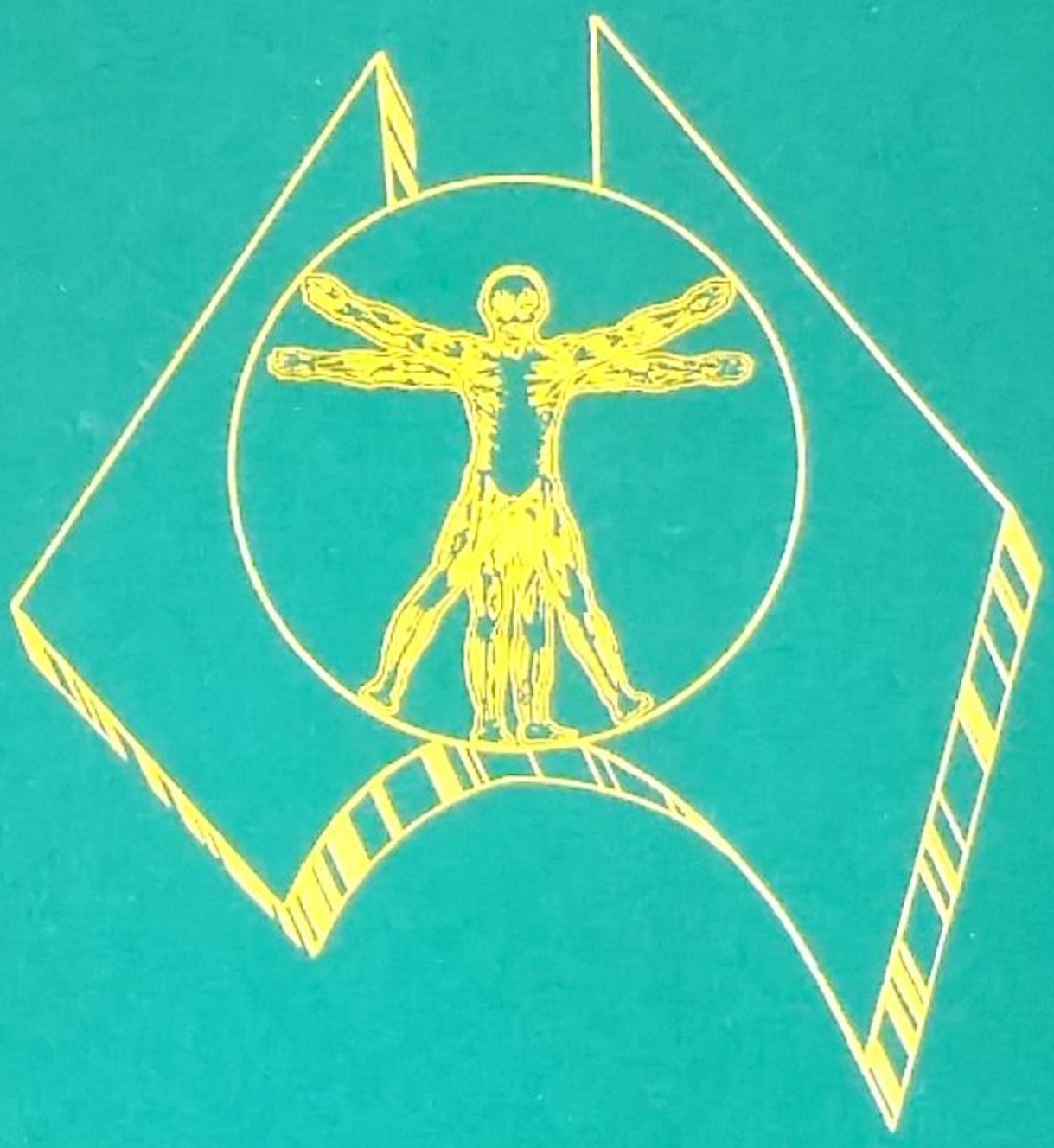
Australian Association of Musculoskeletal Medicine

Bulletin



Zygapophysial Joint Pain

Vol. 6 No. 2 July 1990

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Australian Association of Musculoskeletal Medicine *Bulletin*

Vol.6 No.2 July 1990

CONTENTS

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Published by Belaser Type Services, PO Box 1083, Tamworth, NSW, 2340, telephone (067) 66 6399, fax (067) 66 5440.

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AUSTRALIAN ASSOCIATION OF MUSCULOSKELETAL MEDICINE
OFFICE-BEARERS 1990

The following members were elected to office at the annual general meeting in Sydney on 3rd February, 1990.

PRESIDENT:

Dr. David Vivian MB, BS

441 Bay Street, Brighton, Vic., 3186
telephone (03) 596 7211

HON. SECRETARY:

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7 Brighton Road, Glenelg, S.A. 5045
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82 High Street, Taree, N.S.W., 2430
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Assoc. Prof. Nik Bogduk	Newcastle, N.S.W.	(049) 68 5718
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	Dr. Ian Roddick	(003) 44 7177
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	Dr. Max de Clifford	(03) 873 1764
WA:	**Dr. Arnold Jones**	(09) 390 4444
	Dr. Marius Loeffler	(09) 272 3570

Editorial

The two major articles in this issue of the Bulletin address the topic of zygapophysial joint dysfunction and myofascial pain syndromes. These two entities, occurring singly, together or in various combinations with the third member of a triad, intervertebral disc disorder, account for a large proportion of the chronic pain suffered by members of the community

Chronic pain is one of the major problems of our society, every bit as debilitating as unemployment, economic difficulties and declining standards of living. There are vast numbers of sufferers, one in four or five of the whole population on reliable estimates, doomed to a miserable existence with constant torment from which there seems to be no escape.

Quite apart from the physical morbidity involved, the problem is associated with enormous costs to the community.

In human terms, these costs include all the psycho-social consequences of prolonged suffering and disability. The person with chronic pain has difficulty in participating in his former activites, in expressing creativity and in enjoying recreation. relationships suffer and the quality of life diminishes for his family members as well as for himself as he withdraws progressively into a private existence dominated by the avoidance of exacerbations and the search for relief. As he does so, the community is inevitably the poorer for the loss of his involvement.

The financial costs are also great. Frequently the sufferer loses his ability to generate his own income and must depend instead on financial assistance drawn from the community's aggregated resources. The cost of providing this assistance is compounded by the loss of the sufferertribution to communal productivity. Further costs are involved in the provision of continuing palliative medical treatment and the expensive investigative procedures which tend to be undertaken repeatedly for as long as the prm remains undiagnosed. For example, the annual cost to the Australian health budget of unnecessary C.T. scans of the brains of patients with cervical headaches would be enormous. There must be many ways this money could be better used elsewhere in the health system or in education, housing or some other area of national need.

The answer to the chronic pain problem lies not so much in the provision of increasingly sophisticated treatment modalities as in research and education to improve methods of diagnosis and early interverntion. Chronic pain patients would simply not exist if the problems were diagnosed accurately and treated definitively in thie early stages.

In the case of myofascial pain syndromes, the solution is at our fingertips (literally). Careful palpation will usually reveal the nature and location of myofascial dysfunction and the application of specific treatment is a relatively simple matter once the diagnosis has been established. All that is required is rejection of the unscientific notion that soft tissue injuries are inherently trivial and self-limiting, and its replacement by recognition of the well-documented clinical manifestations of these extremely common conditions.

Zygapophysial joint problems are not so easily diagnosed or treated clinically and their pain often persists or recurs after conservative management. However, a great deal more can be done than to offer the patient the choice between long-term analgesic medication and unwarrantable spinal surgery. The use of diagnostic block injections and radiofrequency denervation holds the promise of safe, definitive treatment.

Not all chronic pain of spinal origin will be overcome by these strategies but they do represent fresh approaches to problems that are not adequately addressed by other methods. The current wastage of resources in this area is something that our country cannot afford and the potential savings, both human and financial, are too great to be ignored.

A Word from the President

The 1990 Conference to be held in Melbourne will have the theme that was enunciated at the Sydney meeting earlier this year.

The first day will concern the pathology of lower back disorders. The first session will be on the origin of pain. This subject was addressed at the recent Adelaide Pain Conference, where the physiology and pathology of pain itself was expanded upon. The importance of the dorsal horn and the pathways ascending from it was emphasised. This discussion on the origin of pain will not only centre on pathology of structures in the back itself, but also consider the pathological processes which contribute to make pain chronic within the spinal cord. After this, the biomechanics of the spine will be considered and from this will follow discussion of pathology related to biomechanical abnormalities, with emphasis on the concept of lumbar instability. I would suggest that registrants who are not sure of the biomechanics of the spine, do some prior reading and this subject is well covered in the book by Bogduk and Twomey titled "Clinical Anatomy of the Lumbar Spine". Another biomechanical abnormality to be discussed is spondylolysis and there will be discussion of scientific evidence collected from around the world in regard to whether or not this condition causes pain. The radiology of the spine, including M.R.I., discogram and C.T. scan correlations will all be discussed and current scientific literature will be reviewed. Registrants should emerge from this session with a better idea of when X-rays should be ordered and have an understanding of the relevance of these investigations, including their sensitivity and specificity. The treatment of spinal conditions will be considered, including injection methods such as trigger point injections, paravertebral steroid injections, facet joint injections, the use of phenol to treat chronic ligamentous pain and dorsal ramus pain, radiofrequency denervation and rhizotomy. There will also be discussion of the surgical management of lower back disorders including the use of chymopapain, percutaneous discectomy, open discectomy, laminectomy and fusion. The last session of the day will consider the biomechanical consequences of spinal surgery, including techniques that cause loss of disc substance and loss of bony structure.

The second day will begin with considertion of neurogenic problems associated with lower back disorders, including sympathetic mediated pain and how it presents in regard to back injuries; its diagnosis with thermography and other techniques will be discussed, as will possible treatment. The use of a spinal cord stimulator to control chronic spinal pain will then be discussed. This is a technique whereby a stimulating device, like a TENS machine, is placed onto the dorsal columns, to control chronic pain derived from the spine or of sympathetic origin. The next session will be on mechanical aspects. This is the most relevant section for those treating the acute pain of musculoskeletal origin in the community. it will include consideration of the techniques that are of greatest use in general practice and a talk on the teaching techniques that are used in France. The discussion on postural influences will include such matters as whether leg length inequalities are important in the genesis of back pain and what can be done about them. A great deal of scientific work has been done in this regard. Exercises are used frequently in the management of back disorders and sometimes for prevention. Stretching exercises, mainly the Williams and McKenzie techniques, are commonly used and these will not be discussed. Instead, attention will be directed to the concepts of estabilishing and strengthening exercises and some examples will be given.

On the last day, the psycho-social elements of back pain will be considered with emphasis on the concept of disability in excess of injury. A review of literature regarding the success or otherwise of pain clinics and rehabilitation programmes will be presented, including studies from America, New Zealand and Australia. A presentation of some of the studies from Australia will follow; in Victoria there are a considerable number of programmes that have been up and running for some years. The various compensation systems of the different Australian states will be considered and compared to that of New Zealand, which will be explained in some detail. The psychological approach to pain management will be presented, summarising the aspects of psychological care relevant to chronic lumbar disorders.

Other less formal sessions will follow. One will be a "two slide two minute" segment on spinal disorders. In addition, a "hypothetical" will be held on the medico-legal system. A number of cases will be presented and the panel will be made up of doctors and lawyers frequently involved in medico-legal matters. Hopefully, this should be both entertaining and informative. Ross Stephenson, a lawyer who conducts an early morning show in Melbourne called Lawyers, Guns and Money, will be the presenter.

The day following the Conference will see a 'Vintage Education Programme', with four lectures by Nik Bogduk, on aspects of anatomy, neurophysiology and pathology relevant to musculoskeletal disorders. This will be a great update session for anyone involved in any aspect of musculoskeletal medicine. It will cover basic knowledge and the latest information available.

Two breakfast sessions will be held, one on Repetitive Strain Injury, in which scientific evidence relevant to this condition will be presented, and the second (a must for any practitioner who uses injection techniques) on the resuscitation of patients in the event of any mishap associated with local anaesthetic administration. The presenter of the repetitive strain session will also be presenting information about back testing machines which are being used more frequently in the diagnosis and management of back conditions, particularly in the chronic pain setting.

For accompanying guests, a cultural programme will be offered and information about this will be posted to registrants. Social functions will include a cocktail party on the first night and a dinner at the end of the last day, so that all the speakers can enjoy the evening without worrying about having to speak next day. Pre- and post-conference courses on different areas of the musculoskeletal system will include courses suitable for beginners as well as those for more experienced practitioners.

I think this comprises the most comprehensive programme that has been offered on back disorders in Australia and I hope that the membership participates in making it a memorable event. I wish to remind you all that there are only about three hundred and fifty seats in the Regent Lecture Theatre and this is all we can take. We already have twenty five registrants (early July, 1990) and this means that the three hundred and fifty should be filled. Please send your registration in early.

From the Hon. Secretary's Desk

Hi. It is very pleasing to report that musculskeletal medicine seems to be thriving both in this country and overseas. I am prompted to say this for several reason:

- *The South Australian plans for a post-graduate diploma in musculoskeletal medicine have advanced a few more steps and the course is likely to be initiated at the Flinders University in 1991. An outline of the proposal is included in this Bulletin. In New South Wales progress is being made for a post-graduate diploma course at the University of Newcastle and as soon as more information is available it will be circulated also.*
- *The international meetings which I attended in Adelaide and Vancouver in recent months emphasised the large number of people actively engaged in research and certainly very interested in the problems which face those involved in treating patients with musculoskeletal pain and dysfunction.*
- *The cost to the numerous compensation agencies throughout the world in treating and rehabilitating patients suffering from musculoskeletal dysfunction is assuming astronomical proportions. Many of these agencies are anxious to encourage research and education which will promote early and precise diagnosis with subsequent savings in treatment costs.*

On the other hand, it is disappointing to note the large degree of fragmentation which some people practising in areas akin to musculoskeletal medicine are creating. Throughout the world there are numerous organisations using various names, such as physical medicine, orthopaedic medicine, physiatry, etc, all concerned with treating musculoskeletal pain. It seems to be a pity that these groups are not united under one banner. Specialties such as orthopaedic surgery, neurology, gastroenterology, etc., are very much united yet our discipline continues to appear divided. This is perhaps because of our relative infancy but it would seem appropriate that efforts be made to correct the impression of disunity and to prevent additonal groups from setting up in ignorance of the activities of bodies such as the AAMM.

While in Canada I talked to many people and presented our syllabus and clinical assessment protocol. I also gave an outline of where musculoskeletal medicine was heading in Australia and New Zealand, with our post-graduate courses etc. It seems that we are streets ahead of organisations in the northern hemisphere in some respects. Many were quite envious that we have become so well organised and have a singular determination which seems to be lacking in the countries of various people to whom I spoke.

One depressing feature of the North American scene was the degree of intervention by the numerous compensation agencies into the treatment of people with musculoskeletal injuries. There is a great variation between agencies; some leave all decisions to the medical profession in the hope that something sensible will result, whereas in some states and provinces agencies are very definite about what will be compensable and what will not be. For example in one province of Canada compensation will be provided for a person who has a diagnosis of lateral epichondylitis which is recognised as a work injury. However, if perchance the patient has tennis elbow and applies for compensation it is considered to be a leisure time injury and not compensable. This is obviously the result of gross ignorance, yet it happens. Other private agencies are telling patients that they will not be compensated for hospital stays or operations that have been performed by orthopaedic surgeons unless these people have first been seen by musculoskeletal medicine specialists and have exhausted all non-surgical avenues of treatment for restoration of function.

As mentioned previously there are many people who are busily engaged in research of various kinds and undoubtedly of various standards. One thing which did impress me was the fact that there are new and different ways of looking at old problems. The paper on myofascial pain presented by Dr. Chan Gunn in Vancouver is one good example of this. There must be numerous people around for whom his treatment would be beneficial. Dr. Gunn related several interesting anecdotes about dry needling, for example, the disappearance of the symptoms of fibroids after dry needling of the S1, S2 areas bilaterally. This may sound fanciful but we must kkeep our minds open. It is only by mingling and talking with people from other countries that such ideas can be openly discussed, with all the potential for improvments in patient management.

Once again I must encourage all members of the Association to take advantage of its activities, especially courses and conferences. As a closing note, it is encouraging to have numerous applications for the Licentiate. All members are encouraged to apply for it and to participate in the associated educational programmes.

AAMM NEWS

Australian Association of Musculoskeletal Medicine

Preparations are well in hand for the Association's twentieth Annual Scientific Meeting, to be held in Melbourne from 29th November to 1st December, 1990. The conference will be the most ambitious ever staged by the Association, with a full programme of scientific papers spread over three days and including a great deal of original work. The meeting's theme will be Lower Back Disorders and subjects to be addressed include pathological, neurogenic and mechanical aspects of lumbar spinal pain and dysfunction, as well as related psycho-social and medico-legal issues. A variety of pre- and post-conference courses will be offered and several interesting satellite meetings are also to be held. Further details will be found in the Meetings, Conferences and Courses section of this Bulletin.

❖

The bitterly cold weather being experienced in most parts of Australia this winter is putting smiles on the faces of those intrepid members of the Association who are waxing their skis in preparation for the annual expedition to Mt. Buller known as the Winter Meeting. More snow has fallen in the high country than for many years past and the skiing season is proving to be one of the best ever. Non-skiing members who are huddling around their radiators in the last week of July might care to spare a thought for those who will be enduring the rigors of crisp mountain air, deep powder snow and copious amounts of gluwein in the interests of forging more strongly the personal links which hold the Association together.

❖

Missing from the Winter Meeting party this year will be stalwart Queensland member, **Bob Michael** and his wife **Sue**. Bob is recovering from a recent bout of illness which interrupted his busy programme and made it difficult for him to take further time off. Fellow Brisbane committee member **Ron Palmer** and his wife **Dianne** have unselfishly volunteered to take the Michael's places in the Buller assault party.

❖

The Association's accreditation programme is gathering momentum and many applications have already been made for the Licentiate. Several members have already qualified in the first part of the accreditation scheme and the writing of theses is in progress. All members are encouraged to become involved. Details of the accreditation criteria are to be found in the March, 1990, issue of the Bulletin and application forms are available from the Hon. Secretary upon request.

❖

Subscriptions for the year 1990-1991 became due on 1st July, 1990. All members are invited to send the prescribed amount of $60 to the Hon. Treasurer at their earliest convenience.

❖

International visitors in Australia at the moment include Di Clofenac from Switzerland. Di is well known for her involvement with various forms of musculoskeletal dysfunction and particularly for her contributions to prostaglandin inhibition. She is presently working in Australia and details of her activities may be found on the centre pages of this Bulletin.

A photograph taken at the foundation meeting of the Association in Melbourne in 1971 has recently been forwarded to the executive committee by **Geoff Toakley**, a foundation member and Queensland neurosurgeon. **Geoff** was kind enough to forward the original for the Association's archives and his thoughtfulness will be appreciated by all members. The photograph will be reproduced in a future issue of the Bulletin, together with some record of the Association's history.

❖

*Former president **Nik Bogduk** is presently on sabbatical leave from the University of Newcastle and is attending even more national and international conferences than usual. He is also managing to spend more time in his university office, working on the multitude of projects in which he is involved, including many on behalf of the Association. It is not known whether special leave is available for those who suffer overwork stress through being on leave.*

❖

Former committee member **Carl Rotkirch** and his wife **Biddy** were recently back in Australia for a few weeks, on leave from the Middle East. Carl is presently working in the United Arab Emirates and they have also been spending some time in Cyprus. Many will be glad to hear that they plan to return here to live at the end of the present contract.

❖

Members are reminded that they can obtain membership lists, reprints of Bulletin articles and some previous issues of the Bulletin from the publishers, Belaser Type Services, P.O. Box 1083, Tamworth, N.S.W., 2340.

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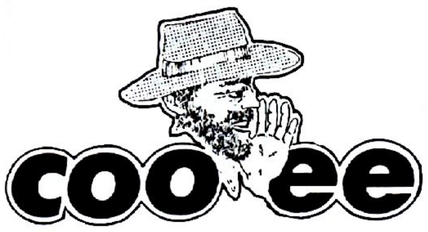

A cheerio call to someone selected at random from the membership list.

The call goes to New South Wales this time, to the leafy suburb of Longueville on the northern side of Sydney Harbour, where **Barry Abeshouse** can be found struggling bravely against the interminable problems of his patients' pains and disabilities. Like all practitioners of musculoskeletal medicine, Barry is kept extremely busy by his work but he he still finds time to play an active part in the affairs of the Association, particularly the meetings held regularly in Sydney. He has been involved in the Association's modular course on patient assessment and is currently organising a weekend workshop where the shoulder module will be presented. He has also been very active behind the scenes, helping with the planning for the Association's accreditation programme and all that goes with it. It is something of a mystery how he manages to achieve all he does and still maintain the flourishing romance he has been conducting with his wife Leonora ever since they met at medical school. Perhaps there's more than we always thought to the old adage about bears liking honey!

Meetings, Conferences and Courses

Local AAMM Meetings

In **Sydney** meetings are held at 7.30pm on the third Monday of each month in the Department of Rehabilitation Medicine, Royal Prince Alfred Hospital. The programme usually consists of a lecture or discussion on a selected topic, followed by case presentations and a practical session of diagnostic and management techniques. The meetings are open to all interested medical practitioners. Those wishing to attend are asked to telephone (02) 550 3837 during the preceding three working days to confirm the arrangement.

In **Melbourne** monthly clinical meetings are held at the Metropolitan Spinal Clinic, 302 Malvern Road, Prahran, usually on the first Tuesday in each month, commencing at 7.30p.m. and running for approximately two hours. These meetings include patient presentations and clinical lectures. Those interested in attending are requested to contact the clinic beforehand on (03) 529 1988.

Regular meetings, practical sessions and courses are conducted in many other centres around Australia by state branches, local groups and individual members of the Association. These activities are mainly for the benefit of members living in a particular area and they will generally be advised by letter or by local notices of dates, times and venues. Anyone who is not receiving information about local activities, or who would like more details about what is going on, should contact one of the local organisers listed below.

In **Adelaide**, Dr. Norm Broadhurst, telephone (08) 295 1890.
In **Brisbane**, Dr. Bob Michael on (07) 345 8999.
In **Canberra**, Dr. Goff Nelson on (062) 95 6773.
In **Hobart**, Dr. Ron Heddle on (002) 34 5990.
In **Newcastle**, Dr. Nik Bogduk on (049) 68 5699.
In **Perth**, Dr. Marius Loeffler on (097) 33 5220.
In **Taree**, Dr. Wade King on (065) 51 0662.
In **Toowoomba**, Dr. Jeff Phillips on (076) 38 4800.
In **Townsville**, Dr. Roger Watson on (077) 71 3084.

Those who live in other areas and who would like to organise or participate in local meetings should contact one of their state representatives, who can arrange publicity and other assistance from the resources of the Association.

AAMM Winter Meeting

The Winter Meeting will be held at the Southern Cross Lodge, **Mount Buller**, again this year, from 29th July to 3rd August, 1990. The meeting is a purely social function, designed to bring members together in a relaxed setting as something of a counter to the increasingly high-powered scientific meetings and courses that are on at other times of the year. The only agenda at Buller is fellowship, fun and skiing.

Members are invited (in fact encouraged) to bring their families, friends, et al to enjoy a week at one of the best lodges in the snowfields for the paltry sum of $250 per head. Bookings will be taken on the basis of first in, first served and as there is only room in the lodge for 40 people members are advised to make their reservations soon. They can do so by sending a deposit of $100 (payable to the Association) to Dr. Wade King, Hon. Treasurer A.A.M.M., 82 High Street, Taree, N.S.W., 2430. Further information will be supplied on request.

Annual Scientific Meeting of the AAMM

Theme: ***"Lower Back Disorders"***

The Association's Twentieth Annual Conference will be held at the Regent Hotel, **Melbourne** on 29th and 30th November and 1st December, 1990. Pre- and post-conference meetings and courses will be held for those interested in taking part. The full programme for the conference will be as follows:

Thursday, 29th November

9.00am	Welcoming address	
	Pathological aspects	
9.10am	Where is the pain coming from?	*Prof N. Bogduk*
9.40am	Instability	*Mr G. Schneider*
10.05am	Biomechanical pathology	*Prof L. Twomey*
11.00am	Spondylolysis - does it hurt	*Prof N. Bogduk*
11.30am	Radiology of disc degeneration	*Dr G. Buirski*
1.30pm	Surgical intervention for back disorders	*Mr G. Speck*
2.10pm	Needle intervention for back disorders	*Dr D. Vivian*
2.30pm	Biomechanical consequences of surgery	*Prof N. Bogduk*
3.00pm	Panel Discussion - Questions/Answers	
4.15pm	AGM of A.A.M.M.	

Friday, 30th November

	Neurogenic aspects	
9.00am	Sympathetic mediated pain	*Mr S. Rosengarten*
9.25am	Thermographic assessment	*Dr D. Cullum*
9.40am	Neuromodulation	*Mr U. Rossi*
	Mechanical aspects	
10.30am	Back pain in general practice	*Prof J. Murtagh*
10.50am	Examination systems for the back	*Dr C. Kenna*
11.10am	Pelvic instability	*Dr N. Broadhurst*
11.25am	Clinical biomechanics of the S-I joint	*Dr W. King*
11.50am	Buttock pain	*Dr N. Broadhurst*
1.30pm	Postural influences on back pain	*Prof H. Burry*
1.50pm	The lifting model - is it precise?	*Prof N. Bogduk*
2.20pm	Backtesting machines - validation, limitations and role	*Dr P. Henke*
3.30pm	2 minutes - 2 slides *	

Saturday, 1st December

	Psycho-social, including medico-legal, aspects	
9.00am	Rehabilitation in chronic back pain - treatment models	*Dr P. Lowthian*
10.00am	A Back Rehabilitation Programme	*Dr. L. Twomey*
11.00am	Imaging algorithm for back pain	*Dr G. Buirski*
11.30am	Medico-legal aspects of spinal practice	*Prof H. Burry*
12.00pm	Does trauma precipitate spondyloarthropathy	*Dr S. Hall*
2.00pm	Lawyer's view: Medico-legal systems	
2.30pm	Hypothetical on medico-legal practice	*Mr R. Stevenson*

* ***One paragraph submissions are required for the "2 minutes - 2 slides" presentation. These should be sent to: Dr David Vivian, 441 Bay Street, Brighton, 3186, no later than 30th September, 1990.***

Conference Secretariat: MCS Conventions, P.O. Box 335, Heidelberg Vic 3084, telephone: (03) 499 6722 facsimile: (03) 499 7137

Associated Meetings

Resuscitation Techniques Applicable to Musculoskeletal Practice - an update
Wednesday, 28th November, 1990
Cost - $75 *Dr A. Ross*

Scientific Basis for "RSI"
Friday, 30th November, 1990
Presentation of the current research findings on the bio-chemistry, pathology, and neuro-physiological basis.
Cost - $40 *Dr P. Henke*

Peripheral and Central Neuroanatomy pertaining to the Musculoskeletal System
Sunday, 2nd December, 1990 - all day. This is a series of four lectures entitled:
- Design of the glenohumeral joint - biomechanics in function; why is the rotator cuff?
- How to remember all the nerves in the body; dermatomes and myotomes of all the peripheral nerves.
- Modern views of the motor system .
- Neuro-anatomy of the spine for musculoskeletal practitioners.

Cost - $150 *Prof N. Bogduk*

Pre - and Post Conference Courses

These courses will run from Sunday, 25th November to Tuesday, 27th November and from Monday, 3rd December to Wednesday, 5th December.

Lumbar Spine I
3 days
Introduction to basic anatomy, physiology, biomechanics and assessment of the lower back. Some aspects of management included. Includes the pelvis.

Lumbar Spine II
3 days
For practitioners who have done basic courses. This will concentrate on greater detail of basic sciences and examination and then deal with all aspects of management as set out in the syllabus. Patient demonstrations. Session of injection techniques.

Cervical Spine I
2 days
Introduction to basic sciences of the cervical spine. Examination and assessment. Some management. Includes shoulder girdle.

Lower Limb II
2 days
For practitioners who have done a basic course on the lower limb. Includes review of pathology, examination and more detailed consideration of management.

The cost, exclusive of meals, is $140 per day.

Cocktail Party
6.00pm Wednesday, 28th November, 1990 - no charge.

Examination and Treatment Upper Limb, Shoulder Girdle and Neck

presented by Australian Association of Musculoskeletal Medicine and The Department of Rehabilitation Medicine

Saturday and Sunday 21st and 22nd July 9 - 5pm each day with lunch provided
Venue Tutorial Room, The Queen Elizabeth Hospital, **Woodville,** South Australia.
The workshop will be a "hands-on" practical exposure for those wishing to gain greater skill in the examination, diagnosis and treatment of musculoskeletal pain arising from pathology of the shoulder, upper limb and neck. Various treatment modalities will be discussed and demonstrated. The protocol for "Clinical Patient Assessment" as outlined in the AAMM Bulletin, September, 1989 will be used as the basis of the workshop.

Further information from Dr Norm Broadhurst, 7 Brighton Road, Glenelg, S.A., 5045, telephone (08) 2951890.

Practical Courses on Spine and Peripheral Joints

conducted by the New Zealand Association of Musculoskeletal Medicine

In 1990 New Zealand Association will conduct four courses open to all registered Medical Practitioners.
Course 1 - Examination of the Vertebral Column (Spine 1). **Auckland**, 17th - 21st April inclusive.
Course 2 - Peripheral Joints - Examination and Mobilisation. **Christchurch,** 30th April - 4th May inclusive.
Course 3 - Mobilisation and Neuromuscular therapy of the Vertebral Column (Spine 2). Attendance at Course 1 is a prerequisite for Course 3. **Christchurch,**30th July - 3rd August inclusive.
Course 4 - Advanced Mobilisation of the Vertebral Column including Impulse Techniques (Spine 3). Course 3 is a prerequisite for this course. **Auckland,** August 6th - 10th inclusive.

All courses include manuals and teaching materials, full catering and a two-day weekend Revision Course.
Any enquiries should be directed to Mrs. M. Johnson, 126 Wade River Road, Whangaparaoa, New Zealand.

Perspectives in Musculoskeletal Pain in Medical Practice

A one day conference will be held at The Queen Elizabeth Hospital, **Woodville**, South Australia on Saturday 18th August, 1990.
Various topics will be discussed including: Acute Pain, Pathways, Effect and Treatment; Chronic Pain, A Psychiatric Dilemma; Osteoporosis, Relevance and Treatment; Treatment Modalities, Which Ones Work and Why?; When Should A Surgeon Be Involved?; Why Does Some Surgery Fail? Panel to include: Prof. Nik Bogduk, Mr. Graham Mercer, Dr. Roy Lee, Prof. Issy Pilowsky, Dr. Pat Phillips, Ms. Ruth Pike.

Further information from Dr Norm Broadhurst, 7 Brighton Road, Glenelg, S.A., 5045, telephone (08) 2951890.

Back Pain and Spinal Manipulation

RACGP course will be conducted at the lecture theatre, Monash Medical Centre, Clayton Road, **Clayton**, Victoria, 24 to 26th August, 1990.
The course includes: three day workshop, course textbook/manual, kit of notes, assessment programme (on theory - through correspondence) lunches and refreshments.

Telephone enquiries: (03) 598 4095 or (03) 890 1314.

First World Congress of Biomechanics

California, USA.
August 30th to 4th September, 1990
Dr Geert W. Schmid-Schonbein, AMES-Bioengineering R-012, University of California, San Diego, La Jolla, CA 92093, USA.

23rd International Congress on Occupational Health of the International Commission of Occupational Health

Montreal, Canada
22nd - 28th September, 1990.

Additional information available from: The Secretariat, 23rd International Congress on Occupational Health, 68 de Brésoles Street, Suite 2, Montreal, Quebec, Canada H2Y 1V5, telephone (514) 499-9835, telex 05-24245, facsimile (514) 288-4627.

Back Pain and Its Management

Friday and Saturday 26th and 27th October, 1990
Directed by Professor H Burry, Department of Medicine, Essendon & District Memorial Hospital, **Essendon,** Victoria.

Topics will include: Pathology, age changes, mechanics of injury, ergonomics, compensation and psychological factors, role of physical, psychological and surgical intervention. This course aims to provide an update on current methods of investigation and management including anatomy, pathology, biomechanics and clinical features, by means of lectures, demonstrations, a quiz and a hypothetical.

Further information may be obtained from Continuing Medical Eduction, School of Medicine, The University of Melbourne, Parkville, 3052, telephone (03) 344 5888.

Pain Programs - A Pracitcal Approach

Twelfth Annual Scientific Meeting of the Australian Pain Society

19th-22nd February, 1991. Wrest Point Federal Hotel, **Hobart**, Tasmania.
For further information and eqnuiries contact: Dianna Crebbin Conferences, PO Box 629, Willoughby, NSW, telephone (02) 417 8525, facsimile (02) 417 8513.

Acupuncture In The Age Of Technology

The Fourth Australian International Congress of Medical Acupuncture

Hotel Conrad, **Gold Coast**, Queensland.
2nd April to 6th April, 1991.

Plenary speakers include: Professor Ronald Melzack, Professor Sven Anderson, Professor Jisheng Han, Professor Pekka Pointinen, Professor Johannes Bischko. The question is: Acupuncture has it arrived in the age of technology? Be there, find out, don't miss out.

Further information is available from: The Convenor, PO Box Box 6045, Gold Coast Mail Centre, Queensland, 4217, telephone 075-911166, international 61-75-911166; facsimile 075-911086 international 61-75-911086.

Management of Zygapophysial Joint Pain by Radiofrequency Denervation

David Vivian

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ABSTRACT

The lumbar zygapophysial joints are responsible for between 5 and 20% of pain from chronic back disorders. Pathology resulting from trauma has been demonstrated in histological studies. A study of the anatomy of the zygapophysial joint and its nerve supply provides possible avenues for controlling this pain.

This paper reviews some previously reported results of radiofrequency denervation (RFD). The techniques of diagnostic joint block and radiofrequency denervation are described and discussed. A proposal for a controlled study is included.

INTRODUCTION

The lumbar zygapophysial joints have been recognized as potential sources of back pain since early this century[1, 2], the term "zygapophysial joint syndrome" was developed in 1933[3]. However, a definitive description of symptoms and signs attributable to zygapophysial joint injury has remained elusive.

Experimental studies of these joints have shown them to be sources of back pain and referred pain into the leg as far as the foot[4, 5, 6].

Although many studies have shown that low back and leg pain can be relieved by local anaesthetic infiltration into the zygapophysial joints[5, 7, 8, 9, 10, 11, 12, 13, 14] it is apparent that these joints are only occasionally the major cause of this pain[15].

Hirsch showed that low back pain could be produced by injecting hypertonic saline into zygapophysial joints[4]. Mooney demonstrated that low back pain and more peripheral pain could be induced by provocative injection and noted that the referred spread was proportional to the intensity of the stimulus. He used joint block injections therapeutically and achieved a 62% rate of relief of chronic pain initially, with 20% at six months[5]. Ogsbury produced only 46% initial relief and 6% long term relief[16]. Selby and Lippitt also published results showing that about 50% of patients received a long term benefit[12]. Moran et al. studied one hundred and forty three zygapophysial joints in fifty four patients with low volume local anaesthetic (1.5ml bupivicaine) and only 16.7% had a zygapophysial joint diagnosis confirmed; they considered that higher results in earlier studies may have arisen because of extravasation of anaesthetic from the zygapophysial joint into other structures, such as the spinal canal[17]. Jackson et al. used the same volume and achieved initial pain relief of 25%, and concluded that "the zygapophysial joints were not commonly the single or primary source for low back pain in the great majority (90%) of patients studied"[15].

The lumbar zygapophysial joints are commonly affected by osteoarthritic or degenerative changes[18, 19, 20, 21, 22]. Generally these changes occur in association with disc degenerative changes but they do occur in isolation in about 20% of cases[19]. The X-ray changes are however independent of back pain, occurring with similar frequency in painless and painful backs[21].

The zygapophysial joints are well visualised on C.T. scans, with changes such as tropism and sclerosis being common[11]. Once again these changes have not been able to be related to back or leg pain.

Low back and leg pain are common afflictions. More than 10% of all adults have had back ache lasting for more than three days in the previous year[23]. The Nuprin Pain Report revealed that on one hundred and one or more days during 1985, 5% of the population suffered head ache, 9% backache, 5% muscle pain and 10% joint pain[24]. These proportions did not consider patients who suffered these complaints less frequently. Sporadic or incidental complaints were tabulated separately. These figures (using 101 days out of a year) were considered to be representative of the prevalence of chronic musculoskeletal pain. A telephone survey of chronic pain in Australia showed the pain prevalence rate to be 19% and that the majority of sufferers had back pain. The pain was generally, either continuous or present daily. The majority had suffered pain for three years or more and 86% had experienced their pain in the previous two weeks[25].

The mode of onset of pain may be relevant to the diagnosis. Any hyperextension component may favour a zygapophysial joint origin. Injuries to the zygapophysial joints have been shown to occur in severe motor vehicle collision. Other causes include a direct fall onto the buttock, or a severe torsional stress. All of these traumas could also injure the intervertebral disc, the sacro-iliac joint or the myofascial structures.

Disc pathology should probably be favoured in chronic pain states that have arisen from a lifting incident. However, if the pain changes with time, this could mean that the changing stresses on the z-joints may be causing pain. It is not uncommon for post-fusion back or leg pain to be derived from an adjacent z-joint.

On examination extension, particularly combined with side flexion, may be the most restricted movement and may reproduce the pain. Local palpation over one z-joint may also reproduce the pain.

The incidence of back pain in the community is high and so, even with low yield, it is worth establishing a satisfactory method for diagnosing and managing pain of zygapophysial joint origin.

Only about 10% of back pain continues after two months and 2-3% after six months[26]. Perhaps many patients with a zygapophysial joint origin recover quickly and get help from conservative regimes including exercises, rest, braces, acupuncture, physiotherapy, manipulation, etc. Generally, invasive diagnostic measures such as zygapophysial joint blocks and invasive joint treatments are used for those patients not responding to non-interventional therapy.

Therapeutic options for pain of zygapophysial joint origin include steriod infiltration into the joint, interruption of the nerve supply to the zygapophysial joint or surgical destruction of the zygapophysial joint, for example by fusion.

EFFICACY OF RFD

The major nerve supply to the zygapophysial joint is via the medial branch of the dorsal ramus. Treatment to this nerve was first reported by Rees in 1971. He used a knife putatively to cut the dorsal rami or their medial branches[27]. Shealey then advocated and performed percutaneous radiofrequency thermocoagulation[28]. A variety of results have been reported since. None of the studies have had any controls. The authors have generally reported favourable results and have claimed that percutaneous radiofrequency denervation is a valid therapeutic tool and generally decreases the need for patients to undergo spinal operations and prolonged physical therapy. Most studies have concentrated on the lumbar spine.

It seems that surgery, particularly fusion, decreases the effectiveness of the radiofrequency denervation procedure and so most reports divide the results in groups of unoperated back, operated but not fused and fused.

Shealy reported on three hundred and eighty patients who had radiofrequency denervation in the lumbar spine. Good results were quoted in 80% of unoperated cases, 40% of operated (non-fusion) cases and 29% of fusion operations[28]. However, he used co-ordinates for the radiofrequency lesion that were most imprecise, and many studies after this used these same incorrect co-ordinates. This fact has probably been the major reason why radiofrequency is not in common use in spinal practice.

Lora and Long reported on one hundred and forty nine patients who had spinal radiofrequency denervation. Good results were obtained in 61% of unoperated cases, 27.5% of patients who had one operation and 0% in patients who had more than one operation[29].

Rashbaum reported on more than one hundred patients and described 82% good relief at one year and 68% sustained relief. He noted that the procedure did help in patients with chronic backache after successful surgery for radicular pain, but that it was ineffective in patients with multiple surgery even if the diagnostic blocks were positive, and in spondylolisthesis[10].

Oudenhoven reported on eight hundred and one patients at six months. Of the six hundred and three who had not had surgery, 57% reported excellent relief, 26% good relief and 17% failures. Of the one hundred and ninety eight who had previous surgery, 15% reported excellent relief, 32% good relief and 43% failures[30].

Lavignolle and Senegar reported on three hundred and fifty two patients at one year and then three years. In the unoperated group, 70% reported good results at one year[31].

Ray reported on one thousand and twenty patients, stating that 65-80% had a good immediate result and 50% had a permanently good result[32].

Katz and Savitz reported on one hundred and fifteen patients. They did not perform diagnostic blocks and reported good results in 66% of all patients, 75% in the unoperated and 58% in the operated group[33].

Ignelzi and Cummins used, as a basis for successful radiofrequency denervation, a reduction in pain of greater than 50% and a maintenance of activity level double or more when compared to the activity level prior to pre-radiofrequency denervation, plus discontinuance of narcotic medication. In sixty one cases they reported a 41% success rate. In addition they reported that at one to two year follow ups, the pain levels increased but

activity level was higher and narcotic analgesia was lower than prior to radiofrequency denervation[34].

More recently Rossi and Pernak reported on three thousand cases of RFD treatment which 55% were in the lumbar region. Overall they reported good results in 60-80% of cases at three to six months and 50% in the longer term. They reported good results in less than 30% of operated cases and noted that the worst results were in patients who had a fusion operation. They achieved their best results in the cervical region[35].

Wedley et al. reported on forty patients with a positive diagnostic joint injection. RFD was performed on half and a sham lesion on the other half. Early results have shown that at six months those patients who underwent RFD experienced superior pain relief when compared to the control group[36].

ANATOMY OF THE LUMBAR ZYGAPOPHYSIAL JOINT

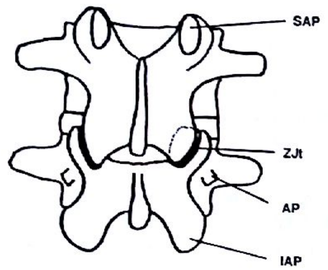

Fig. 1
3rd and 4th Lumbar Vertebrae (posterior view)
SAP - Superior Articular Process
IAP - Inferior Articular Process
Zjt - Zygapophysial Joint
AP - Accessory Process

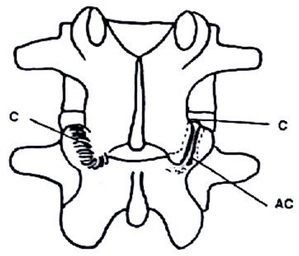

Fig. 2
3rd and 4th Lumbar Vertebrae
C - Capsule
AC - Articular Cartilage
Showing the zygapophysial joint cavity after the posterior capsule has been resected, and the attachment of the capsule is shown by the broken lines.
(after Bodguk N, Twomey L. Clinical Anatomy of the Lumbar Spine).

The zygapophysial joints are formed by the articulation of the inferior articular facet of a vertebra with the superior articular facet of the vertebra below. They are synovial joints with articular cartilage covering the facets, synovial membrane bridging the articular processes and a capsule surrounding this synovium. The capsule is formed antero-medially by the ligamentum flavum and postero-laterally by the capsular ligament.

The articular facets are not simply straight surfaces in the transverse plane; they are generally curved. There is a great variety in this curvature from one lumbar spine to the next. The orientations of the articular facets are of particular importance to the biomechanics of the lumbar spine.

The most typical curve is a gentle C-shape, but the joints can be straight and inclined from fairly close to the sagittal to almost 90° from the sagittal plane. Great differences in the shape of zygapophysial joints at one level can occur: this is called tropism. Tropism derives from the Greek "tropos" (change) and means a change in response to external stimuli. Tropism in the zygapophysial joints is most likely an adaptive change mediated by the summation of forces directed at the joints. These forces would usually produce the changes over a long period.

The articular cartilage of the zygapophysial joint is typical of all articular cartilage. There are four zones recognised in it under histological section[37]. The deepest zone is the calcified zone and it sits on the underlying subchondral bone plate. Age and degenerative changes effect all four zones of articular cartilage and the subchondral bone plate[38].

The zygapophysial joint capsule is a fibrous structure that encloses the joint posteriorly and connects via the inferior joint recess with the ligamentum flavum. The ligamentum flavum attaches to either side of the joint antero-medially and also encloses the superior joint recess. The inner parts of the capsule blend into the articular cartilage near the articular margins. The superficial fibres of the capsular ligament blend with fibres from the multifidus muscles. The capsule in the superior and inferior recesses is lax. There is a small foramen in these regions through which passes fat from the recess to the extra-capsular space.

NERVE SUPPLY OF THE LUMBAR ZYGAPOPHYSIAL JOINTS

The zygapophysial joints receive their nerve supply from the medial branches of the dorsal rami. The L1-L4 dorsal rami generally have a different configuration from that of the L5 dorsal ramus[39].

L1-L4

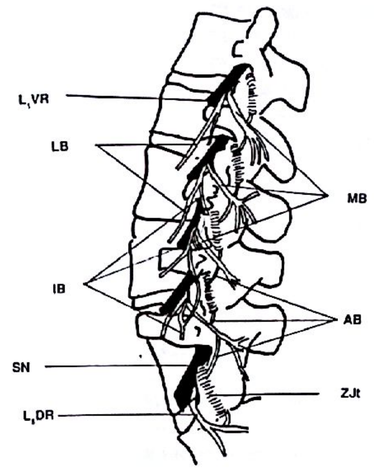

Fig. 3
Diagram of left posterior view of lumbar spine
VR - Ventral Ramus
DR - Dorsal Ramus
IB - Intermediate branch
LB - Lateral branch
MB - Medial branch
A - Articular branch
Zjt - Zygapophysial Branch
(from Bodguk N, Twomey L. Clinical Anatomy of the Lumbar Spine.)

These dorsal rami are short (5mm). They divide in the inter-transverse space, usually into three branches (the medial, intermediate and lateral) and sometimes into two branches.

The medial branch runs in a groove between the root of the transverse process and the superior articular process in a postero-medial direction and then through a groove between the mamillary process and the accessory process, covered by the mamillo-accessory ligament. In these regions the nerve, together with its companion artery, is adherent to the periosteum. Branches then arise to the corresponding superior and inferior zygapophysial joint and the nerve terminates in multifidus and the interspinous region. The fibres do not become cutaneous.

The intermediate branch supplies longissimus thoracis pars lumborum. Communication occurs between the intermediate branches at different levels.

The lateral branch crosses the transverse process lateral to the accessory process, goes through iliocostalis lumborum and the L1-L3 branches are cutaneous over the iliac crest[39].

L5

This nerve is longer and divides into two, the medial and the intermediate. The medial branch supplies the lumbo-sacral zygapophysial joint and the fibres of multifidus arising from the L5 spinous process. The intermediate branch supplies longissimus and communicates with branches from S1[39].

Dissection by Paris showed that other small nerves enter the capsule anteriorly, superiorly and inferiorly[40]. These are branches of the recurrent nerves that innervate the disc and some canal structures[32]. It is apparent that a radiofrequency denervation procedure probably cannot affect all of the nerve supply to a zygapophysial joint, as there is multiple innervation. However, denervation of the medial branch will significantly affect nociception of a zygapophysial joint.

AGE AND TRAUMA CHANGES IN THE ZYGAPOPHYSIAL JOINT

The term "posterior marginal damage" has been coined in recent studies to describe changes seen in the lower quarter of the zygapophysial joint. The changes include[37]:

- Stretching and tearing of the joint capsule
- Articular cartilage tears
- Articular cartilage separation from sub-chondral bone plate
- Incongruity of the bony margin at the articular cartilage (calcified zone) and sub-chondral bone plate interface.

These changes can result in joint instability and deformity of the articular surface, predisposing to osteoarthritis. These changes are seen both in middle or later life and in younger spines as a result of acute trauma. In a study of two hundred and four lumbar spines of all ages, there was a group of thirty one lumbar spines that had been involved in a violent event that had led to death. Lateral and antero-posterior X-rays did not reveal evidence of injury to the zygapophysial joints. Post mortem studies of up to three joint were made (L1-2, L2-3 and L4-5). Of these thirty one spines, only two were free of zygapophysial damage. Eleven had evidence of bony injury; six having fractures of the sub-chondral bone plate and the rest having central infractions of the sub-chondral bone plate. Soft tissue injuries occurred in twenty six of these lumbar spines. The changes included "posterior marginal damage", with capsular tears and articular cartilage splits or separation from the sub-chondral bone plate[37].

The age changes that appear are accelerated and aggravated by instability of a motion segment. The mechanics and loading characteristics of the zygapophysial joint will be influenced by the status of

other supporting structures, particularly the intervertebal disc and the bony supports, the laminae.

Disc injuries that allow excessive movements of any segment will produce greater movement at the zygapophysial joints. Apposition of the joints is controlled postero-laterally by the capsule and is influenced then by multifidus action, as this muscle attaches to the capsule. Excess mobility of an intervertebral segment places the zygapophysial joint at risk to injury, and the paravertebral muscles probably have to work harder to maintain the zygapophysial joint apposition. This may produce back ache due to muscle fatigue.

It is apparent that back pain from the zygapophysial joints can occur. This pain can be in an isolated joint or in a number of joints and will often co-exist with pain of intervertebral disc origin.

DIAGNOSTIC PROCEDURES

The most common problems that produce chronic pain of low back origin are injuries to the disc and the zygapophysial joints. Each structure is innervated and relevant pathology has been demonstrated both in the disc and in the zygapophysial joint.

At present there is no established pattern on history or physical examination to make a definitive diagnosis of pain derived from a disc or from a zygapophysial joint. In addition, most investigations are of little use in determining the origin of pain. There is no correlation between plain X-ray changes and pain origin. The presence or absence of radiological changes is irrelevant in determining the origin and the severity of a mechanical back disorder. C.T. scan is also unhelpful. Changes in the zygapophysial joints, including tropism and degeneration, indicate the effects of forces directed at those joints in the past but do not indicate whether or not the joints are painful. Disc bulging is as yet a non-specific finding that lacks any clinico-pathological correlation.

Disc prolapse is an exception to the above. There is a well established and recognisable pattern of history and examination, and the diagnosis can be confirmed by C.T. scan, myelogram or magnetic resonance image. However, disc prolapse is rare in the setting of chronic pain of low back origin. It probably accounts for between 1-5% of all low back problems[23].

It is almost impossible to differentiate between pain of disc and zygapophysial joint origin on clinical grounds. However, some factors suggest a greater likelihood of a zygapophysial joint injury.

Pain of primary disc origin is more likely in patients who injure their backs when applying torsional and/or axial compressive forces to their spines. Examination often reveals restricted forward bending, often with an arc of pain, pain on returning to the upright position (and this pain can be relieved by passively moving the patient in this way), pain on extension, normal straight leg raise, local lumbar tenderness, and pain exacerbation with sit ups and/or rotation.

It is probable that pains of disc and zygapophysial origin frequently co-exist. The majority of patients will be found to have a mixed pattern of signs and careful discrimination is required in the assessment of their problems.

The management of pain using invasive techniques, however, requires satisfactory elucidation of the origin of pain. Two methods can be used to determine pain of zygapophysial joint origin, local anaesthetic blockade into the zygapophysial joint or into the nerve supply of the joint. These procedures can be delivered precisely but unfortunately do not necessarily implicate the zygapophysial joint as the source of pain for two reasons.

The first is that even if local anaesthetic (and contrast) is injected inside the capsule of the zygapophysial joint, leakage can occur into the spinal canal through the antero-medial capsule of the joint (the ligamentum flavum). In this way a zygapophysial joint injection could produce blockade of spinal canal structures, or of another zygapophysial joint; in fact, most potential sources of spinal pain could be blocked by a single zygapophysial joint injection. Similarly, attempted blockade of the dorsal ramus, even with low volume anaesthetic, could easily block nearby structures, such as multifidus.

The second reason applies to the injection of the dorsal ramus. The anaesthetic could produce analgesia not only by blocking the dorsal ramus but also potentially by defusing a local trigger point in the paravertebral muscles. The putative trigger point, having occurred secondarily to say disc pathology, may have become a potent source of pain and after the block the patient may feel and move much better than before. In this way the zygapophysial joint could be implicated as a major source of pain but the pathology could be elsewhere.

With these provisos in mind, diagnostic procedures for pain of zygapophysial joint origin can be used. Prior to the chosen procedure the patient is examined. Typical aggravating features are established and tested, e.g. sitting pain tolerance, slight flexion pain tolerance, and formal movement tests are applied with pain response (flexion, extension, side bending, rotation). Ten minutes after the procedures the movements are retested and the sitting and slight flexion pain tolerances are measured. If long acting anaesthetic (e.g. bupivicaine) has been used, the patient can then attempt typical aggravating tasks, such as gardening or sitting in a car, and assess the pain response.

If all pain is eradicated, and movements become pain free, then it is likely that the zygapophysial joint is responsible for the pain.

The zygapophysial joint injection can be used as both a diagnostic and therapeutic procedure if a corticosteroid is added to the local anaesthetic. An effective therapeutic response to the zygapophysial joint cortisone injection may well be the best guide to implication of the zygapophysial joint as the source of pain but because of leakage it can still be non-specific.

RADIOFREQUENCY DENERVATION

Radiofrequency denervation (RFD) is used if the diagnostic blocks implicate the zygapophysial joints as the source of pain, and when the therapeutic effect of the corticosteroid has abated.

The facilities required to perform lumbar RFD are:
1. X-ray screening table
2. Image intensifier preferably with C-arm
3. Radionics radiofrequency lesion generator system
4. Radionics needle
5. Anaesthetic and resuscitation equipment.

A pre-medication is usually given (e.g., Pethidine). The patient is placed prone on the table. The lumbar lordosis is maintained to facilitate needle position, as a more vertical needle entry track can then be made to enable the needle tip to run as close as possible to parallel to the dorsal ramus branches.

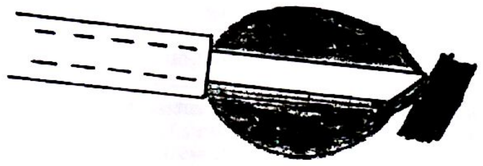

Fig. 4
The needle when heated produces a circumferential lesion in the surrounding soft tissues. It is important for the nerve to run inside the shaded area. If the tip of the needle is placed on to the nerve, very little of the nerve will be successfully denervated.

A light general anaesthetic (e.g. Dipravan) and analgesic is given intravenously. The patient remains conscious and mildly alert during the procedure.

The target points are identified using a direct A-P screen, and the points are marked on the skin. The plan for needle entry points is then made and their positions marked. Consideration must be given here to getting the needle as near as possible to parallel to the dorsal ramus branches[41]; and to avoid striking the articular processes before reaching the transverse process. This is often difficult and it is often important to insert the needle rather inferior and lateral to the target point.

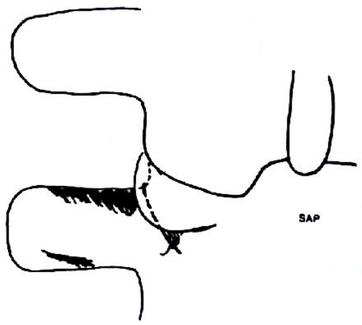

Fig. 5
Path of medial branch of dorsal ramus as it runs posteromedially along the transverse process, in the junction of the root of the transverse process and the root of the superior articular process (SAP). The nerve is shown in a broken line where it lies concealed behind the SAP. Note that in the lower lumbar spine the transverse process is slanting anteriorly from below upwards, and thus as the medial branch emerges it is running almost perpendicular to the skin. Thus, when the RFD needle is inserted, it will be almost parallel to the nerve even when inserted at the horizontal level of the nerve.

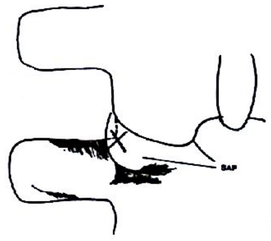

Fig. 6
Diagram showing the target point for a radiofrequency denervation. "X" is on the path of the medial branch of dorsal ramus. On X-ray the superior articular process (SAP) is difficult to see, and as shown it may overlie the target point (X). If the needle is aimed straight down onto point X it will hit the SAP and appear on X-ray to be in correct position. This can be avoided by coming from a lateral position and marching the needle along the transverse process until it hits the base of the SAP (see Figure VII).

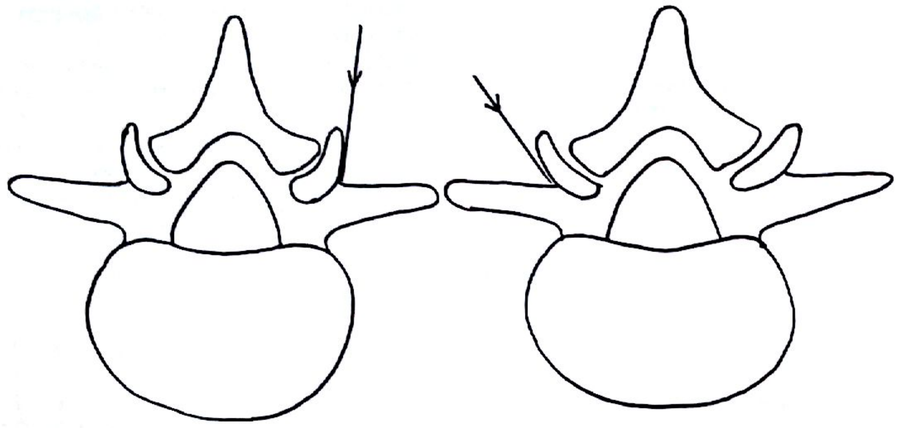

Fig. 7
The orientation and curvature of the lumbar zygapophysial joints will determine the angle of needle insertion. As the alignment cannot be detected on plain X-ray, it is best to insert the needle laterally, running it along the transverse process until it hits the base of the articular facet.

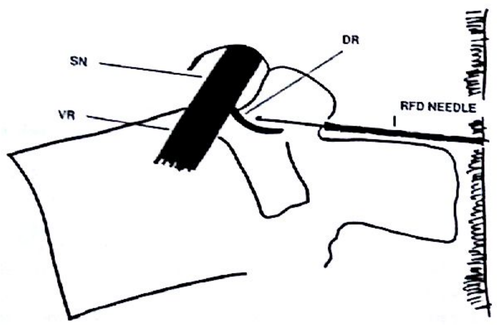

Fig. 8
View of lower lumbar vertebra from a lateral and slightly superior aspect. The RFD needle can be inserted almost perpendicular to the skin in order to run parallel to the medial branch of dorsal ramus.
SN - Spinal nerve
VR - Ventral ramus
DR - Dorsal ramus (medial branch)

The skin is sterilized and draped, and the surgeon gloved. The needle is connected to the lesion generator system and the circuit is completed with a diathermy pad attached to the patient's leg. The needle is then inserted onto the target point and always onto bone. The stimulation system is used. A positive stimulation response occurs if the needle is adjacent to the dorsal ramus or its branches. The response can be sensory, when the patient will experience pain similar in quality and position to the presenting pain, and/or motor, when the paravertebral muscles will contract. If the needle is too close to the ventral ramus, stimulation will cause contraction of the relevant leg muscles.

When the appropriate site is established by the stimulation test, preparation is made to induce the radiofrequency lesion. Xylocaine 1% 1ml is injected through the Radionics needle. The programme is switched to "lesion" and a lesion is applied using about 22 volts. An alternative is to use a temperature monitor on the needle tip, and to heat the tip to between 60° and 80° Centigrade.

Often the diagnostic procedures will give a guide to the dorsal rami that should be denervated. However, as the dorsal rami do communicate, it is prudent to denervate four nerves on the side of pain if either L4-5 or L5-S1 has been implicated; those nerves should be L3, L4, L5 and S1.

POST-OPERATIVE CARE

When the patient is back in the ward he or she is visited by a physiotherapist. The ramifications of the procedure are discussed again. Gentle mobilizing exercises are commenced and a routine for that patient established.

Patients are advised that a pain reaction may occur for up to four to five weeks but that this is rare, with most post-operative pain settling within a day or so. At times mild low back or proximal paraesthesia can occur but, once again, this is usually transient. Patients are further advised that when they begin to feel improved they can return slowly to increased activity but they must remember that their back is injured and they must take care. The patients are reviewed at about four to six weeks.

Patients should also be advised that the effect of the RFD will probably abate in about ten to twelve months and that repeat procedures can be given. The nerve should recover totally in that time. Further RFD will be required for patients with recurrent pain.

THE FUTURE: CONTROLLED STUDIES

It would be very difficult to produce a satisfactory study that will prove that zygapophysial joint pain can be alleviated by radiofrequency denervation. From experience, it seems that a zygapophysial joint is only rarely the single source of back pain, and this is confirmed by other studies[15]. In addition, the blocking techniques have their own limitations in regard to isolation of the zygapophysial joint and its nerve supply.

The correct anatomical sites for blocks and denervations must be observed. Many of the older studies reported good results without specifying accurately where the needles were placed. Also, the approach of the radiofrequency needle to the nerve must be pre-calculated to ensure that nerve and needle are parallel for maximal lesion.

One proposal for an experimental protocol is as follows:

1. The four most painful tasks for each patient in a large group should be designated and scored according to painfulness by the use of a visual analog scale (VAS).
2. The appropriate medial branches should be injected with either 0.5 ml. Marcaine or 0.5 ml. saline on two different occasions (with the operator remaining blind as to the material injected).
3. Total abolition of pain associated with all four activities, (as measured by the VAS scores) following Marcaine injection would be considered as positive indication of the pain having zygapophysial joint origin. No relief should occur following the saline injections.
4. All patients with positive blocks could be treated by radiofrequency denervation and the results assessed, with total relief as a positive result and anything less as a negative result[42].

These criteria may be too severe, as a zygapophysial joint is very rarely the only site of pain genesis and the sampling population would have to be enormous. The suggested classification of results may be too rigid and perhaps success should be measured by return to work, recovery of lifestyle or cessation of analgesia.

CONCLUSION

Radiofrequency denervation of the nerve supply to the posterior structures of the spine can be an effective method of controlling chronic low back pain. As the diagnosis of zygapophysial joint injury cannot be precisely determined by current techniques, case selection is difficult and results may appear less than satisfactory. However, RFD is a safe procedure and when effective provides significant and prolonged relief.

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Appendix

A review of The Principles of Radio Frequency Generation and the Behaviour of Tissue Impedence during R.F. Heating - Cashman E.R.

HISTORY OF DIRECT CURRENT ELECTROLYTIC LESIONS

1895 Golsinger - Unipolar lesions in dogs
1905 Horsley & Clarke - Anodal lesions
1947 Spiegel & Wycis - Stereotactic lesions
1953 Sweet & Monk - Showed that DC lesions were erratic in size and suggested that DC lesions should cease and that radiofrequency might produce better control.
1955 Aronow & Cashman - Produced the first practical R.F. generator.

MECHANISM OF RADIOFREQUENCY ACTION

The Radiofrequency does not work by the needle top heating up. It sets up an electrical field, causing ionic oscillation, and the subsequent friction produces heat.

ADVANTAGES OF RADIOFREQUENCY LESION

1. Lesions well circumscribed.
2. With temperature control the lesion site is quantifiable and no charring or boiling occurs.
3. Excellent target control can be achieved by the use of stimulation, impedence monitoring and recording.
4. It is robust and adaptable.

Reference

1. *Lecture given at the Satellite Meeting for the International Association of Pain, Perth 1990.*

FACTORS TO CHECK ON WITH RADIOFREQUENCY PROCEDURES

1. Erratic behaviour of volt and ampere meters.
2. Body temperature should be normal.
3. Insulation on cable.
4. Have a spare electrode.
5. Have a large dispersive area.
6. Check impedence monitor (200-300 ohms).
7. Raise power smoothly and check power.

MAIL BAG Letters to the editor

"I had written him a letter....."

Dear Sir,

It is commonly stated that spasm of the large muscles of the neck following a motor vehicle accident is due to a protective mechanism guarding damaged deeper structures. Cases which I see cause me to doubt the universal infallibility of this hypothesis.

The patients under discussion belong to two groups; those seen by me years after their involvement in a MVA, and those seen by me three of four days post-accident. The factors common to both groups are rear-end collisions, and onset of severe pain in the posterior muscles of the neck two or three days after the accident.

Let us consider a patient seen within four days of sustaining a whiplash (i.e. the head was jerked backwards and then forwards). When seen by me, there was painful bilateral spasm of the posterolateral muscle mass in the neck, and active and passive neck movements were limited to 5° or less in any direction. Other accident-related symptoms were negligible. On palpation, the muscles in spasm were found to include the levator scapulae and upper fibres of the trapezius. The function of these latter is to act on the scapula, not on the neck. Why then would they go into spasm to protect deep cervical structures?

The head having been forcibly extended without warning, reflexes have now been altered and muscles tensed to resist the anticipated forced flexion. The shoulder girdle has tensed, thereby putting the levator scapulae and trapezius under tension. They are therefore pressed into service in helping the next extensors to resist flexion; i.e. they have become synergists. The result is that extensors and synergists are subjected to eccentric contraction. Let us therefore consider scientific reports on the effects of eccentric as opposed to concentric contractions.

Late onset pain occurring only after eccentric contractions has been described by many workers during the last forty years, and its cause has been elucidated in this decade. Newnham et al. 1982[1] used electron microscopy to reveal structural damage in the human quadriceps after short term eccentric exercise; i.e. using one leg to step up and the other to step down. The damage and pain in the latter leg approached its maximum after 48 hours, the former leg being normal. Similar results were demonstrated by Friden and Ekblom 1987[2].

Returning to my two groups of patients, those seen in the early stages responded quickly and completely to two or three sessions of gentle massage, warmth (e.g. hot water bottle) and self mobilisation using muscle-energy techniques. No residual symptoms have been reported up to five years later.

What of those seen by me two or more years after their MVA? I find marked restriction of neck movement usually at all levels down to T3 or 4 but some being worse than others. There is tenderness and palpable soft tissue thickening over the O.A., A.A. and Z.A. joints of the remaining cervical vertebrae. Some patients even develop intervertebral disc pathology two or more years post-accident.

These are the same degenerative changes which we find in long-standing cases of occupational cervicobrachial disorder (RSI), and the presumed common cause is hypokinetic degeneration. Both groups have undergone prolonged periods of reduced neck movements - the MVA patients being due to pain and the OCD sufferers due to fixed posture during their employment.

Hypokinetic Degeneration of Joints is described thus by Lamb[4], "If joints are kept in improper positions for even a few days, connective tissue in tendons, ligaments, muscle and joint capsules becomes dense and shortened; eventually this connective tissue strongly resists any attempt at stretching to regain the lost range of motion of the joint".

Bland[3], reports on the similarity of all connective tissue and reports on experiments showing that "In the immobilised connective tissue, without continual mechanical stimulation, the tropocollagen fibres are synthesised and exported, but

they fail to aggregate in linear array - instead, they are laid down in random form, as ionic bonds give way to aldehyde bonding; critical and crucial fibre intercept points become locked by inappropriate bonding at these intercept sites. The structure then is limited in its gliding characteristic and becomes locked in a random fibrosis."

Cyriax[5] reports on the painful nature of bulky collagen scars resulting from lack of early mobilisation.

The vital question is - are we creating long term invalids by not mobilising these and other cases of spinal injury early enough and adequately enough; as is the accepted treatment in injuries to joints of the limbs?

(Dr) **Luan Renouf**
3 Oxford Close,
Warranwood, Victoria

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McKenzie Method

Dear Sir,

It was fascinating to read your recent article by Nikolai Bogduk. Bogduk is now saying that annular fissure is a potent cause of lumbar pain. In another article in Spine magazine Bogduk and associates conclude that probably 80% of lumbar pain is generated in the disc.

It seems that after many years of dispute the anatomists are coming down heavily along the lines which Robin McKenzie so bravely postulated so many years ago.

Unfortunately McKenzie's work, in this country, is still very widely misunderstood by physiotherapists and often unheard of at all by doctors.

Would it not be an opportune time to include the basics of the clinical method in our journal? Then maybe all doctors will understand how "annular strain" comes about and how it should be dealt with in practice.

Yours faithfully,

(Dr.) **Michael D. Taylor**
11 Mostyn Street,
Castlemaine, Victoria. 3450

No doubt members would be interested in an article setting out the McKenzie method. It could also be the topic of a Licentiate thesis. How about it, M.T.?

-Ed.

Proposal Submitted to the Flinders University for a Graduate Diploma in Musculoskeletal Medicine

INTRODUCTION

Musculoskeletal medicine is that branch of medical science concerned with the functions and disorders of the musculoskeletal system. This includes the axial and appendicular skeletons as well as those parts of the nervous system associated with them.

The need for such a course of study is apparent when considering:

(i) Topics covered in this course are scattered through various disciplines at the undergraduate level and therefore there is a great need for this subject matter to be co-ordinated in an interdisciplinary systematic way.

(ii) A significant proportion of general practice consultations are in this area.

(iii) General practitioners freely acknowledge that a greater depth of knowledge in this area would make their practices more efficient and satisfying.

(iv) An ageing population will require more attention with problems which arise from the musculoskeletal system.

(v) Competence in diagnosis, assessment and treatment of musculoskeletal injuries occurring in the workplace has the potential for saving millions of dollars.

AIMS

- To provide an opportunity for medical graduates to obtain expertise in the assessment, diagnosis and treatment of musculoskeletal disorders.
- To develop skills in critical assessment of the literature related to musculoskeletal problems.
- To develop skills in the measurement, design, analysis and evaluation of research methodologies related to musculoskeletal medicine.
- To develop a critical approach to the resolution of the numerous problems arising from the musculoskeletal system.

OBJECTIVES

As a result of completing the graduate diploma the candidate will be able :

- To demonstrate a competent understanding of the anatomy, physiology and pathology of musculoskeletal disorders.
- To initiate a course of treatment for musculoskeletal disorders after evaluating the pertinent aspects of the history and examination.
- To justify investigative procedures and treatment programmes.
- To understand the role of other health professionals in the management of musculoskeletal injuries and disorders.
- To undertake further study and/or research in order to expand knowledge and understanding in the area of musculoskeletal medicine.
- To evaluate critically the scientific literature of relevance to musculoskeletal medicine.
- To formulate a research plan.

COURSE REQUIREMENTS

The graduate diploma is offered as a half time commitment over four semesters.

A full time university student will be expected to have 16-18 contact hours per week. This course requires the candidate to commit one whole day a week for four semesters. The course will be made up as follows:

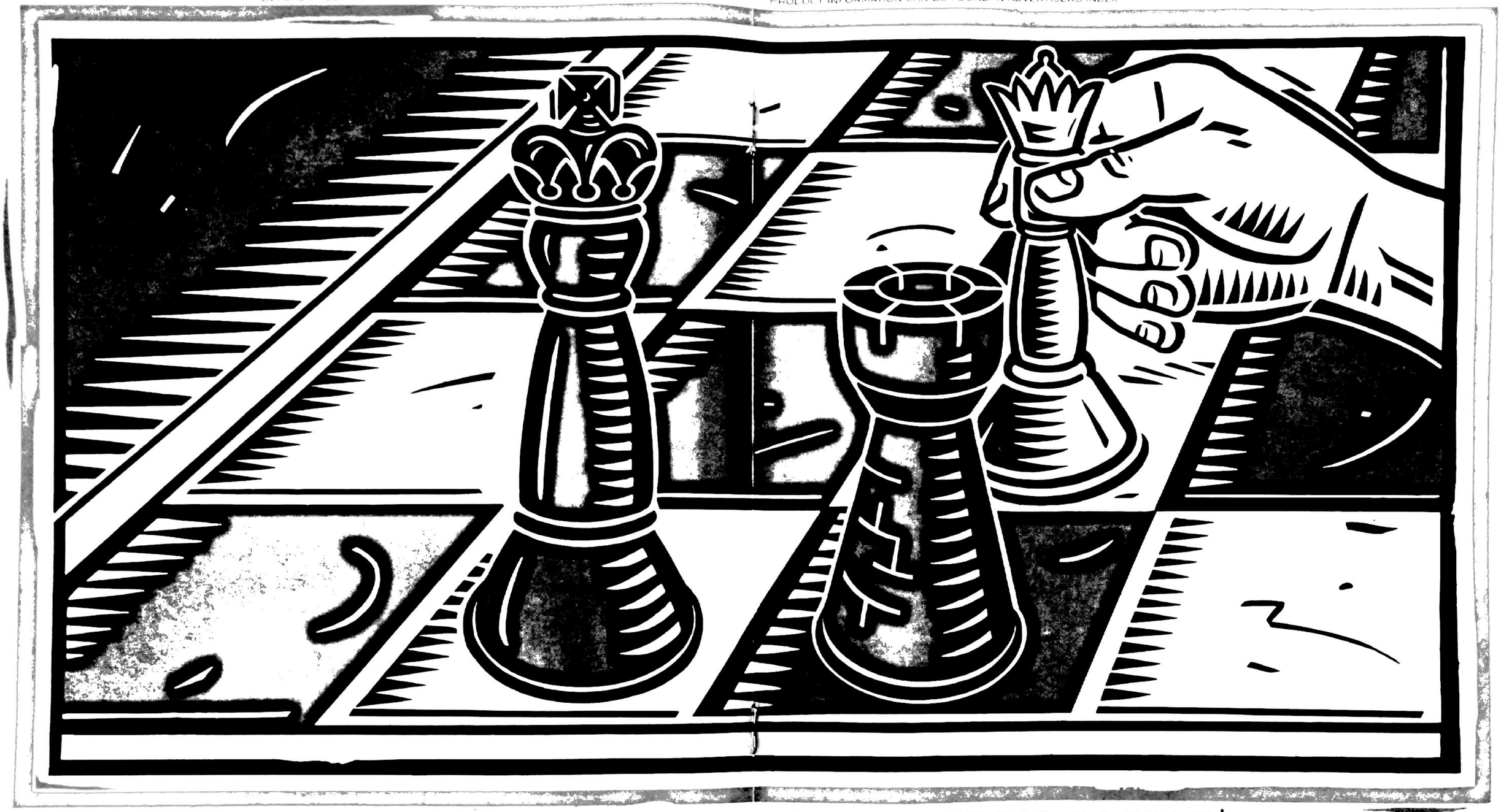
a) Five day inservice workshop at the beginning of each year.
   Year 1 Basic Sciences 1 and Examination techniques.
   Year 11 Basic Sciences 11 Clinical Case Studies and Audits.

b) Sixty supervised clinical sessions, most of which will be in musculoskeletal clinics with four weeks secondment to each of orthopaedics, rheumatology, neurology, neurosurgery and pain clinic.

c) 1 or 2 lectures per week on topics from the syllabus. Topics from the syllabus not covered by lectures will also be examinable.

d) 1 - 2 hour seminar/tutorial per week on assigned topics.

In addition to the above the candidate will be required to keep a log book of 20 patients. The log book will include the usual patient notes with details of musculoskeletal history, diagnosis, assessment and treatment regimes. On-going assessment is expected and the candidates will be required to defend their lines of management with appropriate references.



ARTHRITIS PATIENT RESCUES QUEEN FROM CASTLE!

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(diclofenac sodium)

RELIEVES ARTHRITIC PAIN.

Myofascial Pain Syndromes

Wade King
82 High Street, Taree, NSW, 2430

ABSTRACT

This paper reviews the terminology, clinical phenomena, postulated mechanisms and practical aspects of assessment and management associated with myofascial pain syndromes.

Various forms of terminology are described, with some reference to the historical development of concepts of conditions affecting muscles and other soft tissues. In particular, the terms fibromyalgia and myofascial pain syndrome are interpreted, with consideration of the possible relationships between them. The clinical phenomena of tender points and trigger points are addressed similarly.

Myofascial pain syndromes are described in the light of clinical and pathophysiological findings. Mechanisms believed involved in the development of taut bands and trigger points include the combination of high energy expenditure and inadequate oxygenation. The increased sensitivity of trigger points has been related to the action of bradykinin, prostaglandins and other algogenic substances. Described mechanisms of pain referral involve peripheral branching of nociceptive axons, autonomic connections and central nervous system pathways.

The aetiology of myofascial pain involves factors which cause muscle dysfunction and factors which tend to perpetuate established conditions. These factors also determine the natural history of each condition. Syndromes can be classified by clinical manifestations and by aetiological status.

Clinical features of a myofascial pain syndrome include a history of pain in a characteristic pattern of distribution, other minor symptoms and specific physical signs, such as a palpable taut band in the muscle, tenderness at a trigger point, a local twitch response, the jump sign and symptom reproduction on trigger point stimulation. Pressure algometers have been developed to quantify trigger point assessment. Ancillary tests are not necessary for reliable diagnosis, although thermography may be useful for imaging thermal patterns related to myofascial phenomena detected clinically.

Management of myofascial pain syndromes involves treatment of the cause and treatment of the affected muscles. Many modalities may be useful for the treatment of myofascial dysfunction, including patient education, rest, therapeutic exercise, heat modalities, manual therapy, vapocoolant spray and stretch, trigger point injections, oral medication, neuromodulation (including TENS, acupuncture and biofeedback), psycho-social management and rehabilitation.

INTRODUCTION

Muscular and fascial pains are among the most common afflictions of mankind. Many of these pains are neither intense nor enduring, but the incidence of severe and chronic myofascial pains is still so high as to be of social and economic importance for society as a whole, as well as the cause of considerable morbidity for individual sufferers. Sternbach[1], in the Nuprin study, showed the incidence of chronic musculoskeletal pain in the United States to be 24% or almost one in four of the whole population. A survey conducted along similar lines by Strauss[2] showed comparable figures for the number of people affected in Australia. A large proportion of this very numerous symptomatic population have myofascial pain, either as their sole complaint or as one element of a more complex problem.

An American survey[3] of patients with musculoskeletal pain presenting to a major pain clinic showed that myofascial pain was diagnosed, either alone or in combination with other problems, in 85% of two hundred and eighty three consecutive cases. A similar study[4] at another clinic showed the incidence of myofascial pain to be 76% of two hundred and ninety six consecutive patients.

Despite the high incidence, many aspects of myofascial pain are ill-defined and poorly understood. It is as if the very familiarity of the condition has bred a contempt for its scientific explanation. Vague concepts and confusing terminology abound in the literature and seem to have gained a measure of acceptance in many quarters. If they have done so it has been by their frequent repetition rather than by their intrinsic contribution to the rational explanation of observed phenomena. The literature does contain many pertinent facts and valid descriptions of relevant pathophysiological mechanisms but discernment is required to separate useful information from the many extraneous and even misleading statements in a confused milieu.

TERMINOLOGY

Perhaps the most difficult aspect of understanding material published on the subject of myofascial pain is in interpreting what seems to be a plethora of descriptive terms with confusingly similar sounds and overlapping meanings. Terms which designate clinical entities include (amongst others) fibrositis, the fibrositis syndrome, fibromyositis, myofibrositis, myofasciitis, myogelosis, myositis, polymyositis, polymyalgia, muscular rheumatism, non-articular rheumatism, fibromyalgia and myofascial pain syndrome. Phenomena associated with these entities are known as nodules, tender points, trigger points, taut bands and motor points (and they are only the current terms; many others have been used in the past and are found in older publications). All these terms may be found qualified by adjectives denoting chronicity, aetiologic reference, tissue involvement or anatomic site, and sometimes a combination of these. Eponymous terms, such as Travell points, Ritchie Russell points, Valleix points and Trousseau points may also be encountered.

Fibrositis is a term introduced by Sir William Gowers[5] in 1904 to designate the muscular changes often associated with recurrent or chronic low back pain. Gowers described inflammation of the fibrous tissue within muscles and asserted that such processes were responsible for the tenderness and firmness of muscles observed clinically. This was a landmark in the evolution of thought on such matters in that Gowers attempted to describe the common clinical condition by its pathology, whereas previously it had simply been known by its symptom, as "lumbago". Through the work of successive authors such as Stockman[6], Llewellyn and Jones[7], Kelly[8], Brendstrup et al.[9] and many others, the term fibrositis came into frequent usage to denote a particular muscular condition.

Unfortunately, the concept was accepted uncritically by many and became applied much too widely to "explain" a variety of clinical phenomena. The almost inevitable reaction was the publication of several biopsy series, as by Abel et al.[10], Collins[11] and Valentine[12], which failed to show typical inflammatory changes in a significant proportion of cases. Many authors such as Halliday[13], Moldofsky[14] and Weinberger[15] derided the idea of fibrositis as a useful physical concept and wrote of it as a non-organic diagnosis, using such terms of reference as "psychogenic rheumatism" and "neurasthenic musculoskeletal pain". Medical opinion became divided between the two extremes, with most practitioners adopting the middle ground and developing a healthy degree of scepticism for the emotive arguments of the various proponents.

For many this scepticism extended to the whole topic of musculoskeletal pain in general, and muscular pain in particular, and any condition which could be dubbed fibrositis was regarded with suspicion in some quarters. The term has survived, more recently as **the fibrositis syndrome** and it continues to be applied to physical problems but it is tainted by controversy and is probably best avoided.

Fibromyositis, myofibrositis and **myofasciitis** are used more or less interchangeably in the literature to denote variants of fibrositis. They suffer the same problems as the parent term.

Myogelosis is a term found mainly in the European literature. It was coined in 1921 by the German author Schade[16], who attributed the areas of tenderness and firmness in muscles to localised biochemical changes which caused proteins to gel. In terms of its precision and objectively demonstrable scientific basis it may be considered equivalent to the English fibrositis.

Myositis, polymyositis and **polymyalgia** are some of the names applied by various authors to conditions which were grouped in the older literature under the general heading **muscular rheumatism**. While each has a particular literal meaning, the differences between them are more semantic than scientific. In general they are used to refer to muscular diseases (such as polymyalgia rheumatica) but variations in their usages prevent the single words being related consistently to discrete categories of muscle pathology.

Non-articular rheumatism was introduced in 1938 by Gutstein-Good[17] as a generic term to include all of the aforementioned. There is a certain honesty in its non-specificity but this is as much a weakness as a strength. In essence it describes little more than a broad category of ill-defined entities affecting non-articular components of the musculoskeletal system. It is subject to all the shades of meaning and controversies related to fibrositis, as well as an additional layer of controversy related to allergy, auto-immunity and other postulated mechanisms. Its boundaries are difficult to define.

Fibromyalgia is a term coined by Hench[18] in 1977 and later elaborated by Yunus et al.[19, 20] in response to the controversies surrounding the various concepts of fibrositis. They applied it to distinguish conditions with a demonstrably physical basis from others in the broad category of fibrositis which were either of doubtful aetiology or frankly psychosomatic. In doing so they rescued the notion of a specific dysfunctional state of muscles and fibrous tissue associated with localised pain and tenderness. Yunus et al. defined the entity by criteria which included localised tenderness at five or more of forty specific anatomic sites without other apparent disease, with pain (typically of a dull, aching quality) persisting at three or more sites for three months or longer; at least three of ten "modulating factors" were also required in Yunus's description, including symptom aggravation by weather, by physical activity and by psychological stress, and general features such as sleep disturbance, lassitude, anxiety, headache, irritable bowel syndrome, subjective swelling and numbness. Wolfe[21] argued that tenderness at seven of fourteen specific sites was all that was necessary for a diagnosis of fibromyalgia and that whilst the modulating factors described by Yunus may co-exist they were not only unnecessary for the diagnosis but tended to lead back in the direction of non-specific and psychosomatic states.

Myofascial pain syndrome is a term used in reference to a particular muscle, or group of muscles (as opposed to the more diffuse nature of fibromyalgia). The pathognomonic feature of a myofascial pain syndrome is the presence of one or more sites, within a muscle or its associated fascia, which are painful, tender and when stimulated physically cause referral of pain to other sites in a characteristic pattern. This type of pain referral had been reported in the early literature by several authors and ascribed to sites of origin in various somatic tissues, without adequate explanation. In 1936 Edeiken and Wolferth[22] identified such sites as occurring in muscle and designated them "trigger zones". The concept was developed further in 1942 by Travell and her co-workers[23] who described the treatment of certain types of shoulder and arm pain by the intramuscular infiltration of procaine into what they called "trigger points". The adjective "myofascial" was added in another paper by Travell et al.[24] in 1952 and the points have been known as myofascial trigger points ever since. The characteristic patterns of pain referral from these points became recognised as phenomena distinct from other manifestations of "fibrositis" and the combination of symptoms and signs associated with a particular muscle became known as a myofascial pain syndrome. These syndromes have been described in detail by numerous authors, including Bonica[25], Sola[26], Berges[27], Reynolds[28] and Simons[3, 29]. The collaboration of Travell and Simons, notably as co-authors of a comprehensive textbook on the subject[30], has resulted in a number of significant publications[31, 32, 33] in which aspects of the syndromes are elaborated.

The names applied to the clinical phenomena associated with the various entities also have a degree of similarity which tends to be confusing.

Fibrositic nodules was the name used by the earlier authors to describe their findings of localised tenderness and firmness in the muscles and other soft tissues of patients with conditions recognised as fibrositis. The concept of nodules became widely accepted and numerous suggestions were put forward to explain their precise nature. Various histopathological observations were reported and their diversity reflected both the range of different conditions included in the general category of fibrositis and the heuristic nature of some of the investigations.

Tender points is a more recent term applied to sites of tenderness in the soft tissues of patients with fibromyalgia[34]. As described above, tender points are often found at anatomically consistent locations and the number of such sites which are tender provides the major criterion for the diagnosis of fibromyalgia. A useful abbreviation for tender point is TeP.

Trigger points are points which are both tender locally and associated with reproduction of pain at distant sites. They are found in the bellies and tendons of muscles and are characteristic of myofascial pain syndromes[30]. Trigger points have also been described in ligaments[35], joint capsules[36], periosteum[37], skin[38] and scar tissue[39]. A convenient abbreviation for trigger point is TrP.

Taut bands are palpable bundles of tense muscle fibres found within tight and irritable muscles involved in myofascial pain syndromes. The taut band usually lies in the long axis of an affected muscle and represents the more irritable part of it. Myofascial trigger points are usually located in taut bands.

Motor points are the anatomic sites at which motor nerves enter muscles and were described as early as 1857 by Von Ziemssen[40]. They respond to electrical stimulation rather than to palpation and their location is determined by electrodiagnostic techniques[41].

Eponymous terms are used in some publications to refer to these clinical phenomena. As always, the use of eponyms obscures rather than enhances meaning. Strictly, such terms apply only to their subjects as described in the original papers of the authors whose names are used eponymously. However, they sometimes gain currency and are used in a wider sense; those which follow would come into this category.

Travell points is simply another term for (myofascial) trigger points as originally described by Travell[23].

Ritchie Russell points were described in 1959 as occurring in skin[42] and in the light of later knowledge it seems that they represent one form of trigger points.

Valleix points seem to be another manifestation of trigger points and were probably located in muscles even though when Valleix[43] described them in 1841 he considered them to be in nerves.

Trousseau points were described[44] as tender points in the skin over zygapophysial joints; these "*pointes apophysaires*" were almost certainly another expression of trigger point phenomena.

Whilst some appreciation of each of these terms is required in order to understand the relevant literature, a simplified and systematic usage is essential for effective communication of the pertinent facts and ideas. It is suggested that "fibromyalgia" be used to refer to the diffuse entities of muscular and other soft tissue dysfunction and "myofascial pain syndromes" be used to describe conditions affecting single muscles or particular groups of muscles. In fact such a convention is developing in the current literature; fibromyalgia tends to be described in rheumatological publications and myofascial pain syndromes in the literature of musculoskeletal medicine, rehabilitation medicine and physiatry.

It may well be that fibromyalgia and myofascial pain syndromes each form part of a spectrum of muscular conditions. The relationship between them is subject to debate[3, 19, 21, 34, 45, 46] and certainly they have some features in common. However, the distinction of the terms provides a functional classification of the phenomena encountered clinically and a guide to their understanding for practical purposes.

PATHOGENESIS

The mechanisms leading to the development and activity of a myofascial trigger point are still being explored. Many significant findings have been recorded but some of the ultrastructural and biochemical details are yet to be delineated. There seem to be three stages in the pathogenesis, namely the development of muscle tightness (to form a taut band), the increase in local sensitivity (at the trigger point) and the referral of pain to distant sites. These can conveniently be considered separately.

Development of Muscle Tightness

The pathophysiology of muscle tightness has been investigated by electromyography (EMG), by microscopy and by biochemical analysis.

EMG studies[47] show no electrical activity in a taut band, suggesting that it is composed of muscle fibres in a state of contracture (shortening without action potentials).

Microscopic examinations of biopsy specimens reveal something of the nature of this muscle shortening. Recent biopsy studies of muscular tender points and trigger points[19, 20, 48] have shown, under light microscopy, muscle fibres with a "moth-eaten" appearance. Under electron microscopy, papillary projections of the sarcolemmal membranes have been seen, together with narrowing of the I bands, suggesting that sarcomeres are markedly shortened (hypercontracted); in some specimens necrotic changes have been seen in myofibrils, with local accumulations of mitochondria and glycogen under the sarcolemma.

Biochemical analysis of biopsy specimens has shown unusually low tissue oxygen tension in trigger points[49] and a reduction in the ratio of high energy phosphates (such as adenosine triphosphate or ATP) to low energy phosphates (such as adenosine diphosphate or ADP)[50].

Taken together, these findings suggest that taut bands and trigger points are regions of high energy expenditure and local ischaemia. Simons has postulated[3] a mechanism involving an excessive release of calcium from the sarcoplasmic reticulum (perhaps as a result of injury), producing a maximal contraction locally (with associated high energy expenditure); in turn, the localised contraction would tend to reduce circulation locally, causing relative hypoxia and alteration of local metabolism with depletion of ATP, the very substance required by the calcium pump in the sarcoplasmic reticulum for the recovery of released calcium. Thus a viscious cycle could be established, with muscle persistently shortened and in a state of metabolic distress.

Increase in Sensitivity

The majority of afferent nerve fibres from muscle are either myelinated Aδ fibres or unmyelinated C fibres known collectively as small diameter afferents. The distal ends of these fibres act as nociceptors in muscle tissues and are sensitive to a variety of noxious stimuli, including some naturally occurring chemical compounds (which are then termed "algogenic"). Some substances have been shown to increase the sensitivity of the nociceptors, even to the degree that they cause spontaneous generation of nerve impulses without external stimulation[51]. Bradykinin seems to be the most potent sensitising agent[52], with serotonin, histamine and potassium ions having lesser potency in that order[53]. In addition, prostaglandin E_2 and serotonin have been shown to potentiate the sensitising effect of bradykinin[54].

Some or all of these substances are likely to be involved in the hypersensitivity exhibited by trigger points and very possibly others contribute as well.

Pain Referral

Pain is referred from muscles to other muscles and somatic tissues in clearly defined patterns which have been determined empirically. Each muscle has its own characteristic pattern of pain referral, mediated by its neural connections. Just which pathways are involved is a matter of some conjecture, as numerous relevant neurophysiological mechanisms have been described and any or all might contribute to the phenomena which occur clinically.

Peripheral branching of nociceptive axons is a simple mechanism whereby a stimulus affecting one axon branch is interpreted as affecting other branches which share the same cell body (and its higher connections). Such mechanisms have long been known to occur[55]. They are almost certainly responsible for many of the closer patterns of pain referral but branches have not been described anatomically to explain the more distant sites of referral.

Autonomic connections have been implicated in various ways. These include reflex vasoconstrictive mechanisms leading to ischaemia and mechanisms which affect the levels of algogenic substances at peripheral sites[56]. The role of sympathetic effector

neurons in stimulating somatic afferent fibres[57] may also be significant in autonomic pain referral mechanisms.

Central nervous system pathways involved in the transmission and modulation of pain provide numerous mechanisms of possible significance in myofascial pain referral. The models of **convergence-projection** and **convergence-facilitation** have been proposed[3] as likely explanations of clinical phenomena but further elucidation is required.

AETIOLOGY

The causes of myofascial pain syndromes include factors which act on muscle directly (causing primary muscle problems) and factors which influence muscle indirectly (causing muscle dysfunction as a secondary effect).

A primary myofascial pain syndrome may result from a single instance of excessive loading stress (overload strain), repeated lesser loadings (overuse strain), direct physical trauma, cold injury or from a disease which causes myofascial pathology.

A secondary myofascial pain syndrome may result from injuries of other parts of the musculoskeletal system, exerting their effects either by biomechanical means or by reflex neural pathways. The primary condition may be, for example, an injured intervertebral disc or zygapophysial joint or even another (primary) myofascial pain syndrome.

Perhaps the most numerous myofascial pain syndromes encountered clinically are those related to spinal dysfunction following physical injuries. The vast majority of these muscle problems are either secondary in nature or of mixed aetiology (when the initial injury has affected the muscle directly and also other structures such as joints and discs). Appreciation of the aetiological situation is of great practical importance for the treating clinician. Treatment of the myofascial problem alone will provide only short-term relief of symptoms unless the myofascial pain syndrome is purely primary in nature.

Apart from the actual causes of myofascial dysfunction there are other aetiological factors which may predispose to the development of a myofascial pain syndrome or tend to perpetuate one which is already established. These "perpetuating factors", as they were styled by Travell and Simons[30], include mechanical stresses, intercurrent diseases, nutritional inadequacies and psychological factors. Mechanical stresses represent the most significant factors numerically and include skeletal asymmetry(such as leg length difference), skeletal disproportion (e.g. relatively short upper arms), postural stresses(whether autogenous or due to external influences such as ill-fitting furniture), inappropriate activity, constriction of muscles and prolonged immobility. Intercurrent diseases of relevance include anaemia, electrolyte disturbances, hypoglycaemia, thyroid dysfunction, hyperuricaemia and chronic infections. Nutritional inadequacies which may affect muscle function include deficiences of B group of vitamins, vitamin C and of minerals such as calcium, magnesium, potassium and iron. Psychological factors including anxiety, depression, pain behaviour and secondary gain may also influence the course of a myofascial problem.

NATURAL HISTORY

Once established, a myofascial trigger point may be either symptomatic or quiescent but it will tend to persist for as long as the aetiological factors are present. If the metabolic disturbances in the muscle are severe or prolonged, dystrophic changes will occur in the region of a trigger point, converting the reactive, reversible condition to a permanent local lesion. Hence, the natural history of untreated myofascial pain syndromes is prolonged and some patients suffer chronic or recurrent pain indefinitely. By avoidance of the aggravating factors, symptoms may be minimised but trigger points do not just go away, given time, as patients are often advised. The enormous population of people with chronic myofascial pain[1, 2, 3, 4] attests to the fact that reassurance and masterly inactivity are as ineffective and inappropriate in the management of myofascial pain syndromes as they are in the management of any other active, non-self-limiting medical condition.

CLASSIFICATION

Myofascial pain syndromes and their trigger points have been classified according to their clinical manifestations and their aetiological status. The following categories are now encountered in the literature.

An **active** myofascial pain syndrome is one which is spontaneously symptomatic, with pain, tenderness and other clinical phenomena being either constant or frequently recurrent.

A **latent** myofascial pain syndrome is one in which taut band(s) and trigger point(s) have developed but are not producing clinical manifestations spontaneously. Although not apparent subjectively, latent syndromes can be detected objectively and may become active under the influence of physical stresses or other factors.

A **primary** myofascial pain syndrome is one which has arisen *de novo* as the result of factor(s) affecting the involved muscle directly.

A **secondary** myofascial pain syndrome is one due to dysfunction of other structures, with resultant biomechanical stress or reflex irritability causing the involved muscle to be affected indirectly.

Trigger points can be classified similarly. In additon, they may be described as **satellite** trigger points, if they develop because their muscle(s) lie within the zone of referral of another trigger point, or **associated** trigger points, if they develop in response to trigger points in other muscles; the associated category includes both satellite TrPs and those secondary TrPs for which the primary is another TrP.

CLINICAL FEATURES

History

The cardinal symptom of a myofascial pain syndrome is pain, which occurs in a pattern of distribution characteristic of the particular muscle involved. This pattern will include both local and referred pain, the latter often more prominent in the patient's description. For example, a patient with a sternomastoid pain syndrome may present with troublesome frontal headaches, with neck soreness being a relatively minor associated complaint. Myofascial pain is typically of a dull, aching quality and recurrent in periodicity: patients often report that some degree of pain is present most of every day, with exacerbations related to activities. Aggravating factors include facilitation of the muscle involved (especially strenuous or repeated contractions), prolonged periods of muscle shortening (the pain may be worse on rising if the muscle is held in a shortened position during sleep), passive stretching of the muscle, external pressure on the trigger point and exposure to cold. Relieving factors include short periods of rest and applications of heat.

Other symptoms sometimes reported are subjective sensations of weakness and stiffness, with inability (or at least disinclination) to perform certain movements and activities; further investigation often shows that these are learned responses developed by the patient (generally quite unconsciously) to avoid exacerbations of pain. Proprioceptive symptoms may also occur; they range from unsteadiness or inaccuracy of movements involving the affected muscle(s) to frank dysequilibrium with postural dizziness and even ataxia if the muscle(s) involved have significant postural functions. Occasionally, apparently bizarre symptoms such as intermittent dysmetria (disturbances of the perception of the weight of objects held in the hand) are reported, although they may only be discovered on specific questioning as patients tend not to volunteer what they consider strange behaviour, such as the dropping of objects or intermittent clumsiness whilst performing familiar manual tasks. These problems are readily appreciated and explained when the proprioceptive roles of the neck, shoulder girdle and arm muscles are understood.

Autonomic symptoms are common in myofascial pain syndromes[58], although they are usually of only minor concern compared to the pain. Pupillary reflexes, lacrimation, nasal discharge, sweating and heart rate may all be affected, usually with a relative sympathetic dominance, and the patient may suffer nausea, anorexia and other gastrointestinal symptoms.

The onset of a myofascial pain syndrome may be sudden, if related to a specific injury such as in a motor vehicle accident, or more gradual if the muscle has been repeatedly stressed over a prolonged period. When the myofascial problem is secondary to (say) a spinal joint injury, as is commonly the case, the symptoms of the myofascial pain syndrome may develop gradually some weeks, months or even years after the initial injury. In these circumstances, the superimposition of the myofascial symptoms on those due to the intraspinal dysfunction may result in a very complicated history which the clinician will need to unravel with great care if the contributions of the various factors are to be appreciated fully.

Sometimes the referred symptoms of a myofascial pain syndrome may mimic those of other conditions and in an age of medical specialisation the patient may be investigated and treated in inappropriate ways before the myofascial nature of the problem becomes apparent. To take the example of the sternomastoid again, a patient with an active trigger point in the clavicular division of the muscle may suffer headache, earache, postural dizziness and nausea; he may be investigated extensively (and often even treated on a trial basis) for months or years before the problem is determined, by exclusion, to be myofascial rather than of E.N.T. or neurological origin. Obviously the reverse situation could and probably does also occur. The overall chance of such mis-diagnosis would be lessened considerably if the features of myofascial pain syndromes were taken into account more often at the stage of the initial clinical assessment.

Physical Examination

Muscle shortening is the first objective physical sign elicited in the assessment of a patient with a myofascial pain syndrome. The shortening may be apparent on inspection, either by direct visualisation or by observation of the postural effects of the length change. Testing the ranges of movement of the associated joints will provide further evidence, particularly resisted active movement tests, and the presence of muscle shortening can be confirmed by palpation.

The presence of a taut band within the muscle is the next sign to be sought. A taut band usually lies in the long axis of the muscle and is readily palpable as a cord of tighter fibres which feels firmer than the rest of the

muscular tissue. The muscle should be placed in a position of slight stretch (compatible with the patient's comfort) and palpated either by gentle compression against underlying bone or by gentle squeezing between the examiner's fingers and thumb. As the palpating fingers move over the muscle surface a taut band will be felt to roll beneath them.

Trigger point tenderness can be elicited when a taut band is detected. The examiner should move his finger along the band, slowly and cautiously, and a trigger point will be manifest as a point of marked, often exquisite, tenderness. The identification should be made as quickly as possible (without compromising accuracy) and care taken not to apply either excessive or prolonged pressure, as the examination can be quite uncomfortable for the patient.

A local twitch response may be observed when a trigger point is palpated. The response is due to a momentary contraction of the taut band and is best detected by a second palpating finger placed further along the taut band. Sometimes it may be seen as a slight twitch most visible close to one of the muscle's attachments. The local twitch response may also be evoked by insertion of a needle into a trigger point[36]. Electromyographic studies[59] have shown that the response is a brief muscular contraction, lasting for approximately 20-75 milliseconds.

The jump sign[60] is a more pronounced involuntary flinching than the local twitch response and is not to be confused with it. Stimulation of a trigger point by digital compression causes a sudden flinching of the body part involved, or even of the whole patient. The sign provides a guide to the presence of a trigger point and a useful means of confirming its location; the same stimulus applied to other parts of the muscle will have no such effect.

Reproduction of the patient's symptoms is one of the most significant results of physical examination. Trigger point stimulation will produce pain both locally and in the characteristic pattern of referral. The patient will recognise immediately which symptoms are related to that trigger point and if these correspond to those which have been troubling him the relationship between cause and effect is established. Even though the test procedure is inherently uncomfortable, the reproduction of the typical pain is usually very reassuring for the patient, especially when the symptoms have been present for a prolonged period without adequate explanation. The ability of the practitioner to identify the source of the symptoms so accurately can be the first positive step towards the resolution of what might have hitherto seemed to be a problem without answers.

Weakness can be sought by specific tests of power and the process is simplified when the involved muscle is known. Subjective reports of weakness often are not confirmed by objective testing, as the subjective feeling is frequently due to incomplete facilitation (so as to avoid pain) rather than to an actual loss of strength. Muscular wasting is not generally found.

A compression test described by Travell and Simons[30] may be a useful clinical adjunct when a myofascial pain syndrome is symptomatic during movement but not at rest. If the involved muscle is compressed between the examiner's fingers the offending movement can sometimes be performed without referral of pain. The same inhibitory effect may also be achieved by firm squeezing of a roll of skin overlying the muscle.

The combination of these signs provides a reliable indication of both the nature and the site of the causative condition. Whilst some might argue that there is a large subjective component in the elicitation of signs such as the jump sign and the reproduction of symptoms, comparisons of responses to palpation of a trigger point and of other parts of the same muscle will usually provide the clinician with convincing evidence of the presence or otherwise of a specific myofascial problem.

Attempts have been made to quantify the amount of pressure applied to muscles during clinical assessment so as to gauge the relative degree of tenderness at different points. Various types of pressure recording apparatus have been modified to produce what are termed pressure algometers and these have been shown[34, 61, 62] to provide reliable measurements of the applied pressure. However, the use of such devices is not necessary for satisfactory clinical assessment of patients, as the qualitative aspects of response to palpation are of much greater significance than the quantitative ones.

ANCILLARY TESTS

Phenomena associated with myofascial pain lend themselves readily to clinical methods of assessment and ancillary investigations have little to add to the diagnostic process in practice. Blood test findings are essentially unaltered by the presence of myofascial trigger points, although one study[63] has shown relatively small changes in the serum concentrations of lactate dehydrogenase iso-enzymes. Similarly, electromyographic examinations of involved muscles contribute little in the way of specific positive findings. Radiological studies such as plain X-rays and computerised tomographic scans also do not show myofascial changes. Sophisticated imaging techniques based on nuclear magnetic resonance may do so; however, their use is simply not justified for practical and economic reasons.

Thermography, based on changes in skin temperature, reflects underlying circulatory changes and hence autonomic activity. Thermographic studies have shown characteristic patterns in the presence of trigger point activity, with disc-shaped "hot spots" 5 to 10 centimetres in diameter over the sites of trigger points and increased heat emission in the regions of pain referral[62, 64, 65]. Modern thermographic techniques are non-invasive and quite inexpensive to perform. Whilst their use is unnecessary for the diagnosis of myofascial pain syndromes, they may be useful to provide corroboration of myofascial phenomena detected clinically.

MANAGEMENT

Diagnosis of a myofascial pain syndrome, and its identification as the cause of the patient's symptoms, provides a specific target at which management strategies can be directed.

The first step is the formulation of a management plan, based on an appreciation of the role of the myofascial dysfunction in the overall scheme of the patient's problems. Since most myofascial pain syndromes are secondary in nature, consideration must be given to dysfunction in other structures (such as intervertebral discs and zygapophysial joints) which may be primary causes of the myofascial condition, as well as to other problems which may be secondary to the myofascial pain syndrome itself. Treatment of the cause(s) and other sources of symptoms is essential if the patient is to experience other than partial and temporary relief from the treatment of the myofascial dysfunction.

Many modalities are available for the treatment of myofascial problems and as in all areas of medicine the treatment of choice will be the least invasive method(s) capable of producing a satisfactory outcome in the patient's particular circumstances. The range of choices includes rest, patient education, therapeutic exercise, heat modalities, manual therapy, vapocoolant spray and stretch, trigger point injections (and stretch), oral medications, neuromodulation (including TENS, acupuncture and biofeedback), psycho-social management and rehabilitation. Various combinations of these are usually employed.

Patient Education

Perhaps the most important part of any management programme is the explanation to the patient (in understandable terms) of the nature of the causative problem(s) and the factors which are likely to have some bearing on the outcome. In the case of a myofascial pain syndrome, the patient should be made aware of the mechanisms which cause the pain and the relatively benign but inherently persistent nature of the underlying pathology. Patients often tend to equate severe symptoms with severe disease and some will require reassurance that the problem is not due to a neoplastic disorder or an advanced state of (irreversible) degeneration. The practitioner should explore the patient's beliefs about the condition and tactfully correct any misconceptions (specifically, many a patient with myofascial pain harbours secret and very genuine fears about loss of independance and being confined to a wheelchair). The patient should be given a realistic prognosis of the condition and encouraged to take an active role in self-management, based on an understanding of the factors which he can control. Goals should be set by agreement between the patient and the practitioner and progress towards them monitored at appropriate intervals.

Rest

In this context rest does not mean prolonged immobilisation (which may actually make the problem worse, as already explained) but rather the specific avoidance of mechanical stresses which are likely to aggravate the condition. In many cases the patient may continue working but the work activities should be modified so as to avoid excessive or repeated loading of the muscle(s) involved. The practitioner should give the patient specific instructions, and provide a detailed certificate for the employer, based on the biomechanical factors relevant to the particular circumstances. Similar advice should be given in relation to activities of a non-occupational nature.

Therapeutic Exercise

Exercise has a crucial role to play in the correction of any musculoskeletal disorder. Restoration of function can be facilitated, and hastened considerably, by appropriate exercise and it can be inhibited just as easily by inappropriate exercise. The patient should be taught to develop a concept of exercise which recognises its importance and which includes, in the general sense, all physical activities and, in the specific sense, special manoeuvres designed to affect a particular muscle or muscle group.

Patients with myofascial pain syndromes frequently obtain relief from exercise which encourages stretching and relaxation of the muscles involved. In the specific sense, exercises based on the post-facilitation inhibition (or muscle energy) techniques as described by Lewit[66] and others can be extremely helpful.

The contributory effects of synergists and antagonists should also be taken into account and, if appropriate, some strengthening exercises may be required as well to restore muscle balance.

Particular exercises must be prescribed carefully, with due consideration of the patient's individual biomechanical needs and other circumstances. The programme should be kept as simple as possible, with the minimum number of exercises being prescribed at once so as to avoid confusion. The techniques should be demonstrated precisely and practised by the patient to the practitioner's satisfaction before being undertaken without supervision. The patient should be given a written description of the techniques set out in sufficient detail as to provide a practical guide. Possession of such instructions will also encourage compliance with the exercise programme.

Exercises which are not prescribed specifically may be ineffective or even counter-productive. Those printed on handout leaflets implying that they are suitable for all patients with problems in a particular region will inevitably exacerbate some patients' conditions[67]: they should not be used. Similarly, the advocacy of particular styles of exercise for particular types of symptom, without reference to the patient's individual biomechanical circumstances, is both irrational and dangerous.

Heat

Muscles respond to heat by relaxation and lengthening, and to cold by contraction and shortening. It comes as no surprise that the pain associated with the shortened and tightened state of muscle tissue in a myofascial pain syndrome is usually aggravated by cold and relieved by heat.

Heat may be administered in numerous ways. In the domestic situation, the warm shower or bath, the electric radiator and the ordinary clothes iron (applied with care over a thick, dry towel) are some of the most convenient sources. Special heating devices can also be purchased. More sophisticated heat modalities such as short wave diathermy and ultrasound are suitable for use in a professional therapeutic setting.

Maintenance of body heat is a simple but important measure which can markedly reduce the frequency of myofascial symptoms, especially in colder climates. The wearing of appropriate clothing can be augmented by the use of thermal supports (made of wet-suit material, Chloroprene), which are remarkably effective in maintaining the warmth of the body part enclosed.

Manual Therapy

Taut bands, and the tight muscles which contain them, are amenable to those forms of manual therapy which stretch and relax soft tissues. The gentle, passive, soft tissue stretching techniques used and advocated for decades by many medical practitioners, physiotherapists and osteopaths probably owe much of their efficacy to the reduction of tension in taut bands. The more recent muscle energy techniques, employing post-facilitation inhibition manoeuvres[66] are also very effective and have the added advantage that modified procedures based on the same principles can be taught to the patient as specific inhibitory exercises for self-treatment.

Both these types of treatment are essentially painless when performed skilfully and provide relief of symptoms in a relaxed, comfortable manner.

Trigger points, because of their inherent tenderness, are less amenable to direct manual therapy but the techniques of ischaemic compression and friction massage are described as having some beneficial effects, probably through the mechanism of reactive hyperaemia. These techniques are painful for the patient, even when performed by experienced operators, and are not recommended. They are included here for the sake of completeness.

Vapocoolant Spray and Stretch

The application of a vapocoolant substance by spraying on the skin surface over an affected muscle, followed by passive stretching in the long axis, is one of the most effective forms of treatment of myofascial pain syndromes.

The effect of the vapocoolant was discovered by Kraus[68] and developed by Travell and co-workers[69] in the 1940s. Originally the vapocoolant substance used was ethyl chloride[70] but because of its toxicity (when inhaled) and its potential explosiveness (when mixed with air) a mixture of chlorinated fluorocarbons was substituted. This is still in use but because of the now-recognised hazards of environmental pollution and depletion of the ozone layer of the earth's atmosphere[71, 72] other alternatives are being sought and at least one non-halogenated hydrocarbon preparation is now on the market in Australia.

The technique recommended by Travell and Simons[30] involves placing the patient in a position that is both comfortable and will permit passive stretching of the affected muscle. The patient should be reassured and the cold nature of the stimulus should be explained to him. The vapocoolant container is then held with its nozzle at an angle of about 30° to the skin surface and approximately 45 centimetres from it. The jet is activated and the vapocoolant applied by slow (10cm/sec.), unidirectional sweeps parallel to the taut band, in the direction of the zone of pain referral and just far enough apart so as to cover the whole surface of the muscle and the referral zone; three or four sweeps is usually sufficient. The muscle is then passively stretched in its long axis, within the limits of the patient's comfort. The skin should then be re-warmed to prevent cold injury. The procedure may be repeated two or three times in a treatment session and full passive stretch will generally be achieved. The patient should then be able to perform a full active contraction of the muscle without suffering referred pain.

When properly performed the procedure is not uncomfortable; its effects are immediate and often dramatically successful, at least in the short term.

Trigger Point Injections

Injection of trigger points is a treatment modality combining physical stimulation with pharmacotherapy.

The physical impingement of the needle alone exerts some beneficial effect and the technique of "dry needling" has been advocated[73, 74] as a useful form of treatment. Possible explanations are that the needle physically disrupts the contracture of the trigger point and/or that it cuts nerve fibres responsible for a reflex mechanism. It has also been postulated that the needle initiates the release of vasoactive amines or other chemically active substances.

The injection of a **local anaesthetic** agent adds greatly to the therapeutic effect. When accurately placed, the anaesthetic blocks afferent pathways from the trigger point and abolishes associated pain phenomena. It also causes local vasodilatation, relieves local ischaemia and breaks the viscious cycle implicated in trigger point pathogenesis. The agents most often used are procaine or lignocaine, in lower concentrations (0.5% or 1%); higher concentrations are unnecessary for the effects sought and they increase the risks of complications. Procaine is preferred by some practitioners because it has not been associated with inflammatory and degenerative changes in muscle, as has lignocaine in a 2% concentration[75]; however, these muscle changes are reversible[76] and do not occur with the lower concentrations of lignocaine[77]. Lignocaine is preferred by many as it is more potent than procaine, has a longer duration of action and is less often associated with allergic reactions[78]. Whichever anaesthetic agent is chosen, it is always used without adrenaline to avoid the risk of intravascular injection of that agent.

The injection procedure involves placing the patient in a comfortable recumbent position (so avoiding problems associated with vasovagal syncope which some patients suffer with any injection) and locating the trigger point. The overlying skin is prepared with a suitable antiseptic and a "no touch" aseptic technique is observed at the injection site. The taut band is steadied by the practitioner's non-dominant hand and the needle is introduced into the muscle. As the trigger point is pierced a local twitch response may be observed and the patient may experience a momentary exacerbation of pain. The plunger of the needle should be withdrawn slightly, to check for blood, and then 2-5mls. of local anaesthetic is injected in and around the trigger point. The needle is then withdrawn and the injection site covered with a disposable dressing.

A small amount of **corticosteroid** solution is added to the injection by some practitioners to counteract the post-injection inflammatory effects described experimentally[77]. Many prefer not to use steroids in this way because they consider that the consequences of post-injection inflammation are slight compared to the unwanted effects of injected steroids.

The injection of a non-steroidal anti-inflammatory drug, **diclofenac**, has been reported as having benefits superior to those of lignocaine[79]. The effect is presumably due to the prostaglandin inhibiting action of diclofenac and reflects the role of prostaglandin of the pathogenesis of the condition.

When trigger points have been injected successfully the muscle can be stretched passively without exacerbating pain. Post-injection stretching should be considered part of the procedure, which is sometimes referred to as "injection and stretch".

The pain relief which follows trigger point injections usually lasts much longer than the pharmacological duration of action of the agent injected. Sometimes it will last for days or weeks and sometimes it is permanent. Clearly the effect of the procedure is more than pharmacological and involves reversal of the pathophysiological processes of myofascial pain generation.

Oral Medication

Oral drug therapy has a place in the management of myofascial pain syndromes and three classes of drugs are employed.

Muscle relaxants inhibit the contraction which contributes markedly to the generation of pain from tight, irritable muscles. The most effective of these drugs is diazepam[80] and it may be useful for short periods to assist in symptom relief.

Non-steroidal anti-inflammatory drugs, such as diclofenac, piroxicam, etc., have anti-inflammatory, analgesic and antipyretic properties. Their prostaglandin inhibiting effects suggest that they have a specific role in the treatment of myofascial trigger points and they are useful in the management of myofascial pain syndromes, particularly as adjuncts to the use of physical modalities of treatment.

Analgesic preparations are commonly prescribed

for symptomatic relief. Although their overuse may lead to problems of dependency, their availability often makes the difference between the patient's situation being tolerable and intolerable in the period before definitive treatment is begun.

Neuromodulation

Modulation of pain occurs naturally in the complex neural pathways of the central nervous system, through the interplay of excitatory and inhibitory mechanisms[81]. These mechanisms can be influenced by the selective application of additional stimuli which augment the natural inhibitory processes.

Transcutaneous electrical nerve stimulation (TENS) employs an electrical stimulus with specific characteristics to achieve this effect. Electrodes are attached to the patient's skin in appropriate positions (over a peripheral nerve) and a current is passed between them from a battery operated output device known as a TENS machine. This method of treatment is designed for prolonged or continuous use and the apparatus is compact and easily portable. The patient wears the TENS machine on his belt and can operate small switches to control the characteristics of the electrical pulse produced by it. TENS has been shown to be an effective form of treatment for myofascial pain syndromes[82]. As it is a method of pain control rather than a means of effecting cure, it is best used when more definitive forms of treatment are inappropriate. It is particularly useful as an alternative to the prolonged administration of analgesic medications.

Acupuncture is the application of a physical stimulus, a needle prick, at specific sites called acupuncture points. It also achieves its effect by the augmentation of natural inhibitory mechanisms. When applied at the sites of trigger points it has the additional effect of the physical impingement of the needle on the microstructure, similar to the effect of dry needling.

Biofeedback involves the use of conscious, frontal lobe cerebral mechanisms to modify bodily processes which usually occur unconsciously. It can be an effective means of pain modulation when the patient is well motivated, has the necessary sense of mental discipline and is properly trained in its use.

Psycho-social Management

As well as providing the specific physical and chemical modalities of treatment, the practitioner should be aware of the psycho-social aspects of the patient's problem and should provide appropriate advice and assistance. A few words of reassurance will often go a long way towards allaying the anxiety which is a natural concomitant of the unpleasant experience of severe and/or chronic pain. Simple explanation can relieve patients' fears, especially when those fears are based on false beliefs about a condition and its prognosis. Some patients will benefit from more formal types of treatment such as relaxation therapy and the advice of a suitably qualified clinical psychologist may also be helpful. The psycho-social aspects of the patient's situation should be kept in perspective and the patient should be encouraged to see them as natural sequelae of the physical processes rather than as manifestations of psychological dysfunction. Patients with essentially physical conditions should not be referred to psychiatrists; apart from being simply inappropriate, such referrals tend to undermine the patient's belief system and to create the impression that the pain is at least partially due to some inherent psychiatric disorder.

Litigation is commonly associated with musculoskeletal injuries, especially when they occur at work or in motor vehicle accidents. Prospects of financial compensation add an extra factor to what may already be a complex psycho-social background. The practitioner should evaluate his own role in the situation. He should accept that involvement in litigation is a necessary part of managing the patient's problem but should take care to avoid bias either in favour of or against any party to the legal proceedings. His proper medico-legal function is to express the pertinent medical facts in a scientifically objective manner so as to provide data on which legal decisions can be based. Whilst maintaining an empathetic attitude towards the patient, he should avoid being cast in the role of the patient's legal ally and the patient should be informed accordingly.

Rehabilitation

Restoration of normal function is the objective of all treatment programmes. Whether it can be achieved or not, the patient will require assistance in resuming former activities in an appropriate manner. Occupational therapists and other rehabilitation providers can be extremely helpful in this regard and the practitioner should co-operate with them, and with the patient, to form a rehabilitation team. Ideally, the team should also include the patient's employer.

CONCLUSION

Myofascial pain syndromes are extremely common and are significant causes of morbidity and disability. Their incidence is perplexing in the light of the present state of scientific knowledge of the subject and can only be interpreted as reflecting ignorance, or deliberate disregard, of the established facts by large numbers of medical practitioners. When viewed rationally, the syndromes are seen as straight-forward forms of musculoskeletal dysfunction with readily understandable physical causes and effects.

Diagnosis depends upon an awareness of relevant clinical phenomena, a knowledge of pertinent anatomy and the ability to perform (and to evaluate the results of) simple clinical tests. Sophisticated ancillary investigations are not required and are contra-indicated on economic grounds.

Once the diagnosis of a myofascial pain syndrome is established, definitive management can be started. A wide variety of treatment modalities may be used and most are readily applied in the consulting rooms. In fact, at least some degree of pain relief should be obtainable at the patient's first visit. Continuing treatment, including self-treatment, will generally be required for some weeks before the condition can be considered to be in permanent remission but the treatment requirements are not onerous and are readily accepted by most patients who prior to diagnosis were suffering from chronic pain of uncertain origin and doubtful prognosis.

Most myofascial pain syndromes are secondary to other problems. Detection and treatment of the myofascial component does not necessarily bring the patient's problems to an end. However, it does significantly reduce the burden of symptoms and enables a clearer understanding of the nature and role of the primary problem(s), such as intervertebral disc injuries and zygapophysial joint dysfunction, which should then be easier to manage.

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Foot Arch Manipulation

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ABSTRACT

Mechanical dysfunction of the foot is manifest as pain, flattening of the longitudinal and transverse arches, over-pronation and the formation of calcaneal spurs. Careful clinical assessment will often reveal the nature of the problem and the biomechanical mechanisms involved.

Manipulative techniques may be used to correct the situation and compound orthotic devices provide support in the corrected position.

INTRODUCTION

Peripheral mobilisation and manipulation are manual techniques that have been practiced down through the ages of recorded medicine. As a general principle neither mobilisation nor manipulation should be carried out under anaesthesia unless there are mitigating circumstances. Peripheral mobilisation is a most useful tool in the treatment of many joint conditions and certain physical problems lend themselves ideally to manipulative techniques. The flattening of the foot arches are one such condition.

BASIC ANATOMICAL CONSIDERATIONS

The foot is a complex structure of 26 bones that has two fundamental design functions:
- To support the body weight
- To transport the body

The bones are divided as follows:
- 14 phalanges
- 5 metatarsals
- 7 tarsals

Three functional segments can be distinguished.

Posterior segment

This lies directly beneath the tibia and supports it. It is composed of the talus and calcaneus. The talus not only supports the tibia but is capable of pivoting with a screw-like motion as it articulates with the calcaneus. As the foot flattens the talus rotates medially. This action means the whole leg also rotates as the arch flattens.

Middle Segment

The three cuneiforms and the anterior part of the cuboid form a row with the navicular and the posterior aspect of the cuboid behind.

Anterior segment

Contains the five metatarsals and the fourteen phalanges.

THE ARCHES OF THE FOOT

The longitudinal arch is composed of medial and lateral parts. The medial part is higher and the more important. Functionally, both parts act as a unit with the transverse arch to spread weight in all directions.

The medial arch is composed of the calcaneus, talus, navicular, three cuneiforms, and three metatarsal bones. The head of the talus is located at the summit of the arch and acts as the key stone.

The lateral longitudinal arch rests on the ground and is composed of the calcaneus, cuboid, and the lateral two metatarsals.

The transverse arch is composed of the cuboid, the three cuneiforms, and the bases of the metatarsal bones. The medial and lateral aspects of the longitudinal arch act as pillars for the transverse arch and the peroneus longus muscle helps maintain the curvature of the arch.

The integrity of the arches is maintained by:

a. the shape of the interlocking bones, (and therefore, by definition, their placement).
b. The strength of the plantar ligaments and the plantar aponeurosis.
c. The action of the muscles through the bracing action of their tendons.

Of the three factors mentioned, the plantar ligaments and the fascia are the most important in maintaining the arches when standing.

CLINICAL PRESENTATIONS

Flattening of the foot arches produce problems that frequently present in one of three prominent groups.
1. Metatarsal head pain.
2. Calcaneal spurs (ventral) and plantar fasciitis.
3. Over-pronation of the foot.

Flatfoot is clinically diagnosed if the medial border of the foot is in contact with the ground on weight bearing. The presentation of flat foot is quite common and when symptoms are present it warrants treatment. In a number of cases flat feet are observed on the examination of patients who present with low spinal problems. In this situation both conditions are often interlinked and both may require treatment. Some presentations of flat feet do not require treatment, e.g. in infants where there is found a pad of fat beneath the arch. Similarly, "knock-knees" in children cause a shift in the centre of gravity and can produce weight bearing on the medial margin of the foot.

If a non-weight-bearing foot has adequate arches that disappear on standing it usually implies a laxity in the support ligaments.

Examination should be carried out to exclude a rigid flatfoot. This is a pronated foot with an inflexible depressed arch. Possible causes are:
- soft tissue contracture
- articular damage
- unreduced fracture
- dislocation
- bony ankylosis

Here final diagnosis may require radiology, C.T. scan or isotope studies. A presenting rigid flatfoot should rarely be manipulated and never before full investigation.

METATARSAL HEAD PAIN

When the transverse arch of the foot flattens, the heads of the metatarsals come into contact with the ground as the physical width of the foot increases. The most common cause for this is the loss of tissue resiliance with increasing age. Obvious foot trauma, both accidental and to a lesser degree over-use, and congenital factors are other participants. It is more often the case than not that the longitudinal arch also tends to flatten, thus increasing the overall length of the foot. Therefore, in considering a clinical approach, both arches must be considered.

CALCANEAL SPURS

The aetiology of spur formation may be debated, but the following explanation is based on other areas of body reaction to chronic inflammation, e.g. rotator cuff lesion. It is likely that with the collapse of the longitudinal arch, and thus the lengthening of the foot, stress is placed on the insertion of quadratus plantae, abductor hallucis and the ligaments supporting the arches. The pain factor is most definately related to these stretch phenomena. With this stretch there develops chronic inflammatory changes and then eventually calcification occurs. Eventually a radiological spur is formed. Indeed, the heel spur syndrome is a catch-all term which describes plantar fasciitis, myositis of the abductor hallucis and periositis which occurs at the attachment of the muscles and fascia to the calcaneus.

Current orthopaedic treatment of spurs appears to be directed at the "end result" rather than the aetiology. Injections of steroids and local anaesthetics do not alter the anatomical aetiology although short term pain relief can be obtained. Similarly, surgical removal of the spur is not correcting the underlying fault.

Restoration and maintaining the foot arch will shorten the foot and thereby remove the stretch influence responsible for the inflammatory response. Pain is also then abolished.

OVER-PRONATION

This problem often occurs in athletes, especially long distant runners. They tell of foot pain that interferes dramatically with their training. Examination of the patient should always include foot inspection, gait observation, both walking and running, and examination of their sporting footwear. In over-pronation situations the heel wear is excessive on the lateral posterior side. In extreme cases the lateral side of the shoe sole is also well worn. In all lower limb problems in athletes careful inspection of shoes is essential. A runner's shoe is a "finger print" of his style. In this situation two problems are frequent culprits. Arch collapse can exaggerate style and lead to over-pronation and ankle joint problems can significantly alter the foot strike, again leading to an exaggerated style. Being able to lift the longitudinal arch if often all that is required to overcome the problem.

MANIPULATION CONSIDERATIONS

There are four basic regions to consider when manipulating the foot. These are:

1. The calcaneus
2. The talus
3. The transverse arch as a single unit
4. The longitudinal arch as a single unit.

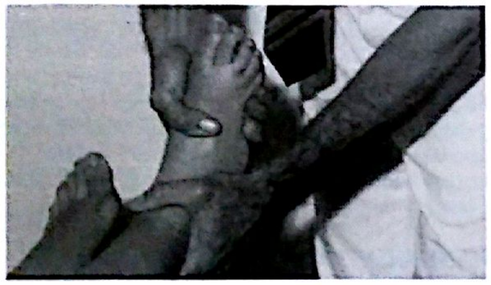
The first two are precise single bone movements and the third and fourth are positioned and manipulated as block areas. While one can adjust single bones in the latter two regions, this is not necessary to achieve excellent results. Before proceeding further, it must be emphasised that correction of structural problems by manipulation is a waste of time if adequate orthotic support is not provided. It is a simple matter to re-position bones and therefore realign arches, but due to the original aetiology, the newly positioned bones will not remain in place Once in the vertical position and

weight bearing the effect of gravity will once more flatten the arches. Thus as a prelude to manipulation the patient should be referred to a podiatrist for non-weight bearing moulds of his feet and subsequent orthotic arch supports. Following this, manipulative procedures are carried out. It is advisable to repeat this treatment weekly for three weeks and then follow it up a month later and again at six months. Further review should be after another six months, or as the patient deems necessary.

MANIPULATION TECHNIQUES

Talus

In a pronated situation the talus is usually displaced laterally. It is readily repositioned by exerting a traction thrust against its lateral aspect. The patient is placed in the supine position. The doctor's thenar eminence contacts the lateral border of the talus while the rest of the hand assumes a natural position around the heel, (viz.Fig. 1 and 2). The other hand then encircles the longitudinal arch. Both hands exert traction to eliminate all joint slack. A sudden traction pull is exerted while the talus is simultaneously thrust medially. Frequently an audible release sound is heard, but as with all manipulative proceedures, this is not necessary for a successful correction.



Figs. 1 and 2
Manipulation of the talus is carried out by one hand encircling the longitudinal arch and the thenar eminence of the other hand being placed in contact with the lateral aspect of the talus. Both hands exert traction to take up all slack. A sudden traction pull is then exerted while the talus is simultaneously thrust medially.

Calcaneus

When the calcaneus moves forwards, the longitudinal arch of the foot tends to increase in height and thus the overall length of the foot tends to shorten. This serves to relieve stretch forces acting on calcaneal attached muscles and ligaments. To achieve this, the patient lies in the prone position and his knee is flexed to 45°,(viz. Fig. 3). The dorsal aspect of the foot is then supported with one of the operators hands while the other hand grasps the calcaneus. The manipulation is carried out by a forward thrust of the upper hand toward the lower one. This is the most difficult of the foot manipulations to carry out successfully, due largely to a thrust type force rather than a traction orientated one. Persistance is required to master this technique but the effort is well worthwhile.

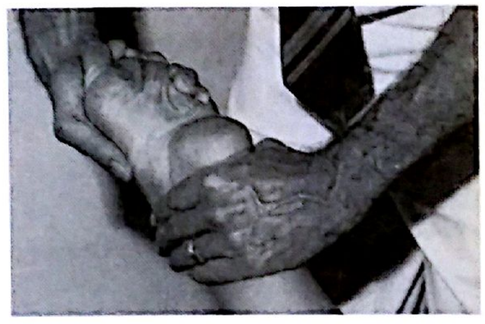


Fig. 3
Calcaneal manipulation is carried out by supporting the dorsal surface of the foot with one hand and grasping the calcaneus firmly between the index finger and thumb of the other hand as it wraps around the heel. Both hands are then thrust toward each other to complete the adjustment.

Arches

A "block" technique is the most useful approach to correcting the anatomical position of the tranverse and longitudinal arches. As stated previously, both arches are usually involved together. Therefore it follows that they should be able to be corrected simultaneously. In practice this is so. The patient is in the prone position, again with the knee flexed to 45°. Standing on the lateral side of the patient the doctor places one hand under the dorsum of the foot, (viz. Fig. 4), then squeezes both margins of the foot toward the midline, at the same time pushing downwards with the thumb of the other hand to increase the gutter effect. This over-compensates the transverse arch. This position must now be maintained firmly. Next, the free hand is placed around the heel and both hands are moved toward each other, (viz. Fig. 5). This will increase the height of the longitudinal arch. The operator should make absolutely certain that all slack is taken up before proceeding. Manipulation is then carried out by moving both hands toward each other in a short sharp action. A loud "crack" sound is usually heard. Without changing sides, the same technique is used to treat the opposite foot. Having done this the practitioner moves to the other side of the patient and manipulates the two feet again. Obviously the hands need to change positions. In other words, each foot is treated twice, once from either side of the patient.

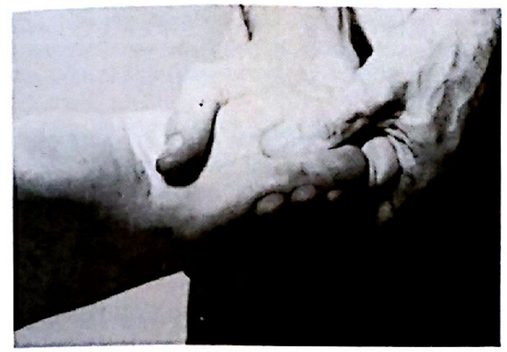


Fig. 4
Standing at the side of the patient place the proximal hand beneath the dorsum of the foot, squeeze both margins of the foot toward each other and at the same time push down in the mid-line with the thumb of the other hand to increase the guter effect. Maintain this position firmly before continuing.

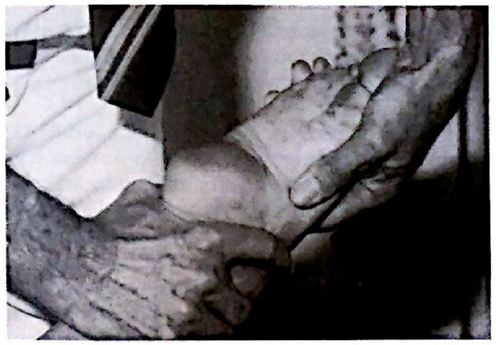


Fig 5.
After forming a gutter at the level of the transverse arch, place the free hand around the heel. Now move both hands toward each other to take up all slack. Adjustment is then carried out by moving both hands toward each other in a short sharp action.

BIOMECHANICAL ORTHOTIC DEVICES

Today's orthotics may ressemble the old fashioned "arch support" but there any similarity ends. True biomechanical orthotic devices do not simply support the arch. Plaster casts are taken to ensure the best functioning position of the foot. The body of the orthotic is contoured over this model. Then additional components called post controls are added to the front, rear or both ends of the body. The compound orthotic is then ground to specific angles in a similar method to spectacle lenses. This then means the orthotic devices create a more normal function of the feet and legs by maintaining the anatomical angular relationships between the segments of the foot and leg while the control posts allow the orthotic device to move into specific positions at specific times in the walking cycle. The effect is to control abnormal motion without restricting normal motion.

ADDITIONAL PROBLEMS OF FLATTENED ARCHES

As the foot arch collapses the talus internally rotates and the whole leg follows anatomically. Excessive rotation produces posterior thigh muscle stretch which in turn tends to tilt the pelvis forwards. Since neither standing nor walking in this position is comfortable, the body responds by "straightening up". This is achieved by increasing the lumbar lordosis. Then the spine compensates by increasing both the thoracic and cervical curves. This increase in overall curvature can mean added tension coming to bear on the relevant zygapophysial joints. In the stress situation there is an increased likelihood of joint dysfunction occurring. Additional long term malalignment can result in excessive wear of the articular surfaces. With patients presenting with spine symptoms it is important to check foot arches. Where flattening of arches has occurred, correction of this problem will also result in less likelihood of recurrence of the spinal problem once it has been treated.

SUMMARY

The above manipulative techniques are easily learnt and together with correct orthotic shoe inserts will greatly benefit the welfare of most patients. In those suffering severe pain the addition of short term NSAIDs is also a useful adjunct. Treating in the above manner has negated the necessity for steroid injections and surgical intervention for plantar fasciitis and spur formation.

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Applied Kinesiology, David Walther.

Subluxation and Locking of the First Rib: A Cause of Thoracic Outlet Syndrome

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CASE REPORT

A 30 year old man presented with a history of right shoulder pain. The pain was of sudden onset, while he was watching television at the end of a normal working day. His regular job involved manipulating a hose from a moving tractor; this required him to lift and rotate his right arm upwards and backwards continually to pull and release the hose.

Initially the pain was not accompanied by sensory symptoms. Movements of the arm were painful, sleep was disturbed and the pain was severe enough to require a narcotic injection.

By two weeks after the onset of pain he had developed symptoms of a "burning" sensation down the right arm, "weakness" and "heaviness" of the arm. Oral non-narcotic analgesia did not provide relief from the symptoms.

Examination showed him to be an otherwise fit man. He had a full range of shoulder movements except for some restriction in internal rotation.

Painful restriction of neck movement was found, especially lateral flexion away from the side of pain. Adson test was positive in the right arm with reproduction of symptoms and obliteration of the radial pulse. The test was negative in the left side. There was tenderness over the right first rib and coracoid process. Brachial plexus tension test produced elbow pain. Neurological examination of both upper limbs was normal.

Plain cervical spine and chest X-rays were normal with no evidence of a cervical rib. E.M.G. was organised but was unable to be performed prior to his recovery. The working diagnosis was of a thoracic outlet syndrome.

Manual therapy consisted of friction massage to pectoralis minor and mobilising the first rib above the clavicle in a cranio-caudal direction. In one of the sessions, there was a sudden "give" of the first rib with immediate relief of symptoms. Adson test was repeated and this was negative.

DISCUSSION

Thoracic outlet syndrome refers to symptoms of pain and paraesthesiae in the neck, shoulder and upper limb due to compression of the neurovascular bundle at the cervico-thoracic dorsal outlet. The neurovascular bundle comprises the brachial plexus, subclavian artery and vein.

Known causes of thoracic outlet syndrome include cervical rib, or fibrous band, scalenus anterior syndrome, costoclavicular syndrome and pectoralis minor syndrome[1]. A fractured first rib and fractured clavicle have also been reported as causes[2]. A rarely described cause is subluxation of the first rib.

McCormick reported brachalgia from upward dislocation of the first rib at the costo-transverse and costo-vertebral junctions[3].

Lingren has reported twenty two cases with a hypomobile first rib causing a thoracic outlet syndrome and postulated the possibility of subluxation[4, 5].

The ribs articulate posteriorly with the vertebrae by means of the costo-transverse and costo-vertebral joints. These are synovial joints which are strengthened by very strong ligaments. The first rib, however, differs in that it articulates at the costo-vertebral joint only with one facet to one vertebral body rather than to two bodies[6] and at the costo-transverse it lacks a superior supporting ligament. In addition, scalenus anterior and medius insert on the superior surface of the first rib and elevate it on deep inspiration. The rib moves like a wing with the anterior and posterior ends fixed[7].

It is conceivable that a backward flinging motion of the externally rotated, raised arm, particularly associated with ipsilateral rotation of the head, could place enough stress upon the first rib to sublux it into an elevated position. Spasm and shortening of the scalene muscles might maintain the rib in the abnormal position. The neurovascular bundle could then be compressed between the first rib and clavicle. The Adson test, abducting the arm, rotating the head towards the painful arm and deep inspiration, causes stretch of the scalenus anterior causing further compression; a positive test results in reproduction of symptoms and loss of the radial pulse.

Head and neck rotation and lateral flexion away from the painful side may be restricted with a bony end-feel. This movement can cause impaction between the transverse process of the first thoracic vertebrae and a subluxed first rib. Palpation of the first rib will demonstrate reduction of movement during inspiration and expiration and this can also be demonstrated by cineradiography[5].

A plain antero-posterior X-ray taken with a cranial angulation, or a C.T. scan of the C8-T1 level, may be helpful particularly if the first rib is dislocated[2].

Electrodiagnostic studies are helpful only in true neurogenic thoracic outlet syndrome and are of less value in the non specific syndrome[2]. Placing the arm in a provocative position may produce abnormalities of ulnar nerve conduction[4].

Mobilisation of the first rib can produce dramatic results, with a sudden "give" as the rib is reduced and immediate reduction of symptoms; all of the above tests then become negative. Alternatively, Lingren describes isometric exercises of the scalene muscles to reduce the first rib subluxation. In the case of a dislocated rib, as described by McCormick, surgical resection was required to resolve the symptoms.

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CONFERENCE REPORT

Spinal Pain: Precision Diagnosis and Treatment

An official satellite meeting of the VIth World Congress on Pain

This conference was held after the Adelaide Pain Conference. It was largely composed of needle techniques directed at a variety of targets. The highlights of this meeting were the initial sessions involving Charles (Chuck) Aprill and Nik Bogduk and the paper presented by Jim Taylor. The anticipated advent of new needle techniques to affect disc pain was not forthcoming.

NEUROLOGY AND PATHOLOGY OF SPINAL PAIN

Nikolai Bogduk

Contemporary evidence implicates the intervertebral discs and the zygapophysial joints as the cardinal sources of spinal pain both in the cervical region and the lumbar region. Experimental provocation of either of these structures produces local and referred pain of a quality and distribution virtually identical to that seen clinically in patients with spinal pain. In such patients anaesthetising these structures relieves their pain.

The zygapophysial joints are innervated by the medial branches of the cervical and lumbar dorsal rami, each joint receiving a dual innervation from the nerves above and below the joint. The intervertebral discs are innervated posteriorly by the sinuvertebral nerves. Anteriorly, cervical discs receive branches from the ventral rami and grey rami communicantes.

The actual pathology of painful zygapophysial joints is not known, but probably encompasses capsular tears, haemarthrosis and subchondral fractures or their long-term sequelae. Lumbar discs are subject to torsion injuries of the anulus fibrosus and internal disc disruption, a painful chemical degradation of the nucleus that eventually afflicts the anulus.

Low back pain is often inflammatory in nature, mediated by chemical, mechanical and ischaemic factors.

Disc pain can be chemical, where inflammation is the nociceptive source. The inflammatory material in the disc becomes like a sterile abscess and produces constant, deep pain. It can be mechanical. This is like a medial knee ligament that is two-thirds cut and then a valgus stress is applied.

Persistent pain can cause changes in the central nervous system and this can cause further pain. So, one important aspect of pain therapeutics is the prevention of input into the spinal cord to prevent the so-called spinal plasticity, where dorsal horn neurones become hypersensitised.

A low density zone seen on C.T. may be a collection of blood, fluid or inflammatory material and if seen, there is an 80% or greater chance of this disc being painful on discogram and showing fissuring (*Yu S. Am J Neurorad, 1988; 9 : 367-370).*

A light signal seen on MRI in the posterior anulus shows on discography as being a circumferential fissure 100% of the time and in 80% of cases this disc will be painful.

CERVICAL DISC PATHOLOGY

J. R. Taylor

The description is based on a histological study of the discs from thirty cervical spines of subjects ranging from infancy to eighty years.

Cervical discs begin like lumbar discs, but during childhood, uncinate processes grow upwards from the lateral margins of cervical vertebrae (C3-7); clefts which appear in the lateral anulus on each side of the disc are called uncovertebral (U.V.) joints. The 45° orientation of cervical facets requires that all movements include a translatory component and since the cervical spine is highly mobile, shearing forces cause the uncovertebral clefts to extend medially towards the nucleus pulposus. By early adult life, fine fissures extend transversely from right to left U.V. joints, across the posterior half of most cervical discs. These are readily demonstrated by cervical discography; contrast spreads from the centre of each disc into both U.V. joints and not infrequently, into the anterior epidural space. After the age of thirty years, unlike lumbar discs, cervical discs do not contain a soft, encapsulated nucleus. Cervical disc pathology must be judged against this pattern of "normal" fissuring, in discs with increased collagen in a relatively hard fibrocartilaginous centre.

With ageing, degeneration involves one or more discs in the C4-5 to C7-T1 region. Degenerative discs are dramatically thinner than adjacent discs, with fragmentation of cartilage plates and irregular vertebral end plates. Fatty change may be seen in the adjacent vertebral marrow. Disc thinning throws increased loads onto the U.V. joints. With compressive loading, the fibrocartilage lining the U.V. clefts changes to hyaline cartilage, which fibrillates, the changes resembling osteoarthrosis in a synovial joint. Large U.V. osteophytes project into the intervertebral foramina. These may deform nerve roots or vertebral arteries, so that movements may endanger the distorted structures or their branches. The U.V. joints prevent direct nuclear prolapse into an intervertebral canal, but degenerate, fissured discs with osteophytic vertebral margins may project as horizontal bars into the anterior spinal canal.

In ten cervical spines from victims of motor vehicle accidents (M.V.A.), horizontal splits were found in the avascular cartilage plates of one or more discs, extending around into the anulus fibrosus. None of the spines showed radiological evidence of fracture and microscopic injuries to the zygapophysial joints were unusual. One case showed prolapse of disc material into the anterior epidural space. It is suggested that similar injuries probably occur in survivors of M.V.A.; cartilage plate splits would be slow to heal and may interfere with disc nutrition.

PATHOLOGY AND DIAGNOSIS OF CERVICAL PAIN SYNDROMES

M. Sluijter

This paper suggested that normally the C5-6 segment is the most mobile and C2-3 the least mobile in the typical cervical joints.

A computer assisted analysis of functional X-rays was suggested in order to detect hypermobility or instability to predict the site for nerve block or treatment. This system did not appear exact. Invasive treatments for failed management includes percutaneous facet denervation, percutaneous partial rhizotomy and percutaneous R.F. block of the stellate ganglion.

ANALGESIC DISCOGRAPHY IN THE DIAGNOSIS OF SPINAL PAIN

Philip M. Finch

Determining the exact origin of discogenic symptoms can be difficult, despite C.T. discography and even M.R.I. Multiple levels can be degenerate or even herniated on M.R.I. and some additional means of locating a symptomatic level is needed. Classical provocation discography can produce confusing results with false positives (image abnormal by C.T. discography, but clinically normal by pain provocation response). Asymptomatic individuals can have an abnormal M.R.I. signal and an abnormal discogram can even be seen with a normal M.R.I. in some cases. Analgesic discography provides an additional and useful method of determining the origin of symptoms.

A group of twelve females and nineteen males diagnosed as having discogenic pain was studied, comparing the results of analgesic discography with M.R.I. A particular spinal level was nominated after consideration of the relevant history, clinical findings and radiology. Following an M.R.I. study, the nominated spinal disc and an adjacent level were injected on separate occasions, usually a week apart. The needle was placed in the anulus fibrosus and not into the nucleus pulposus and on several occasions C.T. studies were performed afterwards to determine the spread of contrast agent.

On many occasions the effect of single level injection of local anaesthetic solution was dramatic, with almost total loss of pain and temporary restoration of normal range of movement. Such an improvement lasted from one to twenty four hours. Distinction between the symptomatic level and the uninvolved level was often clearly apparent to the patient. In almost 50% of those studied the injection of analgesic agents was a substantial aid to diagnosis. This was despite very adequate imaging techniques, using M.R.I. This technique appeared most useful in those with multiple level pathology demonstrated on M.R.I. and neuro-radiology, but where one particular level appeared to be the origin of symptoms. The identification of this particular level has obvious implication in patient management.

PROVOCATION DISCOGRAPHY AND TREATMENT

Charles N. Aprill

Diagnostic disc injection provides accurate anatomic data regarding the nucleus and its envelope, the anulus and end-plates. The reliability of the anatomic data has been documented in vitro by several authors since 1952. The adjunctive use of C.T. scanning has aided in understanding of lumbar disc disease and confirms dispersal patterns in vivo studies. Post-injection C.T. scanning of cervical discs has not proven useful.

Patient responses at the time of injection are more critical in the interpretation of this procedure. The major criticism of discography is based on "experimental evidence" over twenty years old. Such "data", even if correct, is hardly applicable to modern discography. Walsh, et al, in a recent study of asymptomatic volunteers suggest pain response is a valid criterion in the assessment of the painful disc.

Lumbar discography was initially developed to evaluate patients with negative or equivocal myelograms. Modern high resolution C.T. scanning has eliminated the need for discography in the diagnosis of most cases of disc prolapse, extrusion or sequestration. M.R.I. is extremely sensitive to alteration of disc chemistry and

can detect degeneration long before any changes are evident on routine radiographs or C.T. scans.

Major prolapses of cervical discs can be detected by both C.T. and M.R.I. However, both modalities are less sensitive in the cervical region. The paucity of epidural fat reduces contrast in the cervical region. Artifacts associated with patient motion, vascular and C.S.F. pulsation as well as the relative poor spatial resolution of M.R.I. reduce its sensitivity in the cervical spine.

Not all anatomic lesions are symptomatic. Not all symptomatic lesions are evident even on good quality studies. Poor quality scans can miss definite abnormalities.

Discography retains a place in the evaluation of chronic cervical and lumbar pain syndromes because of its unique ability to link pathoanatomy with symptomatology.

The spectrum of pathologic conditions includes painful anular fissures or tears, internal disc disruption, symptomatic end-plate disruption and symptomatic disc degeneration. All may be detected and defined with precise diagnostic disc injections and post-injection C.T. scanning. Discography is a tertiary diagnostic procedure and should follow evaluation by high resolution C.T. scanning and/or magnetic resonance imaging.

APPROACHES TO THE ZYGAPOPHYSIAL JOINT
Nikolai Bogduk

Lumbar zygapophysial joints can be anaesthetised using either intra-articular blocks or lumbar medial branch blocks. Intra-articular blocks are performed under image-intensifier using a posterior or posterior oblique approach along which trajectory the target joint space is best visualised. The needle is directed to the centre of the joint cavity, although if difficulties are encountered with entering the joint, an alternative target can be the capsular recesses immediately above or below the bony joint margin. The target for each lumbar medial branch is the proximal end of the superior border of the transverse process, and the nerve above and below the target joint must be blocked to anaesthetise the joint.

Cervical zygapophysial joints can be anaesthetised using either intra-articular blocks or cervical medial branch blocks, following a posterior or lateral approach for either. The lateral approach is the less tedious, less stressful and faster. For intra-articular blocks, the target is the centre of the joint cavity; for nerve blocks, the target is the centre of the articular pillar. A posterior approach may be used if required for patient comfort or if the joint cannot be entered laterally.

For intra-articular blocks, a pillar view is used, and the needle passed into the joint cavity along an upward and ventral trajectory. This may require a long path and, therefore, an extra-long needle. Medial branch blocks are performed using a direct posterior approach with the target being the "waist" of the articular pillar.

Intra-articular blocks are more tedious to perform and are best reserved for instances where presumptive treatment with intra-articular steroids is contemplated. For screening purposes, medial branch blocks are easier to perform and are less invasive while still yielding the same diagnostic information.

APPROACHES TO THE ZYGAPOPHYSIAL JOINT
Charles N. Aprill

The apophysial joints may be studied by direct injection techniques. Arthrography defines the size and shape of the joint space, as well as the integrity of articular capsule.

Pain patterns provoked by joint injection may implicate or exclude a given joint from consideration as a significant pain generator. Pain patterns have been studied in normal populations as well as in patients with spinal pain syndromes. Analgesic effects following anaesthetisation of the joint is an important diagnostic factor.

Two approaches to the cervical joints have been described. Okada initially described a lateral approach, Dory and others a posterior approach. The lateral approach has been employed in the evaluation of over five hundred patients since 1982 in an outpatient spinal diagnostic practice. It has proven to be safe and relatively simple.

The thoracic facet joints may be studied to evaluate their contribution to thoracic pain syndromes. The posterior approach is the only method of study in this region. The orientation of these joints as well as the surrounding skeletal structures make the thoracic joints inaccessible by any other approach.

Lumbar facet joints are most easily inspected by a posterior or posterolateral approach. Review of C.T. scans or M.R.I. scans (axial images) facilitates this procedure. Occasionally the L3-S1 joints may be almost coronal in orientation. In such instances, a direct posterior approach to the joint space is required, entering the joint through the inferior recess.

Study of the arthrographic patterns in multiple planes enhances understanding of capsular anatomy and facilitates the performance of the examination.

LUMBAR AND CERVICAL FACET DENERVATION
Nikolai Bogduk

Percutaneous radiofrequency medial branch neurotomy (PRFMBN) is a procedure whereby painful zygapophysial joints may be denervated. Because the nerves regenerate, the effect is not permanent, and the procedure should not be viewed as a curative neurosurgical operation, but a form of long-term analgesia with an estimated half-life of between six and twelve months. The principle of the procedure is that by coagulating the nerves that innervate a painful joint, conduction is prevented and pain is relieved. The relief is temporary because the nerve-cell bodies remain intact and the axons "heal". The duration of relief is proportional to the length of nerve that is incorporated into the heat lesion.

The target points for PRFMBN are the same as for cervical or lumbar medial branch blocks, but the trajectories for electrodes must be different from those for anaesthetic blocks. To maximise the size of lesions, the electrodes must lie parallel to the target nerve (not perpendicular as for nerve blocks), because the lesion develops around the circumference of the electrode but not distal to its tip. The electrode must lie against the nerve because the effective radius of the lesion is only about 2mm using SK-10 or owl electrodes (but may be larger with Ray electrodes).

The indications for PFRMBN are complete relief of pain following anaesthetisation of the target nerves. Theoretically, success rates should be 100% in the absence of target failures. However, convincing clinical trials using proper techniques are still lacking.

NERVE ROOT SLEEVE BLOCKS
Charles N. Aprill

Selective nerve sheath injections and blocks are diagnostic and occasionally therapeutic. Specific anatomic relationships dictate the technique at various levels in the spine.

The cervical segmental nerves traverse the lower portion of the intervertebral foramina. The vertebral and carotid arteries and associated main venous structures must always be considered in the performance of this procedure. Inadvertent puncture of the lung with pneumothorax is a foremost consideration in performing thoracic nerve blocks. The approach is almost vertical paramedican. Lumbar nerve blocks are less tedious as the spaces are larger.

In all instances, it is important to avoid dural puncture. Subarachnoid injection results in a total loss of block specificity. Instillation of contrast material is important to verify needle position and dispersal of solutions. Subarachnoid flow can be easily recognised. Venous filling with rapid clearance can occur as a result of the fairly large radicular veins which traverse each canal. Rapid venous clearance can result in a false-negative block if undetected.

The volume of local anaesthetic should be kept to a minimum. Volumes of 2mls or less add to the specificity of blocks.

Nerve blocks are a useful adjunct in the evaluation of patients with radicular complaints and multiple anatomic lesions on imaging studies. Selected blocks are also effective in assessing the chronic pain patient with multiple prior surgical procedures and prior to sensor rhizotomies. Occasionally blocks may be employed for therapeutic effect particularly when performed in conjunction with aggressive rehabilitation therapy.

Precise needle placement can avoid direct contact with segmental nerves. Selective nerve sheath and regional epidural injections are safe and relatively simple and can be done on an outpatient basis in most instances

There are ancillary tests, the results of which should be integrated with clinical and imaging data for more practical diagnosis.

CONFERENCE REPORT

International Symposium of the Physical Medicine Research Foundation.

The Honorary Secretary attended this symposium in Vancouver, Canada, on the 1st, 2nd and 3rd June, 1990.

It was pleasing to find that there is such a vigorous foundation with its prime aims being research and education in musculoskeletal medicine and allied fields.

The organisation has been holding annual meetings in various capital cities in the northern hemisphere since 1986. In 1992 the conference will be in London, then in Prague in 1993 and the suggestion is that in 1994 it should be held somewhere in the southern hemisphere. This would be a great opportunity for the New Zealand and Australian Associations of Musculoskeletal Medicine to join together as co-hosts.

Among many well-known speakers in Vancouver were John Mennell, Robert Salter, Chan Gunn, Vladimir Janda and Karel Lewit.

The main points of some of the papers are recorded below. The work of Janda and Lewit (have not been summarised because their philosophies have been written up in the Bulletin several times and they added nothing new at this particular conference.

CONTINUOUS PASSIVE MOTION
Robert Salter

Professor and Head of the Division of Orthopaedic Surgery, University of Toronto. He well known for his research into the concept of continuous passive motion and its clinical application to healing. His address covered some of the background to, and history of his theory of continuous passive motion. The pertinent points were as follows:

1. There is a limitation of the healing and recuperative power of the articular cartilage, which does not regenerate under normal circumstances. It can be made to regenerate by continuous passive motion. The pertinent characteristics of the articular cartilage are that it has no vascular or nervous supply. Pain comes from stimulation of the nerve endings in the bone below and nutrition is by diffusion.

2. The relative value of rest and movement in the restoration of function is questioned. The fathers of orthopaedic surgery, Potts and Thomas, were adamant that no joint should be moved until the bone had healed. The challenge for continuous passive motion is that joints should be allowed to move as freely as possible so that cartilage is not damaged. Therefore dogma needs to be challenged, questioned and tested.

3. The musculoskeletal system was designed to move joints and it does move continuously throughout life as is shown by the movement of the costovertebral joints throughout respiration. Without respiration there is no life and these joints do not suffer from arthritis.

4. Immobilisation brings with it muscular atrophy, painful joints, swelling, osteoporosis and decreased mobility.

5. All other body parts move following surgery. The integument and viscera are all moving and are not immobilised while the healing process goes on. Therefore it seems irrational that the skeletal system should need to be immobilised for healing to occur.

6. Several experiments were presented which showed how the cartilage regenerates beautifully while the joint is put through its full range of movement in a continuous fashion. One example was the removal of a large piece of bone and cartilage from the knee joint of a rabbit. This bony surface was inverted, such that the trabecular bone was facing upwards, the knee was reconstituted and the joint put through continuous passive movement for six weeks. The animal was then sacrificed; necroscopy showed a complete covering of the area with cartilage, indicating that certain cells were capable of regenerating cartilage and therefore were capable of producing tissue other than that which the stem cell generally produces.

The concept of continuous passive motion(CPM) has also been shown to be effective in septic arthritis; the patient is given an appropriate antibiotic but the joint is not immobilised. The continuation of this theory is that for younger people (those less than 50) who are faced with the prospect of joint replacement should undergo continuous passive movement for up to six weeks to see whether cartilage can regrow; they may not then have to be subjected to joint replacement. Many types of apparatus are available to move joints through their ranges of movements 24 hours a day. The large joints will go through their movement ranges as the person moves about during the daytime and at night the knee and the hip can be put through their ranges of movement continuously by using various types of machinery.

NEUROPATHIC PAIN
Chan Gunn

Director of the Pain Centre, Vancouver, Dr. Gunn gave a fascinating lecture based on identifying the presence of neuropathic tissue. The philosophy espoused by Dr. Gunn is that a large number of musculoskeletal syndromes which give rise to chronic pain occur because there is underlying neuropathic mechanism which is secondary to spondylosis; this then causes painful or hypertonic muscles. The para-spinal muscle shortening compresses and irritates the nerve root, thus perpetuating the pain. The technique which he describes is that of intramuscular stimulation using 30 gauge acupuncture needles of varying lengths.

Chronic pain can occur if any of the following are present:

1. Ongoing nociceptive stimulus or inflammation.
2. Psychological factors such as somatization disorder, depression or operant learning processes.
3. Functional and structural alterations within the central or peripheral nervous system, eg. neuropathic pain.

Dr. Gunn then went on to list the clinical features of neuropathic pain as follows:

1. Pain in the absence of an ongoing tissue damaging process.
2. Delay in onset after the precipitating injury.
3. Abnormal or unpleasant sensations, such as burning or searing pain, or deep aching pain which is more common than aesthetic pain in musculoskeletal syndromes.
4. Pain felt in the region of sensory deficit.
5. Paroxysmal brief shooting, or stabbing, pain.
6. Painful mild stimuli (allodynia)
7. Pronounced summation and after-reaction with repetitive stimuli.
8. Pathological shortening of muscles producing enthesopathy, tenosynovitis and such conditions as chondromalacia.

Very frequently the hypertonicity of the muscle is not measured by current electromyography but the muscle shortening can be palpated as ropey ends within muscles. Changes which are often noted include autonomic manifestations such as vasomotor, sudomotor and pilomotor phenomena.

As far as mobilisation or manipulation is concerned both techniques are appropriate treatment when there is loss of function with respect to a joint. This loss of function is obvious on clinical mobility testing but is not obvious on an X-ray film. The lesion is diagnosable and treatable on clinical grounds without resort to expensive radiological investigations. Therefore, the treatment of pain in the musculoskeletal system can be both cost effective and beneficial to the patient in the hands of a trained practitioner.

FIBROMYALGIA
Frederick Wolfe

Clinical Professor of Medicine at the University of Kansas School of Medicine, Prof. Wolfe, spoke on the problems associated with the diagnosis and treatment of the fibromyalgia syndrome, a topic on which he also spoke at the Adelaide meeting of the International Society for the Study of Pain.

This chronic pain disorder has had varying degrees of acceptance in medical circles. Many attempts have been made to try to define its particular characteristics and recently the following were decided by the fibromyalgic criteria study group, following a multicentre trial in the US. The criteria were as follows:

1. Wide-spread musculoskeletal aching, lasting longer than three months.
2. Localised tenderness in 12-14 areas which have been well mapped after the trigger points of Simons and Travell.
3. Sleep disturbance such that the patient wakes numerous times during the night. In the morning they wake up feeling tired and unrefreshed.
4. There is the presence of tenderness when the skin over the tender spot is rolled.
5. Absence of abnormality in any of the usual medical tests to determine some kind of inflammatory response, eg. ESR, CBP, rheumatoid factor, etc.
6. Muscles have less power and fatigue reasonably quickly.

By using pressure algometers, pain was produced at these various trigger points at a much lower level of tension than the corresponding points in normal people.

Fibromyalgia should be distinguished from myofascial pain syndrome. With regard to myofascial pain of the head and neck, the patients with these syndromes suffer very similar symptoms and it can be difficult to determine whether a person who presents with head and neck pain has fibromyalgia or a myofascial pain syndrome. However, with regard to the trunk and extremities there is a marked difference between the two conditions, with the fibromyalgic patient having a much greater decrease in their capacity for work. Amytriptyline 10-25 milligrams was shown to be helpful for the discomfort experienced by the patient suffering from fibromyalgia. This medication is given once a day for two weeks before the effect was experienced.

Fibromyalgia may be precipitated by motor vehicle accidents as a secondary phenomenan.

SKELETAL STRUCTURE, MUSCLE FUNCTION AND THE POSTURAL COMPLEX

Steven Spark

Dr. Spark is a podiatrist from British Columbia with a special interest in sports medicine. He conducted this workshop on biomechanics and in it gave numerous anecdotal examples indicating that chronic musculoskeletal pain may be caused by a lack of muscle balance in the lower limbs. For example, chondromalacia is likely to be caused by a tight Achilles tendon which produces a poor take-off in gait requiring the quadriceps to be tight and forcing the patella onto the femur.

These conditions can be treated by dry needling the tight ropey strands. Resistance is felt when the needle pierces the fibrosed material; the muscle appears to grasp the needle, which should then be twisted until it is felt to be free. Often the needle has to be left in for up to 10-20 minutes during which time the patient will feel release of pain. The treatment is followed by stretching the muscle using techniques similar to the postisometric stretches advocated by Lewit.

The presence of trophoedema is demonstrated on skin rolling and in particular by the match stick test, which involves sticking the end of a match into the oedematous skin, leaving indentations. This sign is indicative of the presence of trophoedema which is secondary to muscle shortening.

REHABILITATION OF LOW BACK PAIN PATIENTS

Tom Mayer

Prof. Mayer is Medical Director of the Productive Rehabilitation Institue of Dallas for Ergonomics (PRIDE) and also the Clinical Professor of Orthopaedic Surgery at the University of Texas, Dallas. He presented some interesting facts with regard to a special program for the rehabilitation of low back pain. Previous figures on patients in whom disc surgery had been performed indicated that only 43% ever returned to work. He considered that the medical profession was the gate keeper of the compensation system and therefore a more active attitude to rehabilitation should be developed. The PRIDE system for rehabilitation has been effective in returning a much higher percentage of people with chronic low back pain to work.

In a study involving two hundred patients with low back pain of more than four months duration, 95% of the trial group were back at work, whereas the percentage being returned to work for the comparison group was of the order of 40%. An interesting fact was that on follow-up only 10% of the treatment group had additional allied health professional consultations whereas 55% of the comparison group on a follow-up basis, had sought help from other allied health professionals. For those who were in the treatment group and once back at work, only 4% reported recurrent injuries compared with the comparison group in which the number reporting recurrent injuries was four times as great.

It therefore would seem that the programme established by the PRIDE group for the rehabilitation of low back pain is effective. The programme is based on continual stretching and mobilising as well as efforts to improve the psychological disposition of the patient. They have published a book entitled "Functional Restoration of Spinal Disorders" by Mayer and Gotcher, published by Lee and Febiger.

MUSCULOSKELETAL DIAGNOSIS

John Mennell

Dr. Mennell was one of the founding members and a past president of the Physical Medicine Research Foundation. He is now a retired orthopaedic surgeon who has published several books on musculoskeletal conditions.

Dr Mennell stated that chronic musculoskeletal pain only comes about because someone has not managed to diagnose and treat adequately in the early stages. The problem is largely due to poor training in medical school and because of this the consumer is badly treated. It is interesting that the only system in the body in which very little time is spent determining the cause of the pain is the musculoskeletal system. When pain arises in this system patients are given non-steroidal anti-inflammatory drugs, rest, heat or some modality for relief but few attempts are made to determine what the underlying cause of the pain is. This type of approach is not used in any other body systems. (This is what AAMM has been saying for years.)

Other subjects Dr. Mennell addressed included postural imbalance and poor exercise instruction by coaches to junior athletes, which has been recognised as a cause of long term pain in elite athletes. Many training schedules do not take into account the importance of the biomechanics of the exercises which they are doing. A particular example is the stretching of the hamstrings which should never be performed with the subject standing, Also, special attention needs to be given to the biceps component of the hamstring muscles. This is the segment that is usually a problem when the hamstrings are torn but with the exercises of sitting and stretching or touching the toes, this particular part of the hamstring is rarely stretched adequately. A particular exercise for stretching this part of the hamstrings is to keep the knee flexed about 10^0 while the hip is tightly flexed, stretching the particular parts of the hamstring which the patient feels tight. This can be done by varying the angle of stretch.

He also considered that bunions were a function of poor muscle balance whereby the take-off component from the anterior tibialis muscle tends to flare the first metatarsal medially, causing the problem. Orthotics can help but the important treatment is to relax the tight muscles causing the malalignment.

Accreditation Assignment

The administration of oral medication is one modality frequently employed in the management of difficult problems. In the situation depicted below, a relaxant preparation is being used in combination with manual therapy for the relief of irritability due to overload stress.

The practitioner sits on the patient's left side and the patient is encouraged to assume a comfortable recumbent position. Two consultants are on hand to provide any advice or assistance that may be required.

Members are invited to study the various aspects of this situation and to answer the following questions.

1. What is the maximum safe dosage of this preparation and what are the dangers if this dosage is exceeded?
2. Is the traction apparatus in the background likely to be of use in this situation and, if so, how should it be applied?
3. Would you recommend pre-medication in these circumstances and, if so, what preparation(s) and dose(s) would you recommend?
4. What criteria would you accept as denoting that the treatment had been successful?
5. What complications could arise and how could the two consultants assist in dealing with them?

Answers should be confined to one thousand words or less and thirty accreditation points will be awarded for each satisfactory reply received by the Hon. Secretary before 31st August, 1990.

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