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Treating inferior heel pain with vitamin D3 dermal cream

An alternative sacroiliac joint injection technique

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Paraspinal nerve injection
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AFMM website: www.afmm.com.au
FIMM website: www.fimm-online.org
Editorial

Professor Nik Bogduk is retiring from clinical practice in Newcastle and also I believe from the Masters in Pain Medicine at the University of Newcastle. He has advanced the understanding of pain and its assessment and management like no other person, and has been truly a giant in our own time. He has written a vast number of original papers and been on the editorial board of anything worth reading. He has certainly advanced the cause of evidence-based medicine worldwide and has led the good fight in Australia. We all owe him a tremendous debt for his wonderful leadership and his enormous energy and productivity. He will be very sorely missed. We hope that he will still join us when he can, and that we will see him in the very near future.

The Australian Association of Musculoskeletal Medicine (AAMM) had its 38th annual scientific meeting with another combined conference with the New Zealand Association of Musculoskeletal Medicine (NZAMSM), the Australasian Faculty of Musculoskeletal Medicine (AFMM) and the Australian College of Physical Medicine (ACPM) 17-19 October 2008, coinciding with the Melbourne Spring Racing Carnival and the Caulfield Cup.

Melbourne was beautiful as always with a short walk to Fitzroy St, St Kilda for restaurants, Chapel St, South Yarra, for restaurants, cafes, and shopping, or a quick tram ride on St Kilda Rd into the city centre to satisfy any need. I bought my wife some more peppermint tea at T2 in the city to stay in the good books.

The weather was so good I thought I had stayed in Queensland. Beautiful one day, perfect the next, a big surprise really. Melbourne was full of that southern multicultural charm we have come to know and love, with barely an underbelly to be seen.

Back pain and sciatica – New paradigms in management was a huge success all round.

Assessment, conservative evidence-based management, interventional procedures and surgery were all the go and were examined in detail.

The lack of efficacy of public pain clinics was highlighted, with some recent audit results presented from the Newcastle experience. There was massive discrepancy in published results regarding the efficacy of functional restoration touted in studies and systematic reviews.\(^1\)\(^2\)

The purported success rate of functional restoration in terms of return to work is 80%, with 95% confidence intervals of 66% to 94%.

In the Newcastle public pain clinic audit, with data collected by a research nurse not involved in the patient’s care, there was assessment before and then immediately after treatment, and at three months and at six months follow up. This involved using a visual analogue scale for pain, the SF36 for function, a patient-specified functional outcome assessment based on four activities of daily living (ADLs) impaired by pain that the patients most dearly wanted restored, a treatment helpfulness questionnaire, and the need for any other care was recorded.

After treatment, median pain scores did NOT improve. Physical functioning, social functioning, and vitality did NOT improve. One patient out of 30 restored their desired ADLs. The majority of patients (25/30) restored NO activity. These outcomes did not improve at the three- or six-month reviews. Four patients previously unemployed returned to work, but six patients previously employed actually ceased work. This was a net gain of unemployed patients, totally at odds with the results reported in the literature. All patients required some form of continuing care from their general practitioner. Anyone working at the coalface would not be at all surprised by these results.

Importantly the sample size in this latest audit was similar to that used in the original studies that promoted functional restoration as the Holy Grail for return to work in patients with chronic low back pain.

Statistically and clinically the outcomes in the audit are completely dissonant with the published claims of 80% success rate for functional restoration programs. The 95% confidence intervals of a success rate of zero are 0-11%, which fails to reach the lower limit 95% confidence interval of 80%, which is 66%. The results are completely incompatible with the literature supporting functional restoration, which is still recommended by various practice guidelines as the preferred, indeed the only, treatment endorsed for low back pain in many centers.

These results warn that what is achieved in the real world of conventional practice using functional restoration may not even approach the purported outcomes established in the literature as the benchmarks. Evidence from research is certainly not translating into standards of practice, and many of us have been very unimpressed by similar observations with our own patients attending these programs. Obviously, citing the evidence is no substitute for auditing the outcomes of individual programs in different public pain clinic settings or private programs used to treat workers’ compensation patients. Musculoskeletal pain medicine practitioners and general practitioners at the coalface should audit their own local public pain clinic services. Professor Bogduk suggested at the conference that it might be hypocritical to follow such guidelines when doing so condemns patients to failure, despite complying with these widely accepted clinical practice guidelines.

The Sebel Hotel, Albert Park, Queens Road, Melbourne, formerly the Carlton Crest, the scene of the historic 1984 AAMM meeting, is well located, and handy to the local offerings.

An AFMM meeting and winery lunch and afternoon on Tuesday 14 October 2008 kicked off at 9.30 am. The bus up to the bush picked up several stragglers on the way and after a fruitful informal meeting and visiting several wineries to taste the fruit of the vine, we had a wonderful lunch at Yeringberg Station Winery and restaurant, one of the oldest wineries in Australia. We went to the very scenic and impressive TarraWarra Museum of Art in Yarra Glen.
that consists of three galleries – two to display the museum’s extensive collection and one to host temporary exhibitions. We stopped for a palate cleanser and platter on the way home at Domaine Chandon. It’s fair to say that a great time was had by all.

An AFMM workshop on the Wednesday featured interventional techniques at Metro Spinal Clinic. This was followed by drinks and sushi very kindly provided at the Vivian residence for those willing and able to attend. The grand piano got a serious workout, with John Malloy and David Vivian providing some unexpected entertainment and brought the dark horses out of hiding. Thanks very much, David Vivian, for all your trouble for this wonderful day.

A working morning for Faculty with a meeting to discuss amalgamation was held at Mirka at Tolono on Fitzroy Street on the Thursday morning where we were made to feel very welcome. Lunch was enjoyed very much by those not golfing in the afternoon. Mirka was described in *The Age* as a professionally run restaurant with a sense of folly and a place in history.

The pre-conference Thursday afternoon featured golf at Royal Melbourne Golf Course for the lucky few brave enough to accept the challenge. Lunch prior to hit-off was included in the Vivian golfing package and was a highlight for the tried and true and were truly tested. It was a great success.

A group of 20 or so of us descended on Mirka again that night after sampling Fitzroy St and its many delights. The evening menu and the international wine list were irresistible, as it turned out. We knew it was a great place after lunch and they were able to accommodate us at *very short notice* and again did not disappoint, with a great night enjoyed by those imbibing.

The main combined conference started on Friday, and ran until lunch time Sunday. The welcome reception was a blast and a few of us went out to dine and enjoy some live music in the vibrant Melbourne cultural scene. A few diehards but mostly accompanying partners attended the Caulfield Cup. Others descended on the National Gallery of Victoria for some art and colour. Most registrants were transfixed by the concurrent workshops at the conference on the Saturday afternoon. There was a nice roll out of physios and other health professionals and interested parties who served to swell the numbers and make the meeting successful from a social and financial perspective.

The conference dinner was held on the Saturday night at the hotel and was a mixed bag before the power went out. But as usual a great time was had by all, with entertainment by Doctors Harding, Nevin, Vivian, Malloy, and Keightley providing much entertainment before we were consigned to emergency lighting by the storm. It was at times amusing and at others stimulating, with diners joining in for some of the singing as appropriate. The company and comradeship was a highlight of the evening as usual. Thanks to all involved.

The AFMM held a retreat in conjunction with the NZ Pain Society Meeting 12-15 March 2008 in Auckland, with involvement of AFMM fellows in presentations, and was a great success. Other meetings were held in Christchurch with a third planned for November 2008 in Auckland.

There are ongoing plans to again apply to the AMC for specialist recognition under the Rudd government which has now been up and running for some 12 months.

The 2020 submission was made prior to the summit but no reply has been received to date.

There is still interest in forming an alliance of the four organizations to enhance organization and to give secretarial support, at least. This is progressing steadily. Joint educational meetings are planned in the future.

Please read Michael Oel’s AAMM President’s Report for the full details of developments. Michael worked tirelessly with Victor Wilk, David Vivian, Steve Jensen, NZAMSM President Peter McKenzie, and the rest of the executive on the Melbourne program, along with Kate Ryall and Dianna Crebbin from DC Conferences. Thanks to all for a job very well done.

NZAMSM President Peter McKenzie provides an update on the latest developments on the NZ scene across the Tasman. Please read his informative report in this edition of the journal.

The Australian Pain Society (APS) 29th Annual Scientific Meeting will be celebrating the 30th Anniversary of the APS at the Sydney Convention and Exhibition Centre Sunday 5 – Wednesday 8 April 2009. The theme will be *The Pain Continuum: Making Pain History*. Three excellent keynote speakers have accepted an invitation to speak – Rolf Detlef-Treede from Germany, Patrick Mantyh from the USA, and PP Chen from Hong Kong. It should be well worth supporting, so please mark your diaries.3

The AAMM Annual Scientific Meeting is planned for July 2009 on the Gold Coast. A local Queensland subcommittee of co-opted insiders is working with your executive and representatives of all four organizations to plan another memorable and informative combined conference.

James Gaida and Jill Cook from Deacon University have provided a great overview of risk factors for tendinopathy, highlighting that there are intrinsic or predisposing factors and extrinsic mostly environmental factors, usually involving repetitive loading. These combine to make an athlete susceptible to injury, and all it takes is an inciting event, a certain manoeuvre, a collision, or a session of hill running, for presentation with clinical tendinopathy.

Jay Govind, Senior Staff Specialist, Occupational and Pain Medicine, Canberra Hospital, examines issues with personal injury claims and their management that are disturbing, to say the least. The existence of chronic axial pain is denied with the publication of the International Association for the Study of Pain’s (IASP) monograph on *Back Pain in the Workplace*. This monograph has defined
chronic low back pain not as a “medical problem,” but as a problem of “activity intolerance”. Paradoxically, the same monograph advocated that the “medical management” should not be “pain contingent” but rather “time contingent”. The taskforce recommended that those who fail to achieve restoration of function and return to work were to be reclassified as “unemployed.” It seems the IASP Task Force is promoting itself as “a self-appointed surrogate gatekeeper to a non-medical system principally ensconced in claims management, cost containment, and cost reduction.”

Victor Wilk expands further with his critique of the value of discography in diagnosing discogenic pain after the great debate in Melbourne with Nik Bogduk. Local tenderness on physical examination can mislead the unwary. Victor still believes internal disc disruption is a real entity and MRI can certainly be of value in the assessment process. Discography, however, has a high false positive rate and the risk of significant morbidity at least. Please read carefully this interesting and insightful review of the evidence, with some practical case study material included.

John Lyftogt has provided a provocative and thought-provoking article about the relative merits of central nervous system sensitization versus peripheral nervous system autonomy in terms of explaining chronic or persistent pain. He argues against the reigning paradigm of “pain management” for people with chronic neuropathic pain when there are well-documented alternatives available that may offer a cure or at least address the pain scores and attempt to reduce them.

In a subsequent paper, John Lyftogt has explored the use of vitamin D3 dermal cream to treat inferior heel pain, also known as plantar fascitis or even policeman’s foot in some parts, with impressive results. John explains that this may be viewed as an example of a peripheral neuropathic pain syndrome due to persistent neuropathic inflammation of the medial calcaneal branches of the tibial nerve. Vitamin D3 may be viewed as a neurosteroid with neuroprotective properties. John highlights the lack of efficacy and potential morbidity such as plantar fascia rupture associated with the use of corticosteroid injection in this setting. It is a condition resistant to most interventions and this is the first report of two patients responding favorably to twice daily applications of vitamin D3 transdermal cream for this difficult-to-treat condition. Be sure to read this important paper examining the role of vitamin D deficiency in contributing to, and offering a means of treating, some forms of neuropathic pain.

Paul Quin, current president of the Australasian Faculty of Musculoskeletal Medicine (AFMM), has produced a wonderfully clear account of his method of sacroiliac joint injection that should improve the reproducibility of the technique while minimizing patient discomfort and exposure time under fluoroscopy.

Mark Bailey has produced a very nice review of meralgia paresthetica as part of his diploma studies in musculoskeletal medicine, and it is presented in this edition of the journal.

Breck McKay has written a very interesting piece on complementary pain management, emphasizing the importance of taking an exhaustive history and conducting a thorough physical examination, and treating the whole patient.

Breck, Scott Masters, and I have written a paper looking at musculoskeletal pain and the role of paraspinal nerve injection. We have found these injections to be very useful as an office procedure since Stefan Blomberg visited our Melbourne annual scientific meeting in 2002 as keynote speaker. We first heard about using local anesthetic and steroid injections to the parasacrococcygeal paraspinal nerve regions from Stefan’s paper, which was published in Spine in 1994. Paraspinal nerve injections are of value for acute spinal pain, and for chronic spinal pain that may be associated with radiculopathy or radicular pain. Sometimes there are elements of somatic pain and radicular pain and/or features of radiculopathy in a patient’s pain presentation. There are limitations in sensitivity and specificity with history, but it is by far the best guide. Physical examination, and imaging, which does not show pain per se, do not fully overcome diagnostic uncertainty, which remains an issue. There can certainly be mixed presentations. Relieving somatic pain components can provide pain relief and relieve patient suffering and help clarify the diagnosis. Any significant radicular symptoms or signs should be assessed and treated in their own right. There can be somatic components with radicular symptoms and signs as the nerve root sleeves are innervated by the sinuvertebral nerve, a recurrent branch of the ventral ramus, as are the intervertebral discs, and these can be a source of somatic pain.

There are some abstracts from the recent literature and comment which I hope readers will find stimulating and informative. Any feedback on these abstracts and comments is welcome. Please address any correspondence to the editor.

Geoff Harding continues as vice-president, master of ceremonies, and entertainer extraordinaire. Thanks very much Geoffrey. He is still the onsite co-ordinator in Australia for the Otago Diploma of Musculoskeletal Medicine.

Margaret Taylor, our treasurer, has again coordinated educational activities. Thanks for all your wonderful efforts, Margi, ably assisted by your assistant Martin.

David Vivian continues as co-editor of the journal and played a very important role in organizing the Melbourne conference, as did Victor Wilk, Steve Jensen, Michael Oei, and the executive and committee. Thanks David, Steve, and Michael, and all who were involved.

Victor Wilk continues his invaluable role as web master, committee member, and master debater. Thanks very much, Victor, for all of your efforts.

I look forward very much to seeing readers in July 2009
From the AAMM President

I hope everyone attending the combined annual scientific conference of musculoskeletal medicine at Albert Park, Melbourne, on 16-19 October 2008 found it as stimulating and interesting as I did. Unfortunately, I was too unwell to appreciate fully both the conference and the social functions.

From all the feedback, the conference program was very well received by all delegates. Most of the delegates were very impressed by the calibre of the speakers and rated them very highly. The conference was a success in uniting all the musculoskeletal organizations together, with the aim of working more closely as a group. Apart from the educational success, it also achieved a financial profit, which is a great bonus. This is why it’s so important that we should continue to have a joint conference with AFMM, ACPM, and NZAMSM. A successful conference like this will not happen without the hard work of the organizing committee, who have spent countless hours in preparing for it. Once again I would like to thank Victor Wilk, Steven Jensen, David Vivian, Geoff Harding, David Roselt, Margaret Taylor, Peter McKenzie, Jennie Wright, and Rod Ayscough for their contributions. Last but not least, I would like to congratulate Kate Ryall from DC Conferences for doing such a wonderful job and making this conference a success.

At the AGM held on 17 October 2008, it was proposed that we form an alliance between the musculoskeletal organizations rather than an amalgamation. This will have to be worked out by the respective presidents.

The next combined conference is likely to be held at the Gold Coast in July 2009, to allow enough time before the Spine in Action conference in Auckland, New Zealand on 26-30 March 2010.

As the year 2008 is drawing to a close, I wish you all a very peaceful Christmas holiday and a very fruitful and happy New Year 2009. Finally I look forward to meeting all old and new AAMM members at the next conference at the Gold Coast.

Michael Oei
This year has seen the continuing development of musculoskeletal medicine. The role of the NZAMSM continues to evolve and change.

The possibility of becoming part of a larger Australasian musculoskeletal organization is being considered, with discussions between the four organizations underway, that is, NZAMSM, AAMM, AFMM, and ACPM.

What is being considered is the formation of an umbrella organization – possibly called the Australasian College of Musculoskeletal Medicine – to which the above four organizations will be affiliated. With time a merger may occur.

The aim is to find commonality and to speak with one voice on all matters pertaining to musculoskeletal medicine.

The role needs to be clearly defined but could include:
1) Unification of the present separate bodies.
2) Research
3) Organizational and secretarial
4) Registrar training
5) Accreditation and re-accreditation of Fellows
6) Political voice
7) More conjoint conferences.
8) Education, including GPs and colleagues.

Hopefully, the formation of such a body will help achieve Australian specialist recognition.

It could be argued that loss of autonomy may occur. NZ already has specialist status so why bother. However, musculoskeletal medicine in NZ is struggling. Membership of the NZAMSM is dropping, mainly through retirement. The vast majority of active members are Fellows and the distinction between NZAMSM business and AFMM business is increasingly blurred. The paramount concern of both the NZ and Australian Medical Councils is the small numbers. A larger unified body would go some way towards addressing these concerns.

Holding conjoint conferences continues to be very successful. This started in Palmerston North last year and continued in Melbourne this year.

Plans for a flagship Spine in Action conference to be held in Auckland in March 2010 are well advanced, with four keynote European speakers confirmed.

Two AFMM retreats have been held this year, the first in association with the NZ Pain Society (colleagues were asked to contribute) and the second in Christchurch. A third is planned for Auckland in November.

Planning for further practical sessions is under active consideration and members will be notified accordingly.

The NZAMSM remains a CME provider for the RNZCGP and various educational activities have been organized by members in their own regions, the largest being the Auckland Roadshow. Musculoskeletal teaching sessions have also been incorporated into the GP registrar training program.

Jim Borowczyk continues as convener of the Otago Musculoskeletal Diploma. He has developed a new paper MSMX 711 Pain Assessment which is now available. Jim is also looking into the possibility of offering a Masters in Pain Medicine through Otago. This would replace Newcastle’s program as Nik Bogduk is looking towards retirement.

Being involved in and offering academic studies of the highest order helps give musculoskeletal medicine status and credibility. It also helps build bridges with other medical colleagues.

Ian Holding has the position of GP liaison at the Department of Orthopaedics and Musculoskeletal Medicine.

Nationalistic differences continue to beset FIMM, with its continuing existence in doubt. NZAMSM is continuing dialogue but we did not send a delegate to their annual meeting this year as we felt the cost could not be justified with the present uncertainties. A written statement has been sent representing our views, that is, the continuation of a global musculoskeletal organization would be worthwhile but the direction it takes would need to have continuing relevance to us.
Introduction

Tendinopathies limit physical activity, are often long-standing, and are difficult to treat. A thorough understanding of the risk factors for tendinopathy is important for several reasons. First, it makes screening for these factors possible among high-risk populations (that is, athletes). Second, prophylactic treatment can be devised, evaluated and applied among the high-risk individuals. Third, knowing which factors increase the risk of tendinopathy gives insight into the underlying mechanisms. And finally, treatment can be directed toward modifying the factors that have precipitated the injury. The underlying assumption is that addressing these underlying factors will improve treatment outcomes and prevent recurrences.

It is only possible to apply the label “risk factor” if measurement takes place prior to injury. Much of our current knowledge about tendinopathy is derived from cross-sectional and case-control studies. As such, we know little about risk factors for tendinopathy – our knowledge is of factors associated with tendinopathy. The exception is situations where a strong case can be made that the variable of interest cannot change as a result of the injury (that is, genetics).

Bahr model

Bahr and Holm have presented a thorough and comprehensive model of musculoskeletal injury risk factors. While the basics of this model are outlined here, interested readers are directed to the original article. Briefly, factors that increase the likelihood of injury are risk factors for that injury. These factors may either occur within the individual (intrinsic) or they may come from the external environment (extrinsic). In the Bahr model, intrinsic factors make an athlete predisposed to injury. In their paper the authors stress that being predisposed to injury is rarely, in itself, enough to lead to an injury. The next step toward injury involves the extrinsic factors – these are laid on top of the intrinsic factors and create an athlete who is now susceptible to injury. The final piece of the puzzle, once the athlete has been “prepared” for injury by being both predisposed and susceptible, is the inciting event. The inciting event may be, for example, a particular manoeuvre, a collision with another athlete, or a session of hill running. The inciting event encompasses not only the injury mechanism but also the environment – firmness of the surface, interaction with other players, and also factors such as the stage of the game at which the injury occurs (that is, 89th minute of a soccer match).

Predisposing factors (intrinsic risk factors)

Age

Generally the incidence of tendinopathy increases with age. While young athletes often develop tendinopathy as a consequence of overload, with increasing age the loading threshold required to incite a tendon injury decreases. This association is more evident for some tendons than others. For example, in the general community rotator cuff problems are relatively rare below the age of 50 but become common from that age onward. Similarly, Achilles tendinopathy incidence peaks during middle age, particularly among men. It is well known that the mechanical properties of many tissues decline from middle age onward. The increased incidence of tendinopathies may be related to altered tissue properties or to cumulative loading history, which increases with each passing year.

Gender

Gender biases are evident for some tendinopathies, while others are equally represented. In males there are much higher incidences of Achilles, patellar and (hip) adductor tendinopathy. Conversely, gluteus medius tendinopathy presents almost exclusively in women. Upper limb tendinopathies such as medial and lateral elbow and rotator cuff are approximately equally represented. These findings may relate to biomechanical differences between men and women – for example, pelvis shape and gluteus medius tendinopathy. More likely, however, is that for the other tendinopathies, it is the hormonal and metabolic differences that influence tendons and affect the incidence of injury.

Genes

Achilles tendinopathy

Knowledge of genetic factors associated with Achilles tendinopathy is much more advanced than for any other
tendon. Seminal work by Jozsa and co-workers demonstrated associations between tendon injury and ABO blood grouping. It is now thought that the association with ABO grouping is due to genetic linkage between the ABO gene [9q34] and other closely associated genes. The South African group have shown that the gene for the alpha chain of collagen type V (COL5A1, [9q34]) and the gene for tenascin-C (TNC, [9q32-q34]) are linked to Achilles tendon injuries.11-14

Rotator cuff tendinopathy

In comparison, only one study has investigated genetic influences for rotator cuff tendinopathy. A 2004 paper showed a 4.65 relative risk (95% CI 2.42 to 8.63) of symptomatic full-thickness rotator cuff tears in full first-degree siblings of probands compared with spousal controls.15 While this paper did not investigate tendinopathy specifically, it could be argued that painful rotator cuff tears are an endpoint of rotator cuff tendinopathy. Further research is required into the influence that genetics plays in rotator cuff tendinopathy, with COL5A1 and TNC being likely starting points.

Race

A recent study covering 6.8 million person-years of military service and 4,451 tendon ruptures showed the effect of race on these injuries. The increased risk of quadriceps tendon rupture among blacks was 2.89 (95% CI 2.42 to 3.44), for the patellar tendon 4.52 (95% CI 3.94 to 5.19) and for the Achilles tendon 3.58 (95% CI 3.31 to 3.88). The authors hypothesized that these differences may be attributable to racial differences in tendon mechanical properties, ABO blood grouping (higher prevalence of type O in blacks) or body weight.

Estrogen

Following early work which showed a spike in the incidence of Achilles tendon problems in women aged 50 and over (that is, menopause), Cook and co-workers investigated the influence of estrogen on tendons. They compared the tendons of post-menopausal women taking hormone replacement (HRT) with those not taking HRT, who were either active (golfers) or non-active. Active women had more tendon abnormalities than did non-active women, but active women on HRT had less tendon abnormality (p = 0.056) and thinner tendons (p < 0.05) than did active women not on HRT. Using these data, the authors argued that estrogen offers protection against tendon injuries and the spike in tendon injuries with menopause is due to the sudden loss of circulating estrogen.

In contrast, Danish researchers argue that estrogen is harmful to tendons. They note that men’s but not women’s patellar tendons respond to prolonged running by increasing in cross-sectional area. Additionally, they note that post-menopausal women have larger Achilles tendon cross-sectional area than do young females. Their explanation for this apparent contradiction is that once the inhibiting effect of estrogen is removed after menopause, tendons have a greater capacity to adapt to loads.17 Thus, although it is acknowledged that estrogen affects tendons, disagreement exists as to whether the effect is positive or negative.

Biomechanics

Achilles tendinopathy

A host of case-control studies have identified biomechanical factors related to Achilles tendinopathy. These include excessive hindfoot movement, which is thought to cause a whipping mechanism in the Achilles tendon. Arch height as well as increased forefoot varus, knee range of motion and electromyographic patterns of the muscles controlling the foot and ankle also differ between individuals with tendinopathy and controls.

Interestingly, the only two longitudinal studies investigating biomechanical risk factors for Achilles tendinopathy have produced conflicting results. Kaufman studied 334 males enrolled in Navy SEAL training and found that reduced dorsiflexion range was the only factor predicting the 30 cases of tendinopathy. In contrast Mahieu followed 69 military recruits during six weeks of basic training and found that increased dorsiflexion range in conjunction with decreased plantarflexion strength was a predictor for the 10 cases of tendinopathy. A key difference between these cohorts is that the Navy SEAL trainees were athletes who increased their training, while Mahieu’s cohort was unaccustomed to physical training. From a clinical standpoint, the first group is probably most informative. Thus, in trained athletes it is important to address limited dorsiflexion while poor control of dorsiflexion may be a factor when untrained individuals suddenly start running long distances. Again, scientific evidence supporting the efficacy of these interventions is lacking, although clinically good results are seen.

Patellar tendinopathy

A host of small case-control studies have investigated differences in lower-limb biomechanics in relation to patellar tendinopathy. They have shown alterations in running, jumping, and landing mechanics both at the knee and the ankle. At times the findings have been contradictory and currently have limited clinical utility.

A longitudinal study of 138 physical education students provides us with some useful insights. Over two years, 19 students developed patellar tendinopathy. Looking back over the data the investigators found that those who went on to develop tendinopathy had lower hamstring and quadriceps flexibility at the beginning of the study. Thus, a good starting point when treating patellar tendinopathy is to examine and address any muscle inflexibility. We await scientific evidence to support the efficacy of this intervention.
Risk Factors for Overuse Tendinopathy

Another factor linked to patellar tendinopathy is a decreased range of ankle dorsiflexion. As with muscle inflexibility of the thigh, limited dorsiflexion may be improved through intervention and training under physiotherapy guidance.

Lateral epicondyle tendinopathy
When performing the backhand stroke in tennis, novice players strike the ball with a greater degree of wrist flexion compared with expert players. Computer models have shown that this strategy involves substantial eccentric contraction of extensor carpi muscles. It is hypothesized that the repeated eccentric contractions overload the extensor origin.

Diabetes
Achilles tendinopathy
Diabetes is consistently associated with tendinopathy throughout the body. In the Achilles tendon, those with diabetes have thicker tendons and more frequently have disorganized collagen on ultrasound. A recent case series showed that diabetes was more common than expected in young males with painful Achilles tendinopathy.

Knee
At the knee, asymptomatic MRI changes affecting the quadriceps tendon are much more common in diabetes. The clinical significance of these changes was not discussed, and is difficult to determine.

Hand
Tendinopathies affecting the hand and wrist are associated with either poor glucose control or overt diabetes. It has been suggested that a tendon disorder of the hand should prompt the physician to examine glucose tolerance. Thus, tendon problems in the hand may be the first sign of incipient diabetes.

Elbow
At the elbow, a study with more than 10,000 participants found that diabetes was associated with both medial (golfer’s elbow) and lateral epicondylar pain (tennis elbow).

Shoulder
In a study investigating factors associated with rotator cuff tendinopathy among more than 4,000 participants, insulin treatment for diabetes was associated with a relative risk of 12.8 (95% CI 2.6 to 62.7) for tendinopathy among men.

Treatment
Finally, a diagnosis of diabetes is a strong predictor of a poor outcome following the same treatment is a high HbA1c (poorly controlled glucose). The lower rates of success for these interventions in diabetes should be considered when recommending treatments to patients.

Mechanisms

Diabetes
The underlying mechanisms that increase the risk of tendinopathy in diabetes are not well understood. Most likely, elevated glucose levels are responsible through their ability to cause non-enzymatic cross-linking of collagen. This non-enzymatic glycosylation increases the stiffness of tendon and other connective tissue, potentially explaining the increased injury risk. Another possibility is that the metabolic environment associated with diabetes (increased proinflammatory cytokine expression) alters the protein expression of the cells (tenocytes) that build and maintain the tendon extracellular matrix.

Adiposity
Adiposity is a factor that has only recently come to attention as a potential risk factor for tendinopathies. Recent work by both our group based in Melbourne and others internationally highlights the importance of stored fat in relation to tendinopathy. For example, among elite male volleyball players waist circumference (a proxy for abdominal adipose tissue) was the only factor able to discriminate those with patellar tendon abnormalities from those with normal tendons. This factor was superior to body weight, training schedule, and a host of other factors considered. Similarly, high BMI is a very strong risk factor for pathology affecting the rotator cuff tendons and the medial epicondylar attachment of elbow tendons. We have recently outlined the reasons a mechanical hypothesis is insufficient in explaining these findings. Briefly, as only the lower limb tendons are weight bearing, the association between adiposity and tendinopathy in both the upper and lower limbs supports, an alternative, systemic hypothesis. Interested readers are directed to a recent paper discussing this matter.

Susceptible factors (extrinsic risk factors)

Load
The most widely recognized factor that makes individuals susceptible to tendinopathy is repetitive loading. This is why high rates of patellar tendinopathy are seen in...
volleyball players, and Achilles tendinopathy in middle distance runners. It also appears as if training surfaces are important, with much higher rates of patellar tendinopathy among volleyball players who train on asphalt compared with sprung wooden floors. Similarly, runners who perform running sessions in sand, or frequently use hill running for training report higher rates of Achilles tendinopathy. By the same token, tennis elbow is common in tennis; golfer’s elbow common in golfers; adductor tendinopathy common in AFL players; rotator cuff tendinopathy common in pitchers and swimmers.

Also, workers who are exposed to heavy manual labour, repetitive movements, particularly above the level of the shoulder are at high risk of upper limb tendinopathies. Similarly, awkward shoulder postures and vibration appear to be detrimental.

### Tobacco

Tobacco exposure both as cigarettes and snuff/snus seems to play an important role in increasing the risk of tendinopathies. This has been reported for the shoulder, lower limb injuries, wrist and hand symptoms, and for both medial and lateral epicondylar pain.

### Medication

Exposure to certain medications also appears to increase the risk of tendinopathies and associated conditions. In some circumstances it may be appropriate to switch to an alternative medication, while in other cases the medication must be continued and the tendon problem managed as far as possible. For example, if fluoroquinolone antibiotics induce Achilles tendinopathy a different class of medication may provide similar antibiotic cover while avoiding further exacerbation of the tendon problem. Similarly, if tendinopathy is associated with initiation or change in statin medication, avoiding a rapid increase in dosage or changing the class of statin may be appropriate.

Change is inappropriate if patients are being treated with a metalloproteinase inhibitor (such as Marimastat) for inoperable cancers. In these cases the development of musculoskeletal side effects such as frozen shoulder and Dupuytren’s contracture are associated with improved survival.

### Conclusion

It is clear from the evidence presented that our knowledge of true risk factors for overuse tendinopathy is limited. In many cases, we cannot be certain whether the factors cross-sectionally associated with tendinopathy were present prior to injury or whether they developed after the injury. Carefully controlled longitudinal studies are desperately required to address this knowledge shortfall.

A number of the listed factors are non-modifiable (that is, age, gender); however, knowledge of these factors will allow the clinician to identify cases that don’t fit the usual presentation. These atypical cases may prompt the search for underlying diseases such as seronegative arthropathies or other rheumatologic disorders.

Conversely, knowledge of modifiable factors allows the clinician to educate their patients and to encourage them to engage in risk factor reduction. This knowledge also directs the clinician when interviewing the patient with the view to understanding the factors that have led to the injury. And finally, when modifiable risk factors are identified during the examination, these factors can be addressed either by the treating doctor or by appropriate referral.

### References


Risk Factors for Overuse Tendinopathy


Risk Factors for Overuse Tendinopathy


Personal Injury Claims: Quo Vadis?

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takeholders who have a vested interest in the management of personal injury claims and more so in the management of chronic axial pain coincidentally demonstrated an acute attitudinal change with the publication of the International Association for the Study of Pain’s (IASP) monograph on Back Pain in the Workplace. By denying the reality of chronic axial pain, this monograph defined chronic low back pain not as a “medical problem,” but as a problem of “activity intolerance”.1 Yet paradoxically, the same monograph advocated that the “medical management” should not be “pain contingent” but rather “time contingent.” The rationale for this was not articulated, and evidentiary basic science – as one would expect in cardiorespiratory and other medical disorders – was conspicuously absent. The taskforce further recommended that those who fail to achieve restoration of function and return to work were to be reclassified as “unemployed.” Despite its irony, it is a sad commentary for the premier scientific body in pain medicine to deny the existence of chronic axial pain, to encourage unemployment and its psychosocial upheavals and for this Task Force to promote itself as a self-appointed surrogate gatekeeper to a non-medical system principally ensconced in claims management, cost containment, and cost reduction.

Yet the medical profession cannot totally abrogate the need for cost containment. In 1992 the total cost of occupational injuries in California was at least A$20 billion2 and by 2000 Washington State outlaid $472.4 million for medical care only.3 Commensurate with the rapid expansion of Health Maintenance Organizations (HMOs), physicians so affiliated had a greater tendency to classify claims as compensable under workers’ compensation than did other physicians.4 Levied too, is the indictment of self-referral by physicians outside the bounds of “medical necessity”. Yet paradoxically, the same monograph advocated that the “medical management” should not be “pain contingent” but rather “time contingent.” The rationale for this was not articulated, and evidentiary basic science – as one would expect in cardiorespiratory and other medical disorders – was conspicuously absent. The taskforce further recommended that those who fail to achieve restoration of function and return to work were to be reclassified as “unemployed.” Despite its irony, it is a sad commentary for the premier scientific body in pain medicine to deny the existence of chronic axial pain, to encourage unemployment and its psychosocial upheavals and for this Task Force to promote itself as a self-appointed surrogate gatekeeper to a non-medical system principally ensconced in claims management, cost containment, and cost reduction.

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In most Western societies, changes in Workers’ Compensation legislation induced an inflexible system of case management. Defined as a set of “logical steps and a process of interaction within a service network which assures that a client receives needed services in a supportive effective efficient and cost-effective manner”6 case management is primarily pre-occupied with cost containment, including medical care and returning the injured worker to work – be it pre-injury, alternative or even notional work that would expedite case-closure.7 Case managers have multiple roles. Within the ambit of a single claim, they serve multiple stakeholders simultaneously. In their “administrative” role, case managers process claims, pay wages and bills. As “watchdogs” they monitor health care services and the “medical necessity” thereof. In a “supportive” role they liaise and co-ordinate the passage of the claim with the legal fraternity, health care and rehabilitation providers, the employer and the worker. Throughout, case managers are accountable either to the insurer or to government instrumentalities that administer the relevant Act.

Nomogenic disorder is a newer kind of impairment and disability created by such a rigid and inflexible system.8 Analogous to an iatrogenic disorder, nomogenic disorder describes those psychopathologic disorders in which the law and its application play an etiologic role.8 This is further exacerbated by unique pressure placed on health care providers, such that their traditional role as a healer has been transformed into that of a “medical police”. The process also undermines the quality of health care permissible, devalues the (treating) doctor-patient relationship and hinders access to unbiased clinical assessment.9 Many claimants say they have experienced a loss of esteem, self-worth and dignity: a traumatic separation from the workplace and an exposure to an overwhelming range of health care professionals. Inappropriate and ineffectual treatment is said to prolong absence from work, causing financial loss, anger and stress anxiety, whilst adversarial medical consultations could lead to disenfranchisement.10

With its newly found epiphany, some stakeholders including insurers, government instrumentalities and appointed members of the health care profession dictate the management of work related injuries according to “evidence based guidelines”, but fail (or refuse) to appreciate that inherent within such mantra is:

- a failure to define the level or hierarchy of evidence, or that,
- evidence can be conjured to suit self-interests;
- an absence of validation of most published guidelines, and,
- problems associated with insurance-funded research.11

Published guidelines rely heavily on randomized controlled trials for their conclusions, irrespective of quality and validity, and what is not universally known is that policy makers legitimized randomized controlled trials (RCTs) so that the medical profession could be regulated.12 Au-
authors of putative “evidence based guidelines” have carefully omitted “integrating individual clinical expertise ... and ... compassionate use of individual patient’s predica-
ments, rights and preferences” in making decisions about patient care. By a process of selective conceptual-
ization, third party funders and legislators have hijacked the principles of evidence based medicine, principally and solely to contain costs, as aptly exemplified in a recent publication by the Bone and Joint Decade Task Force on Neck Pain.14

Insurers do this by refusing to reimburse the costs of treatment that are not “medically necessary” and by refusing to pay more for the treatment of a particular problem than the predetermined average cost of treating that problem within a particular patient population.15 Rather than judiciously incorporating the current best evidence, pre- eminent in the postulations of guideline authors is the rationing—and not rationalizing16,17 of healthcare18,19 To be trustworthy and accepting, authors of guidelines must demonstrate the legitimacy of their process through clinical governance.20

No system of injury compensation can function without the expert evidence of physicians, irrespective of qualifications.21 Under most workers’ compensation systems, a caveat to reimbursement is that treatment must be appropriate, reasonable and medically necessary. The concept of “medical necessity” actually functions as a principle of allocation and gate-keeping22—because insurance companies fear that funds will be siphoned into a bottomless pit. Thus, pre-emptively, insurers and third party payers rely on “utilization management/review” boards to reduce the consumption of “unnecessary and inappropriate” healthcare services.23 Traditionally, insurers’ decisions are based on the idea that without the service harm will come to the patient and with that service potentially beneficial outcome will result. Setting such threshold is illusionary because patients and providers, insurers and courts have different values and objectives.24 Whilst clinical facts may determine “medical necessity”, ethically and morally, physicians so engaged must demonstrate transparency about the grounds for decisions and procedures for revising the decisions in light of challenges to assure “accountability for reasonableness”.25

In many Australian jurisdictions, such decision-making process is delegated to “approved medical specialists” and in at least one Australian state it appears that medical expertise has been conferred by Parliamentary decree in preference to a university degree. In this way the “hired-gun”26 is legitimized. However, a trend to hold expert witnesses liable for their professional errors is gaining momentum.27

Conclusion

In the USA, approximately 48 million patients suffer from chronic pain and they suffer needlessly, because although the technology is available, 90% cannot access it.10 What is at stake here is the erosion of the real standard of care.28 Recognizing a constitutional right to adequate pain relief has the potential to remedy any inequalities.29 In a throw-away society, when bad evidence happens to good treatments the “injured worker suffers the same fate as the plastic cup”.

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Personal Injury Claims: Quo Vadis?


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Suggested readings


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Introduction

In the November 2007 issue of Australasian Musculoskeletal Medicine we looked at the correlation between lumbar disc morphological changes and pain in the general population. We know that there are certain changes in the disc seen on MRI that do correlate with pain, but are nonetheless present in significant proportions of the normal population. We also examined some of the key evidence questioning the validity of provocative discography as the gold standard in diagnosing discogenic pain.

Since then we have had the “great debate” on discography (between Professor Nikolai Bogduk and me) at the annual scientific meeting of Australasian Musculoskeletal Medicine in Melbourne in October 2008. The debate continues and the jury is still out as to whether there is a clinical role for discography. In this article I will examine the principle of provoking pain to make a specific diagnosis.

Finding the nociceptor

There are a few aspects of musculoskeletal medicine practice that differentiate it from mainstream medicine. The taking of a detailed pain history including the assessment of biomechanical factors leading to pain and assessing the quality of the pain are important. We also put a lot of emphasis on trying to find the source of pain by performing a more careful detailed examination looking for abnormal mobility and tenderness. Over the years, these tender spots near the spine have been described variously as paravertebral muscle spasm, facet joint tenderness, zones of irritation, tenosynovitis and enthesopathies, myofascial trigger points, interspinous ligament pain, iliolumbar ligament strain and, more recently, nerve entrapment (greater occipital nerves in the upper neck, cluneal nerves over the iliac crests).

The diagnosis in each of these examples relies on finding the tender spot and reproducing the patient’s pain. The site and quality of the pain and the texture of the underlying tissues lead to the hypothesis of a likely nociceptor. The reproduction of the exact same pain is said to be proof of the diagnosis. However, what I have discovered over the years (and argued recently) is that provoking pain alone is not specific enough to make a diagnosis. Let me explain by way of example in a real patient I have seen recently – Mr GD, aged 48. Here are my actual notes of consultations and comments in italics below:

01/10/2008 (Wednesday) 9:16 am with Dr Victor Wilk (VW)
Medical initial consultation

HISTORY: WC injury 7/7/08 official date, but gradual onset of low back and left leg pains at work. No specific trauma though, and no time off work. Some tingling was felt in the left foot, dull ache throughout whole leg. No real shooting/electric shock pains in the left leg. Pains bad at night – can’t lie on his back at night. Some back pain in addition to leg pains, but leg pain is worse. Physiotherapy was not making any difference. Pain was there most of time – worse bending forwards, unable to lift anything heavy.

WORK: His job is systems operator – administration job managing all computers in the store and checking pricing. Sitting on and off.

PAST HISTORY: WC injury 12 years ago at Woolworths when doing lots of heavy lifting – order writer at the time – on the day of injury he was squatting down doing orders and waddling along the isles when he stood up and twisted and felt sharp pains across the back worse on the right side, but with no leg pains. He was off work for one month, recovering with lots of physio over 6 months. Occasional pains since.

FAMILY HISTORY: Nil relevant

GENERAL HEALTH: Weight ~ 100 kg – the same over the last 8 years

HOBBIES: Likes bush walking – but he can’t get far, walks the dogs, 3 sons – 14, 15, and 17

TREATMENTS: Traction, interferential (IF), massage, home exercises

MEDICATION: Nil

INVESTIGATION: CT scan lumbar 29/8/08 – moderate left postero-lateral disc prolapse L5/S1

EXAM: Physical: Height: 178 cm; Weight: 110 kg; BMI: 34.72; Abdominal obesity. Walks with slight limp, F=60, E=10, SB = 15 bilaterally, HIPS ok, SLR supine left 40°,
Discography - Making the Diagnosis by Provocative Means

right 60°, slump test positive for left calf pain and tingling in foot on the left side only. Neurology – reflexes ok, power ok, sensation ok, palpation of back – no spasm, but was tender over the left L5 region.

ASSESSMENT: Most likely moderate left L5/S1 prolapse with some radicular pain features, but no radiculopathy.

PLAN: Recommended epidural injection of local anesthetic and corticosteroid.

Interesting points to note: firstly, the slightly atypical history of radicular pain – the leg pain is dull/aching in quality, but on balance with the story of leg pain radiating to the foot, leg pain greater than back pain and associated pins and needles in the foot, it sounds like it may be radicular. The examination findings confirm a moderate restriction of straight leg raise worse on the affected side, with some increased distal leg pain on slumping. Neurological tests reveal no radiculopathy, but there is confirmation on the CT scan of an appropriate disc prolapse. I thought it would be reasonable to proceed to an epidural corticosteroid injection.

09/10/2008 (Thursday) 10:17am with Dr VW
Left L5/S1 Transforaminal epidural injection under fluoroscopy

PROCEDURE: Left L5/S1 Transforaminal epidural injection under fluoroscopy (L5 nerve root) 22 g x 127 mm needle to depth 115 mm. Position checked on AP and lateral films. Some nausea was felt during positioning of the needle, but this settled. Contrast Ultravist 240 showed good position with good epidural spread. 1 ml 1% xylocaine + 1 ml Celestone Chronodose was injected slowly. Pain was 7 out of 10 before injection in the left buttock and leg. After injection the pain eased to level 0 in the left buttock but with only slight relief in left calf. Mr GD was asked to keep a pain diary, and see Dr VW in 2 weeks.

Straightforward epidural injection but his immediate pain response is also atypical – one would expect ? hope for complete relief of the calf pain as well as the buttock pain. One interpretation may be that some of the calf pain is somatic referred pain from the disc itself which we cannot anesthetize or from some other structure.

22/10/2008 (Wednesday) 8:04am with Dr VW, for 25 minutes
Medical review

HISTORY: Was really good for one week post epidural – then pain recurred last 3 days – today almost as bad as before. The tingling present in the foot was now back again.

EXAMINATION: Walking with a mild limp, F=60, E=15, SB = 20 bilat – better than before injection, Hips ok, SLR supine left 50° now, palpation revealed he was quite tender over the left Post Superior Iliac Spine (PSIS), reproducing the ache into the left leg.

PLAN: Injection of a total of 3 ml 1% xylocaine with adrenaline with a 25 g 38 mm needle to the left PSIS 80 mm needle to a depth of 40 mm. This reproduced the exact leg pain on contacting bone – pre-injection pain was level 5 out of 10, and increased to 10 with tingling in the left foot. Post-injection pain after 5 mins was slightly worse. The left leg pain increased to 6 out of 10. Mr GD was asked to keep a pain diary (Figure 1).

Figure 1. Pain chart for injection PSIS with 1% xylocaine.

<table>
<thead>
<tr>
<th>Time</th>
<th>Left buttock</th>
<th>Left calf</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-injection</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>On bone contact</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>5 mins after</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>15 mins</td>
<td>8</td>
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<td>30 mins</td>
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<tr>
<td>90 mins</td>
<td>10</td>
<td>8</td>
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<tr>
<td>2 hours</td>
<td>10</td>
<td>8</td>
</tr>
</tbody>
</table>

When I reviewed him 2 weeks after the epidural injection Mr GD stated that he had good pain relief for one week suggesting the diagnosis of disc prolapse was correct – but the effects were not long lasting. The examination revealed more prominent tenderness at the left PSIS – about 4 cm lateral to the midline at the level of L5/S1 approximately. This area is commonly tender in many patients presenting with low back pain. As his pain was somewhat atypical I thought it may be worth trying a local injection there. In this case when the needle contacted bone it elicited a severe sharp pain response with reproduction of the area of leg pain to a level of 10 out of 10 and bringing on the tingling in the foot. I am quite sure the needle tip was nowhere near any nerve roots having performed an epidural one week previously.

On the basis of provocation testing one would have to rate this latter PSIS injection as being very specific for his pain, and yet the local anesthetic failed to block the pain. Why is this so?

The explanation for this common phenomenon comes from theories of convergence of neural pathways in the dorsal horn. The same pathways that give rise to referred pain patterns are also responsible for convergence of pain from nociceptive sources. In acute inflammation, peripheral sensitization occurs where there is an increase in release of inflammatory mediators and an enlarged field of hyperalgesia results. Typically with any moderate to severe pain there is also likely to be an element of central sensitization where the modulating neurones deep in the dorsal horn become more sensitive to peripheral stimuli. These two phenomena often combine in low back pain syndromes. A regional hyperalgesia results. Aage Indahl published his PhD in 1999 showing the interaction be-
between the disc, zygapophysial joints and the paraspinal muscles and how they interplay to cause regional functional disturbance. It is still very relevant and worth a read.\(^1\)\(^2\)

In some patients, pain can be very localized to one segment and there may be very localized superficial tenderness, some reactive protective muscle tightness and a possible deeper joint/disc problem. In other cases pain may be more widespread. Anesthetizing the pain is the best way to confirm a diagnosis – it is the only true gold standard. However, even this gold standard has to take into account the placebo response.

**Further research**

This lack of specificity of discography has led one researcher, Dr Richard Derby in the USA, to investigate what proportion of provocative positive discs may be responsive to local anesthetic injected into the disc.\(^3\) Of 39 subjects with positive discograms, only 51% gained modest relief with injection of 4% lidocaine into the nucleus pulposis. Criteria for relief included a reduction of pain > 2 on a 0 to 10 pain scale on sitting. However none of the subjects achieved improvement in other functional scores. Some would argue that they didn’t gain pain relief because the anulus may have been largely intact and the local agent couldn’t reach the outer anular nociceptive nerves. This may well be the case – we just don’t know. However, most of these discs had significant grade 3 or above anular fissures and one would expect some anesthetic infiltration into the outer anular tears where the pain is thought to be generated.

I believe this study adds weight to the evidence of a large false positive response to discography. If we use the strict criteria of 90-100% relief of pain used to diagnose zygapophysial joint pain, false positives in the study above approach 100%.

**Conclusion**

As I have stated previously, I am a believer in the concept of internal disc disruption and primary discogenic pain. Some cases may not yet have developed MRI changes – these cannot be diagnosed at present. However, in the majority of cases, discogenic pain will show some morphological changes on MRI scans. There may be some emerging therapies that will help these conditions. I would advocate that any new therapies be trialled on patients with a specific condition diagnosable on MRI scan. In the research setting all patients with the condition would receive the active or control therapy and all could also receive discography. It is only in this way that we can discover whether discography has any extra predictive value over and above MRI. At the present time all we know is that if we see something abnormal in the disc on MRI, then that disc is more like to be painful when provoked with a needle.

We must continue to keep an open mind in relation to the interpretation of findings on physical examination. I have had the odd great success in alleviating pain of several years’ duration by the simple injection of local anesthetic into a tight band of tender muscle. However, I hesitate to make the diagnosis of myofascial trigger points in every patient I see with local muscle tenderness and spasm. I believe it is important to carefully examine for alterations in local tissue texture and to try to reproduce the patient’s pain by palpation or movement. We just need to keep in mind the effects of regional hyperalgesia and the possible differential diagnoses.

In my opinion the only case that can be made for discography in clinical practice is where the surgeon has made the decision to operate irrespective of the discography findings and that they are testing adjacent levels to help plan the extent of surgery. The argument for using discography to avoid surgery does not hold in the real world. A significant number of patients suffer increased pain for up to one year post discography. I have had two patients aggravated by the procedure to the degree that they ended up having fusion operations they didn’t need and they continue to suffer with long-term disability.

**References**

Pain Conundrums: Which Hypothesis?  
Central Nervous System Sensitization versus Peripheral Nervous System Autonomy

Dr John Lyftogt, Christchurch, New Zealand

The two hypotheses, central nervous system (CNS) sensitization and peripheral nervous system (PNS) autonomy are at first glance irreconcilable. CNS sensitization is the reigning paradigm in mainstream pain medicine. It is shared by most members of the medical scientific community.

PNS autonomy is not recognized despite convincing scientific and clinical evidence. It has yet to establish a paradigm status and would require a scientific revolution or paradigm shift for it to gain acceptance, as predicted by Thomas Kuhn.1

However, a growing body of scientific evidence, old and new, is addressing anomalies in the CNS sensitization concepts that have been ignored or dismissed. Historically, ideas culminating in the CNS sensitization theory started with the Melzack-Wall article in 1965 on the Gate Control Theory, which “emphasized the mechanisms of the CNS controlling the perception of a noxious stimulus and thus integrated afferent, upstream processes with downstream modulation from the brain”.3 The current definition of pain endorsed by the International Association for the Study of Pain (IASP) is a logical outcome of this theory: “Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”.4

According to Howard L Fields,5 “The pain pathway we are all familiar with normally begins with tissue damage, inflammation, and traumatic injury. It starts with impulses out in the periphery. These are propagated to the spinal cord, cross in the contralateral spinothalamic tract, delivered to the thalamus and then widely distributed to the cortex. Injury to a peripheral nerve somehow causes an increase of activity in this pathway somewhere along this pathway.” He goes on to say that this increase of activity could come from either greater activity in the nociceptors in the periphery or by removal of some sort of inhibition in the CNS releasing the transmission neurons from inhibition. Further, he argues that these neurons are now spontaneously active and produce a pain signal. Animal studies have shown that these nerves themselves become pain generators. It is also known that damage to selectively large myelinated fibers will cause central fibers to fire more to any peripheral stimulus. There is also evidence that damage to peripheral nerves results in spontaneous activity in second-order neurons. Injury to peripheral nerves can also possibly cause “rewiring” in the spinal cord where fibers from the periphery normally responding to light touch producing the sensation of vibration or tickle now connect to a different second-order cell in the spinal cord. Their activity now may produce a burning pain instead of a tickle. In an article in the Lancet, Loeser and Melzack6 conclude that “The brain contains widely distributed neural networks that create an image of self through genetic programmes and memories of past experience. Afferent inputs act on this neuromatrix and produce output patterns that lead to the report of pain. Stress can change the interactions between the neuromatrix and peripheral stimuli, as can learned experiences and expectation.”

As a consequence of the above concepts, the memory of the injury leading to chronic pain and CNS sensitization are now generally considered useful in the “management” of chronic pain by increasingly complex multidisciplinary teams.

One anomaly in the CNS sensitization theory is its impotence in curing chronic pain, despite more than 40 years of millions of dollars of research unraveling the mysteries of the CNS. Loeser and Melzack6 admit to this when they conclude their article in 1999 by saying, “In both clinical and basic research, we are rapidly gaining useful information that will lead to more effective care for those who suffer pain”. In the introduction to the third edition of The Textbook of Pain, Wall and Melzack6 express the hope that in their next edition they will be able to announce to the world a cure for chronic pain. This inability to cure chronic pain has led mainstream medicine to build up an immutable conviction that any health professional claiming the opposite is deluding himself or herself and his or her patients. Popularized books on the management of chronic pain by reputable and leading specialists thus warn patients seeking a cure for their chronic pain to be wary because offering a cure is akin to deception.7 A further anomaly is the belief there is no demonstrated pathology in the PNS satisfactorily explaining chronic pain. This view is detailed in the introduction to Wall and Melzack’s Textbook of Pain8 in 1997.

The last 40 years of basic research into the PNS has shown the opposite. Since Jancso et al. published their Direct Evidence for Neurogenic Inflammation and its Prevention by Denervation and by Pretreatment with Capsaicin in 1967 a growing number of scientists have systematically unraveled the PNS response to injury. Bennett in 19999 summarized this: “Painful peripheral neuropathies begin with nerve injury caused by disease or trauma. This injury will result in an inflammatory reaction, a neuritis that will mobilize the immune system.” Subsequent changes may result in more slowly developing mechanisms of
abnormal pain that underlie the chronic phase of painful neuropathy.

Douglas Zochodne from the Neuroscience Research Group in Calgary examined the role of the microenvironment and microcirculation of the injured and regenerating peripheral nerve trunk and concluded in his seminal paper on peripheral nerve response to injury: “Better understanding of these and other events in injured nerve trunks is needed to help solve the two cardinal problems of peripheral nerve injuries:

1) functional disability from impaired regeneration, and
2) the development of disabling neuropathic pain.”

Peripheral nerves respond to injury in a unique way. Instead of ischemia, peripheral nerves develop increased endoneurial blood flow. Trauma-induced ischemia in all other tissues may lead to cell death and release of arachidonic acid, stimulating COX I, COX II, and 5-LOX pathways upregulating prostaglandin production and tissue inflammation. Peripheral nerve injury leads to “dumping” of calcitonin gene related peptide (CGRP), substance P (SP) and nitric oxide (NO) from nerves, the extensive innervations of the connective tissue of peripheral nerve trunks, the epi-perineurium. CGRP, SP, and NO are vasodilators with CGRP and SP also potent upregulators of vascular permeability of the vasa nervorum and neighboring blood vessels. The result is a rapid increase in the endoneurial blood flow and neurogenic inflammation of the nerve trunk itself and the surrounding tissues. This forms the basis of the Triple Response described by Lewis in 1927 with the well-known “line, wheal and flare”. Lewis also identified the Axon Reflex, showing axonal impulses travelling in an orthodromic (to CNS) and antidromic direction. In 1901 Bayliss found showing axonal impulses travelling in an orthodromic direction. In 1901 Bayliss found that stimulation of the dorsal root ganglion (DRG) resulted in peripheral vasodilatation. He postulated afferent and efferent conduction. Some 20 neuropeptides and neurotransmitters are known to be involved in neurogenic inflammation. Most of these have been cloned, including their antagonists, their receptors, and receptor antagonists. Evidence of pivotal roles for specific neuropeptides is lacking, hence no drug treatment has yet been developed against neurogenic inflammation and the concept of neuromodulation is rationalizing the impasse. It has also been found that some neuropeptides (CGRP, peptide YY - PYY - and neuropeptide Y - NPY) can be released from non-neuronal cells and also in a paracrine fashion from neurons. Some anti-inflammatory neuropeptides such as melanocyte-stimulating hormone (MSH), vasoactive intestinal peptide (VIP) and pituitary adenylate cyclase-activating polypeptide (PACAP) may be released to terminate inflammation under physiological conditions. Thus neuromediators are not all pro-inflammatory but regulate all phases of the inflammatory response.

The complexity of responses of the peripheral nervous system was also highlighted by Burnstock in 1976. He introduced the concept of co-transmission in the autonomic nervous system with neuropeptides also contained in cholinergic and adrenergic nerves. The intimate connection between sensory and autonomic nerves is particularly poignant in children born with congenital insensitivity to pain in hereditary sensory and autonomic neuropathy (HSAN type I-IV). The absent or abnormal peripheral nerves are the reason for severe disfiguring sequelae of trauma, inability to repair tissue or adequately mobilize the immune system, often leading to death from infection during childhood. The recent finding of opioid receptors on peripheral sensory axons led to some speculation that these μ opioid receptors (MORs) may have an antinociceptive action. This motivated Zochodne et al, to examine the function and expression of local MORs associated with the chronic constriction injury (CCI) model of sciatic neuropathic pain in rats. Low dose morphine was injected percutaneously near the nerve. They concluded that their positive findings “may provide a therapeutic direction for the treatment of certain focal neuropathic lesions in humans”.

Successful treatments of painful peripheral neuropathies or chronic recalcitrant pain have been described in the literature. Whether one goes along with the rationale for these treatments is less relevant. After all, clinicians would consider it unethical to cease treatment with lithium carbonate or chlorpromazine simply because the mode of action is unknown. By 1965 George Hackett had published the results of prolotherapy treatment of 1800 cases of chronic low back pain, with an 82% success rate and a 12-year follow up. He published 16 articles and one book on his treatment. Also in 1965 Melzack and Wall published their Gate Control Theory of Pain. Their Textbook of Pain, published in 1997, does not reference prolotherapy. Neither does it reference neural therapy. This treatment was developed in Germany in the 1940s by Drs Ferdinand and Walter Huneke. Lidocaine is used to treat chronic pain by targeting postulated “interference fields” that cause “blocks” in the autonomic nervous system leading to chronic pain. This treatment is highly successful and practised widely in Germany and Spain by more than 5000 medical practitioners. Most of the literature is in German and this may be the reason there is little knowledge of this treatment in the English-speaking world.

The growing scientific evidence supporting the view that neuropathic pain syndromes are caused by unremitting peripheral neurogenic inflammation involving the autonomic and sensory nerves may lead to renewed interest in prolotherapy and neural therapy as these treatments are effective and seem to target the PNS. The author has now treated more than a 1000 patients with subcutaneous prolotherapy targeting neurogenic inflammation of peripheral nerve trunks in much the same way as Zochodne’s percutaneous near nerve injections with low-dose opioids. Published results are promising for recalcitrant lumbago, shoulder, knee, elbow pain, and achillodynia. Patients with chronic neuropathic pain will continue to suffer needlessly if physicians remain fixed on the reigning paradigm that can suggest only “pain management” when
there are well-documented alternatives available that may offer a cure.

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Treating Inferior Heel Pain with Vitamin D3 Dermal Cream: 
A clinical report on two case histories

Dr John Lyftogt, Christchurch, New Zealand

Abstract

Inferior heel pain, also known as plantar fasciitis, is a peripheral neuropathic pain syndrome due to persistent neurogenic inflammation of the medial calcaneal branches of the tibial nerve. Vitamin D3 is considered to be a neurosteroid with neuroprotective properties. This is the first report of two patients responding favorably to twice daily applications of vitamin D3 transdermal cream for this difficult-to-treat condition.

Introduction

Inferior heel pain, also known as plantar fasciitis or policeman’s foot, is common both in runners and non-runners. Incidence has been estimated at 10-15% of all patients presenting with foot pain and 10% in runners. The incidence peaks between 40 and 60 years in the general population and earlier in runners. In 30% the pain presents bilaterally.1

It is a condition resistant to most interventions and often treated with corticosteroid/local anesthetic injections. This treatment is painful and likely to be ineffective or harmful.2 A number of observational studies found a high rate of plantar fascia rupture and other complications associated with corticosteroid injections, which may lead to chronic disability in some people.2

One telephone follow up survey with a mean length of 47 months on 100 people treated conservatively found that 82 people had resolution of symptoms, 15 had continued symptoms, and three had to change work status and had limited activity.2

The author has treated more than 30 patients with inferior heel pain with subcutaneous prolotherapy with encouraging results (unpublished). However, repeated plantar injections are distressing and infection occurs infrequently unlike treatment with subcutaneous prolotherapy elsewhere.

Vitamin D3 has recently been shown to have suspected analgesic properties in neuropathic pain.3 Inferior heel pain is best viewed as a neuropathic pain syndrome4 caused by persistent neurogenic inflammation of the medial calcaneal branches of the tibial nerve (see Figure 1).

The author postulated that local application of a vitamin D3 transdermal cream may have analgesic effects in the treatment of disabling pain of this self-limiting but recalcitrant condition.

Methods and results

Prospective recovergrams5 were recorded for each patient, monitoring weekly VAS pain scores.

Patients were instructed to massage a small amount of vitamin D3 transdermal cream into the painful area twice daily and after activity, followed by heat application where possible.

The vitamin D3 transdermal cream consisted of cholecalciferol (25(OH) D3) 12,000 IU/gram.

Both patients were monitored for serum vitamin D and calcium levels at the end of treatment. Vitamin D and serum calcium levels were all in the normal range.

At the time of writing this report both patients were running pain free, although still using the cream. Earlier experimental clinical experience suggests that it is prudent to continue the vitamin D3 transdermal cream until all pain and tenderness has resolved.

Patient 1

History: MM, age 46, female, elite masters middle distance runner with 2-month history of right inferior heel pain treated initially with subcutaneous prolotherapy, compli-
Patients 1

History: JM, Male, 23 years, competitive runner with 10 years of running history, complaining of inability to run, difficulty walking, and pain at rest.

Past history of bilateral peripatellar pain treated with failed surgical decompression, Achillodynia.

VAS: visual analogue scale  0= no pain  10= worst imaginable pain

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Patient 2

History: JF, age 24, female, fitness trainer with 8-month history of left inferior heel pain, complaining of inability to run, difficulty walking, affecting work. Has prior treatment with physiotherapy and podiatry without effect.

Past history of bilateral Osgood Schlatter Disease, right medial shin splints and peripatellar pain

VAS: visual analogue scale  0= no pain  10= worst imaginable pain

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Treating Inferior Heel Pain with Vitamin D3 Dermal Cream
Discussion

Policeman's foot or inferior heel pain is a painful peripheral neuropathy precipitated by nerve injury to the medial calcaneal branches of the tibial nerve. The local milieu of the injured nerve will determine the likelihood of regeneration and the development of neuropathic pain. Current research is suggesting a role for vitamin D in nerve repair.

Vitamin D is best known for its role in calcium metabolism and bone health, but new roles are continually being discovered. Vitamin D is now described as a pleiomorphic seco-steroid hormone, with actions on 200 genes in at least 16 different tissues. Vitamin D receptors (VDR) have been identified in the cell nucleus and membrane, giving rise to respectively nuclear and cytoplasmic signalling pathways. Recent investigations have considered the mechanisms underlying the neuroprotective effect of vitamin D and its receptors in the nervous system and the effect of vitamin D on the nervous system and skeletal muscle.

Rheumatologists in the UK have for many years been aware of the extremely high prevalence of hypovitaminosis D in Indo-Asian patients, associated with high scores for musculoskeletal pain and low clinical scores for gait and muscle strength.

A growing number of articles have been published in the last five years connecting vitamin D with musculoskeletal pain.

Most recently a research letter published in the April 14, 2008, issue of Archives of Internal Medicine reported that vitamin D supplementation reduced pain levels in patients with diabetic neuropathy. The authors concluded “Vitamin D insufficiency is under-recognised and may be a significant contributor to neuropathic pain in Type II Diabetes. Vitamin D supplementation may be an effective ‘analgesic’ in relieving neuropathic pain.”

It is now well documented that the skin has a unique role in the vitamin D endocrine system. It is the only site of vitamin D photosynthesis and plays a central role in obtaining a sufficient vitamin D status. The skin is also capable of synthesizing the active vitamin D metabolite calcitriol (1, 25(OH)2 D 3) due to the presence of 1α hydroxylase, although this reaction is heat labile. The skin itself is an important target organ for calcitriol where it regulates growth and differentiation of many cell types.

Vitamin D receptors (VDR) on cutaneous nerves may influence neurotrophin release, Ca2+ regulation and neuroimmunomodulatory effects of calcitriol.

Transdermal creams with bioidentical steroid hormones have a well established use in hormone replacement therapy.

It is postulated that the application of a transdermal cream with the prohormone vitamin D3 (25(OH)D3) near neuropathic cutaneous nerves will upregulate the local production of calcitriol and result in a neuroimmune modulatory effect more effective than oral vitamin D supplementation.

Conclusion

This is the first report of disabling and recalcitrant neuropathic pain of the inferior heel, also known as policeman’s foot or plantar fasciitis, responding favorably to twice daily applications of vitamin D3 transdermal cream. Two patients were treated and no side effects or complications were observed. Further research into this promising and safe treatment is warranted, with particular focus on the long-term beneficial effects of vitamin D3 transdermal cream on peripheral neurogenic inflammation either in combination with subcutaneous prolotherapy or on its own.

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An Alternative Sacroiliac Joint Injection Technique

**Dr Paul Quin, Full-time musculoskeletal and interventional pain medicine, Auckland; current president, Australasian Faculty of Musculoskeletal Medicine**

With the increasing performance of sacroiliac joint injections, both for diagnosis and treatment of sacroiliac joint nociception, there is a suspicion that a significant number of attempted procedures fail in their intent. If the confirmation of intra-articular entry is the demonstration of an arthrogram with contrast medium, it is likely that there is a significant percentage of failures which may not show up in the literature. Anecdotal reporting suggests that 10-50% of sacroiliac joint injections may not succeed which may skew outcome data from sacroiliac joint reports. Rosenberg et al. reported in a double blind study of 39 joint injections in 37 patients that intra-articular injection was successful in 22 joints – a failure rate in technique of 40.5%.

The sacroiliac joints have been shown to be richly innervated and identified as sources of low back pain and referred somatic buttock, groin, and leg pain. Therefore, the injections have face validity. Providing the sacroiliac joint injections are controlled, the technique has also construct validity. If the “gold standard” of diagnosis is injections of local anesthetic into the sacroiliac joint space to demonstrate pain relief, then technique failure becomes relevant.

Owing to its complex configuration, the sacroiliac joint has typically been difficult to enter, especially as a blind office procedure, and even when using fluoroscopic guidance. The plane of the joint is curved in three dimensions, and the posterior aspect of the joint is located medially as compared with the anterior aspect of the joint, which is positioned more laterally. The net plane of the joint is from postero-medial to antero-lateral.

Usual fluoroscopy has utilized obliquity of the fluoroscopic tube in a medial or lateral direction to attempt to obtain a good target for needling. Unfortunately this may give the visual impression that the joint is well aligned, whereas, in fact, only a portion of the joint may be demonstrated, and which portion is demonstrated is uncertain.

**The SIJ injection technique**

The following technique was originally described by Dussault et al. in 2001 in *Radiology* and I have used it for SIJ injections since November, 2003. I describe it as I practise it in Auckland.

The patient is advised in detail about the procedure when it is first considered. He or she is shown what is done on a skeleton of the pelvis, shown a slideshow of the procedure, given a relevant information pack, and queried about allergy to shellfish, crayfish, etc. If an allergy is present, the patient is given a prescription for prednisolone, cetirizine hydrochloride (Razene) 10 mg and ranitidine to be taken over the 24 hours prior to the procedure to obviate cross-sensitivity responses to iodo-contrast.

After informed consent is obtained, the patient is positioned squarely prone on a C-arm fluoroscopic table, generally lying flat with no pillow underneath the trunk. The C-arm x-ray beam is set entirely vertically with no cranial, caudal, or oblique angulation, and the inferior aspect of the SIJ is visualized on the screen (Figure 1).

With the x-ray tube perpendicular to the table, the target is identified with a metal pointer and that position is marked on the skin with a skin pen. The target is the distal 1 cm of the SI joint, and if more than one joint cavity is seen on the image intensifier, the correct target is the medial one (Figure 2).

Usual sterile technique practice is observed and the patient is swabbed appropriately with antiseptic solution – we tend to use iodophor. The skin is anesthetized with xylocaine 1% solution at the site previously marked. Very anxious patients are offered Entenox gas for pain relief and to aid relaxation. A 22-gauge, 5-inch straight or 10° curved-tip spinal needle is advanced perpendicular to the fluoroscopic table through the skin and directed down towards the SIJ for 20-30 mm (Figures 3 and 4).
The tube is then angled about 20°-25° by moving it in a cephalic direction to displace the postero-inferior portion of the SI joint in a caudad direction (Figure 5). This allows the accessible posterior aspect of the joint to be clearly differentiated from the inaccessible anterior aspect of the joint, which moves cephalad on the image.

After moving the tube into the cephalic position (20°-25°), the needle is then directed toward the posterior SI joint, which appears to be easily visible (Figure 6).

If using a curved-tip needle, the tip of the needle is...
An Alternative Sacroiliac Joint Injection Technique

... oriented in a cephalic direction, and the convex portion of the curve is oriented downward (closest to the joint). The curved-tip needle may be advanced either vertically or angled 10° downward to compensate initially for the 10° curve until the needle reaches the joint. As the needle contacts the firm tissues on the posterior aspect of the joint, it is maneuvered through the ligaments and capsule into the joint by advancing it about 5–10 mm, usually by angling the needle tip slightly laterally to follow the natural curve of the joint. It often seems a surprise to feel how far one must traverse to enter the joint. Intra-articular position is confirmed by injecting 0.2–0.5 ml of contrast material through the needle to obtain an arthrogram (Figure 7).

Dussault demonstrated in his original article how the posterior aspect of the SIJ became more accessible with his technique. In his diagrams (Figure 8), a single medial metallic dot (solid arrow, P) is positioned on the postero-inferior SI joints, and then two slightly larger metallic dots located more laterally (open arrow, A) are affixed to the antero-inferior aspect of the SI joints. The view in Figure 8 is that with the x-ray tube perpendicular.

When the image intensifier moves cranially, objects which are nearer or closer to the image intensifier move relatively in an opposite (caudad) direction to those more distant objects which appear to move in the same direction. This will separate the objects in the view. With the x-ray tube angled 20° by moving cephalad, the posterior aspects of the SI joints (solid arrow, P) project inferiorly and are now well outlined (Figure 9). The anterior aspects of the SI joints (open arrow, A) project superiorly and are more difficult to identify. Angling the x-ray beam by moving it in a cephalic direction, the posterior aspect of the caudal end of the SI joint is clearly depicted as separate from the remainder of the joint.

This allows easy placement of a needle directly into this portion of the joint. As the joint is entered posteriorly, the needle may sometimes need to be oriented in a cephalic direction to remain in the joint. This is best obtained with the 10° curved-tip needle, with the curve directed cephalad (Figure 10).

The failure rate of this technique may well be less than that of other techniques. My personal experience suggests that I am failing to obtain an arthrogram with contrast relatively less frequently than before. Some SIJs have...
surrounding osteophytes which may confound the technique.

I suspect that temporary leg weakness lasting 15 minutes or so and up to two hours may be more frequent. It is likely that, as the sciatic nerve is at the same level as the inferior SIJ, some leakage of bupivacaine onto the sciatic nerve may occur at times.

In general, advantages include (a) no premedication required; (b) reproducible technique; and (c) low failure rate, and short x-ray exposure time – 36-328 seconds (mean 108 seconds) in a teaching unit (original article).

My main source of unsuccessful SIJ entry was self-deception. For a while I thought I was getting a superior view at 10–15° of cephalad image intensifier movement but I started to have an increased failure rate. However, I was obtaining enough success to delude myself into thinking I was performing satisfactorily. It took me about six weeks and a few failed arthrograms to re-read and review the technique. The minimum cephalad movement should produce an angle of 20°.

To make a 10° curved tip needle, bend the terminal 1 cm of the sterile 22 gauge needle with the stylus in situ into a 10° convexity while holding the tip with a sterile swab. The bevel must face the convexity (outside) of the curve.

**Summary of sacroiliac joint injection technique**

- Lie the patient on his or her stomach with vertical x-ray and screen.
- Mark the skin over the distal 1 cm of the sacroiliac joint and apply local anesthetic to the skin.
- Insert a 22 gauge x 5 inch needle and start to advance the needle toward the posterior joint without any angling, cephalic, caudad, or oblique.
- Tilt the image intensifier 20-25° down by moving it cephalad but it must not be either medially or laterally displaced. The posterior aspect of the SIJ is then clearly separated from the anterior aspect and is located mediadly compared with the anterior joint which is more laterally placed.
- If using a 10° curved tip, centre the bevel on the convexity of the curve. The tip of the curved needle is orientated in a cephalic direction and the convexity orientated downward (closest to the joint). The curved tip may be advanced either vertically or angled 10° downwards initially to compensate for the 10° curve until the needle reaches the joint.
- Advance the needle about 5-10 mm to pierce the ligaments and capsule. The needle tip is then angled slightly laterally to follow the curve of the joint.
- Inject the contrast slowly to confirm the position in the joint. Contrast runs up and into the anterior sacroiliac joint. If having difficulty with a straight needle, the tip of the needle can be bent about 10° and reinserted with the concavity of the curve pointing cranially, as indicated above.
- If in the joint, inject 0.5-1.0 ml bupivacaine + 1 ml triamcinolone into the joint SLOWLY.

**References**

Meralgia Paresthetica: a Review

Dr Mark J Bailey, Resident Medical Officer, Christchurch, New Zealand

Introduction

Meralgia paresthetica is a syndrome of pain or dysesthesias, or both, in the anterolateral thigh, knee, and sometimes buttock secondary to irritation of the lateral femoral cutaneous nerve. The first case of meralgia paresthetica was described by German neuropathologist Martin Bernhardt in 1878 and then again more fully in 1895. Vladimir Roth, a Russian neurologist also published a paper in 1895 after noticing the syndrome in cavalrymen who wore their belts too tightly. Hence it was originally known as the Bernhardt-Roth syndrome. Roth coined the term meralgia paresthetica from derivatives of “meros” – thigh, and “algos” – pain. Sigmund Freud presented his own affliction and also the first bilateral case report in the same year.

Meralgia paresthetica has a variable presentation with regards to both the nature of the symptoms and the area of skin affected. Patients may complain of pain, burning, aching, tingling, numbness, and dysesthesias with or without paresthesias involving pinprick and touch sensations. Sensory loss is variable and is not usually prominent. The area of involvement varies in size and may affect anywhere from the distal anterolateral thigh/knee region up to the proximal anterolateral thigh/buttock and groin region. It usually presents unilaterally but bilateral afflictions are estimated to occur in 8-20% of cases. Bilateral cases may be seen more commonly in children. Most series have demonstrated a male predominance. Familial disposition has been suggested by some authors, although this does not appear to be a significant factor in most of the literature. Patients often report an aggravating activity such as prolonged standing or positions involving hip extension. Sitting may exacerbate the symptoms in some cases and relieve them in others. Sleep disturbance may feature in more severe cases.

The incidence of the syndrome in the general population is almost impossible to estimate for a number of reasons. Firstly, its variable severity: it is likely that severe cases eventually present to medical services but many milder cases may not seek any help. Secondly, despite the reasonably large collection of literature on the subject, it is likely that the condition is under-diagnosed by medical personal mainly due to ignorance and poor understanding of the condition. Indeed, a review of the literature reveals that the incidence is described as both “common” and “uncommon”! One study estimated the incidence to be 4 per 10,000. It is also interesting to note that the condition may present to family physicians, neurologists, neurosurgeons, orthopedic surgeons, anesthetists with an interest in pain medicine, sports physicians, and plastic surgeons. It would seem very likely that some cases will also be seen by musculoskeletal physicians.

Anatomy

The lateral femoral cutaneous nerve (LFCN) is a sensory branch of the lumbar plexus and is usually derived from the posterior roots of L2 and L3. In addition, an L1 branch may contribute to the nerve, although this is rare. Sympathetic afferent and efferent fibers are also present within the nerve. It emerges at the lateral border of the psoas major just above the crest of the ilium. It then takes an oblique course across the anterior surface of the iliacus before exiting the abdomen near the inguinal ligament and entering the proximal thigh. This exit point has traditionally been described as 1 cm medial to the anterior superior iliac spine (ASIS), but in fact this has been found to be highly variable. A study of 104 LFCNs in 52 cadavers revealed the following five anatomical variations:

Type A (4%)
The nerve exited through the abdominal wall muscles 2-3 cm posterior to the ASIS across the iliac crest. This is the most superficial variation.

Type B (27%)
The nerve was found superficial to the origin of sartorius and directly medial to the ASIS. Ten per cent of type B nerves also had an additional branch that crossed the iliac crest in a similar position to type A.

Type C (23%)
The nerve was located medial to the ASIS and just beneath the inguinal ligament ensheathed in the tendinous origin of the sartorius muscle. Nine per cent of Type C nerves had an additional lateral branch that crossed the iliac crest in a similar position to type A.

Type D (26%)
The nerve passed under the inguinal ligament medial to the tendinous origin of sartorius and then between the sartorius and iliopectos muscles.

Type E (20%)
The nerve was in the most medial location, embedded in soft connective tissue on top of the iliopectos muscle. In addition, the series revealed that only 65% of cadavers had symmetrical LFCN anatomy.

The LFCN divides into anterior and posterior divisions at a variable distance from the ASIS. The anterior branch penetrates fascia lata about 10 cm inferior to the ASIS to supply the skin over the anterolateral thigh down to the knee. The smaller posterior branch innervates skin over the greater trochanter region. Interestingly, in many anatomy texts the area of skin that the LFCN supplies is described as being the proximal two-three of the lateral thigh. However, as described above, more recent research has confirmed that the area of skin...
supplied by the LFCN extends right down to knee level.

Etiology

Meralgia paresthetica may be caused by anything that irritates the LFCN along its course. The classically described lesion is that of entrapment of the nerve as it exits the pelvis in the inguinal region.\textsuperscript{13} It is postulated that this is a site where the nerve is subjected to compressive and possibly traction forces. Table 1 shows suspected causes of nerve irritation at the inguinal region.

Diabetes mellitus may be a risk factor for the development of LFCN entrapment at the inguinal region.\textsuperscript{14,15} The proposed mechanism is either secondary to intrinsic diabetic neuropathy or due to the diabetic nerve swelling and decreased axoplasmic transport, rendering it more susceptible to external compression. However, other series have not observed an increased occurrence of meralgia paresthetica in diabetes compared to that in the general population.\textsuperscript{22} Other associated systemic diseases that have been reported include lead poisoning, alcohol abuse, and “other” neuropathies.\textsuperscript{15}

Dissections have revealed the frequent occurrence of pseudoganglions of the LFCN as it passes under the inguinal ligament.\textsuperscript{23} These pseudoganglions have not been observed in fetal cadavers, so it has been postulated that erect human posture and the angulated course of the LFCN as it exits the pelvis result in mechanical stresses on the nerve leading to their development. However, the relationship of these pseudoganglions to meralgia paresthetica is not entirely clear as they are seen in both symptomatic and asymptomatic individuals.

Although less common, more proximal irritation of the LFCN can also lead to meralgia paresthetica. Potentially any structure that comes into contact with the LFCN along its course from its origin to its exit from the pelvis can lead to compression or traction irritation. To date some of the described causes have included meralgia paresthetica of spinal origin,\textsuperscript{24} malignant tumor of the psoas muscle,\textsuperscript{25} and compression of the LFCN beneath the iliacus fascia secondary to infection or hematoma.\textsuperscript{26} The cause of meralgia paresthetica remains idiopathic in a significant number of cases and isolating an exact cause often proves to be a challenge.

Diagnosis

The diagnosis of meralgia paresthetica can usually be made on the patient’s history of pain and/or dysesthesias in the LFCN territory, exacerbating activities and identification of possible causes. It may be possible to reproduce the symptoms by pressing on the typical location of the LFCN 1 cm inferomedial to the ASIS. Similarly, in a symptomatic patient, it may be possible to abolish their symptoms with an injection of local anesthetic in this spot. Clearly, an awareness of the anatomical variability of the LFCN and consideration of more proximal entrapment is essential when performing this diagnostic test. The pain is generally exacerbated by extending the hip with the patient prone and the knee flexed to 90° as this maneuver places the LFCN under stretch.\textsuperscript{4,15} Other exacerbating activities may include the Valsalva maneuver and anything that increases intra-abdominal pressure. The examination may include assessment of light touch and pinprick sensation to the area of skin supplied by the LFCN but, as objective sensory deficits are often absent, this is not particularly reliable. Similarly, cutaneous stimulation by light touch to the lateral thigh may cause some patients to report a dysesthetic sensation. Some clinicians have also noted an area of hair loss on the lateral thigh that appears to be secondary to the patient’s rubbing or massaging of the leg.\textsuperscript{27} As the LFCN is a sensory nerve, there should be no associated motor weakness or reflex deficits. Nerve conduction studies may be utilized and appear to be reliable with findings of prolonged latency or decreased conduction velocity.\textsuperscript{14} However, they are not usually required except when the diagnosis is in doubt, such as differentiation from lumbar radiculopathy. Standard nerve conduction tests may also miss a proximal entrapment of the LFCN. Recent studies have demonstrated significant alterations in cutaneous silent periods of vastus medialis in subjects with meralgia paresthetica.\textsuperscript{28}
Cutaneous silent periods are brief interruptions of voluntary contraction that follow strong electrical stimulation of a cutaneous nerve and are thought to be a protective reflex. Electromyography has no role in the diagnostic work up as there are no motor manifestations of meralgia paresthetica.

Management
To date, no gold standard treatment has been established for meralgia paresthetica, but a recent series by Haim has established a useful algorithm that appears to be practical and cost-effective. The algorithm has been proposed for spontaneous meralgia paresthetica, that is, not secondary to a traumatic cause and is summarized as in the chart below.

### INITIAL TREATMENT
A course of oral NSAIDs +/- analgesics, rest and avoidance of likely causes of nerve compression (for example, lose weight if obese, avoid tight garments).

(After 3 months of the above and still symptomatic)

### DIAGNOSTIC TEST
Inject 5-10 ml of local anesthetic at a point 1 cm medial and inferior to the ASIS or point of maximal tenderness.

### POSITIVE TEST
Relief of symptoms lasting more than 30 minutes.

### NEGATIVE TEST
Retest 3-4 weeks later as above but to a wider area.

If still negative reconsider diagnosis (or consider intrapelvic causes).

### CORTICOSTEROID THERAPY
Treat the same site with corticosteroid injection (1 ml volume) and repeat on up to 3 occasions every 4-6 weeks as required.

(NON-RESPONDERS)

Further evaluation to rule out a more proximal compression (CT or MRI, additional ultrasound scan of pelvis in females).

### Neurolysis versus transection
The two major types of surgical treatment are neurolysis of the LFCN at the inguinal region and transection of the nerve in the suprainguinal region. Details of the neurolysis and transection procedures have been documented by Aldrich and Williams, respectively. The one randomized series that appears in the literature placed patients into the two options and declared that transection produced superior outcomes. Another series also reported success with nerve transection in 23 out of 24 cases. In a small series,
Ivins reported a high failure rate with neurolysis and also favored transection. However, other operators have found that neurolysis has a high rate of success, and arguments against transection include the potential development of painful neuromas and the resulting sensory loss, although it is a relatively unimportant territory. It has been suggested that some of the anatomical variations of the LFCN render neurolysis as technically difficult and less likely to succeed. In addition, they felt that the presence of a neurinoma results in a higher failure rate with neurolysis and suggested this was an analogous lesion to Morton’s neurinoma. In the presence of either of these findings, transection may be the procedure of choice. Ivins recommended that neurolysis should be offered to all pediatric patients and patients with less than one year of symptoms. He advised that transection should be performed after failed neurolysis and as the procedure of first choice in patients who have been symptomatic for more than one year. One consensus is that alleviation of symptoms from a local anesthetic block in the inguinal region is essential before proceeding to surgical treatment. The choice between the two options seems to be dependent largely on the operator’s preference and familiarity of a particular procedure as well as the patient’s wishes with regards to preservation of sensory function. In the future more successful outcomes from neurolysis may also be facilitated with the assistance of intraoperative somatosensory evoked potentials. A recent case demonstrated that during a LFCN release an intraoperative increase in amplitude of somatosensory evoked potentials in the nerve correlated with a favorable clinical outcome. Further investigation in this area would be useful.

Other treatments

Other treatments that have been described in the literature but their efficacy and role in management is yet to be established include anticonvulsant and nerve-stabilizing drugs such as carbamazepine, gabapentin, and diphenylhydantoin.

Abdominal toning exercises could theoretically reduce pressure over the nerve at the inguinal region, but again this is of unproven value.

One case report in the literature reported the successful treatment of meralgia paresthetica with topical capsaicin 0.025% cream applied to the area of skin supplied by the LFCN. The proposed mechanism is desensitization of C-polymodal nociceptors and depletion of substance P in the nerve. There was no mention of an endpoint in this treatment, but it may be a useful non-invasive adjunctive therapy. Again, further investigation in this area would be useful.

Alcohol rubs, diathermy, and ultrasound have also appeared as therapies in the older literature, but their effectiveness was not described.

With regards to meralgia paresthetica of spinal origin, high rates of a success were obtained with 1-4 epidural injections of dexamethasone. The author developed bilateral meralgia paresthetica while training as an endurance athlete and after three years of symptoms had a failed LFCN neurolysis surgery. The symptoms were eventually brought under control with a yoga-based myofascial stretching program involving the lower back, pelvic, and thigh musculature. To my knowledge this type of therapy has not been described previously in the literature.

References

Meralgia Paresthetica: a Review


Complementary Pain Management
Based on Anatomical, Physiological, and Neurological Concepts

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Introduction

Complimentary pain management has been written about before1,2 but usually as a series of summations of what has been used and the fact that they do work, based on treatment trials. Few, however, have addressed the issue of how they may work, from the basic anatomy, physiology, neurology, etc. To do that one must go right back to the very beginning. What is pain?

“Pain” is a very difficult concept to understand and is made even more difficult through lack of a shared lexicon between individuals. By definition: “Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. Pain is always subjective. Each individual learns the application of the word through experiences.”3

And this is where the problem commences. Pain is a warning system to draw the attention to actual or impending harm to the tissues. Take out the sensory component and there is no pain perception, and take out the emotional component and similarly there is no pain experience per se. So both sensory and emotional components are needed at the same time for the brain to perceive that the afferents are associated with the reactivation of the previous memory of pain.

Based on its learnt memory, pain is only the brain’s perception that starts in utero, but is maximally activated after birth and continues throughout life, where each person has a completely unique experience of pain.

Yet, who can verbally share such sensory and emotionally combined experiences and memories with other people?

The four most common words used to express such an experience are pain, ache, sore, and hurt, but what do these words mean to any individual? Each person has his or her own personal experience of what these words mean, but can they expect or believe that their experience is the same as anyone else? If not, then why not?

The answer is quite simple. Take the example of a gold-colored paper weight. When a patient is asked what color it is, he or she can tell you immediately and say “gold”. So why does this occur?

When they were very young, someone told them that the item that their eye and brain were perceiving was “gold”, and from that point on they hear others saying “gold”, and they tell others that it is “gold”, so the lexical word “gold” has a very specific and continuously shared meaning.

Now when a person suffers a pain, ache, soreness or hurt, there is no similar sharing of the details, context or referential meaning and so we lack any identical shared meaning for the words pain, ache, sore, and hurt. However, there is continuous learning from parents and peers of the difference in attention that is paid to various forms of injury or pain and how they are managed. For example, ignore it, apply a “Band-aid”, try a medicine, see a doctor, or go to bed to rest.

It is obvious that pain is largely the person’s brain perception which has been remembered or reactivated after being sorted out from the massive chaos of afferent stimuli activating all parts of the brain structures continuously during life. Therefore, it is unique to that person.

In medicine we spend a lot of time collecting many shared words and details of the experience of pain from the patient, which forms the patient’s history. Unfortunately, because most health practitioners do not recognize the importance of this stage of history taking and the vagueness of such words, there is often only a brief record of the abbreviated words used to refer to pain, ache, soreness and hurt regarding the pain experience and, as a result, such histories are almost useless.

How can we make this better, simpler, and thus allow the use of appropriate forms of complimentary pain management?

This involves understanding the mechanism of the stimulus to the body part that will finally be perceived as pain. It is activation of the local receptors, then the nociceptive pathways and steps to the spinal cord, with modulation and convergence, upward to the midbrain, and finally to the brain areas. This was clearly detailed in my previous article4 based on the booklet Pathophysiology of Pain produced by Astra-Zeneca,3 available to any doctor requesting it. (Telephone Australia 02 9978 3500, New Zealand 09 623 6300.)

It is now possible to share some understanding of how and why different methods of complementary pain management work in overall pain management.

Pain pathway elements and management options

Most of these have little to no evidence based on blinded or randomized trials. In all cases, however, the most important consideration is that the patient presents with a problem. The doctor or other healthcare practitioner, after taking a history and examination, applies a “black box” (whichever management is utilized) and ideally the patient says “My problem is fully resolved!” That is a successful
Balneotherapy

Considering the peripheral receptors (Figure 1), it can be seen that trauma leads to changes in the cells of the damaged tissues. The very first lecture in physiology told us that if any cell membrane was damaged, K+ would be released from intra- to extra-cellular and this would produce an electric current across the membrane, where adding many together produces an impulse in the specialized cells called nerves. At the same time, other substances, for example, prostaglandin (Pg) E, are also released to help trigger the nerve response.

If this area were to be flooded with extra Na+, the imbalance of K+ would be reduced and less nerve stimulation due to the change would occur. It is not necessary to actually float in salty water because strong salty water applied or sprayed on to the skin can work in the same way. This is the simple principle behind balneotherapy or salt water bathing and is why people feel “refreshed” after swimming in salt water or travel to the Dead Sea to float in the very salty water, or why people get such benefit from Flotation Chamber treatment. How can this be achieved at home? By using 4-8 kg of swimming pool salt (it costs about A$6 for 25 kg bags) in their usual hot bath water, people can soak in this, then pat the water off and finally let the extra salt stay on their skin overnight.

Promethazine: Cheap over-the-counter medication

Everyone knows that promethazine is an ideal treatment for allergy or insects stings, but look carefully at the diagram again (Figure 1). Histamine is released by the mast cell when traumatized, so this is one site for promethazine action, that is, to reduce the dual histamine activation of the A-delta and C fiber nerve endings. The dose is patient dependent, and one should start at a low dose of 10 mg at night initially and increase slowly and gradually each night until the best effect is obtained without excessive dry mouth or somnolence. See later for other uses of promethazine in histamine blockade in pain activated pathways.

Exercise, massage, yoga, tai chi, and related activities – all free too!

Looking at Figure 2, there are different receptor types in tissues. A-alpha fibers relate to control-feedback involving muscle and tendons. A-beta fibers are the fourth input after olfactory cranial...
nerve (CN) 1, optical CN 2, and auditory CN VIII for Pavlov’s Orienting Response5-8 and are fast-conducting impulses at 50 m/sec.

A-delta fibers (myelinated) and C fibers are for nociception, mechanoreception, and thermoreception and are slower conducting impulses at 15 m/sec and 1 m/sec, respectively.

On looking at Figure 3, the input of these fibers to the dorsal root ganglion and spinal cord are clearly illustrated. Further, in considering the function of these nerves, touch A-beta fibers pass primarily and immediately to the higher centers, because survival and function almost always over-rule pain. This is a very fast pathway, but there are also retrograde pathways (Figure 4) that enter the dorsal horn at level V. These act as modulators or inhibitors of the incoming A-delta and C fibers in level V up to level 11 of the Rexed laminae.

This gives rise to simple and effective methods of reducing pain, by utilizing massage, exercise, tai chi, yoga, etc., which all act by increasing A-beta inputs to the dorsal horn layers.

Hydrotherapy

This works for a number of simple reasons. Once the body is two-thirds in water, much of the activity of the antigravity muscles reduces, the attachments of the muscles and tendons to the periosteal areas are less strained (reducing A-delta and C fiber inputs) and muscles can relax more, allowing better flow of the synovial fluid between the joint surfaces. This allows joint spaces to “open more”, with less closely applied cartilage surfaces. In addition, exercise enhances pain reduction, by the clearance of metabolic products, as well as increasing nutrient flow to cartilage normally in close contact and under pressure.

The one caveat for hydrotherapy is that, if the patient has their feet on the bottom of the pool, they should never walk forwards. This results in over-working of the psoas muscles almost in isolation to the posterior back muscles, with overdrive by the quads and other leg muscles. This can aggravate pain and dysfunction, because the proximal psoas muscle fibers effectively surround the exiting lumbar nerve rami. While in water, with feet on the bottom, the person should always walk sideways or backwards to avoid pain exacerbation via excessive psoas muscle activity. Simple anatomy, biomechanics, and whole body function always help understanding of what should or should not be done.

Downward modulation of dorsal horn mechanisms by higher centers

This pathway is via the dorso-lateral funiculus (DLF) as shown in Figure 3. This is the pathway that the current pain clinics try to enhance as their main method of dealing with pain. The patient is taught that the “pain is in the brain and they must learn to ignore it” and they are then taught how to achieve this. Sadly, this is more akin to diversion therapy used in aged care homes, than true pain management.

This is done utilizing classical Pavlovian conditioned reflex activation9 with the amplification of the output via the DLF to the dorsal horn units. The level to which the pain can be attenuated was demonstrated by the solo rock climber who had not notified others regarding his plans. He was trapped in a chasm by a boulder on his right hand. On day three he cut his right hand off with a penknife in order to survive.10 Similarly, in NSW, a young seven-year-old lad came off a quad motorbike and suffered fractures to his shoulder, clavicle, and ribs, and he had a collapsed lung. He walked two kilometers to get help11 because his dad had collapsed near the motorbike.

The power of mind-over-pain occurs when survival and function have to be achieved and pain can be ignored in
the short term if needed. However, when chronic pain occurs due to continuing tissue damage or excessive ongoing nociceptor activity or accumulated damage over longer periods of time, dorsal horn “wind up” may occur. The patient cannot continue this pain suppression pathway. Sleep deprivation often occurs, caused by histamine release in the midbrain areas, giving rise to secondary reactive or relative “depression” due to increased adrenalin and nor-adrenaline compared to the serotonin levels in the autonomic nervous system. Persistent and continuing use of these methods of pain management via the DLF can result in neuroplastic cerebral brain map changes, with increased conscious awareness of some pathways and cerebral recognition of afferents being diminished or ignored altogether in others. This is the amazing new area of brain neuroplasticity. Because pain is a warning system for whole body function, persistent and accumulating pain afferents will eventually override all these pathways modifications and become all-important or overt again.

How the autonomic nervous system is involved, and how to treat that

How many remember the complexity of the autonomic nervous system (ANS) from medical school? If you step back and look at it as a system with two operations, it becomes very simple (Figure 5). One is the parasympathetic nervous system (cranial and caudal) that primarily uses acetylcholine as its final neuromodulator. The other is the sympathetic nervous system that uses adrenalin (flight, fight, fun) or noradrenalin (fear, freeze, fear) as its final neuromodulators at the alpha and beta adrenergic receptors. In all other synapses, preganglionic neurons, ganglia and postganglionic synapses it is acetylcholine that predominates.

On considering the overall mechanism it is easy to use the simplest concepts:

A. Full belly (food) wine, comfort, and relaxation. Time to digest, shift excess heat to the skin and to relax and the blood flow changes to achieve this. **Parasympathetic nervous system.**

Or

B. Threatened, alert, anxious, aroused, ready for flight/ fight or fear/freeze. **Sympathetic nervous system,** via adrenalin – fun, alert, or anxious, or noradrenalin – fearful, scared, and often very jumpy. They have different effects on individual terminal receptors, giving differing outcomes. At the same time all this is happening, information travels to the midbrain areas, where there are immediate midbrain responses, often in less than 200 milliseconds, which activate the autonomic nervous system and whole body function via the midbrain and thalamus. One of these effects is the activation of the sleep centre by neuropeptides activating histamine pathways of the tubero-mammillary bodies and causing wakefulness.

The hormone orexin or hypocretin is released by afferent nociceptive activation of the thalamus and is also associated with wakefulness and increased appetite, historically fulfilling an animal’s need to keep awake and eat to allow rapid repair of damaged tissues after being injured. This is one reason why people in chronic long-term pain tend to put on weight.

So how do we manage these problems by simple methods? Once again, the effect of promethazine becomes relevant. Promethazine blocks the histamine-activated pathways, thus allowing sleep to occur. At the same time promethazine is a general and mild anti-cholinergic so we have a modulation of both the sympathetic and parasympathetic nervous systems by this simple, over-the-counter drug, known best for its use in allergies and also to help children sleep.

So promethazine has a triple action:

1. Working at tissue levels to stop activation of nociceptive fibers by histamine released from the mast cells in injury;
2. Blocking of the histamine pathways that prevent sleep; and
3. General peripheral ganglionic modulation of both sides of the autonomic nervous system.

It is obvious to see why people taking promethazine complain of drowsiness. Their sleep deprivation (caused by acute or persistent pain) be-
comes dominant and, as soon as the peripheral and central histamine effects are blocked by promethazine, the body naturally tries to catch-up on the missing sleep. With continuing use of the promethazine, the dry mouth and drowsiness reduce as the sleep deprivation is also reduced, allowing a dose gradually increasing over time.

Now we go back to the functioning of the autonomic nervous system, as mentioned above. When threatened or in pain, one of the body’s autonomically mediated responses is to reduce blood flow to the skin (pallor, itchiness, aroused erector pilae) and also reduce blood flow to the gut, so that the maximum blood can be shunted to the brain, heart, lungs, and muscles. Reduced blood flow to the gut results in reduced digestive processes, less digestive enzymes, and disturbed peristalsis, often producing bloating, reflux, constipation, or diarrhea.

So it follows logically that oral medication for pain management is often poorly digested, which is necessary before they can be adsorbed. So they are less effective, often hanging around in the gut, causing many side effects (ulceration, nausea, severe constipation, bloating).

Improving drug effectiveness by better digestion

This method resulted from a patient I shared with Dr Daryl Wall (associate professor of surgery, Princess Alexandra Hospital, Brisbane). The patient had gut surgery for a small malignant pedunculated bile duct tumor. It was observed that her Panadeine Forte worked well during the day when she took pancreatic enzymes to assist digestion post-gut surgery, but not at night. So the patient took pancreatic enzymes at night and got much better pain management. The dose is very simple: two capsules of Creon or similar enzyme preparation with each dose of any analgesic medication. There are no dose limits because it is only an enzyme preparation and not a drug and it is used for digestive dysfunction in gall bladder disease, liver diseases, cystic fibrosis, adsorption disorders, etc. They are readily available on general prescription. Many patients find extra doses at meal times also help with their previous poor digestion and gut dysfunction.

How other pain managements work

Exercise

This takes us back to the very first question asked: Why does a muscle not pull out of its attachments when the muscle is very active (Figure 6)? The answer is very simple in principle. The muscle/tendon/ligament is attached to and through the periosteum (via Sharpey’s fibers) to the cortical bone. So when the muscle contracts, the many receptors in the periosteal layer are immediately activated, where the densest distribution of such mechanoreceptors and nociceptors occur17 (Figure 7). This activates feedback inhibition to the motor neurons opposing excessive muscles contraction, both from the spinal cord level and via afferent activation of higher centers, producing secondary efferent motor inhibition.

It was considering autonomic nervous system function in the gut in pain and observing the benefit of the digestive enzymes that led Dr Daryl Wall and me to make longer-term observations on pain management, utilizing regular promethazine and pancreatic enzyme supplements. Dr Wall’s comments, when I asked him if he had tried the combinations, are worthy of publication. He stated that prior to using the promethazine and pancreatic enzyme: “When I did a ward round, I saw tired patients in pain, and when I picked up their charts they were already on maximum analgesic doses and getting side effects. Once we introduced the promethazine and pancreatic enzymes as a regular management, I now see happy patients who have slept well and have little pain, but when I pick up their charts I see that we are using only 1/3 to 1/5 of the pain medication. Another benefit is that our liver and kidney transplant patients are now using 1/3 to 1/5 of their immunosuppressant drugs – a massive financial saving per week per patient.” This would be a wonderful study for some enthusiastic registrar.

Another benefit from this is that many patients may be able to cease the expensive proton pump inhibitors that are not free of side effects and have much better digestion and fewer gut symptoms that were secondary to pain.
Complementary Pain Management

At the same time the receptors in the muscle body (Golgi body, etc.) are squeezed, adding to the feedback inhibition. This compression also impedes both arterial and venous flows, causing a relative lactic acidosis, hypoxoxygenation and forces anaerobic metabolism, resulting in further incremental acidosis. This may eventually cause a reflex increased blood flow as shown by plethysmography which is a whole of limb or body measure.16

The way to reduce those effects is to reduce excessive muscle contraction and cause relaxation and lengthening, thus reversing the above.

How is this done?

Yoga, tai chi, and many other programs work, at least, partly by this method.

Simply consider the arm bending and straightening. As it bends the brain and cerebellum make the flexor muscles contract, but also make the opposing extensors relax. The opposite happens when the arm is again straightened.

So exercise that encourages stretching in both directions by alternate contraction and relaxation helps restore normal lengths and functions of the muscles involved. There is reduction in any excess activity via the A-delta and C-fiber afferents to the dorsal horn. This also helps reduce the afferents to the dorsal horn and higher centers and thus reduces the brain’s perception of pain. This is what so many pain management programs emphasize, but fail to simply and adequately explain – exercise is so important. It also helps reduce relative “depression”. Many other factors are involved as well; for example, endorphin release, total body metabolic function, but most of all there is more balanced whole body function again.

Rubrefactants

These stimulate the A-beta sensory inputs at the periphery and increase A-beta inputs to the dorsal horn thus modulating afferents and pain perception as explained above. The most potent is the commercially available Finalgon® cream.

Deep tissue massage

This activates the A-beta input, reducing increased muscle tone, and thus tension on the attachment receptors, decreasing the A-delta and C fiber inputs to the dorsal horn. As the muscles relax, the accumulated metabolic products (lactic acid, dissolved carbon dioxide, etc.) associated with excessive activity and reduced oxygenation from squeezed tissues, which may be associated with brain level pain perception, gradually find their way out of the muscle tissues and more oxygen and nutrients pass in. This results in restoration of more normal metabolic and functional activity in the muscle. At the same time, deep tissue massage sends a barrage of A-beta input to the dorsal horns at each level, facilitating the blockade of afferent nociception and allowing full muscle length to be restored.

Chinese cupping or equivalent

This acts by causing reduced pressure under the hot cup as it cools, which then sucks out fluid and blood into the tissue. Blood in the tissues is very irritating, as anyone who has had a good sprain or hematoma can attest, and the remaining blood in the tissues is a persisting source of A-beta stimulation.

Prolotherapy

Yelland’s work,19 which showed that injected glucose solution was as effective as saline control begged a simple explanation. In my paper,6 I was faced with the fact that all three variations (needle + steroid + local anesthetic, needle + local anesthetic, and needle alone) gave the same result, even after one month, and in two cases the needle alone was still effective some 2.5 years later. The last cases were effectively simple dry needling. The needle alone is not a placebo effect as there is active physical damage to the entheses being carried out. So what was happening?

In the model published in May 2004,6 the application of Chaos Theory was used to explain how and why the original cases presented and were interpreted. As Bogduk20 suggested: “Chaos Theory explains intricate situations with a complex differential mathematical equation, which includes constants; when a constant changes, everything changes. Restore the constant and everything restores.”

“Pain”, or more correctly, the persistent nociceptive receptor activations due to accumulated tissue damage are the equation “constants”, that is, the damaged receptors in the periosteum, ligaments, tendons, fascia, or muscles are the increasing “constants” or additive damage afferents. These pass with the chaos of information that the brain has to interpret to perceive pain. Not all receptors repair after injury and, over time, the number of damaged, but active receptors increases. By “turning off” these switches or receptors clusters, there is reduction in dorsal horn afferents and thus no amplification in the dorsal horn, and reduced afferents to the brain. This makes further sense, because once the afferents to the midbrain cease, the efferent autonomic outputs (mainly sympathetic adrenergic and nor-adrenergic pathways) associated with increased damaged area neovascularization and other tissue perfusion factors also decreases.

Switching off the nociceptive afferents thus turns off the “cascade of repair processes” in the damaged areas. Perhaps this may explain some of the local changes reported in clinical prolotherapy research.

Reflexology

This is a wonderful example of two extremes. As the
Chinese found many thousands of years ago, if you massage the feet very gently, you increase relaxation. How? Simply by increasing the A-beta inputs to the lower dorsal horns, thus modulating nociception all the way up the spinal levels up to the midbrain. After a while everything settles and is calm.

The opposite was also used by the Chinese. Caning of the feet slowly and continuously was used to torture and kill people. How did this work? By increasing the A-delta and C fiber inputs from receptors (massive nociceptive inputs), the opposite was achieved; death from extreme overload to the autonomic nervous system at midbrain levels. This is one of the natural killers for prey animals in the wild, with pressure on the throat area resulting in autonomic overload after the chase and capture, and death follows much more quickly than by simple strangulation. This rapid death occurs in captured birds, even if quickly released after capture by a predator.

TENS machines
These work by fatiguing the sensory and nociceptive nerves by excessive stimulation at the receptor and primary afferent level. Once a receptor has been forced to fire many times, it runs out of neurotransmitters and the nerve no longer feeds to the dorsal horn. This lasts only until the TENS is turned off and the receptors and nerves have time to metabolically recover their previous functions. The dorsal horn also changes its activity, as described by Melzack and Wall.21

The Likon machine
This is a very interesting machine that came from China and was initially related to acupuncture methods. It has multiple settings that, when managed correctly, actually modulates via low wavelength electrotherapy to the autonomic nervous system in two different ways, modulating the sympathetic or parasympathetic systems. The actual settings required, and how to use the machine in the management of muscle problems and specifically in complex regional pain syndromes, were deduced by simple trial and error over some two years by Adelaide physiotherapist Aileen Jefferis.

My wife and I have extensively used this machine with great benefit since 1990 and its use has allowed our development of the whole body functional models from clinical observations.

It cannot be used without careful training by those who have experience in correct Likon use, as it is very easy to incorrectly modulate the wrong part of the autonomic nervous system, resulting in rapid exacerbation of symptoms and signs.

Cold and heat
Considering the physiology, these are easy to understand. Heat increases the blood flow, thus delivering more substrate and removing metabolic products as well as assisting biochemical reactions. Increased blood flow may also cause more swelling when inflammation is present, which may be counterproductive. The nerves can actually work more efficiently and thus, at times, can make the pain appear worse, due to improved nerve conduction rates and increased afferents to the dorsal horn.

Cold, on the other hand, reduces the blood flow and also decreases the function, physiology, and biochemistry of the muscles and nerves and reduces secondary swelling in the area, as well as decreasing afferent nociceptive activity to the dorsal horn, thus reducing pain perception.

Hypnosis
This involves the ability of the brain to be placed in a sleep-like dissociative state where the pain afferents can be "ignored" both by increasing efferent DLF activity as well as cerebral activity to override the areas associated with pain perception.22

Meditation
This works like hypnosis, but in this case the brain is taught to ignore the nociceptive afferents by activating other more dominant pathways. fMRI studies have shown that many areas of the brain are involved in such activity and brain neuro-plasticity is the key to long term benefit of such meditation.14,22

Aromatherapy
This is a most fascinating area of pain management that is only just starting to be understood. Olfactory inputs pass directly to the midbrain and form an important part of Pavlov’s orienting response, as the result is frequently sub-200 milliseconds in onset.8 From birth onwards, various aromas are related to different environments and are learnt as an association memory.

Later, the presence of that same smell can help recreate the memory and may be calming, alerting, or even repulsive, but the response depends on the original environmental association. This is an underlying feature of Pavlov’s original conditioned reflex development and the ability to moderate this by environmental changes was well demonstrated in Stanley Kubrick’s film “A Clockwork Orange”.

Stationary magnets
Despite all the claims for these, there are no blinded, randomized controlled trials that show any positive benefit, other than the extra warmth and support from the magnet carriers.
Complementary Pain Management

The power of mind over matter

As indicated above, there are many ways in which the brain and cerebral interpretation can be fooled into ignoring or downplaying the importance of the afferent nociceptive input. However, if the incoming pain information continues to increase or accumulate, it will eventually override and redirect the brain and body’s attention to the cause as a method of protection from actual or possible injury.

How powerful is this process? Think of the boxer getting his face and nose pummelled, or the rugby player being heavily tackled, or the person in a fight to the death with anything. It is then that survival and function will override the pain. However, eventually, pain perception will dominate because the afferents from the dorsal horns will cause overload in the midbrain autonomic control area. Either action is taken to address the pain and reduce it, or the whole body function will result in loss of consciousness.

The whole body response is summarized as MOMM.

Monitor (M) the environment;
Orient (O) to the changes;
Memory (M) check for past experience of the same; and
Manage (M) by:

a) Freeze/Fear initially while the situation is considered. Remember it is the excessive Freeze/Fear response that may result in death via autonomic overload.
b) Flight/Fight
c) Return to normal.

These are the steps that Pavlov first identified in 1904 to 1917, and now that we are starting to understand brain neuroplasticity better, newer and better pain management programs may be developed.

As I have tried to show here and in previous articles, in musculoskeletal medicine we do need to go back to the basic principles of anatomical and physiological normality to understand what is going on. We must ensure that not only do we make the right diagnosis, but that our management programs are also aimed at restoration of normal, rather than merely treating symptoms and signs after “labeling” of the problem instead of true diagnosis.

References

20. Bogduk N, McKay AB. Email personal communication.
Paraspinal Nerve Injection: Its use as a presurgical diagnostic disambiguation tool and for the temporary and permanent relief of musculoskeletal pain

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History

Paraspinal nerve injection is an economical and minimally invasive procedure that can be used to treat acute spinal pain, or chronic spinal pain that may be associated with radiculopathy. Radiculopathy can be defined in the broadest sense as pain and dysfunction mediated by the radicles or nerve roots, the final common pathway for nociception into the central nervous system (CNS).

The original work using paraspinal nerve injections was a randomized controlled trial (RCT) by Dr Stefan Blomberg as part of his PhD thesis in the early 1990s. Stefan runs a pain clinic in Stockholm, Sweden.1-5

Stefan Blomberg came to Melbourne in October 2002 as keynote speaker for the annual scientific meeting of the Australian Association of Musculoskeletal Medicine (AAMM). His pragmatic approach is systematic, using his own algorithm.

Because of limited sensitivity and specificity of history, examination, and imaging for diagnosing and managing musculoskeletal pain problems, Stefan believes it is humane and cost-effective to offer patients a pragmatic trial of treatment when there is a chance of “cure” or significant reduction in their pain and associated symptoms.

Musculoskeletal pain is a concept, not a well-defined entity.6 It is the brain’s unique interpretation, based on past learning, of chaotic afferent input to the CNS.

The history is the best guide to diagnosing musculoskeletal pain problems.7-9

It is vital to take a thorough history. The aim is to triage the patient with respect to red flag conditions (such as fracture, tumours benign or malignant, infection, or inflammation such as ankylosing spondylitis and other seronegative spondylarthropathies), radicular pain and radiculopathy, and somatic pain which may be associated with referred pain through convergence.7

The red flag checklist has been validated for acute and chronic lumbar spinal pain, and is applicable to other areas of the body.7

Physical examination can support historical impressions and help engage the patient but lacks diagnostic precision without proven reliability and validity.7 It can reveal tender entheses which may be associated with chronic spinal pain.

Imaging does not show pain per se, and in the absence of red flag indicators usually shows non-diagnostic age-related changes that are no more common in people with pain than those without pain.7-9

This evidence can free the practitioner from the need to image patients repeatedly with XRs, CTs, or MRIs whenever they have an exacerbation of their musculoskeletal pain. In the absence of red flag indicators, the yield of relevant information is very low and should not change management. This can save patients needless exposure to ionizing radiation in the case of XR and CT, and expense for the patient, the insurer, and the tax payer, depending on funding arrangements.

History is the best guide to diagnosis but is not completely reliable. Somatic referred pain and radicular pain with or without radiculopathy can co-exist.10

Neurogenic pain is pain evoked by the stimulation of peripheral axons or their cell bodies rather than peripheral nerve endings as occurs with somatic pain.

Radicular pain is a subset of neurogenic pain, in which pain is evoked by stimulation of the nerve roots, dorsal root ganglion (DRG), or spinal nerve itself.11, 12

In neurogenic pain the pain is perceived as arising in the peripheral area supplied by the affected nerve. As the pain is perceived in a region distal to the actual pain stimulus, neurogenic pain and radicular pain are, by definition, a form of referred pain.7

Lumbar radicular pain (LRP) could be referred to an area innervated by either the dorsal or the ventral rami, but pain in the distribution of the dorsal rami has not been properly defined to date. LRP can also result from irritation of the ventral root or the spinal nerve itself.11-14

LRP differs, however, from somatic and visceral referred pain because it does not involve the stimulation of nerve endings, and convergence onto the same second order neuron at the same segmental level. It is perceived as arising distally because of ectopic activation of the nerves supplying that region by irritation of the nerve roots, DRG or spinal nerve proximally. This manifests as referred radicular pain, that is, shooting, stabbing, lancinating, electric shock like pain, with or without dysesthesia to that region.3 This is in contrast to somatic referred pain, which is constant in location but poorly localized and diffuse, and aching in quality.11, 12

 Burning pain is often a feature of neurogenic pain. Deep burning pain without other features, distribution, or qualities is not necessarily neurogenic in origin. Burning sensations in the skin strongly imply a neurogenic process that may be radicular, or some other neurogenic process.7

The sensory dorsal nerve roots receive fibers from both the ventral and the dorsal rami that combine briefly to form...
Paraspinal Nerve Injection

the spinal nerve just distal to the intervertebral foramen. The sinuvertebral nerves (SVNs) are recurrent branches of the ventral rami that re-enter the intervertebral foramina to be distributed within the vertebral canal, formed by a somatic root from a ventral ramus and an autonomic root from a grey ramus communicans. In the intervertebral foramina, the lumbar SVNs run across the back of the vertebral body just below the upper pedicle supplying the periosteum and also the outer third to half of the intervertebral disc, and the posterior longitudinal ligament (PLL) at that level and also the level above. The SVNs also supply the blood vessels of the vertebral canal, the ventral aspect of the dura mater, the dural sleeves enclosing the nerve roots, and the spinal nerves as far as the intervertebral foramina, where the dura merges with and becomes the epiuerium of the spinal nerve. Ascending and descending meningeal branches travel one segment rostrally and two segments caudally respectively. All the nerve roots and the roots of the cauda equina are covered with their own sleeve of pia mater and bathed in cerebrospinal fluid (CSF) which percolates through the subarachnoid space. 

Therefore, somatic pain could be expected as part of any nerve root irritation or compression syndrome via effects on the dural sleeve enclosing the nerve root. This produces nociception from stimulation of nerve endings in the dural sleeve supplied by the SVNs, which is relayed via the ventral rami to the spinal nerve and then the dorsal root before entering the dorsal horn of the spinal cord. 

It is conceivable that radicular pain from ectopic stimulation of the dorsal root or DRG could be referred to the region supplied by the dorsal ramus. This could produce radicular pain in the distribution of the dorsal ramus which supplies the zygapophysial joints, the paraspinus muscles, and the skin in the paraspinal areas. Similarly, it could refer to the regions supplied by the sinuvertebral nerves, that is, the vertebral body, intervertebral disc, PLL, and meningeal blood vessels. 

Radiculopathy and radicular pain involving referral to areas supplied by fibers from the dorsal ramus could be subtle in terms of clinical presentation and is possible from a basic science perspective. It could conceivably be related to a chemical radiculitis due to the release of demyelinating enzymes from an anular fissure with or without an obvious prolapsed intervertebral disc, but also from foraminal stenosis, or other less common causes affecting the ventral ramus.

Perineural fibrosis is another possible cause of radicular pain that could be present in the absence of gross morphology on imaging. It most commonly occurs after disc herniation or after disc surgery, and seems to respond better to local anesthetic and steroid injections if tried before any surgery is attempted.

In patients presenting with features of somatic spinal pain and LRP together on history, it is important that each pain be defined and delineated where possible, as they arise from different anatomical structures and are caused by different pathological mechanisms. 

Radiculopathy may be more rigorously defined as objective loss of sensory and/or motor function as a result of conduction block. It might include numbness, motor loss, wasting, weakness, and loss of reflexes. Paresthesia and or numbness can occur with nerve root compression or nerve root inflammation and signify conduction block. Ill-defined paresthesia can also occur as a manifestation of somatic referred but raises the possibility of associated radiculopathy.

Any lesion that affects the integrity of the lumbosacral nerve root can cause LRP, radiculopathy, or both. Lumbar radicular pain is caused by more than a mass effect. CT and MRI studies have shown that patients whose symptoms of “sciatica” (radicular pain with or without radiculopathy) have resolved often still show the same mass effects on follow-up imaging. Disc herniations or protrusions evident on CT or MRI may not even be associated with low back pain or LRP, and may be totally asymptomatic.

Ectopic impulses, and hence perception of pain, may be generated as a result of:
- mechanical deformation of the DRG
- mechanical stimulation of previously damaged nerve roots
- inflammation of the roots or DRG, related to chemical radiculitis and/or
- possible ischemic damage to the DRG.

Compression of nerve roots occurs a brief discharge at the time of application but then the root becomes silent. DRG compression can produce sustained activity in nociceptive axons and interestingly also Aβ fibers. Clinical experiments have shown compressing normal nerve roots with urinary catheters evokes paresthesia and numbness but not pain.

Prolapsed nucleus pulposis produces chemical radiculitis via phospholipase A2. This explains conduction block in affected nerve roots, but the mechanism of radicular pain is obscure. Ectopic discharges from DRG ischemia is the most likely cause. This defines radicular pain as neurogenic.

However, the pain of radiculitis may also involve the dural sleeve of the affected nerve roots. This is innervated by the SVN; pain may arise from irritation of nerve endings in the dura, which produces somatic pain. Pain from radiculitis may have been misinterpreted, and may not be intrinsically radicular, but may in fact be referred somatic pain from the inflamed dura of the nerve root sleeve.
Radicular pain and somatic referred pain are not mutually exclusive. They can co-exist. Radicular pain may be superimposed on a background of somatic referred pain. Although not absolute, certain features may assist in distinguishing between somatic pain and radicular pain.10

See table below.

Clinical examination does not diagnose the cause of LRP if present, but may help establish the presence or absence of radiculopathy.10

Although lumbar disc herniation is the commonest cause of LRP, there are no distinctive features either in the history or physical examination that would implicate the intervertebral disc as the cause of pain.10

Definitive diagnosis can be made only by imaging studies that support the clinical diagnosis of LRP based on history, and based primarily on the quality of the pain. The straight leg raise clinical test has the best sensitivity, but a low specificity with an average likelihood ratio of 1.5 which is meagre.10 Other tests such as dorsiflexion of the foot, impaired ankle reflex, sensory deficit and muscle atrophy have modest to poor sensitivities and specificities.10

In younger and middle-aged patients the pretest probability of disc herniation is high, whereas in the elderly, foraminal stenosis or spinal stenosis are more likely causes of LRP,10 with or without associated radiculopathy.

The natural history of LRP is that patients can expect a dramatic reduction in the severity of pain, with treatment limited to simple analgesics, and patients should be encouraged to maintain or resume daily activities. For severe pain, opioids can be used judiciously.10

Bed rest is no more effective than watchful waiting. Depending on the severity of the LRP and the response to medication, the early resumption of daily activities should be actively encouraged.

At 12 months, at least 50% of patients can expect to be free of leg pain, but at least 60–70% will continue to experience low back pain.26

This low back pain has been attributed to somatic pain via stimulation of nociceptive nerve endings in the disc or related structures, but it often arises at the same time as the radicular symptoms or signs. It could conceivably be a manifestation of radiculopathy and radicular pain referring to regions supplied by fibers from the dorsal ramus.

Given the favorable natural history of LRP, authorities recommend that in the absence of other indications such as cauda equina syndrome, progressive motor loss, or other red flag features, imaging is not required unless there is failure to improve 4–6 weeks after the onset of LRP.27

Imaging is best reserved for patients not responding to conservative treatment, and for whom surgery is being considered. In patients with a history of “sciatica” or in whom a red flag condition seems likely, appropriate imaging is indicated. MRI is the investigation of choice. It detects all significant red flag and surgical causes, and is radiation free.

Electrophysiological studies in patients presenting with acute LRP are generally not indicated unless peripheral neuropathy instead is suspected clinically. These tests cannot determine the precise spinal nerve level associated with disc herniation and radicular pain. Electromyogram studies correlate poorly with the anatomical level of a disc herniation.27

Electrophysiological studies may also be indicated to exclude more distal nerve damage, verify suspected muscle weakness by needle electromyogram, or to assess pre-operative baseline muscle status before surgery for radiculopathy related to recurrent disc herniation.27

The efficacy of steroid injection for the treatment of LRP may be due to its anti-inflammatory effects on inflamed nerve roots,28 inherent local anesthetic properties,29 or as a membrane stabiliser suppressing ectopic impulses.30

Steroids injected transforaminally under fluoroscopy may offer significant pain relief for an extended period of time.31, 32 The results are less impressive if given by the interlaminar route, but caudal epidural injection may be tried in the rooms if lumbar radicular pain and or radiculopathy is suggested clinically, or the patient has failed to respond to paraspinal nerve injection in a mixed presentation with elements of somatic and radicular pain.33

A transforaminal epidural injection of long acting local anesthetic and corticosteroid should be considered in this setting, given the potential for significant and lasting relief of pain.10

<table>
<thead>
<tr>
<th>Feature</th>
<th>Radicular pain</th>
<th>Somatic referred pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth</td>
<td>Deep as well as superficial</td>
<td>Deep only, lacks any cutaneous quality</td>
</tr>
<tr>
<td>Quality</td>
<td>Shooting, lancinating, like an electric shock</td>
<td>Dull, aching, like an expanding pressure</td>
</tr>
<tr>
<td>Pattern</td>
<td>Narrow band</td>
<td>Wide area</td>
</tr>
<tr>
<td></td>
<td>Travelling</td>
<td>Relatively fixed in location</td>
</tr>
<tr>
<td></td>
<td>Quasi-segmental, but not dermatomal</td>
<td>Quasi-segmental, but not dermatomal</td>
</tr>
<tr>
<td></td>
<td>Not distinguishable by segment</td>
<td>Not distinguishable by segment</td>
</tr>
<tr>
<td>Distribution</td>
<td>Entire length of lower limb</td>
<td>Anywhere in lower limb</td>
</tr>
<tr>
<td>BUT</td>
<td>below knee &gt; above knee</td>
<td>BUT</td>
</tr>
<tr>
<td></td>
<td>proximal &gt; distal</td>
<td></td>
</tr>
</tbody>
</table>

Paraspinal Nerve Injection

Distinguishing features of LRP and somatic referred pain
A recent systematic review concluded that there is moderate evidence for interlaminar epidurals in the cervical spine and limited evidence in the lumbar spine for long-term relief of radicular pain.

The evidence for cervical and lumbar transforaminal epidural steroid injections is moderate for long-term improvement in managing radicular pain. The evidence for caudal epidural steroid injections is moderate for long-term relief in managing radicular pain and chronic low back pain.33

For somatic spinal pain, paraspinal nerve injection should be considered. It blocks nociception into the dorsal horn of the spinal cord via the dorsal nerve root, and can relieve somatic pain while the patient is on the table, allowing a resumption of usual activities of daily living with stretching and walking that can stimulate A\textsubscript{\beta} fibers which detect gross spinal movements. This input rises to the midbrain, and can help to block incoming nociception by increasing descending pain control mechanisms.

Components of radicular pain and or radiculopathy may also be present but clinical examination lacks sensitivity and specificity.10

Imaging with MRI does not always resolve the dilemma as there is an increasing prevalence of false positive MRI findings with age.8, 9

A trial of treatment may relieve distressing symptoms, and avoid expensive and unnecessary imaging. Red flag features are uncommon, accounting for less than 1% of causes of low back pain in a primary care setting.34, 35

In the original trials by Blomberg on subacute low back pain (pain for 6-12 weeks), including patients with radicular leg pain, the average number of parasacrococcygeal paraspinal nerve injections needed for lasting pain relief was 1-2. In Australia, musculoskeletal pain medicine doctors using this approach have found that people with chronic spinal pain of many years’ duration can benefit from on average 1-4 injections given at weekly or greater intervals, with a cumulative effect that seems not diminished by greater intervals between injections.

The importance of diagnosis, education, and assurance cannot be underestimated, with recommendations for light activity, stretching,36 manual therapy if applicable and a graded home exercise program utilizing walking of great benefit in maintaining improvement.

Indications

Primary indications: somatic with or without referred spinal pain, acute, or chronic, possibly associated with radiculopathy. This may be diagnosed prospectively or retrospectively when revealed after overlying somatic pain that has been clouding the clinical picture has resolved with paraspinal nerve injection.

Secondary indications: after failed epidural injection, when undiagnosed or coexistent somatic spinal with or without referred pain is diagnosed or suspected.

Contraindications

Absolute contraindications: red flag conditions; uncorrected or suspected coagulopathy; known allergy to local anesthetic/corticosteroid, for example, lignocaine, Celestone Chronodose, etc.; local infection.

Relative contraindications: anticoagulation therapy - injection is still possible, with careful technique and fine 25G needles if the INR is within recommended reference ranges; frail elderly patients; those prone to vasovagal episodes. Patients with previous spinal surgery and metal internal fixation can be injected, carefully avoiding infiltration close to the metal using a no-touch or sterile technique, aiming to bathe the dorsal ramus and or other paraspinal nerves with local anesthetic with or without corticosteroid.

Equipment

Paraspinal nerve injections can be done with minimal equipment after marking relevant land marks using a no-touch technique in the rooms. Equipment for treating allergic reactions and vasovagal reactions should be readily to hand.

Procedure

The parasacrococcygeal paraspinal nerve injections for lumbar spinal pain are usually done with the patient lying prone or in the lateral position if this is problematic, and the injections are given paraspinally to block the nerves entering the neuraxis adjacent to the edges of the sacrum and coccyx bilaterally.

The injection site can be at C1-2 (coccygeal) as in Blomberg’s original work and involves infiltration up and down beside the sacrum and coccyx, usually bilaterally to block multiple nerve roots and spinal nerves. Entry site of injection can be at other sites parasacrally, such as at S4 or S2, and infiltration performed up and down the

Level of evidence

There is Level II evidence from Stefan Blomberg’s RCT in the early 1990s, using parasacrococcygeal paraspinal injection.1-5 One of the papers was published in Spine in 1994.5

In Australia, there is level IV evidence from some case series.37-41 A pilot study in Queensland has also been conducted. Data collection has been completed, with results currently being analysed.
parasacrococcygeal region with 15 ml of 0.5% plain lignocaine with Celestone Chronodose or comparable steroid solution typically using 2 ampoules, that is, 2 ml in and around the dorsal nerve rami to block nociception entering the dorsal roots and hence dorsal horn segmentally.

A 22 or 23 G 50-60 mm needle or longer spinal needle is used using a no-touch technique. This has the effect of reversing the wind-up and central sensitization that is the usual cause for persistent spinal pain, acutely, or chronically when it can be associated with radiculopathy.

If patients are needle phobic or in significant pain or markedly tender, consider infiltrating first with 5 ml of lignocaine 0.5% either side at the entry site and infiltrating up and down in the usual directions using a 38 mm 25 G needle. This can be done routinely or in selected patients.

There may be variable lumbosacral tenderness, and diagnosis is best made on the basis of history and or pain diagram.

There may be tenderness paraspinally on digital rectal examination and Stefan Blomberg teaches the injection with a per rectal (PR) guiding digit in the anus to assess for tenderness, to guide the depth of injection, and to stretch the parasacrococcygeal ligaments after paraspinal nerve injection has blocked the nociception and relieved the pain. This was used in the published trials so is the preferred method, though omitting this aspect of the injection may still produce an effective outcome if patients are uncomfortable with the idea after informing them of the trial details and evidence. The use of the PR guide could be reserved as an option for later inclusion or consideration if there is failure to respond to an unguided injection without the associated stretching.

Needling of any tender entheses after blocking the dorsal rami may have additional reflex effects to reset the dorsal horn and reverse the wind-up and central sensitization and persistent pain.

Additional paraspinal nerves may be blocked from L1-5 if needed with lignocaine 0.5% using 1.5-2 ml with or without corticosteroid each side for each involved segment, aiming for the inferior aspect of the attachment of the transverse process to the vertebral arch in order to block the dorsal rami. The history, pain diagram, and spinal tenderness and persistence of pain are the best guide to choosing additional spinal nerves to block. It is important to update and review the anatomy regularly.

Similarly, thoracic spinal pain may be treated by injecting the dorsal rami at the inferior aspect of the attachment of the transverse process to the vertebral arch with lignocaine 0.5% using 1.5-2 ml with or without corticosteroid each side at each involved segmental level. The history, pain diagram, and spinal tenderness and persistence of pain are the best guide to choosing additional spinal nerves to block.

Thoracic radicular pain due to disc prolapse is less common than in the lumbar and cervical regions but other mechanisms could be involved. Chronic thoracic spinal pain is more commonly of zygapophysial joint origin, but thoracic radicular pain still occurs, and can be subtle in its clinical presentation.

The cervical transverse process tips can be infiltrated using lignocaine 0.5% with or without dexamethasone, a non-particulate corticosteroid, using 0.4 ml for each spinal nerve from C3-7 especially if tender unilaterally or bilaterally.

Non-particulate steroids are safer in case of intravascular injection.

A study published in Spine in 2007 on cervical transforaminal epidurals steroid injections (TFEIs) looked at 30 cases of brain or spinal cord infarction (16 brain, 12 cervical spinal cord, and 2 combined brain/spinal cord infarcts), over three times greater than the sum of all published infarcts previously (n = 8 published case reports).

Of all the cases reported, four involved only corticosteroid with no local anesthetic. All four cases involved methylprednisolone and resulted in brain infarction, three of which were fatal. This is the strongest association to date between particulate corticosteroids and brain or spinal cord infarctions.

The authors point out that although co-occurrence of alternative mechanisms of injury (for example, vertebral artery dissection or needle-induced vasospasm) is possible, it supports an embolic mechanism of action.

This was the first study to propose a “top of the basilar” artery mechanism, whereby the steroid embolus travels to the confluence of the distal basilar artery, its thalamoperforate branches, the superior cerebellar artery, and the posterior cerebral artery. Occlusion of these arteries gives rise to midbrain, pons, cerebellum, thalamus, and or temporal and occipital lobe infarctions.

In order to minimize the risk of complications, the authors suggest: 1) using real-time fluoroscopy with non-ionic contrast and digital subtraction to maximize detection of vascular uptake during cervical TFEIs; 2) using a test dose of local anesthetic prior to injecting corticosteroid to prevent irreversible neurologic sequelae; 3) using microbore extension tubing to minimize needle movement while changing syringes; 4) using minimal if any sedation to allow for clinical neurologic monitoring; 5) using a shorter-acting local anesthetic such as lignocaine, preferably at the lowest possible concentration and dose, to minimize high spinal anesthesia occurrence or severity; 6) using blunt needles; 7) screening for arterial dissection risk factors; and 8) using a non-particulate corticosteroid such as dexamethasone.

A recent experimental animal study using a rat model suggested that corticosteroids may add no extra efficacy to local anesthetic injection in treating radiculopathy, suggesting that corticosteroid may be unnecessary for nerve root infiltration (NRI).

The paraspinal injection is meant to be extradural but these points are certainly worth bearing in mind. Cervical injections should be attempted only by experienced doctors with a sound knowledge of the anatomy and the
Paraspinal Nerve Injection

Techniques involved.

Cervical paraspinal nerve injection may succeed in blocking nociception arriving in both the dorsal rami supplying the posterior elements of the spine, and ventral rami which transmit nociception from the intervertebral discs and dural sleeves and dura to the dorsal root via the spinal nerve. It may, therefore, help with discogenic pain.

The patient is placed in the lateral position and the tips of the cervical lateral masses or transverse processes are carefully marked, and then a no-touch technique is used. It is important to be aware of the course of the vertebral artery which is exposed between lateral masses. It is prudent to be very gentle and withdraw if the tip is not contacted at the appropriate depth or if there is uncertainty about needle position.

Utilizing a low dose of local anesthetic such as 0.4 ml of 0.5% lignocaine alone with or without non-particulate steroid such as dexamethasone enhances the safety of the procedure.

Alternatively, a posterior approach can be used to block the dorsal rami or at least the medial branches from behind. The injection is approximately on the same level as the spinous process, the articular pillar, and the tips of the cervical lateral masses or transverse processes. The needle can be inserted slightly inferiorly and 2.5 cm lateral to the spinous process and aimed at the posterior aspect of the articular pillar to be blocked. After bony contact is made, the depth of contact is noted and the needle repositioned aiming towards the most lateral aspect of the articular pillar. The 25 G or larger needle is advanced, but, if it walks off laterally, is withdrawn and redirected slightly medially and advanced to the depth of the previous bony contact.

After the needle is felt to be the optimum position, the hub is observed for blood or cerebrospinal fluid. If neither is evident, gentle aspiration can be attempted. If the aspiration test is negative, 1.5 ml of solution of local anesthetic such as lignocaine 0.5% with or without dexamethasone can be injected to block the medial branch of the dorsal ramus at that level.

The patient is usually shown some gentle stretching exercises to use as part of a home-based rehabilitation program 2-3 times daily and also if there is a flare of pain. It is useful to utilize post-isometric relaxation with deep breathing in accordance with Sherrington's second law to achieve a painless stretch. 

Complications

These include bleeding, infection, pain, scarring, inadvertent epidural or subarachnoid injection producing a high spinal anesthetic if sufficient volume and concentration of local anesthetic is injected too close to the neuraxis. A subdural injection can give similar results to a subarachnoid injection but with a prolonged onset time. However, a small dose of dilute 0.5% lignocaine with or without dexamethasone reduces any substantial risk.

Paralysis is possible with injections in the cervical region, into a vertebral or smaller vessel usually with particulate corticosteroid solution via steroid embolus producing thrombosis, cord or brain stem infarction and or death. Injection directly into the spinal cord itself can produce similar, possibly more localized effects.

Alternatives to paraspinal injection include other conservative management; epidural injection may be considered if there is a poor response to paraspinal nerve injection, as radicular pain or even radiculopathy involving fibers related to the dorsal ramus may be present. However, cervical transforaminal epidural steroid injection produces similar, possibly more localized effects.

Referral for formal cervical or lumbar medial branch blocks, with or without radiofrequency neurotomy if positive with controlled blocks, is supported strongly by the evidence base and is the ideal and only proven treatment for Z joint pain but unfortunately is not always available or done to ISIS standards.

Results

The results have been very good empirically and when followed up in clinical practice, but more formal study is strongly indicated, with case series the best way to proceed in private musculoskeletal or general medical practice.

Conclusions

Paraspinal nerve injection offers a valuable way of managing acute pain or chronic pain that may be associated with radiculopathy. This may be fully revealed only after a trial of treatment. It seems to work best for somatic with or without referred spinal pain but this can coexist with or be associated with radicular pain and/or radiculopathy.

The paraspinal nerve injections can also clarify a mixed somatic and radicular presentation and relieve somatic pain which may be associated with radicular pain and/or radiculopathy. This may allow a more precise diagnosis of specific level nerve root impingement syndromes, which may then be more amenable to surgery with microdiscectomy, etc.

Persistent musculoskeletal pain is likely to have a large neuropathic component. Some 20 years ago Professor Bogduk delivered a lecture at an AAMM or APS meeting in which he threatened a nightmare. The proposition was that there was no such entity as musculoskeletal pain: that it was all neuropathic.

Even osteoarthritis is neuropathic (at the micro level). Radiofrequency neurotomy may work not because it...
denervates a painful joint but because the articular nerves are neuropathic, in that some of the afferents are injured. The pain arises not from the joint but because of afferent imbalance. Denaturing the nerves resets the balance. Could the result of the Bone and Joint Decade be: it’s all neural? All treatments work by resetting the dorsal horn?

This is certainly not an argument for more surgery, etc., as surgery is effective only for intractable radicular pain or for other rare red flag conditions such as cauda equina syndrome, unstable fracture, tumour, or infection.

Paraspinal nerve injections may reduce the volume of back surgery and increase the precision of that which is done once pain treatable by injection is removed and the masking is uncovered.

Public pain clinics in Australia are difficult to access, with extremely long waiting lists for persistent non-malignant pain. They usually have a multi-disciplinary approach which may assist function in the short-term but the Australian experience reveals very small and temporary effects, as revealed at the recent AAMM annual scientific meeting in Melbourne. A recent audit in the Newcastle pain clinic showed no change in median pain, no increase in return to work. Similar results from Stephen J Gibson with the Victorian experience revealed very small and temporary effects, which may assist function in the short-term but the Australian experience reveals very small and temporary effects, which may assist function in the short-term but the Austral-ian experience reveals very small and temporary effects, as revealed at the recent AAMM annual scientific meeting in Melbourne. A recent audit in the Newcastle pain clinic showed no change in median pain, no increase in return to work. Similar results from Stephen J Gibson with the Victorian experience showed that one-third of chronic pain patients ended up worse. These patients commonly represent to primary care doctors for further assistance.

We believe that a trial of paraspinal nerve injections is definitely indicated for acute pain not responding to oral analgesia and simple physical measures, for example, stretching, or chronic spinal pain that is commonly associated with radiculopathy, before referral for a surgical opinion. Surgery is not indicated in the absence of red flag conditions as outlined above. Surgery may still have a role for intractable radicular pain after a trial of epidural injection of local anesthetic with or without steroid, either transforaminally or via the caudal route. Paraspinal nerve injection can help clarify the diagnosis and can offer very effective treatment for somatic spinal pain components. It may also work like a selective nerve block if well placed, avoiding the dangers of a transforaminal epidural injection if the injection is lateral to the intervertebral foramina and epidural space. Surgery is indicated for cauda equina syndrome or progressive motor impairment related to radiculopathy.

References
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Paraspinal Nerve Injection


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BACK PAIN


**Objective.** To determine the effectiveness of lessons in the Alexander technique, massage therapy, and advice from a doctor to take exercise (exercise prescription) along with nurse-delivered behavioural counselling for patients with chronic or recurrent back pain.

**Design.** Factorial randomised trial.

**Setting.** 64 general practices in England.

**Participants.** 579 patients with chronic or recurrent low back pain; 144 were randomised to normal care, 147 to massage, 144 to six Alexander technique lessons, and 144 to 24 Alexander technique lessons; half of each of these groups were randomised to exercise prescription.

**Interventions.** Normal care (control), six sessions of massage, six or 24 lessons on the Alexander technique, and prescription for exercise from a doctor with nurse delivered behavioural counselling.

**Main outcome measures.** Roland Morris disability score (number of activities impaired by pain) and number of days in pain.

**Results.** Exercise and lessons in the Alexander technique, but not massage, remained effective at one year (compared with control Roland disability score 8.1: massage -0.58, 95% confidence interval -1.94 to 0.77, six lessons -1.40, -2.77 to -0.03, 24 lessons -3.4, -4.76 to -2.03, and exercise -1.29, -2.25 to -0.34). Exercise after six lessons achieved 72% of the effect of 24 lessons alone (Roland disability score -2.98 and -4.14, respectively). Number of days with back pain in the past four weeks was lower after lessons (compared with control median 21 days: 24 lessons -18, six lessons -10, massage -7) and quality of life improved significantly. No significant harms were reported.

**Conclusions.** One to one lessons in the Alexander technique from registered teachers have long-term benefits for patients with chronic back pain. Six lessons followed by exercise prescription were nearly as effective as 24 lessons. Trial registration: National Research Register N0028108728.

**Comment.** The study was a large, well-conducted randomized controlled trial comparing the short-term and long-term effects of the Alexander technique with massage and normal care and also the effects of an unsupervised home-based aerobic exercise program (prescribed by a general practitioner with follow-up behavioural counselling from a nurse). The Alexander technique was superior to normal care at three and 12 months and to massage at 12 months. It resulted in moderate improvements in disability and pain-free days. Twenty-four lessons were only marginally better than six lessons combined with the exercise program. Interestingly the exercise program provided modest but useful benefits from a relatively brief GP/nurse intervention.

The results suggest that the Alexander technique is a reasonable choice for the treatment of chronic low back pain. It stands alongside other self-management options with some evidence for effectiveness such as supervised tailored exercise programs. When recommending self-management options, patient preferences and expectations should be considered.

The results also offer a weak endorsement for home-based exercise prescribed by a GP with follow-up by a nurse. Massage therapy may offer short term benefits. – Dr Michael Yelland


**Study Design.** A randomized, double-blind, placebo-controlled trial of patients with radicular low back pain who presented to an emergency department (ED) within 1 week of pain onset.

**Objective.** We hypothesized that a single intramuscular 160 mg dose of methylprednisolone acetate would improve pain and functional outcomes 1 month after ED discharge if the corticosteroid were administered early in disease symptomatology.

**Summary of background data.** Parenteral corticosteroids are not recommended for acute, radicular low back pain, though their role in this disease process is ill-defined. To date, this medication class has only been studied in a highly selected group of patients requiring hospitalization.

**Methods.** Adults between the ages of 21 and 50 who presented to an ED with low back pain and a positive straight leg raise test were enrolled. The primary outcome was change in pain intensity on an 11 point numerical rating scale 1 month after ED visit. Secondary outcomes 1 month after ED discharge included analgesic use, functional disability, and adverse medication effects.

**Results.** Six hundred thirty-seven patients were approached for participation, 133 were eligible, and 82 were randomized. Baseline characteristics were comparable between the groups. The primary outcome, a comparison of the mean improvement in pain intensity, favored meth-
Journal Abstracts


Study Design. A systematic review of randomized controlled trials.

Objectives. To assess the effects of nonsteroidal anti-inflammatory drugs (NSAIDs) and COX-2 inhibitors in the treatment of nonspecific low back pain and to assess which type of NSAID is most effective.

Summary of Background Data. NSAIDs are the most frequently prescribed medications worldwide and are widely used for patients with low back pain. Selective COX-2 inhibitors are currently available and used for patients with low back pain.

Methods. We searched the MEDLINE and EMBASE databases and the Cochrane Central Register of Controlled Trials up to and including June 2007 if reported in English, Dutch, or German. We also screened references given in relevant reviews and identified trials. Randomized trials and double-blind controlled trials of NSAIDs in nonspecific low back pain with or without sciatica were included.

Results. In total, 65 trials (total number of patients = 11,237) were included in this review. Twenty-eight trials (42%) were considered high quality. Statistically significant effects were found in favor of NSAIDs compared with placebo, but at the cost of statistically significant more side effects. There is moderate evidence that NSAIDs are not more effective than paracetamol for acute low back pain, but paracetamol had fewer side effects. There is moderate evidence that NSAIDs are not more effective than other drugs for acute low back pain. There is strong evidence that various types of NSAIDs, including COX-2 NSAIDs, are equally effective for acute low back pain. COX-2 NSAIDs had statistically significantly fewer side effects than traditional NSAIDs.

Conclusion. The evidence from the 65 trials included in this review suggests that NSAIDs are effective for short-term symptomatic relief in patients with acute and chronic low back pain without sciatica. However, effect sizes are small. Furthermore, there does not seem to be a specific type of NSAID, which is clearly more effective than others. The selective COX-2 inhibitors showed fewer side effects compared with traditional NSAIDs in the randomized controlled trials included in this review. However, recent studies have shown that COX-2 inhibitors are associated with increased cardiovascular risks in specific patient populations.

Comment. Here is the latest review on NSAIDs for low back pain for the Cochrane Collaboration http://www.cochrane.org/.

It is consistent with previous reviews, showing short-term symptomatic relief in patients with acute and chronic low back pain without sciatica. Effect sizes are small, and there is no long-term effect on the natural history of chronic spinal pain. Side-effects continue to be an issue and these agents must be used cautiously if at all. Analgesics are often preferable and safer from both GI and cardiovascular perspectives. – Dr David Roselt


Study Design. A randomized controlled study of percutaneous radiofrequency neurotomy was conducted in 40 patients with chronic low back pain (20 active and 20 controls).

Objective. The aim of the study was to evaluate the...
possible beneficial effect of percutaneous radiofrequency zygapophysial joint neurotomy in reducing pain and physical impairment in patients with pain from the lumbar zygapophysial joints, selected after repeated diagnostic blocks.

Summary of Background Data. Facet or zygapophysial joint pain may be one of the causes of chronic low back pain and may be treated by a percutaneous radiofrequency denervation. Patients may possibly be identified by a positive diagnostic block. These blocks need to be repeated as false positive responses to single blocks occur. In all previous studies patients treated with radiofrequency denervation have been selected after single diagnostic blocks resulting in a varying degree of relief.

Methods. All patients were examined by an orthopedic surgeon before and 6 months after the treatment (sham or active). Inclusion criteria were 3 separate positive facet blocks. Denervation was achieved by multiple lesions at active). Inclusion criteria were 3 separate positive facet blocks. Denervation was achieved by multiple lesions at each level in an effort to provide effective denervation.

Results. The active treatment group showed statistically significant improvement not only in back and leg pain but also back and hip movement as well as the sacro-iliac joint test. Pre operative sensory deficit and weak or absent ankle reflex normalized (P < 0.01) and (P < 0.05), respectively. There was significant improvement in quality of life variables, global perception of improvement, and generalized pain. The improvement seen in the active group was significantly greater than that seen in the placebo group with regard to all the above-mentioned variables. None of our patients had any complication other than transient postoperative pain that was easily managed.

Conclusion. Our study indicates that radiofrequency facet denervation is not a placebo and could be used in the treatment of carefully selected patients with chronic low back pain.

Comment. In a point of view, Professor Nik Bogduk in the same copy of *Spine* points out that a previous outcome study by Dreyfuss et al. showed excellent results could be achieved with lumbar medial branch neurotomy. However, that study was criticized for having no controls, for being highly selective in its recruitment criteria, for enrolling only a small proportion of potential patients, and for being too small a study. Later systematic reviews of lumbar medial branch neurotomy demanded randomized controlled trials, but none of the controlled trials that followed used correct surgical technique for this procedure, and none selected patients on the basis of controlled diagnostic blocks.  

This is first study since Dreyfuss et al. to use controlled diagnostic blocks to select patients, and the first to use approved ISIS techniques.

Professor Nik Bogduk points out also that Dr Nath did not select ideal patients, free of comorbidity, with good function, and no depression. His patients were enrolled from a pain clinic population, and they had other comorbidities. Nevertheless, they were able to identify a component of their pain that was completely relieved by controlled medial branch blocks. It was this pain that was treated in a placebo-controlled trial of lumbar medial branch neurotomy.

The results clearly show that the effects of lumbar medial branch neurotomy cannot be attributed to placebo effects. The effects are real. This treatment did not as expected relieve every pain the patients had, that were not the target of the intervention. The index pain was relieved as postulated, and corroborated by improvements in function.

Lumbar medial branch neurotomy as a monotherapy is not indicated for all patients with back pain. However, the study showed that medial branch neurotomy could be a complementary therapy in patients typical of a pain clinic population. It also dispelled accusations that lumbar medial branch neurotomy is a placebo.

Professor Bogduk wonders whether critics will next complain that Dr Nath should have recruited highly selected, ideal patients, with pure zygapophysial joint pain, with no comorbidity, and who would be atypical of patients seen in conventional practice.


— Dr David Roselt


The management of chronic low back pain (CLBP) has proven to be very challenging in North America, as evidenced by its mounting socioeconomic burden. Choosing amongst available nonsurgical therapies can be overwhelming for many stakeholders, including patients, health providers, policy makers, and third-party payers. Although all parties share a common goal and wish to use limited healthcare resources to support interventions most likely
to result in clinically meaningful improvements, there is often uncertainty about the most appropriate intervention for a particular patient. To help understand and evaluate the various commonly used nonsurgical approaches to CLBP, the North American Spine Society has sponsored this special focus issue of Spine Journal, titled “Evidence-Informed Management of Chronic Low Back Pain Without Surgery”. Articles in this special focus issue were contributed by leading spine practitioners and researchers, who were invited to summarize the best available evidence for a particular intervention and encouraged to make this information accessible to nonexperts. Each of the articles contains five sections (description, theory, evidence of efficacy, harms, and summary) with common subheadings to facilitate comparison across the 24 different interventions profiled in this special focus issue, blending narrative and systematic review methodology as deemed appropriate by the authors. It is hoped that articles in this special focus issue will be informative and aid in decision making for the many stakeholders evaluating nonsurgical interventions for CLBP.

Comment. This common abstract applies to this and a series of reviews of these topics in Spine Journal in the first edition this year. This edition looks at evidence-informed management of chronic low back pain with 24 interventions including epidural steroid injections, massage, trigger point injections, functional restoration, and many others. – Dr David Roselt


Background. The sacroiliac joint is a diarthrodial synovial joint with abundant innervation and capability of being a source of low back pain and referred pain in the lower extremity. There are no definite historical, physical, or radiological features to provide definite diagnosis of sacroiliac joint pain, although many authors have advocated provocational maneuvers to suggest sacroiliac joint as a pain generator. An accurate diagnosis is made by controlled sacroiliac joint diagnostic blocks. The sacroiliac joint has been shown to be a source of pain in 10% to 27% of suspected cases with chronic low back pain utilizing controlled comparative local anesthetic blocks. Intraarticular injections, and radiofrequency neurotomy have been described as therapeutic measures. This systematic review was performed to assess diagnostic testing (non-invasive versus interventional diagnostic techniques) and to evaluate the clinical usefulness of interventional techniques in the management of chronic sacroiliac joint pain.

Objective. To evaluate and update the available evidence regarding diagnostic and therapeutic sacroiliac joint interventions in the management of sacroiliac joint pain.

Study Design. A systematic review using the criteria as outlined by the Agency for Healthcare Research and Quality (AHRQ), Cochrane Review Group Criteria for therapeutic interventions and AHRQ, and Quality Assessment for Diagnostic Accuracy Studies (QUADAS) for diagnostic studies.

Methods. The databases of EMBASE and MEDLINE (1966 to December 2006), and Cochrane Reviews were searched. The searches included systematic reviews, narrative reviews, prospective and retrospective studies, and cross-references from articles reviewed. The search strategy included sacroiliac joint pain and dysfunction, sacroiliac joint injections, interventions, and radiofrequency.

Results. The results of this systematic evaluation revealed that for diagnostic purposes, there is moderate evidence showing the accuracy of comparative, controlled local anesthetic blocks. Prevalence of sacroiliac joint pain is estimated to range between 10% and 27% using a double block paradigm. The false-positive rate of single, uncontrolled, sacroiliac joint injections is around 20%. The evidence for provocative testing to diagnose sacroiliac joint pain is limited. For therapeutic purposes, intraarticular sacroiliac joint injections with steroid and radiofrequency neurotomy were evaluated. Based on this review, there is limited evidence for short-term and long-term relief with intraarticular sacroiliac joint injections and radiofrequency thermoneurolysis.

Conclusions. The evidence for the specificity and validity of diagnostic sacroiliac joint injections is moderate. The evidence for accuracy of provocative maneuvers in diagnosis of sacroiliac joint pain is limited. The evidence for therapeutic intraarticular sacroiliac joint injections is limited. The evidence for radiofrequency neurotomy in managing chronic sacroiliac joint pain is limited.

Comment. There is no doubt that sacroiliac joints are innervated and are capable of producing low back and referred pain in the lower extremity. The authors point out that the literature on diagnostic sacroiliac joint injections and non-invasive diagnostic techniques is superior to the literature on therapeutic interventions. Due to the lack of significant literature, the level of evidence was low for therapeutic interventions. It is important that previous studies are replicated and high quality evidence produced. The full text article is available free at www.painphysicianjournal.com.


NECK PAIN

Buitenhuis J, de Jong PJ, Jaspers JP, Groothoff JW.
Catastrophizing and causal beliefs in whiplash. *Spine* 2008;33(22): 2427-33; discussion 2434. Medical Department, Unive Insurance and Department of Social Medicine, University Medical Center Groningen, University of Groningen, the Netherlands. j.buitenhuis@unive.nl.

**Study Design.** Prospective cohort study.

**Objective.** This study investigates the role of pain catastrophizing and causal beliefs with regard to severity and persistence of neck complaints after motor vehicle accidents.

**Summary of Background Data.** In previous research on low back pain, somatoform disorders and chronic fatigue syndrome, pain catastrophizing and causal beliefs were found to be related to perceived disability and prognosis. Furthermore, it has been argued with respect to whiplash that culturally dependent symptom expectations are responsible for a chronic course.

**Methods.** Individuals involved in traffic accidents who initiated compensation claim procedures with a Dutch insurance company were sent questionnaires (Q1) containing the Neck Disability Index, the Pain Catastrophizing Scale, and the Causal Beliefs Questionnaire-Whiplash. Of 1252 questionnaires dispatched, 747 (59.7%) were returned. Only car occupants with neck complaints were included in this study (n = 140). Complaints were monitored using additional questionnaires administered 6 (Q2) and 12 months (Q3) after the accident.

**RESULTS:** Pain catastrophizing and causal beliefs were related to the severity of concurrent whiplash disability. The severity of initial complaints was related to the severity and persistence of whiplash complaints. Attributing initial neck complaints to whiplash was found to predict the persistence of disability at 6 and 12 months follow-up, over and above the severity of Groningen, the Netherlands.

**Conclusion.** The results suggest that causal beliefs may play a major role in the perceived disability and course of neck complaints after motor vehicle accidents, whereas pain catastrophizing is predominantly related to concurrent disability. The current findings are consistent with the view that an early conviction that neck complaints are caused by the medico-cultural entity whiplash has a detrimental effect on the course of symptoms.

**Comment:** This study confirms what musculoskeletal pain medicine practitioners have suspected about the importance of patients' beliefs and catastrophizing in determining outcomes. The importance of early intervention in assessment, with history and examination, education, assurance, maintenance of usual activities of daily living (ADLs), and light activity as much as possible, is apparent. The use of local heat to relax the muscles, and gentle range of movement exercises, using deep breathing in the form of post-isometric relaxation home exercises, along with simple analgesics and/or opioids briefly if necessary, seem to go a long way to manage pain and prevent disability before the lawyers become involved or non-evidence-based input occurs. – Dr David Roselli


**Background:** Chronic mid back and upper back pain caused by thoracic facet joints has been reported in 34% to 48% of the patients based on the responses to controlled diagnostic blocks. Systematic reviews have established moderate evidence for controlled comparative local anesthetic blocks of thoracic facet joints in the diagnosis of mid back and upper back pain, moderate evidence for therapeutic thoracic medial branch blocks, and limited evidence for radiofrequency neurotomy of therapeutic facet joint nerves.

**Objectives.** To determine the clinical utility of diagnostic and therapeutic thoracic facet joint interventions in diagnosing and managing chronic upper back and mid back pain.

**Study Design.** Systematic review of diagnostic and therapeutic thoracic facet joint interventions.

**Methods.** Review of the literature for utility of facet joint interventions in diagnosing and managing facet joint pain was performed according to the Agency for Healthcare Research and Quality (AHRQ) criteria for diagnostic studies and observational studies and the Cochrane Musculoskeletal Review Group criteria as utilized for interventional techniques for randomized trials. The level of evidence was classified as Level I, II, or III based on the quality of evidence developed by United States Preventive Services Task Force (USPSTF) for therapeutic interventions. Recommendations were based on the criteria developed by Guyatt et al. Data sources included relevant literature of the English language identified through searches of Medline and EMBASE from 1966 to July 2008 and manual searches of bibliographies of known primary and review articles. Results of the analysis were performed for diagnostic and therapeutic interventions separately.

**Outcome Measures.** For diagnostic interventions, studies must have been performed utilizing controlled local anesthetic blocks. For therapeutic interventions, the primary outcome measure was pain relief (short-term relief = up to 6 months and long-term relief > 6 months) with secondary outcome measures of improvement in functional status, psychological status, return to work, and reduction in opioid intake.

**Results.** Based on the controlled comparative local anesthetic blocks, the evidence for the diagnosis of thoracic facet joint pain is Level I or II-1. The evidence for therapeutic thoracic medial branch blocks is Level I or II-1. The recommendation is IA or 1B/strong for diagnostic and therapeutic medial branch blocks.

**Conclusion.** The evidence for the diagnosis of thoracic facet joint pain with controlled comparative local anesthetic blocks is Level I or II-1. The evidence for therapeutic facet joint interventions is Level I or II-1 for medial branch
blocks. Recommendation is 1A or 1B/strong for diagnostic and therapeutic medial branch blocks.

Comment. This is good evidence for this common condition and supports the use of medial branch blocks (MBBs) for diagnosis and treatment of thoracic zygapophysial joint pain.

No evidence is available for thoracic intraarticular injections and radiofrequency neurotomy. Intraarticular blocks with local anesthetic with or without steroid are not supported by the same level of evidence as MBBs, and certainly seem to give disappointing results in clinical practice, and appear to be of very limited value.

The full text article is available free at www.painphysicianjournal.com or more simply via the top right hand corner of the Pubmed abstract available at http://www.ncbi.nlm.nih.gov/pubmed/. – Dr David Roselt


Study Design. A double-blind, randomized, controlled trial.

Objective. To determine the clinical effectiveness of therapeutic local anesthetic cervical medial branch blocks with or without steroid in managing chronic neck pain of facet joint origin.

Summary of Background Data. The prevalence of persistent neck pain, secondary to involvement of cervical facet or zygapophysial joints, has been described in controlled studies as varying from 39% to 67%. Intraarticular injections, medial branch nerve blocks, and neurolysis of medial branch nerves have been described in managing chronic neck pain of facet joint origin.

Methods. A total of 120 patients were included, with 60 patients in each of the local anesthetic and steroid groups. All the patients met the diagnostic criteria of cervical facet joint pain by means of comparative, controlled diagnostic blocks, and the inclusion criteria. Group I consisted of medial branch blocks with bupivacaine. Group II consisted of cervical medial branch blocks with bupivacaine and steroid. Numerical pain scores, Neck Disability Index, opioid intake, and work status were evaluated at baseline, 3 months, 6 months, and 12 months.

Results. Significant pain relief (>or=50%) and functional status improvement was observed at 3 months, 6 months, and 12 months in over 83% of patients. The average number of treatments for 1 year was 3.5 +/- 1.0 in the nonsteroid group and 3.4 +/- 0.9 in the steroid group. Duration of average pain relief with each procedure was 14 +/- 6.9 weeks in the nonsteroid group, and it was 16 +/- 7.9 weeks in the steroid group. Significant relief and functional improvement was reported for 46 to 48 weeks in a year.

Conclusion. Therapeutic cervical medial branch nerve blocks, with or without steroids, may provide effective management for chronic neck pain of facet joint origin.

Comment. This study shows therapeutic efficacy from repeated cervical medial branch blocks that could represent another option for treating chronic somatic cervical spinal pain. Paraspinal cervical nerve injections performed in the rooms that seem to have a very beneficial clinical effect may be working along similar lines in a less targeted non-specific fashion. Both treatments may be worthy of further study. – Dr David Roselt

PHI 34.05.2012 18:27


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foraminal (n = 6) and extraforaminal portions (n = 6). CT discography showed a leak of contrast from anular tear to the perianular regions. Pain reproduction at contrast leak level during discography showed concordant pain. There was an apparent correlation between perianular enhancement on MRI and clinical symptoms or provocative epidural nerve root injection in all cases.

Conclusion. The perianular enhancement adjacent to anular tears on MRI may be relevant in the diagnosis of symptomatic chemical radiculitis.

Comment. Many studies document that irritation of adjacent nerve roots by a chemical mediator of inflammation from the nucleus pulposus may result in radiculopathy. Common recognized causes of sciatica include disc herniation and spinal stenosis, central or foraminal, but some patients presenting with radiculopathy can show no evidence of nerve root compression on magnetic resonance imaging (MRI).

Patients with anular tears may experience low back pain and radiation into the lower limb. The posterior aspects of the disc and posterior longitudinal ligament are innervated by the sinuvertebral nerves. The posterolateral aspects of the discs receive branches from adjacent ventral primary rami and from rami communicantes near their junction with ventral primary rami. The lateral aspects of the discs receive other branches from the rami communicantes.

Marshall et al. proposed the concept of chemical radiculitis because of the rupture of the anulus fibrosus and dissemination of disc fluid along nerve roots. If anular tears occur, these nerve endings may be irritated by the acid metabolites, that is, phospholipase (PL) A2 and prostaglandin (PG) E2 contained in the material from the herniating disc.

The pain associated with anular tears has been termed discogenic, and it has been shown to radiate to the legs in the absence of nerve compression. Kayama et al. suggest, based on data from dog models, that leakage of nucleus pulposus material from anular tears, with injury to adjacent nerve roots, might be one pathophysiologic mechanism in patients with low back pain and sciatica without radiologic evidence of disc herniation.

Peng et al. reported that there was a significant positive correlation between the site of anular tear and the side of radiating pain. Anular tears manifest on MRI as a high-intensity zone (HIZ). The presence of HIZs within the posterior anulus seen on T2-weighted MRI has aroused great interest and even controversy regarding whether the HIZs was closely associated with a concordant pain response on awake discography.

However, MRI studies about chemical radiculitis associated with anular tears are rare. Peng et al. investigated the histologic features of 19 specimens of lumbar intervertebral discs from 17 patients with discogenic low back pain during posterior lumbar interbody fusion. They suggested that the zone of granulation tissue with extensive innervations along the tears in the posterior part of the painful disc may be responsible for causing the pain of discography and of discogenic low back pain. Also, they described the adjacent tissues surrounding the anular tears replaced by disorganized and vascularized granulation and scar tissue.

Crock in 1986 raised the concept of internal disc disruption, suggesting that trauma to the intervertebral disc resulted in the production of inflammatory substances within the nucleus pulposus that could have local autoimmune effects causing back pain, and chemical effects on the adjacent nerve roots result in leg pain, but typically no neurologic deficit.

There were several studies for chemical radiculitis, but noninvasive diagnostic imaging studies are rare. In the current study, contrast-enhanced axial T1-weighted images showed enhancement at regions of all perianular inflammations and anular tears. The authors suggest enhancement at perianular regions and anular tears is caused by break-down of the normal vessel wall barrier to the diffusion of gadolinium out of the vessels around vascularized granulation.

Detection of perianular inflammation on noncontrast T1- and T2-weighted images is difficult because there is similar signal intensity at the site of inflammation compared to adjacent normal structures, such as nerve roots and epidural fat. This is why contrast enhanced axial T1-weighted images with fat suppression are important for detection and diagnosis of perianular inflammation.

Non-specific HIZs on T2-weighted images seems to be a reliable marker of painful anular disruption. The authors suggest that perianular enhancement associated with anular tears on MRI could be considered as a reliable new marker of symptomatic anular tears in the diagnosis of radiculopathy. Limitations of the study included the small number of patients. Also provocative CT discography was not performed in all cases. It is possible that no perianular enhancement on MRI can be detected in some cases with chemical radiculitis. Further study may be required to assess the sensitivity of this test. The use of gadolinium adds extra cost and additional time due to extra sequences so gadolinium-enhanced MRI generally is not a routine examination of patients with radiculopathy. However, the authors suggest gadolinium-enhanced study with fat suppression is helpful for detecting perianular enhancement by the inflammatory cytokines if patients presenting with radiculopathy reveal no evidence of nerve root compression or spinal stenosis on noncontrast MRI.

Provocative CT discography is the best examination for diagnosing discogenic chemical radiculitis but is too invasive. Contrast MRI is an excellent modality for replacing invasive provocative CT discography and diagnosing chemical radiculitis. The authors suggest that perianular enhancement adjacent to anular tears on MRI may be relevant in the diagnosis of symptomatic chemical radiculitis.
Journal Abstracts


–Dr David Roselt


Study Design. In a prospective cohort study 532 patients with rheumatoid arthritis (RA) and subluxations of the cervical spine were consecutively collected during 1974-1999. OBJECTIVE: The aims of the study were to assess important factors affecting the mortality rate and the timing of surgical intervention.

Summary of Background Data. The average follow-up time from the first visit to death or to the end of the study was 8.5 (SD, 5.7) years. Of the 217 operated patients 144 (66%) died, and of the 315 nonoperated patients 137 (43%) died.

Methods. Patients were selected for operative intervention based on anterior, vertical and subaxial subluxations, pain, and/or cervical neurology. Survival analyses were used for comparisons between patients with RA and the normal population, and between the operated and those treated conservatively.

Results. The survival rate for all RA patients was significantly reduced when compared with average survival in Norway (P < 0.001). The operated group had a significantly lower survival rate than the nonoperated group. In patients with severe instability of the cervical spine, the defined selection criteria for surgical intervention were specific. By comparison of calculated propensity scores, the operated and nonoperated groups were too different to be directly comparable. After surgery only 11 patients (5%) experienced residual pain in the neck or neurologic symptoms. None of these patients were alive at the end of the study, signifying that residual pain or neurologic symptoms are poor prognostic signs (P = 0.015). In the operated group, anterior subluxation and vertical settling greater than the lower indication limits did not have a significant influence on the survival rate, but there was a reduced survival for patients with subaxial subluxations. A clear association was found between increased vertical settling and sudden death.

Conclusion. RA with neck involvement is a progressive and serious condition with reduced lifetime expectancy. Hence, our interpretation is that operative intervention improves local symptoms and most likely changes the condition from worse to better by increasing lifetime expectancy in high risk patients. Since the peri- and postoperative complications are few, a changed attitude toward more liberal indications for earlier surgery may reduce the symptoms and the mortality rate even more.

Comment. It is important to be mindful of possible cervical spine involvement in rheumatoid arthritis (RA) and to consider early referral for surgical assessment or review in these patients if symptomatic or if clinically significant disease is detected incidentally, such as during pre-operative assessment for other surgery. – Dr David Roselt


Study Design. Retrospective study.

Objectives. To investigate the 10-year survival of a large number of elderly patients who underwent spine surgery for lumbar spinal stenosis, and to identify significant risk factors and compare them with age- and gender-matched controls from the general population.

Summary of Background Data. There have been many studies on treatment options and surgical outcomes for lumbar spinal stenosis. However, survival outcomes after lumbar spinal stenosis surgery have not previously been studied. Because these operations are usually performed for elderly patients, we consider patient survival or life expectancy to be a significant outcome measure.
Methods. Between January 1997 and June 2006, patients underwent spine surgery for lumbar spinal stenosis. The date of death was verified using records from the National Health Insurance Corporation. Cumulative 10-year survival was calculated using the Kaplan-Meier method, and the survival of patients who had undergone spine surgery was compared to that of age- and sex-matched members of the general population. A Cox multivariate regression analysis was used in order to compare the survival rates for different covariates.

Results. Using Kaplan-Meier curves, the overall 10-year survival was 87.8% in patients 60 to 70 years old at surgery, and 83.8% in patients 70 to 85 years old at surgery. The 10-year survival rate of female patients and patients who underwent fusion surgery were higher than those of male patients and patients with nonfusion surgery. Compared to the adjusted corresponding portion in general population, the standardized mortality ratios were 0.21, 0.53, and 0.45 in patients aged 50 to 59, 60 to 69, and 70 to 85, respectively.

Conclusion. Elderly patients who underwent spine surgery for spinal stenosis had reduced mortality compared to the corresponding portion of the general population. Therefore, surgery for spinal stenosis is a justifiable procedure even in elderly patients.

Comment. This study looks at the 10-year survival of 1015 elderly patients who underwent spine surgery for lumbar spinal stenosis and shows pretty favorable outcomes, with substantially reduced mortality compared to that of the general population. It seems quite reasonable to consider referring for a surgical opinion even elderly patients with significantly limiting spinal claudication. – Dr David Roselt


Background. Epidural injection of corticosteroids is one of the most commonly used interventions in managing chronic spinal pain. However, there has been a lack of well-designed randomized, controlled studies to determine the effectiveness of epidural injections. Consequently, debate continues as to the value of epidural steroid injections in managing spinal pain.

Objective. To evaluate the effect of various types of epidural steroid injections (interlaminar, transforaminal, and caudal), in managing various types of chronic spinal pain (axial and radicular) in the neck and low back regions.

Study design. A systematic review utilizing the criteria established by the Agency for Healthcare Research and Quality (AHRQ) for evaluation of randomized and non-randomized trials, and criteria of Cochrane Musculoskeletal Review Group for randomized trials were used.

Methods. Data sources included relevant English literature performed by a librarian experienced in Evidence Based Medicine (EBM), as well as manual searches of bibliographies of known primary and review articles and abstracts from scientific meetings within the last 2 years. Three reviewers independently assessed the trials for the quality of their methods. Subgroup analyses were performed among trials with different control groups, with different techniques of epidural injections (interlaminar, transforaminal, and caudal), with different injection sites (cervical/thoracic, lumbar/sacral), and with timing of outcome measurement (short- and long-term).

Outcome Measures. The primary outcome measure is pain relief. Other outcome measures were functional improvement, improvement of psychological status, and return to work. Short-term improvement is defined as 6 weeks or less, and long-term relief is defined as 6 weeks or longer.

Results. In managing lumbar radicular pain with interlaminar lumbar epidural steroid injections, the evidence is strong for short-term relief and limited for long-term relief. In managing cervical radiculopathy with cervical interlaminar epidural steroid injections, the evidence is moderate. The evidence for lumbar transforaminal epidural steroid injections in managing lumbar radicular pain is strong for short-term and moderate for long-term relief.

The evidence for cervical transforaminal epidural steroid injections in managing cervical nerve root pain is moderate. The evidence is moderate in managing lumbar radicular pain in post lumbar laminectomy syndrome. The evidence for caudal epidural steroid injections is strong for short-term relief and moderate for long-term relief, in managing chronic pain of lumbar radiculopathy and postlumbar laminectomy syndrome.

Conclusion. There is moderate evidence for interlaminar epidurals in the cervical spine and limited evidence in the lumbar spine for long-term relief. The evidence for cervical and lumbar transforaminal epidural steroid injections is moderate for long-term improvement in managing nerve root pain. The evidence for caudal epidural steroid injections is moderate for long-term relief in managing nerve root pain and chronic low back pain.

Comment. This paper from 2007 provides supportive evidence for the use of epidural injection of corticosteroids in managing chronic spinal pain. The authors conclude that there is moderate evidence for lumbar transforaminal epidural steroid injections for long-term improvement in managing nerve root pain. The evidence for caudal epidural steroid injections is moderate for long-term relief in managing nerve root pain and chronic low back pain.

The full text article is available free at www.painphysicianjournal.com or more simply via the top right hand corner of the Pubmed abstract available at http://www.ncbi.nlm.nih.gov/pubmed. – Dr David Roselt
Tachiara H, Sekiguchi M, Kikuchi S, Konno S. Do corticosteroids produce additional benefit in nerve root infiltration for lumbar disc herniation? Spine 2008; 33(7): 743-47. Department of Orthopaedic Surgery, Fukushima Medical University School of Medicine, Fukushima, Japan. tachi@fmu.ac.jp.

**Study Design.** Experimental animal study.

**Objective.** To determine whether corticosteroids produce additional benefit to nerve root infiltration (NRI) for experimental lumbar disc herniation.

**Summary of Background Data.** NRI is used for nonsurgical treatment of radicular symptoms caused by lumbar disc herniation or lumbar spinal canal stenosis. Various studies have shown that NRI using local anesthetic or combinations of local anesthetic and corticosteroid can provide both short- and long-term pain relief. However, whether corticosteroids produce additional benefit to NRI remains controversial.

**Methods.** A total of 174 adult female Sprague-Dawley rats were used in this study. The left L5 nerve root and dorsal root ganglion (DRG) were exposed. For the nontreatment group, autologous nucleus pulposus was harvested from the tail and applied to the DRG. For treatment groups, 1% lidocaine (Lido group), 0.4% dexamethasone (Dexa group), 1% lidocaine + 0.4% dexamethasone (Lido + Dexa group), or saline (Saline group) was injected into the underlayer of epineurium just distal to the nucleus pulposus. At 2, 7, 14, and 21 days after surgery, withdrawal threshold was determined using the von Frey test for mechanical allodynia. Expression of tumor necrosis factor (TNF)-alpha in the DRG was examined by immunohistochemical analyses and immunoblotting.

**Results.** Withdrawal threshold decreased in the nontreatment group from day 2 to day 14. Conversely, Lido, Dexa, and Lido + Dexa groups showed no decreases in withdrawal thresholds, and no significant differences were observed among these 3 groups. Immunohistochemical analyses showed that TNF-alpha was localized in DRG neurons in all groups. Immunoblotting showed that expression of TNF-alpha in the DRG was lower in Lido, Dexa, and Lido + Dexa groups than in the nontreatment group. No significant differences were observed among these 3 groups.

**Conclusion.** NRI prevented mechanical allodynia. However, no additional benefit from using corticosteroid was identified, suggesting that corticosteroid may be unnecessary for NRI.

**Comment.** This rat model suggests that corticosteroids may add no extra efficacy to local anesthetic injection in treating radiculopathy.

Nerve root infiltration (NRI), also known as transforaminal epidural steroid injection (TFESI), is used for nonsurgical treatment of radicular symptoms caused by lumbar disc herniation (LDH) or lumbar spinal canal stenosis.

Paraspinal nerve injections may also have a beneficial effect at this level on somatic components of radiculopathy, and represent in part a non-target-specific ESI that is possible in the rooms.

Various studies have shown that NRI using local anesthetic or a combination of local anesthetic and corticosteroid can provide both short- and long-term pain relief.1-6

Recent studies have reported that, not only mechanical compression due to intervertebral disc protrusion, but also nociceptive and inflammatory mediators originating from the nucleus pulposus (NP) play important roles in the onset of sciatic pain in LDH.7-13

Local injection of corticosteroid to provide anti-inflammatory effects to the affected nerve root appears to be a promising approach. Interestingly, whether corticosteroids produce additional benefit in NRI is still controversial.

A randomized, double-blinded, controlled trial described the effectiveness of corticosteroids, with 67% of patients in the group receiving both local anesthetic and steroid avoiding the need for operative intervention, compared with 28% in the group receiving local anesthetic alone.6 However, another randomized, double-blinded, controlled trial found that corticosteroids produced no additional benefit to NRI using local anesthetic agents for the treatment of chronic radicular pain.4

Another randomized, double-blinded trial showed that the combination of corticosteroids and bupivacaine had short-term effects, but the steroid group experienced a "rebound" phenomenon at three and six months.2

The present results indicate that NRI prevented mechanical allodynia and decreased expression of TNF-alpha in the DRG for experimental LDH. However, no additional benefit from using corticosteroid in NRI was demonstrated.

The therapeutic mechanisms of NRI using local anesthetic have been investigated. Application of NP to the nerve root induces an increase in endoneurial fluid pressure (EFP) and a decrease of blood flow in the DRG.14

Increased pressure is caused by interference with capillary flow and intraneurial edema, followed by breakdown of the myelin sheath and other cytoplasmic components of Schwann cells and the axon.15,16 These changes are thought to represent an important pathogenic mechanism associated with sciatica caused by disc herniation.

Lidocaine reportedly reduces the increase in EFP and pathophysiological changes in the DRG induced by NP.17

Increased intradiscal blood flow has also been observed in compressed spinal nerve roots after NRI with lidocaine.18

NRI with lidocaine may thus exert therapeutic effects by improving EFP and blood flow in the DRG.

Furthermore, a relationship between lidocaine and acid-sensing ion channel 3 (ASIC3) has recently been reported.19

ASIC3 is a sodium channel associated with acidosis and increased inflammatory pain. ASIC3 is up-regulated in DRG neurons following the disc herniation model. Lido-
caine decreases ASIC3 expression in DRG neurons and the pain associated with the disc herniation model. These findings suggest the possibility that lidocaine decreases acidosis by increasing blood flow.

In addition, Hasue suggested that lidocaine breaks up the vicious circle of pain, desensitizing the central and peripheral nervous systems by blocking abnormal impulses from and to the involved nerve root and DRG.20

Corticosteroids may thus have both beneficial and harmful effects on nerve tissue.

Corticosteroids have shown anti-inflammatory properties related to inhibition of prostaglandin synthesis and decreases in regional levels of inflammatory mediators such as interleukin-1, TNF and phospholipase A2.21-23

In patients with LDH, elevated levels of phospholipase A2, prostaglandin E2 production, and inflammatory cytokines may directly or indirectly stimulate the nerve root, and inflammatory reactions may play an important pathogenic role in causing sciatica after disc herniation.24, 25

Recent studies have shown the pro-inflammatory cytokine TNF-alpha in NP plays a vital role in the development of NP-induced inflammatory changes in the nerve root.9, 26

Corticosteroids thus have therapeutic effects on radicular symptoms caused by LDH due to their anti-inflammatory function.

Corticosteroids also reduce increases in early vascular permeability in spinal nerve roots and inhibit reductions in nerve conduction velocity induced by epidural application of NP.27

Corticosteroids exert anesthetic-like actions on nociceptive C-fiber conduction independent of their anti-inflammatory properties.28

Conversely corticosteroids have been shown to possess direct neurotoxic effects on peripheral nerve tissue.29

Dexamethasone causes reduced blood flow in normal nerves and DRG.30

Corticosteroids have some detrimental effects on the function of macrophages, which are thought to play a role in the resorption of herniated intervertebral discs.31

Methylprednisolone contains 40% polyethylene glycol as a buffer, while hydrocortisone contains benzyl alcohol, both buffering agents known to be neurotoxic.32, 33 Corticosteroids may thus have both beneficial and harmful effects on nerve tissue.

The present study focused on TNF-alpha, which plays a very important role in the development of NP-induced inflammatory changes in the nerve root. NRI decreased expression of TNF-alpha in the DRG for experimental LDH. But other factors may contribute to sciatica caused by disc herniation.

The authors feel that further studies focussing on other factors may reveal different findings and provide further information on the pathomechanisms of NRI.

Nonsurgical treatments for radicular symptoms using NRI in clinical situations seem likely to continue to develop.

In this study, combinations of local anesthetic and corticosteroid did not result in multiplicative or synergistic actions in this study, suggesting corticosteroid may be unnecessary for NRI.


– Dr David Roselt
The 12th IASP World Congress on Pain
Glasgow, Scotland, 18-22 August 2008

Dr A Breck Mackay

And so with the farm sold, cashed up on 30 June 2008, we went to Scotland and the 12th International Association for the Study of Pain (IASP) World Congress on Pain in Glasgow.

Yes, we made a few mistakes, like arriving in the United Kingdom during the coldest and wettest summer for 60 years. A couple of days in London to burn off the jet-lag, then to Aberdeen for the pre-conference meeting on Primary Care Management of Back Pain, which did not even get under way (not enough registrants), but next day the visit to Royal Lochnagar Whisky Distillery at Crathie near Balmoral made up in part, especially the bottles of 50-year-old Special Reserve Whisky. (Enough was sent home to last a few years or until the next visit!)

A drive around Scotland to all the scenic bits, such as Pitlochry, Inverness, down past Loch Ness to Glencoe (very eerie), to Loch Lomond and the Robbie Burns memorials, to Thornhill, and back to Glasgow was well worth it. Getting in to the Quality Hotel in Glasgow was an exercise in itself. We drove around in the rain, four times (a long way when you add all the one-way street diversions) and found only one door and no parking anywhere, because it is beside and, in fact, part of the main railway station! We gave up, took the car to the hire place and said fill it up … no petrol stations anywhere near Glasgow (only on the M8 or main roads outside the city) and asked them to take us to the hotel.

Amazingly, as we drove past the side of the railway station the driver suddenly turned sharp right, dived into an obscured street and up into the station to park where you would least expect … beside the railway platform! The main entrance to the hotel is inside the Central Station. Into our Quality Hotel which had the forlorn appearance of Grizabella the Glamour Cat from “Cats”… seen much better days but now very old, tattered, and torn.

And so to the conference.

Held in the Glasgow Conference and Convention Centre (looking like an armadillo perched outside a bloody big square barn) but with a holding capacity way above the 6,500 delegates. So they shut off half of each of the five biggest barn areas and had the presentations in the front half of each as well as the many lesser rooms in the complex. The armadillo held the main auditorium with a maze of steps and passages to get into it and you often ended up nowhere near where you wanted to sit. Oh well, it was only for the plenary sessions.

The facilities were good as long as you liked the coffee and junk food styles available and the nearest other outlets were in Glasgow a six-minute walk and 10-minute tube train ride to central station. It was not at all near the standard of the Sydney Convention Centre where the 11th IASP World Congress on Pain was held in 2005. Bigger but …. Sunday had seen the usual rash of pre-conference refresher courses, all summarized in the available book published by IASP. Monday had the obligatory official opening and then it got underway. The plenaries were excellent and covered many divergent topics from sodium channels and pain, joint pain neuronal mechanisms, stress and chronic pain and fibromyalgia, clinical pain in genetic disorders, neurochemistry of pain, pain and suffering following torture (it missed the torture of patients by doctors not actually listening and jumping to the wrong conclusions without any examination), pain in the developing world (India), understanding pain mechanisms, external validity of randomized trials and to whom they apply (this confirmed that randomized trials apply only to those conforming to the trial patient types … that is, to almost no one else), the neurobiology of itch and pain, inhibition in pain amplification and generation, cytokines in pain, pediatric pain and palliative management quality, pain memory extinction, neuronal spinal cord pain processing, the ubiquitous catastrophizing, neuropathic pain at the spinal level, and traumatic nerve injury.

Now if you were not mind boggled by all those, then there were 90 workshops held between 10.00-11.30 and 15.30 to 17.00 each day, with the special interest groups meeting on the Wednesday afternoon …. or you could join the various tours. Only tragedy was that the organizers had not managed to get it sorted out and, unlike Sydney where every workshop and plenary was recorded and available on CD, only the plenaries were video recorded. What a stuff up! Miss a workshop or 2-5 that you wanted to go to, and there is no way of finding out what was discussed until the abstracts are published in 2009. Surely they should have improved the recording output after the high Sydney standard in 2005. Perhaps 2010 in Montreal Canada might be better.

Of the workshops I attended, the one on fibromyalgia, trigger points, and myofascial pain rehashed all that has gone before and not one new item came up. “The managing low back pain: where do we go from here?” was done by three epidemiologists who waffled on about the statistics and added not one useful or relevant new option. This was a total waste of time compared to our work in Australia and they did not even consider anything other than randomized controlled trials, and they really are a non-entity when one realizes that pain is a unique personal experience and successful management is achieved only in each patient, thus only N=1 trials can give you comparable and useful results. Sorry, the statistical boys and girls presenting could not cope with that idea. They like big numbers per trial.

Functional pain syndrome was going somewhere. I
never found out just where, and CRPS: nerve, immune and endothelial cell interactions was fascinating and mind boggling, with a new laboratory model in the rat by Dr Terrence Coderre from Canada. (I am biased because he agreed our Australian whole body function models were good and need to be followed up.) Pain in older persons was old material and missed the effects of accumulated enthesis damage as one gets older. Preventing chronic pain in primary care was not very useful at all. And performance of the performance measures was analysis of the analysis. I lost their plot. Longitudinal perspective on musculoskeletal pain in the population was very drawn out and then I was done!

Overall? Scotland is very lovely, but I would have preferred to see it without the rain. Aberdeen, Edinburgh, and Glasgow confirmed in my mind why so many Scottish persons emigrate. If that is what it is like in summer, who in their right mind would stick around for very short days and long cold dark nights for the rest of the year?

The whole Pain Congress reminded me that pain is like a new long sausage that has not been twisted into bundles of three. Then it is twisted up and each “expert in pain research and management” is busily beavering away on their own sausage, oblivious to those on either side or anywhere else in the string … and missing the whole pain concept of one unique experience to each patient, involving sensory and emotional fear of, or actual damage.

And yes, we did go to the Edinburgh Military Tattoo. Be sensible, stop at home, be comfortable and warm, have a nice drink and eats beside you, hear it all clearly. Do not get soaked by the very heavy mists (I would hate to see their rain). Do not feel that sardines are very loosely packed in comparison and actually hear the music and performance, for sure as hell we could not as we got uncomfortably jammed into our seats, knees in the person in front, knees behind in our back, looking from left and right like on the side of a king-sized tennis court, elbows hooked each side and the rain saturating everything below the waist. What a waste of money!

Will I go in 2010 to the 13th IASP World Congress on Pain in Montreal Canada from August 29 – September 2, 2010?

Yes, but only because I can add it to a Greek Island cruise and conference, a visit to Bulgaria (to a family physician who is doing what we do and getting the same results), a London visit, time in Montreal, and a visit to friends in Ottawa as well. Must make a wholesome trip out of it to enjoy it fully!
## Educational Activities

### MASTERS, DIPLOMA, AND CERTIFICATE COURSES IN MUSCULOSKELETAL MEDICINE

#### FLINDERS UNIVERSITY DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

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<tr>
<td>23 - 27 July</td>
<td>Due to Norm Broadhurst’s retirement, this course will not be presented until his replacement is appointed.</td>
<td>Flinders Medical Centre</td>
<td>Flinders University School of Health Sciences, Bedford Park SA 5042</td>
<td>Mr Michael McKay McKay, Ph: +61 8 8201 3913; <a href="mailto:michael.mckay@flinders.edu.au">michael.mckay@flinders.edu.au</a></td>
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#### UNIVERSITY OF OTAGO DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

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<td>7-14 March</td>
<td>On campus papers MSMX701 Clinical Diagnosis Pt 1 MSMX701 Pt 2</td>
<td>On-campus course University of Otago, Christchurch</td>
<td>Enrolments: Veronica McGroggan Tel. +64 3 364 1086 Fax +64 3 364 0909 Email: <a href="mailto:veronica.mcgroggan@otago.ac.nz">veronica.mcgroggan@otago.ac.nz</a> or Geoff Harding Ph: +61 7 3269 5522 Fax +61 7 3269 6407 Email: <a href="mailto:drgeoffh@bigpond.net.au">drgeoffh@bigpond.net.au</a> website: <a href="http://www.uoc.otago.ac.nz/departments/msm">www.uoc.otago.ac.nz/departments/msm</a></td>
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<td>17-21 August</td>
<td>MSMX709 Clinical Therapeutics Distance taught papers MSMX704 Pain MSMX711Pain Assessment (new paper)</td>
<td>On-campus course Christchurch Distance taught papers - fortnightly audioconferences ex University of Otago, Christchurch</td>
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<td>MSMX708 Pain Management</td>
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<td>MSMX702 Tissues MSMX703 MSM Disorders</td>
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#### UNIVERSITY OF NEWCASTLE MASTERS IN PAIN MEDICINE

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<td>2008</td>
<td>Masters in Pain Medicine</td>
<td>Internet</td>
<td>Grad school at University of Newcastle, NSW</td>
<td><a href="mailto:Laura.Miller@newcastle.edu.au">Laura.Miller@newcastle.edu.au</a> - administrative liaison at Uni of Newcastle or <a href="mailto:Phillipa.Powis@newcastle.edu.au">Phillipa.Powis@newcastle.edu.au</a> for information about the course</td>
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## AUSTRALIAN COLLEGE OF PHYSICAL MEDICINE DIPLOMA OF PHYSICAL MEDICINE

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<td>Diploma of Physical Medicine Manual Techniques for the treatment of musculoskeletal dysfunction</td>
<td>Sydney</td>
<td>Australian College of Physical Medicine</td>
<td>Shane Maloney, Ph (02) 9438 5088, Fax (02) 9438 5755 <a href="mailto:admin@northsidephysicalmedicine.com.au">admin@northsidephysicalmedicine.com.au</a> or visit the website <a href="http://www.physicalmedicineaustralia.com.au">www.physicalmedicineaustralia.com.au</a></td>
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## OTHER MUSCULOSKELETAL MEDICINE EDUCATIONAL ACTIVITIES

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<td>Adelaide</td>
<td>Margaret Taylor</td>
<td>Ph (08) 8378 1254 <a href="mailto:taylorme@internode.on.net">taylorme@internode.on.net</a></td>
<td>RACGP 40 points category 1</td>
</tr>
<tr>
<td>5-6 Dec 08</td>
<td>AAOM workshop Lumbar sacral spine and lower extremities</td>
<td>Denver, USA</td>
<td>American Association of Orthopedic Medicine</td>
<td>Ph 888 867 1920 email <a href="mailto:aaom@aaomed.org">aaom@aaomed.org</a></td>
<td>For physiotherapists only</td>
</tr>
<tr>
<td>28 Dec - 7 Jan</td>
<td>Med. Challenges Series: Conferences in Antarctica: The Mackenzie approach to musculoskeletal pain</td>
<td>Antarctica cruise from Argentina</td>
<td>World Travel</td>
<td>Ros or Stephanie, World Travel 2/142 Bundall Rd, Bundall 4217 Ph 1800 249 804 Email <a href="mailto:tct@worldtravel.com.au">tct@worldtravel.com.au</a></td>
<td></td>
</tr>
<tr>
<td>5-8 April</td>
<td>The Pain Continuum: Making pain history</td>
<td>Sydney</td>
<td>Australian Pain Society</td>
<td>DC Conferences Ph (02) 9954 4400 <a href="http://www.apsoc.org.au">www.apsoc.org.au</a></td>
<td></td>
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<tr>
<td>3 evenings in 2009</td>
<td>Prolotherapy in Sports Med: 3 x 2.5 hrs</td>
<td>Adelaide</td>
<td>Margaret Taylor</td>
<td>Ph (08) 8378 1254 <a href="mailto:taylorme@internode.on.net">taylorme@internode.on.net</a></td>
<td>RACGP 40 points Category 1</td>
</tr>
<tr>
<td>August</td>
<td>Australian Musculoskeletal Medicine Conference</td>
<td>Sunshine Coast</td>
<td>AAMM, ACPM, AFMM, NZAMM</td>
<td><a href="http://www.dcconferences.com.au/aamm2009">www.dcconferences.com.au/aamm2009</a> DC Conferences PO Box 637 North Sydney NSW 2059, Ph (02) 9954 4400 Fax (02) 9954 0666 <a href="mailto:aamm2009@dcconferences.com.au">aamm2009@dcconferences.com.au</a></td>
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