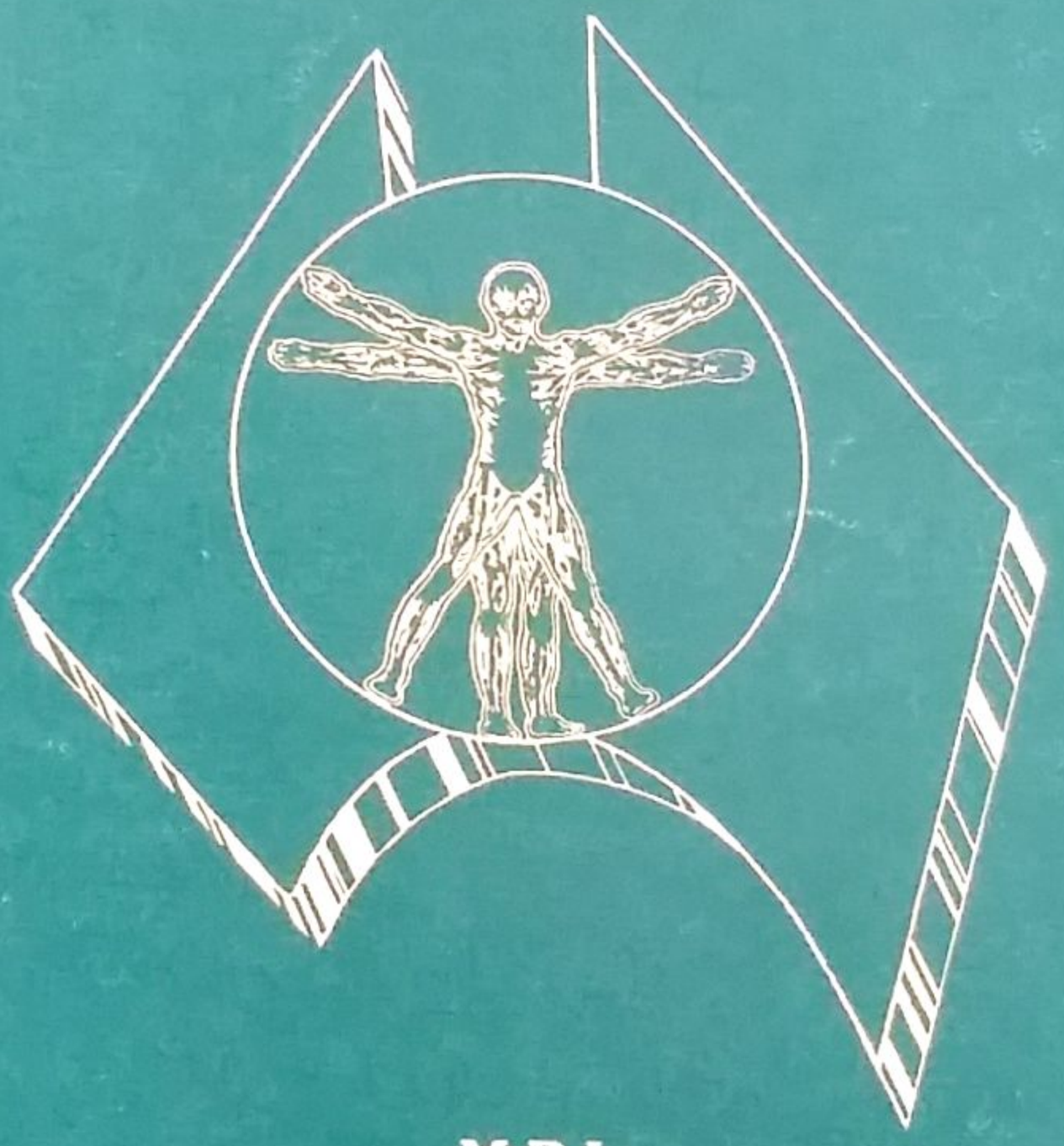
Australian Association of Musculoskeletal Medicine

Bulletin



M.R.I.

R.S.D.

Shoulder Pain

Essays and Case Studies

Vol. 10 No. 1 April 1994

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Australian Association of Musculoskeletal Medicine

BULLETIN

Vol. 10 No.1 April 1994

Contents

The A.A.M.M. Bulletin is produced by the Australian Association of Musculoskeletal Medicine for medical practitioners interested in the aetiology and management of musculoskeletal disorders. Opinions expressed are those of the authors and not necessarily those of the editor or the Asssociation. Editorial comment may reflect the opinions of the editor alone. Contributions on any relevant topic are welecome for submission to the editor, Dr ron Palmer, Suite 24 Royal Brisbane Place, 17 Bowen Bridge Road, Herston QLD 4006. Telephone (07)252 1128 or to any member of the A.A.M.M. Council
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Australian Association of Musculoskeletal Medicine

OFFICE BEARERS 1994

The following members were elected to office at the annual general meeting in Rotorua on 31st July, 1994.

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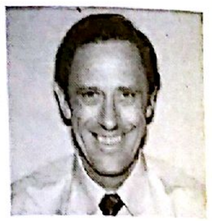


Editorial

The current edition of the Bulletin marks a milestone in the history of the publication and for the Australian Association of Musculoskeletal Medicine itself. From the very humble beginnings of a few like-minded enthusiasts, the Association has blossomed to where we now rate intellectual consideration by our fellow Faculties. Only a very few of the original founding fathers remain in practice today, yet they must surely feel proud of the society they established back in 1971. Acceleration in knowledge, education and politics have taken place in the more recent past years to the point where we are on the threshold of not only forming a "College", but actually needing one to accommodate the rapid expansion that is taking place. The Bulletin celebrates its tenth birthday this year. Yes, it seems hard to believe, but a decade has passed.

This particular edition of the Bulletin has marked a new era in publication. In the past the Bulletin was produced in Tamworth by Belaser. This was initially an obvious location as the founding Editor, Wade, King, was practising at Tamworth. With change in editorship and change in location, Tamworth began presenting some logistical and economical problems. Leonie Bell has worked hard to overcome these areas of concern, but in fairness to all it was felt that a metropolitan produced Bulletin would be a better long term choice. Maintaining some flexibility in this area is essential. When my turn in charge is complete, who knows where the next Editor will reside? Obviously a resident Editor in Perth will have some communication difficulties with a Brisbane based publisher. At that stage, a further change may necessitate. Such problems will only exist while management is in the amateur status. With the formation of a College and professional staff, the role of separate individuals undertaking such tasks as the Bulletin production may well recede into history. Only time will tell. In the meantime we thank Leonie for her dedication over the past decade. On behalf of all our members we wish both her and her Company, Belaser, the very best for the future.

As last the Association has reached a recognized higher plane with the successful graduation of three members from the Flinders University Diploma course. Past President David Vivian, Secretary Wade King, and Editor Ron Palmer received notification of their success in late December. This Graduate Diploma of Musculoskeletal Medicine is currently the highest award that can be achieved in this particular medical field in Australia. The academic success is of course only the icing. There is no doubt that the real achievement is in having established the degree in the first place. This reflects the work of your past committees and the giant undertaking of convincing a university to take on the ominous task of setting up such a course. To recognize that we needed a degree was one thing, to set up the mechanism to achieve it was another. While no single individual is totally responsible, the A.A.M.M. will always be indebted to President Norm Broadhurst for being the driving force in establishing the course at Flinders.

Perhaps, in retrospect, it is just that three of the older and long standing members of the Association became first cabs off the rank. That after all, is what leadership is all about. It is now up to others to follow. The Flinders course has established itself as a legitimate degree in medicine and passing is not without extreme effort and dedication. Fortunately there are thirty others at all stages of their Diploma course and many of these doctors are younger members of the Association.

This is perhaps the most pleasing aspect and represents a healthy future for our Specialty. This year a number of other members have commenced the New Zealand Otago Diploma course. As a result of these two courses we should see something of an explosion of fully qualified musculoskeletal physicians onto the national medical scene. All of this is indicative that as a group we are becoming more recognizable in knowledge and sheer weight of numbers. Together with our Trans-Tasman colleagues we are expanding faster as a specialty group than any other individual group within the medical frame-work. The time to establish ourselves into a closely knitted group of specialists, both medically and politically is near.

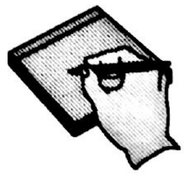
The combined scientific meeting this year will be held in Brisbane and advertising is elsewhere in this edition. Costing of such a conference is quite staggering. The sub-committee undertaking the responsibility of staging the conference is made up of ten Brisbane based members. The format has been set and invitations to speakers has been extended. The conference and the following 5 day workshop promises to be of high calibre and all members are urged to attend. It has become essential that in order to keep all costs to an acceptable level, members should stay at the hotels where the conference and workshops are to take place. The sub-committee has negotiated very attractive tariffs, but failure to meet certain base numbers will result in the Association having to pay for the conference rooms. This will amount to many thousands of dollars. Therefore it is essential that all registering for the conference should also reside "in house". The few dollars you might save by staying at a down market motel could cost the Association dearly. Please think of the future of your association when making this decision.

This Bulletin contains a new section on interesting case studies. Over the years there has been a steadily changing pattern of presentations, upgrading from the early years when the Bulletin was essentially a communication to help weld members together. Gone are the "Cooee" columns that were so essential in the early days, just to let members know that they had colleagues out there somewhere. The influence of a more scientific approach to our specialty is now reflected in more scientific articles. The quality of these submissions has also been under attack to ensure that they reach a standard acceptable for publication. In some cases heavy editing is essential to prune articles to size, correct English, or simply rephrase to make more acceptable reading. To help stimulate members to read more and to help prod them into undertaking a Diploma course, we began reviewing many books suitable for higher education. And so the changes continue. Perhaps we are approaching the stage where we can move on from the "Bulletin" format to an upgraded "Journal" one.

There still remains a lethargy on behalf of members to contribute. This editorial has become somewhat repetitious in stating this. To become a publication of international repute we must have articles of sufficiently high standard to make academics want to read it. To date there has been a paucity in both number and quality of submitted papers. You are urged to send in research papers, case studies, or papers that you feel other members would want to read. There has been little mail to the Editor and we will print this whether it is constructive or unfavourable, if it is forthcoming. This is your publication so let us hear your voice.

This copy of the Bulletin is of limited size due to difficulties associated in relocating the site of publication. There may also probably be only two editions for the year. Once teething problems have settled, the normal practice of producing three Bulletins a year will again eventuate. I trust that you will bear with us through these initial tedious times. There will be a constant striving to improve the Bulletin and if you have any constructive suggestions to aid in this goal, please do not hesitate in forwarding them.

A word from the President



The Annual General Meeting of AAMM held in Rotorua in July elected me as President for the next twelve months. I am grateful to those who expressed confidence in the nomination and the voting and trust that the activities of AAMM in the next couple of years will reflect that confidence.

The discipline of musculoskeletal medicine has advanced considerably from the anecdotal means of arriving at diagnoses to a clear thinking along more scientific lines, involving principles of biomechanics. However we are a long way from being a precise scientific pursuit over the treatment of people with dysfunction in our discipline. Much in the world literature and much of which is being produced in Australia is being channelled along the lines that will scientifically establish a discipline of musculoskeletal medicine in its own right in the years to come.

In the past, AAMM has offered courses for medical practitioners in the mobilising, diagnosing and treating of musculoskeletal dysfunction. We should continue to run workshops along these lines and to this end, the conference in Brisbane in October 1994, will have numerous half day workshops where small groups can update or learn aspects of treating musculoskeletal problems as may be experienced in their various practices.

In addition to running workshops, every state should have members eager to continue ongoing seminars, tutorials, etc. One way of doing this is to have a journal club which may meet quarterly, interspersed with a meeting to discuss difficult cases. This will enable members to keep abreast of what is in the literature as we now have many members able to use the medline research facility in medical libraries as well as having several academics appointed to medical faculties who have an interest in musculoskeletal teaching. Another area in which members should feel free to become involved in is the resurrection of the Certificate of Manual Medicine course which was ably executed through the efforts of John Murtagh and Clive Kenna. Due to numerous circumstances very little has been done in recent years. My suggestion is that this worthwhile course should be reinstituted along the lines in which it was originally taught, ie, weekend workshops with evening tutorials followed by a reasonably lengthy apprenticeship before final assessment of the clinical skills which have been learnt.

At the recent Annual General Meeting of the Royal Australian College of General Practitioners, I was very encouraged by the number of general practitioners wanting to learn more about injection techniques. Each workshop was filled to overflowing with an obvious need being demonstrated. As medical practice becomes more and more specialised there will be less and less for the general practitioner to do unless some additional skills are taught and learnt. The Certificate of Manual Medicine as well the various workshops run by AAMM may go a long way in meeting such needs.

At present the ultimate standard for the practice of musculoskeletal medicine is the acquisition of the Diploma which is offered either by the Flinders University S.A. or the University of Otago. The Flinders University has graduated their first batch of diplomats. Unfortunately, we can at this stage take no more than 8-10 entrants at any one time and this of course leaves a waiting list for people to do the course. I am most encouraged by those who have already participated in the course and

feed back from these people is very positive even though the bulk of them have been engaged in considerable musculoskeletal medicine for many years.

It is obvious that musculoskeletal medicine is alive and well. We are developing a pool of expertise which must be increased if the discipline is increased and not to be engulfed by other specialties or left to languish and wither.

While the bulk of the work previously has been left to a few eager members of AAMM, the growth of musculoskeletal medicine can only be assured when there is a much wider participation by the membership at large. This will be enhanced with the appointment of people into musculoskeletal clinics in large public hospitals as I have encouraged people to do so for quite some considerable time. It is not until we are established in large public hospitals that the concept of an Australian College of Musculosketetal Medicine would become a viable entity. Thus we need a critical mass of members competent and practicing full time in the area of musculoskeletal medicine before a college with fellows is contemplated. This does not mean that we should wait until this critical mass arises before we work on the establishment of a college. Indeed I would see it as a priority in the next 12-24 months. Considerable effort must be directed toward the establishment of a College and then work toward establishing Fellows of such a College.

We already have a good working relationship with our counterpart in New Zealand and the formation of a Faculty of Teachers of Musculoskeletal Medicine at the Norfolk Island workshop is the first step in continuing the medical education of our discipline. This must be capitalised on a regional basis with a view to a trans Tasman College of Musculoskeletal Medicine in the near futurer.

From the Hon. Secretary's Desk

Recently I have been sifting through piles of old notes and cartons of documents, collating and organising the archives of the A.A.M.M. The result is a set of six thick folders containing newsletters, Bulletins, notices and minutes of meetings, conference brochures, discussion papers, financial statements and other documents of various types relating to the Association's activities from its inception in 1971 to the present day.

In the process of sorting these papers I have, in a sense, had the events of the Association's life flashed before me, as those of a drowning man are said to do. Leafing through the collated archives is a thought-provoking experience. They record the words and deeds of those who have been active in the affairs of the A.A.M.M. over the years. As an association we have been, and are, a varied lot. Through it all, however, there are clear threads of a common attitude to the problems associated with musculoskeletal pain and dysfunction. At every stage of the A.A.M.M.'s development the record shows that its primary commitment has been to meeting the needs of those who suffer with musculoskeletal problems.

Accurate diagnosis seems to have been recognised as the key to effective management from the earliest years. The concept has been developed progressively and in recent times the Association's educational programmes have placed greater emphasis on the refinement of diagnostic skills than on diversification of treatment techniques.

Therapeutic fashions shave come and gone, hopefully as dictated by the conventional scientific wisdom of the time. Enthusiasm has waxed and waned over the years for spinal manipulation, traction, cervical collars, spray and stretch therapy, trigger point injections, T.E.N.S., acupuncture, epidural steroid injections and a host of other treatment modalities. The current fashions (such as that in chronic musculoskeletal pain management for the work-hardening approach) seem effective in some ways but things have changed so often and so much that experienced practitioners must have reservations about them.

The real value and place of each of these things has become apparent in time but only to those who have been prepared to question their worth and evaluate the scientific evidence. Members have been greatly assisted in this regard by those active in the fields of information and, more significantly, but the nurturing of concepts of intellectual accountability and academic propriety.

The role and policies of the Association are constantly evolving, as are those of all organisations with a scientific base. As the body of knowledge develops through research, so the practices dependent on it should change. The basic commitment to discovering and , if possible, meeting patients' needs should not. The future of the Association, and of musculoskeletal medicine itself, will depend upon the extent to which it continues to recognise its strengths and weaknesses in serving the needs of those for whom it exists.

NEWS

Brisbane based Kiwi expatriate, Philip Watson, has recently returned from Christchurch where he has been involved in teaching a segment of the Otago diploma course. He is able to report that the course is alive and well.

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Currently there are thirty Australian doctors enrolled in the course. Perhaps unfortunately, all of these are not members of our Association. Together with our members are general practitioners, rehabilitation and occupational medicine doctors. While it is pleasing to see other groups interested in our specialty, one must remain cynical and ask, why? Perhaps they need to expand their horizons; perhaps they misconceive our field as part of their own.

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Philip states that practical sessions of the course will be held on campus sites in Brisbane & Sydney. Some additional weeks will be required to be spent in New Zealand. To date some 50 graduates have successfully passed the diploma examinations and for the next two years there are about ninety registrants.

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Congratulations are extended to Vice President Jim Taylor and to Lawrence Finlay who set out to do the certificate section of the Adelaide diploma course. They have successfully under taken their allotted tasks with excellent results.

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The Association welcomes the services of Janelle Ballantine and John Moffat in the publication of the Bulletin. Janelle is proprietor of "Professional Secretarial Service" and John owns the printing establishment that now produces the Bulletin. Perhaps both have been badgered by the Editor and have bitten off more than they bargained for. We welcome them aboard and hope they can hold on inspite of the initial traumas both have encountered.

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It is interesting to note that the Australasian Faculty of Occupational Medicine is a specialist college that was self formed and still to this day does not have hospital appointments in Australia. It is a recognized specialty under the umbrella of the Royal Australasian College of Physicians. Their faculty offer a formal training program for those wishing to become full members, but they also permit affiliate membership. Perhaps it would be interesting reading for those opposed to an A.A.M.M. College to cast their eye over the very basic requirements that are spelt out for membership in their Faculty "Requirements for Membership."

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The committee organizing this years' Scientific Conference have been able to persuade a number of excellent and prominent speakers to deliver papers at the conference in Brisbane. Among these are Mike Butler from N.Z., Mike Ahern from Adelaide, Peter Myers and Philip Dubois from Brisbane. Mike Butler delivered an excellent address at the Rotarua conference and his topic in Brisbane should generate as much interest. Mike Ahern is one of Australia's most prominent Rheumatologists and was chairman of the Flinders University committee that oversaw the establishment of the Australian diploma course. Peter Myers is Brisbane's leading knee Orthopaedic surgeon and is the specialist resident for the Broncos Rugby League Club. Philip Dubois is in private radiology practice and spent a number of years in the USA in radiology

research. These speakers, together with the principle speaker and all other speakers, should present an outstanding conference. We hope to see you there.

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Discovered at Tamworth following the Bulletin relocation were a large number of back issues of the journal. These will be available for collection at the scientific meeting to any member. This is an opportunity to replace lost issues, or for new members to gain access to some very interesting papers.

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Also found were some original papers that have not been published. After the authors have been contacted it is hoped these will be printed for general benefit. The titles include, "Back Rehabilitation", "The Caudal Injection" & "Biomechanical Pathology". The authors are all extremely prominent in the field of musculoskeletal medicine and the papers are first class. Let us hope they agree to publishing.

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The University of Newcastle has announced its intention to offer a course leading to a Diploma in Musculoskeletal Medicine. Planning for the course is in progress and it is expected that the first intake of students will be enrolled later this year, to begin the course early in 1995. The program is being designed to meet the needs of doctors in practice and especially those living outside the Newcastle area.

Expressions of interest are invited from medical graduates who may wish to do the course. Formal enrolment is not possible yet, but places will be limited and a list will be compiled of those who would like to be contacted when details of the program are finalised. Those who wish to place their names on this list (without commitment at this stage) should contact Prof. Nik Bogduk at the University, telephone (049) 215 608 or the Hon. Sec. on (065) 510 662.

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The National Health and Medical Research Council has just released a comprehensive report on the use of epidural steroids in the management of back pain and sciatica of spinal origin. The report was produced by a working party set up by the N.H.M.R.C. in 1991 and chaired by Prof. Nik Bogduk in his capacity as President of the A.A.M.M. Other members of the working party were representatives of the Royal Australasian College of Surgeons, the Australian Rheumatology Association, the Australian Pain Society, the Spine Society of Australia, the Consumers' Health Forum and the Australian and New Zealand College of Anaesthetists.

The document reviews the relevant scientific literature and 207 references are annotated. The findings and recommendations are too complex to be summarised in a few words but obviously will be of considerable interest to members of the Association, especially as they have been endorsed by the N.H.M.R.C., which means that they carry ethical and medicolegal implications. In view of the significance of the issue, the Council has arranged for a copy of the report to be sent to each member of the Association.

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While the number of patients requiring services continues to grow and waiting lists lengthen, some groups within the medical profession stand accused of attempting to restrict services for reasons of self-interest. A report commissioned by the Commonwealth Department and presented to a recent conference of state and federal health ministers suggested the possible need for legal action under restrictive trade practices legislation to deter the monopolistic attitudes of certain professional groups. Members will not be surprised that management of musculoskeletal disorders was identified as one of the problem areas. The A.A.M.M. was not one of the groups to which the report referred.

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Musculoskeletal Medicine

Postgraduate Certificate & Diploma

Flinders University of SA.

The next intake into this programme will be in November 1994 and begins with the Anatomy, Physiology & Biomechanics segment.

The dates are 7th - 9th November, 1994

There is a yearly intake into the programme with the course beginning in November each year. The reason for this is to enable the best use of staff and facilities provided by F.U.S.A. medical school.

For those who are unaware of the offerings, the programme is based on the following:

a. The university requires the completion of 36 units for a year's work fulltime. All Diploma courses are equivalent to a full year of study. The completion of 36 units for Dip.Mus.Med is required.

b. It is possible to be awarded a postgraduate certificate on completion of 18 units. It is possible to convert to the Diploma at a later date.

c. It is possible to do a Masters and a Doctorate in the field of Musculoskeletal Medicine.

d. All courses are 6 units and the three which constitute the postgraduate certificate are:
   - Anatomy, physiology & Biomechanics (November)
   - Clinical skills (February)
   - Dysfunction of the Vertebral column (July)

   These have to be done in the sequence above for obvious reasons.

e. Having completed the above 3 courses, the following are to be taken to complete the Diploma.
   - Dysfunction of the Appendicular Skeletal (October)
   - Independent Study - to include any in depth study of interest including related courses for other tertiary institutions
   - Principles of Rehabilitation & Occupational Health - or any related topic from the offerings within the Primary Health Care programme.

It is possible to take any of the offerings as an "Irregular enrollee" and not do the exam at the end.

At present the programme is offered in blocks of two weeks intensive study in the case of the first four units. The last two units can be done as distance learning programmes. All expenses involved with the course are tax deductable. The up-front fee is $1600 per course with no extras but does not include accommodation which is available on campus if needed. The cost of the course is fixed until you complete the requirements.

A.A.M.M. strongly supports and encourages members to be involved in this course and keep the Aussie dollar at home. Without being nationalistic, other advantages of doing the Dip. Mus. Med at Flinders are as follows:-

a. There is an intake of 10 students each year.

b. Residential living and study helps the learning process and bulks long lasting friendships.

c. You get the advantage of up to date knowledge on recent advances in matters relating to Musculoskeletal medicine.

d. Regular intensives with case students in between courses helps to keep the student practising new found skills - even for those in a busy country general practice.

e. A measured degree of flexibility enables the student to follow selected interests which would be helpful in their own professional development.

f. Exposure to specialists in allied out patient clinics provides and added dimension to the practice of Musculoskeletal practice.

Further information may be obtained from:

Dr Norman Broadhurst
Co-ordination, Musculoskeletal Medicine Programme
Department of Orthopaedic Surgery
Flinders Medical Centre
Bedford Park SA 5042

Telephone: (08) 204 4289 Fax: (08) 374 0832

Mr Michael McKay
Administrative Officer
Faculty of Health Sciences
Sturt Buildings
Flinders University of South Australia
GPO Box 2100, Adelaide SA 5001

Telephone: (08) 201 3913 Fax: (08) 201 3905

Participants are expected to make their own arrangements for residential accommodation while undertaking course topics. Some accommodation is available in one bedroom sitters attached to the Flinders Medical Centre. Enquires about this accommodation should be directed to:

Mr Doug Griffen
Accommodation Officer
C/- Flinders Medical Centre
Bedford Park
Adelaide SA 4052

Telephone: (08) 204 4954

SHOULDER PAIN: CAN IT BE DIFFERENTIATED?

Norman A Broadhurst, Senior Visiting Medical Specialist

Department of rehabilitation, Queen Elizabeth Hospital, Woodville SA.

ABSTRACT

Presentation of shoulder pain which is not related to trauma, cervical dysfunction or the capsulitis pattern must be assessed for individual structures which may be the source of the dysfunction. In a study of 61 patients presenting to an outpatient clinic, 82% had more than one area of pain. The areas most frequently reported as a source of pain, were supraspinatus tendon/subacromial bursa, infraspinatus tendon and the acromioclavicular joint. The infraspinatus tendon and the AC joint were areas most frequently neglected in the examination of the shoulder.

Introduction

Over a number of years I have seen numerous patients who have had multiple injections into the shoulder. On questioning the patients they have frequently been unhelpful in knowing where the injection had been put and even less helpful in being able to indicate what the diagnosis was which necessitated an injection.

Examination of many of these people revealed that they had several areas of pain which could be the source of the problem. Injection in one area may not resolve the problem because pain is manifest in more than one area.

I though it worthwhile over a period of twelve months to monitor the shoulder pain presentations to the outpatient clinic in musculoskeletal medicine and to see whether some pattern of pain presentation could be elicited. From such data it may then be possible to suggest an explanation as to shy so many injections to the shoulder were unhelpful.

Method

All patients who were presenting with shoulder pain were questioned as to the presence or absence of trauma, whether the pain came on over a period of time or whether there was some other underlying precipitating cause, ie. repetitive strain and what side they went to sleep on. No attempt was made to determine the types of failed treatment used. Patients were excluded from the study on the criteria listed below. The number in brackets represents the number excluded on that basis.

- history of traumas (13)
- frozen shoulder capsulitis 36)
- grossly obese (3)
- poor historians (2)
- inappropriate response to pain on clinical examination (1)
- combination of neck and shoulder pain (3)
- tendon rupture

The *sine qua non* of assessing shoulder pain is to assess the cervical spine to ensure that there were no referred pain patterns which would confuse the diagnosis. Patients who were poor historians and those with inappropriate response to pain were excluded because it was difficult to know exactly what structures were likely to be involved in their symptoms. Patients who were sufficiently obese so that the landmarks and painful structures could not be easily discerned were also eliminated from the study.

The diagnosis of the capsular pattern or frozen shoulder was made on the basis of loss of movement principally in external rotation supported by loss of movement in one or more of the following - flexion, extension and internal rotation. The testing was carried out with the shoulder in the neutral position and the elbow flexed.

The musculoskeletal examination adopted the accepted principle of **Look, Move, Feel.**

After assessment of shoulder dysfunction, the area of areas of pain reproduction were isolated and injected with one ml of 1% local anaesthetic to see whether there was any appreciable diminution of the patients pain. In the first instance this was done under fluoroscopic conditions but once confidence was achieved in being able to isolate the painful areas, the patients were no longer injected under any intensification. Twelve patients were injected under these conditions and there on after this procedure was not followed.

Results

A profile of the patients in this survey is shown in Table 1 while the occurrence of the structure presenting as painful is seen in Table 2.

Of the 61 patients in the study only 11 (18%) had a single source of pain which was a supraspinatus/subacromial bursa dysfunction in 10 patients with only one having a sole cause of shoulder pain being in the A/C joint.

The numbers in the study were too small to make comparisons with the prevalence of right and left handedness and the presence of symptoms. What was very evident was the fact that 53 (87%) indicated that they always went to sleep on that side until prevented from doing so because of the gradual onset of pain.

The supraspinatus tendon was not always the source of dysfunction but was most frequently involved with pain in connection with the infraspinatus tendon or the acromioclavicular joint. These latter structures were most commonly missed when the patient was asked whether the doctor had examined or sought to elicit pain from either or both of these sources.

Although biceps tendonitis of the long head of biceps was present in 7 patients (11%) it was never identified as a source of pain by the referring doctor.

Discussion

This relatively minor survey, but probably reasonably representative, has highlighted several deficiencies in general medical practice in respect to the management of shoulder pain.

It would appear that all too frequently, shoulder pain is thought of a glenohumeral and in particular, supraspinatus tendonitis with little effort to assess thoroughly other structures directly associated with shoulder movement.

Many patients were told that they had a rotator cuff syndrome and this appeared to be equated as the only possible source of shoulder pain. While the term rotator cuff tear is often used synonymously with a supraspinatus tear, we should be at pains to assess the other two substantial muscles which comprise the rotator complex.

Obviously, reproduction of the patient's source/s of pain were not always pursued thoroughly. The importance in musculoskeletal practice is to reproduce the patient's pain, know the anatomy and preferably the biomechanics so that a more thoughtful diagnosis can be formulated. If there is any doubt, the use of a small volume of 0.5% lignocaine can be used to block the painful region. The medical profession is in the best position to make a diagnosis, the management of which may need to be conservative in the first instance, or may need further investigation or invasive procedures if in the first instance the resolution of the pain is not to the patient's satisfaction.

Patients in this study were given the option of conservative treatment, consisting of self treatment exercises plus or minus physiotherapy which mostly consisted of heat, ultrasound and interferential. Those not responding to the conservative measures were then offered injections of triamcinolone and local anaesthetic. The study was not involved with the successful outcome of

treatment, but rather to highlight those structures which although painful were overlooked. The A/C joint pain was particularly refractory to physiotherapy treatment and 28 of the 30 patients elected to have the joint injected with triamcinalone following failed physiotherapy.

Why should 87% of patients presenting with chronic shoulder pain indicate that the side of pain was the preferred side on which to choose to fall asleep? It could be that the aging process is accompanied by vascular changes and with pressure over the shoulder girdle there is a low grade ischaemic reaction which could be the course for the pain. A more detailed study is required to validate this hypothesis but it is worthwhile noting that many patients who elected conservative management along with altered sleeping position, demonstrated resolution of their symptoms in those cases in which the A/C joint was not involved.

Summary

The presentation of shoulder pain must be assessed for the specific contributions to the pain. Structures such as acromioclavicular joint, biceps tendon as well as the insertions of the muscles of the rotator cuff must all be assessed singly. If in doubt, local anaesthetic can be administered to the areas either for assurance for the patient and/or the treating physician.

◆◆◆

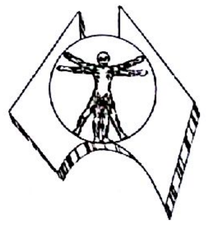
Table 1.

Patient Profile		
age	37-82 yrs	
sex	28F	33M
side effected	R37	L24

Table 2.

Incidence of pain in structures of the shoulder		
	n	**% ***
Supraspinatus	49	80
A/C joint	30	49
Infraspinatus	30	49
Subscapularis	17	28
Biceps tendonitis	7	11

* Percentage is greater than 100 because more than one site was often painful.



AAMM and NZAMSM COMBINED SCIENTIFIC CONFERENCE
Saturday - 22 October 1994; Sunday - 23 October 1994

1. PROGRAM - SATURDAY 22nd

◔	8.15 to 845	**Registration**
◔	8.45 to 9.00	Welcome and Introduction Dr. N. Broadhurst (President - AAMM)
◔	9.00 to 10.30	**Early Intervention Management**
1.	Speaker	Dr. G. Harding **Involving the Patient in Acute Management**
2.	Speaker	Mrs Megan Dalton **Selection Criteria for Early Physiotherapy Intervention**
3.	Speaker	Ms R. Pashen & Dr W Sutherland (WCB) **Early Assessment for Effective Rehabilitation**
◔	10.30	Morning Tea
◔	11.00 to 12.30	**Acute Soft-Tissue Lesions**
1.	Speaker	Dr M Ahern **Acute Presentations of Musculoskeletal Disorders**
2.	Speaker	Dr P Dubois **Effective use of Radiological services in Investigating Acute Musculoskeletal Conditions**
◔	12.30 to 2.00	Lunch
◔	2.00 to 3.30	**SPORTS MEDICINE**
1.	Speaker	Dr P Myers **Acute Management of Shoulder and Knee Injuries**
2.	Speaker	Dr P Friis **Acute Sports Medicine Topic**
◔	3.30	Afternoon Tea
◔	4.00 to 5.30	**CERVICAL SPINE**
1.	Speaker	Dr J Dvorak **Neurological Workup in Cervical Spine Disorders**
2.	Speaker	Prof. J Taylor **Pathology of the Acutely Injured Cervical Spine**

2. PROGRAMME - SUNDAY 23rd

◔	9.00 to 10.30	**THERAPY SESSION**
1.	Speaker	Dr J Dvorak **Current knowledge about Efficacy of Manual Medicine Therapy in dealing with Back Pain and Other Musculoskeletal Disorders**
2.	Speaker	Dr M Butler **Selecting the correct Analgesic method in Acute Musculoskeletal Disorder**
◔	10.30 to 11.00	Morning Tea
◔	11.00	**MINIMIZING RISK IN TREATING MUSCULOSKELETAL DISORDERS**
1.	Speaker	Dr M Butler **The "At Risk" Patient**
2.	Speaker	Dr J Dvorak **Contra-indications to Manual Medicine**

** The morning will close at 12.30 - followed by lunch and then the AGM of the AAMM*

COURSE PROGRAMME

DAY		MORNING 9am - 12.30		AFTERNOON 2pm - 5.30pm
MONDAY **24th October**	B E G	THERAPEUTIC EXERCISE Dr W King	MANAGING FOOT PROBLEMS M Bell, Podiatrist	Late afternoon and evening social event Broadwater Cruise
	A D V	MANAGEMENT OF CHRONIC PAIN BY MUSCULOSKELETAL DOCTORS IN PRIMARY CARE (1) Dr M Butler	FUNCTIONAL RELEASE TECHNIQUE Dr S Choy	as above
TUESDAY **25th October**	B E G	TRIGGER AND TENDER POINTS Dr W King		KNEE Dr R Palmer Dr P Quin
	A D V	MANIPULATION OF SPINE (1) Dr J Dvorak		PERCUTANEOUS NEUROTOMY Dr R Stuckey
WEDNESDAY **26th October**	B E G	HAND and WRIST Dr N Broadhurst		SHOULDER REGION DIAGNOSTIC and THERAPY Dr M Johnston Dr G Harding
	A D V	MANIPULATION OF SPINE (2) Dr J Dvorak		RESEARCH IN MUSCULOSKELETAL MEDICINE Dr M Yelland Dr D Vivien
THURSDAY **27th October**	B E G	NEUROMUSCULAR THERAPY (1) Dr J Dvorak		TREATING SPORTS INJURIES Dr V Wilk
	A D V	PELVIC REGIONAL PAIN SYMPOSIUM		DISABILITY ASSESSING Dr B Tait Dr P Watson
FRIDAY **28th October**	B E G	NEUROMUSCULAR THERAPY (2) Dr J Dvorak		BASIC INJECTION TECHNIQUES Dr N Broadhurst
	A D V	CLINICAL PAIN MANAGEMENT TECHNIQUES FOR THE MUSCULOSKELETAL MEDICINE DOCTOR (2) Dr B Tait		TEACHING THE TEACHERS RACGP Teacher Trainers

THE USE OF MAGNETIC RESONANCE IMAGING IN THE ASSESSMENT OF INTERVERTEBRAL DISC DISEASE AND LOW BACK DYSFUNCTION

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ABSTRACT

Low back pain and pain derived from the lumbar structures are common causes of disability. An accurate diagnosis of the etiology of the dysfunction is often not possible. The advent of magnetic resonance imaging led to an expectation that it was a non-invasive mechanism for the detection of pain. However, it appears that only limited information can be derived from magnetic resonance imaging in this context, and its use should be confined in most cases to pre-operative assessment in the diagnosis of mechanical pain.

This paper deals with the assessment of 'low back dysfunction' when fusion is being considered. In doing so, the literature in regard to the use of magnetic resonance imaging in the diagnosis of somatic origin low back pain and low back dysfunction is reviewed. Magnetic resonance imaging has a very limited role in the management of lower back disorders.

INTRODUCTION

Disorders of the musculoskeletal system are extremely common, and frequently cause impairment and disability. The prevalence of musculoskeletal disease in Australia is high. Twenty nine per cent of Australians have one or more musculoskeletal conditions, and long term back problems are present in 10 per cent of the population, occurring most frequently in the ages 45 to 59.[1] There is no evidence that the prevalence of low back dysfunction (LBD) is decreasing.

There are three possible explanations for the persistence of LBD in the community:

1. Psychosocial factors

It is recognised that compensation and other social and psychological factors play a role in the persistence of LBD.[2,3,4] However, it is possible that this point is overstated.

One paper that is used to demonstrate that compensation itself plays a large factor in ongoing disability was a retrospective controlled cohort study[4] demonstrating that pain, disability, psychological disturbance, unemployment and time off work was greater in a group of compensation patients compared to a control group matched for age, type of injury and follow up period. However, this study was unmatched for socio-economic status as well as the nature of the work undertaken by the injured worker prior to his or her work related low back injury.

It has been established that vibration[5-8] as well as combined lifting and twisting[8-18] are risk factors for the development of LBD. It is possible that patients with LBD that did not respond to conservative treatment may be subject to those occupational factors. Consequently, the conclusions reached in this paper are questionable.

It is not easy to establish the contribution of compensation or other psychosocial factors to a person's LBD. Waddell has devised tests to assess the contribution of psychosocial factors,[19] however, the inter-observer reliability of some of the signs is modest under the best circumstances.[20]

2. Inadequate Technology

Although it is often possible to reproduce the patient's pain and to block such pain with local anaesthetia, radiological investigations often do not show the lesion responsible for the patient's pain. Myelography, CT scanning and MRI have an established role in the detection of postero-lateral disc prolapse, but their role in the detection of the cause of pain in LBD is unclear.

3. Inadequate Treatment

Even if the cause of LBD can be determined, it seems that the optimal method of treatment is yet to be discovered. Prospective, randomised, long-term studies have not been performed to determine if surgery or other treatments are useful for LBD. Other conservative treatments such as exercises and physical therapy, and more invasive treatments such as zygapophysial joint injections, radiofrequency denervation, 'rhizotomy' and epidural injections are in need of validation.

DEFINITIONS

1. LOW BACK DYSFUNCTION (LBD)

LBD could be taken to include any abnormality in the function of the back, whether or not it was painful, but for the purposes of this paper, it is a term used to describe pain derived from any innervated structure of the lumbar spine. This definition excludes pain due to nerve root irritation. In this respect, a useful method for classifying pain is to consider it as either radicular or somatic.

Radicular (or neurogenic) pain, is characterised by sharp, at times severe, lancinating leg pain, often unaccompanied by back pain, and usually concentrating distally.[21] Sciatica is a term that is best applied to radicular pain derived from nerve root compression, or any other problem involving one of the appropriate lumbar nerve roots. Postero-lateral disc prolapse, a common example of a cause of sciatica, is generally well recognised on history and examination, and confirmed by investigations such as myelogram, CT scan, MRI and electromyogram.[22] Sciatica is generally easily managed, either by conservative measures, or by surgery. However, in the total setting of pain derived from the back, sciatica is uncommon, with one recent study suggesting that it accounts for only one per cent of low back problems.[23] Studies on the use of MRI in low back origin pain have mainly concentrated on radicular pain due to nerve root compression by lesions such as disc prolapse and canal stenosis.

In contrast, somatic low back pain with or without referred leg pain is common.[21] Most patients presenting with LBD have either low back pain or a combination of low back and somatic referred pain, rather than true radicular sciatica.[24] Any of the innervated structures of the back can produce pain of somatic origin. Such structures include the annulus fibrosus,[25] zygapophysial joints, dura, ligaments and muscles. Characteristic somatic pain is a deep aching type of pain, spreading at times from the back down the leg, rarely reaching the foot.[21] The pain tends to concentrate proximally, particularly in the back. The physical examination in somatic pain is generally not accompanied by signs of nerve root irritation, such as prominent restriction of the straight leg raising test, or by neurological deficit.

True sciatica (caused by nerve root compression) and referred pain are almost universally confused in the literature. Generally sciatica is defined as any pain in the leg, and no attempt is made to differentiate true radicular pain from the somatic type of pain.[26] Thus, any conclusions about the origins of the more common referred type of pain are flawed. For example, in one study[26] progressive local anaesthesia was used to establish the cause of pain in cases of true sciatica. The involved nerve root and the exposed anterior annulus fibrosus were found to be painful, and most other structures were not. The erroneous conclusion was that zygapophysial joints and other structures were not responsible for low back pain.

Disc prolapse is often erroneously blamed as the cause of pain. In one series 60 per cent of patients attended an orthopaedic clinic with a diagnosis of disc prolapse, although only 11 per cent were shown to have nerve root pain or dysfunction.[27]

2. DISC DEGENERATION (DD)

DD is the process of alteration of structure and function in the intervertebral disc, characterised by loss of protein, dehydration, and deformation of the annulus fibrosus.[28,29] In some instances it appears to be a normal ageing process, and age related intervertebral disc degeneration has been demonstrated from the second decade of life.[30] Tears within the annulus fibrosus may also play a significant role in the process of DD,[31] as well as disc degradation in which there is chemical breakdown of nuclear matrix, particularly proteoglycan.[32]

Plain X-ray can detect the subsequent mechanical changes that arise from the process of DD, and the typical changes of osteophyte formation along the edges of the vertebral bodies are called spondylosis and considered to represent DD.[33] MRI can detect DD at an earlier stage, but there is no correlation between pain and degenerative changes.

TECHNIQUES USED TO DETECT LBD

The problem for the clinician attempting to help a patient with disabling LBD is to decide what tests if any should be ordered and how the tests should be interpreted. It is generally accepted that plain X-rays are necessary in spontaneous onset conditions after a period of about 4 weeks, presumably to exclude any radiologically demonstrable conditions such as bone cancer, infection, and fracture.[34]

The role of CT,[35] MRI[36] and myelogram[37] is accepted in the assessment of sciatica. Their high sensitivity seems responsible for this consideration as disc herniation is demonstrated in 90-98 per cent of patients with sciatica, but notice needs to be taken of the low specificity of these tests in the diagnosis of disc herniation as disc herniation is shown in 28-35 per cent of asymptomatic people.[38] It has yet to be established if these investigations have a role in the detection of the cause of somatic LBD Thus, it seems unlikely that they have a role in the detection of the source of LBD.

1. PLAIN X-RAY

It is fallacious to assume that plain X-ray changes such as spondylosis, spondylitis, spondylolysis, spondylolisthesis, osteophytosis, zygapophysial joint degeneration and disc space narrowing implicates the disc, or indeed the lumbar spine, as a source of pain. X-ray does not provide satisfactory information about the lumbar spinal components as a cause of LBD at the time of X-ray or at some time in the future.[39-48] Plain X-rays should be ordered largely to detect fractures and other bony pathology that might be a cause of pain.

2. MRI

An MRI is a reconstruction of a signal that is emitted from a patient after introduction of a radio wave.[49] The physical basis for MRI involves the interaction of hydrogen bonding, which may be a part of many molecules in many human cells, with an external magnetic field and with an oscillating (radiofrequency) electromagnetic field that is changing as a function of time at a particular frequency.[50] MRI is a useful imaging technique for the lumbar spine as it demonstrates the morhology of the intervertebral disc, which is high in water content in the younger healthy disc, and lower as aging occurs.

MRI can detect DD by a decrease in the signal intensity on some MRI sequences, as DD is accompanied by dehydration.[51-59] DD is best seen on heavily T2-weighted images (TR > 2000m sec, TE 60-90 m sec) using a spin echo technique.[53,60] Under these circumstances the pathological areas are shown up as a dark spot. A normal intervertebral disc shows up as white on a T2 weighted spin echo sequence.[61] This is due to the high water content of the normal nucleus pulposus and annulus fibrosus.[62] A disc with low water content may be represented by a dark signal on a T2 weighted image.

3. DISCOGRAPHY

Discography is used to determine the integrity of the intervertebral disc, and to determine if the disc is likely to be the source of a patient's pain.[63,64] It is particularly useful in the detection of painful annular tears if the tears communicate with the nucleus pulposus.[65,66] Discography involves firstly the insertion of a needle under X-ray control into the nucleus pulposus. The insertion of the needle can be painful, and it is often performed under some sedation. Once the needle is in place, dye is injected. The patient is counselled prior to the procedure on the importance of this part of the examination, as he or she needs to identify the site and nature of the pain produced by this injection. The response can include exact reproduction of pain, or degrees of pain from severe to none that ranges in similarity to the pain from partial to no similarity. The questioning should be done at the time of the procedure.

The morphology of the disc is then studied. Sometimes disc material can be seen to leak into the epidural space, suggesting a communication from the within the disc to this area. The disc is then studied on plain AP and lateral X-ray views, and sometimes CT scan views are taken.

Pain reproduction can occur if there are painful abnormalities in the disc, and if the injection of dye causes a pressure effect in the disc. Thus, symptomatic tears that spread from the nucleus pulposus and are confined within the disc are able to be demonstrated on discogram by elicitation of pain reproduction and demonstration of abnormal morphology. Tears that reach the outer part of the annulus fibrosus and communicate with the epidural space are able to be demonstrated by discogram, but pain reproduction is difficult to detect as there is no pressure effect.

Discography can be very painful, and runs the risk of discitis.[67] It seems that the insertion of antibiotic with the injected contrast protects the disc from discitis.[68]

THE CAUSES OF LBD

The intervertebral disc is considered by some the most frequent source of somatic pain.[24,69] In chronic LBD, the possible origins of pain in chronic LBD can include the zygapophysial joints,[70,71] sacro-iliac ligaments and joints,[72] dura,[73] muscles,[74] interspinous ligaments,[75] entheses and bone. This section only deals with the disc as a cause of pain, as the discussion concerns the use of MRI and discography in the detection of a painful disc.

The nature of the tissue damage that can cause chronic LBD is disputed: some consider that there is no proven organic cause in chronic LBD.[76] However, clinical and pathological studies seem to indicate that the disc can be a source of chronic LBD.[24,31,77] Pain in LBD occurs via the usual nociceptive mechanisms. Chemically induced nociception occurs when the receptors in the peripheral part of the disc are stimulated by released chemicals, and mechanically induced nociception occurs as the small nerve fibres undergo distortion in response to load.[32]

The methods of assessment of patients presenting with LBD include a thorough musculoskeletal history and physical examination and other specialised investigations including plain radiography, CT scan and Magnetic Resonance Imaging (MRI). MRI has advantages over plain radiography and CT scanning in that it does not involve irradiation, and it provides more information about the anatomy, pathology and biochemistry of the spine, and in particular intervertebral disc degeneration.[55]

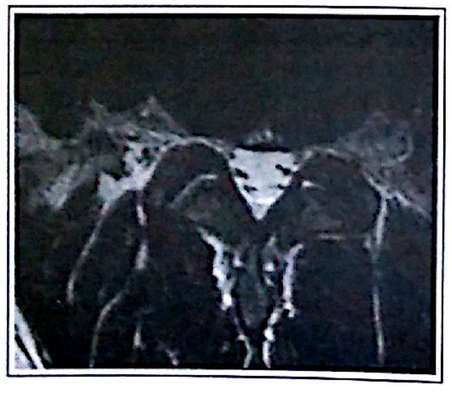
The disc undergoes degenerative changes throughout life, and the process of degeneration itself does not appear to be painful. Some of these changes reflect normal ageing,[79,80] and other changes may be related to increased mechanical stress or trauma. DD in asymptomatic people increases with ageing.[52] Using MRI, about 5 per cent of lumbar discs in people under 20 show signs of degeneration, which increases

with aging so that by 70 most discs are degenerate.[80] Not only does DD increase with age, but the proportion of severely degenerated but painless discs increases with age, as does the proportion of discs with dissimilar pain induced by provocation discography.[81]

MRI has the advantage over myelogram and CT in that it can detect the qualitative and quantitative degree of hydration of the disc. The change in disc morphology that is detectable on MRI largely relates to the changing state of hydration of the intervertebral disc. This state of hydration can be correlated with the level of intact protein within the nucleus pulposus: with ageing the protein in the disc degrades and as a consequence the disc dehydrates. The MRI is able to register this change. The problem with the finding of DD on MRI is that there is no correlation between DD and symptoms.[79,80,82]

Another investigation that measures the changing morphology of the intervertebral disc is the discogram. Degeneration is detected on discogram by the demonstration of tears or clefts in the intervertebral disc. The morphology is demonstrated after dye has been placed in the disc and x-rays (or better CT scan) are taken to demonstrate these abnormalities.

Discogram has another role to play, however. Provocative or analgesic discography can reveal whether or not a disc is likely to be responsible for a person's LBD. A strong correlation exists between the extent of annular disruption and pain reproduction using provocative discography, and over 70 per cent of tears or clefts in the outer one third of the annulus are associated with pain reproduction using provocative discography.[69,83]



Figures: 1,2&3

Degenerated discs with focal "high intensity zones" seen on both axial and sagittal T2W sequences at L5S1 and L45.

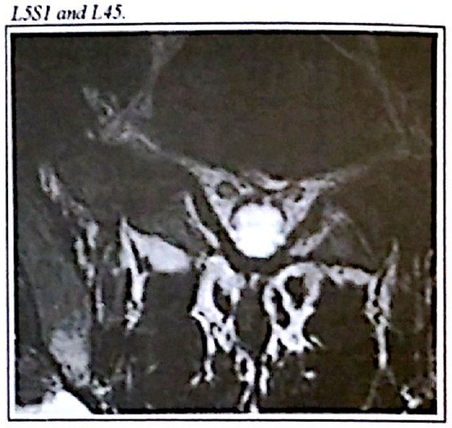


Figure 2.

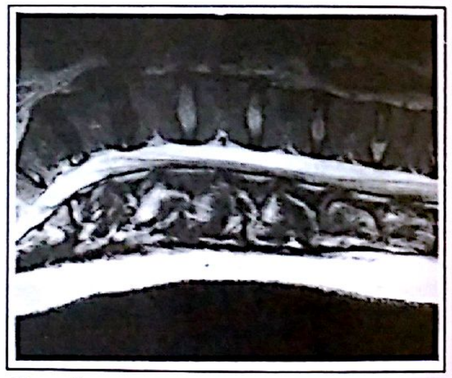


Figure 3.

LITERATURE REVIEW

Early reports on the efficacy of MRI in relationship to determining the origin of LBD were encouraging. However, these earlier studies only compared discography, demonstrating the morphology of discs, with MRI, and did not consider the correlation between the morphology of the discs seen on MRI and discography, and the provocation of typical pain in discography.[56] There is no doubt that MRI seems to be a sensitive and a specific imaging modality for detecting pathologic biochemical disc changes in the spine of a young adult.[84] There is a high

correlation in the identification of the degenerative disc between MRI and discography.[85]

The role of MRI in early identification of DD lead to a school of thought that discography should be abandoned.[86,87] However, recent information has shown that MRI and discography have different roles in the determination of the origin of back pain, and that for a pre-operative work up for discogenic pain, both tests may be required.[88]

It is known that asymptomatic discs may be abnormal on MRI.[79,80,82] One study looked at the correlation between awake discography findings and MRI in the evaluation of symptomatic lumbar disc disease.[88] This study, based on 164 consecutive patients and performed in 1991, produced the following figures:

1. 76% of the abnormal discs reproduced symptoms (by discogram)
2. In 13%, MRI showed abnormal findings and discogram normal findings
3. 37% of the discs classified as abnormal on MRI were asymptomatic
4. MRI showed normal findings and discogram abnormal findings in 7%, of which 5% recreated exact symptoms, and 2% caused no pain.

The conclusion from this study was *"MRI and discography are necessary components for adequate evaluation of back pain of discogenic origin. Dependence on MRI interpretation of disc integrity may result in significant error both in overtreatment of clinically asymptomatic dessicated discs and undertreatment of clinically significant pathology. Awake discography is a more reliable study for determining the symptomatic disc. Correlation of the patient's history and clinical examination with laboratory studies will result in appropriate treatment for the patient."*[88]

A recent study on the usefullness of MRI in the diagnosis of painful lumbar discs has demonstrated a previously unreported correlation between the high-intensity zone (HIZ) in the annulus fibrosus seen on T2 weighted images and discography findings.[24] This finding is of significance because it establishes the first non-invasive method of predicting with a reasonable degree of certainty the presence of a painful internal disc disruption. However, the finding has not as yet been corroborated by other studies,

The HIZ is defined as *'a high-intensity zone (bright white) located in the substance of the posterior annulus fibrosus, clearly disassociated from the signal of the nucleus pulposus in that it is surrounded superiorly, inferiorly, posteriorly and anteriorly by the low-intensity (black) signal of the annulus fibrosus and is appreciably brighter than that of the nucleus pulposus'.* It is suggested that the contrast can be best seen *'by viewing the image at arm's length against bright sunlight'.*

This study concluded *...'(the HIZ) occurred in 28 per cent of 500 patients undergoing MRI for back pain. The presence of a HIZ correlated significantly with the presence of Grade 4 annular disruption and with reproduction of the patient's pain. Its sensitivity as a sign of either annular disruption or pain was modest but its specificity was high, and its positive predictive value for a severely disrupted, symptomatic disc was 86 per cent. This sign is diagnostic of painful internal disc disruption.'*

Another study compared provocative discography with MRI, but no comment was made in respect of HIZ.[77] This may be because it was not recognised as a significant feature, or the MRI machine or settings were insufficient for the detection of the HIZ. All discs shown as abnormal on MRI were degenerate on discography, but of those with a normal MRI, 10 per cent had marked degenerative changes on discogram, and a further 45 per cent had either an inner or outer annular tear.

Although it is considered that the disc is a major cause of LBD, it may be that a

substantial cause of LBD is unable to be detected on both MRI and discography. Pathological studies have demonstrated circumferential tears in the annulus fibrosus that do not communicate with the nucleus pulposus. As discography can only demonstrate morphological abnormality if the injected material can reach the abnormal part of the disc, these lesions may be undetectable by conventional tests that attempt to demonstrate pain reproduction and abnormal morphology.[78]

REASONS FOR APPARENT DISCREPANCIES BETWEEN MRI, DISCOGRAPHY AND PATHOLOGY

A variety of possible investigation results and clinico-pathological correlations can exist. Some of the MRI and discography findings do represent the exact clinical state, but sometimes the results of either can be falsely positive or negative. Some possible combinations are:

1. Normal MRI, normal Discogram, Painless Disc
This combination occurs in most healthy young discs. The findings represent normal discs. At some stage in the life of a nucleus pulposus degeneration occurs. There will be a time when some nuclear protein degradation occurs but the disc shows as being of normal signal intensity on MRI. The degree of degradation that is necessary for detection on MRI is unknown. The discogram is normal if the nucleus pulposus is intact, and from which there are no radiating tears or clefts. The disc should be painless as long as there are no clefts or tears in the innervated part of the disc, (usually the outer third of the annulus fibrosus), and if the internal structure of the nucleus pulposus is sufficiently intact to prevent excess load being borne by an even normal innervated part of the annulus fibrosus.

If a person complains of LBD and there is no tear in the disc (and this is not able to be stated with any certainty using current assessment methods), the pain may come from structures including the zygapophysial joints, the sacro-iliac joints, the muscles and ligaments, or from other as yet unidentified structures.

2. Normal MRI, normal Discogram, painful disc
This is an example of false negative MRI and discogram. Consider a twisting incident leading to a circumferential tear in the peripheral part of the annulus fibrosus without extension from or to the nucleus pulposus. This tear could produce pain. MRI would be normal if the nucleus pulposus had not undergone sufficient degradation, and discography would be normal as the injected dye could not pass into the tear from the intact nucleus pulposus It is possible that this discogram might reproduce some similar pain, due to the pressure transmission from the nucleus pulposus to the annulus fibrosus and its tear, but the pain reproduction would be mild.

3. Normal MRI, abnormal Discogram, painless Disc
This could occur if the disc had undergone little or no protein degradation and a small tear or cleft had opened up in continuity from the nucleus pulposus, but it either had not reached the innervated part of the annulus fibrosus or, if it had, it was painfree. If the tear was of insufficient dimension to stimulate the nociceptive system for either local or central reasons, or if the nucleus pulposus was efficient enough to take excess load from the annulus fibrosus, then the MRI would be normal, the discogram mildly morphologically abnormal but painless, in the presence of a painless disc.

4. Abnormal MRI, normal Discogram, painless Disc
Protein degradation shows as a darkened image on T2-weighted signal. If this has occurred in a disc that has no tear or cleft radiating from the nucleus pulposus, the discogram will be normal and painfree, and the disc will be painless as long as there is no tear of significant dimension in the annulus fibrosus, and if the alteration of function of the nucleus pulposus is of insignificant

dimension to abnormally load the annulus fibrosus.

5. Abnormal MRI, relatively abnormal Discogram
This disc has a degraded nucleus pulposus with tears extending from the nucleus pulposus through the annulus fibrosus to the epidural space. The disc may or may not be responsible for the patient's pain. The discogram will show as degenerate, but there may be no pain on the provocative testing due to the inability of the disc to build pressure because the injected material leaks rapidly into the epidural space. The passage of the needle into this disc may be very painful. This disc could also be totally painfree despite its abnormal morphology.

6. Other permutations
Any combination of findings on MRI and discography is possible, and all diagnostic formulation must take these possibilities into account. If the relevance of the HIZ is validated by other studies, these additional factors will need to be considered when evaluating MRI, provocative discogram and the patient's LBD.

RAMIFICATIONS OF MRI FINDINGS ON TREATMENT

There is no consensus about how to manage the patient with somatic pain of lumbar origin that has not responded to time and conservative management. No treatment method for this type of condition has been scientifically validated.

Spinal fusion is one method used in the management of LBD. One rationale for fusion is that relative stabilisation of a painful disc will affect pain control. For fusion to be successful, the painful disc must be identified, and the offending part must either be resected or totally immobilised. Immobilisation is usually performed via internal fixation using metal and/or bone applied to the posterior elements or the vertebral bodies. It is considered that fusion is best applied only to the painful spinal segment. However, in some circumstances fusion is applied to other non-painful spinal segments, because adjacent segments are seen to be at risk to becoming sources of pain.

It is considered that a degenerate disc adjacent to a segment to be fused is at risk of becoming a source of LBD. Thus, in the work-up of a patient for spinal fusion, it is important to establish which discs are degenerate as well as which discs are responsible for the patient's pain. The results of such a work-up may mean that more than one segment may need to be fused even if only one segment is painful. It is possible that the work-up will establish that fusion is untenable. This occurs when multi-level disc degeneration is discovered.
For example, consider a patient who is sufficiently disabled and psycho-socially acceptable for surgery, and who has had an acceptable course of conservative therapy. If investigation reveals the L5/S1 disc as the source of LBD, and the disc above this to be painless and morphologically normal, then fusion of the L5/S1 disc is performed. However, if the L3/L4 disc was shown to be abnormal, then the surgeon would have to consider one of three options:

- fusing L5/S1 and running the risk of the L3/L4 disc becoming painful at an accelerated rate
- fusing L3/S1 to decrease the chances of L3/L4 becoming painful at an increased rate
- abandoning the procedure due to the problem associated with both leaving L3/L4 unfused and the extent of a triple level fusion.

The decision on what levels to fuse can only be made after determination of both the degree of DD in each disc and the source of a patient's LBD as it relates to pain reproduction.

It appears that the gold standard for the detection of internal disc pain is discography. DD can be detected by MRI or discography. However, even if the disc is shown to be the major cause of pain in a paitient with LBD, fusion, with or without disc excision, does

not always produce wonderful results. Mechanical causes of failed fusion include: complications of surgery such as wound infection, subsequent non-union, fatigue fracture of metal implants, biomechanical insufficiency with posterior fusion, other concommitant sources of pain (eg zygapophysial joints and epidural fibrosis) and pain at other levels.
The diagnosis of a probable cause of LBD should only be attempted if some form of treatment depends on this process. Investigation should only be performed if treatment is likely to lead to improved function. At present the only option when discography is positive is fusion. The first investigative step in the diagnosis of discogenic pain is MRI. Confirmation that the lesion is responsible for the patient's symptoms has to be obtained by discography.

The demonstration of the HIZ opens up a range of possible options for management of the painful annular tear. Although it appears that intra-discal steroids are not successful in the management of disc pain, at least in comparison to marcaine, and only over a 10 to 14 day period,[89] it is still possible that steroid or other anti-inflammatory agents injected into the HIZ, followed by an active period of mobilisation could lead to improvement. Other options for treatment of the HIZ include radiofrequency ablation, percutaneous resection and drug injection.

RECOMMENDATIONS FOR ORDERING MRI FOR LBD

A disc can be a potent source of LBD even if MRI is normal. However, an abnormal MRI is not indicative of a painful disc, unless the HIZ is considered a reliable sign. Further studies in this area are needed before MRI becomes a reliable marker of discogenic pain.

As the results of MRI do not lead to definitive treatment, the indications for MRI in the work-up of a patient with benign, somatic LBD are limited to two circumstances. There may be a place for MRI in a medico-legal setting if the HIZ is validated by other studies as indicative of a high chance of a person having a symptomatic disc. The other indication is as a screening tool in the work-up of a patient for operative intervention such as fusion.

Once a patient is classified as a fit and proper candidate for surgery, MRI is performed to evaluate whether the lumbar spine is suitable for surgery, and thus, to determine if discography is an appropriate investigation. If MRI demonstrates a degree of DD that makes fusion impossible, there is no need for discography. Thus, MRI can obviate the need for some patients undergoing the pain and morbidity associated with discography.

The indication for ordering MRI for LBD is simple. It should only be used in the management of LBD if surgical intervention is indicated.

In the future the role of MRI may change. If the HIZ is validated as a reliable measure of a symptomatic disc, and if a treatment method is established, MRI will become the accepted and first line assessment tool for LBD.

A breakthrough in the management of LBD will occur if:
1. The psycho-social contributions to pain maintainance are minimised by such things as changes to the compensation systems
2. Technology establishes the prime sources of LBD
3. Scientifically proven management techniques are devised to specifically treat these causes of LBD.

Technologies such as MRI should probably not be introduced into the management of conditions such as LBD until such time as tests demonstrate that a particular protocol provides reliable information that leads to some effective treatment.

CONCLUSIONS

1. MRI has a limited role to play in the assessment of LBD, and probably should be confined to the patient scheduled for spinal fusion.
2. New approaches to MRI may make it the investigation of choice
3. A normal MRI is of no clinical relevance in LBD.

***Figures 4 & 5** Nice example of normally hydrated discs in a 19 year old with an incidental (asymptomatic) degenerated L5S1 disc (sagittal T2W sequence)*

Figure 4.

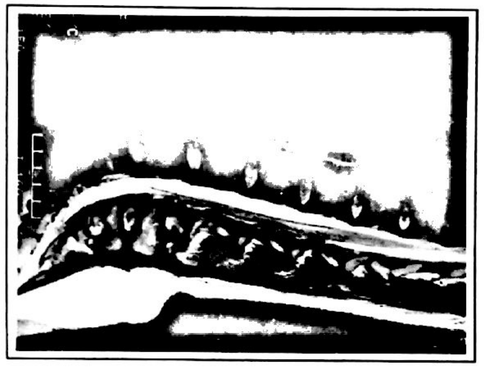
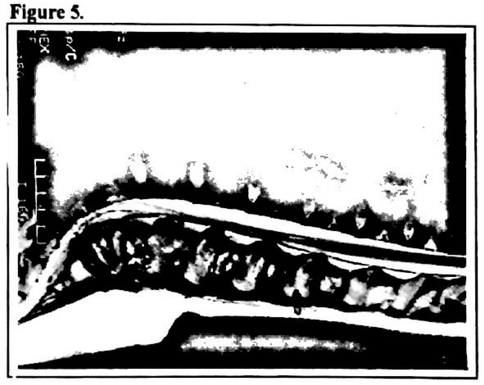


Figure 5.



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"WHAT THE PROFESSOR NEVER TAUGHT ME"

D.P. Anderson
New Zealand

"ART IS LONG, BUT LIFE IS SHORT"

—Hippocrates.

Introduction

Piers Anderson takes a lighthearted, yet serious look, at his own ongoing medical education from an under-graduate to a successful practicing musculoskeletal physician.

The paper was presented as his required Independent Study for the New Zealand Diploma of Musculoskeletal medicine.

In formulating this article for publication it was necessary to delete 28 case studies that were originally tabulated. These deletions have not undermined the relevance, nor the impact of the essay. This excellent paper is thoroughly recommended reading and is guaranteed to provoke interesting discussion in all who do read it.

Editor

My consuming interest in the field of musculoskeletal medicine was kindled on the first day of my first general practice locum. Having completed six years of undergraduate medical education at Auckland Medical School and surviving two years as House Surgeon and then two years Medical Registrar's penal servitude, I looked forward to treating those patients whose wellness was marred by a current problem, not as I had in hospital, subtly altering the inevitable course of end stage chronic disease.

I left the hospital well qualified, I thought, to cope with the diagnostic problems of the wider community. It was to my surprise and chagrin at the end of my first general practice day, that having seen three patients with back pain, I had no idea diagnostically what their problems were. I drew some succour from my internal physician's training which enabled me to confidently tell them they did not have sciatic nerve root entrapment, even if I didn't at that time know that this causes less than 5% of back pain presentations.[1]. But even to myself, and I suspect my patients, my glib phrases of `a deep muscle sprain' or `a torn ligament' had a hollow ring to them. It was my desire from that point to build up a knowledge base and diagnostic and subsequently therapeutic schema for myself as well tested and scientifically valid as the rest of my medical education. Certainly now I feel I am some measure of the way towards a synthesis which is functionally useful and is intellectually satisfying.

Clearly none of this the Professor taught me. We may have endured whole afternoons of detailed subgroups obtruse forms of glomerulonephritis at Medical School, which are either very rare, or which I am missing in droves in my general practice, but we received no undergraduate or postgraduate education in acupuncture, myofacial trigger point therapy, pain theory, functional segmental examination of the spine, joint manipulation or length testing of muscles, all of which are vital everyday components of the practice of my craft. Thankfully this is slowly changing, nowadays Registrars in the Family Medicine Training Programme spend one afternoon each in a year's course discussing segmental spinal dysfunction and sports medicine [2]. Training as they are for a branch of medicine in which it is estimated up to 15% of consultations involve musculoskeletal disorders [3], it would appear the priorities for education in such disorders are relatively low.

After a year of GP locums finding out that general practitioners work involves much effort defining the undefinable and coding the uncodable, my love and I left to explore the world. While on the Greek Island of Kos, we visited the Aesculapion and musing on my profession, sitting beside a fountain in the school of Hippocrates, I realised the healing quality of my work was of great importance to me and that in the future increasing my personal therapeutic skills would mean more to me than prescribing writing and form filling. Throughout all of my musculoskeletal education I have attempted to gleam as much hard scientific support for the therapies or techniques I was acquiring, but it must be said that it is only with the advent of advances in studies in articular neurology [4,5]. pain pathways, endorphin and neurotransmitter research [6,7,8], acupuncture [9,10], myofascial pain [29,32] and biomechanics [11,12], that scientific support can be given to the Empirical techniques of manipulation and of needling, utilised successfully since the time of Hippocrates and Galean [13], the Yellow Emperor [14] and more latterly Osler [15,16].

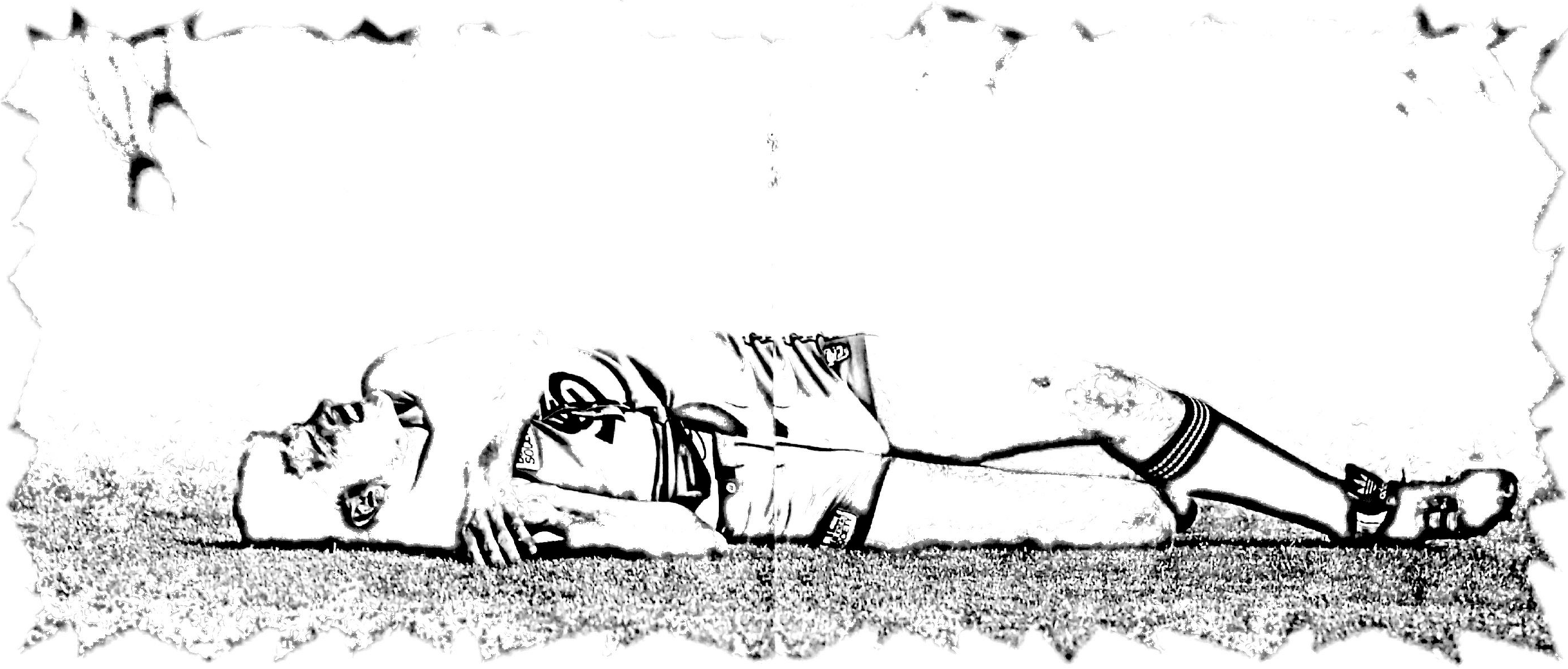
The difficult fact remains that in manual medicine many of ones diagnostic decisions are based on soft physical signs fraught with the difficulties that beset subjective assessment, if infact their existence is believed at all by the more dogmatically hidebound [17]. We have Hippocrates to thank for starting the practice of basing his conclusions on observations of appearances and physical findings [13] and more recent work suggests that amongst manual medicine experts anyway, interobservor error is not marked [18,19] and manual diagnosis shows good co-relation with radiologically guided anaesthetic blocks of the cervical spine [20]. The bald facts of the matter are that manual medicine manipulative techniques, acupuncture and myofascial pain techniques work to relieve symptoms and suffering, have been validated in practice [21,9,10,32,76] and are ideally suited to general practice. Many cases of dropsy, (C.C.F.), were cured by digitalis before its effect on Adenyl Cyclase was demonstrated[22].

For clinicians raised through their medical education in the scientific method embracing the empiricism of manual medicine presents a few philosophical problems, and one is chastened by the works of Bacon:

> "Medicine is a science which hath been more professed than laboured, and yet more laboured than advanced; the labour having been, in my judgment rather in circle than progression."[13].

However, with the comforting recent science just alluded to [4,5,6,7,8,9,10,29,32,11,12,76] and careful intrapersonal observation, it is possible to use Bacon's inductive method [23] to stitch a collar of scientific verisimilitude on the cloak of patient satisfaction. To what end must one go to quiet the superego of Science?

With this preamble exorcised, I intend to describe by which fashion my own study of musculoskeletal medicine has shaped and coloured my practice, and the extent to which it now differs from the orthopaedic soft tissue disruption/sprain and degeneration model that I believe our future practitioners are still being educated in. When I first decided the style of this essay some six months ago, I started to collate cases from my daily practice on file cards where there was a real disparity between what I was happy was the true diagnosis according to my own criteria and that which I would have received on a hospital discharge summary.



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These patient numbers are now greater than 70 cases and I think it neither useful not sensible to describe such cases in exactitude and at length. I intend instead to use the cases to illustrate points of discussion and alternative views to explain a view of dysfunction as the harbinger and eventual handmaiden of disease. In review of the literature it is reassuring to note that one shares company in such view with such greats as Osler [15] and Hippocrates [13] and to learn that the "biopsychosocial model" [24] has not only been pre-empted in time by these greats, but also in the elegance of their style of linguistic expression [15,25].

This discussion will be divided into two broad groups: firstly, diagnosis of more orthodox conditions which my education has heightened my diagnostic sensitivity for, and hopefully improved my treatment of, and secondly, a consideration of more "alternative" diagnostic and therapeutic categories with which I am comfortable and have daily use of, such as joint mobilisation, both spinal and peripheral, trigger point myofascial pain and somatovisceral associations and acupuncture.

It must be stressed at the outset that any diagnosis to be discussed has been made after exclusion of conventional pathological organic conditions by appropriate history, physical examination and investigation both laboratory and radiological, although in no way are they a diagnosis of exclusion. in keeping with the fact that for instance, `myofacial pain syndrome' and `somatic dysfunction', are codifiable diagnosis according to the International Classification of Diseases [26], these conditions have recognisable histories, symptoms on functional inquiry, and physical signs, and have now become part of my differential diagnoses for almost any somatic symptomatology imaginable. Accordingly, my physical examination simultaneously utilises more classical internal medicine palpation, percussion and auscultation and orthopaedic seeing, feeling and moving, but also such concepts as layer palpation [27], asymmetry, range of movement, tissue texture abnormalities [27,28,39,31], a search for the zones of irritation [12,18], abnormal motion barriers [27,28,12,30], and myotendonses as well as the palpable muscle bands [12,18] (all commonly associated with the somatic dysfunction), local twitch responses and jump signs of myofascial trigger points [29], and the hyperaemic vasomotor skin flare [16,29,30] panniculosis or pincer-rouler skin rolling [16,27,12,18,29,30] and sudomotor and trophoedemic changes [16,27,2,18,29,30] common to both conditions.

Amongst the commonest musculoskeletal diagnostic categories encountered in general practice must be the enthesiopathies characterised by such conditions as epicondylar tendonitis (tennis elbow), supraspinatus tendonitis and achilles tendonitis. While in no way disputing that there clearly exists situations in which partial tear or complete rupture occur or cystic degeneration supervenes. I have been struck by the frequency with which the tendon insertion is the principal site of symptom localisation and tenderness following minimal trauma or repetitious movement. These injuries are of such minimal force that direct trauma to the enthesis is commonly explained away as `pulling the tendon off the bone' or `tearing the tendon' clearly cannot be the initiating event. In these instances the concepts of myotendonses (tender muscle tendon insertions related to specific somatic dysfunction)[12], tender tendon insertions relating to specific peripheral joint dysfunction [27,30], and tendonitis changes associated with specific muscle trigger point (myofascial pain syndrome)[16,29] all become important. In some instances many features of all these concepts are present in one individual and treatment success may involve more than one treatment modality. A thorough search for enthesiopathies of muscles not commonly associated with such problems can often resolve a diagnostic dilemma such as a slow to resolve joint sprain or epicondilitis.

Although ultrasound investigation in shoulder and achilles tendon problems is useful [33], conventional exray studies (particularly special views such as the Stryker and West Point) remain a key investigation in recurrent shoulder dislocation [34]. In many cases of diffuse shoulder pain all investigations may be normal and in this circumstance the quality of information provided by ones initial examination becomes extremely important. Cyriax's examination technique for the shoulder remains a gold standard of information gathering [31], cervical movement and translatory movement in association with a search of zones of irritation in the neck will implicate cervical zygapophysical pathology[12,18,15,27,28,30], while Elveys brachial plexus tension test will rule out nerve root traction [35].

Wrights hyperabduction test and its variants points towards trigger point irritation in pectoralis minor and the scalene muscles (and cervical rib pathology) [16,29] and direct muscle palpation my identify specific trigger points around the shoulder [29]. Yeargasons supinations test defines biceps long head pathology and the three impingement tests of the greater tuberosity in resisted flexion, Maitland quadrant, and cave tests stress the subacromial contents of bursa and rotator cuff and maximally stretch the glenohumeral capsule[35].

Having taken a history and built up a seat examining the patient, it is still possible to not have a conclusive diagnosis at hand. Having found myself in this exact circumstance it was with great relief I remembered to test the translatory movements of the shoulder [30,36] and the apprehension test [37] and demonstrated the increased anterior translatory glide and positive reproduction of apprehension and feeling of weakness which characterises the `dead' arm syndrome' of transient glenohumeral subluation [37,38].

In the isolation of general practice it is extremely easy to undervalue ones own clinical aptitude and abilities and the most important ally that a general practitioner has in this regard is his/her own experience. Nowhere is this more obvious than in the interpretation of exrays where commonly one correlates ones own clinical examination with a rapidly available report, presented with all the authority and mana of a Consultant Specialist. It is only recently that I have allowed myself the realisation that the clinician best qualified to be the final arbiter in this decision making process is myself, possessing as I do the luxury of on the spot correlation of radiological and examination findings. The use of bone scan imagining is very important in musculoskeletal medicine; ie. in cryptic limb pain [39,40]. stress fractures [41], bone and joint infections [42], and reflex sympatetic dystrophy [43,44].

Continuing exposure to musculoskeletal education and increasing surety of the validity of ones own findings and conclusions gives one the courage to suggest improbable diagnoses. With increasing confidence in ones diagnostic abilities comes an increasing readiness to perform the therapeutic option must suited to each particular problem. Thus the more commonplace flexor tendon trigger finger injection [31] or elbow haemarthrosis aspiration in radial head fracture [46] are easily dealt with while the more hair raising procedures such as intra articular steroid injection of the hip joint [31,47,48], ,caudal route epidural steroid injection [31,49,50], ,or stellate ganglion blockade [51,52], takes a bit more getting used to. Since developing an interest in manual medicine, I have now attended some three related conferences, five courses in manual technique, and three weeks of live-in requirement for this present diploma. Although this input and my continuing studies have been captivating and interesting, by far the most intellectually and creatively satisfying activity has been writing my essay assignment last year on Autonomic Influences on Musculoskeletal Dysfunction[53]. Being forced to research this vast field and synthesise a coherent account of the interactions between the autonomic nervous system [54] the opiod peptide [55] and

neurotransmitter [56,57] systems and the somatic musculoskeletal system and neuroaxis/nociceptive[58] systems, will I am sure, stand me in good stead for the rest of my professional life. I am no longer able to view any injury from a purely inflammation and repair model perspective and now see all symptomatology from a interactive and modulatory pain theory viewpoint that gives equal credence to the concepts of antidromic neurotransmitter flow down sensory C fibres [59] neurogenic inflammation [60,61,62], spinal cord gating and brain stem inhibitory opoin pathways[63,64] and makes the biopsychosocial systems approach a real phenomenon not a sociologist's daydream.

Nowhere in my clinical work has this new outlook been more noticeable than my heightened sensitivity for sympathetically maintained pain [65] and neurogenic inflammation [61] in all their guises. At times I wonder if my enthusiasm for these concepts is causing self delusion, that the burning quality of pain a patient may complain of may have more to do with their hot water bottle in the bed than their locus ceruleus, but in general these concepts are useful, frequently seen, and go a long way to explain the common everyday troubling `chronic sprains' and `developing arthritis' with which we have been fobbing off patients for years.

Roberts, in a review article about causalgia and related pains, coined the clinically extremely useful concept of `sympathetically maintained pain' [66]. I find this term now appears throughout my case notes as SMP and in my usage describes those patients who may have some, but not all, of the features of a full blown diagnosis of reflex sympathetic dystrophy. It is not uncommon for such patients to have variable combinations of the following:- unusual cold sensitivity of an injury or alteration in pain state with ambient temperature changes, persisting oedema with or without undue tenderness (allodynia), undue clamminess or seating, an acrocyanotic hue to the affected limb, and most important, a burning quality to their pain (hyperpathia). These qualities may only be present in doublets or triplets and when improvement supervenes, leave one at a time. Not uncommonly, although the area of allodynia may be very localised, the acupuncture points of the affected limb are distinctly tender compared to the contralateral limb, not only those overlying the local nerve trunks, but noticeably also those points with known autonomic associations (Liv 3, L1 4, stomach 36, L1 11) 10. In the nine months I have been collating cases for this essay, I have noted eight such cases, four of which would fit the criterion for classical reflex sympathetic dystrophy [33,51] with almost all of the described symptomatology, the rest having SMP, and a brief mental review of the last four years extends these numbers to thirteen, eight of whom had RSD.

Although one case had had a CVA the RSD was secondary to a fall and fractured humerus. In two other cases the RSD developed in a hemipleged limb both about nine months after the stroke. Nearly all the other cases involved minor violence such as sprains to ankles and wrists, one fractured wrist and two involved the chest, one post sternonotomy for C.A.B.G. and one involving a car accident. One case is described because it shows the interesting phenomenon of a potentially serious and chronic pain condition developing in a woman with a strong previous depressive history and apparently being nipped in the bud by appropriate treatment directed at different modalities at once. Only one case involved damage to a peripheral nerve, considered by most of my colleagues with whom I have discussed RSD, to be the only causative agent in, to them, the incredibly rare diagnosis of `causalgia.' The interest in this case is that although a series of Guanethidine blocks has almost totally relieved the `sympathetic' symptoms of hyperpathia and allodynia and her neruogenically mediated synovitis in foot joints initially not involved in her injury, is still very severe and has only responded to manual translatory mobilisations [36,67].

Having developed an awareness of sympathetic interactions in pain state patients with migraine and the long recognised compression sensitivity on their carotid artery during an episode [72] present as an even more interesting symptom group than previously. Migraine is a disorder with many well recognised associations or triggers [17,85], including cervical somatic dysfunction[20,30,35], hormonal and autonomic input[10,73,74] and association with trigger point activation of cervical musculature[29]. With these concepts in mind my fascination with the following patient is not hard to imagine. The patient, to all intents and purposes, could be described as having `an upper quadratic dysautonomia with local myofascial trigger points, carotidynia, tinnitus, cluster migraine headaches and neurogenic inflammation of the face.' She would appear to fall within the category of recurrent carotidynia with related migraine[75], although I have not seen facial swelling or cluster symptoms described in other cases of carotidynia. The whole topic of migraine and its autonomic association has been elegantly summarised in a recent revision of Oliver Sacks classic work, "Migraine". Although space does not allow a full description of their cases, I have seen in one other patient with a similar condition, and two cases of isolated carotidynia of the benign isolated type related to viral illness[75,79]. Both cases in young patients followed a viral illness with elevated ESR's and C. reactive proteins and settled on high dose aspirin without recourse to corticosteroids. One of these cases was a serologically proven case of acute Epstein Barr virus infectious mononucleosis with a concomitant hepatitis and exudative tonsillitis which two months later also presented with a reactive arthritis and subsequently settled with conservative treatment. Another patient who subsequently developed a full blown sympathetic dystrophy related to his stroke, presented with neck pain, amaurosis fugax and contralateral hemiparesis demonstrating that the more serious form of carotidynia associated with carotid artery arteriosclerosis, aneurysm and thrombosis or giant cell arthritis are more common in the older age group[75].

When I come to review my accumulated file cards that relate to myofascial pain, acupuncture and joint mobilisation with and without impulse, I have recorded 50 cases in six months. These are the notable and unusual cases amongst my case load which would include at least two to four examples in each category daily, without having a super selected practice. In much the same fashion that I alluded to with sympathetically maintained pain, I am constantly left wondering whether, as my sensitivity to these conditions has gone up, my specificity has gone down [23] and that Don Quixote - like, I am tilting at myofascial windmills. It would certainly appear these collections of signs exist in association with matients specific and appropriate symptoms and these resolve with directed therapy. The philosophical principal of Ockhams Razor[81],which has always appealed to me, would seem to apply in such instances[77] and now leads me to believe that such phenomena as somatovisceral reflexes and modulation of visceral function by spinal manipulation or needle acupuncture truly exist[14,27,29,30,53,73,78]. Five years ago I would have had great difficulty accepting these tenants but the evidence of my own experience can now lead me to no other conclusion, and having accepted this and unshackled my clinical faculties I see increasing evidence of such activity ever daily.

Thus in the remaining part of this essay rather than detail every muscle or acupoint I have ever needled or joint over mobilised, I intend to focus on those examples that continue to amaze by virtue of their somatovisceral association.

Myofascial trigger points have long been known to have unusual autonomic and somatovisceral association[16,18,29,30,32] and in no one muscle are these associations more marked than the sterno

cleidomastoid[29]. In their text just mentioned, Travell and Simons with some degree of calm equanimity, describe associations between trigger point activity and cough, increased nasal and lacrimal secretions, conjunctival engorgement and a ptosis due to spasm of the orbicularis oculi in the sternal division of the muscle, and proprioceptive dysequilibrium in association with the clavicular division, aside from their more common universal pain reference patterns to throat, ear and face.

When I first read their text four years ago, I scoffed at these clinical syndromes. Now I can say I have seen each one and either reproduced them, cleared them (in the case of cough and dysequilibrium) or cleared them (in the case of conjunctival engorgement, lacrimal hypersecretion and pseudo-ptosis) by trigger point dry needle acupuncture[10.16.29]. I am never sure who is more amazed the doctor or the patient with the prompt pain and symptom reproduction and resolution with needle treatment. Examination of the neck and facial muscles and arteries, in association with the cervical movements and zones of irritation, are now a part of my standard examination of the head and neck. Sternocleidomastoid TP's are very commonly found in the presence of viral illness, recurrent throat pain in association with digastric TP's and almost universally in association with ENT diagnostic catch-all "benign positional vertigo" in the presence of minimal systagmus. This muscle in association with the trapezius are the two chief muscular sources of proprioceptive orientation of the head. This responds to muscle lengthening by neuromuscular techniques[28,30,35,36] and may in fact be the process by which 'extinguishing the vertigo' by recurrent provocation may work as prescribed by some ENT surgeons[80].

I will not begin to delve too deeply into a discussion on investigation and treatment of temporomandibular joint dysfunction as the topic is so vast[29,30]. This diagnosis and its referred pain syndromes alone or in combination with other myofascial TP's (Sternocleidomastoid, trapezius, masseter, medial pterygoid, temporalis, and frontalis) [29,30], comprise eight cases that I have noted but infact many other besides. Acupuncture is my treatment of choice, by dry needling preferably rather than acupuncture laser, unless the symptoms of locking are so prominent as to suggest irredeemable shortening of the superior division of the lateral pterygoid muscle with anterior displacement of the articular disc[16,29]. In the absence of other actiological factors, signs of functional restriction at C0-C1 and C1-C2 level are not uncommonly found in association with TMJ dysfunction.

Amongst the most common myofascial pain syndrome presenting with symptoms mimicking visceral disease, or more usually being interpreted by a fearful patient as representing same, are pectoralis major and minor and other chest wall TP's[29]. Pectoral muscle TP activity commonly present with chest or breast discomfort which may radiate to the shoulder but reassuringly from a cardiac standpoint has association with deep respiration or the perceived restriction of same, arm and shoulder extensions and abduction. The more dramatic whole arm radiation has been associated with specific pectoralis Minor TP activity on two occasions. This trigger point is discussed by Travell and Simons and the "Neurovascular Entrapper" of the axillary structures. The most dramatically unusual somatovisceral reflex I have seen has been the association of right 5th intercostal space pectoral muscle trigger points with frequent premature ventricular beats and supraventricular tachycardia[29]. Needling this frequently asymptomatic but unexpectedly tender TP for three patients has abated frequent symptomatic PVB noted throughout the day not just in repose. One patient is represented because a remarkable ECG was taken at the time of needling which stopped a one hour long episode of P.A.T. resistant to sudden surprise, valsalva, carotid sinus compression and oculocardiac stimulation. This occurred three years ago and unfortunately the ECG original is now misplaced and the xerox copy

has lost some fine detail. Once again, pectoral muscle trigger point examination is routine for me and pectoral muscle TP are commonly found in joggers, weight trainers, manual workers and typists[29]. As most patients are shy of needles, gravity assisted neuro muscular post isometric relaxation in the manner of Lewit[28,30,36] is excellent very effective self treatment which commonly works perfectly to restore muscle length and relieve pain. Another chest wall trigger point which produces symptoms very worryingly similar to visceral pathology is the Serratus posterior superior muscle. This pain syndrome is important as the pain is commonly felt much deeper "in the chest" than is common with other TP's and is strongly referred to the arm. The TP is not palpable and the diagnosis is not made unless the scapular is protracted forward and as this muscle is an accessory muscle of inspiration pain is much worse, "catching" on inspiration and pneumothoraces, pleural effusions, and pneumonias are commonly suspected.

Trigger point activation of the muscles of the abdominal wall and paraspinal area are a well recognised source of pseudovisceral symptoms, referred pain, and somatovisceral responses[10,29,53]. Nowhere else is meticulous care in examination as important as in ascribing potentially serious symptoms to abdominal trigger points. Normal full surgical examination, rectal and/or pelvic examination and urinary sediment and haemtological investigations of WBC and ESR are mandatory. However by utilising gentle minimum pressure layer palpation techniques[28,30] in association with what I annotate as North South East West examination (single finger top 1cm NSEW of an active abdominal TP will produce no pain) and provocation of light TP pressure exacerbation by forward neck flexion or bilateral ankle raise it is possible to ascribe symptoms of a visceral nature to abdominal muscle TP and abdominal scar TP's in a well looking patient who moves easily[29].

Once again as I have become more familiar with TP examination when examining patients abdomens, I routinely find reproducible findings pressure on which induces symptoms of a somatovisceral nature. As all neurones that are excited by visceral afferents have been shown to also receive somatic inputs and all visceral effevents are autonomic in nature the convergence projection theory helps to give scientific grounding to a concept initially very hard to feel very easy with [4,6,14,29,30,53,69]. In fact in my experience these seem the commonest examples of somatovisceral responses commonly seen in general practice and I regularly find and, the patients scepticism allowing, treat bloating and wind, gastric reflex symptoms, urinary frequency in association with sterile urine, nausea and cramp associated with dysmenorrhoea and severe colic associated with irritable bowel syndrome with trigger point acupuncture[9,10,14,29,30,82], with moderate and sometimes, spectacular success.

The associations of somatic dysfunctional disorders of the spine with altered autonomic function has long been recognised, not only as a function of individual spinal segment symptoms, but also through the realm of somatovisceral symptoms[10,12,16,27,30,35,53,73,78,83].

The more prolonged disorders of this nature occur at the junction of the major curves of the spine being occipitocervical, cervicothoracic, thoracicolumbar and lumbosacral and indeed the more chronic and persistent spinal somatic dysfunction commonly implicate one of more of these regions in association with other segmental levels[12,18,30,83]. The association of some specific disease states or symptom complexes with segmental localisation of dyfunction described by Lewit[30] seems to have definite validity. Briefly, these associations include; C0-1 migraine C7-T, autonomic disturbances such as migraine, T3-T5 gallbladder pathology, T12-L1 kidney disease in association with psoas muscle spasm, and L5-S, with gynaecological and bladder disturbance. Would that I could be an impartial examiner of the phenomena, but in

many conditions that involve disorder of function not anatomical form, I commonly look for, and find, these associations. Which is chicken and which egg, is a matter for speculation but many disorders such as migraines, recurrent suboccipital headaches, and sterile urinary frequency without dysuria, do improve with spinal manipulation as the following cases testify.

The patient took a long time before obtaining symptom improvement although the allopathic medical fraternity investigated at some length, (justifiably), to rule out life threatening disease. The manual medicine diagnosis which responded to treatment was short scalene muscles, cervicothoracic and C2-C3 dysfunction and dysfunctional right first rib as a case of unilateral right idiopathic oedema was made by myself two years before osteopathic treatment was obtained with success, following disappointing results from a highly trained physiotherapist manipulator and acupuncturist.

Age is no barrier to manual disorders and treatment of a 6 year old sequentially demonstrated two symptom complexes that were both most unusual, the first because of her age or lack of it and the second because it was the first association of thoracicolumbar and lumbosacral disorders with sterile urinary frequency that I have seen. Subsequently I have seen three other cases all in patients much older, although in their case I doubt that prolonged jumping on a new family trampoline was a important in the aetiology as it seemed to be in this young girl. Although in this slight young girl only thoracicolumbar signs were demonstrated, she did have a very tender symphysis pubis. The full syndrome, which I have only seen in women so far would seem to include long standing sacroiliac and lumbosacral problems[30], pelvic rotations with tender symphysis pubis zones of irritation[12,27] compensatory lumbosacral rotation[18,27] and thoracicolumbar somatic dysfunction with variable amounts of psoas spasm[12,27,30]. The psoas spasm mentioned above commonly can cause intraabomindal and pelvic pain of such severity as to cause concern in two young women of tubal infection, ectopic pregnancy, pyelonephritis or ovarian disease and in one male sportsman prompted his admission in a weekend to a surgical ward with suspected appendicitis. One of the young women mentioned is a ballerina and her psoas hypertrophy was elegantly demonstrated on the ultrasound performed to rule out pelvic pathology, her symptoms of pelvic pain settled promptly with neuromuscular stretching of he psoas and is included because not only does she show many of the features of this symptom constellation but also she had symptoms of meralgia paraesthetica of the thigh. Although always considered idiopathic by many[84] or associated with psoas pathology[30] in her case treatment directed at an anterior innominate rotation ipsilaterally resolved the symptom almost immediately.

Although described in separate groups in this essay, somatic dysfunction and myofascial trigger points are not separate non interactive entities at all and unfortunately there is not room to describe the clinical interactions of these two clinical schemata[29,30] both in theory and in practice[10,12,16,18,27]. The management of chronic pain states such as post herpetic neuralgia with neuromodulation by tricyclic and anticonvulsant/anti-arrhythmic drug therapy and CODERTRON TENS machines has been left untouched, as have fibromyalgia[53], corticosteroid interarticular therapy and the fascinating topic of scar therapy[30].

Of most practical everyday importance to my practice has been the development of my interest and abilities in peripheral joint mobilisation and manipulation by neuromuscular therapy, translatory gliding mobilisation, pain release phenomenon combined compression and gliding, and impulse techniques[27,28,30,31,36,67,85]

It seems inconceivable to me to consider practicing my craft without regularly mobilising such joints as the subtalar,

superior tib.fib, acromioclavicular, radiohumeral and radiolunate joints and carpal tunnel complex yet clearly these techniques and the necessity for them were lost on me less than four years ago.

There is much in the above discussion that the professor never taught me, but I am comfortable with that thought. My practice of the art and science of medicine is markedly different not to that which he taught me. My hope is that I continue to acquire the new skills and grapple with the theories of different disciplines that form a synthesis that furthers a healing art.

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REFLEX SYMPATHETIC DYSTROPHY
A REVIEW

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Reflex sympathetic dystrophy is a complex clinical entity comprising pain, trophic changes and vasomotor disturbances. First described in 1864, pathogenesis of R.S.D. still reamains in doubt and maybe associated with a spectrum of precipitating factors. Diagnosis is purely clinical and treatment should be early and aggressive. Once fully established, pronosis is poor.

Reflex sympathetic dystrophy (RSD) is a complex syndrome of pain, trophic changes and vasomotor disturbance usually affecting part or whole of a limb.

The term algodystrophy is used in the European literature[1].. There are a plethora of other terms, perhaps reflecting the lack of consensus regarding its aetiology and pathogenesis (Table 1).

Table 1.

Names for reflex sympathetic dystrophy
Causalgia
Minor causalgia
Major causalgia
Mimo causalgia
Acute atrophy of bone
Sudeck's atrophy
Post-traumatic osteoporosis
Traumatic angiospasm
Peripheral acute trophoneuritis
Shoulder-hand syndrome
Postinfarctional scleradactyly
Algodystrophy
Algoneurodystrophy
Reflex dystrophy of the extremities
Reflex neurovascular dystrophy
Reflex sympathetic dystrophy syndrome

(adapted from Steinbroker & Argyros, 1958)[10]

The condition was First described by American Civil War surgeons Mitchell, Morehouse & Keen[2] in 1864 and Mitchell in 1972[3] noting that soldiers with gunshot wound injuries of peripheral nerves sometimes had persistent burning pain and progressive trophic changes in the affected limb. The soldiers would fill their boots with water and wrap the affected limbs in wet rats to "extinguish the fire". In 1867, Mitchell[4] named this entity causalgia, from the Greek `kausos' (heat) and `algos' (pain)

Prevalence

There is little epidemiologic data on RSD, no doubt in part due to difficulties in defining inclusion criteria. In several studies the incidence of RSD after peripheral nerve injury ranged from 1015%[5-7]. The occurrence of RSD after myocardial infarction has dropped to less than 1%[8-9]. It occurs at all ages, more commonly in women, and the incidence increases until late middle age [10-13]. In a study of the incidence of RSD in veterans with peripheral nerve injuries, Rothberg et al[6] found a rate of 10-15% in patients aged 17-34 years and 47% in patients 35 years or older. In two similar series of 140 cases[14] and 61 cases[15] no difference was seen between various age groups.

It is less commonly recognised in children although there are now up to 90 reported cases in the literature[16,17]. Most authors feel that the prognosis in children is generally better than in adults. In a recent review of paediatric pain of uncertain aetiology, the final diagnosis was RSD in 11%[18].

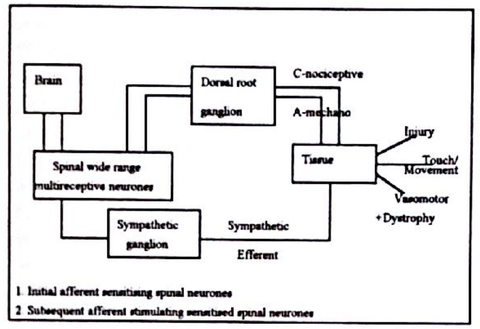
Pathogenesis

Pathogenesis is poorly understood. It is generally agreed, however, that the sympathetic nervous system plays a vital role.

It has been proposed that sensitised wide range multireceptive neurones in the spinal internuncial neurone pool are at the centre of an abnormal reflex, resulting in excessive sympathetic outflow. For unknown reasons, sensitisation occurs after initial nociciptive afferent stimulation, which subsequently results in abnormal pain perception and increased sympathetic afferent activity. Persistent afferent input from damaged soft tissue nociceptive fibres was thought to be important. This input is now known not to continue once a tissue has healed and yet many cases of RSD occur without dystrophic changes. Instead it is postulated

that subsequently abnormal responses to mechanoreceptor afferents occurs[19]. (Figure 1)

Figure 1.



Expansion of the area of sensitized wide range multireceptive neurones within the spinal cord can then give rise to spread of the condition. The influence of descending pathways explain an association with cerebral lesions. Psychological status might affect the condition by this mechanism.

Alternative local tissue mechanisms have also been proposed as being important, whereby a series of vicious circles is established in which vasodilatation, low flow and persistent stimulation of nociceptors all play a part[20].

The authors also suggest that alteration in adrenoceptors may be involved.

Other authors have postulated a role for oxygen free radicals[21], response to infection[22] and neoplasia[23], the latter two indicating possible immunological factors. The role of local vasoactive interstitial polypeptide has also been postulated[24].

This area is a complex one and the exact contribution of the above factors is uncertain. Recognition should be given to the earlier work of Devor[25], Melzack[26], and Melzack & Wall[27] on whose concepts several of the above mechanisms are based.

Clinical Presentation

RSD is associated with a wide variety of precipitating factors. Trauma is the most common, however, associated conditions are protean. (Table 2). In up to one-third of cases there may be no identifiable cause. The means by which these events cause the same clinical syndrome is uncertain, but the common mechanism may be injury to either central or peripheral neural tissue, including peripheral nerve twigs[25].

The Symptoms may begin gradually, days or weeks after the injury, or may manifest within a few hours[2,5,28,29]. The patient suffers greatly and protects the affected area [3,10,11]. Classically it involves a limb starting distally and spreading proximally. However, case reports of RSD include involvement of the entire body[30], one finger[32] and penis[33]

Table 2.

Precipitating factors and diseases associated with reflex sympathetic dystrophy	
Peripheral	Soft tissue injury
	Arthritides
	Infection
	Fasciitis, Tendonitis, Bursitis
	Venous or arterial thrombosis
	Fractures, sprains, dislocations
	Operative procedures
	Malignancy
	Aortic injury
	Myelography, spinal anaesthesia
	Paravertebral alcohol injection
	Post-herpetic
	Brachial plexopathy, scalenus anticus syndrome
	Radiculopathy
	Immobilisation with cast or splint
	Vasculitis
	Myocardial infarction
	Weber-Christian disease
	Polymyalgia rheumatica
	Pulmonary fibrosis
Central	Brain tumour
	Severe head injury
	Cerebral infarction
	Subarachnoid haemorrhage
	Cervical cord injury
	Subacute combined degeneration
	Syringomyelia
	Poliomyelitis
	Amyotrophic lateral sclerosis
Other	Idiopathic
	Prolonged bedrest
	Familial

Adapted from Schwartzman & McLellan, 1987[43].

The dominant symptom is pain, frequently of a burning quality whose severity is out of keeping with the initial insult. Other typical features include hyperaesthesiae, vasomotor disturbance, hyperhydrosis and trophic changes. Classical stages of early hyperaemia and swelling, subsequent ischaemic dystrophy, and eventually and atrophic state vary greatly in time. Early changes are often not seen and progression to dystrophy/atrophy may not occur. In addition, vasomotor changes leading to skin cooling and mottling may be intermittent.

It is difficult to clinically define such a variable polymorphic condition. However, it may be useful to consider RSD in terms of either stage or grade[24]

Clinical Stages of RSD

	Stage 1	Stage 2	Stage 3
Onset after injury	days-weeks	months	months-years
Vasomotor	vasodilation	vasoconstrictions	vasoconstriction
Skin/trophic changes	erythema sweating oedema	pallor, cyanosis sweating indurated swelling patchy osteoporosis	pallor, cyanosis dry, thin skin tissue atrophy diffuse osteoporosis

Clinical Grades of RSD

	Grade 1 (severe)	Grade 2 (moderate)	Grade 3 (mild)
Pain	stabbing, burning Lancinating	dull, aching, diffuse	insidious onset
Vasomotor	Severe vasoconstriction or vasodilatation	skin cold + clammy or hot + dry	mild, but different from unaffected side
Trophic	Fixed atrophy of skin subcutaneous tissue muscles + bone	with or without osteoporosis on x-ray	absent with or without oedema

Psychologically, patients with RSD exhibit a wide range of behavioural changes. They are frequently depressed, anxious and socially withdrawn. Suicidal ideation and drug addiction may occur. Great care must be taken before labelling the pain psychogenic.

Diagnosis

The diagnosis of RSD is primarily clinical[11,34,35]. It is a perplexing syndrome and can be difficult to diagnose as the signs and symptoms are varied, and its multiple manifestations change with time.

It is important to exclude other somatic causes of pain. These include infection, bursitis, atypical inflammatory arthritis, carpal tunnel syndrome, scleroderma, peripheral vascular disease, tenosynovitis, postherpetic neuralgia, peripheral arterial occlusion, spinal nerve and root impingement, peripheral neuropathy, compartment syndrome and malignancy,

Several other diagnostic measures can help confirm one's clinical impression. Plain x-ray studies may show patchy osteoporosis in early cases or diffuse osteoporosis in more advance cases. However, osteoporosis is not universally present. Magnification radiography shows subtle bone changes not detected on plain x-ray films[36]. Isotope bone scanning is as useful and probably superior in early cases[37]. Three phase scans using technetium-99m methylene diphosphonate recording initial (blood flow), early (blood pool) and delayed (tissue uptake as an indicator of active turnover) uptake are advocated, as increased vascularity may be the only abnormality, especially in subjects with early or mild disease. However, reduced rather than increased uptake is commonly the abnormality seen in children and adolescents[38].

Thermography has also been used in the diagnosis of RSD[39]. It is a useful non-invasive method by which vasomotor abnormality of the affected limb can be assessed objectively. A thermogram is considered positive if there is a temperature increase (hypothermia) or decrease (hypothermia) of at least 1°C in the painful area compared with the opposite side. This test however requires a sophisticated thermography system which may not be available.

Abnormal levels of pain are central to RSD and assessment of its severity are important both in diagnosis and in monitoring progress. Multiple measures of pain severity have been proposed[40], though a simple visual analogue scale is generally most useful. The measurement of pressure-pain thresholds using relatively simple and similar instruments such as a dolorimeter[41] and an algometer[42] give a more reliable and objective assessment of tenderness.

There are no consistent abnormalities on routine blood testing although diabetes mellitus, hyperthyroidism, hyperparathyroidism and type IV hyperlipidaemia are predisposing factors[1,37]. Increased urinary hydroxyproline excretion may be found in early disease[1].

American authors recommend sympathetic neural blockade as the most useful means of confirming the diagnosis of RSD[43-45]. The procedure involves placing a needle next to the stellate or lumbar ganglion and infusing 8ml of normal saline into the area (as a placebo). If pain relief is not

obtained after 10-15 minutes, 8ml of 1% procaine hydrochloride is then injected which blocks only the sympathetic fibres. If pain relief is still not achieved, 20-30ml of 1% procaine solution is injected into the brachial or lumbar plexus. If this treatment fails to bring relief of the pain, a central origin (eg cerebral infarction, brain tumour, syringomyelia, cervical cord injury, subarachnoid haemorrhage or poliomyelitis) should be considered.

A similar regional effect can be achieved with a simpler Biers block technique using intravenous guanethidine (not available in USA)[46]. This will be discussed further in the management section.
On the whole, patients with RSD who are diagnosed early and treated promptly have a better outlook. To make the diagnosis one needs to consider RSD in any patient with prolonged pain following an insult. Whilst in the patient with a typical history of minor injury followed by prolonged pain, vasomotor and trophic changes, it may be relatively straight forward in those with atypical or only partial features of the syndrome it is much more difficult. Hence the need for a high index of suspicion, careful history and examination and the appropriate use of ancillary investigations in the diagnosis of RSD.

Management

Over the past 100 years or more there have been a wide variety of therapies advocated for the treatment of RSD (Table 3). This reflects our difficulties with the pathogenesis, lack of comparative therapeutic trials and also the fact that not individual treatment is universally effective.
However, most would agree that diagnosis must be made early, and treatment immediate and aggressive. Treatment must be aimed at the restoration of movement and function to desensitise the abnormal reflex, and so physical therapy is the cornerstone of treatment. This may be effective alone[37,47-49] but the severity of pain often prevents adequate participation, thus the need for other measures[6,50,51].

Apart from analgesics, transcutaneous nerve stimulation (TENS) may be useful[52]. It is postulated to relieve pain by an artificially generated barrage of nerve impulses in large axons[53]

Table 3.

Treatments used in Reflex Sympathetic Dystrophy
Physical therapy
Cold, wet compresses
Hot wax applications
Anti-inflammatory agents
Radiation therapy
Hypnosis
Biofeedback
Muscle re-education
Electroacupuncture
Immobilisation with cast or splint
Diathermy
Transcutaneous nerve stimulation (TENS)
Trigger point injections
Thalamotomy
Ultrasound to the stellate ganglion
Corticosteroids
Propanolol
Phenoxybenzamine
Bier block with guanethadine
Bier block with reserpine
Bier block with lignocaine and corticosteroid
Stellate ganglion block or paravertebral block
Continuous paravertebral sympathetic ganglion block
Periarterial sympathectomy
Tricyclic antidepressants
Psychotherapy
Capsulotomy
Tendon release
Mobilisation performed during anaesthesia
Calcitonin

(Adapted from Schwartzman RJ, Mclellan TL.[43] Reflex sympathetic dystrophy: a review. Arch Neurol 1987; 44: 555061, and Levine DZ.[74] Burning pain in an extremity. Breaking the destructive cycle of reflex sympathetic dystrophy. Postgrad Med 1991; 90: 175-85.)

Various medications such as propanolol[54], phenoxybenzamine[55]m nifedipine[56] and high dose oral corticosteroids[37,57] benefit some patients. Parenteral calcitonin can also be useful[1,7,58], particularly in early ankle and foot disease. A recent study using nasal calcitonin showed no demonstrable effect[59].

Sympathetic blockade is logically the best way of suppressing the sympathetic hyperactivity but is invasive and requires specialist techniques. Multiple blocks are often required and the effect maybe only short-lived[60].Hannington-Kiff[46,61-

[63] developed a method of regional sympathetic blockade using Bier blocks performed with 10-20mg quanethidine sulphate. Guanethidine displaces noradrenaline (norepinephrine) in presynaptic vesicles and prevents its uptake. Excellent results for pain relief utilising this technique have been reported [64,65]. The pain relief usually lasts from 12-36 hours but it may last for up to six months[66]. Repeated blocks with guanethidine have a cumulative effect[67,68]. Similar efficacy had been reported with the use of reserpine[68] which depletes storage of noradrenaline. Other substances, including local steroids plus lignocaine, have been used with less effect.

Prolonged blockade can also be achieved by infusions of local anaesthetic through an epidural catheter and shows promise, often in association with the use of epidural opiates[69]. Surgical sympathectomy and paravertebral ganglionectomy have been used in selected cases[70]. With all these techniques, an active rehabilitation programme is required to optimise their effect.

Manipulative techniques have shown promise in the treatment of peripheral neurological syndromes[71,72] and some authors believe it may be worthwhile in the treatment of RSD[73,74].

Reflex sympathetic dystrophy may be transient and self-limiting but frequently suffering persists for years and may become permanent. Greater understanding of central neurological and local tissue mechanism is needed. Management is hampered by lack of comparative data between different therapeutic regimens. Clearly more research is required into the pathogenesis and management of this syndrome first described by Mitchell in 1864. It remains as great a challenge today as it did to Mitchell and his colleagues during the American Civil War. As in many areas of medicine, whilst much knowledge has been gained, even more questions remain unanswered.

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CASE STUDIES

This new column is being trialed as a teaching aid and hopefully will generate general interest. The first thee reports have been written by diploma undergraduates undertaking the Flinder's Diploma Course.

In Practice we all see diagnostically complex or interesting cases and by sharing these experiences can help other colleagues and ultimately our patients. The aim of this column is not to just have case studies presented by the Association teachers, though these are welcome, but to involve all grass root members. It would be ideal if all A.A.M.M. members forwarded one case study per year.

1. MW - FEMALE - AGED 67

History

A 67 year old widowed female, complaining of right hip and knee pain for nine months. Seen by LMO for xrays which were taken of the hip and pelvic region but showed mild degenerative changes only. The pain persisted and the patient was seen in Casualty at The QEH early January 1994. At the time of presentation, the patient complained of a dull ache in the knee and the hip, especially after sitting and lying down. Also pain in the calves from time to time and pain not relieved by Naproxen, only Panadeine Forte. Cannot weight bear and feels as though she would give away if she continues to walk. There was no neurological deficit but lying on the bed on her side caused pain. There is no history of trauma and no family history of rheumatoid arthritis.

When seen in Casualty, an xray of the lumbar spine was taken. The xray of the lumbar spine showed retrolishthesis of L2 on L3 with marked sclerosis along the inferior border of L2 and the superior border of L3. There was also narrowing of disk space at this level as well as at the L4-5. Other than that there was no other mal-alignment, although there was some marginal osteophyte formation consistent with spondylitis.

Examination

Seen with considerable discomfort in Outpatient Clinic, where the patient hobbled down the passageway and was unable to adequately weight bear. Side-bending to the right and to the left was 25% of normal. Extension was 5% but she could flex quite easily down to her ankles.

Straight leg raise was within normal limits, there was no pain on testing of the sacroiliac joints. Hip movement on the right was relatively painful, as was palpation over the area of the greater trochanter with some tenderness being experienced over the region of the piriform muscle.

On the basis of clinical examination, a diagnosis of greater trochanteric bursitis was made.

Treatment

The area of tenderness was injected with 0.25% Lignocaine and 2 vials of Celestone. Following the injection of a total amount of 10mls, the patient was pain-free, could weight bear without discomfort and moved freely in all directions.

Prognosis

The xrays showing degenerative changes are undoubtedly a red herring and the area of injection should respond to the treatment given. The patient had two weeks relief before onset of symptoms again. Injection has been repeated and the patient given gentle stretching exercises to maintain mobility of the lumbar structures and has been referred back to the LMO.

Points to Note

1. Pain in the hip is not always referred from lumbar spine.
2. A painful gait is not necessarily directly related to weight bearing structures.
3. Always reproduce the patient's pain.

2. DW - MALE - AGED 32

History

At the time of the accident this man was 28 and employed as a catering officer. He was active in sport and had a stable family background. The accident occurred when he was lifting a heavy box from on top of a shelf, when the object he was standing on gave way, he twisted and fell to the ground landing on his left foot in a twisting fashion while still holding the box. The height from which he fell was of the order

of 1.2 metres and he immediately felt pain in the central lumbo-sacral area. There was a rapid onset of pain and he saw his local doctor for conservative treatment consisting of rest, time off work, meld analgesics.

After a period of six weeks without adequate resolution of his pain an orthopaedic surgeon saw him and injected a steroid to some area of the lumbo-sacral structures. The area of injection was not known and the patient was not given any explanation. Following failure to improve the patient proceeded to manipulation under anaesthetic which the patient maintains made him significantly worse to the extent that in addition to his lumbo-sacral pain he had now pain over his left shoulder girdle.

Over the ensuring two years the patient was unable to work because of central lumbo-sacral pain for which he had further xrays, CT scan and MRI of the lumbar spine. The only abnormality indicated a small bulge at L4,5 not pressing on the nerve root but lightly denting the thecal sac. Treatment from physiotherapists and chiropractors was mostly that of mobilising which usually made the patient worse.

At the time of consultation the patient's major complaint was that of severe lumbo-sacral pain which prevented him from walking any distance. He could not participate in the smallest of household chores because of exacerbation of his pain if he did not take large quantities of digesic and at times codeine phosphate. He tried to maintain fitness by swimming, but any excess activity necessitated rest in bed to overcome the induced pain. When the pain was severe in the back there was also some diffuse discomfort into the buttock and down both thighs. There were no relieving factors but he could manage to have his meals by kneeling down and bending forward over a chair, or some other supporting structure. Travelling any distance requires previous analgesia.

Examination
On examination a very solidly built young man who looked mildly depressed. Standing posture indicated a loss of lumbar lordosis but his pelvis was level. There was a minor thoracic kyphosis. Side bending to the right and to the left was 50% of normal with reproduction of pain on both sides. He was unable to stand fully weight bearing on each leg, although when he stood evenly poised on both legs there was considerable discomfort compared with lying down. Extension was possible only to 10 degrees and flexion was possible to knees. Both of these reproduced his central lumbo-sacral pain with the pelvis firmly anchored rotation of the trunk to the right and left reproduced his pain. Quadrant testing to the right and to the left reproduced his pain in the lumbo-sacral area on each side, respectively.

In the supine position straight leg raise was possible to 50 degrees on both sides with reproduction of his central pain. Hips moved equally without discomfort but he was exquisitely tender on testing of right and left sacroiliac joints with tenderness over the symphysis pubis. Hip flexion at 120 degrees bilaterally also reproduced his lumbo-sacral pain. In the prone position the femoral nerve stretch test was negative but there was marked tenderness to PA mobilising of the PSIS and palpation over the lumbo-sacral interspinous space and iliolumbar ligaments bilaterally were very tender. There was not neurological loss and palpation over the region of the erector spine muscles was not tender but tenderness was elicited over the piriformis muscles bilaterally. The slump test was not done because of the patient's discomfort. No abnormalities were found in the cervical spine or shoulder girdle, except for loss of pinprick over a small area on the shoulder in the C5 dermatome.

Assessment & Treatment
A diagnosis of pelvic instability was made with

particular pain arising from the sacroiliac joints bilaterally with possible contribution of his pain arising from the piriformis muscles bilaterally. The placement of a tight trochanteric belt just above the greater trochanters and repeat of the manoeuvres greatly diminished the patient's pain. The patient was informed to wear this belt for the next six weeks and to do pelvic floor exercises as well as mobilising exercises for the lumbo-sacral structures with the belt in place. In the meantime a double blind injection of normal saline and 1% lignocaine into the sacroiliac joints established the fact that this patient's pain arose predominantly out of the sacroiliac joints.

On review at 3 months after initial consult, this patient has made significant improvement, his consumption of analgesics has ceased. He is more active in ADL's.

Points to Note
1. The importance of reproducing the patient's pain is emphasised.
2. Too often central pain is though to be due to a prolapsing or sequestrating disc leading to unnecessary surgery.
3. The sacroiliac joint is frequently ignored as a source of low back pain with diffuse radiation. The reproduction of the patient's pain together with the elimination by selective analgesic blocks is a cost effective and efficient way of arriving at a diagnosis and instituting appropriate treatment. The efficient assessment by a musculoskeletal physician often has more to offer the benefit of the injured worker than does assessment by other specialities.

3. MD - MALE - 30 YEARS

History
Working as a spot welder for Mitsubishi in May 1992 using a machine welding gun attached to an overhead spring-loaded support. This apparatus required two handed operation with the left hand steadying the machine and the right hand used to work a trigger control mechanism against significant pressure. Several weeks after commencing work he noted a painful right wrist with pain extending up the arm involving thumb, volar aspect of the wrist as well as the flexor and extensor musculature of the forearm. He persisted with work but then complained of pain in the right side of the neck and right shoulder.

Reviewed by the Company Doctor who diagnosed a tendonitis and arranged alternative duties. The wrist and discomfort problem persisted and he saw a GP who diagnosed a 'wrist problem' as well as tennis elbow. Physiotherapy was arranged while on light duties but did not produce any resolution of the problem whereupon he was referred to an Orthopaedic Surgeon who performed wrist arthroscopy. A band of thickened synovium was removed and the patient showed significant improvement in the wrist but the shoulder and neck pain persisted.

He continued to work on light duties with the wrist pain settling but now experiencing significant headaches with stiffness of the right shoulder and cervical spine on waking.

At time of presentation to a Musculoskeletal Physician the patient's major complaint was that of frequent occiptal headaches accompanied on occasions with a burning pain in the flexor aspect of the forearm. Movements of the shoulder reproduced pain over the upper aspects of the right scapula and right side of the neck.

Examination
A well built man who stood with good posture. Flexion of the right upper limb was full with pain at end of range, extension was also full with pain at end of range occurring over the deltoid and scapular regions. Abduction to 45 degrees reproduced pain in the shoulder region with some paraesthesia becoming evident to the thenar eminence. Right brachial plexus tension test was positive producing pain and numbness in the C6 dermatome. There was no muscle

weakness in any of the myotomes of the upper limb and all reflexes were brisk. Internal and external rotation of the upper limb with elbow flexed was within normal limits. Dysaesthesia was noted in the radial distribution of the right hand.
Palpation over the right shoulder girdle musculature revealed several tender points in the trapezial, levator scapulae suprapinatus and erector spinae musculature. The cervical spine moved 50 degrees in flexion, 70 degrees in extension with reproduction of pain at end of range. There was 80 degrees of rotation to the left, about 60 degrees rotation to the right again with reproduction of the pain in the right shoulder. Sidebending to the left was 50 degrees with sidebending to the right 30 degrees again reproducing pain in the right shoulder and quadrant testing on the right was also positive. Palpation over the right cervical facets from C3,4 to 5,6 were tender but did not reproduce the neurological findings as indicated above.

Investigations
Because this was a Work Cover presentation plain xrays of the cervical spine and right shoulder were ordered as well as ultrasound of the right shoulder. All these were within normal limits. On referral to a neurosurgeon, a CT scan MRI and CT myelogram were also ordered. The findings of these indicated mild indentation of the posterior longitudinal ligament at the 5,6 and 6,7 levels without evidence of compression of the cord. There was some abnormal filling of the right C6 nerve sheath with no indication of disc involvement.

Assessment and Treatment
Cervicogenic pain arising from the C3,4 - 5,6 cervical segments with possible involvement of neural tethering.

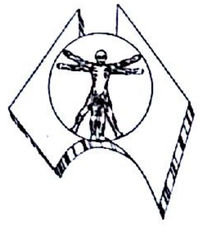
Treatment consists of traction and post isometric stretching exercises along with PA mobilising of possible stiff Z-joints at the levels indicated.

Progress
After two to three weeks of treatment the patient was showing slight signs of improvement with an increased range of movement of the cervical spine and shoulder girdle with improvement in the frequency of headaches.

He will continue to be monitored for improvement.

Points to Note

1. Workers with overhead machinery that requires a large amount of flexion and wiggling to get the machines into position can acquire cervical and shoulder dysfunction.
2. The ordering of expensive investigations is not always rewarding especially in the absence of a concerted trial of appropriate physiotherapy to the damaged structures.
3 Perhaps attention earlier to the discomfort in the shoulder and neck with early treatment could have avoided a series of expensive investigations.



OCCASIONAL ADDRESS

Earl Hackett

The following address is a reprint from the Bulletin of some 8 years ago. There have been several requests to me to reproduce it and the decision to do so was based on the fact that we now have many new members, plus the New Zealand association members who have, in all probability, not been acquainted.
The address was given by Dr. Hackett at the beginning of the morning session of the Adelaide annual conference on 24th November, 1985. The occasion was to mark the safe return of the party dispatched to McLaren Vale the evening before to seek victuals.

They say That God made idiots for practice and then He made after-dinner speakers. I have about thirty years experience of speaking after dinner and therefore I know the futility of it.

Here at this conference though, your Association has offered me something quite new. I have never before spoken after dinner where the chairman took twelve hours to quieten the guests to a point where they could conceivably hear the few words from the speaker.

Last night didn't at all match up with Dr. Broadhurst's pre-dinner instructions to me. Very sedate conservative people he said. They'll appreciate a serious approach. Well, I stuck to those instructions. The serious approach has now resulted in us getting seriously in touch. And that indeed is what I wished to talk about. For I myself spent a medical career in a non-touching area of medicine and I've lived to regret it. You manipulative doctors are all touchers. And good on you, mates.

The nature of all living things on earth is such that all of them are in touch with the environment in which they live and many come into physical contact with one another. We all rub, as we say shoulders; but other surface parts, too, are rubbed as life seeks, avoids, invades, destroys, looks after, mates with, gobbles up, is gobbled up by, or simply blunders into.

Many living things have of necessity developed, all around their boundaries, signalling arrangements which indicate with varying sensitivity, when something, or someone, has been touched. It is particularly important for moving social species to be able to discriminate promptly between a fellow and a stranger.
Touch is very old. Touch is deeply placed. Touch has an all-overness that can make great big touching a very fulfilling bodily experience.
As mewling infants we use it to explore our own selves, to discover where we begin and end; the up and down and fore and aft and side to side of body and space. The touching skins of suckling baby and nursing mother are the feeling origins of being bonded with or separated from another person.

When I was twenty inches long
I could not hear the thrush's song,
The radiance of morning skies
Was most displeasing to my eyes.
For loving looks, caressing words
I cared no more than sun or birds,
But I could bite my mother's breast
And that made up for all the rest

That is Frances Cornford — and here is D.H. Lawrence, a master of the use of words to describe sensations:

Between her breasts in my home, between her breasts.
Three sides set on me space and fear, but the fourth side rests
Sure and a tower of strength, twixt the walls of her breasts.
All things on the move, going their own little ways, and all
Jostling, people touching and talking and making small
Contacts and bouncing off again, bounce! bounce! like a ball!
I am that I am, and no more than that: but so much
I am, nor will be bounced out of it. So at last I touch
All that I am-not in softness, sweet softness, for she is such.

That describes the embracing reassuring approval of a surface caress; and its corollary, the agony and anxiety of being disapproved, outcast, separated from touch and comfort.

Some parts of our bodies are more sensitive than others because they contain greater

densities of those nerve endings that relay the characteristic feelings of touch, pain, deep pressure, temperature and the relative position of a body-part. Thumb and index finger, lips and tongue are strongly represented in the sensory cortex of the brain where the messages coming in from the skin have equivalent representation in the neurones. There they are sorted and interpreted and woven into realities and into the symbolisms, the phrases and the fables about touching and stroking and kissing and caressing, about bonding or separating, about hailing or farewelling, with hand or lip.

Touching on this touching—friends will lose touch, get in touch, and keep in touch, while enemies will not touch one another with barge poles. Seafarers touch at foreign ports. Footballers have touch-downs and touch-lines. Yokels touch forelocks. A con-man touches you for fifty dollars. Fencers cry *'Touche'!* Sentimentalists feel quite touched. Artists touch up pictures. Drinkers with hangovers swear never to touch the stuff again. Your granny has a touch of rheumatism, but no one can touch her at making lamingtons, even though you sometimes think she's a little touched, but of course for anyone to say so would touch her to the quick because she is so touchy, but we all hope she'll have many years yet, touch wood, though it will, of course, be touch and go.

In my youth, aviators used to cry `Contact!', giving out what seemed to be a last cry of ecstasy before they roared off the ground. `Separation!' would have been a more appropriate salute, but to each his own, whatever turns you on: whatever gets your prop going, whatever touches you deeply.

In spite of all that idiomatic metaphor, you will find very little poetry about touching in English literature except in cabinets of erotica and curiosa. The eye seems to be the English poet's prime organ-source of imagery, and the ear the organ of persuasion. The English-speaking world must have decided that touching between grown persons was sexy and dangerous, something to be got around, repressed or ignored, rather than welcomed as a positive part of human nature. Added to this, the imprecision of skin sensation excluded it from the applied science and technology of the industrial revolution. To English minds today, the strong embraces of triumphant hispanic footballers on the field, or the kisses and bear hugs with which overcoated Russian leaders greet one another at snowy airports, are risky, foreign stuff.

As nations vary in the amount of ritual public touching they permit among adults, there is also individual variability engendered as we reinforce self-hood or otherness with personal preferences for privacy or modesty, which then further affect how we shall touch our skins to other skins, in ritual, friendship or love.

This has always complicated medical diagnosis and treatment. We, in the West, owe our direct national hands-on doctoring to the Hippocratic School of classical Greece. Other ancient medical systems, such as those of Egypt, China, India and medieval Europe had more of magic, ritual, elaborate potions, books and numbers, at least as they have come down to us. Although those Hippocratic doctors of two thousand years ago had also their healing temples with mystery visits in the night by priests dressed as Aesculapius bearing baskets of temple snakes, they at the same time advocate a very common-sense, humane approach to diagnosis. On a first encounter with a sick person, they talked carefully about her or his disease. This was the `history', a Greek word meaning inquiry, which is to say that it referred to the personal conversational process by which the doctor elucidated the experiences of the patient. Then they would feel, listen and even taste to find out what was going on in the sick body.

When you're sick in a chronic or wearying way — indeed when you're sick with most diseases, you tend psychologically to regress. To go back to dependent infantile attitudes.

You curl up, miserably, like a baby. You say little, except maybe to complain. Even if you are the heroic type, you almost certainly whinge inwardly to yourself. You become temporarily dependent on those immediately around you. They, alarmed, send for the doctor.

If it were a member of the school of Hippocrates responding to that call, he would be grave and bearded. He would talk to you gently about yourself. Perhaps he would put his hand on yours or on your arm, as he asked sympathetically: When did you have a cough? A pain in your chest? How long ago? What did you cough up? Did the pain go away? Or is it still there?

The inquiry. The history. But as he talks to you there is a feeling of the damn sickness being shared, and this brings remarkable relief. Moreover this Hippocrates is fond of people. He likes them. He seems to like you as much as he is intensely interested in all the patterns of human disease.

You glance at his hands. They are clean and the nails are evenly trimmed. Such hands would have been unusual in ancient Greece, where there was no soap; and bronze scissors were hard to come by. He probably smoothed his nails with a piece of pumice stone. In his writings Hippocrates gives clear instructions about a physician's hands.

When he has learned all the history of your sickness, what sort of life you like to lead, what you eat, and where you have been, Hippocrates proceeds to the physical examination of your body. This continues the intimate encounter. You may find it has a particularly strong effect on you because while you have been lying there sick your family, friends and acquaintances have rather withdrawn from you psychologically, and physically too, if you are a non-touching lot. (There is a natural reluctance, overcome only by discipline and training, on the part of many people to deal with the sick closely. Because sickness alters; it may make you `strange'. Even when others are apparently concerned for you, this can just be a disguised attitude of fearful aggression or domination on their part.)

Hippocrates now has his hands on your chest, your belly, your neck and shoulders. His touch is warm and confident. For him it is commonplace, routine. For you the feel of his hands is soothing as he palpates for lumps and bumps and painful places. He feels your pulse, he taps and presses your public and your private parts. He looks down your throat, into your eyes. Then he comes very close, putting his bearded cheek right on your naked chest, right alongside your breasts if you are a woman, as he listens firmly with his ear against your ribs, front, back and sides. He even dips a finger in some of your urine and tastes it. By now he has been closer to you than any lover at a first encounter by daylight.

Such intimacy without ravishment is overwhelming and very reassuring whatever the outcome of your sickness. For your regressing, sick, strange, infantile mood of dependency was hungering for touch. Skin touch, lip touch, breast touch, private touch — the touch of a strong, health, life-giving, socially approved person that is nourishing, protecting, experienced, powerful, concerned.

That is how you experience it. The healing touch. It can be a great help where it reinforces the positive influence the mind can have in the natural healing process. And today one should seriously ask, has modern medicine lost it? Has modern medicine lost touch?

The healing touch is like a reassuring physical grooming by another person. Horses standing with necks entwined, cats mutually licking fur, monkeys searching each other for `fleas', are all in states of tranquillity because they are caring or curing (same word) one another by touch. Modern scientific medicine has little of it. Mind you, modern medicine works very well where we use it most actively and logically, with its probes, and

scans, and chemistry. But much sickness does not need scientific investigation and scientific treatment. Much disease is self-regulating, and therefore it often 'responds' to Hippocratic caring. Indeed taking all sickness all round most of it does so, and it is the doctor's business to know what kind is which.

In what we regard as the non-medical field we pay professional groomers and touchers high prices relative to the actual technical skills they are using. The hairdresser and the prostitute, for example, get much more money per hour than artisans who may be using far more exacting techniques, but not on the human body. Those who offer body services traditionally ask for fees, not pay. They speak of their clients, not their employers. A client is a dependent, a paymaster not. All of which suggests that professional bodytouchers really have something to offer that is highly esteemed, something that gives them special commercial status and monetary privileges in the market place.

It is also an interesting but of sociomedical history that the chiropractors — the hands-on manipulative practitioners who surfaced only a century ago with that clever readied-up name — appeared at the very moment when scientific attitudes began to overwhelm orthodox medicine.

Now do not misunderstand me. Scientific medicine works well in its scientific place. But if I were starting out as a young doctor today I would not necessarily decide that I had to be overly scientific in my practice. I would make sure, instead, that there was plenty of body contact in the services I offered the sick. I would make sure I like people. I would make sure I knew how to take the history, the inquiry, by intimately talking; that I knew how to examine the sick body by intimately touching. I would keep in mind the lasting market value of the special caring skills of the unlettered bonesetter, of the nurse, of the hairdresser, of the masseur and the masseuse and of the courtesan and the prostitute. Those are all good doctors: good grooms of the surface.

So that is my after-dinner message. You manipulative doctors are all good touchers. Don't lose it. Don't lose touch. Ever. Make a vow now to keep on touching and to keep in touch with touching, for the next one or two hundred years of medicine.

Those who were in the bus lost night excelled themselves in singing "Alouette". I think you should develop it as an Association anthem with expressive musculoskeletal diagnostic and treatment gestures appropriately applied to different parts of the body as the song continues.

And that's all I have to say. Thank you for quietening down to hear me ... eventually.

BOOK REVIEW

Disorders of the Cervical Spine

Edited by Eurig Jeffreys
Second Edition
Published by Butterworth-Heinemann, Oxford, 1993.

The second and substantially revised edition of Jeffreys's book provides a useful overview of cervical disorders, although one from an essentially surgical standpoint. The contributors are three orthopaedic surgeons and a radiologist, all British and all well-known for their contributions to the scientific literature. In fact, all are from Oswestry, the English Midlands centre noted for its contributions to orthopaedic research and home of the Oswestry Disability Index.

The work sets out to survey the broad field of cervical spinal dysfunction and to place in perspective some recent advances in acknowledged (in his preface the editor describes it as an "orthopaedic accent") but so too are the interests of other practitioners. In fact, deficiencies in the text; one wonders to what extent the understatement is intentional. However, the book succeeds in providing a contemporary synopsis of the subject in sufficient detail for its purpose with some special insights into the particular emphases that surgeons place in certain areas.

The topics addressed include applied anatomy, diagnostic imaging, congenital malformations, fractures and dislocations, orthoses, soft tissue injuries, spondylosis, rheumatic disease, osteomyelitis, tumours and surgical techniques. Each of these is dealt with in a separate chapter containing a summary of the pertinent facts and some more detailed information distilled from recent scientific publications. Necessarily in a book of this length, the presentation is germane rather than comprehensive but what emerges is a distinct outline of each topic and the numerous references act as a guide to the scientific literature for those who wish to read further.

Those with interests in musculoskeletal medicine will notice the textual deficiencies to which the editor refers. They are not so much gaps in the material that is presented as other aspects which are not addressed at all, possible because they are not seen from the surgical viewpoint. An example occurs in the first chapter, on applied anatomy. In a discourse on spinal stability and instability, the gross approach of the orthopaedic surgeon is revealed in the broad categorisation of instability into safe and unsafe forms. This ignores segmental instability of the type detected by I.A.R. studies and no mention is made of the significance of these relatively subtle forms of dysfunction in the large number of patients with chronic cervical pain.

BOOK REVIEW

As in any medical book, there are some statements which seem inaccurate in the light of studies published since the text was prepared. In the chapter on diagnostic imaging, disc degeneration due to the normal ageing process is offered as an explanation for disc space narrowing; not surprisingly, the reference quoted in support of that assertion is dated 1960. This implies a lack of knowledge of more recent publications such as those of Twomey and Taylor (1985) and Amonoo-Kuofi (1991) which lead to a totally opposite view. Perhaps the point was overlooked in the revision of the first edition. There are several other examples to be found, particularly in the chapter on soft tissue injuries.

Other points of difference which will be noted by those in musculoskeletal medicine are related more to emphasis than to fact or awareness. The pain maps of Dwyer, Aprill and Bogduk are discussed in the section on "facet injections" but there is no mention of somatic referred pain or its distribution in the chapter on applied anatomy, which tends to perpetuate the impression that spinal pain is referred principally (if not solely) in dermatomal distributions. These points are relatively minor in the scope of the work but they are important to an understanding of contemporary science in this area.

Taken overall, Jeffreys's book is a valuable addition to the literature on cervical disorders and their management. It provides summaries of various aspects of the subject which are well written and clearly set out. The flavour of the work is erudite, with classical and historical allusions (such as a discussion of Richard III's posture and its possible effect on his kingship) which add considerably to the reader's interest. The surgical "accent" also increases the book's value; the orthopaedic surgeons are, after all, a group with considerable power in the field and the text aids understanding of the surgical approach to common clinical problems.

Wade King

164pp R.R.P. $150

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BOOK REVIEW

The Soft Tissues: Trauma and Sports Injuries

Edited by G.R. McLatchie and C.M.E. Lennox
Published by Butterworth-Heinemann, Oxford, 1993

The soft tissue elements of the musculoskeletal system are more complex in form and function than the hard tissue (bone) but they suffer from a bad press. The term "soft tissue injury" with or without the addition of "only", is often used in a pejorative sense, or as an (unwarranted) attempt at reassurance. It almost seems as if a lack of radio-opacity makes the soft tissues unworthy of consideration by some who deal with "more serious' injures. Yet muscle, tendon, enthesis, ligament, fascia, joint capsule, meniscus and cartilage are more easily damaged than bone and, apart from minor problems which resolve spontaneously, their disorders are much more difficult to assess and treat.

McLatchie and Lennox's new book should help to redress the apparent lack of knowledge in this important area. It sets out the essentials of soft tissue function and dysfunction in a fashion which is both readable and informative. The text is a distillation of the contributions of thirty academics, researchers and clinical consultants in several disciplines from both sides of the Atlantic.

The work is divided into three main parts. The first deals with general principles, including the anatomy, physiology and pathophysiology of the different soft tissues, the nature of the injuries they suffer, the epidemiology of these and the economic effects they cause. The central section, which makes up the main part of the book, outlines the practical aspects of clinical assessment, diagnostic investigation (with chapters on imaging techniques, physiological testing and gait analysis), management (including the uses of various modalities) and rehabilitation. The third part is concerned with sports injuries. It addresses the particular risks and injuries associated with sports of different types, with separate chapters on track and field athletics, cross-country running, football, water sports, gymnastics and dance, martial arts and combat sports, and equestrian sports. The text is annotated throughout and each chapter includes extensive lists of references.

The book brings together in one volume a great deal of useful scientific data and sound practical information. Its value will be appreciated by those involved in musculoskeletal medicine, sports medicine, orthopaedic surgery and allied disciplines. It will also be of interest to others who undertake injury assessment and should be required reading for those whose knowledge of soft tissue injuries is apparently limited to bruising.

Wade King

485pp. RRP $145

BOOK REVIEW

Cyriax's Illustrated Manual of Orthopaedic Medicine

by J.H. Cyriax and P.J. Cyriax
Second Edition
Published by Butterworth-Heinemann, Oxford, 1993

268pp. RRP $99

and

Apley's System of Orthopaedics and Fractures

by A. Graham Apley and Louis Solomon
Seventh Edition
Published by Butterworth-Heinemann, Oxford, 1993

736pp. RRP $135

New editions of these two standard works have been published recently. The books concerned are such landmarks in the field that it would seem impertinent to offer opinions on them. Suffice it to say that they belong on the shelves of all practitioners of musculoskeletal medicine. Both are valuable as systematic guides to the assessment and management of musculoskeletal problems (from two distinctly different points of view) and for reference to see what the masters have to say about particular things. Those who do not have them or who have earlier versions, which in both cases could be up to thirty years old, should consider obtaining copies of these new and revised editions.

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BOOK REVIEW

Outcome Measures in Orthopaedics

Edited by Paul Pynsent, Jeremy Fairbank and Andrew Carr
Published by Butterworth-Heinemann, Oxford, 1993

This new book is the first edition of a work which seems destined to become something of a classic. It is very much a book for its time. Measurement and its applications have long been understood, and their value appreciated, by those working in research areas. Concepts of measurement, and in particular measurement of outcome, are less familiar to most clinical practitioners. However, this situation is changing and the trend is likely to accelerate as time goes on.

Modern methods of practice are so dependent on scientific validation that it is dimply not possible to apply them effectively without some appreciation of the measurements on which they are based.

As the ranges of diagnostic and therapeutic options continue to expand, management decisions will depend increasingly on the clinician's understanding of the measures used to assess the advantages and disadvantages of the various interventions available.

Evaluation of outcome has always been desirable in medical practice. Factors are emerging which will make it mandatory. The more obvious of these are the increasing demand for services and the resultant financial constraints imposed by funding authorities, and the explosion of litigation involving doctors either directly or indirectly. Audit, substantiation and accreditation have become accepted parts of practice in recent years and their roles are set to increase in the future.

Pynsent, Fairbank and Carr have produced a guide to outcome measurement that will enable unversed clinicians both to understand the principles involved and to benefit from their practical application. As editors they co-ordinated the work of a large team made up mainly of resident medical officers and younger practitioners with interests in research. The text was written largely by those junior doctors and then refined by senior practitioners recognised for their expertise in each of the relevant fields. In this way the editors have managed to achieve a balance between approaches based on different levels of experience, making the book useful for those with limited knowledge of the subject matter and for those with a solid background in research.

The early chapters are devoted to the principles of outcome measures and their analysis, the measurement of pain, trauma scores and their validation, patient satisfaction and quality of life measures, general outcome measures and complications. These provide outlines of the essential elements of measurement, discussions of various aspects of outcome and surveys of methods used in their evaluation. The various points are illustrated with detailed examples of appropriate instruments of measurement and the editors provide recommendations of what they describe as the "best buys" selected from the wide range of measures available.

BOOK REVIEW

The chapter on pain measurement will be of particular interest to those in musculoskeletal medicine. It discusses the used to measure either pain intensity or pain relief. The "best buys" recommended are the McGill Pain Questionnaire for the evaluation of the state of pain and the Oxford Pain Chart, which includes categorical scales for both intensity and relief, for the analysis of clinical responses to treatment..

Following on from the general section are separate chapters on each of the main musculoskeletal regions, the spine, the shoulder and elbow, the hand, the hip, the knee and the foot. Each of these addresses specific issues related to those body parts, including accurate assessment of impairments, associated disabilities and handicaps, together with evaluations of treatments that might be applied and their effects.

The future of medical practice of all types seems inexorably linked to measurement of outcomes. Claims of diagnostic accuracy and therapeutic efficacy can no longer be sustained without credible evidence based on measurement of the variables involved. The development of therapeutic interventions has long been determined by the measurement of outcomes published in journals. Increasingly, treatment plans are being formulated by individual practitioners on the basis of their perception of likely outcomes and the measures used to evaluate them. Moves towards accreditation are making that style of management a normal expectation. This book will enable clinicians to understand and use outcome measurement as a means of ensuring the highest quality of patient care.

Wade King

273pp. RRP $92

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