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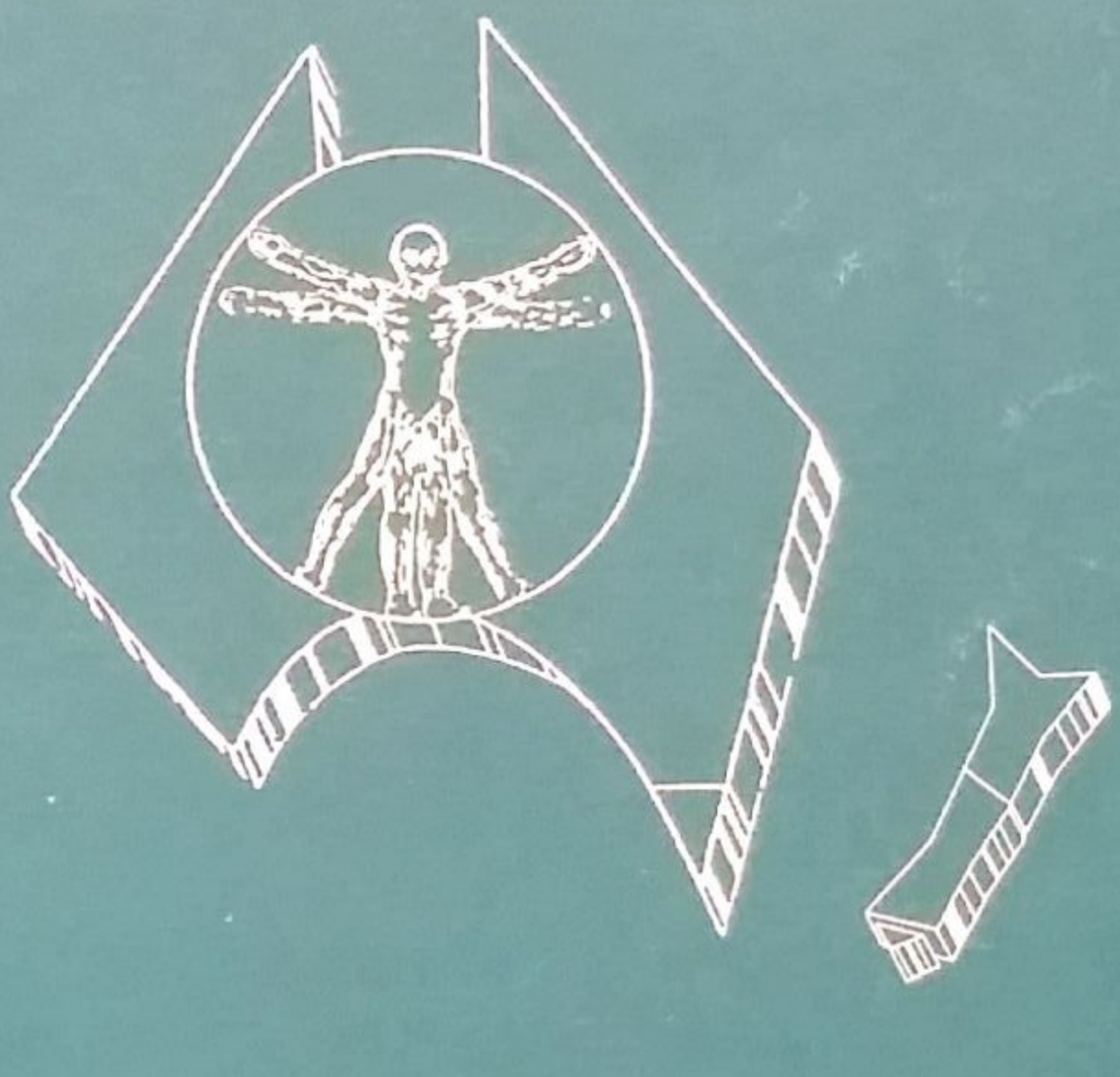

- Mid-Line Thoracic Pain
- Truth in Musculoskeletal Medicine
- Prolotherapy in Lumbo-Pelvic Pain
- Systematic Review of Treatments for "Tennis Elbow"
- Complex Regional Pain Syndrome: Case Study
- Medicolegal Brief
- Case Commentary: Low Back and Leg Pain

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Australian Association of Musculoskeletal Medicine

Office Bearers 1996 - 1997

President
Dr Vic Wilk MB BS, Grad Dip Musc Med
441 Bay Street, Brighton Vic 3186
Telephone: +61 3 9596 7211, +61 3 9690 1052 (h)
Fax: +61 3 9596 7871

Vice-President
Dr Ron Palmer MB BS, Licentiate AAMM, Grad Dip Musc Med
Suite 24 Royal Brisbane Place, 17 Bowen Bridge Road, Herston Qld 4006
Telephone: +61 7 3252 1128 (w), +61 7 5494 9783 (h)
Fax: +61 7 3852 1561

Honorary Secretary
Dr Neil Hearnden MB BS, FRACGP, Cert Man Med (RACGP), FACPM, Licentiate AAMM, Grad Dip Musc Med
2 Nepean Ave, Arana Hills Qld 4054
Telephone: +61 7 3351 6444 (w), +61 7 3351 1747 (h)
Fax: +61 7 3351 1747

Honorary Treasurer
Dr Max de Clifford MB BS, FAMAS, FRACGP, DROCG
36 Heads Road, Donvale Vic 3111
Telephone: +61 3 9873 2537

Councillors and Subcommittee Members

Dr Geoff Harding	
Sandgate Qld 4017	+61 7 3269 1842
Dr Arnold Jones	
Karragullen WA 6115	+61 8 9390 4444
Dr Wade King	
Taree NSW 2430	+61 65 551 0662
Dr Ian McIntyre	
Port Augusta SA 5700	+61 8 8642 6666
Dr Bob Michael	
Sunnybank Qld 4109	+61 7 3345 8999
Dr Norman Broadhurst	
Glenelg SA 5045	+61 8 8295 1890
Dr Michael Yelland	
Inala Qld 4077	+61 7 3275 5444

New Zealand Association of Musculoskeletal Medicine

Office Bearers 1996 - 1998

President / South Island Education Convenor
Dr Jim Borowczyk
79 Wilson's Road, St Martin's, Christchurch
Telephone: + 64 3 337 0991
Fax:+ 64 3 337 0991

President-elect / South Island Assoc. Treasurer
Dr Joe Brownlee
256 Papanui Road, Christchurch
Telephone: + 64 3 355 7038
Fax: + 64 3 355 7038

Secretary
Dr John Malloy
201 Mt Eden Road, Auckland 3
Telephone: + 64 9 631 5863
Fax: + 64 9 630 0146

Treasurer and North Island Convenor
Dr Mark Johnston
16 Moana Avenue, Orewa
Telephone: + 64 9 426 5437
Fax: + 64 9 426 5437

Academic Co-ordinator of Diploma
Dr Barry Tait
19 Stratford Street, Fendalton, Christchurch
Telephone: + 64 3 364 0430
Fax: + 64 3 364 0909

Past President
Dr Angus Johnson
126 Wade River Road, Whangaparaoa
Telephone: + 64 9 424 7912
Fax: 64 9 426 5437

Committee Members

Dr J Watt	
Takapuna	+ 64 9 489 5059/Fax +64 9 486 4973
Dr C Franzmayr	
Christchurch	+64 3 355 7080/Fax + 64 3 355 7080
Dr S Bentley	
Dunedin	+64 3 479 8212
Dr Paul Quin	
Auckland	+ 64 9 520 4761
Dr John Robinson	
Christchurch	+ 64 3 338 6408/Fax: + 64 3 332 9669
Dr Grant Thomson	
Auckland	+ 64 9 435 1269/Fax: + 64 9 435 2634

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Editorial: In Search of the Evidence

Dr Michael Yelland

Since the last issue of this journal many of us have attended the very successful Australian Association of Musculoskeletal Medicine conference in Melbourne. The organisers deserve hearty congratulations on their preparation and presentation of a program which focused on the current state of knowledge and practice in the area of low back pain. This year's program was largely evidence-based and had some sobering messages about some of the clinical practices we feel work well in our hands. It was depressing for many to see the paucity of evidence for the accuracy and efficacy of what we do everyday. However before giving up practice in the belief that you have traded your comfortable old slippers for a pair of new regulation army boots it is worth reading the book *Evidence-based medicine* by Sackett, et. al[1] to put any perceived threat into perspective.

Evidence-based medicine does not replace clinical expertise, rather it acts to augment it. The practice of evidence-based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research. It is pragmatically about improving outcomes for patients. It does not necessarily prove why a particular technique works - simply whether it works. So while there is some evidence (albeit imperfect) that manipulation is useful for acute back pain, we are still not sure whether it is the physical techniques that effect the change or the charisma of the therapist practising the techniques. There are a lot of gurus out there in the health care maze who claim that their techniques work wonders for musculoskeletal disorders, but the common ground for their success may be the confidence with which they practise their art and the reassurance they give to their customers.

The Department of Family Services & Health has recently introduced a new requirement for the provision of evidence of the efficacy and cost effectiveness of new services for which application for an item number is being made. Fifteen million dollars is available over the next four years to fund research projects which examine this area, a clear signal that the Department wants some return for its money. One wonders if the next step will be to remove any existing services for which evidence cannot be provided. The Department has also provided significant funding for the National Musculoskeletal Medicine Initiative to evaluate evidence-based musculoskeletal services in hospital and community settings. It seems whether we like it or not, in this era of budgetary restraint, evidence-based medicine is the way of the future. One hopes that the government's motives are not purely to restrain the burgeoning costs of health care in this country. In identifying and applying the most efficacious interventions to maximise the quality and quantity life for individual patients, the evidence-based approach can potentially increase costs.[1]

Hence there are sound clinical and political reasons to provide the readership of this journal with the best evidence with which to practise. One way of ensuring this would be to upgrade the journal to a refereed publication by appointing an editorial board to review articles. Articles submitted would be passed for review to people with specific areas of expertise to assess their suitability for publication. This suggestion was discussed at the last annual general meeting of the Australian Association of Musculoskeletal Medicine. It was thought that at the present there would not be enough material of a standard that would survive a very rigorous review process. It may be akin to shooting oneself in the foot, if not the heart.

As I stated in my last editorial I am broadening the scope of the journal to promote more debate and provide more practical information. One example of relevance here is the introduction of a medicolegal briefs section in this issue to help readers survive in the increasingly litigious world in which we practise. At the same time I am encouraging the publication of literature reviews and research articles. In this issue we have a systematic review of treatments for tennis elbow which critically appraises the recent literature in this area. The journal should also assist the readers to sharpen their skills in critical appraisal of material in the journal and in other parts of the medical literature. To this end Prof. Nikolai Bogduk has contributed an article on coming to grips with confidence intervals, the first in a series of four articles, "Truth and musculoskeletal medicine". These are long articles but well written, with plenty of examples to facilitate understanding.

When reading this issue one should be constantly questioning whether the material falls in the category of evidence based on sound research, proof extrapolated from a sound basic science base or opinion/conjecture. All are worth reading but the latter two await further rigorous testing in practice.

I hope this will stimulate you to reflect on the basis on which you practice your art and/or science, how you evaluate your practice and finally to contribute to a more lively "Letters to the Editor" section. Read on with a discerning mind!

1. Sackett DL, Richardson WS, Rosenberg W, Haynes RB *Evidence-based medicine. How to practice and teach EBM.* Churchill Livingstone, Melbourne 1997.

From the AAMM President, Dr Vic Wilk

My aim as the incoming president was to re-focus our teaching programs back towards the average general practitioner putting the emphasis back on education and updating of GPs' knowledge on common musculoskeletal problems. This year's conference on Low Back Pain in General Practice was specifically put together with this in mind. An extensive advertising campaign via division newsletters resulted in an expression of interest from over 400, with 75 non-members attending in Melbourne. Hopefully many of these will subsequently join the association for ongoing training and bolster our numbers.

Melbourne Conference. A total of 170 registrants attended of which 73 also attended the practical weekend workshops. From the feedback it seems the workshops were a big hit. Thank you to all those involved in helping make the conference a big success. For those who couldn't attend, there are conference tapes available by filling out the order form inserted with this journal.

Membership. It is of some concern that the association membership has been static over the last few years. More attention needs to be addressed to marketing issues if we are again to grow. One key aspect is the maintenance of comprehensive databases of members and potential members and the adequate advertising of meetings and workshops. Better communication has been made possible via e-mail and the association database is being distributed to all states to streamline contact with members. It has been suggested that we may like to join forces with paramedicals or the sports physicians to broaden our base. Amalgamation with the College of Physical Medicine would be a good start.

Journal. The journal has maintained its high standard under the new editor Michael Yelland, who has done an outstanding job. Michael is keen to establish an editorial board to further enhance the reputation of the journal. Members are encouraged to contribute anything of general interest. This year, in addition to the two journals published, two newsletters have been published with an emphasis on information on upcoming association activities and "tidbits" of information. It is proposed to have six mailings in total each year to keep in closer touch with the members.

FIMM Conference. We have been given the privilege to host the FIMM conference at the Gold Coast over Easter 1998. This is the major international musculoskeletal medicine conference normally held in Europe. Philip Watson together with the conference committee and Carillon the conference organisers have put together an exciting program with many famous international speakers. To be a success it requires participation by all members in every state.

Clinics. The other big news is the establishment of the National Musculoskeletal Health Initiative and the opening of musculoskeletal clinics in public hospitals around Australia. The first of these in New South Wales to be opening about now. Special thanks to Wade King and Nik Bogduk for all their tireless work.

Specialist status. With the increasing number of diploma graduates and the formation of the faculty, specialist status is a real probability. This will depend in part on the success of the clinics and will remain within the realm of the faculty. The Association of Musculoskeletal Medicine must retain its close contact and focus with the average general practitioner, providing a valuable resource and co-ordinating educational programs. All those members involved in teaching and those with post-graduate diplomas in musculoskeletal medicine are encouraged to join the faculty and contribute.

The future. David Collinson has agreed to organise the conference in 1999 in Sydney and is looking for any helpers.

From the NZAMM President, Dr Jim Borowczyk

Times of Change

The nature of musculoskeletal medicine continues to change. With the developments of the late 70s and the early 80s, which saw the Antipodes exposed to a precise but finite view of manual medicine from the Northern Hemisphere, our understanding of matters musculoskeletal has evolved.

In the late 1980s, Barrie Tait, of the Christchurch School of Medicine, and under the auspices of the University of Otago, succeeded against considerable odds in producing the first recognised postgraduate Diploma in Musculoskeletal Medicine - musculoskeletal medicine being at that time a term unrecognised by the medical profession at large.

Since then, there have been major developments. We now find ourselves with well-established and active Associations of Musculoskeletal Medicine on both sides of the Tasman, initially models of the European communities of manual medicine. There are associated colleges within the profession, particularly physical medicine and rehabilitation medicine; we find ourselves annexed to similar bodies of enthusiasts from around the world, the largest being the International Federation of Manual Medicine (FIMM); and there are currently four separate diplomas in musculoskeletal and manual medicine in Australasia alone. In the Northern Hemisphere, journals dealing with musculoskeletal topics and educational courses in manual and musculoskeletal medicine continue to proliferate.

What is our position in this global status, and what now constitutes musculoskeletal medicine? The term and its nature continue to defy precise definition. More particularly, the body of knowledge of the discipline continues to defy accurate description and consensus within our relatively narrow (Australasian) experience. My particular perspectives are:

- Musculoskeletal medicine should deal with all matters related to the neuro-musculoskeletal system
- The promulgation of matters musculoskeletal in the Southern Hemisphere resides with the appropriate (expert) bodies that have sway. These are the Associations (Australia and New Zealand), the Diplomas (Australia and New Zealand), and the Faculty.

These bodies, which call upon considerable man/interest/years, are currently best suited to the further development of the discipline.

Levels of educational hierarchy have evolved. Initially, the Associations were the primary providers of musculoskeletal education, and in particular, manual therapies. The Diplomas then provided an expanded theoretical base to the discipline, and at the same time, real practical skills, both in manual and associated physical techniques. The Faculty now pretends to oversee these two bodies intellectually, if only on a theoretical basis i.e. on a scientific evidence-based basis. In addition the Faculty is developing an instrument to qualify practitioners who are capable of displaying a level of excellence acceptable to our appropriate peer groups.

This is all quite appropriate. However, it will require the devolution of Association educational activities to one of support, to providing basic or introductory skills, and perhaps to furthering specific physical skills gained from some other facility. The Diplomas will continue to provide theoretical and physical instruction at an intermediate level, at the same time providing an entry level to the Faculty for those not already in possession of an appropriate specialist registration.

The Faculty in the meantime, with the Australian National Musculoskeletal Medicine Initiative, will conduct one of the most promising research projects ever undertaken in musculoskeletal medicine. This will almost certainly have a fundamental impact on the nature of the discipline, and on the way it is practised, well into the next millennium. I suspect it may well do for musculoskeletal medicine what Framingham did for cholesterol!

It is up to all of us who profess musculoskeletal skills, to keep abreast of these changes. It is just as important that we continue to unify the discipline, and avoid the fragmentation that has been a frequent source of conflict in the past. On behalf of NZAMSM, may I express our commitment to change, to ongoing research and teaching, and to furthering the association that we already have with the Australian Initiative.

Letters to the Editor

Dear Sir

I would like to make a number of comments with regard articles in the April 1997 Journal. Firstly, I would agree with Dr de Ruyter that a 12 page article on articular cartilage seems excessive but I would disagree with a number of other points he made.

Will we see musculoskeletal medicine become irrelevant to those in general practice or see the demise of AAMM? I have a group of half a dozen GP friends, none of whom had ever heard of AAMM till I joined and who felt musculoskeletal medicine was the realm of orthopedic surgeons. To became irrelevant you have to be known in the first place!

I agree with the comments that we need to influence medical schools more, but before we teach medical students to manipulate, perhaps we should teach them how to examine, to develop soft tissue and articulation skills which are safe and effective and perhaps manipulation skills for those with interest and skills. I realise there are members running GP training courses, but these are not widely known, so could we expand this role.

Many of my GP friends are not interested in manipulation, but would like to be better at examining spines, shoulders or "chronic groin pain/ incipient inguinal hernia", which is why that article is relevant to our journal.

There is more to musculoskeletal medicine than "cracking backs". I think we need a mixture of science and technique in the journal for one without the other turns us into technicians, no more.

This leads me to make some quasi-scientific comments on the case discussions of Dr Devaux. The S/I joint as we all know is a much argued area even among osteopathic practitioners. I found Dr Devaux's case a good summary of textbook cases. I would however suggest that superior innominate shears are not all that common, even in chronic low back pain but reflect often other imbalances.

Greenman[1] in his latest textbook introduces the S/I joint by saying the chapter is not based on any biomechanical or clinical evidence but on clinical observation, and I would add historic belief. He also states that the forward flexion test shows the side of the lesion, and is a test of the thoraco-pelvis - lower limb complex. The list of false positives is long, including leg-length inequality, tight hamstrings, quadratus lumborum, erector spinae or L5/S1 dysfunction, etc.

There have been studies by Denslow,[2] Beal[3] and others[4,5] that show that inter- and intraobserver reliability for S/I tests are terrible, often no better than chance. Osteopathic texts often quote that sacroiliac lesions are more common on the right (depending on which book you read). Brooke,[6] Sashin,[7] Mcdonald[8] and others[9,10] looked at S/I anatomy changes with age and showed the joint (right greater than left) stiffened as early as 30 in males and 50 in females, with 40% to 75% (depending on which study) of males ankylosed by 50. These changes must certainly influence results of S/I testing, but are largely ignored by teachers. These facts do not lessen the need to look for S/I lesions but should be kept in mind when assessing patients along side lists of structural diagnosis findings.

I think positional diagnosis (as against mobility testing) is fraught with difficulty. It was reassuring to see Professor Laurie Hartmann (British School of Osteopathy) in his latest textbook states "original osteopathic thinking was traditionally to reverse the path of a lesion. Although this is an attractive concept, it has become accepted that many cases do not require this type of analysis. A simple breaking of fixation in the direction of the optimum barrier is sufficient to improve range and quality of motion, and positional correction is often unnecessary and excessively complex."

I think this applies to most dysfunctions that occur within the physiological range of motion. Even among nonphysiological dysfunction such as superior innominate shears, suggested corrections are so nonspecific that they influence the entire lumbar spine and associated musculature.

Yours sincerely

PT Cleary

Toowoomba, Qld

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Letters to the Editor

Dear Sir

I recently had the privilege of attending a few talks by Alf Nachemson at a conference in Melbourne organised by the Chiropractic and Osteopathic College of Australasia (COCA). Prof Nachemson is an excellent and most entertaining speaker. Following are some amusing quotes from his talks:

1. Concerning back surgery: "The increase in the use of pedicle screws in the US mirrors the increase in the price of Mercedes Benz cars."
2. Concerning revision back surgery: "You can't unscrew what you've screwed up by screwing it again."
3. Concerning revision back surgery: "This is Colombus surgery - you don't know where you're going, you don't know where you are, you don't know where you've been."
4. Concerning new technology: "We are accelerating our ability to diagnose and treat irrelevant pathology."
5. Advice concerning treatment: "Use new medications quickly while they still work."
6. Concerning the relationship between insurance companies and health care providers: "When they have you by the balls, then your hearts and minds will follow."

Yours sincerely

Robert Gassin Somerville

Victoria

Dear Sir

Musculoskeletal Medicine - Quo Vadis? Where to now? Who does it?

Most of the grey haired members of the musculoskeletal medicine associations around the world will remember the origins of the discipline as mobilising and manipulating various joints and parts of the body in order to effect a cure of musculoskeletal disorders. Proponents of such practices who have considered their techniques to be the best and, when applied correctly, to herald the panacea for most musculoskeletal problems. Alas, journals and texts are replete with accounts of questionable assessment and treatment protocols.

With the advent of modern diagnostic procedures the conditions which we thought we were treating were often seen not to be so, i.e. all referred pain was not solely due to the irritation of a nerve root. Further investigative techniques together with the application of current statistical theory should sharpen our clinical acumen in determining what will be the best treatment that we can offer our patients given certain clinical information. There is a need to attract corporate finance for research into musculoskeletal conditions.

The literature over the last decade or so has shown a tremendous advance in the research of musculoskeletal problems and yet despite the advance there are very few university places where appointments in musculoskeletal medicine are evident.

Most would agree that there is an abundance of patients with musculoskeletal conditions to treat. However with the nonexistent undergraduate and scant postgraduate upskilling programs the majority of doctors are poorly equipped to deal with musculoskeletal problems in general practice. There are many musculoskeletal conditions which can and should be treated by the family physician but instead are being bypassed in favour of our allied health professional colleagues who are increasingly becoming people of first referral.

Undergraduate medical education can only be viewed as a first step to addressing this problem. Many doctors going into general practice quickly recognise that they have very few skills to competently deal with the large variety of musculoskeletal ills. However there seems to be a lack of enthusiasm for the pursuit of postgraduate training in some countries. We have in New Zealand a population of approximately 4 million with a membership of NZAMM of the order of 150 and in Australia the population is 17 million with a membership of AAMM only 300. In the United Kingdom the population is over 60 million and the membership of BIMM is of the order of 300. I do not have figures for the US or Canada but talking to colleagues in these countries the ratio of members involved in musculoskeletal associations relative to population would be significantly higher.

This discrepancy in membership levels in different countries makes us wonder whether we are missing out on a golden opportunity to further the discipline of musculoskeletal medicine?

Our skills have a sound basis in theory and are based on the same concepts of best practice principles as in other branches of modern medicine. Hands-on teaching workshops need to be a regular feature of all our associations. The results of CME needs assessments for GPs in the area of musculoskeletal medicine show the perennial needs foremost to be low back pain followed very closely by pain in the cervical spine and shoulder girdle. There is a groundswell of interest in manual skills to assess and treat these areas. Members of our associations have a responsibility to provide musculoskeletal education to respond to these needs. Teaching takes time and some of this will be at the expense of practice income but we will not progress if we all look at our bank accounts and turn away from the broader picture of what our discipline can offer the medical profession as a whole.

This area of hands-on musculoskeletal education in no way detracts from the practice of inflammatory rheumatology, nor does it detract in any way from orthopaedic surgery as

Letters to the Editor

the latter is becoming more and more involved with the treatment of trauma and degenerative disease in the form of arthroscopy and arthroplasty and cannot cope with the burgeoning area of soft tissue disorders.

A musculoskeletal syllabus and an assessment protocol have been established and revised by both the AAMM and the NZAMM. It is not intended that every aspect of these detailed documents should be accepted without questions but they do represent the grass roots of the discipline and must be given careful consideration as the basis for all of our pedagogical pursuits.

Some tertiary institutions have recognised the deficiency in postgraduate training and have taken steps to correct it by offering full fee paying postgraduate programs to diploma level. Many general practitioners have completed the programs although there is little incentive given to doctors wishing to enrol in postgraduate courses. Motivation to enrol can stem from a recognition that there is a deficiency in their training. There are skills to be learnt and there are ways in which general practitioners can be educated sufficiently in hands-on methods to improve the management of their patients.

The ultimate is for the discipline to be established as a speciality with clinics running in large public hospitals, especially those attached to medical schools whereby both undergraduate students and training registrars in musculoskeletal medicine, rheumatology and orthopaedics can participate in such clinics as part of their training programs.

Now is the time for a profound display of unity. All members must actively work for the discipline or we will fade into obscurity. While this may sound very theoretical and perhaps pie-in-the-sky, unless we have a vision punctuated by attainable goals we will never attain the recognition to which we aspire.

Norman A Broadhurst
Senior Visiting Medical Specialist
Musculoskeletal Medicine Queen Elizabeth Hospital and Flinders Medical Centre
South Australia

Quiz

Professor Jim Taylor
Perth

1. All the lumbar spinous processes are palpable but in the neck only C2, C6 and C7 are palpable. **T/F**

2. The C2-3 level of the articular column is the easiest to identify as it projects back further than C1-2 lateral joint. **T/F**

3. In severe whiplash the discs and ligaments may be injured; in extension, the longus colli tears first, then the anterior longitudinal ligament (ALL) and then the disc. **T/F**

4. Chronic disc pain is more common than chronic facet pain after spinal injuries, in both the lumbar and cervical regions. **T/F**

5. Groin pain may result from lower lumbar disc injuries. **T/F**

See page 12 for answers.

Mid-Line Thoracic Pain - Diagnosis and Treatment

Dr Norman A Broadhurst

Any structure which has a nociceptive nerve supply can be a source of pain. In the case of the cervical and lumbar spine, much attention has been paid to the vertebral disc as a source of discomfort, especially when marked neurological signs can be readily substantiated by investigative techniques such as CT scanning, CT myelogram and MRI.[1,2,3]

Other authors have established the zygaphyseal joints as readily occurring sources of pain in the cervical and lumbar regions.[4,5,6]

It is unfortunate that back pain is synonymous with lumbar pain. It is also unfortunate that low back pain seems to be a diagnosis rather than viewed as a symptom which presents the physician with the challenge to identify the source of the pain.

It is generally accepted that muscles and ligaments can be strained but are generally looked upon as being self-resolving.[7]

A conservative estimate by Schwarzer et al[8] indicated that at least 45% of patients who present with chronic low back pain have pain arising from some unidentifiable structure which cannot be identified by any radiological investigations.

Patients often present with what they consider to be a mid-line pain in the low lumbar area and such pain could be due to the interspinous ligament between L5 and S1 where the supraspinous ligament does not exist.

Mid-line pain arising from the tips of the opposing spinous processes with sclerosis is characteristic of a disease known as Baastrup's disease, a reasonably rare entity.[9,10]

Whiplash patients often indicate that they experience mid-line pain and present with tenderness in the nuchal ligament with either unilateral or bilateral muscle pain.[11] These findings are reasonably common, but what is not very common is the patient presenting with mid thoracic pain arising from pressure over interspinous spaces or the paravertebral musculature between the cervicothoracic and the thoracolumbar junctions.

In the younger person, especially males, Scheuermann's disease often affects the lower thoracic spine, causing diffuse aches and pains which are very difficult for the patient to localise.

In the older patient who presents with thoracic pain, metastatic disease must be considered as the thoracic spine and ribs are common sites for secondary bony deposits, especially as they relate to lung, kidney, prostate and breast.[12]

While sources of pain in the cervical and lumbar regions can be readily identified by radiological investigations, pain arising in the thoracic area is not so amenable to resolution.

Some patients with ongoing mid-line pain and occasional referred pain patterns to the chest may undergo extensive cardiological investigations with no answer to the source of their pain.[13]

Nerve root irritation in the thoracic spine is extremely rare and should not be considered above the paravertebral muscles or the interspinous spaces as the source of referred pain.

Grieve suggested that the peak incident of involvement of dysfunction in the thoracic area is at the cervicothoracic junction as well as at T4/5.[14] Cyriax indicated that the muscles of the thorax and abdomen can suffer strain leading to scarring and persistent problems.[15]

Intercostal pain can readily occur as a result of sneezing, coughing or laughing. This sort of pain is readily identifiable and subcostal blocks can be used effectively to establish the diagnosis.

The region of the interspinous spaces consists of the supraspinous and interspinous ligaments, with the spinalis and rotatores thoracis being the closest muscles to the ligament. The interspinales and the intertransversarii muscles are not evident in the thoracic spine. It might be possible to assume that an enthesopathy could result when these muscles are strained to the extent of causing damage.

The structure and function of the interspinous and supraspinous ligaments is rather intricate and conflicting.

Rissanen studied damage to these ligaments, indicating that the main changes were fatty degeneration, fragmentation and necrotisation of fibre bundles, together with hyalinisation, calcification and proliferation of fibroblasts in the small vessels, together with the accumulation of various mucopolysaccarides and metaplasia into fibrocartilage.[16]

Hukins et al found that the interspinous ligament was fan-like in nature with a structure which presented very little resistance in flexion of the spine.[17]

Adams et al and Posner et al agreed that, while the ligament has no major role in resisting flexion, it may be a source of pain when the spine is maximally flexed by sudden movements or repetitive movements.[18,19]

As early as 1938 Steindler and Luck identified the interspinous ligaments as a likely source of pain in 14 people and they were able to eliminate the pain with injections of procaine hydrochloride.[20]

However, when the ligaments were removed, the pain did not resolve, indicating that it is likely that other structures are the source of the pain.

Aim

This study was designed to determine the source of mid-thoracic mid-line pain by identifying the level of the pain through movement and palpation, blocking the pain by the use of local anaesthetic and to assess the success of a depot steroid as a mode of treatment.

Method

Patients were chosen from those who had been referred for non-resolving mid-thoracic pain of six months' duration or more.

They had had no diagnosis for their pain and no invasive treatments such as injections but had received varying physiotherapy treatments including mobilising and manipulation. All had had plain xrays of the vertebral column which were normal for age.

Clinical examination consisted of the LOOK, FEEL, MOVE sequence to reproduce and isolate the pain.

Patients were asked whether each of the movements of side bending to the right and to the left, rotation to the right and to the left, flexion and extension of the vertebral column in the standing position reproduced their pain.

They were asked to indicate the area of the pain which was marked before lying prone.

In the prone position the following thoracic structures were palpated to elicit the symptomatic pain:

a) paravertebral muscles, especially longissimus and spinalis
b) spinous processes
c) spaces between the spinous processes

It was often necessary to repeat palpation over the tender structure to elicit the area of maximum pain which best reflected the presenting complaint. The spinous processes were never the source of maximum tenderness.

Once the levels of the interspinous space pain or the region of the longissimus muscle were determined as the source of pain, the patient was offered an injection of 0.5% lignocaine both to block the pain and confirm the region of discomfort. Using a 23 gauge 32 mm needle, each of the interspinous spaces was injected with 2 ml of local anaesthetic.

As the needle was introduced into the space, the patient was asked to report any pain and the area was then infiltrated. In this way the mid-line was injected, and as the needle was angulated a few millimetres either side of the midline, if pain was reproduced these areas were also infiltrated with local anaesthetic. No more than 2 ml was used in this procedure at each interspinous level.

With the longissimus muscle, I ml was injected into the muscle at each level of the interspinous space opposite which the muscle was tender.

Depending upon the size of the muscle and the length, the amount of 0.5% lignocaine was varied up to 15 ml.

Following injections of local anaesthetic, patients were retested by palpation and moving. When pain was diminished by 50% within 15 minutes of injection, the blocks were considered to be positive. Patients were then offered treatment at a subsequent consultation.

This treatment consisted initially of 0.5% lignocaine with depot steroid mixed in the ratio of one vial for every 5 ml of local anaesthetic. Hence a patient with pain spread over three levels received 2 ml at each level, giving a total of 6 ml.

If the longissimus muscle was considered the major source of mid-line discomfort, a similar volume was injected at each segmental level.

Each patient was instructed in post-isometric stretching of the tender regions and sent away with an illustrated instruction leaflet. Five stretches twice a day was recommended for each patient no matter what the source.

In the history of the presenting complaint, some patients had experienced trauma while others indicated that the pain was slow in onset.

Roughly 50% of the patients suffered trauma and the rest had slow onset of pain. No attempt was made to correlate the mode of onset of pain with level of dysfunction or response to injection.

Results

The population for the study consisted initially of 49 patients who had presented with mid-thoracic pain. Two of these were excluded when further examination indicated that they met the criteria for fibromyalgia.

Pain arising from the erector spinae group was identified as the primary source in eight patients. It was assumed by palpation that the longissimus was the muscle involved and was tender between T3 and T9 in all cases, ie. at the thoracic kyphosis.

The eight patients with longissimus pain were injected with depot steroid and local anaesthetic supplemented by post-isometric exercises.

At the two week follow-up, four had at least 50% resolution of their pain and did not want further injections but indicated they would continue with stretching exercises.

The other four patients had a second injection, with one patient having a third. All injections were at fortnightly intervals. These four patients considered their pain to be sufficiently resolved for them to cope. The small number of patients in this group prevented any correlations being carried out.

The muscle pain group reported significant relief not to require further treatment, although at follow-up, residual discomfort was still evident.

There were 39 patients (23 females and 16 males) who indicated that their mid-thoracic pain had diminished by 50% or more after the injections of lignocaine into the tender interspinous spaces.

Figure 1 shows the frequency of segmental pain, with a peak at T5,6 and an average of 3.2 levels for each patient.

Three patients refused injections of a depot steroid which meant that 36 patients received injections of the depot steroid at the interspinous space levels where the painful symptoms were

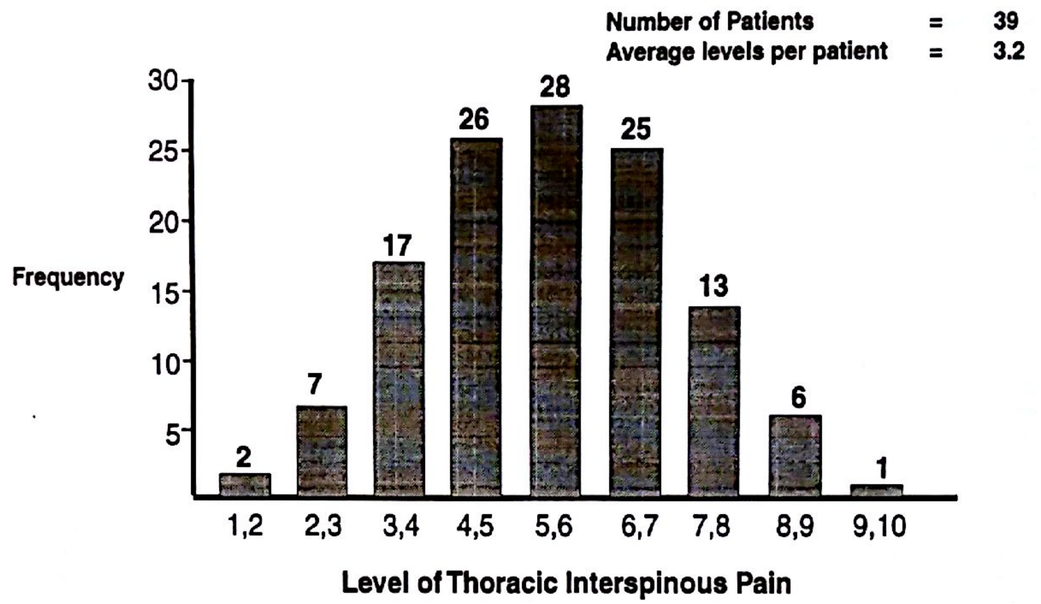

Figure. *The distribution of the levels of interspinous space pain in those suffering from mid-line thoracic dysfunction.*

elicited by palpation.

At the 4-6 week follow-up, 20 patients had improved by 70% or more and did not want further injections. Sixteen patients requested a second injection which gave significant relief in only 10. Three patients said that they had had enough and did not wish to have any more injections. The other three patients in whom the second injection was not helpful, opted to have their painful interspinous spaces sclerosed using 2 ml of 15% dextrose at fortnightly intervals which provided better than 75% pain relief after a series of three injections. This remained so at the three month follow-up.

Discussion

It is difficult to determine the tissue(s) responsible for the manifestation of interspinous space pain. Apart from the supraspinous and interspinous ligaments, pain could be arising from the intrinsic muscles, capsule of the Z joint or even the Z joint itself.

Because the pain was not reproduced until the needle was inserted up to the hilt, it must be postulated that some deep structure or structures are involved.

However Chua reports that "the superior aspect of the capsules tend to blend with the interspinous ligaments."[21] This would suggest that the pain might be due to Z joint dysfunction.

Further investigations, including intra-articular injections to the Z joints at the levels of the interspinous space pain, must be done as well as attempts to diagnose the tissue by injections under image intensification.

If further study proves that the pain is related to the Z joints, then palpation of the interspinous space as opposed to palpation of the thoracic spinous processes may offer a simple test for thoracic Z joint dysfunction.

Summary

The possibility of the interspinous spaces as a source of mid-line thoracic pain should be considered. Appropriate diagnosis and treatment of chronic mid-line thoracic pain resulted in at least 70% resolution of patient's symptoms using a mixture of depot steroid and local anaesthetic.

Only a few patients had complete resolution of their pain after three months, which indicated that further investigations are required to determine the source of the interspinous space pain. This would then enable a more definitive management plan to be devised.

Mid-Line Thoracic Pain

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Answers to Quiz on p.8

1. **False**; all the cervical spines are palpable if the extensor muscles are relaxed.
2. **True**; the zygapophyseal facets are on a more posterior plane that the lateral C0-1 and C1-2 facets.
3. **False**; autopsy studies of extension injuries show that the anterior anulus tears first, then the ALL and the anterior muscles rupture in only the most severe extension injuries. This is because the muscle is the most compliant and the disc annulus the least compliant of the three structures.
4. **False**; this may be true in the lumbar spine but Bogduk et al have shown that facet pain is more common after cervical whiplash.
5. **True**; this clinical observation is supported by the anatomical studies of Nakamura et al in *Spine* 21; 917 & *Spine* 22: 477. They showed that in rats sensory neurons from lower lumbar discs and facets may relay in the L1 or L2 dorsal root ganglia, which also receive input from the groin.

Truth in Musculoskeletal Medicine: I: Confidence Intervals

Professor Nikolai Bogduk, Newcastle Bone and Joint Institute, University of Newcastle

Critical reasoning and biostatistics is not new. The instruments and concepts were developed in the 1950s, and have been elaborated and refined since then. What is relatively new is their application to medicine at large and to musculoskeletal medicine in particular.

Conspicuously absent in the past, and even to this day, has been an appropriate respect for biostatistics and critical reasoning in undergraduate and postgraduate medical curricula. At best, lip service has been paid to biostatistics but the implications of biostatistics have not been integrated into clinical practice. Instead, medical students are typically taught in a way that implies that the various techniques of physical examination and diagnostic tests in which they are trained are reliable and valid, and that the treatments that they are taught are unquestionably efficacious. This pattern continues into postgraduate training. Yet, ironically when these tests and treatments are subjected to scientific scrutiny they often prove not to be reliable, valid or efficacious. There is, therefore, a mismatch between what is taught and the truth.

This series of articles is designed not to constitute some sort of course of instruction in academic material that makes doctors erudite but which is immaterial to clinical practice. Rather, it is intended to help the reader become a better consumer of clinical information, so that they can recognise the reliable, the valid and efficacious, and thereby distinguish truth from mythology, assertion and speculation. The objective is to equip the reader with devices that allow them to check information for themselves rather than relying on what experts say is right and wrong. If nothing else, the reader will learn what questions to ask, what information to demand, before accepting or believing a speaker or the writer of a journal article.

Confidence Interval of a Proportion

This first concept is a preface. It does not lead systematically into subsequent topics but recurs in various forms in other areas of biostatistics, and has some immediate applications to day-to-day practice.

The concept can be introduced by the question: does 3 out of 10 equal 30%?

Mathematicians and philosophers may argue what they please about this question, but in medicine the answer is *no*.

The fraction - 3/10, is a proportion, and in medicine will usually reflect the result of some sort of harvest. An investigator will have studied or surveyed 10 cases and found the index condition in three. They are tempted to proclaim a yield of 30%.

The illegitimacy of this temptation stems from the possibility that if the same investigator, or another investigator, repeated the same experiment, they might encounter a slightly different yield - say, 4/10 or 2/10 or even 6/10. What then is the true frequency?

The principle at hand is that there may be a correct or true proportion, that would be evident if every patient or every person in the universe was surveyed, but this proportion will not necessarily be evident if only a small sample of the total, possible population is surveyed. For any small sample a sampling "error" may occur. Just by accident, the investigator might select a group of subjects who happen to exhibit the feature in question somewhat more frequently than the true proportion or somewhat less frequently.

In order to accommodate this possibility a statistical correction applies.[1] The formula is:

$$p^* = p \pm 1.96\sqrt{\frac{p(1-p)}{n}}$$

where,
p is the observed proportion,
n is the number of subjects,
1.96 is a coefficient that generates a 95% probability, and
p^* is the range within which there is a 95% chance that the true proportion actually lies. Conversely there is a 5% probability that the true value lies outside this range.

If we consider our example,

$p = 3/10 = 0.3$
$(1-p) = (1.0 - 0.3) = 0.7$
$n = 10$

$$p^* = 0.3 \pm 1.96\sqrt{\frac{(0.3)(0.7)}{10}}$$
$$= 0.3 \pm 0.28$$
$$= 0.02 \text{ to } 0.58$$

This result shows that upon sampling 10 subjects and finding an index condition in 3, the prevalence of that condition is not necessarily 30%. The true prevalence could be as low as 2% or as high as 58%. Under these conditions, 30% is not a representative figure. The ambiguity arises because the sample size (n) is small.

Now see the effect of increasing the sample size. Suppose that the investigator studied 100 subjects instead of 10, and found the condition in 30. The prevalence is still not 30%; that figure is still only an estimate because the investigator did not survey every patient in the universe. The confidence interval of the observed proportion must be calculated.

$p = 30/100 = 0.30$
$(1-p) = (1.00 - 0.30) = 0.70$
$n = 100$

$$p^* = 0.30 \pm 1.96\sqrt{\frac{(0.30)(0.70)}{100}}$$
$$= 0.30 \pm 0.08$$
$$= 0.22 \text{ to } 0.38$$

There is still uncertainty but it is less

than when the sample size was 10. The true proportion could be as low as 22% or as high as 38%, but for this range, 30% is now a reasonable, indicative figure.

Increasing sample size decreases the size of the 95% confidence interval of the observed proportion. The decrease is exponential. When n is small the confidence interval is wide. As n increases the confidence interval narrows but never gets to zero. The interval would get to zero only if everyone in the universe is sampled, i.e. n = infinity.

Only you can answer how big the sample should be. The answer translates into a question to you - how close do you want the estimated proportion to be to the true proportion? If you are satisfied with a 20% range you might settle for a small sample, but if you want to be within 5% of the true proportion, you will demand a larger sample. The equation is available to you in order to calculate these answers. This equation is one that you should either memorise or carry in your wallet; you never know when you will need to use it as a consumer of new information.

Examples

The following are a series of examples that illustrate the application of confidence intervals in situations that may befall practitioners in musculoskeletal medicine. They are not esoteric or academic applications but ones that should be of day-to-day concern or interest.

Example 1: success rate of a new treatment

A speaker announces a success rate of 70% for a new treatment. Do you believe him? You should not. You should first ask - what was n?

If n = 10, the success rate is 7/10. You should calculate the confidence interval of this proportion. It amounts to 42% to 98%. In real-life terms this means that if you were to repeat the experiment, i.e. adopt the treatment, you might encounter a result as good as 98% or as bad as 42%. You cannot expect 70%; you should be prepared for 42%.

If n = 100, the confidence interval changes to 61% to 79%. In this case, 70% is a more indicative figure of what you might expect to encounter; but be prepared for a success rate as low as 61% instead of 70%.

This example shows that confidence intervals are not a tool used only by research scholars. They are relevant to you as a therapist. Every time someone advises you to adopt a new therapy, they are effectively inviting you to become an investigator and to repeat their experiment. Therefore, you should have no illusion that reported proportions are absolute. Your experience may be different from that of the first investigator, and the confidence interval formula indicates to you how different your experience might be.

You can also use the equation in reverse to calculate the appropriate n, if you have a certain confidence interval in mind. Let's say you want to ensure that the success you are prepared to accept is anywhere within 15% of the speaker's reported success rate. On what sample size should the speaker's results be based? Under these conditions,

$p^* = 0.70 \pm 0.15$

$1.96\sqrt{\frac{(0.70)(0.30)}{n}} = 0.15$

$\sqrt{\frac{(0.70)(0.30)}{n}} = \frac{0.15}{1.96}$

$\sqrt{\frac{0.21}{n}} = 0.0765$

$\frac{0.21}{n} = 0.0059$

$\frac{0.21}{n} = n$

$n = 35.59$

Thus, for you to expect a success rate in your hands of 70% ± 15%, the investigator should provide you with a success rate of 70% based on 35.59 subjects, i.e. at least 36 subjects. If the investigator's study is smaller than this, you cannot rely on achieving a result in your hands that falls within 15% of 70%; your result might be considerably worse. The same calculation could be repeated if you wanted a tighter range, say 5%, and if the reported success rate were any other figure, say 80% or 60%. The general formula is:

p = the reported success rate
p^* = the range which you would accept, i.e.
$p^* = p \pm z$
where
$z = 1.96\sqrt{\frac{p(1-p)}{n}}$

from which n can be calculated.

Example 2: could it be placebo?

An investigator audits a new treatment. He finds a success rate of 8/10. Is this impressive, or might it be a placebo response? In order to answers these questions, calculate the confidence intervals.

The confidence interval of 8/10 is 55% to 100%. Prima facie this looks like the result is not a placebo, for 55% is well above the conventional estimate of 30% for a placebo response rate. However, one must also calculate the confidence interval of the placebo response rate.

In a sample of only 10 patients, the confidence interval of 3/10 is 2% to 58%. This means that in a sample of only 10 patients, a series of investigators could encounter placebo rates as low as 2% or as high as 58%.

The figure - 58%, is higher than the figure - 55%. Thus, the confidence intervals of 8/10 and 3/10 overlap. This means that statistically it possible for a success rate of 8/10 to overlap the possible placebo range. Thus, prima facie, 8/10 could well be a placebo response rate. The investigator would have to provide you with a larger study, with a narrower confidence interval of the success rate, before you can accept that the result is not placebo.

This sort of calculation does not prove that a reported result is or is not a placebo response; that can only be shown directly in a controlled study. But it does show that statistically 8/10 is not necessarily an impressive result. Calculating the confidence intervals prevents you from being seduced into believing that such an impressive result could not possibly be a placebo.

Investigators wanting to plan a study can use these sorts of calculations in reverse in order to determine what size of study is required to provide prima facie evidence that the observed success rate is unlikely to be due to a placebo effect. Such calculations can be used to determine if the treatment in question is worthy of a controlled trial. There is no point expending effort if the prima facie evidence is consistent with a placebo effect.

What size study should be conducted if the observed success rate is 75%, and the placebo rate is assumed to be 30%?

For there to be prima facie evidence of the treatment not being due to placebo, the upper confidence limit of the placebo rate should be less than the lower confidence limit of the success rate, i.e.

$0.3+1.96\sqrt{\frac{(0.3)(0.7)}{n}} < 0.75 - 1.96\sqrt{\frac{(0.75)(0.25)}{n}}$

$0.3+1.96\sqrt{\frac{0.21}{n}} < 0.75 - 1.96\sqrt{\frac{0.1875}{n}}$

$1.96\sqrt{\frac{0.21}{n}} + 1.96\sqrt{\frac{0.1875}{n}} < 0.45$

$\sqrt{\frac{0.21}{n}} + \sqrt{\frac{0.1875}{n}} < 0.23$

Upon squaring both sides,

$\sqrt{\frac{0.21}{n}} + \sqrt{\frac{0.1875}{n}} + 2\sqrt{\frac{(0.21)(0.1875)}{n}} < 0.053$

$\frac{0.21}{n} + \frac{0.19}{n} + \frac{0.40}{n} < 0.053$

$\frac{0.80}{0.053} < n$

$n > 15.09$

Thus, for this success rate and expected placebo rate, the study must be based on at least 16 subjects. A smaller study would not show a success rate greater than the possible placebo rate. Conversely, if the placebo rate was greater, or the success rate smaller, the sample size would need to be appropriately larger. Remember, however, that such calculations do not prove that the success is not due to placebo; they provide only prima facie evidence that it is unlikely to be due to placebo. The utility of the calculations is not to substitute for a controlled trial, but to prevent controlled trials being wasted on success rates that are possibly within the placebo range. They also protect the consumer from being seduced by figures that numerically look impressive but which are based on too small a study.

Example 3: population studies

In an epidemiological study, an investigator samples 50 individuals with a history of neck pain after a motor vehicle accident and finds that none developed chronic neck pain. He concludes that chronic neck pain after whiplash does not occur. Is this deduction correct?

Assume that the true prevalence of chronic neck pain after whiplash is 6%. Did the author have a large enough sample to exclude this prevalence?

For a sample of 50, the confidence interval of 6% is

$p^* = 0.06 \pm 1.96\sqrt{\frac{(0.06)(0.94)}{50}}$

$= 0.06 \pm 0.065$

$= 0.0$ to 0.125

The figure - 0.0, indicates that with a sample of only 50 the investigator could well be studying a population in which the true prevalence was 6% but would find zero cases. Another investigator, using the same sample size might find 12.5% of 50 = 6.25, i.e. 6 cases. Thus, the study does not exclude a 6% prevalence.

For interest, calculate what prevalence does a sample of 50 reasonably exclude?

$p-1.96\sqrt{\frac{p(1-p)}{50}} > 0$

$p > 0.071$

Thus, a sample of 50 might only exclude a prevalence of more than 7%.

However, remember that this is not an absolute result. The confidence interval expresses only a 95% chance. Thus, a sample of 50 has only a 95% chance of excluding a prevalence of 7%. There remains a 5% chance that a prevalence of 7% would not be excluded by a sample of 50.

Another warning is that towards the extremes, confidence interval calculations come to grief. When the proportions approach 0% or 100%, the conventional formula does not apply and certain mathematical adjustments need to be applied.[2]

Armed with this example, the reader is invited to analyse for themselves, as an assignment exercise, the data and conclusions provided by a recent study in Lithuania on the prevalence of whiplash.[3] [Hint: find how many patients suffered neck pain immediately after the accident and

Truth in Musculoskeletal Medicine

how many of these went on to develop chronic symptoms. Using these figures, calculate the prevalence that would be excluded by this sample size, of chronic neck pain arising in patients who suffer neck pain immediately after an accident. Compare that finding with the prevalence of chronic neck pain in individuals who simply are involved in an accident without suffering neck pain.]

Conclusion

This is the first step towards incorporating biostatistics into everyday practice. The ability to calculate the confidence interval of a proportion equips the reader with a survival technique in the world of medical consumerism. It protects the reader against being hoodwinked by figures that look good but which are based on too small a sample. The confidence interval is one of the devices that helps answer the question - was the study big enough. Instead of appealing to experts, readers can now use the confidence interval formula to answer this question for themselves, whenever the occasion arises. In future articles, we will address truth in diagnosis and truth in therapy.

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The National Musculoskeletal Medicine Initiative: Update

The program of the National Initiative is developing steadily and is about to enter a more obvious phase.

Until recently the major efforts have been devoted to the consolidation of the evidence base and the development from it of clinical practice guidelines.

This work is continuing and will go on indefinitely as new studies are published. However, evidence-based guidelines have now been prepared for all the main regions of the musculoskeletal system and they are about to be put into practice.

The next stage of the program is the opening of the special Musculoskeletal Medicine Clinics in teaching hospitals and community-based settings around the country.

The first clinics are expected to be in operation in October and others will commence over the next two or three months.

Many members of the AAMM and the AFMM will be involved in the conduct of the clinics. Others will be involved in supporting roles.

Unfortunately the program is not large enough to allow all members of both organisations to participate directly at the beginning but all are involved indirectly.

As well as increasing patient access to clinical services, the program represents a multi-centre trial of musculoskeletal medicine and its scientific knowledge base.

The results of audits to be conducted in the clinics will show whether or not the approach taken by practitioners of musculoskeletal medicine has a significant impact upon the burden of illness.

If it does the discipline can be expected to flourish, with expansion of the clinic program and other forms of musculoskeletal practice.

If, on the other hand (which has to be contemplated scientifically), the impact is not significantly positive, we can all save ourselves the effort of daily practice and take up philosophy or some other worthy occupation instead.

No doubt all with some degree of involvement in musculoskeletal medicine will await developments with interest.

Dr Wade King

Prolotherapy in Lumbo-Pelvic Pain

Dr Gurmit S Dhillon, Musculoskeletal Physician, Dernancourt, South Australia

Prolotherapy is defined in Webster's New Collegiate Dictionary as "the rehabilitation of an incompetent structure such as a ligament or tendon, by the induced proliferation of new cells."

It is an innovative treatment for repairing chronic soft tissue injuries. The majority of acute injuries heal naturally. However, some fail to heal completely and go on to cause chronic pain and dysfunction. This usually occurs at the relatively avascular fibro-osseus attachment of a ligament or tendon to bone. Once weakened, this junction or enthesis is prone to repetitive strain and injury. These enthesopathies are responsible for most of the musculoskeletal pain we see in our daily practice. George Hackett[1], who pioneered this treatment, stated that " a joint is only as strong as its weakest ligament".

Prolotherapy can repair and strengthen damaged ligaments and tendons by inducing proliferation of new collagen. Injecting a substance capable of producing tissue damage and inflammation kick-starts the normal wound-healing cascade as described by Banks (see Dhillon[2]). A double blind animal study by Liu et al[3] showed a 30-40% increase in ligament mass and thickness after the injection of a proliferant substance. A statistically significant increase in entheses strength and weight/length ratio was also demonstrated when compared with saline controls. Morphometric analysis of electron micrographs showed a marked increase in collagen fibril diameters in the treated ligaments.

Klein et al[4] demonstrated similar changes in human posterior sacroiliac ligaments three months after prolotherapy injections. Marked fibroblastic hyperplasia was demonstrated on light microscopy. Electron microscopy showed a statistically significant increase in the average diameter of the ligaments.

Leading international authorities acknowledge that although the pathomechanisms of pain are well understood, we still have difficulty in understanding the mechanism of low back pain, based on the prevailing orthopaedic surgical model. Professor John D Loeser[5] stated in Sydney in March 1995[6] that "low back pain is crying out for a paradigm shift".

We need to look at low back pain from a non-surgical perspective. Most non-surgical back pain arises in muscles, ligaments and fascia. Therefore, in primary care, we should concentrate on the non-osseus structures in the lumbopelvic region.

This article focuses mainly on the non-contractile components of soft tissues in the back, and the ligaments in particular.

The Role of Soft Tissues

After the early anatomists dissected, identified and named ligaments in the human body they were largely forgotten and their significance underestimated. However, current researchers in the USA and Europe are taking a fresh look at the role of ligaments in the lumbopelvic region, in particular the posterior sacroiliac ligaments. Some of the newer concepts that have emerged from their studies are discussed below.

Nociception and Soft Tissues

An important relationship exists between the neural components of the lumbosacral region and the soft tissue structures. Several researchers, including Korkala[7] have used immunohistochemical techniques to show that all of these connective tissue structures receive a supply of small calibre, primary afferent fibers, typical of those involved in nociception. Irritation of these primary afferent nociceptive axons initiates the release of neuropeptides that interact with fibroblasts, mast cells and immune cells present in the surrounding connective tissue. The resulting cascade of events, referred to as a neurogenic inflammatory response, is thought to play a major role in the prolongation of low back pain. These chemicals sensitise nociceptive nerve endings in connective tissues, making them more easily activated by mechanical stimuli. This is the mechanism of tenderness.

Nociception detects tissue damage or threatened tissue damage by mechanical or chemical mechanisms. Mechanical nociception occurs in the absence of tissue damage when connective tissues are excessively strained. It is the basis of mechanical pain from ligaments, tendons, joint capsules, periosteum and skin. Nerve fibers weave comfortably between collagen fibrils when a ligament is at rest or at normal tension. When abnormally stretched, the collagen fibrils tighten, squeeze and twist nerve fibers and endings (Bogduk).[8]

Fatigue and Failure in Ligaments

Microscopic damage starts to occur in a ligament when it elongates by more than 4% of its length. Further elongation is made possible by the gradual rearrangement of collagen fibers, proteoglycans and water. This "creep" occurs regularly in our daily lives when we maintain constant postures. The ligament remains temporarily elongated when the strain is removed – this change is called hysteresis. Ligaments require time to reform from hysteresis, during which phase they are vulnerable to injury, and unable to sustain re-applied loads. Repetitive hysteresis within the recovery phase leads to fatigue and eventually, failure of the ligament even at sub-maximal loads. This fatigue-failure phenomenon from sustained repetitive activity explains the occurrence of ligamentous damage in the absence of a history of major or obvious trauma (Bogduk).[8]

Ligamentous Stocking

Kirkaldy-Willis[9] states that the three phases of dysfunction, instability and stabilisation occur at the lumbar motion segment. According to Vleeming[10], disc herniation is not a separate syndrome, but the result of failed stabilisation of the pelvis and lumbar spine. He presents a model showing the effects of shearing forces on the ligaments of the sacroiliac joint both in nutation and in counter-nutation. These abnormal forces if sustained, can lead to abnormal loading of lumbar discs, and in due course to disc herniation.

Although described anatomically as separate entities, the thoracolumbar fascia, ligaments and joint capsules in the lumbosacral region function as a continuous ligamentous stocking or corset. The three osseous elements of the region, the sacrum and two innominate bones, are positioned and suspended within this stocking. The major prime mover muscles of the lower back (multifidus, gluteus maximus and biceps femoris) are anchored indirectly to the underlying osseous elements through this elongated, ligamentous stocking, described by Willard.[11]

Self-bracing Mechanism

These muscular and ligamentous connections are of extreme importance in stabilising the lumbar vertebrae and sacrum during the transfer of energy from the upper body to the lower extremities. Activation of these muscles, according to Snijders,[12] helps tighten the ligamentous stocking, thus stabilising the lumbosacral spine and sacroiliac joints. He describes this as the self-bracing mechanism. Dysfunction of this mechanism is critical to the failure of the lower back. It occurs when these entheses are rendered tender and painful, either after acute trauma or from repetitive injury.

Prolotherapy to these entheso-pathies will produce healing and repair, allowing them to function, without pain, in the bracing mechanism.

Sacroiliac Joint Stability

The Musculoskeletal Research Group, which consists of anatomists, physicists, engineers, and clinicians, at Erasmus University, Rotterdam, developed a biomechanical model identifying the mechanical vulnerability of the sacroiliac joint. Lifting a load asymmetrically, in a stooped posture, results in concentration of the lumbosacral load on one sacroiliac joint. Overload of the dorsal sacroiliac ligaments may also result from forcing the sacrum into counternutation. The integrity of the sacroiliac joints depends on a strong ligamentous system. This model leads to new ideas for diagnosis and treatment (Snijders et al[13]).

Supraspinous and Interspinous Ligaments

Adams[14] found that the supra-spinous and interspinous ligaments are the first to fail and get injured when the spinal motion segment is flexed beyond its elastic limit. The capsular ligaments of the Z-joints follow next.

The work of Heylings[15] suggests that the supraspinous and interspinous ligaments consist mainly of a confluence of tendons, which attach the erector spinae muscles and the thoraco-lumbar fascia to the vertebrae. The supraspinous ligament also provides the spinous processes with a cushion of connective tissue, which protects them from impact during extension.

Yahia[16] studied the ultrastructure of the interspinous ligaments and ligamentum flavum in four normal controls and five subjects with low back pain, who had disc herniation. In normal controls, the interspinous ligaments consisted mainly of fibroblastic cells. Chondrocytes were only seen at the fibro-osseus junctions while proteoglycans were demonstrated between the collagen fibrils and appeared to form a regular interfibrillar linking. In the disc herniation group, chondrocytic cells had replaced the fibroblastic cells in the body of the ligaments, and proteoglycans were only randomly orientated to the collagen fibrils. Necrotic cells were observed in these pathologically altered ligaments.

Cusick[17] suggests that the posterior ligament complex of the lumbar region should be left intact when operating in the region. His study showed that the supraspinous and interspinous ligaments play a very important role as stabilisers of the lumbar spine.

Epicondylitis and Multiple Enthesitis

Histological changes in altered ligaments have been defined by Goldie[18] in his study of patients with lateral epicondylitis of the elbow. He noted oedema and hypervascularity of the extensor aponeurosis. Granulation tissue deep to the aponeurosis was found to be sensitive to pain when this tissue was excised under cutaneous local anaesthesia. Histological examination demonstrated free nerve endings within it. Bradford DeLong[19] states that similar enthesopathies may well occur in damaged ligamentous and aponeurotic structures elsewhere in the body. Inflammation at fibro-osseus attachments of the ligamentous stocking is associated with multiple enthesitis in the lumbopelvic region.

Treatment Protocol

All practitioners already posses the basic skills that are required in prolotherapy. However, the best way to get started and practise prolotherapy safely is to attend a hands-on workshop.

Management of chronic low back pain from the aspect of prolotherapy will be described briefly.

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Prolotherapy in Lumbo-Pelvic Pain

History

In taking the history, after red flags have been excluded, attempts must be made to determine if an injury could have contributed to back pain. It could be a previous motor-vehicle accident, work related injury or a sports injury. In female patients it is pertinent to see if childbirth may have contributed to it. When the history is positive for some form of trauma, it helps to try and analyse the biomechanics that occurred at the time of injury.

Symptoms

Pain from ligament dysfunction can either be local or referred. Most patients can easily direct the examiner to tender enthesopathies contributing to their pain. George Hackett[1], who pioneered prolotherapy 50 years ago, has produced charts showing pain referral patterns arising from ligaments. These charts are a very useful aid to the prolotherapist.

Patients suitable for prolotherapy will usually say that they are unable to stay in any one position for long, whether it is sitting, standing or walking. Sleep is often disrupted by pain when turning over in bed.

Difficulty in putting on shoes and socks is another common symptom reported by many. Patients with ligament laxity avoid long trips in the car. Household chores involving a small amount of flexion, like doing the dishes, vacuuming, sweeping and gardening reproduce the pain.

Examination

The practitioner, in addition to the points mentioned below, should conduct a standard physical examination. Movements must be tested both actively and passively to try and reproduce the patient's pain. The second aspect of the examination is to find and document all tender entheses. The patient may not be aware of all enthesopathies and the examiner will only find them by doing a thorough and systematic examination.

Proliferant Solution

Prolotherapists in the USA use a solution containing glucose, phenol and glycerine with lignocaine. The author has used 15% glucose with lignocaine for 5.5 years and achieved equally good results. This solution is prepared as follows:

3 ml of 50% Glucose
1 ml of 2% Lignocaine
6 ml of Sterile Water.

This will give a solution of 15% glucose with 0.2% lignocaine.

Injection Sites

These will be based on the history and outcome of the examination as described above. Commonly in chronic low back pain, the midline sites for injection are the supraspinous processes of the lower lumbar vertebrae and the interspinous spaces. The posterior sacroiliac ligaments and posterior superior iliac spines are the next most common sites for injection, especially if the pain is unilateral. It is important to customise the treatment for each patient. At each session, between 6–10 sites are injected.

Injection Technique

The cardinal rule is to make bone contact first, so that you know where the tip of the needle is. Withdraw the needle slightly, aspirate and only then inject the solution. Between 0.2 to 1.0 ml can be injected at each site. For some areas the needle can be withdrawn sufficiently to alter the direction and inject different spots within reach of the needle. Depending on the area being treated, needles ranging from 5mm to 60mm can be used. Needle gauges can range from 26G to 21G.

Treatment Plan

In the majority of cases, six injection sessions are scheduled, every two weeks. A follow-up is conducted after two months, when further injections are given if indicated. In a small percentage, a second follow-up may be necessary after a further 2-3 months.

Results

With the above treatment plan you will be able to help the majority of your patients who present with chronic low back pain. It is uncommon for someone not to derive some benefit from prolotherapy.

A randomised double blind controlled study by Klein et al[20] looked at the efficacy of injecting proliferant solution into the posterior sacroiliac ligaments, fascia and joint capsules. 79 patients with chronic low back pain who had failed to respond to previous conservative measures were selected.

They were randomly assigned to receive a double-blind series of six injections, the control group receiving xylocaine in saline. Results were analysed after six months. Patients were observed with visual analogue, disability and pain grid scores. Computerised tri-axial tests of lumbar function were performed before and after treatment. 76.9% (30/39) of those randomly assigned to the proliferant group achieved a 50% or greater reduction in pain disability scores at six months. In the control group only 52.5% (21/40) achieved similar results. Improvements in visual analogue disability and pain grid scores were similarly greater in the proliferant group.

Conclusion

In this paper I have provided an overview of prolotherapy, a simple diagnostic and treatment tool for chronic low back pain. It can be easily incorporated in daily general practice and does not require new skills, nor does it require the purchase of expensive equipment.

Prolotherapy is a safe and effective

treatment modality, not only for low back pain, but also for virtually all musculoskeletal pain, anywhere in the body. Primary care physicians will find it a very useful and invaluable tool.

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A Systematic Review of Treatments for "Tennis Elbow"

Ash Chandani, Debbie Waldron*, Swee Sam Teng*, Dr Paul Glasziou***

**Senior Medical Student, **Senior Lecturer in Clinical Epidemiology, Medical School, University of Queensland, Herston Road, Herston, Queensland, Australia 4006*
Correspondence: Dr Paul Glasziou Email: glasziou@spider.herston.uq.oz.au

Introduction

"Tennis elbow" or lateral epicondylitis (LE) is one of the commonest lesions of the arm and results in considerable morbidity and financial cost.[1] However, the term is a misnomer since it affects populations other than tennis players.[2]

Data show that tennis elbow equally affects both genders,[2] 1-3% of the population [2,3] and peaks in the 30-50 years age group.[1,2,4] It also affects 7.4% of industrial workers [5] and 50% of amateur tennis players.[2,5]

Tennis elbow's onset is gradual although acute pain can recur after exacerbating activities that involve excess pronation of the forearm with the elbow in an extended position (eg. shaking hands, turning door knobs). Consequently, it tends to be seen in those who carry out repetitive work movements of the forearm[2] with over 25 conditions having been suggested as the cause.[3]

Reported costs associated with this condition appear to be significant. Fifty-nine per 10,000 workers are affected[6] with 1981 data from Quebec (Canada) showing that over 1,500 workers claimed $4,442 per case at an average of 62 days work lost.[1]

Treatment

A review of the literature demonstrates the lack of an effective universal treatment. This is highlighted in Labelle's[1] systematic review which found more than 40 different treatments which worked with varyIng success. While research is required into both conservative and surgical treatment [4] "there remains no overall agreement in the literature as to the best steroid preparation, the correct dosage, and the best form of administration. This condition has improved even without treatment suggesting it may be a self-limiting disorder".[3] A greater understanding, therefore, of the pathophysiology at the tissue level is needed in order to improve conservative management.[3] Nevertheless, there is a need to evaluate all types of therapy with properly controlled randomised trials using double-blind methods.[1,2]

Aim of the study

In the meantime, a systematic review offers the most efficient and conclusive method in deciding the most efficacious treatment. Due to the progress in information technology, synthesis of study results becomes essential.

Several new randomised trials on alternative treatments for tennis elbow are available, making an updated systematic review worthwhile. Therefore this study takes a two tiered approach: firstly, to review the current literature in order to determine whether there is any significant scientific evidence to support choice of treatment for tennis elbow; and secondly, to compare the results of this systematic review, with that of Labelle's.

Methodology

A Medline search was made from January 1991 to May 1995 and the most recent issues of relevant journals published in the English language were searched manually. Ninety-eight published articles were located and studied which included reviews, controlled studies, letters to the editors, and single case studies. From these articles 26 were selected for further study. According to the set criteria nine studies were finally selected as suitable for this study. Articles were excluded if they:

- were simply describing the types of treatment available (ie. a review article)
- were simply describing a specific method of treatment
- described the pathology, natural history and/or diagnosis of the condition
- were letters to editors
- described therapeutic activities and preventative measures.

Therefore, 27 studies were selected for this study, the nine previously described and the 18 identified and analysed by Labelle.[1]

One quality appraisal form was used with three individually scored items which allowed a maximum score of 12.[7] The first looks at the method of treatment assignment; the second considers control of selection bias after treatment assigned; the third is related to blinding.

The form was completed independently by three authors (AC, SS, DW) for all 27 articles. Three copies were made of each article so that each author had their own copy of the article and they scored the articles. Differences in scores among the evaluators were minor. It was not considered necessary to take any special precautions to reduce observer's bias as suggested by Labelle.[1] All details of scores and final agreed scores are summarised in Table 1 (page 25).

The assessment period and assessment tools are shown in Table 1. The figures in Table 1 under column heading "Assessment period" represent the percentage of people who showed improvement after

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treatment/placebo. The assessment tools (percentage of patients who showed improvement) were classified under two subheadings, subjective and objective.

Discussion

All of the articles which met the selection criteria are included in the systematic review, however, since several of the articles tended to score quite highly an arbitrary score of 90% was determined as a reference point for a well designed study while those which scored less than 90% were considered of less value. It should also be noted that the appraisal form created for this study may have been too succinct since it did not analyse the subjective versus the objective measurements.

Some studies which scored over 90% used only subjective methods, or very few objective measures,[1,8,9,10,11] which could reduce the importance of their findings. This anomaly, however, could be overcome by blinding procedures. Of course, the question of how comprehensive (or succinct) an appraisal method can be without sacrificing convenience (or accuracy) remains unanswered, but further discussion of this is outside the scope of this study.

Due to the use of a limited number of studies which proposed a variety of treatments, the merits of each article are discussed using six categories:

- ultrasound
- ionisation
- NSAIDS
- steroids
- laser
- other.

Method of Treatment

Ultrasound

Five well designed studies were found which scored above 90%. The findings suggest there may be benefit in using ultrasound as a treatment. Binder,[12] Lundeberg[13] and Stratford[14] all used subjective and objective measures, while Halle[8] did not use objective pairs of measurement. However, the ultrasound studies were amongst the best designed in this analysis so the conclusion can be tentatively drawn that there may be benefit in using ultrasound as a treatment for tennis elbow.

Ionisation

Pharmacological ionisation involves the "use of an electric current to introduce medicaments",[14] and in these three studies, different forms of NSAIDS are delivered in this fashion. As Table 2 (page 26) shows, the studies are few and the results dubious. However, delivering medication via this method seems attractive, and a well designed study with appropriate controls, placebo, sham and oral medication groups seems desirable.

NSAIDS

Four studies were appraised, where two compared two different NSAIDS, one compared an NSAID with a steroid injection, and one compared an NSAID with a placebo. Labelle and Guibert[18] alone have attempted a well designed study. The other researchers seem to have assumed the efficacy of pharmacological treatment in the treatment of tennis elbow, although convincing evidence is lacking. For example, Saartok and Eriksson[19] have assumed the efficacy (in this case) of steroid injection, quoting Day,[20] whose study has been reviewed here, and who scored less (75%) than Saartok and Eriksson.[19] This has resulted in an attitude that obviates the need for control and placebo groups, although other studies have shown definite placebo effects whatever treatment is used for tennis elbow.

Steroid Injections

It is obvious from the low scores that this area is wide open to improvement. There are no definitive studies which demonstrate the efficacy of local steroid injection in the treatment of tennis elbow, therefore it is not recommended. As many practitioners still include these in their range of treatment, there is a strong need for a well designed study to test efficacy in both the acute and chronic conditions.

Laser

As so few studies are available for perusal (and these with equivocal results), laser treatment is not recommended for lateral epicondylitis. Even if future studies prove efficacy in treatment, lack of cost effectiveness would limit its usefulness.

Other Treatments

Table 2 shows that overall, studies on manipulation, DMSO, EM field therapy and REBOX tend not to be effective. However, Percy and Carson's[29] study was fairly well designed and concluded that DMSO was ineffective while Kneer[30] conducted a placebo-controlled double-blind study and found marked improvement in pain of movement under loading and mobility of the joints after treatment with DMSO as compared with the placebo. They concluded that 10% DMSO gel is suitable for topical use in the treatment of acute lateral epicondylitis with little adverse effects to the patient.

Summary

A few general points should be noted. Firstly, although most authors did qualify their results, there were some who reached a positive and significant finding, although they achieved only a low score.[10,20,33] This is of concern since these findings have been quoted in subsequent articles - from review articles,[34] to inclusion as an element for design in new studies.[19,31]

Also, most studies did note a rate of spontaneous improvement whether

Table 1: Various outcome measures used to evaluate the patient's improvement

Authors	Assessment Tools Used: Subjective: Spontaneous pain	Pain on palpation	Ask patient e.g. cured	Tenderness on pressure	Visual analogue scale (VAS)	Pain during resisted dorsiflexion	Objective: Ability to lift weights	Grip strength pain/power	Range of movement	Appraisal Score (%)	Labelle's Quality Score (%)
Binder					+	+	+	+/-		100	44
Lundeberg					+		+	+/+		100	38
Stratford					+	+		+/+		100	73
Vecchini	+	+			+	+			+	100	45
Vasseljen	+		+				+	+/-		100	-
Labelle @	+				+			+/-		100	-
Johannsen			+		+	+	+			100	-
Kneer		+		+		+	+		+	100	-
Krasheninni										100	-
Devereaux *	+							+/-		92	63
Halle	+		+							92	30
Saartok	+				+	+	+	+/-	+	92	37
Haker	+		+	+		+	+	+/-		92	-
Haker (May)	+		+	+		+	+	+/-		92	-
Price		+		+	+			+/+		92	-
Grossi	+		+							83	56
Percy	+			+					+	83	41
Haker (Nov)	+		+			+	+	+/-		83	-
Adelaar	+	+		+		+		+/-	+	75	11
Day	+		+			+		+/-		75	26
Rosenthal	+	+							+	75	44
Burton				+	+			+/-		67	19
Famaey	+	+		+						67	27
Hughes	+			+						67	7
Clarke										58	13
Brattberg	+							+/-		50	17
Kivi		+								50	6

*also used thermography
@also used VAS functional scale (1994 article)

Table 2: Number of patients improved (percentage) as measured during assessment period [Figures presented are treatment/placebo]

	Author and Year	Method	Assessment Period: Week 2-6	Assessment Period: Month 2-6	Assessment Period: Month 12	p-value	Conclusion
Ultrasound	Binder 1985 (12)	Ultrasound vs. placebo	63/29	63/29	90/55	<0.01	Effective short term only
	Lundeberg 1988 (13)	Ultrasound vs. placebo, ultrasound vs. rest	55/42/27	36/30/24		<0.01	No difference between placebo and control
	Stratford 1989 (14)	Ultrasound vs. phonophoresis	25/25				Phonophoresis adds no benefit to ultrasound treatment
	Halle 1986a (8)	Ultrasound vs. phonophoresis vs. TENS vs. steroid injection	69/65,56/63			<0.05	Showed improvement in all 4 groups but no significant difference between groups
	Haker 1991 (15)	Ultrasound vs. placebo	76/77			<0.05	Pulsed ultrasound was not effective
Ionisation	Famaey 1982 (16)	Diclofenac ionisation vs. placebo ionisation	82/38			<0.05	Claim Voltron better than placebo
	Vecchini 1984 (9)	Diclofenac ionisation vs. placebo ionisation	68/20			<0.01	Significant difference between placebo and drug groups
	Grossi 1986 (17)	Sham ionisation vs. placebo ionisation vs. pirprofen ionisation (two dose levels)	50/45			<0.01	Claim effect due to tissue and not systemic effects
NSAIDS	Rosenthal 1984 (21)*	Flurbiprofen vs. piroxicam	18/20				Flurbiprofen better than piroxicam (but design deficiency)
	Saartok 1986 (19)	Naproxen vs. steroid injection	50/45			>0.05	Both treatments equally effective (but no control group)
	Adelnar 1987 (22) *	Difunisal vs. naproxen	128				No difference between 2 groups (but control or placebo group)
	Labelle 1994 (1)* @	Sodium Diclofenac vs. placebo	100 injection		88/20/24	<0.01	Subjective improvement
Steroid Injection	Day 1978 (20)	Methylprednisolone vs. xylocaine vs. saline	60	82/80	70/62		Claim a significant result
	Bratlberg 1983 (24)	Acupuncture vs. steroid injection	50 injection				Claimed improvement
	Clarke 1975 (36)	Methyprednisolone vs. hydrocortisone	50	64/64			Did not measure effectiveness of treatment
	Hughes 1969 (23)	Steroid hypospray vs. steroid injection	88		92/85/90		No conclusion reached
	Kivi 1983 (25)	Oral indomethacin vs. steroid injection vs. immobilisation + indomethacin	145		65/80/88		Could not establish effectiveness of any of treatments
	Price 1991 (11)	Hydrocortisone vs. triamcinolone (10 & 20mg) vs. lignocaine	36				
Laser	Krasheninnikoff 1994 (37)	Lower power laser vs. placebo	30	67/53,80/53			Supported laser treatment
	Vasseljen 1992 (26)	Low level laser vs. placebo	49				Concluded laser efficacious but recommend further studies
	Haker 1991 (27)*	Low energy laser vs. placebo	58				No benefit in mid-laser treatment
	Haker 1991 (28)*	Mid-laser vs. placebo	33				Not stated
Other Treatment	Burton 1988 (31)*	Manipulation vs. manipulation with forearm strap vs. manipulation with topical NSAID	40	47/35#			Ineffective
	Percy 1981 (29)	Dimethylsulphoxide 40% vs. placebo	157	44/9			Marked improvement
	Kneer 1994 (30)	Dimethylsulfoxide 10% vs. placebo	30	53/53	53/53		No benefit
	Devereaux 1985 (10)	Electromagnetic field vs. placebo				<0.01	Beneficial for pain and function but overall improvement minor
	Johannsen 1993 (32)	Rebox vs. placebo					

*unable to extract the data @metaanalysis of this study is based on abstract only #results are based on improvement in pain only

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part of placebo, control or treatment groups. This may reflect the natural history of the disease. As the pathophysiology is still obscure, future studies should take this into account. As well as spontaneous recovery, the chronicity of the condition should be noted, because if the patient's lifestyle has rendered the initial cause of the epicondylitis redundant, then there may be different pathology underlying the recurrence of symptoms.

Conclusion

In conclusion, Labelle's[1] findings are supported, and many others in acknowledging the dearth of scientific evidence for the present treatments of lateral epicondylitis, and in urging the design of better controlled, randomised and blinded studies.

This is especially needed in the area of pharmacological treatment. Steroids and NSAIDS appear to be accepted for use simply because of their anti-inflammatory action, not because of any solid scientific evidence that they are efficacious in the treatment of tennis elbow, specifically. As the rate of spontaneous improvement is significant, conservative treatment may be all that is necessary, though difficult to test. However, if pharmacological treatment can be shown to be of no real benefit, this can at least reduce the associated risks to the patient (eg. side-effects; injection sites), and the costs to patient and community.

While the studies concerning the use of ultrasound appear to show some benefit, more unequivocal results are needed. Though not as cost-effective as simple rest, and avoidance of the causative factor(s), not all of those affected are willing or able to comply with this directive, and ultrasound may be of benefit to those patients.

In addition, this systematic review has served to highlight a different issue. There is a need for a simple, yet accurate method to enable practitioners to evaluate the literature they read - or indeed, for the publishers of papers submitted. The results of this systematic review show that while the appraisal form used was adequate, it would be modified for greater accuracy, but still be easily applied. There could be stricter guidelines for selection criteria, in several areas. Groups should be matched within a relatively narrow age-range; the chronicity of the condition should be similar; the sample size should not be less than 30-40, given the amount of variables that can exist; and assessment follow-up periods should be similar (across studies if review or systematic review is the aim). This should reduce any effect of heterogeneity within results, and enable any pooling of results to reflect truly significant findings.[35] Also, the appraisal form should include information which will show where subjective or objective measurements of the treatment outcomes are used.

Most importantly, patients expect some treatment for tennis elbow from their doctors, whether the symptoms resolve spontaneously over time or not. At present, doctors cannot be sure of the effectiveness of the treatments they offer. Until better designed and controlled studies are implemented, where the treatment of tennis elbow is concerned, the maxim, "do no harm" should still hold sway.

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History of Musculoskeletal Medicine: A Tribute to Michael Kelly (1905-1967) - Australian Pioneer in Musculoskeletal Medicine Research

Dr Philip Watson

Dr Michael Kelly MD (Adel.) has a unique place in the history of medicine in Australia and could well be called the father of the modern concepts of musculoskeletal medicine, as he appears to have embraced myofascial pain ("fibrositis"), the biopsychosocial model of pain, and the multidisciplinary approach to treatments. It is surprising that in this current climate of renewed interest in musculoskeletal conditions, especially the soft tissue complaints, Dr Kelly has fallen into almost obscurity and overshadowed by researchers working in other parts of the world.

Background

Michael Kelly, one of 10 children, was born in 1905 at Mintaro, South Australia. Much of his earlier education, especially in the classics, was provided by his parents, and during his later college years, he excelled in Greek, Latin, English literature, mathematics (including Euclid geometry) and physics. He was also very proficient on the tennis court and as an orator.

After graduating in 1927 with a medical degree from the University of Adelaide, he married, and then between 1931 to 1942 went into general practice in Bunbury, south of Perth.

In 1935 while almost recuperated from an attack of scarlet fever, he developed incapacitating musculo-skeletal pain involving his feet and shoulders, which necessitated a brief stay in hospital. A short time later his symptoms resolved spontaneously, in spite of various remedies, including the suggestion that his pain might be "psychological". Thus his interest in musculoskeletal pain and myofascial trigger points, or "fibrositis" as it was known then, was born.

He became a medical officer with the Australian Military Forces in Perth between 1941 and 1944 and was transferred in 1945 to the Repatriation General Hospital in Heidelberg, Melbourne, as an orthopaedic surgeon. In 1935 having passed his primary examination for Fellowship of the Royal College of Surgeons he became interested in the conservative management of joint injuries and diseases.

In 1947 he entered private practice as a rheumatologist as well as maintaining appointments at the Royal Melbourne Hospital and St Vincent's Hospital. In 1957, resigning from these appointments, he moved his private practice, called the Institute of Rheumatology, to East Melbourne. Also occupying these rooms were secretarial services, physiotherapist, occupational therapist, and a splint maker.

It was said that his practice consisted of three main parts. Firstly, those with fibrositis were treated with Procaine injections; secondly, those with inflammatory arthritis were treated with immobilisation of the inflamed joint, intra-articular injections of Procaine, and phenylbutazone; and thirdly, medicolegal work, as he recognised that trauma was an aetiological factor in the development of "fibrositis" and rheumatoid arthritis.

During the 1960s he became a strong antagonist in the use of corticosteroids in rheumatoid arthritis. Before his death in 1967, he had become quite provocative with regard to medicolegal and political issues. Some of his important earlier contributions to medicine were very significant and are worthy of review. They should not be overshadowed by his later publications.

Fibrositis

Due to Dr Kelly's personal experience with musculoskeletal pain, authors such as Weir, Mitchell, Head, James MacKenzie, Kelgren, Lewis, William Livingstone, and René Leriche, who wrote on the subject, caught his attention. His own extensive research on fibrositis, followed and in 1940 his first paper on treatment by local anaesthetic injections was published.

By 1946 he had published several papers in the *Medical Journal of Australia* detailing his observations of fibrositis (which we now understand as myofascial pain and trigger points), their referred pain patterns and treatment with local anaesthetic injections. Details for treating lumbago, shoulder, headaches and neck, chest, knee, forearm and hand, and thigh areas were included. These articles detail case histories and the diagrams indicate the appropriate injection sites.

In 1946 Dr Kelly wrote a thesis entitled "Interstitial Neuritis and Pressure Theory of Pain: A Critical Review and a Reflex Hypothesis", for which the University of Adelaide awarded him an MD. In his thesis Dr Kelly summarised his perception of fibrositis and the neural basis for soft tissue pain, implying that the central spinal and higher nervous system pathways were involved. He recognised that somatic pain and fibrositis presented as gradations along a spectrum. At one end were the pains without any physical signs, which gave rise to wider areas of diffuse secondary, or referred deep tenderness, and which disappeared when the lesion was anaesthetised. At the other end of the spectrum were somatic tissue lesions (e.g. bursitis, tenosynovitis and other rheumatic inflammations). He was careful to note that fibrositis is characterised by pain, which was usually accompanied by tenderness and muscle stiffness, but no constant pathological lesion. Hence he reasoned that fibrositis could not be considered a disease but a special kind of reaction in fibrous tissue triggered by a number of factors. He thought this may have resulted from a number of different precipitating factors. In the early stages, these manifest themselves as disorders of tissue function rather than

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recognisable structural changes.

He recommended that the term fibrositis be retained to cover all common pains which displayed these constant and recognisable patterns, of all gradations of severity, and seen in daily practice. He concluded that the disordered function in fibrositis involved alterations to the neural pathways of stimulation and inhibition which were concurrently in the spinal cord on the motor and on the sensory side. Therefore he suggested that "a succession of abnormal sensory impulses, such as may proceed from diseased or damaged tissue, could set up a disorder of function in the spinal cord, a functional disturbance in the truest sense of the word, which would manifest itself by abnormal sensory or motor phenomena".

He published further research articles on fibrositis in the *Annals of the Rheumatic Diseases* and in 1947 was awarded the Buxton-Browne Prize by the Harveian Society of London. It was at this time he became aware of other investigators in myofascial pain. One in particular was Janet Travell, with whom he corresponded and met later in the 1960s.

Rheumatoid Arthritis

Dr Kelly did not confine his research solely to the issues of fibrositis but included other aspects of pain. These areas of interest covered nerve injury, non-neural injury, muscle pain, pain due to pressure on nerves, neurogenic arthropathy and other neurological aspects of rheumatic disease. In the latter he developed principles in the management of rheumatoid arthritis, particularly splinting affected joints and active rehabilitation.

In 1951 he presented papers to the European Congress of Rheumatology and again in 1953 to the Eighth World Congress of Rheumatology. In 1959 he won a Ciba-Geigy prize for work in this area. In 1961, at the Tenth ILAR Congress in Italy, his fervent dislike of corticosteroid hormones in the treatment of rheumatoid arthritis was espoused and, following this, he was made an honorary member of the Italian Society of Rheumatology.

Pain

Due to the suggestion that his own experience of pain might have been psychosomatic, Dr Kelly continued to write eloquently on the ease at which doctors could ascribe unexplained pain as being psychosomatic or hysteria. His debates included arguments on issues such as whether fibrositis exists.

In his MD thesis, he enunciated his theories on pain, namely that it often took on a new form and characteristic from the inciting event. These seem just as applicable today. His theories ascribed these to changes in the peripheral nerve. This is in contrast to our current theories where the "lesion" has been shifted more proximally to the dorsal horn and to the higher centres.

In summary, I hope I have brought to your attention some details of the life of a remarkable man to whom little credit has been given, including the teaching curricula of musculoskeletal medicine. I recommend that musculoskeletal medicine practitioners refer to some of Dr Kelly's papers concerning his descriptions of the various regional pain syndromes. It is salutary to be reminded that an Australian too was a pioneer investigator in this field of medicine. It is unfortunate that the clinical knowledge on the management of soft tissue lesions, discovered in the 1930s and 1940s and to which Dr Kelly was a major contributor, is still not being taught at undergraduate level.

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Case Study: A Personal Encounter With Complex Regional Pain Syndrome

Dr Ron Palmer, Musculoskeletal physician, Brisbane

Abstract: This is a personal account of a major traumatic injury that went on to develop the secondary features of complex regional pain syndrome (CRPS) type 1, a traumatic rotator cuff lesion and scattered trigger points.

The development of the significant features, the treatment and the convalescence are taken week by week through the course of the recovery. The former terminology of "reflex sympathetic dystrophy" has now been upgraded by the International Association for the Study of Pain to CRPS. The clinical evidence of this case supports an active early intervention for the treatment of the complicating secondary effects and highlights the difficulty in selecting any single mode of therapy.

Terminology and Definitions

Causalgia was originally described by Mitchell during the American Civil War in 1872. The condition that he observed referred to soldiers who were wounded and went on to develop signs and symptoms that included pain, burning sensations, skin colour changes, hyperaesthesia, glossy skin, loss of limb function and terminating in coldness, pallor and atrophy of a limb. These symptoms over the years have attracted many terminologies, including causalgia, Sudeck's atrophy, sympathetically maintained pain syndrome and more recently reflex sympathetic dystrophy. In 1996 the terminology was again changed to complex regional pain syndrome. There are two subtypes. Type 1 follows a noxious event and pain and hyperaesthesia occur in a regional pattern that is not necessarily the distribution of a single peripheral nerve. Pain can be disproportionate to the inciting event and there are changes in circulation with local oedema. Type 2 is similar to Type 1 but is associated with nerve injury and tends to follow the nerve distribution pattern. Onset of symptoms can be immediate but is often delayed for some time. CRPS is not only found in limb injuries, but can develop with amputations such as mastectomy, visceral diseases, soft tissue lesions and back pain, to name a few. According to Walker and Cousins,[1] CRPS consists of a "constellation of symptoms and signs which may include pain and sensory changes, autonomic dysfunction, trophic changes, motor impairment, and psychological changes".

Pain. An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Hyperaesthesia. Increased sensitivity to stimulation, excluding the special senses. It may refer to various modes of cutaneous sensibility including touch and thermal sensation without pain, as well as to pain.

Dysaesthesia. An unpleasant abnormal sensation, whether spontaneous or evoked.

The Accident

This involved a fall of 30 metres when the edge of a cliff gave way. The loss of vertical height was approximately 30 metres and this involved falling from one ledge to another several times and finally rolling and somersaulting down a steep incline of approximately 70 degrees. The accident occurred in remote bushland inland from Coffs Harbour, NSW, while rock climbing.

Contact with the first ledge was by both feet, then the resultant fall backwards caused extensive bruising to the lower back when this struck the edge of the ledge before somersaulting backwards into space. Contact was made with the next ledge by the R shoulder and a number of bones were heard to break at this point. The next ledge produced direct impact to the R lateral chest wall and again a number of bones were felt to break. I am not sure of the exact sequence of contacts after this, but am aware of further somersaults and finally rolling sideways down a boulder-strewn slope. At no time was the head hit nor was consciousness lost.

On coming to rest I was aware of immense pain and was subconsciously clutching my R arm across my chest wall with my L arm. There was immediate difficulty in breathing. I did not move initially, but did an assessment of my own condition. By this time my climbing partner had climbed down to join me. He suggested he go for help. This was not practical as the terrain was far too rugged and steep for any person to carry me out. The suggestion was then for a helicopter, but this was rejected as the nearest rescue "chopper" was the Gold Coast and would take hours to organize. In addition it would be extremely difficult to locate me in the rugged bush. My breathing was laboured and was worsening. The only alternative was to walk out. I managed to gain my feet without assistance, the pain now so intense that I could not bear to be touched. My climbing partner cleared footholds as best he could and at two steps between rests we managed to climb down the mountain to a track. This took over an hour. He went for help from here. An hour and a half later an ambulance arrived and conveyed me to Coffs Harbour Hospital.

The Injuries

Fractures occurred in the outer third of the R clavicle (spiral), the R scapular was broken completely across just below the spine, the second, third and fourth ribs were fractured anteriorly and the sixth rib was fractured both anteriorly and posteriorly. The R sternoclavicular joint was separated by about 1 cm. The R lung was partially collapsed and the liver was enlarged following contusion. There was haemorrhage into the pancreas and this resulted in temporary diabetes that existed for 4-5 days. There was

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quite massive bruising over the lower half of the trunk posteriorly. Skin loss was also extensive over both buttocks and the R forearm on the ulna surface had lost most skin covering. X-rays also showed some peripheral R lung opacity suggesting bleeding into the pleura. The liver was enlarged, presumably from a contusion and there was a period of hyperglycaemia which settled by day five.

Initial Treatment

Wounds were now cleaned and fully dressed. Pain management for the first two days was by intravenous morphine. I was placed on Indocid tds by the orthopaedic surgeon and this course continued for two weeks. Only an occasional Panadeine forte was taken over the next five weeks.

In spite of the number of fractures it was a little surprising that the most painful site was the sixth rib. This remained acutely painful to movement for about six weeks. Swelling in the R hand and fingers began to appear by the second day. There was also considerable oedema of the R forearm, especially over the extensor surface of the elbow where a sac occurred. I was moving my fingers and was not aware of any sensation loss at this early stage. Colour appeared normal with the exception of some pallor due to oedema.

Ongoing Management

The next four weeks were spent in bed at home. A propped-up position was necessary for pain relief and lying on the back was the only possible position. A pillow beneath the R elbow was necessary to relieve pain from the subluxed R sterno-clavicular joint.

Week Two

I noticed the first sensory changes in my R hand. Dysaesthesia appeared in the R thumb. The ball of the thumb had a quite weird sensation best described as a piece of bone beneath the skin. Touching any object with the thumb produced this sensation, but there was no pain or even discomfort. The oedema of the fingers had increased. The fingers could no longer be closed to a full fist. There was no apparent pain and colour was quite pale. Fingers were moved therapeutically on a regular basis, but this had little effect in reducing the oedema.

Week Three

All fingers began to lose normal sensation. This is best described as paraesthesia and as the swelling was making my fingers look like sausages, I considered the oedema was having an effect on the peripheral nerve fibers. There was stiffness in all interphalangeal joints and the metacarpo-phalangeal joints. There was discomfort when trying to flex any of these joints, but it was more an "end feel" rather than frank pain. Colour had changed in the hand, pallor being replaced by some flushing. This was not marked and I remained unaware of any temperature increase in the digits or hand. I kept up the practice of flexing the fingers on a regular basis during the course of the day. I could still not get out of bed unassisted and apart from toilet necessities remained in bed.

Week Four

Mottling appeared in the R hand. This was intermittent. The digits were becoming painful to move. There was also some low-grade pain in the R wrist. This was more noticeable in ulna deviation. There was some obvious increase in skin temperature. There was no sweating. Swelling remained as before.

Week Five

Wrist pain became acute. Any movement was causing a lot of discomfort to the point that the hand and wrist were x-rayed. Plain radiography was normal. The mottling effect was established and included the whole hand. I noticed the first signs of hyperaesthesia. Touching my face with the R hand was totally different to the L hand. The sensation was one of feeling sharp stubble on the face, similar to a two-day beard growth. In association with this, the glove type paraesthesia still persisted in the whole hand at rest. I was able to get out of bed unassisted and was spending a large portion of the day moving around both inside and outside. The arm remained in the sling and for the first time I could abduct my arm sufficiently to wash the axilla unassisted. The overall pain symptoms from the fracture sites were reducing and this perhaps made me more aware of my hand.

Week Six

The palm of my hand began to sweat, the mottling increased as did the hyperaesthesia. Pain in the wrist joint steadily intensified to an acute level. At rest there was no pain, but any movement was unpleasant. The metacarpo-phalangeal joints were also very painful. The dysaesthesia in the thumb persisted and at this stage I felt I was developing CRPS. By now the fractures were tolerable and some shoulder movements were undertaken. I still had to wear the sling for without it there was a significant strain on the whole shoulder region that was painful. Exercises were carried out periodically and then the sling was replaced.

Week Seven

I telephoned two colleagues for their opinions. (I live on a farm 86 km from Brisbane). They both felt I had developed CRPS. I found car travel very uncomfortable and could not wear a seat belt. Perhaps I should have acted more promptly, but delayed having the problem seen for a further week.

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Week Eight

I consulted an orthopaedic surgeon who works from the same specialist centre at Caboolture as myself. He confirmed CRPS. He also examined my shoulder and ordered plain x-rays of my cervical spine and an ultrasound of my shoulder. He arranged for a consultation with an anaesthetist who does considerable work in the field of chronic pain and CRPS. The cervical x-rays were unremarkable and the ultrasound showed swelling of the supraspinatus insertion, but no tears were visible. Abduction was not more than thirty degrees and external rotation was barely present without considerable pain. I had developed pain in the vicinity of the bicipital grove. This was localized although there did not appear to be significant function loss with the biceps. Wasting of the whole of the R arm and forearm was obvious, due to dysfunction.

Week Nine

Arrangements were made for the following week for a direct block of the stellate ganglion with guanethidine under Bier block cover. In the meantime I was still doing active physiotherapy on my shoulder and hand. This was undertaken at home by myself. Stellate ganglion blocks are usually carried out in theatre due to possible complication that could include pneumothorax.

Week Ten

On Monday of this week the first stellate ganglion block was carried out in morning theatre at the Peninsular Private Hospital at Redcliffe. As I had to be at a funeral by 1 pm, I declined any sedation. To reach the stellate ganglion the injection was introduced from an anterior position just above the clavicle. 15 cc of Marcain were injected. The procedure was not without discomfort. I had almost instant pain in the R auditory canal. This was clearly not at eardrum level and there was no change to sensation in the ear lobe. This passed off within several minutes as the anaesthetic began working. In the initial phase there was also a sensation of significant pressure at the base of the neck. In reasonably quick sequence there was a sensation of the R nostril becoming blocked and some numbness developed on the R side of the face. I was kept in recovery ward for half an hour and by that time had developed ptosis of the R eye. There were no changes to the speech pattern.

Changes occurred slowly over the next few hours by which time the hand felt normal and the colour mottling had gone. There was an overall increase in R upper limb temperature and a "healthy" pink colour had replaced the mottling. Movement in all the small joints of the hand was greatly improved with loss of oedema, but some end range stiffness and pain remained. The wrist felt completely normal. All abnormal hand sensations had abated. Unfortunately this was not to last. Slowly symptoms returned to the previous level and virtually all benefits had been lost by eight hours. At the best there was no more than 20% improvement in general condition after 24 hours.

The second stellate ganglion block took place on Thursday. This time I had 3.5 mg of IV morphine. Symptoms following the block were slightly varied, but more pronounced. On this occasion 12 cc of Marcain were injected. Again the first symptom was pain in the R ear canal. This was followed by a more marked loss of R sided facial sensation, quite similar in intensity to that which is experienced following a dental block. After 15 minutes there was a definite ptosis and some huskiness had developed in speech. There developed a mild tremor in the whole of the R upper limb within half an hour. The onset of R sided headache appeared to coincide with the tremor. Once more there was good improvement in colour and function by three hours. Some limitation of finger flexion with end point range pain remained. Again these effects slowly wore off after eight hours, with the exception of the speech huskiness that lasted for three days. By the next morning there remained a good 40% improvement in function and both colour and sensation were markedly improved. Following the second procedure I felt good progress was made.

Week Eleven

Over the past weeks all fractures continued to heal. There was little discomfort in movement with notable exception of the R shoulder. Here gross loss of movement persisted. Abduction of the arm was no better than horizontal and external rotation still did not permit placing the hand behind my back. The stellate ganglion block benefits continued. Sensation had returned to almost normal in the fourth and fifth fingers. Hand functions had improved with the loss of CRPS symptoms and I could hold a cup and use cutlery efficiently. There remained weakness in both the hand and the arm sufficient to prevent supporting any weighty object such as a pot of tea. There was still an obvious colour change in the hand, however the mottling effect was grossly reduced. The R hand felt warmer than the L although sweating had disappeared. There was a faint paraesthesia in the thumb and index finger and dysaesthesia remained in the ball of the thumb. Hyperaesthesia persisted to some extent.

I was able to drive a car and decided to return to part-time work. At this time I felt the rotator cuff was not responding as quickly as desired, so I commenced a course of prednisolone tablets. Steroids were contraindicated previously due to the presence of multiple fractures. Injection to the shoulder was ruled out as there was

the possibility of introducing infection and the risk of osteomyelitis was best avoided. I curtailed practice to medicolegal work and injection techniques. There was no possibility of being able to do spinal manipulation at this stage. By the end of six hours work my hand had deteriorated and colour had deepened. These features settled overnight.

Week Twelve

By the end of week twelve function of the hand had improved to about 60% of normal. Shoulder range movements had also improved and the arm could be abducted to horizontal without discomfort. External rotation improved sufficiently to permit the fist to reach mid line posteriorly. By now there was some constant low-grade pain in the vicinity of the bicipital grove.

Week Thirteen

Flexibility in the hand continued to improve and swelling of the rays was far less. For the first time skin folds were evident on the palmar surface of the digits. By now ray 3 had returned to approximately 60% of normal function. Perhaps it was because of the overall improvement that I became more aware of the paraesthesia in the thumb and index fingers. I consulted with the orthopaedic surgeon again and he felt that shoulder function was only about 50% of normal. I had started using "trick" movements to elevate the arm and once he stabilized the scapular, movements were decreased. He mentioned the wasting that had occurred to the supra and infraspinatus muscles.

Week Fourteen

I felt that the index finger and thumb had reached a stalemate in recovery. A colleague gave me a paper to read on iontophoretic drug administration (EMDA).[2] Iontophoresis is defined as "the active transport of ionised molecules into tissues by application of an electric current through a solution containing the ions to be delivered." He had a machine supplied by Physion that administered this action. In principle guanethidine would be used locally that evaded the intense pain associated with a Bier block. I was a little undecided as to proceed with further treatment or wait a little longer. I decided to wait a further two weeks and if finger symptoms did not improve a trial course of treatment would be worthwhile. In the meantime physiotherapy for the shoulder continued and I returned to tennis (serving under hand) and played nine holes of golf. Golf was slightly less of a problem as I play L handed.

Week Fifteen

Another colleague called to say a visiting laser acupuncturist had apparently treated many cases of RSD in America and had articles published in *Spine*. At this stage the shoulder was more of a problem than the hand. I arranged to see them the next week.

Week Sixteen

An hour of laser therapy was administered to the neck, shoulder, selected points on the R upper limb and hand. By the end of the session there was an obvious improvement in both shoulder and hand function. On palpation prior to treatment there were a number of acutely tender points found scattered in the muscles of the chest, the shoulder, scapular and upper arm. All these points were subjected to laser. The response to therapy was initially good, but by next day all improvement had returned to the previous state. I had arranged for a follow-up session and kept this appointment.

Week Seventeen

Laser therapy was given. My colleague found an acutely tender trigger point near the insertion of the pectoralis major into the humerus. He injected this with local anaesthetic. As the needle entered the actual trigger point there was a massive trigger effect that can only be described as explosive. I have never witnessed such a response nor had he. Within 10 minutes there was a marked improvement of shoulder movements and these have remained. Abduction alone improved almost immediately by 20 degrees. The acutely tender point near the bicipital grove (the trigger point) has totally disappeared. As good as the response for this was, the laser did little to improve the thumb and index finger.

Week Eighteen

This is the time of writing. Shoulder function is still limited to about 60% of normal. However pain in the shoulder has been reduced significantly. The hand is still affected, but not to the point of restricting activities of everyday living. I can make a fist with a little discomfort, but still cannot flex the index finger fully. Some swelling persists and full flexion of the digits has some discomfort. There is a little hand oedema and after working all day there is minor flushing. I have returned to near full-time work (34 hours) and can manipulate. I have had to adapt some procedures due to weakness in the R arm. By the end of the day the shoulder aches at the site of fracture to the clavicle and scapular. The hand swells and the metacarpo-phalangeal joints ache; however this is bearable. I am on no current treatment, but am continuing tennis, golf and running.

Discussion

There are a number of salient features that have emerged from the above history.

CRPS may develop either quickly as in this instance, or can be of slower onset. The warning signs here were quite subtle, as the first real indication was that of dysaesthesia. In this case

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Case Study

sensory changes may have preceded pain onset. Confusion over this could be contributed to such widespread pain that the localized pain changes were not recognised. I do not think this was the case as most of the distal limb pain that eventually became apparent was localized to the interphalangeal joints, metacarpo-phalangeal joints and the wrist joint. Certainly in the very early stage I could still flex these small joints without pain and the wrist pain became apparent some weeks after the trauma.

CRPS has a diverse set of presenting symptoms and case variations are the norm. My own research indicated a huge number of suggested treatments and the conclusion from this is that there is no one superior approach. This then suggests that a multiple therapeutic approach may be required. In my case two individual treatments were undertaken. The stellate ganglion block had a pronounced positive result, although the first injection had only a short-term beneficial result. Perhaps a series of such injections is indicated if there is a positive gain from the preceding injection. The second mode of treatment via the laser was not of much use in my case.

The use of guanethidine in a Bier block is well described, but on advice of the anaesthetist was only a later choice as apparently it is a most painful procedure. Guanethidine administration via EMDA was not undertaken in this instance as recovery was well underway from other therapy before I had a chance to consider it.

Exercise is recommended in the early stages of the condition and I undertook as much physical movement as was humanly possible considering the fractures present. I have no doubt that this contributed to a quick recovery. Patients frequently complain that exercises can cause discomfort and I confirm that this may be true. However, I remain convinced that it is essential to push to the limit to obtain a favourable outcome and in this instance I believe the recover time was greatly reduced by persisting with exercise in spite of discomfort.

The rotator cuff lesion continues to improve slowly. Early intervention is important in any medical condition yet in this case treatment was restricted by the arm being strapped to my side. Recognition of limited shoulder movement was also not possible due to the sling. Any form of physiotherapy, including laser or ultrasound to the shoulder would have been impossible due to the close proximity of fractures and pain accompanying them. Once the lesion was recognised there was still no possibility of injecting cortisone as this procedure would have created a compound fracture by penetrating the skin in close proximity to the clavicular break. Cortisone taken orally was also discounted, as its presence would have slowed bone healing. I have also found NSAIDs to be of little help in treating rotator cuff lesions and that view remains, as I was on Indocid in the early stages and it had little apparent benefit in curtailing the development of the lesion. Again, exercise played a prominent role in treatment. I spent long sessions under a hot shower lifting and moving the arm in all directions to the point of pain. Once more I believe forcing the issue was responsible for a reasonably quick recovery.

Trigger point presence was a significant factor in limiting arm abduction. The pain at the insertion of the pectoralis major was acute when attempting to abduct the arm. Once this was injected with anaesthetic there was an immediate improvement. A lesson can be learnt here that careful physical examination for all tender points is essential. Simply concentrating on the three principal rotator cuff muscles may in fact prolong recovery.

Conclusion

This has been a personal experience of a number of conditions that all musculoskeletal physicians treat daily. I have learnt some features from a first hand experience that I had perhaps paid only lip service to in the past. Being a patient presents another perspective. While pain is an individual perception, "real" pain is certainly a traumatic event. Being unable to escape is perhaps its most damning feature. Indepth knowledge can also be frightening. On recognising the development of CRPS I became acutely aware of all the possible complications and the possible final disastrous outcome. This more than anything else spurred me to exercise to the point of obsession. It is my belief that recovery is based on a number of factors:

- early intervention
- adequate treatment
- active physical rehabilitation
- a positive mental approach
- a genetic ability to heal quickly.

I thank all my colleagues who assisted in my recovery and this made me aware of how patients view our profession. I certainly have developed a more mature attitude to my own responsibility within my chosen profession as a result of this experience.

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Medicolegal Brief

Dr Bruce Walker, United Medical Protection, Qld

A 54-year-old male presents with seven months' history of back pain that had steadily intensified. He was unable to sleep without disturbance. There was no lower limb referral.

He saw a GP who referred him to physiotherapy after a brief physical examination that did not include CNS or radiology. The examination was basically bending in different directions.

After three weeks of physiotherapy he was no better. He returned to the GP and was prescribed NSAIDS. There was no improvement. The patient then saw a chiropractor who conducted no full examination and took no xrays. He was treated with 12 manipulation sessions. The patient felt significantly worse.

He saw another GP who did not examine him but ordered more physiotherapy. The patient declined and saw a musculoskeletal physician.

His history revealed he had a renal tumour removed four years earlier. Examination showed no lower limb CNS signs, and there was no obvious weight loss or muscle wasting. He was tender to pressure over L3 centrally. An isotope scan was ordered.

L3 vertebral body was found to be virtually eroded completely. The patient was referred to an oncologist with a provisional diagnosis of secondary cancer.

Biopsy confirmed a renal secondary. The patient underwent a course of radiation then a surgical block of bone was inserted to support the spinal column. Chemotherapy followed.

The patient died seven months after referral to the oncologist.

What is the status of patient's family in suing for negligence?

Points:

1. Inadequate history
2. Inadequate examination
3. No investigations
4. Failure to recognise importance of night-time bone pain
5. Failure of "para" medicals to recognise the severity of problem.

In a court case what percentage of blame on medical versus paramedical?

"To be successful in an action in negligence, a plaintiff, the injured party, must be able to demonstrate, on the basis of the balance of probabilities, that he/she

(a) was owed a duty of care by the defendant
(b) that duty was breached by the defendant
(c) the plaintiff was injured, and
(d) the injury was the result of the breach.

Whenever a doctor/patient relationship is established, the doctor always owes a duty of care to the patient.

In this case, whether either doctor breached his/her duty, will depend on whether his/her peers are of the opinion that:

(a) He/she obtained an adequate history. Should the history of the renal tumour have been sought?
(b) He/she evaluated the history appropriately. Is a history of steadily increasing back pain without referral to the legs but accompanied by night pain, enough to require a plain x-ray of the spine?
(c) He/she made a significantly detailed examination, both general and specific spinal examination. How significant is local spinal tenderness?
(d) His/her referral to a physiotherapist was appropriate.
(e) His/her prescription of NSAIDs, without examination was appropriate.

The question of any liability in negligence by the physiotherapist or chiropractor will not be canvassed but the same considerations apply. The standard by which the physiotherapist/chiropractor is to be judged is that of his/her peers.

Assuming the doctors have breached their duty, the question of causation arises: that is, did the breach of duty cause the patient injury?

This question calls for an assessment of the effect of the failure of doctors to diagnose bony renal secondaries seven months prior to the patient's death. This is an area in which we as medical practitioners need to be very wary of our colleagues because someone, somewhere, will assert that the patient lost an opportunity of a "cure" or of significantly increased longevity. When that assertion is made, the best the doctors can hope for is a reduction in the award of damages of 15% to 20%.

"Failure to diagnose" cases are the major growth area in medical defence matters and they are very expensive because judges (and juries) are acutely aware of the "loss of a chance", whether it is scientifically logical or not. I have no doubt that in the present case, the vast majority of doctors would argue that when a 54-year-old male presents with seven months increasing back pain without referral to the legs and without neurological symptoms (or signs), night pain and a history of removal of a renal tumour four years previously, with the body of L3 already replaced by secondary carcinoma, there is a very high chance that he will be dead within 6-12 months.

The doctors who failed to make the diagnosis will be embarrassed but will know that their failure did not influence the eventual outcome, death, in any way. The Court will recognise that as well, in the absence of expert evidence to the contrary. However, once an expert pronounces those fateful words, "loss of a chance", we're doomed!

If the doctors are liable, the paramedicals will not be. Their argument will be that they relied on the referring doctors. It is highly unlikely that an expert will be found who will say paramedicals have more advanced assessment and diagnostic skills than medical practitioners!

Case Commentary

Low Back And Leg Pain

This section illustrates the diversity of approaches to musculoskeletal problems, commencing with the common problem of low back and leg pain. The following case details and question list was sent to six practitioners from a variety of disciplines dealing with musculoskeletal disorders. Three responses appear below. These follow the three published in the last edition of this journal.

History

40-year-old male tyrefitter

- Limped into your surgery two days after developing central low back pain while loading tyres into his ute.
- Continued loading tyres and then drove for one hour.
- Back pain worsened on getting out of ute. It became left sided and radiated down his left leg as far as his calf. Associated burning sensation in left leg, but no numbness or weakness.
- Developed scoliosis, convex to left, and loss of lumbar lordosis as pain spread down leg.
- Unable to bend or straighten without severe pain.
- Pain worsens with bending, extending, getting out of chairs and standing for over five minutes.
- Lying on side is only comfortable position.
- Otherwise fit and healthy with no significant past medical or surgical history.

Examination Findings

- Moderate scoliosis convex to left
- Loss of lumbar lordosis
- Leg lengths equal
- Lumbar movements: Flexion - 25 degrees; Extension - 10 degrees; Left sidebending - 10 degrees; Right sidebending - 30 degrees
- Straight leg raising: Left - 45 degrees; Right - 60 degrees
- "Spasm" left lumbar paraspinal muscles
- Tender regions: L5 and S1 spinous processes; Left L4 - S2 region; Left gluteus medius/minimus; Left piriformis; Left triceps surae
- Neurological examination: Hyperaesthesia left buttock, lateral thigh and calf. Otherwise normal.

Questions

- Are there any other history points you would like?
- Are there any further aspects of examination you would perform? (If so, why would you perform them and what would you expect to find?)
- What is your diagnosis at this point?
- Would you perform any investigations at this point? (If so, why, which investigations and what would you expect to find?)
- What is your preferred method of treatment in this case?
- How effective is your treatment?
- Why do you think your treatment works?
- What do you do when he is no better at - one week? - six weeks?

Dr Norman Broadhurst, Senior Lecturer in Musculoskeletal Medicine

What is listed here is a superficial history and examination findings which are not complete.

In essence, this probably reflects a good history by the average GP but in terms of Fellowship of the Faculty of Musculoskeletal Medicine, a greater depth of understanding and skill needs to be evident.

Points to make with regard to the history are as follows:

1. Need to know something of his social background, ie. married, children, social stresses, marital stresses which may impinge upon the ongoing management.
2. General health, medication, alcohol, cigarettes.
3. What is his usual work habit? Is this a new job that he has taken on, or has he been doing this for some time?
4. Has he had previous back pain of any kind? How is this incident related to previous pain?
5. What was the actual mechanism that precipitated this injury and were the tyres small or large?
6. Forward flexion is a better term than bending as one can bend in any direction, but forward flexion is more specific. Further knowledge of what precipitates his pain and what are relieving factors would be helpful. Can he sit for long periods, roll over in bed, sleep patterns. Is the pain present all the time? How much sleep disturbance does he get?
7. Lying on the side. Which side? Lying on the painful side compared to non-painful side. Some people give a history of having to put a pillow between their legs in a semi-prone position to relieve their pain.
8. Coughing, sneezing, laughing. What is the effect of these and is there any morning pain or stiffness?
9. More information about the back pain - central or lateral in the back and position. Is it superficial or deep - any other adjective to describe it?
10. The leg pain - what distribution - is it dermatomal or crosses dermatomes and the relation of leg pain to back pain?

Case Commentary

Moving on to the examination findings, we have no idea what this patient's body mass index is.

A grossly overweight person is going to be more difficult to manage than a person of normal proportions.

The moderate scoliosis convex to the left doesn't tell us at what level. One assumes it would be lumbar.

We would also like some idea as to the size of the "windows" on either side and whether the iliac crests are of the same level, as well as whether the PSIS are rotated or in a coronal plane.

1. I'm not too sure how the leg lengths were measured, but leg length must be assessed standing. Iliac crests should be measured in the sitting position as well as standing.
2. Flexion 25 degrees is difficult to perceive as a lot of movement occurs at the hips and some people cheat by bending their knees slightly. A 25 degrees flexion could be achieved with a very stiff lumbar spine. Extension of 10 degrees is surprising if this patient has lost lumbar lordosis and has scoliosis. Was this movement at the thoracolumbar junction? While in the standing position, foraminal compression testing should be done especially in view of the fact that he has pain down the leg. Reproduction of his symptoms is what is sought.
3. Slump testing would probably reproduce the patient's symptoms and needs to be done before lying down to do the straight leg raise. Dorsi flexion must be included in the SLR and pain reproduction with symptoms is important especially if there is a cross-over sign. This is suggestive of a problem with the disc and is supported by reproduction of signs from the foraminal compression testing, slump testing and now a positive straight leg raise. One thinks that the disc is the major source of his pain.

   Other testing is usually impossible at this stage because of pain so one can not assess sacroiliac joints or stress other structures in this area such as iliopsoas muscle or the buttock musculature
4. While there is some hyperaesthesia over the left buttock area, it is important to know which part so that the appropriate dermatome can be determined.
5. Palpation of the region to illicit tenderness which obviously involves the paravertebral muscles. However, it is surprising that the spinous processes per se are tender. PA mobes of these would undoubtedly be stressing the damaged disc, ligaments and Z joints.
6. The other tender muscles are probably asymptomatic and not part of his pain profile. Clinically speaking, this patient has pain probably arising from a disc which has been injured with involvement of the nerve root. Other sources of discomfort could involve the iliolumbar ligament and/or Z joint at either the L4,5 L5,SI with secondary hypertonus of the overlying paravertebral muscles. Disc related pain would include (not in any priority):

- Internal disc disruption - discogenic
- Nerve root irritation of disc contents
- Nerve root irritation by venous congestion
- Disc pushing on posterior longitudinal ligament
- Miscellaneous causes such as AV malformation

This patient needs gentle and appropriate mobilising of the tight structures to improve his range of movement together with massage and heat to the area as part of the hands-on therapy.

As this is a work-related injury, physiotherapy ordered along these lines three times a week would be helpful and one should see a reasonable resolution of his problem.

The patient is given self-exercises and should be reviewed in one week and if he says he is no better or worse, then total reassessment of the case needs to follow.

Non-resolving low back pain needs to be considered from two aspects:

a) Are the neurological features getting worse, therefore needs thorough neurological assessment of the lower limbs with appropriate investigations or referral. Epidural is still a contentious issue but given in the acute stage, has the best chance of success. Some would add a depot steroid. My personal preference is 8 ml 1/4% lignocaine with one vial of depot steroid via the caudal root. If the epidural fails, surgical referral is indicated.
b) Non-neurological factors include:
   - mechanical causes - Z joints
   - not coping - inappropriate illness behaviour
   - secondary gains for prolonging the illness
   - assess other related structures ie. SIJ and block under 11
   - occult malignancy
   - infection ie. discitis.

Case Commentary

Dr Greg Day - Division of Orthopaedic Surgery, University of Queensland

Differential diagnosis

This man is in the typical age group to have sustained an acute injury to one of the two lower lumbar discs. The symptoms are sided with leg radiation and I would therefore expect the disc bulge or protrusion to be posterolateral.

One must not assume that there was an absence of pre-existing lumbar pathology, including congenital, neoplastic, inflammatory, endocrine/metabolic or infective disease.

History Points

This is critical in the pathology sieve for pre-morbid conditions.

Intermittent low back pain would point to an already degenerate lumbar disc.

Constitutional symptoms of fever and weight loss and symptoms not relieved by the Fowler's position (curled up on the side) are indicative of more sinister pathology.

Substance abuse or a prior history of malignancy are other obvious pointers.

Other Pointers in Examination

Evidence of muscle wasting in the legs or fasciculation in the muscles would point to more long-standing pathology. A scoliosis is present and it is important to know if the patient has a balanced spine in both planes as measured from the centre of the occiput to the centre of the sacrum. A spine which is off balance must be held by muscles which eventually spasm or fatigue.

The straight leg raising test is critical. It must be ACTIVE firstly, with the patient supine. A passive straight leg raise in this position constitutes assault (say no more).

If the patient is unable to straight leg raise supine, try sitting the patient with the hip flexed at 90 degrees and the knee bent 90 degrees over the end of the couch. Then ask the patient to raise the leg and attempt to straighten it. Neural tension can then easily be gauged. The foot can then be dorsiflexed initially actively, then passively, to demonstrate neural tension (the pain produced must accurately reproduce the same pain complained of down the leg to the foot).

Diagnosis

Lower lumbar disc protrusion with minimal neural compression

Other Investigations

A plain radiograph of the lumbar spine would be a good baseline to exclude other pathology.

A CT scan at this stage is overkill.

Treatment

Insurance companies in the United States of America are the litmus test here. It has been proven statistically that it is economically viable to rest this man in bed (even in hospital) and have him off work for 48 hours with only low back pain and for seven days with low back and leg pain.

The US insurance companies have no problem paying for this and the Australian Workcover Organisations and insurance companies also agree with this treatment.

After this, he may continue to have pain. Anti-inflammatory medication, regular paracetamol and neural stretching and trunk range of movement manouvres are helpful. A trunk static floor exercise programme is helpful.

It has been shown that 90% of patients with similar symptoms and signs to this man will improve over the first six weeks.

If there is no improvement or if neurological signs develop in that time, it is efficacious to organise a CT scan of the lumbar spine. New generation spiral scanners give excellent resolution of intra-canal anatomy and sometimes obviate the need for MRI.

The only absolute indication for a surgical discectomy is a cauda equina syndrome.

All other indications are relative, but strong indications are worsening neurological signs or the appearance of them.

A discectomy will improve leg pain with a 90% certainty but will not help future low back pain. It is critical that patients know the limitations of surgical discectomy.

Outcome

If this man recovers from this episode, he is at risk of having further exacerbations of low back and/or leg pain.

It is important to institute a good floor exercise program for his trunk muscles and I generally ask a physiotherapist to oversee this.

The only indication for a surgical arthrodesis of the spine is to hold it to correct a significant deformity - scoliosis, kyphosis or spondylolisthesis.

A rare cause is the facet syndrome with primary arthritis of the facet joints and conservative management which fails to help.

This man has 20 years left of his working life, on average, doing moderately heavy physical manual work.

If he has dependents, he needs to be given a *long-term* management program to enable him to retire then, financially able to enjoy retirement, with as small a disability in his lumbar spine as possible.

Case Commentary

Dr Peter Jackson, Musculoskeletal Physician, Brisbane

This scenario is a common presentation in a general or musculoskeletal practice.

Flexing, rotating and sidebending the thorocolumbar spine[3,4] whilst moving heavy weights is a frequently heard story as is the gradual peripheralisation of central lumbar pain and the noxious effect of sitting and motor vehicle use.

Important history would be levels of aerobic fitness and flexibility and past history of similar events.

Further examination would include the skin drag test[4,5,6,7] to elicit signs of sympathetic overactivity such as sweating and skin oedema and the "pinch-roll"[6,7] test to elicit allodynia and dermatographia in the dermatomes of the posterior primary rami of T12-L2 and the anterior primary rami of L5-S1.

I would also palpate the myotomes of L4-S1 looking for taut bands, trigger points and local twitch responses and finally I would check for periosteal tender points on the iliac crest, greater trochanter and head of fibula [radicular syndrome].[6,7]

Next I would check the thorocolumbar junction for occult tenderness over the facet joints and attached multifidie, assymetry, reduced range of movement and tissue texture abnormalities.[6]

Following this I would length and strength test the iliopsoas, quadratus lumborum, hamstrings, glutei, adductors, quadriceps, piriformis, tensor fascia lata, ankle flexors triceps surae and peroneal group.

Checking the interdigital skin fold for allodynia between the great and second toe (L5) and fourth and fifth (S1) is a neat trick for identifying a subtle radicular syndrome.[2]

I would expect to find allodynia-hyperaesthesia over the left iliac crest and superior buttock extending down to the greater trochanter and possibly the lateral thigh and also the lateral and more posterior calf.[6,7]

Additionally there would be multifidie trigger points/a thorocolumbar facet joint tenderness and hypomobility at T12-L2, signs of myofascial dysfunction in the glutei including "enthesitis" of the anterior group and similar findings in the short head of biceps, lateral head of gastrocnemius, peroneal group, tibialis anterior and extensor digitorum communis.[6,7]

There would be comorbid tightness in the iliopsoas, hamstring, rectus femoris, tensor fascia lata and iliotibial band, piriformis, and gastrocnemius/soleus. I would expect to find associated weakness in the glutei, vasti and ankle flexors, and a hypomobile left sacroiliac joint and left pubic dysfunction.[3,4]

My diagnosis would be a comorbid minor intervertebral dysfunction at T12-L1 and L5-S1.[6,7]

The only test I might perform at this stage would need a plain thorocolumbar wide view xay in standing with a soft tissue emphasis to look at the psoas shadow, 12th rib, iliac crest heights, pubic symphysis and degrees of scoliosis and kyphosis. I would expect to find all of the above.

Treatment would consist of early intervention with mobilisation with impulse, muscle energy techniques, post isometric relaxation techniques, trigger point injections and spray and stretch in various permutations and combinations depending on the clinical findings.[2,3,4,5,6,7]

I would expect improvement to the degree that the patient could return to work after three visits two days apart. If he had not improved after one week I would begin a cognitive behavioural program with a heavy emphasis on posture and stretching.

If he had not improved by six weeks I would be considering more serious pathology and reflecting on his biopsychosocial milieu and I would be contemplating a CT scan.

This treatment restores normal neuromuscular physiology by stimulating or inhibiting afferent traffic from the peripheral proprioceptors ie. joint mechanoreceptors types 1-4, Golgi tendon organs and muscle spindles through the Aβ, Aδ and C. fibers to modulate the intra spinal aborisations on the Renshaw, group 1 non-reciprocal interneuron, and group 1 a inhibitory interneuron cell pools. These down regulate the pre-ganglionic cells of the intermediolateral column (vasomotor/sudomotor/pain facilitating) and their variants the gamma motor neurons and subsequently the alpha motor neurons. These in turn decrease the efferent traffic to the appropriate segmental muscles that are involved in maintaining the abnormal functional position of the mobile segments.[1,5,8]

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Musculoskeletal Medicine Tips

Tarsal tunnel syndrome*

Dr Norman Broadhurst, Senior Lecturer in Orthopaedic Surgery, Flinders Medical Centre, SA

Pain in the heel is usually due to a plantar fasciitis, but there is an uncommon condition that goes unrecognised. A careful history will tell the examining physician that plantar fasciitis is not likely, especially if the patient has had several injections without resolution.

A common history for plantar fasciitis is for the patient to have little pain sitting but gets significant pain immediately on walking due to swelling, this settles a little and then as walking continues the pain gets worse. There are no neurological signs and it presents in the older age group.

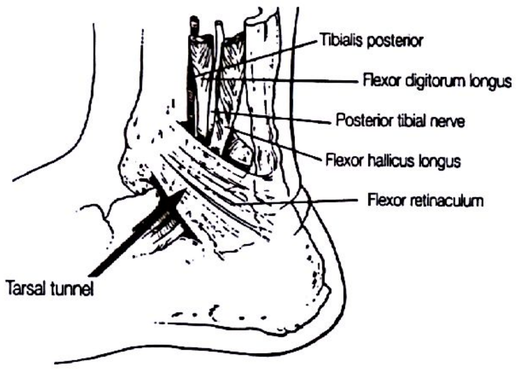

Figure 1

Tarsal tunnel syndrome presents in a similar way to carpal tunnel and is a tenosynovitis caused by an irritation of the posterior tibial tendon. It involves the posterior tibial nerve (Figure 1) and is more likely to be seen in a younger age group.

Diagnosis

The patient is usually pain free in the morning but quickly develops a throbbing pain with referred symptoms to the sole of the foot. Pressure over the posterior tibial nerve just below the medical malleolus produces a tingling in the foot.

Assessment of a foot deformity is important as orthotics may be required; for example, excessive pronation.

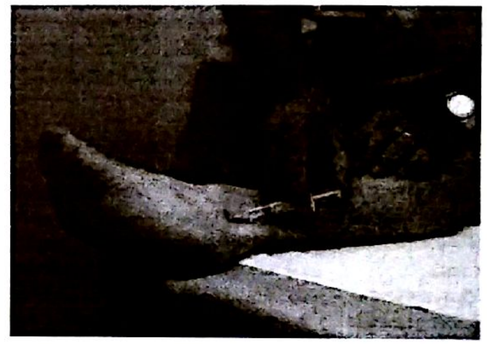

Figure 2. The tarsal tunnel can be successfully injected from above or below. The injection as shown has greater ease of access.

Treatment

Local injection of 2 ml of an equal mix of 1% lignocaine and one vial of a deposteroids is usually successful (Figure 2).

Nerve conduction studies are not usual in this condition because it readily responds to injections. However, ongoing pain in the foot may be due to entrapment of either the medial or lateral plantar nerve distal to the flexor retinaculum. This is a neural situation which might require EMG and nerve conduction studies with the possibility of surgical intervention.

*Reprinted with permission from *Australian Family Physician* 1995; 24: 654-55.

Journal Abstracts

This section introduced in the last issue aims to update the reader with some of the more significant recent research into the diagnosis and treatment of musculoskeletal disorders. Below is a selection of eight abstracts of studies published within the last year found by searching Medline, Current Contents and Rehabilitation and Physical Medicine.

Winters JC, Sobel JS, Groenier KH, Arendzen HJ, Meyboom de Jong B. Comparison of physiotherapy, manipulation, and corticosteroid injection for treating shoulder complaints in general practice: randomised, single blind study. *BMJ* 1997; 314(7090): 1320-25

Objective. To compare the efficacy of physiotherapy, manipulation, and corticosteroid injection for treating patients with shoulder complaints in general practice.

Design. Randomised, single blind study.

Setting. Seven general practices in the Netherlands.

Subjects. 198 patients with shoulder complaints, of whom 172 were divided on the basis of physical examination into two diagnostic groups: a shoulder girdle group (n = 58) and a synovial group (n = 114).

Interventions. Patients in the shoulder girdle group were randomised to manipulation or physiotherapy, and patients in the synovial group were randomised to corticosteroid injection, manipulation, or physiotherapy.

Main Outcome Measures. Duration of shoulder complaints analysed by survival analysis.

Results. In the shoulder girdle group duration of complaints was significantly shorter after manipulation compared with physiotherapy ($P < 0.001$). Also the number of patients reporting treatment failure was less with manipulation. In the synovial group duration of complaints was shortest after corticosteroid injection compared with manipulation and physiotherapy ($P < 0.001$). Drop out due to treatment failure was low in the injection group (17%) and high in the manipulation group (59%) and physiotherapy group (51%).

Conclusions. For treating shoulder girdle disorders, manipulation seems to be the preferred treatment. For the synovial disorders, corticosteroid injection seems the best treatment.

Karas R, McIntosh G, Hall H, Wilson L, Melles T. The relationship between nonorganic signs and centralisation of symptoms in the prediction of return to work for patients with low back pain. *Phys Ther* 1997; 77(4): 354-60; discussion 361-69

Background and Purpose. The purpose of this study was to assess the relationship between the nonorganic signs (Waddell scores) of patients with low back pain, their response to repetitive end-range lumbar spine test movements (centralisation of symptoms), and the rate of return to work at a six-month follow-up.

Subjects. Patients were assessed at five locations of the Canadian Back Institute. A consecutive sample of 126 patients with low back pain, with or without referred leg pain, was selected and reviewed.

Methods. Physical therapists assessed patients' responses to repetitive test movements (centralisation), as described by McKenzie, and tested the patients for nonorganic signs (Waddell scores). Therapists completed a data sheet that classified patients as either those who centralise their symptoms or those who do not centralise their symptoms and recorded their Waddell scores. Although the patients were classified at assessment, they remained in treatment. All patients followed a structured Canadian Back Institute protocol of active exercise, regardless of centralisation status or Waddell score.

Results. The inability to centralise symptoms indicated a decreased likelihood of returning to work, regardless of the Waddell score. A high Waddell score predicted a poor chance of returning to work, regardless of the patients' ability to centralise symptoms.

Conclusion and Discussion. A high Waddell score appears to be the best predictor of outcome, as indicated by return to work.

Malmivaara A, Hakkinen U, Aro T, Heinrichs ML, Koskenniemi L, Kuosma E, Lappi S, Paloheimo R, Servo C, Vaaranen V, Hernberg S. The treatment of acute low back pain - Bed rest, exercises, or ordinary activity? *NEJ Med* 1995; 332/6: 351-55

Background. Bed rest and back-extension exercises are often prescribed for patients with acute low back pain, but the effectiveness of these two competing treatments remains controversial.

Methods. We conducted a controlled trial among employees of the city of Helsinki, Finland, who presented to an occupational health care centre with acute, nonspecific low back pain. The patients were randomly assigned to one of three treatments: bed rest for two days (67 patients), back-mobilising exercises (52 patients), or the continuation of ordinary activities as tolerated (the control group - 67 patients). Outcomes and costs were assessed after 3 and 12 weeks.

Results. After 3 and 12 weeks, the patients in the control group had better recovery than those prescribed either bed rest or exercises. There were statistically significant differences favoring the control group in the duration of pain, pain intensity, lumbar flexion, ability to work as measured subjectively, Oswestry back-disability index, and number of days absent from work. Recovery was slowest among the patients assigned to bed

rest. The overall costs of care did not differ significantly among the three groups.

Conclusions. Among patients with acute low back pain, continuing ordinary activities within the limits permitted by the pain leads to more rapid recovery than either bed rest or back-mobilising exercises.

Aker PD, Gross AR, Goldsmith CH (reprint author), Peloso P. Conservative Management of Mechanical Neck Pain: Systematic Overview And Meta-analysis. *BMJ* 1996; 313 (7068): 1291-96

Objective. To review the efficacy of conservative management of mechanical neck disorders.

Methods. Published and unpublished reports were identified through computerised and manual searches of bibliographical databases, reference lists from primary articles, and letters to authors, agencies, foundations, and content experts. Selection criteria were applied to blinded articles, and selected articles were scored for methodological quality. Effect sizes were calculated from raw pain scores and combined by using meta-analytic techniques when appropriate.

Results. Twenty-four randomised clinical trials met the selection criteria and were categorised by type of intervention: nine used manual treatments; 12 physical medicine methods; four drug treatment; and three education of patients (four trials investigated more than one form of intervention). The intervention strategies were summarised separately. Pooling of studies was considered only within each category. Five of the nine trials that used manual treatment in combination with other treatments were combined. One to four weeks after treatment the pooled effect size was 0.6 (95% confidence interval 0.9 to 0.4), equivalent to an improvement of 16 (6.9 to 23.1) points on a 100 point scale. Sensitivity analyses on study quality, chronicity, and data imputation did not alter this estimate. For other interventions, studies could not be combined to arrive at pooled estimates of effect.

Conclusions. There is little information available from clinical trials to support many of the treatments for mechanical neck pain. In general, conservative interventions have not been studied in enough detail to assess efficacy or effectiveness adequately.

Freemont AJ, Peacock TE, Goupille P, Hoyland JA, Obrien J, Jayson MIV. Nerve Ingrowth Into Diseased Intervertebral Disc in Chronic Back Pain. *Lancet* 1997; 350 (9072): 178-81

Background. In the healthy back only the outer third of the annulus fibrosus of the intervertebral disc is innervated. Nerve ingrowth deeper into diseased intervertebral disc has been reported, but how common this feature is and whether it is associated with chronic pain are unknown. We examined nerve growth into the intervertebral disc in the pathogenesis of chronic low back pain.

Methods. We collected 46 samples of intervertebral discs from 38 patients during spinal fusion for chronic back pain. 30 samples were from pain levels clinically established by discography and 16 samples were from adjacent vertebral levels with no pain. We obtained 34 control samples of intervertebral disc from previously healthy individuals with normal histology within 8 hours of recorded death. We used standard immunohistochemical techniques to test for a general nerve marker, a nociceptive neurotransmitter (substance P), and a protein expressed during axonogenesis (growth associated protein 43 [GAP43]).

Findings. We identified nerve fibres in the outer third of the annulus fibrosus in 48 (60%) of the 80 samples of intervertebral discs. Nerves were restricted to the outer or middle third of the annulus fibrosus in the 34 control samples. Among the patients with chronic low back pain, nerves extended into the inner third of the annulus fibrosus and into the nucleus pulposus in 21 (46%) and ten (22%) samples, respectively. Nerves usually accompanied blood vessels, but in 14 of the samples from back pain patients, isolated nerve fibres were seen in the discal matrix. Both types of nerve fibres expressed substance P, but only non vessel associated fibres expressed GAP43, Deep nerve ingrowth into the inner third of the annulus fibrosus, the nucleus pulposus, or both was seen in four (25%) of 16 biopsy samples from non pain levels and in 17 (57%) samples from pain levels. Of the 16 paired samples from both pain and non pain levels, five pain level samples and one non pain level sample showed deep nerve ingrowth.

Interpretation. Our finding of isolated nerve fibres that express substance P deep within diseased intervertebral discs and their association with pain suggests an important role for nerve growth into the intervertebral disc in the pathogenesis of chronic low back pain.

Moulin DE, Iezzi A, Amireh R, Sharpe WK, Boyd D, Merskey H. Randomised trial of oral morphine for chronic non-cancer pain. *Lancet* 1996; 347(8995): 143-47

Background. The use of opioid analgesics for chronic non-cancer pain is controversial. Some surveys report good pain relief and improvement in performance while others suggest a poor outcome with a propensity to psychological dependence or addiction.

Methods. We undertook a

Journal Abstracts

randomised double-blind crossover study to test the hypothesis that oral morphine relieves pain and improves the quality of life in patients with chronic regional pain of soft tissue or musculoskeletal origin who have not responded to codeine, anti-inflammatory agents, and antidepressants. Morphine was administered as a sustained-release preparation in doses up to 60 mg twice daily and compared with benztropine (active placebo) in doses up to 1 mg twice daily over three-week titration, six-week evaluation, and two-week washout phases. Pain intensity, pain relief, and drug liking were rated weekly and psychological features, functional status, and cognition were assessed at baseline and at the end of each evaluation phase.

Findings. After dose titration in the 46 patients who completed the study, the mean daily doses of drugs were morphine 83.5 mg and benztropine 1.7 mg. On visual analogue scales, the morphine group showed a reduction in pain intensity relative to placebo in period I (p = 0.01) and this group also fared better in a crossover analysis of the sum of pain intensity differences from baseline (p = 0.02). No other significant differences were detected.

Interpretation. In patients with treatment-resistant chronic regional pain of soft-tissue or musculoskeletal origin, nine weeks of oral morphine in doses up to 120 mg daily may confer analgesic benefit with a low risk of addiction but is unlikely to yield psychological or functional improvement.

Beurskens AJ, de Vet HC, Koke AJ, Lindeman E, Regtop W, van der Heijden GJ, Knipschild PG. Efficacy of traction for non-specific low back pain: a randomised clinical trial. *Lancet* 1995; 346(8990): 1596-600

Previous trials to assess the efficacy of lumbar traction for back pain have been methodologically flawed. To avoid these shortcomings, we conducted a randomised controlled trial in which high-dose traction was compared with sham traction. The sham traction was given with a specially developed brace that tightens in the back during traction. To the patient, the experience is that of traction. The patients and outcome assessor were blinded for the assigned treatment. 151 patients with at least six weeks of non-specific low back pain were randomised. Intention to treat analysis showed no differences between the groups on all outcome measures (patients' global perceived effect, severity of main complaints, functional status and pain); all 95% confidence intervals included the value zero. The number of withdrawals from treatment, loss to follow-up, and protocol deviations was low. Consequently, the per-protocol analysis showed results similar to the intention to treat analysis. Subgroup analyses did not show any group for which traction might seem promising. Our data do not support the claim that traction is effective for patients with low back pain.

Kauppila LI, McalindonT, Evans S, Wilson PWF, Kiel D, Felson DT. Disc Degeneration Back Pain and Calcification of the Abdominal Aorta: a 25-Year Follow up Study in Framingham. *Spine* 1997; 22(14): 1642-47

Study Design. A 25 year follow up study of 606 members of the population-based Framingham cohort, who had received lateral lumbar radiographs in 1967/1968 and 1992/1993, and completed an interview on back symptoms at the second examination.

Objectives. To evaluate whether calcific lesions in the posterior wall of the abdominal aorta, the source of the feeding arteries of the lumbar spine, are associated with disc degeneration or back pain, which would suggest that ischaemia of the lumbar spine leads to disc degeneration. *Methods*. The presence of radiographic aortic calcification was ascertained in front of each lumbar segment from L1 through L4, and disc degeneration at intervertebral spaces from L1 L2 through L4 L5. The associations between aortic calcification, disc degeneration, and back pain were tested using logistic regression with adjustment for age and sex.

Results. At the baseline examination, aortic calcification was significantly associated with general disc degeneration, that is, disc space narrowing or endplate sclerosis at any lumbar level (odds ratio 1.6; 95% confidence interval 1.0-2.5; P = 0.034). In longitudinal, level specific analyses, comparing local aortic calcifications with disc degeneration at the matching level, aortic calcifications predicted disc deterioration, that is, a decrease disc space or appearance of endplate sclerosis, between the examinations (odds ratio 1.5; 95% confidence interval 1.3-1.8; P < 0.001). Furthermore, subjects in whom aortic calcifications developed between the examinations had disc deterioration twice as frequently as those in whom aortic calcifications did not develop (odds ratio 2.0; 95% confidence interval 1.2-3.5; P = 0.013). Also, individuals with severe (Grade 3) posterior aortic calcification in front of any lumbar segment were more likely than others to report back pain during adult life (odds ratio 1.6;95% confidence interval 1.1-2.2; P = 0.014).

Conclusions. Advanced aortic atherosclerosis, presenting as calcific deposits in the posterior wall of the aorta, increases a person's risk for development of disc degeneration and is associated with the occurrence of back pain.

Book Review

Back Pain and Spinal Manipulation

Second Edition, John Murtagh and Clive Kenna
ISBN 0 7506 2185 0

During the early 1980s when I was teaching the Murtagh-Kenna course on manual medicine we were using individual "fact" sheets on specific regions. These were basic instruction sheets on examination and manual techniques. For a proper understanding of what was implied, it was absolutely essential to have a practical clinical demonstration of both examination and procedural management.

Both John and Clive travelled around Australia giving weekend seminars and these visits were followed up by locals such as myself. In a very basic manner, the indications for, and the techniques of manual medicine were brought to the attention of many hundreds of general practitioners. Indeed, many practising doctors, especially rural ones, had their only exposure to what has now grown into the specialty of musculoskeletal medicine. Without question, these early courses laid the foundation to the university diploma courses that followed. Those early fact sheets formed the beginning of what is now their text book.

Back Pain and Spinal Manipulation is a good weighty book of 470 pages. It is well illustrated and contains over 100 photographs. The second edition has been extensively revised and includes totally new material on sacro-iliac disorders, muscle energy techniques, muscle stretching techniques and manipulation methods for the thoracic spine. The book is a considerable update on the previous edition.

The format of the text is clearly influenced extensively by Murtagh. His many publications on general practice subjects will be well known by virtually all Australian general practitioners. Easy to read, step-by-step information is his hallmark. This book is no exception. The book should be on the shelf of any general practitioner who even thinks about manually treating patients.

The book is not about detailed anatomy, biomechanics or physiology. For a full understanding of musculoskeletal problems it is necessary to seek information from other sources. The authors do not at any time pretend that this is the case. They state themselves that it is a "practical guide." When reviewing the book simply on this basis it does cover a lot of territory well. This presentation, together with the practical courses still offered, will combine to give a good introduction to basic manual therapy. For those interested in musculoskeletal medicine, it must be pointed out firmly that there is a lot more to the subject than covered in this text.

I found some areas lagged a little behind current thinking. For example the authors frequently talk about "muscle spasm". One would think that a more complete and accurate description would be to use the term "increased muscle tone". Spasm implies a muscle in complete contraction and this is not what is implied by the underlying text. A triggerpoint may be referred to as a localized area of muscle spasm, but the same cannot be applied to a tight trapezius muscle.

The manual manipulative techniques are described as non-specific, semi-specific and specific. Certainly all of these exist, but at this stage of our knowledge it is hard to justify a non-specific procedure. The more non-specific a procedure is, the greater the likelihood of introducing damage from the procedure. Quite frankly I find a non-specific procedure unacceptable, especially when there is a specific procedure for moving any individual facet joint.

In discussing some areas of regional pain, for example the SI joints, no relevance is given to history. The type of underlying damage likely to be found is often semaphored in the actual history. It is difficult to cover everything in a practical manual, but it is also reasonable to point out some of the short falls. Again, one needs to examine the hip joint before any examination is taken on the SI joint. Many of the tests used in musculoskeletal medicine are not absolutely specific and it may require accumulated information from two or more to reach a diagnosis. If there is degeneration in a hip joint, then a FABER test for the SI joint will be positive. This of course does not indicate that there is in fact a dysfunction with the SI joint. Unfortunately this is not pointed out in the book.

While these criticisms are justified, they should not detract from the overall value and thrust of the text. The book is a good introduction manual and some minor tidying up will convert it into a very good manual. For a recommended retail price of $70, it represents good value for money. An understanding of what this book contains serves as a good basic foundation to take the next step in musculoskeletal medicine education, a university diploma course.

Dr Ron Palmer

Report on AAMM 27th Annual Scientific Meeting: Low Back Pain in General Practice

Michael Yelland

This year's annual scientific meeting was held in the comfort of the Hilton Hotel in Melbourne at the end of August. Those coming from warmer climes in the north braced themselves needlessly from the cold as the weather was surprisingly mild. The original title of "Controversies in Musculoskeletal Medicine" made way for the more broadly appealing theme "Low Back Pain in General Practice". However both titles more than adequately described what transpired.

The content of the meeting centred around poor old Bob (convincingly portrayed by Dr Robert Brzozek), a miner from Wollongong, who hurt his back at work. His case presentation was the nidus for presentations on history, examination, investigation and treatment of acute low back pain. His progress (or lack thereof) over the next year was unveiled progressively throughout the meeting and was linked with presentations on a variety of approaches to chronic low back pain. Not surprisingly he ended up at a multidisciplinary pain clinic (portrayed single handedly by Prof. Nik Bogduk).

The proponents of evidence-based medicine, headed by Prof. Bogduk and Dr Wade King, were there in full force delivering some stark and sobering messages about the lack of support for many widely used diagnostic and treatment modalities. Many firm convictions based on clinical experience and intuition were left dented and battered by the absence of supportive evidence. Even many modalities firmly based in the sciences of biomechanics, structural anatomy and pathology were unable to withstand the analytical scrutiny. One was left with a picture of respected practitioners feeling somewhat exposed, perhaps even stark naked at times, trying valiantly to be proud of the wrinkles and warts which gave them their finesse. Prof. Jim Taylor whilst a proponent of the scientific basis of clinical practice cautioned the acceptance of evidence where only one or two good studies are available. Dr Steven Hall, who deserved a prize for the most entertaining presentation, also gave a warning about the acceptance of evidence from meta-analyses based on poor-quality studies - "Shit in - shit out!".

While the evidence-based approach may be an intellectually honest and important process, those present who were relatively new to musculoskeletal medicine were left somewhat confused and even depressed about what they could offer their patients. One such practitioner was urged to put Prozac in his city's water supply. Amongst the more experienced practitioners present, our vice-president quipped that he would become a former practitioner and survive by selling mail order Panadeine.

All joking aside, it must be remembered that evidence-based medicine is not designed to replace or destroy current practice - it is here to educate practitioners about what has been shown to be effective so that they can make informed decisions about which diagnostic and treatment approaches to use. It allows honest patient education about the nature, prognosis and treatment. An evidence-based approach will lead to early recognition of red flag conditions using a minimal number of potentially harmful investigations. Treatments will be chosen on their merits and will not be employed beyond their "use by date".

The meeting concluded with a message that those who prefer to use unproven approaches need not despair - these may indeed be as effective as years of anecdotal experience suggest, even if their effect just stems from the confidence and charisma with which they are conveyed. Whilst the presence of evidence that a certain approach is actually detrimental to patients' health suggests that it should be proscribed, the absence of supportive evidence does not mean it is worthless. It may just be that the studies have not yet been good or powerful enough to provide evidence that it works. This reminds us to be careful to consider the existing evidence when proclaiming the efficacy of our treatments to our peers and our patients. It also raises the challenge for us to become involved in research which critically appraises what we do, or at least to learn how to critically appraise the literature on our current practices. The article entitled "Truth and Musculoskeletal Medicine" by Nik Bogduk in this issue may sharpen the reader's skills in this area.

In spite of the evidence for or against various approaches, one still needs some tools of trade with which to practise. These were amply provided by the workshop program which followed the scientific meeting. A variety of diagnostic and treatment approaches were illustrated and practised at the workshops, often with a healthy critical assessment of their meaning and efficacy. Even if it is only the "charisma and confidence" factor which counts here, the participants at the workshops would have returned to their practices hoping for better outcomes for their patients.

Meetings and workshops like these take an enormous amount of time and energy to conduct. They are the key activity of the AAMM. The organisation of this year's scientific meeting fell on the shoulders of a very dedicated committee comprising Vic Wilk, David Vivian, Steve Jensen and Robert Brzozek with the very able assistance of PR Conference Consultants. They all deserve a huge vote of thanks for what must be one of the most successful meetings in AAMM history. We look forward to the FIMM meeting at the Gold Coast next Easter which promises to continue the evidence-based themes adopted in Melbourne with a more international audience.

Musculoskeletal Medicine Educational Activities

QUEENSLAND

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
23-26/10/97	Basic Acupuncture Pain Management Seminar	ANA Hotel Gold Coast	Continuing Medical Pain Education	Marilyn Strauss Ph 07-55313810	108 plus 25 practice assessment points
23-26/10/97	Part A: The Lumbar Spine	Prince Charles Hospital, Chermside, Brisbane	McKenzie Institute	Kate Soltau Fax 02-97433808	N/A
15-16 & 22-23/11/97	Introductory Medical Acupuncture Course	Sheraton Hotel, Brisbane	Australia Medical Acupuncture Society	Dr David Lee Ph 076-361499	Pending
21-23/11/97	Osteopathic Course: Basic Pelvic and Lumbar Spine - Dr Loren Rex	Kippa-Ring Physiotherapy Centre	Kippa-Ring Physiotherapy Centre	Yvonne Strydom Ph 07-32833721	N/A
12-14/12/97	Osteopathic Courses: 1. Iliotibial Band (12/12/97) 2. T4 Syndrome (13/12/97) 3. Scan Exams (14/12/97) - Dr Loren Rex	O'Reilly's Guesthouse, S.E Queensland	Kippa-Ring Physiotherapy Centre	Yvonne Strydom Ph 07-32833721	N/A
14-18/4/98	FIMM Congress and Workshops	Conrad Jupiters, Gold Coast	AAMM	Carillon Conference Management Ph 07-33682644	Pending
25-28/6/98	Basic Acupuncture Pain Management Seminar	ANA Hotel Gold Coast	Continuing Medical Pain Education	Marilyn Strauss Ph 07-55313810	Pending
24-29/9/98	Acupuncture Pain Management Seminar	ANA Hotel Gold Coast	Continuing Medical Pain Education	Marilyn Strauss Ph 07-55313810	Pending
Ongoing	Practice Assessment Module	Practice based	Continuing Medical Pain Education	Marilyn Strauss Ph 07-55313810	20-25 practice assessment points

Musculoskeletal Medicine Educational Activities

NEW SOUTH WALES

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
Wednesdays 7 - 8 am from 10-12/97	Sports Medicine Meetings	Orthopaedic Skills Laboratory, 34 Chapel St Kogarah	GP Orthopaedic Program	Cathy Hopkins Ph 02-93502830	N/A
11/11/97	GP Treatment of Common Fractures. Paediatric Orthopaedics	Conference Centre 1st Floor, St George Private Hospital 1 South St Kogarah	GP Orthopaedic Program	Cathy Hopkins Ph 02-93502830	4
7/12/97	Cervical Spine	St George Hospital Department of Orthopaedics, 34 Chapel St, Kogarah	RACGP	Vanda Licitis, RACGP Ph 02-98781599	10-12
11/97 (Weekend to be set)	Introductory RACGP/ACPM Manual Medicine Course	Sydney War Memorial Hospital, Waverley	ACPM	Peter Carroll Ph 02-96451581	36
27/5/98	Instructional Course on Back Pain	Sydney Convention Centre, Darling Harbour	Australasian Faculty of Rehabilitation Medicine	DC Conferences Ph 02-94396744	N/A
28-30/5/98	Australasian Faculty of Rehabilitation Medicine Conference	Sydney Convention Centre, Darling Harbour	Australasian Faculty of Rehabilitation Medicine	DC Conferences Ph 02-94396744	N/A
25-27/9/97	International Spinal Injection Society Conference	Sydney Convention Centre, Darling Harbour	International Spinal Injection Society	DC Conferences Ph 02-94396744	N/A
11/97-11/98	One day anatomy update courses: Back to the Back; Shoulder Joint; Head and Neck	Department of Anatomy & Histology, University of Sydney	Department of Anatomy & Histology, University of Sydney	Dr A Neill Ph 02-93651000	N/A

Musculoskeletal Medicine Educational Activities

VICTORIA

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
30/10/97 7.30-10.00pm	Ankle Workshop	Victoria University	AAMM	Vic Wilk Ph 03-95967211	Pending
27/11/97 7.30-10.00pm	Revision	Victoria University	AAMM	Vic Wilk Ph 03-95967211	Pending
6/11/97	Mobilisation of Peripheral Joints. Revision of Basic Manupulative Skills. Dr Hary Pavasaris	268 Waverley Rd, East Malvern	Melbourne Musculoskel-etal Medicine Group	Dr Harry Pavasaris Ph 03-94291811	N/A
27/11/97	New Developments in Low Back Pain Treat-ment: when specific exercises should be used	Cedar Court Rehabilitation Hospital	Dr Julie Hides (PhD), University of Queensland	Sally Pierson or Penny Smallwood Ph 03-9805 4292	
4/12/97	Osteopathic Exam-ination of the SIJ. Managgement Tech-niques for SIJ. Upper Limb Cortisone Injec-tions.- Dr Clive Kenna	268 Waverley Rd, East Malvern	Melbourne Musculoskel-etal Medicine Group	Dr Harry Pavasaris Ph 03-94291811	N/A

SOUTH AUSTRALIA

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
27/2 - 1/3/98	Dr Gurmitt Dhillon and Dr Margaret Taylor	Adelaide	Dr Gurmitt Dhillon	Gurmit Dhillon Ph 08-83361166 Fax 08-8365 0445 Email:dhi @cobweb.com .au	N/A

Contact Dr Norm Broadhurst (Ph 08-82044613) for current information about other activities.

TASMANIA

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
29/3/98 - 1/4/98	Pain & the Poppy - Opioids & Chronic Pain	Wrest Point Hotel, Hobart	Australian Pain Society	DC Conferences Ph 02-94396744	N/A

WESTERN AUSTRALIA
Contact Dr Arnold Jones (Ph 08-93904444) for current information about activities.

NEW ZEALAND
Contact Dr Mark Johnston (Ph 09-4265437) for current information about activities.

Musculoskeletal Medicine Educational Activities

The University of Sydney Postgraduate Diploma in Physical Medicine (Musculoskeletal)

A Postgraduate Diploma in Physical Medicine (Musculoskeletal) is conducted by course work on a part-time basis over two years. A total of six weeks full-time attendance on campus will be required to complete practical and clinical sessions. These sessions will be of one week's duration. The course is designed for general practitioners. A general training in all aspects of musculoskeletal medicine is included. The main emphasis of the course is to acquire knowledge of musculoskeletal anatomy, biomechanics and physical medicine diagnostic skills and treatment, including manual and injection techniques.

Enquiries:
Adam Tierney
Administrative Officer
Department of Anatomy & Histology
The University of Sydney NSW 2006
Phone: (02) 9351 7064 Fax: (02) 9351 6556

Applications close: 9 January 1998
Provisional CME approval - 30 points semester

The Flinders Graduate Diploma in Musculoskeletal Medicine

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Further information and enrolment forms:
Mr Michael McKay
Faculty of Health Science
Flinders University of South Australia
Bedford Park 5042
Phone: (08) 8201 3913
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For further enquires:

In New Zealand:
Mrs Veronica McGroggan
Administrative Assistant
Dept. Orthopaedic Surgery & Musculoskeletal Medicine
Christchurch School of Medicine
PO Box 4345, Christchurch
Ph: (03) 3641 086; Fax: (03) 3640 909
Email: vmcgroggan@chmeds.ac.nz

In Australia:
Dr Philip Watson
Suite 26, 259 McCullough St.,
Sunnybank, Queensland, 4109
Bus: (07) 3345 8999
Fax: (07) 3344 6688
AH: (07) 3344 4940
Email: pnwatson@medeserv.com.au