

Australasian Musculoskeletal Medicine



- Fibromyalgia syndrome
- The patient who couldn't ride his Harley Davidson motorbike
- Literature review for rami communicans block for chronic discogenic low back pain
- Headaches and the cervical zygapophysial joints
- Shoulder pain
- Evidence-based guidelines improve performance measures in orthopaedic outpatients for low back pain
- Personal injury claims

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Editorial

This edition of *Australasian Musculoskeletal Medicine* is dedicated (in the main) to those who have served.

There are two articles by Jay Govind and one by Brian McGuirk which have been reproduced from earlier editions in honour of their contributions to the discipline of musculoskeletal medicine.

Both Brian and Jay have passed away and they represent a great loss to the overall academic force of musculoskeletal medicine. The articles chosen are worthy of reprinting because they are high-quality works which remain topical. The article by Jay "Personal Injury Claims: Quo Vadis?" is particularly worth reprinting because nothing has changed much since it was first printed.

We have reproduced the dedication by Nik Bogduk to Brian McGuirk as well so that we can all gain an understanding of Brian's journey towards musculoskeletal medicine.

No less a loss is the retirement of Professor Norm Broadhurst from active practice and teaching. Norm has been a member of AAMM almost since its inception and over the years has served on the committee in every capacity including the presidency. He was instrumental in setting up the postgraduate diploma course at Flinders University and has organised some of our more memorable scientific conferences in and around Adelaide over the years. Thanks Norm for your contribution. His article "Shoulder pain – What ultrasound imaging reveals" is well worth the re-read.

Finally, there is new material here – a thought-provoking paper by Bill Douglas, a rheumatologist well known to many of us (and a long-standing member of the Association). His paper on fibromyalgia is bound

to promote debate. (He will be presenting his views at the conference in Palm Cove). Also there are two interesting papers by Tom Baster from Brisbane. Tom's current interest in rami communicans blocks and the use of SPECT scanning to diagnose facet joint pain has seen him undertaking a trial with an interventional radiologist to see whether the results can deliver more success in the treatment of chronic back pain.

Tom will also be presenting at the conference, so make sure you get there.

On another note, the AAMM (and the AFMM) have become partners of Pain Australia in its efforts to lobby for support for increased government funding for the treatment of chronic pain in Australia. Whilst most of the impetus for this organisation has come from the pain physicians, there is an obvious role for those practising musculoskeletal medicine since about 60% of chronic non-cancer pain is musculoskeletal in origin. We hope to have an influence on the teaching of GPs to upskill them in the assessment of musculoskeletal pain so that the problem of chronic pain might be avoided with the better management of acute pain.

Finally, we have sent out two press releases recently – one dealing with a court case recommending that doctors performing injections (even for tennis elbow) should be gowned and gloved. This needs to be tackled head on as it would mean the end of injecting anywhere outside of a hospital. The other highlights the need for GPs to be upskilled in the assessment of pain problems to decrease the enormous demand on hospital pain clinics. Hopefully they will stir up some interest.

Dr Geoff Harding
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From the AAMM President

This year we have seen the topic of health raised almost daily in this country. Healthcare costs the community lots of dollars and the politicians are always trying to deal with the ever-increasing drain on the budget. Whilst the focus usually is on hospitals and general practice, there are many other areas where changes are being made.

Health applications have been touted as being one of the main reasons the National Broadband Network will be successful. A cynic might say that healthcare delivery will be improved only when it is actually delivered – by someone on the ground. What we need is better funding for those structures already in place. I believe that the GP is still the cog with all of the spokes attached and that funding of fee-for-service is still the best way to address the access problem. I realise that there are those among us who feel that the fee-for-service approach is susceptible to abuse, and that fund-holding is the most effective way to deliver healthcare.

Whatever is best, this writer feels that with respect to the treatment of musculoskeletal pain problems, there is a growing void between the practical and the esoteric. There is much money spent on approaches to pain which seem to miss the mark. I see many new patients weekly who have been going around in circles for years trying to get a diagnosis for their pain. They have been to see well-credentialed practitioners but have never been given a diagnosis. Insurers have spent thousands on these patients with no improvement. They might be told that there is no surgical solution for the problem or that nothing can be seen on the scans. Or they might be told that they have “tight glutes” (which the patient knew anyway even if he/she didn’t know the name of the muscle).

I believe a diagnosis such as *lumbar spinal pain of unknown origin* or *lumbar spine somatic referred pain* is valid (given what we know we don’t know) but so many patients I see are told that “you have pain because your muscles are weak and need to be strengthened”. (This is often after the patient

has experienced pain of this nature for the first time in their life after falling off a garden wall!). This type of assessment is nonsense. But it is supported by much government or insurance funding.

The discipline of musculoskeletal medicine – as it exists in this country – has much to offer mainstream medicine. The problem is that we are still on the outer because we don’t fit the general practice paradigm and so don’t attract the funding (or even the rebates in the case of the non-VR’d amongst us). Even in the past year there was a feeling that anyone who spent time sorting these patients out (long consultations) was being targetted by Medicare and the PSR because this was “innappropriate practice”. The fact that there has been a respite in the targetting of these doctors is only because of an administrative error in appointing the members of the PSR committees – not because it was seen to be unfair or unreasonable. Expect similar attention in the future because governments and beaurocrats like statistics – and if you lie outside the curve, you are a target.

Those of us who have been practising this discipline for years are getting older. Some, like Jay Govind and Brian McGuirk, are no longer with us. We need young graduates to enter the fold. Unfortunately there is not readily available funding for this type of medicine. There is Medicare funding for dental work for patients in chronic pain, for psychology support, or physiotherapy support, but nothing special for musculoskeletal medicine. So we do what we can.

The thing that motivates us is the response our patients give us. We might not “cure” many, but we do interrupt the vicious cycle of looking for solutions to their pain when often a good explanation for the pathology is all that is needed – or referring off for a block (by someone who follows the guidelines) to localise the pain generator.

Perhaps the National Broadband Network will deliver programs which can do a face-to-face assessment and deliver this sort of management – but I don’t think so. Let’s wait and see.

Geoff Harding

From the NZAMSM President

NZAMSM Charlie Ng is currently overseas, so Secretary Mike Cleary stepped into the breach to provide a report for our journal. Just as we were to go to print, we received a report from Charlie also. Thus we can offer our readers two messages from our NZ committee.

We have been considering our place in the NZ medical system from the point of view of our colleagues in general practice and specialist practice. There has been frustration with the lack of recognition of our existence, experience, and expertise by the Health Workforce tasked with planning for the future of the NZ medical manpower. While we may seethe at this, it seems ignoring musculoskeletal conditions is an international problem with medical school curricula as judged by the recent article in the May 27th *NZMJ*, called **"Basing musculoskeletal curriculum changes on the opinions of practicing physicians"** who sought to survey "...practicing clinicians who manage these conditions, to find out their opinions on which disorders they regard as most important. This information will then be used to inform curriculum design." No prizes for guessing which group of practising clinicians was not surveyed! Charlie has sent the Health Workforce person briefed with musculoskeletal education a CV of our organisation and, as I write, I sense that the *NZMJ* is receiving correspondence on the above article.

We have been benefitting from the presence on our committee of Michael Hewitt and John Gyenge, who as general practitioners are able to remind the Fellows that in NZ "20% of visits to general practitioners are due to musculoskeletal disorders." The Accident Compensation Corporation (ACC) has been using GPs with a special interest in matters musculoskeletal (GPSI) to assess claimants in areas where there are long delays in seeing specialists. Michael Hewitt has been liaising with ACC to offer our expertise to these GPs, but it has been a struggle and changes in ACC personnel as well as a presumed shake up or should that be a shakedown of the ACC after our NZ elections in November have all contributed to very little to show for a lot of effort.

The Auckland contingent has continued to hold an annual educational session for the GPs which is well attended. Their theme this year on Shoulders, developed by Lucy Holtzhausen in collaboration with ACC, will be taken on the road to Wellington in August.

The dwindling membership of NZAMM is a concern for obvious reasons. The enthusiasm and energy of the founding years, with a large and participating membership, seems a long time ago. To conserve our organisational energies we are trying to work out a memorandum of understanding with the NZ branch of the AFMM and avoid burnout with unnecessary duplication. We have spent a significant amount of time at committee level trying to fathom what has changed over the years.

Despite the large burden of musculoskeletal pain in the community (40–67% of the New Zealand population suffers from musculoskeletal pain) and its predicted increase as the population ages, there seems to be no corresponding increase in the clinical curiosity of our medical practitioners to augment the meagre musculoskeletal teaching of our medical schools via the NZAMM. Still we must take heart from the kakapo, that rarest of NZ parrots (for Australians, go to You Tube "Shagged by a rare parrot") which has recently increased to 131 after an intensive breeding program, and let's hope that we will turn the corner and start to build up our numbers again.

Mike Cleary, Secretary

The NZAMM Executive Committee has been reviewing the future direction and activities of the NZAMM. Consideration has been given to what our members want and the goals of the NZAMM – to further the musculoskeletal education of its members, GPs, and allied health professionals in NZ. The vehicle to achieving this has previously been the journal, manual therapy workshops, and conferences. It is planned to continue with two journal publications per year as the journal provides a valuable opportunity for members to contribute for publication. In order to supplement this, the NZAMM has subscribed to the International Musculoskeletal Medicine Journal (IMM) which will give members international exposure.

The next manual therapy workshop will be held on 27 August 2011 at the Chateau Tongariro. The conference this year will be a conjoint one organized by AAMM at Palm Cove, North Queensland, on 30 June to 3 July. NZAMM members will be presenting at the conference and you are encouraged to join the NZ contingent attending. In addition to these educational events, GP workshops have been taking place and more are planned.

Following on from a successful GP shoulder workshop in Auckland in November 2010, members have presented at the Rotorua GP conference in June and will be presenting at a shoulder workshop in Wellington on 13 August. Shoulder and neck workshops will be presented at the RNZCGP conference in Auckland in September. Members are also invited to join musculoskeletal Fellows at their quarterly retreats where a variety of interesting topics and challenging cases are presented for discussion. This is a very good opportunity for peer review and update. The next retreat is on 25–27 August at Chateau Tongariro. Many members hold the Diploma Musc Med. From July 2011, you will have the opportunity to participate in the Master of Pain Medicine, offered by Otago University.

There have been ongoing discussions with ACC about the GP Special Interest (GPSI) scheme which ACC operates in some regions to assess and triage musculoskeletal and orthopaedic ACC cases. The NZAMM has recommended to ACC to use the NZAMM's pool of Diplomates and the education and reaccreditation offered by NZAMM and AFMM.

The success of the NZAMM depends on your support and feedback. I look forward to seeing you at the various educational events.

Charlie Ng, President

In Memoriam: Brian McGuirk

Dr Brian McGuirk was a Newcastle lad who went to Sydney to complete high school, and to study medicine at the University of Sydney. After graduating, Brian spent time in the UK as a house officer, before returning to Sydney to take up general practice. He was awarded a special service medal for being the first medical officer to attend and treat patients injured in the Granville rail disaster in 1977.

In due course, Brian developed an interest in occupational health. He joined the first group of candidates for the Diploma in Public Health (Occupational Health) at the University of Sydney in 1984, and become a founding fellow of the College of Occupational Medicine (later to become Australasian Faculty of Occupational and Environmental Medicine). Under the tutelage of Professor Fergusson, Brian exhibited a talent for developing policy and regulations. After several roles in the NSW public service, and as an international adviser to developing countries in Africa, Brian was appointed Commissioner for Occupational Safety in Western Australia. In that role, he wrote the OHS regulations for that state.

Returning to the east coast, Brian became Chief Medical Officer for the Newcastle division of MMI (later to become Allianz). His role was to assess and approve (or not) plans of management for injured workers on compensation. In that position, he came to learn of the research studies into spinal pain being conducted at the University of Newcastle. When the University was awarded a grant to conduct the National Musculoskeletal Medicine Initiative, he offered to join the Directorate of the Initiative. Foregoing a handsome salary in the private sector, he became Deputy Director of the Initiative with the rank of Senior Lecturer in the University.

Brian assumed a pivotal role in developing the evidence-based guidelines for the treatment of low back pain, and testing their efficacy. He was the senior author of the first study ever to have tested, and shown, the efficacy of evidence-based medicine for low back pain.¹ As the Initiative drew to a close, Brian took on part-time work as a VMO in Staff Health at the Royal Newcastle Hospital, in which he applied evidence-based medicine to the management of injured workers. When his successes were recognised, this role was progressively extended across the Greater Newcastle

Sector, and he became a Staff Specialist in Staff Health. In a landmark study² he showed that evidence-based medicine virtually eliminated workers' compensation claims for back injuries. This work was awarded the prize for best presentation by the Spine Society of Australia in 2007.

Brian worked half-time in Staff Health. The other half he spent in orthopaedic outpatients, where he helped GPs provide evidence-based care for their patients with back pain, and thereby avoid surgery. This work was recognised when Brian became a finalist for the Baxter Awards in 2006.

Brian recorded his work in two textbooks: one on back pain,³ and one on neck pain.⁴ In writing these books, Brian insisted that they be friendly to GPs. The books must not just state what should be done, but describe how actually to do it. He also wrote the chapters on acute low back pain and chronic low back pain in the recently published, 4th edition of *Bonica's Textbook of Pain Management*.

In November 2009, Brian retired from the Hunter New England Area Health Service, having reached the age of 70. His achievements in promoting, testing, and using evidence-based care are unparalleled. Whereas academics preach the word, they do not treat patients, Brian did both. Neither his personal clinical talents nor the outcomes he achieved for his patients are likely to be replaced. On March 3, 2011, he passed away quietly.

Nikolai Bogduk

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Fibromyalgia syndrome:

A review with recommendations for primary care management

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Abstract

This article outlines the development of the current concept of fibromyalgia syndrome (FMS). It reviews current management strategies and discusses the medicolegal implications associated with FMS.

The important, documented psychological associations with FMS have received little recognition by many rheumatologists. Psychological factors are not mentioned in either ACR Criteria. The EULAR 2010 guidelines for management of FMS have brief recognition of this association. Integrating the work done by psychiatry and internal medicine in relation to FMS would only benefit and not disadvantage sufferers with this condition.

Fibromyalgia (FMS) is a common syndrome consisting of a spectrum of multiple unexplained symptoms of which chronic widespread pain (CWP) is the most prominent.

A PubMed search using the key word "fibromyalgia" conducted on 23 September 2010 of papers published in the past five years identified 2058 publications. Despite this large output of literature, the medical profession remains divided over this condition: is FMS a rheumatological disorder or a psychosomatic illness? What is the optimum approach to management of people with FMS? What are the medicolegal implications of different aetiological understandings of FMS?

Historical overview

FMS is a contentious syndrome in terms of terminology, aetiology, and management. Sir William Gowers coined the term "fibrositis" in 1904 in the belief that pain and tenderness was related to underlying inflammatory foci hidden in the fibrous tissue of the muscles. This led to the treatment of "needling" of tender points. "Needling" and the prescription of anti-inflammatory and disease-modifying drugs persist to this day despite the inflammatory theory having been disproved. In the 1960s, fibrositis was also labelled "psychogenic rheumatism".¹ In the mid 1970s, Smythe and Moldofsky suggested the term fibromyalgia.² This term infers the source of pain to be in fibrous tissue and muscle.

In 1990, the American College of Rheumatology (ACR) published criteria for the classification of FMS.³ A panel of 24 rheumatologists studied 293 patients with this condition with 265 controls. The study reported that FMS could be distinguished from other rheumatic conditions with good sensitivity and specificity by the use of simple criteria: (1) history of widespread pain present for three months or more; (2) pain in 11 or more tender sites on digital palpation.

The authors of these criteria found that other prominent symptoms such as unrefreshed sleep was not found in all patients and was therefore not included in the ACR 1990 Criteria. It was noted that the criteria may be useful for the diagnosis and classification of FMS despite the lack of objective abnormalities. Since the 1990 ACR Criteria were

published it has become clear that its concept was flawed, that the tender point count was unreliable and subject to manipulation. This is not surprising as tender point counts are a faulty construct without pathological support.⁴⁻⁶ The ACR 1990 Criteria continued to link FMS to skeletal muscle and in the process reinforced the belief of sufferers, particularly those attending rheumatology clinics, that they were suffering from a serious, disabling rheumatic disease.⁷

It became clear that the ACR 1990 Criteria were not serving its purpose and they are now discredited. Unfortunately many researchers utilised the ACR 1990 Criteria as the "gold standard" and as a consequence many of their publications are probably flawed since the criteria used have been superseded.

A study by 10 eminent rheumatologists found that approximately 25% of FMS patients did not satisfy the 1990 classification criteria.⁸ These authors noted that the important variables were widespread pain, unrefreshed sleep, fatigue, and cognitive symptoms. The ACR has published the 2010 preliminary criteria which propose the diagnosis of FMS be based on: (1) widespread pain index (WPI) assessed by self-report, its distribution and symptom severity; (2) Symptom Severity Score (SSS) based on self-report of fatigue, unrefreshed sleep, and cognitive symptoms; (3) symptoms present to the similar level for at least three months; (4) the patient does not have a disorder that would otherwise explain the pain. Of note is the statement that the diagnosis of FMS can be reached "*without a physical or tender point examination*". The WPI and SSS continue to rely largely on patient self-report to make the diagnosis of FMS.

The ACR 2010 Criteria incorporate recognition of fatigue, unrefreshed sleep, and cognitive symptoms in FMS. Because of the prominence of CWP in FMS, these associated somatic complaints have been largely overlooked. The important, documented psychological associations with FMS have received scant recognition by many rheumatologists. Psychological factors are not mentioned in either the ACR 1990 Criteria or the preliminary ACR 2010 Criteria. In the EULAR 2010 evidence-based recommendations for management of FMS there is brief recognition of this association.⁹

Pain in FMS

CWP and soft tissue tenderness are the universal complaint of patients with FMS. Hyperalgesia and allodynia to light pressure affects the skin and underlying muscles. The pain is not of a dermatome distribution. Patients with anxiety disorders reported the greater number of physical symptoms and highest pain intensity.¹⁰

An editorial in the *Journal of Rheumatology*¹¹ showed a diagram with pain in the centre of a cycle of illness and pain causing disturbed sleep, producing more pain and resulting in depression. The concept that pain is the product of psychological distress is disputed by some clinicians who believe that pain is a cause of psychological problems and not the reverse. Recent research found that co-morbid psychiatric disorders in most cases preceded the onset of FMS.¹²

Sleep in FMS

Unrefreshing and poor quality sleep is virtually universal in FMS sufferers. Moldofsky¹³ hypothesised that pain in FMS was directly related to poor quality, unrefreshing sleep and hence the association between alpha EEG activity and clinical symptoms of FMS.

Polysomnographic findings in FMS include alpha-delta sleep anomaly, an increase in stage 1, a reduction in delta sleep and an increased number of arousals. There is high frequency of alpha intrusions in non-REM sleep with decreased slow wave (delta stage 3 and stage 4 sleep).¹⁴ Some would argue that sleep disturbance is a consequence of pain in FMS; however it has been shown that improvement in sleep quality reduces the intensity of CWP.¹⁵

Experimental studies have shown that CWP and cognitive dysfunction can be reproduced in healthy volunteers by noxious stimuli disturbing sleep over a number of nights.¹⁶ Chronic sleep deprivation has been shown to have adverse effects on neural, behavioural, and physiological functioning with detriment to physical and mental health.^{17, 18}

The biochemical cellular processes occurring during sleep are poorly understood; however studies have reported that some patients with FMS have a decrease in CSF and blood serum levels of serotonin and epinephrine which may be partly associated with the decrease in delta sleep.¹⁸ It is recognised that the use of low dose tricyclic antidepressants in FMS may reduce the intensity of CWP.¹⁹ The beneficial effects of tricyclic medication may be due to its influence on neurologic serotonin metabolism.

Elevated levels of substance P have been reported in the CSF of some patients with FMS.²⁰ Substance P is a neuroactive peptide widely distributed throughout the nervous system and may be concerned with sleep regulation. Whether these elevated levels contribute to arousals requires additional evidence to clarify substance P's role in sleep regulation as elevated levels of substance P are not unique to FMS. A trial blockade of substance P receptors did not relieve pain or anxiety in this condition.²¹

Other symptoms in FMS

Fatigue is almost universal in FMS and may be the consequence of unrefreshed sleep. However, chronic fatigue may be associated with major depressive disorders and there appears to be an overlap between FMS and "chronic fatigue syndrome".

Many other symptoms are commonly reported in FMS such as fatigue and cognitive changes (difficulty concentrating, poor memory) as well as irritable bowel syndrome, temporomandibular pain related to bruxism, restless legs, frequency of micturition, vulvodynia, paraesthesia, and sensations of peripheral swelling. (Psychological associations will be discussed in more detail below.)

Investigations

There are no specific diagnostic tests for FMS. Blood studies and biochemistry are normal and muscle biopsies show normal histopathology.

Basic investigations are required to exclude an organic illness. This would include a full blood count, biochemical profile analysis, erythrocyte sedimentation rate, C-reactive protein and thyroid function. Additional investigations will be determined by specific presenting symptoms and clinical signs.

Because of the prevalence of sleep disorders in the community,²² there is a case for performing polysomnography to exclude sleep pathology in patients where fatigue is a prominent symptom.

Although magnetic resonance imaging and magnetic resonance spectroscopy studies of the brain have been done in the research setting, they have no recognised diagnostic application in FMS at the present time.

Epidemiology and prognosis

Comparisons of the frequency of FMS are confounded by variation in the definition used. Also, studies done in those attending rheumatology clinics are likely to be biased by the prevalence of more complicated and chronic cases in this setting.⁷ With these limitations in mind, in the United States of America, it is estimated that 2-4% of the population suffer from FMS.²³ In Europe, the prevalence ranges from 1.7% in Spanish women to approximately 4.9% in a multicentre report.^{24, 25}

A cross-sectional study in the Finnish population²⁶ screened 7,217 adults, 30 years and over for FMS and found only 54 cases (0.75%). The authors state that "this epidemiological data offered little support for the concept of FMS as an independent syndrome". They also noted no significant association with mental stress at work.

The incidence of FMS in Asian countries has only recently been reported. In Malaysia and Singapore 48 rheumatologists were surveyed.²⁷ They reported that FMS was almost unheard of. FMS is rarely observed in China.²⁸ One study recorded two cases found in Shantou (n = 2,350)

and one case in Taiyuan ($n = 3,915$). The absence of FMS in China may suggest genetic and sociocultural differences. This may reflect the Chinese concept of the unity of body, mind, and health. This scenario could change with the globalisation of the American psyche.²⁹

A Canadian study³⁰ estimated that FMS patients use approximately twice the health services compared to control groups. A European study published in 2008³¹ concludes that FMS imposes a high burden not only on the patient but on all sections of society. There are no reliable Australian data as to the healthcare costs resulting from FMS nor has there been a survey into the frequency of the condition in the Australian community.

Australian observers found that many community patients with mild FMS symptoms improved with time. In a prospective study, 21 of 44 FMS patients treated with minimal intervention no longer fulfilled the ACR 1990 criteria for FMS at two-year follow up. The authors conclude that remission of key clinical features may occur during the natural history of FMS.³²

In the author's private clinical practice, most FMS sufferers continued to work. Their incapacity was seldom severe and many were disinclined to take medication due to its lack of efficacy or to the noted frequency of drug intolerance in this group. Those who were disabled generally had an associated psychiatric condition or were involved in litigation with its repercussions.

Except for those involved in litigation, FMS sufferers rarely recalled any traumatic event. The FMS symptoms came on gradually over a period of years without explanation.

In this practice, most of the patients with FMS were adult women. They rarely admitted to serious psychological symptoms of anxiety or depression and seldom identified significant stressors, either at work or at home, that they considered being the cause or aggravation of their various somatic symptoms. Males who were seen with FMS were generally found to have an underlying depressive illness and/or obstructive sleep apnoea. This observation of gender differences in the presentation of FMS is supported by literature confirming male-female differences in pain sensitivity and representation of chronic pain disorders.^{33, 34}

Psychiatric aspects of FMS

There is a significant body of work regarding FMS in the psychiatric literature. This rarely seems to intersect with the research done by internal medicine and primary care physicians.

Arnold et al. observed that co-morbid psychiatric disorders preceded the onset of FMS in most cases,¹² suggesting that the psychiatric disorder does not develop simply as a consequence of having FMS. They suggested that patients with FMS should be routinely evaluated for the presence of co-morbid psychiatric disorders. The relatives of patients with FMS are more likely to have psychiatric disorders.^{12, 35} Other studies have shown that patients with FMS have a high comorbidity with anxiety and mood disorders—approximately three times higher than the general population.^{10, 12}

A study by McBeth and associates³⁶ showed that subjects ($n = 1,953$) who were free of CWP at baseline were at increased risk of its development over 12 months if they displayed other features of somatisation. This lends support to the hypothesis that CWP can be a manifestation of somatisation and distress. It is noted that this study did not attempt to establish the incidence of FMS.

FMS has been described as “a chronic pain disorder overlapping with other functional illnesses or affective spectrum disorders such as chronic fatigue syndrome and irritable bowel syndrome”.^{10, 35} It was noted that the sufferers had a greater use of maladaptive coping strategies and that psychological stressors may affect the severity of pain with FMS who also show enhanced pain sensitivity when given an array of noxious stimuli.³⁷ This sensitivity is reported to also occur more frequently in the first degree relatives compared to the relatives of healthy people and people with other painful illnesses.³⁸

Many publications report a high percentage of patients with FMS identifying a history of childhood and adolescent sexual and physical abuse.³⁹ One study reported 57% of female FMS patients having a history of mental, sexual, or physical abuse. A significantly greater utilisation of outpatient healthcare services with problems other than FMS was also reported.⁴⁰

Wolfe⁴¹ acknowledges that in the rheumatology clinic it is difficult to assess the patients' psychological status. At the same time, the current recommendations for the diagnosis and treatment of FMS do not take these co-morbidities into account.

An Australian study of somatic symptoms, hypochondriasis, and psychological distress used a questionnaire completed by 10,507 consecutive patients attending 340 general practitioners.⁴² They found that somatisation is common in general practice and more prevalent than depression and anxiety. The authors noted that recognition of depression and anxiety can be hindered by somatic presentation and attribution. 18.5% of patients were classified somatisers while 29.6% of somatisers had high anxiety and depression scores. This study included fibromyalgia as a somatoform disorder.

The editor of DSM-IV-TR under the definition of Mental Disorders writes, “The term *mental disorder* unfortunately implies a distinction between mental disorders and physical disorders. The separation of mental and physical disorder is artificial, but unfortunately the term *mental disorder* persists because an appropriate substitute has not been found.”⁴³

FMS substantially fulfils the criteria for a pain disorder as a subtype of somatoform disorder as defined in DSM-IV-TR:

A. Pain in one or more anatomical sites is the predominant focus of the clinical presentation and is of sufficient severity to warrant clinical attention.

B. The pain causes clinically significant distress or impairment in social, occupational, or other important areas of functioning;

C. Psychological factors are judged to have an important role in the onset, severity, exacerbation, or maintenance

of pain;

D. The symptom or deficit is not intentionally produced or feigned;

E. The pain is not better accounted for by a Mood, Anxiety, or Psychotic disorder and does not meet the criteria for Dyspareunia.

Management

Because of the lack of agreement about the aetiology of FMS, it has been difficult to obtain consensus about how best to treat it.

EULAR recommendations

In 2010, EULAR published recommendations for the management of FMS.⁹ A multidisciplinary taskforce of 19 professionals was formed to undertake a systematic review of the FMS literature. Of the 146 eligible studies, 59 non-pharmacological studies and 39 pharmacological intervention studies were included in the summary tables. The primary outcome measures were change in pain assessed by the visual analogue scale (VAS) and fibromyalgia impact questionnaire (FIQ). Studies were excluded if they did not utilise the ACR 1990 Classification Criteria which have now been superseded. Based on the literature and input from an expert panel the taskforce made nine recommendations for the management of FMS.

The EULAR recommendations suggest that FMS be recognised as a complex heterogeneous condition where there is abnormal pain processing and other secondary features requiring comprehensive assessment of pain, function, and psychosocial content. The paper recommends a multidisciplinary approach with a combination of non-pharmacological and pharmacological treatment modalities. It also noted that associated depression, fatigue, and sleep disturbance should be discussed with the patient.

Non-pharmacological interventions appear to have significantly better effect on function than medications, but were more likely to have used function as an outcome measure. A controlled study of the effects of supervised cardiovascular fitness training showed some therapeutic benefit in selected patients with FMS in improving pain, fitness, and sleep quality.⁴⁴ Based on expert opinion the authors recommended individually tailored exercise programs and hydrotherapy. This is consistent with the Arthritis Australia recommendations encouraging aerobic exercise, strength training, and gentle exercise such as yoga, pilates, and Tai Chi.^{45, 46, 32}

Cognitive behavioural therapy (CBT) was found to be of benefit in some patients with FMS despite the only two studies identified being judged to be of poor quality. Based on expert opinion other therapies that may be of benefit include relaxation, rehabilitation, physiotherapy, and psychological support.

The pharmacological intervention studies focused on newer agents. Simple analgesics such as paracetamol and weak opioids can be recommended. Strong opioids and

corticosteroids are not indicated, based on expert opinion due to the absence of clinical trials identified in FMS and significant long-term side effects.

Although tramadol is recommended, this is qualified with the statement that it be used with caution due to the risk of dependence and the possibility of typical opiate withdrawal symptoms. There was no mention of the significant risk of serotonin syndrome reactions, particularly when tramadol is combined with tricyclic or SSRI antidepressants.

The use of SSRIs and dual reuptake inhibitors are recommended by the EULAR group. The studies included had trials of 6-12 weeks' duration and only four trials were examined. It is not clear whether the antidepressants had been prescribed on a background of major depression or whether the study participants had been assessed by psychiatrists or psychologists prior to the intervention. Because the onset of action of SSRIs occurs gradually over a period of some weeks, given the short duration of the trials, some of the results reported may be unrepresentative. The impact of adverse events was briefly discussed. The strongest evidence is for the use of amitriptyline, the antidepressant with the longest track record.

The EULAR recommendations consider the use of pregabalin and a range of other agents. In June 2007, the use of pregabalin was approved for FMS by the USA Food and Drug Administration (FDA). It is being prescribed off-label in Australia for the treatment of FMS and there seems to be some pressure from industry and consumer groups to achieve Australian Therapeutic Goods Administration (TGA) approval for extended indications.

A large 14-week multicentre trial of pregabalin for patients with FMS reported that pregabalin is an important treatment option for this group of sufferers. The trial was funded by the manufacturer and the article declared that several company employees participated as co-authors.⁴⁷

It is of concern that drugs which are known to carry risks of significant side effects and are likely to be used in the setting of polypharmacy are being advocated for a non-life threatening and at times self-limiting condition.⁴⁸ Also of growing international concern is the role of the pharmaceutical industry. The potential for conflict of interest by researchers or clinicians has been recognised for some time.⁴⁹ Relationships between the pharmaceutical industry and patient support groups are coming under increasing scrutiny.⁵⁰

Medicolegal aspects of FMS

In 2010, the Royal Australasian College of Physicians (RACP) and the Australasian Faculty of Occupational and Environmental Medicine (AFO&EM) released a position statement concerning the health benefits of work.⁵¹ The section about work and musculoskeletal conditions reinforces that early return to work or remaining in suitable work is important and beneficial for the patients' health and well-being.

In Australia FMS is not generally considered to be a work-related condition. However, the question of aggravation arises regularly. It should be noted that work-related pain

alone does not equate to a diagnosis of FMS.

When asked, patients may identify certain stressors such as viral infections, and physical or psychological injury preceding the onset of FMS.⁵² This is not evidence of causation, it is simply association. This is an area of medicolegal importance that requires a well-designed prospective study.

Primary care management of FMS

Family physicians have been shown to diagnose FMS accurately.⁵³ They are in a position to approach the patient as a whole, with strategies directed towards physiological and psychological complaints within the context of the family and society. The family physician is also well situated to curb unnecessary investigations and over-medicalisation of the complaint. Very few people with FMS require specialist evaluation.⁵⁴

A comprehensive medical history is required in all FMS patients. The family history, past medical history, and a careful clinical examination is essential to exclude the presence of inflammatory conditions and other causes of chronic musculoskeletal pain.

Clinical examination is imperative to exclude organic illness presenting as FMS.

The recommended approach is to:

- Be positive and supportive.
- Acknowledge the pain and that it is not "all in your head".
- Reassure the patient they do not have a serious illness or long-term disability.
- Explain that the reason for their symptoms is likely to be related to poor-quality, unrefreshing sleep, with resulting aches and pains, tenderness, and fatigue.
- Encourage the patient to remain in the workforce, negotiating suitable duties if required.

Initial treatment is based on simple, safe analgesia such as paracetamol and directed at improving sleep hygiene. The Arthritis Australia information sheet about exercise and fibromyalgia is a useful resource.⁴⁵ These measures are likely to improve well-being and function. A low dose tricyclic (such as amitriptyline 5-10 mg) seems to improve sleep quality.

Referrals should be considered for some specific groups:

1. To a psychiatrist for patients with psychiatric comorbidities such as anxiety and major depression before prescription of anxiolytics or antidepressants.
2. To a sleep clinic for patients with symptoms suggestive of sleep apnoea.
3. To a pain management clinic for patients who require a multidisciplinary team approach.
4. To the relevant specialist if there appears to be an alternative underlying diagnosis to FMS.

Conclusion

FMS is a common condition of varying severity and duration presenting with unrefreshed sleep, fatigue, cognitive dysfunction and chronic widespread pain. This paper has discussed the history of FMS and the different perspectives taken by physicians and psychiatrists. Cultural and medicolegal aspects have been considered and the EULAR 2010 management recommendations discussed. The paper concludes with some simple, safe empirical recommendations that will assist primary care physicians in treating people with FMS.

Conflict of interest

In the past five years Dr Douglas has attended Journal Club meetings sponsored by Wyeth. He has no financial interest in any healthcare companies.

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The patient who couldn't ride his Harley Davidson motorbike

A case report and a literature review of the use of SPECT scans for the diagnosis of chronic low back pain arising from the zygoapophysial joints

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Case report

Bob (not actual name) aged 48 was not happy, as his chronic low back pain (cLBP) prevented him riding his Harley Davidson motorbike. He had been involved in a serious accident whilst serving in the Armed forces when a heavy tank rear door had struck him, resulting in fractures to his left femur, right femur, dislocated hips and left potts fracture about 20 years previously. He had developed low back pain about eight years ago and had been on the usual treatment circuit: physiotherapy, chiropractic, specialists, acupuncture, and even had tried Blomberg injections.

He described himself as a "grumpy bastard" with an average VAS of 8/10 and had an initial Roland Morris disability (RMDQ) score of 21/24. The pain was sited in his left lower back and buttock and was exacerbated by prolonged sitting and standing, relieved by rest, and not increased with straining. There was no radicular component, no red flags, and moderate analgesic usage of mainly over-the-counter ibuprofen, paracetamol, but also prescribed Panadeine Forte and meloxicam.

Single photon emission CT (SPECT) report indicated "moderately active degenerative change at the right L5-S1 zygoapophysial joint (ZAJ) and left aspect of the L4/5 disc". A trial intra-articular anaesthetic and steroid injection was then given to both the right L5-S1 ZAJ and, after considerable clinical consideration with the patient, the left L4-5 ZAJ. This almost completely relieved his pain, with a follow-up VAS of 2/10 and a RMDQ of 2/24. After about three months when the pain began to recur he elected to proceed to RF ablation of the same two joints (he also had a trial medial branch block as well prior to the RF ablation) and this has also been successful in almost completely relieving his pain. At the time of writing this review, the clinical effect has continued and now exceeds three months.

Literature review

Methods

The literature review is based on a search of Pubmed and Scirus of the terms "spect scan for facet pain OR spect scan for low back pain OR spect sensitivity and specificity for facet pain OR spect scan for zygoapophysial pain".

Results

Search returns numbered 100 of which 26 were selected on the basis of the title being relevant and initial perusal of details. Abstracts were not available for eight, of which three seemed relevant but were not checked further as one was a comment and the other two were from 1995 and 1994.

Summary of literature

There have been several studies indicating that in patients with cLBP between 15% and 40% originates, at least in part, from the ZAJ.¹⁻³ This is demonstrated with use of dual diagnostic double blocks where pain relief is obtained for different periods of time dependent on the anaesthetic given.⁴

However, in 2007 Eubanks et al. in an examination of 647 cadavers showed that lumbar zygoapophysial joint arthrosis is universal (100%) after the age of 60, and indeed one half of males aged less than 30 showed signs of ZAJ arthrosis.⁵ This renders plain x-rays and CT investigation of limited use for diagnosing painful ZAJ.⁶

Thus the clinical problem arises of how to differentiate cLBP patients with symptomatic ZAJ from those with nonsymptomatic arthrosis, and how to determine the anatomical location accurately.

One method is to use SPECT to identify those ZAJ with increased isotope uptake as an indication of increased metabolic activity and by extension as the probable pain generating site.

Literature support for utilising this method of diagnosing symptomatic ZAJ is outlined in Table 1.

Clinical basis for ZAJ pain

Each ZAJ receives dual innervation from medial branches arising from the posterior primary rami at the same level and one level above. Pain can be induced in these joints by injecting such substances as hypertonic saline, or by distension with contrast media in normal patients⁹ and by alleviating pain by injecting local anaesthetic in back pain patients.¹⁰

There is an extensive distribution of small nerve fibers

Table 1. Summary of clinical trials using SPECT as basis for treatment of ZAJ related cLBP

Study	No P	Measure/Investigation	Outcome
Dolan 1996 ⁷	58	SPECT +ve ZAJ injection SPECT -ve - clinical diagnosis ZAJ injection	95% P significant decrease in pain 47% P significant decrease in pain (both at 1 month)
Pneumatics 2006 ⁸	47	GrpA1 - SPECT pos inj GrpA2 - SPECT neg - clinical dx joints inj GrpB - no SPECT clinical dx joints inj	13/15 improved at 1month 2/16 improved at 1month 5/12 improved at 1month

Abbreviations: P = patient, ZAJ = zygoapophysial joint, SPECT = single photon emission CT

and free and encapsulated nerve endings especially in the ZAJ capsule.¹¹ These have been shown to be low threshold mechanoreceptors, mechanically sensitive nociceptors, and silent nociceptors.¹² Inflammation leads to decreased firing threshold and increased baseline discharge in these receptors.¹² Nerve fibres (substance P active) have also been found in the subchondral bone¹³ and the zygoapophysial synovial folds.¹⁴

More recent studies on the neurotrophins and neurotrophin receptors in degenerative ZAJ have revealed the presence of nerve growth factor (NGF) and the receptor for NGF, tyrosine kinase A (TrkA), in 10 patients who underwent surgical treatment for ZAJ pain.¹⁵ NGF is involved in inflammatory processes and has a hyperalgesic effect when administered to rats.¹⁵ Both are thought to play a role in persistent pain.¹⁶ Earlier rat studies showed the presence of substance P and CRGP innervation,¹⁷ and also brain derived neurotrophic factor (BDNF) and vanilloid receptor type 1 (VR1) which are also involved in inflammation.¹⁸

Comparison of planar bone scans versus SPECT

Bone scintigraphy or bone scans have been used medically for many years to detect increased bone metabolic activity and to diagnose such conditions as metastatic tumours, stress fractures, and infection. Increased metabolic activity associated with inflammatory arthritis is also detected by this method.¹⁹ A development from the earlier planar bone scans is a type of scan known as single photon emission CT (SPECT) where a dual head gamma camera is used to create a 3D image. This technique gives improved spatial resolution and provides anatomical localization in the different components of vertebra that is not available with planar scans.²⁰ It also provides improvement in the detection of the number of lesions.²¹

There are several published reports comparing planar scans/SPECT/x-ray/CT, and MRI.

The key study was from Holder (1998) with 43 patients diagnosed with "facet syndrome" on the basis of clinical symptoms and signs and structural findings (x-rays, MRI, and CT).²² This manner of diagnosis is no longer considered adequate, although the final diagnosis of ZAJ pain was reserved until response to injection therapy was assessed. The results of this study (Table 2) can be used to calculate

the accuracy and predictive values of SPECT in the diagnosis of facet joint pain. It has a sensitivity of 100%, a specificity of 71%, a negative predictive value of 1.0, and a positive predictive value of 0.41.

Table 2 SPECT /planar scan result vs ZAJ pain diagnosis (post treatment)

Diagnosis	ZAJ	Not ZAJ
Planar scan: ZAJ abnormal	5	8
ZAJ normal	2	26
SPECT: ZAJ abnormal	7	10
ZAJ normal	0	24

(2 patients refused confirming diagnostic injections thus N=41)

It was previously shown in a study by Ryan et al. in 1992 that SPECT detects about twice as many lesions as planar scans,²⁰ and in a more recent study of 52 patients with cLBP compared x-ray and planar scans with SPECT.²³ Twenty-one patients with normal x-ray had an abnormal SPECT. Of 21 patients with abnormal ZAJ on x-ray only, 14 had abnormal SPECT. Similarly, of 10 patients with an x-ray finding of spondylolysis, five had increased SPECT activity. SPECT detected 60 lesions whereas planar scan detected only 28, a ratio also of about 2:1.

A single case report from 2011 demonstrates that CT and SPECT provide quite different information. The case reported showed gross degenerative change at one level on CT but only minimal SPECT activity, whereas the level of maximal SPECT activity showed only minor CT changes.²⁴

Kanmaz in 1998 also investigated lumbosacral blood flow and blood pool imaging along with SPECT and planar scans.²⁵ He found that 44% of lesions were detected by both SPECT and planar, 24% were better on SPECT, 31% present only on SPECT, and with 0.4% present only with planar scans. The lesions were equally distributed between the vertebral body, disc, ZAJ, and laminae. Once again the conclusion was that SPECT was significantly superior to planar scans.

SPECT has also been used in prevalence studies of cLBP. In a 2010 study of 534 hospital patients with cLBP there was a finding in 486 of at least one SPECT abnormality. There was 42.% prevalence at the ZAJ, 29.8% at the vertebral end plate/vertebral body and 5.9% at the SIJ.²⁶ It is interesting that this corresponds roughly to previous published prevalence

rates determined by the "gold standards" of double blocks for ZAJ and provocative discography.

Compared to planar scans, SPECT can determine whether the lesions are in the vertebral body such as fracture, infection, and metastatic deposits, in the disc space where discitis and disc degeneration can be detected, or the posterior arch where laminae, ZAJ, and pedicles can all be differentiated.²⁷

There have been other reports of using SPECT to assess response to surgery for spondylolysis,²⁸ assessment of post-surgical back pain,²⁹ and for the assessment of discogenic pain.³⁰

The conclusion that can be drawn from these studies is that SPECT will detect considerably more lesions than planar scans alone and can provide better anatomical siting.

Clinical use of SPECT

There were only two trials of note specifically reporting the use of SPECT as the basis of treating ZAJ related cLBP. These are summarised in Table 1.

The study by Dolan et al. involved 58 patients with cLBP suspected of ZAJ basis (pain increased by extension, standing, and sitting, relieved by rest and not increased by straining).⁷ Exclusions included radicular pain, previous surgery, and any of the usual red flag conditions. SPECT was positive in 22 patients and negative in 36. Two patients were subsequently excluded after developing radicular symptoms, and a further two for technical reasons. Therapeutic injections involved 0.5 ml local anaesthetic and steroid mix into the joint space or adjacent if insertion of the needle into the joint was unsuccessful. The results for those patients with positive SPECT scans was a 94% reported improvement at one month (VAS, MPQ, and analgesic intake) versus 47% in the SPECT negative group. Any difference between the two groups had disappeared after six months. Importantly only three of 22 patients with SPECT positive scans had a site of tenderness that corresponded clinically with the scan, illustrating the clinical difficulty of determining anatomical siting.

The next study to report involved 47 patients also with a diagnosis of ZAJ related cLBP on a similar basis to the former study (pain increased by extension, etc.).⁸ There were two groups in a ratio of 2:1. The larger group had a SPECT scan, whereas the smaller control group did not. SPECT positive patients were given a targeted ZAJ steroid and local anaesthetic injection, SPECT negative patients had a clinically determined ZAJ injection, and the control group who did not have a SPECT scan were also given a clinically determined ZAJ injection. Results were significantly improved in the SPECT positive group in terms of number of patients reporting pain relief and reduction in pain scores. The changes in pain scores were statistically significant at one month and three months. There was also a reduction in the number of ZAJ injections administered to the SPECT group versus the other two groups clinically determined. Any statistical difference between the groups had dissipated

by six months. One critique of this study has been that the volume of local anaesthetic of 2.5 ml and 0.5 ml steroid used. Some was injected into the ZAJ joint space and the remainder adjacent to the joint potentially anaesthetising other nearby structures.

This search did not discover any studies addressing the following questions:

1. Do patients without back pain exhibit positive SPECT scans?
2. Do SPECT scans "improve" after treatment with steroid and local anaesthetic or medial branch neurotomy?

Conclusions

Two clinical studies have shown SPECT useful in detecting ZAJ activity as a marker for inflammation and thus a potential pain generating site in cLBP. Patients treated on the basis of SPECT activity seem to have a superior outcome compared to those treated only on a clinically determined basis. It seems to provide a significant improvement on planar scanning in terms of providing useful anatomical siting of active lesions that can then be targeted by therapeutic injections or radiofrequency ablation.

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Literature review of rami communicans blocks for chronic discogenic low back pain

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This paper reviews the current literature relating to the use of anaesthetic blocks and radiofrequency ablation of the rami communicans at the L2 level. It is based on searches of Pubmed and Scirus of keywords "rami communicans", "L2 block", "DRG block L2", "Radiofrequency ablation lumbar back pain", and "lumbar disc innervation". It was prepared with the assistance of the library service at Griffith University.

Summary

Human studies and extensive rat investigations have determined that the innervations of the lumbar intervertebral disc is sympathetic, with segmental and nonsegmental components. There is some evidence that blocking the rami communicans at the L2 or L4/5 level helps relieve chronic low back pain, probably of discogenic origin. One well-designed study did not find any clinical benefit, possibly due to the use of a combination of bupivacaine and clonidine having an antagonistic effect. The treatment has been recommended as suitable for clinical use in appropriate cases.

Introduction

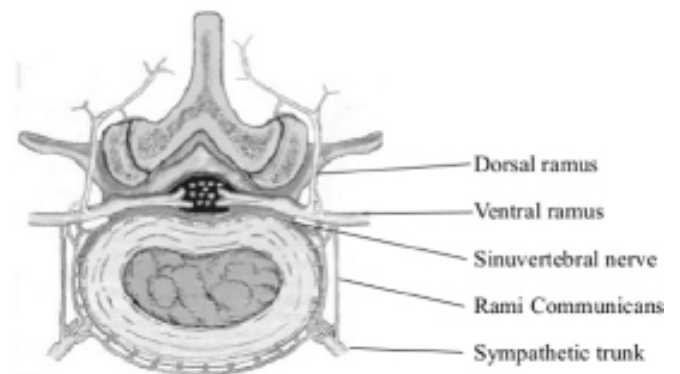
It has been shown in several studies that the prevalence of chronic lumbar back pain due to discogenic causes is approximately 40% using provocative discography.¹ Management of such cases is problematic for many physicians, with treatment options limited to conservative, surgical fusion or disc replacement for severe cases. More recent developments have included intradiscal electrothermal therapy (IDET), with a success rate of about 68% in the latest reports in terms of pain reduction and improved function.² There have also been several reports of successful alleviation of symptoms by anaesthetic block and radiofrequency ablation of the rami communicans both at the L2 level^{3, 4, 5, 6} and also L4 and L5.⁷ There has been one negative study.⁸ The rationale for such treatment is that it has been found in several rat studies that some of the afferent fibres from the lumbar intervertebral discs traverse to the L2 level via the sympathetic chain before re-entering the spinal cord.⁹⁻²⁰ This provides a potential target for treatment via anaesthetic blockade and radiofrequency denervation akin to the techniques used for chronic zygoapophysial lumbar back pain via medial branch block.

Intervertebral disc innervation

The nerve of Luschka, or sinuvertebral nerve, and the innervation of the lumbar intervertebral disc, has a long and colourful history and has attracted the attention of many

notable anatomists.

A simplified diagram of the innervation of the lumbar intervertebral disc (IVD) follows.



Whilst dissection and anatomical description is essential, information as to the actual pain pathways from the disc is not clearly evident. Traditionally, it was considered that the afferent pain fibres returned segmentally but Bogduk et al. in 1981 clearly described the presence of sympathetic fibres innervating the lateral and anterior aspects of the disc.²¹ This raised the possibility of viscerogenic rather than somatic nociception from the disc which would be transmitted via the sympathetic trunk. Much research has continued into the pathways of the afferent innervation of the lumbar discs using modern labelling techniques and there have been a progression of reports mainly from Chiba University in Japan based on the innervation of the rat intervertebral disc. Such studies cannot be undertaken in human subjects for obvious ethical reasons. The rat has a different number of intervertebral discs than the human but it is a useful and readily available animal for research purposes and is considered a good model in terms of mechanical properties,²² neurotrophins,²³ and overall structural arrangement.²⁴

Rat lumbar IVD innervation studies

There are more than 20 published scientific studies on the innervation of the rat lumbar IVD, including anatomical, histochemical, and surgical investigations. Some studies report on the anterior or ventral disc, others on the lateral side and still more on the more clinically relevant dorsal aspect.

The first report is by Kojima in 1990, who reported that the innervation of the posterior longitudinal ligament (PLL) consisted of a superficial and a deep plexus within the intervertebral portion of the PLL and superficial layers of the annulus fibrosus (AF). Whilst there were interconnections between the superficial and deep plexuses on the PLL, the

deep fibres seemed to be unrelated to the meningeal branch, thus indicating a purely sympathetic origin. A follow-up report involved surgical removal of dorsal root ganglia (DRG) or ventral roots.¹⁰ DRG removal resulted in loss of the PLL nerve plexus, whereas removal of the ventral root had no effect. This was confirmed in a later study.¹⁴

The next study demonstrated that the L5-6 disc was innervated by the L2 DRG.¹¹ Administration of Evans blue stain and then injection of capsaicin into the L5-6 disc resulted in extravasation of the dye in the groin area or L2 distribution. The reverse of this, using horse radish peroxidase placed adjacent to the anterior portion of the L5-6 disc, resulted in labelling at the L1 and L2 DRG.¹³

Calcitonin gene related peptide (CRGP) and tyrosine hydroxylase are considered to be markers for nociceptive and noradrenergic fibres, respectively. Staining for these receptors in the PLL suggested a dual innervation – a polysegmental autonomic and a unisegmental somatic.¹²

Further studies using CRGP-immunoreactive (CRGP-ir) staining found that neurons from the Intervertebral Disc (IVD) passed through the sinuvertebral nerve into the rami communicans and not into the segmental spinal nerve.¹⁵ Non-sympathectomised rats had labelling from T13-L6 whereas the sympathectomised rats only had labelling L2-L6. That indicated L5-6 disc innervation from T13, L1, L2 via the sympathetic trunk and L3-6 via sinuvertebral nerves.

This was confirmed in 2001 by Ohtori also using the Fluorogold method.¹⁶

The next question concerned the percentage of the nociceptive neurons which pass to the DRG at L2 level or higher. Using Fluorogold and staining for four types of receptors (CRGP-

ir, substance P-ir, brain-derived neurotrophic factor, and vanilloid receptor type 1), two studies determined that the percentages of neurons in L1 and L2 innervating the L5-6 disc were not different from those at L3, L4, or L5.^{17,18}

In a different centre in 2008, Chen et al. performed selective L2 rami communicans resections in the rat model whilst staining for SP-ir neurons.¹⁹ Using the Fluorogold method labelling of SP-ir neurons decreased after L2 rami communicans resection. SP is also well known to mediate pain transmission.

The latest studies from Chiba further support the multisegmental autonomic innervation model.²⁰ From these multiple rat studies it seems that there is nociceptive transmission from ventral, lateral, and dorsal parts of the L5-6 disc via the sympathetic nervous system up to the T13 level. Between L3 and L6 nociceptive neurons return to the spinal cord segmentally. From L2 and above the rami communicans is the probable route. Neither pathway seems predominant for the more clinically relevant lateral and dorsal disc.

Clinical trials of rami communicans blocks

The first description of the usage of radiofrequency ablation of the rami communicans for discogenic pain was in 1988 by Sluitjer et al.²⁵ He reported a series of 20 patients with ongoing severe low back pain after spinal surgery. He reported a 60% “success” rate based on patient judgement.

Other trials are summarised in Table 1.

Table 1. Summary of trials using rami communicans blocks for chronic low back pain

Study	n	Diagnosis	Process	Measure	Outcome
Nakamura ³	33	Clinical, MRI	Unilateral L2 NR 1.5ml LA	VAS pain with forward flexion	Average VAS decrease 4/10 Effect lasted for 3 hrs to 1 month
Oh & Shim ⁷	49	+PD, failed IDET	Trial block L4 or L5 RC. RFA L4 or L5 Control: LA only	VAS pain SF36 pre and 4/12 post	Decrease VAS 46.5% Increase SF36 49.7% Control: no significant change
Simopoulos ⁴	5	Clinical MRI, +PD	Trial block RFA RC L2 unilateral or bilateral	VAS pain function, Pain medication	Each patient had about 50% decrease pain and improved sitting tolerance and function. Four had reduced analgesic intake Effect lasted for average 4 months
Ohtori ²⁶	68	Clinical MRI	L2 block vs L4 or L5 block	VAS pain	L2 - VAS av 8 -> 4.4 at 1/12 L4 or L5 - VAS 7.8 -> 6.4 at 1/12
Murata ⁵	246	cLBP x 3 yrs - no attempt to diagnose disk	122 patients L2 block 124 patients (control)- IM injection	VAS pain on 100 point scale at 0 & 5 minutes & 1week	Average VAS L2 block: 69 - 14 - 54: Controls: 68 - 62 - 59 In 13 L2 block patients there was some effect up to 24 weeks
Richardson ⁸	12	+PD	DRG block L1 and L2	VAS ADL	No effect at 1 month

Abbreviations

n = number of patients, +PD = positive provocative discography, RFA = radiofrequency ablation, NR = nerve root, cLBP = chronic low back pain Study, RC = rami communicans, LA = local anaesthetic, IDET = intradiscal electrothermal therapy, ADL = activities of daily living, ODI = Oswestry disability index, VAS = visual analogue scale, SF36 = Short Form 36

Chiba University human trials

There have been several trials in patients from Chiba University in Japan. Starting in 1996, Nakamura using clinical criteria and MRI findings, injected 33 patients with 1.5 ml lignocaine at the L2 level.³ In patients with unilateral pain there was a reported decrease in VAS from average of 10 to 1.7. In patients with bilateral pain there was alleviation in ipsilateral pain in five out of ten patients and bilateral pain also in five out of ten. Leg pain was not generally relieved. The effect lasted up to three weeks. In 2008, Ohtori compared L2 nerve root blocks with L4 or L5 blocks in 68 patients.²⁶ The diagnosis of discogenic pain was not according to ISIS criteria using provocative discography as in most of the trials from this centre.²⁷ It consisted of clinical examination with painful forward flexion, and MRI indicating disc degeneration. After nerve root block of 1.5 ml of lignocaine in each of the groups VAS was recorded over three weeks. Each group recorded about a 50% average decrease initially but with a longer effect in the L2 group of about three weeks, as in the Nakamura study. Murata in 2009 administered a L2 block or superficial intramuscular control block to 242 patients with chronic LBP with no attempt at diagnosis of discogenic cause.⁵ There was a noticeable decrease in VAS in the L2 group and not the control group. A few patients in both groups reported VAS decrease for up to six months. Finally, from this group in 2010 was a report of comparison of outcomes following interbody fusion for cLBP.⁶ Prior to fusion each patient was given a L2 block and the effect recorded. At follow-up two years after fusion there were two groups. The group in which fusion was successful in relieving pain had an 80% positive response to the L2 block prior whereas the group in which the fusion did not relieve pain had a 40% response to the L2 block prior to operation.

Unfortunately all of the studies from Chiba, apart from the last, have not confirmed the diagnosis of discogenic back pain with provocative discography and it makes it difficult to assess the significance fully, apart from a general conclusion that injecting at L2 seems to have a clinical effect that might last three weeks or longer.

Other trials

There are four other studies to report. Oh and Shim in a trial, with diagnosis based on positive provocative discography and failed IDET, administered trial blocks and then radiofrequency ablation (RFA) of the rami communicans at the L4 or L5 level.⁷ They recorded significant reductions in VAS (mean reduction 3.3) and increases in SF36 (mean increase 11.3).

The next significant trial was by Simopoulos et al. in 2005.⁴ He performed L2 trial blocks, then RFA, on five patients, with proven discogenic low back pain. There was a >50% improvement in pain and function that lasted for about four months. Each patient had the procedure repeated at least once, so patient satisfaction seems to be evident with the procedure.

Mendez compared unilateral L2 block with provocative

discography in an attempt to determine whether it would be a worthwhile investigative technique.²⁷ Using a 50% reduction in pain as the measure of a positive result for the block there was at best 50% specificity and 43% sensitivity. Bilateral blocks were not performed in this latter study.

Negative report

The trial by Richardson et al. published in 2009 was well designed using patients with proven discogenic pain.⁸ He reported that DRG block at L1 and L2 was completely ineffective up to one month. Each patient was given a combination of bupivacaine, clonidine and methylprednisolone. Clonidine was not used in the other studies. Whilst clonidine is usually considered synergistic in action with local anaesthetics, it is possible that an antagonistic effect was evident here. Hiruma et al. in 2008 demonstrated in cultured rat DRG that clonidine increased and lidocaine decreased axonal transport.²⁸ The inhibitory effects of lidocaine were reduced by simultaneous treatment with clonidine. This might explain the negative effect of the Richardson study. It is unfortunate that this trial did not simply use bupivacaine/lidocaine as patient selection was in accordance with ISIS guidelines and this trial is probably the best designed of the literature discovered by this search.

Reviews

An editorial review of the innervation of the lumbar intervertebral disc by Edgar in 2007 concluded that the sensory pathway is dual, with some segmental and nonsegmental via the sympathetic trunk.²⁹

Kallewaard et al. in a 2010 review rated RFA of the rami communicans at level 2B+ or positive recommendation for clinical use and IDET at 2B+/- which is a level recommended for use preferably related to further study purposes.³⁰

Kosharsky in 2007 whilst not formally rating RFA of the rami communicans opined that it could be offered once a diagnosis of discogenic pain had been reached.³¹

Conclusion

From human studies and extensive rat investigations it seems apparent that the innervation of the lumbar intervertebral disc is sympathetic with segmental and nonsegmental components.

There is some evidence that blocking the rami communicans at the L2 or L4/5 level helps relieve chronic low back pain, probably of discogenic origin.

If further studies confirm the clinical usefulness of L2 rami communicans RFA it could be a useful addition to treatments options for chronic discogenic low back pain if other options are unavailable or not appropriate.

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Headaches and the cervical zygapophysial joints

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Introduction

According to the World Health Organization, patients with severe headaches or migraine suffer the same degree of disability as a patient with quadriplegia or dementia.¹ Population-based studies confirm that the impact of severe headaches on the quality of life is substantially burdensome.^{2, 3} Severe and persistent pain is often associated with profound personal suffering and the increased risk of suicide is not uncommon.^{4, 5} In the Australian context, headaches generate some two million consultations (13%) annually⁶ at a cost of greater than \$700 million per annum.⁷

Definition

Cervicogenic or cervical headache is pain perceived in the head whilst the primary source resides in any structure of the cervical spine. Cervical headaches are due to a referral of pain from the neck and do not imply nerve compression. Facilitated by a system of neuronal convergence within the upper segments of the cervical cord, cervical or cervicogenic headaches typify a form of somatic referred pain that may originate from the zygapophysial joints, intervertebral discs, muscles, ligaments, or other components of the cervical spine.^{8, 9} Hence, unlike migraine or other variants of primary headaches, cervicogenic headache does not represent a distinct neurological entity.

Synonyms

Cervical headache,^{10, 11} cervico-encephalic syndrome, cervical spine syndrome, cervicocephalic syndrome,¹² cervical migraine,^{13, 14} occipital neuralgia,^{15, 16} occipital myalgia-neuralgia syndrome,¹⁷ vertebragenic or spondylogenic headache,¹⁸ post-traumatic muscle contraction headache,¹⁹ residual occipital neuritis,²⁰ great occipital trigeminus,²¹ painful intervertebral dysfunction,²² cervical occipital pain, neuralgic headache,²³ migraine cervicale,²⁴ post-traumatic headache,²⁵ basilar migraine,²⁶ cervical-occipital neuritis/neuralgia²⁷ and reflex tension myalgia.²⁸

Characteristics

Epidemiology

There is very limited validated epidemiological data on either the incidence or the prevalence of cervicogenic headaches. Non-uniform diagnostic criteria confounded by

idiosyncratic ambiguities have severely eroded reliability studies. It is likely that the reported prevalence of 16-60%,^{29-32, 33} irrespective of IASP classification³⁴ is reflective of the contextual environment within which the patients were reviewed. None of the studies implicated a specific component of the cervical spine as the primary source of nociception.

Scientifically, best and most rigorously studied are the cervical zygapophysial joints. A substantial body of anatomical and pathological data now complement the clinical evidence that implicates the cervical zygapophysial joints as the most common source of chronic post-traumatic headache.^{35, 36} By way of comparative diagnostic blocks, Lord et al.³⁷ specifically investigated the co-existence of headaches in patients presenting with post-traumatic cervical zygapophysial joint pain. In what must be the only published randomized double-blind control study, the C2/3 zygapophysial joint yielded a prevalence of 49%, the C5/6 and C4/5 contributed 13% and 4% respectively, 7% was attributed to the C3/4 and 2% to C6/7. Amongst patients in whom headache was a dominant feature, the C2/3 synovial joint accounted for 53%. These results were replicated in a recent independent study.³⁸ Where the cervical disc was the putative source, more than 60% of respondents undergoing surgical fusion of the cervical spine reported that headache had been the dominant symptom.³⁹

Causes and differential diagnosis

Heterogeneous group

By far the commonest cause is trauma to the cervical spine either recent or remote.^{35, 39-43} Headache may occur prodromally in vertebral artery dissection,⁴⁴ vascular compression of C2 cervical root,⁴⁵ meningioma,⁴⁶ cervical cord infarction,⁴⁷ spontaneous dissection of the carotid artery,⁴⁸ and Chiari type malformation.⁴⁹

Neuralgias

The common perception that *occipital neuralgia* is due to entrapment of the greater occipital nerve has been challenged. Anatomical studies have disputed the putative pathomechanism,^{50, 51} and hence surgical extrication of the nerve and its ganglion may not be justified.²⁷ Unlike occipital neuralgia, however, C2 neuralgia is a distinctive form of headache caused by lesions affecting the C2 spinal nerve (for example, venous or arterial anomalies),^{52, 53} A separate disorder, neck tongue syndrome is characteristically recognized by acute unilateral occipital pain associated with simultaneous ipsilateral numbness of the tongue.⁵⁴ This

occurs because the second cervical root is compressed in the atlanto-axial space by sharply rotating the neck.⁵⁵

Cervical intervertebral discs

As a source of cervicogenic headache, the intervertebral discs have been implicated in a few observational studies. Following cervical fusion, a proportion of patients reported complete relief of their headaches whilst in others relief had been incomplete.⁵⁶⁻⁶⁰

There are no clinical features by which the disc could be implicated as a source of pain to the exclusion of other structures. As a diagnostic utility, provocation discography lacks specificity,⁵⁸⁻⁶¹ and has a high false positive rate.^{62,63} Not only does discography reveal symptomatic discs at multiple levels, it is also uncommon to find discogenic pain limited to a single level.⁶⁴⁻⁶⁵

Muscle pain

Despite the lack of anatomical, histological, biochemical, and electrophysiological evidence,^{65, 67} notions such as “muscle spasm” and “pain-spasm-pain cycle” have been widely publicized as potential causes of headaches.⁶⁸⁻⁷⁰ Contrary to published evidence,⁷¹⁻⁷⁶ as a cause of headache, muscle spasm, either as an autonomous clinical entity or as a manifestation of myofascial syndrome, currently enjoys an increasing resurgence amongst practitioners who advocate bacterial toxin as a therapeutic agent.⁷⁷⁻⁷⁹ Several of the tender spots, ascribed to “trigger points” of the myofascial variety, overlie cervical zygapophysial joints.⁸⁰ Hence, the alleged muscle tenderness or hyperalgesia is most likely a manifestation of central sensitization, with the zygapophysial joints being the continual source of primary nociception.⁸¹⁻⁸⁶ Consequently, to avoid misdiagnosis and hence inappropriate treatment, the distinction between “trigger points” and painful zygapophysial joints is paramount. In the absence of any plausible biological mechanism, the treatment of “trigger point syndrome” is entirely empirical. Intralesional administration of local anaesthetic may offer short-term palliation but there are no scientific studies that would attest to its long-term efficacy. A systematic review found that neither “dry” nor “wet needling” – irrespective of the injectate – was better than placebo.⁸⁷

Zygapophysial joints

Zygapophysial joints: the evidence

To be a source of headache, cervical spinal structures should

- have a nerve supply
- evoke headaches upon stimulation,
- relieve the headache when anaesthetized
- be ideally described in anatomical, biomechanical, radiological, and pathological terms;⁸⁸ and,
- treatment should confer complete and prolonged relief of pain.⁸⁹

Neuroanatomy

Peripheral. Archetypical of somatic referred pain (that is, somatic origin of pain as distinct from visceral origin of

referred pain) headache may arise in any of the cervical structures and the pattern of referral is primarily dependent upon the peripheral distribution of the upper three cervical nerves; hence, any structure innervated by the first three cervical nerves can be a potential source. Known sources include the suboccipital and upper posterior neck muscles,^{90,91} the intervertebral discs,^{92,93} the synovial joints including the C2/3;^{90,91} the joints and ligaments of the median atlanto-occipital joint and the lateral atlanto-axial joints,⁹⁴ the spinal dura mater,^{95,96} the vertebral artery,²⁴ the C2 nerve root/ganglion,⁹⁷ the synovium and facet joint capsules,⁹⁸⁻¹⁰² and structures within the posterior cranial fossa.¹⁰³ Neurohistochemical techniques have confirmed the presence of small diameter nerve fibres (nociceptors) and mechanoreceptors in the synovium and the synovial joint capsules,⁹⁸⁻¹⁰² thus providing the neuronal circuitry not only for proprioception but also for the transmission of pain impulses.

Central connections. Fundamental to the genesis of cervical headache is the trigemino-cervical nucleus, a column of grey matter formed by the pars caudalis of the spinal nucleus of the trigeminal nerve and the grey matter of at least the upper three cervical spinal cord segments (Fig. 1).⁸ The supratentorial pain producing structures are innervated by the first division of the trigeminal (ophthalmic) nerve, the fibres of which pass through the trigeminal nerve root and descend to the level of C1/2 spinal cord segment to synapse on second order neurons in the trigemino-cervical nucleus.¹⁰⁴ Infratentorial and cervical structures are largely innervated by the C2 and upper three cervical nerves respectively.^{8,90,105} The latter in turn synapse over the same range of trigemino-cervical nucleus as the ophthalmic nerve. Prior to entering the substantia gelatinosa, the C2 afferents extend caudally to the third spinal segments and proximally to the pyramidal decussation, whilst the C3 afferents may extend superiorly as far as the corresponding fibres of C2.¹⁰⁵ This arrangement allows nociceptive afferents from the trigeminal nerve and from the first three cervical spinal nerves to form multiple collateral channels which converge onto common second order neurons¹⁰⁶ and it is this neural

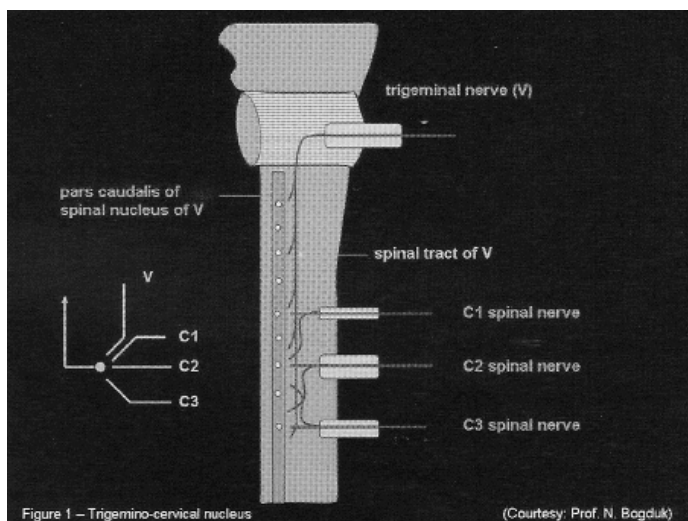


Figure 1 – Trigemino-cervical nucleus

(Courtesy: Prof. N. Bogduk)

circuitry of “convergence” that facilitates the perception of referred pain. Hence, stimulation of one group of afferents may be perceived as arising from a region innervated by another group. Referred pain therefore is a *sensory illusion* that results from such convergent circuitry and does not imply spinal nerve compression.¹⁰⁷ Perceptually the brain is unable to distinguish whether the incoming information from the second order neuron was initiated by the vertebral afferent or other convergent fibres and hence attributes its origin to both.¹⁰⁸ Spinal referred pain is nociceptive in nature, that is, the pain is initiated by stimulation of nerve endings of afferent fibres and this peripheral stimulation distinguishes it from neurogenic and radicular pain.¹⁰⁸ Afferent fibres from the region of referred pain are not stimulated by the causative agent.¹⁰⁸

Once they have passed through the Gasserian/trigeminal ganglion, trigeminal afferents that convey pain and temperature do not synapse with second order neurons at the pontine level of the brain stem, but instead all three divisions turn inferiorly within the brain stem as the descending tract of the trigeminal nerve and synapse with the second order neurons principally within the upper cervical spinal cord.^{109,110} Whilst fibres from all three divisions can be found as low as the second cervical segment,¹⁰⁶ fibres from the ophthalmic division within the pars caudalis can be found as low as the fourth or fifth cervical segment.¹¹¹ Furthermore, cervical afferent fibres from as low as C4 and probably C5 are found in the dorsal horn at the second cervical level.^{106,111}

The ophthalmic division extends most caudally and is most densely represented. Such a distribution facilitates greater convergence between the cervical and trigeminal afferents. Hence, somatic referred pain is more likely to be perceived as frontal headaches.

Although not a distinct anatomical entity, there is electrophysiological evidence that establishes the interaction between the upper cervical dorsal root and the trigeminal system. Neurons in the C1 and C2 segments correspond to a stimulation of afferents in both the upper cervical spinal nerves and the trigeminal nerve.^{112, 113}

The spinal trigeminal tract also receives a small complement of fibres from the facial (7th), glossopharyngeal (9th) and the vagus (10th) nerves.¹⁰⁹ Cumulatively, such an arrangement establishes the trigemino-cervical nucleus as the essential nociceptive nucleus of the head, throat, and upper neck.⁸

Pain provocation

Patterns of referred pain described in clinical studies have been replicated in normal volunteers. By injecting hypertonic saline into the suboccipital muscles, Cyriax¹¹⁴ reported that the pattern of pain referred to the head is contingent upon the site of stimulation: the more cranial the site of injection the closer to the forehead the pain projected, whereas stimulation below the occiput confined projection of pain to the upper end of the cervical muscles. Other investigators reproduced patterns of referred pain in the orbital and frontal regions by either dry-needling the periosteum of the occipital condyle or by injecting hypertonic saline.^{115,116} Stimulation of the greater occipital nerve in experimental

animals increased the metabolic activity in the ipsilateral caudal brain stem, upper cervical cord, and in the dorsal horn at the level of C1 and C2.¹¹⁷ The neuronal activation appeared contiguous with the pars caudalis and was in the same distribution when trigeminally innervated structures were stimulated.¹¹⁸

Referred pain of a similar pattern can be evoked when certain cervical joints are stimulated. Normal volunteers who had their atlanto-occipital, lateral atlanto-axial,¹¹⁹ the C2/3, and their C3/4 zygapophysial joint capsules¹²⁰ distended with contrast medium perceived pain to originate in the occipital and suboccipital regions. All joint referral patterns were ipsilateral. The C2/3 pattern was distinguished from that of C3/4 by the former's extension into the head. The onset of pain coincided with capsular distension. Stimulation of the lower zygapophysial joints failed to refer pain to the head. Fukui et al.¹²¹ not only replicated the pain pattern first described by Dwyer et al.¹²⁰ in a similar manner, but, in the same cohort of normal volunteers, reproduced the same patterns of pain by electrically stimulating the medial branches of their respective cervical dorsal rami.

Relief of pain

Whilst many of the earlier reports attested to the efficacy of local anaesthetics in relieving headaches ostensibly originating in the cervical spine,^{22, 124} none identified or implicated a specific cervical structure as the primary locus of somatic referred pain. Irrespective of this, the zygapophysial and atlanto-axial joints were often prime contenders.^{112, 125} A semblance of target specificity was reported by Hunter et al.¹²⁶ Patients who had local anaesthetic injected directly onto the second cervical root for suspected occipital neuralgia reported greater relief of pain than those in whom local anaesthetic was infiltrated around the peripheral distribution of the greater occipital nerve.

Fluoroscopy now permits the precise deposition of minute amounts of local anaesthetic either directly into the joints or selectively onto the nerves that innervate them. A number of clinical studies in which headache has been relieved by selectively anaesthetizing the intervertebral joints complement observations in normal volunteers. Identified sources of joint pain include the atlanto-occipital joints,^{119,127, 128} the lateral atlanto-axial joints,^{127, 131} and the C2/3 zygapophysial joint.¹³²⁻¹³⁵ Similar outcomes were reported in patients presenting with upper cervical rheumatoid arthritis¹³⁶ and osteoarthritis of the atlanto-odontoid joints.¹³⁷⁻¹³⁹ A controlled study identified C2/3 zygapophysial joint to be the most common source of headache after whiplash injury.¹⁴⁰ Collectively, of all the possible causes of cervicogenic headache the cervical zygapophysial joints have been most extensively studied and remain the commonest source of cervical pain referred to the head.

Biomechanical aberrations

Evidentiary confirmation that components of the cervical spine undergo considerable kinematical and biomechanical aberrations following simulated whiplash injury have been secured from studies in cadavers¹⁴¹⁻¹⁴² and in normal human

volunteers.¹⁴³ Rapid sequence cineradiography confirmed that nearly all structures of the cervical spine are subject to a complex array of injurious perturbations¹⁴³ long before the protective function of the cervical musculature is called into action.¹⁴⁴ Within a few milliseconds of simulating a rear-end collision, physical forces impacting upon the thoracic spine are cranially deflected. Such a deflection initially causes the lower cervical segments to extend, which in turn cause the upper segments to flex, thus creating an “s-shaped curve”.

With continuing application of whiplash loading, the inertia of the head catches up with the cervical spine and “whips” the entire head-neck complex into extension,¹⁴⁵ compressing the lower zygapophysial/facet joints as it slides anteriorly. The additive effects of simultaneous distraction (avulsion), compression, translation (sliding) and torsion have the potential to generate subtle injuries to both the intra- and extra-articular components of the cervical spine^{141, 146, 147} injuries, which may not be visualized radiographically.¹⁴⁸

Radiological evidence

Plain radiographs are generally insensitive for detecting subtle injuries including osseous lesions.¹⁴⁸ Several retrospective and contemporary clinical studies have verified that fractures of the articular pillars might have been overlooked and hence under-reported.¹⁴⁹⁻¹⁵⁴ Both conventional^{152, 153} and high-resolution computerized tomography¹⁵⁴ is more reliable in detecting occult fractures than plain x-rays.

Improved sensitivity with corresponding reduction in the false-negative rates could be achieved by adopting specific projections, for example, “pillar views”.¹⁵⁴ Similarly, reconstructing the injury mechanism with its biomechanical implications may predict the nature of lesions; for example, compressive fractures to the atlanto-axial complex with possible subluxation following acute lateral flexion of the cervical spine.¹⁵⁵

With the advent of magnetic resonance imaging (MRI), injuries previously deemed elusive and “non-demonstrable” are becoming more evident. Magnetic resonance imaging is invaluable in demonstrating pre- and paravertebral haemorrhage, oedema including cord oedema, occult end-plate fractures, tears of anterior and posterior longitudinal ligaments, and tears of the anterior anulus.^{156, 157} The fibres of the anterior longitudinal ligament are firmly fixed to the intervertebral disc and the periosteum of the vertebral body;¹⁵⁸ hence sudden distraction may cause simultaneous avulsive tears to the fibres of the anterior anulus, the anterior longitudinal ligament and vertebral end-plates fractures with intra discal haemorrhage.^{156, 157}

Surgical evidence

Surgery for the treatment of cervicogenic headaches has not been subjected to randomized studies. Logistically and ethically, devising such studies would be severely hampered by epidemiological and other constraints. Irrespective of this, a not-too-generous interpretation of the “N=1” study design¹⁵⁹ does allow meaningful information to be harnessed from the available data. As such, the published observational

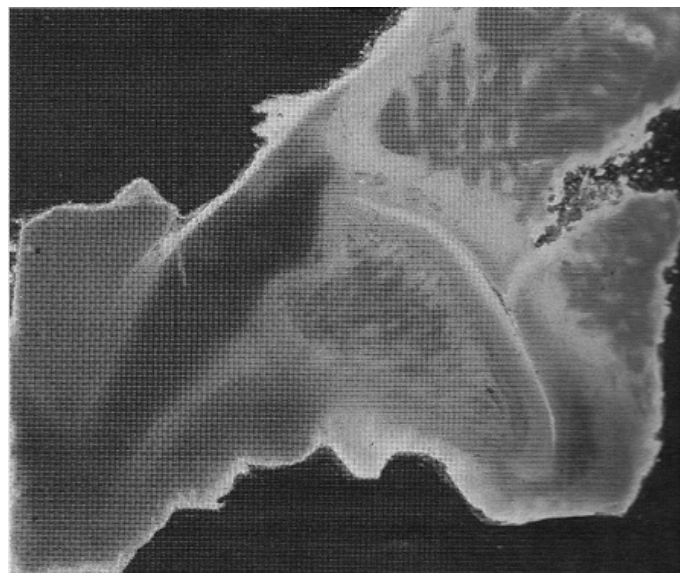
studies are neither distracting nor discouraging. A surgico-anatomic-pathological relationship between the C2/3 zygapophysial joint, the third occipital nerve and occipital headaches was first reported by Trevor-Jones.¹⁶⁰ Patients who had their third occipital nerve surgically decompressed reported complete relief of their occipital headaches. At operation, the third occipital nerve was seen to be entrapped by an osteophytic mass. Similar outcomes were reported by Poletti¹⁶¹ some 19 years later.

In unrelated studies where occipital headaches were attributed to osteoarthritis of the lateral atlanto-axial joints,¹⁶²⁻¹⁶⁵ a significant proportion of patients secured complete relief of their headaches for as long as seven years following surgical fusion of these joints. In all cases, the suspected anatomical locus of pain was radiologically evident. Presurgically, total relief of the index pain was achieved with intra-articular deposition of local anaesthetic and surgical fusion conferred long-term relief. Clinically, diagnostically, and therapeutically, these outcomes fully satisfy the revised criteria and caveats for the diagnosis of cervicogenic headaches, recently enunciated by the International Headache Society.⁸⁹

Pathology

Even with the advent of MRI, certain injuries to the soft tissue elements of the cervical synovial joints may not be overtly evident. Tears of the joint capsules,¹⁶⁶ loss of articular cartilage with eburnation, and proliferation of subchondral bone¹⁶⁷ have been confirmed at operations in symptomatic individuals. The aggregate data from experimental studies, cadavers, radiological and postmortem findings constitute a substantial body of evidence which confirms that the cervical zygapophysial joints and surrounding soft tissues are damaged in whiplash injury. Autopsies on victims of motor vehicle accidents who had died from unrelated causes revealed injuries to the joint capsules and their respective meniscoids; neither these nor the small fractures were identified on imaging (Plate 2).¹⁶⁸⁻¹⁷⁰ Complementary

Plate 2. Facet fracture not seen on x-rays. Courtesy Prof. J. Taylor, ref. 171.



studies confirmed that no tissue structure was immune from injury. Equally, independent observers reaffirmed that in some instances, fractures of the articular pillars, tears of the anterior annulus, bruising of the vascular synovial folds (menisci), haemarthrosis, various grades of vascular injuries, haemorrhage within the C2 spinal nerve and/or the dorsal root ganglion^{169, 173} could neither be confirmed nor discounted either clinically or radiographically.

Current understanding on pain pathophysiology would irrevocably implicate such injuries as potent sources of chronic nociception, capable of generating somatic and neuropathic pain.^{174, 175} There are no valid clinical signs, electrophysiological or radiological abnormalities by which such diagnoses can be refuted. Excessive reliance on x-rays to isolate a pain focus is erroneous and fallacious, and the concept of the so-called, "nondemonstrable injury" is best abandoned. Where organicity is elusive or inconspicuous, it behoves every practitioner to exercise considerable restraint rather than to impugn the patient's veracity and invoke a psychiatric diagnosis of a dubious nature.^{176, 177}

Diagnosis

In 2004, the International Headache Society (IHS) revised its diagnostic criteria for cervicogenic headache.⁸⁹ It required, *inter alia*, that the suspected cervical lesion be identified either by a biological marker or by imaging, and that complete relief of headache be secured by anaesthetizing – either the structure itself or the nerves which innervate it. Prior to its publication, the two major competing philosophies relied entirely on clinical features.¹⁷⁸⁻¹⁸⁰ Neither has been validated,¹⁸¹⁻¹⁸⁶ nor are the features described unique to headaches of cervical origin. Similar features including allodynia¹⁸⁷ have been described in migraine, tension headache, cluster headaches,¹⁸¹⁻¹⁸⁸ and drug-induced headache.^{184, 188} In one study, 44% of patients attending a pain facility had more than one headache diagnosis whilst more than 70% of the cervicogenic group fulfilled diagnostic criteria for migraine.¹⁸⁵ In sharing the same physiological mechanisms, it is not surprising that primary

headaches and headache of cervical origin have similar clinical presentations.¹⁸⁶

To date, only controlled diagnostic blocks have been validated by which cervical sources of headaches can be isolated. Local anaesthetic may be injected into the putative pain source, or onto the nerves that innervate it. The primary objective is to locate the source by relieving the pain.⁴⁰ Bogduk and Marsland¹³² were the first to devise and describe the concept of comparative diagnostic nerve blocks. When properly executed, two important variables emerge, namely, the relief of pain and the duration of such relief. Under normal circumstances, injecting a short-acting agent (for example, lidocaine) would relieve pain for a short duration, whilst a long-acting agent (for example, bupivacaine) would relieve pain for a longer duration, a paradigm ratified in subsequent studies.^{37, 189} Under fluoroscopic guidance, minute amounts of local anaesthetic can be injected into any of the upper cervical joints or onto the nerves that innervate them (Plate 3).^{37, 189} To minimize false-positive responses or other placebo effects, the use of controls is inescapable.

Neither the history, physical examination nor the imaging studies can distinguish between the various sources of cervical pain. Comparative diagnostic block is the only valid modality that can precisely localize the cervical source of pain,¹⁹⁰ including the atlanto-occipital joints,^{119, 127} the lateral atlanto-axial joints,^{119, 127-130} the C2 spinal nerve in C2 neuralgia,^{53, 191} and the C2/3 zygapophysial joint.^{132, 133}

There are limitations. Not every structure is accessible to diagnostic blocks. False positive responses generated by patients' expectations or physician bias can be avoided by having the patient assessed by an independent observer who is blinded to the nature of the injectate. Some patients have a paradoxically long response to short-acting anaesthetics; for example, with lignocaine pain relief may last for days.¹⁹²

Treatment

Physical therapies

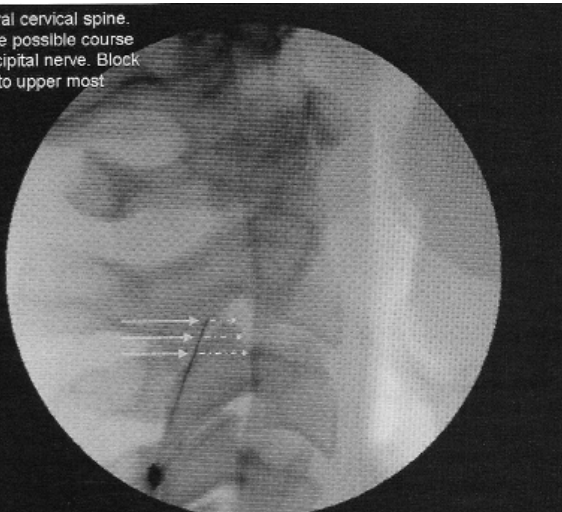
Although routinely prescribed and generously administered, physical modalities such as ultrasound, laser, or interferential do not exert any lasting therapeutic effect. The biological mechanism by which these modalities putatively operate awaits scientific validation, but what does emerge consistently is that these modalities are no better than placebo.^{193, 194}

Manual therapy

For manual therapy, the evidence is inconclusive, ambiguous, and inconsistent. Studies are replete with methodological shortcomings.¹⁹⁵ One study reported that in the short-term manipulation was more effective than massage in reducing the frequency and severity of headaches,¹⁹⁶ and when combined with a non-steroidal anti-inflammatory, the modest gains did not extend beyond two weeks.¹⁹⁷

More recently, a controlled trial of six weeks' duration randomly assigned participants to receive manipulative therapy, specific exercises or both.¹⁹⁸ A week after completion, 76% reported at least 50% reduction in headache frequency

Plate 3 – Lateral cervical spine. Arrows indicate possible course of the third occipital nerve. Block needle points to upper most position



and 35% reported a complete relief of headache. Although at 12 months 72% reported at least a 50% reduction in headache frequency, 24% sought additional or alternative treatment during the study period and 25% failed to achieve any benefit.¹⁹⁹ The physiological mechanism by which improvement was secured was not explained. Claims that spinal manipulation is an effective treatment for cervicogenic headaches is undermined by considerable methodological limitations and it remains unclear to what extent the reported positive effects can be explained by manipulation or other non-specific factors.²⁰⁰

Injection therapy

Diagnostic

Comparative diagnostic blocks may assist in isolating the source of pain and hence in establishing an anatomical diagnosis. In certain instances, blocks may determine the nature of treatment, predict its outcome and test the validity of clinical inferences.²⁰¹

Therapeutic

Local anaesthetics. That local anaesthetics can confer substantial and long-lasting relief has been widely reported,^{23, 38, 113, 202} but formal studies validating its adoption as a primary therapeutic agent are wanting. Irrespective of this, the relief of headache by peripheral nerve blocks not only questions the legitimacy of neuronal convergence but also imputes a newer and different but undefined neuromodulatory effect.²⁰³

Steroids. For occipital tenderness, steroids have been injected pericranially, ostensibly for its anti-inflammatory effect.^{30, 204} However, the evidence for an inflammatory reaction has not been demonstrated. In-vivo studies have confirmed that tender points lack the typical inflammatory mediators and metabolites and are not sites of ongoing inflammation.²⁰⁵ Intra-articular steroids on the other hand are target specific and seem to exert a modest but appreciable beneficial effect. A small-uncontrolled study showed that patients who suffered chronic daily headaches, and in whom diagnostic blocks were positive, steroids resulted in total abolition of their headaches in 11% for as long as 19 months.¹³⁴ In a further 50%, frequency of headaches was reduced to three per month, and these responded to oral analgesics.

From a biological and epidemiological perspective, a number of confounders negate the use of epidural steroids either diagnostically or therapeutically.^{206, 207}

Botulinum toxin. Its mechanism of action is dissonant with the known pathophysiology of cervicogenic headaches. For the treatment of cervicogenic headache there are no supportive data.^{208, 209}

Prolotherapy. There are no data specifically for the management of cervicogenic headaches.

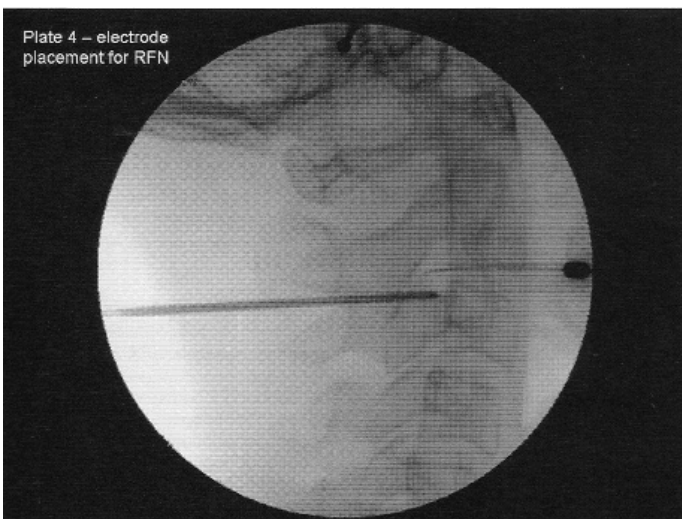
Radiofrequency neurotomy

As a therapeutic agent, thermal radiofrequency neurotomy (RFN) creates a mechanical barrier to the transmission of pain impulses emanating from a known source of pain. It achieves the desired effect by coagulating the nerve/s which innervate that structure. Thermal RFN is not cauterization. It involves placing the exposed tip of a Teflon-covered electrode adjacent and parallel to the targeted nerve. High frequency low energy electricity conducted through the electrode generates heat in the surrounding tissues, which in turn coagulates the nerve.²¹⁰ Radiofrequency does not offer a permanent cure. After a while, the nerve regenerates and pain may return. Relief may be reinstated by repeating the procedure. It is thus far the only established valid means of conferring complete relief of pain for a durable period.

One form of cervicogenic headache where RFN has been most successful is in the treatment of third occipital headache.¹³² Third occipital headache or the headache of Bogduk²¹¹ is so named because the headache perceived is transmitted by the superficial branch of the C3 dorsal ramus, the third occipital nerve. This nerve crosses the lateral and posterior aspect of the C2/3 zygapophysial joint and furnishes articular branches to the underlying joint. It is the sole source of innervations of this joint.⁹⁰ Anaesthetizing the third occipital nerve with minute aliquots of local anaesthetic can be used as a test for pain emanating from the C2/3 zygapophysial joint. Unlike the dorsal rami at lower levels, the third occipital nerve is a large single nerve and it is the only dorsal ramus that crosses the joint line. The C2/3 zygapophysial joint has been shown to be the commonest source of post-traumatic cervicogenic headache.³⁷

There are no clinical features by which pain from the C2/3 joint can be diagnosed and diagnostic blocks are the only means by which a diagnosis could be secured with certainty.

For the treatment of third occipital headache, RFN offers the best-published outcomes (Plate 4). When meticulously executed, complete relief pain can be achieved in at least 88% of patients, in whom the medium duration of complete relief was 297 days,²¹² with some patients reporting continuing relief at the time of review. Once the nerve regenerates and



should pain return, the procedure could be repeated and relief reinstated. In some patients in whom the procedure was repeated, the sustained relief of headache was achievable for more than two years. Unlike the primary headaches, third occipital headache is one form of cervicogenic headache that can be diagnosed with certainty and for which valid treatment is available. The cardinal indication for the procedure is complete relief of pain following controlled diagnostic blocks,²¹³ which makes RFN a logical procedure.²⁸

Conclusion

Amongst the reasons headache treatment fails, incorrect diagnosis, physician bias and the failure to recognize secondary disorders predominate.^{214, 215} Cervicogenic headache is not a neurological disorder. It is one form of somatic referred pain (*sensory illusion*) and not unlike arm pain associated with cardiac ischaemia.²¹⁶ It has a distinct pathomechanism. This convergence of cranial and upper cervical afferents into a common synaptic region provides a logical explanation for the phenomenon of *referred pain* in the head and neck.²¹⁷ Primary headaches (for example, migraine) on the other hand are driven from the brain,²¹⁸ and the headache perceived reflects the interaction between nerves and blood vessels.²¹⁸

Such *neurovascular headaches* have a unique pathophysiology and their clinical manifestations including contra-lateral limb allodynia point to a higher neuronal involvement.^{219, 220} Given this fundamental difference, the insistence that cervicogenic headache be validated by objective physical signs, electrophysiological and/or radiological abnormalities²²¹ is spurious and no longer tenable. Morphological changes seen on imaging studies correlate poorly with symptoms and this inconsistency has been replicated.²²²⁻²²⁶

Compared with primary headaches, cervicogenic headaches and in particular the third occipital headache is best understood anatomically and physiologically. A common disorder, contemporary evidence implicates the cervical zygapophysial joints as the most common source of post-traumatic headaches. Similar data for intervertebral disc, muscles, and other structures that constitute the cervical spine are not available. Cumulatively, data derived from experiments, clinical observations, radiological and post-mortem studies are irrefutable. Clinical features are neither valid nor reliable and as a diagnostic criterion, only controlled diagnostic blocks have survived scientific scrutiny. To ignore the evidence is to deny patients legitimate and valid treatment. It is likely that such iatrogenic behaviour may invoke certain legal imperatives.²²⁷

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Shoulder pain – What ultrasound imaging reveals

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This is the final report of stage I of the Shoulder Imaging Project financed by the Commonwealth Department of Health and Ageing.

Preamble

Shoulder pain is third behind back and neck pain as a musculoskeletal reason for presenting to general practice, with 10% of the adult population presenting with shoulder pain at least once in a lifetime.^{1,2} Despite the fact that 50% of acute shoulder pain resolves in 8-10 weeks many patients present with the anticipation of having some kind of imaging. The reason pain may persist beyond three months is strongly related to personality traits, coping style, and occupational factors.^{3,4}

Plain radiography is uninformative in the assessment of acute shoulder pain, with questionable worth for detecting neoplasia.^{5,6} Ultrasound is highly dependent on the operator and cannot be interpreted effectively by subsequent viewing of the films, but is accurate in detecting tears even though many tears are asymptomatic.^{7,8}

Introduction

Over recent years the Department of Health and Ageing has identified a marked increase in the use of ultrasound imaging for problems relating to the shoulder. In the first quarter of 2001 shoulder ultrasound was half of all musculoskeletal ultrasound. The reason for this sudden increase is likely to be multifactorial, but the important question to ask is whether such an increase is justified in terms of the management outcome for shoulder pain.

A project devised in three parts was initiated by the Musculoskeletal Ultrasound Project Team and operated through the auspices of the Musculoskeletal Foundation of Australia. Stage I was to see what was written on the request form and then to compare this with the radiologist's report. Stage II compared 100 shoulder pain presentations in general practice who had no imaging with 100 patients who had imaging. Several parameters were used for comparison between the two groups including the outcomes of management therapies. Stage III is to involve academic detailing to GPs in an urban Division of General Practice. Subsequent ordering of imaging modalities will then be measured and compared against the profile of a similar socioeconomic division.

This paper reports in detail the findings of Stage I, which was undertaken with a view to:

- Assessing the degree of information on the referral request which might reflect the reason for the referral;

- Correlating the clinical data on the request form with the findings from the ultrasound assessment;
- Providing recommendation to referrers on means to reassess the reasons for ultrasound musculoskeletal imaging.

Method

Approximately 400 shoulder ultrasound request forms and reports were randomly selected from four radiology practices over the same three-month period. Two practices were in Adelaide and one each from Melbourne and Sydney. Both the request and report were anonymous for the patient, referring doctor, and reporting radiologist. The analysis included the patient's sex and age, as well as whether the referral was from a GP or a specialist.

Reports were excluded if the patient was sent solely for injection of a joint or some shoulder structure, under ultrasound control. Requests were rejected when it was not possible to determine what was written on the request form. No other exclusion criteria were applied. The data were put on spreadsheets and judgements made to categorise the report findings, as terminology varied from state to state.

Results

Although 386 requests and reports were received, several were of other anatomical areas and so deleted. In all, 329 requests and reports were analysed, 176 being female and 150 male, while three showed neither age nor gender. The average age overall was 54.6 years, with the average age for females being 56.7 years and that for males 52.1 years.

For the most part, pathology was found in the subacromial space associated with the rotator cuff and subacromial bursa with 80% of reports indicating a finding of some pathology in the structures imaged.

There was one fracture of the greater tuberosity, three patients with adhesive capsulitis, four with disruptions of the A/C joint, and eight with swellings around the long head of biceps.

It was assumed that a clinical diagnosis of dysfunctional shoulder was made which precipitated the request for an ultrasound assessment. However, the analysis revealed that 20% (66) of the reports could not find any structural abnormality to explain the patient's discomfort while 34%

(113) requests contained no contributory history for the radiologist. This latter group included 29 requests (9%) in which “shoulder pain” was all that was written. For this group of 113 requests, 90 (80%) had noticeable abnormality, a proportion that was true of the study as a whole and also true in the case where a significant history was included on the request form (see Table 1).

Pathology Found

History helpful	Yes	%	No	%	Total	%
Yes	173	53	43	13	216	66
No	90	27	23	7	113	34
	263	80	66	20	329	100

Table 1 compares whether requests with examination findings or diagnosis have more pick-ups on pathology than an ineffective request form.

A diagnosis was suggested on 169 (51%) of the request forms, with pathology shown in 80% of these patients. The accuracy or correctness of the diagnosis from the request form was 136/263 or 52%. A chi-square value of 0.028 is non-significant as seen in Table 2.

Pathology Found

History helpful	Yes	%	No	%	Total	%
Yes	136	41	33	10	169	51
No	127	39	33	10	160	49
	263	80	66	20	329	100

Table 2 provides a correlation of suggested diagnosis versus the dysfunctional anatomy.

The correlation between the correctness of the diagnosis by the specialist and GP is shown in Table 3. This demonstrates that a specialist is correct 31/77 or 40% of the time, while the GP is correct on 24/111 or 22% of occasions. Despite the apparent difference between the specialist and GP diagnosis, the chi-square value was again not significant.

Request Type	No Diagnosis		Correct		Not Correct		Total	
	n	%	n	%	n	%	n	%
Unknown	78	24	31	9	32	10	141	43
GP	63	19	24	7	24	7	111	34
Specialist	19	6	31	9	27	8	77	23
	160	49	86	25	83	25	329	100

Table 3 identifies the proportion of correct referrals between GPs and specialists.

In terms of tears, 123 (34%) overall were reported, of which 89 (72%) were partial and 34 (28%) complete, the average age of the patient with a partial tear being 63.1 years, and the complete tear being 73.6 years. Incomplete tears were shown equally between males and females while complete tears were in the ratio 2:1 females v. males.

An additional note of interest is that an MRI was recommended in 13 of the 329 reports and all of these were

in Sydney. The study is too small to allow any conclusions, only speculation.

Discussion

In discussing the results it was beyond the scope of the study to attempt to correlate cause of pain/dysfunction with pathology found on ultrasound imaging.

Nearly one-third (34%) of requests for ultrasound imaging of the shoulder contained no tangible information to assist the radiological examination. There was a noticeable sparsity of information on the request forms as regard the results of a clinical examination such as whether the pain is anterior, posterior or superior, or history of onset, or ranges of movement in flexion, extension and abduction, etc.

The data would indicate that pathology is found in 80% of all reports whether adequate clinical data is provided or not. Given more clinical information it would be reasonable to argue that a more definitive examination could be undertaken by the sonographer/sonologist that may then provide cause for improved clinical management.

When a clinical diagnosis is provided, the degree of accuracy with the ultrasound findings is 40% in the hands of the specialists compared to 86/329 or 26% overall and 22% for GPs. This begs the question as to whether the clinical examination has been rushed and insufficient care taken to complete the findings. However, if an informed history accompanied the request, the proportion of the presence of pathology was the same. The important question to ask is how much do pathological findings correlate with the patient's pain. Perhaps a more important question to ask is, how reliable are the tests commonly used by practitioners to then produce a diagnosis upon which a management plan can be derived? Both these questions need answers so that a rational approach to managing acute shoulder pain can be implemented.

Ultrasound is a reliable imaging modality in the hands of one who has made this his or her speciality. What this means is that the various abnormalities can be delineated, but the big question is how do these relate, if at all, to the patient's pain or complaint, or loss of function, etc.

The major reason that a patient presents to a medical practitioner with a musculoskeletal problem is pain. The clinician must reproduce the patient's pain in order to make a diagnosis. It is apparent that none of the radiologist's reports indicated that the dynamic ultrasound reproduced the patient's pain by such-and-such a manoeuvre or over a specific region or structure. What would be of use would be a finding that an area of defined pathology reproduced the patient's pain or presenting complaint. Would it not be reasonable to suggest that in the process of the investigation defined pathology be related to a specific manoeuvre or sign? This would give more direction for the management of the patient rather than giving a list of pathologies, which may or may not be relevant.

This qualitative study raises several issues, the most important of which would be to determine what further

clinical action was taken by the practitioners on the basis of the report and how frequently were patients referred on to specialists or allied health practitioners and not managed by their GP. The National Musculoskeletal Medicine Initiative (NMMI) found that evidence-based medical practice referred 8% of shoulder pain for imaging compared to 58% for vocationally registered clinics.⁹ This study and the results of the NMMI would suggest that there is a need for general practitioners to be made aware of what is the best cost-effective musculoskeletal practice in regard to managing acute shoulder pain.

Conclusion

This study provides good evidence that:

- An improvement can be made in the amount of relevant information that must be put on the request form for diagnostic imaging.
- The clinical tests used to determine a clinical diagnosis for shoulder dysfunction in specialists' hands only provides a 40% accuracy as regards the observed pathology.
- A means for accurately diagnosing what structures are responsible for shoulder pain and guidelines for when imaging is to be ordered are long overdue.
- The only significant pathology shown was rotator cuff tears which are not likely to be the cause of the symptoms for an elderly age group that are unlikely to pursue surgical correction.
- At no time during the investigation did the radiologist report that the pathology found reproduced the patient's pain.

Recommendations

1. More detailed information is needed on request forms. This information should include what has been found on examination and then to be specific as to what the requesting practitioner wants investigated.
2. Further study is needed as to what the requesting practitioner does with the information and how much imaging results in the management of the shoulder problem.
3. The assessment and management of acute shoulder pain appears to be in need of some upskilling which would enable more in-house treatment.
4. Radiologists should be encouraged to comment on whether pain is reproduced with movement or pressure of the transducer over the painful area and that such pain relates to the clinical findings recorded on the request form.

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Evidence-based guidelines improve performance measures in orthopaedic outpatients for low-back pain

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Introduction

General practitioners and the public have the impression that back pain is a condition for which there is a surgical treatment and which, therefore, warrants treatment by orthopedic surgeons. Consequently, patients with back pain constitute a substantial proportion of patients referred to orthopedic outpatients. This load limits the time available for consultants to provide comprehensive and non-surgical care. Meanwhile, evidence-based guidelines for the management of back pain emphasize the need for explanation, assurance, and activation, and the avoidance of passive treatment and the use of investigations.^{1,2} The required management is distinctly medical, and not surgical, in nature.

The present study reports the results of an innovation in which a physician was appointed to provide care for patients with back pain referred to orthopedic outpatients.

Methods

A physician in musculoskeletal medicine conducted four outpatient sessions per week, seeing four new patients and two review patients per session. He conducted the sessions under the auspices of the Director of Orthopaedics, with whom he discussed patients who might require surgical intervention. He provided care according to published evidence-based guidelines.^{1,2}

Data were compared, before and after the intervention, for administrative performance measures such as waiting lists for consultations, duration of waiting time, and growth of waiting lists for surgery. Between September 2003 and August 2008, the physician saw 712 new patients.

Results

Of the 712 patients, only 62 required investigations or surgical opinion. Of these only 19 were placed on the waiting list for surgery: five with hip pain, treated successfully with arthroplasty; four with knee pain also treated surgically; five with internal disc disruption, treated by arthrodesis; three with spinal stenosis, treated by decompression; and 13 with disc herniations, treated by discectomy.

Five other cases were referred to other units because of red flag conditions, including one aortic aneurysm; three with prostatic carcinoma; and one, recently, with a very enlarged uterus due to fibroids (as revealed on MRI).

The remaining 27 patients were referred for interventional pain procedures, and avoided the need for surgery.

The majority of patients (650) did not require surgical

consultation or surgery, and were returned to their GP with a plan of management. Upon discharge, these patients expressed their satisfaction with the approach to their management, and were particularly relieved that they did not require surgery. No complaints were received by disgruntled GPs. Rather, when they have been contacted, GPs have consistently expressed their support for the approach used, particularly the reduction in waiting times. Waiting times for appointments were reduced from 5-6 months to 1-2 weeks. Accrual to the waiting list for surgery reduced from 25 to 6 per annum. No patients were returned by their GPs because of inadequate management or for further management.

Interestingly, I have been monitoring a group of elderly patients – over 60 years of age, whose CT scans, as ordered by their GPs – show nothing more than widespread degenerative changes in their lumbar Z joints. While purely anecdotal at this stage, it appears that their back pain is aggravated by activities of daily living that require repetitive twisting activities; for example, sweeping, mopping, vacuuming, hanging out the washing, ironing, as well as social activities such as golf.

Of this group, comprising over 70 patients, I have performed three-month follow-up after giving advice that they should carry out such activities for short periods only. I have so far reviewed 40 of these patients and in over 90% of cases, they have acknowledged that their pain levels are significantly reduced. Additionally, they are able to manage the pain with paracetamol, which is a marked change from the drugs initially prescribed by their GP, including tramadol, and in some cases oral opioids.

Discussion

The burden on orthopedic outpatients from patients with back pain can be reduced successfully by engaging a physician who can provide evidence-based care. This measure satisfies patients and their referring doctors, and substantially improves administrative performance measures. It frees orthopedic surgeons to concentrate on patients who require surgical treatment.

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Personal injury claims: Quo vadis?

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Stakeholders who have a vested interest in the management of personal injury claims and more so in the management of chronic axial pain coincidentally demonstrated an acute attitudinal change with the publication of the International Association for the Study of Pain's (IASP) monograph on *Back Pain in the Workplace*. By denying the reality of chronic axial pain, this monograph defined chronic low back pain not as a "medical problem," but as a problem of "activity intolerance".¹

Yet paradoxically, the same monograph advocated that the "medical management" should not be "pain contingent" but rather "time contingent." The rationale for this was not articulated, and evidentiary basic science – as one would expect in cardiorespiratory and other medical disorders – was conspicuously absent. The taskforce further recommended that those who fail to achieve restoration of function and return to work were to be reclassified as "unemployed."

Despite its irony, it is a sad commentary for the premier scientific body in pain medicine to deny the existence of chronic axial pain, to encourage unemployment and its psychosocial upheavals and for this Task Force to promote itself as a self-appointed surrogate gatekeeper to a non-medical system principally ensconced in claims management, cost containment, and cost reduction.

Yet the medical profession cannot totally abrogate the need for cost containment. In 1992 the total cost of occupational injuries in California was at least A\$20 billion² and by 2000 Washington State outlaid \$472.4 million for medical care only.³ Commensurate with the rapid expansion of Health Maintenance Organizations (HMOs), physicians so affiliated had a greater tendency to classify claims as compensable under workers' compensation than did other physicians.⁴ Levied too, is the indictment of self-referral by physicians outside the bounds of "medical necessity".⁵

Self-referrals were associated with costly and excessive administration of inappropriate multimodal treatments, generating positive revenue enhancement but negative medical outcomes – causing Congress to pass legislation prohibiting the referral of Medicaid and Medicare patients for any of the 11 designated health services – from clinical labs to prosthetic supplies – with which the referring physician had a financial relationship.⁵

In most Western societies, changes in Workers' Compensation legislation induced an inflexible system of case management. Defined as a set of "logical steps and a process of interaction within a service network which assures that a client receives needed services in a supportive effective efficient and cost-effective manner"⁶ case-management is primarily pre-occupied with cost containment, including medical care and returning the injured worker to work – be it pre-injury, alternative or even notional work that would expedite case-closure.⁷

Case managers have multiple roles. Within the ambit of a single claim, they serve multiple stakeholders simultaneously. In their "administrative" role, case managers process claims, pay wages and bills. As "watchdogs" they monitor health care services and the "medical necessity" thereof. In a "supportive" role they liaise and co-ordinate the passage of the claim with the legal fraternity, health care and rehabilitation providers, the employer and the worker. Throughout, case managers are accountable either to the insurer or to government instrumentalities that administer the relevant Act.

Nomogenic disorder is a newer kind of impairment and disability created by such a rigid and inflexible system.⁸ Analogous to an iatrogenic disorder, nomogenic disorder describes those psychopathologic disorders in which the law and its application play an etiologic role.⁸ This is further exacerbated by unique pressure placed on health care providers, such that their traditional role as a healer has been transformed into that of a "medical police".

The process also undermines the quality of health care permissible, devalues the (treating) doctor-patient relationship and hinders access to unbiased clinical assessment.⁹ Many claimants say they have experienced a loss of esteem, self-worth and dignity: a traumatic separation from the workplace and an exposure to an overwhelming range of health care professionals. Inappropriate and ineffectual treatment is said to prolong absence from work, causing financial loss, anger and stress anxiety, whilst adversarial medical consultations could lead to disenfranchisement.¹⁰

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

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

Published guidelines rely heavily on randomized controlled trials for their conclusions, irrespective of quality and validity, and what is not universally known is that policy makers legitimized randomized controlled trials (RCTs) so that the medical profession could be regulated.¹² Authors of putative "evidence based guidelines" have carefully omitted "integrating individual clinical expertise ... and ... compassionate use of individual patient's predicaments, rights and preferences"¹³ in making decisions about patient care. By a process of selective conceptualization, third party funders and legislators have hijacked the principles of

evidence based medicine, principally and solely to contain costs, as aptly exemplified in a recent publication by the Bone and Joint Decade Task Force on Neck Pain.¹⁴

Insurers do this by refusing to reimburse the costs of treatment that are not “medically necessary” and by refusing to pay more for the treatment of a particular problem than the predetermined average cost of treating that problem within a particular patient population.¹⁵ Rather than judiciously incorporating the current best evidence, pre-eminent in the postulations of guideline authors is the rationing – and not rationalizing^{16,17} of health care^{18,19}. To be trustworthy and accepting, authors of guidelines must demonstrate the legitimacy of their process through clinical governance.²⁰

No system of injury compensation can function without the expert evidence of physicians, irrespective of qualifications.²¹ Under most workers’ compensation systems, a caveat to reimbursement is that treatment must be appropriate, reasonable and medically necessary.

The concept of “medical necessity” actually functions as a principle of allocation and gate-keeping²² – because insurance companies fear that funds will be siphoned into a bottomless pit. Thus, pre-emptively, insurers and third party payers rely on “utilization management/review” boards to reduce the consumption of “unnecessary and inappropriate” health care services.²³ Traditionally, insurers’ decisions are based on the idea that without the service harm will come to the patient and with that service potentially beneficial outcome will result.

Setting such threshold is illusory because patients and providers, insurers and courts have different values and objectives.²⁴ Whilst clinical facts may determine “medical necessity”, ethically and morally, physicians so engaged must demonstrate transparency about the grounds for decisions and procedures for revising the decisions in light of challenges to assure “accountability for reasonableness”.²⁵

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

Conclusion

In the USA, approximately 48 million patients suffer from chronic pain and they suffer needlessly, because although the technology is available, 90% cannot access it.¹⁰ What is at stake here is the erosion of the real standard of care.²⁸ Recognizing a constitutional right to adequate pain relief has the potential to remedy any inequalities.²⁹ In a throw-away society, when bad evidence happens to good treatments³⁰ the “injured worker suffers the same fate as the plastic cup”.³¹

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Study Pain and Pain Management at Otago

Overview

The endorsement in Pain and Pain Management is open to medical practitioners and allied healthcare professionals and can be studied part-time.

It is designed to present an understanding of the importance of pain to the individual and to society, and how best to optimise its management. It provides candidates with the necessary skills to better understand and manage pain problems that pertain to their particular discipline within healthcare. At Master's level, it provides a knowledge and understanding of research methodology and its practical application.

Qualifications

- Pain and Pain Management is available as an endorsement for:
- Postgraduate Certificate in Health Sciences (PGCertHealSc)
- Postgraduate Diploma in Health Sciences (PGDipHealSc)
- Master of Health Sciences (MHealSc)

A candidate for these qualifications must be a graduate or possess an appropriate health professional qualification requiring at least three years' full-time tertiary study, and must have experience of, or be currently working in, the relevant field of healthcare.

These qualifications are available through distance learning. Some papers may have an on-campus component.

Programme requirements

- Postgraduate Certificate in Health Sciences (PGCertHealSc) endorsed in Pain and Pain Management
- Postgraduate Diploma in Health Sciences (PGDipHealSc) endorsed in Pain and Pain Management
- Master of Health Sciences (MHealSc) endorsed in Pain and Pain Management

Further information

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PGCertHealSc endorsed in Pain and Pain Management
<http://www.otago.ac.nz/christchurch/study/postgraduate/otago012371.html>

PGDipHealSc endorsed in Pain and Pain Management
<http://www.otago.ac.nz/christchurch/study/postgraduate/otago012370.html>

MHealSc endorsed in Pain and Pain Management
<http://www.otago.ac.nz/christchurch/study/postgraduate/otago011631.html>