make firm recommendations for long-term treatment.

Manheimer et al. concluded that the pooled effects of acupuncture are statistically superior to those of sham treatment. They also found that acupuncture provided clinically relevant improvement when compared with usual

care or waiting list; but in that context, there is no or minimal improvement compared with sham acupuncture. On this basis, they suggested that the effects of acupuncture may be due to placebo, although they conceded that superficial penetrating sham acupuncture was so similar to true acupuncture that it may have weak physiologic activity and may not have been true placebo controls.

Both groups recommended that further large scale studies are required. They further suggested that physiologically inactive but credible sham acupuncture should be used. Long-term studies with maintenance treatment; and comparative studies with other non-pharmacological interventions should be carried out.

Discussion on the findings of the two systematic reviews

In the pursuit for evidence-based medicine, guidelines were developed for use as tools in the evaluation of clinical information. The National Health and Medical Research Council (NHMRC) regards an intervention to have level 1 evidence if the intervention is supported by a systematic review of level 2 studies (randomized controlled trials).²² Other institutions and scientific organizations developed similar hierarchy for evaluation of clinical evidence and strength of recommendation.

However, RCTs were developed in the context of pharmacological treatment. Studies on non-pharmacological interventions (including surgical interventions) commonly encounter methodological issues, including:

- Control intervention – placebo or sham intervention can be difficult or impossible to perform for ethical or technical reasons.
- Blinding – blinding of patients and care providers is often impossible. For example, patients commonly know which rehabilitation program they have undergone and surgeons commonly know whether a patient has undergone a true or sham surgical procedure.
- Standardization and care providers' effects – non-pharmacological interventions are usually complex, multi-factorial and individualized. The active component is commonly difficult to identify, and the intervention is difficult to standardize and replicate. It is also commonly dependent on the care providers' skills.

A comparative analysis of articles on pharmacological and non-pharmacological interventions of hip and knee osteoarthritis showed that non-pharmacological articles scored lower in terms of quality.²³ It may not be possible to demonstrate level 1 evidence for some non-pharmacological interventions, although they are commonly and consensually considered as effective. For example, joint replacement therapy is generally accepted as effective for treatment of osteoarthritis of the knee and hip, in terms of pain relief, functional improvement, quality of life and cost effectiveness. Yet it is not evaluated with RCTs because of ethical and

methodological considerations. Instead its acceptance as an effective treatment is based on large numbers of observational studies and some cohort studies. On the hierarchy of evidence, it qualifies only for level 3.²⁴

With regards to assessment of the effectiveness of acupuncture in the treatment of osteoarthritis of the knee, similar methodological issues are encountered. This was acknowledged by both authors in their respective articles.

Many authors now consider that sham acupuncture using penetrating needles, at a non-acupuncture distal point, cannot be considered a true placebo.²⁵ It appears to have analgesic effects on 40-50% of patients. A possible explanation of sham acupuncture's analgesic effect is via the descending pain inhibitory mechanism of diffuse noxious inhibition control (DNIC).²⁶

Lunde and Lundeberg further proposed that the various forms of acupuncture control may activate unmyelinated C afferent, which generates activity in the insular of the limbic system (and not necessarily in the somato-sensory cortex), resulting in emotional and hormonal reactions commonly seen in caressing. They further proposed that this limbic response may alleviate the affective component of unpleasantness of pain and therefore would be equally effective as true acupuncture in the treatment of pain conditions with affective component.^{27,28}

Considering the above, it is not surprising that when Peter White et al. conducted a single blind, randomized cross-over pilot study on patients in hip and knee replacement lists, using true acupuncture and the "Streitberger" needle (the needle pricks the skin then withdraws into its handle, causing a pricking sensation without penetrating the skin). The study produced a fascinating mixed result. Most patients were not able to discriminate the needles by penetration, but 40% could detect a difference in treatment type. No major differences in outcome were found between the two groups.²⁹

Adrian White, who conducted one of the above systematic reviews, and Jorge Vas, who conducted the trial which was included in both meta-analyses and had the best positive result, conducted an "exploratory review" of four recent well-designed sham controlled RCTs of acupuncture on osteoarthritis and White's systematic review of 13 RCTs.³⁰ They seek to understand why a particular study provided a better positive result compared to the other three and to find some indication of what constitutes optimal acupuncture treatment. They arrived at the following speculative factors that might contribute to optimal results:

- Climatic factors – high temperature.
- High patient expectations.
- Minimum of four needles – there may not be much more benefit from adding more needles. Furthermore, needling distal points may not improve the patient's response.
- Electro-acupuncture rather than manual acupuncture, especially strong electroacupuncture to needles placed in muscles.
- A course of at least 10 sessions. Most trials gave acupuncture at least twice weekly at the outset, but

the only trial that gave acupuncture once weekly had the best result.

All trials using manual acupuncture followed the practice of eliciting de qi; therefore, the authors were not able to draw conclusions regarding de qi. All trials left needles in situ for at least 20 minutes. The studies that left needles in situ for 30 minutes did not provide better results.

However, experienced acupuncturists commonly described the traditional notion of “strong reactors” and “normal reactors”. Felix Mann stated in his chapter in *Medical Acupuncture: A Scientific Western Approach* that “I think by far the commonest cause of failure in acupuncture is the failure to distinguish the Strong Reactors from the Normal Reactors.”³¹

Similarly, Peter Baldry³² described the aggravation of pain when a Strong Reactor was overstimulated, and Anthony Campbell³³ described the Strong Reactor as one that gained therapeutic effect regardless of the site of needling. Hence the traditional approach proposes that the “optimal amount” of acupuncture is a very individualized and subjective notion based on the assessment of the experienced clinician who takes into consideration a large number of factors such as:

- Patient characteristics
- Condition being treated
- Environmental factors, such as season of the year, time of day, prevailing climatic factors.

In summary, the scientific evaluation of acupuncture for its role in the management of knee osteoarthritis is marred by:

- The difficulty in finding a truly inert control intervention that is creditable
- The complexity of treatment, and the lack of an evidence-based “optimal acupuncture dose and regime”.

The results were positive for acupuncture, but there are more questions that need to be answered, such as:

- Is there an optimal dose and regime?
- Should an acupuncture regime with a maintenance program incorporating treatment every 1-3 months be evaluated?
- Is there a subset of target population that will benefit more from acupuncture, and if so how do we identify that subset?

Acupuncture in the management of knee osteoarthritis: current consensus

The use of acupuncture in the management of osteoarthritis has been evaluated extensively. Guidelines developed by many health organizations incorporated recommendations on the use of acupuncture in the management of osteoarthritis.

EULAR recommendations 2003³⁴ did not include acupuncture in the “final set of 10 recommendations based on both evidence and expert opinion”. Of the 10 recommendations, two related to general principles of

management, seven related to pharmacological agents (including glucosamine and chondroitin) and only one related to non-pharmacological treatments. Of the non-pharmacological recommendation, four modalities were mentioned: education, exercise, appliances and weight reduction. Acupuncture was not mentioned in that recommendation. However, it was acknowledged that acupuncture was one of the treatment modalities used by healthcare professionals for knee osteoarthritis. In the table of “Level of evidence based on the literature search, and strength of recommendation based on both evidence and expert opinion”, acupuncture was considered to have Category 1B evidence in the management of knee osteoarthritis and its strength of recommendation was rated as Category B. Comparatively, the four modalities mentioned above were rated as follows:

	Level of evidence	Strength of recommendation
Education	1A	A
Exercise	1B	B
Appliances	1B	B
Weight loss	1B	B
Acupuncture	1B	B

OARS recommendations for the management of hip and knee osteoarthritis³⁵ included acupuncture as one of the 25 recommendations. It is considered “may be of symptomatic benefit in patients with knee OA”, with Category 1a level of evidence and with Strength of Recommendation (SOR) at 59%.

Of the 25 recommendations, one related to general principles of management, 11 related to non-pharmacological modalities, eight related to pharmacological modalities, and five related to surgical modalities. Of the modalities similar to the EULAR recommendations mentioned above, the respective level of evidence and strength of recommendation are as follows:

	Level of evidence	Strength of recommendation
Education	1a	92%
Exercise (knee)	1a	85%
Appliances - walking aids	IV	90%
Appliances - knee brace	1a	76%
Appliances - insoles	1a	92%
Weight loss	1a	96%
Acupuncture	1a	59%

It is noteworthy that the OASRI “strength of recommendation” is generated via a Delphi exercise involving a panel of experts taking into consideration research evidence, treatment

factors such as safety and cost effectiveness, and the experts' perception on patient tolerance, acceptability, and adherence. While acupuncture scored highly in terms of level of evidence, it scored only fairly in terms of strength of recommendation. Additionally, it scored only 69% in terms of "level of consensus", indicating a higher degree of disagreement among the experts.

The Royal Australian College of General Practitioners (RACGP) Clinical Guidelines on Hip and Knee Osteoarthritis³⁶ recommended that there is Grade C evidence to support a GP recommending acupuncture for the treatment of knee osteoarthritis. Included in the guidelines are 34 recommendations (26 positive recommendations and eight negative recommendations).

Of the 26 positive recommendations, four related to general principles of management, 15 related to non-pharmacological modalities, and eight related to pharmacological modalities. Surgical interventions were not mentioned as the guideline also included algorithms which included referral to orthopaedic surgeons for consideration towards surgery. Comparing a similar group of non-pharmacological interventions as above, the grading of recommendations was as follows:

	Grading of recommendation
Education	C
Exercise - land based	B
Appliances	*
Weight loss	B
Acupuncture	C

* Braces and orthoses were included in the negative results group and considered to be of little or no benefit with Grade B recommendation. Walking aids were not mentioned.

The ACR 2000 update on the recommendations for the medical management of osteoarthritis of the hip and knee considered acupuncture as too difficult to evaluate and recommend.³⁷ The college proposed to make recommendations on acupuncture when the result of a large randomized controlled trial being conducted by the National Institute of Health (NIH) is published.

The Royal Australasian College of Physicians (RACP) does not have a current management guideline on osteoarthritis, but it is in the process of implementing the "Arthritis and Musculoskeletal Quality Improvement Program (AMQIP)" which will address best practice for the optimal management of osteoarthritis. It is not clear whether acupuncture is being considered or discussed in the program.

A Cochrane systematic review is currently being revised to assess the efficacy of acupuncture for osteoarthritis. In particular, it proposes to assess:

- The efficacy of acupuncture alone compared to standard medical treatment
- The efficacy of acupuncture plus standard medical treatment compared to standard medical treatment alone

- The efficacy of true acupuncture compared to sham acupuncture.⁹

Conclusion

Both of the more recent systematic reviews on acupuncture in the management of knee osteoarthritis yielded positive results. But both authors were careful in their interpretations and recommendations. Review of some Australian and international guidelines on the management of osteoarthritis reflected a similar sentiment – reasonably strong levels of evidence but cautiously moderate strength of recommendation. Other socially more recognized and more acceptable non-pharmacological treatment frequently enjoyed higher strength of recommendation despite similar level of evidence when compared with acupuncture.

More large scale and better-designed trials are required to provide good quality data to support the use of acupuncture in knee osteoarthritis. However, better understanding and acceptance by the community and healthcare professionals is equally important if acupuncture is to become a recommended frontline treatment modality for knee osteoarthritis.

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Efficacy of low-level laser therapy in the management of neck pain: a systematic review and meta-analysis of randomised placebo or active-treatment controlled trials*

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Summary

Background. Neck pain is a common and costly condition for which pharmacological management has limited evidence of efficacy and side-effects. Low-level laser therapy (LLLT) is a relatively uncommon, non-invasive treatment for neck pain, in which non-thermal laser irradiation is applied to sites of pain. We did a systematic review and metaanalysis of randomised controlled trials to assess the efficacy of LLLT in neck pain.

Methods. We searched computerised databases comparing efficacy of LLLT using any wavelength with placebo or with active control in acute or chronic neck pain. Effect size for the primary outcome, pain intensity, was defined as a pooled estimate of mean difference in change in mm on 100 mm visual analogue scale.

Findings. We identified 16 randomised controlled trials including a total of 820 patients. In acute neck pain, results of two trials showed a relative risk (RR) of 1.69 (95% CI 1.22–2.33) for pain improvement of LLLT versus placebo. Five trials of chronic neck pain reporting categorical data showed an RR for pain improvement of 4.05 (2.74–5.98) of LLLT. Patients in 11 trials reporting changes in visual analogue scale had pain intensity reduced by 19.86 mm (10.04–29.68). Seven trials provided follow-up data for 1–22 weeks after completion of treatment, with short-term pain relief persisting in the medium term with a reduction of 22.07 mm (17.42–26.72). Side-effects from LLLT were mild and not different from those of placebo.

Interpretation. We show that LLLT reduces pain immediately after treatment in acute neck pain and up to 22 weeks after completion of treatment in patients with chronic neck pain.

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Introduction

Chronic pain is predicted to reach epidemic proportions in developed countries with ageing populations in the next 30 years.¹ Chronic neck pain is a highly prevalent condition, affecting 10–24% of the population.^{2–5} Economic costs of this condition are estimated at hundreds of millions of dollars,² creating an imperative for evidence-based, cost-effective treatments. Low-level laser therapy (LLLT) uses laser to aid tissue repair,⁶ relieve pain,⁷ and stimulate acupuncture points.⁸ Laser is light that is generated by high-intensity electrical stimulation of a medium, which can be a gas, liquid, crystal, dye, or semiconductor.⁹ The light produced consists of coherent beams of single wavelengths in the visible to infrared spectrum, which can be emitted in a continuous

wave or pulsed mode. Surgical applications of laser ablate tissue by intense heat and are different from LLLT, which uses light energy to modulate cell and tissue physiology to achieve therapeutic benefit without a macroscopic thermal effect (sometimes termed cold laser). LLLT is non-invasive, painless, and can be easily administered in primary-care settings. Incidence of adverse effects is low and similar to that of placebo, with no reports of serious events.^{10,11}

Research into the use of LLLT for pain reduction^{12,13} and tissue repair^{14,15} spans more than 30 years. However, reports do not identify this therapy as a potential treatment option,¹⁶ possibly because of scepticism about its mechanism of action and effectiveness.¹⁷ Research from the past decade suggests that LLLT produces anti-inflammatory effects,^{18–21} contributing to pain relief. Cochrane reviews of the efficacy

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of LLLT in low-back pain²² and rheumatoid arthritis²³ have been unable to make firm conclusions because of insufficient data or conflicting findings. However, effectiveness depends on factors such as wavelength, site, duration, and dose of LLLT treatment. Adequate dose and appropriate procedural technique are rarely considered in systematic reviews of electrophysical agents. Research into the dose response profile of LLLT suggests that different wavelengths have specific penetration abilities through human skin.^{17,24,25} Thus, clinical effects could vary with depth of target tissue. We have shown the importance of accounting for dose and technique in systematic reviews of transcutaneous electrical nerve stimulation²⁶ and LLLT,^{11,21} and our approach is an acknowledged means of establishing efficacy.²⁷

The only systematic review focusing solely on LLLT in treatment of neck pain included four randomised controlled trials, and concluded that there was evidence of short-term benefit of LLLT at infrared wavelengths of 780, 810–830, and 904 nm.²⁸ A Cochrane review of physical medicine for mechanical neck disorders, since withdrawn because much time had passed without an update, included three LLLT trials, for which outcomes did not differ from those of placebo.²⁹ The same investigators did a meta-analysis³⁰ of 88 randomised controlled trials of conservative treatments for acute, subacute, and chronic mechanical neck disorders, which included eight trials using LLLT. They concluded that LLLT has intermediate and long-term benefits.

These reviews did not identify treatment variables associated with positive outcomes, include non-English language publications, or quantitatively assess data.^{28,30} We have therefore undertaken a new systematic review and meta-analysis of LLLT in neck pain to establish whether LLLT relieves acute and chronic neck pain and to systematically assess parameters of laser therapy to identify treatment protocols and dose ranges (therapeutic windows) associated with positive outcomes.

Methods

Search strategy and selection criteria

We did a search of published work without language restriction using Medline (January, 1966, to July, 2008), Embase (January, 1980, to July, 2008), Cinahl (January, 1982, to July, 2008), the Physiotherapy Evidence Database (January, 1929, to July, 2008), Biosis (January, 1926, to July, 2008), Allied and Complementary Medicine (January, 1985, to July, 2008), and the Cochrane Central Register of Controlled Trials (second quarter of 2008). Keywords used for neck pain and related conditions were: “neck pain/strain”, “cervical pain/strain/syndrome”, “cervical spondylosis/itis”, “cervicobrachial (pain/disorder/syndrome)”, “myofascial (pain/disorder/syndrome)”, “trigger points”, “fibromyalgia”, “whiplash/WAD”, “osteoarthritis/arthritis”, and “zygapophyseal/ZG joints”. We combined these keywords with synonyms for LLLT: “low-level/low-energy/low reactive-level/low-intensity/low-incident/low-output/infrared/diode/semiconductor/soft or cold or mid/ visible”; “laser therapy”, “(ir)radiation”, “treatment”;

“low-energy photon therapy”; “low output laser”; “LLLT”; “LILT”; “LEPT”; “LELT”; “LILI”; “LELI”; “LPLI”; “biostimulation”; “photobio/stimulation/activation/modulation”; “light therapy”; “phototherapy”; “narrow band light therapy”; “904 nm”; “830 nm”; “632 nm”; “1064 nm”; “GaAs”; “GaAlAs”; “HeNe”; and “defocused CO₂”. We consulted experts and searched reference lists of retrieved reports and textbooks for additional references.

Citations were screened and full reports of potentially relevant studies obtained. We applied inclusion and exclusion criteria, assessed methodological criteria, and extracted data including trial characteristics, demographic data, laser parameters, pain outcome measures, and cointerventions. Non-English language studies were translated by JMB.

We included randomised or quasi-randomised controlled trials of LLLT for acute or chronic neck pain as defined by trial investigators, and identified by various clinical descriptors included under the term non-specific neck pain.³¹ These diagnostic labels included neck strain, neck sprain, mechanical neck disorders, whiplash, neck disorders, and neck and shoulder pain. We also used surrogate terms for neck pain, such as myofascial pain and trigger points.^{32,33}

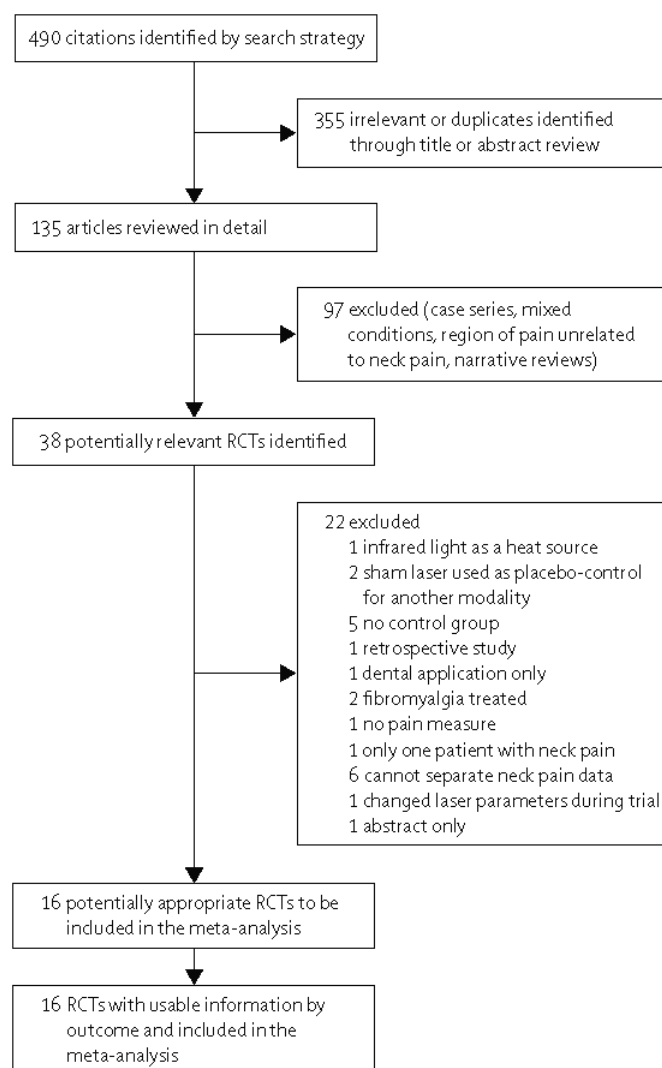


Figure 1. Selection process
RCT = randomised controlled trial

Study participants were restricted to those aged 16 years and older. We excluded studies in which specific pathological changes could be identified, such as systemic inflammatory conditions—eg, rheumatoid arthritis, localised or generalised fibromyalgia, neck pain with radiculopathy, and neck pain related to neurological disease. We excluded abstracts and studies for which outcome measures for neck pain could not be separated from data for other regions of the body. Two reviewers (RTC, JMB) independently undertook the search of published work, screened studies, and extracted data. Any disagreements between reviewers were resolved by consensus with other team members acting as arbiters (RABL-M, MIJ).

Investigators had to have used a laser device that delivered irradiation to points in the neck identified by tenderness, local acupuncture points, or on a grid at predetermined points overlying the neck. Control groups had to have been given either placebo laser in which an identical laser device had an active operating panel with the laser emission deactivated or an active treatment control (eg, exercise). We also included trials in which an active control was used as a co-intervention in placebo and real laser groups.

To be eligible for inclusion, a study had to compare pain relief

along a 0–100 mm visual analogue scale, a numerical rating scale, or by patient-reported improvement (eg, categorical report of no change to complete relief of pain) as a primary outcome measure before and after laser therapy. Functional measures of disability (eg, neck pain disability questionnaire) were assessed as secondary outcome measures. We also examined adverse events where reported, although did not specify these a priori. Duration of follow-up was assessed and defined as short term (<1 month), medium term (1–6 months), and long term (>6 months).

Assessment of methodological quality and heterogeneity

Reviewers assessed all studies for methodological quality on the basis of Jadad criteria (maximum score 5).³⁴ Jadad criteria allocate a point each for randomisation, double-blind design, and description of dropouts. If randomisation and double-blind concealment are assured, an additional 2 points are added. If randomisation or double-blind concealment is not assured, a point is deducted for each. A trial with a score of 3 or more is regarded as high quality. Data from

	n	Design	Diagnosis	Jadad score	Control	Sites treated	Cointerventions	Primary pain outcome measure
Ceccherelli et al (1989) ⁴⁹	27	DB, RCT	Cervical myofascial pain	3	Placebo	Tenderpoints in neck and distal acupuncture points	NR	VAS
Flöter et al (1990) ⁴⁵	60	DB, RCT	Cervical osteoarthritis	3	Placebo	Tenderpoints in neck	NR	VAS
Taverna et al (1990) ⁵²	40	DB, RCT	Chronic myofascial pain	3	Placebo	Tenderpoints in neck	NR	Graded subjective assessment: no change to optimum
Toya et al (1994) ⁵³	39	DB, RCT	Cervical pain complex	5	Placebo	Site not specified	No physical or medical therapy allowed	Graded subjective assessment: exacerbation to excellent
Soriano et al (1996) ⁵⁰	71	DB, RCT	Acute cervical pain	3	Placebo	Site not specified	No NSAIDs or other medical or physical therapy allowed	Graded subjective assessment: exacerbation to excellent
Laakso et al (1997) ⁴⁰	41	DB, RCT	Neck pain with triggerpoints in neck	3	Placebo	Three most painful trigger points	Simple analgesic drugs allowed as needed; NSAIDs, corticosteroids, tricyclic antidepressants excluded; no physical therapies	VAS
Özdemir et al (2001) ⁵⁰	60	DB, RCT	Neck pain related to neck osteoarthritis	3	Placebo	Six arbitrary points over neck muscles	NR	VAS
Seidel and Uhlemann (2002) ⁵¹	48	DB, RCT	Chronic cervical syndrome	3	Placebo	Local neck points and distal acupuncture points	Acupuncture not allowed less than 6 months before inclusion; drug therapy unchanged during trial	VAS
Hakgüder et al (2003) ⁴⁷	62	DB, RCT	Neck pain with one triggerpoint	3	Exercise with LLLT and exercise alone	One active triggerpoint in levator scapulae or trapezius	NR	VAS
Chow et al (2004) ⁴²	20	DB, RCT	Neck pain (non-specific)	5	Placebo	Multiple tender points in cervical spine and attachments	Simple analgesic drugs allowed; no physical therapies	VAS
Gure et al (2004) ⁴⁶	60	DB, RCT	Chronic myofascial pain in the neck	5	Placebo	Up to ten trigger points	NR	VAS
Ilbuldu et al (2004) ⁴⁹	40	DB, RCT	Myofascial pain syndrome	2	Placebo and needling	Trigger points in upper trapezius	Simple analgesic drugs as needed; exercise to all groups	VAS
Altan et al (2005) ⁴¹	53	DB, RCT	Cervical myofascial pain syndrome	3	Placebo	Three triggerpoints bilaterally and one trigger point in trapezius	No NSAIDs or analgesic drugs; exercise in both groups	VAS and graded assessment
Aigner et al (2006) ⁴⁰	45	SB, RCT	Acute whiplash injury	0	Placebo	Local and distal acupuncture points	Both groups wore cervical collar; paracetamol and chlormezanone	Assessment of subjective pain symptoms
Chow et al (2006) ³⁹	90	DB, RCT	Non-specific neck pain	5	Placebo	Local tender points	Simple analgesic drugs allowed; no physical therapies	VAS
Dundar et al (2007) ⁴⁴	64	DB, RCT	Cervical myofascial pain syndrome	3	Placebo	Three triggerpoints bilaterally	No NSAIDs or analgesic drugs	VAS

n=number of patients. DB=double blind. RCT=randomised controlled trial. NR=not reported. VAS=visual analogue scale. NSAIDs=non-steroidal anti-inflammatory drugs. SB=single blind.

Table 1: Study design and outcome measures

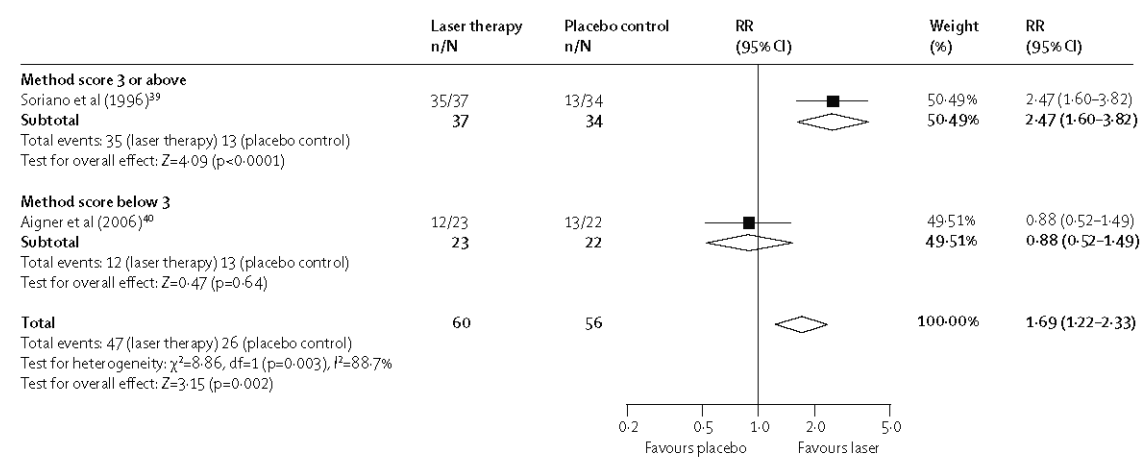


Figure 2: Relative risk of improvement in acute neck pain in laser-treated versus control groups in two randomised trials reporting categorical data
RR=relative risk.

trials with scores of 3 or more were grouped and analysed separately from those scoring less than 3. We assessed clinical heterogeneity by considering population difference in age, sex, duration of symptoms, and outcomes. Clinical judgment was used to establish whether trials were sufficiently similar to allow pooling of data. The specific parameters of laser devices, application techniques, and treatment protocols were extracted and tabulated by laser wavelength. Details for power output, duration of laser irradiation, number of points irradiated, and frequency and number of treatments were listed. When specific details were not reported, calculations were made from those described in the report when possible. When crucial parameters were not reported, we contacted manufacturers of laser devices and trial investigators to obtain missing information. Not all data were available because of the time elapsed since publication of some studies. Heterogeneity was qualitatively assessed for these factors by an expert in laser therapy (JMB).

We used five levels of evidence to describe whether treatment was beneficial: strong evidence (consistent findings in several high-quality randomised controlled trials); moderate

Statistical analysis

Effect size for the primary outcome, pain intensity, was defined as a pooled estimate of the mean difference in change in mm on a 100 mm visual analogue scale between the mean of the treatment and the placebo groups, weighted by the inverse of the SD for every study—ie, weighted mean difference of change between groups. Variance was calculated from the trial data and given, with 95% CI, in mm on visual analogue scale. For categorical data, reported pain relief was dichotomised into two categories (improvement or no improvement), and we calculated relative risk (RR) of improvement, with 95% CI. For the secondary outcome, disability, effect size was defined as the standardised mean difference, which was a combined outcome measure without units—ie, the standardised mean difference in change between active laser groups and placebo groups for all included trials, weighted by the inverse of the variance for each study.³⁶

Mean differences of change for laser-treated and control groups and their respective SDs were included in the statistical pooling. If variance data were not reported as SDs, they were calculated from the trial data of sample size

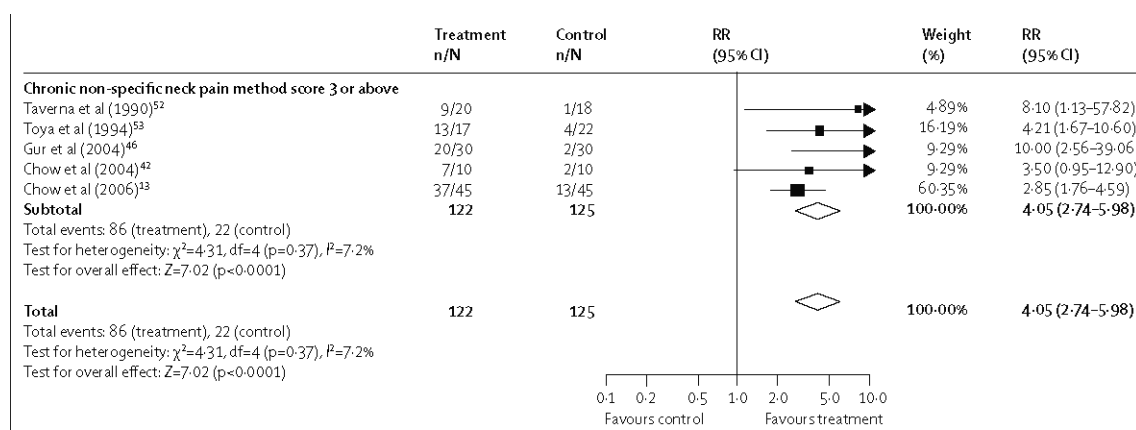


Figure 3: Relative risk of global improvement in laser-treated versus control groups in five trials reporting categorical data for improvement in chronic pain
RR=relative risk.

evidence (findings from one high-quality randomised controlled trial or consistent findings in several low-quality trials); limited evidence (one low-quality randomised trial); unclear evidence (inconsistent or contradictory results in several randomised trials); and no evidence (no studies identified).³⁵

and other variance data values such as p values, t values, SE, or 95% CI. Results were presented as weighted mean difference between laser-treated and control with 95% CI in mm on visual analogue scale—ie, as a pooled estimate of the mean difference in change between the laser-treated and control groups,

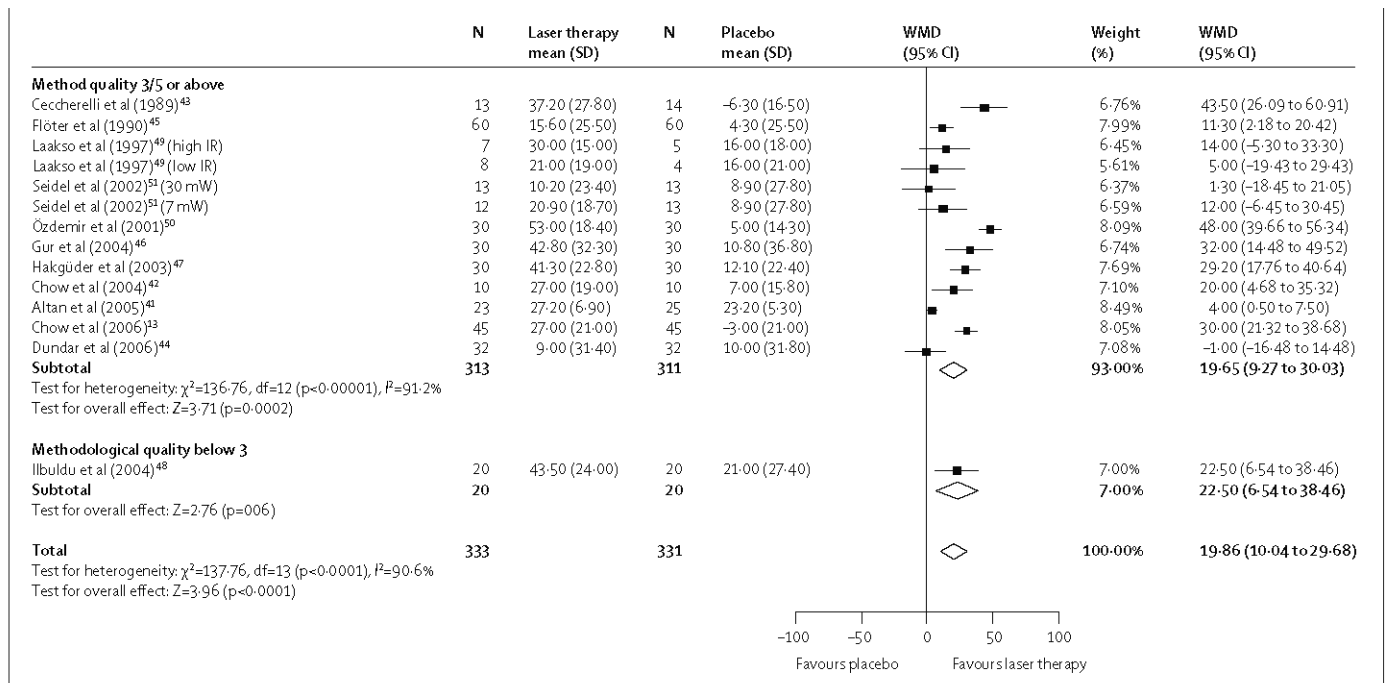


Figure 4: Weighted mean difference in chronic pain reduction on 100 mm visual analogue scale between laser-treated and placebo-treated groups from 11 randomised trials grouped according to Jadad criteria

WMD = weighted mean difference. IR = infrared.

weighted by the inverse of the variance for each study.³⁷ Statistical heterogeneity was assessed for significance ($p<0.05$) with Revman 4.2, and χ^2 and F values greater than 50%. For categorical data, we calculated combined RRs for improvement, with 95% CI. A fixed effect model was used unless statistical heterogeneity was significant ($p<0.05$), after which a random effects model was used. Publication bias was assessed by graphical plot.³⁸ Revman 4.2 was used for statistical analysis and Microsoft Excel 2003 (version 11) to plot dose-response curves.

Role of the funding source

There was no funding source for this study. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

We identified 16 randomised controlled trials of a possible 38 that were suitable for inclusion, and that included 820 patients (figure 1). Two trials^{39,40} provided data for laser therapy of acute neck pain, one treating acute whiplash-associated disorders and one treating acute neck pain of no defined cause. The other 14 trials reported response of chronic non-specific neck pain without radiculopathy to laser therapy.^{13,41–53} Of the studies included, 648 (79%) of the sample of patients with chronic neck pain were women, and patients had a mean age of 43 years (SD 9.8), mean symptom duration of 90 months (SD 36.9), and mean baseline pain of 56.9 mm (SD 7.5) on a 100 mm visual analogue scale in

any trial. Co-interventions were inconsistently reported (table 1). Ten trials reported co-interventions, and six studies did not report or limit co-interventions. Of the studies reporting co-interventions, five groups of investigators explicitly excluded use of concurrent physical therapies, and four excluded use of non-steroidal anti-inflammatory drugs. Four studies allowed use of simple analgesic drugs as needed. Methodological quality assessment values for the trials by Jadad scoring ranged from 0 to 5 (table 1).

Analysis of categorical data for immediate before and after LLLT effects showed that LLLT groups in the two trials^{39,40} of acute neck pain had a significant RR of 1.69 (95% CI 1.22–2.33) for improvement immediately after treatment versus placebo (figure 2). Methodological quality varied between these two studies. Five trials of chronic neck pain reported categorical data, and all were high-quality trials with methodological scores of 3 or more. RR of pain improvement with LLLT was 4.05 (2.74–5.98) compared with placebo at the end of treatment (figure 3).

Analysis of data from visual analogue scale showed that in patients in 13 groups in 11 trials, irrespective of methodological quality, pain intensity was reduced by a mean value of 19.86 mm (10.04–29.68) compared with placebo groups (figure 4). Seven trials with eight LLLT groups provided follow-up data for 1–22 weeks after end of treatment (figure 5). The pain-relieving effect in the short term (<1 month) persisted into the medium term (up to 6 months). Five studies provided evidence for improvement in disability at end of the LLLT treatment (figure 6). Several questionnaire-based outcome measures were used—specifically, the neck pain and disability scale,⁵⁴ Northwick Park neck pain questionnaire,⁵⁵ short form^{36,56} Nottingham health profile,⁵⁷ and neck disability index.⁵⁸

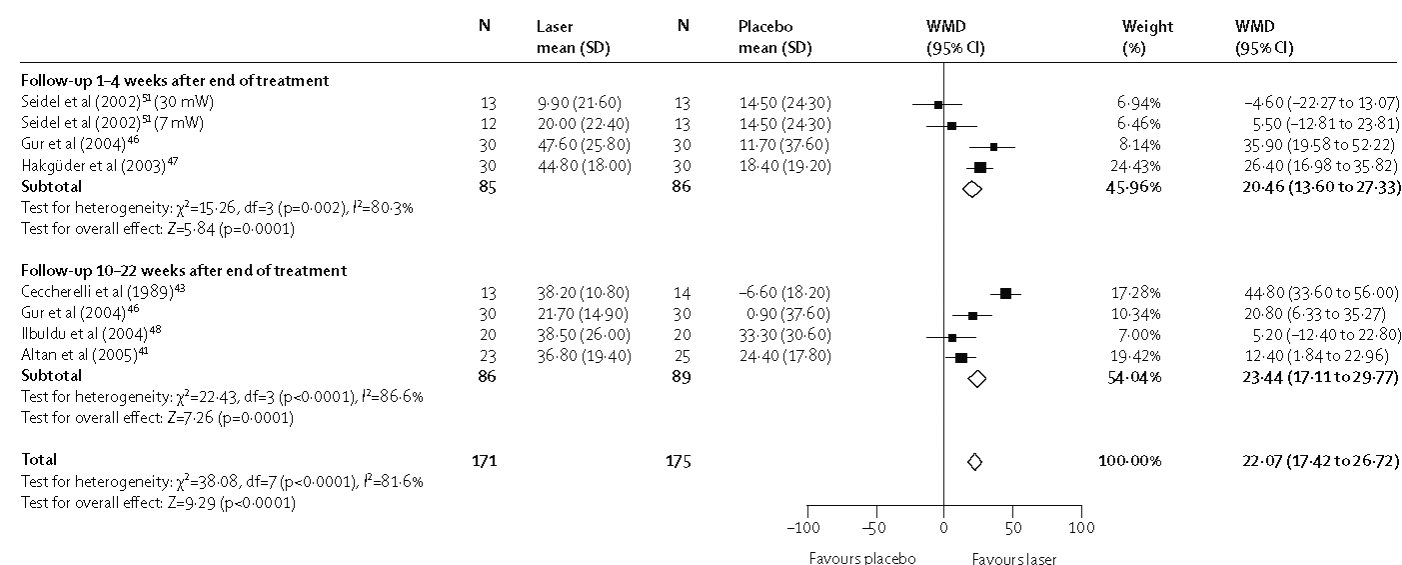


Figure 5. Weighted mean difference in pain reduction on 100 mm visual analogue scale between placebo-treated and laser-treated groups in seven trials reporting follow-up data
 WMD=weighted mean difference

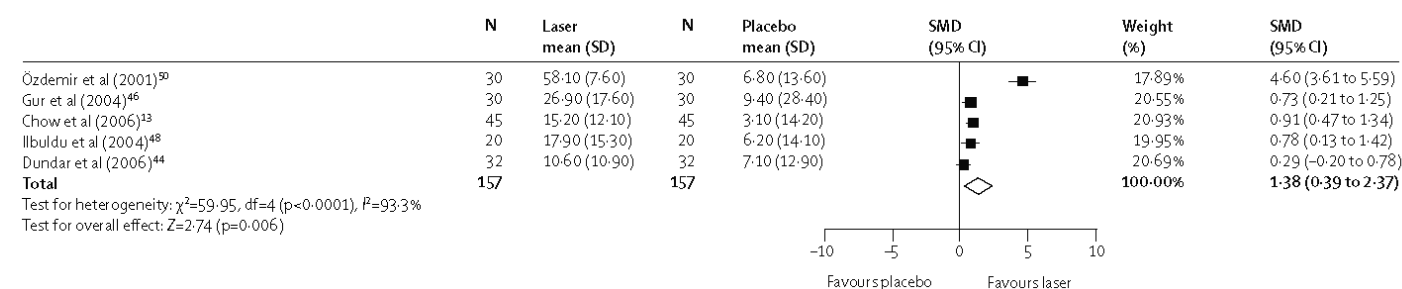


Figure 6. Standardised mean difference in disability scores between placebo-treated and laser-treated groups from five trials
 SMD=standardised mean difference

Positive publication bias, which tends to exclude negative studies, was not apparent on testing (figure 7).³⁸ The plot has an aggregation in the lower left quadrant of several small studies with results showing no or only small changes in visual analogue scale.⁵⁹ If publication bias towards only positive studies was present, few studies would lie in this position and small studies would have exaggerated positive outcomes. The slight asymmetry might be partly due to

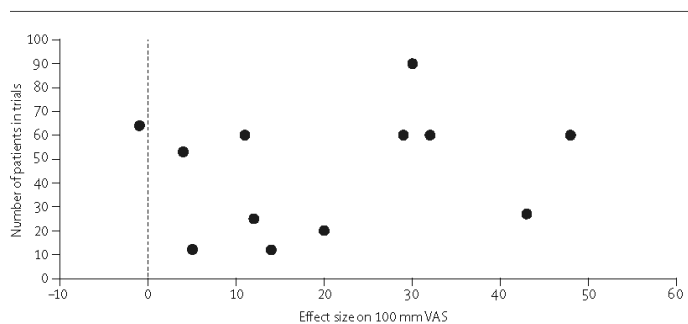


Figure 7. Publication bias plot
 Plot of effect size between placebo and real laser groups within each trial versus their respective sample sizes. Circles show one trial. VAS=visual analogue scale

a negative publication bias, the small number of studies, and because we have included the most reported studies so far.

We subgrouped trials according to a-priori protocol in acute and chronic categories for the statistical analyses. Within these categories, we noted small variations between trials in patient characteristics such as baseline pain, symptom duration, age, and sex, and we did not detect any clinical heterogeneity (data not shown). Laser parameters and application techniques, including treatment protocols, were heterogeneous (table 2). Laser irradiation was applied to an average of 11 points (range 3–25) in the neck. Energy delivered per point ranged from 0.06 to 54.00 J, with irradiation durations of 1–600 s. Patterns of treatment ranged from a one-off treatment to a course of 15 treatments, which were administered daily to twice a week. On average, participants received a course of ten treatments. Visible (632.8 and 670.0 nm) and infrared (820–830, 780, and 904 nm) wavelengths were used at average power outputs ranging from 4 to 450 mW, in pulsed and continuous wave mode.

When trials with significant results in favour of LLLT were subgrouped by wavelength, doses and irradiation times seemed fairly homogeneous within narrow ranges (table 3). We noted a distinct dose-response pattern for each

	Wavelength (nm [mode])	Average output (mW)	J per point	Total time per point (s)	Frequency of treatment	Number of repetitions
Ceccherelli et al (1989) ⁴⁸	904 (p)	~25	1	~40	Three times per week on alternate days for 4 weeks	12
Flöter et al (1990) ⁴⁵	904 (p); 632.8 (cw)	20.5 (9.5 IR; 11.0 red HeNe)	1	600	Twice per week for 3 weeks	6
Taverna et al (1990) ⁵²	904 (p)	24	2	180–300	Six times per week for 2.5 weeks	15
Toya et al (1994) ³⁹	830 (cw)	60	NR	NR	One application only	1
Soriano et al (1996) ³⁹	904 (p)	40	4	100	Five times per week for 2 weeks	10
Laakso et al (1997) ⁴⁹	820 (p)	25	0.06; 0.40	1; 6	Three alternate days per week for 1.5 weeks	5
Laakso et al (1997) ⁴⁹	670 (p)	10	NR	4; 18	Three alternate days per week for 1.5 weeks	5
Özdemir et al (2001) ⁵⁰	830 (cw)	50	0.75	15	Five times per week for 2 weeks	10
Seidel and Uhlemann (2002) ⁵¹	830 (cw)	7	0.42	60	Twice per week for 4 weeks	8
Seidel and Uhlemann (2002) ⁵¹	830 (cw)	30	1.8	60	Twice per week for 4 weeks	8
Hakgüder et al (2003) ⁴⁷	780 (cw)	5	1	196	Five times for week for 2 weeks	10
Chow et al (2004) ⁴²	830 (cw)	300	9	30	Twice per week for 7 weeks	14
Gur et al (2004) ⁴⁶	904 (p)	11.2	0.18– 1.80	180	Five times per week for 2 weeks	10
Ilbuldu et al (2004) ⁴⁸	632.8 (cw)	NR	2	NR	Three alternate days per week for 4 weeks	12
Altan et al (2005) ⁴¹	904 (p)	4	0.5	120	Five times per week for 2 weeks	10
Aigner et al (2006) ⁴⁰	632.8 (cw)	5	0.075	15	Three times per week for 3 weeks	9
Chow et al (2006) ¹³	830 (cw)	300	9	30	Twice per week for 7 weeks	14
Dundar et al (2006) ⁴⁴	830 (cw)	450	54	120	Five times per week for 3 weeks	15

p=pulsed, cw=continuous wave, IR=infrared, HeNe=helium-neon, NR=not reported.

Table 2: Laser parameters and treatment regimen

wavelength for which LLLT is effective within a narrow therapeutic window. For 820–830 nm, mean dose per point ranged from 0.8 to 9.0 J, with irradiation times of 15–180 s. For 904 nm doses, mean dose per point was 0.8–4.2 J, with irradiation times of 100–600 s. Investigators who used doses outside the minimum (0.075 J and 0.06 J)^{40,49} and maximum (54 J)⁴⁴ limits of these ranges did not show any effect of LLLT, lending further support to a dose-dependent response for LLLT in neck pain.

Significant heterogeneity exists in categorical data for improvement from two studies^{39,40} of acute neck pain ($p=0.003$, $\chi^2=8.86$, $I^2=88.7\%$). This finding could be attributable to the low dose per point used in one study.^{40,62} We noted no heterogeneity between trials of chronic neck pain reporting on categorical data ($p=0.37$, $\chi^2=4.31$, $I^2=7.2\%$).

For continuous data from 100 mm visual analogue scale in chronic neck pain, we detected significant heterogeneity across all wavelengths ($p<0.0001$, $\chi^2=137.76$, $I^2=90.6\%$). However, when heterogeneity was addressed separately by wavelengths, most heterogeneity could be accounted for by variations in doses and application procedures. Removal of the study⁴⁴ that used a very high dose from the disability analysis eliminated statistical heterogeneity ($p=0.31$, $\chi^2=3.61$, $I^2=16.9\%$). For pain intensity on 100 mm visual analogue scale for 820–830 nm wavelength, this study caused heterogeneity together with results of a second study⁵⁰ that showed a highly significant effect, without obvious reasons for heterogeneity. After removal of both studies from the 820–830 nm analysis, statistical heterogeneity was eliminated ($p=0.12$, $\chi^2=10.20$, $I^2=41.2\%$), but the overall effect remained similar, with narrower confidence intervals after (22.0 mm [14.5–29.6]) than before (21.6 mm [10.3–32.9]) removal.

For 904 nm wavelength, statistical heterogeneity was evident for analysis of pain intensity on 100 mm visual

analogue scale ($p=0.00001$, $\chi^2=28.37$, $I^2=89.4\%$). The only study in the review using a scanning application procedure in contact with the skin had weaker than average results.⁴⁵ Contrary to other laser application procedures, this method irradiates the target area intermittently. Few studies compare scanning irradiation with stationary irradiation, and most LLLT studies have used a stationary laser beam. Another study using 904 nm wavelength⁴¹ with non-significant results has been criticised for absence of laser testing and

	Mean dose per point (J)	Mean irradiation time per point (s)
632.8 nm ⁶⁸	2	200
780 nm ⁶⁷	1	196
820–830 nm ^{13,42,50,53}	5.9 (3.4)	39.8 (30.3)
904 nm ^{39,41,43,45,46,52}	2.2 (1.6)	238 (184)

Data are mean (SD, when applicable). LLLT=low-level laser therapy.

Table 3: Mean dose per point and irradiation times for wavelengths of LLLT used in studies with statistically significant results

calibration, and the actual dose used remains uncertain.⁶³ Removal of these two trials from the 904 nm analysis of pain reduction on 100 mm visual analogue scale increased the overall effect from 20.6 mm (95% CI 5.2–36.2) to 37.8 mm (25.4–50.1).

50% of trials did not report side-effect data. Side-effects reported included tiredness, nausea, headache, and increased pain, but were mild and, apart from one study in which unusual tiredness occurred more in the laser group than in the placebo group ($p>0.01$),⁴² did not differ from those of placebo.

Discussion

Our results show moderate statistical evidence for efficacy of LLLT in treatment of acute and chronic neck pain in the short and medium term. For chronic pain, we recorded an average reduction in visual analogue scale of 19.86 mm across all studies, which is a clinically important change.^{64,65} Categorical data for global improvement also significantly favoured LLLT. From our analysis, 820–830 nm doses are most effective in the range of 0.8–9.0 J per point, with irradiation times of 15–180 s. At 904 nm, doses are slightly smaller (0.8–4.2 J per point), with slightly longer irradiation times (100–600 s) than at 820–830 nm.

Our findings build on those of previous reviews of LLLT^{28,30} by including non-English language studies, laser acupuncture studies in which local points were treated, and a quantitative analysis. Our search strategy has identified a greater number of studies than have previous reviews, and draws attention to the intrinsic difficulties in searching the topic of LLLT. Specifically, no accepted terminology exists for laser therapy. We have overcome this limitation by using as wide a range of synonyms as possible.

Moreover, many apparently disparate diagnostic terms are applied to patients presenting with neck pain. These terms suggest distinct clinical entities; however, there is strong evidence that a definitive diagnosis of the causes of neck pain is not possible in a clinical setting.^{66,67} By using the term non-specific neck pain, which encompasses many descriptors,³¹ we have addressed the clinical reality that patients presenting with neck pain can have several concurrent sources of pain from joints, muscles, and ligaments.

In addition to aggregating all included studies, irrespective of diagnostic label, we also combined data irrespective

of the intended rationale for treatment, as long as neck muscles and spinal joints were exposed to laser irradiation. Transcutaneous application results in laser-energy scattering and spreading into a three-dimensional volume of tissue, up to 5 cm for infrared laser.⁶⁸ Since the same effect would be achieved with application of laser energy to acupuncture points, we also included data from studies in which local points in the neck were treated as part of the protocol. Evidence suggests that trigger points in the neck coincide with the location of acupuncture points in 70–90% of patients (eg, BL10, GB 20, GB21, and Ah Shi points).^{69,70} Since trigger points and acupuncture points are characterised by tenderness, the treatment effect of laser irradiation to tender points, trigger points, or acupuncture points is likely to be the same. We did not distinguish any differences in subgroup analyses between these techniques. Thus, when treating neck pain with LLLT, irradiation of known trigger points, acupuncture points, tender points, and symptomatic zygapophyseal joints is advisable.

Dose assessment is crucial for interpretation of outcomes of LLLT studies, for which failure to achieve a dose in the recommended range has been identified as a major factor for negative outcomes.⁷¹ The direct relation between positive outcomes of trials with adequate doses of laser irradiation for the appropriate condition has been shown in acute injury and soft-tissue inflammation,²¹ tendinopathies,⁷² rheumatoid arthritis,⁷³ lateral epicondylitis,¹¹ and osteoarthritis.¹⁰

Several crucial parameters of laser devices are needed to assess dose of laser irradiation, but these doses were inconsistently reported in the studies that we reviewed. No study provided all parameters identified as important by the Scientific Committee of the World Association of Laser Therapy.⁷⁴ In neck pain, however, there is little reason to believe that factors other than a plausible anatomical target, dose per point, and irradiation times are essential for efficacy of class 3B lasers (5–500 mW). We had sufficient data relating to each of these components of therapy, when combined with manufacturers' specifications, to identify a dose-response pattern for the number of joules per point and wavelength used and positive outcome. Subgrouping of studies by wavelength and ascending doses reduced apparent heterogeneity in treatment protocols and laser parameters, and showed a dose-response pattern with distinct wavelength-specific therapeutic windows. Most statistical heterogeneity disappeared when we excluded trials with small doses or flaws in treatment procedure from efficacy analyses. Additionally, a very high dose (54 J) of 830 nm LLLT used in one trial did not cause beneficial nor harmful effects.⁴⁴ This finding suggests not only that doses of this magnitude are higher than the therapeutic window, but also that LLLT is safe even if such an overdose is delivered. Frequency of treatments varied from daily to twice a week, raising questions about optimum treatment frequency.

Our analysis suggests that the optimum mean dose per point for 820–830 nm was 5.9 J, with an irradiation time of 39.8 s, and for 904 nm, 2.2 J delivered with an irradiation time of 238 s. We recommend a multicentre, pragmatic trial in an appropriately powered study to test the effectiveness

of parameters of this order, with both pain intensity and functional improvement as outcome measures.

Data from seven trials were available for up to 22 weeks after the end of treatment, suggesting that positive effects were maintained for up to 3 months after treatment ended. Trials of knee osteoarthritis,⁷⁵ tendinopathies,^{61,76} and low back pain reported similar long-lasting effects of LLLT.^{77,78} These results contrast with those for nonsteroidal anti-inflammatory drugs in arthritis and spinal disorders, for which the effect ends rapidly when drug use is discontinued.⁷¹ Reduction of chronic neck pain at the end of treatment of 19.86 mm and at follow-up of 23.44 mm on a visual analogue scale of 100 mm represents clinically significant pain relief.^{64,65} This result compares favourably with those of pharmacological therapies that are widely used in treatment of neck pain, for which investigators have shown no conclusive evidence of benefit.³² Intake of oral analgesic drugs was not systematically reported; however, randomisation within trials would keep the confounding effect of this factor to a minimum.

Half the studies obtained data for side-effects,^{39,42,44–46,49,52,53} with tiredness reported in the laser-treated group in three studies,^{42,46,49} which was significant in one study.⁴² Since LLLT does not generate destructive heat, safety relates mainly to potential eye damage, dependent on class of laser device (classes 1–4), which is defined by analysis of several parameters. Safety glasses are required for classes 3B and 4 to eliminate this risk, and would be required for use in all studies. Systematic reporting of side-effects in future studies would also be recommended to clarify short-term and long-term safety aspects of LLLT.

Mechanisms for LLLT-mediated pain relief are not fully understood. Several investigations exploring the pleiomorphic tissue effects of laser irradiation provide plausible explanations for the clinical effects of LLLT. Anti-inflammatory effects of red and infrared laser irradiation have been shown by reduction in specific inflammatory markers (prostaglandin E₂, interleukin 1 β , tumour necrosis factor α), in in-vitro and in-vivo animal studies and in man.⁷⁹ In animal studies, the anti-inflammatory effects of LLLT are similar to those of pharmacological agents such as celecoxib,⁸⁰ meloxicam,⁸¹ diclofenac,⁸² and dexamethasone.⁸⁰ Chronic neck pain is often associated with osteoarthritis of zygapophyseal joints,⁸³ which is manifested by pain, swelling, and restricted movement as clinical markers of local inflammation. Laser-mediated anti-inflammatory effects at this joint could result in decreased pain and increased mobility. The distance between skin surface and lateral aspect of the facet joint is typically 1.5–3.0 cm without pressure, and less with contact pressure (measured with ultrasonography [unpublished data, JMB]). Since 830 nm and 904 nm lasers penetrate to several centimetres,^{24,84} anti-inflammatory effects at zygapophyseal joints are a plausible mechanism of pain relief.

Another possible mechanism of LLLT action on muscle tissue is a newly discovered ability to reduce oxidative stress and skeletal muscle fatigue with doses similar to those delivering anti-inflammatory effects. This effect has been

reported in an animal study⁸⁵ and in human studies with biceps humeri contractions and different wavelengths.^{86,87} Because muscle fatigue is usually a precursor of muscle pain, and chronic trapezius myalgia is associated with increased electromyograph activity during contractions and impaired microcirculation,⁸⁸ reduction of oxidative stress and muscular fatigue could be beneficial in patients with acute or chronic neck pain.

Inhibition of transmission at the neuromuscular junction could provide yet another mechanism for LLLT effects on myofascial pain and trigger points.^{89,90} Such effects could mediate the clinical finding that LLLT decreases tenderness in trigger points within 15 min of application.⁹¹ Laser-induced neural blockade is a further potential mechanism for the pain-relieving effects of LLLT.^{92,93} Selective inhibition of nerve conduction has been shown in A δ and C fibres, which convey nociceptive stimulation.^{94,95} These inhibitory effects could be mediated by disruption to fast axonal flow in neurons⁹³ or inhibition of neural enzymes.⁹⁶

These tissue effects of laser irradiation might account for the broad range of conditions that are amenable to LLLT treatment. Whether specific treatment protocols are necessary to elicit different biological mechanisms is unknown. Heterogeneity of treatment protocols might be due partly to variation in LLLT parameters and protocols, eliciting different effects. Whatever the mechanism of action, clinical benefits of LLLT occur both when LLLT is used as monotherapy^{13,43} and in the context of a regular exercise and stretching programme.^{46,47} In clinical settings, combination with an exercise programme is probably preferable. The results of LLLT in this review compare favourably with other widely used therapies, and especially with pharmacological interventions, for which evidence is sparse and side-effects are common.^{16,32}

Contributors

RTC participated in the literature search, development of inclusion and exclusion criteria, selection of trials for inclusion in the analysis, methodological assessment, data extraction and interpretation, and writing of the report. MIJ participated in data analysis and interpretation, critically reviewed the report with special expertise in pain management, and contributed to writing of the report. RABL-M participated in data interpretation and analysis, and critically reviewed the report with respect to the mechanism of action of laser, and relevance to neck pain.

JMB participated in development of inclusion and exclusion criteria, translation of non-English language articles, methodological assessment, data analysis and interpretation, writing of the results section of the report, and supervised writing of the report as a whole.

Conflicts of interest

RTC is a member of the World Association for Laser Therapy (WALT), the Australian Medical Acupuncture College, the British Medical Acupuncture Society, the Australian Pain Society, the Australian Medical Association, and the Royal Australian College of General Practitioners. MIJ is a member of the International

Association of the Study of Pain. RABL-M is funded by Fundação de Amparo do Estado de São Paulo (FAPESP, Brazil) and is scientific secretary of WALT, from which he has never received funding, grants, or fees. JMB is a member of the Norwegian Physiotherapy Association, Norwegian Sports Physiotherapy Society, Norwegian Society for Rheumatological and Orthopedic Physiotherapy, and has received research awards and grants from the Norwegian Manual Therapy Association, the Norwegian Neck and Back Congress, the Norwegian Research Council, the Norwegian Fund for Postgraduate Training in Physiotherapy, and the Grieg Foundation. He is also president of WALT, a position for which he has never received funding, grants, or fees.

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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.

Sucu HK, Gelal F. Lumbar disk herniation with contralateral symptoms. *Eur Spine J* 2006 May;15(5):570-4.

The aim of the study is to determine if leg pain can be caused by contralateral lumbar disk herniation and if intervention from only the herniation side would suffice in these patients. Five patients who had lumbar disk herniations with predominantly contralateral symptoms were operated from the side of disk herniation without exploring or decompressing the symptomatic side. Patients were evaluated pre and postoperatively. To our knowledge, this is the first reported series of such patients who were operated only from the herniation side. The possible mechanisms of how contralateral symptoms predominate in these patients are also discussed. In all patients, the shape of disk herniations on imaging studies were quite similar: a broad-based posterior central-paracentral herniated disk with the apex deviated away from the side of the symptoms. The symptoms and signs resolved in the immediate postoperative period. Our data clears that sciatica can be caused by contralateral lumbar disk herniation. When operation is considered, intervention only from the herniation side is sufficient. It is probable that traction rather than direct compression is responsible from the emergence of contralateral symptoms

Comment: A small case series of neurosurgical patients with referred lower limb pain associated with contralateral disc herniation. Operative resection of the disc herniation led to improvement in symptoms in all five patients. The article asserts that this proves a causal link, although this may be overstating the situation somewhat. – *Dr Chris Homan*

Kiter E, Karaboyun T, Tufan AC, Acar K. Immunohistochemical demonstration of nerve endings in iliolumbar ligament. *Spine (Phila Pa 1976)*. 2010 Feb 15;35(4).

Study design. Immunohistochemical study on fresh cadaver specimens.

Objective. Assessment of mechanoreceptor and nociceptor levels and distribution in iliolumbar ligament.

Summary and background data. The function of iliolumbar ligament and its role in low back pain has not been yet fully clarified. Understanding the innervation of this ligament should provide a ground which enables formation of stronger hypotheses.

Methods. Bilateral 30 iliolumbar ligaments of 15 fresh cadavers were included in the study. Morphologic properties were recorded and the ligaments were examined by focusing on 3 main parts: ligament, bone insertions, and tendon body. Assessment of mechanoreceptor and nociceptor levels and their distribution in iliolumbar ligament were performed on the basis of immunohistochemistry using the S-100 antibody specific for nerve tissue.

Results. Iliac wing insertion was found to be the richest region of the ligament in terms of mechanoreceptors and nociceptors. Pacinian (type II) mechanoreceptor was determined to be the most common (66.67%) receptor

followed by Ruffini (type I) (19.67%) mechanoreceptor, whereas free nerve endings (type IV) and Golgi tendon organs (type III) were found to be less common, 10.83% and 2.83%, respectively.

Conclusion. Immunohistochemical staining has shown that iliolumbar ligament had a rich nerve tissue. Those results indicate that ILL plays an important role in proprioceptive coordination of lumbosacral region alongside its known biomechanic support function. Moreover, the presence of type IV nerve endings suggest that the injury of this ligament might contribute to the low back pain.

Comment: A “basic science” study which looks at the type and distribution of nerve receptors within the iliolumbar ligament. The authors find a variety of mechanoreceptors, as well as free nerve ends. The presence of the latter provides support for the iliolumbar ligament as a potential source of nociception. – *Dr Chris Homan*

Chappell AS, Ossanna MJ, Liu-Seifert H, Iyengar S, Skljarevski V, Li LC et al. Duloxetine, a centrally acting analgesic, in the treatment of patients with osteoarthritis knee pain: a 13-week, randomized, placebo-controlled trial. *Pain* 2009 Dec;146(3):253-60.

Pain is a common cause of disability in osteoarthritis. Duloxetine, a serotonin and norepinephrine reuptake inhibitor (SNRI), has demonstrated analgesic effects in diabetic peripheral neuropathy and fibromyalgia. Considering its central mechanism of action, duloxetine may be effective in other pain states with evidence of central sensitization. Herein, we report the results of a 13-week, randomized, double-blind, placebo-controlled trial of duloxetine (60–120 mg/day) versus placebo in the treatment of knee pain in 231 patients meeting clinical and radiographic criteria for osteoarthritis of the knee. Duloxetine was superior to placebo on the primary efficacy measure (weekly mean 24-h pain scores) beginning at Week 1 and continuing through the treatment period ($P = .05$). There was also a significant improvement in the WOMAC physical functioning subscale and several other secondary outcomes. Adverse-event rates did not differ significantly between treatment groups (49.5% for duloxetine 60–120 mg/day, and 40.8% for placebo).

Comment: This study was of Duloxetine 60–120 mg per day vs. placebo for 13 weeks in a multicentre randomized controlled double blind study of 231 patients who suffered pain from osteoarthritis of the knee. Duloxetine is a centrally acting compound that is a serotonin and noradrenaline reuptake inhibitor. This medication caused a statistically significant pain reduction of pain scores beginning in the first week of treatment and sustained through the 13 weeks of therapy.

The pain reduction was due to a direct analgesic effect not antidepressant effect. Not only was there a significant improvement in pain scores but also functional scores. Interestingly the average pain reduction score was greater in those aged over 65 than below. Patients were not required to increase their normal daily activities to exclude the beneficial

effect mild-to-moderate exercise. The drug was beneficial for various durations and severity of pain symptoms.

The greatest barrier to treatment in Australasia is convincing patients that an antidepressant will be beneficial for their pain. – *Dr Peter Jackson*

Mannion AF, Helbling D, Pulkovski N, Sprott H. Spinal segmental stabilisation exercises for chronic low back pain: programme adherence and its influence on clinical outcome. *Eur Spine J* 2009 Dec;18(12):1881-91.

Exercise rehabilitation is one of the few evidence-based treatments for chronic non-specific low back pain (cLBP), but individual success is notoriously variable and may depend on the patient's adherence to the prescribed exercise regime. This prospective study examined factors associated with adherence and the relationship between adherence and outcome after a programme of physiotherapeutic spine stabilisation exercises. A total of 32/37 patients with cLBP completed the study (mean age, 44.0 (SD = 12.3) years; 11/32 (34%) male). Adherence to the 9-week programme was documented as: percent attendance at therapy, percent adherence to daily home exercises (patient diary) and percent commitment to rehabilitation (Sports Injury Rehabilitation Adherence Scale (SIRAS)). The average of these three measures formed a multidimensional adherence index (MAI). Psychological disturbance, fear-avoidance beliefs, catastrophising, exercise self-efficacy and health locus of control were measured by questionnaire; disability in everyday activities was scored with the Roland-Morris disability scale and back pain intensity with a 0–10 graphic rating scale. Overall, adherence to therapy was very good (average MAI score, 85%; median (IQR), 89 (15)%). The only psychological/beliefs variable showing a unique significant association with MAI was exercise self-efficacy ($Rho = 0.36$, $P = 0.045$). Pain intensity and self-rated disability decreased significantly after therapy (each $P < 0.01$). Adherence to home exercises showed a moderate, positive correlation with the reduction in average pain ($Rho = 0.54$, $P = 0.003$) and disability ($Rho = 0.38$, $P = 0.036$); higher MAI scores were associated with greater reductions in average pain ($Rho = 0.48$, $P = 0.008$) and a (n.s.) tendency for greater reductions in disability ($Rho = 0.32$, $P = 0.07$). Neither attendance at therapy nor SIRAS were significantly related to any of the outcomes. The benefits of rehabilitation depended to a large extent on the patient's exercise behaviour outside of the formal physiotherapy sessions. Hence, more effort should be invested in finding ways to improve patients' motivation to take responsibility for the success of their own therapy, perhaps by increasing exercise self-efficacy. Whether the "adherence–outcome" interaction was mediated by improvements in function related to the specific exercises, or by a more "global" effect of the programme, remains to be examined.

Comment: The study of a small group size was carried out in a hospital rheumatology clinic which recruited chronic low back patients from tertiary care providers. The authors' information implies that the treatment was provided at no cost to patients by staff physiotherapists which makes this cohort

different to that of private musculoskeletal practitioners in Australia and probably New Zealand where a fee-for-service is required and thus expectations are more demanding and the therapeutic relationship is in the patients' favour.

The study identified that exercise self-efficacy or high locus of control ("I know I can do it") was the strongest factor in adherence to an exercise program of core strengthening that reduced pain and disability. That this was so would be no great surprise to Australasian musculoskeletal medicine practitioners.

Apparently, attendance rates to the clinic were high. Could this be in part due to free treatment and thus be different sociocultural context to Australasia?

The study also noted that the adherence to home-based exercise was lower than in the clinic, an unremarkable finding. This finding is explained by the "Hawthorne Effect" which states that individuals temporarily change their behaviour in response to being observed.

Interestingly, men had higher scores of home exercise adherence than did women but not attendance at clinic therapy.

The much vaunted fear avoidance behaviour and catastrophising did not appear to adversely affect adherence to exercising.

Another counterintuitive observation was that as compliers became more aerobically fit there was no concomitant improvement in VAS scores.

Overall, though, this study was unrealistic for everyday clinical settings of the readers of this journal and not recommended reading. – *Dr Peter Jackson*

Fritz JM, Hebert J, Koppenhaver S, Parent E. Beyond minimally important change: defining a successful outcome of physical therapy for patients with low back pain. *Spine (Phila Pa 1976)*. 2009 Dec 1;34(25):2803-9.

Study design. Prospective, longitudinal cohort study.

Objective. To examine the validity of a threshold that has been used to define a successful outcome for patients with low back pain (LBP), undergoing nonsurgical rehabilitation based on a 50% improvement on the Modified Oswestry disability index (ODI).

Summary of background data. Making research findings interpretable is a goal of evidence-based practice. One attempt to improve interpretability is reporting treatment results as the percentage of patients achieving a threshold level of improvement within treatment groups along with mean between-group differences. The most recommended threshold is the minimum clinically important difference of the outcome tool. For clinical conditions with favorable natural histories such as LBP, thresholds requiring more than minimal improvement may be preferable for defining success.

Methods. Patients with LBP receiving 4 weeks of physical therapy were examined. The ODI and measures of pain, fear-avoidance beliefs, and demographic characteristics were recorded at baseline and after treatment. A 15-point global rating of change was also completed after treatment. The percent ODI change with treatment was computed and compared between groups known to have different

prognoses. The percent ODI change was compared to the global rating of change to determine the accuracy of various thresholds of success based on the percent ODI change.

Results. A total of 243 subjects (mean age 37.2 +/- 11.4 years, 44.9% female) were included. Mean percent ODI change was 43.1% (+/-40.5), and 109 subjects (44.9%) had a successful outcome ($\geq 50\%$ ODI improvement). As hypothesized, baseline factors with known prognostic importance were less likely to be present in subjects with a successful outcome. The 50% ODI improvement threshold for success had high sensitivity (0.84; 95% CI: 0.79, 0.88) and specificity (0.89; 95% CI: 0.85, 0.93) when compared with success based on the global rating of change. No other percent improvement threshold for the ODI had a higher accuracy than the 50% threshold when compared to the global rating of change.

Conclusion. A threshold of 50% improvement on the ODI may be a valid measure for defining a successful outcome for patients with LBP.

Comment: Jaeschke¹ has defined the minimum clinically important difference (MCID) as "the smallest difference in score in the domain of interest which patients perceive as beneficial and would mandate, in the absence of troublesome side effects and excessive cost, a change in the patient's management". There are a number of ways to calculate this, but most commonly it is derived by comparing the change in a patient's outcome with their global impression of change (GIC). It is usually anchored to those who rate their GIC as somewhere between "a little better" to "moderately better". A recent consensus group of the world's top back pain researchers pegged this at a reduction of 10 points or 30% for the Oswestry Disability Index. They also set this 30% threshold for the MCID for other commonly used pain and disability measures.

However, in this paper, Fritz et al. have set the bar much higher at 50% because this cut off is most accurate for predicting and they reason that it should be larger than the improvement due to time alone. A change of 50% corresponds to a GIC of "quite a bit better," to "a very great deal better", hardly a minimally important clinical change. Perhaps they should acknowledge this benchmark for what it is and rename it a "substantial clinical change". – *Dr Michael Yelland*

1. Jaeschke R, Singer J, Guyatt GH. Measurement of health status. Ascertaining the minimal clinically important difference. *Control Clin Trials* 1989 Dec;10(4):407-15.

Ostelo RWJG, Deyo RA, Stratford P, Waddell G, Croft P, Von Korf M, Bouter LM, de Vet HC. Interpreting Change Scores for Pain and Functional Status in Low Back Pain: Towards International Consensus Regarding Minimal Important Change. *Spine* 2008;33:90-94

Takasaki H, Hall T, Jull G et al. The Influence of Cervical Traction, Compression, and Spurling Test on Cervical Intervertebral Foramen Size. *Spine* 2009.34(16):1658-1662.

Objective. To evaluate the functional changes in the cervical foramen during distraction (DT) of 12kg in neutral position, axial compression (ACT) of 7 kg with neck in neutral, and

the cervical spine extended, rotated, and lateral flexed, in a standardized manner (SST–Spurling Test). There had been no previous studies observing the in vivo MR characteristics of these foramen.

Method. A comparative measurement design investigating the foramen cross-sectional area (FCSA) and foramen shape (ratio of foramen height to FCSA), on the right side from C4 to T1, were measured using 3D sequence of MRI in 23 asymptomatic participants, under 4 different conditions. These were control resting in supine, DT, ACT and SST.

Results. During DT all levels except C7-T1 increased around 120% of control. ($P < 0.05$). During SST, FCSA decreased approx. 70% of control at all levels. ($P < 0.05$). Significant differences ($P < 0.05$) in foramen shape occurred between ACT and SST conditions, but only at C4-C5, and C5-C6 levels. The FCSA did not increase at C7-T1.

Limits of the study include the small number of young asymptomatic subjects, (making comparison with symptomatic elderly subjects impossible), the inability to study adaptive changes of the nerve root and ganglions within the foramen, and, tests were not done in the sitting position. Also the important parameters of the SST (extension, lateral flexion and rotation) were not studied individually.

Comment. The Spurling or SST test is used as a clinical test for cervical nerve root compression within the foramen. The nerve root and ganglion occupy the foramen, with the larger sensory roots above and behind the smaller motor roots.

Cervical radiculopathy affecting these nerve roots (causing sensory changes or weakness respectively) may be expected as a result of the foramen shape and dimensions changing (at critical levels).

This study suggested this to be the case – but at C4-C5 and C5-C6 only. The authors point out that mobility, and the coupled movements of lateral flexion (side bend) and axial rotation is highest at these levels. The forces used in the study may not have been sufficient to test lower levels.

– *Dr Philip Watson*

Bogduk N. On the definitions and physiology of back pain, referred pain, and radicular pain. Topical review. *Pain* 2009; 147(1-3):17-19.

In this review we are reminded that, in spite of the efforts of the International Society for the Study of Pain, confusion persists amongst clinicians using the definitions of back pain, somatic referred pain, radicular pain and radiculopathy. Basic scientists could inherit this confusion when using animal models of back pain.

He reminds us that we have not learnt from studies undertaken as long as 70 years ago (e.g., studies by J H Kellgren in 1938, B Feinstein et al. in 1954).

Nociceptive back pain

Pain that is evoked by noxious stimulation of structures in the lumbar spine. These include muscles of the back, interspinous ligaments, lumbar zygapophysial joints, sacroiliac joints, dura mater, and the posterior surface of

the lumbar intervertebral disc. Noxious stimulation causes dull aching in the back.

Somatic referred pain

In addition to back pain, noxious stimulation to the structures listed above can produce referred pain. This spreads into the lower limbs perceived in regions innervated by nerves other than those innervated at the site of the noxious stimulation. The source of the spinal referred pain lies in the somatic tissues of the lumbar spine, hence the term "somatic referred pain". This distinguishes it from visceral referral pain and radicular pain. It does not involve stimulation of nerve roots. In general, somatic referred pain is perceived in regions that share the same segmental innervation as the source. The proposed mechanism is convergence of nociceptive afferents on second order neurons in the spinal cord that happen also to subtend regions in the lower limb. There are no neurological signs. It is felt as a dull aching, gnawing, and sometimes described as an expanding pressure sensation. It is difficult to localize but when established tends to be fixed in location. The patterns of referral may not be consistent amongst subjects or between studies. They are not dermatomal. The pattern corresponds more with segmental innervation of deep tissue in the lower limb, e.g., muscles or joints. More commonly found in the gluteal region and proximal thigh it may extend as far as the foot.

Radicular pain

Physiologically this is evoked by ectopic discharge emanating from a dorsal root or its ganglion. Radicular pain appears to be a heterospecific discharge in the affected nerves involving A β , A δ and C fibres. Inflammation of the affected nerve seems to be the critical pathophysiological process. The quality of pain is lancinating, shocking or electric, travelling along the posterior length of the lower limb in a band two to three inches wide or less. Mechanically stimulating the nerve roots does not produce radicular pain unless they have been previously inflamed. For compression alone to be painful it seems it must involve the dorsal root ganglion. The term "sciatica" is deemed to be arcane and IASP recommends replacement by the term "radicular pain".

Radiculopathy

This occurs when the conduction is blocked along a spinal nerve or its roots. If sensory fibres, numbness is a symptom and sign, if motor fibres, weakness is the sign. Diminished reflexes occur with either block. The numbness is dermatomal in distribution, the weakness is myotomal. These objective neurological signs define radiculopathy, not pain. However radiculopathy and radicular pain can occur separately or together. It is the occurrence of radicular pain with radiculopathy having a dermatomal distribution of numbness that allows the segment of origin to be determined.

Discussion

Distinguishing the terms "radicular pain" from "somatic referred pain" has significant clinical manifestations. Whilst back pain and somatic referred pain are common,

radicular pain is not, being 12% or less, if defined strictly. Imaging investigations may be justified for radicular pain or radiculopathy but not for somatic referred pain. These are unable to reveal the source of somatic pain in the majority of cases but more importantly carry the risk of false-positive interpretations of the other radiological observations being incriminated instead. Nociceptive back pain and somatic referred pain do not involve nerve injury. Neurological symptoms and signs are therefore unexpected including allodynia. The latter is more often seen with true nerve damage and neuropathy rather than compression or inflammation. Patients with straightforward diagnosis fulfilling the above definitions are less challenging than those with combinations.

Causes of nociceptive back pain may refer into the lower limb causing somatic referred pain. There may also be inflammatory irritations to nerve roots that cause radicular pain. Radiculopathy may also be present if conduction block occurs. It is therefore important to recognize these separate components, as the underlying cause, mechanism, investigation and treatment for each are separate. In particular, distinguishing somatic referred pain and radicular pain would lead to less mismanagement and iatrogenic problems.

Comment. This is a timely article for all those involved in musculoskeletal and spinal medicine. Readers of this journal are encouraged to use these terms as extolled by Professor Nik Bogduk. The term "sciatica" conveys little when on the same day, I saw a patient complaining of "sciatica", it that is, "low back pain to the gluteal fold" as well as a letter received from a spinal surgeon detailing a patient's leg symptoms as "sciatica". It behoves us to heed Professor Bogduk's advice in attempting not only to distinguish these pain patterns so that management can be improved, but also to use these terms correctly in our correspondence with colleagues. – *Dr Philip Watson*

Jensen M P. Topical Review. Hypnosis for chronic pain management: A new hope. *Pain* 2009;146: 235-237. Department of Rehabilitation Medicine, University of Washington School of Medicine, Seattle, USA.

Hypnosis for treating chronic pain has showed renewed interest, possibly due to three reasons.

Firstly, the increasing shifts in knowledge from peripheral to central neurophysiological mechanisms in the experience of pain. This allows clinicians opportunities to use interventions that alter cortical activity, hypnosis being one them.

Secondly, imaging studies show hypnosis alters activity in CNS sites. In one study, suggestions for feeling pain in healthy subjects resulted in (1) reports of pain, and (2) increased activity in many areas of the brain. Both intensity and cerebral activation were stronger following hypnotic induction, than suggestion alone. Other studies whilst confirming these findings also show that reduction in the intensity of pain can also be observed, following suggestion, but is more with hypnotic induction.

Further studies demonstrate that unpleasantness and intensity of pain can be altered similarly, but that each result

in different cortical activation patterns. This suggests hypnotic suggestion can selectively target different cortical areas.

Thirdly, the findings of older controlled trials – the majority having focused on headache – have been confirmed in four recent controlled trials that demonstrated that hypnosis was as or more effective than no or standard treatment in the management of chronic pain. (Conditions investigated were idiopathic orofacial pain, chronic widespread pain, multiple sclerosis and chronic pain, and, spinal cord injury and chronic pain). In addition some of the studies encouraged the participants to practise self-hypnosis, with 62-80% continuing to do so, without or with audio recordings, 60-85% continued using the tapes. Only 22% of spinal cord injury and 40% of multiple sclerosis participants reported >30% decrease in daily pain intensity. Those who practised self-hypnosis found pain relief lasted several hours. This suggest two types of benefits (1) a significant decrease in chronic daily pain that lasted up to a year, and (2) a skill patients can learn even if pain relief is only temporary

Although hypnotic suggestion rarely cures chronic pain, for those in whom pain involves neurophysiological processes, it can produce marked decrease in pain intensity. A subgroup requires long-term maintenance, while some do not respond at all.

Three strategies to enhance the efficacy of hypnosis have been suggested. (1) Using virtual reality hypnosis. The 3D computer-generated environment allows the subject to dissociate from the actual environment. One study has shown its effectiveness in acute painful medical procedures, and another for chronic pain management. As distinct from hypnosis, response to VR is not related to hypnotic ability, and may be automated so that a technician provides the service. This may be practical in the future where clinical hypnotherapists are unavailable. (2) Research has identified EEG pattern changes occur with hypnosis. Biofeedback can also alter EEG activity. It may be possible to train individuals to perform “EEG-biofeedback” exercises, achieving a “hypnotic-like state” just prior to hypnosis. (3) Many patients have reported that they had wished they had learnt self-hypnosis earlier, even before their pain had become chronic. Chronic pain can have long-term detrimental effects on brain structures. Hypnosis may have a role in preventing these negative effects. Studies involving soldiers with war injuries benefit from hypnosis. There may be benefits in limiting the development of chronic pain and of post-traumatic pain disorders in those who are injured.

Hypnotic analgesia may not be for everyone, nor does it provide complete pain relief. Evidence to date suggests its usefulness in patients with chronic pain who would like to pursue this modality in their treatment program, including techniques in self-hypnosis.

More research is needed into hypnosis to develop methods, and subject selection, that will enhance its efficacy. – *Dr Philip Watson*

Baogan Peng, Shuxun Hou, Wenwen Wu, Chunli Zhang, Yi Yang. The pathogenesis and clinical significance of a high-intensity zone (HIZ) of lumbar intervertebral disc

on MR imaging in the patient with discogenic low back pain *Eur Spine J* (2006) 15: 583–587.

Recently, the presence of a high-intensity zone (HIZ) within the posterior annulus seen on T2-weighted MRI has aroused great interest and even controversy among many investigators, particularly on whether the HIZ was closely associated with a concordant pain response on awake discography. The study attempted to interpret the correlation between the presence of the HIZ on MRI and awake discography, as well as its characteristic pathology. Fifty-two patients with low back pain without disc herniation underwent MRI and discography successively. Each disc with HIZ was correlated for an association between the presence of a HIZ and the grading of annular disruption and a concordant pain response. Eleven specimens of lumbar intervertebral discs which contain HIZ in the posterior annulus from 11 patients with discogenic low back pain were harvested for histologic examination to interpret the histologic basis of a nociceptive response during posterior lumbar interbody fusion (PLIF). The study found that in all of 142 discograms in 52 patients, 17 presented HIZ. All 17 discs with HIZ showed painful reproduction and abnormal morphology with annular tears extending either well into or through the outer third of the annulus fibrosus. The consecutive sagittal slices through the HIZ lesion showed that a notable histologic feature of the formation of vascularized granulation tissue in the outer region of the annulus fibrosus. The current study suggests that the HIZ of the lumbar disc on MRI in the patient with low back pain could be considered as a reliable marker of painful outer annular disruption

Comment: This study was conducted in Beijing between April 2000 and August 2003. Its main role seemed to be determining histopathological characteristics of the high intensity zone (HIZ) seen on MRI scans. HIZs in people with persistent low back pain have a high specificity for indicating the affected disc as the source of pain. It is strongly predictive of internal disc disruption with a grade 3 or 4 fissure.

In this study, 52 patients with persistent low back pain underwent MRI and discography. Seventeen patients had HIZ discs, 11 of these undergoing fusion surgery. Those 11 all had positive discography, that is, “reproduction of his or her usual pain response on injection of the contrast”. No information of the pressure needed or the state of the disc above and below was given in this study. Some people would argue strongly this limits the ability to justify the particular disc as being the pain generator. The role of discography has previously been discussed in this journal (*Australas Musculoskeletal Med* 2008;13:69-71, *Australas Musculoskeletal Med* 2009;14:9-21).

In the 11 patients who underwent surgery, disc biopsies were performed. The histological studies indicated that the HIZ in the patients with low back pain represented the ingrowth of the vascularized granulation tissue into the tears in the posterior part of the painful disc. Neovessels were present within the annular tears, presumably with accompanying nerves thus presenting another source of nociception along with the nociceptors already existing in the outer one-third of the annulus. – *Dr Scott Masters*