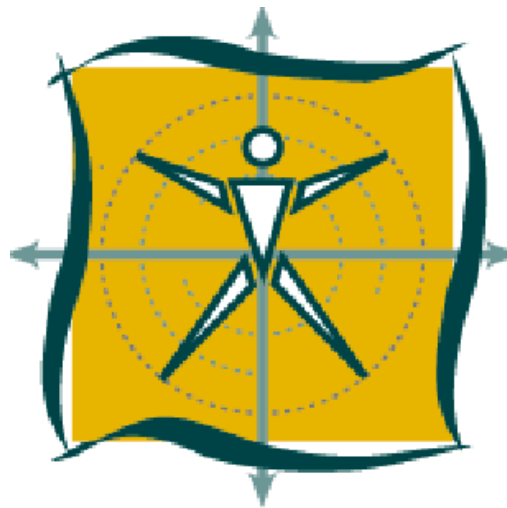


Australasian Musculoskeletal Medicine



- Prolotherapy and Achilles tendinopathy
- Indications for low back prolotherapy
- Chronic low back pain
- Treatment of complex regional pain syndrome with peripheral nerve blocks
- Spinal fracture in epileptic fits
- The frozen shoulder
- Post-herpetic neuralgia
- A systematic review of the literature of low-level laser therapy
- Piriformis syndrome
- Therapy for low back pain
- Reproducibility studies in manual/musculoskeletal medicine

Australian Association of Musculoskeletal Medicine Office Bearers 2004-2005

President

Dr Scott Masters MBBS, Dip Musc Med, FRACGP, FAFMM
Caloundra Spinal & Sports Medicine Centre
39 Minchington St, Caloundra, Qld 4551
Ph: +61 7 5491 1144
Fax: +61 7 5491 1253

Vice-President

Dr Michael Yelland MBBS, Dip Musc Med, FRACGP, FAFMM
64 Wirraway Pde, Inala, Qld 4077
Ph: +61 7 3275 5444
Fax: +61 7 3278 9987

Honorary Secretary

Dr Brian Lovell MBBS, Dip Phys Med, M Med Phys Med
Metropolitan Spinal Clinic, 302 Malvern Rd, Prahran, Vic 3181
Ph: +61 3 9529 1988
Fax: +61 9529 1844

Honorary Treasurer

Dr Esther Langenegger MBBS, Dip Musc Med, FAFMM
482 Macauley St, Albury, NSW 2640
Ph: +61 2 6041 4494
Fax: +61 2 6021 6373

Committee Members

Dr Steve Jensen MBBS, Dip Musc Med, FAFMM; Immediate Past President
5 Stanlake St, Footscray Vic 3011
Ph: +61 3 93185233
Fax: +61 3 93186630

Dr Derek Davey MBBS, Dip Obs, Dip Occ Health, Dip Musc Med, FAFMM
29 Craigie Rd, Newtown Vic 3220

Dr Geoffrey Harding MBBS, Dip Musc Med, FAFMM
1st Flr, 67 Brighton Rd, Sandgate, Qld 4017
Ph: +61 7 32695522
Fax: +61 7 3269 6407

Dr Michael Oei MBBS, Dip Phys Med, M Med Phys Med, Cert Man Med
213 Darlinghurst, NSW 2010
Ph: +61 2 8302 1180, +61 2 9969 2198
Fax: +61 2 8302 1195

Dr David Roselt MBBS, Dip Musc Med, FRACGP, FAFMM
26 Crofton St, Bundaberg 4670
Phone +61 7 4152 2888
Fax +61 7 4153 3245

Dr Margaret Taylor BSc, MBBS, FACNEM, FIBCT
PO Box 570, Fullarton SA 5063
Ph/fax: +61 8 8338 3778

Webmaster

Dr Victor Wilk MBBS, M Med Pain Med, Dip Musc Med, FAFMM
441 Bay St, Brighton, Vic 3186
Ph: +61 3 9596 7211
Fax: +61 3 9596 7871

New Zealand Association of Musculoskeletal Medicine Office Bearers 2004-2005

President

Dr John Robinson BSc, MBChB, M Med Pain Med, Dip Musc Med, Dip Occ Med, FAFMM, Cert Spinal Inj
256 Papanui Rd, Christchurch
Ph: +64 3 3550342, Fax: +64 3 3100516

Vice-President

Dr Peter McKenzie BSc, MBChB, Dip Obs, Dip Musc Med, FRNZCGP, FAFMM
217 Bridge St, Nelson
Ph: +64 3 5483455

Honorary Secretary

Dr Gary Collinson MBChB, Dip Musc Med, FAFMM, Cert Spinal Inj
4 Kinross St, Blockhouse Bay, Auckland
Ph: +64 9 6271024, Fax: +64 9 6271181

Honorary Treasurer

Dr Clemens Franzmayr Specialist for Conservative Orthopaedics 1975
15 Frank St, Christchurch
Ph: +64 3 3527761, Fax: +64 3 3527762

Committee Members

Dr Jim Borowczyk BSc, MBChB, Dip Musc Med, MRCP, FAFMM
256 Papanui Rd, Merivale, Christchurch
Ph: +64 3 3550342, Fax: +64 3 3557071

Dr Steve Bentley MBChB, Dip Obst, Dip Musc Med, Dip Sports Med, FAFMM
72 Newington Ave, Dunedin
Ph: +64 3 4672046, Fax: +64 3 4672042

Dr Elizabeth Elliott, MB, ChB, FRNZCGP, Dip Occ Med, Dip MSM
Ph: +64 3 352 6882, Fax: +64 3 532 7762

Dr Mark Johnston MBChB, M Med Pain Med, Dip Mus Med, FAFMM, Cert Spinal Inj
394 Hibiscus Coast Highway, Orewa
Ph: +64 9 4261260, Fax: +64 9 4261136

Dr Charlie NG MBChB, Dip Musc Med, Dip Sports Med, FAFMM, Cert Spinal Inj
8 Archibald Rd, Kelston, Auckland
Ph: +64 9 8185208, Fax: +64 9 818 5208

Dr Grant Thomson MBChB, Dip Mus Med, MLCOM
Bush Rd Medical Centre, Kamo, Whangarei
Ph: +64 9 4350692, Fax: +64 9 435 2634

AAMM website: www.musmed.com

NZAMSM website: www.musculoskeletal.co.nz

AFMM website: www.afmm.com

FIMM website: www.fimm-online.org

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Contributions on any relevant topic are welcome for submission to the editor, Dr David Roselt
26 Crofton St, Bundaberg 4670
Phone +61 7 4152 2888
Fax +61 7 4153 3245
Email droselt@aberdovy.com.au

Guest editorial

From FIMM Scientific Committee International Academy of Manual/Musculoskeletal Medicine

Chairman of the Science Board, Dr Jacob Patijn, MD, PhD, Neurologist, University Hospital Maastricht, Pain Management and Research Centre, Dept. Anaesthesiology, Maastricht, The Netherlands. Phone 0031 43 3876543, fax 0031 43 3875457, email jpat@sane.azm.nl

History

FIMM is an international association of 29 national societies of manual or musculoskeletal medicine. Its activities include the development and improvement of standards of practice internationally.

In 1997 FIMM General Assembly (GA) decided to split the existing Scientific Advisory Committee into an Education Committee (EC) and a Scientific Committee (SC) to meet the increasing needs of FIMM, and to pay more attention to evidence-based science and education in M/M medicine.

The SC chairman, in his first report to the GA in Australia in 1998, stated that science must have a prominent place in FIMM and that the composition of the SC must be independent of national society interests and representation. A well-defined problem in M/M medicine was illustrated by the sentence "There are many (arguably, too many) different approaches in M/M medicine in different countries with many different diagnostic procedures and many different therapeutic modalities."

At first the SC had to create conditions for information exchange between different schools in M/M medicine, and to stimulate scientific work within M/M medicine.

In the longer term, the SC had to develop a structure, clearly related to FIMM, in which more scientists and educationalists from the different schools in M/M medicine (in diverse countries) could be involved.

The SC comprised invited scientists and researchers in M/M medicine, recommended by the chairman and ratified by the FIMM GA. In the early SC meetings, plans were further elaborated and it was decided that the first scientific activities of the SC were to be focused on efficacy and reliability in

M/M medicine. At the subsequent SC meetings, the different areas of the locomotion system (lumbar, cervical, shoulder/thoracic, and extremities) were discussed in the framework of efficacy and reliability. The SC decided also to provide the international community of M/M medicine with proper tools to perform scientific work.

Reliability and efficacy protocols and a protocol for 13 Good Clinical Practice (GCP) rules were developed and published. To create an international platform to discuss scientific work, a first SC conference was organised in cooperation with a national society for M/M medicine (Denmark, Odense 2003). At this SC conference, scientists presented and discussed their (preliminary) results, and proposals for scientific protocols.

At the SC meeting in 2004, a start was made to discuss basic research in M/M medicine. A consensus model, in which different approaches of M/M medicine could be integrated, was elaborated. Such a model was needed to provide M/M medicine with outside evidence for efficacy and reliability, with a general theoretical background that included the different approaches in M/M medicine. Also, a protocol was developed to evaluate the reproducibility studies of diagnostic procedures in M/M medicine, with the ultimate goal of providing the educational boards of the societies of FIMM with the state-of-the-art, evidence-based diagnostic procedures. The same was true for the state-of-the-art efficacy in M/M medicine. A paper will be published by the SC about the present status of efficacy in M/M medicine.

In the past seven years, the chairman and 11 SC members undertook the work of the SC. From a practical point of view, this composition of the SC created a basis for future science in FIMM. However, it became clear that

the SC had reached a point at which it became necessary to involve more scientists and educationalists in its work. This idea was strengthened by the fact that many requests were received from individuals to become members of the SC. The financial status of FIMM and the format of the SC prevented such an enlargement. In an early stage of the SC, ideas were developed to establish an International Academy of M/M medicine, and were subsequently discussed with the FIMM Policy Committee. The Policy Committee supported these ideas, and at the GA of Bratislava 2004 the plans and statutes of a FIMM International Academy for M/M Medicine were ratified by a substantial majority of the FIMM national societies.

FIMM Academy of M/M Medicine

The work of the SC over the past few years resulted in a firm position of science in FIMM. By defining the main problem in M/M medicine and providing solutions in the form of protocols and publications, the SC has slowly influenced a change in attitude of the international community of FIMM towards a more evidence-based M/M medicine. However, the present composition of only 12 SC members cannot guarantee further development.

The diversity of approaches in M/M medicine is not reflected in the present SC. The results of scientific work undertaken in countries with a different approach cannot be discussed and integrated internationally.

M/M medicine is by definition a multidisciplinary and eclectic profession, in which many medical disciplines such as neurology, internal medicine, etc., and preclinical disciplines such as neurophysiology and clinical biomechanics are integrated, but the present format of the small SC cannot guarantee such a multi-

disciplinary character.

In previous years the national societies indirectly, by their fees, financed the work of the SC. Because of the politically independent character of the SC, the national societies could not influence the activities of this committee.

Although this political independency was the best condition for the activities of a SC in FIMM, the disadvantage of such a format was that many scientists and educationalists from the national societies could not be involved in the work of the present SC. As a consequence, not all available knowledge within M/M medicine can be assimilated and discussed on its evidence-based merits.

At present, two different institutes (the Scientific Committee and the Education Committee of FIMM) generally operate independently. In keeping these two institutes apart, educational programs will not professionalize with a firm evidence base.

By transforming a SC to an International Academy, many of these drawbacks can be overcome. A future Academy, by involving more scientists and educationalists from the national societies, can better guarantee increased professionalism of M/M medicine in a more evidence-based way. The Academy as an international organisation will provide the national societies with a larger platform on which to interact and discuss the different approaches in M/M medicine.

Academy format

The present SC has worked very closely with FIMM, and the FIMM General Assembly ratified SC proceedings and plans. There must be a similar close relationship with an International Academy. In addition to medicopolitical issues, FIMM has an overall responsibility for scientific and educational aspects of M/M medicine.

The organisational format of an Academy, with an Executive Board and a Science Board, and the election of officers by the FIMM General Assembly, reflects in a constitutional way this mutual relationship. Furthermore, both the chairman of the Executive Board

and the chairman of the Science Board are obliged to present their annual reports to the General Assembly.

An Academy without a connection to the national societies through FIMM is meaningless. Participation and support of the national societies of FIMM is essential for the future of an Academy.

By ratifying the founding of the Academy, the national societies not only confirm the need for a strong relationship between an Academy and FIMM, but they subscribe to the goals of the previous SC, which also forms the basis for future activities of the Academy. More precisely, the national societies subscribe to the need for improved professionalism of and increased evidence basis for M/M medicine.

The Academy

The Academy has two separate structures: logistic and scientific. The Executive Board of the Academy is responsible for the logistic structure. This Board comprises a chairman, scientific director, administrative officer, and finance officer. The Executive Board is also responsible for Academy finance.

The Science Board comprises a scientific director (chairman) and nine Science Board (SB) members. Additionally, the chairman of the FIMM Education Committee participates in the activities of the Science Board together with the administrative officer. The Science Board is responsible for all science and education.

All members of the Academy have full voting rights at the annual general meeting. They choose the scientific director and the members of the Science Board. Members of the Academy are expected to make presentations to the Academy conferences, and to uphold the principles of FIMM, the parent organisation. Interactions and contributions to discussions and the development of consensus papers on diverse topics will be open to all members.

Individual SB members are responsible for areas of special attention, such as efficacy, reliability, basic re-

search, complications, education, paediatric M/M medicine, and additional diagnostics.

Individual SB members are charged with developing plans and involve scientists and educationalists to establish a special Academy working group. In these Academy working groups, future Academy members can be involved such as those proposed by national societies. What was previously the SC conference will become an Academy conference, held every second year. At this conference, Academy members can present their papers on scientific and educational issues. Participation in the Academy conference is intended for both Academy members and others who are interested. The Academy will also be involved in the scientific organisation of FIMM congresses.

So far a rough outline only has been formulated for scientific and educational plans. Nevertheless, the activities of the Academy will be a logical continuation of the work of the previous SC. The area of responsibility of the previous SC and its proposed problem solving will form the basis of future Academy activities, although with the involvement of more scientists and educationalists.

Education

The starting point for the Academy is the view that science is nothing without education, and vice versa. This means that education must have its place in the Academy. This relationship is illustrated by the fact that the chairman of the FIMM Education Committee (EC) will participate in the Science Board. In this way the connection between the Academy and the educational boards of the national societies is guaranteed.

Secondly, a section of educationalists must be formed with responsibility for the level of international educational standards in M/M medicine and development of new formats for education programs.

Further plans will be elaborated in cooperation with the EC chairman. Although science plays a major role, educational concerns are also essential.

Education guarantees the implantation of scientific results and consensus produced by the Academy. Conditions for membership will be elaborated in cooperation with the EC.

Future plans

The future work of the Academy will be a logical continuation of the work of the former SC. Areas with special interest will be defined by Academy working groups, in which Academy members will be involved and/or invited under the responsibility of a member of the Science Board.

Projects which have not been completed by the former SC will be finalised. Plans for a first Academy conference will be developed, and the possibility of establishing an international journal for M/M medicine in cooperation with existing journals will be explored.

A logistical structure will be devel-

oped to guarantee maximal transparency and maximal information dissemination to the national societies, scientists, educationalists, and FIMM as an organization.

A public relations policy will be elaborated to involve all the FIMM national societies in the work of the Academy and to stimulate membership.

Support by national societies

For the work of the SC, support of the FIMM national societies is essential. For the future of the Academy, substantial participation in its development and support of its activities by FIMM and its national societies is even more necessary.

The ratification of the Academy and adoption of its Articles of Association by a large majority of the national societies at the FIMM GA in Bratislava 2004 not only reflects a substantial support of the idea of an Academy, it

also obliges FIMM in general and its national societies in particular to contribute wholeheartedly to the success of the Academy.

As science is nothing without education, so an Academy is nothing without the national societies of FIMM. The work of the Academy is firstly to professionalize M/M medicine in a more evidence-based way, and secondly, to be an international platform for mutual discussions and cooperation between schools and/or approaches of national societies.

FIMM has, by creating an International Academy for M/M medicine, taken a step towards a new future of its organisation whereby scientists, educationalists, and practitioners in M/M medicine can meet each other in a more personal way.

This Academy will also provide FIMM with the necessary knowledge to address future medicopolitical problems.



The New Zealand Association of Musculoskeletal Medicine

Annual Conference 23rd-26th June 2005

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Editorial

If you look around the arrivals gate at Sydney airport before the APS and IASP conference in August you'll see people arriving and being greeted by fathers and sons, mothers and daughters, husbands and wives, boyfriends, girlfriends, old friends.

The **26th Annual Scientific Meeting of the Australian Pain Society (APS)** will be held in conjunction with the **International Association for the Study of Pain (IASP) 11th World Congress on Pain, August 21-26, 2005**, and should be well worth attending. Be there, or miss out on a truly inspirational goodwill event in the world of pain management. Mark it in your diaries, and keep the time free. Details at <http://www.iasp-pain.org/05Cong.html>.

Dr Stefan Blomberg from Sweden is coming to Australia and will run a workshop in Brisbane on August 27-31 following the Sydney meeting on his full comprehensive **Stayac Algorithm**. Further details are in the Educational Activities section of this journal, kindly compiled by Associate Professor Michael Yelland. This is sadly for the last time, as Michael moves on to new challenges.

In this issue there is featured a guest editorial from Dr Jacob Patijn, chairman of the Science Board for the International Academy of Manual/Musculoskeletal Medicine of FIMM to bring readers up to speed with promising international developments.

The New Zealand Association of Musculoskeletal Medicine (NZAMSM) will be holding their annual conference at Otago University June 23-26, 2005. **Shoulder Pain and Related Disorders – the Full Monty** promises to be a no-holds-barred account of the latest developments in the evidence base. There will be an emphasis on assessment and pragmatic treatment both in the plenary sessions and the workshops in this beautiful part of the South Island. Be sure to book early to ensure your flights and accommodation.

The 35th Annual Scientific Meeting of the Australian Association of Musculoskeletal Medicine was held at Novotel Twin Waters Resort in Mudjimba, Sunshine Coast, south of Noosa on March

3-6. The topic **Pain in the Elderly** was highly relevant and practically orientated and had not been highlighted previously.

The sunshowers cleared on the Thursday afternoon, giving way to three days of absolutely pristine early autumn weather as speakers and delegates arrived for the welcome reception. The academic sessions were very stimulating and informative, with wonderful speakers doing full justice to their topics. The workshops were well received, with feedback highly complimentary all around.

The resort with its spacious picturesque grounds is a great setting to achieve a balance between work and play. The social program was a truly great success. The sunset conference dinner on the banks of the Maroochy River at the alfresco Picnic on the Rocks at Yandina was the highlight of the stay, with the band rising to the occasion with a vengeance.

The ladies from DC Conferences Pty Ltd once again attended to our every need, metaphysically speaking.

The conference closed early on the Sunday afternoon as the sky turned a little overcast but did not detract from unwinding by the pool with family and friends to cap off a perfect few days at the beautiful Sunshine Coast.

This edition of the journal features important reports from the presidents of both Australasian associations, Drs Scott Masters and John Robinson.

Highlighted is an illuminating debate in Letters to the Editor that initially arose online between protagonists of prolotherapy and paraspinal injections and has been featured here with permission.

Dr John Lyftogt from Christchurch NZ has written a very timely piece on his pilot study on the use of the resurgent prolotherapy for the treatment of Achilles tendinopathy, a common malady especially in the active sports person.

There is a related paper that is thought provoking and reflective on the indications for low back prolotherapy by Dr Robert Kidd from Ontario, Canada, kindly reprinted from the *Pain Journal*. It examines further whether local ten-

derness is truly an indication of local ligamentous laxity, or perhaps other factor(s) such as neuropathic pain with central sensitization.

Dr Breck McKay has provided another instructive contribution on functional leg length and low leg length inequality as being critical factors that if not assessed and addressed can lead to recurrence of chronic low back pain that has successfully responded to parasacroccocygeal injection(s) and related multimodal treatment.

There is a case series, another pilot study, on the use of peripheral nerve blocks in complex regional pain syndrome (CRPS) by Dr Gireesh Kanji, musculoskeletal pain physician from Wellington, NZ. This approach using peripheral nerve blocks with local anesthetic injections

to block somatic afferent and sympathetic efferent pathways provided effective pain relief for CRPS when combined with other treatments for neuropathic pain (NP) such as trigger or tender point injections and oral medication in the form of opioids and carbamazepine. Further controlled studies to validate this protocol are in the pipeline.

There is a very interesting paper on spinal fractures as a result of epileptic seizures by Dr Graham Corbett who works for the Accident Compensation Corporation in NZ. This was written in completing his Diploma of Musculoskeletal Medicine through Otago University.

We have an erudite paper by Dr Bill Douglas, rheumatologist from Brisbane, who has followed on from his letter to the editor in the May 2004 edition of the journal on adhesive capsulitis. Bill emphasizes the importance of early presentation and prompt treatment to achieve good outcomes for idiopathic frozen shoulder (IFS). In his extensive experience, there is a window of opportunity for treatment using intra-articular and oral corticosteroids in the first four months, but as usual the earlier treatment can be instituted the better. He is very wary of surgical intervention, including the use of hydro-dilatation in this group, especially for workers' compensation cases.

A systematic review on low level laser therapy (LLLT) in neck pain from Dr Roberta Chow from Castle Hill Medical Centre Sydney has been published recently in *Lasers in Surgery and Medicine*. It was written with Associate Professor Les Barnsley from the Faculty of Medicine, University of Sydney, and Department of Rheumatology, Concord Repatriation General Hospital in conjunction with Roberta's PhD thesis. She has kindly obtained permission for a reprint from the publishers Wiley-Liss, Inc.

There is also a review of the evidence base for treatment and prevention of post-herpetic neuralgia (PHN) by your editor. This is a growing problem as the population ages. Most people have been exposed to wild varicella-zoster virus (VZV) and so are at risk of its reactivation. Up to half of all people living to age 85 years will be affected by herpes zoster (HZ).¹ Even with early anti-viral treatment, up to 20% of patients aged 50 and over will still be afflicted by PHN six months after the onset of rash.²⁻⁴ The absolute importance of the early use of antiviral medication and optimum pain relief cannot be overemphasized.

There is a reprint from the *Journal of Orthopaedic Medicine* of an important article on piriformis syndrome by Dr Lars Remvig who recently visited Australia, and co-authors Drs Richard Ellis and Patijn. This is highly relevant with respect to the management and treatment of low back pain and the recent interest in parasacroccygeal and paraspinal injections.

Dr David Squirrell from Adelaide, South Australia, has kindly written up his presentation from the Adelaide conference on the pragmatic management of low back pain highlighting the use of local anesthetic steroid injections and stretching using post-isometric relaxation in the emergency department.

An article on reproducibility studies from Dr Patijn is very topical with respect to conducting trials but also in the day-to-day assessment and treatment of musculoskeletal pain.

Our president Dr Scott Masters, Dr Michael Yelland, and I have reviewed

some journal articles again for your interest. I hope you enjoy reading the abstracts and comments provided for your entertainment.

So enjoy yourself as you feast at this magnificent buffet of musculoskeletal pain medicine information.

Thanks once again to all contributors for their fine efforts in this May edition, and as usual I would love to encourage more journal articles, and more discussion and feedback in the form of contributions to the Letters pages.

Be sure to get over to New Zealand in July and down to Sydney in August for a veritable bonanza of vital information and knowledge. I'm certain you will see that as far as the world of musculoskeletal pain medicine is concerned, if you look for it you'll find that love actually is all around.

1. Johnson RW, Dworkin RH. Treatment of herpes zoster and postherpetic neuralgia. *Br Med J* 2003; 326: 748-50.

2. Dworkin RH, Boon RJ, Griffin DR et al. Postherpetic neuralgia: impact of famciclovir, age, rash severity and acute pain in herpes zoster patients. *J Infect Dis* 1998; 178(Suppl 1): S76-80.

3. Beutner KR, Friedman DJ, Forszpaniak C et al. Valaciclovir compared with acyclovir for improved therapy for herpes zoster in immunocompetent adults. *Antimicrob Agents Chemother* 1995; 39: 1546-53.

4. Tyring SK, Beutner KR, Tucker BA et al. Antiviral therapy for herpes zoster: randomized, controlled clinical trial of valacyclovir and famciclovir therapy in immunocompetent patients 50 years and older. *Arch Fam Med* 2000; 9: 863-69.

David Roselt

From the AAMM President

I hope all members have recovered from the gruelling and intensive three days of brainstorming at Twin Waters in March. It was another first for the Sunshine Coast, according to Associate Professor Stephen Gibson, who could not recall a previous conference that had been devoted entirely to pain in the elderly. The feedback from the conference both formal and informal was extremely positive. Delegates found the speakers were on target with their practical approach and differing slants on management issues. The panel discussions were brisk, with many interesting questions and answers flowing from the audience and chair. Again the workshops were popular, with delegates enjoying the chance for a hands-on approach to solving clinical dilemmas. The only criticism was that workshops should have been longer! Once again I'd like to thank the scientific committee for their hard work in putting the program together and arranging the workshops. Hats off to Philip Watson, Michael Yelland, and David Roselt, and to the script writers for "Darby and Joan", Geoff Harding, Mark Craig, and the mercurial Dr Roselt.

Again it was interesting to hear the feedback from the invited speakers at the conference. The recurrent theme from the invited speakers is how impressed they are with the level of interest shown and the quality of questions in the panel discussions. Delegates should be congratulated for doing their homework!

On the Queensland front, AAMM and AFMM have been in discussions with Q-comp, the controllers of work insurance for Queensland. Input over fee structures, medical review of injured workers, and input into training of doctors were all discussed. Dr Roselt and I presented a submission to the board regarding the role of musculoskeletal medicine practitioners with insurance providers. They were very interested in the results of the National Musculoskeletal Medicine Initiative and the resource of information available with AAMM and AFMM. Our one weakness was the number of active members throughout the state available and

qualified to teach other health practitioners.

In Victoria, some of our members are recognized as specialists in musculoskeletal medicine, paid as such for their time and reports, and asked to sit on boards making decisions regarding injured workers. Ideally this should be the situation in every state. The prospect of a win-win situation is obtainable in the work injury arena. The initiative spelt out the enormous cost savings that are there to be made in the management of back pain. The system set up at Newcastle Hospital and run by Dr Brian McGuirk has also shown that it is possible to greatly reduce costs and sick leave by implementing the evidence-based approach of the Initiative. The Newcastle system has been so successful that it has been exported to other places. The practical side of this program is being presented at the IASP meeting in Sydney during the latter part of August and will be a must for all with an interest in this area of medicine.

Unfortunately the insurance system is often largely adversarial, prolongs disability, and creates anger and frustration in the injured, resulting in increased costs to the insurance company. We have all witnessed patients on endless roundabouts of specialist reviews while they await their insurers' acceptance of a management plan. Meanwhile the recovery window for the patient shrinks, with the likelihood of a satisfactory outcome diminished. All too often we pick up these patients anywhere from six months to two years after their initial injury. They are confused, have lost faith in the system, are often depressed and experiencing relationship difficulties, are withdrawn from their usual social activities, and have fallen into despair. Their case managers seem unable to deal efficiently with even the simplest requests. Surely in the 21st century it is time for a complete overhaul. It would be worthwhile at our next AGM for the Association to consider making further representations to the parties involved in the insurance of musculoskeletal injuries.

Congratulations are in order for one of the stalwarts of the Association. Dr

Michael Yelland has recently been appointed Associate Professor in Primary Care at the new Griffith University Medical School. I would like to take this opportunity to wish Michael all the best in his new position. It is a tremendous opportunity to have a significant influence in the development of medical graduates in Queensland.

Finally, I'm looking forward to meeting with members at the IASP conference in Sydney late August. The week has some fascinating speakers and topics and should be a feast for pain aficionados. It will also be the scene for our AGM, since our annual conference was held early on in the year.

Scott Masters

From the NZAMSM President

It has been an interesting year thus far for the New Zealand Association of Musculoskeletal Medicine.

We now have 23 vocationally registered musculoskeletal physicians. Approximately three-quarters of the Fellows are now in full-time MSM practice (mostly private practice), with the others not far behind.

We have two Fellows in hospital consultant positions, and several involved in lecturing to undergraduate and postgraduate students and registrars, as well as holding courses for GPs and other interested parties.

We have made progress in our association with the ACC. We now have regular three monthly meetings to discuss contract issues. In addition to regular consultation and treatment, we are funded for diagnostic as well as for therapeutic injection procedures including radiofrequency neurotomy.

We are, however, being seriously scrutinized with regard to the performance of these procedures, and are being asked to supply evidence of efficacy far greater than that required for other procedures, surgery, or indeed for CBT.

Progress is being made with Southern Cross, the major medical insurance company in New Zealand, but this is slow as one would expect.

On the manual medicine front, a weekend retreat was held in March to update the instruction manuals, and to discuss the application of different manual medicine techniques. There is a great deal of interest in and enthusiasm for this type of treatment and the retreat was well attended and successful. There are plans to run a workshop on developmental kinesiology this year with Professor Parvel Kolar.

Last year Steve Bentley attended the FIMM conference in his role as president of the NZAMSM, and found this very useful, both in terms of increasing our profile and for establishing contacts. Funding of attendance at this conference is to become an annual occurrence.

Jim Borowczyk continues his tireless work with the diploma course which doubles as the part one for the Fellowship.

The registrar training program is moving ahead, the main difficulty being the current review of vocational registration by the medical council which could cause uncertainty for potential registrars. We are in the process of drawing up a formal agreement with registrars, and there are several very interested doctors waiting in the wings. We will proceed with this in the near future.

A weekend meeting of the education committee is planned for June to draw up a training manual for registrars. Many of the Fellows are keen to be involved in this as well as in the training program.

The New Zealand Medical Council is currently reviewing vocational registration for the various branches of medicine, and musculoskeletal medicine (which achieved vocational registration in 2000) is one of these. Vocational registration itself is not under threat, but the council has expressed some concerns regarding our recertification program, as well as our training program. There were some major inaccuracies in their report. This is being clarified and attention given to our auditing and peer review processes.

I feel hopeful that a successful outcome will be achieved. Our annual conference is to be held in Dunedin 23 - 26 June 2005. This is entitled "Shoulder Pain and Disorders - The Full Monty". It has been organized almost single handedly by Steve Bentley and promises to be highly successful.

Next year is the 25th anniversary of the New Zealand association, and we look forward to continued growth and influence.

We need to continue to demonstrate the benefits we can achieve by documenting outcomes in a meaningful way and publishing our results whenever possible. In this way it will be very difficult for our group to be ignored as our results will speak for themselves.

John Robinson

Letters to the editor: Prolotherapy debate

Dear Editor

I feel it's definitely time to put Blomberg and prolotherapy together and come up with one single, simple use this or use that, as a combination therapy..... Then to work out what and where each is best... but I am very sure that the *needle* is the most important factor... how many times and where it hits!

I start at the parasacrococcygeal ligaments (PSClg) and go along the edge of the sacrum then really "sewing machine stitch" the area medially to that.

The injection is rapid up and done, of very short range, tapping on to the bone so that the enthesis is affected. Repeat on the opposite side. Yes: I have gone into the dorsal ramus orifices on occasion, *but* you know immediately you have done this and you do not penetrate very far. If you hit the nerves, it reproduces the patient's pain beautifully!

Then I inject at the centre of the PSIS and go centrally, to the iliolumbar ligaments and all around the upper half of the sacroiliac joint (SIJ), and "into" the SIJ if tender.

Following my findings with the 33 patients (equivalent effect in the short-term of needling alone versus LA alone vs LA/steroid injection), and the confirmation by Michael Yelland of the needle being the most important part of the process (glucose = saline results), I make sure I jab ++++ in, on, and around the areas.

One week later the most common sites that need re-injecting are the bits between that are usually the S1, 2 areas not quite hit the first time and the PSIS laterally.

I think we all should get together and talk about what we each do and find what works. That was how John Murtagh did it for the manipulation. Everyone learns from others, yet we all have our own favorite ways of getting results.

Yours sincerely

Dr Breck McKay, Brisbane

Dear Editor

In response to Breck's letter:

If the needle is the most important part of the process – that is, creating an injury, then the process we are setting off is the inflammatory response of: release of cytokines, attraction of polymorphs, then macrophages, then fibroblasts which lay down new collagen which eventually strengthens the enthesis. Even if the result is due just to alteration of the pain response in some way, the steroid is not necessary.

Can we have a discussion about putting some solution that is less inhibiting of the inflammatory response in the needle, for example, saline or lignocaine, which would be preferable to my way of thinking, because of the decades of work in neural therapy in Europe, using local anesthetics. Breck MacKay's technique sounds like the technique of the West Coast prolotherapy doctors who are pretty aggressive (strong?) in their needling. You should read Tom Dorman's technique in his articles, for example:

- Re whiplash - *J Orthopaed Med* 1999; 21(1): 13-21
- Ligament instability of the knees - *Manual Med* 1988; 3.
- The low back technique is probably best described in Klein et al, *J Spinal Disorders* 1993; 6(1): 23-33, but it may be better in Dorman's book available from his website www.dormanpub.com for US\$80.

The articles are all available from my website (www.drmtaylor.com.au), via links to printable versions on Dorman's other website. Also see a nice discussion of the inflammatory process and its relevance to prolotherapy by Allen Banks called "A rationale for prolotherapy" which is also accessible from my website.

Yours sincerely

Dr Margi Taylor, Adelaide

Dear Editor

In response to Margaret Taylor's letter:

I have no problems with your *local* effect of the needle.

I go to the next step *up to* and *in* the posterior horn, which is neatly shown in the Astra booklet, *The Pathophysiology of Pain*.

The needle is actually "damaging"/ "destroying"/ "reducing" the *total* inputs to the posterior horn, thus blocking the cacophony of inputs causing "wind up", thus allowing all the afferent pathways to calm down.

The established patterns then can "restore" and Chaos theory says the whole system down steps. (Restore the constant = no pain to restore the upset system to the previous state ... Nik's incredible input.)

Hence the reference to auditory feedback of a microphone close to a speaker causing the noisy feedback. To stop that feedback, you do one of three things;

1. Remove/switch off the microphone (injection effects on receptors, etc.)
2. Turn the amplifier off (drugs and posterior horn effects)
3. Turn down the volume = (a) increased A-beta inputs by movement, massage, etc.; or (b) via the dorsolateral funiculus (DLF) efferent pathways that modulate from the CNS higher centres downwards.

I have a CD Rom of the models that I had in Adelaide with this all pictorialized!

I returned to using the steroid + LA + needle because those treated with the needle alone or needle + LA reverted much sooner (after 3-6 months), and they came back for further injections. With the addition of the steroid they seem to last a lot longer. Also correction of leg length made a big difference too, with much longer periods before returning for more treatment.

However, there were some (very few) who had needle only or needle/LA who went 12-15 months before returning.

Just too many factors present – chaotic!

We need to work out *how* to determine who needs/will respond to what.

What do you have against good old steroids? Brief action (2-3 weeks), minimal total body effect (as measur-

able in Type 1 diabetes BGL levels), they work, etc.

Perhaps they prevent too much excessive effect from the needling.

Who else has experience and is prepared to discuss this interesting topic?

Yours faithfully

Dr Breck McKay, Brisbane

Dear Editor

I agree with Breck that the most important effect of the needle is probably neurophysiological in the dorsal horn. The effect of the steroid is more likely via effects on C-fibers than any anti-inflammatory affect. I'm in the Yelland camp of using steroids first then reverting to proliferant or LA solution if the patient needs multiple injections.

The next step is to clarify the algorithm, who suits what. Alas, more studies fellow musculoskeletal medicine denizens and fewer proclamations.

Yours faithfully

Dr Scott Masters, Caloundra

Dear Editor

This is a most interesting area of discussion as we see two apparently opposing treatments having a similar effect. That makes me wonder whether the neurophysiological effects on central sensitization may be the key mechanism of action. Trauma induced by needling with or without solution seems to have some effect on pain thresholds after the storm of irritation passes. I agree that Breck's approach is akin to the heavy needling used by the West Coast, but probably more aggressive. Is that why he reports such good results with so few treatments? The addition of steroid may help in three ways:

- (i) By reducing transmission in the C- and A-delta fibers
- (ii) By reducing peripheral sensitization (if neurogenic inflammation plays a significant role in spinal pain)
- (iii) By elevating mood - this seems to occur fairly frequently even with

one ampoule of Celestone.

Then there is a suggestion from the trials of Ongley et al and Klein et al and a small, unpublished trial by US neurosurgeon Wilkinson (comparing phenolic prolotherapy with lignocaine) that more irritant solutions containing phenol may be more effective for pain and disability than saline or lignocaine. This could be explained by the neurolytic effect of phenol. But then prolotherapists assert (from experience) that sodium morrhuate, which is also more irritant but not neurolytic, is more effective. Perhaps Margi could comment here.

Then there is the question, does it matter that much where you irritate a segment, as long as you irritate the right segment. The results from our trial on chronic low back pain using glucose/lignocaine on tender points concordant with pain diagrams were similar to the lignocaine group in the trial by Klein et al. They routinely treated the L4-S1 facet joint capsules and iliolumbar ligaments whereas I rarely ventured into those areas.

I am very interested in refining the predictors of success for prolotherapy.

Yours sincerely

Dr Michael Yelland, Brisbane

Dear Editor

Nigh on 20 years ago now I delivered a lecture at an AAMM or APS meeting in which I threatened a nightmare.

The proposition was that there was no such entity as musculoskeletal pain: that it was all neuropathic.

Even osteoarthritis (OA) is neuropathic (at the micro level). Radiofrequency neurotomy (RF) works 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. Cooking nerves resets the balance.

The arguments being raised about needle therapy are reminiscent of the 1940s and 1950s when counter irritation was all the go. In the 1990s sterile

water by IC injection was the rage in Sweden, until they banned it.

Could the result of the Bone and Joint Decade be: it's all neural. All treatments work by resetting the dorsal horn?

Yours sincerely

Professor Nikolai Bogduk, Newcastle

Dear Editor

This gives us all food for thought and reconsideration.

I have a paper in preparation called "Tennis Elbow Everywhere" possibly for November's green journal.

Scotty indicated that Michael Yelland had raised the point of segmental injections to achieve the same result.

I think that the injections are/should be given at the point of origin of the neuropathic message that causes feedback/wind up at the dorsal horn. So by injecting the tender/causative points it turns off the switch, the current ceases and the dorsal horn resets, thus all other responses diminish.

So what segments would be injected and where?

What are the criteria for segment selection?

The basic anatomy/function/physiology is critical to understanding, so segment choice must also be critical.

Any help?

Yours sincerely

Dr Breck McKay, Brisbane

Dear Editor

Breck's reference to tennis elbow (epicondylitis lateralis) highlights the different prevalent paradigms of the etiology of chronic pain.

While George Hackett coined the term *prolotherapy* in the 1950s, based on extensive animal studies and human clinical experience, Wall and Melzack published their "Gate Control Theory" in 1965. These two different views are diametrically opposed and poor old George lost the debate. A few hardy prolotherapy followers kept the practice going against

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much opposition. There is now a resurgence in the USA and Australia in prolotherapy. NZ is lagging behind, however.

There are two opposite views. Hackett claims most chronic pain arises in the periphery as a result of incompetent/weak connective tissue, that is, tendons and ligaments. Wall and Melzack support the CNS sensitization concept. Incidentally, Wall and Melzack abandoned their gate control theory in the 1980s.

Scientific support for the Hackett view is now coming from the sports medicine researchers. There is now international agreement on the histopathology of tendinosis/ligamentosis with the characteristics of hypercellularity, collagenolysis, revascularization, and increased abnormal ground substance all in the absence of inflammation.

Tendinosis is often wrongly called a degenerative process but Christopher and Maffuli and others in a review article in December 2004 on the etiology of tendinosis described the condition as an "exaggerated dysfunctional repair response" in the absence of inflammation.

Karim Khan et al urged medical practitioners in an editorial in the *BMJ* in 2002 to "abandon the myth of tendinitis." In it they argue that most tendon/ligament pain and dysfunction is due to an "osis"-process with far reaching consequences for diagnosis and treatment. For example, "the use of corticosteroids and NSAIDs are scientifically irrational and should be reviewed."

Swedish sports medicine researchers Haakon Alfredson and others at the University of Umea published in February 2005 the results of a double blind controlled crossover trial on the treatment of Achilles tendinosis with Polidocanol, a widely used European sclerosing/local anesthetic solution.

The study proves their theory that chronic pain associated with tendinosis arises from neovascularization/neoneurogenesis complexes identified with Color Doppler/USS.

It seems that medicine has come full circle and we are back where George

Hackett left off in the 1950s with the additional scientific findings of chronic pain arising from "tendinosis complexes" which are eminently treatable with sclerosing injections.

Prolotherapists have always claimed that a hypertonic glucose solution does the same, that is, a reintroduction into the dysfunctional tendon/ligament of immune competent cells and the start of a proliferative process resulting in a functional repair response leading to stronger functional connective tissue.

Those wishing to practise prolotherapy effectively now know the underlying pathology and etiology of "weak" tendons and have a rationale for their treatment.

The sooner we abandon the myth of CNS sensitization the better we can serve our patients with chronic pain.

With regard to lateral epicondylitis, it is important to inject over a large enough area to initiate an inflammatory reaction in the area affected by tendinosis. I don't find it necessary to needle any periosteum or enthesis per se, just a subcutaneous peritendinous/periligamentous deposition of diluent is enough. Directly needling an Achilles tendon is extremely painful for days afterwards and has an adverse effect on the outcome, so a similar technique is warranted.

I have submitted the results of my prospective pilot study on prolotherapy and Achilles tendinosis to the journal and the references to the above mentioned articles are in there.

Prolotherapy has taught me that most locomotor pain is in the periphery and diagnosable with palpation and treatable with prolotherapy.

This has profoundly altered my practice after nearly 30 years of sports/musculoskeletal medicine in a general practice setting in the conventional way. I am now a fulltime prolotherapist and this is the most rewarding phase of my medical career in terms of relieving patients of their pain, chronic or otherwise, and in all ages. The youngest, aged 12, with disabling Osgood Schlatter Disease and the oldest, a 91-year-old with four years of intense chronic knee pain following a total knee reconstruction, have had their

pain effectively relieved.

My next article will be on a pilot study on prolotherapy and compartment syndrome and I am also compiling a prospective audit of all my prolotherapy patients over 2005 with recoverogram monitoring with follow up.

Yours sincerely

Dr John Lyftogt, Christchurch

Dear Editor

I'd like to thank John for joining in this productive debate and for his great work in documenting his responses to treatment. I hope he will present them at the 2006 NZMM conference "Spine in Action".

I'm not totally convinced with his argument on mechanism because:

- 1) Many tendinoses are not painful.
- 2) Not all chronic pain responds to prolotherapy.
- 3) The mechanism of prolotherapy has not been fully elucidated. Michael Yelland's study suggests that the needle may be the most important factor, that is, via its influence on the dorsal horn.
- 4) Corticosteroids have an effect other than anti-inflammatory - their effects on modulating C-fibers. That is probably why they are effective in CRPS. The use of NSAIDs I agree needs review but the COX-2 situation is helping people reflect on their use and misuse.
- 5) Polidocanol is an emerging treatment but again its effect in part could still be on the dorsal horn - further studies will flush this out.
- 6) We should design our own studies to try to answer some of these questions.

Yours sincerely

Dr Scott Masters, Caloundra

Dear Editor

It is great to hear that John's study on the treatment of Achilles tendinosis and prolotherapy is nearing publication and that he has more results in the pipeline. I look forward to reading them soon. I have adopted his technique for

treating a few Achilles tendinosis patients with good results so far. The debate about the mechanism of action of prolotherapy is a very interesting one. It will be ongoing as it is very hard to prove conclusively whether it works by strengthening weakened ligaments/enthesees or by resetting the dorsal horn (or by some other mechanism). I have been asked by FIMM to write an article on this general area and hope to publish it in *Australasian Musculoskeletal Medicine* late this year.

I agree with John's assertion that tendinitis is often a myth and that histological studies show more a pattern of tendinosis. I'm not aware of biopsy studies comparing the histology of painful tendons with that on the opposite (non-painful) side, but they would be helpful in understanding the link between tendinosis and pain. Ultrasound studies offer some insights.

Most would be aware of the poor correlation between tendinopathy and pain in the shoulder, but this correlation could be different for other tendons. If tendinosis is indeed the cause of pain in the periphery, the evidence for tendinosis or ligament pathology in the spine is still lacking.

There is a biopsy study of sacroiliac ligaments by Klein et al¹ of three patients with chronic low back pain that showed increased fiber thickness and fibroblastic activity in the dorsal sacroiliac ligaments following prolotherapy compared with the contralateral ligaments before prolotherapy, but there is no mention of tendinosis in the untreated ligaments.

More work needs to be done in this area. This lack of good evidence and the observation that many different treatment methods ranging from prolotherapy to steroid injections to exercise, manipulation and CBT can have some effect on chronic pain leads me to speculate whether resetting the dorsal horn could be a common pathway for many different treatments. I don't think we should reject this mechanism, but it too needs more research. The handout I give my patients mentions both strengthening of ligaments and entheses and resetting the pain threshold as possible mechanisms of action

of prolotherapy.

Regardless of the mechanism of action of prolotherapy, I think the way forward is to refine the predictors of response so that prolotherapy will be targeted on those most likely to benefit. In our study on prolotherapy for chronic low back pain,² we found only four predictors in the lumbosacral spine. Those with high initial depression scores and those with previous use of over four types of treatment showed poorer pain and disability responses. Smokers and those with high initial anxiety scores showed poorer disability responses.

Demographic and other clinical variables including duration of pain, presence of leg pain, physical demands or work, and x-ray findings did not predict response. Allen Hooper, from Canada, recently published a case series of 177 patients with chronic spinal pain that hinted at some predictors of response.³ Based on this, he has suggested the following selection criteria for further studies:

- 1) concordant pain diagram drawing,
- 2) local tenderness over the affected ligament,
- 3) positive response to bracing or taping,
- 4) exclusion of other organic cause for the symptoms, systemic illness, and vitamin or hormone deficiency.

Comparing spinal regions, he observed a better response in the lumbar and thoracic spines than in the cervical spine, but this could be explained by the higher prevalence of cervical pain patients being involved in MVAs.

We are now looking at whether the historical features of so-called "lumbar instability" or the "theater-cocktail party syndrome" (that is, pain induced or exacerbated by prolonged standing or sitting and relieved by activity) may be a predictor of response to prolotherapy.

Kind regards,

Dr Michael Yelland, Brisbane

1. Klein RG, Dorman TA, Johnson CE. Proliferant injections for low back pain: histologic changes of injected ligaments and objective measurements of lumbar spine mobility before and after treatment. *J*

Neurolog and Orthopaed Med and Surg 1989; 10: 123-126.

2. Yelland MJ, Glasziou PP, Bogduk N, Schluter P, McKernon M. Prolotherapy injections, saline injections, and exercises for chronic low-back pain: a randomized trial. *Spine* 2004; 29:9-16.

3. Hooper RA and Ding M. Retrospective case series on patients with chronic spinal pain treated with dextrose prolotherapy. *J Altern Complement Med* 2004; 10(4): 670-674.

Dear Editor

Scott and Michael's responses to my email illustrate the general lack of awareness of the basic research taking place in the world of sports medicine. Hence I'll add some more of my interpretations of this research.

Rolf and al. from the University of Hong Kong conducted the only (known to me) controlled histological trial on patellar tendinosis comparing normal patellar tendon surgical specimens from ACL reconstruction with surgical pathological specimens from surgery to patellar tendons.

Amongst other findings they confirm yet again the histopathology of tendinosis, that is, increased cellularity, collagenolysis, increased and abnormal ground substance, and neo-vascularization, all in the absence of inflammatory infiltrates. Their comment on the general microscopic picture was that the condition resembled "a halt in the early phase of healing". These researchers have written a number of very good articles on the pathology of tendons, which are well worth reading.

Khan et al. in their *BMJ* editorial in 2002 further comment on the histopathological events that may lead to tendinosis. As there are no human studies, for obvious reasons, on the sequence of events following injury, insights in this area depend on studies of surgical trauma to rabbit tendons. These show that the initial inflammatory phase peters out after 12-15 days and that healing stops. This is followed, the authors speculate by an "exaggerated, dysfunctional repair

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response" (Christopher and Maffuli) called tendinosis described above.

Both above-mentioned descriptions "a halt in the early healing response" and the "exaggerated, dysfunctional, repair response" suggest impaired healing.

H Alfredson, an orthopedic surgeon from The University of Umea in the north of Sweden, had a bet some years ago with a colleague that he could beat him in an upcoming 30 km run. While training for this he developed a non-insertional Achilles tendinosis which threatened to thwart his efforts. He attended the university physiotherapy department and was put on an intense, painful eccentric strengthening exercise program and 12 weeks later he completed the 30 km run. Curious about the pathophysiology underlying this, he and others decided to do more basic research. The results were a controlled trial on intense, painful eccentric strengthening exercises, the only one of its kind so far. Then a controlled biopsy study on lactic acid levels in normal versus tendinosis affected Achilles tendons showed that tendinosis is associated with anaerobic metabolism.

This was followed by color Doppler/US studies evaluating neovascularization. A theory was developed that the neovessels may well be associated with unmyelinated afferent nociceptors, a common biological response in tissue repair or in tumors. A widely used sclerosing/anesthetic solution Polidocanol was injected under color Doppler guidance until flow in the neovessels ceased. The pilot study on this was published three years ago and the follow-up double blind controlled study published last month.

They concluded that the disappearance of pain was associated with the sclerosing of the neovessels and the associated neurons.

Here we have good scientific support for the view that pain starts at the peripheral level.

Incidentally, I have treated several patients with extremely painful Achilles tendinosis associated with extensive areas of allodynia, all strongly suggestive of complex regional pain syn-

drome (CRPS). These patients without exception have responded with complete resolution of their allodynia and tendinosis to prolotherapy.

Possibly to confuse things even further, Christopher and Maffuli in their review article in December 2004 on "the etiology of tendinosis" (reprinted in the ADIS review) maintained that the scientific evidence now supports the view that tendons are subject to differential strains, that is, some parts of the tendon are stressed more and other parts are "stress shielded". As tendons adapt to workload, albeit slowly, some parts of the tendon strengthen and some parts weaken. Significantly, it is the "stress shielded" areas that first develop tendinosis. Hence they argue that tendinosis is a result of "underuse" not "overuse".

The above research results indicate that we now know the histopathology of tendinosis. There is strong scientific evidence for the etiology of tendinosis as an "under use" phenomenon. There is separate evidence of the etiology of pain in the periphery associated with tendinosis from Sweden.

The clinical findings in Achilles tendinosis vary and the US grading (1, 2, and 3) has been proposed in at least two papers (also referenced in my Achilles tendinopathy/tendinosis paper). Patients present only when pain starts to interfere with their activities and Khan and others argue that the process of tendinosis begins some time before the pain starts. Hence there are different degrees of tendinosis and some are associated with pain. It is the pain that patients want treated!

I am not a born-again prolotherapist and I know first hand that mesotherapy and neural therapy are also effective in treating pain, chronic and otherwise. Sclerotherapy with Polidocanol seems attractive although there are some unresolved issues about the possible sclerosing effects on nearby larger sized nerves. One study has shown this traveling 6 cm from the site of injection.

The advantage of glucose is its effectiveness and absence of side effects. It is cheap, although not easy to learn.

Rest me to say that pursuing the CNS for the etiology of pain confuses the etiology of the perception (consciousness) of pain with the etiology and pathology of the source of the pain itself. Hence "management of pain" is pursued, and not "treatment of pain". Consciousness of pain is a fascinating subject of course, but academic and of little consequence to the patient.

The treatment of pain is what patients expect, particularly by clinicians who choose to work at the coal face of medicine. We have had 50 years of focusing on the perception of pain without any results that matter to our patients. It is now time to look the other way and come up with treatments of pain that work. Prolotherapy, mesotherapy, neural therapy, and sclerotherapy with Polidocanol are a good and sound start.

May the debate be vigorous and unrelenting.

Cheers

Dr John Lyftogt, Christchurch

Dear Editor

In reply:

Thanks John for those insights. I keep an eye on the sports medicine literature and was aware of the Swede's 2002 pilot study but was unaware of their recent RCT publication. Was that in the *Br J Sports Med*?

I'm still not sure what you have against the dorsal horn being an amplifier? The periphery starts the signal but why does it persist and why do we get the allodynia as you also have noticed with your patients? Why do some patients get neovessels and others don't? Are all neovessels painful? Does the injection have to be US guided?

I notice the Swedes are also promoting their injections for insertional pain (presumably enthesopathies) at the calcaneum as they have seen neovessels there as well. They stated that they achieved good results when there was associated tendon or bursal pathology on the US but poor results if bony pathology (spurs, bony fragments) was present.

We need to design our own trials to attempt to further clarify the best treatments

Yours sincerely

Dr Scott Masters, Caloundra

Dear Editor

In reply:

Dear Prolotherapists

I have just visited my musculoskeletal medicine veterinarian mate in Melbourne who has been doing prolotherapy for 20 years using iodine in almond oil which he has made up by a contemporaneous pharmacist. He uses the solution in racehorses and dogs with good results.

The same source told me that he was sitting beside Professor Blomberg at the Melbourne conference dinner in 2002 and asked him what was the reason that he used steroids in his solution, and he said words to the effect "just because I can".

My recent reading informs me that Sir William Osler, at the beginning of last century was plunging needles into lumbar spine muscles for the relief of pain (without injecting any solution).

Does prolotherapy work in areas other than the pelvis, for example, neck, shoulder, upper extremity, lower extremity, and thorax? If so, what are the weak ligaments that are injected?

Why is it that so many people who complain of painful stiffness have weak ligaments?

How do people get weak ligaments?

When I have used a prolotherapy solution some people have improved immediately (and some did not improve at all).

If prolotherapy works by laying down fibroblasts - collagen to strengthen a ligament which I would imagine would take quite a number of weeks why did the treatment work so quickly, that is, in a matter of minutes to hours rather than the expected weeks? Did the weak ligaments strengthen fairly instantaneously or did the pain threshold in the dorsal horn virtually immediately increase after being depressed for quite a considerable time with all the perhaps permanent/semipermanent mo-

lecular and cellular changes that take place in the dorsal horn and the descending inhibitory pathways.

These are the problems that I am wrestling with.

Thank you for these answers in anticipation.

Dr Peter Jackson, Brisbane

Prolotherapy and Achilles tendinopathy: A prospective pilot study of an old treatment

John Lyftogt, New Zealand

Abstract

Background. Prolotherapy has been successfully used for over 60 years in the treatment of a large variety of musculoskeletal conditions. No studies on prolotherapy and Achilles tendinopathy have been published.

Objective. This prospective pilot study assessed: (1) the clinical effectiveness of prolotherapy in the treatment of Achilles tendinopathy in a general medical/sports medicine setting with three-month follow up; (2) a postulated positive relationship between initial visual analogue scale (VAS) scores and number of treatments.

Patients and Methods. All 16 patients with 19 Achilles tendinopathies presenting over a 16-week period were included in the study. All patients were treated weekly with a standard prolotherapy solution of 20% dextrose and 0.1% lignocaine. Results were monitored with individual prolotherapy recovergrams which were compiled in a study recovergram.

Results. Fourteen patients were satisfied with the results of the treatment and returned to pre-injury levels of activity. One patient was referred for bilateral Haglund exostostomy and decompression surgery. One patient was not satisfied but was not available for follow up. Of the 14 Achilles tendinopathies reaching VAS=0 at the end of treatment, 11 remained at VAS=0 at follow up. The study-recovergram showed a positive correlation between initial VAS score and number of treatments.

Conclusions. Prolotherapy is a safe, effective, and cheap treatment for Achilles tendinopathy in this pilot study. Recovergrams are an effective clinical tool for monitoring progress, evaluating effect, and predicting duration of treatment.

Background

In the *British Medical Journal* editorial (16 March 2002)¹ the authors alert all medical practitioners dealing with

lateral and medial elbow pain, rotator cuff problems, achillodynia, patellar tendinopathy, and others, with the banner "Time to abandon the 'tendinitis' myth."

These conditions should be called "tendinosis" (or tendinopathy), as was first described in the *American Journal of Sports Medicine* in 1976.²

The authors finish the editorial by saying that "adopting the tendinopathy paradigm is essential if general practitioners are to practise evidence-based medicine. However, there remain many unanswered questions, particularly with respect to treatment."

Other researchers have responded with treatment trials and research in line with the new paradigm.^{3,4} Hence there is now a growing literature showing the beneficial effect of eccentric strengthening exercises in Achilles tendinopathy and also a new treatment by Swedish researchers with ultrasound-guided sclerosis of neovessels in painful chronic Achilles tendinopathy.⁵

Decompression surgery could also be considered to address tendinopathy along this paradigm, although it is generally viewed as a last resort treatment.

George Hackett,⁶ who first coined the term "prolotherapy" in the 1940s, carried out extensive animal studies on rabbit tendons, with proliferants showing an average increase in tendon diameter of 25-40% after 4-6 weeks of treatment. This resulted in an increased strength and load to failure, particularly at the fibro-osseous junction.

Like many prolotherapists, the author believes that prolotherapy is also an appropriate response to the paradigm of tendinopathy.

Patients and methods

All 16 patients (four women and 12 men) with 19 Achilles tendinopathies presenting over a 16-week period to Active Health, a sports medicine and rehabilitation clinic, were included in the pilot study.

All patients were either self-referred or referred by a sports medicine colleague specifically requesting prolotherapy. All patients except one had received extensive physiotherapy/eccentric strength exercises/acupuncture/podiatry, and many other treatment modalities without resolution. The median duration of symptoms was 14 months (range 0.5-600). Median age was 48 years (range 37-59).

In two patients the condition threatened their occupation (both police officers) and in 10 patients their participation in masters triathlon and running had ceased, causing an adverse effect to their overall health and well-being. The remaining four patients experienced difficulty with walking and/or reported night pain.

A distinction between insertional (3) and non-insertional (16) Achilles tendinopathy was made on clinical grounds. Insertional tendinopathy may well have a different etiology, as a common underlying Haglund exostosis causes compression injury to the anterior entheses, ultimately requiring surgical removal of the exostosis. Confirmation of this diagnosis is with a lateral x-ray of the os calcis. One patient was referred for bilateral Haglund exostostomy. Non-insertional Achilles tendinopathy traditionally affects the 2-7 cm area proximal to the os calcis.

Prolotherapy was administered weekly where possible. The treated area was identified by palpation of the swollen and painful areas. Injection sites were marked 15 mm apart on the postero-lateral and postero-medial side of the tendon. A "bleb" of local anesthetic (lignocaine 2%) was injected in marked sites. Then 1 ml of prolotherapy diluent consisting of dextrose 20%/lignocaine 0.1% was injected very slowly into each site subcutaneously, carefully avoiding the paratenon. Even minor needle trauma of the paratenon causes prolonged pain, discouraging patient and practitioner alike. The aim is to achieve complete local anesthesia at the time of treatment as this indicates

Prolotherapy and Achilles tendinopathy

that all areas causing pain are treated.

Each patient was monitored with a recovergram recording pain VAS scores with 0 = no pain and 10 = worst imaginable pain. Once familiar, most patients volunteered their VAS score at the beginning of each consultation. Most VAS scores declined in a linear fashion, the exception usually caused by accidental needle trauma of the paratenon the previous week.

Results

Fourteen patients with 16 Achilles

tendinopathies were satisfied with the treatment.

One patient was not satisfied although his VAS score went down to 0. He was not available for follow up due to being stationed in Antarctica.

One patient with bilateral insertional Achilles tendinopathy was referred for surgery after receiving four treatments reducing his pain level from VAS 7 to 4 while waiting for surgery.

Of the satisfied 16 Achilles tendinopathies, 13 VAS scores went down to 0, with three at follow up going

respectively to a VAS score of 1, 2, and 3, and without return of previous disability. One patient with a 50-year history chose to stop after six treatments at VAS score 3 (down from 8) with no change at follow up and one patient interrupted her treatment for six months due to overseas travel. At the time of writing she had resumed her treatment and is now nearly pain free (VAS=1). One patient went from VAS 5 to 1 with same at follow up.

The study recovergram graphically demonstrates the outcome of

Prolotherapy Recovergram

(after Dr P Watson Aust Mus Med Nov 2000)

Name: GI

Date of onset: September 2003

Address:

Hx : Training for Masters World Champs Triathlon. Injured L Achilles. Has had several previous episodes of Achillodynia since age 20. Usually settled with rest and stretching. This episode treated with Physio x 30 and Acupuncture x 6.

Phone:

Age: 56

Sex: male

Occupation:

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

F/U

Year:	Month:	4		4		5	5		5	5	6	6	6	6	7				12
Day:	6		20	27	4	11		19	25	1	8	16	22	6					7
10																			
9																			
8																			
7	x																		
6																			
5			x	x															
4					x	x													
3								x	x										
2										x									
1											x	x	x						
0														x					x

ACTIVITIES LIMITED

Running

Walking

2		1	1	1	0		1	1	1	0	0	0	0					0
1		1	1	0	0		0	0	0	0	0	0	0					0

Disability Rating:

0 = no limitation, 1 = can do slightly limited, 2 = can do with difficulty, 3 = can't do at all

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prolotherapy in this pilot study. It also illustrates the clinical experience that the initial pain VAS score is a good guide for assessing length of treatment, that is, the higher the initial VAS score the longer the expected duration of treatment. It does not, however, predict success of treatment.

Discussion

A recent review article in *Sports Medicine*⁷ by Constantinos at al. on the biomechanics and pathophysiology of overuse tendon injuries has identified the presence of intratendinous differential strains, with some parts of the tendon preferentially loaded and other parts “shielded”. In general it is the “shielded” areas that are initially affected by tendinopathy. Alternatively, they say compression factors between bone and tendon at the enthesis, or thermal damage and injury may also be involved in the etiology of tendinosis. Exercise temperatures above the 42.5°C threshold viability for fibroblast have been recorded.⁸

The authors also argue that the traditional view of a tendon overuse injury as a result of tensile overload does not stand close scientific scrutiny and that mechanical “underuse” may be important in the etiology of tendinopathy which they describe as “an exaggerated dysfunctional repair response”. This view of tendinopathy is similar to what Rolf et al. describe in the first and

only controlled study on the histopathology of patellar tendinopathy in *Rheumatology* in 2001.⁹ These authors interpret the hypercellularity, disturbance of the collagen matrix and increased proteoglycans found in tendinopathy as strongly suggestive of a “halt in the early phase of tendon healing, except for the absence of the inflammatory response”. They also point out that although some authors regard tendinopathy as a degenerative disorder it also occurs in young people.^{10,11} In this respect it is interesting that patient TS, age 57 in this pilot study, sustained his initial Achilles tendinopathy at age seven when flying down hill on his tricycle, a birthday present. The right tricycle paddle hit his Achilles tendon at full speed causing a major contusion of the tendon at the time and chronic intermittent pain and disability for the next 50 years. He responded well to six prolotherapy treatments with considerable thinning of the tendon and reduction of his VAS score from 8 to 2 (3 at follow up).

Neovascularisation in Achilles tendinopathy was suspected to be part of the pain mechanism by the Swedish researchers Ohberg and Alfredson in their article “Ultrasound-guided sclerosis of neovessels in painful chronic Achilles tendinosis: pilot study of a new treatment.”¹⁵ Nociceptive afferent ingrowth associated with the neovascularisation is postulated to be the

etiology of pain in tendinopathy.

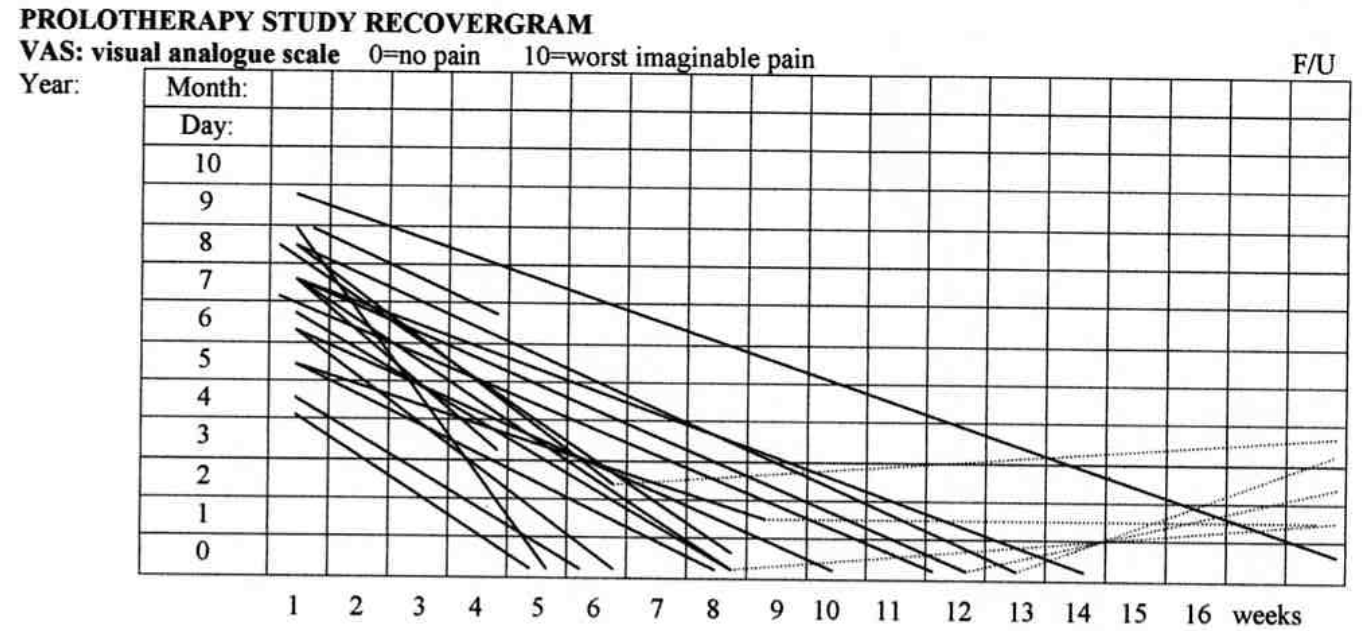
The new vessels were located on the ventral and distal side of the Achilles tendon in the “stress shielded” areas⁷ of the tendon. These were sclerosed with Polidocanol resulting in significantly reduced pain levels.

Since the inception of prolotherapy by George Hackett⁶ in the 1940s, practitioners have maintained that rehabilitation of an incompetent structure, such as ligament or tendon by the induced proliferation of new cells, constitutes an effective treatment for musculoskeletal disability. Hence this pilot study’s premise that inducing a physiological inflammatory/proliferative/remodeling healing response with the standard prolotherapy diluent dextrose 20% and lignocaine 0.1% will effectively strengthen an “incompetent” Achilles tendon, resulting in a lasting resolution of pain and dysfunction.

Discussion on the etiology of tendinopathy will no doubt be ongoing. The histopathology is, however, generally accepted.^{1, 9, 12}

The weakness of this study is the lack of a control group and short follow up. A suggested comparative trial of eccentric strengthening exercises, Polidocanol sclerotherapy, prolotherapy, and surgery with one-year follow up should answer many outstanding questions.

As the incidence of Achilles tendinopathy increases with age¹³ rec-



ommended treatment protocols could be different for different age groups with different color Doppler flow imaging grading 0-3¹⁴ or ultrasonography grading 1-3.¹⁵

It is quite feasible that younger athletes with low grade tendinopathies may well respond better to an eccentric exercise regime than masters' athletes with an ultrasound grade 3 tendinopathy.

Surgery could then be delayed until all medical options were exhausted, as rehabilitation from surgery requires a lengthy rest followed by an intense exercise regime often lasting more than six months.

The use of recovergrams in clinical monitoring was first proposed by Dr Philip Watson in Australia in 2000.¹⁶ Pain VAS scores and disability scores are validated parameters in clinical monitoring.

This pilot study also demonstrated in the study recovergram an interesting connection between the initial pain VAS score and the number of required treatments, that is, the higher the initial VAS score the more treatments were needed. This was particularly evident in the patients with bilateral Achilles tendinosis with different initial VAS scores for left and right. The author has observed the same phenomena in bilateral lateral epicondylitis and knee pain and found this helpful in advising new patients on how many treatments they might need.

In conclusion, this pilot study shows the effectiveness of prolotherapy in Achilles tendinopathy and the benefits of clinical monitoring with a recovergram.

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Indications for low back prolotherapy*

Dr Robert F. Kidd, Private Practice, Renfrew, Ontario, Canada

Assessment is not simple, but prolotherapy usually provides satisfactory and permanent results if patients are selected carefully.

Prolotherapy is an injection therapy used to treat ligament, joint capsule, fascial, and tendinous injuries. It is used to stimulate proliferation of new connective tissue at the site of injury, thereby restoring strength to injured and weakened connective tissue. Solutions such as dextrose 12.5% are commonly injected creating a controlled inflammatory response. The proliferating substance provokes an inflammatory cascade, the later stages of which include the deposition of collagen. This new collagen is identical in every way to the pre-existing collagen.

Prolotherapy is also known as sclerotherapy, a term still used in Britain and by many osteopathic physicians in the United States. Prolotherapy is preferred by some because it avoids suggesting the hardening of tissues implied in the Greek root *scleros*. Other terms sometimes used in lay publications include ligament or joint reconstructive therapy.

From ancient times, prolotherapy has been practised sporadically in different forms. Hippocrates recommended the use of hot cautery to treat recurring shoulder dislocation; however, the modern era of prolotherapy began with the injection of synasol into the temporomandibular joint in 1937 by Schultz, a dentist.¹ In the same year, an osteopathic physician, Gedney, reported injecting the sacroiliac ligaments with neoplasmoid and McDonald's solution, neither of which are currently used.² Gedney taught at the Philadelphia College of Osteopathic Medicine, where he sowed the seeds of sclerotherapy. Teaching and research have continued to this day through the American Osteopathic Academy of Sclerotherapy.

In the early 1940s, George Hackett, a trauma surgeon in Canton, Ohio, began to study ligament injury in his patients. Drawing upon the observations of Head, Baer, and perhaps

Kellgren, he mapped pain referral patterns that occurred in his patients with post-traumatic chronic pain.³⁻⁵ If pain were reproduced by the irritation of injection and relieved by local anesthetic, he deduced that the injected site was the source of the patient's pain. Since many of these pain sources occurred in ligaments, he also concluded they were actual sites of injury and that the ligaments had been made lax by trauma.

Hackett coined the term *prolotherapy* and wrote a monograph describing these pain patterns and methods of diagnosing and treating ligament laxity.⁶ Updated in recent years by Hemwall and Montgomery, it remains the most widely known text on prolotherapy.

After two widely publicized medical disasters involving prolotherapy in the 1960s, prolotherapy fell into disuse. The ideas and techniques were kept alive during this period by a handful of British physicians and the Prolotherapy and Sclerotherapy Societies.⁷ The latter two societies were made up largely of American medical doctors and doctors of osteopathy. During the 1980s, a modest revival occurred, resulting in the publication of several key studies on prolotherapy. Among these was an animal study that demonstrated that collagen was indeed formed by injections of sodium morrhuate, which significantly strengthened the fibro-osseous junction.⁸ Another controlled clinical trial involving a large group of patients showed that low back prolotherapy was of lasting benefit.⁹

Published studies have continued to demonstrate the value of prolotherapy in low back and knee pain.¹⁰⁻¹² In the United States, the Hackett Foundation, the American Association of Orthopaedic Medicine, and the American Osteopathic Academy of Sclerotherapy teach prolotherapy regularly, offering 2- and 3-day courses. Approximately 600 physicians now practise prolotherapy in North America.

Ligament and muscle anatomy

Ligament, fascia, joint capsules, and muscles are all of mesodermal origin.

Evolutionary history shows that muscle is the most primitive of these tissues. This is a counterintuitive observation, since it might seem that tissues necessary for active motion are more highly evolved than passive tissues. Nevertheless, ligaments only appear in mechanically more evolved animals where strength and speed are important. A phylogenetic example is the differentiation of the lower part of the quadratus lumborum into the iliolumbar ligament in early adulthood in man.

The evolutionary origin of muscle and ligaments has both mechanical and clinical implications and is intertwined with function. Tension on ligaments is modulated by muscle attachments directly onto ligaments (for example, gluteus maximus onto the sacrotuberous ligament) or by muscles running parallel to ligaments (paraspinal, multifidus, and intertransversarii muscles and intervertebral ligaments). Muscle may even modulate the tension of a joint capsule. This should not be surprising if the tensegrity model of mechanics is accepted.¹³ Ligaments and muscles are tension elements essential to the strength and stability of the whole.

Ligaments protect muscles from injury and allow them to rest under certain circumstances, for example, locking of the knee and the hip when standing easy. Ligaments not only transmit forces generated by muscles, but also may, through their elastic properties, have an energy-storing effect. Dorman has hypothesized that this is a key function of the pelvic ligaments in gait.¹⁴

Because ligaments are stronger than muscles and cannot give way when overloaded, they probably bear the brunt of external trauma, especially when large forces are involved. Commonly a cascade of ligament injury occurs in which the number of injured ligaments in a region is proportional to the severity of the trauma. This phenomenon is well recognized in the shoulder and forms the basis of the classification system of acromioclavicular injuries; the coracoclavicular

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ligaments become involved only in the more severe (class 3 and 4) injuries. Hackett believed that the posterior sacroiliac ligaments were the most commonly injured. The interosseous, sacrospinous, and sacrotuberous ligaments appeared to be involved in more serious injuries. With single ligament injuries, however, the iliolumbar ligament is most often implicated. This may explain why pain from the iliolumbar ligament has been described as a distinct syndrome, whereas pain from the other low back ligaments has not.¹⁵

Although in vitro studies had shown that peripheral ligaments most commonly fail in the midsubstance when stressed,¹⁶ this is not necessarily true in vivo particularly for the more central ligaments of the low back. Clinical experience seems to suggest that the fibro-osseous junction is the more common site of injury, at least in those instances that become chronic.

This is the working hypothesis from the point of view of prolotherapy, where permanent laxness is believed to occur at this spot. Trigger points then develop because the sensory nerve endings are more vulnerable to the otherwise innocuous strains of everyday life.

Mechanics

Low back mechanics are exceedingly complex. Despite many years of research on the topic, clinicians cling tenaciously to different schools of thought. This reflects not only poor standards of examination, but also a lack of communication between clinicians and researchers.

Probably no group of clinicians has carried clinical assessment of low back mechanics as far as the osteopathic profession. From the early 1900s, Lovett and later Fryette described the mechanics of the spine in great detail.^{17,18} Mitchell, Greenman, and Kuchera have demonstrated clinical methods of examining the pelvis and have outlined the mechanical assumptions that accompany them.¹⁹⁻²¹ These assumptions include recognition of multiple axes of motion, both physiologic and nonphysiologic somatic dysfunctions and a close interaction with surrounding muscles and total body mechanics.

Although these examination meth-

ods are widely accepted by the osteopathic profession and many physiotherapists, interexaminer reliability remains a problem. Also, the observations are unintelligible to those not trained in manipulation. Technology does not yet exist that can demonstrate to the non-initiated the mechanics postulated by the osteopathic profession.

The allopathic understanding of low back mechanics has for the most part remained primitive. In the early part of the last century, sacroiliac strain was a common diagnosis and some research implicated the pelvic ring.^{4,22} Mixer and Bar's paper on disc herniation in 1934 changed all this, however, and most low back mechanical research has since centered on the disc and the surrounding structures.

A small but steady stream of papers on sacroiliac anatomy and mechanics continued in parallel through the 1950s, 1960s, and 1970s, mostly by nonclinicians.²⁴⁻²⁶ This research added little to the knowledge of physicians already familiar with sacroiliac joint examination; however, the prevailing view that the sacroiliac joints are immobile proved irrational.

In the early 1980s, a number of new developments began to challenge this stagnant situation. Osteopathic and medical physicians began to interact, first in the North American Academy of Manipulative Medicine and soon after in the American Association of Orthopaedic Medicine.

Manual medicine flowered in Europe and trans-Atlantic communication increased. Physiotherapy and chiropractic became increasingly sophisticated and cooperation between these and the medical professions improved. Consequently, the need for better research, especially in mechanics, became apparent.

In the 1990s, two international conferences on the sacroiliac joint were held, bringing together physicians, physiotherapists, anatomists, engineers, and other researchers from North America and Europe. First-class research on all aspects of sacroiliac joint function and dysfunction was presented. Of particular interest to those interested in prolotherapy was the relative importance of ligaments and mus-

cles in joint stability.

The engineering terms "form closure" and "force closure" were introduced to differentiate the passive (ligament and joint) mechanisms versus the active (myofascial) mechanisms providing joint stability.²⁷ Although no consensus yet exists as to their relative importance, both appear to be important in sacroiliac joint stability.

Although the understanding of pelvic ring mechanics has advanced greatly in the last decade, the central challenge of demonstrating the relationship between abnormal mechanics and pain remains unmet.²⁸⁻³⁰

Low back stability

Instability can be defined as a loss of the functional integrity of a system that provides stability. It is a broad term that can be applied to any dynamic system. When applied to the low back, it can mean anything that consistently interferes with normal function.

Hypermobility and hypomobility are mechanical terms that directly apply to orthopaedic medicine.

Hypomobility refers to the restriction of motion of any body part. Hypermobility refers to excessive range of motion and is of special interest to physicians practising prolotherapy.

Joint hypermobility may be categorized into two types: *primary* and *secondary hypermobility*. Primary hypermobility results from a weakening of the joint capsule and ligaments. Peripheral joint hypermobility occurs commonly following injury, but central axial joint hypermobility may occur as well. The most obvious examples are sacroiliac joint hypermobility and intervertebral hypermobility after motor vehicle accidents. Hypermobility of this kind may be difficult to detect clinically as muscles will compensate in many different ways. Piriformis syndrome, quadratus lumborum syndrome, and psoas syndrome are just a few of the muscular patterns that may develop in response to hypermobility in the pelvic or lumbar intervertebral joints.

Secondary hypermobility may develop as a result of abnormal neuromuscular control of a joint or group of joints. Weakness of passive supporting structures may not exist, but

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the mechanical effect may be identical to that of a primary joint hypermobility.³¹ An example of secondary joint hypermobility would be sacroiliac joint hypermobility caused by a change in muscular tension of the gluteus maximus muscle acting upon the sacrotuberous ligament. The sacrotuberous ligament is a major support of the sacroiliac joint.

The origin of secondary hypermobility may be quite complex, since the neuromuscular abnormality may be part of a much larger postural pattern. Abnormalities emanating from mechanical disturbances in the lower extremity, trunk, neck, and cranium are common and make treatment of the local problem inefficient or impossible if not addressed.

The autonomic nervous system may affect muscular balance by facilitating muscles or activating muscle trigger points. Sources of autonomic nervous system destabilization include visceral disturbances (viscerosomatic reflexes) and foci of electrophysiologic instability ("interference fields") such as scars, teeth, and nerve entrapments.³²

Clinical assessment of low back instability

Both a medical history and physical examination are important. Radiologic studies of the pelvic ring may demonstrate mechanical abnormalities when stressed in different ways,³³ but their clinical significance is not known. Lumbar spine dynamics have been studied radiologically with little success. The Spinoscope, a device that tracks intervertebral motion using light-emitting diodes taped to the skin, shows promise for the lumbar spine, but is ineffective for the pelvic area.

In an unstable back, pain may be induced or exacerbated by prolonged standing or sitting, and relieved by activity—the so-called "Theatre-Cocktail Party syndrome," coined by Barbor.⁷ Clinical experience has shown that prolotherapy benefits these patients. Some have concluded that patients with unstable backs have ligament laxity with or without secondary muscular trigger points and pain.

Another symptom of low back instability is recurrent episodes of acute back pain associated with unguarded

movements of the trunk possibly resulting from lack of ligamentous mechanical support. Patients with this problem benefit from prolotherapy, but less so than those with "Theatre-Cocktail Party syndrome."

There is no consensus about which physical signs indicate low back instability. The osteopathic concepts of physiologic and nonphysiologic somatic dysfunction imply that the soft tissue supports of the sacroiliac joints may, under certain circumstances, decompensate and cause a true subluxation of the sacroiliac joint. Others consider recurrent pelvic asymmetry or "asymlocation" to be significant. Whether this apparent asymmetry results from displacement of bones or from changes in the overlying soft tissues has been called into question.³⁴

The sitting and standing flexion sign (or variations of these tests) are widely used to assess pelvic ring mechanics, but their reliability and significance have not been established. Presumably they demonstrate abnormal motion of the sacroiliac joint, but this does not necessarily indicate joint instability.

Various techniques may be used to isolate large low back ligaments and test their sensitivity to stretch. Stretching the ligament to reproduce the patient's pain is thought to be a sign of ligament pain and laxity. For persons with above-average manual skills, passive motion palpation of the pelvic ring joints and the vertebral segments is direct evidence of relative joint hyper- or hypomobility.^{31,35,36} This probably reveals more about the stability of the joints than does assessment of symmetry and active motion testing. These techniques, however, suffer from three major limitations: (1) the required skills are highly subjective, difficult, and time-consuming to acquire; (2) inter-examiner reliability is often too poor to be valuable in research; and (3) although changes in motion characteristics can be detected at the time of the examination, they may not represent the stability of the joint in everyday life. This is where the modulating effect of muscle activity on ligament tension comes into play (secondary hypermobility). Changes in posture and re-

cruitment of different supporting muscles may completely alter the patterns of joint stability.

There is another method for diagnosing low back instability that rests on the assumption first proposed by Hackett about chronic pain emanating from a ligament trigger point (always at the fibro-osseous junction) that results from ligament laxity. The best evidence to support this assumption is that repeat injections of a proliferating solution frequently do abolish the pain and the trigger points.

A number of difficulties arise, however, when the theory of ligament laxity is examined more carefully. The first is the question of whether pain at a ligament trigger point is always, usually, or only sometimes due to unresolved injury. In ligaments supporting central joints, this is particularly difficult to determine, but examination of peripheral joints may provide some theoretical answers. It should first be noted that joint instability by itself does not necessarily mean that lax ligaments supporting a joint produce pain even when stretched to a certain degree. A weakened anterior cruciate ligament can be demonstrated to be lax, without provoking pain when it is stretched. Clinical experience certainly suggests that lax ligaments may render the joint and its supporting ligaments more vulnerable to injury, but with everyday usage they are generally painless.

Not only are lax ligaments not necessarily painful, but also healthy ligaments may produce pain under certain circumstances. This may be easily demonstrated in a finger interphalangeal joint. If a proximal interphalangeal joint is gently abducted and tension put on the opposite collateral ligament for a minute or so, pain will gradually develop. Even when the tension is removed, some pain will remain and a repetition of the strain will induce the same pain more quickly than before. In other words, the ligament becomes sensitized by prolonged tension and readily produces pain.

In most peripheral joints, active muscle contraction does not produce abnormal tension on ligaments. In central axial joints, however, this almost certainly does occur. How often it occurs is open to conjecture, but abnormal

postural patterns resulting from injury, illness, degenerative processes, or emotional stress cannot help but place prolonged abnormal tension on central ligaments. This might be the cause of at least some of the ligamentous pain blamed on ligamentous laxity.

Another cause of ligament pain is heightened sympathetic nervous system tone. Ligaments in the lumbar and pelvic regions are richly supplied with sympathetic nervous system efferent fibers. Sympathetic efferent fibers may activate primary afferent fibers or potentiate inflammatory processes by releasing neuropeptides and catecholamines.³⁷ This may explain the sudden, lasting, response to low back prolotherapy in some patients. Almost certainly this is a neural therapy effect from the local anesthetic that is always mixed into proliferant solutions.

Ligament pain does not necessarily indicate ligament laxity, and ligament laxity does not necessarily cause pain. Evidence of pain coming from a ligament alone is, therefore, an inadequate indication for prolotherapy.

Overview of low back prolotherapy indications

Gedney's original paper in 1937 described prolotherapy of both the sacroiliac joint and the knee. His indication was "joint instability due to elongated ligament structure following trauma from whatever cause." He also used the term *hypermobile joint* and referred to "lacerated" ligaments, but did not explain how he made these assessments. In 1951, he described a technique of testing vertebral segments for passive motion (Dandy's sign).³⁸

Hackett described in great detail methods of detecting lax ligaments.³⁹⁻⁴³ History was very important, as was palpating potential ligament trigger points. He felt it important not just to find trigger points, but also to have the patient agree upon the particular point.

Perhaps Hackett's greatest contribution was the mapping of pain referral patterns from ligamentous trigger points. His textbook is rich in clinical pearls such as the observation that pain down the posterior leg is ligamentous in origin if it skips the back of the knee, and is true sciatica if it does not.

The detection of ligamentous trigger points was essential to Hackett's method. He believed that trigger points could develop only from ligamentous laxity. Naturally prolotherapy was the treatment. For many years he paid scant attention to muscles, believing that muscle spasm was mostly a secondary phenomenon. In acute low back pain, he recommended waiting for the muscle spasm to settle before examining for the underlying ligamentous laxity.

By 1955, Hackett realized, however, that laxity at the osseotendinous junction could also occur, and he began treating these trigger points with prolotherapy. From our current vantage point, it seems surprising that nowhere does he consider that pain at a fibroosseous junction could be caused by chronic excess tension. Perhaps this is because his knowledge of mechanics was limited, and there was no technique allowing him to assess joint mobility.

The British tradition of prolotherapy was probably best represented by Barbor who examined the causes of lumbar instability and divided them into four categories: (1) disc protrusion; (2) sacroiliac strain or subluxation; (3) ligamentous insufficiency; and (4) spondylolisthesis.⁷ Prolotherapy was the treatment or part of the treatment for all of these conditions with the exception of disc protrusion. The term *disc protrusion* as described by Cyriax is a syndrome characterized by the restriction of gross lumbar motion in one or more, but not all directions. No x-ray or other imaging evidence or neurologic deficit is required to confirm a diagnosis of disc protrusion. As a clinical term, disc protrusion is similar to, but not identical to the osteopathic term somatic dysfunction. Consequently, Barbor believed that prolotherapy should not be performed unless there is normal range of motion in the lumbar spine.

Barbor's diagnosis of ligamentous lesions depended almost entirely on symptomatology. Barbor recommended taking a meticulous history, noting the quality of pain, its location at the time of examination and at its onset, and the effect of posture and activity on the pain. Physical examination re-

quired normal range of motion and pain on stretching ligaments. He did not describe his techniques for stretching ligaments in his paper, but emphasized the importance of maintaining stretch for an adequate length of time. In some cases, he used Hackett's technique of injecting suspect ligaments with local anesthetic and observing the response.

Dorman extended the ideas of his predecessors in the British school by incorporating ideas of mechanics derived from osteopathy.⁴⁴ He concurred with Cyriax in citing intervertebral disc fragments as a source of pain in the lumbar spine; however, he believed the disc to be more vulnerable to injury when the intervertebral ligaments were insufficient. He was able to describe many situations implicating ligaments as a source of pain in the lumbar spine and in the pelvic ring.

Central to his thinking is the concept of *asymlocation* of the sacrum, a term coined to describe the static expression of disturbed sacroiliac mechanics. Sacral asymlocation not only puts strain on its supporting ligaments and muscles, but also distorts the mechanics of the lumbar spine. In his view, most disturbed mechanics have underlying ligamentous insufficiency as its basis.

Because this paradigm is ligament-centered, the possibility of painful ligaments (either lax or tense) from abnormal muscle balance is not considered. His treatment protocol (Ongley's method), named after his mentor Milne Ongley, combines manipulation and injections to relieve pain and relax muscles.¹²

Summary

The main purpose of prolotherapy is to strengthen and tighten ligaments around hypermobile joints. The challenge remains to: (1) determine when joint hypermobility is a cause of pain; (2) identify the affected joint or joints; and (3) decide whether ligament laxity is the cause of the hypermobility.

For most prolotherapy practitioners, ligament tenderness is synonymous with ligamentous laxity. It is assumed that when pain demonstrably emanates from other sources, for example, muscle trigger points, the underlying

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cause is ligament laxity; however, these assumptions rest on minimal evidence.

Deciding if ligament pain results from laxity, excessive tension, a combination of the two, or some other cause requires a reliable method of assessing primary joint hypermobility, that is, the impact of form and force. Such a method does not yet exist.

Until primary hypermobility can be diagnosed accurately, the indications for rational low back prolotherapy should include (1) a history of injury; (2) physical findings consistent with joint hypermobility; and (3) a failure to maintain joint stability after skilled treatment for all potential causes of secondary hypermobility.

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Chronic low back pain: functional leg length may be a critical factor

Dr A Breck McKay, On Doctors Orders, Carina, Brisbane, mckayabATbigpond.com.au

Introduction

The importance of leg length measurement has been controversial. The number of views expressed is similar to the number of health carers involved.¹ There are many ways of measuring leg length, the most accurate being a full length x-ray from the lumbar spine to the feet. However, few of the measurements take into account the functional leg length when the person is standing with equal weight on each foot. None considers the patient's ability to monitor whole body function and self-measure subjectively but accurately.² In the 1960s physiotherapists at the University of Queensland were taught to use differing thickness plates to assess differences, but this method has lost vogue with the apparent need for highly sophisticated measurement by modern medicine. There is acceptance that differences up to 12.5 mm in leg length can be accommodated. In modern podiatry, even with the wizardry of video recording, foot pressure patterns and computer-guided, manufactured orthotics, similar thickness plates are still used and tolerance of leg length differences persist.

Background

For 25 years, my Brisbane medical-physiotherapy practice had a set of variable thickness plates, but they were not used very often. Health professionals were taught that a difference under 12.5 mm is acceptable. Following my modification of Blomberg's para-sacroccygeal ligament injections in the form of the McMaropi-Wasubo protocol, I noted that a number of patients returned after relatively short periods (1-3 months) with exacerbations of their chronic low back pain, usually on one side, when previously it had been bilateral.

Looking for possible causes, I resurrected the leg length measuring plates. As a result of the findings, I now combine the slump test (which helps identify nerve irritation by disc protrusion

which may also affect leg length) and functional leg length tests during the initial consultation and assessment.

The use of such plates was addressed during discussion at the Melbourne 2002 AAMM conference. Other doctors were using similar methods, but there was no process formally considered or accepted. At the Adelaide 2004 conference Blomberg workshops, considerable surprise was expressed when I demonstrated that doctors could discriminate between 12.0 and 12.5 mm thicknesses, with subjects unaware of the actual differences until after the measurements were completed.

Method

Leg Length Plates (Fig. 1)

A series of combination wooden/Laminex plates, 100 mm x 300 mm, with thicknesses of 2 mm (two pieces of glued Laminex), 4 mm (mixed plywood/Laminex), 6 mm (plywood), 12.5 mm (plywood), 19 mm and 25 mm (standard pine timber) were used.

Measurement is performed by asking the patient to stand on the plates in bare feet, and balance with equal weight on each foot. Starting with the 12.5 mm plate under the left and then the right foot, the patient can determine which leg is shorter or if both are equal. All other measurements are then on the shorter leg.

In combination these plates allow 2 mm variations to be determined. When using the 12.5 mm plywood compared to the 2 mm + 4 mm + 6 mm plates, many patients are able to discriminate precisely between the two with the difference being only 0.5 mm.

Observations

My last 125 consecutive patients assessed for the McMaropi-Wasubo Protocol have been reviewed. Eighty-

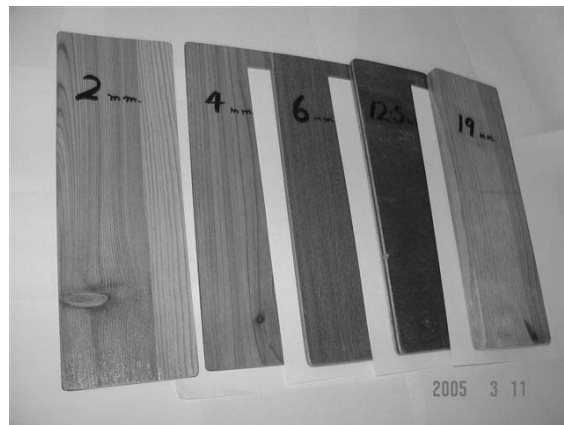


Figure 1. Leg length plates

eight (70.4%) had recorded leg length differences, always with the shorter leg being corrected to the longer leg length. Only 8 (6.4%) had positive slump tests.

The following results were obtained. Each measurement was made of the shorter leg.

Right Leg Shorter		Left Leg Shorter	
40 (45.5%)		48 (54.5%)	
Leg Length Differences			
2 mm	8	9.0%	
4 mm	18	20.5%	
6 mm	15	17.0%	
8 mm	17	19.0%	
10 mm	15	17.0%	
12 mm	6	6.8%	- These 10 (11.3%) patients could accurately discriminate 0.5 mm differences in leg length.
12.5 mm	4	4.5%	
Others	4	4.5%	-16.5 mm, 18.5 mm, 23.0 mm, 25.0 mm

Following the McMaropi-Wasubo Protocol, the functional leg length changed in four patients:

- A. from 8 mm to 12 mm in one (the only increase seen)
- B. from 10 mm to 6 mm in two, and
- C. from 8 mm to 2 mm in one.

These were all reproducible at follow up one week later.

Discussion

Following these clinical observations,

Chronic low back pain: functional leg length may be a critical factor

I now recommend that the functional leg length should be measured at the initial assessment of all CLBP patients and reviewed following any treatment intervention. It was due to the unexpected recurrence of CLBP in some treated patients that causation was considered. Leg length difference was the only obvious factor. The simple correction of the leg length, from full length shoe inserts, external changes (such as double thongs for 8 mm of difference), external full length footwear elevation, up to expensive computer generated orthotics, for amounts ranging from 2 mm to 12.5 mm, prevented recurrence of CLBP symptoms. This was functionally perceived and reported by the patients to remove effectively or prevent recurrence of their CLBP.

I am unaware of any equally accurate and simple method for measuring the functional leg length of patients.

From birth to five years of age, a child develops the perception of being upright and located in a three-dimensional world. Multiple directional movements and positions are perceived by visual, vestibular, sensory, and proprioceptive inputs, which create the pattern against which all future inputs are compared. This is achieved by the human body functioning as a single entity.²

All visual, proprioceptive, and other inputs are collated subconsciously, as the body moves in space and time, to produce the upright human position. Should any of these inputs change later in life, then the learnt posture control must compensate and adjust the body position. Such compensation occurs readily for up to 12.5 mm differences in younger persons. As the body/brain ages, the increased inputs and decreased capacity to cope results in less compensation by the brain-body interaction and chronic low back pain is one of the common clinical presentations.^{3,4}

Correction of leg length

The methods of correction have been many and varied. Less than 8 mm of difference can usually be corrected inside the footwear, while greater than 8 mm usually requires external adjustment for part of the correction.

Podiatrist Greg Dower (www.gregdower.com) has been most helpful and his results have been the best to date, with computer-cut orthotics and correction of gait and foot problems at the same time.

Patients without private health insurance have been able to use two thongs glued together (8 mm raise), full-length shopping centre boot maker shoe adjustments, and for lesser amounts (2-4 mm) some patients have achieved satisfaction with multiple simple shoe inserts. One old "bushy" uses one shoe on and one off and gets excellent results!

The ability of patients to discriminate 0.5 mm of difference was initially thought to be due to chance, but time has shown that it occurs very frequently and is reproducible. Once patients had their leg lengths corrected, even for 2-12.5 mm differences, they constantly reported better standing, walking, and mobility functions. Some patients with difficulty walking on uneven footpaths and surfaces reported better exercise tolerance and increased weight loss following correction of their leg length differences, and less CLBP. It is now essential to reconsider the invalid assertion that up to 12.5 mm difference in functional leg length is acceptable and should be accommodated and ignored.

The patients reported the worst results from podiatrists using plasticine or plaster moulds to create special inserts, or pharmacists selling partial length inserts, arch supports or similar specific purposes items. This may be due to under- or over-correction of other factors (for example, incorrect arch support, or partial leg length correction in part of the foot) that change the whole stance and gait, aggravating the CLBP. To further define such causes would require detailed video gait analysis and pressure measurements not generally available outside university and research centres.

Leg length correction is especially important with respect to the increasing incidence of back, hip, and knee surgery, where new afferents can overload the vestibulo-autonomic postural control mechanism, causing altered stance and gait and increasing the risk of falls and musculoskeletal pain.

Elderly patients already at increased risk of falls could conceivably be affected by smaller leg length differences. Perhaps it is now time to start measuring the subjective functional leg length in the elderly and follow-up interventions to reduce the risk of falls and pain.

This would be a worthy subject for study in multiple age groups, using the simple, functional leg length method, which may further help to dispel the 12.5 mm assertion. There are many available elderly communities in the blossoming retirement village settings who might benefit significantly from such study.

I have now designed a simple measuring unit comprising two bathroom scales and a variable height system for one leg, which allows a continuous range of 0.5 mm differences to be measured from +30 mm to zero to -30 mm. When the patient turns around, the longer leg is measured, correcting for any scale errors. The patient's weight must be equal on each scale at the time of measurement.

The construction is simple and cheap and may allow any doctor, anywhere, simply by weighing a patient twice, to measure functional leg length difference more accurately.

Perhaps the era of functional measurement of the human musculoskeletal system has arrived, casting out the old assertion that anything less than 12.5 mm be accommodated.

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Treatment of complex regional pain syndrome with peripheral nerve blocks: A case series of nine patients

Dr Giresh Kanji, Musculoskeletal Pain Physician, Southern Cross Specialist Centre, Wellington*

Summary

This case series describes nine patients suffering from complex regional pain syndrome (CRPS) who were treated with a combination of peripheral nerve blocks, myofascial injections of Marcaine into trigger points and medications for neuropathic pain such as carbamazepine and opiates.

Patients were treated from April 2002 to August 2003 in a private clinic outpatient setting in Invercargill and Wellington. Allodynia, hyperalgesia, and sleep disturbance subsided in eight of nine patients. The average Visual Analogue Scale (VAS) scores improved from 9/10 to 2/10. This case series indicates that peripheral nerve blocks in combination with appropriate medications can provide good relief of CRPS. Further studies to validate this treatment protocol are planned.

Introduction

Complex regional pain syndromes (CRPS) cause significant pain and suffering and are often poorly controlled by conventional therapies.¹ Clinical features of CRPS include allodynia, pressure hyperalgesia, vasomotor changes, sudomotor changes, temperature changes, trophic changes of the skin, motor impairment and osteoporosis.² CRPS can often progress to permanent impairment.

CRPS Type I (previously known as reflex sympathetic dystrophy) is present when no apparent nerve injury precedes pain, allodynia or pressure hyperalgesia. Pain, allodynia, or hyperalgesia present is out of proportion to the injury. CRPS Type II (previously known as causalgia) is present when a nerve injury precedes pain, allodynia, or pressure hyperalgesia. The pain is not necessarily limited to the distribution of the nerve. The International Association for the Study of Pain (IASP) diagnostic criteria also require

evidence of edema, changes in skin flow or abnormal sudomotor activity in the region of pain.² The absence of another diagnosis to account for the pain is also a diagnostic criterion.

Abnormal transmission of pain from the periphery to the brain is thought to be responsible for the exaggerated pain experience. Changes at one site in the pain transmission pathways are unlikely to explain all cases of this bizarre pain syndrome. Some cases may involve central nervous system plasticity in the spinal cord or brain to account for symptoms and some patients are likely to have changes in peripheral nerves² that account for symptoms. Both central and peripheral mechanisms may be operating. Four possible explanations for the mechanisms of pain have been offered in review articles² – the Ephapse model, the model of sympathetic afferents, neuromas, and ectopic signal generation in the dorsal horn.

The natural history of CRPS may be derived from a randomized prospective single-blinded study¹ comparing occupational therapy, physical therapy, and a control group in patients with RSD (complex regional pain syndrome type I). One hundred and thirty-five patients with symptoms of less than 12 months' duration were randomly allocated into the three groups. Impairment using the American Medical Association (AMA) Guides to the Evaluation of Permanent Impairment was rated at inclusion and after 12 months.

In all three groups the impairment was approximately 20% whole person impairment at inclusion and 20% whole person impairment at one year after treatment without a difference in each group. The natural history was of no improvement in impairment using the AMA Guides to the Evaluation of Impairment after 12 months in these 135 patients.

Literature on nerve blocks for treatment of CRPS

A Medline search for case series or trials using peripheral nerve blocks for treatment of CRPS from 1966 to 2003 revealed very few papers. The keywords "complex regional pain syndrome", "causalgia", "reflex sympathetic reflex dystrophy", and "local anesthetics" were used. Case reports of single patients were not included. Kingery³ in his review of controlled clinical trials for CRPS and neuropathic pain found no controlled trials using peripheral nerve blocks. Robinson⁴ reviewed treatment of CRPS Type I and also did not find any case series using peripheral nerve blocks. There were some case series using local anesthetics and opiates which are described below.

A case series⁵ using brachial plexus blockade with an infusion pump of Bupivacaine (0.5%, 3 ml/hour) with six patients reported that three patients responded favourably. The treatment interval between infusions varied from three to six months. The time between diagnoses and treatment was from two to seven months for five patients, with one patient having an interval of 25 months.

Azad et al⁶ reported a pilot study using morphine through an axillary brachial plexus catheter. Nine patients with upper limb CRPS (mostly under 12 months' duration) were given an average of 17 days of infusion. All patients were kept in hospital for treatment and received physiotherapy. Follow up at five months found a reduction in VAS at rest and during motion of 50%.

Linchitz and Raheb⁷ wrote a case series on nine patients treated with continuous subcutaneous lidocaine infusions for 4-8 weeks. Five patients completed the infusions and responded positively with reduced pain, allodynia, colour, temperature changes, and

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changes in hair and nail. VAS pain scores were reduced by approximately 50% in the five patients who completed the treatment. Another study,⁸ however, found little effect of intravenous lidocaine on allodynia and mechanical pain in a double blind, placebo-controlled trial with 16 patients.

A trial⁹ looking at the treatment effects of high dose topical capsaicin used regional nerve blocks prior to applying capsaicin due to the burning that develops on applying high dose capsaicin. They found patients receiving more than one treatment obtained additional relief with subsequent treatments. The treatment effects may have

been due to the peripheral nerve blocks rather than the large dose of capsaicin applied.

Methods

Seven patients were referred to a private clinic in Invercargill and two patients to a private clinic in Wellington. Each patient was diagnosed with complex regional pain syndrome after satisfying IASP diagnostic criteria. The first few patients were treated with myofascial trigger point injections of Marcaine but this proved to be painful and not well tolerated. Peripheral nerve blocks using Marcaine 5-6 ml proximal to the site of pain was better tolerated

and seemed to alleviate allodynia usually within a few treatments.

The demographics of the patients are outlined in Table 1. Five females and four males with an average age of 41 years (range 22-61) were treated. Two patients were referred with lower limb pain and seven with upper limb pain.

The nature of trauma, date of injury and the duration of pain at presentation are seen in Table 2. Four suffered major trauma and five suffered minor trauma. The duration of symptoms ranged from two months to 12 months (average six months). At the time of presentation, five were off work, one

Table 1. Patient demographics

Patient	sex	Age	Ethnicity	Occupation	Pain location
1	F	45	European	process worker	R arm
2	F	22	European	teacher	R knee
3	M	61	European	farmer	L elbow
4	F	54	European	home help	R thumb
5	F	33	European	office	L hand
6	F	20	European	mechanic	R elbow
7	M	51	European	retail	L knee
8	M	47	European	office	L elbow
9	M	36	European	haulage	L elbow

Table 2 Trauma site and mechanism of injury

Patient	Trauma		Pain location	Description of ppt cause	DOI	Pain Duration	Time off work
	Major	Minor					
1	x		Right arm	Forklift hit elbow	18/09/2002	4 months	off
2		x	R knee	Twisting injury	4/05/2002	12 months	nil
3	x		L elbow	Crutching machine	29/03/2003	2 months	off
4	x		R thumb	Injection	1/10/2001	15 months	half hour
5	x		L hand	Door 4x4	9/02/2003	2 months	off
6		x	R elbow	Forceful gripping	9/08/2002	2 months	off
7		x	L knee	Fall onto knee	2/01/2003	7 months	nil
8		x	L elbow	Nil remembered	Feb-03	6 months	nil
9		x	L elbow	Blunt trauma	28/05/2001	11 months	off
						average 6.7 months	

Table 3. Clinical features prior to treatment

Patient	Allodynia	Hyperalgesia	Erythema	Sweating	Heat/cold	Pins/needles	Numb/tingling	Sleep disturb
1	x	x	x	x		x	x	x
2	x	x	x		x	x	x	x
3	x	x	x			x		x
4	x	x	x					x
5	x	x	x			x	x	x
6	x	x	x	x		x	x	x
7	x	x	x					x
8	x	x	x					x
9	x	x	x	x				x

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was working half time, and three were working full time with limitations.

Table 3 outlines the clinical features of the nine patients prior to treatment. Table 4 provides a summary of the clinical features present. Allodynia, pressure hyperalgesia, erythema, and sleep disturbance were present in all patients.

Table 4. Summary of clinical features

Feature	Out of 9 Patients
Allodynia	9
Hyperalgesia	9
Erythema	9
Sweating	3
Heat/cold	1
Pins and needles	5
Numbness and tingling	4
Sleep disturbance	8

The medical personnel seen prior to their first appointment are outlined in Table 5. More than one cross is present if more than one healthcare provider was seen in that group. The investigations ordered prior to the first appointment are outlined in Table 6. Table 7 lists the medications that were tried prior to the first appointment.

The patients were all treated with peripheral nerve blocks as identified in Table 8. Five to six ml of Marcaine 0.5% was used when performing the nerve blocks. It soon became apparent that it was less painful for the patients when the nerve blocks were performed proximal to the site of neuropathic pain than in the regions affected by allodynia and hyperalgesia.

The nerves blocked in the upper limb were always the closest nerves proximal to the site of the CRPS that would anesthetize the site of the CRPS. Thus if the pain was at the hand, regional blocks would be performed at the elbow region. If the pain was at the elbow region, then the brachial plexus was blocked using the axillary approach.

The lower limb blocks were performed initially with tibial nerve blocks which although they do not supply the skin over the anterior knee still alleviated the pain and dysaesthesia over the knee area. Femoral nerve blocks were not required for the patients with knee CRPS although they would seem a

Table 5. Medical personnel seen prior to treatment

Patient	GP	ED	Physio	Ortho	Surgeon	Rheum	Occup Therapy
1	x		xxx	x			
2	x	x	xxx	x		x	
3	x	x	x	x			
4	x	x	x	x			
5	x	x	x	x			
6	x	x	x				
7	x		xx	x			
8	x		x				x
9	x		x	x			

Table 6. Investigations carried out on patients seen prior to treatment

Patient	MRI	CT scan	NCS	Bone scan
1			x	
2	x			
3				
4				x
5		x		
6			x	
7	x			
8				
9				

Table 7. Medications/injections tried by patients prior to treatment

Patient	NSAID	Panadol	Steroid injection
1	x	x	
2	x		
3			
4	x		x
5	x	x	
6	x	x	
7	x	x	
8	x		
9	x		x

more logical choice. Trigger point injections were tried on some patients after the case series began. Follow up was carried out using the questionnaire below. Seven patients were followed up by phone and two at consultation. Three patients are still continuing with treatment every 3-4 weeks.

Results

The time since treatment ended is outlined in Table 9. Three patients have not been discharged from the clinic and are being followed three weekly to monthly. For those still undergoing treatment, 0 months is taken as the time since treatment.

The clinical features of the patients are seen in Table 10, with a summary of the clinical features before and after

treatment outlined in Table 11. The visual analogue scores for pain before and after treatment are outlined in Table 12.

Patients responded well to the treatment, with over 80% reduction in the average VAS scores for pain. Allodynia and sleep disturbance resolved in 8 out of 9 patients. Pressure hyperalgesia subsided in seven of nine patients. The reduction in symptoms also coincided with the decrease in disability among patients. Three patients were working full time at the start of the series, with seven patients working full time at the follow-up period. Of those working full time, their tasks at the workplace became easier to perform.

Conclusions

Patients with CRPS Type I present

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Table 8. Treatment received after referral

Patient	Tegretol	Paradex	Tramadol	DHC	First visit	Second visit	Third visit	Fourth visit
1	x		x			TP	TP	NB
2	x		x		NB	NB TP	NB TP	Nil
3			x		NB			
4	x	x			NB/TP	NB/TP	Nil	
5	x		x	x	NB	NB TP	NB TP	
6					NB TP	NB TP	Nil	
7	x		x		NB/TP	NB/TP	Nil	
8	x	x			NB	NB	NB	NB/Steroid
9	x		x			NB	NB	

NB = Nerve Block, TP = Trigger point injections, Steroid = Kenacort/Marcaine injection

Phone questionnaire follow-up

- Use of pain medication: Are you taking any medications for pain at the site of the CRPS?
- Daily pain experienced: What is the best and worst pain experienced during the day out of 10?
- Disturbed sleep: Is sleep disturbed by pain?
- Allodynia: Is it painful to lightly touch the area?
- Pressure hyperalgesia: Is it painful when pressure is applied to the area?
- Limitations: What activities are you limited in at present from the site of CRPS?
- Are you working your full hours?
- Are redness/mottling still present?

Table 9. Time from last appointment to follow up

Patient	Follow-up	Time since treatment (months)
1	29/08/2003	0
2	20/08/2003	0
3	26/08/2003	3
4	27/08/2003	5
5	27/08/2003	2
6	26/08/2003	9
7	28/08/2003	0
8	26/08/2003	0
9	26/08/2003	8

Table 11. Summary of clinical features at follow up

Feature	Pre-treat	Follow-up
Allodynia	9	1
Hyperalgesia	9	2
Erythema	9	3
Sweating	3	1
Heat/Cold	1	1
Pins/Needles	5	2
Numb/Tingling	4	2
Sleep Disturbance	9	1

Table 10. Clinical features at follow up

Patient	Allodynia	Hyperalgesia	Erythema	Sweating	Heat/cold	Pins/needles	Numb/ting
1	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2	No	No	No	No	No	No	No
3	No	No	No	No	No	No	No
4	No	No	No	No	No	No	No
5	No	Yes	Yes	No	No	?	?
6	No	No	No	No	No	No	No
7	No	No	Yes	No	No	No	No
8	No	No	No	No	No	No	No
9	No	No	No	No	No	No	No

Table 12. VAS pain scores at first consultation and at follow up

Patient	Pain at start	Pain at end	Follow-up (months)
1	9	4	0
2	10	0	0
3	7	2	3
4	9	2	5
5	9	5	2
6	8	0	9
7	10	7	0
8	10	0	0
9	10	0	8
Total	82	20	
Average	9	2	

Treatment of complex regional pain syndrome with peripheral nerve blocks

Follow up March 2005

Patient	Follow up	Pain at start	Pain at end	Time since treatment (months)
1	03/2005	9	6	18
2	03/2005	10	5	18
3	03/2005	7	0	21
4	03/2005	9	2	23
5	03/2005	9	1	25
6	03/2005	8	0	34
7	04/2005	10	0	18
8	03/2005	10	0	18
9	03/2005	10	0	26
Total		82	14	
Average		9	1.5	

Patient	Sleep disturb	Allodynia	Erythema	Medications	Sought treatmt	Limitations	Working
1	yes	yes	yes	Tried gabapentin, epilim	yes	using hand	no
2	no	no	no	nil	nil	walking	full time
3	no	no	no			nil	full time
4	no	no	no	nil	nil	nil	full time
5	no	no	no	nil	nil	nil	full time
6	no	no	no	nil	nil	nil	full time
7	no	no	no	nil	nil	nil	full time
8	no	no	no	nil	nil	nil	full time
9	no	no	no	nil	nil	nil	unemployed

with neuropathic pain that is exaggerated. The heightened response is likely to reflect changes in the peripheral and central nervous system pain pathways. In six of the nine cases, once the neuropathic pain, allodynia, and hyperalgesia settled, there was underlying somatic pain present. A deep, dull ache remained after treatment. Three patients fulfilled the diagnostic criteria for lateral epicondylitis and were treated with an injection of steroid and Marcaine into the lateral epicondyle with marked relief of somatic pain. One patient responded to a steroid injection into the knee with relief of somatic pain.

Two patients with CRPS of the knee continued to experience a deep, dull ache after the neuropathic pain, allodynia, and hyperalgesia settled. Both had decreased weight bearing because of the severe pain experienced. One of these patients experienced morning stiffness for 10 minutes. With the lack of weight bearing for extended periods, peri-articular osteoporosis and cartilage deterioration are likely to develop. This process may contribute to ongoing somatic pain after neuropathic pain subsides.

Patients with CRPS are often not diagnosed. Only two patients of the

nine presented with a diagnosis of CRPS. Inappropriate medications were also prescribed at presentation. NSAIDs (nonsteroidal anti-inflammatory drugs) have been shown to be ineffective in neuropathic pain⁴ but eight of nine patients presented taking NSAIDs. One patient of nine presented taking membrane-stabilizing medication.

The pain is out of proportion to the injury in most of the cases of CRPS. The severe pain and often crippling disability is often difficult to comprehend for healthcare providers. Most patients were unable to tolerate having the site of pain within their sheets when sleeping and kept the site of pain over the edge of the bed. Many of the cases of upper limb CRPS were limited in brushing teeth, writing, and using a hairbrush. One patient with lower limb CRPS was walking with two crutches. The disabilities subsided when the pain subsided.

The treatment effects are several:

1. Patients receive an explanation of their condition which gives them a logical reason for the exaggerated pain response and reduces patient anxiety.
2. The treatment is based on a logical and believable premise that is easily

understood by most people.

3. The analgesic medication and neuropathic pain medication improves patients' sleep and pain.
4. The nerve block eliminates all pain for a certain period of time, giving acute relief.

The results of this case series show nerve blocks are promising for the treatment of CRPS. Although the neuropathic pain has responded in eight of the nine cases, there has been residual somatic pain present. Disability has also declined significantly with treatment and four of six patients who were off work returned to work after treatment.

This case series is a preliminary report and has not been subjected to scientific scrutiny. A follow-up case series with questionnaires and/or interviews administered by a person other than the treatment provider before and after treatment may help validate this treatment protocol. This would address the question of bias in reporting favourable results, that is, the patient telling the doctor what the doctor wants to hear. A multicentre case series/controlled trial may be appropriate due to the limited number of cases seen in our population.

Peripheral nerve blocks with bupivacaine and appropriate medication for neuropathic pain seems to be a promising treatment for the neuropathic symptoms of CRPS type I in patients with symptoms for less than 12 months. Seven of nine patients had markedly reduced pain with allodynia, hyperalgesia, sleep disturbance, and disability reduction. The VAS scores also decreased from an average of nine to 1.5. Further studies need to be performed to validate the treatment of this exquisitely painful and debilitating condition.

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*Dr Gireesh Kanji, dr.kanji@xtra.co.nz
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Appendix: Patient advice regarding condition and treatment

The advice given to patients regarding this treatment is based loosely on scientific facts to give the patients a framework upon which to conceptualize what is happening to them. Often patients are reassured that there is an explanation for their exaggerated symptoms. Other explanations can be used but I have found this one easily understood by most patients.

Complex regional pain syndrome (CRPS) Type I is an unusual pain pattern that develops when the pain and sensitivity experienced is out of proportion to the injury sustained. Often a minor sprain may precede the development of CRPS.

The nerves transmitting pain to the brain have exaggerated the message.

In a normal nerve, sodium enters the nerve and potassium leaves the nerve in channels present along the length of the nerve. This creates a message along the nerve that is transmitted to the central nervous system. In the normal nerve channel one potassium leaves the nerve and one sodium enters the nerve. In CRPS there are 10 times the normal amounts of sodium entering the nerve and 10 times the number of

potassium leaving the channels. This creates an increased pain experience and increases the sensitivity of the skin.

The treatment of nerve blocks aims to make the area pain free for a certain period of time giving some relief. The nerve blocks are performed central to the area of sensitivity at a convenient point. The purpose of the nerve blocks is to block all the channels transmitting the nerve messages so that when they wake up they will be back to normal and transmit only one sodium and one potassium through the channel. This is similar to turning off a computer when it crashes. Switching the computer off and then back on has the effect of resetting the channels and returning to normal. It may take a few attempts to completely change the channels and the nerve block may be repeated at 2-3-weekly intervals for a maximum of three nerve blocks depending on response.

Medication for neuropathic pain is also used as this may help both sleep and also help dampen the nerve channels. Medication is usually taken for a short period of a few months or less and then can be withdrawn.

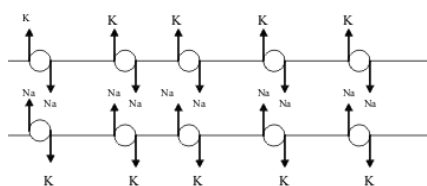


Fig. 1. Normal nerve conduction. A nerve fibre is shown with the sodium (Na) and potassium (K) channels.

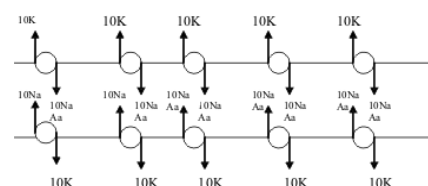


Fig. 2. Nerve conduction in CRPS. The numbers of sodium (Na) and potassium (K) ions entering and leaving the nerve are increased, resulting in an increased pain response.

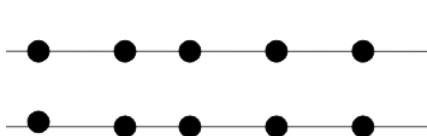


Fig. 3. Effects of a nerve block with local anesthetic. All channels are blocked and there is no conduction of signals.

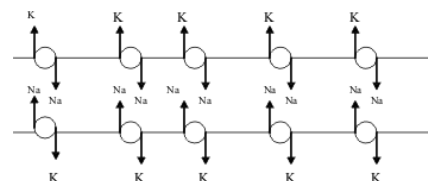


Fig. 4. Nerve conduction returns to normal, resulting in decreased pain signals.

Spinal fracture in epileptic fits

Dr Graham Corbett, Branch Medical Adviser, Accident Compensation Corporation, NZ

The question of the mechanism by which spinal fractures might come about during an epileptic fit arose recently, when a 25-year-old linesman presented to an emergency department with the story of having woken up to find himself in severe pain located in the back of his chest. Four years before he had been diagnosed with type 1 diabetes and this was well controlled on insulin and diet. He was unmarried and lived and slept alone. Neurological examination did not reveal any abnormalities but plain x-rays revealed high thoracic vertebral body fractures. CT and MRI scans showed blow-out fractures of T3-6 vertebral bodies.

X-rays

Should one perform spinal x-rays on a case of acute back pain where there is no history of trauma? This is thoracic back pain, but the New Zealand Acute Low Back Pain Guide¹ influences a lot of our thinking about back pain and the relevance of doing x-rays in the acute situation. A review of the Red Flags for acute back pain gives us this list:

- Features of Cauda Equina Syndrome:
 1. Severe pain at night
 2. Weight loss
 3. Fecal incontinence
 4. Widespread neurological symptoms
 5. Signs in the lower limbs
 6. Gait abnormality
 7. Saddle area numbness
 8. Lax anal sphincter
- Significant trauma
- Weight loss
- History of cancer
- Fever
- Intravenous drug use
- Patient over 50 years
- Severe, unremitting night pain
- Pain that gets worse on lying down

In the absence of these red flags, we are advised not to x-ray in the first instance. Furthermore, the utility of routine x-rays of the spine in making a diagnosis in cases of acute back pain is low. Scavone² records that in a survey of 871 films taken without the

presence of red flags, though of little use in making a diagnosis, only one in four were actually reported as normal and so could be quite misleading.

Additionally, note needs to be taken of the danger of ionizing radiation from such investigations. The annual recommended safe dose limit of radiation is 1 mSv, but a lumbar spine series will expose a patient to 2.4 mSv on top of the normal background radiation of 1-2 mSv. The consequent risk in a population the size of Australia for the current rate of lumbar x-rays is of an extra 62 fatal malignancies per year.³

CT and MRI are less readily available, more expensive, and therefore not so likely to be vicariously employed as screening tools; but CT scans do expose the patient to even higher (3.6 mSv) levels of radiation. MRI does not involve the use of ionizing radiation but the results of both these investigations need to be interpreted with caution.

Wiesel⁴ showed an incidence of herniated nucleus pulposus in those aged less than 40 years of 19.5% in a group of asymptomatic subjects who had CT scans. In those aged over 40, herniated nucleus pulposus was found in 26.9%, degenerative joint disease in 10.4%, and spinal stenosis in 3.4%. All in asymptomatic people!

In another study involving Wiesel,⁵ MRI in asymptomatic subjects demonstrated an incidence of 24% herniated nucleus pulposus and 4% spinal stenosis across all age groups. In the 20-39-year-old group that this young man fits into, there was 20% herniated nucleus pulposus, 54% disc bulge, and 34% disc degeneration.

Nevertheless, CT and especially three-dimensionally reconstructed CT is invaluable for its excellent portrayal of bone for assessing the stability of spinal fractures. MRI scan, whilst not as useful in visualizing bone, portrays soft tissue very well and is of particular use where the neurological picture does not quite fit with the findings on CT scan.⁶

Notwithstanding the above, in this particular case, the rarity of the body site for spinal pain, its severity, the youth of the patient, and the onset during sleep, a limited trauma series of

x-rays was performed. Positive findings of spinal fracture caused this to be followed up with CT and MRI scans. The orthopedic consultant expressed the opinion that these fractures were more likely than not the result of an epileptic seizure, cause unknown. An application was then made to the Accident Compensation Corporation (ACC) for insurance cover for the vertebral fractures.

Accident Compensation Corporation (ACC) cover for injuries

The issue of whether or not one receives ACC cover for an illness is an important one in New Zealand. For those with ACC cover, there is an extensive array of entitlements, including compensation for loss of wages, lump sum payments for impairment, and a wide range of private healthcare options both in hospital and at home. For those without ACC cover, there is only the sickness benefit and the public health care system.

At a simplistic level, most fractures attract cover from ACC and in the public mind all fractures are associated with a right to ACC cover. Denial of cover to a fracture patient is therefore almost certain to provoke a legal challenge. However, ACC cover is provided only for injuries caused by accident.

One definition of an accidental injury is damage caused by a force external to the body. Damage to the body caused by forces intrinsic to the body does not generally qualify for cover. Therefore, if this individual's fractures were caused simply by the internal muscle contractions resulting from an epileptic fit, they would not receive cover. An exception to this might occur if the epileptic fit itself had been caused accidentally such as by an accidentally administered electrical discharge or an accidentally ingested chemical.

Causes of seizures

Chadwick⁷ gives a long list of possible causes of seizures. Disorders of electrolyte and fluid metabolism may well have played a part here as both hyper- and hypo-natremia may occur in diabetes, as can hypoglycemia.

Capillary blood sugar in the emergency room would give a clue here, though the morning blood sugar may subsequently have been driven back up again by the effect of glucagon. There is apparently no evidence to support a diagnosis of hypo- or hypercalemia, hypo-magnesemia, or hypophosphatemia. Thyroid disease, porphyria, liver disease, and renal failure are other possible causes that seem unlikely but not yet ruled out.

A large number of drugs has been implicated in seizure activity. This can be either in the nature of promoting seizures by their introduction, in overdose, or in their withdrawal. Whilst many of these are infrequently seen outside the hospital environment, such as anesthetic agents and radiographic contrast media, others more likely to appear in the community are anti-dysrhythmic agents, anticonvulsants, antidepressants, antipsychotics, and drugs of abuse such as amphetamines, codeine, pethidine, alcohol, etc. Less likely to spring to mind are antibiotics such as a variety of penicillins and quinalones, isoniazid, and naladixic acid. Aminophylline and ephedrine can also be implicated, though they are less frequently encountered.

If there were evidence that the claimant had previously been the victim of cerebral trauma, either from birth defect, previous hypoxia, head injury, or previous brain surgery, certain of these might impart cover as previously registered accidental injuries. Cerebrovascular disease such as cerebral tumour and infection are other possibilities. Aboukasm's⁸ comments that onset after age 20 is due largely to acquired focal causes and that nocturnal epileptic seizure is particularly common in those who have partial epilepsy bears note at this stage. The cause of the seizure in this particular case is still under investigation, but since a space occupying cranial mass has been detected, cover from accidental causes of fitting is less likely.

Possible mechanism of vertebral fracture in epilepsy

Why then should we believe that vertebral fractures found after an epileptic fit are a result of the muscle

contractions generating forces solely within the body? No one was there when this young man was hurt. If he did have a seizure, it might be argued that he suffered his injuries in a fall from the bed or in crashing into a piece of furniture or the wall. If he had then climbed back into bed during his post-ictal state, he might now be quite unaware of the episode. In so doing, it could be argued that he was in fact the victim of an accidental injury due to force external to the body and so entitled to cover from ACC. With so much at stake for them financially, claimants who are declined ACC cover require a full and convincing explanation of the reasons for that. It is not uncommon to have to defend such a cover decision from legal challenge.

In discussing the forces involved in lifting, Bogduk recounts that an average vertebral body can endure 10,000 N before its endplates fracture, but that the lower end of the normal range is about 4,000 N. By contrast, the maximum tension in the back muscles is about 4,000 N and this is what is exerted in the effort to lift a 30 kg weight from a stooped position.⁹ The length of the lever arm of the posterior spinal muscles has been variously estimated,¹⁰ and since it is so short, a change of even 1 cm makes a large difference in the calculated extensor moment. Nevertheless, whatever length is selected from those offered, a major effect is that the muscle contraction is being exerted on such a short lever arm that most of this force is transmitted as a compressive force along the axis of the spine.

Clearly there is some overlap in the forces quoted here, such that the available power of muscular contraction is of an order that might crush a vertebral body in some individuals; but strangely, it is also being used at this power level in an everyday way without causing vertebral fractures. Even more strangely, our era is one in which sporting activity and physical fitness is very popular. Some authors estimate that about 4% of the population is involved in weightlifting as a sport.¹¹ It seems likely that there is some other protective factor involved, because our waiting rooms and emergency departments are not experiencing an epi-

demic of spinal fractures from all this competitive sporting endeavour.

There are intrinsic difficulties in investigating spinal injuries from epilepsy. The spine is a deep-seated structure and unless pain is complained of, or there is an obvious visible deformity such as a new kyphosis, spinal injury may go undetected.¹² Limb injuries have been more amenable to investigation by virtue of being more easily accessible and some studies have suggested that external forces cannot be ignored. One study of 276 patients¹³ was able to exclude external trauma in only five (2%) of the patients. How then, in the face of these findings for limb injuries, can we be so certain that these spinal fractures are due simply to crushing by intrinsic forces?

History of spinal fractures in epilepsy

The first report of a spinal fracture resulting from a seizure appears to have come from Lehndorf in 1907, who described a boy of 12 with a kyphosis that came on after a fit caused by tetanus. Finding no evidence of osteomalacia, he suggested that the strong muscular contractions of the fit had damaged the vertebrae. This view acquired some supporters as well as some detractors, each publishing small surveys to support their views. A review by Erlacher in 1921 compiled all such similar reported cases and supported Lehndorf's view.¹²

The addition of electroconvulsive shock therapy (ECT) to the psychiatric therapeutic armamentarium, and its use without anesthesia and neuromuscular blocking agents, permitted the collection of large series of vertebral fractures in circumstances where the fact of observation of the seizures permitted the assumption that they could have been caused only by the intrinsic force of muscular contractions.

The site of the fractures was up to that time thought to correspond to that of traumatic fractures, that is, at the thoracolumbar junction.⁶ However, a study of 2,200 patients with 37,000 induced convulsions by Kelly¹⁴ showed that in induced convulsions at least, the most common sites were T4, T5, T6, and T7. Fractures below T7 were un-

Spinal fracture in epileptic fits

common in this series.

The study by Vasconcelos¹² was the first involving large numbers of patients known to have epileptic grand mal fits in which a search was made for vertebral fractures. One thousand four hundred and eighty-seven patients were investigated from their history and 70 were x-rayed. Fractures were found in 15% of patients; more frequently in those whose epilepsy started in adult life and in those whose seizures occurred in their sleep, or those who had experienced a grand mal status. The site of fracture was again found to be most commonly in the upper thoracic spine and next commonly in the mid thoracic spine. Males were more commonly affected by 3:1 than females.

The percentage (15%) of epileptics with vertebral fractures found by Vasconcelos has been confirmed in a further x-ray survey.¹⁵ Pedersen's survey involved epileptic patients who had no history of back pain, showing this to be a frequently missed condition.

A French study in 1993¹⁶ discussed eight subjects with spinal fractures. The thoracic spine was most commonly involved, three cases involving the upper thoracic spine and three cases the intermediate thoracic spine. Osteoporosis was not apparent from their patients' x-rays, but of their cases, four were alcoholic, one was on anti-epileptic drugs, and one was on corticosteroids. All are known to carry the possibility of bony demineralization.¹⁷⁻¹⁹ In six out of eight cases, this was a first fit and males predominated 6:2. The youngest of Dubost's patients was aged 41 years, which is rather old compared with other studies.

Aboukasm⁸ described a 21-year-old with compression fractures of T7, T8, and T9 and a 36-year-old with compression fractures of T5 and T6, both of whom were woken from their sleep by pain. Subsequent fits in these individuals were preceded by an aura and a diagnosis of partial epilepsy with secondary generalization was made. They noted that when there is onset of epilepsy after age 20 it is largely due to acquired focal causes and that nocturnal epileptic seizure is particularly common in those with partial epilepsy. Two other papers also make note of the incidence of focal seizure activity and

adult onset with spinal fractures in young adults.^{20, 21}

Against the tendency to thoracic fracture is a paper²² describing burst fractures at L1 and L2 in a schizophrenic who experienced a witnessed tonic clonic seizure. They do, however, note that they were unable to find any other instances of lumbar fractures incurred in an epileptic fit in the literature.

Thus, a picture emerges of the typical victim of vertebral fracture from an epileptic fit. Young adults are most commonly affected, with males favoured 3:1 over females, and the victims are most often having their first seizure.

Vertebral fractures are more common in those with strong muscles (that is, males), which intuitively makes sense. They are more common in those with denser bones to start with and additionally, before age and antiepileptic drugs can weaken them. Nor are the fracture sites located where they are commonly found in trauma cases, that is, either above the thoracic spine, or below at the thoracolumbar hinge.^{23, 24} Instead they are located in the upper and middle thoracic spine, suggesting a much different mechanism of injury.

Biomechanics of the fractures

Perhaps there is something wrong with the model which compares an epileptic seizure with weightlifting, simply in terms of the nature of the participants.

Musculoskeletal tissue is capable of adaptation to the loads placed upon it, though limited in its speed of response by the rate of supply of nutrients in the blood supply. Muscle having a very good blood supply responds quickest. It replaces its proteins with a half life of 7-15 days.²⁵ An increase in muscle mass is immediately obvious in weightlifters, but bone also has a fairly good supply of nutrients from its abundant blood supply and, whilst not as quick in adapting as muscle, it is nonetheless reasonably speedy. Turkey bones, for example, can increase their mineral content by 40% in six weeks when exposed to stimulatory loading.²⁶

Elite weightlifters have been shown to have an exceptionally high mineral

content in their vertebrae,²⁷ as have tennis players in their dominant forearms.²⁸ There is a close relationship between bone mineral content and bone strength.²⁹

However, whilst this hypertrophic effect must have some effect on the resistance of weightlifters to vertebral injury, let us not forget that the injured person of this story is more than just any young male adult, but a linesman. This is an occupation which is closely associated with considerable daily exertion and physical fitness.

Finding ourselves steered towards investigating those with stronger muscles, one might expect to see more vertebral fractures in adolescent males at a period when muscle development was more pronounced but skeletal growth was incomplete. Trying to separate spinal fractures due to intrinsic muscular effort rather than the external forces involved in impact during teenage sporting activities in the literature is difficult. I have found only one sports-related spinal fracture where impact could clearly be ruled out - a lumbar ring apophysial fracture in an adolescent weightlifter.³⁰ However, I note that Schmorl's nodes are reputed to be the result of end plate fracture allowing minor intrusion of nucleus pulposus material into the vertebral body in young athletes.³¹ They are micro fractures of vertebrae, limited in their extent by the action of protective reflexes, for example, Golgi tendon organs.

Is there something intrinsic to a seizure which allows the application of more muscle power? Perhaps the absence of inhibitory reflexes such as those from the Golgi tendon organs to prevent damage by limiting power is a factor. Or, is it because of a series of violent, opposite, and unopposed motions? Whilst the idea of violent motion back and forth is attractive at first, in practice as Youssef²² has described, seizures most commonly bend the trunk forwards in a series of shuddering spasms.

The amount of muscle power applied to voluntary motion is influenced in many ways other than the Golgi tendon organ reflex that are not applicable to epileptic contractions. Coupled motion is involved in normal spinal motion, so that the spine bends, sways, and

twists to achieve the minimum resistance to a primary motion. Not indulging in these coupled motions in a seizure could conceivably increase the forces coming to bear on the spine, thus tending to cause damage in a seizure that is not likely to happen in a conscious person.

Input from other parts of the conscious body may also affect how the power of the muscles is applied. Interestingly, this can happen below the conscious level even when the conscious mind is aware of the information. In a study in Toronto³² using a 6 kg industrial tote bin divided into nine compartments inside and loaded with a 7 kg and an 11 kg mass randomly placed inside. The force on the bin's handles and electromyographic studies of the erector spinae, latissimus dorsi, and external oblique muscles were recorded. No differences in the patterns were found between when the subjects knew where the loads were placed and when they didn't. Activity appeared to be influenced mainly by proprioceptive information from sensations of where the load was positioned coming from the wrists.

In actual measurements on a trained weightlifter, it appears that the total force at the disposal of the muscles is not actually employed. Graceovetsky³³ showed that the moments employed by a trained weightlifter do not exceed 67% of the ultimate strength of the tissues.

Additionally, the muscle contraction in a seizure is potentially of all muscle groups, not just the para-vertebral musculature. As described by Youssef²² in their discussion of a lumbar fracture, the contraction bends the abdomen forwards, imposing axial loading on the anterior and middle columns of Denis,⁶ suggesting that the abdominal muscles are exerting a greater moment on the spine by virtue of either a longer lever arm or greater force or both.

However, the lumbar fracture reported by Youssef is the exception rather than the rule. In the case of the majority of fractures, which are in the mid and upper thoracic spine, there is a "fourth column", the sternum.³⁴ Berg contends that the sternum confers stability to the thoracic spine protecting it

from compressive fracture in trauma (except where the sternum-rib complex is broken). This arrangement would also tend to protect the mid and upper thoracic spine from the effects of the abdominal musculature in terms of flexing the upper and mid thoracic spine.

In the case of lifting, Daggfeldt¹⁰ in their paper and the ensuing letter to the editor and response in 2004, report that the muscles likely to flex the spine such as rectus abdominis and external oblique in fact contribute little in the way of contraction as judged by EMG measurements. Internal oblique and transverses on the other hand are used very much more but are not oriented (in a flexed torso is the important point for internal oblique) so as to cause forward flexion to any great degree.

The mid and upper thoracic spine has little in the way of directly attached anterior musculature to impart flexing and compressive forces, a few fibres across the uppermost vertebrae from the lower end of longus cervicis only. However, there is a considerable quantity of respiratory muscle, the intercostals, which when contracted would tend to cause axial compression of the thoracic spinal unit.

In the case of an epileptic seizure and an open glottis, the effect of these muscular contractions would be translated directly into an axial compressive force. With a closed glottis, however, the mechanics would be different as I attempt to show below, due to the raised intra-thoracic pressure.

Bogduk⁹ has considered raised intra-abdominal pressure in relation to lifting, with regard to its potential for pushing up on the underside of the diaphragm and helping to extend the spine. He commented that the hoop tension required to make a significant contribution exceeded the potential of the abdominal muscles and would incidentally occlude the abdominal aorta. He also considered that the flexion effect of the muscles would negate any extension effect. Daggfeldt's view¹⁰ is contrary. These viewpoints regarding the ability of the abdominal muscles to impart an extensor force to the lumbar spine is, however, quite a different issue from thoracic fractures in epileptic fits.

Lifting involves taking a deep breath, closing the glottis and performing a Valsalva manoeuvre.³⁵ It is also a matter of common experience that the heavier the lift, the more likely one is to go purple in the face. In other words, the force of the Valsalva appears to have a direct relationship to the weight of the lift.

Bogduk and others have suggested that imbalance of a load is a threat to spinal stability and that the abdominal muscles are important in controlling such rotatory effects.^{36, 35} If maintaining rotary stability of the spine requires such great force in case of imbalance of the load occurring when lifting, then one might also expect to see it employed in other situations where there are even more severely unbalanced forces on the spine. As a test, one may perform a "push-up" from the floor and then balance on one arm and the contra-lateral foot. Performed either face up or face down, and so applying either flexion or extension forces at the same time as a large rotation force, the ability to converse and hence to demonstrate the absence of a Valsalva is easily retained.

It might be argued that this Valsalva could act protectively on the vertebral bodies. The vertebral bodies' venous drainage is connected without valves to the vena cavae, thus permitting a back flow of blood into them on performing a Valsalva. This hydraulic pressure might then be effective in strengthening the vertebral bodies against a compressive force. Calculating this, we note that it is easily possible to blow into a desk sphygmomanometer to a pressure of 80 mm Hg. Studies have shown that pressures of up to 150 mm Hg can be attained.³¹ Converting this to SI units:

Atmospheric pressure is approximately 760 mm Hg.

Therefore, 150 mm Hg is approximately 0.2 atmospheres

1 atmosphere = 101.325 kPa

Therefore, during a maximal Valsalva manoeuvre, the pressure within the vertebral bodies could be raised by about 20 kPa.

The pressure within the lumbar discs (which are more accessible) can be measured by inserting a needle. Stand-

Spinal fracture in epileptic fits

ing, the disc pressure is 70 kPa, but holding a 5 kg weight raises the pressure to 700 kPa.³⁶ Clearly, the pressure change in the vertebrae from performing a Valsalva manoeuvre alone is of little relevance in relation to the pressure changes involved in lifting even small weights.

However, within the thorax, we have a closed oval cylindroid (figure at right) resisting the axial compression not only of itself, but also the thoracic spine, to which it is intimately attached as pointed out by Berg.³⁴ The force that this shape exerts to resist axial compression can be calculated from the internal pressure and the area at right angles to the axial compression, that is, the cross-sectional area of the chest. In considering the case of an imaginary adult male weightlifter, with a chest circumference of 110 cm and by making some assumptions about his chest shape, such as that its transverse diameter is double its antero-posterior diameter, we might calculate the forces involved.

Using the formula for an ellipse:
Circumference = Mean diameter $\times \pi$

The mean diameter for this ellipse = $110 \div 3.14 = 35.03$, rounding to 35 cm

Using two formulae implicit from the assumptions made regarding the chest shape:

1. Mean diameter = $(Da + Db) \div 2$
And:

2. $Da = 2 \times Db$

Where: Da = external transverse diameter of chest and
 Db = external antero-posterior diameter of chest

Then, $2 \times 35 = 3 \times Db$.

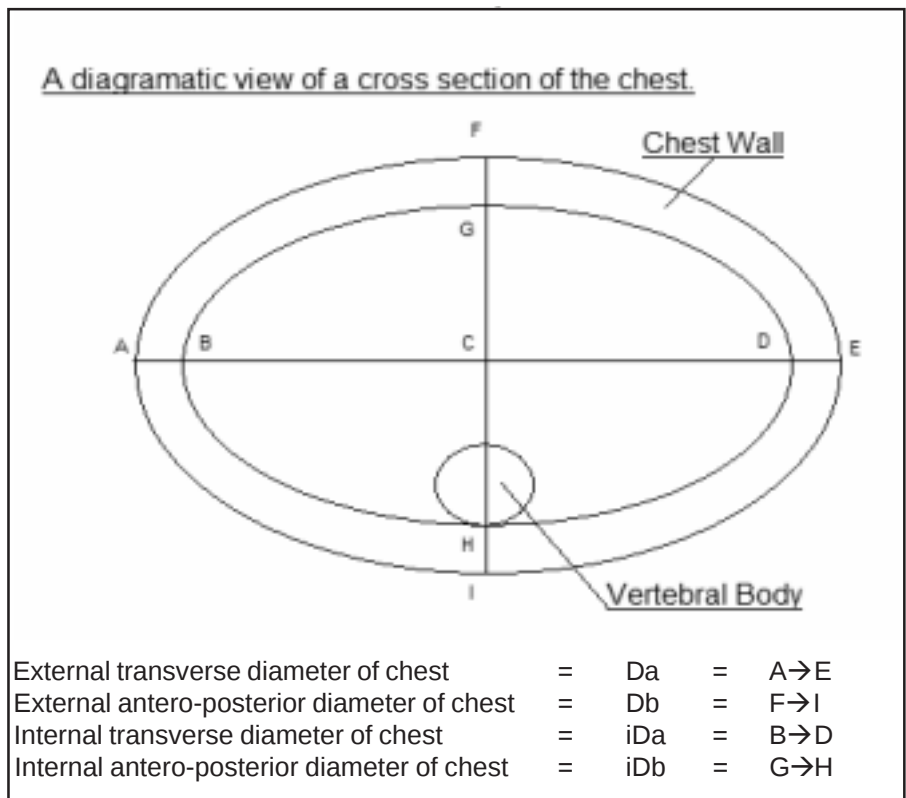
Therefore $Db = 70 \div 3 = 23.33$ cm

Therefore $Da = 46.66$ cm

& $Db = 23.33$ cm

Presuming that the thoracic wall plays little part itself in resisting axial compression of the chest and thoracic spine, it is necessary to subtract the thickness of the thoracic wall to estimate the size of the oval cylinder that is involved.

Though the vertebral bodies project into the oval of the thoracic cavity, because of the valve-less nature of their veins, they can be assumed to



behave as though they were part of the thoracic contents in terms of their internal pressure, rather than part of the thoracic wall and so the simplicity of the oval is maintained.

Allowing values of 7.66 cm for the antero-posterior diameter thickness and 5.33 for the transverse diameter thickness (the difference being to account for the thickness of the paravertebral musculature and posterior spinal elements), the internal diameters could be rounded to

$iDa = 39$ cm

$iDb = 18$ cm

where:

iDa = internal transverse diameter

iDb = internal antero-posterior diameter (including the vertebral bodies)

The internal cross-sectional area of the chest would then be:

$(\text{Mean radius})^2 \times \pi$

$= \{[(iDa \div 2) + (iDb \div 2)] \div 2\}^2 \times 3.14 \text{ cm}^2$

$= \{[(39 \div 2) + (18 \div 2)] \div 2\}^2 \times 3.14 \text{ cm}^2$

$= \{[19.5 + 9] \div 2\}^2 \times 3.14 \text{ cm}^2$

$= \{14.25\}^2 \times 3.14 \text{ cm}^2$

$= 203.06 \times 3.14 \text{ cm}^2$

$= 637.62 \text{ cm}^2$

$= 637.62 \div 100^2 \text{ m}^2$

Which rounds to 0.064 m^2

Returning to the calculation of internal pressure within the thoraco-abdominal cavity during a maximal Valsalva manoeuvre of 150 mm Hg.³¹

$1 \text{ Pa} = 1 \text{ N m}^{-2}$,

Therefore, $20 \text{ kPa} = 20,000 \text{ N m}^{-2}$

Force (N) = Pressure (N m^{-2}) $\times$ Area (m^2)

$= 20,000 \times 0.064 \text{ N}$

$= 1280 \text{ N}$

Therefore the force resisting axial compression of the thorax during a maximal Valsalva $\approx 1280 \text{ N}$.

As a point of comparison, at the 2004 Athens Olympics record holder Hossein Rezazadeh lifted 263.5 kg in the men's +105kg snatch. That's a force of $263.5 \times 9.8 = 2582.3 \text{ N}$. The calculated force from the Valsalva acting in opposition is thus potentially a useful 50% of that promoting axial compression even in the world record holder. It was 61% compared to his Sydney Olympic record in 2000 and is likely to be an even more significant fraction for the rest of humanity.

Type of fracture

The anatomy of the thoracic spine and in particular the intervertebral disc is not the same as in the lumbar spine,

which has been more closely studied. The lumbar intervertebral disc in a young adult has a mucoid nucleus pulposus which assists in withstanding axial compression by applying radial force to the encircling annulus fibrosus. Failure of the vertebral endplate with consequent injection of nuclear material into the vertebral body under conditions of extreme compression is therefore a possibility.

In contrast, the cervical intervertebral disc has only a crescentic annulus fibrosus anteriorly and another thin band posteriorly. The nucleus is not mucoid, but instead is quite fibrous by the young adult age of those suffering spinal fracture from epileptic seizure.³¹ Thus the mechanics of failure for the two will be different.

The thoracic intervertebral disc is not yet well studied, but it appears that in the upper and middle portion it may well resemble the arrangement in the neck and then go through a transition in the lower third, coming to resemble the arrangement in the lumbar intervertebral disc.⁹ This might rule out the possibility of high-pressure injection of mucoid nuclear material into the vertebral discs during an epileptic seizure. The rate of rise of pressure during a seizure would have an effect on whether a crush fracture or a burst fracture of the vertebral body would take place under axial load, taking into account both the viscosity of the liquid contents of the vertebral body and the speed with which the venous spaces could be emptied in relation to the rate of shortening once structural failure began.

Conclusions

It has been recorded that fractures of the vertebral bodies can occur as a result of seizures. Whilst most limb fractures are not solely the result of forces internal to the body, it appears quite likely that the thoracic vertebral fractures that are characteristic of epileptic seizures are often solely the result of forces intrinsic to the body.

These fractures are of the vertebral body and historically have been described as crush fractures, though burst fractures have been described also. The commonest location is the upper thoracic spine followed by the

mid thoracic spine.

They occur mainly in young male adults having a first seizure in their sleep and are found surprisingly commonly in 15% of epileptics studied, which rather suggests that they are looked for too infrequently.

The muscular forces involved in an epileptic seizure are greater than those involved in lifting for a number of reasons. The spinal extensors and the muscles of the abdominal wall and the intercostal muscles are all potentially in use at once in an epileptic fit. The contractile forces generated are not tempered by protective reflexes such as those from Golgi tendon organs or by coupled motion.

The axial compression present in lifting can be opposed in the thoracic spine by a significantly large force generated by the Valsalva manoeuvre commonly observed in lifters, which appears to be proportional to the weight of the lift and is not present in an epileptic fit. Whilst this force is not of any importance in actually extending the spine during lifting or in contributing to the force of a lift, it is significantly large in relation to the loads of lifting for maintaining the integrity of the thorax in the face of axial compression.

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The frozen shoulder – A practice update

Dr William A Douglas, Physician Rheumatologist, Brisbane

In the Brisbane *Sunday Mail* of 24 April 2005, columnist Dr Wright replied to a question about “frozen shoulder”. His answer encapsulates the problems of frozen shoulder treatment today: the etiology is unknown and many treatments are suggested. Fortunately he concludes that, “with proper management the outcome is positive for most people.”¹

Frozen shoulder is a significant cause of disability. It is a condition whose pathogenesis remains unclear and there is no consensus regarding optimal medical treatment. It is a fundamental principle of good medical practice that you have to understand the pathogenesis of a condition before you can successfully treat it. This applies to frozen shoulder as much as to any other pathology.

This paper outlines the current knowledge about frozen shoulder, its diagnosis, and management.

History of frozen shoulder

In 1872 Duplay² described scapulohumeral peri-arthritis, which encompassed a spectrum of pathological conditions causing a painful, stiff, dysfunctional shoulder. These conditions were various and probably included rotator cuff tendonitis, rotator cuff tears, bicipital tendonitis, calcific deposits, and severe degenerative arthritis of the shoulder.

The term “frozen shoulder” was introduced by Codman in 1934.³ He explained that the entity was “difficult to define, difficult to treat, and difficult to explain”. He felt that it may be related to tendonitis of the rotator cuff.

In 1945, after surgically exploring a number of cases with frozen shoulder, Neviasier proposed the term “adhesive capsulitis”.⁴ Surgically, he identified “a chronic inflammatory process involving the capsule of the shoulder causing a thickening and contracture of the structure which, secondarily, becomes adherent to the humeral head”.

Neviasier and Neviasier later (1987) described four arthroscopic stages of frozen shoulder and proposed that these stages should be used in treatment planning⁵ (see later). Despite the

Definition and current classification of frozen shoulder

Periarthritis (Duplay 1872)²

Frozen shoulder (Codman 1934)³

Adhesive capsulitis (Neviasier 1945)⁴

Idiopathic Frozen Shoulder (Harryman 1998)⁷

Lacks specific diagnostic criteria

Clinical description of presentation

Does not always reflect arthroscopic findings

Use synonymously with “frozen shoulder”, should differentiate from other forms of frozen shoulder

time elapsed since this work was done, these stages still apply.

Sir Reginald Watson Jones wrote, “In the early acute stage, the worst treatment is manipulation under anaesthesia or frequent passive and forcible stretching by a masseuse. Forcible treatment tears the tissues which are already inflamed and increases serofibrinous exudation. The one treatment of paramount importance is active exercise performed for a few minutes hourly throughout the day... successful treatment may be summed up in two words – active exercise”.⁶

Most readers will recall the common occurrence of shoulder-hand syndrome not many years ago. This condition occurred after prolonged immobilization in bed following myocardial infarction. It also occurred after a stroke where the paralyzed upper limb was left dependent or immobile for some weeks. Shoulder-hand syndrome unfortunately still occurs after chest surgery and axillary node dissection.⁷

Idiopathic frozen shoulder (IFS) can be defined as progressive painful global stiffness of a shoulder joint that comes on in the absence of the following exclusion criteria:

1. Systemic inflammatory joint disease including rheumatoid arthritis and polymyalgia rheumatica;
2. Diabetes mellitus, both type 1 and type 2;
3. Radiological evidence of severe osteoarthritis or trauma to the shoulder;
4. Extensive calcification in the rotator cuff mechanism;
5. Suspicion or demonstration of complete rotator cuff tear(s) demonstrated on ultrasound or MRI;
6. Recent surgery to the shoulder;
7. Acute calcific tendonitis with subacromial bursitis.

This paper focuses on the medical aspects of frozen shoulder.

Pathogenesis of idiopathic frozen shoulder

Various investigators have been unable to answer the question “why is the frozen shoulder frozen”? Over the last 60 years, there have been many proposed pathological mechanisms but none has been proven.

Stages of frozen shoulder described by Neviasier and Neviasier⁵

1. A mild erythematous synovitis
2. Acute synovitis with adhesions in dependent folds of the synovial lining
3. Maturation of adhesions with less reactive synovitis
4. Chronic adhesions without synovitis

One constant factor is age. Idiopathic frozen shoulder rarely occurs before the age of 50 years. The peak incidence of IFS is between 50 and 70 years. Thus, it is reasonable to assume that degenerative processes within the shoulder, in some way, are directly related to the condition.

It has been suggested that microscopic calcium hydroxy apatite crystal deposition increases vascularity and interaction between the collagenous structures and microcrystalline complexes containing calcium.⁸ It is postulated that this mechanism produces a low-grade but progressive inflammatory synovitis.

One can compare the normal epithelial lining of the shoulder to that of the peritoneum. The peritoneum, under normal circumstances, is smooth and pink but with relatively minor irritation such as surgical procedures, bleeding, or contamination, the peritoneal membrane becomes inflamed and may

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progress to a reactive fibrosis with chronic adhesions.

Arthroscopy in the early stages of frozen shoulder shows mild to moderate inflammatory synovitis of variable thickness under the rotator cuff capsule, along the biceps tendon root, posterior capsule and superior labrum.⁹

Magnetic resonance imaging (MRI) has improved our understanding of the frozen shoulder by showing a specific pattern of post-gadolinium enhancement during the first few months after symptom onset.¹⁰ Dr Paul O'Connell of Queensland Diagnostic Imaging has performed MRIs without gadolinium during the severe painful phase of frozen shoulder. These show abnormal T2 weightings superiorly (the rotator cuff interval) and in the inferior glenohumeral region including the inferior pouch of the shoulder and the long head of biceps recess (personal communication). These findings have also been recorded by others using gadolinium.^{11, 12}

Thus, in all probability, IFS is the result of a localized inflammatory disorder secondary to underlying degenerative changes occurring within the shoulder joint, such as in the humeral cartilage or rotator cuff tendons.

Clinical evaluation of a patient presenting with a frozen shoulder

In clinical practice, these patients commonly present with a global loss of shoulder motion, with marked restriction of all active and passive movements.

External rotation and abduction are reduced by 30 - 50% compared to the unaffected arm.⁷ The shoulder is painful and stiff. Night pain is a major feature of IFS.

There is little or no localized soft tissue tenderness. Some associated muscle wasting may already be present. To confirm a diagnosis of IFS, these findings should be present for at least three weeks.

There is often no precipitating injury or incident. However, it is not uncommon for the patient to report having pre-existing discomfort in the shoulder without loss of movement.

Investigations for IFS

Blood studies

Routine blood studies are normal. If

the inflammatory markers (WCC, ESR, and C-reactive protein) are elevated, the diagnosis of idiopathic frozen shoulder must be questioned.

The HLA B27 histocompatibility antigen is no more prevalent in patients with frozen shoulder than in controls.¹³

Plain x-rays

Plain x-rays seldom show a gross abnormality and are generally normal for the patient's age. Minor degenerative changes on the greater tuberosity of the humerus and small areas of calcification in the supraspinatus tendon are commonly observed.

Radioisotope bone scans (99 m technetium diphosphonate)

Binder et al¹⁸ using radionuclear scans in 38 patients found that 90% of the patients with frozen shoulder had increased uptake on the symptomatic side before treatment. They were unable to show an association between bone scan activity and the disease severity, duration of symptoms, arthrographic findings, or ultimate outcome.

Arthrography

Arthrography is not a necessary investigation for the diagnosis of frozen shoulder. Contracture of the axillary pouch has been well documented and intra-articular volume and pressure measurements have been correlated with the restriction of shoulder range. Some authors have noted that arthrographic findings fail to differentiate between a true frozen shoulder and a post-traumatic stiff shoulder.⁷ There is no correlation between arthrographic findings and treatment outcomes.

Ultrasound

The main sonographic features of adhesive capsulitis are a constant limitation of a sliding movement of the supraspinatus tendon against the scapula. Sonography, in expert hands, may identify degenerative changes or rupture of the rotator cuff mechanism, however it is of little use in assessing or diagnosing the frozen shoulder.

Magnetic resonance imaging (MRI)

MRI is the most useful non-invasive

diagnostic investigation for IFS. Given the limitations on access to MRI, it is best reserved for situations where the differential diagnosis is unclear; for example, when needing to document or exclude a mechanical lesion.

Previous studies

Contemporary management of IFS is constrained by the lack of well-

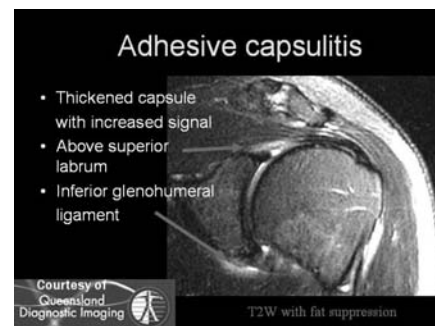


Fig. 1. Adhesive capsulitis - thickened capsule with increased signal.



Fig. 2. Abnormal signal within the rotator interval

designed studies.^{14, 15} Many studies of frozen shoulder have included patients suffering from long-standing painful, stiff shoulders, diabetes mellitus, and severe degenerative shoulder changes. This has led to conflicting and disappointing results.^{16, 17}

It is a challenge for practitioners working in the area of musculoskeletal medicine to address this deficiency and to develop recommendations for the management of this condition based on sound scientific research.¹⁴

Management and outcomes of IFS

In the absence of evidence-based guidelines for the treatment of IFS, I present my personal experience from my rheumatology practice.¹⁸ The key finding from this series is that early intervention is essential. Once the shoul-

der has been frozen for greater than four months, the likelihood of successful medical treatment decreases considerably.

Dr Douglas's recommended regime for IFS

- Intraarticular (IA) injection of hydrocortisone acetate suspension or its commercial equivalent with 2 ml xylocaine 2% using appropriate aseptic techniques;
- Oral prednisone 15-20 mg per day (dose relating to body weight);
- Weekly review to assess progress;
- Further IA injections may be given (total three) depending on response. More than three injections will be ineffective and add to the risk of adverse effects;
- Prednisolone is taken for two weeks and then phased out by a mean of five weeks;
- A home exercise program using a pulley is advised. This is to stretch the shoulder capsule passively. This should be done at least twice a day along with a light general exercise program.

The first sign of positive response to this regime is relief of night pain and improved sleep. This is followed by the gradual return of a full range of movement. In my experience, most people show improvement in terms of relief of pain and range of movement within a fortnight. More severe cases (those needing three IA injections) will take up to eight weeks before reporting freedom from pain and marked improvement in range of movement.

To date there have been no failures with this regime, except in patients who have presented late (greater than four months from the onset of stiffness). They may report some relief of pain following the above treatment but little or no improvement in the range of movement.

From my perspective, surgery and invasive techniques such as manipulation under anesthesia or arthrographic joint distension (hydro-dilatation) have no place in the management of IFS.

Prognosis of idiopathic frozen shoulder

Schaeffer¹⁹ and Reeves²⁰ report high

morbidity and prolonged recovery times. This is the view propagated by internet websites linked to orthopedic practices and associations. These present a pessimistic view of the condition, *"you should learn to live with the condition for 12 - 24 months (provided the pain is tolerable and you can cope with the activities of daily living)"*.²¹ *"Frozen shoulder will generally get better on its own. However this takes some time, occasionally up to two - three years."*²² Treatment options discussed invariably involve orthopedic interventions.

It is my experience that early presentation and prompt treatment of IFS will result in satisfactory recovery.¹⁸

The results challenge those working in this area to change the current mind set and create better outcomes for those suffering from IFS.

Other causes of stiff and painful shoulder

Diabetes mellitus

The stiff shoulder is a common occurrence in both type 1 and type 2 diabetes and is often included in studies for the treatment of frozen shoulder as it is considered by some authors to have the same pathogenesis as that occurring in IFS.

Musculoskeletal disorders are more common in patients with type 1 diabetes than those with type 2 diabetes. The study by Fisher et al²³ showed a prevalence of frozen shoulder in type 1 diabetes with cherioarthropathy of 44.8%. This was significantly more than matched type 1 diabetes with normal hands where the incidence of stiff shoulder was only 7.1%.

Stiff shoulder in both diabetes groups has certain characteristics contrasting with the clinical description of frozen shoulder in IFS. The mean age of onset in diabetes with cherioarthropathy and frozen shoulder was 44 years compared to IFS where the peak incidence is between 50 and 70 years of age at onset.²⁴ The marked loss of shoulder movement in diabetes comes on slowly and progressively and the severe pain which occurs in the second stage of IFS is generally not great in the diabetes group. Few diabetes patients regained normal shoulder movement despite a mean

interval from the onset of symptoms to assessment of eight years.^{25, 26}

Various authors conclude that there is an increased prevalence of frozen shoulder in diabetes with microvascular disease and cherioarthropathy of the hands, wrists, and hips. It is suggested that both rheumatological conditions are related to underlying abnormalities in glycosation of collagen rather than to an inflammatory synovitis.²⁵

Shoulder stiffness in diabetes patients generally responds to a program of daily gentle stretching, resistance exercises, and weekly hydrotherapy. The use of corticosteroids (IA and/or oral) is relatively contraindicated. Invasive surgical procedures are not recommended.

Rheumatoid arthritis

Stiffness of the shoulders in rheumatoid sufferers may occur in the acute phase of the illness or over a period of years. In the initial stages, it is due to acute rheumatoid synovitis. In the latter stages, it is due to a chronic synovitis of the shoulder joint which may lead to gradual stiffness of the shoulder resulting from a gradual destruction of the rotator cuff mechanism, tendon sheaths, and erosion of the humeral head cartilage.

In chronic long-standing rheumatoid arthritis, shoulder stiffness comes on gradually and generally is more of an inconvenience because of limited movement. Most people with rheumatoid arthritis adjust to fairly severe rheumatoid shoulder damage including complete rotator cuff tears.

Surgery to repair the rotator cuff is of doubtful benefit in most people with rheumatoid arthritis, although surgery is sometimes indicated in the younger person.

Polymyalgia rheumatica (PMR)

PMR rarely occurs before the age of 60. Men and women are affected equally. The onset of the condition comes on over a matter of days or weeks and affects both shoulders more or less equally.

The shoulder pain and stiffness may be extremely severe and are remarkably similar to that occurring in IFS. The sufferers complain of shoulder pain at night associated with marked

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stiffness and sleep disturbance. There is generally major functional loss of shoulder movement interfering with the ability to dress and self-care.

Arthroscopic and histological investigations of patients with PMR have shown that the condition is associated with a mild synovitis of the shoulder joint.²⁷ Shoulder pain and stiffness always improves dramatically within 24 hours of administration of intra-articular corticosteroids with or without the addition of oral corticosteroids. With these measures, resolution of shoulder pain and stiffness is rapid and complete.

PMR is always associated with a rise in the inflammatory markers (ESR and CRP), albeit delayed in some individuals for some weeks.

Osteoarthritis

Severe osteoarthritis of the shoulder is relatively uncommon and generally is secondary to repeated trauma over many years. One sees this condition in manual workers such as boilermakers, sheet metalworkers, and home removalists. Shoulder stiffness occurs gradually and progressively over some years and is rarely associated with severe pain. Radiologically, there is marked narrowing of the glenohumeral joint and it is commonly associated with osteophyte formation on the inferior head of the humerus.

Ankylosing spondylitis (AS)

Occasionally severe pain and stiffness of both shoulders occurs with this condition. It tends to parallel the inflammatory process occurring in the spine. The pain and stiffness of the shoulders initially behaves like that of the early stages of idiopathic frozen shoulder (IFS), with severe night pain and stiffness consistent with an underlying synovitis. The shoulder pain and stiffness is best treated with intra-articular corticosteroids and hydrotherapy. Seldom is full shoulder movement regained. The use of the recently introduced anti-TNF preparations for the treatment of AS may dramatically and favorably alter this.

Scleroderma

Articular involvement, particularly in the CREST variant of scleroderma,

has been described and is associated with widespread periarticular calcification.²⁸ I have seen one case of a woman with advanced bilateral calcification of the rotator cuff. She had mild CREST-type scleroderma. Her shoulder movement was moderately impaired and was associated with severe degeneration of the rotator cuff.

In primary systemic sclerosis (PSS), stiffness of the shoulders occurs at times but is generally due to the tethering effect of the overlying skin rather than to direct involvement of the shoulder joint or rotator cuff. However, underlying tendonitis or myositis may be responsible for the shoulder pain.

Acute calcific bursitis

Acute calcific bursitis is relatively common. Radiologically, there are fluffy deposits of calcium within the supraspinatus tendon adjacent to the sub-acromial bursa. A sudden release of calcium apatite crystals produces an acute, extremely painful shoulder initially with total loss of movement.

Plain x-rays of the shoulder confirm the presence of fluffy calcium deposits. There is little or no rise in the ESR or CRP. One should exclude the presence of chondrocalcinosis elsewhere by checking for hypercalcemia and conditions such as hyperparathyroidism and hemochromatosis.

Injection of xylocaine and hydrocortisone into the sub-acromial bursa, combined with immobilization of the arm in a sling for 12-24 hours and the application of ice, is generally sufficient to give the person complete and lasting relief within 48 hours.

Idiopathic frozen shoulder in the medicolegal setting

It is not uncommon to be asked to review the case of an injured worker where a surgical procedure appears to have resulted in the aggravation of the injury. Generally, they have suffered a soft tissue injury to the shoulder region and have undergone some form of shoulder surgery on the basis of a worker's compensation injury. These individuals keep the affected arm immobile and dependent, are emotional and show an exaggerated response to pain on attempted movement of the arm. As one would expect, their re-

sponse to various forms of treatment including pain management and rehabilitation is often limited.

Medical practitioners should be very careful in advising surgical procedures for people presenting with work-related shoulder injuries which are apparently causing chronic upper limb pain not responding to conservative measures. The impact of future litigation may affect these people's ability to respond favorably to surgical procedures. The reasons for this are complex and beyond the scope of this article.

Summary

- IFS is the result of a low grade inflammatory synovitis leading to adhesions and fibrosis of the shoulder capsule.
- Diagnosis is directed at excluding other causes of a painful, stiff shoulder.
- Early active treatment of IFS leads to good outcomes.
- Other conditions causing painful stiff shoulder are managed according to the underlying pathology.

What still needs to be done

- Research into the pathogenesis of IFS
- Well-designed studies to establish the best treatment of IFS in its early stages

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Post-herpetic neuralgia (PHN): Review of the evidence base for treatment and prevention

Dr David Roselt, Musculoskeletal Pain Medicine, Bundaberg, Queensland

Herpes zoster (HZ) is a recrudescence of latent varicella zoster virus (VZV) from dorsal root or cranial nerve ganglia, present since primary infection with varicella. HZ presents as a rash of 2-3 weeks' duration in immunocompetent patients, often accompanied by moderate to severe dermatomal pain. The pain may have a prodromal component, and precede the appearance of the rash by several days. In some patients pain does not resolve when the rash heals, but may continue for weeks, and sometimes for months or years. This persistent pain has been termed "post-herpetic neuralgia" (PHN).^{1, 2}

The risk of PHN is significantly reduced with antiviral therapy for patients with HZ both clinically and statistically, but it does not prevent PHN in all patients. Almost 20% of patients aged over 50 years continue to have neuropathic pain (NP) six months after rash onset, despite treatment with anti-viral agents famciclovir or valacyclovir beginning within 72 hours of the onset of rash.³⁻⁵ The absolute importance of the early use of antiviral medication and optimum pain relief cannot be overemphasized.

The virus is believed to reactivate frequently but competent cell-mediated immunity (CMI) usually prevents symptoms and signs of clinical disease. The commonest cause of presentation is decline in CMI with age.⁶

Reduced immunity can also be due to:

- malignancy, for example, lymphoma
- its treatment with chemotherapy or radiotherapy
- HIV infection
- immunosuppressant drugs, such as after organ transplant surgery, or for disease management, for example, steroids.

As the age of the population, these illnesses, and their treatments all in-

crease, the incidence of HZ is expected to rise. HZ may also increase as less childhood varicella in the population provides fewer opportunities for re-exposure, which is believed to boost specific immunity and help maintain latency.⁶

Three phases of post-herpetic pain have been described:

- Acute herpetic neuralgia (AHN) accompanies the rash and lasts for up to 30 days from the onset of HZ rash
- Subacute herpetic neuralgia (SHN) lasts 30-120 days from the onset of rash
- Postherpetic neuralgia (PHN) is defined as pain that persists for greater than or equal to 120 days from the onset of rash.

The significance of the three phases is that patients with subacute herpetic neuralgia (SHN) who did not develop PHN were significantly younger with less severe acute pain than PHN patients. They were more likely to have severe and widespread HZ rash than patients whose pain did not persist longer than one month from rash onset.⁷⁻⁹

Risk factors in HZ patients for development of PHN include:

- Older age
- Female sex
- Presence of a prodrome of dermatomal pain
- Greater rash severity
- Greater acute pain severity.¹⁰

Recent major advances in the treatment of PHN

Based on results of randomized controlled trials (RCTs), there have been five recent major advances in the treatment of PHN:

- Gabapentin
- Pregabalin
- Lignocaine patch 5%
- Opioid analgesics
- Tricyclic antidepressants (TCAs), with nortriptyline preferable to amitriptyline.⁶

Gabapentin

This second generation anticonvulsant was recently shown to provide significant benefits over placebo for PHN in two large RCTs. Gabapentin is a structural analog of γ -aminobutyric acid (GABA). The mechanism of action has not been fully elucidated, but appears not to involve binding to GABA receptors. Recent evidence suggests gabapentin modulates $\alpha_2\delta$ calcium-channel subunits important in NP.^{11, 12}

Gabapentin analgesia is unaffected by opioid antagonism, working via different mechanisms. Repeated administration of gabapentin does not lead to analgesic tolerance.¹³

Doses of 1800-3600 mg daily produced statistically significant reductions in daily pain ratings, and improvements in sleep, mood, and quality of life.^{11, 14}

In a systematic review of published studies, the NNT (number needed to treat)¹⁵ for a 50% reduction in NP was 4.39; the NNH (number needed to harm) was 4.07 for minor harm and 12.25 for major harm; for example, necessitating withdrawal from a clinical trial.^{16, 17}

Adverse effects of gabapentin include:

- Dizziness
- Somnolence
- Less commonly, mild peripheral edema.

These may require dose adjustment but treatment can usually continue. The elderly are also prone to ataxia, gait, and balance problems, and cognitive impairment.

Dose reduction is necessary with impaired renal function based on creatinine clearance. This may be calculated according to the Cockcroft-Gault formula:

Men:

$$\frac{\text{Body wt (kg)} \times (140 - \text{age in years}) \times 0.0885}{72 \times \text{se Cr (mmol/L)}}$$

Women: Above calculation $\times 0.85$

Gabapentin is generally well tolerated and safe. It lacks significant drug interactions. Treatment can be initiated with 100 mg tds or 100-300 mg n, with titration by 100mg tds, as tolerated, to 1800-3600 mg daily. Complete relief may occur rarely, but unacceptable adverse effects that do not resolve over a few weeks limit the dose.⁶

The efficacy of gabapentin in NP was further evaluated in a randomized, double-blind, active placebo-controlled four-period crossover trial published in the *New England Journal of Medicine* in 2005.¹⁸ Patients were randomized to received gabapentin, sustained-released morphine, a combination of gabapentin and morphine, and an active placebo lorazepam.

Fifty-seven patients were randomized to the different arms of the trial, 35 with painful diabetic neuropathy (PDN) and 22 with PHN, and 41 completed the trial.

In a linear mixed model, treatment contrasts were adjusted for all observed carryover effects related to the crossovers. Mean daily pain intensity (+/- standard error SE) on an 11-point Likert pain numerical rating scale (NRS) at baseline and at maximum tolerated doses of the study drug(s) was 5.72 +/- 0.23 at baseline, 4.49 +/- 0.34 for placebo, 4.15 +/- 0.33 for gabapentin, 3.70 +/- 0.34 for morphine, and 3.06 +/- 0.33 with the gabapentin-morphine combination. This was statistically significant for the gabapentin-morphine combination versus placebo, gabapentin, and morphine. The combination produced the best results in terms of pain reduction, but the maximum tolerated doses were lower with the combination, and there was a higher frequency of constipation than with gabapentin alone, and a higher frequency of dry mouth than with morphine alone.

Recent evidence suggests that a clinically important reduction in pain is usually present with a change from baseline on an 11-point NRS of 30% or 2 points.^{19, 20, 21}

Pregabalin

A new anticonvulsant similar to its developmental predecessor gabapentin, pregabalin showed greater analgesic activity in rats in studies of

NP mechanisms and treatment. The exact mechanism of action is unclear but pregabalin is thought to reduce excitatory neurotransmitter release by binding to the $\alpha_2\delta$ protein subunits of voltage-gated calcium channels.²²

Orally administered pregabalin in doses of 150-600 mg per day, in 2-3 divided doses, was superior to placebo in relieving NP and related sleep disturbance in three randomized, double-blind, placebo-controlled multicenter trials of 8-13 weeks' duration in a total of 776 patients with PHN.²³⁻²⁶

Weekly mean pain scores in all three trials and weekly mean sleep interference scores assessed in two studies were significantly improved after one week of treatment with pregabalin. In two studies significant improvements in daily mean pain scores were evident on the first or second day of treatment.

Pregabalin was well tolerated when force-titrated over one week to fixed dosages of 150-600 mg per day in clinical trials that enrolled mostly elderly PHN patients. Dizziness, somnolence, and peripheral edema of mild-moderate degree were the most common treatment-associated adverse effects.

Pregabalin is rapidly absorbed orally and has linear pharmacokinetics. Food delays the rate but not extent of absorption. It is not significantly metabolized or bound to plasma proteins, so there are minimal drug interactions. Renal excretion is predominant, with 98% excreted unchanged in the urine.²²

Pregabalin was approved by the European Union (EU) in July 2004 for the treatment of peripheral NP, including PHN and PDN, as well as adjunctive treatment for partial seizures in epilepsy. It was approved by the Food and Drug Administration (FDA) in the US in September 2004 for PHN, PDN, and partial seizures in adults with epilepsy. In Australia, pregabalin is in the process of being approved for registration by the Australian Drug Evaluation Committee (ADEC) for the Therapeutic Goods Administration (TGA) as adjunctive therapy in adults with partial seizures with or without secondary generalization, and for treatment of neuropathic pain.

Lignocaine patch 5%

Rowbotham and colleagues published results of several double-blind vehicle-controlled studies in which topical lignocaine in either gel or patch form relieved pain in patients with PHN with allodynia.²⁷⁻²⁹

Use of the 5% lignocaine patch is supported by two published RCTs in patients with PHN and prominent allodynia (pain in the affected dermatome in response to innocuous stimuli) who experienced statistically greater pain relief with 5% lignocaine patches compared with vehicle controlled patches without lignocaine.^{28, 29}

An open-label, nonrandomized, effectiveness study was performed in 332 patients, using up to three patches applied to the area of greatest pain for 12 hours per day for 28 days.³⁰ The mean time from the onset of HZ to treatment with the 5% lignocaine patches was 28 months. The patches produced reductions in all mean pain intensity, pain interference with quality of life (QOL), and composite scores at all time points ($P = 0.0001$). Overall, 66% of patients reported improvement in pain intensity, and 74% reported improved QOL by day seven. Approximately 43% who did not respond by day seven experienced improvements in pain intensity by day 14. For all measures of pain intensity, relief, and interference with QOL, improvements from baseline were equally significant regardless of time since HZ onset. Overall, approximately 60% of patients reported moderate to complete pain relief at final evaluation. The 5% lignocaine patch was very well tolerated.

Based on previous RCTs and the current study designed to gauge response in the clinical setting, the 5% lignocaine patch should be first-line therapy, alone or in combination with other agents for PHN. It is efficacious and safe to use with minimal systemic adverse effects and drug interactions, and easy to administer. Although equally effective in long-standing PHN, it would be prudent to begin therapy as early as possible in the course of PHN. The patch is becoming available in Australia, with reports that it is available through the pain clinic at St George Hospital in Sydney.

Application is usually made of a

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maximum of three patches per day for a maximum of 12 hours daily, applied directly to the area of maximum pain and allodynia. It is not approved for application in HZ or patients with open lesions, as it is not sterile. No significant systemic absorption occurs. Relief is usually apparent within two weeks. There is minimal systemic uptake and no need for dose escalation. These qualities are very attractive, with minimal concerns about contraindications and drug interactions. The onset of relief is relatively rapid, and should be evident within 1-2 weeks.³⁰

Topical lignocaine is also available in Australia as a 5% gel, and so may be an alternative if the 5% lignocaine patch can not be obtained.²⁷

Lignocaine administered intravenously (IV) was shown to produce pain relief in patients with PHN equivalent to morphine and superior to placebo, although the mechanism of action is unclear.³¹ This analgesic effect of systemic lignocaine is believed to involve blocking sodium channels and may occasionally be reproduced by oral mexiletine or flecainide. However, these drugs are often rejected by patients because of adverse effects.

Opioid analgesics

For many years it was claimed that NP was unresponsive to opioids. Intravenous and nonblind studies have suggested that patients with PHN can obtain significant pain relief from opioid analgesics, and these medications have become more widely used in the treatment of PHN and other NP syndromes. Opioid analgesics have an important role in the treatment of PHN. There is now compelling evidence that NP responds to opioids.^{18, 31-34}

The efficacy of opioid analgesics in PHN was first demonstrated in the RCT comparing IV morphine with IV lignocaine and placebo.³¹ This suggested that longer-term oral medication may be effective. Three RCTs of controlled release (CR) oral opioid treatment for PHN have now been published.

CR oxycodone titrated to a maximum dose of 60 mg per day provided significant benefits with respect to pain, disability, and allodynia compared to placebo.³³

CR morphine titrated to a maximum

dose of 240 mg per day provided statistically significant benefits with respect to pain and sleep, but not physical functioning and mood, compared to placebo.³⁴

This study was a three-period crossover trial comparing opioid analgesics to tricyclic antidepressants (TCAs) as well as placebo. Patients preferred the opioids despite adverse effects.

The recent *New England Journal of Medicine* study¹⁸ was prompted by preclinical studies suggesting that additive benefits may occur between morphine and gabapentin.³⁵⁻³⁷ There is evidence that opioid tolerance can be prevented by the concurrent use of gabapentin.³⁸

This *New England Journal of Medicine* study provides further evidence of the efficacy of opioids for PHN and NP.¹⁸

The most common adverse effects of opioid analgesic therapy are:

- Constipation
- Sedation
- Nausea.

In elderly patients, there is greater risk of cognitive impairment, mobility problems, and hip fracture from falls. Opioids must be used cautiously in patients with a history of substance abuse, overdose, and attempted suicide. Patients treated with opioid analgesics may develop analgesic tolerance (reduction in analgesic benefits over time) but a stable dose can often be achieved. All patients develop physical dependence (withdrawal symptoms with abrupt discontinuation or rapid dose reduction) and should be advised about this. The risk of substance abuse is thought to be very low without past history of this, especially in patients with PHN who are usually elderly. This is not usually a problem when treating pain as opposed to recreational use. Concerns about abuse certainly do not justify refraining from the use of opioid analgesics for PHN.⁶

Treatment can begin with short-acting oxycodone or morphine at the equivalent dose of 5-15 mg orally given 4 hourly. Conversion to a CR form of oxycodone, morphine, methadone, or even transdermal fentanyl after one to two weeks is usually optimal with short-acting opioid continued as required for

breakthrough pain. There is no maximum dose of opioid analgesics with careful titration and monitoring, although pain medicine referral should be considered if morphine equi-analgesic doses of greater than 120 mg per day are needed.⁶

Tramadol

Another analgesic with recently demonstrated efficacy in NP is tramadol, the weak opioid μ -receptor agonist and monoamine (that is, noradrenaline and serotonin) reuptake inhibitor.³⁹

In a pilot study of patients with PHN, tramadol was compared with clomipramine alone and combined with levomepromazine. In the tramadol group, nine of 10 patients reported their pain relief was satisfactory or better.⁴⁰

A subsequent multicenter, randomized, double-blind placebo-controlled, parallel-group trial published in *Pain* in 2003 evaluated 127 outpatients with PHN in Paris, France. The mean duration of NP at inclusion in the study was six months. Sustained release tramadol could be increased from 100 mg per day to 400 mg per day in patients less than 75 years of age, or to 300 mg in those older than 75 years. Treatment was given for six weeks. Mean pain intensity measured on Visual Analogue Scale (VAS) was significantly lower on tramadol than placebo. The mean (SD= standard deviation) dose in the tramadol group was 275.5 (89.7) mg per day. There was no significant difference in the tramadol and placebo groups in terms of adverse effects, with the main treatment associated adverse effects being nausea and constipation. The number needed to treat (NNT) to obtain one patient with more than 50% pain relief was 4.76 (3.51-6.01).⁴¹

Tricyclic antidepressants (TCAs)

There are many RCTs demonstrating the efficacy of TCAs for PHN and PDN.⁴²

Amitriptyline is still the most widely used TCA for PHN and other NP syndromes as it is the most studied. However, it is poorly tolerated, and relatively contra-indicated in the elderly. It has the highest incidence of adverse

effects such as sedation, anticholinergic effects such as dry mouth, constipation and urinary retention, and postural hypotension.

ARCT demonstrated equivalent efficacy for nortriptyline, but fewer adverse effects. Nortriptyline is the preferred TCA for PHN.⁴³ Cardiac toxicity and adverse effect profiles require considerable caution when treating older patients with PHN. Dry mouth is the most common adverse effect, occurring in up to 40% of patients treated with amitriptyline and 25% of patients treated with nortriptyline. Constipation, sweating, dizziness, disturbed vision, and drowsiness are reported by as many as 30% of patients treated with amitriptyline and 15% of those treated with nortriptyline.

TCAs must be used very cautiously in patients with a history of cardiovascular disease, glaucoma, urinary retention, and autonomic neuropathy. Screening electrocardiogram to check for cardiac conduction abnormalities is recommended by some authors before beginning TCA treatment for patients over 40 years of age or with a past history or risk factors for heart disease.⁶

TCAs must be used cautiously when there is a risk of suicide or accidental death from overdose. They may cause balance problems and cognitive impairment in elderly patients. TCAs can interact with drugs metabolized by the hepatic cytochrome enzyme P450 2D6 (for example, cimetidine and type 1C antiarrhythmics). All serotonin reuptake inhibitors (SSRIs) inhibit P450 2D6, so caution is necessary if co-administering TCAs and SSRIs to prevent toxic TCA plasma concentrations. This combination is generally not recommended.⁶

To decrease adverse effects, all TCAs should be initiated at low dosages (10-25 mg in a single dose taken at bedtime) and should then be slowly titrated as tolerated. It has been claimed that the analgesic effects of TCAs occurs at lower dosages than the antidepressant effects, but there is no evidence of this from controlled clinical trials. TCAs should be titrated to a dose of 100-150 mg per day as tolerated for an adequate analgesic trial. It should be explained to the patient that the TCAs

have analgesic effects independent of their antidepressant effects.⁶

Choices for treatment and combination treatments

There are few clinical trials in which these medications have been directly compared.^{18, 34, 43} More direct head-to-head comparisons regarding efficacy, safety, and tolerability would be very useful. Analgesic responses to opioid analgesics and TCAs were uncorrelated in the three-period, placebo-controlled crossover trial.³⁴ Patients not responding to one of these types of medication may still respond to another.

The RCTs of the five first- and second-line treatments for PHN examined the efficacy of single medications versus placebo or a comparison drug. Combination therapy, however, is the norm in the clinical setting. Until recently there were no Level II data regarding the additive or synergistic benefits of combination treatment.

It is not yet fully known which patients are most likely to benefit from different medication combinations. Disadvantages of combination treatment include an increased risk of adverse effects as the number of medications is increased and difficulty identifying which medication is responsible should they occur.

Ideally, combination therapy would involve a commonsense approach based on an understanding of the mechanisms of action and adverse effect profiles of the individual agents and on individual patient characteristics. This may become easier as the understanding of pain mechanisms in PHN increases.^{44, 45, 49}

The maximum tolerated doses of drugs such as gabapentin and opioids administered individually often reduce NP by only 26-38%.^{11, 33, 34} This is because of incomplete efficacy, dose-limiting adverse effects, or both. The hope is that the combination of mechanistically distinct agents may result in additivity or synergism and may improve efficacy at lower doses, and with fewer adverse effects. Except for sedation, the adverse effect profiles of gabapentin and the opioids do not significantly overlap, so the combination is promising in this regard.

Sedation is mediated only supraspinally, whereas both drugs have been demonstrated to have analgesic effects at supraspinal, spinal, and peripheral sites of action.^{46, 47, 48} There is the chance of more additivity for analgesia than sedation.⁴⁹

Gilron et al revealed that the maximum tolerated doses of gabapentin and morphine were lower with combination treatment which is consistent with an additive interaction.¹⁸ This supports findings in a previous study in healthy subjects which suggested that the addition of morphine to gabapentin resulted in higher serum concentrations of gabapentin than are seen with gabapentin alone.³⁶

Also examined was the use of combination therapy titrated concurrently rather than the more common option of sequential therapy. This allowed more flexibility in titration and balancing between analgesic and adverse effects. It allowed lower doses with greater pain relief and tolerable adverse effects. This approach warrants further investigation.⁴⁹

An unknown percentage of patients with PHN will not respond to these first- and second-line treatments when used alone or in combination. For these unfortunate patients, other treatments deserve consideration, and referral to a pain management center should be contemplated sooner rather than later.

First-generation anticonvulsants such as carbamazepine and phenytoin have traditionally been used for the management of NP and have beneficial effects in trigeminal neuralgia (TN) and PDN.^{50, 51}

Evidence for their benefit in PHN is lacking however, and their adverse effects particularly in the elderly and frail can be unpleasant.^{52, 53, 54} Watson concluded that the benefits of treatment for PHN with first generation anticonvulsants had often been unimpressive or difficult to interpret because of concomitant use of antidepressants.⁵⁵

Oral NSAIDs seem to be of little benefit in acute HZ pain or in PHN, although paracetamol combined with weak opioids is often prescribed.⁵⁶

Other agents

Baclofen, a GABA-B receptor ago-

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nist used primarily as an antispasticity agent, has been beneficial in patients with TN and may be useful in other disorders, although evidence is lacking.⁵⁷ Sedation, hypotonia, and confusion in the elderly may be problematic. Effective in the treatment of dystonia associated with complex regional pain syndrome (CRPS) and refractory spasticity, intrathecal baclofen has anecdotal support as an adjuvant treatment for NP.^{58, 59}

Clonidine, an α_2 -adrenergic receptor agonist, may be an effective analgesic in some patients. Neuraxial clonidine has been shown to be effective in treatment of NP associated with cancer and may be better tolerated than when systemically administered. It appears to work by mimicking the effects of endogenous pain-inhibiting monoaminergic neurotransmitters (for example, noradrenaline).⁶⁰ However, its long-term efficacy in NP not associated with cancer has not been validated.⁶¹ The reported analgesic benefit of tizanidine, another α_2 -adrenergic receptor agonist and antispasticity agent, also awaits confirmation.

NMDA receptor antagonists

The N-methyl-D-aspartate (NMDA) receptor is complex and involved in peripheral and central pain pathways. Centrally acting drugs are available, though unwanted adverse effects including ataxia, somnolence, short-term memory loss, and other psychological symptoms often preclude their use.

In central sensitization states, it is possible that potentiation of opioid analgesics may be a useful benefit of NMDA receptor antagonists. NMDA receptors are involved in the development and maintenance of changes in neuronal excitability with respect to the development of sensitization, allodynia and persistence of NP following neural damage.

Ketamine is known to produce analgesia at least partially as a result of blocking these receptors. Recent studies have shown that ketamine reduces NP in some patients with PHN. However, adverse effects limit its use.⁶²⁻⁶⁶ Ketamine is not licensed in Australia for oral use.

Based on these initial reports, increasing attention is being paid to the

role of NMDA antagonists in the treatment of NP, with the challenge being to find agents with a more favorable therapeutic ratio than ketamine.

Dextromethorphan is an antitussive, and weak opioid μ -agonist, and an NMDA receptor antagonist with analgesic properties. There is limited evidence of its efficacy in NP. Dextromethorphan in relatively low doses of 20 mg three times daily has not produced lasting analgesia and adverse effects are a problem.⁶⁷

In a recent double-blind, placebo-controlled trial, dextromethorphan was titrated to mean dosages of 439 mg per day in 13 patients with PHN and to 381 mg per day in 13 patients with PDN.⁶⁸

There was evidence of statistically significant benefit from dextromethorphan treatment in the patients with PDN, but not in those with PHN. The role of NMDA receptor antagonists in the treatment of PHN needs to be clarified by the results of further studies of dextromethorphan and other NMDA receptor antagonists, for example, amantadine and memantine.^{69, 70}

Methadone both blocks the NMDA receptor and is a potent long-acting μ -opioid analgesic. It would be valuable to further examine whether this agent may have a unique role to play in the management of PHN and other NP syndromes.

A double-blind randomized controlled crossover trial examined the analgesic effectiveness of low-dose methadone in NP.⁷¹ It followed 18 patients with a variety of chronic NP syndromes, who had previously responded poorly to various other agents. The 20 mg daily dose (10 mg bd) produced statistically significant reductions in maximum pain intensity and average pain intensity on VAS compared to placebo. The dosing was second daily to allow for the very variable biological half-life of methadone, quoted as 10 to over 75 hours.⁷² The analgesic effects extended for over 48 hours. The 10 mg per day (5 mg bd) starting dose used for three weeks, before increasing to 20 mg per day, was somewhat less effective. This is the first double-blind RCT to demonstrate that methadone has a significant analgesic effect in NP, and the trial was designed to optimize the analysis of this effect. The

trial design was experimental, not pragmatic as would be applied in clinical practice. However, it does not yet answer the question of whether in NP the analgesic effects can be explained purely by opioid μ -agonist activity, or whether it is attributable to nonopioid properties of methadone (NMDA receptor antagonist activity and ability to inhibit the reuptake of biogenic amines) offering specific advantages *per se*.

There is significant danger of methadone accumulation leading to delayed onset of adverse effects with chronic administration. Fixed interval dosing conducted over several days is associated with the risk of significant morbidity and possibly mortality. The complex and highly individual pharmacokinetics of methadone require experienced clinicians to take responsibility for initiating, titrating, and monitoring methadone use.⁷³

Other medications have been tried but not subjected to RCT, and have been the subject of single case reports and uncontrolled studies. Many of these drugs may provide relief in a few patients with PHN, but the natural history of HZ pain, placebo effects, and regression to the mean must be considered in any evaluation of their effectiveness.⁷⁴

Pain management centers

These tertiary referral centers aim to reduce pain, improve function, decrease psychological distress and increase quality of life (QOL). Medications currently available are rarely associated with the complete elimination of PHN. Evidence for their beneficial effects on QOL is limited. Pharmacological treatments of patients with PHN should be considered one component of a more comprehensive treatment approach.

Further assessment with history, physical examination, and investigations and specialist consultations as necessary is usual. Education and supportive counseling is an important part of the treatment process. Other measures such as trials of transcutaneous electrical nerve stimulation (TENS), relaxation therapy, and possibly biofeedback or hypnosis may be utilized if indicated.

Cognitive behavioral therapy (CBT) assesses and addresses underlying cognitions, beliefs, and behaviors regarding pain. Occupational therapy assessment focuses on function and vocational rehabilitation. Physical therapy is used as considered appropriate. Invasive treatments may be considered if patients have not obtained relief from other treatment approaches.

Neuraxial and sympathetic nerve blocks have been evaluated for HZ and PHN in recent reviews.^{76, 77}

There is one good quality RCT with 600 patients supporting the use of **epidural administration of local anesthetic (LA) with steroid for pain control during the acute phase of PHN.**⁷⁸

It was not placebo controlled and compared epidural 0.25% bupivacaine with methylprednisolone to intravenous and oral acyclovir and prednisolone as the control group. It is good evidence none the less. The value of epidural LA or steroid alone for HZ still needs to be examined.⁷⁷

Evidence for the use of sympathetic blocks with LA is supported by one RCT⁷⁹ with limited sample size and needs validation in a larger RCT.⁷⁷

The use of intrathecal (IT) LA with steroid for PHN is supported by two RCTs.^{80, 81} The first compared IT 2% lignocaine with methylprednisolone and epidural administration and showed a clear benefit with IT use. Evaluation of treatment effect¹⁵ showed that one out of two patients (NNT = 2 [95% CI, 1.0–2.0]) will benefit from IT LA + steroid. They also found no evidence for adhesive arachnoiditis from IT administration of steroids by monitoring cerebrospinal fluid levels of interleukins.⁸⁰

The second study compared IT 2% lignocaine with methylprednisolone, IT 2% lignocaine alone, and no injection as the control for PHN.⁸¹ It found significant improvement in pain of PHN (after one year) and no evidence for adhesive arachnoiditis in 89 patients treated with LA with steroid (3 ml of 2% lignocaine with 60 mg of methylprednisolone) by the IT route every week for four weeks and followed for up to two years. This group was compared with patients treated with IT LA alone (3 ml of 2% lignocaine; n = 91) or no injections (control; n = 90). This trial

showed that 91% (81 of 89) of patients treated with IT LA with steroid showed more than 50% pain relief at the end of the study ($P < .001$) compared with 15% (14 of 91) treated with IT LA alone and 4% (4 of 90) without any intervention. Evaluation of treatment effect¹⁵ showed that one in two patients will benefit from IT LA with steroid (NNT = 2 [95% CI, 1.1–1.3]) and one in 10 patients will benefit from IT LA alone (NNT = 10 [95% CI, 5.1–41.8]) compared with controls. This study offers additional support for combining steroid with LA by the IT route. No similar comparison study could be found for epidural administration of LA and steroids, either for HZ or PHN.⁷⁷

Both these studies used methylprednisolone with propylene glycol as preservative. Although propylene glycol has been shown to be neurotoxic in animal models,⁸² no evidence for similar effects was seen at the concentration of propylene glycol used in these studies. The levels of interleukins in the cerebrospinal fluid decreased more than 50% in the group treated with IT steroid and the authors attributed this to the beneficial effect of steroid.^{80, 81} The risk of arachnoiditis after IT steroids continues to be a controversial issue at this time.⁸²

Evidence for use of epidural LA with steroid and sympathetic blocks for PHN is supported by nonrandomized trials only. Hence, they may be considered useful but lack good quality RCTs to date.⁷⁷

In general, evidence for use of nerve blocks (epidural⁷⁸ and sympathetic blocks⁷⁹) in the acute phase of HZ in the prevention of PHN appears to be strong when the blocks were administered within two months of acute HZ onset, based on the two RCTs.^{78, 79} However, this aspect needs to be directly evaluated for different injectates (LA, steroid, LA with steroid) administered through each mode (epidural, IT, sympathetic block) of drug delivery.⁷⁷

Spinal cord stimulation (SCS) can be effective and offers a therapeutic option for pharmacological non-responders with PHN, acute HZ pain, and other NP conditions. Effects are dependant on anatomically intact pathways.

Antidromic activation of dorsal col-

umn and root fibers may induce analgesic mechanisms via pre- and post-synaptic inhibition of the afferent barrage from injured peripheral neurons via GABA-ergic interneurons, and suppression of sympathetic overdrive.

The aim is to get ideal distribution of paresthesia over the painful area and affected neuronal structures. This can be difficult in PHN as the pain may expand into surrounding unaffected regions. With careful placement of leads under verbal communication and optimization of settings, regular reviews every three months, immediate revision if lead migration occurs, and good patient selection, benefit has been demonstrated in more than 80% of patients in one case series.⁸³

General and non-pharmacological measures

Because allodynia is common in PHN, decreased stimulation to the periphery may be valuable in reducing symptoms. Natural fiber clothing is said to be preferable to artificial fibers. A protective layer between the skin and provoking stimuli may be helpful. Cling film, cut to size and shape, or a layer of "plastic skin" may be applied intermittently.

Transcutaneous electrical nerve stimulation (TENS) is occasionally helpful in established PHN.⁸⁴ One study reported no benefit in a series of 17 patients.⁸⁵ Ultrasound had a poor record in a few small series of patients with PHN.^{86, 87}

Acupuncture seems to provide little benefit in PHN,⁸⁸ although early treatment may be more effective.⁸⁹ Cold pack application can provide short-term relief and may be worth trying. A small packet of frozen peas can be molded to the needed shape.

Topical agents such as capsaicin may have a minor role in the treatment of patients with PHN. Capsaicin, the active ingredient in chili peppers, opens a heat-activated ion channel (vanilloid receptor subtype 1) modulating substance P in peripheral axon terminals. It is occasionally effective in patients who tolerate its initial burning effects. But compliance with this treatment is low because of at times intense burning after application, which may lessen with time, however.⁹⁰

Post-herpetic neuralgia

There are two systematic reviews examining the use of capsaicin for PHN. Volmink et al. reported that the 0.075% preparation of capsaicin provided a statistically significant benefit.⁹¹ However, McQuay and Moore found that there was no evidence of significant improvement following capsaicin treatment in patients with PHN.⁵⁰

In some countries, a 0.025% preparation of capsaicin has recently become available. Some patients reportedly find this preparation helpful and it may be better tolerated. Placebo-controlled studies of capsaicin are problematic because of the difficulty in blinding due to the burning sensation with active treatment.

This is of less relevance at present as topical capsaicin has been unavailable in Australia for some time.

Nonsteroidal anti-inflammatory drug (NSAID) creams have been investigated in several studies and may help some patients with PHN, but the evidence is inconclusive.⁹²⁻⁹⁴

Limited studies of traditional suspensions of aspirin in chloroform, ether, or acetone have been reported. There is doubt regarding the extent of clinical benefit of these treatments, and concern regarding the safety and stability of the mixtures. They may help some patients but are not recommended as first-line treatments.⁹⁵⁻⁹⁸

Prevention of post-herpetic neuralgia

PHN can be refractory to first- and second-line treatments and also to all other therapies. Prevention is a very important goal. Older age, female sex, prodromal dermatomal pain, greater acute pain during HZ, and greater rash severity have been identified by independent investigators as risk factors in HZ for developing PHN.^{10, 99, 100}

There is greater risk of PHN in patients with more severe acute HZ infection accompanied by greater neural damage. This neural damage in patients with HZ contributes to the development of PHN, and risk of PHN is reduced by reducing the severity of the HZ infection. Timely treatment with the antiviral agents valacyclovir, famciclovir, or acyclovir by inhibiting viral replication attenuate the severity of the acute HZ infection, the duration

of viral shedding is decreased, rash healing is hastened, and the severity and duration of acute pain is reduced. The results of RCTs and meta-analyses have demonstrated that antiviral therapy in HZ significantly reduces the risk of prolonged pain.^{3-5, 101-104}

There is strong support for the use of antiviral agents in the treatment of HZ. Antiviral therapy has been recommended in several recently published literature reviews and treatment guidelines for patients with HZ, especially those who are older, have moderate or severe rash, have moderate or severe pain, or have ophthalmic involvement.¹ It needs to be initiated as early as possible, and ideally within the first 72 hours of the onset of HZ rash which is the peak time of viral replication. It may be of value if the vesicular rash is still erupting.

Reduction in the risk of PHN with antiviral therapy in patients with HZ is both clinically and statistically significant, but it does not prevent PHN in all patients. Almost 20% of patients aged over 50 years of age continue to have pain six months after rash onset, despite treatment with famciclovir or valacyclovir beginning within 72 hours of the onset of HZ rash.³⁻⁵

The results of a number of studies to date that have examined the long-term benefits of the use of corticosteroids and TCAs in patients with HZ are either equivocal or in need of replication. Use of antiviral agents,^{3-5, 101-104} corticosteroids,^{105,106} TCAs,¹⁰⁷ opioid analgesics, neuraxial and sympathetic blocks,⁷⁶⁻⁷⁹ and even SCS⁸³ in intractable cases of HZ is still recommended as needed in the acute stages to control acute HZ pain as it is a major risk factor for progression to PHN.⁹⁹⁻¹⁰⁸ Obviously pain relief should be sought on humanitarian grounds.

There are compelling reasons to predict that combining antiviral therapy with effective relief of acute HZ pain will further lessen the risk of PHN beyond that achieved by antiviral therapy alone. The basis for this hypothesis is provided by the very close relationship between acute pain severity and PHN and by research on the pathophysiological mechanisms of PHN.^{44, 99, 100,}

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Prevention of herpes zoster

Another possibility for the prevention of PHN is with varicella vaccine to prevent varicella and thus HZ in the first place. Live, attenuated varicella vaccine is effective in protecting against varicella and its complications.¹⁰⁹

Vaccine uptake in the US for children aged 19-35 months in 2001 was 76%, and varicella epidemic curves were significantly reduced where the vaccine was accepted.^{110, 111}

The incidence of HZ and PHN may significantly decrease as vaccinated children become older adults and as adults who are latently infected with wild-type varicella-zoster virus (VZV) die. The vaccine virus appears to be less likely to establish latency and reactivate than wild-type VZV. This is supported by observations that HZ was less common among leukemic children vaccinated than among leukemic children with a past history of wild-type varicella.¹¹²

Some investigators are concerned that the incidence of HZ and PHN could paradoxically increase among individuals latently infected with wild-type VZV as the incidence of varicella decreases.¹¹³ A decrease in the incidence of varicella will reduce population exposure to VZV, prevent subsequent immune boosting to VZV, and increase the risk of VZV reactivation.¹¹⁴

Most individuals are latently infected with wild-type VZV and at risk for HZ. A basic epidemiological feature of HZ is the significant increase in incidence with increasing age.⁹⁹

The most notable increase in HZ incidence is in the 50-60-year age group, related to decline in VZV-specific cell-mediated immunity. Use of live, attenuated VZV vaccine in older adults not previously afflicted by HZ led to increased mean anti-VZV antibody levels and increased VZV-specific cell-mediated immunity.¹¹⁵

HZ might be prevented or minimized by use of VZV vaccine in older adults.¹¹⁶

If this reduces the incidence or severity of HZ, reduction in the incidence or severity of PHN is expected. Ongoing trials will reveal whether immunization of older adults with VZV vaccine is efficacious in this regard.¹¹⁷

If so, and if vaccine use among older

adults is recommended and accepted, PHN may hopefully become a rarity.

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A systematic review of the literature of low-level laser therapy (LLLT) in the management of neck pain*

Roberta T Chow,^a General Practitioner, Castle Hill Medical Centre, NSW; Les Barnsley, Associate Professor, Faculty of Medicine, University of Sydney and Department of Rheumatology, Concord Repatriation General Hospital, NSW

Abstract

Background and Objective. Low-level laser therapy (LLLT) is widely used in the treatment of musculoskeletal pain. However, there is controversy over its true efficacy. We aimed to determine the efficacy of LLLT in the treatment of neck pain through systematically reviewing the literature.

Study Design/Materials and Methods. A search of computerized bibliographic databases covering medicine, physiotherapy, allied health, complementary medicine and biological sciences was undertaken from date of inception until February 2004 for randomized controlled trials of LLLT for neck pain. A comprehensive list of search terms was applied and explicit inclusion criteria were developed *a priori*. Twenty studies were identified, five of which met the inclusion criteria.

Results. Significant positive effects were reported in four of five trials in which infrared wavelengths ($\lambda = 780, 810-830, 904, 1064 \text{ nm}$) were used. Heterogeneity in outcome measures, results reporting, doses and laser parameters precluded formal meta-analysis. Effect sizes could be calculated for only two of the studies.

Conclusion. This review provides limited evidence from one RCT for the use of infrared laser for the treatment of acute neck pain ($n = 71$) and chronic neck pain, from four RCTs ($n = 202$). Larger studies are required to confirm the positive findings and determine the most effective laser parameters, sites, and modes of application.

Introduction

Despite receiving less attention than low back pain, neck pain is a highly prevalent condition, with cross-sectional studies reporting that 10-24% of

the population are troubled by neck pain at any one time.¹⁻⁴ Such frequent morbidity incurs significant costs to the community and to the individual. In the Netherlands for example, total cost of neck pain management was estimated to be \$US686 million in 1996.⁵ Standard general-practitioner-initiated treatment includes simple analgesics, anti-inflammatory medications or referral to a physiotherapist.^{6,7} Non-invasive treatments for neck pain lack a strong evidence base.^{8,9}

A potential non-invasive treatment for neck pain is low level laser therapy (LLLT). The term LLLT encompasses a heterogeneous group of applications varying from local point treatment to scanning techniques covering large areas. The putative effects of LLLT results from the photochemical and photophysical effects of light occurring with less than a 0.5°C increase in temperature of the exposed tissue.¹⁰ Output power of "low-level" lasers varies from 1 mW to 500 mW in the continuous mode, with considerably higher peak powers when pulsed. Wavelengths used extend from the visible ($\lambda = 400 \text{ nm}$) to the infrared ($\lambda = 1064 \text{ nm}$) end of the spectrum. LLLT lasers are grouped into Classes I, II, IIIa, and IIIb according to international standards. Class IV lasers, which produce their effects by heating (that is, thermic lasers) are not considered further in this review.

Extant systematic reviews of physical medicine modalities which include an evaluation of LLLT have been hampered by using restricted search terms and relatively narrow database searches, potentially leading to some trials being missed.^{9,11} Moreover, studies of related but different techniques such as laser acupuncture have been "lumped" with trials of LLLT, risking

distortion of true effects. Similarly, some reviews have included studies with a crossover design or the use of the contralateral body side as a control that may have compromised their outcomes. These latter two reviews reported no positive effect of LLLT. However, the heterogeneity of the small number of included trials was recognized as a confounding factor in assessing laser therapy.

Systematic reviews of LLLT in which trials of neck pain were included with other painful conditions are confounded by a different set of factors. "Non-specific" neck pain was lumped together with systemic inflammatory conditions; for example, rheumatoid arthritis and/or chronic pain syndromes, such as chronic orofacial pain, though the pathophysiology of these conditions is different. Multiple laser parameters were lumped together in the review of these diverse conditions,¹² and it was concluded that LLLT had no effect on musculoskeletal pain. A similar review, incorporating a subgroup analysis based on wavelength and doses used, reported that laser therapy had a "substantial specific therapeutic effect" in rheumatoid arthritis, post-traumatic joint disorders, and myofascial pain.¹³

Factors affecting conclusions of reviews of LLLT have been addressed in two reviews.^{14,15} Their analyses incorporated careful consideration of technical parameters, and noted that adherence to accepted dose, wavelength and other application issues, based on accepted published parameters, increased the likelihood of a positive outcome.

An alternative approach to reviewing LLLT literature has been to select a wavelength for review, thereby limiting the heterogeneity of parameters.¹⁶ This

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review included wound healing studies in a review of musculoskeletal disorders but nevertheless concluded that there was some evidence for efficacy in knee and myofascial pain.

Mindful of the issues in existing reviews, and with a particular focus on the common clinical problem of neck pain, we chose to review the efficacy of LLLT in the treatment of non-specific neck pain. A secondary aim was to determine whether there were any specific laser parameters or techniques of application that were more likely to yield a positive outcome.

Methods

Criteria for considering studies for this review

A priori inclusion/exclusion criteria were developed based on methodological criteria and a current understanding of the principles of application of LLLT. Only prospective randomized controlled trials of LLLT were included.

Inclusion criteria

Inclusion in this review was restricted to trials with adult participants over 16 years of age suffering from acute or chronic mechanical (non-specific) neck pain (including conditions described

variously as "myofascial pain," "trigger points," or "localised fibromyalgia"). Trials had to be randomized controlled studies of LLLT of any wavelength which reported some measure of neck pain as an outcome. Abstracts of randomized, controlled trials were also included if there were sufficient data for analysis.

Exclusion criteria

Publications in languages other than English were excluded. Laser acupuncture studies in which acupuncture points were stimulated by laser were excluded because the principles of laser acupuncture differ from those of LLLT. Studies which used a crossover design were excluded because of the potential for a cumulative systemic effect of LLLT.¹⁷ Similarly any studies using the contralateral body side as the control were excluded, as there is experimental evidence that LLLT may have effects distant from the treated area.¹⁸

Conditions excluded

Systemic inflammatory conditions, such as rheumatoid arthritis, were excluded, as the natural history and pathophysiology of these conditions differs from those of mechanical neck pain. Trials treating widespread pain, such as fibromyalgia were excluded *a*

priori as these conditions are thought to reflect generalized alteration in nociception.

Search strategy

A comprehensive list of currently used and formerly used synonyms for athermic lasers was tabulated from all the available reviews of LLLT, in order to undertake as broad a keyword search as possible (Table 1).

The keyword list for neck pain and conditions associated with neck pain was generated, then combined with the LLLT synonyms listed in Table 1: neck pain/strain, cervical pain/strain/syndrome, cervical spondylosis/itis, cervicobrachial (pain/disorder/syndrome), myofascial (pain/disorder/syndrome), trigger points, fibromyalgia, whiplash, WAD (whiplash associated disorder), osteoarthritis/arthritis, zygapophysial joints, za joints, facet joints.

Computerized bibliographic databases were searched using these keywords, without language restriction, from 1966 or the earliest year available (depending on the database searched) to February 2004 (Table 2). Additional references were sought from consulting experts in the field and hand-searching reference lists of retrieved

Low-Level Laser Therapy, (Ir)radiation, Treatment
Low-Energy Laser Therapy, (Ir)radiation, Treatment
Low Reactive-Level Laser Therapy, (Ir)radiation, Treatment
Low-Intensity Laser Therapy, (Ir)radiation, Treatment
Low-Incident Laser Therapy, (Ir)radiation, Treatment
Low-Energy Photon Therapy, (Ir)radiation, Treatment
Low-Output Laser Therapy, Low Output Laser
LLLT, LILT, LEPT, LETT, LILI, LELI, LPLI,
Infrared Laser Therapy, (Ir)radiation, Treatment
Diode Laser Therapy, (Ir)radiation, Treatment
Semiconductor
Biostimulation
Photobioactivation
Photobiomodulation
Photobiostimulation
Laser therapy
Light Therapy
Phototherapy
Soft or Cold or Mid Laser Therapy (Ir)radiation, Treatment
Narrow band light therapy
Visible Laser Therapy
904nm, 830nm, 632nm, 1064nm, GaAs, GaAlAs, HeNe, defocussed CO₂

Table 1. Keywords for search strategy

Medline (1966-2004)
Premedline (2004)
Embase (1974-2004)
Cinahl (1982-2004),
Biological Abstracts (1984-2004)
Cochrane Database of Systematic Reviews (CDSR) (2004 update)
Cochrane Central Register of Controlled Trials (CCTR) (2004 update)
ACP Journal Club (2004 update)
Database of Abstracts of Review of Effects (DARE) (2004 update)
PEDro (earliest ref 1929)
Science Citation Index (1980-2004)
BIOSIS (1980-2004)
AMED (1985-2004)

Table 2. Databases searched

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and known articles and of appropriate textbooks.

Although resources did not permit the inclusion of trials published in languages other than English, the search strategy was not limited to the English language only, as it was intended to identify trials which could be translated at a later stage, should resources become available.

Assessment of methodology

It was decided, *a priori*, to assess methodological quality using the Jadad criteria.¹⁹ Following identification of trials potentially suitable for inclusion, the methods sections of all identified trials, were reviewed independently by the authors.

Assessment of technical aspects of the trials

A data extraction form was developed for collation of laser parameters used, methods of application and treatment protocols from each of the studies to assess the heterogeneity of the trials (Table 3). Authors scored the technical aspects of the laser application, with a possible maximum score of

18 if all the parameters were reported or could be calculated, permitting replication of the trial.

Differences or difficulties with inclusion of reviews, trials and data extraction were resolved by consensus.

Outcome measures

The primary outcome measure sought was an observed change in pain scores before and after treatment. Other clinically relevant outcomes of pain measurement such as range of movement, function/disability measures, quality of life, and adverse effects were recorded.

Results

We identified 20 potentially eligible randomized controlled trials using the *a priori* inclusion and exclusion criteria (Table 4). Of these, five studies fulfilled the criteria for review (Table 5). The 15 excluded trials and their reason for exclusion are detailed in Table 6.

Methodological quality

Four of the five studies were randomized controlled studies of satisfactory quality (Jadad score >3/5)

(Table 5). One study was single-blind and of low methodological quality (Jadad score 2/5).

Summary of included trials

Soriano et al studied 71 patients with acute neck pain randomized to 10 treatments using 904 nm laser (average power 40 mW at 10,000 Hz) with an energy density (ED) of 4 J/cm² or sham laser.²⁰ A significant improvement was reported at the completion of treatment, and six months later, in the treated group.

Toya et al compared neck pain intensity on a five-point scale in a double-blind RCT of 39 patients. Each received a single treatment session with active 830 nm laser (60 mW, cw) or sham laser. Those receiving the LLLT had a statistically significant improvement in their pain compared with sham laser.²¹

Ozdemir et al studied 60 patients with chronic neck pain associated with cervical osteoarthritis.²² Patients were randomized to 10 treatments with 830 nm laser (50 mW, cw), or a sham laser. The treated group had a significant improvement in neck pain, disability

Laser beam parameters

Output power (W or mW)
Power at skin surface (W or mW)
Mode (continuous wave or pulsed)
(If pulsed: pulsing parameters: frequency [Hz] & duration of pulse [nsec])
Lasing medium (for example, diode, gas)
Wavelength (nm)
Type of probe (single, multi-head)
Calibration of laser device at appropriate points in the study

Dose parameters

Energy density (J/cm²)
Power density (W/cm² or mW/cm²)
Area treated or spot size (cm² or mm²)
Duration of treatment (seconds)
Number of points treated
Number of joules/point (J)
Total number of joules per treatment

Application technique

Mode of application (contact, scanning)
Site of application

Treatment schedule

Frequency of treatment (daily, etc)
Number of treatments

Trigger points and myofascial pain

Lewith et al 1981 (37) - Local trigger points
Snyder-Mackler et al 1986 (38) - Trigger points
Waylonis et al 1988 (39) - Chronic myofascial pain
Airaksinen et al 1989 (18) - Trigger points
Ceccherelli et al 1989 (40) - Cervical myofascial pain
Snyder-Mackler et al 1989 (41) - Trigger points
Thorsen et al 1991 (42) - Chronic myofascial pain
Thorsen et al 1992 (43) - Myofascial pain in the neck and shoulder girdle
Laakso et al 1994 (36) - Myofascial trigger points
Laakso et al 1997 (24) - Myofascial trigger points
Logdberg-Andersson et al 1997 (31) - Tendonitis and myofascial pains
Hakguder et al 2003 (23) - Myofascial pain syndrome

Neck or chronic pain (including neck pain)

Gallacchi et al 1981 (44) - Cervical syndrome
Toya et al 1994 (21) - "Selected pain groups" - "Cervical pain"
Soriano et al 1996 (20) - Acute cervical pain
Fukuuchi et al 1998 (45) - Chronic pain
Slattery et al 2002 (46) - Chronic neck pain (and shoulder pain)
Seidel et al 2002 (47) - Chronic cervical syndrome

Cervical osteoarthritis

Taverna et al 1990 (48) - Cervical osteoarthritis
Ozdemir et al 2001 (22) - Cervical osteoarthritis

Table 4. Retrieved studies of randomized controlled trials of laser therapy for neck pain

Table 3. Laser parameters, dose, and mode of application in the data extraction form

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and functional outcome measures.

Hakguder et al. reported significant benefits of exercise with LLLT to a single trigger point in cervical muscles compared to exercise alone, as the control, in a controlled trial of 62 chronic neck pain patients using 780 nm laser (10 mW, cw) with an ED of 5 J/cm².²³

Laakso et al reported a positive post-treatment benefit for laser on cervical trigger points²⁴ when compared with sham laser. She studied 41 patients divided into six groups: 820 nm laser at two doses, visible laser at two doses, and two sham laser control groups. However, these outcomes were based on within-group analyses, and as no comparison was made between groups, the study was categorized as inconclusive.

Size of effect

The heterogeneity of outcomes and measures reported in these trials precluded formal pooling of data. For example, two studies reported only the level of the statistical significance (p value).^{21,20} For the remaining three studies and to establish a unitary but dimensionless measure of outcome, we attempted to calculate effect sizes. For comparison between intervention and control group, small effect size is defined by a value > 0.2-0.4, moderate if > 0.5-0.7 and large if > 0.8.²⁵

An effect size was able to be computed from the available data for the outcome measures in two of the trials.^{25,23} In the study by Ozdemir et al and Hakguder et al, effect size for pain reduction was large, 3.9 and 1.8, respectively.

As no between-group data were available for analysis in one trial, no effect size calculation could be performed.²⁴

In the study of acute pain by Soriano et al, a self-reported improvement of 60% or more was defined as an effective treatment, with 94.59% in the treated group versus 38.24% in the placebo group achieving this outcome.²⁰ Complete pain relief was achieved in 67.59% of patients in the LLLT active group and 17.65% in the placebo group.

In the report by Toya et al, the treatment of chronic pain with a single session of LLLT achieved effective pain relief (defined as "excellent", "good" or "fair" response to treatment) in 82% of patients compared with 42%

in the placebo group.²¹

Technical assessment

Scores of the technical quality of the trials varied from 12 to 17, using the evaluation criteria listed and developed from those described by other authors.¹⁰ The laser parameters used in each study are detailed in Table 5.

Side effects

The presence and frequency of side effects was sought in three trials.^{20,21,24} No side effects were found by Soriano or Toya, but Laakso et al reported symptoms occurring only in the active group (nausea, faintness, tiredness, shakiness, euphoria, weakness, stomach distension, and increased pain). The occurrence of side effects was not reported in two trials.^{22,23}

Discussion

This is the first systematic review, that we are aware of, to specifically evaluate LLLT in the management of neck pain. We reviewed the literature with clearly defined *a priori* inclusion and exclusion criteria and found four positive trials and one equivocal study for evaluation in our review.

We found limited evidence for a short-term benefit of 820-830 nm laser in two of the trials for chronic neck pain for up to three weeks.^{21,22} There was limited evidence for a beneficial effect of 904 nm laser in acute neck pain, with limited evidence for reduction in recurrence at six months follow-up in a single trial.²⁰ There was limited evidence for the increased efficacy of exercise when combined with 780 nm laser in patients with chronic neck pain associated with trigger points.²³

The efficacy of a visible wavelength ($\lambda = 670$ nm) was studied in a single trial. Although reported positive at an energy density of 1 J/cm² ($p < 0.02$) (but not 5 J/cm²) as between-group analysis data was not available, the outcomes of this trial are regarded as equivocal, and efficacy of visible laser in neck pain is not established.²⁴ Side effects were reported in only one of the three studies in which they were sought, and occurred in the laser group only.

We attempted to address the second aim of our review, that is, to identify optimal parameters of treatment by

adopting the approach taken by Bjordal et al. in which the authors assess outcomes of studies based on whether or not an effective dose of laser was administered to an appropriate site, using available published data.¹⁴ In doing so we identified the parameters of the study and quantified the technical aspects of the study, rating each study for inclusion of specific data.

The most consistent finding in our review was the use of infrared lasers in all five trials. However, even within the infrared spectrum three different wavelengths ($\lambda = 780, 820-830, 904$ nm) were used. Infrared laser penetrates more deeply than visible wavelengths enabling higher doses to be delivered to deep-seated structures which are potential sources of pain.²⁶ The specific infrared wavelength may be less important than the ability to reach a depth at a dose which is physiologically more relevant.

Marked heterogeneity between the trials in all other parameters was identified. The range of average output power varied from 5 mW to 60 mW (a factor of 12x), in pulsed and continuous modes. The reported energy density (ED) varied from 0.9 J/cm² to 1800 J/cm², a variation by a factor of 2000x. This represents a huge variation in dose for any biological system, but is likely to represent different conventions in ED calculation as well as the reporting of the total ED administered during a treatment, rather than at any one point. In four of the trials a course of treatment, between five and 10 sessions on a daily or second daily basis, was administered. Treatment sites varied from a single trigger point to 12 arbitrary points over the neck, with one trial not identifying the sites treated, making it difficult to identify a common anatomical or pathophysiological principle on which to construct a rationale for treatment.

Given such heterogeneity, two issues emerge. Firstly it is difficult to advocate a "unifying theory" of plausible biological mechanisms with such diversity of parameters and protocols. Secondly, it is meaningless to perform any pooling of data such as a meta-analysis and impossible to identify optimal parameters, using the approach by Bjordal and collaborators.

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Author & year	N	Design	Incl. criteria	Jadad score	Tech score	Control	Sites treated	λ (nm)	Power (mW)	Energy density	No. & frequency of Rxs	Primary outcome measure	p values
Toya et al 1994 (21)	39	DB RCT	Cervical pain complex	5/5	12/18	Sham	Site not specified	830	60 mW (cw)	900-1800 J/cm ²	1 Rx	Graded subjective assessment "Exacerbation to excellent"	p<0.01
Soriano et al 1996 (20)	71	DB RCT	Acute neck pain	4/5	14/18	Sham	"painful area": laser applied using 2 cm ² grid	904	Average power: 40 mW (pulsed)	4 J/cm ²	10 Rxs daily (Mon-Fri) for 2 weeks	Graded subjective assessment: "bad" to "excellent"	p<0.0019 (after treatmt.) p<0.05 (@6 mos)
Laakso et al 1997 (24)	41	DB RCT	Neck pain with TPs in neck	3/5	13/18	Sham	3 most painful trigger points	820 670	25 mW (pulsed) 10 mW (pulsed) for each wavelength	1 J/cm ² & 5 J/cm ²	5 Rxs, every second day over two weeks	VAS	Inconclusive
Ozdemir et al 2001 (22)	60	DB RCT	Neck pain rel. to neck OA	3/5	14/18	Sham	6 arbitrary points over neck muscles	830	50 mW (cw)	0.9 J/cm ² (reported) 95.5 J/cm ² (calc.)	10 Rxs daily for 10 consec. days	VAS	p<0.001
Hakguder et al 2003 (23)	60	SB RCT	Neck pain with 1 trigger point	2/5	17/18	Exercise with & LLLT & exercise alone	1 active trigger pt. in levator scapulae or trapezius	780	5 mW (cw)	5 J/cm ²	10 sessions daily for 10 consec. days	VAS	p<0.001 for all outcome measures

Table 5. Laser parameters, outcome measures, and side effects.

Author and Year	Reason for exclusion
Gallacchi et al - 1981 (44)	German/laser acupuncture
Lewith et al - 1981 (37)	Trial of heat treatment generated by light, that is, not athermic lasers
Snyder-Mackler et al (38)	Did not use pain outcome measure
Waylonis et al - 1988 (39)	Laser acupuncture
Airaksinen et al - 1989 (18)	Crossover design
Ceccherelli et al - 1989 (40)	Laser acupuncture
Snyder-Mackler et al - 1989 (41)	Cannot separate out neck pain data
Taverna et al - 1990 (48)	Italian
Thorsen et al - 1991 (42)	Danish
Thorsen et al - 1992 (43)	Crossover design
Laakso et al - 1994 (36)	Did not use pain outcome measure
Logdberg-Andersson et al - 1997 (31)	Cannot separate out neck pain data
Fukuuchi et al - 1998 (45)	Cannot separate out neck pain data
Slattery et al 2002 - (46)	Abstract only - no data available
Seidel et al 2002 - (47)	German/laser acupuncture

Table 6. Excluded trials and reason for exclusion.

While the absence of negative trials is unusual and might be criticized for publication bias,²⁷ we do not believe that our search strategy and rules for inclusion were flawed. Moreover, the methodology of four out of the five included trials was satisfactory.

The tendency to positive findings of our review are at odds with two other systematic reviews where LLLT for neck pain showed no effect when

evaluated within a study of other modalities.^{9,11} Differences emerge on several fronts when our review is compared with these reviews. We rigorously compiled an extensive list of synonyms used for LLLT over the last 20 years searching a variety of databases including those within complementary medicine. We included conditions, such as myofascial pain likely to be associated with neck pain,

though not explicitly stated as such, in the keyword search and included subgroups of neck pain in chronic pain studies. These strategies provided a list of 20 trials, compared with the reviews using less rigorous or inclusive strategies which yielded less than five LLLT studies. In addition, our exclusion criteria were developed on the basis of expert knowledge of the principles of LLLT trial design, so that trials

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included in other reviews were excluded from ours. Though the value of technical expertise within a review group has been subject to some criticism,²⁸ it is acknowledged that content experts in a field can bring important perspectives to a review.²⁹

Crossover design and laser acupuncture, in particular, are exclusion criteria not applied in other reviews. A cumulative, and therefore delayed effect of laser is known to occur,¹⁷ most probably associated with alteration of gene expression.³⁰ If crossover occurs within that as-yet-unknown period of time then a "carry-over" effect may mask a positive outcome within a study.³¹ Laser acupuncture trials are also excluded, as the principles of laser acupuncture differ significantly from LLLT.³² In the former, laser is used to stimulate nominated points consistent in anatomical location from patient to patient, with specific effects mediated by activation of ascending and descending spinal tracts. In contrast LLLT relies on the direct effect of light on tissue, with dose-dependent effects, which may be different from patient to patient. It is therefore more appropriate for trials of laser acupuncture to be included in a review of acupuncture trials rather than LLLT trials.

While acknowledging the possible bias of excluding trials in foreign languages,^{33,34} resources did not permit translation and hence inclusion, of primary studies into English. However, three of the four trials excluded on the basis of language would have been excluded on other criteria. The fourth trial which was potentially suitable for inclusion had positive outcomes. We do not therefore believe that language exclusion would have materially altered our interpretation of results.

We attempted to correlate the methodological findings of the included studies with the technical score using this as an estimate of trial reproducibility. In doing so we suggest that the inclusion of methodologically sound but technically poor trials results in flawed conclusions of reviews.³⁵ Moreover, we found in our review that methodological and technical assessment were not congruent as the trial with the highest technical score (17/18) had the lowest methodological score (2/

5).²³ Conversely, the trial with the highest methodological score (5/5), had the lowest technical score (13/18).²¹ The lack of congruence between methodology and technical description confounds the assessment of LLLT trials.

Because of the heterogeneity of trials we are unable to answer the question as to what is the optimal ED, or the ideal anatomical site for LLLT in the treatment of neck pain. This is at odds with Bjordal et al.'s findings where it was possible to identify an optimal treatment range of EDs for a specific pathology, based on laboratory estimations of an effect.¹⁴ Lack of heterogeneity of EDs applied or specific pathophysiology of neck pain identified suggests the possibility that other mechanisms of action of laser, such as systemic effects³⁶ may be acting to relieve pain.

Conclusion

Notwithstanding the heterogeneity of the studies identified within this review, LLLT with infrared wavelengths appears to be efficacious for the treatment of neck pain with limited evidence being provided from the reviewed trials. Details of the most effective energy densities, sites of treatment, and mechanisms of action remain unresolved and further research is warranted to address these questions. The treatment is relatively simple to apply and side effects appear mild and transitory. The reduction in pain levels was modest in patients with chronic neck pain, and although limited by short-term follow up were supported by positive functional changes. Further empirical research into LLLT, particularly larger studies with long-term follow up, would seem to be justified on the basis of this review.

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^a rtchow@bigpond.net.au

Piriformis syndrome – definitions, reproducibility, and validity of diagnostic procedures and results of efficacy trials*

*For the Scientific Committee of FIMM.** First author: L Remvig. Assistant authors: RM Ellis, J Patijn*

Abstract

A position statement of the FIMM Scientific Committee is presented about the piriformis syndrome. It concludes that the piriformis syndrome has yet to receive an agreed definition, although the commonest feature described is pain in the buttock without clear primary, structural pathology. The literature is reviewed and tabulated, and does not determine whether the syndrome exists as a primary entity or is secondary to disease or dysfunction elsewhere.

Introduction

The Scientific Committee of the International Federation of Manual/Musculoskeletal Medicine has reviewed knowledge of the different areas of the locomotion system, especially with respect to reproducibility/validity studies and efficacy studies. This review is based on a literature search on Pubmed based on the index words “piriformis syndrome” – and diagnosis and treatment, and on references cited in this literature. The journal *Manuelle Medizin*, and various textbooks in or-

thopedic and musculoskeletal medicine have likewise been reviewed.

Definition of the piriformis syndrome

A “syndrome” is by definition a set of frequently related symptoms, but in medical literature one often finds objective signs included in the definition of a syndrome. This is also the case with respect to the piriformis syndrome, which most often is characterized by certain concurrent symptoms and clinical signs. The syndrome was not defined either by Yeoman¹ or by Freiberg^{2,3}

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Table 1. Definition of the piriformis syndrome as mentioned in publications

	Definition
Yeoman W, 1928 (1)	No syndrome definition. Focused on inflammatory reaction in the piriformis muscle causing sciatica.
Freiberg AH, Vinke TH, 1934 (2)	No syndrome definition. Mention three indications of piriformis-caused sciatica.
Robinson DR, 1947 (4)	A type of sciatica which is due to an abnormal condition of the piriformis muscle, and which is usually traumatic in origin.
Edwards FO, 1962 (8)	A neuritis of branches of the sciatic nerve, caused by pressure of an injured or irritated piriformis muscle.
Retzlaff EW, Berry AH, et al, 1974 (9)	The piriformis muscle syndrome is the result of an injury-induced contracture of the muscle.
Pace JB, Nagle D, 1976 (13)	A clinical syndrome characterized by pain and disability.
Solheim LF, et al, 1981 (10)	A term applied to an abnormal condition of the piriformis muscle, characterized by symptoms and signs due to sciatic nerve entrapment at the greater sciatic notch.
Steiner C, et al, 1987 (7)	A clinical condition with low back pain, general muscle guarding, limited vertebral mobility, sciatic pain, sciatic paresthesia and a positive straight leg-raising maneuver.
Jankiewicz JJ, et al, 1989 (17)	A rare condition characterized by pain and paresthesias located in the buttock, often radiating to the posterior thigh.
Barton P, 1991 (18)	A little-known entity in which injury to the piriformis muscle results in buttock pain often associated with leg pain.
Fishman LM, Zybert PA, 1992 (11)	A loose cluster of symptoms arising from entrapment of one or both divisions of the sciatic nerve as they pass the sciatic notch
Silver JK, Leadbetter WB, 1998 (5) McCrary P, 2001 (24)	There is no clear consensus on what clinical presentation is characteristic. The piriformis syndrome is usually described as a cramping or aching pain in the buttock and/or hamstring.
Fishman LM, et al, 2002 (19)	Defining piriformis syndrome as a three-standard-deviation prolongation of the H-reflex in the leg placed in 90° of hip and knee flexion, adduction, and internal rotation (FAIR test).

even though they believed that the piriformis muscle was a causative factor in sciatica. Robinson⁴ has been credited with the naming of the piriformis syndrome. He described the syndrome citing six typical features that included both subjective and objective items. Since then various definitions have been published, some by description of diagnostic criteria (Table 1). However in a brief report, Silver and Leadbetter⁵ pointed out that there is no consensus on the diagnosis or treatment of the piriformis syndrome, a statement based on a review of the literature and a survey of 75 physiatrists. Broadhurst⁶ advocated another name: "the external rotator syndrome" due to lack of definitive proof of the involvement of the piriformis muscle.

Pathogenesis and pathomorphology of the piriformis syndrome

According to Yeoman,¹ the symptoms connected with sciatica could specifically be attributed to (or associated with) sacroiliac degenerative joint disease causing "peri-arthritis in the anterior sacroiliac ligament, the piriformis muscle and the adjacent radicals of the sciatic nerve". Steiner et al⁷ presumed the same pathogenesis, as they referred to a "sciatic neuritis caused by biochemical agents released from an inflamed piriformis muscle".

Other authors suggest "the possibility of the mechanical effect of pressure upon the nerve as the result of continuous spasm of the muscle".² It was hypothesized that the effect could be caused by circulatory disturbances. Several others have supported the view of a mechanical effect.⁸⁻¹²

The authors argued that an abnormal condition of the piriformis muscle is the reason for the symptoms, as the condition causes entrapment of nerve trunks and/or blood vessels thereby interfering with the function of the structures supplied by these nerves and vessels. However, they didn't provide us with basic studies documenting any nerve interference except for one study.¹¹ The other authors draw attention to the fact that pain arises in muscles during ischemia.

Robinson⁴ mentioned "a history of trauma to the sacroiliac and gluteal

region" as the first of six cardinal features of the piriformis syndrome, and had the opinion that spasm of or disease in the muscle could affect the sciatic nerve and/or the first, second, and third sacral nerve.

This is partly supported by TePoorten¹² who mentioned acute trauma as one of five etiological factors. TePoorten seems to believe that there are two pain mechanisms, one due to nerve entrapment, and the other due to a trigger point in the muscle causing referred pain.

Pace and Nagel¹³ also put forward that the symptoms are due to a deep muscle trigger point and not to an inflammatory process in the muscle, and they did not believe that there is any "discernable common causative factor in piriform muscle syndrome".

Foster¹⁴ finds numerous case reports describing "problems intrinsic to the muscle or primary piriformis" and other reports where "piriformis may be secondary to sacroiliac irritation or a mass near the sciatic notch", thus distinguishing between primary and secondary piriformis syndrome.

Diagnosing the piriformis syndrome

As there is no consensus regarding the definition of piriformis syndrome, nor the etiology, it is obvious that there are almost as many sets of diagnostic criteria as there are authors (Table 2). All types of diagnostic procedure have been used, except for auscultation. Many different descriptions have been used not only regarding the history but also regarding the performance of objective examinations and provocation tests.

Some authors mention several tests for the syndrome, but do not indicate how many they require to be positive in order to diagnose the syndrome.¹⁵

Both bone scintigraphy¹⁶ and CT/MRI-scanning^{17,18} have in single cases been able to illustrate affection of the piriformis muscle, but the H-reflex is the only paraclinical test that has been used in a clinical efficacy study in order to diagnose the syndrome.¹⁹

Only about 50% of the authors demand exclusion of lumbar and/or sacroiliac pathology in order to diagnose the syndrome (Table 2).

Reliability and validity of diagnostic tests

The more crucial aspect of these various diagnostic tests and criteria is that neither the palpatory findings nor the pain provocation procedures have been analysed with respect to reproducibility and only in one occasion have they been validated.¹⁶

Using two positive tests out of three as a criterion for the piriformis syndrome and using the delayed H-reflex as gold standard, Fishman demonstrated a rather high sensitivity and specificity (Table 3). However, these figures are highly debatable for at least four reasons:

1. The figures were based on the results from only 688 patients, despite the fact that a detailed history was recorded on 918 consecutive patients complaining of low back pain and/or sciatica, in whom 1014 lower limbs were involved. The authors did not explain why they did not include the remaining 326 patients. Altogether 665 patients had a delayed H-reflex and 339 did not.
2. The sensitivity and specificity values presented in the paper cannot be calculated from the figures given in the text.
3. The authors have used a paraclinical test – the delayed H-reflex – as gold standard without documenting the validity of the test.
4. The figures show nosographic sensitivity and specificity and not what one needs in daily practice – the positive and negative predictive value of the tests.

Efficacy studies

Through the years there have been various suggestions regarding how to treat patients with a piriformis syndrome, no matter how the syndrome was defined. Surgical release has been suggested and carried out by several authors (Table 4), and the effect of this treatment has also been taken as an indication of the pathogenesis of the syndrome – nerve entrapment.

Others have suggested and illustrated the effect of intramuscular injection with local anesthetic and corticosteroid^{13,17,16} and some have advocated for manual/osteopathic treatment,^{9,19,6}

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Table 2. Diagnostic criteria for piriformis syndrome used in the literature

History
History of trauma to the sacroiliac and gluteal region
Pain in the region of the SI joint, greater sciatic notch and piriformis muscle extending down the leg and causing difficulty in walking
Acute exacerbations of the chronic pain brought on usually by stooping or lifting and relieved by traction
Complaint of a severe pain radiating from the sacrum to the hip joint
Aching pain for one year
Low back and hip pain with pain radiating down the back of the leg
Low back pain and/or sciatica
Difficulty walking on uneven ground
Sitting intolerance
Serious sexual problems in both sexes (N. pudendus affectation)
Persistent buttock pain not caused by lumbar spine dysfunction including disc prolapse and sciatica, or by sacroiliac joint dysfunction
Pain in the buttock when the patient turns in bed from side to back
Gluteal pain radiating down in the leg
Inspection
Gluteal atrophy depending of the duration of the condition
External rotation of the affected leg ("positive piriformis sign")
Rotation of the sacrum over its oblique axis and rotoscoliosis of the lumbar vertebrae and increased lordosis
Palpation
Palpable sausage-shaped mass over the piriformis muscle markedly tender to pressure
Palpation of the piriformis tendon near the trochanter major will elicit severe pain
Tenderness and reproduction of the patient's complaints by digital pressure over the belly of the piriform muscle
Palpation of a firm piriformis with typical trigger points in the lateral and medial third of the piriformis muscle
Tenderness to palpation along the piriformis (horizontally)
Tenderness at the sciatic notch
Tenderness at the intersection of the piriformis muscle and the sciatic nerve (mechanical pressure replicates the pathogenic mechanism)
Digital palpation of the piriformis muscle for reproduction of sciatic pain
Buttock tenderness
Rectal or pelvic examination to rule out lateral pelvic wall tenderness and reproduce sciatica
Rectal examination shows a tight and painful muscle by palpation, and pain provocation by isometric abduction
Diagnostic tests
Positive Lasègue's sign
Negative Lasègue's sign
Positive supine Lasègue's sign, applied as 15° reduction in straight-leg raise on the affected side versus the unaffected side, or less than 65°
Pain and weakness on resisted abduction-external rotation of the thigh with the patient in seated position (positive Pace's sign)
Pain reproduction by resisted external rotation of the flexed hip
Pain reproduction by resisted abduction of the hip when adducted and flexed
Pain on passive internal rotation of the femur in prone position (positive Freiberg sign)
Aggravation by prolonged hip flexion, adduction and internal rotation
Absence of low back or hip findings
Having the patient lie, with the painful side up, the painful leg flexed and the knee resting on the bench, buttock pain is produced when the patient lifts and holds the knee several centimetres off the bench
Isometric abduction force of femur in seated position is reduced due to pain
Passive internal rotation of a straight leg, passive adduction at 70° hip flexion, and passive external rotation at maximum flexion is reduced due to pain
Pain at the intersection of the sciatic nerve and the piriformis muscle on flexion, adduction, and internal rotation (FAIR)
Paraclinical examinations
Prolongation of the H-reflex of at least three standard deviations (1.86 msec) by flexing, adducting, and internally rotating the leg
Transient relief of symptoms by any type of injection was accepted as confirmatory of the piriformis diagnosis

Piriformis syndrome

[illegible]

Piriformis syndrome

Table 2, continued

Exclusion criteria
Other pathological conditions of the lumbar, sacral and hip joint areas should be ruled out by examination and x-ray
True neurological deficit should never be found
Negative radiological findings
Patients with persistent buttock pain were screened for evidence of lumbar spine dysfunction including disc prolapse and sciatica, and of sacroiliac dysfunction
EMG excluded neuropathy and myopathy
The standard workup for a spinal cause of pain should be negative
Any significant findings during lumbar examination would exclude patients

Table 3: Validity studies of palpatory findings and of pain provocations tests used for diagnosing the piriformis syndrome with delayed H-reflex as gold standard (Fishman¹⁹)

	N		
	≥ 2 pos of 3	<2 pos of 3	total
H-reflex delayed ≥3 SD	468	22	490
H-reflex delayed <3 SD	69	129	198
Total	537	151	688

Sensitivity: 0.881 and specificity: 0.832

(Calculating sensitivity and specificity from the figures given in the publication (identical with the figures in the table) one reaches Sensitivity: 0.871 and Specificity: 0.854).

or for a combination of injection and manual therapy.^{7,18} However, until 2002 there were no published results of controlled studies.

Fishman et al¹⁹ conducted an interesting study comparing the effect of a single injection of lidocaine plus triamcinolone (L/T) with botulinum toxin (B) and with normal saline (S), – all injections given with EMG guidance to the myoneural junction in the piriformis muscle. Patient selection was based on symptoms and presence of the delayed H-reflex (Table 2) and the result was based on a 50% reduction in pain VAS (Table 4). The result of the study was clearly in favour of the botulinum treatment, but it was not mentioned how many of the patients became pain free.

Discussion

This review illustrates that within the scientific literature there is no clear-cut definition of the piriformis syndrome. Likewise, there is no consensus with respect to diagnostic criteria, and none of the many different clinical tests for this syndrome have been tested for reproducibility or validity. Nor, when looking in different text-

books in orthopedic or musculoskeletal medicine, can one find a clear definition of the syndrome, even though tension in the muscle²⁰ or the syndrome is mentioned.^{21, 22}

In their publication, Fishman et al¹⁹ used the delayed H-reflex as a validated test for the piriformis syndrome. However, the test was not validated in their previous paper,¹¹ but used as a gold standard for evaluating the clinical tests for the syndrome. Consequently, we have no knowledge concerning the validity of the delayed H-reflex as part of the definition of the syndrome. It is known that the H-reflex also is delayed in sciatica with clinical signs of lumbar root involvement.²³ However, is this delay increased when the patient's leg is placed in the straight leg raise? Or when it is placed in the hipflexion, adduction, and internal rotation (FAIR) position? Similarly, we do not know the H-reflex response with the leg in FAIR position in other pathological situations causing sciatica – for example, dysfunction of the sacroiliac joint, or trigger points in the gluteus medius muscle. The author did not mention how radicular syndromes were excluded, based for example on addi-

tional electromyographic investigations in their patients.

Basically, quantitative diagnostic procedures, such as electromyography and H-reflex, should first be analysed for their reproducibility (test-retest) in the same subjects at different times and subsequently be tested for validity against accepted clinical diagnostic criteria. If the reliability is high, then the new method, in this case the H-reflex, can be defined as the gold standard for the piriformis syndrome.

Presuming that the clinical tests in Fishman's article were reproducible and accepted as the diagnostic criteria for the syndrome, and that the delayed H-reflex consequently was tested against these clinical criteria, one would find that the nosographic sensitivity of the test was: 0.955 and the specificity was just 0.652, reflecting no discrimination between other syndromes in which the root and/or nerve is compressed

The predictive value of the test will probably be even lower making the test almost useless in daily practice, when examining unselected low back pain patients.

With one exception the results of the

Robinson ⁴	TePoorten ¹²	Retzlaff ⁹	Pace ¹³	Durrani ¹⁵	Jankiewicz ¹⁷	Barton ¹⁸	Broadhurst ⁶	Fishman ¹¹	Beatty ²⁵	Frölich ²⁶	Fishman ¹⁹	Fishman ¹⁶	Foster ¹⁴
+													
			+										
				+									
							+						
								+					
									+				
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various publications concerning treatment efficacy are to be regarded more as case reports and pilot studies. The result of the Fishman¹⁹ study is interesting. However, it would have been nice to see raw data of the study: What was the level of the individual pain score at entrance? How high was the pain reduction in individual patients in the groups? VAS-score as a single outcome measure is insufficient. At least a quality of life scale should have been added to the outcome measures. What were the reasons for patient dropouts? Were there any side effects of the treatments? What happened if injections were repeated twice every second week? How were the pain scores after six and 12 months?

Conclusion

First of all there is definitely a need for an international agreement of a definition of the piriformis syndrome. Having achieved that definition, studies regarding test reproducibility – clinical as well as paraclinical tests – and validity should be conducted and, based on that, one could suggest the criteria for the piriformis syndrome. When those are accepted, one should examine the effect and potential side effects of different treatments. Further research is needed to determine whether the piriformis syndrome is a clinical entity and/or it is a consequence of identifiable conditions of the lower back.

**Authors

L. Remvig, Clinic of Orthopaedic Medicine and Rehabilitation, Rigshospitalet, Copenhagen University Hospital, Denmark, Member of the Scientific Committee FIMM; R.M. Ellis, Department of Rheumatology, Southampton General Hospital, England, Member of the Scientific Committee FIMM; J. Patijn, Pain Management and Research Centre, University Hospital Maastricht, The Netherlands, Chairman of the Scientific Committee FIMM

Members of the Committee

O Airaksinen, J.v Beek, L Beyer, S Blomberg, B Buser, RM Ellis, J-Y. Maigne, R Palmer, J Patijn (chairman), L Remvig, J Vacek, R Ward

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Table 4. Piriformis syndrome, efficacy studies

	N	Treatment	Method	Effect
Freiberg AH, 1937 (3)	several cases	section of the piriformis	case report	one case described
Robinson DR, 1947 (4)	2	section of the piriformis	case report	two cases described
Pace JB, Nagel D, 1976 (13)	39	intramuscular injection with lidocaine 1% and kenalog 10 mg/cc	review of records	all had immediate and dramatic benefit and relief
Solheim LF, et al, 1981 (10)	2	the piriformis muscle sectioned at its musculotendinous junction	case report	total pain relief
Barton P, 1991 (18)	4	Management schema: 1. correction of underlying biomechanical factors 2. stretching exercises 3. steroid injections 4. surgical piriformis release	case report	four cases described
Broadhurst NA, 1991 (6)	24	Post-isometric stretching exercises: The muscle is facilitated, relaxed, and then stretched. 3-4 stretches x 2 daily, for 5-7 days	unblinded and uncontrolled	all patients indicated a significant improvement after one week; 6-month follow-up phone calls: pain resolved but returned at times
Fishman LM, Zybert PA, 1992 (11)	12	Biweekly myofascial-release technique to the lumbosacral paraspinal region, McKenzie exercises, and stretching of the external rotators	unblinded and uncontrolled	11 reported more than 60% subjective relief after 2-6 months
Fishman LM, et al, 2002 (16)	918 pts. 1014 limbs	1. injection at the initial visit with lidocaine + triamcinolone acetate (note 1) 2. concentrated physical therapy (note 2)	unblinded and uncontrolled three patient groups treated 1. H-test delayed >3SD in FAIR position; 2. $\geq 2/3$ pos. criteria; 3. where clinical suspicion is high	>50% pain improvement: 1. criteria pos + FTP: 83.1%; 2. criteria neg + FTN: 67.7%; 3. FTP: 79%; 4. FTN: 54.8% (note 3)
Foster MR, 2002 (14)	7	Surgical technique: Identification and release of piriformis with subsequently reattachment in shortened position	unblinded and uncontrolled	All 7 returned to work within 1-6 months
Fishman LM, et al, 2002 (19)	N=67 pts N=72 cases	1. botulinum toxin A 2. lidocaine + triamcinolone 3. placebo (note 4) All patients received physical therapy 2-3 times weekly for 12 weeks (note 2)	successive patients with buttock tenderness, sciatica and positive FAIR test; randomised, double-blinded and controlled study; exam. every 2nd week for 12 wks	50% pain improvement on VAS: 1. Botox (n=21): 65% 2. L/T (n=31) 32% 3. Placebo (n=15) 6% B versus P: p=0.001 B versus L/T: =0.044

Note 1. Injection therapy:

Injection with 1.5 ml of 2% lidocaine + 0.5 ml triamcinolone acetonide (20 mg) at a point one-third the distance from the greater trochanter to the area of maximum tenderness in the buttock at a depth of approximately 3-5 cm.

Note 2. Physical therapy protocol:

Place the patient in contralateral decubitus and FAIR position.

1. Ultrasound 2.0-2.5 W/cm² applied in broad strokes longitudinally along the piriformis muscle from the conjoint tendon to the lateral edge of the greater sciatic foramen for 10-14 minutes.
2. Wipe off ultrasound gel.
3. Hot packs or cold spray at the same location for 10 minutes.
4. Stretch the piriformis muscle for 10-14 minutes by applying manual pressure to the muscle's inferior border, being careful not to press downward, rather directing pressure tangentially towards the ipsilateral shoulder.
5. Myofascial release at lumbosacral paraspinal muscles.
6. McKenzie exercises.
7. Use lumbosacral corset when treating patient in the FAIR position.

Duration: 2-3 times weekly for 1-3 months.

Note 3. FTP = FAIR test positive, FTN = FAIR test negative.

Note 4. Injections were administered under EMG guidance.

Therapy for low back pain

Dr David Squirrell, Director General Practice Teaching and Training and ED Consultant, Noarlunga Health Services; Senior Lecturer Flinders University, Flinders Rural Community School

When asked to write a paper based on the presentation I gave at the Australian Association of Musculoskeletal Medicine conference held in Adelaide, South Australia, in October 2004, I felt both honoured and challenged. It has been but a few months since the Australian government and the National Health and Medical Research Council distributed their “bible,” the *Evidence-based Management of Acute Musculoskeletal Pain – a Guide for Clinicians*. On page 27, there is a statement regarding injection therapy:

“There is insufficient evidence demonstrating the effectiveness of injection therapy (facet joint, epidural or soft tissue) in the treatment of acute low back pain.”

I will attempt to give a discourse on my journey through the jungle of entangled branches I had to navigate on my quest to find a technique to assist the patients entrusted to my care when I was working in an emergency department. My scope was not a research project but a humble practitioner's journey and findings.

Professor Norm Broadhurst and I have several things in common – medical school graduation together, early postgraduate years in the same areas, general practice backgrounds, and rehabilitation physician training. Thanks to his persistence, I enrolled in the Graduate Diploma which led to the development of the techniques I will present, as I most likely would not have developed this approach without participating in the course.

A journey is never complete without a description of the factors that led to the adventure. During 13 years as a proceduralist (anesthetics, surgery, and obstetrics, combined within rural general practice) I had often found myself facing the frustration of dealing with musculoskeletal pain, especially low back pain. Along the way I was frequently invited to embark on the Graduate Diploma in Musculoskeletal Medicine pioneered by Professor Broadhurst via Flinders University in

South Australia. My previous practice was marred by the classic mistakes of the unenlightened – poor history, poor examination, unprofitable use of x-rays, referral to the wrong health professions, and not changing or challenging my presuppositions as to causation. The lack of locum relief was an added factor that impeded knowledge I could have gleaned by earlier enrolment in this course.

Migration back to the metropolitan area and acceptance into rehabilitation medicine as an advanced trainee once again revealed my therapeutic impotence in the area of low back pain. But when offered a position as deputy director and consultant in an Adelaide emergency department, I had my real awakening. Professor Broadhurst persisted in his evangelical outreach. I was meandering down my road to Damascus and struck with the reality that I was now dealing with a significant percentage of musculoskeletal presentations and needed to empower myself with the ability to manage them effectively. I lost my apathy and rebellion and I enrolled. I have now been filled with the spirit of enthusiasm and challenge that the course offered and I embarked upon the adventure of rethinking and changing behaviour with respect to the management of many conditions.

Prior to presentation of the development of the technique I use I should discuss the environment in which I work and its clientele. Noarlunga is located on the southern fringe of the Adelaide metropolitan area. It is characterized by a mixture of middle class strugglers, unemployed, intellectually challenged, and retiring persons. Cash flow is significantly limited for a large sector of the 160,000 persons residing in the Noarlunga basin. The local medical workforce is significantly under resourced, with between 20 and 50 extra general practitioners needed. Consequently, the population tends to gravitate towards the emergency department for both primary medical care and emergencies. Noarlunga Health

Services is a level 1 hospital which functions on a fee-for-service basis for inpatients, and apart from the academic admission unit and mental health unit, visiting accredited GPs or specialists utilise the beds. The emergency department is staffed principally with medical officers from rural or metropolitan general practice backgrounds, career medical officers, and a few interns and GP registrars. The hospital is on major trauma diversion, with patients being taken to the Flinders Medical Centre 18 km north. The clinical throughput for the emergency department consists of 44,000 - 45,000 patients per annum.

Upon graduating with the Graduate Diploma of Musculoskeletal Medicine, I felt the strong desire to change my practice, but to what – manipulation, mobilization, torsional release therapy, trigger point therapy? The following is a chronological overview of the changes I made and the outcomes of the process I have developed.

As with all graduates of a new course I was guilty of believing I had found a new panacea, but then realized each treatment option has a role but in itself is not the total answer. Through trial and error I tested each of the techniques I had learnt. As time went on I found that I developed a feeling and preference for one specific technique that appeared to work and this is what I desire to present to open up discussion.

My referral base was acute presentations, often brought on a stretcher by the South Australian Ambulance Service, community and ED colleagues, injured hospital staff, and acquaintances of those whose had found restoration of function by the method I used. When a pattern of success appeared to be on the horizon I decided to keep a statistical database and monitor outcomes in a slightly more rigorous manner.

Prior to this discourse I should relay my indebtedness to the visionary and pioneering work of Professor Broadhurst. What had this achieved

for my clinical practice?

- Improved and more focused history taking and examination
- Appreciation of biomechanics
- Improved understanding of therapeutic modality options
- Significantly improved discriminatory use of radiographic evaluation (my use of lumbosacral films in persons without red flags decreased from 90% to 1-2%)
- Seeing better outcomes as measured by reduction in pain, improvements in activities of daily living (ADL), restoration of recreational and vocational activity, and faster return to work, especially for patients on the Workers' Compensation nightmare roller coaster.
- Reduced ongoing demands upon the health system, with resultant improved quality of life and increased productivity of those presenting with an assortment of conditions.
- Reduction in opioid utilization for pain management
- Ability to offer patients an alternative to the merry-go-round that many chronic pain sufferers end up on as they seek relief from myriad medical and non-medical providers, enduring collected surgical and non-surgical investigations and procedures, often becoming embittered towards the world at large and towards doctors specifically.

History

Apart from the standard emergency department history, I take a much more vigorous biomechanical history from the patient, considering ADLs, related issues, chronological sequences, treatment modality options tried and the results, etc.

I seek to clarify whether the discomfort is somatic, somatic referred, visceral referred, radicular, complex regional pain syndrome, or a combination, and whether the chronology is acute or chronic. I seek the source of the pain anatomically, and investigate what attitudes and beliefs the person has regarding their pain and its manifestations. The recognition of red and yellow flags is most important. Is the pain mechanical, does it have features of psychological overlay, or are there

indicators of possible infective, inflammatory, neoplastic or metabolic components?

Examination

Standing

- Posture, alignment, scars, arm window, flat feet (assessed with the feet 10 cm apart) and suitable exposure
- Passive movements
 - Flexion – distance of hands to ground (and end feel)
 - Extension – watch for compensatory measures (and end feel)
 - Side-bending – hands to knees (and end feel)
 - Axial compression test – 10° extension, 10° rotation, and 10° side flexion for 30 seconds (zygapophysial joint, nerve, ligament)
- Hop on each leg up to 10 times to exclude skeletal issues – if unable to hop use Trendelenburg test (sound side sags = positive)
- Check gait and shoe wear
- Check PSIS and iliac crests for symmetry (tilt and rotation)
- Sit on the edge of the table
- Slump test – flex neck. Flex trunk. Elevate leg via heel. If this reproduces pain the head may be taken off flexion to see if symptoms resolve. Place hands on thighs and straighten legs first to see if tolerated.

Lie on edge of bed

- Iliopsoas test – flexed leg on the subject's chest. Push the other leg down then use resisted extension.

Lie on back (supine)

- Feel iliopsoas – above pelvis and at attachment at femur
- Check for psoas irritability and if present monitor improvement with torsional release manoeuvres
- SLR with dorsiflexion at 30° from the horizontal
- Check hamstrings for tightness
- Hip rotation
- SIJ – FADE or FABER test (C/I with hip prosthesis – if present do REAB test – resisted abduction of abducted leg)
- Palpate greater trochanter for bursal tenderness

- Dermatome sensation and reflex reactions

Lie on front (prone)

- Lumbar spine tenderness to palpation
- Paraspinal muscles tenderness to palpation and any spasm activity
- PSIS tenderness
- Buttock – gluteal muscles starting laterally and palpate medially
- Sciatic notch for tenderness
- Piriformis palpation and pump test
- Ischial tuberosity for tenderness
- Iliopsoas insertion on the lesser trochanter on the posteromedial femur
- Femoral nerve stretch with flexed knee and stabilized sacrum

Lie on side

- Iliotibial tract – palpate and test
- Hold in hip abduction and let go to see if pain occurs in sudden adduction.

Other physical examination as necessary.

One of the useful guides I have found is to get the patient to press on my back as to where they experience their discomfort – frequently I find they have placed their finger over my PSIS.

Over this time I have changed my approach from:

BC (before confrontation)

- X-ray, analgesia, and referral back to their local doctor and or physio (if they can afford it)

AD (after deliverance)

- Mobilization and or manipulation (unless contraindications)
- Torsional release with or without manipulation/mobilization
- Trigger point location (progression with cocktail used)
 - Lignocaine alone
 - Lignocaine with Celestone Chronodose
 - Marcaine with Celestone Chronodose

Findings

Of the over 150 in my sample to date using the technique, I now principally use the characteristics of the population as follows:

Male 48%: female 52%

Age range 18 – 80 years, with mean

Therapy for low back pain

average age 44 years

Principal mechanisms - mechanical activity combined with faulty biomechanics or failure to use manutention aids.

- Four had radicular features with CAT confirmation of pathology, with three referred back to their local doctor, and one I referred (a staff member) who subsequently underwent microdiscectomy.
- Two failed to respond and were referred to Professor Broadhurst – pelvic instability, improving with the use of a support belt.
- One had piriformis syndrome and improved with injecting the piriformis femoral attachment with Marcaine and Celestone Chronodose.
- One had abnormal illness behaviour for which I had to attend court. There was no abnormal outcome, but I had to give supportive evidence about a domestic issue before the courts – she was going through a custody battle and part of her chronology of presentations to the medical world was collecting procedures. She was a brilliant actor of whom I became aware after a phone call from the prosecuting police officer as to her whereabouts on a certain day. She was supposed to be attending court and he desired to confirm that she had a significant reason for the case to be postponed due to inability to attend.
- No red flags were missed.
- One hundred and forty-seven were exposed to the technique I will discuss, and were followed up with a phone call the following day and given my permission to communicate with me if there were any ongoing issues or their discomfort had not settled. I held as a standard that if I had to see them three times then my diagnosis was incorrect, my treatment had failed, or the patient was noncompliant with instructions given about exercises and lifestyle changes. To date I have seen only three persons more than twice, none more than three times, and most only once.
- Two have declined to embark on the technique I now use, when I believe

it may have been of assistance. Both had needle phobia.

- At times the clinical examination is not as comprehensive as documented above due to individual circumstances, such as with those brought in by ambulance because of inability to entertain independent mobility and are barouche- or bed-bound.
- I was asked to see one 76-year-old lady admitted to the ward. After she was deemed to be a suitable candidate with no red flags found by my colleagues, explanations given and consent obtained, she was subjected to my technique. I mobilized her and then beckoned her to mobilize from her three-day bed-bound loss of freedom with lack of physiotherapy improvement. Once she gained confidence that she could mobilize and was pain free she was most encouraged. In jest I stated that, if she could catch the security guard walking by, she could be discharged later that day. Taking me literally she ran along the corridor, caught him, turned around and stated she had not felt so good for years. One month later she was still free of the pain that had plagued her intermittently and she was compliant with daily stretches and a changed ADL profile.

Technique

- Once red and yellow flags have been considered and excluded on history and examination I discuss with the patient the biomechanics of the lower back and associated structures. This entails a discussion of pain pathways and the fact that all lower back pain is not zygapophysial joint, disc prolapse, and sciatica. Diagrammatic displays are shown and therapeutic modalities I can offer are discussed. Questions are permitted and answered as honestly as possible. If they are willing to embark on the "Squirrell technique" then verbal consent is secured and documented in the case notes.
- I discuss trigger point activity with the patient and reassure them that they are in control. They are assured that if any trigger activity is

experienced I will stop, anesthetize that area, and wait for their permission to proceed.

- I draw the low back anatomy on their skin so as to consistently guide my approach and assist the understanding of those watching the technique – medical and nursing students or ED staff.

Equipment

This consists of a 22 gauge spinal needle, 20 ml of 0.5 % Marcaine, 2 ampoules of Celestone Chronodose, and topical skin preparation for aseptic technique. The Celestone Chronodose is drawn up into a 10 ml syringe and then made up to 10 ml with Marcaine.

Technique

The spinal needle entry point is just medial to the PSIS where a natural dimple usually is located.

The needle is introduced slowly in an inferior direction aiming at the posterior sacroiliac ligaments, and then angled anteriorly towards the iliolumbar ligament, followed by the iliac crest where the aponeurosis and iliocostalis lumborum attaches to the posterior crest. Then it is aimed anterosuperiorly to where the longissimus thoracis and multifidi are located.

These constitute the four usual areas I have found to be the origin of pain in most people presenting with acute or chronic pain in the lumbar region. At each region 2.5 ml is administered from the 10 ml syringe, not in a single depot manner but in a regional manner.

This is administered bilaterally because I found that treating only one side prevents maximal stretching. Also residual pain is sometimes unmasked on the untreated side.

Once this is complete, the patient is encouraged to change posture from prone to supine ready for mobilization.

Mobilization

I utilize a few stretches that appear not only to help, but the patient can take them away with them and do three times a day in their own home, either alone or with a helper according to the mobilization exercise. To give less appears to downplay the issue of biomechanical fitness and to give more

tends to overwhelm them and lead to poor compliance. I encourage them to do the exercises for life as part of readjustment to respecting their body and attaining a healthier lifestyle.

Supine exercises use the "breathing out – relaxation association" to gain extra stretch until the end point is achieved, and are held for 15 seconds followed by the stretch being released gradually and the lower limb brought back to its normal position. I attempt to get the patient to discuss with me where they feel the stretch anatomically as a biofeedback technique, whether there is any discomfort, and whether they feel they could have done this prior to the injection.

1. The knee is flexed, followed by the hip as the knee is approximated towards the ipsilateral shoulder. Barriers are stiffness, residual discomfort (which is usually absent in my experience), abdominal adiposity, or large breasts.
2. The same is repeated towards the contralateral shoulder.
3. The knee is flexed so that the sole of the foot is resting on the bed adjacent to the contralateral knee. Standing on the non-flexed knee side, I place one of my hands on the patient's shoulder (flexed knee side) and using my other hand rotate the flexed knee towards me, and attempt to mobilize it towards the surface of the bed. Alternatively, this can be done standing behind the patient as illustrated.

Standing exercises

1. Standing upright and performing a sidebend in an attempt to bring the hand to the ipsilateral knee then repeating this on the other side.
2. I then get the patient to stand with their feet 30 cm apart and ask them to rotate their body. In so doing I point out they have in fact rotated using components of each joint from the ankles upwards and use this to teach them an alternative. Keeping the pelvis straight and looking forward I ask them to point their elbows outwards with 90° abducted shoulders. I use the analogy that this is like a broomstick going from elbow to elbow. I then get them to rotate their vertebrae without head

or pelvis rotation. Once again biofeedback discussion is used. If they really struggle with pelvic immobility during this exercise I discuss doing it whilst seated. I advocate doing this whilst watching television or looking in a mirror to get fixed vision to assist head immobility.

Gait

I then encourage them to go for a walk along the corridor and to return lifting their knees in an exaggerated manner (to hip flexion of 90°). This is not a home exercise but to demonstrate the degree of relief they now feel and to assess their gait.

Feedback is requested. This is usually a flurry of thanks, surprise, and motivation to change any lifestyle risk factors that may be components in their presentation. It is essential to capitalize opportunistically at this point on biomechanics and maintenance, lifestyle, daily exercise rituals to maintain flexibility, weight loss, and other issues which are evident.

I demonstrate the exercises again and get the family member (usually one is present) to show me how they will assist with the exercises or supervise them, answer any new questions, and advise them of my usual follow up. Because of the location in an emergency department I have limitations that may not exist in a private setting. Consequently reliance is on phone contact at 24 hours from me to them, and then within one month from them to me via my mobile.

I found that Marcaine decreased the incidence of any flair phenomena, which used to occur at the 8-hour mark using lignocaine 1%. To limit this further I usually offer a Voltaren suppository or oral NSAID for 24 hours, assuming there is no contraindication.

Issues that stem from this research

- The sample is biased by the nature of their presentation and the location of my practice.
- There is no scope to use a random or double blind approach.
- Follow up is limited by the constraints of working in an ED of a public hospital.
- Persons labelled with a variety of conditions of a chronic nature often

find that not only does the acute phase subside but the chronic symptoms do so as well.

- There is value in thorough and early assessment of patients presenting with acute or acute on chronic lower back pain, using appropriate musculoskeletal treatments with explanations as to biomechanics, etc., and the use of opportunistic lifestyle counselling.

Conclusions

For adults presenting with non-radicular acute lower back pain and the absence of red and yellow flag features, there is a close to 95% improvement with Marcaine and Celestone injections and mobilization, followed by daily stretching. This is seen with 90% within 24 hours and another 5% within 48 hours. There appears to be benefits both for acute and chronic low back pain sufferers. There is substantial benefit to be made from investing in thorough training of primary medical care physicians about biomechanical history taking, examination, and the correct use of investigative modalities. I cannot emphasize enough that early intervention for back pain has significant gains in reducing progression from acute to chronic pain, and the loss of productivity and vocational and recreational activity that follows. Each patient takes me approximately one hour to manage and educate. This is outside what is acceptable to most primary healthcare physicians. Medicare does not reward this quality time input. I also state my concern for the workers' compensation system that appears to favour referrals to specialists for back pain patients that in turn creates multiple delays, productivity loss, and many unnecessary tests. Managing nursing staff with low back pain presentations has seen all but one return to work within a week without LS spine radiation or CAT scan use, except for one referred to above who required microdiscectomy for true radicular features that failed to respond to conservative management over six months.

Further reading

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Noarlunga Health Services & ED



Review of anatomy



Examination & Surface anatomy



Check waiting room behaviour



Surface anatomy is important

Injection sites – I have 4 favourites locations via one entry point per side



Iliac Crest



Sacro-iliac



Thoraco-lumbar
fascia



Ilio-lumbar
Ligament



Stretches in standing position



Side bend



Truncal rotation using
fixed pelvis and head

Stretches lying down

Knee to shoulder –
ipsi and contra lateral
using
breathing relaxation
technique



Spinal
Rotation
with
fixed shoulder



Reproducibility studies in manual/musculoskeletal medicine: a new method for kappa independence from prevalence

Jacob Patijn, Pain Management and Research Centre, University Hospital Maastricht, Maastricht, The Netherlands; Elien Pragt, Pain Management and Research Centre, University Hospital Maastricht, Maastricht, The Netherlands; Ruud Brouwer, Institute for Manual/Musculoskeletal Medicine, Eindhoven, The Netherlands

Abstract

In manual/musculoskeletal medicine, it has become increasingly important that diagnostic tests are reproducible. The kappa statistic is the measure most frequently used to define the interobserver agreement of diagnostic procedures. The main disadvantage of the kappa statistic is its dependence on the prevalence, making a good kappa value at the end of every reproducibility study always unpredictable. A previous, published theoretical protocol proposed solving this problem by obtaining a prevalence near 0.50. This was evaluated in the present study of the passive hip flexion test. A prevalence of 0.44 was found with a good to excellent kappa value of 0.75. It is concluded that when imple-

menting the proposed method in the protocol format for reproducibility studies, using kappa statistics, a prevalence P near 0.50 can easily be obtained, avoiding unexpectedly low kappa values.

Introduction

In the past, a number of schools have developed in manual/musculoskeletal medicine (M/M medicine) using many different diagnostic procedures sometimes for the same joint.¹ Estimates of the reproducibility of most tests in this field are generally inadequate or in some cases completely lacking.¹ It has become increasingly important that diagnostic tests are reproducible. Only then can these procedures be used to define specific syndromes. Specific

syndromes are necessary to define homogeneous study populations for efficacy trials. Reproducible diagnostic procedures are also essential for the professionalisation of educational systems in M/M medicine.² Many of the earlier reproducibility studies show methodological flaws such as lack of definitions of source population, absence of selection procedures, absence of detailed description of diagnostic procedures and their process of final judgement. In most studies, training phases were not performed to standardise the procedures. Frequently, blinding procedures were lacking.

Cohen introduced the kappa coefficient as a measure of agreement between two observers' recording of the same diagnosis in a random sample of patients from a well-defined population.^{3,4} The advantage of the kappa statistic was its adjustment of the overall agreement for the expected agreement by chance. Therefore, kappa statistics are the method of choice for evaluation of concordance between two clinicians for nominal categories.⁵⁻⁷ Since then, many kappa studies of the locomotor system have appeared in the medical literature. However, contradictory results sometimes occurred in kappa studies for the same diagnostic test. For instance, in the low back, different groups of diagnostic procedures showed a wide range of kappa values. Based on a previous literature study,¹ the ranges of these different groups of diagnostic testings are shown in Figure 1.

Normally, kappa values can range from -1 to $+1$.³ Several schemes are available to draw the line defining good concordance.⁸⁻¹⁰ The most widely used scheme is that of Landis and Koch.⁹

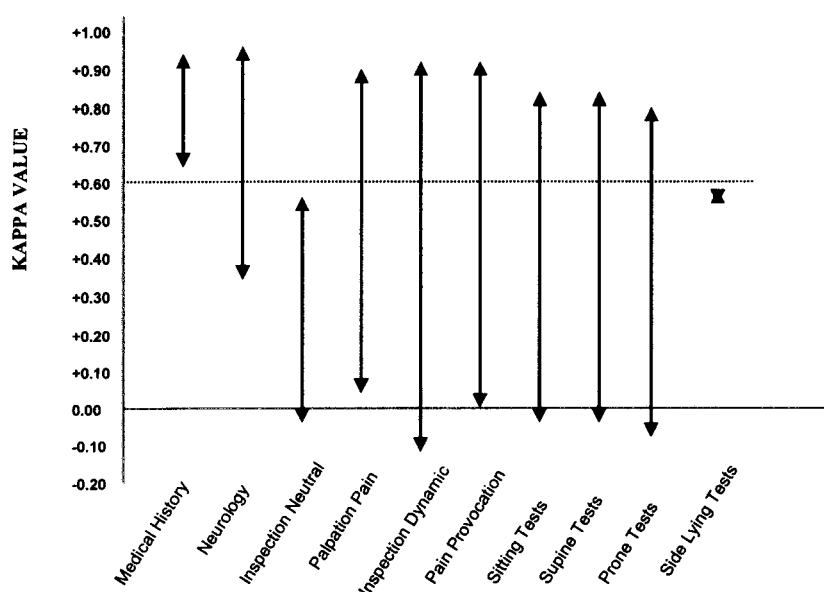


Fig. 1. Overall view of the range of kappa values in different diagnostic groups of the low back, based on a previous literature study.¹² The cut-off line for good agreement is at the level of a kappa value of 0.60.

Reproducibility studies in manual/musculoskeletal medicine

These authors stated that kappa values above 0.60 represented good to excellent agreement beyond chance between two raters. In contrast, kappa values between 0.40 and 0.60 reflected a fair to good agreement. Kappa values of 0.40 or less represented a poor agreement beyond chance. Frequently, kappa studies using the scheme of Landis and Koch conclude that many diagnostic tests are invalid for clinical practice, because their kappa showed values less than 0.40 or sometimes negative values. Comparison of kappa studies of the same diagnostic procedure is frequently impossible because vital information such as overall agreement and the prevalence of the index condition (the frequency of positive tests in the study population) were not presented. Moreover, a 2x2 contingency table with the raw data in combination with the calculated kappa value of an evaluated diagnostic procedure is seldom shown.

In particular, the overall agreement and the prevalence of the index condition can strongly influence the final kappa value of a reproducibility study.¹¹ Overall agreement reflects the total proportion in which two observers agree about positive and negative diagnosis

or test procedure. Too few (or too many) subjects showing the positive sign reduces the power of the kappa statistic to yield an adequate assessment.

It appears from many reproducibility studies¹ that the investigators have not always been aware of the influence of the prevalence on the size of the kappa value. Similarly, the extensive medical literature, providing kappa statistics with their studies, fail to mention this problem, despite the fact that the statistical literature has recognised this problem for a long time.¹²⁻¹⁶ and proposed solutions such as the PABAK-method (Prevalence-Adjusted Bias-Adjusted Kappa).¹²

Figure 2 shows the relationship between kappa values and prevalence (P) for different overall agreements (P_o).¹¹ It appears that in both very low prevalences and very high prevalences a kappa value can become very low and sometimes even negative. Dependent on the level of overall agreement P_o , the kappa/prevalence curve can shift upwards or downwards. The top of the curves, reflecting the maximal possible kappa value ($\hat{e}_{max}$) of overall agreement, is always associated with a prevalence of 0.50. If for in-

stance an overall agreement P_o is 0.77 the top of the kappa/prevalence curve ($\hat{e}_{max} = 0.54$) does not reach the cut off line of a kappa value of 0.60. The list of different overall agreement values P_o in Figure 2 also shows the higher the overall agreement the larger the range of prevalences with kappa values above 0.60.

Adapting the cut off level of 0.60 of Landis and Koch⁹ for a good to excellent agreement beyond chance between two raters, Figure 2 also shows that an overall agreement of 0.80 or more is preferable to obtain a substantial kappa value in a reproducibility study.

Since the prevalence of the index condition in reproducibility studies is never known in advance, there is always a risk of obtaining an inappropriately low kappa value.

To avoid most of the flaws for reproducibility studies as mentioned above, a protocol format has been developed as a guideline for investigators performing this kind of study.² The protocol distinguishes five different phases for the reproducibility study, through which the investigators have to pass in order to obtain a best estimate of kappa value.

In the first phase of the protocol, a sufficient training program has to guarantee that both observers agree in detail about the performance and judgement of the diagnostic procedure. In the second, overall agreement phase observers have to obtain an overall agreement larger than 0.80. In the third, test phase, a study format is provided to obtain a prevalence approximating 0.50.

How to influence the prevalence in the direction of a value of 0.50 is shown in an example in Figures 3a and 3b. Observers (A, B) have already reached an overall agreement of 85%. To get substantial numbers of positive tests in this example, 100 subjects were recruited. In normal kappa studies only 40 subjects are needed. From the source population, each observer selects 25 subjects with a positive test and 25 subjects with a negative test (nominal data). Both observers are unaware of their mutual results and subjects don't know the judgement of their own diagnostic test. The observers send

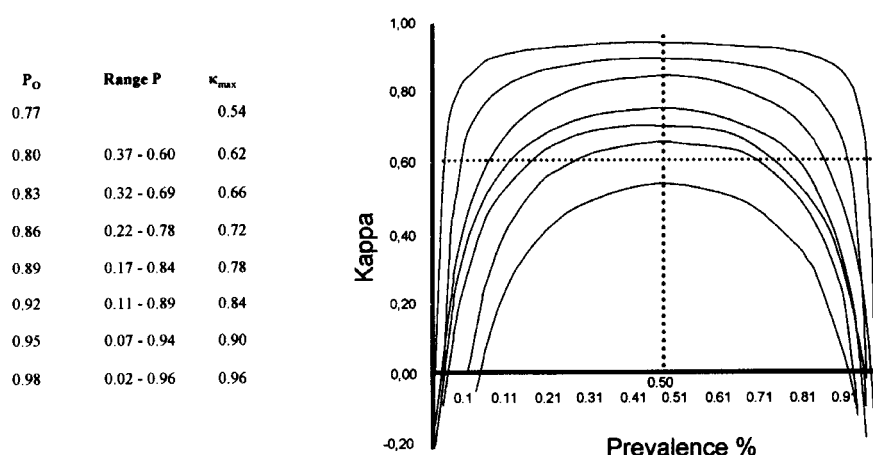


Fig. 2. Kappa/Prevalence curve for different levels of overall agreement P_o . The cut off line for good agreement is at the level of a kappa value of 0.60. Kappa/prevalence curves passing this cut off line reflect an overall agreement > 0.80. The maximal kappa values K_{max} , shown in the list, are always related with a prevalence P of 0.50, the top of the curves.

their respective selections to each other. Based on the overall agreement of 85% and therefore a disagreement of 15% an observer will judge the 25 positive tests of the other observer in 21.25 (0.85x25) cases as also positive. 3.75 (0.15x25) cases will tend to be negative. The same will tend to happen to the other observer. Based on these theoretical figures, a prevalence P_o of 0.51 was obtained with a kappa value of 0.70.

Since the proposed method of influencing the prevalence towards a level of 0.50 was purely theoretical, the main aim of this study was to evaluate the proposed 50%-prevalence method in practice.

Material and Methods

The study was performed according to the international FIMM protocol format on kappa studies in M/M medicine.² All patients of the "study phase" and the "overall agreement phase" were selected on entrance from a daily population of the university pain clinic. All patients were informed about the aim of the study. The passive hip

flexion test was chosen as the test to be evaluated for its reproducibility. In the preparation phase, two observers (P and E) discussed the most effective way to arrange the study, so as not to interfere with the daily patient outdoor clinic program. The observers' outpatient clinic timetables were mutually attuned to select as many patients as possible per day. In the training phase of the protocol observers discussed in detail all aspects of the performance of the hip flexion test. In 10 joint sessions, both observers examined the same patients to standardize their performance of the hip flexion test and its final judgement. Their consensus was written down in a study logbook and an evaluation form was developed to record the test results. In the same training phase of the protocol, the observers agreed that the hypothesis of the hip flexion test was mainly a testing of the muscle tone of the different muscles around the hip and not the range of motion of the hip joint itself.

The hip flexion test had to be performed in a supine patient. Observers were always standing on the left side of

the patient. Next, the observer passively flexed the hip with his right hand, positioned on the knee, in the direction of the shoulder on the same side. The passive flexion was continued until the observer judged the maximal point of resistance was reached. The pelvis was not allowed to move from the examination table. When this point of maximal resistance was reached, the observer positioned his left hand (perpendicular on the body) at a point on the body where five fingers fit in the space between the body and the surface of the upper leg (see Figure 4). If this space was too small, the maximum number of fingers was placed between the lower margin of the patella and the surface of the body. Subsequently, the left hand was moved and horizontally shifted over the body surface to the opposite side and the hip flexion test was performed on the left side. The number of fingers was estimated for both sides. A difference of more than one finger was judged as decisive for left/right difference. The side of the passive hip flexion test showing the bigger gap (more fingers) was judged

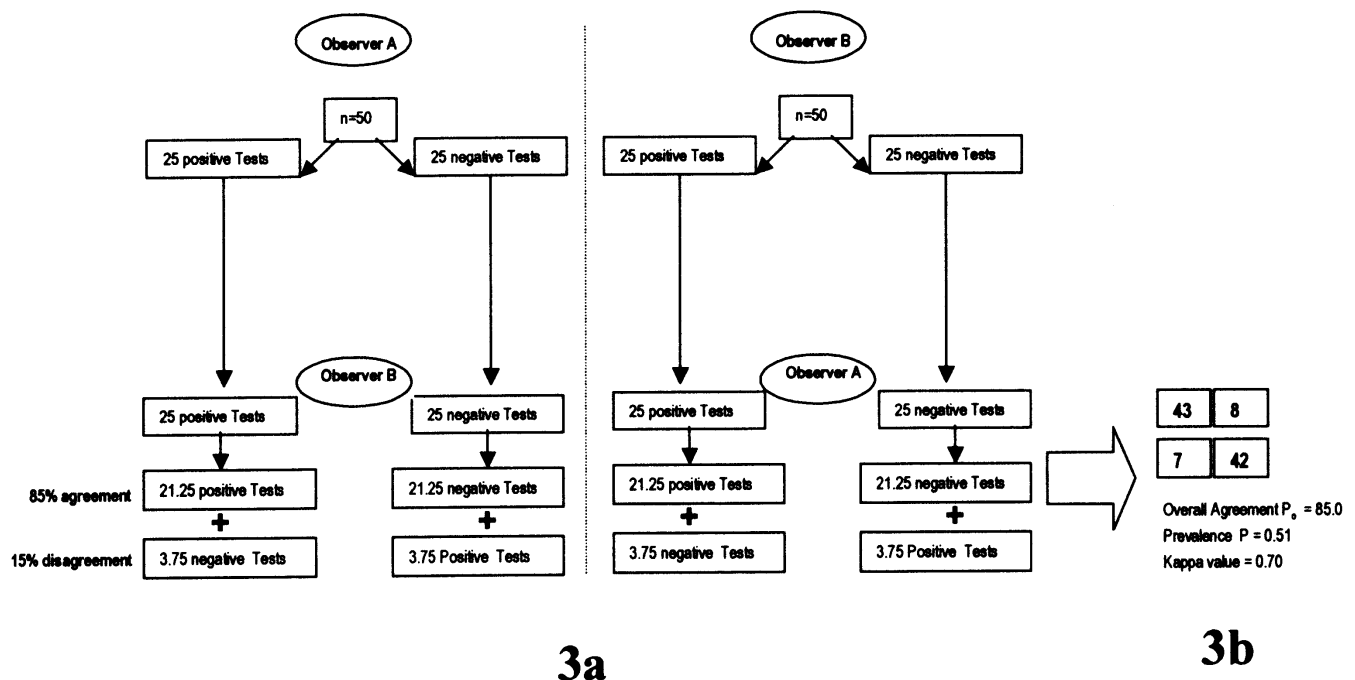


Fig. 3. a. Theoretical format to accomplish a prevalence P near 50% (see text). b. 2x2 contingency table and calculated prevalence P and kappa value.

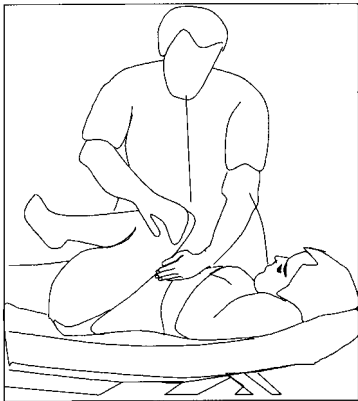


Fig. 4. Passive Hip Flexion Test. At the point of maximal resistance of the passive hip flexion, the observer positioned his left hand (perpendicular on the body) at a point of the body on which five fingers fit in the space between the body and the surface of the upper leg.

		Observer P	
		Yes	No
Observer E	Yes	11	1
	No	3	20

Prevalence P : 0.37
Overall Agreement P_o : 88.6
Kappa Value: 0.74

Fig. 5. 2x2 contingency table based on 35 included patients, resulting in a prevalence P of 0.37.

		Observer P	
		Yes	No
Observer E	Yes	15	2
	No	3	20

Prevalence P : 0.44
Overall Agreement P_o : 87.5
Kappa Value: 0.74

Fig. 6. 2x2 contingency table based on 35 included patients and the remaining not included 5 patients of observer E. The supposed judgements of observer P of these 5 patients are based on constant overall agreement in this study. In the completed population of 40 patients a prevalence P of 0.44 was obtained.

as the side of the positive test.

After the training phase observers entered the so-called “overall agreement, fourth phase” of the protocol in which observers had to accomplish an overall agreement better than 0.80 in 20 patients.² Each observer examined 10 patients and sent them to each other. In the final, fifth “study phase” of the protocol⁹, the observers had to evaluate the hip flexion test in 40 patients (2x20) for its reproducibility with prevalence approximating 0.50.

To guarantee optimal blinding procedures, observers P and E were not allowed to communicate with each other during the evaluation of the patients. Only one observer was present in the examination room at any one time. The observers were not allowed to communicate with the patient who was sent to him by the other observer. Patients were not intentionally made aware of the result of their test.

Results

In the first “overall agreement phase” of the protocol, the observers (P and E)

agreed only on 50% of the hip flexion tests. To analyse the reason for this low overall agreement, it was decided to return to a second “training phase” to search for differences in performance and judgement of their hip flexion tests. It became clear that the agreement about the judgement of the hip flexion test, using a difference of one finger or more, was not suitable. It appeared that the size of the hands and therefore the thickness of the fingers differed substantially between observers! One finger of observer P was two times thicker than one finger of observer E. Subsequently, it was agreed to redefine the judgement of the test, whereby for observer P a difference of more than one finger was decisive for positive test and for observer E more than two fingers. It was further decided not to restrict the performance of the agreed hip flexion test only to the study patients. To enhance their performance skills, both observers applied the agreed test procedure in every patient who visited the pain clinic during the study.

In the second “overall agreement

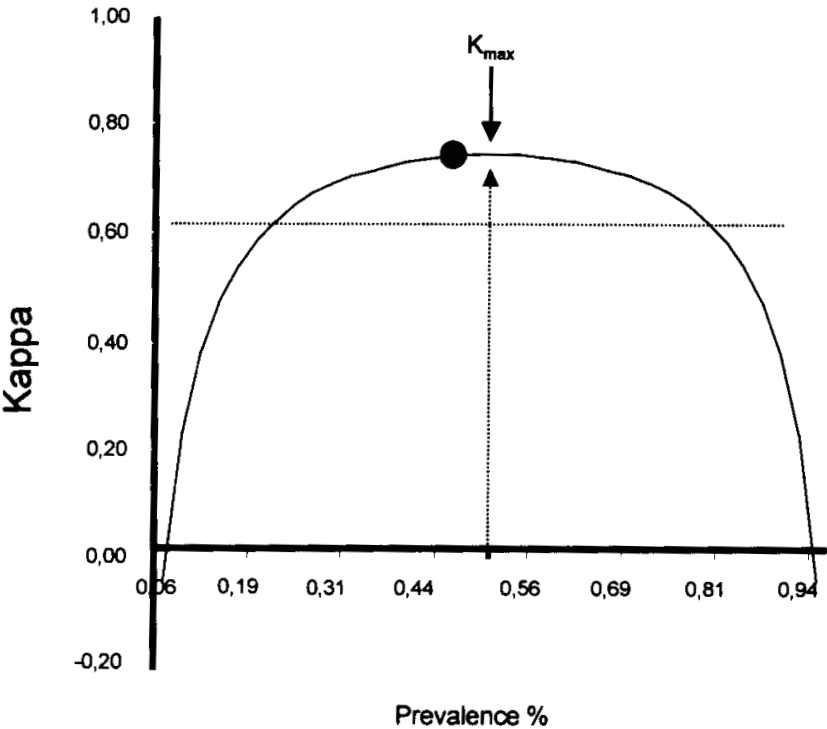


Fig. 7. Kappa/ Prevalence curve with an overall agreement P_o of 87.5%. The cut off line for good agreement is at the level of a kappa value of 0.60. The black dot on the curve represents the found kappa of 0.74 in the present study. The maximal kappa value K_{max} of 0.75 at the top of the curve reflects a prevalence of 0.50.

phase" observers obtained a far better agreement of 88.2%. In the subsequent "study phase" of the protocol, almost the same overall agreement of 88.6% was found. In Figure 5 the 2x2 contingency table is shown. An observed kappa value ($\hat{\kappa}_o$) of 0.74 was found, with a prevalence of the index condition of 0.37.

However, these results were calculated in a study population of only 35 patients instead of the agreed 40 subjects. The reason for this difference was unforeseen logistic circumstances. Observer E was not able to include her last five patients in the study. During the study, both observers had to mark the registration forms with their own name if it was a patient they had selected themselves for the study or with the name of the other observer if it was a patient sent by the other observer. In this way, both observers kept themselves informed of how many positive and negative tests they had sent to the other observer. After collecting the registration forms at the end of the "study phase" of the protocol, it became clear that the remaining five patients were of observer E. Counting her own positive and negative tests of patients she had included in the study, it became clear that observer E still had to include five patients with a positive test to send to observer P.

Based on the consistent overall agreements found in both "overall agreement phase" and "study phase" of this study (respectively 88.2% and 88.6%) and which is in agreement with the literature⁶, it can be assumed that observer P would have judged the five remaining hip flexion tests of observer E in four cases as positive and in one case as negative. The final 2x2 contingency table with a total of 40 patients is shown in Figure 6. The prevalence increased from 0.37 to 0.44 with a kappa value of 0.74 and an overall agreement of 87.5%.

Based on the numbers in Figure 6 a prevalence/kappa curve (see Figure 7) could be made. Such a curve can be constructed in every kappa study by changing the numbers in the cells of agreement (15 and 20) from 0 to 35. The figures in the cells of disagreement (2 and 3) are kept constant. Under our study conditions (an overall

agreement of 88.0), the highest obtainable kappa value K_{max} was 0.75 for a prevalence of 0.50.

Discussion

In M/M medicine, reproducibility studies of diagnostic procedures are crucial for its further professional development. In clinical practice, the indication for every therapeutic intervention must be based on reproducible diagnostic procedures. Therefore we must educate our discipline to standardize procedures and ensure that they are at least reproducible.

The present study was performed based on a recently published protocol format in which different phases can be distinguished.² The most important phase of the protocol is the "training phase". In this phase, observers have to agree about the smallest details of the performance of the whole diagnostic test procedure. Initially, we found a very poor interobserver agreement of 50.0%. In the second phase, a small detail of difference in finger thickness between observers was to blame. A simple adaptation was required to achieve an acceptable overall agreement of 88.3%. Adapting the scheme of Landis and Koch to draw the line on good concordance at a kappa level of 0.60, an overall agreement of at least 80% is necessary to provide a better chance of obtaining a kappa value greater than 0.60¹⁷ (see Figure 2). With an overall agreement of less than 0.80, the top of the kappa/prevalence curves, reflecting the maximal kappa values of overall agreement, will never cross the cut off line of a kappa value of 0.60. In the "training phase" of the protocol, it is essential for observers to agree about the hypothesis underlying the test. In two former reproducibility studies,^{18,19} changing the hypothesis of the sacro-iliac tests in the first study¹⁹ supposed to test the mobility of the SI-joint into one measuring muscle tone of the hip/SI-joint in the second study,¹⁸ resulted in an increasing of the kappa value from -0.09 to 0.70. In the present study observers agreed that the left/right difference of the hip flexion tests was due to differences in the tone of the hip muscles and not a result of difference in mobility of the hip joint itself.

As can be seen in the kappa/prevalence curve of Figure 2, the dependency of the kappa on prevalence is a serious limitation of the kappa method.

The results of our study, with an obtained prevalence of 0.44 and thereby a good-to-excellent kappa value of 0.75, showed that the proposed theoretical format to influence the prevalence approximating to 0.50 is successful. From our experiences with the entire protocol, it became clear that the "training phase" is the most vital phase in the reproducibility study. Apparently minor details of the diagnostic procedure, such as a difference in finger thickness, can have major consequences for the interobserver agreement. The present study shows the researcher how to achieve a more robust kappa value by arranging prevalence to around a level of 50%. The disadvantage of this method is that only one diagnostic procedure with nominal data can be evaluated per study.

The first task of all schools in manual/musculoskeletal medicine is to make their specific diagnostic procedures at least reproducible. However, even if we define a reliable and repeatable test, we must then prove the validity of the test to determine its inherent meaning. Only then can the specificity and sensitivity of a diagnostic procedure can be estimated.

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Journal Abstracts

This section aims to update the reader with some of the more significant musculoskeletal research published in the last year which is listed on the Medline and CINAHL databases.

BACK PAIN

Barker KL, Shamley DR, Jackson D. Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine* 2004; 29(22): E515-9.

Study Design: Prospective, cross-sectional observational study.

Objectives: The aim of this study was to determine if there was an association between wasting of psoas and multifidus as observed on MRI scans and the presenting symptoms, reported pathology, pain, or disability of a cohort of patients presenting with unilateral low back pain.

Summary of Background Data: Current physiotherapy practice is often based on localized spine stabilizing muscle exercises; most attention has been focused on transversus abdominus and multifidus with relatively little on psoas.

Method: Fifty consecutive patients presenting to a back pain triage clinic with unilateral low back pain lasting more than 12 weeks were recruited. The cross-sectional surface area (CSA) of the muscles was measured. Duration of symptoms, rating of pain, self-reported function, and the presence of neural compression were recorded.

Results: Data analysis compared the CSA between the symptomatic and asymptomatic sides. There was a statistically significant difference in CSA between the sides ($P < 0.001$). There was a positive correlation between the percentage decrease in CSA of psoas on the affected side and with the rating of pain ($\rho = 0.608$, $P < 0.01$), reported nerve root compression ($\rho = 0.812$, $P < 0.01$), and the duration of symptoms ($\rho = 0.886$, $P < 0.01$). There was an association between decrease in the CSA of multifidus and duration of symptoms.

Conclusions: Atrophy of multifidus has been used as one of the rationales for spine stabilization exercises. The evidence of coexisting atrophy of psoas and multifidus suggests that a future

area for study should be selective exercise training of psoas, which is less commonly used in clinical practice.

Comment: The psoas has been the centre of the musculoskeletal medicine (MSM) universe for some practitioners for many years. This intriguing study suggests it may be worthwhile exploring further. Some methodological idiosyncrasies had me puzzled (excluding spondylolithesis patients, measuring nerve root compression) and there would be much speculation as to the meaning and relevance of this finding. The limits of the study include: the patients were supine for the measurements, there was no allowance for fatty infiltration of the muscles, and no functional tests of the muscles were performed.

We still await guidance as to optimal exercise programs for low back pain (LBP), perhaps a goal that will never be met, but probably worthwhile pursuing for a bit longer. This study points to incorporating psoas into the tested exercise regimes. – *Dr Scott Masters*

Shaw WS, Pransky G, Patterson W, Winters T. Early disability risk factors for low back pain assessed at outpatient occupational health clinics. *Spine* 2005; 30(5): 572-80.

Study Design: Inception cohort (≤ 14 days after pain onset) with 1-month follow-up.

Objective: To determine whether disability risk factors provided by patients and clinicians at a first medical visit for acute occupational low back pain predict outcomes.

Summary of Background Data: Improving health and work outcomes for patients with occupational low back pain may require early identification of risk factors for persistent pain and disability. Previous studies of back pain prognosis have not assessed patients at the time of initial provider contact, and many have not differentiated between occupational and non-

occupational injuries.

Method: Patients (183 female, 385 male) presenting to occupational health clinics with recent onset occupational low back pain (≤ 14 days duration) completed a 16-item survey of potential disability risks including demographic, injury, workplace, psychosocial, and symptom factors. After the initial visit, clinicians completed an additional 10-item questionnaire of symptoms and initial prognosis. Outcome variables of functional limitation and work status were assessed 1 month after pain onset.

Results: In multivariate analyses, functional improvement and return to work were more strongly predicted by employer factors (job tenure, physical work demands, availability of modified duty, earlier reporting to employer) and self-ratings of pain and mood than by health history or physical examination. A logistic regression model had a sensitivity of 74.3% to predict those remaining out of work and a specificity of 70.1%.

Conclusions: Early screening for disability risk factors may be helpful to identify those patients at greatest risk for delayed recovery from occupational low back pain. Intervention strategies for high-risk patients might be improved by focusing on job factors, pain coping strategies, and expectations for recovery.

Comment: Prognostic risk factors for occupational LBP (OLBP) offer an insight into secondary prevention. This cohort study looked at OLBP at presentation through simple tests that would be viable to implement in a practice setting.

The findings confirm previous implications re the importance of non-medical factors in predicting disability. Workplace physical and psychosocial environment, perception of injury severity and expectation of recovery were the big three predictors of disability. These are all modifiable factors.

Next step, combine intervention to high risk patients in an attempt to

improve outcomes. – *Dr Scott Masters*

Long A, Donelson R, Fung T. Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine* 2004; 29, 2593-602.

Study Design: Multicentered randomized controlled trial.

Objectives: To determine if previously validated low back pain (LBP) subgroups respond differently to contrasting exercise prescriptions.

Summary of Background Data: The role of “patient-specific” exercises in managing LBP is controversial.

Methods: A total of 312 acute, subacute, and chronic patients, including LBP-only and sciatica, underwent a standardized mechanical assessment classifying them by their pain response, specifically eliciting either a “directional preference” (DP) (i.e., an immediate, lasting improvement in pain from performing either repeated lumbar flexion, extension, or sideglide/rotation tests), or no DP. Only DP subjects were randomized to: 1) directional exercises “matching” their preferred direction (DP), 2) exercises directionally “opposite” their DP, or 3) “nondirectional” exercises. Outcome measures included pain intensity, location, disability, medication use, degree of recovery, depression, and work interference.

Results: A DP was elicited in 74% (230) of subjects. One third of both the opposite and non-directionally treated subjects withdrew within 2 weeks because of no improvement or worsening (no matched subject withdrew). Significantly greater improvements occurred in matched subjects compared with both other treatment groups in every outcome (P values <0.001), including a threefold decrease in medication use.

Conclusions: Consistent with prior evidence, a standardized mechanical assessment identified a large subgroup of LBP patients with a DP. Regardless of subjects’ direction of preference, the response to contrasting exercise prescriptions was significantly different: exercises matching subjects’ DP significantly and rapidly

decreased pain and medication use and improved in all other outcomes. If repeatable, such subgroup validation has important implications for LBP management

Comment: In keeping with the recommendations of the Cochrane Back Review Group, this study emphasises the importance of identifying subgroups to improve results with so-called “non-specific low back pain”. By using the McKenzie method of testing for a directional preference in exercises, the results were better for all outcomes when using these exercises as treatment compared with using exercises in the opposite direction or non-directional “evidence-based” exercises. This may come as no surprise as the ultimate selection bias was applied in the selection process. There was also a possible bias in the treatment process that the authors acknowledge—that the treating therapists in the study were trained in the McKenzie method. It is stated that they tried to be equally as enthusiastic with all three groups of participants, but this must have been a hard act to do.

The most interesting result reported was that 74% of those who underwent a mechanical assessment for the study actually had a directional preference. This suggests that these results may have quite a wide application in the low back pain population. The selection process can be done in the surgery without painful or expensive needles or investigations as long as the appropriate training has been done.

Two questions not addressed by this study are the effects of non-directional exercises on those who were excluded and the long term outcomes of those who were included. Follow-up was only for two weeks, so there is no certainty that the effect was lasting. Elsewhere the McKenzie “centralisation phenomenon”, one aspect of directional preference, has been shown to be a predictor of better outcomes at one year. This makes it potentially useful in the routine assessment of low back pain.

Werneke M, Hart DL. Centralization phenomenon as a prognostic factor for chronic low back pain and disability. *Spine* 2001; 26: 758-64; discussion, 765.

– *Dr Michael Yelland*

Niemisto L, Rissanen P, Sarna S et al. Cost-effectiveness of combined manipulation, stabilizing exercises, and physician consultation compared to physician consultation alone for chronic low back pain: a prospective randomized trial with 2-year follow-up. *Spine* 2005; 30(10): 1109-15.

Study Design: A prospective, randomized controlled trial.

Objective: To examine long-term effects and costs of combined manipulative treatment, stabilizing exercises, and physician consultation compared with physician consultation alone for chronic low back pain (CLBP).

Summary of Background Data: An obvious gap exists in knowledge concerning long-term efficacy and cost-effectiveness of manipulative treatment methods.

Methods: Of 204 patients with CLBP whose Oswestry Disability Index (ODI) was at least 16%, 102 were randomized into a combined manipulative treatment, exercise, and physician consultation group (i.e., a combination group), and 102 to a consultation alone group. All patients were clinically examined, informed about their back pain, and encouraged to stay active and exercise according to specific instructions based on clinical evaluation. Treatment included 4 sessions of manual therapy and stabilizing exercises aimed at correcting the lumbopelvic rhythm. Questionnaires inquired about pain (visual analog scale (VAS)), disability (ODI), health-related quality of life (15D Quality of Life Instrument), satisfaction with care, and costs.

Results: Significant improvement occurred in both groups on every self-rated outcome measurement. Within 2 years, the combination group showed only a slightly more significant reduction in VAS ($P = 0.01$, analysis of variance) but clearly higher patient satisfaction ($P = 0.001$, Pearson χ^2) as compared to the consultation group. Incremental analysis showed that for combined group compared to consultation group, a one-point change in VAS scale cost \$512.

Conclusions: Physician consultation alone was more cost-effective for both health care use and work absen-

teeism, and led to equal improvement in disability and health-related quality of life. It seems obvious that encouraging information and advice are major elements for the treatment of patients with CLBP.

Comment: This prospective randomized trial from an orthopedic hospital in Helsinki, Finland, showed marginal reduction in pain on VAS of doubtful clinical benefit with the addition of manipulation and stabilizing exercises to an evidence-based physician consultation alone with a stay-active emphasis. In addition to conventional statistical analysis, the minimum clinically important difference can be used; that is, the smallest difference that patients perceive as beneficial. For VAS this is a change of 2 on a 0-10 scale.^{1,2} Based on this result, the observed statistical difference in the present study at two-years follow-up (2.4 in the VAS scale of 0-100) is not clinically significant.

Cost-effectiveness of these other components was doubtful in this high-quality study when follow up was extended for two years. Disability and HRQOL improved equally in both groups. The authors concluded that information, advice, and personal communication served adequately as the treatment of choice for most patients with chronic non-specific low back pain. This is consistent with earlier studies by Indahl et al.^{3,4}

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- Dr David Roselt

Galiano K, Obwegeser AA, Gabl MV et al. Long-term outcome of laminectomy for spinal stenosis in octogenarians. *Spine* 2005; 30(3): 332-35.

Study Design: Cohort study with follow-up after at least 1.5 years.

Objectives: The purpose of this study was to determine long-term safety and efficacy of laminectomy in octogenarians.

Summary of Background Data: This is the first study evaluating the outcome in octogenarians with well-defined lumbar spinal stenosis. This study was designed to provide some guidance in clinical-practical decisions in the treatment of aged patients with lumbar stenosis.

Methods: We evaluated long-term outcome after laminectomy in 23 consecutive patients affected by lumbar spinal stenosis. Comorbidity was assessed using the Cumulative Illness Rating Scale for Geriatrics. At follow-up, all patients completed a questionnaire containing the Visual Analog Pain Scale and the Oswestry Disability Index. The use of analgesics was assessed from chart review of their family physician.

Results: The average age at the time of surgery was 82.2 +/- 2.6 years; the mean follow-up was 2.7 +/- 1.2 years. The mean of the Cumulative Illness Rating Scale for Geriatrics total score was 7.7 +/- 4.3, reflecting the normative comorbidity-values of octogenarians. At follow-up, 4 patients had died. The Oswestry Disability Index for the remaining patients was 36.4 +/- 28%. The daily nonsteroidal anti-inflammatory medication had decreased from 1.9 to 0.1 equivalent analgesic doses and the amount of morphine from 0.6 to 0.2 equivalent narcotic doses. The Pain Score on the Visual Analog Pain Scale decreased from 85 to 39. After surgery no patient had claudication.

Conclusion: On the long-term, decompressive laminectomy in selected octogenarians results in decreased disability, decline of analgesics usage, and increased quality of life.

Comment: Lumbar spinal stenosis has become the most frequent indication for spinal surgery in the over 65 age group. This study is reassuring for

patients with spinal canal stenosis who are suffering pain, usually in the form of spinal claudication, and disability not responding to trials of conservative treatment including caudal epidurals of local anesthetic and corticosteroid and oral opioids. This is a cohort study, and lacks a control group, but it is very unlikely that an RCT would be acceptable to most patients in this situation. There were no major surgical complications, and the four deaths at follow up is consistent with the average death rate in industrialized countries in this age group. Only 65% of patients reported a general satisfaction with surgery and the authors speculate that this could be related to five patients (21%) being diagnosed with comorbid depression which can coexist with cognitive impairment, with evidence of this combination in up to 25% of patients over the age of 85 years.¹

With respect to prognostic factors, the only organ system to negatively influence the post-operative Oswestry Disability Index (ODI) score was musculoskeletal comorbidity, which also correlated with multilevel decompression.

This study shows that laminectomy can be a safe and effective option in this older age group, that it can provide sustained improvements in pain, disability, and quality of life (QOL) for those not responding to conservative treatment. Some subgroups may have only limited benefit from surgery, especially those with multilevel stenosis and coexisting musculoskeletal disease.

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- Dr David Roselt

Taylor RS, Van Buyten JP, Buchser E. Spinal cord stimulation for chronic back and leg pain and failed back surgery syndrome: a systematic review and analysis of prognostic factors. *Spine* 2005; 30(1):152-60.

Study Design: Systematic review.

Objectives: To assess efficacy and safety of spinal cord stimulation in

patients with chronic leg and back pain and failed back surgery syndrome and to examine prognostic factors that predict spinal cord stimulation outcome.

Summary of Background Data: A previous systematic review of spinal cord stimulation in patients with chronic back and leg pain and failed back surgery syndrome by Turner et al in 1995 identified 39 case studies and no controlled studies.

Methods: A number of electronic databases were searched through January 2002. Citation searching of included papers was undertaken, and gray literature was sought through contact with clinical experts. No language restrictions were applied. All controlled and non-controlled study designs were included. Study selection was carried out independently by two reviewers. Prognostic factors (age, sex, duration of pain, time post surgery, follow-up duration, publication year, data collection year, indication, data collection country, study setting, and quality score) responsible for pain relief outcome across case series were examined using univariate and multivariate meta-regression.

Results: One randomized controlled trial, one cohort study, and 72 case studies were included. The randomized controlled trial reported a significant benefit ($P = 0.047$) in the proportion of patients with failed back surgery syndrome reporting 50% or more pain relief with spinal cord stimulation (37.5%) compared with patients undergoing back re-operation (11.5%). There was evidence of substantial statistical heterogeneity ($P < 0.0001$) in the level of pain relief following spinal cord stimulation reported across case series studies. The four principal prognostic factors found to be predictive of increased level of pain relief with spinal cord stimulation were poor study quality score, short follow-up duration, multicenter (versus single center) studies, and the inclusion of patients with failed back surgery syndrome (versus chronic back and leg pain). Overall, 43% of patients with chronic back and leg pain/failed back surgery syndrome experienced one or more complications following spinal cord stimulation implantation, although no major adverse events were reported.

Conclusions: Despite an increase in the number of studies over the last 10 years, the level of evidence for the efficacy of spinal cord stimulation in chronic back and leg pain/failed back surgery syndrome remains "moderate." Prognostic factors found to be predictive of the level of pain relief following spinal cord stimulation were study quality, follow-up duration, study setting, and patient indication.

Comment: This study from the University of Birmingham, UK, is a systematic review. There is one randomized controlled trial (RCT) available, not placebo controlled, but it showed a statistically and clinically significant improvement against the control group of more surgery. This is a moderately high incidence of minor complications with spinal cord stimulation (SCS) but it is safe in skilled hands. Careful attention to follow-up is important as with most things medical. It is a clearly a valuable addition to the armamentarium especially for the failed back surgery syndrome (FBSS) which is despairingly common. However it is not first- or second-line treatment and should not be embarked upon without an adequate trial of more conservative, less invasive approaches such as employed by the dedicated musculoskeletal medicine practitioner. A trial of paraspinal injection of local anesthetic with or without corticosteroid or glucose solution and consideration for medial branch blocks as highlighted in this and recent editions of *Australasian Musculoskeletal Medicine*¹⁻¹⁹ should be mandatory first.

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- Dr David Roselt

Ng L, Chaudhary N, Sell P. The efficacy of corticosteroids in periradicular infiltration for chronic radicular pain: a randomized, double-blind, controlled trial. *Spine* 2005; 30(8): 857-62.

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

Objectives: To determine the treatment effect of corticosteroids in periradicular infiltration for chronic radicular pain. We also examined prognostic factors in relation to the outcome of the procedure. **Summary of Background Data:** Various studies have examined the therapeutic value of periradicular infiltration using treatment agents consisting of local anesthetic and corticosteroids for radicular pain, secondary to lumbar disc herniation and spinal stenosis. There is currently no randomized trial to determine the efficacy of a single injection of corticosteroids for chronic radicular pain.

Methods: Eligible patients with radicular pain who had unilateral symptoms who failed conservative management were randomized for a single injection with bupivacaine and methylprednisolone or bupivacaine only. Outcome measures included the Oswestry Disability Index (ODI), visual analogue scale (VAS) score for back pain and leg pain, claudication walking distance, and the patient's subjective level of satisfaction of the outcome.

Results: We recruited 43 patients in the bupivacaine and methylprednisolone group and 43 patients in the bupivacaine only group. The follow-up rate is 100%. Five patients had early termination of the trial for discectomy and further root block. There is no statistically significant difference in the outcome measures between the groups at 3 months (change of the Oswestry Disability Index [$P = 0.68$],

change in visual analogue score [back pain, $P = 0.68$; leg pain, $P = 0.94$], change in walking distance [$P = 0.7$]). Duration of symptoms has a statistically significant negative association with the change in Oswestry Disability Index ($P = 0.03$).

Conclusion: Clinical improvement occurred in both groups of patients. Corticosteroids did not provide additional benefit.

Comment: This randomized double-blind controlled trial without a placebo arm from University Hospitals of Leicester, UK, had 100% follow up at three months. Radicular pain is thought to be due to ectopic firing from a nerve root.^{1, 2}

Periradicular infiltration with local anesthetic and steroid delivered to targeted pathology under fluoroscopic guidance is an alternative to the translaminal epidural route. This allows neurograms illustrating satisfactory contrast distribution at the target disc nerve or foramen nerve interface. It is also called alternatively transforaminal epidural steroid injection and selective nerve root block. It has been utilized for radicular pain secondary to lumbar disc herniation (LDH) and peripheral foraminal stenosis. Experimental studies have shown that radicular pain occurs from mechanical compression and chemical radiculitis.³⁻⁵

Both phospholipase A2 (PLA2) and prostaglandin E2 have received attention with respect to the development of clinical radiculopathy.^{6, 7}

Local delivery of corticosteroid with both anti-inflammatory⁸ and local anesthetic properties⁹ to the affected nerve root appeared to be a reasonable option.

Triamcinolone acetonide, a corticosteroid, has been shown to provide rapid inhibition of ectopic firing provoked by PGE2.¹

In view of the rapid effect, a direct action of corticosteroids on the neural membrane rather than an anti-inflammatory effect has been postulated.¹⁰

A randomized controlled trial with 160 patients has shown that a single injection lasts no more than four weeks.¹¹

Other studies have shown long-term results with multiple injections.¹²⁻¹⁴

This study failed to show any extra benefit from addition of corticosteroid to local anesthetic bupivacaine alone for periradicular epidural block for chronic radicular pain. There was a modest reduction in VAS for both groups at three months. The authors thought this may be due to the fact that the majority of patients had chronic symptoms. There was no difference in outcome between the patients with radicular pain secondary to LDH as compared to foraminal stenosis. Multiple regression analysis showed a negative association between duration of symptoms and change in ODI at three months. A prolonged duration of symptoms also was associated with poorer outcomes of the procedure in other studies.

Chronic nerve root irritation can induce peripheral and central sensitization of the nervous system and lead to neurogenic pain.¹⁵

The study by Riew demonstrated effectiveness of corticosteroids in a double-blind randomized controlled trial.¹³

This study found 67% of patients receiving local anesthetic and steroid avoided surgery compared to 28% in the local anesthetic only group. The positive treatment effects may be from cumulative injections as the patients received further treatment if the response was judged inadequate.

In this study periradicular infiltration provided a sustained reduction in radicular pain and disability at three months. Addition of corticosteroids did not produce additional benefit, but this could be related to the chronicity of the radicular pain in the patients studied. It is possible that benefit from addition of steroid may still be achieved in cases of more recent onset.

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- Dr David Roselt

Chiu TT, Lam TH, Hedley AJ. A randomized controlled trial on the efficacy of exercise for patients with chronic neck pain. *Spine* 2005; 30(1): E1-7.

Study Design: A randomized controlled trial with single-blind outcome assessments.

Objective: To evaluate the efficacy of a neck exercise program in patients with chronic neck pain. **Summary of Background Data:** The effect of exercise for patients with chronic neck pain has been investigated in a number of studies. The efficacy is, however, questionable.

Methods: A total of 145 patients were randomly allocated into an exercise (n = 67) and a non-exercise (control) group (n = 78). Patients in the control group were given infrared irradiation and neck care advice. In addition to infrared irradiation and advice, patients in the exercise group had undergone an exercise program with activation of the deep neck muscles and dynamic strengthening of the neck muscles for 6 weeks. Subjective pain and disability and isometric neck muscle strength were measured at baseline, 6 weeks, and 6 months. Analysis was by intention-to-treat.

Results: At week 6, the exercise group had a significantly better improvement in disability score (P = 0.03), subjective report of pain (P = 0.01), and in isometric neck muscle strength (P = 0.57-0.00) in most of the directions than the control group. However, significant differences between the two groups were found only in the subjective report of pain and patient satisfaction at the 6-month follow-up.

Conclusions: At week 6, patients with chronic neck pain can benefit from the neck exercise program with significant improvement in disability, pain, and isometric neck muscle strength in different directions. However, the effect of exercise was less favorable at 6 months.

Comment: Exercise therapy has not been a huge success story in chronic pain although it normally scores well regarding patient satisfaction. This study compared isometric and deep flexor exercises supervised 2 x week for six weeks to infrared and neck care

advice. Physiotherapists supervised the exercise and an air-filled pressure sensor was used for feedback. Both groups showed significant improvements in the first six weeks with the exercise group superior. After cessation of the program, there was no discernible difference between the two groups at six months. Whether the cost of the intervention is justifiable for short-term gains only is debatable.

- Dr Scott Masters

Bergman GJ, Knoester B, Assink N et al. Variation in the cervical range of motion over time measured by the "flock of birds" electromagnetic tracking system. *Spine* 2005; 30(6): 650-54.

Study Design: Observational longitudinal study.

Objective: To establish the normal variation over time for active and passive cervical range of motion (ROM) measured with the Flock of Birds electromagnetic tracking system (FOB).

Summary of Background Data: Data about normal variation of cervical ROM over time are scarce but important for the interpretation of study results.

Methods: Forty-eight subjects without a manifest dysfunction in neck and shoulder region (asymptomatic group) and 58 subjects with a dysfunction in the neck and shoulder region (symptomatic group) participated in this study. Cervical active and passive ROM was assessed in three different sessions 6 weeks apart. The following movements were measured: flexion-extension, lateral bending, and axial rotation in neutral, flexed, and extended position.

Results: A wide range of variation of active and passive cervical ROM was found at the 6- and 12-week measurement in the asymptomatic group as well as in the symptomatic group. Highest variation was found during passive ROM testing as compared with active ROM testing. The symptomatic group showed larger variation than the asymptomatic group.

Conclusions: Cervical range of motion varies considerably over time. This variation should be taken into account when results of therapeutic trials with respect to cervical ROM are

interpreted.

Comment: This is a very important study with respect to reliability of assessment of range of movement (ROM) measurements which have been used as outcome measures especially in studies of neck pain assessment and treatment. It confirms my clinical impression that reproduction of pain and tenderness may be more reliable and useful. As pointed out by Bogduk and Mercer, "A lower range today, a higher range tomorrow, or *vice versa*, could be only the normal, diurnal variation and not something attributable to a disease or a therapeutic intervention."¹

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- Dr David Roselt

Gun RT, Osti OL, O'Riordan A et al. Risk factors for prolonged disability after whiplash injury: a prospective study. Spine. 2005 Feb 15;30(4):386-91.

Study Design: A prospective study of 135 patients with whiplash injury.

Objectives: To identify factors predictive of prolonged disability following whiplash injury.

Summary of Background Data: Although patients with whiplash associated disorders lack demonstrable physical injury, many exhibit prolonged disability. Disability appears unrelated to the severity of the collision.

Methods: A total of 147 patients with recent whiplash injury were interviewed for putative risk factors for disability, and 135 were reinterviewed 12 months later to assess degree and duration of disability. Bivariate and multivariate analyses were undertaken to measure the association between putative risk factors and measures of outcome (change in Neck Pain Outcome Score [NPOS] and visual analogue pain score [VAPS], return to work, still requiring treatment, settlement of claim).

Results: The bodily pain score and role emotional scores of the Short Form-36 health questionnaire showed a consistent significant positive association with better outcomes. After

adjustment for bodily pain score and role emotional scores, consulting a lawyer was associated with less improvement in NPOS ($P < 0.05$), but there was no association with change in VAPS. Consulting a lawyer was associated with a lesser chance of claim settlement ($P < 0.01$) and a greater chance of still having treatment ($P < 0.01$) after 1 year, but there was no significant association with a return to work. The degree of damage to the vehicle was not a predictor of outcome.

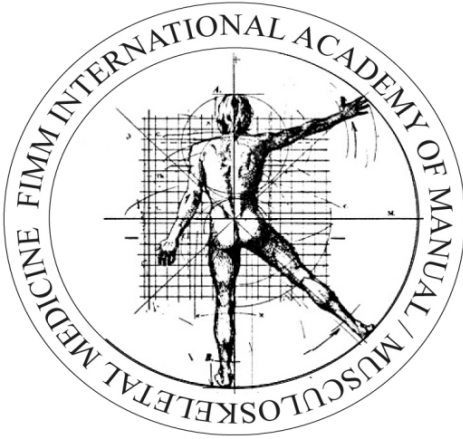
Conclusions: Short Form-36 scores for bodily pain and role emotional are useful means of identifying patients at risk of prolonged disability. The findings support the implementation of an insurance system designed to minimize litigation.

Comment: This interesting study from the Department of Public Health at the University of Adelaide, South Australia, provides sound evidence for what many working in the pain field have sensed regarding the effects of litigation on prolonging pain, disability, and suffering.

Multivariate analysis revealed consulting a lawyer was associated with a 6-point lower NPOS at the end of one year ($P < 0.05$), a seven-fold lesser chance of claim settlement ($P < 0.01$) and a seven-fold greater chance of still having treatment ($P < 0.01$) after one year after adjustment for bodily pain and role emotional. This is good evidence that lawyers have an independent adverse effect on prognosis that is at least partly causal.

Patients treated by a physiotherapist or chiropractor showed statistically less improvements in NPOS and VAPS. They were more likely to still be receiving treatment, medical or otherwise, but there was no association with return to work or settlement of claim after one year.

The importance of reassurance, education, adequate analgesia, continuation of usual activities of daily living (ADLs) as much as possible, and early mobilization with simple stretching utilizing post-isometric muscle relaxation from deep breathing, cannot be overemphasized for neck pain and whiplash injury. - Dr David Roselt



FIMM International Academy of Manual/ Musculoskeletal Medicine

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Jacob Patijn, Neurologist, Physician
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Chairman Executive Board

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The *FIMM* Academy, instituted in September 2004, provides a forum for collaboration and consensus debate in manual/musculoskeletal medicine (M/MM).

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Applications for membership are invited from medical practitioners engaged in the practice of manual, musculoskeletal, orthopedic, or osteopathic medicine, or in medical practice that impacts on M/MM, and university graduates in disciplines allied to M/MM.

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The New Zealand Association of Musculoskeletal Medicine

ANNUAL CONFERENCE 23rd - 26th June 2005

St. David St Lecture Theatre, Otago University, Dunedin, New Zealand

Shoulder Pain and Disorders

The Full Monty

Keynote Speakers

Mr Bruce Twaddle	Auckland NZ
Dr Sue Mercer	Dunedin NZ
Dr Quentin Reeves	Auckland NZ
Assoc Prof John Cronin	Auckland NZ

Programme

Thursday 23rd June

8.00 am-10.00 am	12.00pm – 6.00pm	Conference Registration
8.30 am - 11.00 am	NZ Association of Musculoskeletal Medicine meeting (NZAMSM members only) Anatomy Dept Otago Medical School. Anatomy Shoulder Practical.	
11.15 am - 1.00 pm	NZAMSM AGM. St David St Lecture Theatre seminar room.	

Thursday 23rd June

2.00 pm	Conference Opening Welcome address: Dr John Robinson
2.15 pm	ACC and Shoulder Injury: Dr Kevin Morris
2.35 pm	Shoulder Anatomy/Functional Anatomy: Dr Sue Mercer
3.35 pm	Afternoon Tea
3.55 pm	Myofascial Triggerpoints in Relation to Muscle Balance. Posture & Respiration affecting the Shoulder: Ietje van Stolk
4.45 pm	Prognostic Risk Factors in Shoulder Disorders. Psychosocial Factors: Dr Jonathan Kuttner
5.15 pm	Panel Discussion

Friday 24th June

08.30 am	Shoulder Biomechanics: Dr Sue Mercer
09.30 am	Acute Traumatic Shoulder Injuries: Dr Ra Durie
10.30 am	Morning Tea
11.00 am	The Origin of Shoulder Pain. Neurophysiological relationships between the Cervical Spine and Shoulder, Viscerosomatic Shoulder Pain: Dr Gireesh Kanji
11.45 pm	Myofascial Shoulder Pain: Diagnosis and Treatment: Dr Peter Jackson
12.30 pm	Lunch
1.30 pm	Tendinopathies and Rotator Cuff Disorders. A/C joint Pathologies: Mr Bruce Twaddle
2.20 pm	Impingement Syndromes. Is there a place for corticosteroid injection?: Dr Mark Johnston
3.15 pm	Afternoon Tea
3.35 pm	Neurological Disorders Affecting the Shoulder, Peripheral Neuropathies: Dr Vic du Plessis
4.30 pm	Neurophysiological Testing of Shoulder Problems
5.15 pm	Panel Discussion

05.30- 6.30pm Munslow's Otago Wine tasting

Saturday 25th June

08.30 am	Imaging of the Shoulder. Part I Plain Radiology: Dr Quentin Reeves
09.30 am	Evidence on Shoulder Diagnosis and Treatment, Reliability/Validity: Dr Wade King
10.30 am	Morning Tea
10.50 am	Imaging of the Shoulder. Part II: Ultrasound and MRI Scan Shoulder: Dr Quentin Reeves
11.50 am	Rheumatological Shoulder Disorders, Frozen Shoulder, Osteolysis Clavicle: Dr Guy Taylor
12.30 pm	Lunch
1.30 pm	Shoulder Instability: Mr Bruce Twaddle
2.30 pm	Post – op Shoulder Rehabilitation: Andrea Mosely
3.10 pm	Afternoon tea
3.30 pm	Shoulder Strength and Conditioning: Assoc Prof John Cronin
4.30 pm	Thoracic Outlet Syndrome: Dr Steve Bentley
5.15 pm	Panel Discussion

Sunday 26th June

09.00 am	Workshops. 2 streams 30 mins each
1.	Biomechanics & Examination of the Shoulder: Dr Jonathan Kuttner
2.	Shoulder Strength and Conditioning: Assoc Prof John Cronin
10.00 am	Morning Tea
10.30 am	Workshops. 3 streams 30 mins each
3.	Myofascial Triggerpoints in Relation to Muscle Balance: Ietje van Stolk
4.	Diagnosis and Treatment of Myofascial Shoulder Pain: Dr Peter Jackson
5.	Treatment of Frozen Shoulder: Dr Clemens Franzmayr

12.00pm Close: Dr John Robinson

Conference enquiries

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Dr Sue Mercer. Dept of Anatomy & Structural Biology, University of Otago, Dunedin NZ.

Mr Bruce Twaddle. Orthopaedic Surgeon. Auckland. NZ

Dr Quentin Reeves. Radiologist. Auckland

Assoc Prof John Cronin. Director of Sport Performance Centre. AUT. Auckland NZ

Dr Wade King. Musculoskeletal Physician. Newcastle Australia

Dr Peter Jackson. Musculoskeletal Physician. Brisbane. Australia

Dr Vic du Plessis. Neurologist. Dunedin

Dr Ra Durie. Sports Physician. Palmerston North NZ

Dr Mark Johnston. Musculoskeletal Physician. Auckland NZ

Dr Johnathan Kuttner. Musculoskeletal Physician. Auckland NZ

Dr Guy Taylor. Rheumatologist. Wanganui. NZ

Dr Steve Bentley. Musculoskeletal Physician. Dunedin NZ

Dr Giresh Kanji. Musculoskeletal Physician. Wellington NZ

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Shoulder Pain and Disorders – The Full Monty

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- NZAMSM
- AFMM
- RNZCGPs

If you require a CME certificate of attendance please circle **YES** if not circle **NO**

Conference Registration Fees

	Conference Days Attending (Tick)	NZAMSM Member	NZAMSM Member Register before 1/5/05	NZAMSM Non Member	NZAMSM Non Member Register before 1/5/05	Students (ID required)	Students (ID required) Register before 1/5/05
Thursday 23/6/05		\$125.00	\$110.00	\$145.00	\$130.00	\$75.00	\$65.00
Friday 24/6/05		\$160.00	\$145.00	\$175.00	\$160.00	\$100.00	\$90.00
Saturday 25/6/05		\$160.00	\$145.00	\$175.00	\$160.00	\$100.00	\$90.00
Sunday 26/6/05		\$125.00	\$110.00	\$145.00	\$130.00	\$75.00	\$65.00
All 4 days Whole Conference		\$550.00	\$500.00	\$600.00	\$550.00	\$300.00	\$275.00

Total Conference Fee: **NZ\$**

Notes:

- All fees are in NZ Dollars.
- Overseas delegates are requested to send registration fee as a bank draft in NZ Dollars.
- Discounted conference fee (see table) applies **if payment is made before 1/5/05**.
- Students must supply written documentation of current full time University enrolment to qualify for the reduced fee
- NZAMSM members qualify for a reduced fee (see table).
Membership of NZAMSM is restricted to Registered NZ Medical Practitioners.
For membership enquiries contact:
Dr Gary Collinson. Secretary NZAMSM. 4 Lynbrooke Ave, Blockhouse Bay Auckland. Ph: Bus 09 6279205. Fax: 09 6271181

Please send your registration form completed with payment to:

The NZAMSM Conference Organiser: Dr Steve Bentley. Attention "Joanne"

Postal Address: Suite 19 Marinoto Clinic, 72 Newington Ave, Dunedin, NZ

Ph: (+64) 03 4672046, **Fax:** (+64) 03 4672042

Educational Activities

MASTERS, DIPLOMA, AND CERTIFICATE COURSES IN MUSCULOSKELETAL MEDICINE

FLINDERS UNIVERSITY DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
22-26/6/05	Dysfunction of the vertebral column	Flinders Medical Centre	Flinders University	A/Prof Norm Broadhurst Ph: +61 8 8295 1890 Fax: +61 8 8295 6808 Email: norm.broadhurst@flinders.edu.au	tba
14-18/6/05	Dysfunction of the appendicular skeleton	Flinders Medical Centre	Flinders University	A/Prof Norm Broadhurst	tba

UNIVERSITY OF OTAGO DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
21/7/05-27/10/05	MSMX 702 - MSM tissues & MSMX 703: MSM disorders	Distance taught paper - fortnightly audioconferences	University of Otago	New enrolments for second semester 2005 close 1 June Veronica McGroggan Tel. +64 3 364 1086 Fax +64 3 364 0909 Email: veronica.mcroggan@chmeds.ac.nz or	Mixture of points including small group points
8-12/8/05	Pt 2 - MSMX701 on campus course	Christchurch School of Med. & Health Science	University of Otago	Geoff Harding Tel. +61 7 32695522 Fax +61 7 32696407 Email: geoffharding@uq.net.au website: www.chmeds.ac/departments/msm/	
?Late Sept/early Oct	Pt 2 - MSMX701 on campus course	Brisbane			

UNIVERSITY OF NEWCASTLE MASTERS IN PAIN MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
2005	Masters in Pain Medicine	Internet	University of Newcastle	Email Cath.Williams@newcastle.edu.au - administrative liaison at Uni of Newcastle or Phillipa.Powis@newcastle.edu.au for information about the course	N/A

OTHER MUSCULOSKELETAL MEDICINE EDUCATIONAL ACTIVITIES

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
23-26/6/05	Annual conf. of NZAMM - Shoulder Pain and Disorders: The Full Monty	St. David St Lecture Theatre, Otago University, Dunedin, NZ	NZAMM	The Conference Organiser. Dr Steve Bentley or Joanne Suite 19 Marinoto Clinic 72 Newington Ave, Dunedin Ph: +64-3 4672046. Fax: +64-3 4672042 NZAMSM website: www.musculoskeletal.co.nz	N/A for Australians
24-26/6/05	Prolotherapy Workshop	Adelaide	Margaret Taylor	Phone 0404092899; email taylorme@internode.on.net website: Prolotherapy for Drs www.drmtaylor.com.au	
9-10/7/05	Complementary Medicine for Musculoskeletal Disorders Workshop	Adelaide	Margaret Taylor	Phone 0404092899; email taylorme@internode.on.net website: Prolotherapy for Drs www.drmtaylor.com.au	
21-26/8/05	11 th World Congress on Pain	Sydney Convention Centre, Darling Harbour	International Association for the Study of Pain	IASP Secretariat, 909 NE 43 rd St, Suite 306 Seattle, WA 98105-6020 USA Ph: +1-206 547 6409 Fax: +1-206 547 1703 Email: iaspdesk@juno.com URL: www.iasp-pain.org	N/A
27-28/8/05	International Conference on orofacial pain and tempero-mandibular disorders	Sydney Convention Centre, Darling Harbour		DC Conferences, PO Box 571, Crows Nest, NSW 1585 Ph 02 9954 4400 Fax 02 9954 0666	N/A
27-31/8/05	Blomberg course on the Stayac algorithm	Riverglenn Conference Centre Indooroopilly Brisbane	AAMM	Rebecca Fielding, Qld Conferencing & Events, rffielding@qce.net.au Ph: +61 7 3236 9673 Fax: +61 7 3831 0999	Application to RACGP for 2 CPD points per hour
8-9/10/05	Chiropractic & Osteopathic College of Australasia (COCA) national conference	Rydges Melbourne, 186 Exhibition St Melbourne, Vic 3000		Mr Alan Ralph, Executive Secretary, PO Box 1010 Ringwood, Vic. 3134 Ph: 1300 13 99 50 Fax: 1300 88 66 90; email: info@coca.com.au http://www.coca.com.au/conferences.htm	