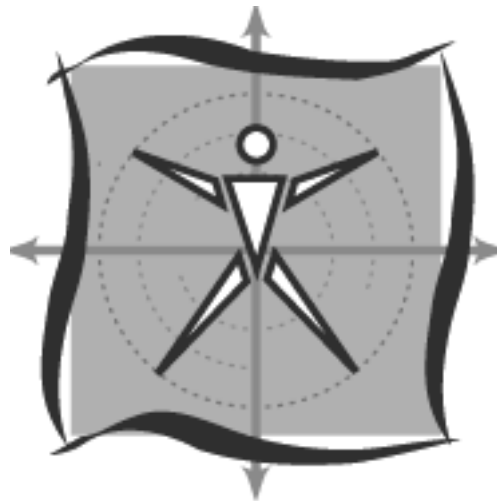


# *Australasian Musculoskeletal Medicine*



- **Mechanisms of complex pain syndromes**
- **AFMM standards: Lumbar medial branch blocks**
- **AFMM standards: Cervical medial branch blocks**
- **Muscle cramp**
- **Chronic Achilles tendinosis**
- **Articular cartilage defects**
- **Stiff painful shoulder**

## Australian Association of Musculoskeletal Medicine Office Bearers 1999 - 2001

### President

**Dr Steve Jensen** MB BS, Grad Dip Musc Med  
5 Stanlake St  
Footscray Vic 3011  
Ph: +61 3 93185233  
Fax: +61 3 93186630

### Vice President

**Dr Scott Masters** MB BS, FRACGP, FAFMM  
Caloundra Medical Centre  
39 Minchinton St, Caloundra, Q 4551  
Ph: +61 7 54911144  
Fax: +61 7 54911253

### Honorary Secretary

**Dr Michael Yelland** MBBS, FRACGP, FAFMM, Grad Dip Musc Med  
64 Wirraway Pde, Inala, Q 4077  
Ph: +61 7 32755444  
Fax: +61 7 32789987

### Honorary Treasurer

**Dr Derek Davey** MB BS, D Obst, RCOG, Grad Dip Occ H, Grad Dip Musc Med, FAFMM  
29 Craigie Rd, Newtown Vic 3220  
Ph: +61 3 52223677  
Fax: +61 3 52213104

### Committee Members

**Dr Robert Gassin** MB BS, FAFMM, Grad Dip Musc Med  
Cranbourne North, Vic 3977  
Ph: +61 3 59956999  
Fax: +61 3 59956700

**Dr Geoff Harding** MB BS, FAFMM, Grad Dip Musc Med (immediate past president)  
Sandgate, Q  
Ph: +61 7 32695522  
Fax: +61 7 38693288

**Dr Des Schimeld** MB BS, Grad Dip Musc Med, BSc(Hons)  
Dulwich, SA 5065  
Ph: +61 8 83612769

**Dr Phil Watson** BSc, MB, ChB, FRACGP, Dip Obst, Dip Mus Med, CIME  
Sunnybank, Q 4109  
Ph: +61 7 33457117  
Fax: +61 7 3216 9052

## New Zealand Association of Musculoskeletal Medicine Office Bearers 2000 - 2001

### President

**Dr James Watt**  
308 Lake Rd, Takapuna, Auckland  
Ph: + 64 9 489 5059  
Fax: + 64 9 486 4937  
jameswatt@clear.net.nz

### Secretary

**Dr John Robinson**  
256 Papanui Rd, Christchurch  
Ph: + 64 3 355 0342  
Fax: + 64 3 355 7071  
jonr@xtra.co.nz

### Treasurer

**Clemens Franzmayr**  
256 Papanui Rd, Christchurch  
Ph: + 64 3 355 7080  
Fax: + 64 3 355 7071

### Past President

**Dr Mark Johnston**  
16 Moana Ave, Orewa  
Ph: + 64 9 426 5436

### Academic Co-ordinator

**Dr Jim Borowczyk**  
256 Papanui Rd, Christchurch  
Ph: + 64 3 355 0342  
Fax: + 64 3 355 7071

### Committee Members

**Dr Peter Airey**, Christchurch  
Ph: + 64 3 352 6882

**Dr Steve Bentley**, Dunedin  
Ph: +64 3 474 1899

**Dr Gary Collinson**, Auckland  
Ph: + 64 9 624 1024

**Dr Alastair Fraser**, Taupo  
Ph: + 64 7 378 4080

**Dr David Jacks**, Havelock North  
Ph: + 64 6 877 7555

**Dr Andrew Moynagh**  
Ph: + 64 4 388 7018

**Dr Peter McKenzie**  
Ph: + 64 3 541 8911

**Dr Grant Thomson**, Whangarei  
Ph: + 64 9 435 0692

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# Editorial

*Dr Scott Masters*

This edition of the journal is a landmark publication. We have published the first guidelines in the world on medial branch blocks and radiofrequency ablation to the lumbar and cervical spine. Even more special is that much of the research into these techniques was forged here in Australia. Thus it seems more than appropriate that they are released in *Australasian Musculoskeletal Medicine* under the Australasian Faculty of Musculoskeletal banner.

It was also rewarding to see other musculoskeletal luminaries having work published in the general practice arena. Our new president Steve Jensen wrote a timely article for *Australian Family Physician* (9/01) on musculoskeletal causes of chest pain. In the same journal Michael Yelland, research guru, published his original research on spinal signs in back, chest, and abdominal pain. Recommended reading for anybody with a passing interest in matters

musculoskeletal.

Geoffrey Harding and I have also had feature articles published in successive *Medical Observer* issues. There seems to be a demand out in the medical literature for informed critical analysis and pragmatic summaries on musculoskeletal topics. In the past, much of the written material in the journals or the verbal offerings at sponsored meetings has been dominated by non-critical comment on problems that present to tertiary care rather than primary care. The tide seems to be slowly turning now as editors of medical publications start to seek out material of more relevance to both primary care doctors and patients.

We need to take this opportunity to avail these people of the AAMM database and encourage its dissemination. Members should notify their divisions and local medical associations about our resource. If they want informed comment, then

they should be directed to the president or one of the committee members for further help.

Recently two new members have joined the editorial team. Dr David Roselt and Dr Esther Langenegger. Both have over 10 years' experience in postgraduate study and both are fellows of the AFMM. David hails from Bundaberg and has a strong background in medical acupuncture. Esther works in Albury/Wodonga in a pain clinic and tutors for the Otago musculoskeletal medicine diploma course. Their professional expertise will enhance the quality of the journal well into the rest of this decade.

Finally, I'd like to pass on my appreciation to the outgoing committee members for all their hard work over the last two years. It is heart warming and inspiring to see the dedication these members devote to the cause. All for the love, not for the money. Hats off.

*Scott Masters*

## From the AAMM President, Dr Steve Jensen

**A**t the Hunter Valley Annual Scientific Meeting held in July 2001 I had the honour and privilege to be elected to the office of President of AAMM. I perused my old copies of the Green Journal, or the Bulletin as it was known way back when, and found the following list of ex-presidents: Geoff Harding, Vic Wilk, Norm Broadhurst, David Vivian, and Nikolai Bogduk. If you look at this list, there is a plethora of talented and dedicated people to whom we are all indebted because, in their own way, they have all helped musculoskeletal medicine move from the realms of alternative medicine to a position whereby it is a force in mainstream medicine. There is still a lot of turf to till, but I perceive that we now have a solid plot of land to develop. Indeed, when I reflect on those who have held this office before me, and what they have done for musculoskeletal medicine in Australasia, to say that I have some hard acts to follow is the proverbial understatement. Nonetheless, I will do my best to aspire somewhere towards their heady heights.

Increasing acceptance of musculoskeletal medicine by higher authorities, at least in the Workcover environment, is perhaps best demonstrated in Victoria, where those of us with faculty fellowships are recognised as specialists in our own right. Some of us have been invited to be members of Medical Panels, the final arbiter of disputed Workcover and transport accident claims. Most recently I have been invited to sit on a clinical advisory group for the Victorian Workcover Authority, which is trying to implement a new model for management of "sprain and strain" injuries. I hope to report to you on the outcome of this initiative in the not too distant future. Suffice to say that early signs are that, if it becomes implemented, there will be openings for many interested musculoskeletal practitioners, not only those with fellowships, to be actively involved in the

scheme, for which remuneration will be well above the current GP rate.

On contemplating this report, I reflected on how I came to be involved in AAMM. Like many members my introduction to matters musculoskeletal was via the course run by John Murtagh and Clive Kenna. I first attended one of these courses because I had moved from the protected environment of hospital practice to the frontline of general practice. Confronting me day in and day out were numerous people with musculoskeletal pain, and particularly low back pain. Published figures suggest that around 20% of GP consultations are for musculoskeletal problems, while low back pain alone represents about 5%. My undergraduate and postgraduate training, including that run by RACGP, had failed to provide me with any skills to assess or examine these patients, let alone manage these poor unsuspecting individuals!! The advert, and I forget where I came across it, implied that I would be able to learn all of this in 2 weekends! Sounded too good to be true! And although it did not provide me with a panacea, it did plant a seed which grew into a small forest until it consumed over 60% of my general practice consultations. With the help of a Flinders Diploma, I ultimately decided to take the plunge and move into full time msk medicine practice. I have absolutely no regrets apart from the fact that, if anything, I am *too* busy. Nearly all of my work is referred from either GPs or other medical specialists, with a smattering from paramedical practitioners. I read this as a sign that there is a huge need in matters musculoskeletal that is not currently being met by established groups.

But AAMM is not for specialists like me. That is where the Faculty fits in. AAMM is for primary care practitioners. I believe AAMM needs to continue to disseminate the skills and knowledge that collectively our organisation has in order to meet this need. Yes, we

need to be cognisant of the limitations in terms of reliability and validity of our physical examination, and other facets (no pun intended) of the evidence base. But even just examining someone confidently can be therapeutic. Thus, one of my aims of office is to expand on the work started by Murtagh/Kenna and admirably continued by the likes of the Quattro Amigos in Queensland, and Norm Broadhurst in South Australia and take our message and skills to general practitioners throughout the country. We need to find a way to get our foot in the door of institutions like the GP divisions in order to achieve this. So if there are any divisional leaders out there, please contact any member of the committee and we will endeavour to run a workshop in your area. If anyone has any helpful hints in enabling us to open these and any other doors, please forward them.

The rural doctors, I believe are a special needs group, given that they often do not have ready access to paramedical services. We should certainly be targeting that group to upgrade their skills in order to better serve the people of the rural and remote communities.

So this is also a call to all of you teachers out there. Even if you haven't been involved in teaching before! If you have been practising musculoskeletal medicine for a number of years, then you most certainly have many skills to pass on to your GP colleagues. So let us know who and where you are and we can try to involve you in teaching. It is a most rewarding experience. And I for one have found that it is a very sobering experience in that it is a sure way to find out how well you know a subject.

There are often unexpected benefits from teaching. The Quality Patient Education (QPE) CD enclosed with this issue of the journal was devised and produced by Dr Ronnie Moule, who was one of my students at the Swinburne University of Technology,

**From the AAMM President, Dr Steve Jensen**

Graduate School of Integrative Medicine. Having been bitten by the musculoskeletal bug, Ronnie conceived and developed this CD herself, and I think you will agree that the finished product will be a very useful tool on every GP's desk. As a service to members, the committee of AAMM has negotiated a very heavily discounted price for this CD so that it may be distributed to all members at no cost to them. The recommended retail price of this CD is almost half of your annual subscription.

Of course one of the highlights every year is the annual scientific meeting of AAMM. As I write this, the next meeting, to be held in Melbourne on the weekend of October 20 2002, is already being planned. The conference committee, and Vic Wilk in particular, is already working towards making this the best conference ever in terms of the scientific content, practicality of the workshops and entertainment within the conference framework. This time of year in Melbourne is a very busy but vibrant one with the spring horse racing carnival, as well as the Melbourne Festival for all of you culture buffs, and also the Melbourne Fringe Festival for all of you alternative bods out there. So I suggest that you write this date in your diary now.

Hope to see you there, if not beforehand.

## From the NZAMM President, Dr James Watt

# Coming of Age

**M**usculoskeletal medicine has come of age in New Zealand with achievement of vocational registration, inclusion on the Specialist Register and consequent change in funding, and acceptance by most insurance companies for payment of specialist fees. We have a contract with ACC for ordering examinations requiring specialist approval as well as an assessment contract.

What has led to this recognition? It has involved persistent hard work in a number of key areas. Initially we developed an educational program designed to promote musculoskeletal medicine amongst GPs. This started in the 1970s when an enthusiastic group arranged courses taught by Danish musculoskeletal physicians, Fossgreen and Pripp, and later, Rasmussen. Subsequently Barrie Tait arranged for renowned American osteopath, Philip Greenman, to visit for a semester and advise on the establishment of the Otago Diploma of Musculoskeletal Medicine.

Then in the mid 1980s Jiri Dvorak, a professor of neurology from Zurich, worked with a group who established a series of teaching courses and manuals which were used for teaching the practical part of the Otago diploma.

The group involved at this stage developed a close working arrangement with Australian counterparts (such that one of our number emigrated), working initially on a syllabus and later a Fellowship with its associated exam. This step has proven critical in acceptance of specialist recognition as it established a standard by which Fellows are judged.

Now that we have achieved this recognition, we are acutely aware of the need to provide evidence of our usefulness and cost effectiveness in order to further the professional recognition by colleagues and funders alike. Consumers already seem satisfied overall (as judged by attendance and satisfaction questionnaires).

There is little support in the literature as yet for much of what we do. Articles continue to be published showing poor intra-, and very poor inter-observer, reliability in clinical tests, which are often used for diagnosis. There is considerable nihilism regarding the worth both of such tests and of examination. A series of recent articles in journals of manual therapy<sup>1-6</sup> attest to the lack of both inter- and intra-observer reliability over a number of different tests performed often by very experienced practitioners. There are occasional articles<sup>7,8</sup> which demonstrate a degree of concordance, but these mainly test fairly coarse measures.

There is some evidence that a sophisticated measuring instrument, the lumbar motion monitor, could be used to differentiate between those with back pain and those without.<sup>9,10</sup> (While some may prefer merely to ask the question, at least this shows that there is a quantifiable difference.)

How then, do manual therapists expect to diagnose, let alone treat musculoskeletal problems? This, presumably, is not achievable by random manipulation.

Despite the bleak picture painted by much of the literature, there are a large number of therapists who have reputations that cause patients to seek, and be willing to pay for, their help. The possibility of accuracy of clinical diagnosis, was shown by Jull<sup>11</sup> in a paper which was later held to be open to scientific criticism.

The literature shows that manipulative therapy is helpful at least in acute cases, and the popularity of manual therapists suggests that they provide some benefit (even though natural history is a very handy ally). In order to achieve this benefit, therapy needs to be appropriate both in form and direction. This at least requires an educated guess.

The major cost to society, both in social as well as fiscal terms, comes

from the chronic cases – those whose symptoms have been present for longer than three months. It is this group which places the great demand on society both in lost productivity and in seeking relief from their chronic pain and impairment. It is also this group that the literature shows has a very small prospect of recovery. Thus, it is this group that the skills of a specialist musculoskeletal physician are most likely to benefit. The broad base of knowledge and skills, ranging from anatomy and biomechanics, including physical modalities and rehabilitation, to pain management using drugs, injection techniques and blocks, offer the greatest prospect of relief and recovery in the large proportion of cases unlikely to be helped by surgery.

It is imperative that we all keep records and review our patients' progress as required by our re-accreditation program, and that we then, either individually or in groups, publish our results. It is our performance by which we will be judged. If we demonstrate the benefit we offer, our specialty will flourish.

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## Glucosamine

Dear Editor

Thanks very much for your review of glucosamine. I think you have certainly covered the main issues involved comprehensively.

Some observations on the topic are as follows:

1. Unfortunately, there is no evidence that glucosamine sulphate and glucosamine hydrochloride are bioequivalent. Yet I think it will be the hydrochloride salt the majority of Australian patients will be taking. Even though there is reasonable evidence that glucosamine hydrochloride is absorbed, it's the sulphate salt about which most of the fabulous evidence has been constructed. So, can we really - in all good conscience - apply the same conclusions from one to the other?

2. Perhaps it would help physicians' insight into the complementary medicine industry if you told them how glucosamine sulphate is a *patented* salt and that's why *one* manufacturer is behind so much of the evidence (Rotta Pharmaceuticals), and that's also why you are likely to see other salts, like the hydrochloride, being marketed, riding on the sulphate's coat-tails. Some people believe that the sulphate salt is superior because it provides sulphate groups that bind glycosaminoglycan molecules together.

3. I don't think it's quite accurate to say (on page 2) that "In Australia there is only one TGA approved glucosamine preparation." There is only one glucosamine preparation which carries an AUST R number - and it happens to be glucosamine as hydrochloride. Nutrasense's product Arthro-Aid carries an AUST R number, but that does *not* mean that it is "approved" by TGA. It means it is a *registered* product, as opposed to *listed*. This is not to be confused with prescription only products for whom the TGA approves *indications*. They wouldn't have done that with Arthro-Aid. Incidentally, be-

cause all Australian drug manufacturers (unlike in the US) must comply with the Code of Good Manufacturing Practice, it is unlikely that labelling discrepancies will occur in our country. You could make that comment about products that are obtained from overseas. However, our products are regulated as foods not drugs. But all therapeutic goods in Australia - whether treatment or over-the-counter or complementary medicines - must be either listed or registered and made according to the Code of GMP or they have their manufacturing licence withdrawn.

4. With respect to *safety*, you should contact ADRAC for a print out of the adverse reactions they've had reported to glucosamine salts. We've had reported to our Medication Helpline rashes, urticaria, nausea, and so on. Boring and common though those reactions are, they still show that adverse reactions can occur and glucosamine is not benign. (I understand that the rigour of those reports is less than perfect, but that's the nature of postmarketing surveillance.)

5. I think there are more data for this drug than some of the medicines that get on the PBS, e.g., Zyban.

**Geraldine Moses**, BPharm, Postgrad DipClinPharm  
Mater Pharmacy Services  
Mater Misericordiae Hospitals  
South Brisbane 4101

# Mechanisms of Complex Regional Pain Syndromes

by Nikolai Bogduk, Newcastle Bone and Joint Institute

Some patients who suffer an injury to a peripheral nerve, and some patients who suffer a relatively trivial musculoskeletal injury, develop a bizarre and seemingly unique pain syndrome. In its most florid state this syndrome is characterised by the following:

- Pain
- Hyperalgesia
- Allodynia
- Vasomotor, sudomotor and temperature changes
- Trophic changes in the skin
- Motor impairment
- Osteoporosis.

A further feature is that the symptoms and signs seem disproportionate in severity to the nature of the precipitating injury, and occur in a region considerably larger than the one affected by the original injury. Thus, in the case of a nerve injury, the changes occur outside the territory innervated by the affected nerve. In the case of a musculoskeletal injury, the changes affect anatomical regions beyond that of the injured part. More curiously, the same symptoms can develop after visceral injuries (e.g., myocardial infarction) or central nervous system injury (e.g., stroke) and be manifest in a limb that is remote from the site of injury.

The pain in question is an unpleasant sensory experience but has no unique or singular quality. In some cases it may be burning in quality; in others it may be deep and aching. It may be dysesthetic; it may be spontaneous or present only when evoked by palpation of the affected part.

Hyperalgesia is an exaggerated or increased response to a stimulus that is normally painful.<sup>1</sup>

Allodynia (meaning foreign energy) is pain evoked by a stimulus that normally does not produce pain.

Vasomotor changes include vasodilation or vasoconstriction manifest respectively as reddening and swelling or cyanosis of the affected part.

Sudomotor changes include excessive sweating or dryness of the affected part.

Temperature changes mean warming or cooling of the affected part.

Trophic changes include keratosis, brittle nails, hair loss and brawny induration of subcutaneous tissues.

Motor impairment includes, muscle spasm and contracture of muscles each of which resist and interfere with voluntary movement.

Osteoporosis means elution of calcium from bones ostensibly because of increased osseous blood flow.

There is debate, confusion and controversy concerning the distinction between hyperalgesia and allodynia. Some regard the two as complementary aspects of the same phenomenon and mechanism, i.e., a shift to the left of the response curve of sensory nerves<sup>2</sup> (Fig. 1). Under these conditions, stimuli of an intensity that normally would be painful are perceived as more painful than usual. This constitutes hyperalgesia. Stimuli of intensities that would normally not be painful become painful. This constitutes allodynia.

This interpretation, however, ignores the essential meaning of allodynia, which is that the stimulus that evokes pain is qualitatively, not quantitatively, different from those that normally evoke pain. The cardinal example is brushing the skin, i.e., a mechanical stimulus that is delivered tangential to the skin surface and which does not involve pressure and deformation of the skin. This type of stimulus never evokes pain under normal conditions, regardless of its magnitude.

Confusion arises when touch perpendicular to the skin surface is used as the stimulus. Touch involves pressure, and pressure of sufficient magnitude can under normal conditions be painful. A shift to the left of the response characteristics of high threshold mechanoreceptors would render them low threshold mechanoreceptors. However, in that event, the nature of the modality involved does not change. Receptors normally capable of being nociceptive are simply rendered more sensitive. In contrast, when strictly defined, allodynia requires a switch in the modality.

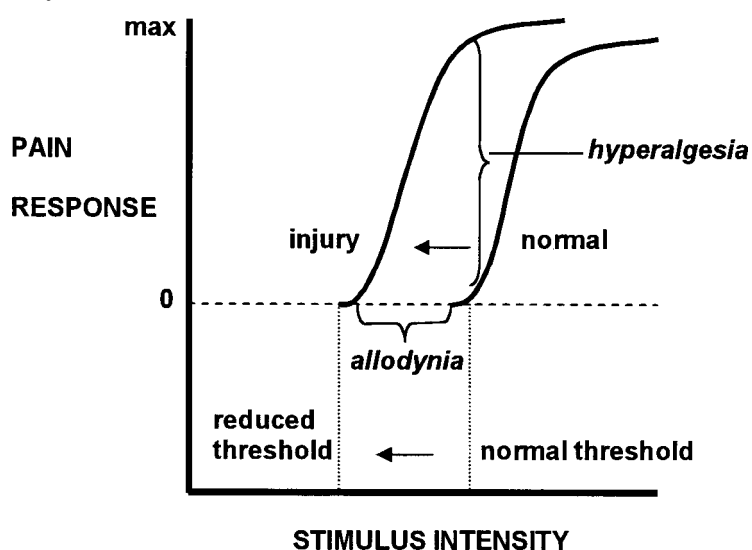


Figure 1. A definition of hyperalgesia and allodynia in terms of a shift to the left of the response curve of a sensory neurone. Under normal conditions the neuron is activated by a stimulus intensity that constitutes a normal threshold for nociception. After injury the response curve shifts to the left. Allodynia is the pain evoked by stimuli of intensity less than normal threshold. Hyperalgesia is the greater response to stimuli of an intensity that normally would be painful.



## Mechanisms of Complex Regional Pain Syndromes

For such reasons some authorities<sup>3</sup> have objected to allodynia being defined on the basis of a shift to the left. They prefer hyperalgesia to refer to the increased sensitivity of (normally) nociceptive afferents. On the other hand, Price et al<sup>4</sup> distinguish two types of allodynia. One they describe as low-threshold A $\beta$  allodynia, which is evoked by gentle brushing with a cotton swab. The other they describe as high threshold allodynia, which is evoked not by gentle stimuli but by intense static stimuli, like pressure, that normally are not painful. The latter would be what Campbell<sup>3</sup> refers to as hyperalgesia. Others<sup>5,6</sup> recognise similar distinctions. They consider brushing to be a dynamic (moving) stimulus, and refer to pain evoked by such stimuli as brush-evoked allodynia or dynamic allodynia. Pain evoked by static pressure they refer to as static hyperalgesia.

The danger of misusing the term allodynia lies in the inference that might be drawn. If allodynia is simply a shift to the left of the response curve of otherwise potentially nociceptive afferents, all that is required is a mechanism that lowers the threshold of activation of their pathways. This could readily be achieved by facilitating or disinhibiting their second-order neurones. However, if allodynia requires a change in modality, the mechanism cannot involve simply a lowering of threshold, it must involve a switch, in which non-nociceptive afferents gain access to nociceptive pathways, be that by developing totally new connections, or opening latent or previously suppressed connections.

In the present article, when quoting previous and especially older literature the term *allodynia* is used without further qualification to mean whatever the original author felt it to mean. Otherwise, when considering the mechanisms of this clinical feature, the terms *brush allodynia* and *pressure hyperalgesia*, as defined above, are used.

### Historical Perspective

In the past, patients presenting with a constellation of neurologic, vasomotor and trophic features attracted diagnostic labels<sup>7</sup> that:

	Example
Described the region affected	Shoulder-hand syndrome
Described the circumstances of onset	Post-traumatic pain syndrome Post-infarctional sclero dystrophy
Reflected one or more of the component features	Post-traumatic spreading neuralgia Post-traumatic painful arthrosis Chronic traumatic oedema Post-traumatic oedema Acute atrophy of bone Peripheral acute trophoneurosis Traumatic angiospasm Post-traumatic osteoporosis Traumatic vasospasm Reflex neurovascular dystrophy
Implied the mechanism	Sympathetic reflex dystrophy about 2-5% of such nerve injuries.

Two terms that arose into most common usage were *causalgia* - meaning burning pain, and *reflex sympathetic dystrophy (RSD)*. The term *causalgia* was applied to cases in which nerve injury was the precipitating event. RSD was applied to cases in which a nerve injury was not evident.

On clinical grounds, the vasomotor, sudomotor, temperature and trophic changes, were inferred to indicate sympathetic overactivity or underactivity, and it was the presence of these features that distinguished the syndromes from other painful conditions due to nerve injury, musculoskeletal injury, or visceral disease. Classical or archetypical descriptions of the two conditions were developed that grouped the clinical features as injury, neurological features and sympathetic features.

### Causalgia

#### Injury

Partial nerve injury was regarded as the cardinal aetiological factor in *causalgia*. The most frequently affected nerves were said to be the sciatic, the median, and the brachial plexus.<sup>7</sup> *Causalgia* was reported to occur in

### Neurological Features

In addition to the sensory loss resulting from the primary nerve injury, the patient suffers from pain and other sensory disturbances. The pain is usually burning in quality, intense, continuous, with episodes of more severe pain; and is usually felt distally in the affected limb.<sup>7,8</sup>

The other sensory disturbances are hyperalgesia and allodynia. These terms were used to refer to the phenomenon that the patients found that touching, or even brushing the skin of the affected part to be painful. These features were regarded as due to sensitisation of intact nerve endings in the affected limb by sympathetic activity, rendering them more easily activated by normal and subliminal stimuli. Evidence brought to bear in support of this inference was that:

- ♦ sympathetic features were otherwise prominent in the syndrome;
- ♦ sensitivity could be abolished by interrupting sympathetic activity by sympathectomy or by sympathetic nerve blocks,<sup>7-10</sup> or by the infusion of guanethidine.<sup>11,12</sup>

## Mechanisms of Complex Regional Pain Syndromes

- ♦ In patients successfully relieved of their pain and sensitivity, the injection of noradrenaline intradermally immediately reproduced the causalgic symptoms.<sup>13</sup>

### Sympathetic Features

The sympathetic features of causalgia were believed to evolve through an early and a late phase.<sup>7</sup> In the early phase, the vascular changes consist of vasodilation and consequent warmth with sweating and redness. Later, the vascular changes consist of vasoconstriction with consequent cooling and cyanosis of the skin. The skin undergoes atrophy and becomes glossy. Hair loss occurs. Initially the subcutaneous tissues are oedematous, but later they stiffen. Similarly, joints swell but later stiffen. In parallel, muscles initially spasm but later atrophy. Bones progressively become demineralised.

An attractive synopsis is that there is an early "angry" phase with vasodilation, warmth, redness, swelling, and spasm, followed by an atrophic phase of vasoconstriction, coldness, cyanosis, induration, stiffness and osteoporosis.

### Reflex Sympathetic Dystrophy (RSD)

RSD shares many of the features of causalgia, and differs essentially only in the nature of the precipitating cause.

### Injury

The trauma is often trivial. RSD has been reported after simple sprains,<sup>7</sup> dislocation,<sup>7</sup> fracture,<sup>7,14</sup> a crush injury,<sup>7</sup> a surgical procedure,<sup>7,15</sup> and even simple venepuncture.<sup>16</sup> Other causes include spinal injury, cerebrovascular accidents, spinal cord injury, myocardial infarction, diabetic neuropathy, and central nervous system disease such as multiple sclerosis (see Appendix I).

### Neurological Features

The cardinal feature of RSD is pain

that is continuous and burning in quality and usually felt distally in the affected limb. The pain is accompanied by hyperaesthesia and hyperalgesia.<sup>7,17</sup> The major difference between RSD and causalgia is the lack in RSD, of obvious sensory loss. Otherwise the neurological features of the two conditions are remarkably similar. Indeed, there is no detectable difference in the description of pain given by patients with causalgia and those with RSD.<sup>18</sup>

This lack of difference could be interpreted as suggesting that nerve injury does occur in RSD, but that the injury affects nerves that lack a cutaneous distributions such as muscles nerves and articular nerves, and therefore, escapes clinical detection.

As in causalgia, the neurological features of RSD were believed to be due to facilitation of peripheral nerve endings by sympathetic efferents and noradrenaline, for they could be relieved by sympathetic blockade<sup>7,9,10,19</sup> or intravenous guanethidine.<sup>11,20,21</sup>

### Sympathetic Features

The sympathetic features of RSD were grouped into three phases (Table 1).<sup>7</sup> As in causalgia an initial "angry" or "inflammatory" phase was typically followed by a cold, dry, stiff and atrophic phase. The involvement of the sympathetic nervous system in these changes was inferred because sympathetic blocks or guanethidine infusion could reverse the changes, at least in the early phases.<sup>7,9,10,11,20,21,22</sup> The joint stiffness and muscle atrophy seen in the late phase could not be reversed by neural blockade.

Histological studies of the joints of patients with RSD, revealed various degrees of synovial oedema, proliferation of synovial cells and capillaries, fibrosis of the sub-synovium, and some periarticular infiltration by chronic inflammatory cells.<sup>23</sup> Bone scans revealed a predominant localisation of nuclides in the juxta-articular region of bones suggesting a focal increase of blood flow to these areas.<sup>24</sup> This in-

TISSUE	TEMPORAL PHASE		
	EARLY	INTERMEDIATE	LATE
VASCULAR	Warm Dry	Cold Sweating	Cold
SKIN	Red	Cyanotic Glazed	Pale Smooth Glossy
HAIR		Loss	Denuded
NAILS		Brittle Grooved	Brittle Ridged
SUBCUTANEOUS	Edema	Brawny	Atrophy Fat loss
JOINTS	Swollen Tender	Thick Stiff	Fibrosis Ankylosis
MUSCLES	Spasm	Wasting	Atrophy
BONES		Osteoporosis	Atrophy

**Table 1.** The sympathetic features of reflex sympathetic dystrophy grouped in temporal phases to describe the phases of the conditions. Based on Bonica.<sup>7</sup>

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creased blood flow was inferred to be the mechanism of demineralisation seen in RSD.<sup>24</sup>

Thermographic studies showed that affected limbs may be warmer or colder than the unaffected limb but more commonly colder in chronic cases.<sup>25</sup> Temperature asymmetry, however, is not unique to RSD, for it can occur in other pain states, by asymmetries greater than 2°C, and particularly when greater than 3°C are more frequent in RSD than in other disorders.<sup>25</sup> However, although skin temperature in RSD may not be significantly different from that of the uninvolved limb, muscle blood flow and resting blood flow are significantly increased.<sup>26</sup>

### Extension

Perhaps the most bizarre feature of RSD is its extension to regions well beyond the initially affected area. Scintigraphic<sup>23,24,27</sup> and neurologic studies have shown that subtle and substantial changes can be detected in the opposite limbs of patients with RSD and there has been one case report of RSD affecting the whole body after surgery for low back pain.<sup>28</sup>

### Problems

Many problems befell the continued or wider recognition of causalgia and RSD. Foremost was the definition of liminal cases. Although the classical and archetypical descriptions rendered the recognition of florid cases straightforward, they did not define early or minimal cases. Critics asserted that:

- ♦ the label of RSD is quite practitioner dependent, ranging from a hyperalgesic, sweaty, oedematous, cool appendage to simply any surgical outcome that fails to meet the expectation of the operating surgeon.<sup>29</sup>
- ♦ of patients labeled as having RSD, perhaps 85% had nothing that even approached RSD, and clearly had other diagnoses such as neuralgias, peripheral vascular disease, and

even myofascial pain syndromes.<sup>29</sup>

Otherwise, critics<sup>30-35</sup> have noted that:

- ♦ sympathetic features are not consistent,<sup>33,36</sup> skin temperature changes are variable and may be the same, warmer, or cooler on the affected side;<sup>37</sup> therefore, this cannot be a discriminating, diagnostic criterion;<sup>30</sup>
- ♦ the cutaneous features of RSD do not necessarily imply abnormal activity of sympathetic nerves;<sup>33</sup> they could be manifestations of normal responses to injury;<sup>30</sup> coldness and cyanosis could be due to hypersensitivity to circulating amines<sup>33,35</sup> and warmth and redness could be due to neuropeptides possibly released antidromically from sensory nerves;<sup>35</sup> trophic changes can be ascribed to disuse<sup>30</sup> or immobilisation;<sup>31</sup>
- ♦ abnormal skin temperatures can occur in the absence of any noradrenergic vasomotor innervation;<sup>35</sup>
- ♦ pain does not correlate with vasomotor or sudomotor activity, and causalgic pain can occur in the absence of vascular changes;<sup>32,33</sup>
- ♦ microneurographic studies have detected no abnormal sympathetic activity in patients with RSD;<sup>32,33,38-41</sup>
- ♦ the effect of sympathetic blocks is unpredictable, and does not predict the effect of sympathectomy;<sup>33</sup>
- ♦ pain relief after blocks does not correlate with the duration of effect of sympathetic blocks;<sup>11,33,42</sup>
- ♦ pain relief after blocks is independent of the thermal effects of blocks;<sup>11,33</sup>
- ♦ sympathetic blocks relieve pain even when the causative lesion is proximal to the block;<sup>32</sup>
- ♦ pain is relieved by blocking the stellate ganglion with morphine which does not produce block of sympathetic efferents;<sup>32,43</sup>

- ♦ intravenous clonidine interrupts sympathetic transmission but has no effect on pain;<sup>33,44</sup>
- ♦ the effects of stellate ganglion blocks have never been controlled in any studies of causalgia;<sup>30,33,45</sup> one study found that only 15 out of 54 blocks satisfied criteria for an effective block;<sup>46</sup>
- ♦ stellate ganglion blocks are not target specific; very little of the injectate reaches the area of the stellate ganglion and much spreads elsewhere;<sup>29</sup>
- ♦ when compared to saline controls, intravenous guanethidine or reserpine has no diagnostic or therapeutic efficacy;<sup>47-50</sup>
- ♦ saline is just as effective as phenolamine in relieving pain;<sup>51-53</sup>
- ♦ investigations of the purported sympathetic and noradrenergic basis of RSD have found decreased, rather than increased, levels of catecholamines in the venous blood of limbs affected by RSD;<sup>30,33,54,55</sup>
- ♦ the intra-cutaneous injection of noradrenaline evokes pain in only a minority of patients but few patients remain sensitive to such injections when re-examined 12-16 years later;<sup>56</sup>
- ♦ with respect to taxonomy, critics have asked how to classify patients who lack sympathetic features or patients who have the sympathetic features but no pain.<sup>31</sup>

These observations strike at the heart of the traditional, clinical models of causalgia and RSD and their diagnosis. Denied sympathetic blocks and intravenous guanethidine, proponents are left with clinical features of questionable specificity upon which to make the diagnosis.

### A Resolution

At a conference held in 1993, proponents of RSD<sup>29,57</sup> agreed that:

- ♦ the term (RSD) had lost any clinical or research utility because of wide-

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- spread, indiscriminate use, with no diagnostic or descriptive criteria;
- the reflex that is required by the term has never been demonstrated;
- the linkage to the sympathetic nervous system is inconsistent and inconsistent;
- the term *dystrophy* is used imprecisely and the features may not be present consistently.

They resolved to create a nomenclature that was based on a descriptive method which was clinically useful but did not imply any particular mechanism.<sup>29</sup> They arrived at the term *complex regional pain syndrome* (CRPS) on the grounds that

**Complex:** recognised the intellectual and clinical complexity of the symptoms and signs encompassed by this rubric

**Regional:** described the distribution of the symptoms which is the hallmark of the conditions

**Pain:** is the sine qua non of the condition.

**Syndrome:** recognised that the condition was not ascribed to a single aetiology, and represented a cluster of symptoms and signs.

This nomenclature was adopted for the second edition of the taxonomy of the International Association for the Study of Pain.<sup>1</sup>

The condition previously known as RSD was reclassified as CRPS type I. Its diagnostic criteria were to be:<sup>1</sup>

1. The presence of an initiating noxious event, or a cause of immobilisation;
2. Continuing pain, allodynia or hyperalgesia with which the pain is disproportionate to any inciting event;
3. Evidence, at some time, of oedema or changes in skin flow, or abnormal sudomotor activity in the region of pain;
4. The diagnosis is excluded by the existence of conditions that would

otherwise account for the degree of pain and dysfunction.

The condition previously known as causalgia was reclassified as CRPS type II. Its diagnostic criteria were to be:<sup>1</sup>

1. The presence of continuing pain, allodynia or hyperalgesia after a nerve injury, not necessarily limited to the distribution of the injured nerve.
2. Evidence at some time of oedema, changes in skin blood flow, or abnormal sudomotor activity in the region of pain.
3. The diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

These revisions addressed several criticisms that had been raised about RSD and causalgia. The emphasis on "sympathetic" features was reduced. Instead, the emphasis lied on the presence of pain and allodynia or hyperalgesia. Oedema, changes in skin blood flow, or sudomotor activity needed to be present only at some time in the course of the condition. A link to the sympathetic nervous system was not implied<sup>29</sup> and, in particular, there was no implication that the sympathetic nervous system was responsible in any way for the pain.

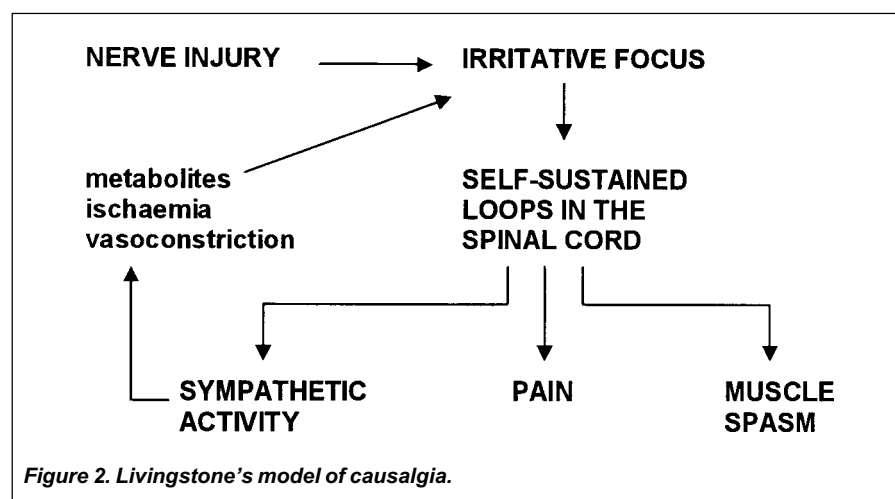
CRPS	
Type I	Type II
SMP	SMP
SID	SID

Table 2. The four types of complex regional pain syndrome (CRPS). SMP; sympathetically maintained pain. SID; sympathetically independent pain.

Indeed, a further dimension was added that did not prejudice the primary diagnosis. It was recognised that the pain of CRPS might be relieved by sympatholytic procedures or it might not. Pain not so relieved was classified as sympathetically independent pain (SID), whereas pain relieved by sympathetic blocks was classified as sympathetically maintained pain (SMP). Whether or not the pain could be relieved by sympathetic blocks was not considered an essential criterion for any condition. It was simply a feature that extended the classification to four basic conditions (Table 2).

### Mechanisms

In the past, authorities ventured to explain all the features of CRPS by singular, comprehensive models. These models, however, were essentially heuristic. They linked the various features descriptively into a single disorder, but afforded little or no insight into the specific mechanisms of each



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feature. One of the earliest models, that of Livingstone,<sup>58</sup> serves just as well today as it did when it was first conceived (Fig. 2). Authors of later models acknowledge that theirs are essentially based on that of Livingstone.<sup>59</sup>

The Livingstone model maintains that nerve injury creates a peripheral "irritative" focus that, in turn, generates "self-sustained loops" in the spinal cord that generate muscle spasm, pain, and sympathetic activity. The latter causes vasoconstriction and ischaemia in the periphery, forming metabolites that are responsible both for the sympathetic features of the condition and maintenance of the irritative focus.

While encapsulating the essential features of CRPS such models do not offer insights into the mechanisms involved. They do not explain the nature of the "irritative focus" or how it generates "self-sustained loops" or what these actually are. Moreover, these models accept that sympathetic activity is an essential part of the condition and mechanisms involved, which modern research has brought into question. Nevertheless, such models have served to direct attention towards individual components of the syndrome in the pursuit of the explicit mechanisms involved.

#### Brush Allodynia

Of all the features of CRPS, brush-evoked allodynia is the best understood. For allodynia there is a satisfying model supported by experimental evidence both in humans and in laboratory animals.

Brush allodynia is mediated by A $\beta$  fibres. The evidence for this is that:

- ♦ the reaction time for this sensation is consistent with the conduction velocity of large myelinated afferents;<sup>4,60-63</sup>
- ♦ the pinprick threshold for brush-evoked allodynia is equal to, or nearly equal to, that of low threshold mechanoreceptors in healthy skin;<sup>4,60-63</sup>

- ♦ electrical stimulation evokes pain from symptomatic tissues at stimulus intensities that evoke only tactile sensations in normal skin;<sup>4,40,60,63-65</sup>
- ♦ brush-evoked allodynia is abolished by nerve blocks at a time when tactile sensations are but other sensations remain unaffected.<sup>5,38,40,62,66</sup>

Brush allodynia also involves central neuronal plasticity. The evidence for this is indirect in humans but direct in laboratory animals.

In humans, the application of capsaicin to skin lowers the threshold for activation of tactile mechanoreceptors in nearby skin unaffected by the capsaicin.<sup>5,60,66-69</sup>

The mechanism of this sensitisation is central for it is evident upon electrical stimulation of peripheral nerves, which bypasses any putatively sensitised peripheral nerve endings.<sup>5,67</sup>

Similar phenomena have been observed in animals, and are associated with expansion of the receptive fields of WDR neurones and lamina I neurones in the dorsal horn.<sup>70-75</sup>

The expansion of receptive fields explains the extension of allodynia to regions beyond the immediate site of injury in CRPS.

Blocking peripheral nerves relieves allodynia in regions beyond the territory of the affected nerve.<sup>65</sup>

Primary nociceptive input initiates and maintains brush allodynia. The evidence for this is circumstantial.

Some studies have shown that nociceptive primary afferents are sensi-

tised to mechanical, thermal, and chemical stimuli in patients with brush allodynia, which implies ongoing activity in these afferents.<sup>6,39</sup> However, others have found no evidence of sensitisation of C fibres or A $\delta$  fibres.<sup>76</sup>

Nevertheless, other studies have shown that blocking afferent input from sources of nociception, either by using local anaesthetic blocks or by compressing nerves, abolishes both spontaneous pain and allodynia.<sup>5,60,65,77</sup>

It would, therefore, seem that ongoing input from primary afferents is essential for the maintenance of allodynia. The implication is that this input triggers central sensitisation. However, the mechanism by which nociceptive input initiates and maintains central sensitisation has not been established. One conjecture is that nociceptive input facilitates second-order neurones through the action of glutamate acting on NMDA receptors, and through the sustained action of substance P and neurokinin A.<sup>60</sup> Neurokinin A has been shown to spread beyond its immediate site of release following noxious stimulation,<sup>78</sup> and may well thereby excite distant neurones, rendering them more sensitive to peripheral input. An alternative conjecture is that central sensitisation could be due to loss of inhibition of second-order neurones resulting from trans-synaptic degeneration of inhibitory inter-neurons caused by nociceptive excitotoxicity.<sup>4,60,74,79</sup>

In essence, the mechanism of brush allodynia can be summarised as shown in Figure 3. There is no evidence that

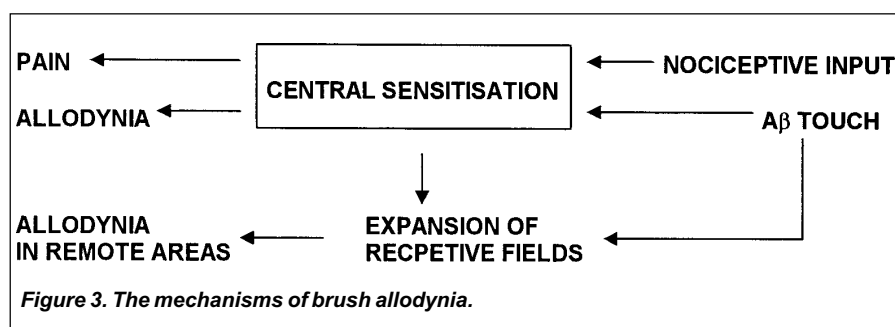


Figure 3. The mechanisms of brush allodynia.

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brush allodynia is sympathetically mediated. Experiments have shown that in patients in whom allodynia is relieved by sympathetic blocks, electrical stimulation of A $\beta$  fibres, and vigorous rubbing of the previously affected skin does not re-evolve allodynia.<sup>80</sup> This argues against peripheral sensitisation. Any role of the sympathetic nervous system must relate only to the sensitisation of primary nociceptive input or to the maintenance of central sensitisation. However, any such role is dependent on the validity of the data concerning sympathetically maintained pain.

### Static Hyperalgesia

The available evidence indicates that static, or punctate, hyperalgesia is due to a shift to the left of the response characteristics of nociceptive afferents, due to central sensitisation.

Experimental injury to the skin produces mechanical hyperalgesia in normal volunteers<sup>5,60,66,8</sup> that is mediated by nociceptive afferents<sup>6,39,66,68</sup> that exhibit increased sensitivity.<sup>82-84</sup>

Central sensitisation must be operating because punctate hyperalgesia is not abolished by peripheral blocks of the injured site<sup>68</sup> and outlasts the spontaneous pain induced by capsaicin injury to the skin.<sup>66</sup>

In animal experiments, the extension of hyperalgesia to areas remote from the original site of injury is associated with expansion of the receptive fields of second-order nociceptive neurones.<sup>72,85-87</sup>

The inability of peripheral blocks to relieve static hyperalgesia indicates that the central sensitisation involved differs from that which underlies brush allodynia. Whereas sensitisation to A $\beta$  input requires ongoing peripheral nociceptive activity, sensitisation to nociceptive input seems to be induced by a noxious stimulus but outlasts that stimulus. What is not known is how long that sensitisation lasts: whether it is self-limited or permanent; or whether it

is rekindled by periodic nociceptive input in order to appear long-lasting. In animals, features of hyperalgesia resolve spontaneously,<sup>88</sup> therefore, there are no models, at present, of the long-lasting hyperalgesia seen in humans.<sup>88</sup>

### Spontaneous Pain

In the past, basic scientists who have sought to explain the pain of CRPS have explored not only the mechanisms of the pain but also its relationship to sympathetic activity. Their investigations, however, predated the doubts that have been cast on the validity of sympathetic blocks and, therefore, the necessity of linking pain to sympathetic activity.

Accurate figures on the prevalence of SMP are hard to find, but some studies suggest that only 45%<sup>89</sup> or as few as 36%<sup>52</sup> or 33%<sup>77</sup> of patients with CRPS have SMP. Even fewer patients have genuine SMP if responses to blocks are discounted for placebo effects.<sup>52</sup> Phentolamine<sup>51-53</sup> and guanethidine<sup>47,48-50</sup> infusions are just as effective as saline infusions and so cannot be regarded as specific tests of sympathetic mediation. The only unchallenged hallmark of sympathetic mediation have been local anaesthetic blocks of the sympathetic trunk. However, a recent study now calls even them into question.

In a cross-over study, Price et al<sup>90</sup> performed stellate ganglion blocks or lumbar sympathetic blocks using either normal saline or local anaesthetic. In terms of immediate pain relief and relief of allodynia and hyperalgesia, the two agents were indistinguishable. Local anaesthetic differed from normal saline only in that it afforded longer-lasting relief. Consequently, the immediate response to sympathetic blocks cannot be held as a diagnostic criterion for sympathetically mediated pain.<sup>90</sup>

Consequently, the significance of sympathetic mediation of pain may have been overestimated in the past,

and the pursuit of a sympathetically mediated mechanism of pain applies to only a minority of patients. Nevertheless, the mechanisms that have been proposed serve equally for SIP as they might for SMP.

Review articles have suggested four possible mechanisms of the pain of CRPS. They are ephapses,<sup>90,92</sup> sympathetic afferents,<sup>32,33,90,92</sup> neuromas,<sup>92,93</sup> and ectopic activity in dorsal root ganglia.<sup>88,92,94</sup> Each of these requires an injury to a peripheral nerve and, therefore, serves to explain the pain of CRPS type II. No explanations have been proffered for CRPS type I. However, the pain (and other features) of CRPS type I can be explained if it is assumed that this condition involves occult (i.e., clinically unapparent) nerve injury.

The **ephapse model** requires that, after nerve injury and at the site of injury, connections develop between peripheral axons such that impulses along one are transmitted to the other. The connections could be between sensory afferents such that normal stimuli along the distal segment of an intact and non-nociceptive afferent are communicated to the proximal segment of a nociceptive afferent, resulting in non-noxious stimuli being perceived as painful. The connections could be between efferent sympathetic fibres and nociceptive afferent fibres, such that efferent activity is reflected as nociceptive activity.

Arguments against this model are that:

- ♦ such ephapses as do occur after nerve injury are not between the appropriate axons required by the model;<sup>90</sup>
- ♦ ephapses between sympathetic and afferent fibres have not been identified;<sup>45,59</sup>
- ♦ if ephapses occur between distal non-nociceptive axons and proximal nociceptive axons, the opposite should also occur such that peripheral noxious stimuli would be

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- perceived as not painful (this has not been observed);<sup>63</sup>
- ♦ ephapses take time to develop and, therefore, cannot explain the early onset of pain;<sup>8,65,92</sup>
  - ♦ local anaesthetic delivered to the putative site of such ephapses does not relieve pain,<sup>92</sup> and
  - ♦ afferent activity from ephapses is not synchronous with sympathetic activity.<sup>90</sup>

The model of **sympathetic afferents** has been promoted by one author<sup>32,33</sup> largely on the grounds that other models inadequately explain the pain of CRPS. Although the author refers to earlier anatomical literature on the existence of sympathetic afferents, this work has not been corroborated by modern studies; nor is there any convincing physiological evidence of afferent activity in sympathetic nerves in patients with CRPS. Earlier reports that morphine injected around the stellate ganglion relieves the pain of CRPS without affecting vasomotor activity<sup>43</sup> have been contradicted.<sup>94</sup> This model remains only a conjecture available for pursuit if other explanations are less satisfying.

**Neuroma-formation** is the one proposed mechanism of pain in CRPS that has most often been invoked in the literature on CRPS.<sup>35,45,90-93</sup> However, this does not necessarily argue that it is the most favoured or the best explanation. Rather, it may be only that neuroma formation is the most studied and best understood pathophysiological phenomenon of nerve injury. Therefore, when authorities are called upon to offer explanation for CRPS they gravitate to what is most studied and best understood.

Neuromas occur when peripheral nerves are transected. Within hours of transection, axon sprouts appear from the cut end of the proximal segment. Between two and 30 hours after injury, a small proportion of these axons exhibits spontaneous discharges.<sup>95</sup> With

the passage of time a greater proportion of axons discharge spontaneously and become mechanosensitive.<sup>96-100</sup> Moreover, the sprouts are sensitive to circulating adrenaline and noradrenaline, and the excitation of neuromas by amines can be blocked by phen-tolamine.<sup>91</sup> This latter feature rendered neuromas particularly attractive as a source of SMP.

The neuroma model is attractive in that it provides a pathology consistent with nerve injury and capable of producing spontaneous pain. However, while directly applicable to CRPS type II, it is not applicable to CRPS type I, unless it is acknowledged that in CRPS type I neuromas are formed on deep nerves, and have hitherto been clinically inaccessible. Moreover, the neuroma model predicts that the pain of CRPS would be relieved by blocking the neuroma, but peripheral blocks or neurectomy do not always succeed in relieving the pain of CRPS.<sup>8,32</sup> The neuroma model has also been rejected, at least for SMP, on the grounds that:

- ♦ there is no correlation between pain and vasomotor activity;<sup>32</sup>
- ♦ sympathetic activity is normal in CRPS;<sup>32,101</sup> and
- ♦ substances other than adrenaline and noradrenaline are equally capable of exciting neuromas, including these include acetylcholine, histamine and prostaglandin E.<sup>32</sup>

An adaptation of the neuroma model is the **constriction model**.<sup>102</sup> In experimental animals if ligatures are applied to a peripheral nerve so as to constrict it but not transect any of the axons, the animal develops pain, allodynia and hyperalgesia.<sup>102</sup> At the site of ligature, the axons are compressed by the ligatures and by the oedema that occurs.<sup>103</sup> Distally, axons degenerate. Virtually all the myelinated axons degenerate and nearly all the unmyelinated axons.<sup>103,104</sup> Physiologically, however, myelinated and unmy-

elinated fibres become spontaneously active, both distal and proximal to the site of injury.<sup>103</sup> The activity of C fibres and A $\delta$  fibres is presumed to be the basis for pain induced by this type of lesion. The source this activity has not been established for certain but one interpretation is that it arises from growth cones from the axons at the site of injury.<sup>103</sup> The injured axons develop an increased number of sodium channels and an increased number of  $\alpha$ -adrenergic receptors, which renders them susceptible to spontaneous discharge and to stimulation by amines.<sup>9,91</sup> In effect, the injured axons behave like neuromas, and the condition is sometimes regarded as a neuroma-in-continuity.<sup>91</sup>

The constriction model offers an explanation of CRPS type without requiring frank transection of a nerve, as in the case of neuroma. It can also be adapted to explain CRPS type I. Somatic injuries might fail to injure a peripheral directly but focal swelling of injured tissues surrounding a peripheral might nonetheless constrict it.

Ectopic impulse generation in **dorsal root ganglia**<sup>88, 105, 106</sup> is an appealing alternative to the neuroma or constriction models in that it explains why peripheral blocks, in some cases, fail to relieve the pain of CRPS. Unfortunately, this model has barely been explored in experimental animals and not at all in clinical studies. The circumstantial evidence is that:

- ♦ after transection of a peripheral nerve, not only do neuromas develop but dorsal root ganglion cells become spontaneously active;<sup>88, 105-107</sup>
- ♦ dorsal root ganglion cells also become active after constriction of a peripheral nerve;<sup>108</sup>
- ♦ the spontaneous activity that develops after nerve constriction is not abolished by transecting the affected nerve proximal to the site of injury or just distal to the dorsal root ganglion, but it is totally abolished if

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- the dorsal root is transected proximal to the dorsal root ganglion;<sup>108</sup>
- after nerve injury, dorsal root ganglion cells receive a neo-innervation by sympathetic efferent fibres;<sup>109</sup>
- spontaneously active dorsal root ganglion cells are activated by adrenaline,<sup>88,106</sup> and are suppressed by phentolamine.<sup>109</sup>

The latter phenomena render the dorsal root ganglion model an attractive explanation of SMP. Moreover, the dorsal root ganglion model offers an explanation of pain that is relieved by stellate ganglion blocks but not by regional intravenous blocks of the upper limb.

The model that has attracted the greatest acclaim is that of **Roberts**.<sup>110</sup> Indeed, it was hailed by Bonica as "brilliant".<sup>111</sup> This model proposed that at the time of injury, C fibres activate and sensitise wide dynamic range (WDR) neurones in the spinal cord. These neurones remain sensitised by normal inputs from large diameter afferents whose activity is perceived as painful and is maintained by sympathetic activity. In support of this model, Roberts and colleagues showed in animal experiments that only WDR neurones were activated by sympathetic stimulation,<sup>112</sup> and that such stimulation drove hair afferents and slowly adapting peripheral afferents.<sup>113</sup>

Arguments raised against this model are that:

- there is no correlation between pain and vasomotor activity.<sup>32,33</sup>
- the frequency of stimulation required to activate peripheral receptors by sympathetic stimulation is large and in excess of what is normally encountered in sympathetic nerves.<sup>59</sup>
- sympathetic activity is normal in CRPS.<sup>32,101</sup>
- if WDR neurons were sensitised, sensitivity should be also be evident for other modalities such as heat,

but this is not always the case.<sup>64</sup>

- the model requires that the sensitisation of WDR is maintained not by nociceptive input but by input from large diameter afferents. It predicts that sympathetic blocks would eliminate this sensitisation by normalising the activity of large diameter afferents. Were that the case, then stimulating large diameter afferents, electrically or by vigorous rubbing of the skin, should reinstate the pain and allodynia after a sympathetic block. This is not the case.<sup>4,64</sup> Successful sympathetic blocks eliminate hyperalgesia, and protect the patient from re-activation of their pain.<sup>64,80</sup>

### Central Mechanisms

Where all the foregoing models fail is in the explanation of CRPS that develops following lesions in the central nervous system (Appendix I), in which there is no peripheral injury, and no basis for the formation of neuromas or the development of spontaneous activity in the dorsal root ganglia. Indeed, the occurrence of CRPS after central lesions has repeatedly been raised as a criticism of all peripheral-based models of the pain of CRPS.<sup>32,33,92</sup> For this reason, several authors have gravitated towards a "central" mechanism for the pain of CRPS, although without elaborating any particulars.<sup>8,92,93</sup> Sunderland<sup>8</sup> introduced the notion of a "turbulence" hypothesis, in which causalgia was caused by disordered activity in the spinal cord induced by retrograde and trans-synaptic degeneration following peripheral nerve injury. Nathan<sup>93</sup> referred to the work of Denny-Brown<sup>114-116</sup> as an explanation of the spread of pain and hypersensitivity.

The studies of Denny-Brown<sup>114-116</sup> revealed that the organisation of the spinal cord and brainstem is far more complex than the peripheral models of CRPS currently admit. In the normal state, segmental nerves ramify over multiple spinal cord segments and elicit

both excitatory and inhibitory influences over multiple segments through the dorsolateral tract. Normal sensation involves not simply the response of a single neurone at the level of entry of a peripheral afferent, but a profile of excitatory and inhibitory activity over several segments. Transecting a peripheral nerve results in quite bizarre sensory changes. These changes do not involve ongoing peripheral activity, but occur as a result of loss of peripheral input. They include development of areas of numbness and areas of hyperaesthesia but most strikingly, these areas are not fixed; they change size, and can be made to shrink or enlarge by manipulating the tonic inhibitory functions of the dorsolateral tract either by injections of strychnine or by selectively transecting the tract.<sup>114-116</sup>

These observations indicate that the wiring of the spinal cord is such that simple loss of input from the periphery can result in hyperaesthesia, not because of sensitisation of the dorsal horn, but through loss of inhibition. Others have studied the same phenomenon more explicitly.

Studies in cats have shown that, following peripheral deafferentation, receptive fields of dorsal horn neurons increase<sup>117,118</sup> but the extent of expansion is too great to be accounted for by axon sprouting.<sup>118</sup> Rather, the investigators reasoned that the expansion was due to unmasking of latent synapses, ostensibly through loss of inhibition.<sup>117,118</sup> Furthermore, earlier work by Hillman and Wall<sup>119</sup> had shown that the peripheral receptive fields of low threshold and high threshold receptors overlap extensively, and have different excitatory and inhibitory effects on dorsal horn neurons. More significantly, they showed that these receptive fields and their effects were subject to descending modulation. Blocking descending inhibition increases the activity of dorsal horn cells and increases the sizes of their recep-



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tive fields.<sup>119</sup>

Meanwhile, other studies have shown that deafferentation causes spontaneous activity in nociceptive neurons in the dorsal horn or trigeminal nucleus.<sup>120-122</sup> This activity is not driven by peripheral input; indeed it can be exacerbated by spinal anaesthesia. Denied their accustomed peripheral input, these neurones behave as if they have unstable membranes and discharge spontaneously. Moreover, they lack receptors to conventional transmitter substances, and are unreceptive to iontophoretic application of GABA, glycine, glutamate and homocysteine.<sup>120</sup>

Collectively these observations allow for a central model of the pain of CRPS. The pain is not caused by peripheral nociceptive input but either by peripheral deafferentation or by loss of descending inhibition. Thus, the pain of CRPS could be a form of "central" pain, caused, in some cases, by peripheral deafferentation or, in other cases, by central lesions. Such a mechanism is the only one that can account for both peripheral and central causes of CRPS. Allodynia and hyperalgesia occur in company with the pain not because of excitation or facilitation, but as a result of loss of inhibition of surrounding segments.

### A Synthesis

Just as peripheral models do not explain the pain suffered by patients with central causes of CRPS, the central model does not explain those cases in which peripheral somatic blocks still relieve their pain. A diplomatic synthesis could be that there is no singular explanation for the pain of CRPS. Rather, it might be that different patients suffer injuries at different sites along a common pathway. As a result, patients may resemble one another clinically, but the mechanisms of their pain are slightly different. Another modification is that perhaps as patients evolve through different phases

of their condition, the mechanisms change. Thus, it might be that peripheral mechanisms operate early, but more central mechanisms operate later, when the condition becomes refractory to peripheral interventions.

### Sympathetic Features

The so-called sympathetic features of CRPS almost defy explanation. The confounding factors are the variation between and within patients, and selection bias in studies of these patients. For example, Baron and Maier<sup>123</sup> studied only patients with cold limbs, whereas Kurvers et al<sup>124</sup> studied patients with warm limbs.

Traditional descriptions of the phases or stages of CRPS (Table 1) are idealised and have not been corroborated. When tabulated according to duration of symptoms, the "sympathetic" features of CRPS type I do not differ<sup>125</sup>. Early in the course of the condition, a somewhat greater proportion of patients (86%) exhibit oedema, but oedema is present in 55% of patients at 12 months. Osteoporosis on x-ray is uncommon in patients with a history shorter than two months, but is evident in some 40% of patients with a history longer than two months. The incidence of other features such as colour difference, temperature difference, hyperhidrosis, trophic changes in hair or nails, as well as well and neurological features, does not differ with time<sup>125</sup>.

When tabulated according to whether the affected limb is warm or cold, the "sympathetic" features do not differ. Oedema occurs somewhat more frequently in patients with warm limbs and a short history; and trophic changes are more common in patients with a cold limb and a longer history. However, the incidence of hyperhidrosis, abnormal nail growth or hair growth, motor features or sensory features does not differ.<sup>124</sup>

Modern evidence clearly discounts sympathetic overactivity as the basis

for the "sympathetic" features of CRPS.<sup>123,126</sup> At rest, skin blood flow and skin temperature may be greater, lower, or the same as on the unaffected side,<sup>127</sup> but if patients are acclimatised to a warm environment, they exhibit essentially normal sympathetic reflexes. At most, the evidence suggests that in the early phases of CRPS, vasoconstrictor drive is deficient.<sup>123,126</sup> Moreover, the deficiency lies in the central nervous system and not at spinal or peripheral levels.<sup>126</sup>

Such deficiencies as do occur are selective for certain aspects of vasomotor control. Whereas vasoconstrictor drive may be decreased, sudomotor activity is normal or may be enhanced.<sup>128</sup> Although thermoregulatory skin blood flow may be increased in early CRPS, nutritive skin blood flow is not. Yet both are decreased in later CRPS.<sup>124</sup> These irregularities indicate that mechanisms other than, or in addition to, sympathetic activity affect the vasomotor state of the affected limb, particularly in the later stages of the condition.

Among the mechanisms suggested are:

- ♦ hypersensitivity or upregulation of peripheral adrenoreceptors on blood vessels;<sup>33,35,59 123,124,126</sup>
- ♦ increased vascular permeability due to inflammatory mediators;<sup>45,129</sup>
- ♦ antidromic activity in C-fibres causing vasodilatation.<sup>35,130</sup>

Accordingly, the "sympathetic" features of CRPS may involve a mixture of various mechanisms at different times or at different stages of the condition. Decreased vasoconstriction might complement antidromic or inflammatory vasodilatation, but when vasoconstrictor drive returns it might compete with antidromic or inflammatory vasodilatation, resulting in unstable and variable features.

Regardless of the mechanism of vasomotor disturbances contemporary authorities agree that there is no corre-

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lation between sympathetic dysfunction and pain.<sup>32,33,123,126</sup>

With respect to central causes of CRPS (Appendix I), peripheral mechanisms of the sympathetic features cannot be invoked. The only explanation must be disturbed descending control of sympathetic drive.

### Summary

Given the available evidence, Livingstone's model can be elaborated as shown in Figure 4. The model allows for either a peripheral nerve injury to initiate the process, or a central lesion of the nervous system. The model presumes that in CRPS type I an occult nerve injury occurs.

Nerve injury might cause deafferentation and/or neuroma formation, or involve a constriction injury of the nerve. Neuroma formation or constriction injury causes spontaneous activity in C fibres and A $\delta$  fibres, either at the site of injury or in dorsal root ganglion cells. This activity is transmitted to the nervous system where it excites and facili-

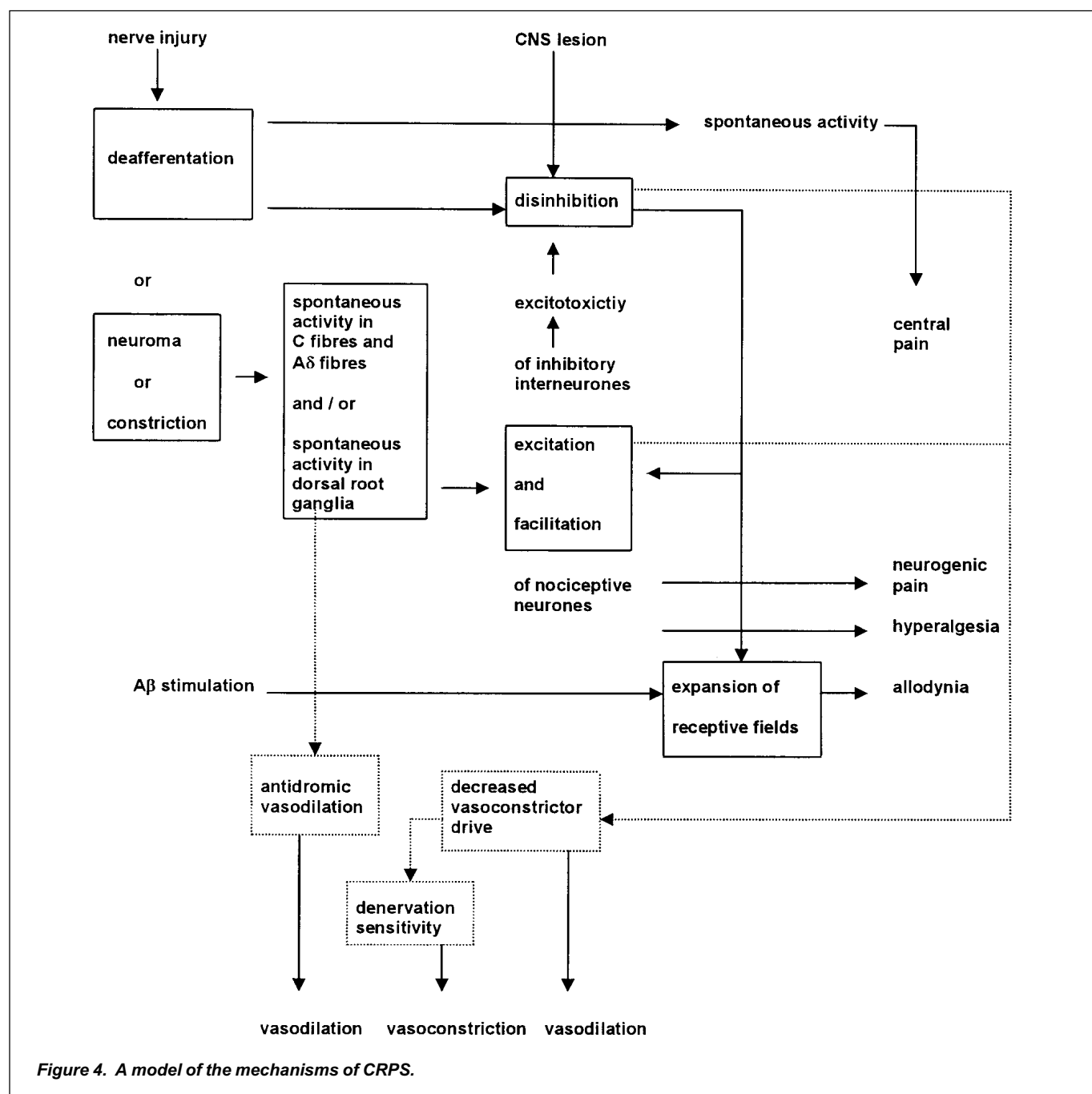


Figure 4. A model of the mechanisms of CRPS.

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tates nociceptive neurones in lamina I and in lamina V. That activity is perceived as pain, which by the mechanism involved is neurogenic pain. Facilitation of the central neurones becomes the basis for hyperalgesia, and also causes expansion of the receptive fields of adjacent neurones. The expanded fields capture evoked activity in A $\beta$  fibres which is received by the facilitated neurones, and is perceived as allodynia.

As well, or alternatively, inhibitory interneurons are stimulated by afferent activity and undergo excitotoxicity. Loss of inhibitory interneurons results in disinhibition of nociceptive neurones and in expansion of receptive fields.

On the other hand, additionally or alternatively, deafferentation alone may result in disinhibition of interneurons, and thereby facilitation of nociceptive neurones and expansion of receptive fields. Meanwhile, deafferentation may result in spontaneous activity in nociceptive neurones, thereby causing central pain.

A CNS lesion could evoke the same processes by causing disinhibition directly within the central nervous system. In order to accommodate visceral causes of CRPS, the model must assume that visceral disorders involve an injury to one or more of the nerves of the affected organ, or deafferentation of that organ.

Central to the generation of "sympathetic" features is disinhibition. This could be caused by central lesions or by deafferentation, and results in decreased vasoconstrictor drive, in the first instance. Subsequently, blood vessels develop denervation sensitivity. Meanwhile, in the case of peripheral lesions, spontaneous activity in nociceptive neurones may also cause antidromic vasodilation, which supplements or competes with sympathetically mediated vasodilatation or vasoconstriction.

The model expects and requires no

reinforcing effect of sympathetic activity on the processes that generate pain and other features. Such effects require more compelling data on the role of sympathetic nerves in CRPS.

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# Australasian Faculty of Musculoskeletal Medicine Practice Standards and Protocols: Lumbar Medial Branch Blocks

*The following guidelines have been officially endorsed by the Australasian Faculty of Musculoskeletal Medicine. These are the first published guidelines on medial branch blocks and radiofrequency ablation for the lumbar and cervical spine. They represent the standard that the AFMM expects of its members or professionals to whom they refer for these services.*

## Definition

**L**umbar medial branch blocks are a diagnostic procedure designed to test whether a patient's pain is mediated by one or more of the medial branches of the lumbar dorsal rami. They involve anaesthetising the target nerve with a tiny volume of local anaesthetic in an effort to relieve the patient's pain.

By convention, lumbar medial branch blocks are used to test whether a patient's pain stems from a given lumbar zygapophysial joint. For that purpose, the nerves that innervate the joint are anaesthetised.

This convention is based on the argument that, of all the structures innervated by the medial branches of the lumbar dorsal rami, the zygapophysial joints are the only ones that might harbour a discrete, focal source of chronic pain.<sup>1</sup> No pathology capable of producing chronic pain is known to affect the segmentally specific muscles innervated by the dorsal rami. For this reason, and because the ensuing term is shorter and more obvious in meaning, lumbar medial branch blocks can be, and have been referred to as (one of the means of achieving) zygapophysial joint blocks.

## Historical Background

The development of lumbar medial branch blocks was prompted by claims in 1971 that back pain could arise from the lumbar zygapophysial joints, and that this pain could be treated by severing the nerves that innervated these joints. Rees claimed that the nerves could be severed percutaneously with a special scalpel,<sup>1,2</sup> and later Shealy<sup>3-6</sup> claimed that they could be coagulated with a radiofrequency electrode.

It was subsequently shown that neither the Rees technique nor the Shealy

technique succeeded in severing the nerves to the lumbar zygapophysial joints;<sup>7-10</sup> but the concept of zygapophysial joint pain remained an attractive explanation for some cases of low back pain. Anatomical studies showed that the articular branches to the lumbar zygapophysial joints could not be accurately targeted for percutaneous procedures but their parent nerves, the medial branches of the lumbar dorsal rami, did constitute a valid and accessible target.<sup>9-12</sup> It also appeared logical that, if neurotomy was the basis of treatment for zygapophysial joint pain, local anaesthetic blocks of the medial branches would be the basis of diagnosing this pain and predicting response to neurotomy. For this purpose, target points for anaesthetising the lumbar medial branches under fluoroscopy were defined, and diagnostic blocks of these nerves were advocated as the appropriate diagnostic test for zygapophysial joint pain that was to be treated by medial branch neurotomy.<sup>7</sup>

The subsequent history of lumbar medial branch blocks became swamped by and confused with the use of intra-articular injections for the diagnosis of lumbar zygapophysial joint pain. Indeed, papers describing intra-articular injections dominated the literature on zygapophysial joint pain during the 1980s. However, once it emerged that intra-articular injections of steroids did not provide lasting relief from lumbar zygapophysial joint pain, lumbar medial branch neurotomy remained the singular means of possibly providing relief from this pain. Consequently, the utility of lumbar medial branch blocks became bound with the issues of the prevalence of zygapophysial joint pain and the efficacy of lumbar medial branch neurotomy.

In 1994 Schwarzer et al<sup>13</sup> established that in younger aged, injured workers with chronic low back pain, the prevalence of lumbar zygapophysial joint pain was about 15%. The next year, Schwarzer and others<sup>14</sup> established that its prevalence in older, non-injured, rheumatology patients was 40%. These studies showed that zygapophysial joint pain was common. Questions remained, however, concerning the validity of medial branch blocks for the diagnosis of this pain. These were answered by Dreyfuss and colleagues in 1997 and 1998.

Dreyfuss et al<sup>15</sup> showed that lumbar medial branch blocks were target specific, provided that precise target points were accurately used, and that needles were introduced in a particular direction. Structures other than the target nerves were not anaesthetised by lumbar medial branch blocks. Kaplan et al<sup>16</sup> showed that normal volunteers were protected from experimentally induced lumbar zygapophysial joint pain if the appropriate medial branches were anaesthetised. Together, these studies showed that lumbar medial branch blocks were target specific and were a valid test of zygapophysial joint pain.

Subsequently, van Kleef et al<sup>17</sup> demonstrated that lumbar medial branch neurotomy was not a placebo, and Dreyfuss et al<sup>18</sup> showed that dramatic and lasting relief from back pain could be achieved with lumbar medial branch neurotomy in patients carefully diagnosed with controlled diagnostic blocks of their lumbar medial branches. Accordingly, lumbar medial branch blocks were shown to have both diagnostic utility and therapeutic utility.

Given this background, lumbar medial branch blocks have replaced, or should replace, intra-articular inject-

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tions in the diagnosis of lumbar zygapophysial joint pain.

- ♦ Medial branch blocks are relatively easier to perform
- ♦ Medial branch blocks are safer and more expedient
- ♦ Medial branch blocks are more easily subjected to controls
- ♦ Intra-articular blocks, if positive, lack a valid subsequent treatment
- ♦ Intra-articular blocks lack proven therapeutic utility and predictive validity
- ♦ Medial branch blocks, if positive, can be followed by radiofrequency neurotomy
- ♦ Medial branch blocks have therapeutic utility and predictive validity.

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### Principles

The explicit purpose of lumbar medial branch blocks is to test whether the patient's pain is relieved by anaesthetising the nerves targeted. They are not a test of the patient's veracity. They test the hypothesis raised by the treating doctor that perhaps the pain is mediated by the nerves specified.

**If pain is not relieved**, the target nerves cannot be regarded as mediating the patient's pain. A new hypothesis about the source of pain is required. The pain may perhaps be mediated by other medial branches, or it may arise from a source not innervated by the lumbar medial branches.

**If pain is relieved**, the response constitutes prima facie evidence that the targeted nerves are mediating the patient's pain; but steps need to be taken to ensure that the observed response is not false positive.

It is possible that a patient may have several sources of pain. For example:

- ♦ they may have pain bilaterally at a given segmental level, in which case anaesthetising the left nerves should relieve the left side of their pain but not the right side (and vice versa);
- ♦ they may have pain from more than one segmental level on the one side, in which case anaesthetising the upper one or two of a series of nerves may relieve only the upper half of their pain.

In either instance, complete relief of all pain cannot be expected. Indeed, complete relief of all pain is contrary to what should be expected. Rather, a positive response can be entertained if there is complete relief of pain in a distinct topographical region that constitutes part of the patient's total complaint, but which corresponds to the area from which pain could be expected to be mediated by the nerves anaesthetised. Although this may constitute only partial relief of all of the patient's pain, it is more accurately and more informatively viewed as **complete relief of pain in the region targeted**. Remaining areas of pain may be targeted separately, or blocks might be extended (within reason) to include additional nerves that subtend those remaining areas.

Lumbar medial branch blocks have diagnostic utility. If positive, they identify the source of pain. Establishing a



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positive diagnosis protects the patient from the futile pursuit of other and competing diagnoses, and from undergoing presumptive treatment or treatment that is not appropriate for pain mediated by the lumbar medial branches.

Lumbar medial branch blocks have therapeutic utility. A positive response predicts a good chance of obtaining complete relief of pain from percutaneous radiofrequency neurotomy.

### Control Blocks

Control blocks are essential to exclude false positive responses, and to maximise the confidence of a securing a true positive response. The most rigorous form of control is the use of a placebo injection of normal saline under double blind conditions, but logistic and ethical considerations militate against the use of normal saline in conventional practice.

The injection of saline ethically would require informed consent. Impromptu, single blind injections of normal saline are unethical.

If saline is used it would have to be in the context of three blocks of the same joint. The first block would have to be with an active agent in order to establish, *prima facie*, that the joint is symptomatic. (There is no point in performing routinely a series of three, controlled blocks in a patient in whom there is no objective indication that medial branches are at all mediating the patient's pain.) The second block could not be the normal saline control, for a mischievous patient would know that "the second injection is always the dummy" and could respond appropriately. In order to maintain the controlling effect of chance and blinding, the second block would have to be either normal saline or an active agent, and the third block would need to be the reciprocal agent.

Comparative local anaesthetic blocks constitute a more practical form of control, that can be readily incorpo-

rated into routine and conventional practice.

A true positive response to comparative local anaesthetic blocks is one in which the patient reports complete relief of pain for a shorter duration when a short-acting agent is used, and for a longer duration when a long-acting agent is used. Such a response is referred to as "concordant" in that it is concordant with the expected action of the agents used.<sup>1</sup>

If a patient obtains complete relief of pain but does not report an appropriate differential response in terms of duration of relief, the joint is not excluded as the source of pain. Such paradoxical responses may still be consistent with the hypothesis being tested.

What should be the criteria for a positive response to comparative local anaesthetic blocks is a matter of judgement for the physician. However, the following principles apply.

If the consequences of a false positive diagnosis are relatively innocuous, less stringent criteria can be used. Complete relief of pain on each of two occasions, regardless of the duration of relief, will ensure that all patients with zygapophysial joint pain will be detected, i.e., "ruled in", but the cohort so identified will include false positive cases. This may not matter if irreversible therapies are not being considered. But physicians should be aware that the inclusion of false positive cases will undermine the success rate of any treatment.

If the consequences of a false positive diagnosis are serious, more stringent criteria should be used. In such circumstances, the response must be reliably true positive. The criteria for a concordant response are appropriate for this purpose.

If the consequences of a false positive diagnosis are dire, it would be imperative to perform triple blocks with saline controls, in order to be certain that the response is beyond doubt a true positive response.

What constitutes "innocuous", "serious" and "dire" is a matter of consideration and decision between the physician, the patient, and any other parties involved such as an ethics committee or institutional review board.

Notwithstanding these intellectual and clinical considerations, controlled diagnostic blocks are also cost-effective.<sup>2</sup> Failing to confirm a diagnosis with controlled blocks results in patients with false positive responses to uncontrolled blocks undergoing treatment. This not only results in a high rate of treatment failures, but also waste resources by having practitioners expend their time and skills treating patients who will not benefit. Controlled blocks reduce this wastage.<sup>2</sup>

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### Indications

The fundamental indication for lumbar medial branch blocks is the desire to know whether the patient's pain is mediated by the medial branches of the lumbar dorsal rami. This principle emphasises the fact that back pain, *per se*, is not an indication for diagnostic blocks. Not all patients need diagnostic blocks, so not all patients should undergo blocks.

Legitimate reservations can be raised about the need to perform blocks if the patient is going to be treated conservatively. In that event, the response to blocks does not affect management, and performing blocks can be viewed as superfluous. The only proposition that might be entertained is that medial branch blocks have diagnostic utility

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and, therefore, establishing a positive diagnosis might curtail the futile pursuit of other diagnoses. That proposition, however, needs to be weighed carefully against the likelihood of obtaining negative responses. Proving that patients do not have a condition that they are unlikely to have is a waste of the skills and resources of the investigator. Those skills could be better channelled into procedures and actions that have a greater chance of improving patient outcome. For this reason, attention should be paid to careful patient selection (see below).

The only validated treatment for pain mediated by the lumbar medial branches is percutaneous radiofrequency neurotomy. If this treatment is not available, the conduct of medial branch blocks can be justified only on the grounds of their diagnostic utility. However, if radiofrequency neurotomy is available, medial branch blocks are essential prerequisite before entering radiofrequency neurotomy.

Medial branch blocks are not indicated for acute back pain. There is a high chance of acute back pain recovering, regardless or even despite conservative treatment, and radiofrequency neurotomy is not indicated for acute pain. Therefore, it is not necessary to perform diagnostic blocks in patients with acute back pain. In principle, therefore, medial branch blocks are indicated only for patients with chronic pain. However, it could be conceded that there is merit in investigating patients with subacute pain, whose pain is not improving or responding to conservative management. Although it has not been demonstrated, the proposition is attractive that pinpointing the source of pain in these patients and promptly treating it could avert the onset of chronic pain behaviour.

### Patient Selection

A fundamental criterion for the selection of patients for lumbar medial branch blocks is that serious possible

causes of back pain, such as infection, tumours, vascular disease, and metabolic disease, have been excluded by careful and thorough history and examination, laboratory tests, and medical imaging. The work-up should provide a diagnosis of lumbar spinal pain of unknown origin, uncomplicated by associated features other than perhaps restricted motion.

There are no clinical features that allow a physician to predict with confidence that a patient with back pain will respond positively to lumbar medial branch blocks. Multiple studies have shown a lack of correlation between the results of conventional clinical examination and the response to controlled blocks.<sup>1-4</sup> Nor are features seen on CT scan predictive of response to blocks.<sup>5</sup>

There are, however, certain features described by Revel<sup>4</sup> that increase the likelihood of zygapophysial joint pain. They are age greater than 65, pain relieved by recumbency, and absence of aggravation of pain by coughing, by forward flexion, by rising from flexion, by hyperextension, or by extension-rotation.<sup>4</sup> If five or more of these features are evident, the likelihood ratio for a positive response to blocks is three.<sup>6</sup> Consequently, the presence of these features triples the likelihood of zygapophysial joint blocks being positive.<sup>6</sup> Even so, given the low prevalence of lumbar zygapophysial joint pain, the likelihood of a positive response is barely one in three.<sup>7</sup> If the clinical features are not evident, one needs carefully to consider the propriety of pursuing diagnostic blocks. Their yield is likely to be low, and the patient should be forewarned of that, rather than being lulled into optimism by the enthusiasm of the investigator.

Somatic referred pain into the lower limb is not a contra-indication for lumbar medial branch blocks. Pain referred as far as the leg and foot has been relieved by anaesthetising lumbar zygapophysial joints.<sup>7,8</sup>

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### Contraindications

#### Absolute

The absolute indications for lumbar medial branch blocks are conditions in which the conduct of a needle procedure under x-ray control might jeopardize the patient's health. These include, but are not necessarily limited to the following:

- bacterial infection, systemic or lo-

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calised in the region of the blocks to be performed;

- bleeding diathesis, due to haematological disease or anticoagulants
- possible pregnancy.

### Relative

Relative contraindications are conditions that do not preclude the conduct of lumbar medial branch blocks but which require special consideration because of the risks they pose. In the face of these conditions the investigator may elect not to perform medial branch blocks, or if they do undertake blocks special precautions are required. These conditions include, but are not necessarily limited to the following:

- allergy to contrast media, which may require cover with corticosteroid and H1 and H2 antagonists;
- allergy to local anaesthetics, which may require identification and use of a class of anaesthetic to which the patient is not allergic;
- concurrent treatment with non-steroidal anti-inflammatory medications, including aspirin, that may compromise coagulation, in which case, medication may need to be suspended for an appropriate period prior to the conduct of blocks.

Neurological signs are a relative contraindication for lumbar medial branch blocks, inasmuch as the neurological disorder should be managed first. If a patient also has back pain, that pain could be investigated with medial branch blocks once the neurological disorder has been managed or is being managed.

In that context, radicular pain is not a contraindication for medial branch blocks, but the investigator should not be under any misapprehension that medial branch blocks will relieve radicular pain. Medial branch blocks are indicated if the diagnostic hypothesis is that the patient's back pain and any somatic referred pain might be medi-

ated by lumbar medial branches. It is a concomitant but separate problem to their radicular pain.

### Facilities Required

#### *Radiological Equipment*

Fluoroscopy is mandatory for the conduct of lumbar medial branch blocks. The preferred equipment is a C-arm fluoroscope that allows the x-ray beam to be directed at any angle. Furthermore, in order to document the accurate placement of needles and the spread of contrast medium a device must be available to obtain either hard-copy films or an image on specialised paper.

#### *Needles, Gowns, Drapes, etc.*

A 90 mm, 25 gauge needle is optimal, for it is minimally painful when passed through the skin and muscles overlying the target joint.

The needle may or may not have a Luerlock, but such a lock is preferable.

A standard preparation tray may be used, which comes with cotton balls and gauze, but more elaborate trays can be custom made to come with needles and local anaesthetic.

Solutions for skin preparation may be an iodine-based solution (e.g., Povidone-iodine), chlorhexidine, or alcohol-based antiseptic (e.g., chlorhexidine 0.5% in 70% alcohol).

Sterile gloves are used.

Local anaesthetic agents may be injected directly from a syringe attached to the spinal needle, or minimal volume extension tubing may be interposed between needle and syringe, according to operator preference.

Given that only a small volume of agent is injected, a 2 ml syringe is all that is required.

#### *Medications*

Intravenous solutions, sedation or antibiotics are not required.

### Agents

Any conventional local anaesthetic can be used for medial branch blocks. Agents most commonly used are bupivacaine 0.5% and lignocaine 2%. Other concentrations that can be used are bupivacaine 0.75% and 0.25%, and lignocaine 1% and 4%. Because of the small volumes used, high concentrations should generally be used in order to obtain the most effective anaesthetic effect. For medial branch blocks, no more than 0.3 ml is required to block the nerve adequately.

Contrast medium is required if the operator wishes to test for intra-vascular placement of the block needle.

### Preliminary Procedures

#### *History and Physical Examination*

A history and physical examination are required to exclude pain likely not to be of zygapophysial joint origin and to identify or exclude contraindications to blocks. Otherwise, a history and physical examination are required to record baseline data concerning the location and extent of pain, including a visual analogue score, and the movements and activities of daily living that are customarily prevented by the pain.

On obtaining this baseline history, the patient should be briefed as to how their response to blocks will be measured. They should be instructed in the use of any pain diaries or visual analogue scales that might be used.

#### *Informed Consent*

Informed consent must be obtained. Although medial branch blocks should be safe procedures, like any invasive procedure they carry the nominal risk of infection, bleeding and allergic reaction.

The patient should be advised that the procedure is a diagnostic one, and should not be confused with a therapeutic procedure. They should be advised that they may or may not obtain relief, and that in particular they

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should be prepared for no relief ensuing. They should be advised that expecting any particular result confounds the purpose of the test, and that they should report the result honestly.

Other than to expect either relief or no relief, the patient should not be informed of the duration of relief to expect. They should be prepared only to the effect that if relief ensues they will need to monitor and record its duration and extent.

### *Premedication*

No premedication is required.

### **Technique**

#### *Preparation*

Neither physiological monitoring nor intravenous access is required.

#### *Positioning*

Although a posterior approach is possible, the most convenient and technically least demanding approach for lumbar medial branch blocks is an oblique. For this, either the patient lies prone and an oblique view is obtained by rotating the C-arm of the fluoroscope, or an oblique view is facilitated by the patient lying semi-prone with a pillow under their abdomen to tilt the target side upwards.

#### *Sterility*

The skin of the back must be adequately prepared as for an aseptic procedure, using one of the solutions listed above. The prepared area must be allowed to dry in order to ensure sterility. A fenestrated drape made of cloth, paper or plastic should be applied to cover the non-sterile areas surrounding the prepared area.

#### *Target Identification*

For the L1-L4 medial branches, the target point will be at the junction of the superior articular process and the transverse process which the target nerve crosses, midway between the superior

border of the transverse process and the location of the mamillo-accessory notch. This target point has been shown to be least associated with inadvertent spread of injectate into the intervertebral foramen or epidural space.<sup>1</sup> On oblique views, the target point lies high on the "eye" of the "scotty dog".<sup>1</sup> At the L5 level, the target nerve is not the medial branch but the L5 dorsal ramus proper. This nerve crosses the ala of the sacrum in a manner analogous to the passage of upper lumbar medial branches across a transverse process.

For a given lumbar zygapophysial joint both of the two nerves that innervate the joint will need to be anaesthetised. Caution should be taken in labelling and recognising the appropriate nerves, because the nomenclature of the nerves and the respective joints is out of phase; the joints and nerves do not take the same segmental numbers.

The L5-S1 joint is innervated by the medial branch of the L4 dorsal ramus (which crosses the L5 transverse process) and by the dorsal ramus of L5 (which crosses the ala of the sacrum). The L4-5 joint is innervated by the medial branches of L3 and L4 which cross the L4 and L5 transverse processes respectively. Note how the identity of the joint is numerically the same as the transverse processes onto which needles shall be placed, but that the names of the nerves are one segment less.

In identifying and recording a target point care should be taken to specify whether the segmental numbers refer to the joint, the transverse processes or the target nerves; otherwise confusion will emerge.

#### *Needle Placement*

*For L1-4 Medial Branch Blocks.* A puncture point on the skin is selected above and lateral to the target point, usually just above the tip of the target transverse process as seen of AP view. However, if an oblique view of the target area has been obtained, such

that the outline of the "scotty dog" is clearly evident, the puncture point is automatically selected by placing the tip of the needle on the skin directly in line, along the x-ray beam, with the target point on the "eye" of the "scotty dog".

The needle is then passed quickly through the skin and slowly through the back muscles in a straight line towards the target point. The progress of the needle is monitored by periodic intermittent screening to ensure that it does not stray from a direct course to the target point. Insertion is terminated once the tip of the needle strikes bone. This should be high on the "eye" of the "scotty dog".

Correct placement is confirmed by obtaining a postero-anterior view. In this view the tip of the needle should be at least opposite the lateral margin of the silhouette of the superior articular process, and preferably slightly medial to this margin. This is because the superior articular process often bulges laterally, overlapping the target point dorsally. If the tip lies lateral to this margin, it has struck the base of a thick transverse process instead of the superior articular process, the original target point having been judged too low on the "eye" of the "scotty dog". In which case, the needle should be re-adjusted dorsally, i.e., higher on the "eye", until correct position is obtained, and confirmed on PA views.

Once the needle is in correct position, the bevel should be directed caudally so as to avoid spread of the injectate to the intervertebral foramen.<sup>1</sup> This having been done, 0.1-0.3 ml of contrast medium can be injected to test that venous uptake does not occur. If it does, the needle must be readjusted by a millimetre or two and the test repeated. If there is no venous uptake, 0.3 ml of local anaesthetic is injected onto the target nerve. Both nerves that innervate the target joint are anaesthetised in the same way.

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*For L5 Dorsal Ramus Blocks.* The protocol is the same as for blocks at higher levels. The differences are that the target nerve is not the medial branch but the dorsal ramus itself, and that the target point is the junction of the ala of the sacrum with the superior articular process of the sacrum. This target point is recognised on PA views, *prima facie*, as a notch between these two bones. The target point lies opposite the middle of the base of the superior articular process and hence, slightly below the silhouette of the top of the ala of the sacrum. A higher placement is associated with spread of injectate into the L5-S1 epidural space, and a lower placement is associated with spread to the S1 posterior sacral foramen.<sup>1</sup>

A puncture point on the skin is selected just lateral to the target point so that the course of the needle will be in a ventral and medial direction, towards the target point, but medial to the adjacent iliac crest. Insertion is monitored to ensure that the needle does not stray over the top of the sacrum. A security in this regard is that, at all times, the tip of the needle must be below the upper margin of the sacrum.

Insertion is terminated once the needle strikes bone at the target point. If bone is reached but not precisely at the target point, the needle is readjusted until correct position is obtained. Once the needle is in correct position its bevel is directed to face medially. This reduces the risk of inadvertent spread of injectate to either the L5 intervertebral foramen or the S1 posterior foramen.<sup>1</sup>

#### Records

An image demonstrating the needle position must be obtained whenever a substance is injected. A plain radiograph may be obtained using conventional film or specialised paper. Such a record protects the operator in the event of alleged misadventure.

#### Reference

Dreyfuss P, Schwarzer AC, Lau P, et al. Specificity of lumbar medial branch and L5 dorsal ramus blocks: a computed tomographic study. *Spine* 1997; 22: 895-902.

#### Post-procedural Care

Upon removal of the needle, the skin is cleaned to remove the antiseptic and any blood. A small adhesive patch can be applied to the puncture sites, but is probably unnecessary.

If the patient complains of any untoward side effects following the procedure, appropriate action must be taken. A common reaction is a vaso-vagal response. This is managed with pulse and blood pressure observations and rest in the supine position. Rarely is any further intervention indicated.

Otherwise the procedure is usually well tolerated, and the patient may be allowed to dress and await evaluation and discharge.

Discharge instructions include:

- ♦ to contact the doctor who performed the procedure if there is any unusual symptom or pain following the procedure. Fever and tenderness greater than that which might be ascribed to a needle track may be signs of infection. To this end, an instruction sheet with a name and telephone number is useful. This sheet should be separate to any other data sheets given to the patient.
- ♦ to monitor the extent and duration of any relief that ensues. To this end a pain diary is helpful. However, critical is the time when the pain starts to return and the time that it returns to its former, accustomed intensity.
- ♦ if relief occurs, the patient should carefully attempt the movements and activities of daily living that customarily are restricted by pain, in order to determine whether these movements and activities can be

resumed while pain free. They should record movements and activities that they have been able to resume.

#### Evaluation

The singular reason for performing diagnostic blocks is **to obtain information**. That information depends on a reliable evaluation of the patient's response to blocks. Although performing the diagnostic block is an essential first step, the block itself does not make the diagnosis. Unless the patient's response is carefully evaluated and controlled for false positive responses, the act of performing the block is a waste.

There are several potential sources of error in the assessment of a response to a diagnostic block.

- ♦ Patients who expect and want a block to work may suffer a placebo response, and obtain or report relief for reasons other than the pharmacological effects of the block.
- ♦ A doctor who expects or wants the block to work may overtly or subconsciously coach the patient to report a positive effect even when one is not truly achieved.
- ♦ An assessor who wants the block to work may exercise observer bias, and report as positive a block whose effect has not truly been positive, or report as completely effective a block that has been only partially effective.
- ♦ Blocks may be performed at a time when the patient's pain is minimal, or even absent. In that event it is difficult to argue that any supposed relief obtained was due to the effects of the block, and not simply a reflection of the patient's low level of pain at the time.
- ♦ If the response to a block is evaluated immediately and only upon completion of the block, a false impression may arise. Having rested during the performance of the block the patient may obtain relief of the

## Lumbar Medial Branch Blocks

pain. If asked if there is any relief upon completion of the block the patient will correctly respond that there has been relief, but when subsequently they resume activities of daily living it may become apparent that the block has, in fact, not produced a positive effect.

- ♦ If a patient is discharged following completion of a block and their response is assessed at some time later, be that by telephone interview or at a subsequent consultation, they may suffer recall bias. They may not remember accurately how much relief they obtained and for how long. Furthermore, their report is entirely subjective, no independent trained observer having corroborated objectively the validity of their response.
- ♦ Although patients in absentia might be asked to complete a graphic record of their pain levels, this process is confounded by the patient having access to what they previously recorded. Guidelines for the completion of serial visual analogue scales for pain maintain that patients should not see their previous entries.<sup>1</sup>
- ♦ An untutored patient may fail to recognise that a block has been successful. This can occur when a patient has multiple sources of pain. Although a block may successfully anaesthetise one of their sources of pain it may not relieve other sources. Consequently, when asked, in absentia, if their pain was relieved the patient, having not obtained total relief of all of their pain, may report that it wasn't.

Such errors have potential ramifications with respect to both medicolegal proceedings and treatment. Blocks subject to error may lead to false conclusions about the veracity of a source of pain. Liability, therefore, may subsequently be misattributed. A false conclusion may lead to inappro-

priate therapy that is destined to fail. It is therefore, imperative that information based on diagnostic blocks be reliable and valid, i.e., free from error.

Certain errors can be reduced by performing diagnostic blocks under double-blind conditions. When the patient does not know which agent is being used, they cannot conform to an expected response. Simultaneously, the double-blind paradigm prevents the doctor, or an independent assessor, from coaching the patient as to what response to expect. Unless a diagnostic block is performed under double-blind conditions, the risks of response bias, observer bias, and reporting bias, remain eminent, regardless of how honest and objective a doctor claims, or insists, they are. The elimination of other sources of error require other measures, as outlined below.

### Towards An Optimal Protocol

At a Master Class conducted by the International Spinal Injection Society at the University of Newcastle in 1998, participants discussed the issues raised above. They agreed that the significance of diagnostic blocks for spinal pain lay in the information obtained, not in the execution of the block. They recognised the potential sources of error that obtained when blocks were not performed under controlled conditions.

The meeting resolved that the optimal means of reducing error and securing reliable diagnostic information was **real-time assessment**. Under this protocol, the response to a diagnostic block is evaluated immediately after the block, and for some time afterwards, at the clinic at which the block was performed, and by an independent observer using validated and objective instruments or tools.

Under the protocol, the doctor who is to perform the diagnostic block introduces the patient to an independent observer, typically a registered nurse

The doctor describes the pain that is being targeted by the forthcoming block, and if appropriate, highlights how this pain is distinguished from any other pain that the patient might concurrently have.

Both the doctor and the assessor should determine and agree that the patient's level of pain is sufficiently intense for any response to the intended diagnostic block to be credible and meaningful.

In this regard, a reasonable guideline is that the patient's present pain should be no less than 50% of their pain at its worst. Serious consideration should be given to the propriety of proceeding with blocks either in patients whose typical pain is less than 40 on a 100 mm scale, or in patients whose pain at the time when the block is to be undertaken is less than or equal to 20 on a 100 mm scale, for the natural diurnal variation in pain may be of this magnitude; and a decrease in pain by only 20 points may not be legitimately ascribable to the intervention.

Separately with the patient, the assessor records baseline measures pertaining to the patient's pain.

Separate from the assessor, the doctor performs the diagnostic block. Once the doctor is satisfied that the block has been adequately and safely completed, and that the patient has no resulting side effects that require immediate medical attention, they return the patient to the registered nurse for assessment and evaluation. The doctor takes no part in this evaluation, and is free to continue with other patients.

The assessor evaluates the patient's response to the block, administering the instruments that have been selected for this purpose. (See Schedule A, below.)

The assessment continues in principle for the duration of the patient's response to the block, or until the effects of the block have been reasonably established beyond doubt.

## Lumbar Medial Branch Blocks

- ♦ If the patient's pain has not been relieved, the patient can be prepared for discharge once it has been clearly established that there has been no relief.
- ♦ If the patient reports relief, this should be monitored and corroborated by the assessor for at least two hours or until the effects of the local anaesthetic agent wear off, whichever is the sooner. If possible logistically, the relief should be monitored for longer. In this regard, the period of two hours is nominated as a minimal period that seems practicable in general. Patients who need to travel or who need to return to work or to other duties may find it inconvenient to remain for a longer period.

The patient's response should be recorded independently by the assessor at prescribed periods. A reasonable schedule is to record the level of pain before the block, immediately after the block, 30 minutes after the block, and hourly thereafter. The assessment of pain should be complemented by an assessment of any improvement of disabilities, and by a narrative description of either how the patient feels about the relief obtained, or any difficulties that they may have concerning the response. The narrative serves to corroborate the response inferred from the visual analogue scores for pain. When a patient scores zero on the VAS but is not pleased by the effects of the block, doubts can be raised about the effectiveness of the block. Conversely, the assessor can be more confident in the response if the patient reports feeling the best that they have felt in years, or other such descriptions. Reciprocally, a patient who verbally reports that they obtained complete relief but fails to indicate that on their visual analogue scale should explain the discrepancy.

Once the assessor has completed the assessment it should be reported to

the doctor who should review it. The doctor's responsibility in this regard is to ensure that the assessment has been properly conducted and thoroughly recorded. The doctor cannot dispute the assessment. They can, however, identify whether there has been any misinterpretation, for example if the patient and the assessor both misconstrued which of a patient's several pains was targeted by the block undertaken. Any corrections to the record in this regard should be described in narrative. In order to avoid ambiguity, misinterpretation, or misrepresentation, the original assessment form should not be amended; instead, a revised assessment form should be appended to the original form, along with an explanation of why and how the revision was undertaken.

Once the doctor has reviewed the assessment, the patient can be discharged.

Subsequently, the assessor should review the patient in order to determine and record the patient's longer-term response to the block. This could be done by telephone interview on the following day. This review should record the ultimate duration of any positive effect of the block, in terms of when the patient's pain returned, how they felt during the period of relief, and any side effects or reservations about the effect. The use of telephone enquiry does not offend the reservation outlined above about recall bias, because the patient's primary response has already been recorded. The telephone enquiry simply obtains supplementary information concerning the patient's subsequent course and welfare.

### Reference

Carlsson AM. Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain* 1983; 16: 87-101.

## Schedule A. Instruments for Assessing Response

A variety of instruments might be used to assess the effects of a diagnostic block. There is a risk however, of overloading a patient with enquiries, questionnaires, and other tools. A pragmatic approach is to use three instruments.

- ♦ Relief of pain can be recorded by using serial visual analogue scales.
- ♦ Relief of disabilities can be recorded by having the patient nominate four, or as many as possible, activities of daily living that (1) are impeded or prevented by their pain; (2) which are likely to be restored, or should be restored, if the pain is relieved; (3) and which can practically be assessed in a clinic setting. (Examples might include bending, lifting, turning, sitting, walking. Impractical examples include return to work, sleeping, and having sex.) To this end, the assessor should record the nominated activities before the execution of the block, and observe and note the demonstrated degree of disability. After the block, and repeatedly throughout the period of assessment, these disabilities should be assessed and the degree of restoration recorded.
- ♦ To corroborate the assessor's record of the assessment, a powerful tool is to videotape the patient executing activities before and after the block. Such a record could also include the patient's mood and facial expression.

Appendix A, shows an example of a form that succinctly records an assessment.

### Interpretation

A positive response to a block is,

## Lumbar Medial Branch Blocks

prima facie, one in which there is complete relief of that part of the patient's pain which the blocks might be expected to relieve, for a duration commensurate with the expected duration of action of the local anaesthetic used. Partial reduction of that pain does not constitute a positive response. The only exception can be that the patient's accustomed pain is completely relieved but they complain of pain from the needle track, which would not be relieved by a medial branch block.

If the patient's pain is mediated by the nerves anaesthetised, they should obtain complete relief of their pain.

If the patient's pain is mediated by nerves other than the ones anaesthetised, they should obtain no relief of their pain.

If the patient's pain is mediated by several nerves, including but more than the ones anaesthetised, the patient will obtain relief of that part of their pain that is mediated by the nerves anaesthetised, but no relief of pain mediated by the other nerves. Examples include:

- ♦ in a patient with bilateral pain, if only the left side is blocked, the pain on that side will be relieved but the pain of the other side will not be relieved.
- ♦ in a patient with pain mediated by three consecutive nerves, if only the upper two are blocked, the patient may obtain relief of the upper part of their pain, but no relief of the lower part. The converse applies if the lower nerves are blocked.

Such responses nevertheless constitute a positive response, for the pain targeted by the blocks was completely relieved. The responses may be partial topographically but they are complete physiologically, in the targeted area.

In the event of such responses, a comprehensive or systematic approach may be undertaken for a complete and accurate diagnosis.

If the patients' pain is bilateral and

corresponds to the pattern for a particular segmental level,

- ♦ the two nerves on each side of the same segment may be blocked, or
- ♦ the patient could be assessed as if they have two pain sources, one on each side, and each side is addressed systematically but independently.

If controlled blocks on each side relieve the pain on their respective sides, a subsequent block may be undertaken simultaneously bilaterally, if it is necessary to show that all of the patient's pain can be relieved at the one time.

If blocking one side relieves all of the patient's pain bilaterally, there is no need to proceed with investigation of the opposite side.

The latter approach is intellectually more efficient and less subject to "diagnostic noise", for it allows the demonstration of bilateral pain stemming from just one side, if that is the case. However, in some settings it might not be practical or convenient for the patient to return for systematic investigation, in which case it might be preferable to block both sides of an apparently unisegmental pain simultaneously; or, once a single block on each side has been found to relieve the pain on the respective side, a confirmatory block may be undertaken simultaneously on both sides.

If the diagnostic hypothesis is that the patient's pain is mediated by multiple, consecutive medial branches, and if they obtain relief of the upper half of their pain when the upper nerves are blocked, it should transpire that blocking the lower nerves will relieve the lower half, but not the upper half, of their pain, and all of their pain should be relieved when all nerves responsible are blocked. Although it takes one more procedural session than it does to diagnose a unisegmental pain, it is preferable to diagnose multi-level pain as a staged procedure. The first block

relieves the upper region of the patient's pain, a second block relieves the complementary region of pain, and the third, confirmatory, control block can address all levels at once. A staged procedure in this manner, secures a valid diagnosis. It avoids wrongly presuming, *ab initio*, that the pain is mediated by multiple, consecutive nerves, and blocking all of those nerves. Doing so may lead to false positive results when more nerves are incriminated than warranted.

How these various combinations can be accommodated efficiently, using the minimum number of procedures is address below under "An Algorithm for the Investigation of Back Pain".

### Performance Parameters

Although experienced and expert operators may be more efficient and faster than the following standards require, the parameters that define a minimum level of competence and proficiency for the performance of lumbar medial branch blocks are:

- ♦ not more than eight adjustments or corrections of the course of the needle from insertion to reaching the target point;
- ♦ not greater than 1.5 minutes total radiation exposure time to block a given nerve, which includes exposure time to identify the target point and puncture point, prior to insertion of the needle.

## An Algorithm for the Investigation of Back Pain

### Introduction

The following algorithm for the investigation of low back pain is based on the best available evidence on the epidemiology of various identifiable sources of chronic low back pain, and is designed to promote the efficient use of invasive investigations. It is not de-



## Lumbar Medial Branch Blocks

NAME OF PRACTICE OR INSTITUTION			
DIAGNOSTIC BLOCK EVALUATION SHEET: LUMBAR BLOCKS			
PATIENT'S NAME: .....		DOB: ...../...../.....	
PROCEDURE: .....		DATE: ...../...../.....	
DOCTOR: .....		ASSESSOR: .....	
DESCRIPTIONS		PAIN MAP	
INDEX PAIN: .....			
CONCURRENT PAIN: .....			
.....			
.....			
.....			
FOUR ACTIVITIES LIMITED BY INDEX PAIN:		INSERT BODY CHART OF LUMBAR SPINE AND LOWER LIMBS	
1: .....			
2: .....			
3: .....			
4: .....			
VAS: Worst pain ever experienced: /10			
Worst ever index pain: /10			
Index pain today: /10			
RESPONSE: ADLs RESTORED:      1 2 3 4			
PATIENT'S REMARKS: .....			
100 .....			
90 .....			
80 .....			
V 70 .....			
A 60 .....			
S 50 .....			
40 .....			
30 .....			
20 .....			
10 .....			
0 .....			
Pre Post 30m 1hr 90m 2hr 3hr 4hr .....			
INTERPRETATION OF RESPONSE:		PLAN OF ACTION	
.....			
.....			
SIGNED: DOCTOR .....			
DATE: ...../...../.....			
ASSESSOR: .....			
...../...../.....			
CORRECTIONS/COMMENTS			

## Lumbar Medial Branch Blocks

signed to achieve a diagnosis in every patient. Indeed, the algorithm contains several nodes that call either for a cessation of investigations or for careful reconsideration of the propriety to proceed. the algorithm is based on the principles that:

- ♦ once an ambiguous or contradictory result has been encountered, it is wasteful of resources to venture to correct, overcome, or reverse that ambiguity by repeating procedures.
- ♦ the resources of a physician are better committed to investigating new patients who have a greater likelihood of obtaining a diagnosis than pursuing a diagnosis in patients in whom ambiguous or spurious results have been encountered.

In this regard, cardinal amongst the resources that can be so squandered are the skills and time of the physician who undertakes the investigations.

Otherwise, the algorithm is designed to provide a disciplined approach to the use of invasive investigations for lumbar spinal pain, and to avoid haphazard behaviour or investigations being undertaken essentially at the whim of a physician. In this regard, the algorithm is predicated by the pre-test probabilities of various conditions, and invites investigation of the more common conditions first, rather than pursuing any condition arbitrarily.

### *Part 1: Discogenic Pain*

The algorithm commences with and requires an MRI of the lumbar spine. This serves two purposes.

First it provides a screening test for red flag and exotic causes of back pain, such as tumours, infections, and metabolic disorders. Because of the high sensitivity of MRI, not only will such a screening test detect these rare conditions, it will also exclude them. That being the case, invasive investigations can be undertaken with-

out fear of missing an undisclosed or unsuspected serious condition.

The second step requires a decision as to whether the intervertebral discs are pristine. The purpose of this decision is to direct investigations to or away from the intervertebral discs.

Although discogenic pain can arise from discs that are normal on MRI this is uncommon.<sup>1</sup> In the interests of efficiency, therefore, it is recommended that investigations of the disc not be undertaken in the first instance in patients with normal discs on MRI. Although this ostensibly disenfranchises the few patients who might have discogenic pain but with normal discs, the recommendation is critical. Without it, the absurd situation arises in which every patient becomes entitled to undergo discography. Because of the low yield of discography in patients with normal discs, this constitutes a waste of resources. Physicians concerned about their patients with normal discs being disenfranchised are nevertheless free to make a case for investigating the discs of these patients. For the most part, however, it is inefficient to do so.

If on MRI the intervertebral discs are normal, it is unlikely that the patient will have discogenic pain, but reciprocally it is more likely that they have some other source of pain. Therefore, the pre-test likelihood of pain from one or other of the synovial joints of the lumbar spine and sacrum becomes greater than base rates. Accordingly, the algorithm invites investigation of the synovial joints.

If on MRI one or more of the lumbar discs is abnormal, the decision to investigate should be predicated on whether reasonable and appropriate treatment is available, should the investigation of the discs prove positive. The investigation of discogenic pain, using presently available techniques, is potentially harrowing for the patient, and carries a risk, albeit very low, of infection.

There may, however, be reasonable cause to test for a diagnosis of discogenic pain even though treatment is not available. Establishing a diagnosis of discogenic pain may prevent the futile pursuit of other diagnoses. The algorithm permits the use of discography for such purposes but calls for an active and conscious consideration of this indication.

If reasonable and appropriate treatment is available, the discs should be investigated. This recommendation is based on the best available evidence which indicates that amongst patients with chronic low back pain, internal disc disruption is the single most common cause; it accounts for at least 40% of cases, and is far more prevalent than any other identifiable condition.<sup>2,3</sup> In a patient with abnormal discs on MRI, internal disc disruption is the most likely diagnosis, and in the interests of efficiency should be the diagnosis first pursued. It constitutes a waste of effort and resources to undertake other investigations only to prove them negative in patients in whom those other investigations were never likely to be positive.

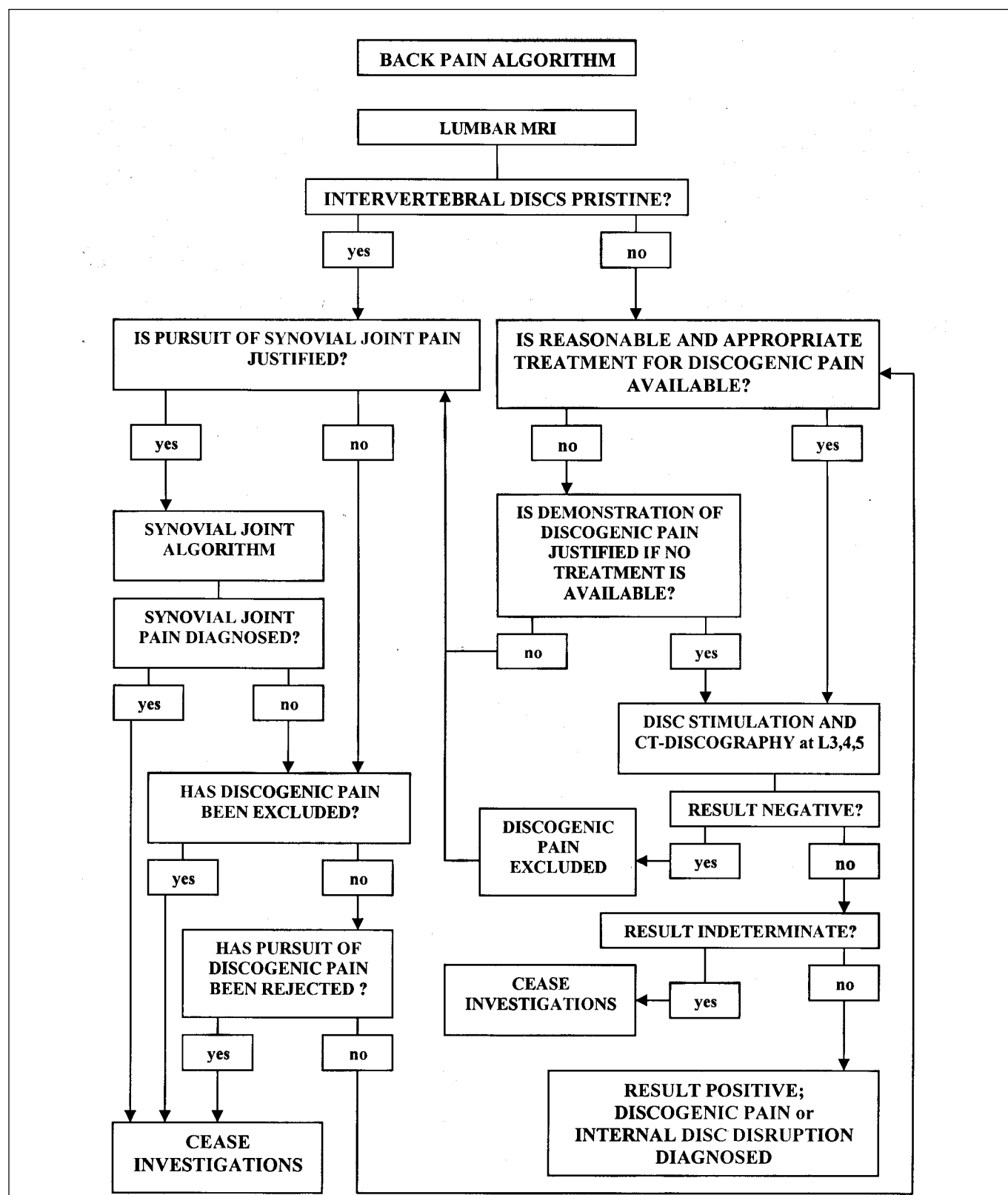
Disc stimulation and CT-discography is the only established means of pursuing discogenic pain. The techniques involved have been described in the literature,<sup>1</sup> and criteria for a positive diagnosis have been established.<sup>2,4</sup>

If the results of disc stimulation are negative, discogenic pain is excluded.

If discogenic pain is excluded, the question should be raised as to whether further pursuit of a diagnosis is justified. This decision relies on the judgement, inclination, or intuition of the physician involved. If further pursuit is not justified, investigations cease. Otherwise, the algorithm allows the patient to be investigated for sources of pain amongst the synovial joints of the lumbar spine and sacrum.

If the results of disc stimulation are not negative, they may be indetermi-

## Lumbar Medial Branch Blocks



### Algorithm Part 1: Discogenic Pain

## Lumbar Medial Branch Blocks

nate, i.e., not convincingly positive. This pertains to situations where many or all discs are positive to stimulation, or when no control disc is negative. Under these circumstances the algorithm recommends cessation of investigations. The patient may have discogenic pain, possibly at multiple levels. In that event, however, there is no valid treatment that might responsibly be prescribed. If the painful disc cannot be confidently identified, it cannot be targeted for treatment. If multiple discs are painful, there is at present no dependable treatment for multi-level discogenic pain. On the other hand, the patient's response may be false positive. In that event, the indeterminate result constitutes a cue that any additional or further investigations may also be liable to false positive results. Since there are no valid means of overcoming this possibility, the algorithm recommends cessation of investigations.

If the results of disc stimulation are neither negative nor indeterminate, by definition they will be positive. In that event a diagnosis of discogenic pain will have been made, and if the appropriate morphological features are evident on CT-discography, the diagnosis may be internal disc disruption.

### *Part 2: Synovial Joint Algorithm*

The algorithm for investigating the synovial joints of the lumbar spine and sacrum, commences with a set of clinical questions. The first is whether or not the patient's pain is located in the very midline. The available evidence indicates that patients with this sort of back pain defy the investigations encompassed by this algorithm.<sup>1,5</sup> The yield from zygapophysial joint blocks or from disc stimulation is essentially nil. Therefore, the algorithm invites reconsideration of the propriety of pursuing investigations, and implicitly recommends that they cease.

Whether the pain is bilateral is an intermediate question. Patients with

bilateral back pain are unlikely to have bilateral sacroiliac joint pain. They are more likely to have bilateral zygapophysial joint pain. Therefore, the algorithm asks whether there is good reason to believe that the patient might have bilateral sacroiliac joint pain. The expected, default answer is "no", and the algorithm proceeds to assessment of the zygapophysial joints. Nevertheless, the algorithm does allow for consideration of bilateral sacroiliac joint pain.

The third question of the algorithm is whether the patient's pain is entirely caudal to the L5 level of the lumbar spine. The basis for this question is that in patients proven to have sacroiliac joint pain, in all instances the pain is perceived caudal to the L5 level. Conversely, no patient with proven sacroiliac joint pain has been described who had pain extending above the L5 level.<sup>6</sup> Pain below L5 does not necessarily implicate the sacroiliac joint as the source,<sup>6</sup> but pain above L5, renders sacroiliac joint pain unlikely, and by implication, promotes the likelihood of zygapophysial joint pain. Consequently, this third question is pivotal to the efficiency of the algorithm. It is worth pursuing sacroiliac joint pain if the pain is entirely caudal to L5, but not if it extends above L5.

In the event that the patient's pain does not stem from the sacroiliac joint, that will be established at the expense of only one block. It is more efficient, therefore, to exclude the sacroiliac joint in the first instance, than to exclude or pursue zygapophysial joint pain, for the latter may require multiple investigations.

If the patient's pain is entirely caudal to L5, a sacroiliac joint block should be undertaken.

If the block is negative, the patient is considered for zygapophysial joint blocks.

If the block is positive, a confirmatory block should be undertaken.

If the confirmatory block is positive,

the diagnosis of sacroiliac joint pain is established.

If the confirmatory block is negative, a conundrum arises. Either the first block was false positive, or the second block was false negative. Since this conundrum cannot be resolved without multiple, repeated investigations, the algorithm recommends cessation of investigations, with the diagnosis remaining indeterminate.

Before allowing zygapophysial joint blocks the algorithm asks if the patient is negative to Revel's tests.<sup>7</sup> These tests do not establish that the patient has zygapophysial joint pain, but they do increase the likelihood to a modest degree. The tests, however, are essentially negative in nature. They require the absence of certain features. If any of the tests are positive, the likelihood of zygapophysial joint pain drops. The algorithm requires careful reconsideration of the propriety of summarily proceeding to zygapophysial joint blocks in a patient who does not satisfy Revel's criteria.<sup>7</sup> In that event, the likelihood of obtaining a positive result from blocks is low. To proceed with blocks should be justified on grounds greater than a guess or a whim.

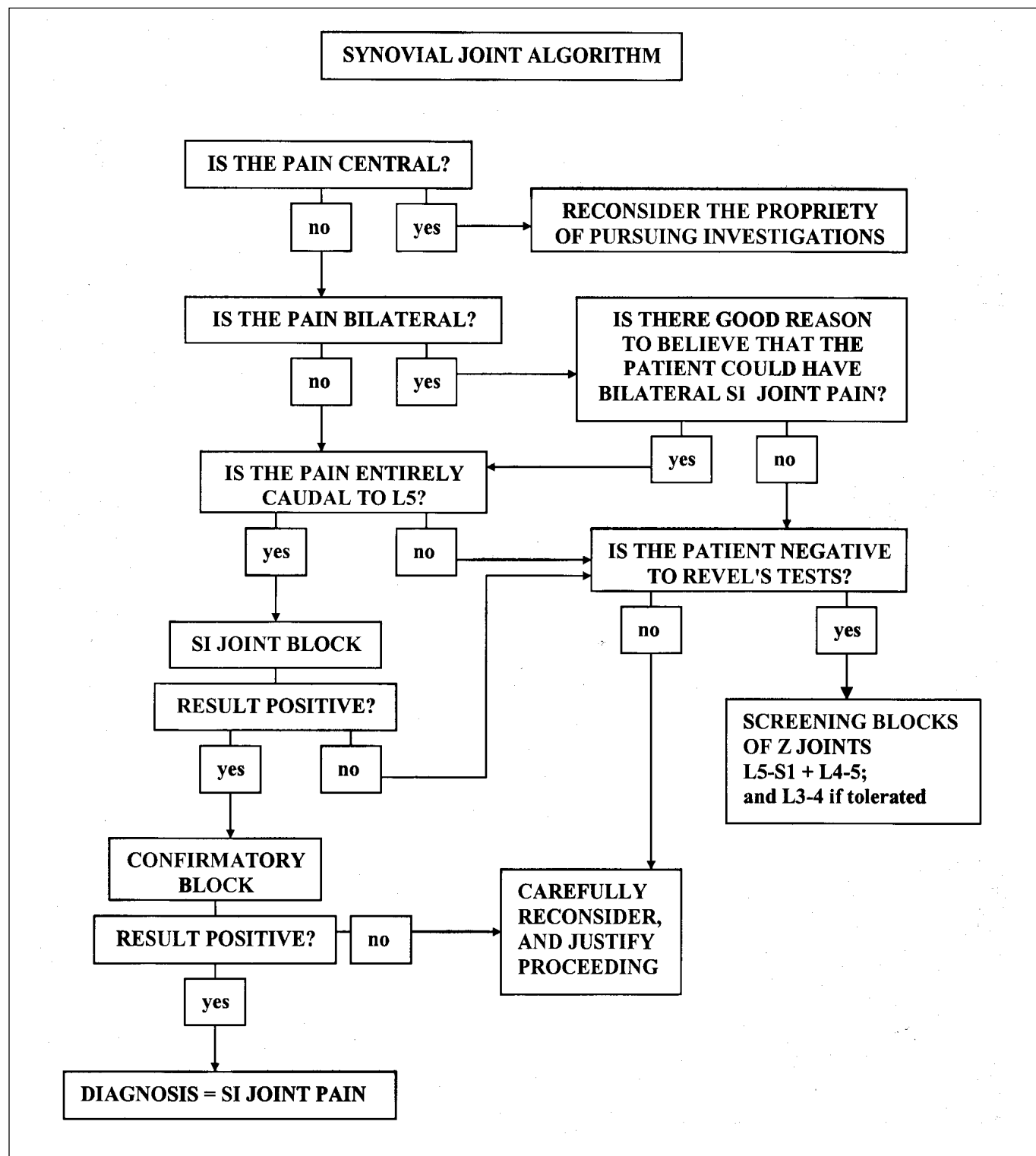
If a patient is negative to Revel's tests, their zygapophysial joints can be investigated.

### *Part 3: Zygapophysial Joint Blocks*

The zygapophysial joints are entertained last in the algorithm because they are the least likely sources of chronic back pain in the working age population, with a prevalence of less than 15%,<sup>5</sup> and probably closer to only 10%.

Different figures, however, apply to an older population without a history of injury. In those patients the pre-test likelihood of zygapophysial joint may be 40% or higher.<sup>8</sup> In that population, zygapophysial joint blocks become a prime investigation, ahead of disc stimulation, and possibly ahead of

## Lumbar Medial Branch Blocks



Algorithm Part 2: Synovial Joint

## Lumbar Medial Branch Blocks

sacroiliac joint blocks.

The low prevalence of lumbar zygapophysial joint pain predicates the design of the algorithm. Because zygapophysial joint pain may arise from any of a number of segmental levels, multiple investigations may be required to detect a symptomatic joint. However, the low prevalence of this condition means that the majority of such investigations will be negative and fruitless. It, therefore, becomes inefficient to pursue zygapophysial joint pain, one joint at a time, only to exclude all joints in the majority of cases. For this reason the algorithm recommends a multi-level screening test.

The virtue of a screening test is its negative predictive value. If the likelihood is that the majority of patients will not have zygapophysial joint pain, it is efficient to establish this expeditiously. Not only are resources conserved but the patient does not need to suffer repeated invasive tests in vain.

At a single sitting, both joints at L4-5 and L5-S1, bilaterally if indicated, can be anaesthetised. If the patient can tolerate the additional steps required, the L3-4 joints can be added.

If the result of this screening test is negative, zygapophysial joint pain will have been excluded, and investigations can cease.

If the result of the screening test is positive, zygapophysial joint pain is implied, but its exact source is not evident. That requires anaesthetising joints one at a time.

The algorithm recommends commencing arbitrarily at L5-S1 in order to pinpoint the exact joint that is the source of pain.

If blocks of L5-S1 are negative, the L4-5 joint should be considered and blocked.

If L4-5 blocks are negative, the question is posed whether it is reasonable to suspect or to test L3-4. The basis for this question is that L3-4 is an uncommon source of pain.<sup>5</sup> The physician

should, therefore, have good cause to suspect this joint, lest they perform investigations that prove that it is not painful.

If at any time a block is positive, it should be followed by a confirmatory block.

If that confirmatory block is negative, investigations should cease. A negative response raises a conundrum. Either the first block was false positive, or the second block was false negative. Since this conundrum cannot be resolved without undertaking multiple further blocks, the algorithm recommends cessation of blocks, with the diagnosis remaining indeterminate.

If the confirmatory block is positive, a diagnosis of zygapophysial joint pain is established.

If at any time a block of a single joint or of both joints at a single segment is partially positive, in that it provides complete relief of pain but only in part of the patient's region of pain, yet the patient responded to multi-level screening blocks, the response indicates zygapophysial joint pain at multiple levels. In that event, the next most likely joint should be blocked, in an effort to relieve that part of the patient's pain that was not relieved by the previous, single-level block.

If the response to the block of the second level is negative, the patient's responses to blocks should be carefully reconsidered, for their responses to screening blocks are not concordant with their responses to blocks at single levels. Their response to screening blocks may have been false positive.

If the response to the block of the second level is positive, in that it relieves that part of the patient's pain that was not relieved by the first block, a diagnosis can be entertained *prima facie* that the patient has two-level zygapophysial joint pain. That diagnosis can then be confirmed with a control block in which both symptomatic levels are simultaneously anaesthe-

tised, so as to reproduce and confirm the effect of the original screening block.

If the response to the control block is positive, the diagnosis of two-level pain is confirmed.

If the response to the control block is negative, the patient's responses to blocks should be carefully reconsidered, for their responses to blocks are inconsistent with two-level pain. Their responses to blocks may have been false positive.

### *Part 4: Efficiency*

Under the operation of this algorithm, discogenic pain is excluded or confirmed within one step. Sacroiliac joint pain is excluded within one block, or confirmed within two blocks.

In patients in whom sacroiliac joint pain is not suspected, zygapophysial joint pain is excluded within one step - a screening block, or diagnosed within four steps: one screening block that is positive; one or two blocks at single levels to pinpoint the responsible joint; and one confirmatory block.

In patients in whom sacroiliac joint has been entertained but excluded, zygapophysial joint pain is excluded within two steps: the negative sacroiliac block, and the negative screening block for zygapophysial joint pain. Zygapophysial joint pain is established within a total of five steps: one to exclude sacroiliac joint pain, one screening block to implicate zygapophysial joint pain, one or two blocks to pinpoint the responsible joint; and one to confirm the response.

Given the pre-test probabilities that

- ♦ internal disc disruption accounts for 40% of cases of chronic low back pain;
- ♦ sacroiliac joint pain accounts for up to 20% of cases; and
- ♦ zygapophysial joint pain accounts for 10%;

most patients under the algorithm would undergo investigations of their discs, with 40% proving positive and

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requiring no other investigations. Of the 60% remaining, not all will require sacroiliac joint blocks, but perhaps half will prove positive, and will not require zygapophysial joint blocks. Zygapophysial joint blocks will therefore be indicated in perhaps only 30% of the original population. Perhaps half of these will prove negative on screening blocks. Only the remaining half should be subjected to multiple tests of the zygapophysial joints.

Accordingly,

- ♦ in about 30% of cases sacroiliac joint pain will be diagnosed within one block plus a confirmatory block;
- ♦ in about 15% of cases investigations will exclude sacroiliac joint pain and zygapophysial joint pain within two blocks;
- ♦ only 15% of cases may require up to four or five blocks to pinpoint a painful zygapophysial joint.

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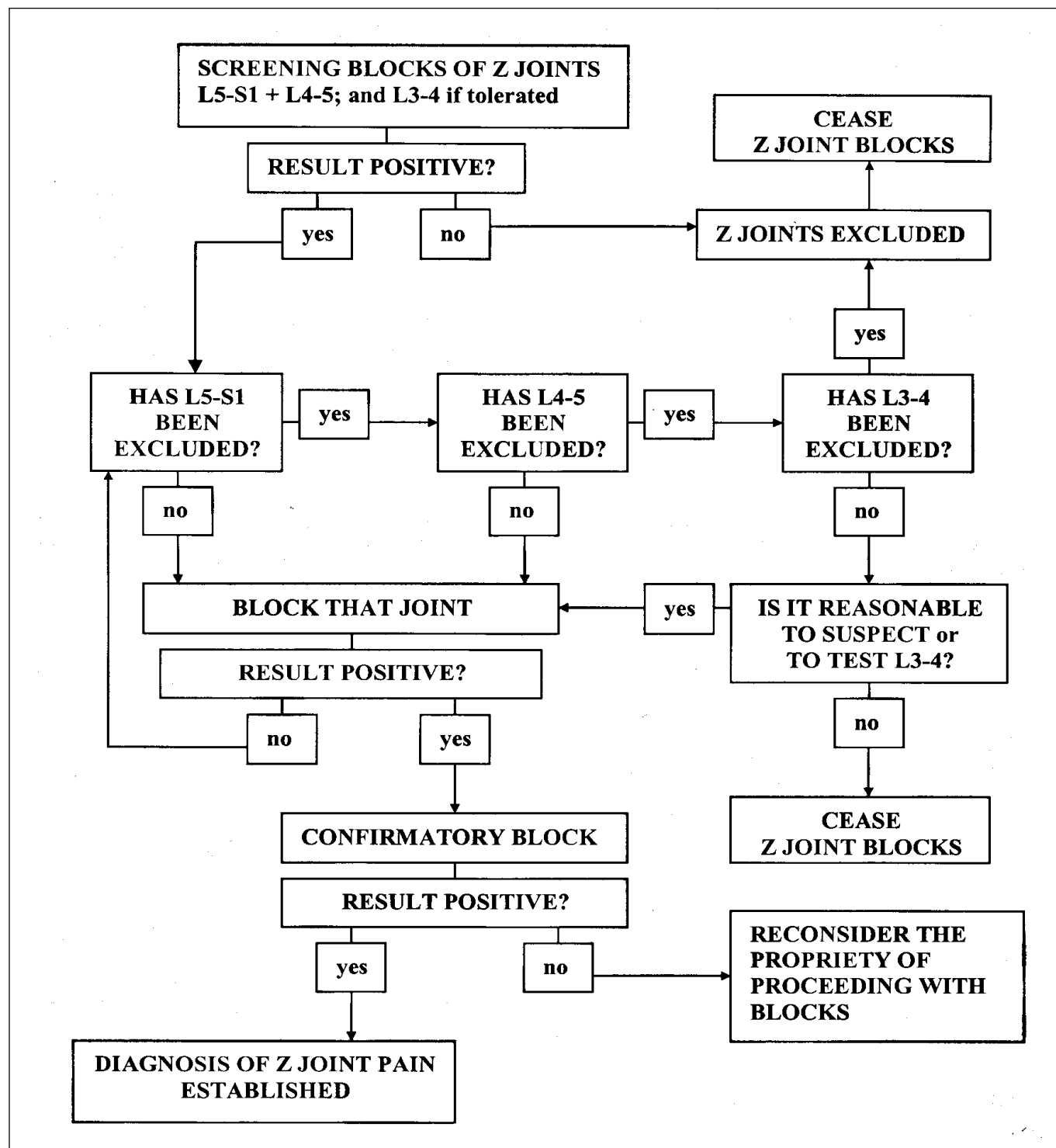
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## Lumbar Medial Branch Blocks



Algorithm Part 3: Zygapophysial Joint Blocks



# Australasian Faculty of Musculoskeletal Medicine Practice Standards and Protocols: Cervical Medial Branch Blocks

## Definition

**C**ervical medial branch blocks are a diagnostic procedure designed to test whether a patient's pain is mediated by one or more of the medial branches of the cervical dorsal rami. They involve anaesthetising the target nerve with a tiny volume of local anaesthetic in an effort to relieve pain.

By convention, and on the basis of theoretical argument,<sup>1</sup> but not on the basis of explicit, objective evidence, cervical medial branch blocks are used to test whether a patient's pain stems from a given cervical zygapophysial joint. For that purpose, the nerve or nerves that innervate the joint are anaesthetised.

This convention is based on the argument that, of all the structures innervated by the medial branches of the cervical dorsal rami, the zygapophysial joints are the only ones that might harbor a discrete, focal source of chronic pain.<sup>1</sup> No pathology capable of producing chronic pain is known to affect the segmentally specific muscles innervated by the dorsal rami. Because the ensuing term is shorter and more obvious in meaning, cervical medial branch blocks can be, and have been referred to as (one of the means of achieving) zygapophysial joint blocks.

## Historical Background

The conduct of cervical medial branch blocks arose for the investigation of neck pain as an analogue of use of lumbar medial branch blocks in the pursuit of back pain. The investigation of cervical zygapophysial joints as a possible source of neck pain was originally performed using intra-articular blocks of these joints. However, intra-articular blocks were progressively supplanted by medial branch blocks because:

- ♦ medial branch blocks are relatively easier to perform and therefore more expedient inasmuch as they

involve a single pass of a needle, with little adjustment, whereas with intra-articular blocks, difficulties may be encountered with entering a narrow joint-space. Moreover, medial branch blocks can always be performed, whereas osteophytes may preclude entry into a joint.

- ♦ medial branch blocks are safer inasmuch as bone prevents the over-penetration of the needle into the spinal canal, whereas it is possible for a needle to pass through a target joint into the spinal canal and spinal cord.
- ♦ medial branch blocks are more easily subjected to controls, in that the target nerves can be anaesthetised with different agents whose duration of effect on peripheral nerves is known, whereas the differential effect of different agents inside joints is not known.
- ♦ intra-articular blocks, if positive, lacked a valid subsequent treatment.
- ♦ intra-articular blocks, therefore, lacked therapeutic utility and predictive validity.
- ♦ medial branch blocks, if positive, could be followed by radiofrequency neurotomy.
- ♦ medial branch blocks, therefore, had therapeutic utility and predictive validity.

Nerve blocks for the investigation of neck pain were first suggested in 1980 by Sluijter and Koetsveld-Baart<sup>2</sup> who advocated blocking the cervical dorsal rami near their origin. Others in Europe adopted this procedure.<sup>3,4</sup>

Based on anatomical studies of the branches of the cervical dorsal rami and the innervation of the cervical zygapophysial joints, Bogduk advocated a more selective approach by targeting the medial branches of the dorsal rami, rather than the dorsal rami themselves.<sup>5</sup> The medial branches could be targeted easily and safely where they crossed the articular pil-

lars, whereas the dorsal rami had to be targeted near the intervertebral foramen and spinal nerve.

The first report of the diagnostic utility of cervical medial branch blocks was in the context of headache. In 1985, Bogduk and Marsland<sup>6</sup> reported complete relief of headache in eight out of 12 patients following anaesthetisation of the medial branch of the C3 dorsal ramus, the third occipital nerve. An earlier report, but published later in 1986, described relief in seven out of 10 of the same patients.<sup>7</sup> The first report of medial branch blocks at all cervical levels appeared in 1988. Bogduk and Marsland<sup>8</sup> reported relief of neck pain and headache, or neck pain and shoulder pain, in 17 out of 24 patients, following diagnostic blocks of the C3 or lower medial branches. They also published maps of the distribution of the pain relieved. These maps suggested a consistent segmental pattern.

Although never published, doubts and reservations were expressed concerning the implications of these studies. Prevailing wisdom maintained that the cervical zygapophysial joints could not be a source of pain, and that studies in patients with neck pain were unreliable. This prompted studies in normal volunteers to determine whether these joints could be a source of pain, and epidemiologic studies to determine whether zygapophysial joint pain was no more than a rare, idiosyncratic phenomenon.

Dwyer et al<sup>9</sup> stimulated the cervical zygapophysial joints in normal volunteers by distending the joints with injections of contrast medium. They found that the referred pain patterns from individual joints followed a distinctive segmental pattern. In a companion paper, Aprill et al<sup>10</sup> found that the pain patterns could be used to predict the segmental location of painful joints. Later, Fukui et al<sup>11</sup> used intra-articular injections and electrical stimulation of medial branches to confirm the segmental patterns.

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Bogduk and Aprill<sup>12</sup> performed various investigations in 318 consecutive patients with neck pain, and found the prevalence of cervical zygapophysial joint pain to be at least 25%. Because not all patients underwent blocks of these joints, the possibility existed that the prevalence of zygapophysial joint pain was even higher.

In 1991, Barnsley, later joined by Lord, commenced a systematic series of investigations of the validity and utility of medial branch blocks. They showed that cervical medial branch blocks had face validity, i.e., they were target specific.<sup>1</sup> Material injected onto the target nerves consistently bathed the location of the nerve, and did not spread in a manner so as to affect any other structure that might be an alternative source of pain or to any other nerve that might be mediating the patient's pain. Specifically their studies refuted the criticism that medial branch blocks anaesthetised non-specific muscle spasm, or that they anaesthetised the spinal nerves or their roots.

Next they addressed the construct validity of cervical medial branch blocks. All previous studies had used single diagnostic blocks, which did not control for false positive responses, i.e., placebo responses. Construct validity requires that diagnostic blocks correctly discriminate true responses from false responses, at least to an acceptable level of statistical certainty. To this end, Barnsley et al<sup>13</sup> tested a paradigm of comparative local anaesthetic blocks that had been advocated in the pain medicine literature, but promoted on the basis of theory alone. The paradigm maintained that a placebo response could be identified or excluded by repeating the same diagnostic block with local anaesthetic agents with different durations of action. Under double-blind conditions, Barnsley et al<sup>13</sup> performed cervical medial branch blocks in patients with neck pain, using lignocaine and bupivacaine in a random manner. They

identified and defined four patterns of response:

*Concordant:* in which patients obtained long-lasting relief following bupivacaine but short-lasting relief following lignocaine, with relief in both instances lasting not longer than the expected duration of action of the agent used.

*Prolonged concordant:* in which patients obtained longer-lasting relief following bupivacaine than that following lignocaine, but the duration of relief with either or both agents exceeded the expected duration of action of the agent used.

*Discordant:* in which relief following lignocaine was longer than that following bupivacaine, but relief in either instance was within the expected duration of action of the agent used.

*Discordant prolonged:* in which relief following lignocaine was longer than that following bupivacaine, but relief following either agent was longer than the expected duration of action of the agent used.

*Discrepant:* in which patients failed to obtain relief when the same nerves were blocked on a second occasion.

In order to sustain a sound epidemiologic argument Barnsley et al<sup>13</sup> considered only concordant and concordant prolonged to constitute a true-positive response. Even with this restriction, they found a high prevalence of positive responses, which was extremely unlikely to have occurred by chance alone, i.e., by the patients having guessed which agent they received on each occasion. Not only did this validate the paradigm of comparative local anaesthetic blocks, it suggested that the prevalence of cervical zygapophysial joint pain might be high.

In a contemporary paper, Barnsley et al<sup>14</sup> demonstrated that single diagnostic blocks were not valid. A large proportion of patients who responded to an initial block failed to respond to a subsequent block. A diagnosis based

on a single block, therefore, could not be relied upon to secure a correct diagnosis. This result foreshadowed the critical need for controlled blocks in every patient who underwent diagnostic blocks.

A later paper<sup>15</sup> tested the validity of comparative local anaesthetic blocks by comparing diagnoses made on the basis of comparative blocks with those based on placebo-controlled blocks. It established that the criteria for concordant and prolonged concordant responses, when combined, had a sensitivity of only 54% but a specificity of 88%. This meant that a diagnosis based on a concordant response to comparative blocks was very unlikely to be false, but that not all patients with zygapophysial joint pain would be correctly detected if these criteria were applied. Many patients who were not placebo responders had discordant responses. Consequently, for research purposes, the criteria for concordant responses to comparative blocks were robust and would not overestimate the prevalence of cervical zygapophysial joint pain. For clinical purposes, the criteria could be relaxed, if desired, to include patients with discordant responses provided that they obtained complete relief of their pain whenever the medial branches were anaesthetised, regardless of the agent used and regardless of the duration of relief obtained. In the event of the latter, the sensitivity of the criteria increases but the specificity drops to 65%. In other words, a diagnosis based on discordant responses detects more patients as positive, and is more often correct than it is not, but does have a substantial chance of being wrong.

Three epidemiologic studies followed. The first two used comparative blocks under double-blind conditions to determine the prevalence of cervical zygapophysial joint pain in consecutive patients presenting with chronic neck pain after whiplash. The first focussed on patients with headache,<sup>16</sup>

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and found that, overall, the prevalence of headache stemming from the C2-3 zygapophysial joint was 27%, but that in patients in whom headache was the dominant symptom, this prevalence was 53%. The second study<sup>17</sup> reported 50 consecutive patients with neck pain after whiplash. It included some patients from the first study but focussed on neck pain as well as headache. It found a prevalence of zygapophysial joint pain of 54%. The joints most commonly involved were those at C2-3 and at C5-6. The third study,<sup>18</sup> used placebo-controlled diagnostic blocks and focused exclusively on neck pain. It reported the results of blocks at levels below C3. In 68 patients, not previously reported, it found a prevalence of cervical zygapophysial joint pain of at least 49%, with the possibility that it could be as high as 60%.

Collectively these studies showed not only that cervical zygapophysial joint pain was common, but that it was the single most common basis for chronic neck pain after whiplash. These results implied that physicians who did not employ zygapophysial joint blocks in the investigation of their patients risked missing a valid diagnosis in over 50% of cases.

A later study, found a much higher prevalence in a specific subgroup of patients.<sup>19</sup> Amongst drivers in motor vehicle accidents injured at high speeds, the prevalence of cervical zygapophysial joint pain was found to be as high as 88%.

The next phase of research addressed the therapeutic utility and predictive validity of cervical medial branch blocks, i.e., if blocks were positive, did they lead to a useful treatment, and did they accurately predict response to that treatment.

A randomised, double-blind, placebo controlled trial<sup>20</sup> showed that injecting painful cervical zygapophysial joints with corticosteroids did not offer any greater chance of relief than simply

anaesthetising the joint. Moreover, it showed that very few patients obtained gratifying relief for longer than a few days, regardless of the agent used. Intra-articular steroids did not provide a therapeutic answer to zygapophysial joint pain.

In a pilot study, Lord et al<sup>21</sup> assessed the efficacy of percutaneous radiofrequency neurotomy, in which the medial branches to a painful zygapophysial joint are coagulated to stop the pain. They found that results were poor when the third occipital nerve was targeted, and they recommended a moratorium on third occipital radiofrequency neurotomy until better techniques were developed. However, for neurotomy at lower cervical levels, they found encouraging results. This prompted a randomised, double-blind, placebo controlled study.<sup>22</sup> This established beyond doubt that cervical medial branch neurotomy was not a placebo, and suggested that 70% of patients could obtain complete relief of their pain if treated by this procedure. Furthermore, the study established the therapeutic utility and predictive validity of cervical medial branch blocks. Patients who obtained complete relief from controlled diagnostic blocks of the cervical medial branches stood a good chance of obtaining complete relief of their pain if the same nerves were coagulated.

A later study<sup>23</sup> indicated that following an initial neurotomy, long-term relief, in excess of 200 days, could be achieved in the majority of patients, and that relief could be reinstated by repeating the treatment. Moreover, effectiveness was independent of litigation, the nature of the electrode used, and whether patients had been diagnosed using comparative blocks or placebo-controlled blocks.

Two review papers summarise the historical background and technical aspects of cervical medial branch blocks<sup>24</sup> and cervical radiofrequency neurotomy<sup>25</sup> until about 1998.

The development of the theory and practice of cervical medial branch blocks has attracted recognition beyond just publication in peer-reviewed journals.

- ♦ The early work on third occipital headache<sup>6</sup> was awarded the Prize for Best Poster at the 2nd International Headache Congress in 1985.
- ♦ The work on pain patterns in normal volunteers<sup>9</sup> was awarded the Prize for Outstanding Cervical Spine Research by the Cervical Spine Research Society in 1988.
- ♦ The controlled study of radiofrequency neurotomy<sup>22</sup> was awarded the Research Prize of the Spine Society of Australia in 1996.
- ♦ For her collection of studies, Dr Lord received the Research Prize of the International Association for the Study of Pain at the World Pain Congress in 1999.

This international recognition by the scientific community stands in contrast to reservations still maintained, but never publicly published, by insurance companies and their advisers. Those reservations do not involve scientific criticism but constitute social rhetoric.

Cervical medial branch blocks are condemned privately to patients and their treating doctors as procedures that have not been embraced by "mainstream" medicine, or that the research has come from only one unit (in Australia), and has not been replicated by others.

Both accusations are false. Independent of any of the original investigators, in the latest textbook of pain medicine (amongst others),

- ♦ the use of comparative local anaesthetic blocks is not only endorsed but emphasised;<sup>26</sup>
- ♦ cervical medial branch blocks are advocated and illustrated;<sup>26</sup>
- ♦ the prevalence of cervical zygapophysial joint pain is recognised;<sup>27</sup>

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- ♦ as is its treatment with radiofrequency neurotomy.<sup>27</sup>

One study corroborating use of cervical radiofrequency neurotomy has been published in abstract form,<sup>28</sup> and a prevalence study of cervical zygapophysial joint pain will soon appear in the *Medical Journal of Australia*, and a report of a trial of cervical medial branch neurotomy in the USA will appear in *Spine*.

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### Principles

The explicit purpose of cervical medial branch blocks is to test whether the patient's pain is relieved by anaesthetising the nerves targeted.

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They are not a test of the patient's veracity. They test the hypothesis raised by the treating doctor that perhaps the pain is mediated by the nerves specified.

**If pain is not relieved**, the target nerves cannot be regarded as mediating the patient's pain. A new hypothesis about the source of pain is required. The pain may perhaps be mediated by other medial branches, or it may arise from a source not innervated by the cervical medial branches.

**If pain is relieved**, the response constitutes *prima facie* evidence that the targeted nerves are mediating the patient's pain; but steps need to be taken to ensure that the observed response is not false positive.

It is possible that a patient may have several sources of pain. For example:

- ♦ they may have pain bilaterally at a given segmental level, in which case anaesthetising the left nerves should relieve the left side of their pain but not the right side (and vice versa);
- ♦ they may have pain from more than one segmental level on the one side, in which case anaesthetising the upper one or two of a series of nerves may relieve only the upper half of their pain.

In either instance, complete relief of all pain cannot be expected. Indeed, complete relief of all pain is contrary to what should be expected. Rather, a positive response can be entertained if there is complete relief of pain in a distinct topographical region that constitutes part of the patient's total complaint, but which corresponds to the area from which pain could be expected to be mediated by the nerves anaesthetised. Although this may constitute only partial relief of all of the patient's pain, it is more accurately and more informatively viewed as **complete relief of pain in the region targeted**. Remaining areas of pain may be targeted separately, or blocks might be extended (within reason) to

include additional nerves that subtend those remaining areas.

Cervical medial branch blocks have diagnostic utility. If positive, they identify the source of pain. Establishing a positive diagnosis protects the patient from the futile pursuit of other and competing diagnosis, and from undergoing presumptive treatment or treatment that is not appropriate for pain mediated by the cervical medial branches.

Cervical medial branch blocks have therapeutic utility, in that a positive response predicts a good chance of obtaining complete relief of pain from percutaneous radiofrequency neurotomy.

### Control Blocks

Control blocks are essential to exclude false positive responses, and to maximize the confidence of a securing a true positive response. The most rigorous form of control is the use of a placebo injection of normal saline under double blind conditions, but logistic and ethical considerations militate against the use of normal saline in conventional practice.

The injection of saline ethically would require informed consent. Impromptu, single-blind injections of normal saline are unethical.

If saline is used it would have to be in the context of three blocks of the same joint. The first block would have to be with an active agent in order to establish, *prima facie*, that the joint is symptomatic. (There is no point in performing routinely a series of three controlled blocks in a patient in whom there is no objective indication that medial branches are at all mediating the patient's pain.) The second block could not be the normal saline control because a mischievous patient would know that "the second injection is always the dummy" and could respond appropriately. In order to maintain the controlling effect of chance and blinding, the second block would have to be

either normal saline or an active agent, and the third block would need to be the reciprocal agent.

Comparative local anaesthetic blocks constitute a more practical form of control that can be readily incorporated into routine and conventional practice.

A true positive response to comparative local anaesthetic blocks is one in which the patient reports complete relief of pain for a shorter duration when a short-acting agent is used, and for a longer duration when a long-acting agent is used. Such a response is referred to as "concordant" in that it is concordant with the expected action of the agents used.<sup>1</sup> A concordant response confirms that the joint is the source of pain with a confidence of 85%.<sup>2</sup>

If a patient obtains complete relief of pain but does not report an appropriate differential response in terms of duration of relief, the joint is not excluded as the source of pain. Such paradoxical responses may still be consistent with the hypothesis being tested. Indeed, some 65% of patients who report such responses withstand challenge with placebo.<sup>2</sup> This paradox seems to arise because, in some patients with chronic pain, lignocaine has a long duration of action, ostensibly due to its action on "open" sodium channels.

What should be the criteria for a positive response to comparative local anaesthetic blocks is a matter of judgement for the physician. However, the following principles apply.

If the consequences of a false positive diagnosis are relatively innocuous, less stringent criteria can be used.<sup>2</sup> Complete relief of pain on each of two occasions, regardless of the duration of relief, will ensure that all patients with zygapophysial joint pain will be detected, i.e., "ruled in", but the cohort so identified will include false-positive cases.<sup>2</sup> This may not matter if irreversible therapies are not being considered. But physicians should be aware

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that the inclusion of false positive cases will undermine the success rate of any treatment.

If the consequences of a false positive diagnosis are serious, more stringent criteria should be used. In such circumstances, the response must be reliably true positive. The criteria for a concordant response are appropriate for this purpose.<sup>2</sup>

If the consequences of a false positive diagnosis are dire, it would be imperative to perform triple blocks with saline controls in order to be certain that the response is beyond doubt a true-positive response.<sup>2</sup>

What constitutes "innocuous", "serious", and "dire" is a matter of consideration and decision between the physician, the patient, and any other parties involved such as an ethics committee or institutional review board.

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### Indications

The fundamental indication for cervical medial branch blocks is the desire to know whether the patient's pain is mediated by the medial branches of the cervical dorsal rami. This principle underscores that neck pain, per se, is not an indication for diagnostic blocks. Not all patients need diagnostic blocks, so not all patients should undergo blocks.

Legitimate reservations can be raised about the need to perform blocks if the patient is going to be treated conservatively. In that event, the response to blocks does not affect management,

and performing blocks can be viewed as superfluous. The only proposition that might be entertained is that medial branch blocks have diagnostic utility and, therefore, establishing a positive diagnosis might curtail the futile pursuit of other diagnoses. That proposition, however, needs to be weighed carefully against the likelihood of obtaining negative responses. Proving that patients do not have a condition that they are unlikely to have is a waste of the skills and resources of the investigator. Those skills could be better channelled into procedures and actions that have a greater chance of improving patient outcome. For this reason, attention should be paid to careful patient selection (see below).

The only validated treatment for pain mediated by the cervical medial branches is percutaneous radiofrequency neurotomy. If this treatment is not available, the conduct of medial branch blocks can be justified only on the grounds of their diagnostic utility. However, if radiofrequency neurotomy is available, medial branch blocks are an essential prerequisite before entertaining radiofrequency neurotomy.

Medial branch blocks are not indicated for acute neck pain. There is a high chance of acute neck pain recovering, despite conservative treatment, and radiofrequency neurotomy is not indicated for acute pain. Therefore, it is not necessary to perform diagnostic blocks in patients with acute neck pain. In principle, therefore, medial branch blocks are indicated only for patients with chronic pain. However, it could be conceded that there is merit in investigating patients with subacute pain, whose pain is not improving or responding to conservative management. Although it has not been demonstrated, the proposition is attractive that pinpointing the source of pain in these patients and promptly treating it could avert the onset of chronic pain behaviour.

### Patient Selection

No studies have been published that report empirical data on the validity of clinical criteria that optimize the diagnostic yield of cervical medial branch blocks, or that might be used to exclude patients from undergoing blocks. These criteria can be based only on theoretical considerations, and perhaps on advice from those who have contributed to the development of these blocks, based on their experience to date.

A fundamental criterion is that serious possible causes of neck pain, such as infection, tumours, vascular disease, and metabolic disease, have been excluded by careful and thorough history and examination, laboratory tests, and medical imaging. The work-up should provide a diagnosis of cervical spinal pain of unknown origin, uncomplicated by associated features other than perhaps restricted motion.

Cervical medial branch blocks are optimally performed in patients with discrete areas of neck pain that correspond to one or other, or one or more, of the areas known from studies in normal volunteers to be associated pain produced from a given joint or mediated by a given nerve or nerves. It should be recognised that pain in a certain area does not implicate the zygapophysial joint as the source, nor does it implicate the corresponding medial branches. Referred pain maps only indicate the likely segmental innervation of the source of pain. That source might not be a zygapophysial joint, and could lie outside the territory of the cervical medial branches. Indeed, it has been shown that discogenic pain refers in patterns almost identical to those of the zygapophysial joints of the same segment.<sup>1</sup>

A critical point is that the recognition of a pain map does not rely on the total extent of the pain, which may vary from day to day with the severity of the pain. The critical feature for identifying the area of pain is to determine the centroid of the pain, i.e., where the pain starts,

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where it occurs most consistently and with the greatest intensity, and from where it seems to spread to more peripheral areas. Metaphorically, the physician should identify the epicenter of the pain, and use that (not the total extent of the pain) to identify its segmental location.

If the patient's pain pattern is unilateral and corresponds to the pattern of a given motion segment, the two medial branches of that segment should be blocked.

Some patients may have neck pain from more than one source. Their overall distribution of pain will be a composite of more than one segmental pattern. The most common combinations are bilateral pain at the same segmental level, pain from consecutive motion segments (most often C5-6 and C6-7), or pain from displaced sites (most often upper neck pain and headache from C2-3 and neck-shoulder pain from C5-6).<sup>2</sup> For these patients certain precautions should be taken to avoid inaccurate, inefficient, and excessive investigation. These steps are outlined later under Interpretation and under Algorithm.

As yet unpublished data indicate that patients whose pain does not correspond to a single motion segment, or whose distribution of pain defies reduction sensibly into a composite of discrete segmental patterns, are very unlikely to respond to medial branch blocks. Such patients have very widespread pain and extensive tenderness, and cannot identify centroids for their pain. In such patients, medial branch blocks cannot be proscribed until valid data are published, but lest it give the use of medial branch blocks a bad reputation, it is probably unwise to squander medial branch blocks in these patients. The diagnosis is unlikely to be zygapophysial joint pain, or the sources of pain will be so extensive as to defy responsible treatment with radiofrequency neurotomy.

## References

1. Grubb SA, Kelly CK. Cervical discography: clinical implications from 12 years of experience. *Spine* 2000; 25: 1382-89.
2. Lord S, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash: a placebo-controlled prevalence study. *Spine* 1996; 21: 1737-45.

## Contraindications

### Absolute

The absolute indications for cervical medial branch blocks are conditions in which the conduct of a needle procedure under x-ray control might jeopardize the patient's health. These include, but are not necessarily limited to the following:

- ♦ bacterial infection, systemic or localized in the region of the blocks to be performed;
- ♦ bleeding diathesis, due to haematological disease or anticoagulants;
- ♦ possible pregnancy.

### Relative

Relative contraindications are conditions that do not preclude the conduct of cervical medial branch blocks but which require special consideration because of the risks they pose. In the face of these conditions the investigator may elect not to perform medial branch blocks, or if they do undertake blocks special precautions are required. These conditions include, but are not necessarily limited to the following:

- ♦ allergy to contrast media, which may require cover with corticosteroid and H1 and H2 antagonists;
- ♦ allergy to local anaesthetics, which may require identification and use of a class of anaesthetic to which the patient is not allergic;
- ♦ concurrent treatment with non-steroidal anti-inflammatory medications, including aspirin, that may compromise coagulation, in which

case, medication may need to be suspended for an appropriate period prior to the conduct of blocks.

Neurological signs are a relative contraindication for cervical medial branch blocks, inasmuch as the neurological disorder should be managed first. If a patient also has neck pain, it could be investigated with medial branch blocks once the neurological disorder has been managed or is being managed.

In that context, radicular pain is not a contraindication for medial branch blocks, but the investigator should not be under any misapprehension that medial branch blocks will relieve radicular pain. Medial branch blocks are indicated if the diagnostic hypothesis is that the patient's neck pain and any somatic referred pain might be mediated by cervical medial branches, but is a concomitant but separate problem to their radicular pain.

## Facilities Required

### Radiological Equipment

Fluoroscopy is mandatory for the conduct of cervical medial branch blocks. The preferred equipment is a C-arm fluoroscope that allows the x-ray beam to be directed at any angle. Furthermore, in order to document the accurate placement of needles and the spread of contrast medium a device must be available to obtain either hard-copy films or an image on specialised paper.

### Resuscitation Equipment

Although misadventure attributable to the agents injected is extremely unlikely, given the small doses injected for cervical medial branch blocks, it is probably wise to perform the procedure in a facility equipped with proper resuscitation facilities. At the very least, the operator is thereby equipped to deal with possible allergic reactions to

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local anaesthetic agents.

### *Needles, Gowns, Drapes, etc.*

A 90 mm, 25 gauge needle is optimal, for it is minimally painful when passed through the skin and muscles overlying the target joint.

The needle may or may not have a Luer lock, but such a lock is preferable.

A standard preparation tray may be used, which comes with cotton balls and gauze, but more elaborate trays can be custom made to come with needles and local anaesthetic.

Solutions for skin preparation may be an iodine-based solution (e.g., Povidone-iodine), chlorhexidine, or and alcohol-based antiseptic (e.g., chlorhexidine 0.5% in 70% alcohol).

Sterile gloves are used.

Local anaesthetic agents may be injected directly from a syringe attached to the spinal needle, or minimal volume extension tubing may be interposed between needle and syringe, according to operator preference.

Given that only a small volume of agent is injected, a 2 ml syringe is all that is required.

### *Medications*

Intravenous solutions, sedation or antibiotics are not required.

### *Agents*

Any conventional local anaesthetic can be used for medial branch blocks. Agents most commonly used are bupivacaine 0.5% and lignocaine 2%. Other concentrations that can be used are bupivacaine 0.75% and 0.25%, and lignocaine 1% and 4%. Because of the small volumes used, high concentrations should generally be used in order to obtain the most effective anaesthetic effect. For medial branch blocks, no more than 0.3 ml is required to block the nerve adequately.

## Preliminary Procedures

### *History and Physical Examination*

A history and physical examination are required to exclude pain likely not to be of zygapophysial joint origin and to identify or exclude contraindications to blocks. Pain maps are used to select which should be the target nerves in the first instance (Fig. 1). Otherwise, a history and physical examination are required to record baseline data concerning the location and extent of pain, including a visual analogue score, and the movements and activities of daily living that are customarily prevented by the pain.

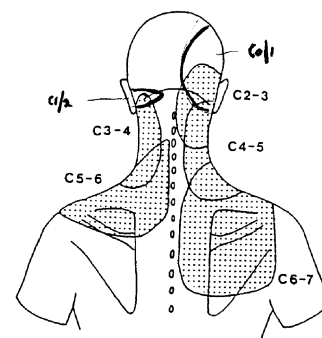
On obtaining this baseline history, the patient should be briefed as to how their response to blocks will be measured. They should be instructed in the use of any pain diaries or visual analogue scales that might be used.

### *Informed Consent*

Informed consent must be obtained. Although medial branch blocks should be safe procedures, like any invasive procedure they carry the nominal risk of infection, bleeding and allergic reaction. After upper cervical blocks the patient is likely to suffer temporarily a sense of ataxia. These risks should be explained to the patient who should be advised of the precautions to be taken.

The patient should be advised that the procedure is a diagnostic one, and should not be confused with a therapeutic procedure. They should be advised that they may or may not obtain relief, and that in particular they should be prepared for no relief ensuing. They should be advised that expecting any particular result confounds the purpose of the test, and that they should report the result honestly.

Other than to expect either relief or no relief, the patient should not be informed of the duration of relief to expect. They should be prepared only to the effect that if relief ensues they will need to monitor and record its duration and extent.



**Figure 1. Map of referred pain from the cervical zygapophysial joints.**

### *Premedication*

No premedication is required.

## Technique

### *Preparation*

Neither physiological monitoring nor intravenous access is required.

### *Positioning*

Although a posterior approach is possible, the most convenient and technically least demanding approach for cervical medial branch blocks is a lateral approach. For this the patient lies on their side with the painful side uppermost. A device to hug, such as a pillow or a teddy-bear, serves to accommodate their arms, if required.

### *Sterility*

The skin of the lateral neck must be exposed, and the patient's hair should be prevented from falling into the field of the procedure. Patients with long hair could wear a surgical cap.

Once the patient is in position the proposed entry point and surrounding area must be adequately prepared as for an aseptic procedure, using one of the solutions listed above. The prepared area must be allowed to dry in order to ensure sterility. A fenestrated drape made of cloth, paper, or plastic should be applied to cover the non-



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sterile areas surrounding the prepared area.

*Target Identification*

As near as perfect, a true lateral view of the cervical spine must be obtained. In such a view, the silhouettes of the articular pillars of both sides at a given segment are superimposed. Tilting the x-ray beam slightly, around the long axis of the patient, should split the images of the superimposed silhouettes, which confirms superimposition. This confirmatory step is essential, for failure to obtain a confirmed, true lateral view risks aiming a needle towards the contralateral side of the neck.

The fluoroscope should be aligned so that the target point is at the center of the x-ray beam, i.e., so that it appears on center-screen. This avoids any errors due to parallax.

*1. For medial branch blocks C3-C6*

The target point is the centroid of the articular pillar with the same segmental number as the target nerve. This centroid is found at the intersection of the two diagonals of the diamond-shaped pillar.

*2. For medial branch blocks C7*

The target point lies high on the apex of the superior articular process of C7. This is because the base of the C7 transverse process occupies most of the lateral aspect of the C7 articular pillar and thrusts the medial branch relatively higher than typical cervical medial branches. The presence of this transverse process should be realised, for it is not readily apparent on lateral views, but it may confound accurate needle placement. If too low a target point is selected, the needle may strike bone and appear to rest on the superior articular process but it will instead be resting on the transverse process which points, end-on, towards the operator. If the silhouette of its base can be perceived superimposed on the

superior articular process, the target point for a C7 medial branch block will lie substantially above this silhouette.

*3. For third occipital nerve blocks*

Three target points are used in order to ensure adequate infiltration of the third occipital nerve, which is thicker than the medial branches of typical cervical dorsal rami, and which is embedded in the pericapsular fascia of the C2-3 joint, and which has a variable location in relation to this joint.

The target points lie on a vertical line that bisects the C2-3 joint. The high target point lies opposite the level of the apex of the C3 superior articular processes. The low target point lies opposite the bottom of the C2-3 intervertebral foramen. The middle point lies midway between these foregoing points, usually on the subchondral plate of the superior articular process of C3.

*Needle Placement*

A puncture point on the skin is selected overlying the target point, checking that a subcutaneous vein is not about to be penetrated. If a vein lies in the intended path of the needle, the puncture point is relocated slightly so as to avoid venipuncture. Also, in order to minimise discomfort during the passage of the needle, particular at upper cervical levels, it is preferable to avoid piercing the sternocleidomastoid muscle, if possible. If the puncture point initially selected overlies the sternocleidomastoid, the patient can be asked to rotate their head slightly, into the pillow, in an effort to draw the muscle forwards, so that it no longer lies under the puncture point.

Prior to penetrating the skin, the tip of the needle should be placed on the intended puncture point, and the shaft aligned parallel to the x-ray beam. This allows the needle to be directed straight towards the target point without having to judge the magnitude of angles, which is required if an oblique insertion is used. The needle is then inserted

quickly through the skin, and carefully through the neck muscles but only to a depth sufficient to obtain purchase for the needle, to stop it swaying if released. Its position and orientation should then be checked to ensure that it overlies and is pointing towards the target point within a tolerable error zone. If this is not the case, the needle should be reinserted at a point at which it does satisfy these criteria.

*For medial branch blocks at C3-C6.*

The tolerable error zone for insertion and passage of the needle should be no greater than the middle half of the area of the articular pillar across which the target nerve runs. When the needle is inserted, its tip should not stray outside this zone. This standard ensures that needles are not directed too obliquely so as to require multiple insertions, withdrawals, and over-corrections. The needle is progressively inserted towards the target point, using periodic screening to check its course, and undertaking any corrections required to its course. As the needle passes progressively more deeply, its tip should appear to overlie the target point progressively more closely. In effect, coarse corrections to the path of the needle should be undertaken only while the needle is relatively superficial, and only fine corrections should be required as its tip nears the depth of the target point. Once the needle rests on the target point 0.3 ml of local anaesthetic can be injected in order to anaesthetise the target nerve. In order to anaesthetise a typical cervical zygapophysial joint both the medial branches that innervate the joint should be anaesthetised.

*For medial branch blocks at C7.* The tolerable zone for insertion and passage of the needle should be no greater than triangular silhouette of the superior articular process of C7. When the needle is inserted, its tip should not stray outside this zone. This standard

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ensures that the needle does not pass deeper than the depth of the superior articular process, into the C8 intervertebral foramen, or through the C6-7 zygapophysial joint. The needle is inserted carefully and slowly, with frequent screening to check progress, such that the tip never strays beyond the tolerable error zone. If at any time the needle tip strays beyond this zone, it should be withdrawn sufficiently to allow it to be reorientated so that tip lies within the error zone. Once the needle appears to have contacted the superior articular process, a postero-anterior view should be obtained to confirm that the needle tip lies right against the lateral margin of the superior articular process. If instead, it transpires that the tip has struck the upper surface of a thick C7 transverse process and lies short of the superior articular process, the needle should be readjusted under lateral views to a higher location on the superior articular process, and its position checked and confirmed once again on PA view.

Once the needle is in correct position, 0.3 ml of local anaesthetic can be injected in an attempt to infiltrate the target nerve. Once that is done, however, the needle should be withdrawn about 4 mm and a further aliquot of 0.3 should be injected. The step caters for the variation in which the C7 medial branch, instead of running across the surface of the articular process, is displaced away from bone by a bundle of the semispinalis capitis. If required, postero-anterior screening can be used to gauge the depth of withdrawal of the needle.

*For third occipital nerve blocks.* The tolerable error zone is a rectangular area bounded by the anterior edge of the superior articular process of C3, upper and lower lines perpendicular to this edge passing posteriorly from the apex of the superior articular process and from the bottom of the C2-3 intervertebral foramen, and a posterior

line approximately through the posterior edge of the inferior articular process of C2. When the needle is inserted, its tip should not stray outside this zone. This standard ensures that the needle does not pass into the C2-3 intervertebral foramen, or too far away from the target points.

The needle is inserted carefully towards the middle of the three target points, using periodic screening to check its course. If at any time, the needle strays out of the error zone, it should be withdrawn sufficiently to reorientate it so that it points towards the target point. The needle is progressively inserted until it rests on the low target point. Once the needle is in position and rests on bone, it should be withdrawn slightly, by a length equal approximately to the thickness of the capsule of the joint. This step ensures that the needle has not penetrated the joint capsule, and that an injection will not be intra-articular instead of perineural. Once the needle has been withdrawn, 0.3 ml of local anaesthetic can be injected.

The needle is then readjusted to either the upper or lower target point, at which 0.3 ml of local anaesthetic can be injected.

The needle is finally readjusted onto the remaining target point where 0.3 ml of local anaesthetic can be injected.

### Records

An image demonstrating the needle position must be obtained whenever a substance is injected. A plain radiograph may be obtained using conventional film or specialised paper. Such a record protects the operator in the event of alleged misadventure.

### Post-procedural Care

Upon removal of the needle, the skin is cleaned to remove the antiseptic and any blood. A small adhesive patch can be applied to the puncture sites, but is probably unnecessary.

If the patient complains of any untoward side effects following the procedure, appropriate action must be taken. Vaso-vagal episodes may occur. These are managed with pulse and blood pressure observations and rest in the supine position. Rarely is any further intervention indicated.

Patients who undergo upper cervical procedures, particularly third occipital nerve blocks, are very likely to report a sense of ataxia. This occurs because the blocks anaesthetise upper cervical proprioceptors which are critical for tonic neck reflexes. To compensate for this they should be instructed to engage and rely on visual cues. This is achieved by having the patient always focus on objects horizontal, regardless of whatever they do. Such objects include window frames, door frames or the horizon itself. They should be warned that if they look downwards or if they look sideways to a companion they will incur the sense of unsteadiness and so must avoid these actions. They can be reassured that if they follow these precautions they should not meet with misadventure. Furthermore, they are very likely to adjust to the strange sensation within 15-30 minutes. They should be reassured that the sensation will be only temporary.

Because of the giddiness, patients must be advised not to drive a motor vehicle, because sudden loss of contact with the horizon, such as upon sudden turning of the head, may result in temporary loss of control of the vehicle.

Otherwise the procedure is usually well tolerated, and the patient may be allowed to dress and await evaluation and discharge.

Discharge instructions include:

- ♦ to contact the doctor who performed the procedure if there is any unusual symptom or pain following the procedure. Fever and tenderness greater than that which might be ascribed to a needle track may be

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signs of infection. To this end, an instruction sheet with a name and telephone number is useful. This sheet should be separate to any other data sheets given to the patient.

- ♦ to monitor the extent and duration of any relief that ensues. To this end a pain diary is helpful. However, critical is the time when the pain starts to return and the time that it returns to its former, accustomed intensity.
- ♦ if relief occurs, the patient should carefully attempt the movements and activities of daily living that customarily are restricted by pain, in order to determine whether these movements and activities can be resumed while pain free. They should record those movements and activities that they have been able to resume.

### Evaluation

The singular reason for performing diagnostic blocks is **to obtain information**. That information depends on a reliable evaluation of the patient's response to blocks. Although performing the diagnostic block is an essential first step, the block itself does not make the diagnosis. Unless the patient's response is carefully evaluated and controlled for false-positive responses, performing the block is a waste.

There are several potential sources of error in the assessment of a response to a diagnostic block.

- ♦ Patients who expect and want a block to work may suffer a placebo response, and obtain or report relief for reasons other than the pharmacological effects of the block.
- ♦ A doctor who expects or wants the block to work may overtly or subconsciously coach the patient to report a positive effect even when one is not truly achieved.
- ♦ An assessor who wants the block to work may exercise observer bias,

and report as positive a block whose effect has not truly been positive, or report as completely effective a block that has been only partially effective.

- ♦ Blocks may be performed at a time when the patient's pain is minimal, or even absent. In that event it is difficult to argue that any supposed relief obtained was due to the effects of the block, and not simply a reflection of the patient's low level of pain at the time.
- ♦ If the response to a block is evaluated immediately and only upon completion of the block, a false impression may arise. Having rested during the performance of the block the patient may obtain relief of the pain. If asked if there is any relief upon completion of the block the patient will correctly respond that there has been relief, but when subsequently they resume activities of daily living it may become apparent that the block has, in fact, not produced a positive effect.
- ♦ If a patient is discharged following completion of a block and their response is assessed at some time later, be that by telephone interview or at a subsequent consultation, they may suffer recall bias. They may not remember accurately how much relief they obtained and for how long. Furthermore, their report is entirely subjective, no independent trained observer having corroborated objectively the validity of their response.
- ♦ Although patients in absentia might be asked to complete a graphic record of their pain levels, this process is confounded by the patient having access to what they previously recorded. Guidelines for the completion of serial visual analogue scales for pain maintain that patients should not see their previous entries.<sup>1</sup>
- ♦ An untutored patient may fail to recognise that a block has been

successful. This can occur when a patient has multiple sources of pain. Although a block may successfully anaesthetise one of their sources of pain it may not relieve other sources. Consequently, when asked, in absentia, if their pain was relieved the patient, having not obtained total relief of all of their pain, may report that it wasn't.

Such errors have potential ramifications with respect to both medicolegal proceedings and treatment. Blocks subject to error may lead to false conclusions about the veracity of a source of pain. Liability, therefore, may subsequently be misattributed. A false conclusion may lead to inappropriate therapy that is destined to fail. It is therefore, imperative that information based on diagnostic blocks be reliable and valid, i.e., free from error.

Certain errors can be reduced by performing diagnostic blocks under double-blind conditions. When the patient does not know which agent is being used, they cannot conform to an expected response. Simultaneously, the double-blind paradigm prevents the doctor, or an independent assessor, from coaching the patient as to what response to expect. Unless a diagnostic block is performed under double-blind conditions, the risks of response bias, observer bias, and reporting bias, remain eminent, regardless of how honest and objective a doctor claims, or insists, they are. The elimination of other sources of error require other measures, as outlined below.

### Towards An Optimal Protocol

At a Master Class conducted by the International Spinal Injection Society at the University of Newcastle in 1998, participants discussed the issues raised above. They agreed that the significance of diagnostic blocks for spinal pain lay in the information obtained, not in the execution of the block. They

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recognised the potential sources of error when blocks were not performed under controlled conditions.

The meeting resolved that the optimal means of reducing error and securing reliable diagnostic information was **real-time assessment**. Under this protocol, the response to a diagnostic block is evaluated immediately after the block and for some time afterwards at the clinic at which the block was performed, and by an independent observer using validated and objective instruments or tools.

Under the protocol, the doctor who is to perform the diagnostic block introduces the patient to an independent observer, typically a registered nurse. The doctor describes the pain that is being targeted by the forthcoming block, and if appropriate, highlights how this pain is distinguished from any other pain that the patient might concurrently have.

Both the doctor and the assessor should determine and agree that the patient's level of pain is sufficiently intense for any response to the intended diagnostic block to be credible and meaningful.

In this regard, a reasonable guideline is that the patient's present pain should be no less than 50% of their pain at its worst. Serious consideration should be given to the propriety of proceeding with blocks either in patients whose typical pain is less than 40 on a 100 mm scale, or in patients whose pain at the time when the block is to be undertaken is less than or equal to 20 on a 100 mm scale, for the natural diurnal variation in pain may be of this magnitude; and a decrease in pain by only 20 points may not be legitimately ascribable to the intervention.

Separately with the patient, the assessor records baseline measures pertaining to the patient's pain.

Separate from the assessor, the doctor performs the diagnostic block. Once the doctor is satisfied that the block has been adequately and safely

completed, and that the patient has no resulting side effects that require immediate medical attention, they return the patient to the RN for assessment and evaluation. The doctor takes no part in this evaluation, and is free to continue with other patients.

The assessor evaluates the patient's response to the block, administering the instruments that have been selected for this purpose. (See Schedule A, below.)

The assessment continues in principle for the duration of the patient's response to the block, or until the effects of the block have been reasonably established beyond doubt.

- ♦ If the patient's pain has not been relieved, the patient can be prepared for discharge once it has been clearly established that there has been no relief.
- ♦ If the patient reports relief, this should be monitored and corroborated by the assessor for at least two hours or until the effects of the local anaesthetic agent wear off, whichever is the sooner. If possible logistically, the relief should be monitored for longer. In this regard, the period of two hours is nominated as a minimal period that seems practicable in general. Patients who need to travel, or who need to return to work or to other duties may find it inconvenient to remain for a longer period.

The patient's response should be recorded independently by the assessor at prescribed periods. A reasonable schedule is to record the level of pain before the block, immediately after the block, 30 minutes after the block, and hourly thereafter. The assessment of pain should be complemented by an assessment of any improvement of disabilities, and by a narrative description of either how the patient feels about the relief obtained, or any difficulties that they may have

concerning the response. The narrative serves to corroborate the response inferred from the visual analogue scores for pain. When a patient scores zero on the VAS but is not pleased by the effects of the block, doubts can be raised about the effectiveness of the block. Conversely, the assessor can be more confident in the response if the patient reports feeling the best that they have felt in years, or other such descriptions. Reciprocally, a patient who verbally reports that they obtained complete relief but fails to indicate that on their visual analogue scale should explain the discrepancy.

Once the assessor has completed the assessment it should be reported to the doctor who should review it. The doctor's responsibility in this regard is to ensure that the assessment has been properly conducted and thoroughly recorded. The doctor cannot dispute the assessment. They can, however, identify whether there has been any misinterpretation, for example if the patient and the assessor both misconstrued which of a patient's several pains was targeted by the block undertaken. Any corrections to the record in this regard should be described in narrative. In order to avoid ambiguity, misinterpretation, or misrepresentation, the original assessment form should not be amended; instead, a revised assessment form should be appended to the original form, along with an explanation of why and how the revision was undertaken.

Once the doctor has reviewed the assessment, the patient can be discharged.

Subsequently, the assessor should review the patient in order to determine and record the patient's longer-term response to the block. This could be done by telephone interview on the following day. This review should record the ultimate duration of any positive effect of the block, in terms of when the patient's pain returned, how they felt during the period of relief, and any side

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effects or reservations about the effect. The use of telephone enquiry does not offend the reservation outlined above about recall bias, because the patient's primary response has already been recorded. The telephone enquiry simply obtains supplementary information concerning the patient's subsequent course and welfare.

### Reference

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## Schedule A. Instruments for Assessing Response

A variety of instruments might be used to assess the effects of a diagnostic block. There is a risk, however, of overloading a patient with enquiries, questionnaires, and other tools. A pragmatic approach is to use three instruments.

1. Relief of pain can be recorded by using serial visual analogue scales.
2. Relief of disabilities can be recorded by having the patient nominate four, or as many as possible, activities of daily living that (1) are impeded or prevented by their pain; (2) which are likely to be restored, or should be restored, if the pain is relieved; (3) and which can practically be assessed in a clinic setting. (Examples might include bending, lifting, turning, sitting, walking. Impractical examples include return to work, sleeping, and having sex.) To this end, the assessor should record the nominated activities before the execution of the block, and observe and note the demonstrated degree of disability. After

the block, and repeatedly throughout the period of assessment, these disabilities should be assessed and the degree of restoration recorded.

3. To corroborate the assessor's record of the assessment, a powerful tool is to videotape the patient executing activities before and after the block. Such a record could also include the patient's mood and facial expression.

Appendix A, shows an example of a form that succinctly records an assessment.

### Interpretation

A positive response to a block is, *prima facie*, one in which there is complete relief of that part of the patient's pain which the blocks might be expected to relieve, for a duration commensurate with the expected duration of action of the local anaesthetic used. Partial reduction of that pain does not constitute a positive response. The only exception can be that the patient's accustomed pain is completely relieved but they complain of pain from the needle track, which would not be relieved by a medial branch block.

If the patient's pain is mediated by the nerve or nerves anaesthetised, they should obtain complete relief of their pain.

If the patient's pain is mediated by nerves other than the ones anaesthetised, they should obtain no relief of their pain.

If the patient's pain is mediated by several nerves, including but more than the ones anaesthetised, the patient will obtain relief of that part of their pain that is mediated by the nerves anaesthetised, but no relief of pain mediated by the other nerves. Examples include:

- ♦ in a patient with bilateral pain, if only the left side is blocked, the pain on

that side will be relieved but the pain of the other side will not be relieved.

- ♦ in a patient with pain mediated by three consecutive nerves, if only the upper two are blocked, the patient may obtain relief of the upper part of their pain, but no relief of the lower part. The converse applies if the lower nerves are blocked.

Such responses nevertheless constitute a positive response, for the pain targeted by the blocks was completely relieved. The responses may be partial topographically but they are complete physiologically, in the targeted area.

In the event of such responses, a comprehensive or systematic approach may be undertaken for a complete and accurate diagnosis.

If the patients' pain is bilateral and corresponds to the pattern for a particular segmental level,

- ♦ the two nerves on each side of the same segment may be blocked, or
- ♦ the patient could be assessed as if they have two pain sources, one on each side, and each side is addressed systematically but independently.

If controlled blocks on each side relieve the pain on their respective sides, a subsequent block may be undertaken simultaneously bilaterally, if it is necessary to show that all of the patient's pain can be relieved at the one time.

If blocking one side relieves all of the patient's pain bilaterally, there is no need to proceed with investigation of the opposite side.

The latter approach is intellectually more efficient and less subject to "diagnostic noise", for it allows the demonstration of bilateral pain stemming from just one side, if that is the case. However, in some settings it might not be practical or convenient for the patient to return for systematic investigation; in which case it might be prefer-

## Cervical Medial Branch Blocks

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## Cervical Medial Branch Blocks

able to block both sides of an apparently unisegmental pain simultaneously; or, once a single block on each side has been found to relieve the pain on the respective side, a confirmatory block may be undertaken simultaneously on both sides.

If the diagnostic hypothesis is that the patient's pain is mediated by multiple, consecutive medial branches, and if they obtain relief of the upper half of their pain when the upper nerves are blocked, it should transpire that blocking the lower nerves will relieve the lower half, but not the upper half, of their pain; and all of their pain should be relieved when all nerves responsible are blocked. Although it takes one more procedural session than it does to diagnose a unisegmental pain, it is preferable to diagnose multilevel pain as a staged procedure. The first block relieves the upper region of the patient's pain; a second block relieves the complementary region of pain; and the third, confirmatory, control block can address all levels at once. A staged procedure in this manner, secures a valid diagnosis. It avoids wrongly presuming, *ab initio*, that the pain is mediated by multiple, consecutive nerves, and blocking all of those nerves. Doing so may lead to false positive results when more nerves are incriminated than warranted.

A third circumstance is where the patient has two, displaced sources of pain, e.g., upper cervical pain and headache stemming from C2-3, and lower cervical pain and shoulder pain stemming from C5-6. In those circumstances, blocking the third occipital nerve will relieve the headache but not the neck-shoulder pain; and conversely, blocking C5,6 will relieve the neck-shoulder pain but not the headache. In such cases, the two components of the patient's complaint should be addressed separately, blocking one at a time. By this measure, it can be shown beyond doubt that the two sites of pain have independent sources.

Investigating both sources separately also allows cases to be identified in which upper cervical as well as lower cervical pain arise from only an upper cervical joint, or in which lower cervical as well as upper cervical pain arise from a lower cervical joint. Such cases will not be identified if it is presumed that the two sites of pain have separate sources. Using such a presumption to proceed summarily with blocks at upper and lower levels simultaneously may result in more nerves being incriminated than warranted.

For patients with multiple and bilateral sources of pain mediated by the cervical medial branches, it may not be possible to anaesthetise all sites simultaneously. If a comprehensive diagnosis is required, each site might have to be confirmed separately, or in combinations that are both sensible and practical.

### Performance Parameters

Although experienced and expert operators may be more efficient and faster than the following standards require, the parameters that define a minimum level of competence and proficiency for the performance of cervical medial branch blocks are:

- ♦ not more than eight adjustments or corrections of the course of the needle from insertion to reaching the target point;
- ♦ not more than six adjustments to move from one target point to another in the conduct of a third occipital nerve block;
- ♦ not greater than 1.5 minutes total radiation exposure time to block a given nerve, which includes exposure time to identify the target point and puncture point, prior to insertion of the needle.

## An Algorithm

### *Part 1: Initial Assessment*

The first part of the algorithm is an overview. It invites practitioners first

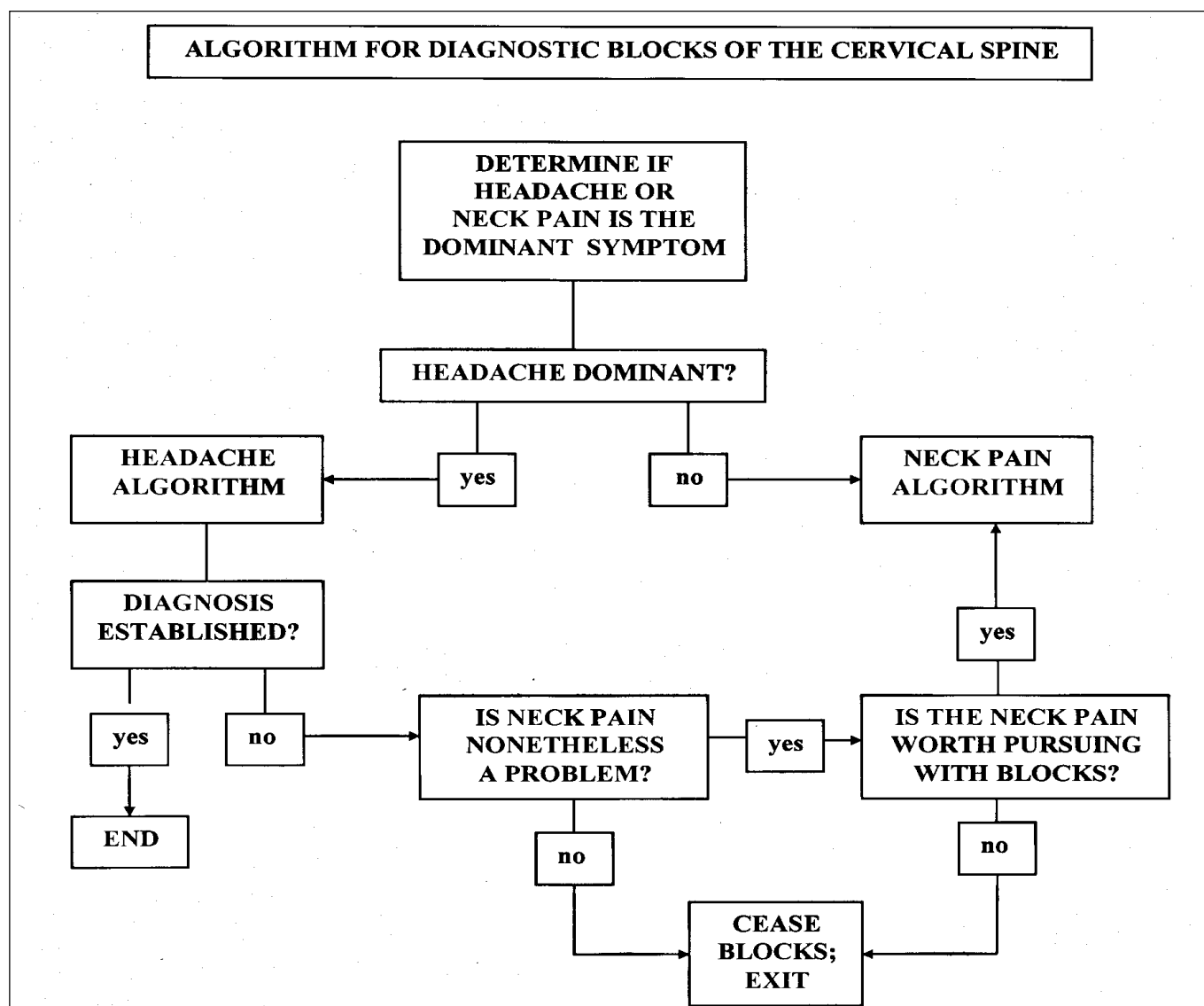
and foremost to determine whether headache or neck pain is the patient's dominant symptom. This question does not disqualify a patient from eventually being investigated both for their headache and for their neck pain. Indeed, the algorithm allows for a patient with headache to return for investigation of their neck pain in the event that investigations of their headache should prove negative.

The purpose of the initial question "Is headache dominant?" is to direct investigations to appropriate levels in the cervical spine. The available epidemiologic data indicate that cervicogenic headache most often stems from the C2-3 and adjacent joints.<sup>1</sup> Only rarely does it arise from C4-5, and it has not been proven to arise from joints lower in the neck.<sup>2</sup> In a patient with headache greater than neck pain, it becomes more likely that a positive diagnosis will be established by starting investigations at C2-3. To start at other levels defies the pre-test probabilities, and renders the diagnostic process inefficient.

If a diagnosis is not established by pursuing the headache, the algorithm allows for a return to pursue the neck pain. However, it poses two questions: whether the neck is nonetheless a problem, and whether the neck pain is worth pursuing with blocks. These questions rely upon the practitioner's discretion and intuition, because there are no objective data by which these questions might be answered.

In this regard, the algorithm is potentially educational. Practitioners can audit their experience and determine whether positive responses to these questions prove profitable in their experience. If they are not, the practitioner should reflect on the validity of the criteria that they have been using to establish affirmative responses to the questions, and correct those criteria in the interests of improving the efficiency of their practice.

## Cervical Medial Branch Blocks

**Algorithm Part 1: Initial Assessment****Part 2: Headache Algorithm**

The algorithm commences with a surprising question, "are confirmatory blocks positive?" The virtue of this idiosyncrasy is revealed later, once the algorithm operates. In essence, it is an invitation to stop. In the first instance, the logical answer to this question for a patient who has not yet commenced investigation is "no". That answer permits entry deeper into the algorithm.

The next question constitutes a node of accountability. It asks the practitioner intuitively to answer whether

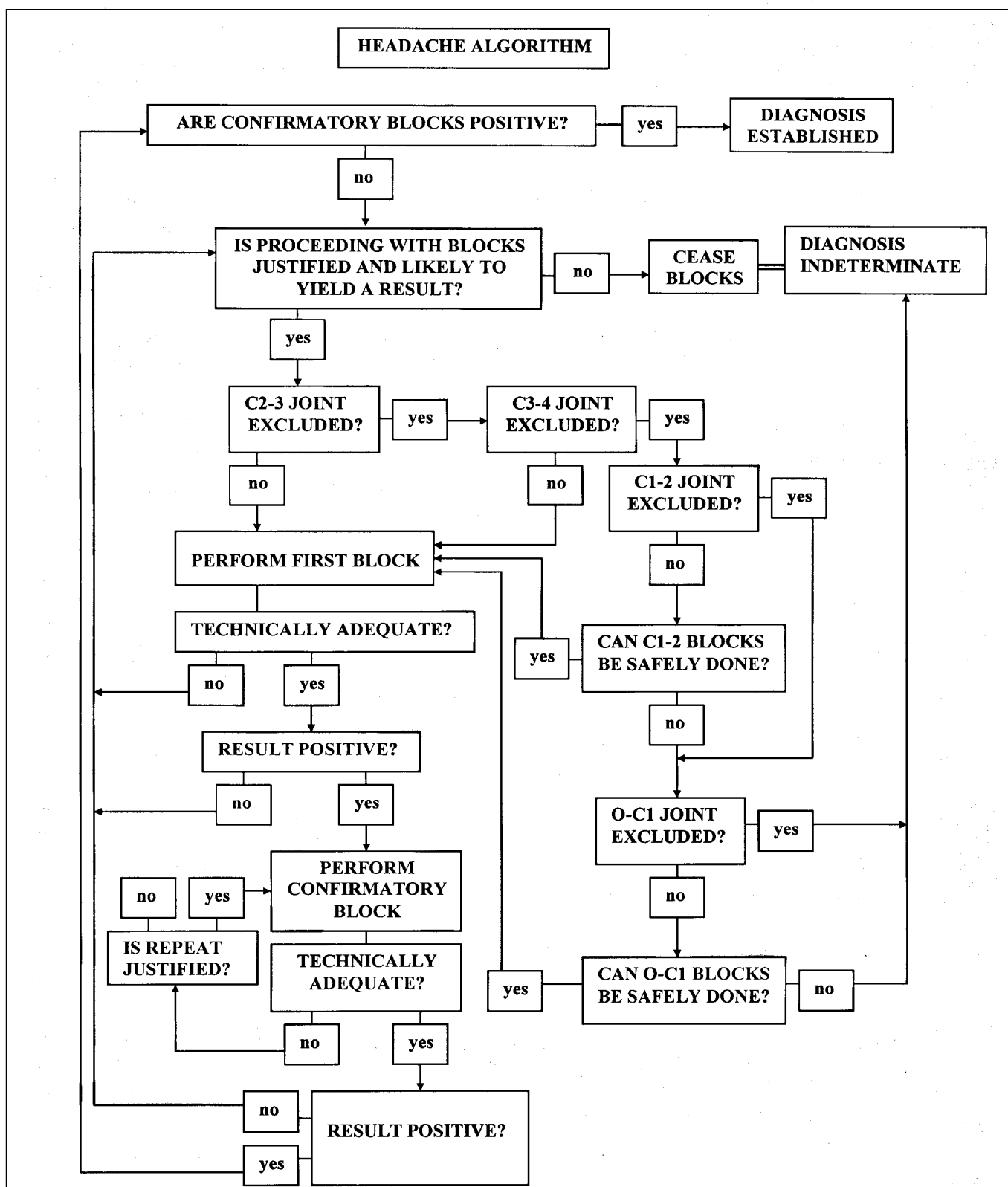
proceeding with blocks is justified and likely to yield results. For a patient who has not commenced blocks the implicit answer is obviously "yes", which permits entry further into the algorithm. Later, in the face of negative results, the answer to this question may be "no". In that event, the algorithm invites cessation of investigations. This question is not based on absolute criteria. Rather, it offers the practitioner the opportunity to decide that enough is enough. It is placed in the algorithm largely to accommodate negative responses and technical failures of

blocks. When practitioners encounter one or more technical failures, the question invites them seriously to consider why they should persevere.

The definitive section of the algorithm asks whether the C2-3 joint has been excluded. This joint is specified as the starting point because the epidemiologic evidence indicates that, of patients with neck pain in whom headache is the dominant feature, the chances of C2-3 being the source are 54%.<sup>1</sup> Most patients, therefore, will have pain stemming from this level. Investigating patients for this condition



## Cervical Medial Branch Blocks



Algorithm Part 2: Headache

## Cervical Medial Branch Blocks

is the most efficient step. There is no evidence to support beliefs that a practitioner can tell whether a patient has headache stemming from a different level. Therefore, starting at any other level is not justified.

The established test for C2-3 joint pain is a third occipital nerve block.<sup>1</sup> This should be performed in the manner described above (see Technique).

A real and genuine problem that does occur with the conduct of third occipital nerve blocks is the failure to anaesthetise the target nerve adequately. This may occur in up to one-third of cases. It is, however, immediately evident. The patient fails to obtain anaesthesia in the cutaneous territory innervated by the nerve. This event invites repetition of the block, either on a subsequent occasion, or as soon as practicable on the day of the first procedure once the failure is recognised. For this reason, the algorithm asks whether there has been a technical failure. If not, the patient proceeds deeper into the algorithm. But if the block has been technically inadequate, the practitioner is asked to consider whether persevering with a repeat block is justified. In most cases the response would be "yes", but in some instances it might be that the practitioner recognises irregularities, such as osteophytes in the target region, that render it unlikely that repeating the block will avoid another technical failure.

If the result of the first block of the C2-3 joint is positive, the algorithm requires a confirmatory block.

Furthermore, the algorithm allows for a possible technical failure and repetition of the confirmatory block. This step essentially presumes that there have been no previous technical failures. However, practitioners should consider seriously whether they encounter technical failures more than once in a given patient, or whether technical failures are inordinately common in their practice. Perhaps they

are not performing the procedure correctly.

If the response to the confirmatory block is positive, a diagnosis of C2-3 joint pain is established, and investigations cease. This should be the most common outcome of the algorithm. It is reached within two blocks, but perhaps three, if there has been a technical failure. Some 50% of patients, therefore, should be subjected to no more than three procedures, and most of these to only two procedures.

If the response to C2-3 blocks is negative, the algorithm calls for reflection.

If the first block of C2-3 is positive but the confirmatory block is negative, a conundrum arises. This is a paradoxical response. Either the first block was false positive or the confirmatory block was false negative. Without further and multiple investigations these possibilities cannot be distinguished. The answer does not lie in simply repeating the block until another positive response is produced. This constitutes illusory logic. Obtaining further positive response does not wipe out the negative response, and may itself be another false positive response. It might be argued that a patient could undergo two or three repetitions in order to establish by averaging that the one and only negative response was false negative, but doing so invokes the possibility of coaching the patient into having a positive response. In the absence of an efficient means of resolving the conundrum, the algorithm recommends that no further investigations be pursued. The course of two or more further investigations, simply to resolve the conundrum under dubious conditions denies new patients access to the resources of the practitioner.

If the first block of C2-3 is negative, the investigation can proceed to other segmental levels.

The algorithm invites consideration next of the C3-4 joint. This joint should be blocked using standard techniques

for medial branch blocks of typical cervical zygapophysial joints (see Technique above).

Blocks of the C3,4 medial branches should not be subject to technical failures. Accordingly the questions concerning technical adequacy should be ignored when evaluating the response to these blocks.

If C3,4 blocks prove positive the diagnosis is established. On the average, patients with headaches stemming from C3-4 would be diagnosed within three procedures: the first being a third occipital nerve block whose result is negative and which excludes C2-3, a second to establish *prima facie* a positive response to C3,4 blocks, and a third to confirm the response.

If the results of the confirmatory block are negative, investigations should cease, for the same reasons outlined above in the context of negative response to confirmatory blocks of C2-3.

If C3,4 blocks are negative, the C1-2 joint should be considered. However, a major caveat applies. C1-2 blocks are not easy to perform, and carry risks greater than other cervical joint blocks. Consequently, the algorithm asks whether C1-2 blocks can be safely done. If the answer is "yes", the algorithm invites performance of the appropriate block.

C1-2 blocks require an intra-articular injection, for which safe techniques have been described.<sup>3-5</sup> The questions of technical adequacy do not apply when these blocks are performed.

If C1-2 blocks are positive and confirmed, a diagnosis is established. According to the algorithm, this is achieved within four steps: one block each to exclude C2-3, and C3-4, one to establish *prima facie* that C1-2 is positive, and one to confirm the response.

If the response to C1-2 blocks is negative, or if C1-2 blocks cannot be safely performed, the algorithm asks

## Cervical Medial Branch Blocks

whether atlanto-occipital blocks (O-C1) can be performed safely. If not, all investigations cease, and the diagnosis is indeterminate. If O-C1 blocks are available, they can be performed as the final possible step in the investigation of headache.

The prevalence of O-C1 headaches is not known, but is presumably low. For that reason it is the last joint to be entertained by the algorithm. If it is the responsible joint, the algorithm finds it positive within five steps: one each to exclude C2-3, C3-4, and C1-2, one to establish *prima facie* a positive response at O-C1, and one to confirm it. Given the rarity of O-C1 headaches, and given the scarcity of individuals with skills to perform blocks at this level, proceeding to this point in the algorithm should be a rare occurrence.

In essence, most cases of headache should be identified within two blocks, with perhaps up to one-third of patients encountering a technical failure. In other words 50% of patients will require 2-3 blocks, on the average. Of the other 50%, a small proportion will prove positive to C3-4 blocks, and will require no more than three blocks to achieve the end-point. Another proportion will prove positive at C1-2, and will require four blocks to reach that end-point. The efficiency of the algorithm is such that patients with headache should have a diagnosis established, on the average, within three blocks. Investigations might be abandoned after fewer than this number of blocks in some cases. The need for four blocks should be unusual, and would pertain to the rare instances of pursuing O-C1. Otherwise, the need for more blocks than average suggests a lack of skill on the part of the practitioner, or an undisciplined approach to the problem.

### Part 3: Neck Pain Algorithm

The initial step in investigating neck pain is to interpret the distribution of the patient's pain. If it does not conform within reason to a typical pattern of

pain attributable to a zygapophysial joint, investigations should not be undertaken, for the probability is low of determining a confident diagnosis in patients whose pain is not so distributed. In particular, the yield of positive zygapophysial joint blocks is essentially nil in patients with widespread pain with no focal epicenter.

If a pattern is identified according to the pain maps for zygapophysial joint pain<sup>6</sup> the corresponding segment should become the target for investigations.

The algorithm proceeds with an unexpected question, "are confirmatory blocks positive?" This is a point to which the algorithm returns if investigations prove positive. If no blocks have yet been performed the answer is "no", which allows the practitioner to proceed to deeper layers of the algorithm.

The next question constitutes a node for reflection. It relies on the practitioner's judgement and intuition to cease blocks if, after other steps in the algorithm, there are grounds for doubt about the propriety of proceeding with investigations. At the commencement of investigations the default answer is "no", which allows progress to the next step.

The algorithm asks whether the first joint selected has been excluded. If it has been excluded, the option arises to investigate other joints that might reasonably be responsible for the patient's pain. This step of the algorithm allows for acceptable errors in judging the segmental location of the patient's pain from the pattern of distribution of their pain. In this regard, the acceptable error is  $\pm$  one segment. Thus, a C6-7 pain may be misread as a C5-6 pattern, or a C5-6 pattern might be read for a C4-5 pain. For practical purposes, the joints that might reasonably be expected to account for lower cervical pain are the C5-6, C6-7, and C4-5 joints, in that order of prevalence. Pursuit of a joint outside this range

would constitute extenuating circumstances, and is not covered by this algorithm.

If all joints have been excluded, further investigations are unlikely to yield a positive result, and investigations should cease.

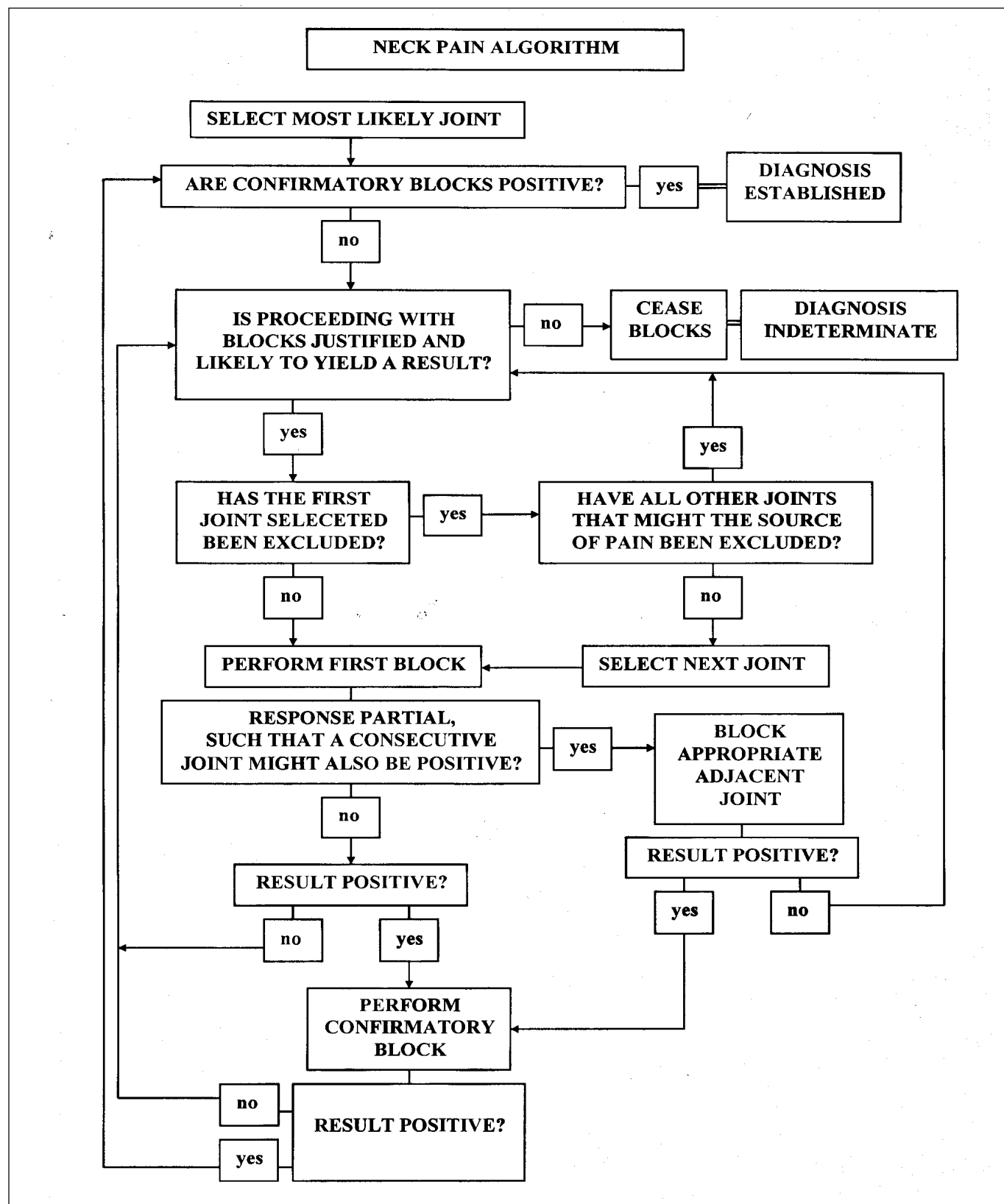
If all joints have not been excluded, but previously selected joints have been excluded, the next most likely joint should be selected and investigated.

Whether the joint is the first to have been selected, or is a joint subsequently selected, the first definitive step of the algorithm is to block that joint. The block should be performed according to published standards (see Technique above).

The first step in evaluating the response asks whether the response is partial. This question allows for the possibility of a patient having two consecutive joints painful. In that event the patient reports complete relief of pain in one half or other of their region of pain, but no relief in the complementary half. This is the only and strict meaning of partial (see Interpretation above). Partial does not mean incomplete relief of all of their pain. It requires complete relief of pain but only in part of the anatomical region in which the pain is felt. In practice, the patient will report complete relief in the upper half of their painful region, or in the lower half. The implication is that a consecutive joint, below or above the one anaesthetised, is still painful.

If the response is partial, the algorithm invites a block of the appropriate adjacent joint: the one below the first joint blocked if only the upper half of the patient's pain was relieved, or the joint above the first joint blocked if only the lower half of the patient's pain was initially relieved. This second block carries the same status in the algorithm as a first block. For the purpose of allocating agents under double-blind protocols, the same agent should be used for this block of the adjacent joint as was used for the block of the first joint.

## Cervical Medial Branch Blocks



Algorithm Part 3: Neck Pain

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If the response to the adjacent block is positive, i.e., it relieves the pain not relieved by the first block, a confirmatory block is undertaken. This block should be performed at both the previously anaesthetised levels, and for both joints the second agent allocated under a double-blind protocol should be used.

If the response to the confirmatory block is positive, a diagnosis is established of double-level pain at consecutive joints. In this regard, the most common of double-level patterns is C5-6, C6-7. Less commonly, C4-5, and C5-6 pain may occur together.<sup>7-9</sup>

If the response to the confirmatory block is negative, the investigator should reflect on the propriety of proceeding with further blocks. Implicitly, the patient has reported either false-positive responses to the first blocks or a false-negative response to the confirmatory block. The two possibilities cannot be distinguished with information available. Nor is it legitimate to repeat blocks in an effort to secure a positive response. On the one hand this subsequent positive response does not negate the negative response. Nor can it be refuted that the patient was coached into having a positive response. Since this conundrum cannot be resolved without multiple investigations starting afresh, investigations should cease in the interests of public efficiency. Pursuing multiple blocks in order to resolve a conundrum in one patient denies access to investigation to other patients who might prove clearly positive.

If the response to the initial block is not partial, its result will be either negative or positive.

If the response to the first block is negative, the algorithm invites consideration of whether pursuing investigations is justified. Given a negative response at the only level that the practitioner considers likely to be the cause of pain, the practitioner might be satisfied that no further investigations are required. If, however, the practitioner

believes that they may have made a mistake in selecting the joint to be blocked, the algorithm allows other joints that might reasonably be the source of pain to be investigated.

If the response to any block is positive, a confirmatory block is performed.

If the confirmatory block is positive, a diagnosis is established.

If the confirmatory block is negative, the algorithm calls for reflection. As argued above, under these conditions, proceeding with blocks is inefficient, and the default recommendation is to cease blocks.

#### Part 4: Efficiency

Under the operation of this algorithm, the most common source of lower cervical pain, C5-6, can be diagnosed within two blocks: one to establish *prima facie* that the joint is painful, and one to confirm the response. In the event that a patient has two consecutive joints that are painful, the diagnosis is established within three blocks: one each to establish that each of the two joints is *prima facie* positive, and one to confirm the response. If an error is made in judging from the distribution of the patient's pain which joint is responsible, the algorithm allows for another joint to be selected. In that event, a diagnosis should be established within three blocks: the first negative block, the second block to establish *prima facie* that the second joint selected is painful, and one to confirm the response. At its limit, the algorithm allows for two errors in judgement, and the testing of three joints. Under those conditions, either the third selected joint is diagnosed within four blocks, or all reasonably possible joints are excluded within three blocks.

Given that errors in judgement should be uncommon, the algorithm allows a diagnosis to be established, on the average, within three blocks. The rare instances of needing to proceed to four blocks should be outnumbered by the occasions when the diagnosis can

be made within two blocks.

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# Muscle Cramp

*Dr Steve Bentley MBChB, Dip Obst, Dip Sports Med, Dip MSM, FAFMM, Musculoskeletal Physician, Dunedin NZ*

## Abstract

Muscle cramp is a common phenomenon experienced by most people at some time in their life. Cramp occurs in a wide variety of conditions and circumstances such as; during prolonged exercise, pregnancy, metabolic disorders (e.g., hypocalcaemia), central nervous system disorders and in certain cases of toxicity or poisoning, reflecting a multifactorial pathogenesis.

Since Denny-Brown and Foley demonstrated increased frequency of action potentials on EMG during cramp in 1948 and Norris et al induced cramp using a maximum voluntary contraction with the muscle in a shortened position in 1957, there has been very little worthwhile research on the subject.

Muscle fatigue has been shown to affect spinal reflexes, increasing muscle spindle activity and reducing 1b afferent activity from golgi tendon organs. Fatigue also slows the relaxation phase of muscle contraction predisposing to tetany if the firing frequency is maintained or increased. However cramp can still be induced when spinal reflexes are abolished by peripheral nerve block. Cramp may start at several different places simultaneously in a muscle, not within the same motor unit. It is unclear whether muscle cramp may originate at the terminal branches of motor axons, the motor end plate or within the muscle fibres themselves. In some cases the disturbance arises within the CNS.

Cramp is a self-limiting condition but can be treated by active or passive stretch. Quinine reduces the frequency of night cramps by up to 43% and also the severity of cramp. Botulinum toxin injection and myofascial trigger point injections may have a role in treating cramp.

## Definition

**M**uscle cramp is the localised, involuntary, sustained and painful contractions of skeletal muscle, generally of sudden onset and characterised by high frequency discharges of muscle action potentials on the electromyogram, reflecting increased motor unit activity.

## Classification of Muscle Cramp

1. Night cramps, especially in the elderly (limb position)
2. Exercise-induced muscle cramp
3. Occupational cramp (e.g., writers, miners, musicians)
4. Cramps of pregnancy
5. Metabolic disorders
  - ♦ extracellular hypoosmolality, hyponatraemia
  - ♦ hypocalcaemia, tetany or carpopedal spasms
  - ♦ hyperventilation, respiratory alkalosis (transient fall in free calcium)
  - ♦ hypokalaemia (diuretics, liquorice, carbenoxolone, amphotericin B)
6. Neurological disorders
  - ♦ spinal cord injury
  - ♦ multiple sclerosis, motor neuron disease
  - ♦ neoplasia of CNS

- ♦ encephalomyelitis, poliomyelitis
7. Suspected neurological disorders
    - ♦ "Stiff Man" syndrome (? CNS disorder). Cramps can be arrested by curare or peripheral nerve block
    - ♦ Denny-Brown AND Foley syndrome (benign fasciculations and cramp)
  8. Toxicity or poisoning
    - ♦ tetanus (peripheral nerve), black widow spider bite
    - ♦ drugs: strychnine (inhibits glycine), danazol, labetalol, beta 2 agonists, phenothiazines, lithium carbonate)
  9. Peripheral vascular disease

## Historical Studies on Muscle Cramp

1940. Quinine was reported by Moss and Herman<sup>1</sup> to be effective in treating 15 elderly people for nocturnal cramp.

1948. Denny-Brown and Foley<sup>2</sup> demonstrated increased frequency of action potentials during muscle cramp on electromyogram.

1957. Norris et al<sup>3</sup> reported that 16% of healthy young adults experience night cramps >2x/month.

Cramp could be induced using a maximum voluntary contraction while the muscle was in a shortened posi-

tion. Relief of cramp can occur by stretching the muscle. The authors commented: "It is still uncertain whether cramps have their origin in muscle, peripheral nerve or in the spinal cord."

There has been very little research on cramp over the last 50 years. Muscle cramp pathophysiology has yet to be determined. Myths such as increasing salt intake to prevent cramp have no scientific basis. Electrolyte disturbances at the muscle cellular level during cramp have not been measured. Muscle cramp may occur in hyponatraemia due to excessive intake of hypotonic fluids during endurance sport.

1986. Maughan<sup>4</sup> measured serum electrolytes (sodium, potassium and bicarbonate), haematocrit and plasma volume in 82 runners before and after a 42.2 km marathon. There were no differences in the 18% who suffered cramps and those who did not.

1988. Sontag and Wanner<sup>5</sup> proposed that modern lifestyle and sitting causes loss of flexibility in the lower limbs. A series of case studies is used to illustrate the benefit of stretching in patients with chronic muscle cramp symptoms.

## Muscle Cramp

**Observations on Muscle Cramp**

1. Most individuals experience cramp at some time in their life.
2. Certain individuals experience cramp often. Cramp is less common in children.
3. Twitches or fasciculations increasing in frequency may precede cramp.
4. A voluntary contraction of a muscle in a shortened position can cause cramp.
5. Muscle cramp can be relieved by stretch, but not after prolonged exercise to fatigue.
6. Exercise related cramp occurs in endurance events near the end or after the race.
7. Exercise related cramp occurs during intense competition rather than in training.
8. Athletes may on occasions "run through" cramp.
9. Cramp occurs during a concentric contraction (e.g., in the calf or toes during a kick turn in a pool).

Various theories on the pathogenesis of muscle cramp include CNS excitation, increased motoneuron excitability and local muscle metabolic, and electrical or motor end-plate disturbances.

Research supporting different theories is scant. Physiological studies on muscle may give indirect evidence of possible mechanisms.

**Muscle Proprioceptors and Spinal Reflexes**

- ♦ Nelson and Hutton<sup>6,7</sup> (1985/86) examined proprioceptor activity in cat gastrocnemius muscle stimulated to fatigue. The excitatory drive to motor units ( $\alpha$  and  $\gamma$  motoneurons) increases as force production falls during fatigue. Golgi tendon organ (GTO) activity is depressed or abolished in a fatigued muscle and recovers slowly. This results in unopposed  $\alpha$  and  $\gamma$  motor neuron stimulus to motor units.

- ♦ Romano and Schieppati<sup>8</sup> (1987) demonstrated during concentric muscle contraction, particularly when the muscle is in a shortened position and contracting rapidly, increased spindle activity and motor unit recruitment.
- ♦ It is likely that chemically induced nociceptor stimulus from muscle damage or metabolic stress will cause increased reflex  $\alpha$  and  $\gamma$  motoneuron activity.

**Actin-Myosin Contraction Coupling**

$H^+$  ions produced during intense anaerobic exercise from glycolysis (particularly type IIB muscle fibres) have been postulated to cause muscle fatigue by:

1. Interfering with phosphofructokinase activity in glycolysis (negative feedback)
2. Competing with  $Ca^{2+}$  for troponin binding sites, reducing actin-myosin coupling
3. Reducing calcium release from the sarcoplasmic reticulum (SR).

Calcium re-uptake to the SR is energy (ATP) dependent requiring calcium ATPase. Individuals with calcium ATPase deficiency experience muscle pain following exercise and delayed muscle relaxation but not cramp.  $H^+$  ions may possibly inhibit calcium ATPase activity, reducing the reuptake of calcium to the SR, prolonging muscle contraction (hypothesis).

In a fatigued muscle there is a slower relaxation phase of contraction, muscle spindle activity and motor unit recruitment increases, GTO activity is depressed or abolished. If the firing frequency is not reduced, fused summation and tetany will result.

**Evidence for a Peripheral Mechanism of Cramp**

1. Bertolasi et al<sup>9</sup> (1993) used EMG, and induced muscle cramp by voluntary contraction of the muscle in a shortened state. Cramp was re-

lieved by stretch. Following peripheral nerve block, cramp was induced by stimulation distal to the block and could be relieved by stretch despite the nerve block (GTO 1b afferent blocked).

2. Roeleveld et al<sup>10</sup> (2000) used a spatial surface EMG recording muscle activity during calf cramp which was induced by a maximum voluntary contraction. The results showed:

- ♦ Cramp can start at several places simultaneously in a muscle, may involve only a small part of the muscle and spread slowly activating neighbouring muscle fibres, not necessarily whole motor units.

- ♦ The firing rate of motoneurons in cramp is higher than during a maximum voluntary contraction in which the whole muscle is active. Large motor units have fast twitch fibres with shorter action potentials. The high firing frequency during cramp suggests selective recruitment of fast motor units during cramp. The self-limiting character of cramp may be due to rapid fatigue of fast twitch fibres.

Is the action potential during cramp generated at the level of the terminal branches of motor axons or at the muscle fibres themselves, rather than by way of whole motor unit activation?

**Treatment of Muscle Cramp**

1. *Passive or active stretch.* Norris et al<sup>3</sup> 1957; Bertolasi et al<sup>9</sup> 1993.
2. *Quinine.* Quinine reduces the frequency of nocturnal cramps by 21-43% and reduces the severity of cramps. Side effects of tinnitus and other rare but serious side effects such as cardiac arrhythmia, optic neuritis, thrombocytopenia, and convulsions may limit its use. Quinine reduces motor end-plate excitability and increases refractory period of skeletal muscle contraction. Man-Son-Hing et al<sup>11</sup> (1998).

## Muscle Cramp

3. *Transcutaneous Nerve Stimulation (TNS)*. Mills et al<sup>12</sup> (1982), in a case study, reported the successful application of TNS in aborting widespread chronic cramp of uncertain origin. TNS applied between the scapulae aborted cramp in the lower limbs and elsewhere indicates a likely central mechanism for cramp in this individual.
4. *Botulinum Toxin*. Botulinum toxin blocks presynaptic release of acetylcholine at motor end plates and has been used to treat muscle hypertonicity and cramp. A prospective uncontrolled trial by Bertolasi et al<sup>13</sup> (1997), on people with a benign cramp-fasciculation syndrome who had botulinum injections to the calf and toe flexors, reduced the severity of cramp and increased the cramp threshold frequency (to peripheral nerve stimulation) for up to three months.
5. *Injection of Myofascial Trigger Points*. In a randomised uncontrolled trial, trigger point injections with xylocaine had a similar effect as quinine on reduction in frequency, severity and intensity of nocturnal cramps. The effects were prolonged for over four weeks after cessation of treatment (Prateepavanich et al<sup>14</sup> 1999).
6. *Magnesium*. Magnesium is not effective in the treatment of nocturnal leg cramps. In a randomised, cross over, double-blind placebo controlled trial of 45 subjects with nocturnal leg cramps, treated with oral magnesium citrate 900 mg bid for four weeks and placebo four weeks, there was no difference in the two groups in frequency duration or severity of cramps (Frusso et al<sup>15</sup> 1999).
7. *Vitamin E*. Vitamin E is ineffective in reducing leg cramp frequency, severity or sleep disturbance (Connolly<sup>16</sup> 1992).
8. *Calcium in Pregnancy*. Calcium has been used for cramps during

pregnancy, but there is a lack of evidence of efficacy. In a double-blind randomised study of 60 pregnant women who were given either 1 g calcium gluconate bid or Vitamin C 1 g bid for three weeks in the third trimester, all had normal serum calcium, magnesium and albumin throughout the trial irrespective of treatment. There were no differences in symptoms of cramp between the two groups, and cramp symptoms improved in 60% of both groups (Hammar et al<sup>17</sup> 1987).

### Summary

- ♦ Muscle cramp is common and self-limiting, affecting most people some time in their life.
- ♦ A diverse range of conditions affecting different parts of the nervous system may cause muscle cramp. Disturbances in the CNS, spinal reflexes, peripheral nerves and muscle may all contribute to the occurrence of muscle cramp. Muscle fatigue is important in exercise-induced muscle cramp.
- ♦ There is a lack of scientific knowledge on muscle cramp pathophysiology.
- ♦ Proven effective treatments of muscle cramp include stretching, quinine (moderate efficacy but side effects). There may be a place for botulinum toxin, TNS and trigger point injection in some cases. More research is required.

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# Management of Chronic Achilles Tendinosis: An Update for Drafting Clinical Guidelines

*Dr Charles Ng, MB ChB, Dip Sports Med, Dip MSM*

## Introduction

**A**chilles tendinosis is a common condition affecting sportsmen and active people as well as the less active. It often affects the large group of middle-aged (35 years+) male recreational sportsmen. It has a tendency to become chronic, that is, lasting more than 1-3 months, and is therefore difficult and frustrating to manage for both doctor and patient. The more chronic the condition, the worse the prognosis. Surgery may be resorted to, with unsatisfactory or poor results. A plethora of treatments has developed but there are no satisfactory clinical guidelines demonstrating the appropriate indications for the variety of treatments proposed.

This review sets out to define Achilles tendinosis as distinct from other conditions affecting the Achilles tendon, namely tendinitis, paratendinitis, retrocalcaneal bursitis and tendon rupture. In doing so, tendinosis may be distinguished from these other conditions and therefore treated appropriately.

A review of studies on Achilles tendinosis was carried out from a Medline literature search of studies published since 1995.

## Definitions

**Achilles tendinitis** is a term used by doctors to describe any condition presenting with pain and swelling in the Achilles tendon area. Therefore, treatment tends to be empirical using anti-inflammatory medication (NSAIDs) and physical therapies which are often unsuccessful. The terms tendinosis, tendinitis, paratendinitis, retrocalcaneal bursitis need to be distinguished.

Achilles tendinitis implies an inflammatory process of the Achilles tendon. However, biopsies have failed to demonstrate the presence of inflammatory cells.<sup>1</sup> Alfredson et al,<sup>2</sup> in a study using microdialysis in tendon tissue, did not find high levels of prostaglandin E2 to suggest inflammation. So although tendinitis is a widely used term, it does

not actually describe any specific histopathological diagnosis and hence should not be used.

**Achilles tendinosis** describes degenerative changes in the tendon. There is degeneration and disordered arrangement of collagen fibres, focal hypercellularity, and vascular proliferation.<sup>3,4</sup> Thus it is a histopathological diagnosis. This correlates well with ultrasound and MRI appearances. Tendinosis and *partial rupture* may be hard to distinguish.

**Paratendinitis** refers to inflammation of the paratenon sheath through which the tendon slides. It therefore does not imply any abnormality of the tendon itself, although tendinosis and paratendinitis can occur together.<sup>1</sup>

The terms **Achilles tendinopathy** and **achillodynia** are clinical labels for pain and/or swelling in the Achilles tendon. They could be used initially until a more specific diagnosis is made clinically or with the use of diagnostic imaging or histologically.

This review will address the diagnosis and management of chronic Achilles tendinosis only.

## Diagnosis

Chronic tendon injuries are significantly more common in ageing athletes.<sup>8</sup> The peak incidence appears to be between ages 30 and 50 years.<sup>3,5</sup> The ageing tendon is more vulnerable at a given level of physical loading.<sup>3</sup> However, Gibbon et al<sup>12</sup> found no relationship between the age of the patients and the site of disease, suggesting that the predisposing factors to tendon disease are not age related.

Male predominance is partly explained by a greater participation in sports. However, in men there is a tripled risk of symptoms developing regardless of the level of physical activity.<sup>3</sup> This condition is also seen in patients who do not participate in sports activities or other strenuous leg-loading activities.<sup>7</sup> Astrom<sup>3</sup> found that the extent and severity of the lesion were

unrelated to the degree of physical loading. Although traditionally thought to be related to overuse, few studies have focused on the role of overuse in the development of chronic tendon injuries.<sup>8</sup> Most studies are retrospective and fail to have a control group.

There is usually a gradual onset in Achilles tendon pain and swelling. An acute onset suggests a partial tear of the tendon. However Astrom<sup>3</sup> found partial tendon ruptures were present in 19% of patients with chronic Achilles tendinopathy. The partial ruptures always occurred in areas afflicted with tendinosis. It is plausible that where a degenerative tendon exists, whether symptomatic or asymptomatic, a partial rupture of the tendon can develop. Present data demonstrate that tendinosis and partial rupture are clinically and histopathologically indistinguishable.<sup>3</sup> However *total* Achilles rupture cannot be attributed to pre-existing tendinosis.

In this review **chronic** tendinosis referred to a minimum symptom duration of one month; the majority of study cases being in a 12-250 weeks' range of duration.

## Symptoms

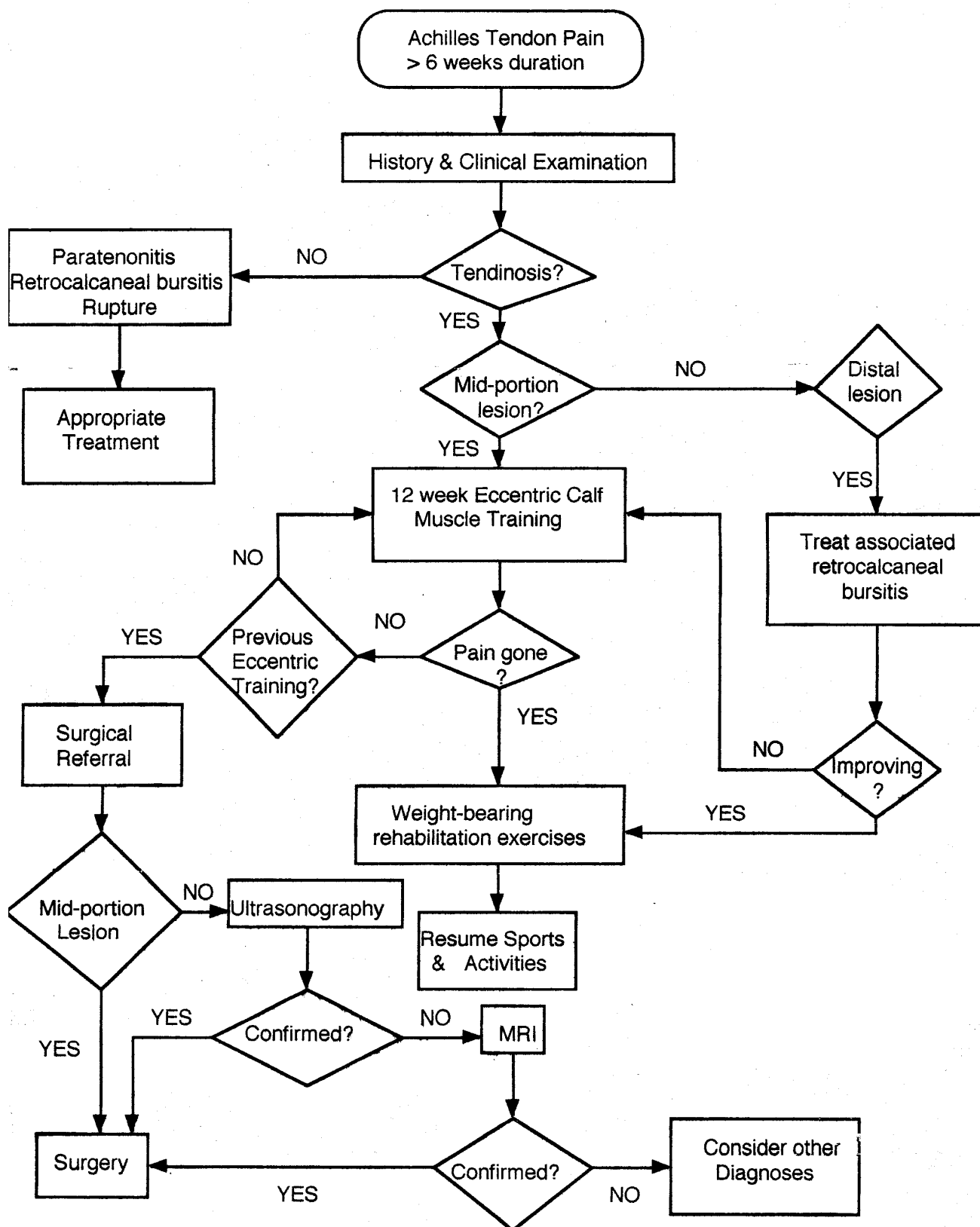
Pain occurs most often in the mid-portion of the Achilles tendon. If severe, pain occurs with walking, otherwise its onset is with increasing weight-bearing activity, walking, running and jumping and landing. There is morning stiffness or stiffness after inactivity.

## Signs

Tenderness and swelling is found in the mid-portion of the Achilles tendon, ranging from 1.5 cm to 7 cm proximal to its insertion in the calcaneus.<sup>1,6,9,11</sup> It can also exist in the proximal and distal (tendon-bone junction) parts of the tendon. Pain and tenderness are consistent presentations. Swelling was variable: 80% of subjects had a soft tissue swelling at the site of pain.<sup>3</sup>

Achilles **paratendinitis** also presents

## ALGORITHM FOR TREATMENT OF CHRONIC ACHILLES TENDINOSIS



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with pain, tenderness and swelling in the same portion of the Achilles tendon. However, the swelling with paratendinitis tends to be more diffuse and remains in a fixed position when the foot is plantar or dorsiflexed. With **tendinosis** the more focal swelling and area of tenderness moves beneath the palpating fingers during plantar and dorsiflexion of the foot.

Pain, swelling, and tenderness in the distal part of the Achilles tendon could also be caused by tendinosis.<sup>1,3</sup> However, **retrocalcaneal bursitis** needs to be excluded and can occur concurrently. Karjalainen et al<sup>6</sup> performed MR imaging of Achilles tendon overuse injuries and demonstrated retrocalcaneal bursitis in 83% of patients with insertional tendinosis. Gibbon et al<sup>12</sup> using ultrasonography found 82% had associated retrocalcaneal bursitis.

Based on the clinical presentation and examination findings, a working diagnosis can be made on which to formulate a management plan. Any tendinosis of less than 12 weeks' duration and which has been untreated would benefit from a conservative management program. If the patient had received some form of conservative treatment and continued to have Achilles tendon pain or if there was any doubt over the diagnosis or other tendon disorders were suspected, then proceed to diagnostic imaging.

### Imaging

Achilles tendinosis has a characteristic appearance on imaging with ultrasonography and MR. However the indications for which imaging technique to use and when to use them remain controversial.

Astrom et al<sup>10</sup> found that chronic Achilles tendinopathy is almost always caused by tendinosis. Therefore ultrasonography and MRI are of limited use in decisions regarding surgical treatment since the lesion is easily identified clinically, but they may become valuable prognostic instruments by

indicating the severity of the lesion. They found that imaging was unreliable in assessing the paratenon and that MRI is more sensitive, but both give similar information.

Karjalainen et al<sup>6</sup> state that, as lesions in the Achilles tendon and in the peritendinous structures can have similar clinical presentation, MRI detects and characterises these changes. A more specific diagnosis and prognosis can be made with the use of MRI than with clinical examination alone. They studied 118 painful Achilles tendons with MRI and detected abnormalities in 111. Of the 21 who went to surgery and had proven foci of tendinosis, 20 were detected on MRI. In all cases with true-positive MRI findings, the lesion had a disorganised tendon fibre structure.

Movin et al<sup>11</sup> performed MRI on 20 patients. All were biopsied and showed increased noncollagenous extracellular matrix and altered fibre structure in the lesions corresponding to the contrast-enhanced areas. There was a high level of extracellular glycosaminoglycans. In summary, Movin felt that clinical symptoms and signs usually correlate well with the area of increased signal abnormality seen at MRI.

Ultrasonography, however, is cheap and reliable in experienced hands and therefore should be the first method of choice.

Gibbon et al<sup>12</sup> performed a retrospective study of 118 symptomatic heels. They found the ultrasound appearance of enlargement and decreased echogenicity of the tendon consistent with the histopathological finding of tendinosis. They stated that high frequency sonography has greater spatial and contrast resolution for superficial tendon structures than does MRI. Ultrasonography is useful in distal third Achilles tendon disease as 82% of the heels had associated retrocalcaneal bursitis. This suggests either a common mechanism of injury or a common causal relationship for

distal third tendinosis and retrocalcaneal bursitis. Paratendinitis was also found to be associated with retrocalcaneal bursitis.

Soila et al<sup>13</sup> conducted a MRI study of 100 normal Achilles tendons and described the appearance of tendon and peritendinous tissue in asymptomatic individuals. The average AP diameter was 5.2 mm. An area of tendon degeneration was detected in four cases. Retrocalcaneal bursae contained a prominent fluid collection in 15 cases. They were able to show the paratenon in great detail, whereas in abnormal tendons Astrom<sup>10</sup> found the method unreliable. Therefore normal Achilles tendon anatomy is variable and may feature degenerative changes and retrocalcaneal bursae fluid collections in the absence of symptoms. Hence there is a risk of misinterpreting findings in symptomatic tendons. Consideration of the clinical presentation needs to be given in order to determine the significance of any abnormal findings.

Some authors suggest ultrasonography and MRI to monitor a tendon's healing response, but Astrom<sup>10</sup> feels that healing is best indicated by relief of pain. His results in healed tendons showed that abnormal imaging was compatible with excellent clinical results. Astrom even doubted that there is any advantage in preoperative imaging as the lesion is easy to identify clinically and chronic Achilles tendinopathy is almost always caused by tendinosis. This is not in concordance with other studies stating that the clinical diagnosis may not identify other Achilles tendon problems.<sup>6</sup>

### Histopathology

Biopsies of abnormal Achilles tendons<sup>2,3,6,9,10</sup> show the following common features:

1. disorganised, irregular tendon fibre structure
2. separation and lack of continuity of the collagen fibres

## Management of Chronic Achilles Tendinosis

3. light collagen staining
4. roundness of tenocyte nuclei
5. active capillary proliferation in some tendons
6. absence of inflammatory cells
7. increased staining for glycosaminoglycans located extracellularly between the collagen fibres.

Movin et al<sup>9</sup> postulate that the increased glycosaminoglycans content may be a reactive cell response to mechanical overloading of the tendon. The glycosaminoglycans may reduce the interfibre cohesion of the collagen bundles resulting in the observed changes in the fibre structure and arrangement. Therefore tendinosis may represent an imbalance between matrix synthesis and degradation.

Astrom's<sup>10</sup> studies in post-mortem material have indicated that tendon degeneration is present in up to 30% of asymptomatic individuals. Minor abnormalities in the tendons of healthy controls have been noted with ultrasonography. Although the gold standard for diagnosis of tendinosis is a histopathological one, the aforementioned MRI and ultrasonography studies have demonstrated an excellent correlation between imaging and histopathological findings.

### Management

Physical therapies like ultrasound, electrical stimulation, cold therapy, heat therapy, frictioning and massage have all been proposed for chronic Achilles tendinosis. However there is an absence of randomised controlled trials to demonstrate their effectiveness.

The general consensus from the limited number of studies available recommend conservative (nonsurgical) treatment initially and that surgical treatment be used if there is no improvement after 3-6 months of conservative treatment.<sup>14-17</sup> The average duration of symptoms from onset to surgery was 12 months.

Johnston et al<sup>14</sup> defined Achilles ten-

don pain as being chronic if of more than six weeks' duration. This distinction was important as more than 90% of acute cases will recover without surgical intervention.

### Conservative Treatment

Despite the long-standing use of these conservative treatments, once again there is a lack of randomised controlled studies of their effectiveness.<sup>8</sup>

#### *Biomechanical Abnormalities*

Astrom's (unpublished) data of 362 patients with Achilles tendinopathy and 147 control patients showed that biomechanical abnormalities were not important in chronic Achilles tendinopathy. Therefore the use of orthotics was of questionable value.

#### *General Recommendations*

These include wearing adequate supporting footwear, improving flexibility by stretching and correcting training errors. Relative rest is recommended as there is little point in persisting with the same type or intensity of exercise if Achilles tendon pain persists or increases. A modified exercise program with reduced or non-weight-bearing exercises would be recommended until the tendon pain disappears.

### Medications

#### *NSAIDs*

These are frequently used for the treatment of soft tissue inflammation, in particular conditions of "tendinitis". However, the histopathological appearance of Achilles tendinosis shows the absence of inflammatory cells,<sup>9</sup> and microdialysis technique showed normal prostaglandin E2 levels.<sup>1</sup> Based on this consistent finding from many studies, there would appear to be no indication for the use of NSAIDs for their anti-inflammatory effect. Any benefit would be from their analgesic effect.<sup>1,18</sup> However, as they are associated with frequent gastrointestinal side effects,

safer medications should be used for analgesia. There may be a theoretical benefit for the use of NSAIDs in Achilles tendinosis where there is associated paratendinitis or retrocalcaneal bursitis. Gibbon et al,<sup>12</sup> showed that 82% of the heels with distal third Achilles tendon disease had associated retrocalcaneal bursitis.

#### *Corticosteroid Injections*

Once again, as there is no demonstrable inflammation in Achilles tendinosis, there is no justification for the use of corticosteroid injections. Corticosteroid injections have been associated with tendon ruptures. However the relationship between steroid injection and rupture remains controversial. Following the use of corticosteroid injections to treat chronic painful conditions of the Achilles tendon, frequent partial ruptures have been observed.<sup>19</sup> Astrom<sup>20</sup> in a retrospective study of 342 patients with chronic tendinopathy found that treatment with corticosteroid injections was shown to predict a partial rupture. Therefore the concern is whether corticosteroid injections cause partial ruptures of the Achilles tendon, or is this merely an association, as the more severe Achilles tendinopathies are the ones that tend to be injected. The more severe cases are the ones that have severe degenerative changes and pre-existing partial tears. Achilles tendon tears following corticosteroid injections are likely to occur if the patient mobilises excessively or too soon following an injection. If corticosteroid injections are given for paratendinitis and retrocalcaneal bursitis, there may be a case for immobilising the ankle joint in a functional brace after an injection. The duration of bracing and its efficacy would need to be confirmed in controlled trials.

### Exercise therapy

Almekinders<sup>8</sup> reviewed studies using exercise therapies for chronic ten-

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don injuries but could not find any prospective controlled studies. Recently however, Alfredson et al<sup>5</sup> reported excellent results from a prospective trial studying the effect of heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. They studied 15 recreational athletes (12 men and three women, mean age 44.3 years  $\pm$  7 years) who had a diagnosis of chronic Achilles tendinosis with a long duration of symptoms (18.3 months, range 3-100). They had pain on running and had tried conventional conservative treatment consisting of rest, NSAIDs, biomechanical correction, physical therapy and ordinary training programs, with no effect on the Achilles tendon pain. The comparison group consisted of 15 recreational athletes (11 men and four women) with a younger mean age of 39.6  $\pm$  7.9 years. This group also differed in having a longer duration of symptoms (33.5 months, range 6-88), so it was not an identical control group. This group had tried the same conventional treatments as the study group without any relief of the Achilles tendon pain. They were rested from treatment for the three-month period prior to undergoing surgery. All patients had Achilles tendon pain for at least three months and an area of tendinosis located 2-6 cm above the Achilles tendon insertion. The diagnosis of tendinosis was confirmed by ultrasonography.

Calf muscle strength and the amount of pain during activity (recorded on a 100 mm visual analogue scale) were measured before the onset of training and after 12 weeks of eccentric training. At week 0, all patients had Achilles tendon pain preventing running and there was significantly lower eccentric and concentric calf muscle strength on the injured compared with non-injured side. After the 12-week training period, all 15 patients were back at their pre-injury levels with full return to running. There was a significant de-

crease in the pain score during activity (from 81.2  $\pm$  18.0 to 4.8  $\pm$  6.5). The calf muscle strength on the injured side had increased significantly, not differing much from the non-injured side.

Despite the small study numbers (n=15), the favourable results from the heavy-load eccentric calf muscle training were statistically significant.

### *Heavy-load Eccentric Calf Muscle Training*

Due to its effectiveness, a more detailed description of the training program is warranted.

Patients were instructed on how to do the eccentric training by physical therapists. There were also written instructions. Three sets of 15 repetitions were performed twice a day, seven days per week for 12 weeks.

From an upright body position and standing (facing a step) with all body weight on the forefoot and the ankle joint in plantar flexion, the calf muscle was loaded by having the patient lower the heel beneath the forefoot/step level. The calf muscle was loaded eccentrically only, no following concentric loading was done. Instead, the noninjured leg was used to get back to the start position. Patients were to continue the exercises even with pain, stopping only if the pain was disabling. When they were relatively pain free doing the exercise, they increased the load by adding weights.

### *Other Exercise Programs*

The results of this exercise program were superior to that of Angermann and Hovgaard.<sup>15</sup> Their graduated training program followed a period of heel elevation, massage and triceps surae stretching. The duration of the training program was until the patient was able to return to sports or a maximum of six months. The exercise program included heel-raises as in the Alfredson study. However, it was not specific in emphasising the eccentric phase of the heel-raise exercise, isokinetic calf muscle

strength was not measured, and there was no comparison or control group. However the Angermann study did have a longer-term follow-up of five years (range 33-72 months), whereas Alfredson had only a 12-week follow-up period.

### *Eccentric Exercise Training*

From the Alfredson study, some questions need to be answered. Why did these patients benefit significantly from their eccentric calf muscle training? What is the theoretical benefit of eccentric muscle training?

Research suggests that eccentric exercise may be of benefit in rehabilitation of musculoskeletal injuries.<sup>20</sup> During exercise, the maximum load is placed on a tendon during the eccentric phase, making it likely that injury to a tendon occurs during eccentric loading.<sup>5</sup> Eccentric loading promotes collagen formation throughout the tendon, thereby increasing the elastic and tensile properties of the tendon.<sup>20,23</sup> Remodelling of the tendon, whether healthy or pathological, as in tendinosis, is induced by eccentric loading. This reference also suggested that a combination of eccentric and concentric activations would be more effective. Concentric activations should be used prior to eccentric training, which should be started when the muscles improve their strength, due to the high tension development during eccentric activations. Also eccentric training at fast angular velocities compared with slow speed training was recommended, as slow speed eccentric loading generated excessive tension and regulatory demand. However, Alfredson<sup>5</sup> did not feel that eccentric exercise was velocity dependent and achieved excellent results with loading applied at slow speed. Alfredson is regularly performing follow-up ultrasounds on his study group in order to detect structural changes in the tendon related to eccentric training.

## Management of Chronic Achilles Tendinosis

### *Recommendations from Eccentric Loading*

From this study, a number of recommendations can be made for the management of chronic Achilles tendinosis.

Patients with long duration of symptoms (mean 18.3 months, range 3-100 months) responded well to the training program.

This suggests that it is still worthwhile persevering with conservative treatment for at least three months from the time of presentation before considering any surgical treatment. During these three months, the patient should undertake the prescribed exercise program described above. This recommendation is in contrast to other studies<sup>14-16</sup> where the longer the duration of Achilles tendinosis symptoms, the less favourable the response to conservative treatment, and surgery was recommended if no progress was being made after 3-6 months. The surgical studies stated that if surgery was not undertaken by the 3-6-month stage, then it was less likely that there would be a favourable response to surgery.

The training program is safe and easy to perform. It is cost effective, not requiring medication or intensive therapist input, and it allows the patient to be in active control of their treatment. The program also fits in well as a logical step in the rehabilitation ladder prior to the patient returning to the impact-loading of walking and running.

### **Surgical Treatment**

Surgery is needed in about 25% of cases of chronic painful conditions of the Achilles tendon - not specifically tendinosis.<sup>21</sup> Surgery is more likely for the older patient, longer duration of symptoms and the presence of tendinopathic changes.

The surgical technique usually involves a straight longitudinal skin incision medial to the Achilles tendon. The paratenon is incised. Any abnormal paratenon tissue is excised. A central longitudinal tenotomy then exposes any

abnormal tendon tissue which is excised. A side-to-side suture is then used to repair the area of excision.

The result from surgery is variable. Kvist<sup>21</sup> reported 20% of surgically treated patients requiring re-operation, with 3-5% forced to give up their athletic careers due to persistent debilitating pain. Johnston et al<sup>14</sup> reported that all 17 study patients who had soft tissue tenolysis and/or excision of degenerative tendon cysts were able to return to unrestricted activity after 31 weeks. Maffulli et al<sup>16</sup> performed surgical decompression of chronic central core lesions of the Achilles tendon. Their study group of 14 athletes had a long duration of symptoms, average 87 months. At follow-up, only five had a good result. They recommended that surgery be performed earlier.

Maffulli et al<sup>17</sup> in a different study performed percutaneous longitudinal tenotomy in runners. Multiple longitudinal incisions were made in the area of maximal swelling. This is thought to be beneficial in improving the local circulation of the tendon. However, only 17 of the 52 patients in the study had tendinosis demonstrated on ultrasound and the results do not specify the outcome of the tendinosis group.

The surgical studies recommend surgical treatment of chronic Achilles tendinopathy if symptoms persist after 3-6 months of conservative treatment, claiming that further delay in surgery results in a less favourable outcome. However as shown by Alfredson et al,<sup>5</sup> conservative treatment with eccentric calf muscle training was very effective for patients with an average duration of symptoms of 18 months. Bearing in mind the comparatively poorer results from surgery and the possible complications, even the more chronic cases of Achilles tendinosis should undergo at least three months of an eccentric calf muscle training program before proceeding to surgery.

### *Complications of Surgery*

The complication rate from surgery has been reported between 4.7 and 13%.<sup>1,7</sup> Skin edge necrosis, wound infection, haematoma, deep venous thrombosis and Achilles tendon rupture have occurred. Alfredson in another study<sup>22</sup> found that 12 months following surgery for chronic Achilles tendinosis, bone mineral density of the calcaneus was found to be 16.4% lower at the injured side.

### **Summary of Management Guidelines**

The aim of this review is to formulate a draft clinical practice guideline for the treatment of chronic Achilles tendinosis. The reason for this is that the condition is relatively common, yet clinical management of it is unsatisfactory and at times inappropriate. Unsatisfactory treatment results in chronicity of symptoms, disability, and less favourable outcomes. Inappropriate treatment includes persisting with some forms of physical therapies and anti-inflammatory medication when there is no proven benefit or evidence of inflammation. This also represents a waste of valuable resources and a risk of side effects from use of anti-inflammatory medication.

Most of the time chronic Achilles tendinosis can be diagnosed clinically. Chronic tendinopathy clinically correlates well with tendinosis demonstrated on ultrasonography, MRI and histopathological examination. Because of this, it is unnecessary and once again a waste of resources performing ultrasonography and MRI merely to diagnose chronic tendinosis. Imaging should be performed only if the condition has failed to respond after at least three months of eccentric calf muscle training and surgery is being contemplated. In tendinosis affecting the distal third of the tendon, imaging is useful to identify frequently associated retrocalcaneal bursitis which may respond to other forms of

## Management of Chronic Achilles Tendinosis

conservative treatment. Prior to surgery, ultrasonography is the most cost-effective form of imaging to localise the area of tendinosis and to detect any associated retrocalcaneal bursitis.

Some authors suggest ultrasonography and MRI to monitor healing response but Astrom<sup>10</sup> feels healing is best indicated by relief of pain. His results in healed tendons showed that abnormal imaging was compatible with excellent clinical results.

Tendinosis is not peculiar to the Achilles tendon, so an understanding of this condition will assist in the management of tendinopathies at other sites like the shoulder, elbow and patella. The principles of management of chronic Achilles tendinosis may apply equally to the other sites.

Formulation of clinical practice guidelines is limited by the quality of evidence-based clinical studies. Many of the studies are merely retrospective clinical observations. Some are prospective trials, but lack adequate control groups or the size of the study group is too small for any useful conclusions to be made.

The level of evidence of all the available studies in the references is either level III-3 or level IV.<sup>24</sup> Prospective randomised controlled clinical studies (providing level I evidence) are required to allow a better comparison of the various treatment methods. This will help in optimising outcome and resource utilisation.

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# Autologous Chondrocyte Implantation in the Management of Articular Cartilage Defects

*Mr Ian Henderson, Orthopaedic Surgeon, Director, Orthopaedic Research Department, St Vincent's & Mercy Private Hospital, Melbourne*

## Introduction

Tissue engineering has been described as the manipulation, manufacture or alteration of cells, tissues or organic substances for human implantation.

Autologous chondrocyte implantation (ACI) is a tissue engineering approach to the treatment of full thickness articular cartilage defects and is the first clinically proven application of tissue engineering in orthopaedics.

The structure of articular cartilage is well known. It consists of chondrocytes surrounded by a complex extra cellular matrix of type II/IX collagen, with large water retaining aggrecan molecules creating a unique structure ideally suited for the lining of articular surfaces. This structure is capable of withstanding significant forces and has regional variation in thickness and stiffness, according to functional requirements in the joint.

Articular cartilage has no blood supply, lymphatic drainage, or neural elements and articular chondrocytes are ineffective in responding to injury. Whilst there may be transient cell replication and local increased matrix synthesis in response to injury, at the margin of the lesion, there is no effective reparative process unless the subchondral bone is penetrated allowing an inflammatory response with cell recruitment from the marrow elements. This response is usually incomplete, fails to restore normal hyaline cartilage but results in the formation of fibrocartilage which fails as an articular surface in the long-term.<sup>1</sup>

Damage to articular cartilage of the knee is common, including the large area of loss caused by osteochondritis dissecans, which can lead to premature arthritis.<sup>1</sup>

Whilst elderly patients with low demand can be successfully treated symptomatically and functionally by joint replacement, there are many patients in the young and middle age groups for whom there is usually no

acceptable or reliable treatment for articular cartilage injury, resulting in inevitable deterioration and the onset of osteoarthritis.

Pharmacological agents for intraarticular injections, such as corticosteroid, hyaluronic acid and cross-linked hyaluronic acid (Synvisc®) have been studied; however, none of these has shown positive effect beyond short-term symptomatic improvement.

Techniques have been developed to attempt to initiate repair in articular cartilage; however, these are ineffectual in the long term and result in fibrocartilage formation only.

Pluripotential cell recruitment by microfracture appears to be effective in terms of pain relief in the short term, but produces fibrocartilage of limited durability.<sup>2,3</sup>

Local joint replacement using osteochondral grafts (mosaicplasty) has proven technically challenging with incomplete incorporation of grafts and subsequent replacement of surface hyaline cartilage by fibrocartilage of limited efficacy.

The preferred process is to regenerate articular cartilage using isolated autologous chondrocytes or whole tissue with chondrogenic potential, such as perichondrium or periosteum.<sup>4</sup> No long-term studies on periosteal grafting have been published to date and this procedure is often associated with complications such as graft hypertrophy, chondrification, and ossification.

## Autologous Chondrocyte Implantation

Autologous chondrocyte implantation (ACI) as a technique for regenerating articular cartilage in humans was developed from the early animal studies of Grande<sup>5</sup> and Brittberg,<sup>6</sup> which verified the efficacy of cultured chondrocytes in enhancing the repair of articular cartilage defects treated with periosteal grafting in rabbits.

Subsequently Brittberg and Peterson in 1994<sup>7</sup> and 1998<sup>8</sup> reported clinical

use in humans and confirmed clinical efficacy with follow up to nine years. Peterson demonstrated that isolated femoral condyle defects gave the best results with less success for osteochondritis dissecans and patients with lesions associated with ACL rupture and subsequent reconstruction. There was no evidence of deterioration with time and improved results were seen with patella defects when alignment was corrected.

Subsequently Peterson reported the clinical results of 59 ACI patients at the 1997 AAOS meeting.<sup>9</sup> Most of these patients had failed prior conventional surgery of either debridement or microfracture. The ACI patient results were either excellent or good for 95% of femoral condyle defects, 89% of osteochondritis dissecans and 75% of femoral condyle defects in association with ACL reconstruction. Biopsy revealed the presence of "hyaline-like" repair and biomechanical assessment using the arthroscopic indentometer of Lyyra (Artskan 1000)<sup>10</sup> demonstrated that the stiffness of hyaline repair (2.77N) was comparable to that of normal articular cartilage (3.08N) and substantially greater than that of fibrous repair (1.23N).

These patients were treated by what has become known as the Peterson Technique. This consists of arthroscopic harvest of articular cartilage from non-weightbearing areas of the knee or the periphery of the lesion to be treated, release and expansion of chondrocytes in the laboratory, reimplantation of free cells at arthrotomy after preparation of the lesion and coverage using periosteum harvested from the proximal tibia or distal femur. The periosteum is sutured in place and sealed with fibrin adhesive. Cell populations appropriate to the volume of the lesion are cultured and instilled beneath the periosteal patch in a volume of medium acceptable to the lesion and containing the optimum number of cells (see Figs. 1a, 1b, and 1c).



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Figure 1(a). Native lesion.

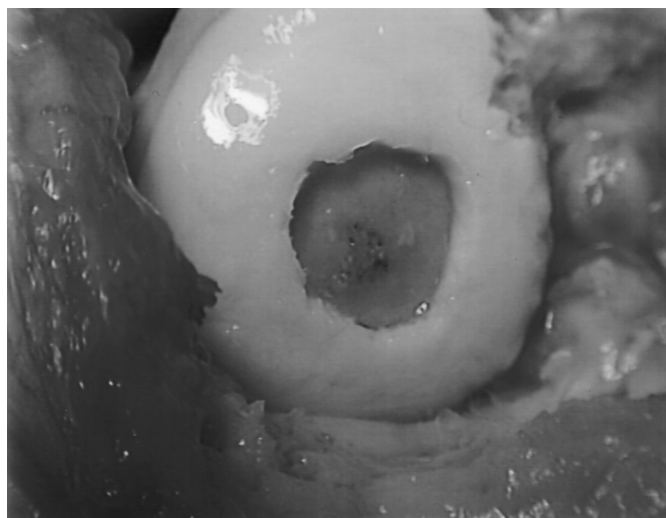


Figure 1(b). Prepared lesion.

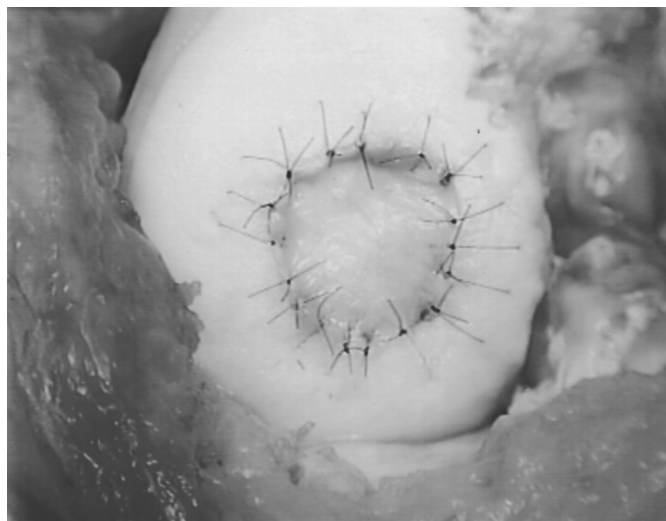


Figure 1(c). Sutured patch.

Following reimplantation, the limb is rested for 24 hours to allow adherence of the cells, followed by a progressive rehabilitation program beginning with continuous passive motion and progressing to ambulation with regulated restricted weightbearing and a progressive activity program over 12 months. Patients with tibiofemoral lesions must remain non-weightbearing for six weeks, followed by a six-week period of protected weightbearing in an "un-loading" brace. At three months normal daily activities can be resumed; however, high demand activities are restricted for six months. Return to sport is permitted at 12 months. Patients with patellofemoral lesions can weightbear as tolerated in an extension splint, but must avoid bent knee activity for three months. The rehabilitation program requires carefully supervised physiotherapy.<sup>11</sup>

While patients largely self-select by presentation the procedure is recommended for patients between the ages of 15 and 55 who have a well-circumscribed lesion or lesions in an otherwise essentially normal joint, with surrounding articular cartilage suitable for suture of the periosteal patch. Full thickness articular cartilage lesions greater than 1 cm<sup>2</sup> on the femoral condyle, trochlea or patella where subchondral bone is intact are most suitable. Multiple lesions are acceptable and femoral condyle and trochlea lesions commonly treated at the same time. Patellofemoral lesions are acceptable and "Kissing" lesions are acceptable, providing the opposing surface defect is no greater than grade II Outerbridge. Tibial plateau lesions provide technical difficulties at this time due to access restrictions for periosteal patch suture. However, some anterior lesions have been treated successfully with or without associated ligamentous release.

The procedure is contraindicated in patients with generalised osteoarthritis or inflammatory disease, crystal disease, or chondrocalcinosis. Angular deformity resulting in medial or lateral compartment overload must be corrected and patella femoral dysfunction or instability must be addressed. Morbid obesity, defined as >150% ideal body weight, is a contraindication.

The clinical use of ACI has been developed in several centres in Europe and the United States. There are now several companies actively developing a commercial product, including Verigen Tissue Sciences in Denmark, Codon in German, Educell in Slovenia and Genzyme Corporation (Carticel®), Advanced Tissue Sciences, Integre Life Sciences Corp. and Lifecell Corp. in the USA.

### ACI Clinical Results

Clinical data are available from both European and North American patient series. Those in the North American studies have been assessed using a modified Cincinnati Knee Score with patient and clinician assessment. The

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European experience has assessment using modified Cincinnati Knee Score and four other validated clinical scoring systems (Lyschholm, Wallgren, Tegner, Brittberg.) The European results have also included arthroscopic "second look", biopsy for histology, biochemical analysis and biomechanical assessment using indentometry.<sup>10</sup>

### Swedish Experience May 2000<sup>11</sup>

The clinical arthroscopic and histological results were reported from follow up of 110 implantations in the first 101 patients treated in Sweden for full-thickness chondral defects of the knee ranging from 1.5 cm<sup>2</sup> to 12 cm<sup>2</sup> following the introduction of ACI in 1987. Of this group 93 patients were clinically evaluated, 65 underwent arthroscopic assessment and 37 of these agreed to core biopsy of repair tissue and histological assessment.

The assessment of ACI in the study consisted of clinical outcome quantified by the five validated grading scales listed, macroscopic characteristics of the repair tissue assessed at arthroscopy and histological examination of the repair tissue from core biopsies.

Histological assessment was performed on objective criteria by three pathologists blinded to treatment outcomes, with the majority opinion used to classify the repair tissue. Seventeen out of 21 (81%) of all femoral condyle biopsies demonstrated hyaline-like cartilage repair. This group included solitary condyle lesions as well as OCD and multiple lesions. The results of histologic analysis showed a positive correlation between "hyaline-like" repair tissue (defined as hyaline matrix containing morphologically normal chondrocytes, staining positive for type II collagen and lacking a fibrous component) and good to excellent clinical results. Conversely, only fibrous tissue was seen in patients who suffered graft failures.

Hypertrophy of the repair site produced clinical symptoms in seven cases (6%). This was treated by arthroscopic debridement which did not affect the final outcome.

Peterson subsequently presented two year results of 213 ACI patients in March 2000.<sup>12</sup> The results on modified Cincinnati Knee Score were good/excellent for 90% of femoral condyle lesions, 74% of femoral condyle + ACL, 84% of OCD, 69% of patella, 58% of trochlea and 75% multiple lesion. Patella lesions were dependent upon normal mechanics and the results of trochlea lesions were influenced by size of the lesion.

Forty-six repair sites were inspected at "second look" arthroscopy and evaluated on the ICRS scale, allowing four points each for the three parameters, level with surrounding cartilage, complete integration and arthroscopically normal appearance of the surface. The mean score was 10.5 out of a possible 12.

Nineteen repair site biopsies were carried out with histologic hyaline repair tissue in 74% of patients, with a strong correlation (0.73) between hyaline repair and good/excellent clinical rating.

Indentometry was carried out on 14 repair sites demonstrating a stiffness of 3.1N in normal cartilage, 2.7N in hyaline-like repair tissue and 1.2N in fibrous repairs. More importantly, 31 of the patients graded as good/excellent were re-evaluated at 5-10 years, average 7.5 years, and all retain good/excellent grade.

### ACI and OCD

Peterson subsequently reported in May 2001<sup>13</sup> the 2-10 year results for 42 patients with OCD treated with ACI. 35 of these patients had an average of 3.2 failed prior surgical procedures, the average duration of symptoms was 7.5 years with average defect size of 5.7 cm<sup>2</sup>. Thirty-three patients were assessed clinically and 10 arthro-

scopically. Clinical assessment showed a modified Cincinnati Knee Score of good/excellent in 28 patients (86%) and ICRS score for degree of defect repair at arthroscopy of 10.2/12. Two patients were classified as failures.

### North American Experience February 2001

Data from the International Cartilage Repair Registry (ICRR) presented at the AAOS February 2001<sup>14</sup> now include 1300 patients. Of these 42 are at 60 months follow up and data for 36 of these were available.

The patients had been assessed on four criteria without arthroscopy or biopsy. Patient and clinician evaluation using modified Cincinnati Knee Score with pre- and post-operative assessments, assessment of patient's symptoms and knee examination.

The 36 patients with five-year follow up, on MCKS showed improvement on clinician and patient evaluation of 3.1 to 7.2, and 3 to 6.8 respectively. There was no change in these patients between 12 and 60 months follow up, indicating that the initial improvement was maintained for at least five years.

Patient symptoms showed dramatic improvement in pain score by an average of 3.2 points ( $P < 0.001$ ), swelling by an average of 3.3 points ( $P < 0.001$ ), and giving way by an average of 2.7 points ( $P < 0.001$ ). Again, no change in patient symptoms was noted between 12 and 60 months.

Clinical examination showed decrease in joint line tenderness from 81% to 30% and effusion from 78% to 11% at 60 months. Treatment failure, indicated by a further procedure, occurred in 3% at five years and 94% of cases were considered successful. A small number of these did require further treatment, but this usually consisted of a minor arthroscopic procedure.

Articular cartilage surgery in the previous five years was noted in 60%, with half of these involving abrasion,

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drilling or microfracture procedures, previously considered the best treatments available.

The most common adverse events (<2%) were adhesion or overgrowth of periosteal tissue, or loosening of the graft complex.

This report was not a controlled study, had no arthroscopic evaluation or histological assessment, and the mechanical properties were not assessed by indentometry.

#### ACI Compared with Non-Implanted Patients

A three year prospective controlled study of ACI cases versus 86 cases of debridement only with no cell implantation for full thickness articular cartilage lesions of femoral condyle or trochlea was presented at the Orthopaedic Research Society 2001 meeting by Browne.<sup>11</sup> Evaluation by modified Cincinnati Knee Score showed improvement in 89% of ACI patients but only 55% of debridement only cases. Overall clinician score at follow up was 7.6 for ACI versus 5.8 for debridement only. This was statistically significant ( $P < 0.001$ ). Overall modified Cincinnati Knee Score was unchanged in ACI cases by lesion size, but decreased with increasing lesion size in debridement only patients.

This is an important study as it demonstrated significant statistical improvement in patients undergoing autologous chondrocyte implantation when compared to lesion debridement only.

There is now an ever-increasing volume of data on outcomes of ACI in the knee showing clinical effectiveness, but more importantly an increasing volume of data on objective assessment at arthroscopy including histological and biomechanical evaluation of the repair tissue.<sup>11,12,14-21,28-32</sup>

MRI has been demonstrated as effective in determining the filling of the defect.<sup>24-27</sup>

A correlation between histological structure of articular cartilage tissue in the knee and indentation stiffness has been demonstrated and can be recorded using the indentometer of Lyyra.<sup>9,10,30</sup> The biomechanical evaluation of normal articular cartilage in the human knee has been carried out<sup>33</sup> and these data will be essential in the assessment of the result of articular cartilage repair. The geographic variation of articular cartilage stiffness in the knee ranges from approximately 4N at the femoral condyle to 1.2N at the femoral trochlea. Therefore the regional normal readings are required to determine the efficacy of articular cartilage repair.

Although autologous chondrocyte implantation has developed an accepted place in clinical practice in Europe and North America in the last 12 years, the procedure has not been available in Australia until now, as there has not been a laboratory licensed by the Therapeutic Goods Administration (TGA) to produce autologous chondrocytes as living tissue for human implantation.

The development of the Mercy Tissue Engineering Laboratory by the Sisters of Mercy and Mercy Health and Aged Care at Mercy Private Hospital, Melbourne, has now made available this treatment to Australian patients, and the cell product produced by the laboratory (Cartogen®) is now listed on the Prosthesis Benefit Schedule. It is therefore available at no cost to privately insured patients, patients covered by third party insurers such as WorkCover, DVA and TAC, etc., and is also available to public patients in public hospitals in the same manner as any other prosthesis.

The Mercy Tissue Engineering Laboratory is the first human use living implant laboratory licensed by the TGA and the product is the first approved by the Commonwealth Department of Health for inclusion on the Prosthesis Benefit Schedule, based on cost ben-

efit analysis.

Whilst the current application has been predominantly to the knee, extension of indications for ACI is occurring to other joints particularly the talar dome of the ankle.

Peterson,<sup>34</sup> Giannini,<sup>35</sup> and Cherubino<sup>36</sup> have all reported a small series of talar dome lesions with good results.

Current results indicate that autologous chondrocyte implantation using the Petersen Technique with periosteal patch retention of chondrocytes is clinically effective, reliable and safe. Yet, development will occur to improve techniques of cell delivery, including all arthroscopic reimplantation, improved methods of retention of chondrocytes in situ and improved bonding of regenerating tissue to surrounding normal articular cartilage and subchondral bone.

Cell placement and maintenance can be improved by the development of appropriate matrix scaffolds, cell carriers and adhesives using synthetic biodegradable polymers such as Dexon, Vicryl, Polydioxanone, Polylactic, and Polyglycolic acid, decalcifying matrix, hydroxylapatite, agar, methyl cellulose, carbon fibre, fibrin adhesive, and collagen fibre gels (type I and II).<sup>37</sup>

Articular cartilage regeneration can be enhanced by local and temporal delivery of growth factors and cytokines including fibroblast growth factor (FGF), transforming growth factor (TGF) and insulin growth factor (IGF). Synthetic matrixes for invitro tissue culture can be developed. Type I and II collagen gels allow satisfactory proliferation and simultaneously enhance bovine chondrocyte differentiation.

Adjunctive techniques (e.g., enzyme-aided digestion) are required to improve bonding of regenerated articular cartilage to adjacent cartilage and for the restoration of subchondral bone prior to articular cartilage regeneration.<sup>38,39</sup>

## Autologous Chondrocyte Implantation

There are future requirements for the establishment of rigorous clinical trials, preferably randomised, controlled and blinded, with inclusion of further assessment criteria using clinical, MRI, arthroscopic, histopathologic, and biomechanic techniques.

The basic science of ACI is well defined, the clinical application is now becoming accredited, and the nine year results are superior to alternative treatments for articular cartilage injury. There appears to be minimal adverse events or potential complications of this procedure. Clinical progress needs to continue to be assessed with the use of detailed outcomes databases.

Whilst articular cartilage regenerated by current techniques does not appear to exactly reproduce hyaline cartilage, the "hyaline-like" articular cartilage that is produced demonstrates satisfactory mechanical load characteristics with good function and demonstrated long-term durability.

Whilst current methods of application have limitations, this is the basis for future development, and the availability of autologous chondrocyte will enable progress in this field as tissue engineering application in orthopaedics develops.

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# The Stiff Painful Shoulder:

## Capsular Contracture Or Hyperactivity of Shoulder Musculature

*Greg Schneider, Southern Tablelands Specialist Manipulative Physiotherapist, Braidwood NSW*

**C**ontracture of the capsule of the shoulder is a recognised cause of gross movement restrictions of passive shoulder motion in all planes.<sup>1</sup> The fibrous contracture is most marked in the region of the subscapularis muscle and bursa,<sup>2,3</sup> causing lateral rotation of the shoulder to be the most restricted movement.<sup>4</sup> This condition with fibrous contracture is termed frozen shoulder.

Contracture and adhesions are not present in every case of clinically diagnosed frozen shoulder.<sup>5</sup> Misdiagnosis and confusion occur in cases resembling contracture. These patients may present with global restriction of shoulder mobility, but detailed examination reveals causes other than contracture.<sup>3</sup>

This study observed five patients with stiff painful shoulders diagnosed as frozen shoulders. The cases were separated into two groups on the basis of a subtle difference in the pattern of movement restrictions. There was also a difference between the groups in their response to cervical joint blockade. The outcomes form the basis for a research proposal.

### Method

Five patients with stiff painful shoulders were selected. Each patient had a global restriction of passive shoulder movements in all planes. Subtle differences in movement patterns were observed and the patients were identified as two groups.

Two patients were classified into Group I. The range of lateral rotation for both patients was less than two-thirds, or 60°. The range of medial rotation, measured by the hand behind back test (HBB) was also less than two-thirds. This two-thirds limit was set arbitrarily as the radial styloid of the patient's wrist reaching to the level of S1 on the vertebral column. This pattern of movements was identified as an equivalent rotation pattern (ERP). The measured ranges of motion for both

GROUP I ERP			GROUP II non-ERP		
Case	LR	MR	Case	LR	MR
1	10°	G.T.	1	35°	L3
2	50°	PSIS	2	(-5°)	L5
			3	0°	L5

**Table 1. Individual scores for lateral rotation (LR) and medial rotation (MR) for the two cases in Group I with equivalent rotation patterns (ERP), and the three cases in Group II with non-equivalent rotation patterns (non-ERP).**

patients are shown in Table 1.

Three patients were classified into Group II. The range of lateral rotation for each of these patients was also less than two-thirds. There was a subtle difference in the range of medial rotation in these Group II cases compared to those in Group I. The range of medial rotation was restricted, but it exceeded the two-thirds S1 limit. Each patient was able to reach his radial styloid cephalad beyond the S1 level. This restricted movement pattern was termed a non-equivalent rotation pattern (non-ERP) (Table 1).

Each of the five patients, two in Group I and three in Group II, was given an ipsilateral C5/6 zygapophysial joint block, of 10 ml of Bupivacaine under radiological image intensification. The ranges of lateral rotation and medial rotation for each patient were measured at two weeks and immediately preceding blockade. Measurements were repeated at three intervals after blockade.

### Results

The two patients in Group I had an improvement in the ranges of LR over 24 hours, but there was a decline to pre-blockade status by seven days, and these two patients failed to progress (Table 2). Subsequent arthroscopic investigation of these two cases determined that the cause of the movement restriction of the shoulder was contracture of the joint capsule.

Each patient in Group II, with non-equivalent restrictions, gained a significant improvement in the range of LR over 24 hours, and maintained the improvement at seven days.

The individual scores at seven days show gains of 45°, 70°, and 40° (Table 3).

The three patients in Group II became pain free and recovered normal shoulder mobility within one month. This was a highly significant outcome. Each of the three cases had a nine-month history of considerable pain and disability, unresolving, and had

INTERVAL	CASE 1	CASE 2
PRE	10	50
2 HR	60	60
24 HR	60	70
7 DAYS	10	65

**Table 2. Individual lateral rotation scores for the two cases in Group I with confirmed contracture. The scores were recorded immediately preceding blockade (PRE) and at 2 hours, 24 hours, and 7 days post cervical blockade.**

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INTERVAL	CASE 1	CASE 2	CASE 3
PRE	35	(-)5	0
2 HR	75	55	40
24 HR	75	60	50
7 DAYS	80	65	40
GAIN	45	70	40

**Table 3. Individual lateral rotation scores for the three cases in Group II with non-equivalent rotation patterns (non-ERP). The scores were recorded immediately preceding blockade (PRE), and at 2 hours, 24 hours, and 7 days post cervical blockade. Gains in the ranges of LR of 45°, 70°, and 40° were recorded for cases 1, 2, and 3 respectively.**

been told to wait two years for recovery.

### Discussion

The two Group I patients, with confirmed contracture, presented with equivalent restrictions (ERP) of medial and lateral rotation. Both cases failed to respond to cervical blockade.

The three Group II patients, who presented with non-equivalent rotation patterns (non-ERP) had a good response to cervical blockade.

These outcomes suggest that factors other than the capsule contributed to the movement restrictions seen in the Group II patients with non-ERP, and also suggest a relationship with the cervical spine. The limitations of this introductory study permit no conclusions. The findings can be discussed only in a theoretical sense.

Some research findings are pertinent to this discussion. The cervical ZJs are capable of producing pain and somatic referred pain.<sup>6</sup> C5/6 pain extends from the base of the neck, across the supraspinous fossa of the scapula, to the region of the deltoid muscle.<sup>6</sup> Anaesthetisation of the C5/6 ZJ relieved patients of lower neck pain and referred pain to the shoulder.<sup>7</sup> The actual prevalence of cervical ZJ point could be as high as 63%.<sup>8</sup> Spasm in diverse shoulder muscles has been elicited by stimulation of cervical ZJs.<sup>9</sup> Pain arising from lumbar ZJs can limit

SLR by producing hamstring spasm.<sup>10</sup> If these principles are applied to the findings in the present study, a hypothetical model can be proposed.

Through the mechanism of convergence, noxious stimuli from a symptomatic C5/6 ZJ may be interpreted as pain arising from the shoulder region.<sup>11</sup>

The motor neurone pools from the shoulder muscles acting across the scapulo-humeral joint, and supplied by the C5 and C6 cervical nerves may be facilitated, producing hyperactivity or spasm, which could restrict shoulder movement. Tonic spasm of subscapularis would limit LR.<sup>12</sup>

Anaesthetisation of the C5/6 ZJ decreases of blocks discharges from the joint afferents.

This blockade relieves the somatic referred pain to the shoulder, allows relaxation of the subscapularis activity, permitting an increase in LR of the shoulder.

This hypothetical proposal has a weak link. It invokes a model for a neuromuscular mechanism for movement restriction at the shoulder which has not yet been validated.

### Conclusion

The findings from this observational study of a small sample of patients prompt further research, which will be undertaken within the following format.

A sample of patients with global re-

strictions of shoulder movement will be selected. Each case will be classified according to movement restrictions. The patients will be differentiated into two categories.

- Those patients presenting with an equivalent rotation pattern, having both LR and MR restricted to less than two-thirds, with LR being the more restricted.
- Those patients identified with a non-equivalent rotation pattern with LR limited to two-thirds, and MR also being restricted but exceeding two-thirds.

Each case will receive an arthroscopic investigation of the shoulder joint.

Subsequently each case will be given an ipsilateral C5/6 medial branch block with the ranges of shoulder movement assessed before, and at set intervals after blockade.

Two hypotheses will be tested using a two by two contingency table.

H1 a non-equivalent pattern signals negative capsule involvement.

H2 non-equivalent cases respond to C5/6 blockade.

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# Thoughts on Soft Tissue Pain

*Dr Peter Jackson, Musculoskeletal Physician, Brisbane\**

## Introduction

It doesn't take long for naive musculoskeletal physicians to realise that gapping spinal facet joints, as important as this is in management of acute spinal pain or for providing a window of opportunity for managing chronic pain, is not the solution for most patients who seek care. They gradually become aware of the huge problem that they have been taught nothing about, namely, soft tissue pain. We and our other medical colleagues are standing knee deep in this problem. We read about it in medicolegal reports and hear other medical practitioners dismiss it in vaguely disparaging terms.

So, what is soft tissue pain? Well, two different but overlapping conditions are the source of soft tissue pain. These are the fibromyalgia syndrome and myofascial syndromes of individual or regional groups of muscles.

## Nomenclature

Soft tissue pain, fibromyalgia, and myofascial pain aren't very good terms because they are vague, a bit old-fashioned, and don't appeal to the scientifically minded practitioner. Possibly fibromyalgia should be called central sensitisation. Myofascial pain should be called functional muscle nociceptor sensitisation syndrome. Until better names come along, however, I shall use these older terms. It's a pity that just as these names are gaining common currency they be deleted from the medical lexicon.

A few years ago I was talking to a *Medecin sans Frontieres* doctor in Morocco about fibromyalgia. He told me that in Africa fibromyalgia is called "There Disease". When an African tribesman is asked, "where do you hurt" he would indicate, "there, there, and there," pointing to unrelated areas of the body.

A recent survey<sup>1</sup> of American Pain Society members, who regularly care for patients with body pain, was con-

ducted to assess current opinion about the myofascial pain syndrome (MPS). The objectives of the study were to determine whether MPS was a legitimate disorder, whether it was distinct from the fibromyalgia syndrome (FMS), and whether the participants believed that a limited core of symptoms or signs could readily characterize it. Of the 403 respondents, 88.5% believed that MPS was a legitimate diagnosis. Eighty-one percent believed that MPS and FMS are distinct from each other. The survey further indicated that the signs and symptoms essential to the diagnosis of MPS were a regional location pattern of symptoms, the presence of trigger points (TrP), and a normal neurological examination. These findings imply that the study respondents were properly distinguishing the specific condition MPS, defined by trigger points, from the improper generic usage of the term "myofascial pain," which has come to loosely mean all "soft tissue pain" conditions. To codify avoidance of this common form confusion, it has been recommended that the term "soft tissue pain" (STP) be adopted as the relevant generic term.

## Discussion

Soft tissue pain has had numerous descriptions over history that were a reflection of the time and culture. Rheumatism, cellulitis, fibrositis, and myodysneuria are a few examples.

Currently, the myofascial pain syndrome component of soft tissue pain can be considered a regional pain syndrome and a true neuromuscular disorder.

Myofascial pain syndrome can be tested by specific needle EMG, surface EMG, ultrasound, and algometry.

Modern understanding of myofascial pain began in the 1930s when Professor John Kellgren, working in London, investigated the effects of stimulating nociceptors in fascia and its subtending muscle by mechanical and chemical means after first anaesthetising the

overlying skin and subcutaneous tissue. He found that mechanical and chemical stimulation of fascial nociceptors caused well-defined localised pain referred just distally to the area of stimulation. Mechanically stimulating subtending muscle nociceptors caused slight but more diffuse pain, which was greatly enhanced in terms of perception and distal referral by chemical stimulation.

Kellgren found that chemically stimulating muscle nociceptors caused both localised and more diffuse referred pain and certain of these muscles referred pain to joints. The *infraspinatus* caused deep shoulder joint pain whereas the *vastus medialis* caused (the enigmatic) knee pain. Furthermore he was able to induce visceral pain. When he injected the *multifidus* at L1/2 his subject felt testicular pain. Kellgren believed that this pain was referred via intramuscular septa, as he was unaware of substance P, sensitisation of dorsal horn transmission neurones, and expansion of receptive fields.

The medical profession, not being able to verify these observations with laboratory tests and x-rays, ignored these observations and felt that psychogenic rheumatism was the most appropriate rubric. This time was also the "decade of the disc" under the influence of Cyriax and Mixter and Barr, which diverted attention to bone and joints as the source of musculoskeletal pain.

An Australian, Michael Kelly, was one of the insightful few to acknowledge Kellgren's observations and made contributions to understanding myofascial pain until his last paper in 1962. I like to think of this as the first flowering of musculoskeletal medicine in Australia, the second episode, starting with Murtagh and Kenna in 1985. In New Zealand, Professor Barry Tait and the organised medical profession's battle with the ACC to prevent lay practitioners becoming preferred pro-

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viders of musculoskeletal medicine were similar influential events.

Concurrently, Janet Travell, a sufferer of shoulder muscle trigger points found similar problems in bed-ridden tuberculosis and chest pain patients which she and her cardiologist colleague, Rinzler, treated with procaine injections and/or spraying the overlying skin with ethyl chloride spray (to stimulate cutaneous temperature receptors) and passively stretching the underlying muscle. They simply described the problem as "tender spots" in muscles.

It was a professor of orthopaedic surgery from Iowa who nominated tender points in muscles that referred pain as (the gluteal) myofascial syndrome around 1940.

Later, Travell collaborated with Simons, a person with postgraduate electromyographic training, in progressing the understanding of the pathophysiology of myofascial TrP pain.

Studies indicate that the prevalence of TrP pain in selected patient populations is quite common. The problem in a general medical population has been found to be 30%, in a pain medicine centre 93%, in a head and neck pain clinic 55% and in a lumbogluteal orthopaedic clinic 21%.

It is believed that direct stimuli by acute or chronic muscle overload, radiculopathy and trauma stimulates group 4 (similar to cutaneous C fibres) muscle nociceptors to produce an afferent drive into the dorsal horn convergent transmission neurones. These neurones have active and inactive synapses connected to primary afferent neurones. When substance P is released at the active or open synapses and sensitises the cell wall of the convergent transmission neurone the inactive or silent synapses open up, which results in the development of new receptive fields in distal muscles. The exception is in the neck where the receptive fields are proximal.

It is possible that the dorsal horn

neurones and the myofascial cells developed from common embryological stem cells. This could explain why the convergent transmission neurones "see" pain in distal muscles. Furthermore there can be a vague myotomal distribution of pain evoked from trigger points. For example, scalene trigger points can produce referred pain similar to C6/7 radiculopathy or gluteus minimus anterior trigger points can cause referred pain similar to L5 radiculopathy.

### Needle Electromyography

Various studies in humans and rabbits have confirmed low amplitude "noise" activity that is highly characteristic of myofascial trigger points but not pathognomonic. When this "noise" is associated with high amplitude spikes it is a strongly confirmatory finding.

### Ultrasound

Two researchers have visualised a local twitch response in taut band fibres with the use of high-resolution ultrasound. In one study the twitch was elicited by needle penetration of a trigger point in a taut band of an infraspinatus muscle. The transient contraction coincided with the patient's verbal report that he felt his typical pain and experienced a referred pain to his shoulder and arm.

### Surface Electromyography

Trigger points are associated with three disturbances of muscle function, which can be assessed by surface electromyography.

1. Increased responsiveness
2. Delayed relaxation
3. Increased fatigueability.

In addition, trigger points can produce referred spasm (induced muscle activity) and referred inhibition in other muscles via central reflex mechanisms. This observation supports earlier observations by Janda of a predictable stereotypical response of muscles to nociceptor traffic with either tightness

or inhibition.

Spasm or inhibition can be referred to other muscles independent of pain referral and this can be identified with surface electromyography. A trigger point in the infraspinatus can strongly inhibit the anterior deltoid during shoulder flexion, which can nevertheless be normally recruited during shoulder abduction. Another common example is active trigger points in the quadratus lumborum causing neurological inhibition of gluteal muscles. Surface electromyography indicated a return to normal function after the trigger points were deactivated and can facilitate retraining by biofeedback.

### Algometry

Algometry does not indicate the cause of tenderness, does not provide an absolute value, requires great palpatory skill to position the footplate over the point of maximal tenderness, and requires a standardised rate of application because fibromyalgia patients tend to report pain at less pressure.

Algometry helps with acquiring information about pain thresholds of active and latent trigger points and normal muscles. There is considerable overlap of these three types of pain threshold measurements.

### Interrater Reliability

Studies of interrater reliability for the myofascial pain syndrome component of soft tissue pain have been poor. These need to be deconstructed to provide a more insightful view. The first study by Wolfe et al involved four very experienced examiners but who were self taught, i.e., not formally trained. They had no chance to agree on a technique for examining trigger points and in fact the design of the study prevented them from discussing their techniques prior to the study.

The second study by Nice et al involved examination of three sites in the thoracolumbar paraspinal muscles of 50 patients with low back pain by 12

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experienced physiotherapists. Before the patient examination, a practice session was held to allow the therapists to practise this method on each other until they felt capable. There was no evaluation of uniformity of technique. The examiners were experienced but inadequately trained.

The third study by Njoo et al reported the examination of the quadratus lumborum and gluteus medius by two examiners. An experienced general practitioner trained four medical students for three months. From the pool of the trainer general practitioner and his students, two were chosen to perform the examination. Essentially, four of the five examiners were well trained but inexperienced. The kappa scores were better but not good.

Gerwin et al conducted a study with four experienced examiners under the assumption that they employed the same examination techniques. They achieved the same poor results. They undertook a second study but this time participated in a three-hour training session and achieved statistical agreement before proceeding. They then repeated the study and achieved good kappa scores (.44-.88). A more recent well-designed study of headache<sup>2</sup> achieved kappa scores of .74-1.0.

Palpating for myofascial trigger points requires the same skill as listening for heart sounds or palpating foetal parts in a pregnant uterus. Could one imagine the kappa scores of cardiologists or obstetricians if they did not have a standardised training scheme?

To obtain reliable clinical results studies should employ both experienced and trained examiners who have been tested for interrater reliability before the study is conducted. The necessary skill can be learned and Friction, in a diagnostic study of masticatory myofascial pain, found that experienced raters were more reliable than inexperienced ones and concluded that palpatory findings are technique sensitive.

### Distinguishing Features

The female: male ratio for fibromyalgia sufferer years is 4-9: 1 and for myofascial pain syndrome 1: 1. The female predominance in fibromyalgia may reflect this gender's increasing likelihood to seek help and higher incidence of hypermobility. Fibromyalgia pain is widespread and general, including all four quadrants of the body plus the "fifth quadrant" which encompasses visceral pain, such as reflux oesophagitis, irritable bowel syndrome, irritable bladder syndrome, and dysmenorrhoea.

Pain of myofascial trigger points is regional. The difference between the two can be observed from a pain diagram. The markings made by the fibromyalgia are all over the body representation and even outside it in a diffuse non-dermatomal and non-myotomal way. Markings on a myofascial pain diagram are very specific, commonly just a line or a dot or a recognisable pain pattern. Fibromyalgia patients have widespread tenderness whilst TrP tenderness is very focal. The muscles of fibromyalgia patients are soft and doughy whilst those of MPS are tense and ropey with a restricted range of motion, and whereas the joints of fibromyalgia patients are distinguished by their hypermobility those of MPS are the reverse. Fibromyalgia patients are examined for the mandatory tender points whilst myofascial pain syndrome patients are examined for stereotypical trigger points that are predominantly in the middle of the muscle belly or entheses of muscles. These trigger points commonly have an immediate beneficial response to injections of local anaesthetic or local anaesthetic with glucose. Fibromyalgia patients frequently have an adverse response to focal injections and then a delayed and poorer response. Seventy-two per cent of these fibromyalgia patients have active trigger points, whilst 20% of myofascial pain syndrome patients

have comorbid fibromyalgia.

The concentration of substance P in the CSF of fibromyalgic patients is up to four times normal, although this is not so in myofascial pain syndrome patients. The former patients are equally tender in locations other than the mandated 18 tender points, whereas non-trigger point sites in myofascial pain patients have normal pain thresholds. They are abnormally tender only at sharply circumscribed TrP sites and specific sites of referred tenderness.

### The Neuromuscular Junction

Sensory nerves, autonomic nerves and blood vessels accompany motor nerve fibres. The junctional endplate can be localised by an EMG recording an initially negative deflection. Both sides of the endplate have positive first deflections. They are found in the mid-region of each muscle fibre.

Dysfunction of the neuromuscular junctions is now thought to be the cause of trigger points. High amplitude, high-speed EMGs using specialised needle techniques have revealed two significant components to the electrical activity. Intermittent and variable high amplitude spike potentials superimposed over a consistently present lower amplitude noise-like component has been identified at trigger points. These spike potentials had negative initial deflections. Muscle adjacent to trigger points is electrically silent. When a voluntary contraction is initiated, muscle fibres that are initially recruited nearly always exhibit the spontaneous electrical activity outlined above. These fibres are known as "Cinderella fibres". This would suggest the possibility of excitable motor neurones.

Excessive acetylcholine has been identified as the cause of this increased electrical activity, which has also been called "acetylcholine noise". It has been estimated that the noise-like electrical discharge is caused by 1000-fold increase in the rate of release of acetylcholine from a motor nerve ter-

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minal.

It has been proposed that clinically identified trigger points consist of multiple abnormal endplates evidencing spontaneous electrical activity and are scattered among uninvolved normal endplates.

Spikes occur when a sufficient number of acetylcholine packets are released and summate to depolarise the post junctional membrane sufficiently to initiate a propagated action potential in the muscle fibre.

### Distribution

The endplate zone of a muscle can be electrically identified. Trigger points in taut bands can be clinically identified. Using spontaneous electrical activity with or without spikes, it has been shown that dysfunctional endplates are four times more common where taut bands and the endplate zone intersect than elsewhere in the muscle. No dysfunctional endplates were observed in taut bands outside the endplate zone.

### Histopathology

Trigger points in dog muscle have been studied and have revealed isolated fibres and groups of enlarged round muscle fibres on cross sections. Longitudinal sections of the same TrP areas revealed segments of muscle fibres with extremely contracted sarcomeres, which showed a corresponding increase in diameter of the muscle fibre and abnormally decreased muscle fibre diameter on either side of it. The sarcomeres on either side of the contraction knot showed compensatory elongation and narrowing when compared to adjacent normal muscle fibres. Sarcomeres within a contraction knot are markedly shorter and wider than the in neighbouring normal muscle fibres which are free of contraction knots. Sarcolemma between contraction knots is empty of contractile elements. These "contraction knots" are scattered among normal muscle

fibres.

A trigger point can be considered a complex containing sarcolemmic contraction knots in a taut band, which exhibit electrically active loci. They are found at the motor point or endplate zone of the muscle. A taut band of muscle fibres extends from this central TrP to the attachment at each end of the involved fibre, which with sustained tension induces a localised enthesopathy, or attachment TrP. This is where patients point to their pain, for example, the lateral epicondyle or greater trochanter. They don't point to the middle of the wrist extensors or gluteal group.

If enthesopathic attachment trigger points alone are treated without treating the Central TrP, recurrence of symptoms can be expected. Attachment trigger points have not been studied properly and their nature is not yet understood and cannot be assumed to display similar characteristics as the central TrPs.

### The Pain System for Dummies

Nociceptive information travelling upstream from the dorsal horn is transmitted through two separate pathways that have evolved over time, the old spino-reticular and the new spino-thalamic tract. The former arises from lamina 5, 7, and 8 and carries noxiously generated information arriving at the dorsal horn by C and the similar group 4 sensory afferents which initially terminate in transmission neurones in lamina 1 and 2 then via polysynaptic pathways to the above-mentioned deep lamina. From here information ascends in the old tract until it reaches the reticular system and thence to higher centres such as the hypothalamus, thalamus, limbic system (pain tolerance), and frontal lobe (attitude to pain). The parasympathetic nervous system which controls rest, digestion, reproduction and "freezing" also originates in this area and I often wonder whether overstimulation of this

system results in the chronic fatigue seen after traumatic episodes.

Neurones in the reticular system have bifurcating axons, which project downwards to the ventral horn motor neurons and upwards to the higher centres. This may set up increased tone in muscles, which may activate the "Cinderella fibres" in which trigger points develop. There is evidence that trigger points develop in pre-existing tight bands rather than create them.

In the reticular formation the old tract stimulates the nuclei reticularis gigantocellularis and raphe magnus to produce serotonin, the locus coeruleus to produce noradrenaline and the periaqueductal grey to produce opioid peptides. These substances, via descending pathways, exert a calming effect on the dorsal horn transmission cells, preventing spontaneous activity and keeping unwanted receptive fields closed.

This old tract carries noxious information from myofascial tissue (syn = deep somatic tissue, fibromuscular tissue) but not from skin and subcutaneous tissues. It is this system (myofascial tissue, old spino-reticular tract and reticular formation) that is associated with the development of chronic pain. This may occur by primary failure or acute nociceptor implosion (whiplash) or chronic nociceptive traffic (internal disc derangement).

By contradistinction, the newly evolved pathway arises from laminae 1 and 5 where A-d fibres terminate, cross the cord without poly-synapsing and ascends directly to the thalamus and thence to the topographically organised somatosensory cortex to produce an "ah ah" experience. For example, "I have stubbed the tip of my left little toe and I think it's going to hurt (from my past experience)". This system, A-d cutaneous nociceptors, spino thalamic tract and somatosensory cortex is not involved in the process of chronic pain. Have you ever heard of a chronic pain syndrome resulting

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from a chronic skin ulcer?

### Conclusion

Soft tissue pain may be thought of as two different but associated conditions. The most challenging from a clinical point of view is the fibromyalgia syndrome. The other, which is much more satisfying to treat, is myofascial pain syndromes of muscles or groups of muscles.

Fibromyalgia may be seen as primary and secondary. Primary fibromyalgia occurs spontaneously without a history of physical or emotional trauma and may be associated with failure of the descending inhibitory pathway. There seems to be a genetic component to fibromyalgia. Secondary fibromyalgia appears to develop after sudden unexpected or chronic afferent nociceptor drive onto the dorsal horn from deep somatic tissues including myofascial tissue. This afferent drive seems to overpower the tonic inhibitory endogenous pain system. Probably the commonest cause of this afferent drive is neglected or untreated myofascial trigger points. Whilst there is no peripheral pathology in the fibromyalgia syndrome there is an increase in substance P in the interstitial spaces of the spinal cord and CSF. There may also well be cellular changes such as sprouting, remodelling, and apoptosis but who is going to perform a spinal tap or cord biopsy to find out?

Some people with fibromyalgia do respond to physical medicine techniques and it is probably the abolition of peripheral afferent drive from a myofascial trigger point that is the cause of this improvement. Otherwise one is left with trying to activate the descending inhibitory system by cognitive behavioural therapy (synonym = "Indahling"), small doses of tricyclic antidepressants to increase the endogenous noradrenaline and improve sleep and relying on aerobic exercises to stimulate activity in the "pleasure

fibres", the fast conducting Ab fibres. This has been described as "fast blocks slow".

Myofascial trigger points are satisfying to treat. One only needs to mechanically deactivate the Central trigger point and if one also treats the attachment trigger points, so much the better. The mechanical deactivation can occur with any means at ones disposal such as dry needles, wet needles, Beaver blades, medial branch burning devices and emerging technologies such as IDET. After all, nucleus fibrosis laminae are a form of fascia. There is no inflammation so that steroids are of no help and local anaesthetic, whether short, medium or long acting, cheap or expensive is more for the benefit of the doctor than the patient. Tight bands of myofascial tissue surrounding facet joints are most likely the cause of "minor intervertebral dysfunction", "the osteopathic lesion" and "the chiropractic lesion" resulting in the clinical observation of tenderness, asymmetry, reduced motion and increased tissue tension abnormality. Mobilisation with impulse, osteopathic muscle energy techniques and post-isometric relaxation techniques may have their effect by stretching these tight bands. For example, if the multifidie and short intersegmental fibres of the psoas are relieved, if only temporarily, the enigmatic "joint blockage" will vanish. If afferent drive from myofascial trigger points can be treated properly and promptly, thus preventing central sensitisation, the subsequent need for repetitive "bone cracking" and multidisciplinary pain clinic attendance may well be dramatically reduced.

### Resources

Mense S, Simons D, Russell IJ. *Muscle Pain: Understanding Its Nature, Diagnosis, and Treatment*. Lippincott Williams & Wilkins, 2001.

Baldry PE. *Myofascial Pain and Fibromyalgia Syndromes: A Clinical*

*Guide to Diagnosis and Management*. Churchill Livingstone, 2001.

### References

1. Harden RN, Bruehl SP, Gass S, et al. Signs and symptoms of the myofascial pain syndrome: A national survey of pain management providers. *Clin J Pain* 2000; 16: 64-72.
2. Marcus DA, Scharff L, Mercer S, Turk DC. Musculoskeletal Abnormalities in Chronic Headache: A Controlled Comparison of Headache Diagnostic Groups. *Headache* 1999; 39: 21-27.

\* Email: [jacksonp@bigpond.net.au](mailto:jacksonp@bigpond.net.au)

# Journal Abstracts

***This section aims to update the reader with some of the more significant musculoskeletal research published in the last year which is listed on the Medline and CINAHL databases.***

## HIP

**Fink MG, Kunsebeck H, Wipperman B, Gehrke A. Non-specific effects of traditional Chinese acupuncture in osteoarthritis of the hip. *Comp Ther Med* 2001; 9: 82-88.**

**Objectives.** The effectiveness of acupuncture treatment in patients with osteoarthritis of the hip was tested. **Design:** This is a prospective, randomized, controlled, patient- and investigator-blinded clinical trial.

**Patients and Setting.** The study was performed at a university department for physical medicine and rehabilitation. Sixty-seven patients were separated into two treatment groups.

**Interventions.** Group 1 (treatment) had traditional needle placement and manipulation, whereas in group 2 (control) needles were placed away from classic positions and not manipulated. In both groups needles were placed within the L2 to LS dermatomes. Outcome parameters were: pain (VAS), functional impairment (hip score), activity in daily life (ADL) and overall satisfaction before treatment, and 2 weeks and 2 months after treatment.

**Results.** For all parameters there was a significant improvement versus baseline in both groups 2 weeks and 2 months following treatment, but no significant difference between the two treatment groups.

**Conclusions.** We conclude from these results that needle placement in the area of the affected hip is associated with improvement in the symptoms of osteoarthritis. It appears to be less important to follow the rules of traditional acupuncture techniques.

## KNEE

**Matsui N, Kobayashi M. Application of MR imaging for internal derangement of the knee. *Seminars in Musculoskel Radiol* 2001; 5(2): 139-41.**

Magnetic resonance imaging is a non-invasive imaging modality with clear contrast and superior spatial resolution. These characteristics are especially useful for detecting pathology of the soft tissue of the knee joint, such as the menisci, ligaments and articular cartilage, which are difficult to diagnose using plain x-ray examination. MRI has become one of the first choice diagnostic modalities for the internal derangement of the knee joint, and is generally replacing some invasive arthrographic or arthroscopic examination. Pathology of the articular cartilage cannot yet be depicted clearly by MRI. We expect further development of the spacial resolution of MRI to make possible the detection of chondral lesions more clearly and precisely in the near future.

## SPINE

**Kasch H, Bach FW, Jensen TS. Handicap after acute whiplash injury. A 1-year prospective study of risk factors. *Neurol* 2001; 56: 1637-43.**

**Background.** Exposure to a whiplash injury implies a risk for development of chronic disability and handicap, with reported frequencies ranging from 0% to 50% in follow-up studies. The exact risk for development of chronic whiplash syndrome is not known.

**Objective.** To prospectively determine the sensitivity and specificity of five possible predictors for handicap following a whiplash injury.

**Methods.** In a 1-year prospective study of persons with acute whiplash injury (n = 141) and control subjects who had acute ankle distortion (n = 40), pain intensity, number of nonpainful neurologic complaints, cervical mobility, workload during extension and flexion of the neck, and results of psychometric assessment were recorded. The consecutively sampled injured persons were assessed with

structured and semistructured questionnaires, and underwent neurologic examination after 1 week and 1, 3, 6, and 12 months. After 3 to 4 years, participants with whiplash injury were questioned about legal issues.

**Results.** After 1 year, 11 (7.8%) persons with whiplash injury had not returned to usual level of activity or work. The best single estimator of handicap was the cervical range-of-motion test, which had a sensitivity of 73% and a specificity of 91% ( $p < 0.01$ , Cox regression analysis). Accuracy and specificity increased to 94% and 99% when combined with pain intensity and other complaints. This increase was gained at the expense of a reduced sensitivity. Initiation of lawsuit within first month after injury did not influence recovery.

**Conclusion.** The cervical range-of-motion test has a high sensitivity in prediction of handicap after acute whiplash injury. The value of cervical range-of-motion test is further improved by additional recording of symptoms.

## PAIN

**Brockhow T, Dillner A, Franke A, Resch KL. Analgesic effectiveness of subcutaneous carbon-dioxide insufflations as an adjunct treatment in patients with non-specific neck or low back pain. A pragmatic, open, randomized controlled trial. *Comp Ther Med* 2001; 9: 68-76.**

**Objectives.** To evaluate the analgesic effectiveness of subcutaneous carbon dioxide insufflations in addition to standard physical treatment in patients with non-specific neck or low back pain.

**Design.** A pragmatic, randomized controlled trial. **Setting:** Rehabilitation hospital inpatients. **Interventions:** Patients received either subcutaneous carbon dioxide insufflations (10 treatments) and standard physical treatment or standard physical treatment only. **Outcome measures:** Affective

pain perception (42-point scale), sensory pain perception (30-point scale), pain intensity (100 mm visual analogue scale).

**Results.** Between-groups differences were -2.2 [95% CI -5.2; +0.9] (affective pain perception), -1.2 [-3, 0; + 0.7] (sensory pain perception), and -6.5 [-14; + 1.0] (pain intensity) respectively in favour of subcutaneous carbon dioxide insufflations.

**Conclusions.** Subcutaneous carbon dioxide insufflations do not seem to be a worthwhile adjunct in the given setting of inpatient rehabilitation. Trials in a monotherapeutic setting, which aim more at the efficacy of subcutaneous carbon dioxide insufflations, might help to solve this issue.

**Espeland A, Baerheim A, Albrektsen G, et al. Patients views on importance and usefulness of plain radiography for low back pain. *Spine* 2001; 26(12): 1356-63.**

**Study Design.** Quantitative and qualitative cross-sectional interview study.

**Objectives.** To investigate how patients who are referred for plain radiography because of low back pain perceive the importance and usefulness of the examination.

**Summary of Background Data.** Up to 50% of plain radiography examinations for low back pain may be unnecessary based on clinical criteria. However, many patients have great confidence in these examinations. A further exploration of the patients' views may indicate how their needs can be met without unnecessary use of radiography.

**Methods.** Ninety-nine patients (65 women, 3 men) 14-91 years of age who were referred from Norwegian general practitioners for plain radiography of the lumbo-sacral spine were asked to rate the examination as slightly/fairly or very important (93 responded). Chi-squared tests were used

to evaluate differences in rating according to age, gender, clinical history, and clinical appropriateness of the examination, as determined by comparing information in the referral form with Norwegian (NR) and British (BR) recommendations for use of radiography. Each of the 99 patients also underwent a semistructured interview that was based on questions about importance, usefulness, and reasons for the radiography referral. Answers were categorised and described using a qualitative method (template analysis).

**Results.** Seventy-two percent (68 of 93) of patients rated radiography as very important. The proportion was higher for men than women (85% vs 65%,  $p = 0.04$ ), higher for those with worsening than those with improving/unchanged symptoms worsen (86% vs 65%,  $p = 0.03$ ), and higher for inappropriately than appropriately referred patients (NR: 76% vs 61%,  $p = 0.17$ ; BR: 81% vs 56%,  $p = 0.01$ ). The qualitative analysis showed that the patients related their views on the importance and usefulness of receiving radiography to seven different issues: symptoms and clinical history, information and advice (especially from health care providers), need for emotional support from the physician, need for certainty and reassurance, need for symptom explanation and diagnosis, reliability of radiography compared with clinical evaluation, and expected practical consequences of the radiological examination.

**Conclusions.** The finding that inappropriately referred patients tended to rate their radiography referral as more important than appropriately referred patients indicates that the patient's view may be a substantial barrier to appropriate use of radiography. The study identified seven issues underlying the patients' views on importance and usefulness of receiving radiography. Strategies to prevent unnecessary use of plain radiography for low

back pain that address these issues are suggested.

**Gottschalk A, Smith DS. New Concepts in Acute Pain Therapy: Pre-emptive Analgesia. *Am Fam Physician* 2001; 63: 1979-86.**

Pain, which is often inadequately treated, accompanies the more than 23 million surgical procedures performed each year and may persist long after tissue heals. Pre-emptive analgesia, an evolving clinical concept, involves the introduction of an analgesic regimen before the onset of noxious stimuli, with the goal of preventing sensitisation of the nervous system to subsequent stimuli that could amplify pain. Surgery offers the most promising setting for pre-emptive analgesia because the timing of noxious stimuli is known. When adequate drug doses are administered to appropriately selected patients before surgery, intravenous opiates, local anaesthetic infiltration, nerve block, subarachnoid block and epidural block offer benefits that can be observed as long as one year after surgery. The most effective pre-emptive analgesic regimens are those that are capable of limiting sensitisation of the nervous system throughout the entire peri-operative period.

**Leclaire R, Fortin L, Lambert R, et al. Radiofrequency Facet Joint Denervation in the Treatment of Low Back Pain: A Placebo-Controlled Clinical Trial to Assess Efficacy. *Spine* 2001; 26: 1411-17.**

**Study Design.** A prospective double-blind randomised controlled trial was performed.

**Objective.** To assess the efficacy of percutaneous radiofrequency articular facet denervation for low back pain.

## Journal Abstracts

**Summary of Background Data.** Uncontrolled observational studies in patients with low back pain have reported some benefits from the use of facet joint radiofrequency denervation. Because the efficacy of percutaneous radiofrequency had not been clearly shown in previous studies, a randomized controlled trial was conducted to assess the efficacy of the technique for improving functional disabilities and reducing pain.

**Methods.** For this study, 70 patients with low back pain lasting of more than 3 months duration and a good response after intraarticular facet injections under fluoroscopy were assigned randomly to receive percutaneous radiofrequency articular facet denervation under fluoroscopic guidance or the same procedure without effective denervation (sham therapy). The primary outcomes were functional disabilities, as assessed by the Oswestry and Roland-Morris scales, and pain indicated on a visual analog scale. Secondary outcomes included spinal mobility and strength.

**Results.** At 4 weeks, the Roland-Morris score had improved by a mean of 8.4% in the neurotomy group and 2.2% in the placebo group, showing a treatment effect of 6.2% ( $P = 0.05$ ). At 4 weeks, no significant treatment effect was reflected in the Oswestry score (0.6% change) or the visual analog pain score (4.2% change). At 12 weeks, neither functional disability, as assessed by the Roland-Morris scale (2.6% change) and Oswestry scale (1.9% change), nor the pain level, as assessed by the visual analog scale (-7.6% change), showed any treatment effect.

**Conclusions.** Although radiofrequency facet joint denervation may provide some short-term improvement in functional disability among patients with chronic low back pain, the efficacy of this treatment has not been established.

**Comment.** It is patently obvious that the authors of this article and Dr Deyo (Point of View) have not read the AFMM guidelines on lumbar medial branch blocks. If they had they would have soon realised that this study was a waste of time due to the use of intra-articular facet injections to predict response to radiofrequency ablation (RFA) of the medial branch. The conclusions that the authors and Deyo make from the study are thus erroneous and just perpetuate misinformation about this useful (in the right hands) procedure. These authors need to read Dreyfuss' article on radiofrequency denervation to understand correct methodology, i.e., using controlled medial branch blocks before considering RFA. - Dr Scott Masters



# FIMM Update: Thirteenth Triennial International Congress, Chicago, USA

*Dr Ron Palmer, Vice-President, FIMM*

The thirteenth International Congress of the Federation of Manual/Musculoskeletal Medicine was held in Chicago, USA, on July 23-27, 2001. The title for the conference was Integrative Manual Medicine. The meeting was held at the Chicago Marriott Downtown Hotel, a massive structure of 45 levels. The congress was sponsored by the Kirkville College of Osteopathic Medicine, the University of Wisconsin Medical School in cooperation with the American Academy of Osteopathy (AAO) and the American Association of Orthopaedic Medicine (AAOM). Congress chairman was Dr Michael Kuchera.

The International Federation of Manual/Musculoskeletal Medicine (FIMM) is an organisation comprising 26 member nations which have a common goal in advancing scientific and educational awareness and training. Via select multinational committees, it aims to establish an International Collegium for Musculoskeletal Medicine. While most aims of the member national organisations are reasonably uniform, there are some philosophical and education differences that separate a small number of the member countries. One of the aims of FIMM international meetings is to work closely together and bridge the current gulfs. The Chicago congress was most successful in narrowing many of these ideological differences. Attendance at the congress was similar in size to the last triennial congress held at Surfers' Paradise, Australia, in 1998.

Advances in technology, both medical and communication, together with a shrinking global base, lend support to the concept of international cooperation and a single organisational body to overview the advance of musculoskeletal medicine. This international environment of cooperation is conducive to expanding the evidence base for this aspect of medicine and will serve to raise the standards of the basic skill core and ultimately patient care. As

Michael Kuchera said, "This congress is about *you and your patients*. Integrative Manual Medicine is about sharing the safe and effective application of manual medicine techniques integrated according to the most current evidence base and clinical experience of physicians dedicated to maximizing health and the function of each individual."

The format of the conference was fundamentally formal lectures in all the morning sessions and practical workshops in the afternoon periods. The afternoon workshops were of about two hours' duration and were repeated. There were also pre- as well as post-conference workshops. Lecture and workshop duties were evenly divided between MDs and DOs (doctors of osteopathic medicine). Australian musculoskeletal medicine was well represented with contributions by Norm Broadhurst, Phillip Watson, Michael Yelland, and myself. There were nine Australians at the conference, including AAMM Past President Dr Conrad Winer.

The standard of paper presentation was somewhat mixed. There were many excellent papers and unfortunately several that were not up to the standard that should have been expected from such an illustrious presentation group. All official FIMM presenters performed well and the scientific input from Stefan Blomberg and Jacob Patijn was excellent – in fact, a highlight of the conference. It was my opinion that the standard of research work is advancing well within our group and the evidence-based approach to patient management is also progressing in a favourable forward direction. There are still some differences in management between MDs and DOs and, although the differences were obvious, there does appear to be a narrowing of the different philosophies. The "total" patient approach was emphasised by the osteopathic clinicians and personally I have

no objection to this overall approach. Simply, treat the patient as a unit rather than look at only a single presenting condition. On this theme, I was most impressed by a workshop given by Robert Irvin and Michael Kuchera on Postural Balance Strategies using Orthotics. This workshop approached many of the anatomical changes that will occur when there is an overall body imbalance produced by foot changes. It was singularly the most constructive and informative lecture I have attended on the use and function of orthotic devices. Bob Irvine will have a textbook published on this subject next year. If the book is as good as his presentation then I fully recommend it.

At the general assembly (AGM) it was agreed that the next triennial congress should be held at Bratislava, Slovakia, in 2004. The next annual general assembly will be held in Kuopio, Finland, on 6-7 September 2002. All executive positions will be decided at this meeting. The Finnish Association of Manual/Musculoskeletal Medicine will be responsible for staging the event. Kuopio is a city 400 km north of Helsinki, with a population of 90,000. It is situated close to the Russian border and can be reached by plane, train, or bus from Helsinki. The city has a medical school on the shores of Lake Kallavesi, one of hundreds of lakes located in the surrounding district. In conjunction with the general assembly, the education and scientific committees will meet prior to the conference.

It was passed at the recent AGM that the work of the education and scientific committees should continue, as the standard being achieved by them is of a very high quality. It is hoped that from the work being undertaken by these two committees that a teaching program will develop. FIMM sees the importance of lifting the standard of practitioner knowledge and patient treatment techniques before the establishment of an International Collegium. At present the standards are not uniform

## FIMM Update

in all the 26 member countries. Australia has representation on both committees. Professor Norm Broadhurst has replaced Dr Phillip Watson after his retirement from the education committee and I am the representative on the scientific committee. These committees meet once a year and have a continuous dialogue through the Internet and email connections. The

next scientific committee meeting will be held in Prague at the Charles University in early May 2002.

The FIMM website is again up and running. The website was initially developed in Queensland and, following my accident, was taken over by the English. It has been modified and has a new address: [www.fimm-online.org](http://www.fimm-online.org).

If there are any queries, questions or

suggestions regarding the website please feel free to contact me via by email address:

[Palmer@campac.net.au](mailto:Palmer@campac.net.au).

Norm Broadhurst and I are happy to convey any thoughts or queries to the respective committees if you care to contact us. The strength of any organisation is only as strong as its collective membership.

# A Research Perspective on the FIMM Congress

*Dr Michael Yelland*

Reflecting on my time in Chicago at the FIMM congress, I would endorse many of Ron Palmer's comments above. In particular, FIMM as an organization seems to be moving towards an emphasis on the evidence base for manual/musculoskeletal medicine, and to a more structured and uniform teaching program for musculoskeletal medicine throughout the world. At the same time it provides a forum for teachers and practitioners of the large number of systems of diagnosis and treatment which exist within musculoskeletal medicine. These systems range from the traditional biomedical ones, like orthopaedics, to others which are strong on mystique and weak on science, such as neural therapy. From my perspective as someone interested in research, this offers a fertile ground for research into claims that "it works in my hands". And there was evidence at the FIMM congress that this research is happening.

The congress program, having been organised by the American Osteopathic Medicine Association, naturally had a strong osteopathic flavour. It was encouraging to see some genuine attempts at critical evaluation of the osteopathic approach. For example, there was a very good poster presen-

tation by Brian Degenhardt et al on the interexaminer reliability of osteopathic palpatory evaluation of the lumbar spine. This began by rejecting some totally unreliable signs and going on to investigate the levels of reliability that can be achieved by training examiners in the "potentially reliable" signs. I was told that this research was actually influencing the content of the curriculum at osteopathic medical schools.

There were some interesting presentations on the effect of osteopathic manipulative treatment of both visceral and musculoskeletal conditions. One study showed that, while it had no effect on the serological response to the influenza vaccine in the frail elderly, it was associated with a lower incidence of respiratory and urinary tract infections and a reduced usage of antibiotics. Another study showed that osteopathic manipulation following knee or hip arthroplasty actually increased the length of hospital stay and reduced the rehabilitation efficiency. In contrast, a small randomized controlled trial on its effect on osteoarthritis of the knee showed it to be superior to control treatment for most outcome measures. The largest and most publicised study was a comparison of osteopathic spinal manipulation with standard care for acute low

back pain. This was published in the *New England Journal of Medicine* in 1999, despite some significant flaws in the collection and analysis of data. Robert Kappler, the study's treating physician, presented the results, but the best summary was by a Chicago reporter, who proclaimed that it showed that osteopathic manipulative care was just as effective as standard care. An intention to treat analysis may have shown it to be superior but this was not done. On the positive side, the study did demonstrate that patients treated with manipulation had lower usage of medication and physical therapy and had an improvement in nearly all of their physical findings. However suggestions of recruitment bias into the osteopathic group and observer bias in data collection both cast doubt on the validity of these findings.

The osteopathic manipulation study was easily overshadowed both in quality and positive results by the work presented by the next speaker, Stefan Blomberg, from Sweden. He presented the results of his three RCTs on manual therapy and steroid injections for acute and chronic low back pain, showing them to be consistently superior to standard care for a wide range of outcomes. The quality of the studies was easily the highest at the confer-

ence – quite inspirational to other researchers. It would be good to get him out to Australia to teach us his evidence-based algorithm for low back pain treatment.

For prolotherapists, like myself, there was quite a lot on offer, even though the preconference weekend workshop was cancelled due to lack of registrations. Tom Dorman spoke eloquently on the history, theory, and practice of prolotherapy and the trial evidence for its efficacy. Björn Eek talked about the indications and limitations of prolotherapy, including his research into intradiscal prolotherapy. This was complemented by an excellent short workshop given by Jeff Patterson and Tom Ravin with a very good video on technique. Perhaps the most fascinating presentation was given by Steven Levin from Virginia. He talked about

the concept of “tensegrity” or tension integrity which addresses the inability of conventional biomechanical models to explain how muscles move joints with such short lever arms without fracturing bones. He views the musculoskeletal system as a complex system of trusses with tension and compression elements spreading the load and conserving energy.

Despite the excellent venue, there were a few disappointments. There was quite poor attendance at some of the research sessions, although they did have to compete with concurrent practical workshops. Another reason was the low number of registrations overall. It was surprising that, in a big city like Chicago, the congress attracted no more registrants than we did here at FIMM in the Antipodes in 1998. Still, there was no shortage of interest-

ing people to meet from around the world. Finally, some of the “State of the Art” lectures on various disciplines within musculoskeletal medicine were so superficial and broad ranging as to be of little use to anyone. Those dealing with specific conditions, such as carpal tunnel syndrome, were much more useful.

Would I go to the next congress in Bratislava in 2004 – yes I would certainly like to. I do hope that is long enough for the Ozzie dollar to drag itself out of the doldrums. Chicago was a lot of fun but very expensive for Australians. At the end of the congress I popped into an art shop and found a painting for sale that was going for roughly what our house and land are worth. Perhaps Chicago’s gangsters from last century have just donned suits in this one and moved to the other side of the cash registers!

## Comments about the Chicago Triennial Conference of FIMM

*Professor Norm Broadhurst*

I share the impressions of those of Ron Palmer and Michael Yelland concerning the recent FIMM Conference and wish to congratulate the organisers on a well-organised program. I felt very privileged to be invited to present a workshop on pelvic pain and in addition to have a free paper accepted on the morphology of the piriformis muscle. I can say my sojourn in Chicago was both enjoyable and memorable and I look forward to going to Bratislava in three years’ time.

It was regrettable to arrive and see about 200 registrants when 10 times that number was hoped for. Lack of support for this conference especially by the host nation, and the one on the Gold Coast three years ago, raises the question whether we are “losing the plot”. I was informed by some of the older members that thousands attended

conferences when they were held in Europe a decade or so ago. Similarly, workshops in Australia a few years ago attracted scores of practitioners but now such workshops are either poorly attended or not programmed at all.

While the plenary sessions were a mixed bag I felt that the workshops provided an excellent means to see how the numerous modalities were used to treat similar dysfunctions with equally positive outcomes according to the various presenters. These modalities included Pain Free Maigne’s Method, Treatment of Still, Myofascial Trigger Points, Treatment of Sutherland, Facilitated Positional Release, Myofascial Release, Counter strain of Jones, Muscle Imaging Techniques, Zones of Sell, etc. This then begs the question as to what is reliable and valid in the way musculoskeletal pain is

managed and how can we arrived at a consensus - or do we need to?

It is without any fear of contradiction that we in Australasia are making every effort to teach and implement musculoskeletal medicine from an *evidence base*. This is reflected in the postgraduate diploma programs and the final report of the National Musculoskeletal Medicine Initiative. Thus we are in the position to support and contribute to the proceedings of the educational committee of FIMM, which is chaired by Glen Rasmussen. This committee is compiling a database on what is taught in this field throughout the world. This database is assessed against the evidence base, which should culminate in a few years in the body of knowledge and practice of musculoskeletal medicine that most certainly should lead to specialist recognition.

# Musculoskeletal Medicine Educational Activities

## MASTERS, DIPLOMA AND CERTIFICATE COURSES IN MUSCULOSKELETAL MEDICINE

### FLINDERS UNIVERSITY DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
18-24/11/01	Module one - Anatomy, Physiology and Biomechanics.	Flinders Medical Centre, Adelaide	Flinders University, South Australia	A/Prof Norm Broadhurst Ph 08-82951890	50 per module
23/2/02 – 3/3/02	Module two - Clinical Skills	Flinders Medical Centre, Adelaide	Flinders University, South Australia	A/Prof Norm Broadhurst Ph 08-82951890	50 per module

### UNIVERSITY OF NEWCASTLE MASTERS IN PAIN MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
2002	Masters in Pain Medicine	Internet	University of Newcastle	Prof Nikolai Bogduk Ph +61-2-49236172 Fax +61-2-49236103 <i>mgillam@mail.newcastle.edu.au</i>	N/A

## Musculoskeletal Medicine Educational Activities

## UNIVERSITY OF OTAGO DIPLOMA/CERTIFICATE IN MUSCULOSKELETAL MEDICINE

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
24/2/02-2/3/02	MSME 701 Part 1– Clinical Diagnosis (Aust.)	On campus, Australian venue in Queensland to be advised	University of Otago	V. McGroggan Ph. +64 3 364 1086 Fax +64 3 364 0909 Email: <i>veronica.mcgroggan@chmeds.ac.nz</i>	On application
16-24/3/02	Part 1- Clinical Diagnosis (New Zealand)	NZ venue - Christchurch School of Medicine & Health Sciences	As above	Christchurch	50 points*
29/7/02 - 2/8/02	Part 2- Clinical Diagnosis (New Zealand)	Christchurch		Ph +61-7-32695522 Fax +61-7-32696407 Email: <i>geoffharding@uq.net.au</i> website - <i>www.chmeds.ac.nz/go/dept-orthop</i>	(*total Pt1 & 2)
9/02	Part 2- Clinical Diagnosis (Aust.)	Queensland			
14/2/02 - 6/02	MSME 704 – Pain	Fortnightly teleconferences on Tuesdays	As above	Same contacts as above	NZ – on application Aust.-50 points
14/2/02 - 6/02	MSME 708 – Pain management	Fortnightly teleconferences on Tuesdays	As above	Same contacts as above	NZ – on application Aust.-50 points
21/2/02 - 3/3/02	MSME 709 – Clinical Therapeutics Australia	On campus Aust. venue in Qld to be advised	As above	Same contacts as above	NZ – on application Aust.-50 points
16-22/03/02	New Zealand	NZ venue Christchurch School of Medicine & Health Sciences	As above	Same contacts as above	NZ – on application Aust.-50 points
21/2/02 - 6/03	MSME 705 – Regional Disorders - Spine and MSME 706 - Regional Disorders - Limbs	Fortnightly teleconferences on Tuesdays	As above	Same contacts as above	NZ – on application Aust.-50 points
7-10/02	MSME 707 – Musculoskeletal Rehabilitation	Fortnightly teleconferences on Tuesdays	As above	Same contacts as above	NZ – on application Aust.-50 points
7-10/02	MSME 710 – Recreational and Sports Injuries	Fortnightly teleconferences on Tuesdays	As above	Same contacts as above	NZ – on application Aust.-50 points

## Musculoskeletal Medicine Educational Activities

### OTHER MUSCULOSKELETAL MEDICINE EDUCATIONAL ACTIVITIES

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
24-26 /3/02	Beyond the Horn - Australian Pain Society Ann. Scientific Meeting	Sydney Convention Centre	Australian Pain Society	DC Conferences Ph 02-94396744 Fax 02-94392504 Email <i>mail@</i> Website: <i>www.apsoc.org.au</i>	To be announced
Weekend March/ April '02	Prolotherapy Workshop	Fullarton, Adelaide, SA	Australasian Prolotherapy Association	Dr Margaret Taylor Ph: 61-8-8338 2899 Fax: 61-8-8338 2911 Email: <i>taylor@health.on.net</i>	Nil
7-10 /5/02	Rehabilitation: A Global Perspective – Ann. Scientific Meeting of Australas. Faculty of Rehabilitation Medicine (Preconference course: State of the Art Review of Back Pain on 7/5/02)	Sheraton Hotel, Brisbane	Australasian Faculty of Rehabilitation Medicine	DC Conferences Ph 02-94396744 Fax 02-94392504 Email <i>mail@</i> <i>dcconferences.com.au</i>	To be announced
18-20 /10/02	A Pain in the Butt: Groin, Hip and Pelvic Pain. Annual Scientific Meeting of the AAMM and AFMM	Melbourne – venue to be announced	AAMM	Vic Wilk Ph +61-3-95967211 Fax +61-3-95967871 Email: <i>vicwilk@smart.net.au</i>	To be announced
15-19 /6/03	Pain in Childhood: The Big Questions International Symposium on Paediatric Pain	Sydney Convention Centre	Paediatric Pain Medicine Unit, Sydney Children's Hospital	DC Conferences Ph 02-94396744 Fax 02-94392504 Email <i>mail@</i> <i>dcconferences.com.au</i> Website: <i>www.sydneypain.net</i>	To be announced



# AUSTRALIAN ASSOCIATION OF MUSCULOSKELETAL MEDICINE (AAMM)



## MANAGEMENT OF LATERAL ELBOW PAIN (TENNIS ELBOW) (Information for Patients)

### What is lateral elbow pain?

Lateral elbow pain refers to pain felt on the outside of the elbow. It is a common complaint, usually resulting from repetitive use or sustained contraction of the forearm muscles.

Many of the muscles that move the wrist and fingers are found on the lateral side of the elbow where they attach to bone. Lateral elbow pain is usually due to strain in one or more of these muscles close to their bony attachment.

Lateral elbow pain has been given many different names in the past. These include tennis elbow, lateral epicondylitis, and extensor tendinosis.

### What are the symptoms?

Lateral elbow pain is usually deep, dull, and aching. This pain can spread down the forearm to the hand, and/or up the arm to the shoulder. Pain can be present at rest but more commonly is brought on or aggravated by activity, especially lifting, twisting and grasping.

### What tests are useful?

Lateral elbow pain is diagnosed on the basis of the medical history and physical examination. It cannot be diagnosed by any blood test or on x-ray.

### What is the treatment?

The goals of treatment are to relieve pain and allow resumption of normal activities with as little discomfort as possible. Following are some simple measures that will help you achieve these goals.

1. *Avoidance of aggravating activities* is the most important aspect of treatment. This includes work, domestic, and sport activities. The avoidance may not have to be complete. A decrease in frequency or rate of the activity may be sufficient. Learning to modify the way some activities are performed may also be helpful. In general, performing activities with the palm up will be less stressful than performing them palm down.
2. *Heat and ice* are good for controlling pain. Heat may be applied on a regular basis. Heat or ice may be applied to the outside of the elbow before activity to reduce discomfort. Ice is best used to relieve pain after an aggravating activity.
3. *Painkillers* are useful. Your doctor will advise you about the best and safest ones to use. Anti-inflammatories have not been shown to have any extra benefit over other analgesics.
4. *Stretching and strengthening exercises* for the forearm muscles are often helpful. A specific exercise program can be devised by a musculoskeletal physician to maximise your functional recovery.
5. *Massage* of the painful muscle(s) is often helpful.
6. A *counter-force brace* is a tight non-flexible strap worn just below the elbow. It is believed to relieve the strain on the injured muscle(s) at the elbow. It is particularly helpful when used whilst performing aggravating activities.
7. An *injection* of steroidal anti-inflammatory with local anaesthetic is the most effective treatment for lateral elbow pain. This is not very painful and is well tolerated. Side effects can occur, but are rare. This is your best option if the above suggestions have not helped. For best results it is suggested to do strengthening and stretching exercises in the weeks following the injection.
8. *Total rest* with your arm in a sling is best avoided. It can be used for very severe pain, but even then only for a very short period.
9. *Extracorporeal shock wave therapy* is a promising new treatment for more chronic cases.

The above information should allow you to understand the nature of your condition and how to manage it. The information provided is based on the currently available scientific evidence. You will notice that this approach does not involve the use of expensive tests or gadgets, and does not involve the patient attending for treatment several times a week. Instead, you are given the information and thus can help develop the management plan. You are encouraged to play a proactive role in the management of your problem. Active patient participation in their treatment is known to improve results.

Musculoskeletal physicians are doctors who have completed extra postgraduate training in the management of a wide range of musculoskeletal conditions including lateral elbow pain. Our aim is full restoration to a functional and healthy state as quickly as possible.