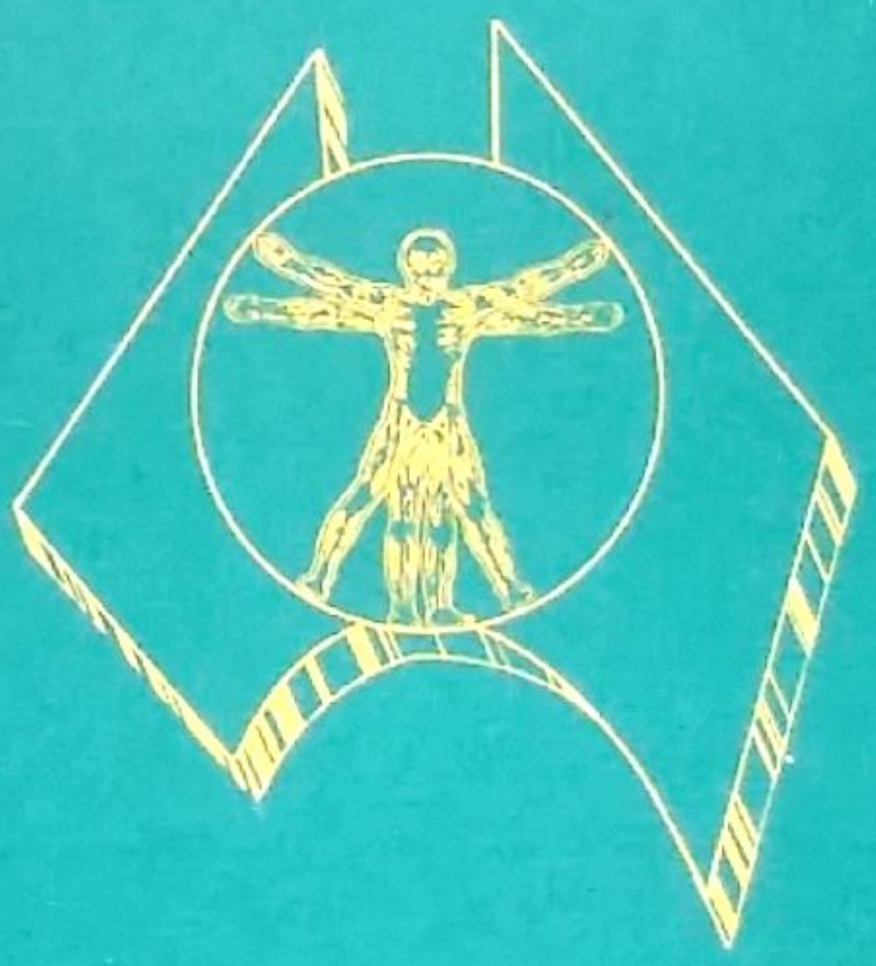
# Australian Association of Musculoskeletal Medicine

# *Bulletin*



## Anatomy and Physiology of Nociception

## Epidural Spinal Electrical Stimulation

## Leg Length Inequality

## A Diagnostic Approach to Back Pain

Vol. 7 No. 1 March 1991

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# Australian Association of Musculoskeletal Medicine

# *Bulletin*

Vol.7 No.1 March 1991

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The A.A.M.M. Bulletin is produced by the Australian Association of Musculoskeletal Medicine for medical practitioners interested in the aetiology and management of musculoskeletal disorders. Opinions expressed are those of the authors and not necessarily those of the editor or the Association. Editorial comment may reflect the opinions of the editor alone. Contributions on any relevant topic are welcome for submission to the editor, Dr. Wade King, 82 High Street, Taree, NSW, 2430, telephone (065) 51 0662, or to any member of the A.A.M.M. Council.
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# AUSTRALIAN ASSOCIATION OF MUSCULOSKELETAL MEDICINE
## OFFICE-BEARERS 1991

The following members were elected to office at the Annual General Meeting in Melbourne on 29th November, 1990.

### PRESIDENT:

**Dr. David Vivian** MB, BS

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

### VICE-PRESIDENT:

**Assoc. Professor Nikolai Bogduk** BSc (Med) (Hons), MB, BS (Hons), PhD, Dip Anat, Hon MMTAA

Faculty of Medicine, University of Newcastle,
Newcastle, N.S.W., 2308
telephone (049) 21 5608

### HON. SECRETARY:

**Dr. Norman Broadhurst** MB, BS, MSc, PhD, DipEd, DipRACOG, FRACGP

7 Brighton Road, Glenelg, S.A. 5045
telephone (08) 295 1890

### HON. TREASURER:

**Dr. Wade King** MB, BS

82 High Street, Taree, N.S.W., 2430
telephone (065) 51 0662

### COUNCILLORS AND SUB-COMMITTEE MEMBERS:

| | | |
|---|---|---|
| **Dr. Max de Clifford** | Donvale, Vic. | (03) 873 2537 |
| **Dr. Geoff Harding** | Sandgate, Qld. | (07) 873 2537 |
| **Dr. Gary Hopkins** | Largs Bay, S.A. | (08) 347 0400 |
| **Dr. Marius Leoffler** | Pinjarra, W.A. | (09) 531 1658 |
| **Dr. David McGrath** | Fadden, A.C.T. | (06) 292 6574 |
| **Dr. Ron Palmer** | Herston, Qld. | (07) 252 1128 |
| **Dr. Simon Rosenbaum** | Prahran, Vic. | (03) 525 1285 |
| **Dr. Allan Saltau** | Miles, Qld. | (076) 27 2000 |
| **Dr. Vic Wilk** | Brighton, Vic. | (03) 596 7211 |

### SUB-COMMITTEES:

| | |
|---|---|
| **Accreditation:** | Gary Hopkins *(convenor)*, Nik Bogduk |
| **Bulletin:** | Ron Palmer *(convenor)*, Wade King, Simon Rosenbaum, Allan Saltau |
| **Education:** | Max de Clifford *(convenor)*, Geoff Harding, Norm Broadhurst |
| **Membership and Publicity:** | Vic Wilk *(convenor)*, David McGrath |
| **Overseas Liaison:** | Marius Loeffler *(convenor)*, David Vivian |

# Editorial

*If a tree falls in a deserted forest, is there any noise? This question has long been posed as something of a conundrum but for the scientifically minded the answer is simple. Noise is an experience rather than a physical phenomenon. For noise to be experienced there must be a receiving apparatus (such as that in the ear) to convert sound waves to a form of neurological transmission, which the cerebral cortex perceives as the experience of noise.*

*So it is with pain. For pain to be experienced there must be reception of a stimulus and transmission via neurological pathways to the cerebral cortex, where the final form of the incoming message is interpreted. That interpretation will depend upon the modifying effects of other cortical factors such as mood, motivation and cultural conditioning, each of which in turn will be influenced by other considerations such as secondary gain, financial options, career prospects, educational status and other psychosocial determinants.*

*In recent years there has been a tendency for attitudes to pain management to become polarised. Recognition of the roles played by cortical influences has had a profound effect on methods of chronic pain management and attempts to modify the experience (and the resultant behaviour) at a cortical level are seen by many practitioners as the best approach to the problem. Others take an opposing view, claiming that psychological programmes have little effect on the physical phenomena associated with pain; instead, they place emphasis on particularisation of the noxious stimulus and its effects on the functions of the structures involved. Management regimes based on this approach rely heavily on physical modalities of treatment aimed at restoration of function.*

*Each of these attitudes represents an undue focus on one end of the nociceptive process. Each is valid but only if placed in the perspective of the whole process (and they are certainly not mutually exclusive). In fact, each represents one extreme of a broad spectrum of approaches to pain management, which should properly address all aspects of each particular sufferer's problems.*

*The main difficulty in the management of pain, and especially chronic pain, is lack of understanding of the complex physiological mechanisms involved. The situation has been compounded until recently by lack of reliable scientific information about the neurophysiology of nociception and the micro-anatomy and biochemistry by which it is effected. Much is yet to be revealed but enough is now known to make redundant forever the simple models of pain perception which had been taught for generations.*

*An outline of current knowledge of the anatomy and physiology of nociception is provided by one of the articles in this issue of the Bulletin. The paper presents the salient facts in sufficient detail for those involved in musculoskeletal medicine to place the problems with which they deal into an appropriate scientific context. It is a timely review and one which will challenge members to re-evaluate their therapeutic strategies.*

*Another article in this issue describes epidural spinal electrical stimulation (ESES), one of the neurosurgical techniques of pain modulation recently introduced for control of certain types of pain problem. ESES is an example of the practical application of the science of nociception. Whilst it will not in itself solve many of the problems which musculoskeletal physicians see, an understanding of it will serve to widen their therapeutic horizons and to deepen the perspective in which other treatment modalities are seen.*

*Efforts to modify the direct effects of noxious stimuli continue to be of fundamental importance, as do efforts to modify the cortical influences which tend to increase or prolong the experience of pain. Musculoskeletal practitioners are encouraged to continue using both approaches, as appropriate in particular circumstances. They are also encouraged to increase their understanding of the nature of nociceptive mechanisms, both as a means of refining their indications for the use of particular treatment modalities and as a guide to the evaluation of the newer forms of patient management to which research into nociception will inevitably lead.*



# A Word from the President

*My kitchen is full of gadgets. My garage is full of gadgets. My musculoskeletal practice is full of gadgets.*

*Most of my gadgets gather cobwebs. Some even have cobwebs on the cobwebs. They are obviously superfluous.*

*By far the most expensive gadgets are the ones that sit in the musculoskeletal practice. The others are merely souvenirs of capricious forays into being a handyman, chef, etc.*

*The gadget that takes the cake, however, is the laser acupuncture machine. Now, I've always thought that needle acupuncture seemed to be a reasonably good idea. The needles are cheap, even if used only once, they have at least an excellent placebo value, and acupuncture seems theoretically sound in treating pain along the lines of the gate control theory; but laser acupuncture?*

*It was with great interest that I read articles written on the effects of laser acupuncture in the journal Pain, Volume 43, November, 1990, and in a March edition of the Australian Medical Journal.*

*The sort of papers published in the Pain journal are well reviewed in a guest editorial entitled "What's in a Laser Beam for Pain Therapy?" by M. Devor. He points out the following facts about a helium-neon laser therapy gadget:*

1. *Lasers produce light.*
2. *Helium-neon lasers produce red light.*
3. *Lasers used as cutting implements, e.g. in surgery, provide energy at the infra-red wave lengths and delivering heat.*
4. *In laser acupuncture devices, the energy is about 10,000 times weaker.*
5. *These laser acupuncture machines are much the same as the laser pointers used in lectures.*
6. *The articles on treatment of acute pain with laser acupuncture suggest that they have absolutely no effect.*
7. *If laser acupuncture is effective as a placebo machine, then why spend thousands of dollars buying one, when you can buy a flashlight and put a piece of red cellophane paper over the bulb.*

*There was a time when I nearly bought a laser acupuncture gadget. A salesman brought one in and introduced it to the clinic for a trial period. I could never get really enthusiastic about it. I could not even see it as a good placebo gadget. Now I might just resurrect that little pocket torch that I had back in the 1970s, call in at the newsagent to buy some of that red cellophane paper and book myself a $2,000 ticket to the 1992 Gadgets Convention in New York.*

### *References*


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3. *Brockhaus A, Eiger C E. Hypoalgesic efficacy of acupuncture on experimental pain in man. Comparison of laser acupuncture and needle acupuncture. Pain 43:2, November 1990.*
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5. *Haker E, Lundeberg T. Laser treatment applied to acupuncture points in lateral humeral epicondylalgia. A double-blind study. Pain 43:2, November 1990.*
6. *Tandon M, Soh P, Wood A. Acupuncture for bronchial asthma? A double-blind crossover study. Med Journal of Australia, Vol 154:6, March 1991.*

# From the Secretary's Desk

The Annual Conference in Melbourne last year was greatly appreciated by the large number of members who attended. Several other doctors indicated their interest in matters musculoskeletal by joining the Association at the meeting. As more and more doctors are contacted, many express their amazement at the amount of scientific information which is available to help in the management of musculoskeletal problems.

Acceptance of the new constitution and election of a new committee offers potential for greater activity in the area of continuing education. With added enthusiasm and motivation from other sources it is hoped that regular teaching sessions and workshops will be conducted in each state. All efforts should be directed towards involving more people in the activities of the Association which will culminate this year in the Annual Scientific Meeting in Adelaide, advertisements for which were circulated at the Melbourne meeting. Details of the annual meeting and the regular meetings held in the various states will be found in each issue of the Bulletin. Every member is encouraged to invite as many colleagues as possible to participate in the Association's educational programme.

The difficulties of establishing outpatient clinics and postgraduate courses in musculoskeletal medicine are not of recent origin. In reviewing the literature I found that James Cyriax had similar problems, as he indicated in a letter to the British Medical Journal in 1972, from which I would like to quote the following:

* "I have yet to meet an orthopaedic surgeon who does not complain of the boredom of seeing endless cases of backache."

* "The void which orthopaedic medicine closes is there for all to see. This limbo comprises, what is more, the most quickly remedial fraction, did the doctor and patient but know it. However, medical students cannot learn what their teachers do not tell them. And where in our training schools is the physician responsible for the non surgical side of orthopaedic tuition. The graduate becomes embarassed then frustrated at his inability to recognise and deal with these common disorders and is only partly consoled when the hospital in his district proves equally unable."

* "This hiatus in medical education has led to the emergence of all sorts of laymen happy to step in where doctors neglect to treat. The patient then lies at the mercy of a large variety of cultists - nature curers, faith healers, bone setters, masseuers, chiropractors, osteopaths. There are no nerve setters, cardiopractors or diabeticopaths, for in all these diseases the patient feels the interest and competence of his family doctor and consultant".

Cyriax obviously was anxious for the establishment of hospital outpatient clinics and postgraduate courses in musculoskeletal medicine. His efforts did not meet with much success at the time, for reasons open to conjecture, but the seeds sown by him and other pioneers of the discipline are certainly beginning to bear fruit now. We must carry on the work and should heed the final words of Cyriax's letter:

* "We must not be in the position where we can say that nothing can be done because there is no one there to do it."



# MAIL BAG Letters to the editor

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


## An open letter to the A.M.A.

Dear Sir,

I have been a member of the A.M.A. for nearly forty years. In that time, a growing interest in musculoskeletal medicine has meant that patients now consult me primarily for these and related problems.

As I understand it, I am therefore not eligible to apply for inclusion as a V.R.G.P. Now I am 64 years old and this is not a serious financial consideration but there are many younger, struggling doctors significantly disadvantaged at present and probably more severely in the future. Even our medical reports attract a smaller fee.

I understand the A.M.A. still has reservations about vocational registration but this does not appear to be one of them.

If, as you claim, our Association is concerned with the rights of every individual, you should be able to correct this obvious anomaly without too much trouble.

If you are unable or unwilling to do this, continued membership would not seem appropriate for us.

Yours faithfully

(Dr.) Egow Auerbach
Elizabeth Bay, NSW.

*Dr Auerbach's point is well made and many members will share his concerns. Vocational registration of G. P.'s has certainly added an extra anomaly to what was already inadequate remuneration for services in musculoskeletal medicine.*

*The Council of the A.A.M.M. has been negotiating for some time to overcome this strong disincentive for provision of comprehensive services in the discipline. However, it maintains that more must be achieved than admission to the register for the current V. R. descriptors. The policy being pursued is that descriptors should be related to the service provided rather than to the providor. The A.A.M.M. is not in the business of trying to create a new speciality for an elite few. It is (and always has been) in the business of advocating higher standards of patient care and of gaining recognition (including adequate recompense) for services conforming to those standards. The objective is to achieve levels of remuneration which will encourage provision of more effective services, whether on an occasional basis by those in general practice or on a regular basis by those in full-time musculoskeletal practice.*

- Ed.

# AAMM NEWS

Australian Association of Musculoskeletal Medicine

The twentieth Annual Scientific Meeting of the A.A.M.M., held in Melbourne at the end of November, 1990, was an outstanding success. The programme included three days of lectures and demonstrations on a wide range of musculoskeletal topics from basic sciences to clinical assessment and management strategies. Many of the papers presented were based on original work and the large amount of new material put forward at the meeting reflected the wealth of activity in musculoskeletal medicine in Australia.

The sessions were obviously appreciated by the many who attended and even the associated breakfast and luncheon meetings attracted capacity audiences. Several pre- and post- conference courses were also held and they too were deemed successful by those who attended. Altogether, with two weeks of activities and a wide range of educational opportunities catering for all levels of expertise, the conference set a new standard for the discipline.

❖

*An Extraordinary General Meeting was held immediately prior to the Annual General Meeting on 29th November, 1990, to consider a change of constitution with a view to incorporation of the Association. The E.G.M. adopted a new constitution, which embodies the spirit of the previous constitution but conforms to the guidelines established by the New South Wales Associations Incorporations Act of 1984. Formal registration of the A.A.M.M. as an incorporated body is now in progress and will provide legal safeguards which all members should appreciate. Copies of the new constitution (now designated "Articles of Incorporation") were distributed to all members prior to the E.G.M. and are available from the Hon. Secretary.*

❖

The committee is henceforth to be known as the Council of the association, in accordance with the incorporation guidelines and the new constitution. New members were elected at the A.G.M. in November, 1990, and each was assigned a "portfolio" so as to increase the efficiency of the Associations' governing body. There are now five standing sub-committees, which include Accreditation, Bulletin, Education, Membership and Publicity and Overseas Liaison. The new councillors' names and areas of responsibility are to be found on the Office Bearers page of this issue and members are encouraged to approach them directly for assistance with relevant matters.

❖

*The culmination of the Association's cultural programme for 1990 was an open air performance of Bogduk's "Dance of the Myotomes" and "Dance of the Dermatomes", choreographed and directed by the composer, in the Treasury Gardens, Spring Street, Melbourne on the afternoon of Sunday, 2nd December, 1990. The performance was presented as a cultural gift to the people of Melbourne and as an expression of thanks for their hospitality to the members of the A.A.M.M. during the annual conference. Forty members of the Association took part in the performance, which was so well received that the members of the company were cheered to three encores before being allowed to leave the stage.*

❖



One of the very interesting presentations at the twentieth Annual Scientific Meeting was that by **Professor Lance Twomey** of his work in conjunction with **Professor Jim Taylor** on the histopathology of zygapophysial joint and intervertebral disc injuries following motor vehicle accident trauma. Although many members had read their early reports in the literature, being able to see their slides in colour gave members an opportunity to appreciate the work in a way that would not otherwise have been possible. The lesions they demonstrated repeatedly in a series of motor accident victims with negative radiological findings should greatly increase understanding of the problems of those who suffer spinal pain after motor vehicle accidents and other forms of spinal trauma.

❖

*Some membership subscriptions for 1990/91 are still outstanding and members who are currently unfinancial will be receiving a reminder notice with this Bulletin. Early return of these (with the appropriate cheques) to the Hon. Treasurer will be much appreciated.*

❖

Licentiates were awarded on 1st December, 1990, to the first group of members who had satisfied the requirements of the Association's accreditation scheme. Several theses have been submitted since and the number of those accredited is rising steadily. Negotiations are still proceeding for vocational registration of those involved in the discipline. Members who are not yet accredited are encouraged to consider making application in the near future and to contact the Hon. Secretary for further information and the necessary application forms.

Meanwhile, because of the number of theses received, for the first time since the inception of the Bulletin the editor is embarrased by the amount of copy available for publication. Authors who are waiting for their works to appear in print are asked to be patient.

❖

*Due to the length of articles in this issue the promised item on the history of the A.A.M.M. was not able to be included. Interested members are assured that it will be included in a future issue when space permits.*

❖

This issue of the Bulletin was produced under some difficulty, due to the fact that the Bulletin sub-committee convenor, **Ron Palmer**, is currently overseas pursuing higher learning. Just what he is learning is open to speculation but it is certainly at a high level. Ron is currently a member of a mountaineering team making an assault on Mt. Everest. We look forward to hearing of his adventures when he returns (hopefully with all appendages intact).

❖

*Less adventurous mountaineers will be planning their assault on Mt. Buller, at the Association's fifth annual Winter Meeting in July-August, 1991. Details will be found in the Meetings, Conferences and Courses pages of this issue. Members thinking of going to Buller this year should be making bookings now so as to avoid disappointment. Application forms are enclosed with this Bulletin.*

❖

The Association's twenty-first Annual Scientific Meeting will be held in Adelaide from 24th to 26th October, 1991, shortly before the Adelaide Grand Prix. The theme will be "Injuries of the Cervical Spine" and details of the programme are to be found on the Meetings, Conferences and Courses pages. Some members have already made bookings so it is obviously not too early to contact the convenor, Dr. Norm Broadhurst, 7 Brighton Road, Glenelg, S.A., 5041 to ensure a place at the meeting.

# 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** the 1991 meetings will be held in conjunction with Sports Medicine. The venues and some of the topics are listed below; all meetings commence at 8pm. Those interested in attending are requested to contact the clinic beforehand on the telephone numbers listed.

| | |
|---|---|
| April 9 | **'Headache - differential diagnosis' Dr. Paul McRory and Mr. David Groom**<br>Malvern Sports Medicine Centre, 330 High Street, Ashwood<br>telephone (03) 885 8961 |
| May 14 | Alphington Sports Medicine Clinic, 339 Heidelberg Road, Northcote<br>telephone (03) 481 5744 |
| June 11 | **'Foot Pain' Mr. David Young and Mr. Matthew Appleton**<br>Croydon Sports Medicine Centre, 383 Dorset Road, Croydon<br>telephone (03) 725 2444 |
| July 9 | Olympic Park Sports Medicine Centre |
| August 13 | Metropolitan Spinal Clinic, 302 Malvern Road, Prahran<br>telephone (03) 529 1988 |
| September 10 | **'Rotator Cuff Injuries' Dr. Mary Jane Fitzpatrick, Dr. Frank Burke and Ms. Lynne Watson** Prahran Sports Medicine Centre, 316 Malvern Road, Prahran<br>telephone (03) 529 8899 |

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**, Prof. Nik Bogduk on (049) 68 5749.
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.



## Annual Scientific Meeting of the AAMM

Theme: ***"Cervical Spine"***

The Association's Twenty first Annual Conference will be held at the Grand Hotel, **Glenelg** on 24th to 26th October, 1991. 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, 24th October**

| | | |
|---|---|---|
| 08.30am | Registration | |
| 09.00am | Opening address and Conference Perspectives | |
| 09.30am | Whiplash Injury - The Cause and The Lesion | *Prof J. Taylor* |
| 10.10 - 10.40am | What Causes the Pain? | *Prof N. Bogduk* |
| 10.40 - 11.10am | Refreshment Trade Exhibits | |
| 11.10 - 11.45am | Biomechanics of the Neck | *Prof M. Allen* |
| 11.45 - 12.15am | Why don't people get better? | *Dr M. Awerbach* |
| 12.15 - 13.30pm | Lunch | |
| 13.30 - 14.00pm | 3D Cervical Segmental Movement | *Dr M. Pearcy* |
| 14.40 - 14.40pm | Pain Pathways of Neck and Shoulder | *Prof N. Bogduk* |
| 14.40 - 15.05pm | Ophthalmological Aspects of Whiplash | *Dr R. Renton* |
| 15.05 - 15.30pm | Radiofrequency Denervation | *Dr D. Vivian* |
| 15.30 - 16.00pm | Refreshments and Trade Exhibits | |
| 16.00 - 17.30pm | Annual General Meeting A.A.M.M. | |

**Friday, 25th October**

| | | |
|---|---|---|
| 09.00 - 09.45am | Radiology of the Neck | *Dr N. Sandu* |
| 09.45 - 10.30am | Chronic Pain from Whiplash | *Dr D. Cherry* |
| 10.30 - 11.00am | Refreshments and Trade Exhibits | |
| 11.30 - 12.00am | Neck Pain Syndromes | *T.B.A.* |
| 12.00 - 12.20pm | T.M.J. - Pain and Treatment | *Dr P. Duke* |
| 12.20 - 12.40pm | Sympathetic Pain - R.S.D. | *Dr J. Pfitzner* |
| 12.40 - 14.00pm | Lunch | |
| 14.00 - 14.45pm | Psychiatric view of Chronic Pain | *Prof I. Pilowsky* |
| 14.45 - 15.15pm | S.G.I.C. - A.C.CC. Experience | *Mr R. Daniels* |
| 15.15 - 15.45pm | Refreshments and Trade Exhibit | |
| 16.15 - 16.30pm | Free Papers | |
| 16.30pm | Depart for Annual Dinner on board Falie | |

**Saturday, 26th October**

| | | |
|---|---|---|
| 09.00 - 09.45am | Myofacial Pain Syndromes | *Dr W. King* |
| 09.00 - 10.00am | Dry Needling Experience | *Dr N. Broadhurst* |
| 10.00 - 10.30am | Cervical Injuries in Sport | *Prof M. Allen* |
| 10.30 - 11.30am | Surgical Intervention - When? | *Mr P. Reilly* |
| 11.30 - 12.00am | Free Papers | |
| 12.00 - 12.30pm | 2 minutes/2 slides | |
| 12.30 - 14.00pm | Lunch | |
| 14.00 - 14.35pm | Whiplash Research Project | *Dr N. Broadhurst* |
| 14.35 - 15.00pm | Rehabilitation Profile | *Dr R. Lee* |
| 15.00 - 15.30pm | What directives in Research | *Prof J. Taylor* |
| 15.30 - 16.00pm | Refreshments and Trade Displays | |
| 16.00 - 17.00pm | How to treat the Lesion - Panel Discussion involving N. Bogduk, M. Allen, J. Taylor and P. Reilly | |

**The Conference Secretariate:**
*7 Brighton Road,*
*Glenelg, 5041.*
***Phone (08) 295 1890.***

## Pre - and Post-Conference Courses

These courses will run from Tuesdsay, 22nd October to Thursday, 24th October and Sunday, 27th October to Monday, 28th Octoberl

**Biomechanics of the Musculoskeletal System** with Assoc Prof. Murray Allen from Simon Fraser University, B.C.

**Shoulder and Upper Limb** with Dr David Vivian.

**Cervical Spine and Shoulder Girdle** with Dr. Norm Broadhurst.

**Lower Limb - hip, knee, ankle and foot** with Dr. Wade King.

Breakfast and luncheon sessions will be organised with topcs and speakers arranged to suit. Details to be advised at a later date.

Please refer to conference brochure included with this publication or contact the Conference Secretariat for further details.

**The Conference Secretariat:**
*7 Brighton Road,*
*Glenelg, 5041.*
***Phone (08) 295 1890.***

## AAMM Winter Meeting

The Winter Meeting will be held at the Southern Cross Lodge, **Mount Buller**, again this year, from 28th July to 2nd August, 1991. 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 $275 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.

## 1991 International Congress of Pain Clinicians

*"New Trends and Techniques in Pain Therapy"*

September 19 to September 22, 1991, **Davos,** Switzerland.

under the auspices of the World Society of Pain Clinicians and the Swiss Pain League in co-operation with the World Health Organisation.

Your enquiries, suggestions, contributions, etc., will be gratefully received, and free papaers and posters are invited. Contact: **A. R. Lut**, secretary of the Swiss Pain League, Medicura, Medical Consulting, Florastrasse 30, CH-8008 Zurich, telehpone [0(041)1 383 02 02].



## XI World Congress of the International Federation of Physical Medicine and Rehabilitation (IFPMR)

*"Trends in the Physical Medicine"*

September 14th - 18th, 1992, Kulturpalast, **Dresden**, Germany.
For further information contact the secretariat Professor Jurgen Kleditzsch, Secretary of the XI World Congress of IFPMR, Medical Academy "Carl Custav Carus", Clinic of Orthopaedics, 74, Fetscherstrasse, Dresden GDR - 8019.

## Transport Accident Commission Rehabilitation Centre

*Lecture Series*

All sessions are to be held at 499 Springvale Road, Glen Waverley, **Victoria**, Australia.

April 24th, 1991.
Seminar - Knee Pain – Mr. David Young, Orthopaedic Surgeon and Dr. Rob Adler, Rehabilitation Specialist, Royal Talbot Hospital and Lee Eadie, Physiotherapist, TACRC.

May 29th, 1991.
Lecture - CT and MRI Scanning – Dr. Harold Fabrikant, Radiologist, Royal Melbourne Hospital.

June 26th, 1991.
Seminar - What has happened to RSI – Dr. Geoffrey Littlejohn, Rheumatologist, Monash Medical Centre, Dr. William Stone, Rehabilitation Specialist, Vocational Rehabilitation Services.

July 31st, 1991.
Lecture - Rehabilitation Research – Prof. Hugh Burry, Professor/Director of Rehabilitation Medicine, RMH/Essendon Hospital.

## The Spine Society of Australia

***Second Annual Scientific Meeting***

June 4 - 5, 1991.

For further information contact Robert D. Fraser, C/- Spinal Unit, Royal Adelaide Hospital, North Terrace, **Adelaide**, South Australia 5000.

## Back Pain – *Current Concepts and Recent Advances*

***Fourth International Meeting***

June 6 - 9, 1991. **Calgary**, Canada.

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# The Anatomy and Physiology of Nociception

**Nikolai Bogduk**
*Faculty of Medicine, University of Newcastle, N.S.W., 2308.*

## ABSTRACT

***Pain is defined as an unpleasant sensory and emotional experience associated with tissue damage. It is a psychic or cortical phenomenon evoked by information conveyed to the cortex by the neurological process known as nociception.***

***Nociception involves the detection of tissue damage ("transduction"), the transmission of nociceptive information along peripheral nerves, transmission in the central nervous system and modulation.***

***The anatomy and physiology of nociceptive mechanisms are described and management techniques based on them are discussed.***

## INTRODUCTION

Pain is the leading complaint for which patients attend medical practitioners or for which they are referred to physiotherapists. In some cases, a definite diagnosis may be made but in many others, particularly when the pain is related to the spine, a precise pathological diagnosis may not be possible. The practitioner is then left to deal with the pain symptomatically. To do this effectively requires knowledge of the anatomy, physiology and pathophysiology involved. This knowledge must be sufficient to identify those problems amenable to physical treatment modalities, to understand how such modalities relate to the mechanisms involved and to be able to decide when patients' problems are beyond the province of the practitioner's range of management skills.

## DEFINITION OF PAIN

In defining pain, the Taxonomy Committee of the International Association for the Study of Pain enunciated a critical concept: pain is not a sensation, it is an experience. The Taxonomy Committee defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".

Pain is thus a psychic or cortical phenomenon. Technically it does not exist unless and until the cerebral cortex receives information that evokes the experience of pain. Under normal circumstances this information arrives along peripheral nerves and the spinal cord from sites of actual or perceived tissue damage. This information, however, is not pain. Although it may lead to the experience of pain, until it does so it is only information about tissue damage. It is information that is evoked by noxious stimuli, and the process by which it is detected and transmitted is referred to as nociception.

Conventional physical treatment modalities do not deal with pain as such, unless the practitioner chooses to become involved with behavioural therapy, whereupon he enters the realm of psychologists and psychiatrists who deal with pain. What physical treatment does address, generally, is nociception. Techniques are aimed at the disorder causing pain or the transmission of nociceptive information from it. To many practitioners this distinction may seem only semantic or academic, but international authorities on pain take the distinction seriously and their approach should be heeded by those involved "at the coal-face".

Musculoskeletal medicine has evolved in the traditional medical model that maintains that if a patient's disorder can be cured or if nociception can be blocked, the patient's pain will be relieved. Notwithstanding the faith, conviction and good intentions of practitioners operating in this model, it is based on an incomplete picture of pain. Particularly in chronic pain, factors may operate that are beyond simple nociception. These include the patient's reaction to pain and its symbolic or material meaning to them; reactions of anxiety, anger and depression; the consequences of disability, loss of employment and self esteem; the frustration of not finding a "cure"; the seemingly perverse yet understandable advantages of a sick-role; the value of "pain" as a marketable commodity in a medical and legal market place; and the complexities of abnormal illness behaviour as a consequence of pain.

These dimensions of the pain problem are beyond the scope of this paper, but unless the treating practitioner is cogniscent of them he or she risks falling into the trap of seeing only the nociceptive perspective of a patient. By focussing exclusively on the physical, the practitioner may, through their own conviction, dedication and energy, unwittingly serve to reinforce the patient's somatosisation and disease conviction in situations where a physical approach is actually futile and where emphasis should instead be laid on the patient's coping abilities and responsibility for their own physical, psychological and social problems.

These comments notwithstanding, there is nevertheless a major place for dealing with nociception. Physical treatment has inherited a reputation of being relatively futile in the management of chronic pain but this reputation may not be due to the inappropriateness of a physical approach, but rather because nociceptive therapy has been applied poorly, irresponsibly and without insight. Thus, instead of deferring to contemporary pressures to transfer the treatment of chronic pain patients to psychologists, there is scope for musculoskeletal practitioners to improve their performance at a physical level based on a thorough understanding of nociception and its treatment.

## NOCICEPTION

The process of nociception involves several components: the detection of tissue damage (referred to as transduction); the transmission of nociceptive information along peripheral nerves; its transmission in the spinal cord; and its modulation. It is furthermore important to recognise that not all nociception involves peripheral tissue damage. Nociceptive signals can be initiated in damaged or diseased peripheral nerves or in the spinal cord whereupon, although the patient complains of pain in a particular part of the musculoskeletal system, its origin is not in that part but in some component of the peripheral or central nervous system that normally innervates that part.

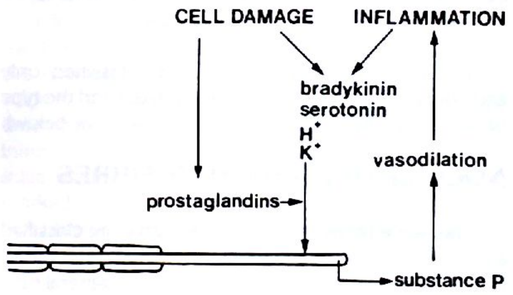


**Fig.1** *Chemical nociceptive transduction. A free nerve ending can be stimulated by chemicals released by damaged cells or inflammatory cells. Prostaglandins facilitate the effect of algogenic chemicals on nerve endings. Substance P, released from nociceptive terminals promotes vasodilation and reinforces the inflammatory response to tissue damage.*

## TRANSDUCTION

There are only two known mechanisms whereby nociception can be initiated by tissue damage or threatened tissue damage. These are chemical and mechanical mechanisms.

Chemical nociception is a process in which algogenic (pain-producing) chemicals are released in the region of nerve endings capable of detecting noxious stimuli. Chemicals which have this capacity include histamine, serotonin, hydrogen ions, potassium ions, bradykinin and adenosine diphosphate (Fig. 1). The common feature of these chemicals is that they are ones that typically are released by damaged tissue-cells or by inflammatory cells. Thus, chemical nociception occurs only in the presence of actual tissue damage. Related chemicals are prostaglandins and substance P.

Prostaglandins are synthesised from arachidonic acid and their synthesis is initiated by phospholipase A, an enzyme found in cell membranes and activated by membrane damage. However, in general, prostaglandins are not algogenic. The application of prostaglandins onto nociceptive nerve endings does not elicit pain but prostaglandins have the effect of facilitating the action of other algogenic chemicals on nerve endings.

Substance P is involved in nociception as a transmitter substance co-transmitter released from the central terminals of nociceptive axons (see below). However, it is also released from the peripheral terminals of nerves involved in nociception, i.e. when a nociceptive nerve is activated, substance P is released from both of its ends.

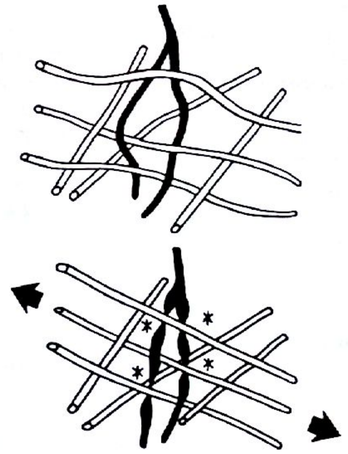

**Fig.2** *Mechanical nociceptive transduction. A: in a relaxed network of collagen fibres nerve terminals weave comfortably between the fibres. B: when the network is tensed, the collagen fibres are approximated and squeeze the nerve fibres which are activated at sites of compression (*).*

In the periphery, substance P does not activate nerve endings, but what it does do is cause vasodilation. In doing so, its role is to promote inflammation and tissue healing. In this respect, it is regarded as part of the nocifensive system, that component of the reaction to tissue damage designed to promote healing. In this way, nociceptive nerves are not simply transducers of tissue damage but are also actively involved in the repair process.

Mechanical nociception is a less well understood phenomenon because of the technical difficulties involved in studying it. It underlies pain that occurs in the absence of actual tissue damage but when tissues are being excessively strained. At a microscopic level, mechanical nociception occurs whenever collagen is excessively strained. Thereby, it is the basis for mechanical pain from ligaments, tendons and joint capsules, and from periosteum or skin if and when this is stretched.

The actual mechanism of mechanical nociception is unknown but would appear to be analagous to the operation of a Golgi tendon organ. In a network of collagen fibres at rest, nerve fibres and nerve endings weave comfortably through the interstices of the network (Fig. 2A). When the network is stretched, it is deformed and collagen fibres are approximated. This results in nerve fibres and nerve endings being squeezed between them (Fig. 2B). Presumably, this pressure activates the nerve fibres.

Mechanical nociception and chemical nociception may co-exist and each of the processes may account for different clinical features. Damaged tissue becomes inflamed and swells. This in turn stretches any surrounding collagen networks. Examples include an abscess, whose swelling stretches the dermis and epidermis, or an inflamed joint with an effusion that stretches the joint capsule. The chemicals released by the tissue damage or inflammation result in chemical nociception which accounts for sustained pain. If the tissue swelling reaches a critical threshold, mechanical nociception may be superimposed. Mechanical nociception is furthermore enhanced in two ways. First, the presence of algogenic chemicals sensitises nociceptive nerve endings rendering them more easily activated by mechanical stimuli. Secondly, although tissue swelling might of itself cause mechanical nociception by prestressing the collagen network, it renders the nerves in the network more easily stimulated by any additional stimulus. This becomes the mechanism of tenderness.

At rest, normal collagenous tissues have to be deformed greatly before they become painful, e.g. by an arm-lock in wrestling. However, if the tissue is prestressed by swelling, and/or if the nerve endings are sensitised by algogenic chemicals, even the slightest extra, external mechanical stress may activate the nociceptive nerve endings. This is why a tense abscess is painful and exquisitely tender but once it is lanced, tissue pressure is relieved and the site is markedly less tender. Similarly, swollen joints become less painful and less tender once aspirated.

The above combinations can be viewed as mechanical nociception and chemical nociception in series, i.e. chemical nociception predisposes to mechanical nociception. However, the two processes may also operate in parallel. Tissue damage may occur and attract an inflammatory response. Chemical nociception is established. Meanwhile, uninjured adjacent collagenous tissues remain intact. They are not necessarily swollen or tensed by the inflamed, injured tissues nearby, but their normal function is compromised. This phenomenon typically occurs in partially injured ligaments. The uninjured collagen fibres in the ligament are called upon to continue to bear the load normally borne by the entire, intact ligament. If this load is beyond their physiological capacity, mechanical nociception will ensue. A ligament that has suffered 75% damage has only 25% of its collagen intact. The threshold for mechanical nociception for these remaining fibres is consequently four times less than what it would have been for the entire ligament when it was intact. Under these circumstances, chemical nociception occurs at the injured part while mechanical nociception occurs in parallel at the uninjured, normal part of the ligament.

## NOCICEPTORS

In histological terms, there are no specialised receptors involved in either mechanical or chemical nociception. Specialised receptors are involved in detecting touch, temperature and pressure, but the nerve endings involved in nociception are free nerve endings that consist of either a single, tapering terminal or at most, a relatively simple arborisation of simple terminals.

To date, nociceptors have been classified only according to their response characteristics and the type of nerve fibre from which they are derived (see below).

## NOCICEPTIVE AFFERENT FIBRES

The nerve fibres in a peripheral nerve are classified according to their diameter and conduction velocity. Larger fibres conduct more rapidly and as a general rule, amongst myelinated axons the conduction velocity in metres per second is about six times their diameter in microns. When a mixed nerve is experimentally stimulated with an electrode, a compound action potential can be recorded at an arbitrary site some distance away from the stimulating electrode. The compound action potential is generated by the sum of the action potentials of the individual axons that constitute the nerve and is referred to as a neurogram. An archetypical neurogram is illustrated in Figure 3.



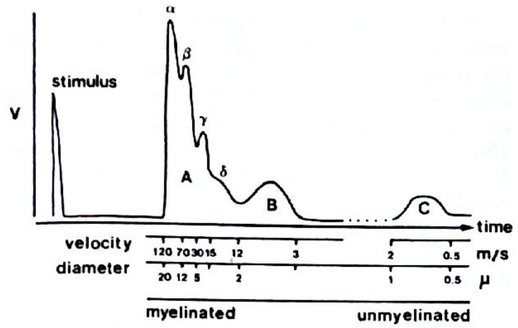


**Fig. 3** *A neurogram of a mixed peripheral nerve. The recording shows the amplitude of the compound action potential arriving at a recording electrode after an electrical stimulus that depolarises the nerve. Three waves of activity are detected (A, B and C) each generated by a respective population of axons characterised by different conduction velocities and fibre diameter. The A wave is subdivided into secondary peaks labelled α, β, γ and δ. The time axis is interrupted because C fibres are very slowly conducting and their action potential arrives much later than the A and B waves.*

The first action potentials to arrive at the recording electrode are those generated by rapidly-conducting axons; the last are those generated by slowly-conducting, unmyelinated nerve fibres. The neurogram exhibits three peaks, reflecting three populations of axons each with different conduction velocities. These are known as the A, B, and C waves. The C wave arrives very late after the A wave because of the much slower conduction velocity of unmyelinated fibres, and for this reason the time scale at the base of the neurogram has to be interrupted if the A and C waves are to be displayed to scale on the same diagram (Fig. 3). The A wave is marked by four secondary peaks generated by subclasses of A fibres known as Aα, Aβ, Aγ and Aδ fibres, each with a slightly slower conduction velocity.

B fibres are preganglionic, sympathetic neurons. Aα and Aγ fibres are efferent, motor fibres to skeletal muscle. Sensory fibres create the A β, Aδ and C waves.

Aβ fibres are typically sensory axons conveying sensations of touch, vibration, pressure and proprioception. They are not involved in nociception. Nociceptive axons are found only in the Aδ and C class of fibres, although these classes are not exclusively nociceptive.

The Aδ class of fibres includes axons that are not nociceptive. These are axons that innervate low threshold mechanoreceptors (receptors that respond to mechanical stimuli such as light pressure or touch) and thermoreceptors that respond to temperatures that are not perceived as noxious (Fig. 4). These neurons exhibit a graded response to non-noxious stimuli, i.e. as the strength of the stimulation increases, their frequency of discharge increases, but at certain thresholds short of what would be noxious, they either cease to be activated or they do not increase their frequency of discharge further (Fig. 4). Consequently, they cannot code for stimuli in the noxious range.

Nociceptive neurons of the Aδ class differ from these aforementioned fibres with respect to their threshold of stimulation and their response characteristics. They innervate receptors referred to as high threshold mechanoreceptors or high threshold thermoreceptors, meaning that they are activated by strong mechanical or thermal stimuli. What renders them nociceptive is that they continue to increase their frequency of discharge as the stimulus becomes noxious, i.e. they exhibit a graded response into the noxious range (Fig. 4). Nociceptive neurons thus operate in both the noxious and non-noxious range. No particular type of fibre is exclusively nociceptive. Furthermore, a variety of fibre types is capable of being nociceptive, depending on the type of stimulus to which it is sensitive and its threshold (Fig. 4).

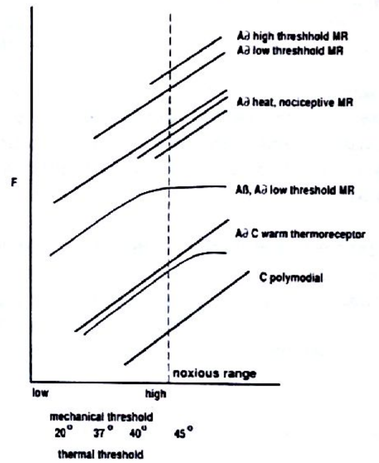


**Fig. 4** *Response characteristics of selected sensory fibres. The graph shows the thresholds and relative frequency of discharge (F) of nerve fibres connected to different types of receptors. The abscissa is calibrated for both mechanical thresholds and thermal thresholds. Low threshold mechanoreceptors (MR) are activated by light mechanical stimuli. High threshold receptors are activated by strong stimuli. Nociceptive fibres are those that exhibit a graded frequency response in the noxious range regardless of the nature of the stimulus or the threshold of the receptor. Non-nociceptive axons do not exhibit a graded response in the noxious range.*

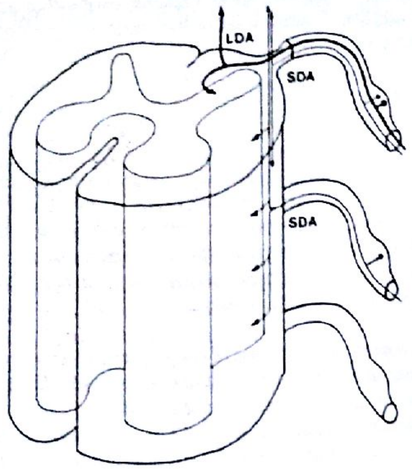


**Fig. 5** *The projections of primary afferent fibres in the spinal cord. Large diameter afferent fibres (LDA) are located dorsally (i.e. medially) in the dorsal root as it approaches the spinal cord. They sweep around the medial aspect of the dorsal horn to pass up the posterior columns but collaterals enter the base of the dorsal horn. Small diameter afferents (SDA) are located laterally in the dorsal root. Each divides into ascending and descending branches that travel in the dorsolateral tract and which send collateral branches into the grey matter of the dorsal horns 1-3 segments above and below the segment of entry of the primary afferent.*

Thus, there is no such thing as a "pain fibre" in the peripheral nervous system. All nociceptive neurons in the Aδ class subserve some other sensory function such as pressure, touch or temperature in addition to being nociceptive.

In experimental animals, C fibres occur with different response characteristics. However, in humans all C fibres have been found to be polymodal, i.e. they respond to a variety of stimuli including mechanical, thermal and chemical stimuli. These fibres are activated by innocuous and noxious stimuli in a graded fashion and are therefore nociceptive. C fibres are the smallest of axons in peripheral nerves but they are the most numerous. Thus, C fibres are the predominant type of nociceptive neuron.

## CENTRAL CONNECTIONS

The majority of sensory nerves enter the central nervous system through the dorsal root of a spinal nerve. However, some enter through the ventral root, their impulses travelling in the opposite direction to those of efferent fibres that constitute the major proportion of this root. Ventral root afferents have only started to be studied in recent years and the details of their central connections remain largely unexplored, but the connections of conventional dorsal root afferents are well determined.

Within a dorsal root, as it approaches the spinal cord, small diameter afferent fibres (Aδ and C fibres) segregate from large diameter afferents (Aβ fibres). Small diameter afferents assume a more lateral position in the terminal portion of the dorsal route leaving the large diameter afferents more posteriorly (Fig. 5). The large diameter afferents sweep around the posterior aspect of the dorsal horn continuing mainly up the posterior columns but also sending collateral branches into the middle of the dorsal horn at the levels of laminae IV, V and VI of the grey matter of the dorsal horn.

Small diameter afferents assume a more elaborate course. First, each fibre divides into ascending and descending branches that respectively pass rostrally and caudally along the outer surface of the apex of the dorsal horn in what is known as the dorsolateral tract (of Lissauer) (Fig. 5). Each branch ascends or descends for 1-3 segments. Along the entire intersegmental course of each branch, multiple collateral branches pass into the grey matter of the adjacent dorsal horn (Fig. 5). As a result of this extensive ramification, a given small diameter afferent fibre relays not just to one but to multiple segments at, above and below the segment at which it enters the spinal cord.

Within the spinal cord, second-order neurons capable of transmitting nociceptive information to higher centres are located at two main sites: in lamina I of the dorsal horn and in lamina V (Fig. 6). Other nociceptive neurons are located in lamina VII but have not been studied in any detail.

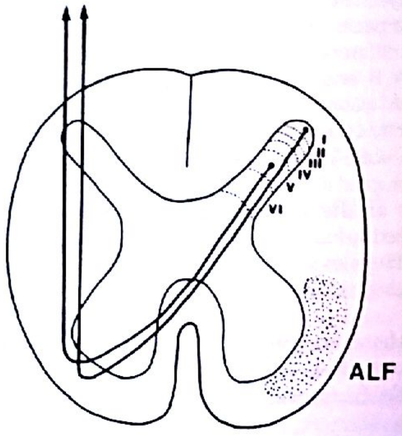


**Fig. 6** *Second-order nociceptive neurons. Neurons that transmit nociceptive information in the spinal cord have cell bodies located in lamina I and lamina V of the dorsal horn. Their axons cross the midline in the anterior white commissure and ascend through the spinal cord in the anterolateral funiculus (ALF).*



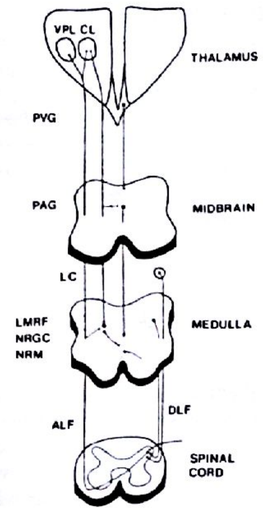


**Fig. 7** *Nociceptive tracts in the spinal cord and brainstem. Axons of nociceptive neurons in the dorsal horn pass upwards in the anterolateral funiculus to reach the ventral posterior lateral nucleus (VPL) and centralis lateralis nucleus (CL) of the thalamus. Collaterals from the ascending axons pass to the nucleus reticularis gigantocellularis (NRGC) in the medulla which in turn also projects to the CL nucleus. Descending pathways reach the dorsal horn along the dorsolateral funiculus (DLF) and arise in the nucleus raphe magnus (NRM), the lateral medullary reticular formation (LMRF) and the locus coeruleus (LC). Their activity is controlled by the NRGC, the peri-aqueductal grey matter (PAG) and periventricular grey matter (PVG).*

The nociceptive neurons of lamina I are large neurons. They are called marginal neurons because they occupy the marginal lamina of the dorsal horn and lie close to the junction of lamina I and lamina II. These neurons form axons that pass up the anterolateral funiculus of the spinal cord to the thalamus and reticular formation, as do the axons of nociceptive neurons of lamina V (Fig. 6 and 7).

Lamina V neurons are not exclusively nociceptive. They respond to a variety of stimuli including innocuous, mechanical stimuli as well as noxious stimuli. For this reason they are called wide dynamic range (WDR) neurons. The marginal neurons of lamina I are relatively more specific. Most are nociceptive-specific, responding only to noxious stimuli.

Connections are made between small diameter afferents and the neurons in lamina V and lamina I in a variety of direct and indirect ways (Fig. 8). Direct connections are made on the dendrites of lamina I neurons by small diameter afferents largely of the Aδ class that respond to mechanical stimuli. C fibres and other Aδ fibres terminate on small interneurons in lamina II.

Lamina II neurons consist mainly of two types: islet cells and stalked cells. Stalked cells are excitatory interneurons that receive the terminals of small diameter afferents either directly in the form of conventional axo-dendritic synapses, or in a more complicated fashion in complex structures known as glomeruli (Fig. 8).

The axons of stalked cells are distributed within their segment of origin and between segments. Some axons leave the dorsal horn and ascend or descend in the dorsolateral tract to enter lamina I of adjacent segments.

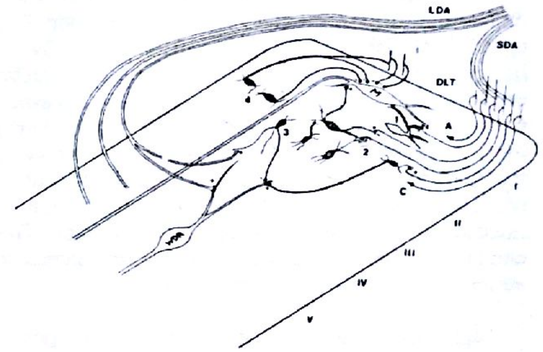


**Fig. 8** *Nociceptive connections in the dorsal horn. Large diameter afferents sweep around the medial aspect of the dorsal horn and enter lamina IV, V and VI. They terminate on dendrites of wide dynamic range neurons (WDR) located in lamina V. After travelling along the dorsolateral tract, small diameter afferents enter the dorsal horn. Ad fibres terminate on dendrites of marginal neurons located in lamina I. Other Ad fibres and C fibres terminate on dendrites of excitatory interneurons: stalked cells (s), in lamina II. The axons of stalked cells relay the marginal neurons and to WDR neurons. Inhibitory neurons in lamina I (1) provide post-synaptic inhibition of marginal neurons and presynaptic inhibition of primary afferents. Inhibitory neurons in lamina II: islet cells (i), control the interaction between primary afferents and stalked cells in complex structures known as glomeruli (2). Other inhibitory interneurons (3) are activated by large diameter afferents and inhibit the dendrites of WDR neurons that receive nociceptive information. Further interneurons (4) exert inhibitory effects on the cell bodies of marginal neurons both within the same segment and at adjacent segments.*

Either way, the axons typically terminate on the dendrites of a marginal neuron, thereby completing the connection to a second order nociceptive neuron.

Other stalked cells relay to the dendrites of WDR neurons of lamina V which spread from lamina V into the outer reaches of lamina III (Fig. 8). The same WDR neurons also receive terminals from large diameter afferents sweeping in from the posterior columns. Consequently, the WDR neurons receive both nociceptive and non-nociceptive information, and the neuron is capable of transmitting either type of information. The difference in information is coded by frequency; nociceptive information typically is transmitted as a high frequency discharge, while non-nociceptive information has a lower frequency. Thereby, higher centres receiving the axon from a WDR neuron can distinguish the nature of the information being relayed to them on the basis of frequency.

WDR neurons are connected so that they preferentially transmit non-nociceptive information. The terminals of a large diameter afferent aiming for a WDR neuron send collaterals to interneurons which inhibit those dendrites of the WDR neuron that receive axons from nociceptive stalked cells (Fig. 8). By inhibiting those dendrites the large diameter afferent fibre prevents the WDR neuron from receiving nociceptive information while still allowing the WDR neuron to respond to non-nociceptive information along other dendrites. This circuit provides a means for non-noxious stimuli to inhibit the transmission of noxious stimuli.

Islet cells in lamina II and other interneurons in lamina I have inhibitory functions related largely to descending modulatory pathways (see below). Lamina I interneurons exert post-synaptic inhibition of marginal neurons and pre-synaptic inhibition primary nociceptive afferents (Fig. 8). Inhibitory interneurons of lamina II send axons within their segment that synapse with the cell bodies of marginal neurons. Other branches of these axons enter the dorsolateral tract to reach marginal neurons at adjacent segmental levels.

These seemingly complex connections have an important role to play in sensory discrimination (see below) but they also underlie the mechanism of pain relief of certain therapeutic techniques such as acupuncture, transcutaneous electrical nerve stimulation and vibration (see below).

## TRANSMITTER SUBSTANCES

Within the central nervous system, small diameter afferent fibres release a variety of transmitter substances. It is not clear whether nociceptive axons release a single or a variety of transmitter substances nor what this substance is. However, it is known that they release a variety of neuropeptides, chemicals consisting of chains of amino acids of different lengths and composition. Some authorities believe these neuropeptides to be the transmitter substances of nociceptive axons; others believe they are only co-transmitters and the principal transmitter substance has yet to be discovered. The reluctance to acknowledge neuropeptides as the principle transmitter substances of nociceptive axons is based on the fact that when released onto second-order neurons these substances have a slow onset of action, insufficiently fast for them to account for the speed of detection of noxious stimuli. Nevertheless, neuropeptides have profound affects on nociceptive neurons that suggest they are involved in the nociceptive process in some way.

The most studied neuropeptide is substance P, a chain of 11 amino acids. This substance is found in many primary afferent fibres and when released onto the dendrites of second-order nociceptive neurons it excites them strongly. Other neuropeptides putatively involved in nociception include cholecystokinin, somatostatin, calcitonin gene related peptide, vaso-active intestinal polypeptide and neuropeptide Y, and there is some evidence to suggest that nociceptive afferent fibres from different locations and responding to different stimuli contain different peptides. Thus, chemoreceptive afferent fibres from viscera will have a different neuropeptide from that found in mechano-receptive nociceptors from skin. This proposition is still being explored.

## ASCENDING PATHWAYS

Traditional teaching has maintained that "pain" travels in the spinal cord along the lateral spinothalamic tract whereas the anterior spinothalamic tract conveys only pressure and touch sensations. In the face of contemporary knowledge of physiology this distinction is no longer appropriate. The axons of lamina I and lamina V neurons are scattered throughout the regions occupied by what formally would be known as the lateral and anterior spinothalamic tracts. They occupy most of the anterolateral quadrant of the spinal cord and constitute what is referred to as the anterolateral funiculus (Fig. 6). Within this funiculus the axons of WDR neurons convey nociceptive information as well as non-nociceptive information. There is no segregation of the two types of information. The difference is dictated only by the frequency of discharge in the axon and not by the type or location of the axon.

Most axons of lamina I and lamina V cross the midline in the anterior white commissure before passing rostrally along the spinal cord, whereupon nociceptive information passes along the spinal cord on the side opposite to that on which it entered the central nervous system. However, some axons do pass ipsilaterally, so that nociceptive transmission is not exclusively contralateral.

Other pathways for nociceptive information exist in the spinal cord, located in the depth of the lateral funiculus and in the posterior columns, but the details of these pathways have not been explored; nor has their clinical significance been determined beyond noting that pathways other than the anterolateral funiculus exist.

The anterolateral funiculus contains axons destined to reach the brain stem reticular formation and others directed to the thalamus. The former are referred to as spino-reticular fibres and the latter spino-thalamic fibres. In addition to reaching the thalamus, spino-thalamic fibres send collaterals into the reticular formation and these collaterals may also be referred to as spino-reticular (Fig. 7).

Within the thalamus, spino-thalamic fibres have two principal destinations: the ventral posterior lateral (VPL) nucleus and the nucleus centralis lateralis. The VPL nucleus is not particularly involved with the aversive, noxious aspects of the information received. This nucleus projects to the parietal lobe of the cerebral cortex which is responsible for determining the location of the origin of the stimulus. The aversive and emotive nature of pain is triggered by the centralis lateralis nucleus whose connections are not well known but are presumably to the limbic system of the brain, that portion responsible for pleasurable and aversive sensations and their associated visceral and emotional features. In other words, the projections of the centralis lateralis nucleus are responsible for the suffering aspects of pain.

Spino-reticular fibres have diverse connections within the brainstem. Some of these involve activation of descending pathways (see below). Others involve activation of brainstem centres that control respiratory and cardiovascular function, and are responsible for the cardiovascular reactions to nociception. Still others constitute upward, polysynaptic relays to the centralis lateralis nucleus that reinforce the effects exerted on this nucleus by spino-thalamic tracts.

Some authorities maintain that the anterolateral funiculus may be divided into an outer, lateral spinothalamic tract or neospinothalamic pathway and a deeper, medial spinothalamic tract or paleospinothalamic pathway. The principal distinction is that the neospinothalamic pathway predominantly has direct connections to the thalamus whereas the paleospinothalamic pathway maintains polysynaptic connections via the reticular formation. This distinction is largely conceptual for it does not correlate with any identifiable segregation of nociceptive axons on anatomical grounds. Rather, the distinction emphasises

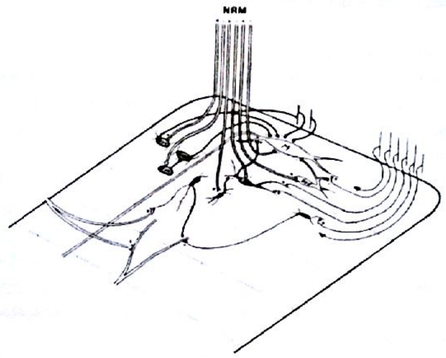


**Fig. 9** *Descending inhibition of nociceptive neurons. Descending neurons from the nucleus raphe magnus (NRM) establish various connections in the dorsal horn. Some axons directly inhibit marginal neurons. Others stimulate inhibitory neurons in lamina I that use dynorphin (DYN) as their transmitter to inhibit marginal neurons and primary nociceptive afferents. Descending inhibitory axons inhibit excitatory stalked cells (s) that relay primary nociceptive information to marginal neurons. Excitatory axons stimulate islet cells that inhibit stalked cells. Inhibitory axons inhibit enkephalinergic interneurons (ENK) that control other inhibitory neurons (GABA) that use gamma-aminobutyric acid as their transmitter to control marginal neurons. Excitatory axons stimulate enkephalinergic interneurons that inhibit marginal neurons.*

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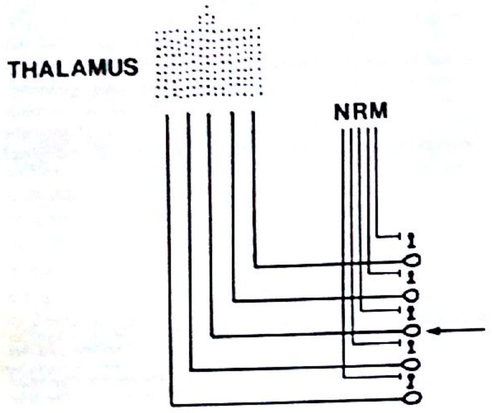
| β-endorphin | Tyr-Gly-Gly-Phe-Met-Thr-Ser-Glu-Lys-Ser-Gln-Thr-Pro-Leu-Val-Thr-Leu-Phe-Lys-Asn-Ala-Ile-Val-Lys-Asn-Ala-His-Lys-Gln-Gln |
| Met-enkephalin | Tyr-Gly-Gly-Phe-Met |
| Leu-enkephalin | Tyr-Gly-Gly-Phe-Leu |
| Dynorphin | Tyr-Gly-Gly-Phe-Leu-Arg-Arg-Ile-Arg-Pro-Lys-Leu-Lys-Tyr-Asp-Asn-Gln |

**Fig. 10** *The amino-acid sequences of the endorphins and enkephalins. Note the similarity in the first four and five amino-acids.*

the two routes by which axons in the anterolateral funiculus may reach the thalamus: a direct one and an indirect one via the reticular formation.

The reason for maintaining this distinction is that it is believed that the neospinothalamic pathway receives information predominantly from skin and from Aδ fibres, whereas the paleospinothalamic pathway receives information from deep structures and predominantly (although not exclusively) from C fibres. The paleospinothalamic pathway is the phylogenetically older pathway (hence its name). It subserves nociception from internal structures. The neospinothalamic pathway is more recently evolved and subserves nociception from those parts of the body that encounter the external environment.

External noxious stimuli delivered to skin pose an avoidable threat to the body. It therefore makes sense that information about such stimuli would be delivered directly to the thalamus through the neospinothalamic pathway. Furthermore, this pathway is highly organised somatotopically, i.e. there is faithful coding within the entire system for the origin of the stimulus. Consequently, the information is well localised and reaches the brain rapidly, whereupon it can be analysed and any external threat avoided.



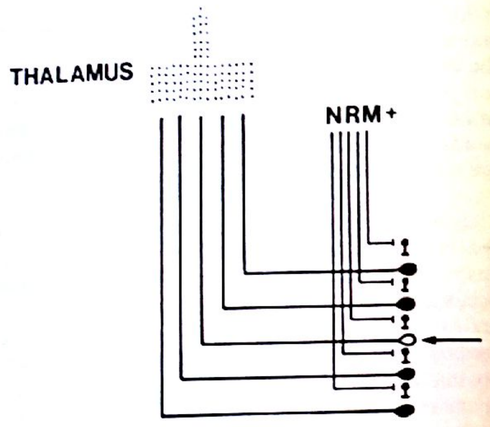




**Fig. 11** *The sensory discrimination mechanism. A: The diagram schematically illustrates an array of nociceptive neurons (open circles) whose axons project to the thalamus. These neurons are controlled by inhibitory interneurons (black circles) whose activity is controlled by the nucleus raphe magnus (NRM). A noxious stimulus delivered to the middle nociceptive neuron (arrow) evokes activity in the thalamus (dots) which is barely discernible amongst the background activity of surrounding neurons. B: If the NRM inhibits all neurons surrounding the one transmitting the nociceptive signal, their activity in the thalamus is suppressed whereupon the nociceptive signal stands out in contrast.*

In contrast, the paleospinothalamic pathway is poorly organised somatotopically. It involves multiple synapses as a result of which the origin of the stimulus is lost, although its aversive nature is nevertheless preserved. There is no need for accurate localisation within this system, for there is little benefit to be gained in creating and maintaining a complex system that codes the exact location of an internal noxious stimulus. After all, the organism can do little to avoid it. Moving simply takes the affected body part with it. This mechanism underlies the harrowing nature of visceral pain and musculoskeletal pain, for there is nothing the patient can do to escape the pain. No matter where they turn the pain stays with them. The only purpose of the paleospinothalamic pathway is to warn that the body is injured internally and should avoid stressing the affected part; the disease itself cannot be avoided.

## PAIN MODULATION

Within the brain stem are several centres that exert descending inhibitory influences on nociceptive primary afferents and on second-order nociceptive neurons. The principal centres are located in the periventricular grey matter, peri-aquaductal grey matter, the nucleus reticularis gigantocellularis, nucleus raphe magnus, the locus coeruleus and the lateral medullary reticular formation. Stimulation of these centres inhibits the transmission of nociceptive information between primary afferents and second order neurons in the spinal cord. Axons projecting to the spinal cord arise from the nucleus raphe magnus, the locus coeruleus and the lateral medullary reticular formation. These centres themselves are controlled by the peri-aquaductal grey matter and the nucleus reticularis gigantocellularis (Fig. 7).



Descending axons pass along the dorsolateral funiculus of the spinal cord and reach all segments of the spinal cord, entering lamina II of the dorsal horn (Fig. 7). Here they exert excitatory and inhibitory influences on nociceptive neurons both directly and indirectly via islet cells and stalked cells in lamina II and inhibitary interneurons in lamina I (Fig. 9).

Direct connections inhibit marginal neurons and excitatory stalked cells. Indirect connections excite inhibitory islet cells that postsynaptically inhibit marginal neurons. Other axons excite inhibitory interneurons in lamina I that presynaptically inhibit primary afferents reaching the dendrites of marginal neurons (Fig. 9).

These connections involve a variety of transmitter substances. Descending axons from the nucleus raphe magnus use serotonin as their transmitter substance. Axons from the lateral medullary reticular formation use noradrenaline. Inhibitory neurons in lamina II use enkephalin and GABA. Inhibitory neurons in lamina I use dynorphin.

## ENDORPHINS AND ENKEPHALINS

Particular transmitter substances which occur in the central nervous system belong to a class of chemicals known as endorphins and enkephalins. They are all polypeptides. Endorphin itself is a large molecule consisting of 31 amino acids. It is found largely in the pituitary gland where it is synthesised from pro-opiomelanocortin (the precursor of ACTH and MSH). Endorphin has analgetic properties but it is not a conventional neurotransmitter. It is released largely in conjunction with ACTH into the blood stream and presumably has a role in providing analgesia during periods of major stress. It is therefore more a hormone than a transmitter substance.

The enkephalins are short chain peptides consisting of only 5 amino acids. The two main varieties are leucine-enkephalin and methionine-enkephalin. Their sequences are illustrated in Figure 10. What is striking is that the first four amino acids of enkephalin are identical to the terminal sequence of endorphin and it is this set of amino acids that confers analgetic properties to both endorphin and the enkephalins.

The enkephalins are transmitter substances. They are found in numerous locations throughout the body and not just in the nociceptive system. They occur in the cerebral cortex, the basal ganglia, in sympathetic ganglia and in the myenteric plexus of the gut. In the nociceptive system they occur in the peri-aquaductal grey matter, the nucleus raphe magnus and in lamina II of the dorsal horn. Whatever their location, enkephalins act as inhibitory transmitter substances causing hyper-polarisation of post-synaptic membranes.

Dynorphin is another peptide consisting of seventeen amino acids, the terminal five of which are in the same sequence as methionine-enkephalin. It too is inhibitory and has analgetic properties.

The analgetic properties of the enkephalins and dynorphin arise because they are transmitter substances in the nociceptive system. They occur at sites where the inhibitory influences they exert inhibit pain perception or block nociceptive transmission. The naturally occurring and synthetic narcotics such as morphine and pethidine act as analgesics because when they enter the central nervous system they mimic the inhibitory effects of enkephalins and dynorphin and thereby block "pain" (see below).

## SENSORY DISCRIMINATION

The existence of descending pathways capable of inhibiting nociception raises questions as to their natural function. It is tempting to believe that the body is equipped with its own analgetic system. However, this is manifestly not the case. Were it so, the body would automatically inhibit pain but the existence of prolonged pain and chronic pain reveals that this is not the case. The function of the descending pathways is more sophisticated. It is part of the body's sensory discriminatory mechanism designed to filter out unwanted sensory information and to highlight information in which the brain might be interested.

At rest, the descending pathways are tonically active; they maintain a certain level of inhibition of the connections between primary afferent fibres and second-order neurons in the spinal cord. When nociceptive information enters the spinal cord it competes for recognition with other sensory information reaching the brain. Activation of the spino-thalamic tract is not enough in this regard (Fig. 11A). Unless the information is somehow highlighted it may be missed. In the case of a nociceptive signal, the body cannot afford to miss information about a threatening stimulus, so the central nervous system enhances the perception of the signal.

In addition to reaching the thalamus, nociceptive information uses spino-reticular axons or the collaterals of spinothalamic axons to activate the nucleus reticularis gigantocellularis. This in turn activates the nucleus raphe magnus and the lateral medullary reticular formation either directly or via the peri-aquaductal grey matter. The net result is that the tonic influence of the descending pathways is modified. The modification is such that the tonic inhibition of the spinal cord segments receiving the nociceptive information is reduced while adjoining segments are inhibited further. The effect is to highlight the incoming signal by enhancing its transmission while suppressing surrounding inputs (Fig. 11B). This latter effect suppresses information that

constitutes "background noise" and which might otherwise obscure perception of the incoming signal. This entire process is an example of centre-surround inhibition, a process used in many other sensory systems such as the eye and the posterior columns to enhance a desired signal by surrounding it with a field of inhibition. In other words, the central nervous system enhances the contrast of the incoming signal.

The role of the descending pathways is thus to subserve sensory discrimination and not to provide the body with analgesia. It is only fortuitous that various therapeutic techniques can tap into this system and provide analgesia. They do this not by recruiting or mimicking the body's own pain relief system but by artificially disturbing its sensory discrimination system (see below).

## REFERRED PAIN

Clinicians have laboured under a misconception about the perception of pain if they have expected that the patient suffering pain should be able to indicate where it is coming from. The natural consequence of this misconception has been that when referred pain occurs it suggests that something different, something special, is happening.

Referred pain is pain perceived as arising in a location other than that of the source of pain. For example, pain may be perceived in the umbilical region when its source is in the appendix; pain may occur in the shoulder when its cause is blood irritating the abdominal surface of the diaphragm. In the case of musculoskeletal pain, pain may be felt in the buttock when its source is in the lumbar spine; pain may be felt in the head when its source is in the upper cervical spine. These seemingly bizarre associations invite an explanation. They imply that some special mechanism must be operating to generate referred pain but the mechanism of referred pain has for many years remained elusive. Yet, the problem lies not in failing to find a mechanism but in having a misconception about pain and its localisation. The only form of pain that is felt locally is pain from skin. All other pain (from deep tissues) is referred pain to a greater or lesser extent.

When a noxious stimulus is delivered to skin it is accurately localised for two reasons. First, the neospinothalamic pathways that mediate cutaneous nociception are highly organised somatotopically. The pathways code for the location of the stimulus and the information is delivered to the VPL nucleus of the thalamus and the parietal lobe (see above). Secondly, very few external noxious stimuli selectively affect only nociceptors; touch receptors and pressure receptors are also activated by mechanical noxious stimuli. Impulses from these non-nociceptive receptors are delivered to the VPL nucleus of the thalamus by the posterior columns which again are highly organised somatotopically, more so than the neospinothalamic pathways. Thus, the thalamus and brain receive two complementary sets of information that incorporate codes as to the location of the source of the noxious stimulus, whereby the brain registers virtually the exact point of stimulation on the skin. Indeed, the role of the posterior columns is such that without them the ability to identify the source of noxious cutaneous stimuli is reduced by half.

It is appropriate for cutaneous noxious stimuli to be well localised for they pose a threat to the organism. By identifying the exact source of stimulation the organism can take action to avoid the stimulus. The same does not apply for deep, internal noxious stimuli.

From a point of view of design, an organism does not need to accurately localise internal noxious stimuli for there is little it can do to avoid them; it cannot turn or run away; it cannot brush the stimulus away. All its requires is a general idea of the source of the pain so that it can avoid aggravating the pain. This is provided by the paleospinothalamic pathway.

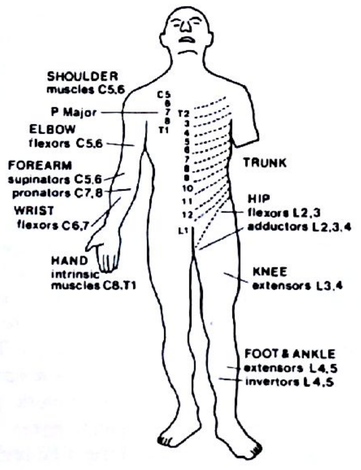
The spino-reticulo-thalamic connections of the paleospinothalamic pathways provide information to the thalamus and brain that a noxious event is occurring. This establishes the appropriate alarm and concern. The localisation they provide is relatively poor but nevertheless is sufficient for the concerns of the organism. As a general rule, the spino-reticular system provides localisation of the stimulus to within about one neural segment. Thus, instead of being able to identify the exact point of stimulation, the brain registers the signal as arising from somewhere within those structures supplied by a certain spinal cord segment.

Thus, for example, a patient may feel pain coming from the knee joint but cannot specify that it arises specifically from the anterior cruciate ligament. As a rule, deep muscle pain is not perceived from the site of stimulation within the muscle but usually as if it is coming from the joint that the muscle moves. The deeper and more obscure and unfamiliar the source of pain, the less distinct its localisation. The pain may be centred around the source but the boundaries of the pain are indistinct and extend beyond the limits of its source. This is not so much a characteristic of referred pain as it is of musculoskeletal pain and other deep pain in general, be it referred or not.

The reasons for this are twofold. First, the spino-reticular system does not code for the location of deep noxious stimuli as well as the neospinothalamic pathways do for cutaneous stimuli. Secondly, and moreover, deep pain does not involve the activation of non-nociceptive cutaneous receptors which of their own accord provide localising information to supplement that carried by the nociceptive pathways. In other words, deep pain is recognised as unpleasant but lacks accompanying information as to where it is actually coming from.

It is only in the case of joints that additional information about location may be provided. Proprioception travels along the posterior columns and is well organised somatotopically. Thus, a patient will be able to localise joint pain because moving the joint aggravates the pain. Whereas the nociceptive pathway provides information about the nature of the noxious stimulus, it is the posterior columns that provide parallel information as to its origin. In registering movement at the same time as aggravation of the pain, the brain is able to deduce that the structure that is moving is the source of the pain. This facility is less available for muscles and not at all for bones, for bones do not move and are not endowed with proprioceptors. As a rule, the more a structure lacks cutaneous and proprioceptive innervation the less apparatus it has whereby to localise pain, and the more indistinct the perception of noxious stimuli arising from it.

Patients have the ability to explore their injured parts. This can provide sensory cues. If the pain arises from a deep structure near to skin, palpation of the part evokes tenderness. Consequently, the source of pain may be localised on the basis of localising information from the skin that was palpated. By simultaneously receiving nociceptive information and touch information the brain is able to deduce that the source of the pain must be near the site that was touched. This facility is not available for deeper structures. No amount of touching skin aggravates the pain. The patient is frustrated in their exploration and is left with an unclear message as to the source of the pain.




Perhaps the most striking example in this regard is the pain of sphenoid sinusitis. The sphenoid sinus is the deepest portion of the head; it cannot be palpated and anyone who has not studied anatomy would not know what the sphenoid sinus is, let alone where it is. The bone does not move and so has no proprioceptive innervation. Consequently, the pain is poorly localised. The patient can only report that it is somewhere in the field of the trigeminal nerve, i.e. somewhere in the head. The complaint is therefore solely one of headache.

Similar remarks apply to varieties of spinal pain. If the pain stems from a deeply located joint that cannot be palpated, whose whereabouts are unfamiliar to the patient and whose proprioceptive output is only one part of the diffuse proprioceptive output from the vertebral column, there simply is no means whereby the patient can specify the actual origin of the pain. The best he can report is that the pain arises somewhere from within the neural segment or segments that happen to innervate the affected joint.

In these terms, it is evident that no special mechanism operates for referred pain; it is simply a reflection of the lack of localising information accompanying deep nociception in general. The deeper and more inaccessible the source of pain is, the more indistinct are noxious stimuli arising from it ,whereupon, the more widespread is the complaint of pain. In such situations it is the role of the clinician to use a knowledge of anatomy to identify the possible sources of pain and in this regard a knowledge of the segmental innervation of the body becomes paramount.

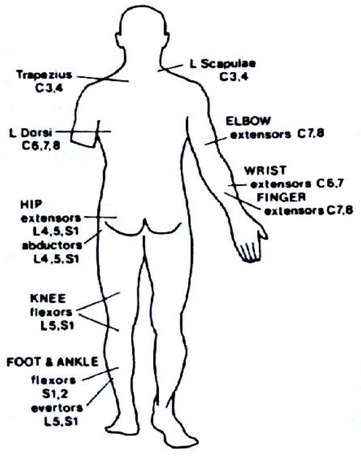



**Fig. 12** *A pictorial summary of the segmental innervation of the trunk and the major muscles and joints of the limbs.*

The segmental innervation in question is not that of the dermatomes. Deep pain is not perceived in the skin; it is perceived deeply. What is therefore required is a knowledge of the deep segmental innervation of the body. In the case of the trunk, the pattern is straightforward. Virtually the entire trunk is supplied by intercostal nerves. The body parts (muscles, bones, joints and ligaments) innervated by an intercostal nerve are those lying in or limiting its intercostal space (Fig. 12). Thus, tracing the T4 intercostal space for example, detects all the structures innervated by the T4 nerve.

The lower six intercostal nerves leave the chest and continue into the abdominal wall, but do so in parallel bands. The location of these bands is indicated by the direction of the respective ribs (although not the costal cartilages). Whereas the lower costal cartilages turn upwards towards the sternum, their respective ribs point downwards and forwards towards the abdominal wall. It is this same direction that the lower intercostal nerves follow. Thus, the 10th rib points towards the umbilicus and the umbilical region is innervated by the T10 intercostal nerve. The 12th rib points downwards towards the groin and the suprapubic region is innervated by T12. The L1 segment is simply the next in sequence and innervates a band of tissue ending in the inguinal region (Fig. 12).

Deep pain arising in any of the thoracic or L1 segments will project into the corresponding segment of the trunk wall as described above. Thus, when a patient indicates pain in a particular trunk segment or segments the diagnostic exercise requires first recognising which segments are being indicated and then a deduction of what other tissues are innervated by the same segment, and which therefore might be the actual source of pain. This may be any of the bones, ligaments, muscles or joints innervated by the segment, including structures in the vertebral column as well as in the trunk wall, but in the case of trunk pain it also includes viscera. The interpretation of trunk pain therefore requires a knowledge of the segmental innervation of viscera.

The innervation of viscera is not constant nor is it accurately known, but sufficient knowledge is available to enable the formulation of worthwhile clinical rules (Fig. 13).

All thoracic viscera are innervated by T1-T4 whereupon pain from any of these viscera will be perceived over the T1-T4 segments of the trunk, i.e. centrally over the chest or over the upper lateral chest wall.

The innervation of the abdominal viscera is essentially that of the alimentary tract. This tract is innervated sequentially from proximal to distal regardless of the actual location of the viscus. The thoracic oesophagus is

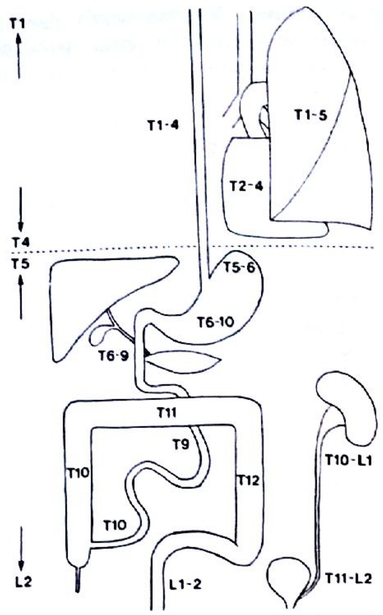



**Fig. 13** *A pictorial summary of the segmental innervation of the thoracic and abdominal viscera.*

innervated by T1-4. The gastro-oesophageal junction is innervated by T5-6. The stomach itself is innervated by segments T6-10. The duodenum and its derivatives (liver, bile ducts, gall bladder and pancreas) are innervated by T6-9. The rest of the small intestines is innervated by segments T9-10 with the T10 segment focussing over the terminal ileum and ileocaecal junction (hence the appendix belongs to T10). From proximal to distal, the ascending, transverse and descending colon is innervated by segments T10-12, while the sigmoid colon and rectum is innervated by L1-2. The kidneys, ureter and bladder are innervated by segments T10-L2 with the more proximal portions of the urinary tract receiving a higher segmental innervation (T10-L1) than the terminal ureter and bladder (T11-L2). Other pelvic organs are innervated by L1 and L2.

Given this pattern of innervation, it is evident that gastric pain will be perceived over segments T7 $\pm$ 2 of the trunk, i.e. in the epigastric region. In this case the organ in question happens to underlie the region of pain but this is fortuitous. In the case of the transverse colon, the structure may lie in the epigastric region or the umbilical region but its innervation is about T11, whereupon its pain is perceived lower in the abdomen and not in the epigastrium. Renal and ureteric pain occurs in the loin and travels to the inguinal region. This reflects the course and distribution of the T12 and L1 nerves in the trunk wall.



In the case of the limbs, the pattern of segmental innervation is topographically more complicated, for in the course of the development of the limbs what were once segmental bands in the embryo become twisted, mixed and convoluted in the formed limb. Nevertheless, a reasonable guide to the segmental innervation of limbs can be gained from a knowledge of myotomes (the segmental innervation of muscles). These are summarised in Figure 12.

From this guide it is evident that the "top" of the shoulder is formed by trapezius (C3,4) and levator scapulae (C3,4). Note also that the diaphragm is innervated by the phrenic nerve (C3,4,5) whereupon it is evident that pain from the diaphragm will be perceived in the shoulder, not because the shoulder is covered by the C3,4 dermatome (as is commonly taught) but because the deep tissues in this region belong to C3 and C4.

Pain stemming from cervical structures innervated by C5, 6 will be referred to any of those portions of the upper limb that receive a similar innervation, i.e. the shoulder, the arm, the elbow and the forearm. Lower cervical segments may refer pain to the arm, forearm, wrist and hand.

In this context it is noteworthy that the pectoralis major muscle is innervated by segments C5,6,7,8 and T1 but the muscle itself lies over the anterior chest wall. Therefore, quite apart from visceral pain referred to the chest wall (T1-4), chest pain invites a consideration of neck pain from segments C5,6 and below referred to the pectoralis major.

In the lower limb it is evident that lower lumbar segments of the vertebral column share the same innervation as most of the muscles of the gluteal region and posterior thigh whereupon the buttock and thighs are the most common sites for referred pain from the lower lumbar spine. The L5 and S1 segments are also represented in the leg and ankle whereupon it is possible for low lumbar pain to be referred distally into the lower limb.

## NEUROGENIC PAIN

Under normal circumstances, nociception involves the detection of damage or threatened damage in tissues innervated by the peripheral nervous system. However, in certain circumstances pain may be caused by nociceptive activity generated within nerves themselves. Peripheral tissues are not damaged and nerve endings are not stimulated but nociceptive nerves are activated somewhere along their course. An archetypical example of neurogenic pain is that arising from a traumatic or surgically-induced neuroma. When a peripheral nerve is cut or otherwise disrupted, within hours axon filaments sprout from the proximal end of the nerve. Their purpose is to reach the distal stump of the nerve and to re-establish the connection. When this is successful the nerve regenerates and function is restored. However, the reconnection may be thwarted. The gap between the proximal and distal stumps may be too large, because of retraction of the two ends, or the gap may become filled with scar. Under such conditions the axon sprouts grow aimlessly forming a tangle of filaments that constitute a lump on the nerve: the neuroma. The critical requirements for neuroma formation are that the continuity of axons must be broken and the epineurial sheath must be breached. The latter allows axon sprouts to wander beyond the confines of the nerve. If the epineurium remains intact it confines the axon sprouts and directs them along and within the sheath to the distal stumps of the axons.

Not all neuromas become painful. Accurate figures are not available but perhaps some 10 - 15% become painful. The mechanisms for the pain are several. First, neuromas are exquisitely sensitive to mechanical stimuli. They are also sensitive to circulating noradrenaline. Furthermore, they can become spontaneously active. Whatever the mechanism, activity in the damaged neurons is generated at the site of injury. However, although generated in the neuroma the activity is perceived as arising from the tissues that normally would have been innervated by the affected nerve. This is because the activity generated uses the same nerves and the same central connections that would be used by nociceptive information arising from peripheral tissue damage but the brain has no way of determining that the origin of the information is actually from a neuroma and not from the peripheral tissues.

Other sources of neurogenic pain are dorsal root ganglia. Impulses generated in a ganglion will be perceived as having arisen in those structures innervated by the spinal nerve to which the dorsal root ganglion belongs. In the case of spinal nerves that contribute to the brachial or lumbosacral plexuses the pain is perceived in the corresponding limb. Pain involving thoracic dorsal root ganglia is perceived around the chest wall or abdominal wall.

Ectopic activity in dorsal root ganglia may be evoked by a variety of means. Dorsal root ganglia are sensitive to mechanical stimulation, so that compression of a dorsal root ganglion by a disc prolapse, for example, may elicit bursts of neural activity. Alternatively, the ganglion may be pressed against a prolapse or an osteophyte as the nerve root moves during flexion or extension movements of the spine. Ganglion cells may be affected by inflammatory disorders, the archetypical example of which is herpes zoster or "shingles". A ganglion may become involved secondarily in inflammatory reactions to herniated disc material. If rendered ischaemic, dorsal root ganglion cells may fire spontaneously. This occurs in tabes dorsalis and when

arteritis affects radicular vessels. It is also believed that nerve root ischaemia rather than simple mechanical pressure may be an operant factor in radiculopathies due to disc prolapse and foraminal stenosis.

To distinguish its origin, pain stemming from a dorsal root ganglion may be referred to as radicular pain and it differs in quality from musculoskeletal pain. Radicular pain is lancinating in quality and is perceived as travelling through the tissues supplied by the affected nerve, typically along a narrow band. This contrasts with the constant, deep, dull aching pain of musculoskeletal origin. Furthermore, referred pain is not purely nociceptive. Axons other than nociceptive axons are also triggered by the causative disorder. Consequently, the sensation has components beyond those purely of pain but the patient is usually unaware of these. The massive discharge in many axons of different types is so alarming and overwhelmingly unpleasant that the entire sensation is described as painful, although close enquiry of the patient can reveal that the sensation is different from what they would ordinarily describe simply as pain.

Traditional wisdom maintains that nerve root compression can be a source of pain but contemporary evidence refutes this. Mechanical stimulation of normal nerve roots does not evoke nociceptive activity. Only if nerve roots have previously been damaged are they capable of being painful. Otherwise, those pain states conventionally ascribed to nerve root compression are more likely to represent radicular pain due to some disorder affecting a dorsal root ganglion.

## CENTRAL PAIN

Another form of neurogenic pain can arise from cells within the central nervous system. It occurs when second or third-order neurons in the nociceptive system lose their accustomed afferent input. This is reflected by the alternative nomenclature of "deafferentation pain", but the term "central pain" is preferred because it specifies the origin of the nociceptive signal as being in the central nervous system.

When neurons lose their afferent input it appears that they undergo several physiological changes. They cease to maintain receptors and their membrane characteristics change, whereupon they become spontaneously active and unresponsive to excitatory or inhibitory inputs. It is as if the neuron responds to the lack of input by no longer maintaining receptors. In more physiological terms, it is likely that transmitter substances released by peripheral nerves play a humoral role in maintaining receptors on second-order neurons. When peripheral nerves are destroyed or disconnected from the spinal cord, their transmitters are no longer supplied to the receptors and the maintaining, humoral effect is lost.

Central pain is the mechanism for phantom limb pain, paraplegic pain, the pain of brachial plexus avulsion and later post-herpetic neuralgia. The common feature of all these conditions is that peripheral nerves are destroyed or disconnected from the central nervous system, whereupon cells in the central nervous system lose their accustomed input. Another example is thalamic syndrome, usually caused by an infarction, in which it seems that neurons in the thalamic nuclei of the nociceptive system become spontaneously active as a result of loss of an accustomed inhibitory input or loss of an accustomed input from nociceptive tracts.

Central pain is notoriously difficult to treat for it is not due to a peripheral nociceptive input. It represents spontaneous activity within the central nervous system due to lack of peripheral input. Furthermore, once central neurons cease to maintain receptors they become unresponsive to pharmacological therapies that rely on mimicking the effect of inhibitory transmitter substances. In particular, if cells cease to maintain opiate receptors for enkephalin and dynorphin, they become unresponsive to morphine and other narcotics.

Of particular concern to practitioners of musculoskeletal medicine and physiotherapy is growing speculation that central pain may be the operant mechanism in conditions other than those involving major nerve injury. There is increasing evidence that some forms of joint pain to a greater or lesser degree involve changes in WDR neurons similar to those seen in central pain. Thus, some of the features of the pain of arthritis may not be nociceptive in the classical sense but involve an element of central pain. It behoves practitioners in the field to stay abreast of developments on the neurophysiology of joint pain for it may transpire that classical interpretations and therapeutic approaches may need to be modified, focussing less on peripheral nociception and more on central mechanisms.

## SEROTONINOPATHY

There is increasing speculation that another form of central pain may occur. Nociceptive pathways are kept tonically suppressed by descending inputs from the brainstem and amongst these inputs are ones that use serotonin as their transmitter substance (see above). Although details have not been established, in principle it seems plausible that failure of serotonin to maintain this inhibition may create a disturbance in the sensory discrimination mechanism that controls the perception of pain such that an illusion of pain is produced. In physiological terms this pain is illusory for it does not represent actual nociceptive activity entering the central nervous system. Rather, the imbalance in the central nervous system creates a pattern of activity that the brain misinterprets as pain. To the patient, however, the pain is nonetheless real and distressing, if not severe, for even though there is no peripheral nociceptive input, the pattern generated within the nervous system is still real neuralgia.

The basis for this disturbance appears to be some form of chronic or periodic disturbance of serotonin metabolism or that of a closely related substance. As levels of serotonin in neurons fall or as serotonin becomes less effective, its inhibitory influence on nociceptive neurons decreases, thereby decreasing the balance of tonic inhibitory effects on nociceptive pathways.

There is evidence to suggest that this mechanism underlies common complaints such as tension headache and migraine. In the case of migraine, it appears that headache occurs because of periodic failure of the locus coeruleus and raphe nuclei to suppress activity in the C2-3 segments of the spinal cord. The vascular features of migraine are simply a parallel phenomenon, reflecting disturbances in the control of cerebral and cranial blood vessels by the locus coeruleus and raphe nuclei, but these vascular changes themselves do not cause the pain.

Serotonin is implicated in the pathogenesis of migraine not only by its presence in nociceptive pathways and the response of migraine to serotonergic drugs but also by the associated features of migraine such as mood disturbance, changes in appetite and vomiting, all of which processes involve serotonin or a related substance.

A similar relationship may apply for the mysterious yet seemingly common condition of fibromyalgia. This complaint is characterised by pain seemingly arising from muscle, associated with disturbances of mood, sleep and other functions. The common link is serotonin. Investigations of such patients have failed to reveal any primary disturbance in the affected muscles, yet the patients show disturbances of serotonin metabolism. A marker of this disorder is an increase in serotonin receptors on platelets. Increases in receptors are a sign of chronic deficiency of the transmitter substance that acts on them, which suggests that fibromyalgia is not a peripheral nociceptive problem but a pain state due to deficiency of the level of serotonin within the central nervous system.

Similar inferences can be drawn about tension headache. Investigations have failed to reveal consistent abnormalities of personality or muscle activity to justify ascribing tension headache either to psychological problems or to chronic muscle contraction. Yet, there is mounting evidence that tension headache is associated with abnormalities of serotonin metabolism. Tension headache may therefore constitute one extreme of a spectrum of disorders including fibromyalgia and migraine which are caused by central metabolic disturbances and not by peripheral nociception.

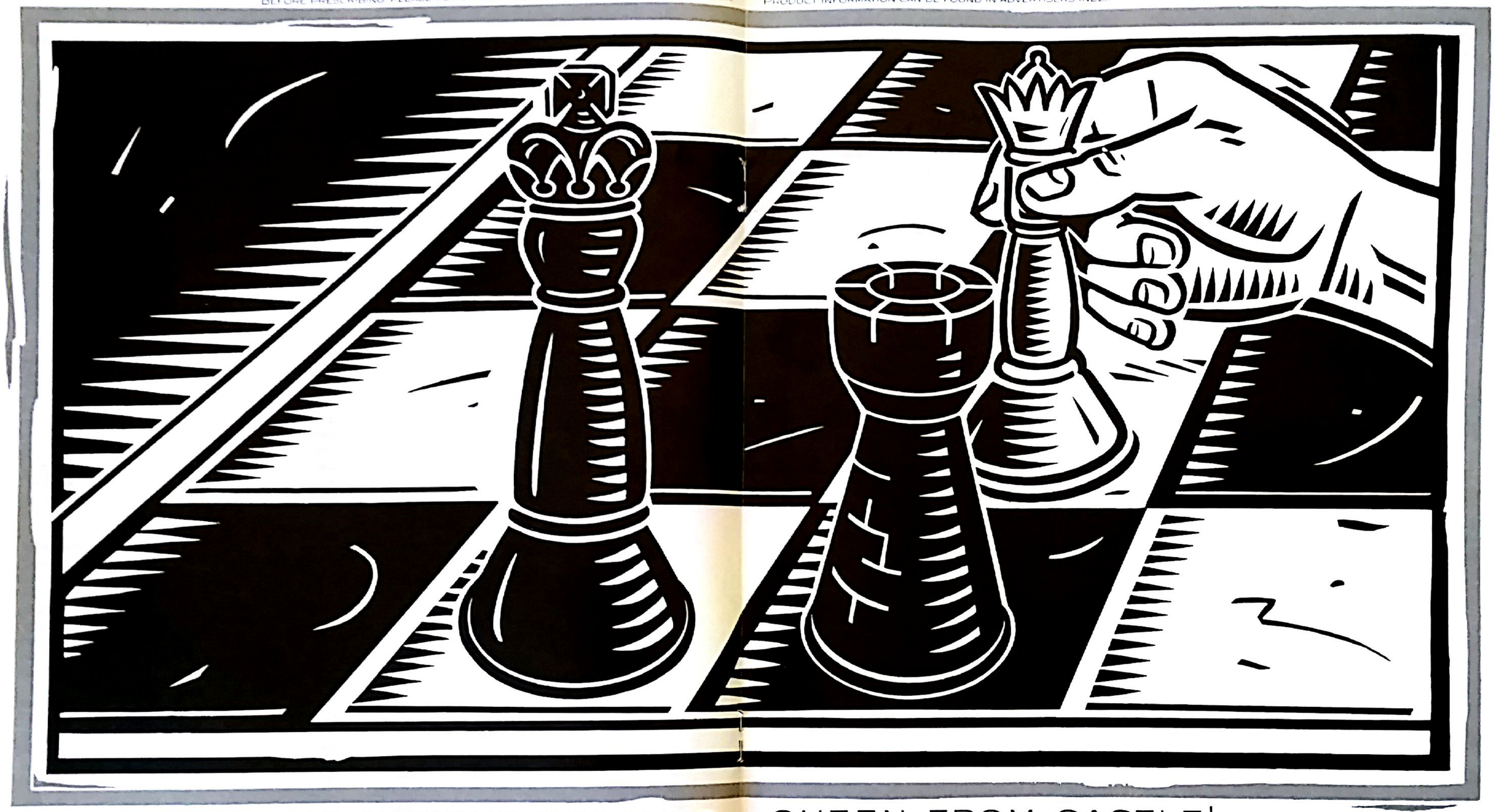
## REFLEX SYMPATHETIC DYSTROPHY

Apart from causing pain, tissue damage evokes a repair response. At the site of injury, this involves a cellular response that constitutes inflammation. Inflammatory cells such as neutrophils and macrophages invade the region to remove cellular debris. Cells are attracted to the site of injury by chemotactic factors and their delivery is enhanced by vasodilation. At a local level, vasodilation is promoted by chemotactic factors and by substance P released from nociceptive nerve endings. Vasodilation is further promoted by the sympathetic nervous system which increases blood flow to the injured region. However, in certain pain states this sympathetic response can be disturbed. For reasons not yet understood, certain injuries evoke a bizarre reaction in the sympathetic nervous system consisting in its early phases of excessive vasodilatory response followed later by a severe vasoconstrictive response.

Pain problems involving the sympathetic nervous system are known by a variety of names; the two most commonly used are reflex sympathetic dystrophy (RSD) and causalgia. Causalgia typically occurs following a partial injury to a major peripheral nerve such as the median nerve or the sciatic nerve. In contrast, RSD typically follows some form of musculoskeletal injury which itself may be relatively quite trivial, like fingers being jammed in a door. Otherwise, the two conditions are virtually identical in their subsequent manifestations and associated features (Table 1).

The patient complains of severe burning pain over a wide area of the affected limb or body part, associated with increased sensitivity to stimuli in the skin; the threshold for noxious stimuli is lowered and even innocuous stimuli become painful (allodynia). The affected limb exhibits vasodilation; it becomes red, hot and sweaty; muscles become tender and spastic; joints become swollen and bones become hyperaemic especially towards their ends. Biopsies reveal proliferation of synovial cells and fibroblasts in the affected joints. This "angry" phase of sympathetic activity lasts for some weeks or months only to be replaced by a cold, deathly vasoconstrictive phase. The limb becomes colder, blue and withered; muscles atrophy and become fibrous; joints stiffen, bones becomes osteoporotic; and trophic changes in the skin, hair and nails set in.

The pain in these conditions is most likely a form of central pain. In the case of causalgia the nerve injury causing deafferentation is obvious. In the case of RSD, nerve injury has not been documented but could well be present in deep sensory nerves that innervated the original site of musculoskeletal injury but which lacked a



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cutaneous distribution, wherefore the nerve injury is not apparent on clinical testing. In some cases, RSD may occur after injury or diseases involving the central nervous system such as multiple sclerosis or infarctions. In some cases it may follow ischaemic heart disease.

The associated features all seem to be mediated by the sympathetic nervous system. The cutaneous hypersensitivity is produced by noradrenaline released from sympathetic nerves sensitising peripheral nociceptors and touch receptors, rendering them more easily and more strongly activated. The changes in skin, muscle, joints and bones are due to excessive vasodilation in these tissues but may also reflect a direct trophic effect of sympathetic nerves. The later, atrophic features are the converse of this vasodilation.

The role of the sympathetic nervous system in these conditions is indicated by the fact that if performed early in the condition, regional sympathetic nerve blocks or sympathectomy can abolish the neurological sensitivity and vasodilatory features. However, in established cases the changes may be irreversible.

In these conditions, although the sympathetic nervous system is involved, it does not transmit the pain. The pain occurs independently and appears to involve WDR neurons in the spinal cord. However, the sympathetic nervous system perpetuates the pain by reinforcing the sensitised peripheral input to these neurons.

## NOCICEPTIVE THERAPY

Nociception can be managed at several levels, either singly or in combination. Therapy can be directed to the disease process, injury or cause of pain; to the transduction process; to the peripheral transmission of nociceptive information; to its central transmission; and to its modulation (Fig. 14).

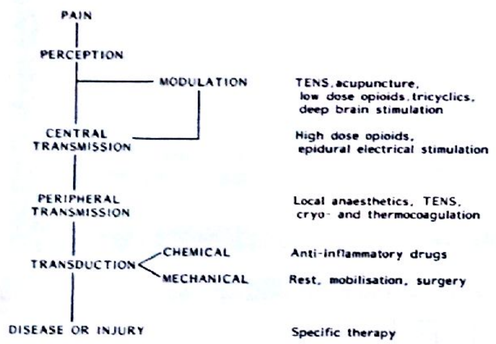


**Fig. 14** *A tabular summary of the process of nociception and pain perception and the sites at which different forms of therapy can be applied.*

### Disease Process

Therapy directed at the disease process is the ideal, for if tissue damage can be resolved the trigger for nociception is removed. This can be done for relatively simple problems like sports injuries where in time tissue-healing occurs spontaneously. A therapist may then only have a role in attempting to accelerate or to optimise natural healing. In infectious diseases, a physician may assist the natural healing process by administering antibiotics, by lancing an abscess or by aspirating a septic joint. However, in many instances there may be no means by which the disease process can be reversed.

Some conditions lend themselves to radical excision, such as cholecystectomy for gall bladder disease, appendicectomy for appendicitis, or excision of a peptic ulcer. However, such options apply largely for visceral diseases. In musculoskeletal disorders the option for radical surgery still applies but is not necessarily as universally successful as for visceral diseases. Painful joints may be debrided, fused or excised and replaced. In the case of joints of the appendicular skeleton, joint surgery is reasonably successful but nevertheless is attended by limitations and significant risks of morbidity. In the case of spinal pain, arthrodesis has an unproven and controversial status.

### Transduction

When a disease or injury cannot itself be addressed, or can only be treated partially by medical or surgical means, the option arises of treating the pain at the transduction process.

For chemical nociception, pharmacological tools are available. Drugs like aspirin and its cogeners inhibit prostaglandin synthesis. By eliminating prostaglandins they remove their facilitatory effect on other algogenic chemicals. This may be useful in reducing chemical nociception but it does not eliminate it altogether. Corticosteroids have a more profound effect because they stabilise cell membranes thereby not only preventing the activation of phospholipase A (see above) but also inhibiting the release of lysosomal enzymes from inflammatory cells. However, because of their side effects, corticosteroids are indicated for only certain inflammatory pain conditions.

Apart from aspirin cogeners and corticosteroids, there are no drugs in general use whose action is on the transduction process of nociception. There is perhaps a place for the development of drugs that might inhibit bradykinin or serotonin in the periphery but none is yet available.



There are no drugs available that can inhibit the transduction of mechanical nociception. It is therefore futile to attempt to treat mechanical nociception with peripherally-acting drugs. Mechanical transduction can only be treated by correcting the mechanical abnormality triggering nociception.

### Peripheral Transmission

If nociception cannot be blocked at the transduction level there is scope for blocking its transmission along peripheral nerves. The aim is to stop conduction of action potentials. This can be done using drugs like local anaesthetics but their usefulness is limited by their short duration of action. The major role of nerve blocks using local anaesthetics is diagnostic: to establish which particular nerve or nerves are mediating the pain, whereupon other forms of therapy might be directed specifically at the nerves involved. However, some pain problems (and typically those involving muscles) can exhibit a lasting response to a single or to a series of injections of local anaesthetic. The temporary relief afforded by the local anaesthetic appears to provide a temporary respite during which the cause of pain mysteriously and spontaneously resolves. Such conditions are probably ones in which some minor mechanical disturbance causes muscles or parts of muscles to go into spasm and become painful. The spasm perpetuates the mechanical disturbance but if the pain is relieved the spasm abates and the mechanical disturbance spontaneously resolves. If it does not, the pain reappears and is only temporarily responsive to local anaesthetics.

Transcutaneous electrical nerve stimulation (TENS) is another means of blocking peripheral transmission. The exact mechanism by which TENS operates is still controversial. Both central and peripheral mechanisms may be involved. At the periphery, the dispute concerns whether nociceptive nerves are saturated or fatigued by the electrical stimulation rendering them incapable of nociception, or whether the electrical stimulus simply disturbs the frequency of discharges in peripheral axons of all types thereby decoding the nociceptive signal and replacing it with "noise" that is perceived as a tingle instead of pain.

Whatever the exact mechanism, TENS can be a powerful tool for relieving pain. It works best when the electrodes can be placed over a nerve at a site between the source of pain and the central nervous system. It can still work in other situations by stimulating branches of the same spinal nerve as the one that innervates the source of pain but if the electrodes are peripheral to the source of pain the analgetic potency is much less. It has also been demonstrated that TENS exerts good effects when applied over acupuncture points.

Peripheral nociception can be blocked surgically. In the past, neurosurgeons were wont to transect nerves in an attempt to relieve nociception. This approach is no longer favoured because of the risks of inducing neuroma formation or central pain, which essentially replace the original pain with what is usually a worse form of pain. Instead of peripheral nerves being cut, they can be frozen or coagulated using needle-like electrodes inserted percutaneously onto the nerve.

Freezing nerves (cryo-neurotomy) turns intracellular water into ice which fractures the axon membranes. This interrupts conduction in the nerve for about six weeks or so, after which time the membrane heals and conduction is restored. Such treatment is not particularly useful for chronic pain but it can play a valuable role in conditions that are painful but destined to resolve in a matter of weeks. In the case of fractured ribs, intercostal nerves may be frozen to provide prolonged analgesia over the period it takes for the rib to heal.

Nerves can be thermo-coagulated using electrodes that transmit a radiofrequency current (percutaneous radiofrequency neurotomy). The electrode heats the nerve over a small area and denatures its proteins. Conduction remains blocked until the nerve repairs which, depending on the length of nerve coagulated, may take up to twelve months or more. This form of therapy has the prospect of providing valuable long term analgesia, although it does not constitute a permanent cure for the pain.

### Central Transmission

The classical approach to stopping central transmission of nociceptive information is spinothalamic tractotomy, a procedure in which the anterolateral funiculus is interrupted surgically (either by incision or by radiofrequency neurotomy). This form of therapy has been reserved largely for cancer pain in patients with a limited life expectancy, for after about twelve months the effects of the operation wear off. The reasons for this are unclear but hypotheses include nociceptive transmission along pathways other than the anterolateral funiculus (see above) or the onset of thalamic pain (see above).

Related to TENS is epidural electrical stimulation, a technique in which electrodes are introduced like a pacemaker into the epidural space and used to stimulate the spinal cord. The effect of the electrical current is to block nociceptive transmission. Its mechanism is unclear but may involve either the recruitment of descending inhibitory pathways or simply disturbing the frequency code of nociceptive information. Clinically, the effect is that of blanketing the region of pain with a sensation of tingling. This form of therapy has a limited efficacy for muscloskeletal pain but appears more efficacious for neurogenic pain.

## Pain Modulation

The strongest and most widely applied forms of nociceptive therapy operate by interfering with the sensory discrimination mechanism for pain. These include drug therapy, acupuncture, deep brain stimulation and possibly also TENS and dorsal column stimulation.

Morphine and other opiates are the strongest, most widely available analgesic drugs. They operate in either of two ways depending on the dose and route of administration.

In conventional, small systemic doses (taken orally or by injection), morphine interferes with the supraspinal tonic inhibitory control of nociceptive transmission. It inhibits the tonic inhibition, thereby raising the "background noise" of sensory information entering the spinal cord. This does not block peripheral nociceptive input but obscures it. The nociceptive signal is still present but the brain cannot discriminate it from amongst the heightened level of background activity in the spinal cord, whereupon pain ceases to be evoked.

In higher doses, or if morphine is delivered by injection into the epidural or subarachnoid space, the drug reaches synapses in the spinal cord that involve enkephalin or dynorphin. Here, the morphine mimicks the inhibitory action of these transmitter substances on nociceptive neurons. In essence it acts like a false transmitter substance. It is able to do so because although enkephalin and dynorphin are chemically different to morphine, the three-dimensional shape of morphine is virtually identical to that of enkephalin and dynorphin, i.e. their active radicals are spaced in exactly the same configuration. Consequently, enkephalin and dynorphin receptors on nociceptive neurons cannot distinguish whether it is morphine or enkephalin acting on them and they proceed to hyperpolarise the neurons thereby blocking nociceptive transmission.

Tricyclic anti-depressant drugs, like amitriptyline, are believed to offer an analgesic effect through their action on serotonin. They block the re-uptake of serotonin after it has been released from descending inhibitory axons in the spinal cord. Consequently, the serotonin remains in the synapse longer and exerts its inhibitory affect for longer. This appears to be the mechanism by which these drugs provide pain relief in conditions such as tension headache, migraine and fibromyalgia, and why tricyclics can be a useful adjunct in the treatment of non-specific back pain.

Research into the physiology of acupuncture indicates that it has an effect similar to that of morphine. Acupuncture operates largely by the mechanism of diffuse noxious inhibitory control. This is a process by which a new, noxious stimulus triggers the sensory discrimination mechanism to enhance its own input but in so doing it inhibits any antecedent input to other spinal cord segments. In a patient with ongoing pain mediated by a particular spinal cord segment, the sensory discrimination mechanism will be operating to highlight that input. When an acupuncture signal is then delivered to some distant site, its input switches the sensory discrimination mechanism to enhance the acupuncture signal, and in so doing it suppresses all other activity in the spinal cord including the pre-existing pain.

Since it involves the sensory discrimination mechanism, acupuncture involves the serotonergic and noradrenergic descending pathways and enkephalinergic interneurons. Obliterating these constituents of the nociceptive system or blocking their transmitter substances abolishes the effects of acupuncture.

The supreme form of pain control is deep brain stimulation. Electrodes may be introduced into the periventricular grey matter or periaquaductal grey matter and used to activate the entire descending inhibitory system of the brainstem. This provides a profound analgesia but this form of therapy is not widely practised because of the delicate surgery involved, the risks of morbidity, the costs involved and the after care required.

A variant of deep brain stimulation is a procedure in which the electrode is directed not at the periventricular grey matter but into the thalamus near the terminals of the spinothalamic tract. Stimulation at this site blocks the reception of nociceptive information by the thalamus. This form of stimulation is particularly useful for central pain problems.

## Neurogenic Pain

The treatment of neuromas is difficult and controversial. Over the years, various conservative and surgical therapies have been explored including hammering the neuroma, injecting it with alcohol, phenol, steroids and other sclerosants, surgical excision, burying the neuroma in bone, and encasing it in perspex or the like. Burying the neuroma reduces its exposure to mechanical stimuli but does not address its spontaneous activity. Sclerosants can have only a temporary effect for the neuroma regenerates. Excising the neuroma simply invites a fresh neuroma at the site of transection of the nerve. Microsurgical ligation has been advocated and is logical in the sense that it aims to enclose the proximal stump of the nerve in its own epineurial sheath to prevent outgrowth of axon filaments; but despite encouraging reports by its originators this procedure has not attracted widespread endorsement.

Radicular pain is difficult to treat conservatively. Ideally, the cause of pain should be eliminated if possible.



Typically, this can be done surgically in the case of osteophytes, foraminal stenosis or disc prolapse. Chemonucleolysis constitutes an alternative, less invasive means of treating certain forms of disc prolapse. It operates by dissolving the prolapsed material. There is no evidence that traction, aimed at "opening up" narrowed intervertebral foramina, has any value in the treatment of radicular pain other than providing a temporary relief (if that) while the patient is under traction. Any foraminal enlargement provided by traction is summarily reversed upon resuming the upright posture.

If the cause of radiculopathy cannot be reversed, radicular pain is virtually intractable. Some authorities advocate the use of membrane stabilising drugs like carbamazepine, dilantin or clonazepam in an attempt to reduce spontaneous activity in dorsal root ganglion cells but the efficacy of these drugs is not reliable.

Radiculopathy due to auto-immune arteritis or inflammation is amenable to corticosteroids which may be taken orally or injected epidurally on to the affected nerve root. In the case of post-herpetic neuralgia, amitriptyline is the only drug of the many that have been tried that has been shown to have any substantial beneficial effect.

In general, physical therapy, acupuncture and electrical therapy (other than epidural stimulation) are notoriously ineffective for neurogenic pain.

## Central Pain

Central pain is difficult to treat because it is not due to peripheral nociceptive input. Consequently, therapy directed at nociceptive transduction or peripheral transmission is futile. Furthermore, if the spontaneously active neurons in the central nervous system cease to maintain receptors, central pain will be unresponsive to any of the pain modulation therapies (see above). If the nociceptive neurons lack opiate receptors and serotonin receptors they will be unresponsive to morphine, tricyclics, acupuncture and TENS. This is particularly relevant to practitioners who may be called upon to deal with patients suffering from central pain but who have been misdiagnosed as having some form of obscure musculoskeletal pain.

From a conservative perspective, distraction appears to be the single most effective treatment for central pain. The more the patient dwells on their pain, the more they suffer. If patients can be distracted from their pain and gainfully employed, they become less aware of the pain and suffer less. Temporary relief from severe central pain can at times be achieved by intravenous infusion of lignocaine and sometimes the relief can be long-lasting.

From a surgical perspective, central pain can be treated by epidural electrical stimulation or by deep brain stimulation if facilities for these procedures are available. A relatively new surgical therapy is dorsal root entry zone lesions. In conditions like brachial plexus avulsion and post-herpetic neuralgia, where central pain stems from spontaneously active second-order neurons in the outer layers of the dorsal horn, the pain may be stopped by coagulating these cells with fine radiofrequency electrodes introduced under direct vision into the spinal cord in the region of the entry zone of the dorsal root of the affected spinal cord segment.

## Reflex Sympathetic Dystrophy

RSD and causalgia are miserable pain problems. Not only does the patient suffer severe burning pain, he cannot bear for the affected part to the touched, and meanwhile the musculoskeletal elements and skin undergo progressive trophic changes culminating in a withered, useless limb.

Physical methods are not applicable for the treatment of the actual pain (the mainstay are sympathetic blocks and drug therapy aimed at the sympathetic overactivity). However, physical therapy has a crucial role to play in maintaining the integrity of the affected limb: to prevent muscle atrophy and joint contractures, so that if the pain can in due course be relieved, the patient is not left with a useless limb.

The difficulty faced by the practitioner, however, is the inaccessibility of the limb. It is too sensitive and painful to be touched, even brushed, let alone manipulated. The cardinal recourse is to institute physical therapy while the affected limb is anaesthetised or under some form of sympathetic blockade. This reduces the sensitivity and allows the muscle and joint problems to be treated. This combined approach requires co-ordination and co-operation between the musculoskeletal therapist and the rest of the medical team. When undertaken, the results are most rewarding, yet tragically, because of logistic difficulties or because of uninformed reluctance on the part of some doctors, this type of therapy is all to often denied to patients.

## EPILOGUE

It is perhaps fitting to close with the preceding example of RSD, for no condition better illustrates the utility of a musculoskeletal practitioner having insight into a pain problem. By understanding the mechanism of RSD, he should recognise that physical modalities have little value for the pain itself, but nonetheless have a crucial role in preserving the limb. Moreover, he should realise that other medical assistance may be required to institute local anaesthetic blockade to enable physical treatment. Uninformed colleagues may not have considered this. Wherefore, the responsibility may

fall to the musculoskeletal practitioner to become the patient's advocate. In order to do this effectively, he must be conversant with all of the dimensions of the problem and be able to explain this to the patient and to colleagues. On the basis of a thorough knowledge of the physiology of pain, the musculoskeletal practitioner should be able to indicate in a rational and logical manner what can and needs to be done, and not simply plead that something should be done by someone who knows more about the problem than themselves.

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**TABLE 1**
**CLINICAL FEATURES OF**
**CAUSALGIA AND REFLEX SYMPATHETIC DYSTROPHY**

**NEUROLOGICAL FEATURES**
burning pain, hyperaesthesia, hyperalgesia, allodynia.

**SYMPATHETIC FEATURES**

| | EARLY | INTERMEDIATE | LATE |
|---|---|---|---|
| SKIN | warm | cold | cold |
| | red | cyanotic | pale |
| | | glazed | smooth |
| HAIR | | loss | denuded |
| NAILS | | brittle | brittle |
| SUBCUTANEOUS | oedema | brawny | atrophy |
| JOINTS | swollen | thick | fibrous |
| | tender | stiff | ankylosed |
| MUSCLES | spasm | wasting | atrophic |
| BONES | hyperaemic | osteoporotic | atrophic |

# Modulation of Spinal Pain by Epidural Electrical Stimulation

**Umberto Rossi**

*Knox Hospital, 262 Mountain Highway, Wantirna. Vic. 3152*

## ABSTRACT

***New therapeutic modalities have emerged from the exploitation of the neuromodulation mechanisms which regulate the endogenous antinociceptive systems.***

***Neuroaugmentive surgery induces amplification of normally existent inhibitory activity by electrical stimulation of the nervous system at peripheral, spinal or cerebral levels.***

***This presentation focuses on the use of epidural spinal electrical stimulation (ESES) as symptomatic treatment of intractable lumbar pain syndromes.***

***A retrospective study of seventy-six implanted cases with a follow-up between three months and seven years suggests that this treatment modality is effective and relatively safe. The overall results were good to excellent in 60% of cases and fair in 13%. The failure rate was 26%.***

***There was a strong correlation between aetiology, location of pain and success rate. Preferred cases have neurogenic pain with leg predominance.***

***From a limited experience it appears that sympathetically maintained lumbar pain responds well to ESES.***

***Key words: Pain modulation - Neuroaugmentation - Spinal cord stimulation - Sympathetically maintained pain - Failed back surgery syndrome.***

## INTRODUCTION

Sensory modulation is a stabilising factor in nervous system function. It ensures homeostatic balance in most, if not all, sensory systems. From common observations and anectodal reports sensory modulation can also be implicated in the experience of pain.

An individual may perceive more or less pain from a given stimulus depending on the situation in which it occurs or the state of mind at the moment. A soldier wounded in battle or an athlete injured in competitive sport may feel little or no pain. Different individuals may display variable tolerance to the same painful stimulus depending on cultural learning. Non-painful factors such as skin rubbing, massage, vibration and manipulation may reduce or abolish pain.

It used to be taught that pain is a modality of sensation conducted along a specific linear transmission system. Based on this concept classic surgical treatments were aimed at interrupting putative pain pathways. However, the destructive procedures did not produce persistent results. The influence of extrinsic or intrinsic processes on the awareness of pain found no explanation within the "specificity" theory.

In recent times considerable basic scientific and clinical research has been devoted to the neuronal mechanisms of pain and has led to a new conceptualisation which emphasises dynamic controls of pain transmission and perception subserved by "gating" systems.

This new concept not only provides a basis for the understanding of how cognitive activites may influence somatic sensory input but also helps to interpret the control of pain by non-painful events[1].

It is now recognised that spinal neuronal systems which are excited by pain signals are also facilitated or inhibited by non-painful stimuli. This competitive interaction is under the influence of descending inhibiting mechanisms which are mediated by morphine-like substances synthetised in the brain[2].

Neuromodulation is regarded as the resulting amplification or dampening of nervous system functioning that occurs with the activation of the anti-nociceptive systems. In these systems there is a preponderance of inhibiting activity and modulation principally operates by activating inhibitory circuits.



A new therapeutic dimension has emerged from the intentional manipulation of the modulatory mechanisms. It is called neuro-augmentation. This denotes amplification of normally existant inhibitory activity. It is induced by electrical stimulation of the nervous system at peripheral, spinal or cerebral levels.

Within the limits of the assigned topic, this presentation focuses on the symptomatic treatment of intractable spinal pain syndromes with epidural spinal electrical stimulation (ESES).

## HISTORICAL BACKGROUND

In 1967 the American neurosurgeon, Norman Shealy, implanted the first dorsal column stimulator for inhibition of pTin. this innovative technique was based on experimental studies in animals which suggested that spinal cord transmission of responses evoked by noxious stimuli was suppressed by electrical stimulation. It evolved directly from the gate control theory of pain[3] which postulated that quantitative superiority of non painful stimuli at spinal gates would overload the circuit and prevent pain signals from reaching the brain.

Initial technical problems were overcome by the improvement of techniques and complications were reduced by the placement of electrodes in the epidural space. The method gained wider acceptance in the early 1980s with the availability of wire-type electrodes which could be introduced percutaneously for intraoperative testing and trial stimulation.

Several thousand implants have been performed to this date worldwide for different indications. A recent review has revealed that in 65% of the cases the indication for the use of ESES was low back pain[4].

## MATERIALS AND METHODS

The purpose of this retrospective study was to evaluate the efficacy of ESES; to define its therapeutic benefits in relation to the diagnostic categories; to assess the complications and to delineate the determinants of success.

The study population included eighty cases from a personal series of patients who received a permanent implant between July, 1983, and August, 1990. Pertinent demographic data is presented in *Table 1*.

Four patients were lost to followup, leaving seventy-six cases for the final evaluation.

In all cases chronic disabling low back and/or leg pain, refractory to various treatments, had been present for at least twelve months. The majority of patients had undergone previous spinal operations or surgical treatments for pain. Persistent nerve root compression or spinal stenosis had been excluded clinically and radiologically. Stabilising procedures had been considered in a few cases but had eventually been declined by the referring orthopaedic surgeons.

*Table 1*

**STUDY POPULATION**

| | | |
|---|---|---|
| • | Number of cases implanted | 80 |
| • | Number of cases evaluated | 76 |
| | Male | 30 |
| | Female | 46 |
| • | Age (in years) | |
| | Range | 19-71 |
| | Mean | 43 |
| • | Pain duration (in years) | |
| | Range | 1-42 |
| | Mean | 7.8 |
| • | Previous surgeries for pain | |
| | Range | 1-9 |
| | Mean | 2.5 |
| • | Follow-up (in months) | |
| | Range | 3-84 |
| | Mean | 28 |

About one third of patients had become drug dependent. The majority could not be considered "employable".

## OPERATIVE TECHNIQUES

The operative techniques described by Ray[5, 6], with minor modifications, were adopted throughout.

An implant procedure includes three basic steps:

1.) placement of the electrode
2.) trial stimulation
3.) internalisation

Two basic approaches to the epidural space are used:

a. through a Tuohy needle for the placement of wire-type electrodes
b. through a laminotomy and flavotomy for the placement of plate-type electrodes.

The initial phase of the operation at least is performed with the patient awake but sedated.

The epidural space is probed with a Seldinger wire or a curved plastic dissector before inserting the electrode.

The correct location for the final placement of the electrode is identified by the response to the trial stimulation, which is performed at various levels until a good pattern of evoked paraesthesia is perceived in all painful areas.

In all cases of this series the interlaminar space was surgically exposed and the electrode was anchored to the yellow ligament in order to prevent displacement during the early post-operative phase.

The internalisation of the complete system which includes the electrode, the connecting leads and the stimulator, can be performed in one stage. Alternatively extension wires can be externalised through the skin for a continuation of the trial stimulation over a few days and the system can then be internalised at a second stage.

In all but three cases of this series, Medtronic equipment was used. Two thirds of the electrodes were of the plate-type *RESUME*, one third were of the wire-type *QUAD*. Until recently, radiofrequency linked externally powered stimulators of the type SE4 were utilised but now the fully implantable pulse generator type *ITREL II* is preferred.

## RESULTS

The data for the evaluation was collected by a registered nurse using a patient-completed questionnaire and supplementary telephone interviews. The criteria for scoring the results were: pre- and post-implant pain profile, drug intake, sleep pattern and ability to perform daily activities.

The efficacy was assessed in terms of percentage of pain relief as perceived by the patient. The result was classified as excellent if the patient had reported 75% to 100% relief of pain; good 50% to 75%; fair 25% to 50%; failure 0 to 25%. Cases with good relief of leg pain but inadequate relief of low back pain were allocated to the group of "fair result". The validity of assessment was confirmed if improvement of pain intensity was corroborated by an equivalent change in the dose, frequency and type of medication.

There was diversity in the aetiology and distribution of pain; however, the pre-implant intensity of pain was uniformly severe. The largest subgroup consisted of sixty-nine patients with failed spinal surgery, including arachnoiditis, peridural fibrosis and intraneural fibrosis. Forty of these cases (58%) had a good to excellent result *(Table 2)*. Interestingly in the same subgroup only a few patients with a fair result had stopped stimulation altogether, suggesting that even a low degree of pain reduction was perceived as helpful.

The overall results were good to excellent in 61% of cases and fair in 13%. The failure rate was 26% *(Table 3)*.

*Table 2*

**RESULTS IN RELATION TO AETIOLOGY**

| Diagnostic Category | Number of cases | Pain Relief % Excellent | Good | Fair | Failure |
|---|---|---|---|---|---|
| Failed Back Surgery Syndrome | 69 | 38% | 20% | 13% | 29% |
| Idiopathic Low Back Pain Syndrome | 2 | - | 50% | 50% | - |
| Sympathetically Maintained Lumbar Syndrome | 4 | 75% | 25% | - | - |
| Post-surgical Lumbar Neuralgia | 1 | 100% | - | - | - |



*Table 3*

**CLINICAL RESULTS**

| Time Since Implant | Number of cases | Pain Relief * Excellent | Good | Fair | Failure |
|---|---|---|---|---|---|
| 3 to 6 months | 16 | 8 | 6 | 1 | 1 |
| 6 months to 1 year | 6 | 3 | 1 | 1 | 1 |
| 1 year to 2 years | 21 | 8 | 4 | 4 | 5 |
| 2 years to 7 years | 33 | 11 | 5 | 4 | 13 |
| | 76 | 30 (39.47%) | 16 (21.05%) | 10 (13.15%) | 20 (26.30%) |

* Excellent pain relief defined as 75% to 100%; good as 50% to 75%; fair as 25% to 50%; failure as 0 to 25%.

*Table 4*

**RESULTS IN RELATION TO LOCATION OF PAIN**

| Distribution of Pain | Number of cases | Pain Relief (%) Excellent | Good | Fair | Failure |
|---|---|---|---|---|---|
| • Buttocks & Hips | 10 | 30% | 20% | 20% | 30% |
| • Low back only | 1 | 100% | - | - | - |
| • Low back > 60% Both legs < 40% | 16 | 25% | 44% | - | 31% |
| • Low back > 60% One leg < 40% | 28 | 39% | 10% | 22% | 29% |
| • Both legs > 60% Low back < 40% | 9 | 56% | 22% | 11% | 11% |
| • One leg > 60% Low back < 40% | 12 | 42% | 33% | 8% | 17% |

From an aetiological standpoint the patients with best result were those with "sympathetically maintained pain".

With regard to the distribution of pain there is definite evidence that patients with predominantly leg pain fare better than those with predominantly low back pain *(Table 4)*.

## COMPLICATIONS

There was no mortality. The operative morbidity was limited to prolonged incisional pain which settled spontaneously in all cases but required extended use of analgesics in some instances.

Infection was a rare complication since the adoption of antibiotic prophylaxis and internalisation of the system in a single operation. Explantation of the system was not necessary in the two infections of this series.

A few frustrating technical problems called for surgical revisions *(Table 5)*. The most frequent problem was a loss of stimulation due to fracture of the electrode or a change in distribution of the evoked paraesthesia due to displacement of the electrode. Replacement of the wire-type electrodes with fixed arrays of the *RESUME* type corrected the problem in all cases.

Three systems were explanted elsewhere. In two cases the pain spontaneously disappeared after a few months of stimulation. In another case the explantation was deemed necessary due to an intercurrent psychiatric problem.

## DISCUSSION

Conflicting results on the use of ESES in chronic low back pain have been reported in the literature. A recent survey by Gybels[4] has revealed that the success rate ranges from 20% to 91% with a mean of 58%.

A comparison of the published results is difficult due to lack of uniformity in the presentation of the data.

In general the variation in success rate may be attributed to difference in the selection of patients, surgical techniques or simply the criteria used for the assessment of success. Return to work for example, is a poor measure of outcome in individuals whose rehabilitation is precluded not only by chronic pain but also by permanent physical disability.

The clinical results of this series are comparable to those ones of recent reports with equivalent populations of patients, techniques of implant and scoring methods [6,7,8,9,10,11,12,13]. These results are unanimously interpreted as being indicative of proven efficacy of ESES. To a critical observer the failure rate of 26% in this series may appear excessive.

*(Table 5)*

### SURGICAL REVISIONS

| Problem | Number of Cases | Corrective Action | System Now in Use |
|---|---|---|---|
| Incorrect electrode position | 2 | Reposition of same electrode | Yes |
| Incorrect receiver position | 2 | Reposition of same receiver | Yes |
| Wire-type electrode displacement | 2 | Replacement with plate-electrode | Yes |
| Wire-type electrode fracture | 6 | Replacement with plate-electrode | Yes |
| Infection | 2 | Debridement, antibiotic and primary closure | Yes |
| Pain disappeared after implant | 2 | System explanted | - |
| Subsequent psychiatric problem | 1 | System explanted | - |



A detailed analysis *(Table 6)* indicates that nine out of twenty causes of failure are related to technical problems which could possibly be corrected, given the opportunity. Malfunction of the equipment was responsible for erratic upsurge of stimulation which occurred unrelated to movement or position of the body in three cases *(Neuromed systems)*. Fracture of the *QUAD* electrodes (model *3487 R*) was the cause of failure in three further cases and in the final case the *QUAD* electrode was displaced.

*(Table 6)*

### ANALYSIS OF FAILURES

| Problem | Number of Cases |
|---|---|
| Primary non-responder | 4 |
| Secondary non-responder | 2 |
| Wrong position of electrode *(not corrected)* | 2 |
| Displacement of electrode *(not corrected)* | 1 |
| Malfunction of equipment | 6 |
| Drug addiction | 4 |
| Psychiatric condition | 1 |
| TOTAL | 20 |

In two patients the stimulation induced cooling of the legs and reduced skin blood flow. This could be related to a wrongly positioned electrode. Miles[14] has recently reported on an apparent relationship between the anatomic level of spinal cord stimulation and the segmental autonomic outflow, inferring that a repositioning over the conus could eliminate the abnormal sympathetic response.

In this context the results in the cases of sympathetically maintained lumbar syndrome are of particular interest. A lumbar pain syndrome generated within the disc and the paradiscal structures, mediated predominantly within the sympathetic nervous system has been recently discussed by Jinkins et al[15]. These authors mention limbus vertebra[16] among the causes of this syndrome. In two of the four cases of the present series limbus vertebrae had been confirmed by discography and post discogram CT scan. All patients with sympathetically maintained pain had an optimal result. This is probably the first report on the use of ESES for such an indication. The number of cases is too small to venture a conclusion but the limited experience is certainly encouraging.

Cost of treatment with ESES is at times mentioned as a deterrent. Unlike other expensive treatments for chronic pain, ESES appears to be cost-effective. One of the two patients with pain generated from limbus vertebrae had been on a regiment of ninety-two items of medication, including opiates, prior to the implant with an estimated total cost of A$42,000 over a twelve month period. He is now on four to six tablets of minor analgesics/day.

Cicala et al. have evaluated the cost effectivness of ESES in ten patients with chronic low back pain. The pain related health care costs during the twelve months prior to the implantation, totalling more than US$500,000, was reduced by approximately 60% in the twelve months following the implant. This included the costs of the stimulation devices[17].

Among the determinants of results some factors deserve a special comment *(Table 7)*. A well documented history should indicate an organic cause of pain unresponsive to treatments directed at the causative factors. There is a strong correlation between distribution of pain and success rate *(Table 4)*. An ideal responder to ESES has predominantly leg pain, preferably due to a mechanism of deafferentation with monoradicular distribution and minimal or no low back pain[6]. Patients with significant emotional problems or drug-seeking behaviour are poor candidates. Age appears to have no bearing on outcome; however elderly or unskilled patients may lack the mental alertness to operate the equipment correctly. The patient's expectations ought to be realistic. ESES is not a cure. It does not prevent pain. It can only alleviate pain as it occurs. If the intensity of pain increases stimulation may become ineffective.

A most significant determinant of result is the location of the electode, because stimulation is strictly site-specific. The results improve with the surgeon's experience and refinement of operating skills. Anchoring of the electrode to a deep midline structure is a proven method of preventing displacement.

Wire-type electrodes are easier to insert but more prone to cause mechanical failures. There was no displacement or spontaneous fracture of any *RESUME* electrode used in this series. Decline of effectiveness over time can be improved by replacing wire-like electrodes with plate-like electrodes[18].

*(Table 7)*

### DETERMINANTS OF RESULTS

| PATIENT RELATED FACTORS | TECHNIQUE RELATED FACTORS |
|---|---|
| Cause of pain | Electrode location |
| Distribution of pain | Surgical skills |
| Inappropriate drug use | Wire–type vs plate-type electrodes |
| Ability to operate the equipment | One stage vs two stage implant |
| Reasonable expectations | External stimulator vs fully implanted system |

Permanent implant in one stage reduces the infection risk but a prolonged trial stimulation provides the opportunity to predict the response in cases of doubtful aetiology.

Fully implantable pulse generators (*ITREL*) are more acceptable to active patients than the external stimulators. However, systems with internal power need periodical replacement and increased technical supervision for the telemetric control of parameters. This is of particular concern for patients who live in remote areas.

## CONCLUSION

The information presented offers a frame of reference for considering an innovative treatment modality.

The available data supports the conclusion that ESES has a definite place in the neurosurgical armamentarium for control of intractable lumbar pain syndromes. It has the advantage of being non-destructive, non-addictive and reversible.

Risk factors are few in experienced hands.

A result of good to excellent pain relief in 61% of patients is more than acceptable if one considers that ESES is usually a last resort when nothing else will help.

The final comment is on the patient's acceptance of this treatment: out of seventy-one who volunteered an answer to a supplementary question forty-one (57.8%) said that they would undergo the same treatment again for the same relief, nineteen (26.8%) would not, and eleven (15.4%) were undecided.

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# Assessment, Significance and Management of Leg Length Inequality

**Jeffrey Phillips**
*270 Margaret Street, Toowoomba. Qld., 4350*

## ABSTRACT

***A simple, safe and inexpensive method of detecting leg length inequality is described and compared with other methods. The suggested method is proposed as being reliable, non-invasive and easy to use.***

***The significance of leg length inequality is discussed. This paper does not purport to settle arguments about the possible ill-effects of leg length discrepancy. However, some of the voluminous literature on the subject is reviewed and a considered opinion is expressed.***

***A management strategy is outlined for the problems commonly encountered in practice.***

## INTRODUCTION

The measurement of leg lengths and the significance of any inequality detected have long been subjects of controversy. No clinical method of measuring leg lengths has gained universal acceptance and those methods which are generally agreed upon are too invasive for regular use. Even when leg length discrepancies are reliably detected, opinions differ as to their clinical significance and indications for management.

## ASSESSMENT OF LEG LENGTHS

The traditional method of measuring leg length was by use of a tape measure to assess the distance between either the anterior superior iliac spine or the umbilicus and the medial malleolus. Difficulty in pinpointing these landmarks makes the method unreliable and inaccurate.

A method which is more reliable in detecting leg length inequality involves examining the subject lying supine with the observer at the foot of the couch. The observer's thumbs are applied firmly below the medial malleoli, the right thumb being placed below the left malleolus and vice versa. The examination is then repeated with the subject prone to verify that the findings agree in both positions. While this method is very reliable in determining whether there is a discrepancy or not, only a rough estimate of actual inequality can be made. In order to eliminate error caused by pelvic obliquity sustained while mounting, or lying down on, the examination couch, before making the observations two manoeuvres may be carried out. First, in the supine position, the subject is asked to flex his hips and knees and then to extend them, second, in the prone or supine position, the observer applies gentle traction to the ankles while, at the same time, shaking the legs gently from side to side.

At the opposite extreme, measurement by means of Lineagrams and Scanograms would appear to involve more exposure to radiation than is acceptable for routine use in treatment, and certainly for screening studies.

The radiological method of measurement (Fig. 1) in which the patient stands erect with his back to the cassette with a radio opaque plumbline included in the picture is accurate provided that certain criteria are met, viz: the X-ray tube must be aimed exactly horizontally at the level of the femoral heads, the subject must have his feet approximately 6 inches (15 cm) apart and knees must be absolutely straight. This is easily achieved in a research setting, but not in practice, where films are being taken by different technicians.

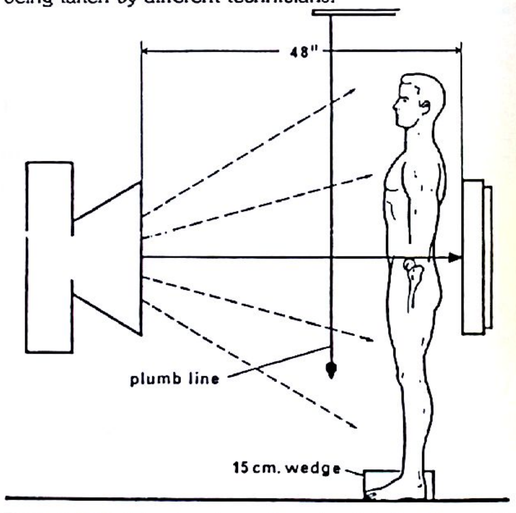



**Fig 1.** Method of erect-posture radiography. Note the 15 cm wide block placed between the ankles, the X-ray tube centered at the femoral heads, and the radiopaque plumbline.

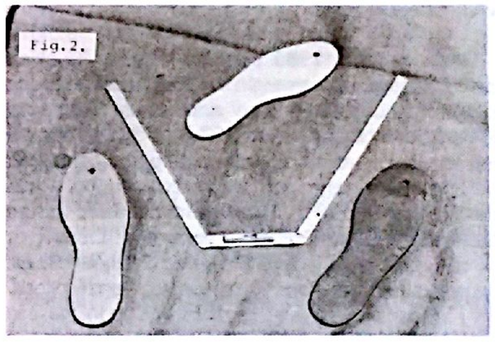


The author has used this method since the early 1960's. It was described by Clarke[1] in 1972 and at an A.A.M.M. meeting in Melbourne by Fisk in 1980.

X-ray exposure can be reduced by suitable screening as described in 1983 by Friberg[2] who claimed a safe gonadal dose of 11.4mR and an estimated bone marrow dose of 13.6 mR. Friberg claims that "this method is radiation safe and is suitable for screening studies even in young and symptom free subjects".

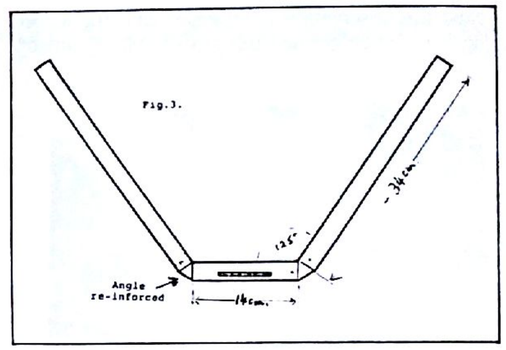
In clinical practice it is possible to make an assessment of pelvic obliquity by sighting the tips of the index fingers of each hand placed on the iliac crests of the patient, who stands erect in front of the observer with his back to him and with knees straight and heels firmly on the floor. This is a valid observation, but it does not allow for actual measurement of the difference in height of the iliac crests.

In order to overcome these deficiencies and disadvantages, the following method has been used.



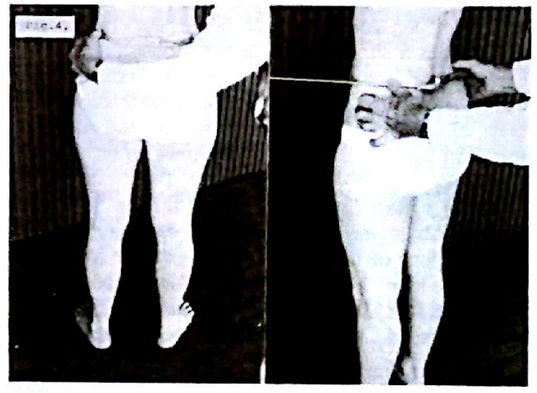

## Equipment

The instrument (Fig. 2) consists of a central body with two arms projecting forwards and outwards at an angle of 125 degrees. The dimensions are shown in the diagram (Fig. 3). On the central body is mounted a line spirit level. The original instrument was made of aluminium, pop riveted at the angles and the spirit level was attached by means of epoxy glue. It has been in use for ten years and has been found to be of satisfactory dimensions for all subjects except small children. Blocks of various thicknesses (12, 6 and 3mm) (Fig. 2) are used to correct the tilt and enable an estimation of the actual difference in height of the femoral heads to be made.




## Method

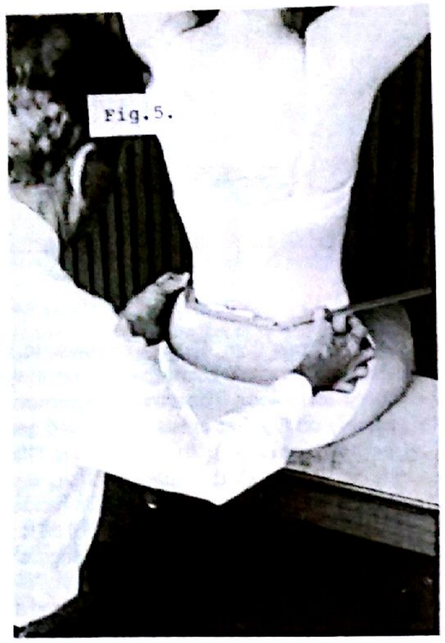
The subject stands (Fig. 4) with his back to the observer, feet approximately 15cm apart, knees straight and heels firmly on the floor. The arms of the instrument are placed firmly on the iliac crests and moved gently until "the feel" is the same on both sides. This is difficult to describe in words, but, with practice, easily accomplished. If a tilt is found the subject is handed a block, thought to be about the right size and asked to put it under one foot. In order to avoid a leading suggestion he is never told to place it under a particular foot. When the block is in place he is asked "how it feels" and then told to place it under the opposite foot and is again questioned. During the testing, the instrument is either kept in place or removed completely each time a block is placed under a foot in order, again, not to give a clue to the patient.




There are three possible replies:

1. He may say that it feels much better under the foot on the side of the pelvis which is lower.
2. He may say that it is more comfortable under the foot on the side of the pelvis which is higher.
3. He may say that he cannot feel any difference or cannot decide.

Next the observation is repeated with the subject sitting (Fig. 5) in order to exclude gross pelvic asymmetry.




## Interpretation

If the subject gives the first answer, that building up the "short" leg side feels much better, the chances are that he has a short leg. A gentle mobilisation of the lumbo-sacral spine will not alter the findings. To estimate the build up required to achieve a level pelvic brim, blocks of different thicknesses are placed under the short leg until the spirit level shows no tilt.

If the subject finds that building up the "long" leg (answer 2) is more comfortable, he may have a derangement of the lumbo-sacral spine or a blocked sacro-iliac joint. Mobilisation of the lumbo-sacral spine and sacro-iliac joints is carried out and pelvic obliquity again assessed. In many cases the pelvic tilt will have disappeared and any limitation of spinal movement noted during the accompanying examination will have been reduced or abolished when assessed again.

If the subject is unable to decide which build up is more comfortable, again, a very gentle mobilisation is used and the pelvic tilt is re-assessed. If the tilt remains and the patient is still unable to decide which side is more comfortable, or if he says that he finds the build up more comfortable on the long side as in answer 2, he is re-assessed after an interval of several weeks.

## SIGNIFICANCE OF LEG LENGTH INEQUALITY

Much has been written concerning management of subjects with leg length discrepancy and opinions vary from taking no action, through correcting only differences of more than an arbitrary figure, most commonly a half inch (12.5mm), to correcting differences as little as one eighth of an inch (3mm). Opinions also differ concerning the management of leg length discrepancy acquired as a result of some abnormal occurrence such as hip disease, fracture or polio-myelitis and inequality which seems to be developmental in origin.

Andersen[3] stated that inequalities of three quarters to one and a half inches (19-38mm) need a heel lift and, or, a foot correction, but that inequalities of less than three quarters inch (18mm) require no correction.

Subotnick[4] claimed that differences as little as one eighth of an inch (3mm) require correction if symptoms of imbalance are present. Wooden[5] stated that a true leg length difference of more than one quarter of an inch (6mm) discovered at pre-season screening of athletes require correction if extensive running activities are to be undertaken.

Dieck et al[6] in 1984 published the results of an epidemiological study on eight hundred and seventy one asymptomatic women who entered an American women's college between 1957 and 1959. These women were assessed clinically and by photograph on entry to the college and were reviewed by postal questionnaire in 1981. They found that there was no statistically significant increase in cervical, thoracic or low back pain in subjects with a pelvic tilt, though there was a definite correlation between pelvic tilt and scoliosis. They further pointed out that there was no increase in back pain in subjects with scoliosis, a view supported by Hunt. However, Dieck et al did point out that the response rate to their questionnaire was poor, which perhaps casts doubt on their conclusions. Also the follow up time was only about twenty years which may be too early for degenerative changes to become manifest in the joints of subjects of this age. Further, their assessment was only by means of photography of the back with the patient standing erect.

Froh et al[7] endeavoured to show a correlation between leg length inequality and lumbar zygapophysial joint orientation in a study of forty patients aged 17 - 79 years (mean 43.8 years) attending a back pain clinic. Leg length differences ranged from 0.5mm to 15mm and they failed to show any change of zygapophysial joint orientation, in other words, structural change. However, they did suggest an hypothesis to explain a correlation between leg length inequality and low back pain involving asymmetrical loading of the spine.



Giles and Taylor[8] reviewed one hundred patients aged 19 - 61 years (mean 40 years) with chronic low back pain, fifty of whom had a leg length difference greater than 9mm and fifty a difference of less than 3mm. Measurements were made radiographically as described by Clarke[1] in 1971 and Giles and Taylor[9] in 1981. They recorded the contour of the end plates of the first to fourth lumbar vertebrae and found concavities which were not symmetrical in 28% of the subjects with a leg length inequality of 8mm or more, these changes being more pronounced in the lower than the upper end plates and most marked in the vertebrae at the apex of the postural scoliosis. Similar changes were found in only 2% of the subjects with a leg length inequality of less than 3mm. They also measured the fifth lumbar vertebrae and found wedging in the AP view in 10% of the subjects with more than 9mm of leg length inequality and none in the subjects with less than 3mm leg length inequality.

Finally, they looked for lateral traction spurs and osteophytes. These occurred in 83% of the subjects with more than 9mm of leg length inequality and only 25% of subjects with less than 3mm leg length inequality. They state that these spurs are an indication of motion segmental instability and further suggest that small spurs are of great clinical significance in this regard when seen projecting horizontally from a point 1mm above the vertebral body edge. They further state, quoting McNab[10], that the larger, claw shaped, osteophytes extending from the rim of the vertebral body and curving upwards or downwards are growing round a bulging degenerate disc and may indicate actual, or impending, stability at that level.

Farkas[11] stated that asymmetrical degeneration of intervertebral discs occurs when they are stretched on the convex side of a scoliosis.

Farfan[12] has stated that, if the anulus becomes sufficiently stretched and torn, a significant degree of distortion may be produced and that tortional stresses of a magnitude encountered in daily activities may play a major role in initiating degeneration of lumbar inter-vertebral discs.

It is logical to conclude that a postural scoliosis with disc wedging might therefore accelerate lumbar inter-vertebral disc degeneration.

Friberg[2] in a review of subjects with a leg length inequality of 5mm or more complaining of low back pain, hip pain or "sciatica" (by which is meant referred leg pain rather than actual sciatic nerve involvement), following correction of the inequality, recorded abolition of symptoms in over 70%, reduction in 15% and no relief in 13%. Interestingly, the 70% had an average age of 42 years, the 15% 46.7 years and the 13% 53 years. Further, the "sciatic" symptoms occurred in 78.5% of subjects and hip joint symptoms in 89% on the long leg side.

Subotnik[4] surveyed four thousand athletes and long distance runners seen over six years and found some form of leg length inequality in almost 40%. He went on to differentiate between the causes of leg length inequality from the simple anatomical short leg to low back derangements and limb abnormalities such as external rotation of the hip leading to a pronation deformity of the foot or caused by it. He further stated that leg length inequality was often associated with injury on the short side or could be the primary cause of injury. In his article, Subotnik discusses the management which includes building up for true leg length inequality, orthotic devices in cases of pronation deformity of a foot and manipulative treatment of spinal causes.

Stoddard[13] found seventeen of one hundred patients with low back symptoms had leg length inequality of a half inch or more compared with 8% of symptomless control subjects.

Nichols[14] found leg length shortening in 22% of British airmen with backache compared with only 7% in controls.

Rush and Steiner[15] found 77% of soldiers with low back pain had an average leg length inequality of 7mm.

Dixon and Campbell-Smith[16] have shown that in patients with 25mm of leg length inequality for over one year the knee joint on the short side is subject to greater mechanical damage.

Schuit, McPoil and Mulesa[17] surveyed a small group of randomly selected students at the University of Illinois and found a surprisingly high incidence of leg length discrepancy and a fairly high incidence of asymptomatic sacro-iliac joint malalignment. They advised that when a leg length discrepancy is detected during a musculoskeletal assessment, the examiner should be alert to the possibility of sacro-iliac joint malalignment even though the patient may not complain of symptoms related to that joint. They further suggest, on personal advice from J.A. Pasterfield, that these subjects will most likely experience some symptomatology in the future and are "accidents waiting to happen".

## SUGGESTED MANAGEMENT STRATEGY

The management will depend upon the reason for the subject seeking advice in the first instance and on the history obtained at consultation.

For a patient complaining of long standing low back symptoms who is found to have a short leg and who gives the "correct" answer during experimental building up at examination, a trial period of 4-6 weeks with a build up of the heel is worthwhile. How much to build up will be discussed later as will the actual method of achieving this.

For a patient who gives the "incorrect" answer and whose tilt disappears after mobilisation, no further immediate action is taken, but re-assessment is recommended after one week if symptoms persist. If symptoms disappear, and do not recur during this time, no further action would appear to be necessary until such time as symptoms do again appear.

When a pelvic tilt is discovered during examination of a patient with no low back symptoms, for example, during the overall assessment of a person complaining of shoulder pain, the question of advising correction is more difficult and depends upon factors such as age and, particularly, sporting activities.

A useful rule of thumb is to build up only those subjects who are sure that correction of the "right" side is more comfortable than correction of the "wrong" side when a pelvic tilt is found in an adolescent who has been noted at a routine school medical examination to have a mild scoliosis. Additional findings may be noted during the examination. There may be asymmetry of the height of the shoulders, which has often been noted by a parent and, on the short side, the flank will be less hollow and the hip less prominent. Such patients are usually asymptomatic, presumably because the mobile young spine adapts quite easily. If the direction of the scoliosis corresponds with the tilt and if the "correct" answer is given during experimental building up, then build up should be suggested with re-assessment every 4-6 months.

Depending upon the clinical signs an initial x-ray examination with the patient erect to measure the degree of scoliosis and tilt will sometimes be necessary and is probably advisable as clinical assessment of the degree of scoliosis is not easy or reliable. Such films should be taken with the patient erect as should all films of the spine or weight bearing joints.

### Method of Shoe Modification

During a trial period it is quite practical to use a hard insert of rubber, leather, or even cardboard up to three eighth inch (10mm) of thickness inside the heel of a "sensible" shoe. Later a build up on the outside of the heel is better, but this must be wedge shaped to avoid strain at the junction of the heel and sole of the shoe. By this means a correction up to half inch (12.5mm) can be made and is not obvious. Above this size, reduction of the opposite heel is necessary. Also for greater corrections consideration should be given to carrying the build up through to the toe, e.g. 12mm at the heel, 6mm at the ball of the foot and 3mm at the toe, and this is essential in sports shoes to avoid ankle joint strains and pronation of the foot.

In older persons, correction should be in stages if more than three eighth inch (10mm) is necessary. There is no definite age cut off, but caution is advisable in persons over 35 years and in persons who have marked changes on x-ray. Caution must also be observed in correcting leg length inequality in a subject with a severe fixed scoliosis in the older age group as doing so may aggravate upper spinal symptoms.

Finally, if after a short trial period a patient states that a build up causes aggravation of symptoms, it should be removed immediately.

## DISCUSSION

It is the author's contention that any measure which might reduce asymmetrical strain on the spine is worthy of consideration. It would appear that leg length inequality must, and does except in very exceptional circumstances, throw such asymmetrical strain on the spine.

Each individual case must be judged on its own merits rather than being subjected to a hard and fast ruling. For example, a 10mm difference in leg lengths will cause a greater pelvic tilt in a small adult with a narrow pelvis, or in a child, than in a large subject who is much wider across the hips.

Also, activities must be considered, especially if running is involved as the rule of three applies. This rule states that a leg length inequality of $x$ mm is as important in an athlete as a difference of $3x$ mm in a non-athletic person (Subotnick [4] ).

Similarly, individual susceptibility to harm must vary from person to person and it is surely a wiser course to correct leg length inequality by means of a simple non-invasive method rather than to await possible damage at a later date in the hope that it will not occur.

## SUMMARY AND CONCLUSIONS

A method of assessing leg length discrepancy or inequality has been described which has been found reliable and easy to use over a period of ten years. The method is inexpensive and does not involve any irradiation whatsoever. An overview of some of the literature has been presented which suggests that correction of leg length discrepancy is usually advisable, if not absolutely essential. The method of achieving this correction by modification of shoes has been described. A strategy for management of subjects with leg length discrepancy has been outlined.



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# Low Back Pain: A Diagnostic Approach

**John Murtagh**

*Department of Community Medicine, Box Hill Hospital, Box Hill, Vic 3128.*

## ABSTRACT

***The outstanding causes of low back pain are vertebral dysfunction, involving the intervertebral disc and the zygapophysial joints, and spondylosis. It is important to differentiate between the pain of inflammation (worse at rest, relieved by activity) and that of mechanical dysfunction (worse on activity, relieved by rest). The key to the diagnostic method is to understand the daily patterns of back pain.***

## INTRODUCTION

Low back pain accounts for at least five per cent of the problems seen in general practice. The most common cause is minor soft tissue injury but such patients do not usually seek medical help because the problem settles quickly within a few days.

Most back pain presenting in general practice is due to dysfunction of elements of the mobile segment - that is, the two zygapophysial joints, the intervertebral joint (with its disc) and the ligamentous and muscular attachments. This problem, often referred to as mechanical pain or traumatic joint derangement, will be referred to as vertebral dysfunction - a general term, which, while covering radicular and non radicular pain, mainly includes dysfunction of the joints of the spine.

## CAUSES OF LOW BACK PAIN

In order to develop a comprehensive diagnostic approach the practitioner should have clear understanding of the possible causes of low back and leg pain including a perspective of their relative frequency and clinical presentations.

Table 1 presents the major causes for such pain presenting as a primary symptom by several hundred patients to the general practice of the author.

**Major causes of low back and leg pain presenting in general practice**

| | Patients % |
|---|---|
| Vertebral dysfunction | 72.3 |
| Lumbar spondylosis | 8.5 |
| Depression | 3.0 |
| Urinary tract infection | 2.2 |
| Spondylolisthesis | 2.0 |
| Spondyloarthropathies | 1.7 |
| Muscle strains/tears | 1.0 |
| Malignant disease | 0.8 |
| Arterial occlusive disease | 0.6 |

***Table 1***

## A DIAGNOSTIC APPROACH

Using the safe diagnostic strategy model (Table 2) the five self posed questions can be answered as follows:

**1. Q. What is the probability diagnosis?**

**A.** The answer, of course, is vertebral dysfunction, which then has to be further analysed. Muscle or ligamentous tears of similar soft tissue injuries are uncommon causes of back pain alone: they are generally associated with severe spinal disruption and severe trauma such as that following a motor vehicle accident.

In the lumbar spine most problems originate from the joints of the spine, either the zygapophysial joints or the intervertebral disc and its connections, or from both simultaneously. The disc can cause pain, either intrinsically from internal disruption or extrinsically by presssure on adjacent pain-sensitive structures, leading to radicular pain (if the nerve root is involved) or non-radicular pain. However, frank disc prolapse is not the common cause of back pain it was once believed to be.

Degenerative changes in the lumbar spine (lumbar spondylosis) is commonly encountered in the older age group.

**2. Q. What serious disorders must not be missed?**

**A.** It is very important to consider malignant disease, especially in an older person. It is also vital to consider infection such as acute osteomyelitis and tuberculosis which tend to be encountered in recent immigrants especially those from Asia. For pain or anaesthesia of sudden onset, especially when accompanied by neurological changes in the legs, consider cauda equina compresssion due to a massive disc prolapse and also retroperitoneal haemorrhage. It is important to ask the patient if they are taking anticoagulants.



**Low back pain: summary of diagnostic strategy model**

**1. Q. Probability diagnosis?**
- **A.** • Vertebral dysfunction especially of facet joints and discs
  - Spondylosis (degenerative OA)

**2. Q. Serious disorders not to be missed?**
- **A.** • Malignant disease especially metastases, myeloma
  - Bone infection, eg. osteomyelitis, tuberculosis
  - Cauda equina compression )
  - Retroperitoneal haemorrhage ) acute
  - Ruptured aortic aneurysm )

**3. Q. Pitfalls (often missed)**
- **A.** • Spondyloarthropathies
  - ankylosing spondylitis
  - Reiter's disease
  - psoriasis
  - bowel inflammation
  - Sacro-iliac dysfunction
  - Spondylolisthesis
  - Claudication
    - vascular
    - neurogenic

**4. Q. 7 masquerades check list**

| | | |
|---|---|---|
| **A.** | Depression | ✔ |
| | Diabetes | - |
| | Drugs | - |
| | Anaemia | - |
| | Thyroid disease | - |
| | Urinary infection | ✔ |
| | Spinal dysfunction | ✔ |

**5. Q. Is the patient trying to tell me something?**
- **A.** • Quite likely. Consider lifestyle stresses, work problems, malingering, conversion reaction.

***Table 2***

**3. Q. What questions are often missed?**

**A.** The inflammatory disorders must be kept in mind, especially the spondyloarthropathies which include psoriatic arthropathy, ankylosing spondylitis, Reiter's disease and inflammatory bowel disorders such as ulcerative colitis and Crohn's disease. The old trap of claudication in the buttocks and legs (due to a high arterial obstruction) being confused with sciatica must be avoided.

**4. Q. Could the patient have one of the seven masquerades?**

**A.** Of these conditions depression and urinary tract infection have to be seriously considered. For the young woman with upper lumbar pain, especially if pregnant, the possibility of a urinary tract infection must be considered. These patients may not have urinary symptoms such as dysuria and frequency.

Depressive illness has to be considered in any patient with a chronic pain complaint. This common psychiatric disorder can continue to aggravate or maintain the pain even though the provoking problem has disappeared. This is more likely to occur in people who have become anxious about their problem or who are under excessive stress. Many doctors treat such patients with a therapeutic trial of antidepressant medication, for example, amitryptiline or doxepin taken at night.

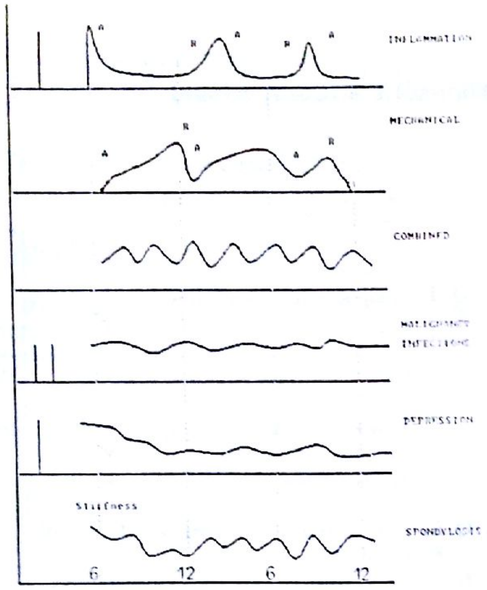
**5. Q. Is the patient trying to tell me something?**

**A.** Low back pain following lifting at work is the classic problem that causes considerable anguish to doctors especially when patients' whose problems become chronic and complex. Such an event may be the 'final straw' for a patient who has been struggling to cope with a personal problem, and their fragile equilibrium may be thrown into disarray by their episode of back pain. Many patients who have been dismissed as malingerers turn out to have a genuine problem. The importance of a caring, competent practitioner with insight into all facets of the patient's suffering, organic and functional, becomes obvious. The tests for non organic back pain are very useful in this context.

## USING DAILY PAIN PATTERNS TO ASSIST DIAGNOSIS

A careful history that maps the diurnal variations of pain facilitates the diagnosis (Figure 1). It is especially important to note the intensity of the pain and its relation to rest and activity. In particular ask whether the pain is present during the night, whether it wakes the patient and whether it is present on arising or associated with stiffness.

Continuous pain present day and night is suggestive of neoplasia or infection. Pain on awaking also suggests inflammation or depressive illness. Pain provoked by




A = activity

R = rest

A R A R A Inflammation

A R A A R Mechanical

Combined

Malignancy infections

Depression

Stiffness Spondylosis

6 12 6 12

**Fig 1.** *Typical diurnal patterns of pain for conditions causing back pain. Note conditions that can wake patients from their sleep.*

activity and relieved by rest suggests mechanical dysfunction while pain worse at rest and relieved by moderate activity is typical of inflammation. In some patients the co-existence of mechanical and inflammatory causes complicates the pattern.

Pain with standing or walking but relieved by sitting is suggestive of spondylolisthesis.

Pain in the calf which travels proximally with walking indicates vascular claudication while pain in the buttock which descends with walking indicates neurogenic claudication. This latter problem is being encountered more frequently in the older population who have a tendency to spinal canal stenosis associated with spondylosis.

## THE PHYSICAL EXAMINATION

The basic objectives of the physical examination are to reproduce the patient's symptoms, detect the level of the lesion and determine the cause (if possible) by provocation of the affected joints or tissues. This is performed using the time honoured method for joint examination - look, feel, move and test function. The patient should be stripped to a minimum of clothing so that a careful examination of the back can be made. A neurological examination of the lower limb, should be performed if symptoms extend below the buttocks.

A most useful screening test for a disc lesion and dural tethering is the slump test.

## INVESTIGATIONS

Investigations for back pain can be classified into three broad groups namely front line screening tests, specific disease investigations and procedural and pre-procedural diagnostic tests.

The author finds that screening tests are most important in the patient presenting with chronic back pain where it is important to exclude serious disease such as malignancy, osteoporosis, infection and spondyloarthropathy.

The screening tests for chronic pain are:

- plain X-ray
- urine examination (office dipstick)
- erythocyte sedimentation rate
- serum alkaline phosphatase
- serum acid phosphatase (in males).

# Postgraduate Programme in Musculoskeletal Medicine
# Flinders University, South Australia

The Department of Orthopaedic Surgery and Primary Health Care have arranged a postgraduate programme to increase the skills of medical practitioners in assessing, diagnosing and treating musculoskeletal dysfunction.

The University requires 36 units of study to be completed before the Diploma is awarded and to this end, 6 x 6 unit courses have been approved. A Certificate may be awarded after completion of 3 x 6 unit courses.

After consultation with numerous interested parties it was decided that such a programme would best serve rural and city practitioners if it were offered in intensive in-service blocks of two weeks duration as follows:

- ** Anatomy, Physiology and Biomechanics of the musculoskeletal system
- ** Clinical Skills in managing non-surgical and non-rheumatological musculoskeletal dysfunction
- ** Musculoskeletal dysfunction related to diseases of the vertebral skeleton
- * Musculoskeletal dysfunction related to diseases of the appendicular skeleton

** N.B. These three courses are compulsory for the postgraduate certificate.

Emphasis in the Anatomy, Physiology and Biomechanics course will be on diagnosis. The Clinical Skills course deals with managementand includes specific treatment modalities.

In addition to the above, two additional units must be taken to complete the requirements for the Diploma:

Independent Study - to include indepth literature survey, critical assessment of a treatment modality/ examination or procedure, etc., to be made in consultation with the course co-ordinator.

Rehabilitation Studies **or** any related unit from the offerings within the Master of Science Primary Health Care Programme.

In determining the fees for such a course cognisance must be taken of:

a. The charge for each year of an undergraduate medical course is of the order of $26,000 per year for the full-fee paying student, e.g. overseas students.
b. There is no mechanism whereby university or government funds can be put toward the costs of the course.

The fairest way is to charge $2,000 for each unit or $12,000 over a two to three year period until the Diploma is completed.

The first of the two week intensive inservice courses is planned for 18th to 30th November, 1991 at the Flinders Medical Centre, Adelaide, S.A.

Participants are expected to find their own accommodation. The Flinders University Halls of Residence may have some vacancies.

| | |
|---|---|
| *Course:* | Anatomy, Physiology and Biomechanics of the Musculoskeletal System. |
| *Dates:* | Monday 18th November to Saturday 30th November |
| *Venue:* | Flinders Medical Centre, South Australia |
| *Cost:* | $2,000 |
| *Limit:* | 12 |

Note that Continuing Medical Education can be claimed as a tax deduction.

Further information about course offerings can be obtained from: Dr. Norm Broadhurst, Department of Primary Health Care, Flinders Medical Centre, Bedford Park, S.A. 5042.

# BOOK REVIEW

## Disorders of the Lumbar Spine

edited by Yizhar Floman

Publisher:

Aspen Publishers, Rockville, Maryland, 1990

In the preface to this book its editor lays the ghost of the ruptured intervertebral disc as the major cause of low back pain and goes on to suggest the need for a much broader view of the problems of lumbar spinal dysfunction. The book then sets out to review current knowledge and practical considerations in the numerous areas of relevance, from basic sciences to treatment and rehabilitation. It is an ambitious project but one in which this book succeeds.

Beginning with chapters on anatomy, biomechanics, biochemistry and epidemiology, the book moves through the realms of clinical assessment, ancillary investigations, the various modalities of conservative management and the different types of surgical treatment. Each topic is addressed in a chapter written by authors with particular expertise and the list of contributors is impressive. Floman is a distinguished spinal surgeon and there is some surgical bias in the emphasis given to certain topics but this does not detract from the book's usefulness. In fact, it provides something of a bonus for the non-surgeon, as the less familiar subjects are treated in the greatest detail.

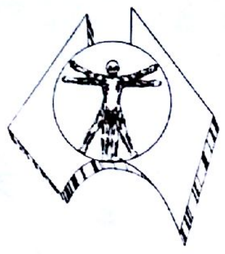
The information presented forms a summary of current knowledge on each topic and the book's many illustrations enhance the understanding of the practical aspects described. For example, the 28 page chapter on magnetic resonance imaging of the lumbar spine contains 33 illustrations, providing an overview of the subject which is more than adequate for the clinical practitioner wanting to understand the appropriate use of this particular investigative modality. There are similar chapters on radiology, myelography and computerised tomography, bone scintography, electrophysiological evaluation and evoked potential studies, all containing the essential elements of their subjects in sufficient detail for the interested clinician.

The section on conservative management strategies will seem sketchy to musculoskeletal physicians but this is understandable in such a wide-ranging work. On the other hand, the nine chapters devoted to specific surgical techniques are clearly written and very informative, providing valuable insights into the work of spinal surgeons.

In addition to the general descriptions of treatment strategies, the book has several chapters devoted to specific pathological entities including fractures of the lumbar spine, arthritic diseases of the spine, diffuse idiopathic skeletal hyperostosis, metabolic diseases of bone, lumbar spine sepsis and tumours of the lumbar spine and sacrum. Each provides a use reference for practitioners who encounter such conditions only rarely. There are also interesting chapters on back pain in children and low back pain in pregnancy.

All in all, Floman's book is a very useful single volume reference on many aspects of lumbar spinal disorders and should find a place on the bookshelves of all those interested in the management of these problems.

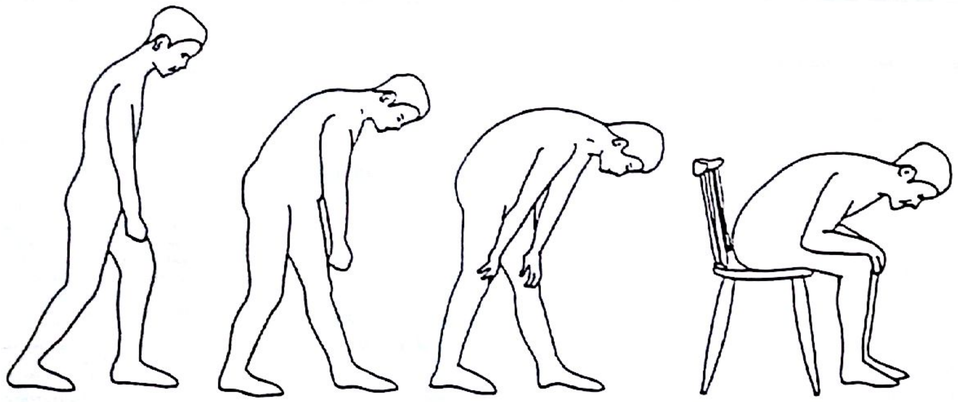
*996 pp.; distributed in Australia by Mosby-Williams and Wilkins Pty. Ltd.*



# Accreditation Assignment

The figure below is reproduced from the clinical assessment section of Yizhar Floman's new book "Disorders of the Lumbar Spine" (which is reviewed in this issue of the Bulletin).

Members are invited to study the situation depicted and to select the most appropriate caption from the alternatives which follow.



1. Patients should be encouraged to enter the consulting room and approach the physician in a supplicant manner.

2. Patients may suffer episodes of priapism due to autonomic dysfunction associated with sacral entrapment radiculopathy.

3. Nausea and vomiting are always distressing features of thoracic dysfunction especially when they occur in the waiting room.

4. Stoope's dyke test is a useful technique in the clinical assessment of patients whose pain is aggravated by straining at stool.

5. Dyck's stoop test assists in the interpretation of spinal claudication associated with lumbar entrapment radiculopathy.

Ten accreditation points will be awarded for the first correct entry received by the Hon. Secretary.



# ADVERTISERS' INDEX



There is space on this list for many other companies with interests in the field of musculoskeletal medicine. The Bulletin welcomes advertisements for any products or services considered worthy of members'