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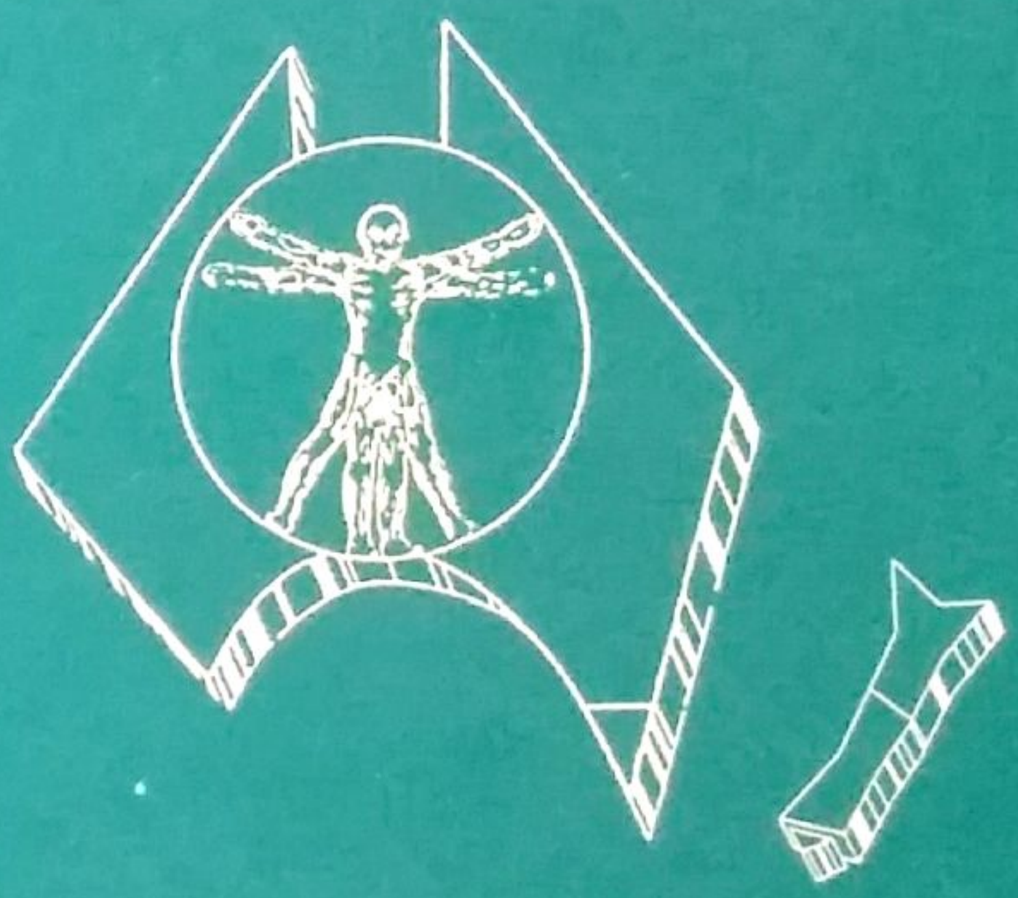

- **Effects of Exercise & Loading on Lumbar Discs**
- **Structural & Functional Changes in Articular Cartilage**
- **Thoracic Spinal Pain**
- **Malalignment Syndrome**
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Vol. 1 No.5 November 1996

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Australasian Musculoskeletal Medicine

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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:

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or to any member of the A.A.M.M. Council.

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Australian Association of Musculoskeletal Medicine

OFFICE BEARERS 1996 – 1997

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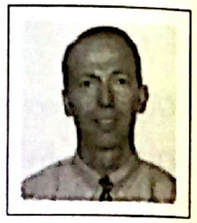

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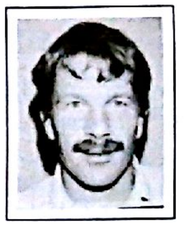

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EDITORIAL

The combined Australian and New Zealand Associations annual scientific meeting held in Fiji was a successful exercise of co-operation and learning. Numbers may have been higher if the meeting had not coincided with the Otago and Newcastle Diploma Courses. While economically the meeting did not meet expectations, the quality of lecture presentations and the practical tutorials were of excellent standing. It is unlikely that any delegate would have gone away disappointed.

"Off Shore" conferences do have the reality of being less profitable as it is less likely that paramedical groups will attend in any significant numbers. Combined Association conferences on the other hand supply both a stimulus to learning and a bonding of the two Trans Tasman association members. There does appear to be merit in alternating annual conferences between the two countries and thereby members could enjoy an "offshore" conference every second year as well as maintaining closer contacts. As the teaching at Diploma level is compatible and now with the formation of the Australasian Faculty, it is worth the consideration of the two Associations to run such alternating venues as suggested. In the past there has been some dispute over the division of revenue raised from joint conferences. Ideally, each country would run it's own conference economically independent of the other. This then avoids the past squabbling over finances. A successful economic conference every second year for each Association would be far better than poorly attended conferences that seem to be the current pattern.

The Australian Association's AGM resulted in the election of a new executive. The past executive oversaw the establishment of university courses and the detailed, but long drawn out negotiations with successive Federal governments to have musculoskeletal units established at major teaching hospitals. Both these directions have to a large extent been fulfilled. While the Faculty continues to deal directly with the government for the establishment of clinics, the incoming committee needs to re-focus the more broader aspect of post graduate medical teaching. This most important aspect in the advancement of your specialty will never fall on the shoulders of the Faculty and the practical aspects can never be taught adequately in a university Diploma course. Now is not the time for the new committee to rest on past laurels. Indeed, a greater push in the broader teaching field is needed.

The past executive has been chaired by Norm Broadhurst for three years. He has now stood aside, but remains on the committee. Norm has contributed greatly to the advancement of musculoskeletal medicine, not only in Australia, but overseas as well. His personal involvement and the selfless donation of time has perhaps been greater than any past president. This association remains indebted to him as does indeed the newly formed Australasian Faculty. This journal, on behalf of all association members, expresses it's gratitude and thanks to him for such an outstanding effort.

We also thank all other executive and committee members. There has been some lateral shift as well as elevation in positions. Vic is now president and keen to add his own brand of leadership. I move from editor of the journal to vice president, while David takes over the new role of "Net" co-ordinator. Max moves back into treasury and Neil moves up from the committee to secretary. Michael now takes over full responsibility for the journal. We wish them all well in the coming year.

The F.I.M.M. world conference is to be held outside Europe for the first time in 1998. Queensland has the arduous task of staging this conference and essentially the same committee that staged the A.A.M.M.'s most successful annual scientific conference is in charge of proceedings. This will be a tremendous opportunity to advance musculoskeletal medicine both here and on the world scene. It is essential that all A.A.M.M. members support this conference. Your support is best given by your attendance. Adequate long term notice of the event means you should be able to allocate time to be in Brisbane for this event.

There is still opposing discussion on the qualifications required for entry as "Fellows" to the Australasian Faculty of Musculoskeletal Medicine. It has been advanced that there is a need for peer group recognition and thereby an approved examination system. This has some merit, but also possesses drawbacks. The alternative of admitting all without scrutiny is just as potentially damaging. It would seem prudent that some flexibility is in order. No past college has ever been founded by consideration of other peer groups. Indeed, most colleges have had significant, if not total, "grandfather" clauses. As has already been pointed out in the Faculty newsletter, there is a danger of losing many of the senior teachers who have seen the advancement of musculoskeletal medicine from virtually a non-entity to its current university Graduate Diploma standards. This editorial suggests that those persons in charge of organising the faculty should consider the matter of flexibility very carefully. Our specialty does not hinge on academic knowledge alone. There needs to be a balance of academia with practical expertise. Neither can exist without the other. Double blind trials and facet joint injections do not affect many patients directly. The crunch to any medical treatment is, "Did the patient get better?" Perhaps there is a place for some seniors as Fellows who perhaps do not feel the desire or capability of pursuing arduous registrar type examination aimed at much younger and more recent graduates primed in the skill of passing examinations. Conversely, having anaesthetists, rheumatologists etc., (examples only for the purpose of discussion), as full Fellows leaves a weakness to the Faculty and the specialty as a whole. Manual methods of treatment are quite foreign to many of these other groups and completely absent in their training. Other Faculties have only members who are trained solely in that specialty. On this basis, is a further look at where we are heading in order?

Ron Palmer, Co-editor

A WORD FROM THE PAST PRESIDENT OF THE A.A.M.M.

Since the last AGM in July, 1995 there have been three full committee and four executive hook-ups. In addition I have communicated on several occasions with Angus Johnston, the president of the NZAMM with regard to our combined Fiji meeting. Our committee meetings have dealt further with our perennial problems which do not seem to have any better solutions, than they have had over the past decade.

A. Advertising for our journal

Members will notice that there has been an absence of advertising in recent issues. The major reason for this is that nobody has had time to look into this further. It now becomes an issue as to whether we employ somebody to chase advertising or allow the journal to be produced from members' subscriptions. With all the other activities we are required to fulfil as members of the medical profession requiring registration, practice profiles etc., there is little time for such unrewarding activities as chasing advertising.

B. Articles for the journal

Review and research articles are always needed. Despite the fact that we have several diploma courses running, there is not much in the way of good material which can be published which can then reflect the status of the discipline while also contributing useful information to our members.

Grateful thanks to Ron Palmer and his team of willing members who keep the journal afloat.

C. Continuing our medical education

The buzz word is 'upskilling'. It is always pleasing to hear of the great amount of enthusiasm which comes from our banana bender members. It is a great shame that this enthusiasm is not infectious and does not travel south of the border. Outside Newcastle, that state with the most members apparently sleeps on. However, it is still pleasing to note that some activity is evident is WA, SA Vic, Tas and ACT. I did mention previously that there were funds from the divisions of General Practice which members could tap into to run interesting and worthwhile programs. It is feared that the goose that laid the golden egg will soon vanish and it could be that divisions of General Practice will contract considerably. Now is the time to structure a musculoskeletal program for your region.

D. Specialist status

We are continuing with efforts to have the discipline recognised as a specialty in its own right. It would be very nice for a separate stream within the faculty of Rehabilitation Medicine to be introduced. However it does appear that if specialist status is to be achieved we must do it alone. This is not a task of AAMM but rather of the Australasian Faculty of Musculoskeletal Medicine.

This year has seen some positive outcomes for our endeavours which are as follows:

a. The long awaited Government acceptance of funding for Outpatient Clinics in Musculoskeletal Medicine has been realised in the recent Federal Budget. Some members may be aware of the fact that it was Liberal policy to have Musculoskeletal Medicine clinics in public hospitals as part of their new health program. In the recent budget papers there is confirmation that monies have been set aside for establishing 6 centres for out patient clinics throughout Australia. These clinics will be staffed by members who have the post graduate diplomas in Musculoskeletal Medicine and it is hoped that we will be able to run such clinics in association with our Orthopaedic, Rheumatological and Rehabilitation & Occupational Medical colleagues.

b. This year we have for the first time, have a permanently paid business manager. This is a part time position filled by Linda de Clifford who monitors our activities quite closely. It is pleasing to have such an efficient person running the day to day affairs of the Association. Last count we had 296 members with about 220 of these paid up for the ensuing financial year. Unfinancial members will be approached in due course.

c. The long awaited off shore meeting with the Kiwis has finally materialised. Advertising with this met with a considerable degree of enthusiasm from members, but unfortunately this did not materialise in confirmed bookings. At the time of writing this report we have 26 Australians and 15 Kiwis attending. This number of 41 is supplemented by families, but are not paying the $250 conference fee which is needed to supplement the activities of the Association.Unfortunately last year's Canberra conference made a small profit and it is obvious that the Fiji conference will make only a small profit. We

therefore need to address the issue of the 1997 conference to highlight hands on teaching and recent advances in the discipline.

As President of the Association over the past year I have actively promoted the association with all my teaching activities. This year I have conducted weekend workshops in Hobart, Scotsdale, Kalgoorlie, Perth, Busselton, Canberra and Mt Gambier. The total number of participants was 75 and the feed back from these is always encouraging. Therefore it is difficult to understand why more members are not organising workshops. As well as the weekend workshops I have doctors sitting in on my outpatient clinics at the Flinders Medical Centre and the Queen Elizabeth hospitals and I run a Tuesday evening group on the first Tuesday of each month. This is attended by 8-10 doctors on a regular basis as we go through examining the various aspects of the musculoskeletal system. All this is in addition to running the diploma course and the requirements of attendance at the four 8 day modules which are associated with the diploma.

A pleasing note recently was the invitation to speak at the Annual General meeting of BIMM in April 1997. What this means to me is that Musculoskeletal Medicine is becoming an acceptable worldwide activity and our diploma courses based on the AAMM syllabus and protocol are widely accepted. This means that we are at the forefront of teaching and research in the area, but unfortunately the bulk of this burden is carried only by a few. There are many problems still before us and I would like to address these next.

(i) What is going to be the ongoing role of AAMM versus the Australasian Faculty of Musculoskeletal Medicine? Will we see a demise of AAMM? If there are no keen members to fulfil the teaching roles that the association has been pursuing over the last decade then the discipline will cease to be relevant to those in general practice. What is urgently needed are heirs apparent, which theoretically should come from the diploma courses. However, many of the diploma graduates are in rural practice where they are already heavily committed.

(ii) The whole of our membership needs to get firmly behind the arrangements of FIMM. As mentioned previously, it is gratifying to see such a small group of dedicated and enthusiastic musculoskeletal physicians in Brisbane and we look forward to seeing more of their challenging organising capacity in the next 18 months. It would be most encouraging if every member made the effort to be present at this historic meeting of masters in the knowledge and treatment in matters musculoskeletal. The FIMM conference will be held in the days immediately after Easter in 1998 on the Gold Coast. Further details of this will appear in this addition of the Journal.

Phil Watson and his faithful team are to be congratulated for their innovations and initiatives.

(iii) It has been suggested by the British and the New Zealand Presidents that a travelling scholarship fund be set up such that members involved in teaching and administering the associations be able to travel from one country to another so that the exchange of ideas will result in an informed and unified approach to the discipline. This will need to be discussed further by the new committee.

(iv) Last year we made the decision that the 1997 annual conference would be held at a ski resort in NSW. In view of that fact that the membership has poorly supported the Fiji concept it becomes necessary for us to rethink this decision. It might be better to have a 2 day conference in Sydney or Melbourne with an international speaker. This matter needs further discussion and the 1996 AGM should be entrusted with the final decision on this matter.

I would like to express my gratitude to all those executive and council members over the past seven years for their help, encouragement and co-operation in working together to forward the objectives of AAMM and the discipline. During this period I have learnt much while helping to lay some of the foundations upon which the future Musculoskeletal Medicine will be built. I plan to retire in 5 years so it is time for young and enthusiastic members to continue the building.

My final plea is to encourage those with diplomas and who are in full time Musculoskeletal practice to pursue the realisation of a Fellowship of the AFMM. Without Fellowship status we will not go anywhere. Although Fellowship within the Faculties of Rehabilitation and Occupational Medicine are available whereby one could practice the discipline full time, the ideal is for the discipline to stand alone. Endurance and perseverance are needed to overcome the current impediments.

Norman Broadhurst

A WORD FROM THE PRESIDENT OF N.Z.A.M.S.M.

Valedictory

As this is my last such message as President I would like to share my thoughts on the situation facing Musculo-skeletal Medicine today. Our Association was formed in response to a damning Report from the 1979 "Commission into Chiropractic in New Zealand", which made recommendations to correct the "gross neglect of Medical Schools and Physiotherapy Schools in teaching of spinal manipulation at undergraduate and postgraduate levels". Sadly with a change of Government the commission's report and its recommendations were ignored, presumably because of lobbying from the Establishment embarrassed by the report. In any event no change was made regarding the lack of training in MSM in the Medical Schools. As only one Hospital in N.Z. (Christchurch) has a Dept. of MSM, doctors are graduating with no exposure to MSM as a discipline within General Medicine. Not surprisingly, they are not willing to accept that their training is deficient, so they accept the view of *orthodox* medicine that MSM is *"alternative"* or *"fringe"* medicine and feel vaguely threatened by it. Efforts to raise the profile of MSM within *orthodox* medicine founder at this point. One recent initiative to raise the profile has been the formation of The Australasian Faculty of MSM, with the intention of legitimising MSM as a speciality in its own right. This may be assisted by affairs in Europe where Dr Michael Hutson, President of the British Institute of Musculoskeletal Medicine has introduced an initiative within FIMM to have MSM made a monospecialty within the E.C. If he is successful in this then all Europe will have to follow suit and a standardised European diploma of MSM will be set up. With the next FIMM Congress to be held on the Gold Coast in 1998, this will be where the decision to proceed may be made. If we could facilitate this, it would be a major step forward in achieving the objective of a speciality of MSM in our part of the world as well as ensuring that our own standardised diploma, the first in the world, will get universal recognition. The ball is in our court and we should work steadfastly towards this end; it would be a fitting achievement for the first FIMM Congress in Australia to elevate our discipline to specialty status.

A perennial problem within MSM is that of terminology. We speak of "dysfunction" as if it were a definitive diagnosis instead of merely saying that there is a problem at that level; "lumbo-sacral dysfunction" tells us no more than "lumbago". Until we can make an anatomical and pathological diagnosis, any treatment we advise is empirical. Any practitioner calling bronchitis or pneumonia "lung dysfunction" and treating the condition on the basis of such a diagnosis would be practising poor medicine. We should expect no less precision in diagnosis in MSM. As one who believes that if you can confidently exclude fractures (and other gross traumatic lesions), inflammatory, neoplastic and infective conditions, all other musculoskeletal conditions are caused by disordered muscle physiology. Our role is to identify the affected muscle and correct the disorder.

To ascribe the dysfunction to the joint involved without reference to the muscles controlling that joint is only partially stating the case. A "sacro-iliac joint dysfunction" can originate in quadratus lumborum, any of the lumbar paraspinal muscles, glutei or hamstrings and piriformis or any combination of these. Treating the dysfunction involves treating the affected muscle by any of a number of modalities at our disposal.

An osteo-arthritic hip is not primarily a disease of the hip but has its origin in the contracted external rotators of the hip which caused the attrition of the articular cartilage. This can readily be diagnosed and treated before there is loss of cartilage and thus MSM is capable of reversing the increasing incidence of joint replacement surgery of the hip or knee. Obviously this will be a major advance.

I regret that more of our members could not see their way clear to attend the Combined Meeting in Fiji. The unsettling influence of the medical reforms have caused practitioners to be very selective about where they obtain their continuing medical education. The Executive is investigating how we might award CME points towards accreditation for attendance at our conferences and courses without sacrificing our autonomy to the College of G.P.'s As yet the Medical Council has still to reply to our request for the registration of MSM as a separate discipline for the purposes of vocational registration.

In parting I would like to place on record my enjoyment at having served with such a fine group of practitioners as I have met from both associations. It seems that MSM attracts an agreeable type of follower.

Farewell to you all.

Angus Johnston.

FROM THE AAMM PAST HON SECRETARY'S DESK

The last year has indeed been an exciting time for Musculoskeletal Medicine in Australia. The recognition of the discipline by the federal health minister and the commitment to fund musculoskeletal outpatient clinics in our teaching hospitals is encouraging to say the least. At the recent annual general meeting of the Australasian Faculty of Musculoskeletal Medicine, Wade King and Prof. Nik Bogduk outlined the history of negotiations with the government over the past 7 years.

The Faculty has been commissioned to establish Musculoskeletal Medicine clinics in 13 major hospitals around Australia and to conduct audits and controlled trials of evidence based practice for the management of musculoskeletal health problems of the regional pain syndrome type. The project will be managed by a steering committee made up of representatives of the Faculty, the Department of Health, the A.M.A. and patient interest groups. Treatment protocols have been drawn up which still allow some flexibility of individual practitioner style. All treatment strategies will be under the microscope.

Speaking of assessing treatment strategies, the recent conference in Fiji was very interesting in a number of ways. Dr. Wolfgang Schamberger presented a system of examination looking at 'Malalignment of the pelvis and spine'. He claimed that most of us have some degree of malalignment that predisposes us to injury and pain. As I was having some difficulty detecting the abnormalities he was demonstrating, I designed a simple trial of inter-observer reliability. This involved 7 experienced practitioners examining 6 subjects for differences in iliac crest and posterior superior iliac spine (PSIS) height in standing and sitting, forward flexion and stork tests (assessing sacro-iliac joint movement) and anterior superior iliac spine and pubic symphysis heights in supine.

The results are not all tabulated as yet, but will be published in full in the next edition of this journal. In essence there was poor correlation. There was not one single sign in any subject that all examiners could agree on. Elsewhere in this issue appears another study undertaken at the same venue with similar results. Of major concern is that even the experienced examiners can not agree on the signs. The main lesson I gleaned from this is that we must re-evaluate our teaching methods and programs. We must critically assess the validity and reliability of each examination technique and standardize their teaching. This requires co-ordination and co-operation on all fronts.

On a further note about research, I would like to publicly thank Dr. David Collinson for sending in his completed headache survey. Two other co-opted members have since replied, but this is not enough to base a research project on. The details are in the last journal issue. If you want something to read in the next issue, please help.

Vic Wilk

LETTERS TO THE EDITOR

SCIENCE OR NOT SCIENCE - THIS IS THE QUESTION

In January this year Christchurch hosted the International Society for the Study of the Lumbar Spine (I.S.S.L.S.) Conference. Here, highly qualified Surgeons, Neurologists and Orthopaedic Surgeons presented interesting new operative technology and techniques for the lumbar spine that were far in advance of those we had available to us 25 years ago.

To become a member of the I.S.S.L.S Club you have to be elected and the Society has only 200 members. Once elected, you must perform to the Society's high standards of research science, otherwise you will be asked to resign. This means for each member the privilege of membership is required to be justified by highly scientific performance.

As a result no study presented at the Conference failed to achieve the status of a controlled and double blinded trial. This applied even to the multi-centre studies that were presented. The results of cases were reported only where a scientifically accepted diagnosis was able to be made and where an operation was able to be performed.

The literature records several estimates of operation rates for low back pain. Krämer[1] reports that operations are carried out on 0.25% of his low back pain patients. Gordon Waddell[2] of Edinburgh operated on 1% of his patients and finds 95% of his low back pain patients have no pathology. In the 1980s, Wood[3] followed up on 10,000 patients attending their General Practitioner with low back pain and arrived at a rate of 1.6 per thousand who finally underwent surgery. According to Melzack & Wall[4], 60-78% of low back pain patients have no clinical findings.

In other words, with regards to low back pain the members of this bastion of modern science do not mind talking for 5 days exclusively about 5% of all low back pain sufferers. On 'the rest' there is silence - does this then mean that 95% of the world's low back pain patients are malingerers, drug seekers, compensation seekers or people too lazy to work and who try to live on the insurance system of their countries? There may indeed be a few such patients, but the bulk of these patients are people such as you and I, apart from the fact that they are walking around for years or decades with the pathology ofdysfunction. Dysfunction is the most common pathology for low back pain, but is not regarded as the scientific diagnosis. This pathology, although a treatable condition is still today widely unknown and, as a consequence, not accepted by modern established medicine.

Its non-acceptance leads to the repeatedly heard statements: 'We have examined and investigated you - there is nothing wrong', or even: 'This is all in your mind'. (The latter being the most common 'diagnosis' when we fail to find a reason for a patient's complaints).

Apparently, science and medicine is a golden calf which we are bound to dance around and if science is able to make a diagnosis in only 5% of low back pain patients then the rest are not allowed to exist. Scientists trust their x-rays, their laboratory findings, myelograms, MRIs, nerve conduction studies and so on, yet are unwilling to trust their own clinical examination techniques. However, you cannot make a diagnosis of a dysfunction without a segmental examination of the spine.

The techniques for clinical examination and treatment have been available for decades. There are objective signs of dysfunction which we are able to see with the naked eye or with a camera and there are subjective signs which we can palpate with the examining finger. Together these form very effective empirical methods, which however are considered non-scientific.

Have our scientists forgotten that every new development in medicine has been empirical and not scientific? We are, after all, sitting on a huge mountain of knowledge which the generations before us have piled up on the basis of trial and error.

A good example for this was given by Melzack & Wall themselves. They referred to an observation recorded in 1763 by English Country Clergyman, Edward Stone that the extract from willow was good for rheumatism and fever. It wasn't until 1827 that Leroux isolated the active component from the willow (Salix alba) and named it Salicin. Still later, in 1899, Dreser produced acetyl salicylic acid which was then marketed by Bayer under the trade name of aspirin. Even today, scientists are troubled about how aspirin really works. In spite of the purely empirical knowledge of willow extract, millions of patients using it over the centuries have experienced relief from pain and fever. On the one hand modern scientific medicine does not accept, or has difficulties accepting, that dysfunction is the most common reason for low back pain. On the other hand medical science fails completely to give an answer to 95% of all low back pain sufferers. We are sitting in the scientific trap.

The only way yet known out of this trap is the empirical way. Musculoskeletal Medicine, and the different philosophies of Osteopathy and Chiropractic each offer this way. Every dysfunction, no matter for how many years it has been present, remains a treatable condition (with only a few exceptions). The results of the treatment are dependent only on the experience and skill of the treatment provider. I encourage my experienced Musculoskeletal Medicine colleagues not to feel domineered by scientists, who do not know any better. Continue to treat the remaining 95% of low back pain, daily, to the benefit of the pain suffering patient.

I also encourage my younger colleagues to train their eyes and their palpating fingers in these examination and treatment techniques. Empirical medicine needs you urgently to be able to cope with the ever growing problem of pain in modern society.

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Yours sincerely,

Clemens Franzmayr, Christchurch, New Zealand

10th October 1996

The Editor,
The Australasian Journal of Musculoskeletal Medicine,

Dear Sir,

CAREER PATHWAYS IN MUSCULOSKELETAL MEDICINE

After many years of Musculoskeletal Medicine Practitioners working alone, and "out on a limb", clearer career pathways have emerged.

The 1990's will be looked on as the decade that changed the face of Musculoskeletal Medicine in Australasia.

With the development of the Diplomas in Musculoskeletal Medicine, firstly in New Zealand and later in Australia, Doctors are able to obtain skills and knowledge at University level above or beyond other short courses that are currently being offered. Doctors completing the Diploma are equipped with expertise needed to manage patients that are referred by colleagues.

Thus G.P.s with a Diploma in Musculoskeletal Medicine are now able to provide an extremely valuable and useful medical service to patients. As the G.P.s interest and experience in Musculoskeletal Medicine increases the practice profile may also change. Another option for G.P.s therefore has been the slanting of their practice more and more towards this discipline and even for some G.P.s to practice this full time. Initial G.P. training (eg. Fellowship of R.A.C.G.P.) then the later development of a special interest in Musculoskeletal Medicine is now endorsed by the R.A.C.G.P.

The formation of the Australasian Faculty of Musculoskeletal Medicine in 1994 was another major landmark. Members of this Faculty are either graduates of a Diploma in Musculoskeletal Medicine or Fellows from other recognised Specialist Colleges who have a major interest in Musculoskeletal Medicine.

The Faculty has emerged relatively quickly with confidence, vigour, direction and purpose. The proposed development of The National Musculoskeletal Medicine Health Initiative is the result of the Federal Government injecting $7.2m into the A.F.F.M.M. to run a three year trial of Musculoskeletal Medicine practice in Hospital Outpatients. The Outpatient staff will have evidence based guide-lines. A continuing education could develop from the audit reviews forming a Fellowship training programme. It is planned to hold the first Fellowship examination in 1998.

By this time, the A.F.M.M. will have had more time to mature, have an established presence in hospitals for treating patients, and for training (e.g. Registrars), practising evidence based medicine and will have incorporated a stage one examination, (i.e. the Diploma), followed by a Fellowship training programme, and examination. The Faculty may then be in a position to be considered for Specialist recognition?

This is of course, a personal viewpoint. My intention is to draw your readers to the possibilities of a more structured career in Musculoskeletal Medicine. The Doctor can choose his or her involvement at several distinct levels. These are exciting times and I urge the younger General Practitioners in particular to engage in post graduate medical education in Musculoskeletal Medicine and to take advantage of the opportunities now available which were only dreamt of a short time ago.

Yours sincerely,

DR P.N. WATSON.

THOMAS A. DORMAN, M.D.
INTERNAL AND ORTHOPAEDIC MEDICINE
A Professional Corporation

May 10, 1996

Diplomate of American Board of Internal Medicine

Dr. Ron Palmer
Editor
Australasian Musculoskeletal Medicine
Suite 24 Royal Brisbane Place
17 Bowen Bridge Road
Herston QLD 4006

Dear Doctor Palmer:

I was interested in reading Dr. Broadhurst's report from the San Diego conference on Low Back and the Sacroiliac Joint in Vol.1 No.4 (April) of your *Australasian Musculoskeletal Medicine* journal from 1996, pages 23-24. In it, Dr. Broadhurst comments on the use of sclerosing and sugar solutions and states, "As yet, there are no good scientific trials to support these anecdotal findings..."

Would you allow me to point out to the readers of your journal that two double-blind control studies have been performed in California establishing the benefit of these treatments. The treatments are called *prolotherapy*. The studies are Ongley MJ, Klein RG, Dorman TA et al, *A new approach to the treatment of chronic back pain*, Lancet 2:143-146, 1987, and Klein RG, Eek BC, DeLong WB, Mooney V, *A randomized double-blind trial of dextrose-glycerin-phenol injections for chronic low back pain*, J Spinal Disord 6:23-33, 1993. The whole subject has been reviewed extensively in *SPINE: State of the Art Reviews: Prolotherapy and the Lumbar Spine and Pelvis*, edited by the undersigned, published by Hanley & Belfus, Inc., 210 S. 13th Street, Philadelphia, PA 17107, telephone (213) 546-4995, fax (213) 790-9330, in their Vol.9 No.2 issue from May 1995.

Respectfully yours,

Thomas A. Dorman, M.B., Ch.B., MRCP(UK), FRCP(C)

TAD/cbp

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ORTHOPAEDIC MEDICINE CLINIC
(DONALD M. FRASER, M.D., P.C.)
Diplomate, American Board of Neurological and Orthopaedic Surgery in Orthopaedic Medicine.

4/3/96

Letter to the Editor:
Australian Association of Musculoskeletal Medicine,

Dear Sir :

Re: Prolotherapy (a.k.a. Sclerotherapy in Canada and U.K).

In your Newsletter No.1, 1996, you refer to Prolotherapy which is a procedure which I learned in 1975 from the late James Cyriax and the late Ron Barbor. Anecdotally, I have had great results, approximately the same as the study published by Ron Barbor(seen enclosed reprint which I give to my patients for their information).

Recently, there has been a book published by Hanley and Belfus in the "STATE OF THE ART REVIEWS" edited by Tom Dorman of California. The book is subtitled "SPINE" and "PROLOTHERAPY IN THE LUMBAR SPINE AND PELVIS".

For your members who might be interested in expanding their therapeutic armamentarium, the ISBN # is 1-56053-187-8. I shall enclose a photocopy of the publisher for your information.

I am looking forward to your Newsletter and Journal, as I have recently joined the organization.

Fraternally,
D.M. Fraser M.D.
Diplomate:
Society of Apothecaries (UK) in Musculo-Skeletal Medicine

Editor:
An enclosed article titled "Sclerosant Therapy" by the late Dr. R. Barbor, U.S.A., was also forwarded. This was then reviewed by the editorial panel and was excluded from our publication on the basis that it would have required a massive re-write and that the scientific basis of the article itself was questionable.

EFFECTS OF EXERCISE & LOADING ON LUMBAR DISCS & ARTICULAR CARTILAGES: DOES HARD WORK OR STRENUOUS EXERCISE DAMAGE LOAD-BEARING CARTILAGES?

A mini-review: James R Taylor

Structure & Function in Cartilage

The cartilages of the hip joints, knee joints and the lumbar discs, share the functions of load bearing and movement. The cartilage cells produce proteoglycans and collagen fibres to serve these functions. The diarthroses provide free, sliding movements on smooth lubricated surfaces; the compliance of the articular cartilages bears loads and attenuates impacts. In the lumbar discs, the collagen fibres and proteoglycans provide for load bearing and shock absorption in strong joints where tissue compliance allows limited movements.

Articular Cartilages

Movement is essential for articular cartilages nutrition. Movement circulates synovial fluid between the joint cavity and the avascular, cartilage matrix (Stockwell, 1979; Salter, 1989). Articular cartilages have a deep calcified zone, which joins the avascular cartilage to the vascular subchondral bone plate; this zone is almost impermeable to diffusion of nutrients. It is thin in young people, allowing some diffusion, but thick in middle aged and older people, preventing significant diffusion. Collagen bundles, binding the cartilage together, are arranged perpendicular to the subchondral bone plate; they pass through the deep and mid-zones, then arch and radiate to run tangential to the articular surface.

Intervertebral Discs

Lumbar intervertebral discs are the largest avascular structures in the body. The anulus fibrosis and cartilage plates enclose the nucleus pulposus. The 12-15 fibrous lamellae are arranged in two distinct zones, an outer ligamentous zone which joins the vertebral rims and an inner, PG-rich zone, whose fibres are continuous with the cartilage plates forming an elliptical envelope for the nucleus (Finch & Taylor, 1996).

Strength and Load Bearing in Discs

The outer, ligamentous anulus withstands tensile forces. The inner anulus and the nucleus share the function of axial load bearing. The inner cartilaginous annulus bears compressive loads, because each collagen fibril is supported by a water-attracting, inflatable PG collar. In addition, the enclosure of the incompressible but deformable PG-rich nucleus by the anulus and the cartilage plates has a load bearing function. The PGs attract water into the nucleus, creating a swelling pressure, which resists externally applied, compressive loads. Prolonged external compression causes creep shrinkage, as water is squeezed out of the disc.

Intermittent Loading, Nutrition and Cell Function

Intermittent loading and unloading promote fluid exchange between the synovial fluid and articular cartilages, and also, to a limited extent, between the intervertebral discs and the vascular vertebral spongiosa. In addition, when living cartilages are intermittently loaded at physiological loads, the cells respond by producing PGs. The PG concentrations are higher in the nucleus of lower lumbar intervertebral discs in humans, where axial loading is greatest and lower at the centres of cervical discs where axial loading is least (Scott et al, 1994). Animal experiments show that moderate exercise increases the PG concentration in ACs while repeated, very prolonged and strenuous exercise causes site dependent decreases in PGs in the superficial zones of the weight bearing summits of the femoral condyles (Sammanen et al 1994 Aronski et al, 1994).

Nutrition & Age

Articular cartilages and intervertebral discs are avascular in adults. During infancy and early childhood the discs show progressive decrease in vascularity so that by 4 years there are few blood vessels left and by adult life a few vessels penetrate the calcified layer joining the cartilage plates to the vertebral bodies (Taylor, 1974). In young articular cartilages a few small blood vessels penetrate the deep, calcified zone but in older cartilages, the thickening of the calcified zone means that nutrition can only be from synovial fluid exchange (Stockwell, 1979).

In the discs, the vascular vertebral end plates remain the principal sources of nutrition for the avascular cartilage. From about 10 years onwards the size of lower lumbar discs is such that the central disc cell population falls off dramatically and the production of GAGs is by an anaerobic mechanism so that Keratan sulphate becomes the common GAG rather than the chondroitin sulphates found in young disc cartilages (Scott et al, 1994).

Joint movement enhances articular cartilage nutrition and continuous passive motion aids in repair of injured cartilages (Salter, 1989). The nutrition of lower lumbar discs is more critical and it is probably aided less by movement, as the almost continuous daytime load bearing causes creep of fluid out of the discs rather than entry of fluid into the discs. Loading in fixed postures redistributes water and electrolytes within the discs (Twomey & Taylor, 1982). When the discs are unloaded at night, in a prone or supine body position, fluid re-enters them. In addition to these diurnal, mechanical changes there is constant passive diffusion of water and

solutes in and out of the discs. Some studies suggest that movement enhances disc nutrition in experimental animals (Holm, & Nachemson, 1983); other studies do not support this view (Katz et al., 1986).

Moderate exercise with regular loading and unloading is beneficial to all cartilages. Prolonged bed rest, on the other hand, is harmful, leading to long term swelling changes in the discs, with delayed recovery on loading; prolonged loading in one posture has a detrimental effect on nutrition of the loaded part of a cartilage and creep shrinkage reduces its biomechanical efficiency.

Osteoarthritis

Many statements in the literature, based on retrospective studies, suggest that prolonged strenuous exercise predisposes to OA in the large weight bearing joints, especially the knees in females and the hips in males (Lane, 1995). Animal experimentation supports the view that prolonged strenuous running exercise favours the development of OA, with depletion of PGs and softening of cartilage, though it also shows that moderate, short term running exercise increases the PGs in AC without damage to the joint (Saamanen et al 1994; Aronski et al, 1994). In recent reviews, Lane (1995) states that there is no sound evidence from controlled prospective studies that lifetime sport such as endurance running leads to early osteoarthrosis in humans. It is accepted, that in injured or abnormal joints, OA may result from such repetitive, strenuous loading of the joints. On the other hand regular movement of synovial joints is essential to their health and regular use with low loading promotes healing after injury. Salter's experiments in animals (1989) show clearly that certain types of perforating injury to articular cartilages heal more effectively with continuous passive motion while immobility delays and inhibits healing. There is more limited evidence that the same effects of continuous passive motion apply to humans.

Disc Degeneration:

Epidemiological studies of the effects of exercise and loading or intervertebral discs show that lifetime handling of heavy materials, working in fixed bent twisted postures and intensive sporting exercise with risk of repeated minor injuries as in weight lifting or soccer can lead to early lumbar disc degeneration (LDD) while other activities like running are neutral in their effect on LDD and life time sedentary employment is relatively protective of intervertebral discs (Horal, 1969; Lawrence, 1969; Riihimaki, 1989 & 1990; Videman, 1995; Battie, 1995).

Some types of exercise or occupational activities have effects on specific joints. Boxing on hand joints, baseball pitching on shoulder joints, ballet on foot joints, weight lifting on upper lumbar discs, fast bowling on lumbar laminae and discs and soccer on lower lumbar discs and on ankle and possibly on knee joints; mining affects knee joints, some textile work affects hand joints, while heavy manual work and fixed bent postures as in sheep shearing or brick laying, imperil all lumbar joints.

It is of equal interest, that while there is a moderately good, though inconsistent correlation between lumbar disc degeneration and low back pain (Lawrence 1969; Sachs 1987; Vanharanta et al 1989; in the study of athletes with disc degeneration, athletes with LDD who remained active had less back pain that sedentary controls who were presumably inactive Videman 1995). In a review and prospective study of the effects of leisure time physical activity and low back disorder, Leino (1993) found a good effect of exercise in preventing low back pain in men.

Slow healing in Discs:

Observations of surgically injured discs show that repair is absent or slow and the surgical lesions promote increased degeneration (Osti et al, 1990). Observations of injuries to cervical discs support this view (Taylor & Twomey, 1993).

In summary, avascular cartilages depend on movement for nutrition and for the continuing production of the collagen and PGs essential to their load bearing and movement functions. Repeated low impact or low loading movements favour cartilage health while overloading and high impacts lead to premature degeneration. Moderate regular exercise is good for bones, joints and muscles but overuse and lifetime overloading are damaging.

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Structural and Functional Changes in Articular Cartilage with Ageing, Mechanical Loading and Osteoarthritis

Chris Little and Peter Ghosh

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Introduction

The specialised bio-mechanical composition of articular cartilage is discussed in detail.

The synthesis for PG's by articular chondrocytes decreases with increasing age and chondrocytes from more superficial zones synthesise less PG's than deeper cells. Mechanical loading tends to increase PG synthesis from the articular chondrocytes. Conversely, a lack of loading results in a depletion of PG's, thus mechanical compression acts as a stimulant for PG synthesis.

Osteoarthritis is a disease of arthodial joints characterised by progressive destruction of articular cartilage. Degeneration of articular cartilage may occur through continuation of abnormal synthesis and catabolism of various matrix components. There is evidence that chondrocytes from OA cartilage have an altered phenotype and that chondrocytes from different joint regions may vary in defininative responses. Mechanical trauma, either as a single event or from repetitive stress may lead to phenotypic changes.

The key to understanding OA changes, and their future management, may lie in the early identification of "pre-osteoarthritic" abnormalities.

Articular cartilage covers the ends of bones in synovial joints and in conjunction with synovial fluid provides a resilient weight bearing surface essential for normal articulation. The structure-function relationship of articular cartilage has been well described in a recent article by Mow et al., [125]. Articular cartilage serves three main functions:

(1) it minimises contact stress by distributing load over a larger contact area;

(2) it dissipates some of the energy associated with weight bearing; and

(3) it permits almost frictionless movement between the two articulating surfaces of a joint.

In the adult joint, articular cartilage is avascular, aneural and alymphatic. The cells of this tissue (the chondrocytes), which account for approximately 10% of the tissue wet weight, are responsible for the synthesis and catabolism of the matrix components and derive their nutrition by diffusion of nutrients from the synovial fluid.

The articular cartilage matrix is composed of hydrophilic proteoglycans (PGs) which account for roughly 5-10% of the wet weight, entrapped within a network of inextensible collagen fibres (10-30% of the wet weight). The bulk of the water content (60-80% of the wet weight) of articular cartilage is maintained in a gel by the osmotic force of the highly negatively charged PGs. The water content of the tissue is important in maintaining the tissue's resilience under compressive deformation. A disruption of the normal three dimensional biochemical architecture of articular cartilage renders the tissue susceptible to mechanical damage.

Normal Articular Cartilage

The thickness of articular cartilage varies between species, joints in an individual, topographical location within a joint and between individuals of different ages [48,82-84,173]. In general, articular cartilage is thicker in the larger joints, in regions exposed to the highest weight bearing stress and in joints of younger individuals. The articular cartilage of sexually mature individuals has been divided into four zones on the basis of depth from the surface [14,40]. The superficial zone or tangential layer contains flattened chondrocytes and tangential collagen fibres oriented parallel to the cartilage surface (Figure 1). The intermediate or transitional zone is characterised by larger, rounded, single or paired chondrocytes and randomly oriented collagen fibres. The broadest layer, known as the radiate or deep zone, contains chondrocytes arranged in vertical columns separated by collagen fibre bundles that have an overall radial arrangement and a larger diameter than those of the upper two zones [137]. The basophilic "tidemark" separates the radiate from the calcified cartilage layer which abuts onto the subchondral bone. The calcified zone is sparsely populated with large hypertrophic chondrocytes and the matrix is mineralised with crystals of calcium salts. The collagen fibres

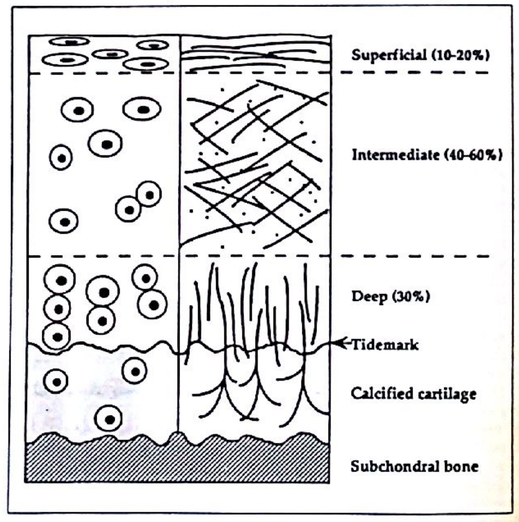


Figure 1.
Schematic representation of articular cartilage demonstrating the cellular (left panel) and collagen (right panel) morphology within the different zones.

in the deep zone cross the tidemark and divide repeatedly in the calcified cartilage zone to form an interlocking network which anchors the cartilage to the subchondral bone [143].

Cartilage Collagens

The collagen fibres in articular cartilage serve to resist tensile forces and maintain the three dimensional shape of the tissue. In adult articular cartilage approximately 80-90% of the collagen molecules are of type II [27,90]. The other so called "minor collagens" of cartilage consist of types IX, XI [27,49,50,VI 63,140] and in calcified cartilage type X 55. Recently type III collagen has also been demonstrated in the pericellular region of normal human articular cartilage [198,202]

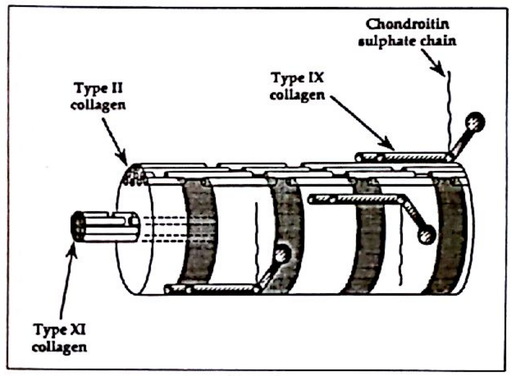


Figure 2.
Schematic representation of the cartilage collagen fibril. A core of quarter-staggered type XI collagen molecules is surrounded by type II molecules. Type IX molecules are attached to the surface of the fibril in an antiparallel manner. (molecules are not drawn to scale. Adapted from Bruckner et al.27)

The major collagen fibrils in articular cartilage are a heterogeneous polymer of collagens II, XI and IX with a very defined architectural arrangement [27] (Figure 2). In this model, a core of quarter-staggered collagen type XI molecules is coated by type II molecules. Collagen type IX is attached to the surface of this composite fibril and together with type XI likely plays a role in controlling fibril diameter. The stability and cross-linking of the articular cartilage collagen network is important for maintaining the structural integrity of the tissue. Type VI collagen, which makes up the fine fibrillar network of the chondron, has several cell binding sites [4], interacts with hyaluronan [79] and specifically binds to type II collagen and a small PG decorin (DS-PG II) 16. Thus type VI collagen may act as an intermediate binding molecule between the chondrocyte and the surrounding interterritorial matrix. The type IX collagen molecules which reside on the outside of the type II/XI fibres have an attachment site for a glycosaminoglycan (GAG) side chain [27,49]. In articular cartilage this GAG chain on type IX collagen contains chondroitin sulphate although not all of the type IX collagen molecules are glycosylated. It has been proposed that type IX collagen is a source of covalent links between separate type II/XI fibrils [126,200]. These interactions would enhance the mechanical stability of the fibril network, a property necessary to resist the swelling pressure of the entrapped proteoglycans. Enzymatic degradation of collagen types II and/or IX reduces the elastic stiffness and increases tissue swelling in articular cartilage which is consistent with the proposed biomechanical role of these molecules in the three dimensional organisation of cartilage[11,19,190].

Proteoglycans

Proteoglycans are specialised macromolecules which consist of a protein core to which glycosaminoglycan (GAG) chains and N- and/or O- linked oligosaccharides are covalently attached. PGs are a family of molecules which exhibit great variation in composition with regard to both their core protein sequences and the number and type of attached GAGs. In articular cartilage the three predominant PG associated GAG subtypes are chondroitin sulphate (CS), keratan sulphate (KS) and dermatan sulphate (DS), although small amounts of heparin sulphate (HS) have recently been isolated from bovine articular cartilage [175]. The non-proteoglycan GAG of articular cartilage, hyaluronan (HA), is an important component of the extracellular matrix of cartilage, where it represents the core filament for aggregation of PG monomers.

Aggrecan: The large PG which aggregates with HA, "aggrecan", is the most abundant species in terms of mass in articular cartilage [66]. Aggrecan has a core protein of approximately 230 kDa to which both CS and KS chains are attached (Figure 3). These GAGs make up 90% of the total mass of the secreted PG [65]. The core protein is a single gene product which contains several distinct domains [47,88]. The N-terminal region of the core protein has 2 globular domains (G1 and G2) separated by a 21 nm extended segment (E1). The major extended segment (E2) is approximately 260 nm long and bears much of the KS and all of the CS chains. The E2 can be divided into the N-terminal KS rich region followed by a longer CS rich region. At the C-terminal of the aggrecan molecule lies a third globular domain (G3). In immature cartilage 55% of the aggrecan molecules contain the G3 domain while this figure falls to about 35% in adults [52].

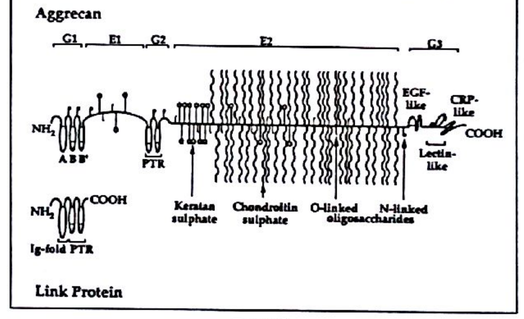


Figure 3.
Diagrammatic representation of aggrecan and link protein. (Adapted from Hardingham et al.[66]) Abbreviations: G1 = globular domain 1, E1 = extended domain 1, G2 = globular domain 2, E2 = extended domain 2, G3 = globular domain 3, A, B and B' = disulphide bonded loops, Ig-fold = immunoglobulin-like motif present in the A loop, PTR = proteoglycan tandem repeat of the B abs B' loops, Lectin like = region with similarity to mammalian type C lectin, CRP-like = region with similarity to a compliment regulatory protein, EGF-like = region with similarity to epidermal growth factor, COOH = carboxyl-terminus, NH2 = amino terminus.

The G1 domain consists of an immunoglobulin-like (Ig-like) fold and two disulphide bonded loops (the proteoglycan tandem repeat or PTR) (Figure 3). This region is responsible for the binding of aggrecan to HA and to link protein which stabilises this interaction. Link protein, a 39 kDa protein, has marked homology with G1, containing an Ig fold and a PTR. The G2 domain has extensive homology with G1 but its function is unknown. The G3 domain has homology with a

hepatic cell surface lectin, specific for galactose and fucose but whether this imparts matrix binding properties to this portion of the aggrecan molecule is not known [44]. The E1 region has been extensively studied in recent years as it appears to be the primary site for proteolytic cleavage of aggrecan in both normal matrix turnover and pathological degradation [66].

The binding of link protein and or G1 to HA causes a defined spatial organisation of the HA molecule [119]. A limiting factor in the size of the aggregates is the length of the HA chain, with decasaccharides being the smallest HA fragments able to bind aggrecan [47]. Buckwalter et al.,[28] have demonstrated on the basis of sedimentation velocity that two populations of aggregates exist in adult cartilage. These differences were associated with the number of aggrecan molecules per aggregate (15 compared with 44) rather than the length of the HA chain. This result was consistent with the findings of Müller et al.,[124] who suggested that the higher density aggregates may allow for "packing" more PGs into cartilage in areas where greater compression resistance is required.The proportion of aggrecan monomers that are aggregated with HA and the number of monomers per aggregate declines with increasing age [29]. In-vitro studies have suggested that the decreased percentage of aggregates may be partly associated with a decrease in synthesis of link protein [136]. The percentage of aggregates has also been shown to be reduced as depth from the surface increases [54,124].

The E2 region carries more than 100 CS chains and about 20-50 KS chains in addition to O-linked oligosaccharides [30]. However, variations in the chemical composition of aggrecan monomers with both age and cartilage depth have been extensively described. With both increasing age and depth the percentage of KS increases [71,77,204]. The length of the CS chains is similar in all cartilage zones although controversy exists as to whether the chain length decreases with age or remains relatively constant [54,77,149]. The ratio of C6:C4 sulphation of the CS chains increases with age [71] but decreases with increasing depth from the surface [86]. The size of the aggrecan monomers decreases with increasing age and depth, associated with increased cleavage of the C-terminal portion of the E2 and associated loss of portions of the CS rich region [54,77,186,191].

The primary function of aggrecan within the cartilage matrix appears to be in providing the tissue with its elastic resilience under compressive load [123]. The anionic hydrophilic GAG chains of aggrecan absorb water into the matrix but they are maintained in an under-hydrated state within the constraining collagen network. When a compressive load is applied to the tissue there is an instantaneous increase in hydrostatic pressure within the cartilage. When the load is removed there is an elastic recoil of the tissue. If the load is continuously applied there is extrusion of water from the matrix and deformation of the tissue until a new equilibrium is reached ("creep" response). When the load is removed water is resorbed and the original tissue shape regained. An essential requirement of the functional response of articular cartilage to compressive loading is the immobilisation of aggrecan within the matrix. This can only be accomplished by the aggregation of PGs with HA and their entrapment within the fibrous collagen network as described earlier.

*Biglycan and Decorin:*In addition to the space-filling aggregated PGs, articular cartilage from a number of species has been shown to contain several small PGs that do not aggregate with HA 172. Two of these PGs, biglycan (DS-PG I) and decorin (DS-PG II) belong to the family of small interstitial PGs which have highly homologous leucine-rich core proteins (Figure 4). In articular cartilage the DS-PGs represent 1-4% of the total mass of PGs present within the tissue [150,157]. However because of their small molecular size they may be present in similar molar concentrations to aggrecan [172].

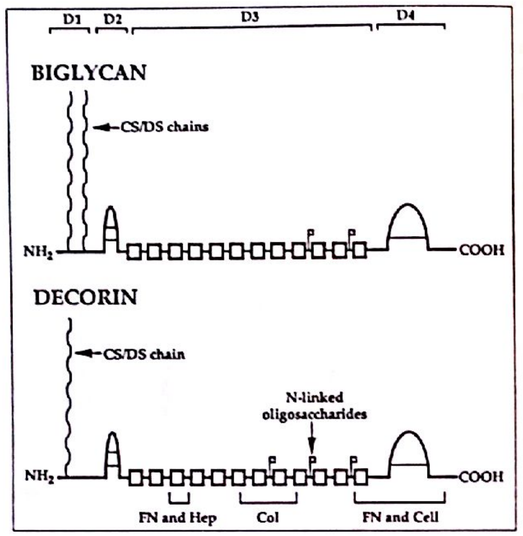


Figure 4.
Schematic representation of biglycan and decorin showing the putative active regions on the core proteins, and attachment sites for GAG chains and oligosaccharides. Abbreviations: NH2 = amino terminus, COOH = carboxyl-terminus, D1 = amino terminus and GAG attachment region, D2 = disulphide bonded region, D3 = extended, leucine rich repeat region, D4 = carboxyl terminus with a single disulphide bond, FN = fibronectin binding regions, Col = collagen binding region, Hep = heparin binding region and Cell = cell binding region.

The core proteins of the DS-PGs have molecular weights of approximately 37 kDa (37.3 kDa for DS-PG I and 36.4 kDa for DS-PG II) [145]. The core proteins of the two DS-PGs show extensive homology (approximately 55%) even between different species, however the first 20 amino acids at the NH2-terminus of DS-PG I and DS-PG II are completely different [41,51,89,127,164]. DS-PG I contains 2 GAG side chains attached to serine residues at positions 5 and 10 for human or 5 and 11 for bovine molecules [51,127,150]. DS-PG II possesses only one GAG chain on the serine residue in position [4 37]. The GAGs of the DS-PGs from both human and bovine articular cartilage contain approximately 40% iduronic acid residues [116].

The iduronic acid moieties of the DS-PG GAG chains are C4 sulphated in human articular cartilage [116]. The DS chains are a mixture of iduronic and glucuronic acids however and the latter may be sulphated at C4 or C6. It has been demonstrated that the concentration of DS-PG II in human articular cartilage increased between 5-20 years of age, remained relatively constant between 20-40 years and then declined in individuals over 55 years [157]. Melching and Roughley[116] demonstrated that in fetal human cartilage DS-PG I was the major DS-PG synthesised while in adults DS-PG I synthesis was barely detectable and DS-PG II synthesis was increased.

The distribution of DS-PG I and DS-PG II within articular

cartilage shows both age and zonal variations. Immunohistological studies have demonstrated that in adult bovine and human articular cartilage both DS-PGs show a decreasing concentration with increasing depth from the surface [117,139,169] In the case of DS-PG II there is a slight increase in staining in the deepest cartilage zone however this is still less intense than in the superficial cartilage [117,139]. The distinct zonal distribution of DS-PG II was not apparent until 10 months of age [139]. It has been demonstrated with electron microscopy that both DS-PGs could be immuno-localised to specific but separate sites on collagen fibrils [117].

The function of the DS-PGs in articular cartilage has not been fully elucidated and many of the proposed roles for these molecules have been derived from in-vitro studies and their putative functions in other tissues. Nevertheless, it appears that the DS-PGs function within articular cartilage is to modulate:

(1) collagen fibril size and three dimensional organisation;

(2) cell adhesion to matrix proteins and

(3) cell proliferation [88,145,172].

The effect of DS-PG II/collagen type I interaction has been studied in detail by Rosenberg et al. [146]. These workers concluded that DS-PG II inhibits the lateral association of collagen monomers in all stages of collagen fibril assembly, including the lag phase. However DS-PG II did not inhibit the end-to-end association of monomers with cross-linked oligomers to form microfibrils. In contrast to the earlier work of Brown and Vogel [75] and Hedbom and Heinegård [70], it has recently been demonstrated that DS-PG I does indeed specifically associate with type I collagen fibres both in-vivo [117] and in-vitro [165]. The DS-PG GAG chains appear to have importance in the three-dimensional organisation of collagen fibrils [166]. It is proposed that the GAG chains of DS-PG II molecules associated with the exterior of adjacent collagen fibrils, aggregate in an antiparallel duplex. The interacting GAG chains act as cross-linking bridges where the length of the DS chains will determine the separation between fibrils. This hypothesis has been supported by the observation that GAG chains from DS-PGs of the inner temporo-mandibular disc are longer than those from the peripheral regions of this structure and that the inter-fibrillar distance of the inner disc is greater than that of the outer disc [167].

The attachment of cells to fibronectin is mediated via specific cell membrane integrins binding to a distinct cell-binding domain on the protein, containing the amino acid sequence RGD [172]. An auxiliary mechanism involves cell surface heparin sulphates binding to a heparin binding domain on fibronectin [151]. The effect of DS-PGs on cell binding to fibronectin have been extensively studied by a number of laboratories. It has generally been shown that binding of DS-PGs to fibronectin inhibits subsequent cell attachment to the RGD domain of this protein [17,96,195]

The L-iduronate-rich GAG chains of the DS-PGs have been shown to inhibit cell replication in the presence or absence of growth factors[192,193]. Further growth modulating activities of the DS-PGs reside in the specific binding of transforming growth factor-b (TGF-b) to their core proteins [72,177,201]. This specific TGF-b binding activity is shared by fibromodulin, another member of the leucine-rich small PG family [72]. The intact PGs do not bind TGF-b as effectively as the GAG-free core proteins, suggesting that the GAG chains may hinder interaction between the core protein and growth factor [72]. Collagen associated DS-PG II retains its ability to bind TGF-b, although the binding is of lower affinity [69]. The result of DS-PG/TGF-b binding has been shown to variably inhibit, enhance or not change the activity of TGF-b depending on the cell system studied [69,177,201].

From the above it is clear that the DS-PGs have potentially diverse functions within the extra-cellular matrix of articular cartilage. Rather than acting as mechanical load-resisting structures like hydrated aggrecan, the DS-PGs appear to function as modulators of tissue organisation, cell proliferation and matrix adhesion. In this regard both the core proteins and GAG chains of the DS-PGs appear to play significant roles.

Articular Cartilage Metabolism

It has been suggested from both in-vitro and in-vivo studies that aggrecan in the extracellular matrix resides in two distinct pools with different rates of turnover [68,102,118]. These results suggest that aggrecan molecules in the pericellular and territorial matrix have a much faster rate of turnover that those further removed from the cell in the inter-territorial matrix. Autoradiography has demonstrated that PG turnover in the pericellular and territorial matrix occurs uniformly throughout the depth of the cartilage [109].

The major region of aggrecan core protein cleaved by proteinases is E1 between G1 and G2. Use of monoclonal antibodies to protein epitopes on aggrecan core protein generated by a variety of enzymes have implicated the MMPs (stromelysin and collagenase) in the turnover of aggrecan in the cartilage matrix [53,171]. However when aggrecan fragments released into the culture media or synovial fluid were examined it was evident that cleavage occurred at a site distinct from that attacked by the MMPs [96,101,159]. The identity of the proteinase(s) responsible for this E1 cleavage is not known at present. Furthermore it is unclear whether this enzyme, named "aggrecanase", is involved in constitutive turnover of aggrecan in normal joints or only assumes importance under pathological conditions[78].

In normal articular cartilage there is a balance between PG biosynthesis and catabolism such that a "steady-state" of matrix composition is maintained. The rate of turn-over of PGs, particularly aggrecan, within articular cartilage varies with both cartilage zone and age. Immature articular cartilage has a slower rate of PG turn-over than mature tissue which may be partly associated with a decrease in formation of link-stable aggregates in mature cartilage[21,136]. However the percentage of PGs released from the matrix that retain the ability to aggregate with HA (20-25%) does not vary with age[21]. Aydelotte et al.,[9] have shown that the half life of PGs is significantly longer for cultures of deep zone compared with superficial zone chondrocytes. Whether these age and zonal differences in PG turnover are related to variable synthesis of proteinases or the susceptibility of the PGs synthesised by the different cells is not clear. Superficial zone chondrocytes and chondrocytes from immature cartilage have been shown to be more sensitive to IL-1 than deeper cells and cells from mature animals respectively[10,120]. IL-1 increases the breakdown of PGs in articular cartilage by increasing the secretion of MMPs and decreasing TIMP synthesis[120].

The synthesis of PGs, as with their turnover, is modified by

age and cartilage zone and generally reflects the compositional differences observed in these cartilages. The synthesis of PGs by articular chondrocytes (as measured by 35SO42- incorporation) has generally been shown to decrease with increasing age [60,74]. The aggrecan monomers synthesised by chondrocytes from immature cartilage are larger than adults, the GAG chains are longer and the C6S/C4S ratio is lower[21,74].

Chondrocytes from the superficial zones of articular cartilage synthesise less PGs than deeper cells[6,9,60,74,85,86,163]. Maroudas and co-workers[60,163] have demonstrated that the incorporation of 35SO42- decreases slightly in the deepest compared with middle layers but is still higher than superficial cells. The size of the newly synthesised aggrecan monomers and their GAG chains were found not to vary with depth[6,9,86]. The size of endogenous aggrecan monomers decreases with increasing depth, suggesting that increased post-synthetic modification may occur in deeper layers[86].

The ability of the newly synthesised aggrecan monomers to aggregate with HA has been shown not to vary, or to increase slightly with depth from the surface[6,9]. This contrasts with the change in the percentage of endogenous PGs found in aggregates with depth from the surface[54,124]. This latter finding may be related to the lower synthesis of HA by deep zone chondrocytes[74]. Chondrocytes from the superficial cartilage zone have been shown to synthesise less KS in-vitro than deeper cells[6,203]. In contrast, Korver et al.,[86] found that the relative content of KS in newly synthesised aggrecan monomers did not vary with cartilage depth despite an increase in KS content of endogenous PGs with depth. These latter authors also found a decrease in the C6S/C4S ratio of newly synthesised GAG chains with depth, which was consistent with the differences in endogenous PGs.

The synthesis of small non-HA-aggregating PGs is increased in chondrocytes from the superficial zone compared to deeper cells[9,85,86]. These small PGs have subsequently been shown to be DS-PG I and DS-PG II187,188. This synthetic profile was consistent with the distribution of endogenous DS-PGs in adult articular cartilage. The synthesis of DS-PGs also varies with age, with DS-PG I synthesis being predominant in immature cartilage while DS-PG II synthesis predominates in adults[116,187,188].

Mechanical Loading and Proteoglycan Metabolism

Numerous in-vitro studies have demonstrated that articular chondrocytes respond to cyclic mechanical loading by increasing the synthesis of PGs as measured by incorporation of 35SO42- [81,97,132,156]. The nature and the magnitude of the applied mechanical force influences the response of chondrocytes. When loads are applied with low frequency or continuously and/or supraphysiological levels of dynamic loading are applied, PG synthesis is depressed [97,156]. Depressed PG synthesis with continuously applied loads has been associated with the development of reversible structural abnormalities in the golgi apparatus of chondrocytes [131]. The synthesis of LP and HA appear to be relatively unaffected by static compression [80].

The effects of mechanical loading on PG synthesis appear to result, at least in part, from changes in the ionic environment surrounding the chondrocytes [163,179]. The changes induced by mechanical compression can therefore be mimicked by hydrostatic pressure [95,131]. In these latter studies it was determined that there was no change in aggrecan monomer size, C6S/C4S ratio, GAG chain length or ability of the PGs to aggregate with HA induced by cyclic compression. However static compression increased aggrecan monomer size and GAG chain length.

The stimulation of PG synthesis in response to mechanical loading is more pronounced in the superficial rather than deep layers of articular cartilage [65,132]. A similar finding was reported by van Kampen et al.,[181] when cartilage was compressed with high loads (50 kg), however at lower loads (5 kg) the increase in PG synthesis was uniform throughout the depth of the cartilage. The selective increase in synthesis by superficial cells may explain the increase in synthesis of small PGs observed with mechanical loading [85]. Visser et al.,[187,188] demonstrated that mechanical loading of immature and mature bovine articular cartilage increased DS-PG II but not DS-PG I synthesis.

The results of these in-vitro studies are consistent with in-vivo evidence of the effect of mechanical stress distribution across a joint on PG synthesis. A lack of load-bearing results in depletion of PGs from the articular cartilage and the remaining aggrecan monomers have a reduced size and ability to aggregate with HA [15,34,82]. The C6S/C4S ratio of GAGs decreased with immobilisation [155]. The loss of PGs was most marked in the superficial zone [84]. The depletion in PGs was considered to be associated with a decrease in synthesis by chondrocytes [15,34]. Mechanical compression appeared to be the major determinant of chondrocyte PG synthesis, as joint motion without loading did not maintain cartilage PG content. The cartilage changes induced by reduced loading appear to be at least partially reversible with subsequent mobilisation and weight bearing [15,63,155]

Increased joint loading in-vivo generally increases cartilage PG content associated with an increase in PG synthesis [34,82,154]. In these studies it was found that there was an increase in C6S/C4S of GAGs in the cartilage [154]. However it was apparent in this same study that while the level of aggregating PGs did not change there was an increase in the non-aggregating PG in association with running exercise. There was no change in HA or LP content associated with running exercise [154]. The level of exercise has important implications on the adaptation of articular cartilage as strenuous exercise has been shown to result in decreased PG content especially in the superficial layers of articular cartilage [7,153].

Distinct regions or topographical locations within joints are exposed to different levels of mechanical stress. Given the response of cartilage to mechanical loading it is not surprising that topographical differences in cartilage composition and metabolism exist. In joint regions exposed to higher weight-bearing stress the PG content is increased in all zones of the uncalcified articular cartilage [25,60,82,163]. The aggrecan monomers from high weight-bearing cartilages are smaller than those from other regions, have a lower chondroitin sulphate content and a higher C6S/C4S ratio[62,154]. The ability of the aggrecan monomers to form aggregates with HA does not seem to vary with joint location [62]. The content of HA has been reported to be higher in cartilage from joint regions exposed to increased weight-bearing stress [105]. We have demonstrated previously that the synthesis of DS-PG II is increased in cartilge from high weight bearing areas of normal ovine (sheep) joints [99].

Osteoarthritis - Osteoarthritis (OA) is a disease of diarthrodial joints characterised by progressive destruction of articular cartilage. While degeneration of the cartilage is the hallmark of OA, it is variably accompanied by remodelling of the subchondral bone, marginal osteophyte formation, hypertrophy and mononuclear infiltration of the synovial membrane and changes in the synovial fluid. Abnormalities in any one of the joint tissues (cartilage, bone, synovium and synovial fluid) will affect the others. The following discussion will focus on the changes in articular cartilage composition and metabolism associated with OA and the pathogenesis of these changes.

Composition of Articular Cartilage in Osteoarthritis

The morphological appearance of OA cartilage depends on the degree of degeneration. Characteristic pathological features include disruption of the collagen fibrillar network and increased hydration, areas of chondrocyte replication (cloning) and regions of cell death, disruption of the cartilage surface with fibrillation and deep clefts, vascular invasion of the zone of calcified cartilage and tidemark and loss of PGs from the cartilage matrix [108,107] (See Figure 5).

Along with these morphological abnormalities in OA articular cartilage, there are marked changes in the biochemical composition of the matrix. Type X collagen which is normally restricted to the zone of calcified cartilage is synthesised by chondrocytes throughout the depth of OA cartilage [5,190]. Several authors have described the synthesis of type I collagen by human OA chondrocytes in-vitro [1,129] and type I collagen has been extracted from severe OA cartilage in rhesus monkeys[61]. However no synthesis of type I collagen was detected in cartilage derived from experimentally induced OA in dogs [50,76]. Furthermore using in situ hybridisation techniques Aigner et al.,[4] were unable to demonstrate type I collagen mRNA in human OA cartilage, although they did detect the presence of type III message in the superficial zones. McDevitt et al.,[115] have found an increase in cartilage type VI collagen in experimental OA. The concentration of collagen (total) does not change appreciably until the very late stages of OA [110,114]. However Hollander et al.,[73] found that type II collagen content was decreased and there was increased denaturation of the type II fibres especially in the superficial zone of OA cartilage. Furthermore the content of collagen types IX and XI decrease in OA cartilage [100].

Loss of PGs from the matrix is characteristic of OA cartilage and the degree of loss has been positively correlated with the severity of the degeneration [59,108]. The composition of the PGs remaining within the tissue has also been shown to change with OA. Inerot et al.,[77] found that there was an increase in the extraction yield of PGs, the KS/CS ratio was unchanged, the PG monomers were smaller, the CS chain length was unchanged and the ability of the aggrecan monomers to aggregate with HA was decreased in OA compared to age matched control human articular cartilage. Other workers have found a decrease in the CS chain length in OA cartilage[18] and a decrease in the KS content[29,106,176]. The C6S/C4S ratio on OA PGs has also been shown to decrease compared with normal [29,106]. Monoclonal antibodies to epitopes on the GAG chains of PGs have recently been used to demonstrate subtle changes in the chemistry of the GAGs (see reviews by Caterson et al., [35,36]). These changes appear to involve alterations in the sulphation and termination of GAG chains.

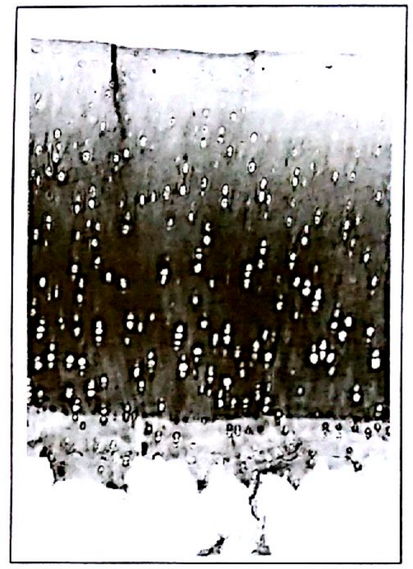
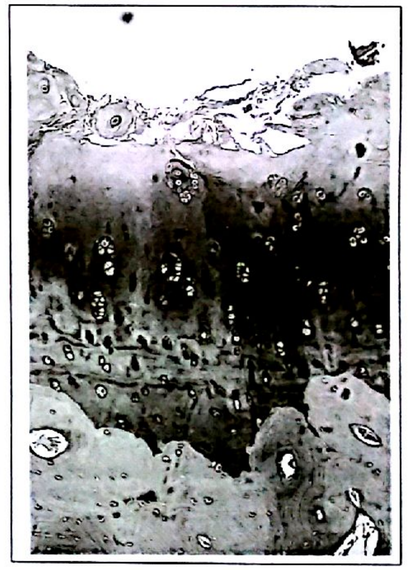


Figure 5.
Histological examination of articular cartilage and subchondral bone from the femoral condyle of (ABOVE) a normal and (BELOW) an osteoarthritic joint. Note the loss of matrix PG staining, surface fibrillation, chondrocyte cloning and multiple tidemarks in the osteoarthritic specimen.
(Toluidine blue/fast green stain. Original magnification x 10).



A decrease in the ability of aggrecan monomers to aggregate with HA in OA cartilage has been demonstrated by some authors[77,114,122] while others have found no change[25,148]. These disparate results may be explained by the species of animal examined and the stage of cartilage degeneration. Palmoski and Brandt[130] demonstrated no change in aggregation ability in extracted PGs from less diseased areas, while those from extensively degenerate areas showed decreased aggregation. A depletion of HA has been reported in - cont P.

24OA articular cartilage from both humans and experimentally induced disease in dogs [104,178]. Little difference in the content of LP or its ability to stabilise aggregation has been found between OA and normal articular cartilage [148,152]. Non-dissociative extraction of PGs from normal and OA cartilage has demonstrated that there is a decrease in the presence of aggregates in OA compared to normal cartilage in-vivo [124,125].

In extracts of OA cartilage there is an increase in the presence of small PG species which do not aggregate with HA [32,169]. These authors could not determine whether these PGs represented non-aggregating degradation products of aggrecan or a distinct synthetic product. More recently the presence of increased concentrations of DS-PGs in OA human cartilage has been reported [29,78]. The latter paper demonstrated that the content of GAG substituted DS-PG I and DS-PG II increased in OA but there was also an increase in the content of GAG-free core proteins. The GAG-free core proteins of both DS-PGs showed evidence of proteolytic cleavage. In previous studies of polyarthritis in humans and IL-1 treated rabbit knees, it was reported that the content of both DS-PGs was decreased and that core-protein fragmentation of DS-PG II but not DS-PG I had occurred[196,197]. Rostand et al.,[148] found no increase in DS GAGs in spontaneous OA in mice.

Metabolism of Articular Cartilage in Osteoarthritis

The changes in OA cartilage composition described above could occur through a combination of catabolic and anabolic processes. Increased mRNA levels and synthesis of type II collagen have been demonstrated in spontaneous human OA and a canine model of this disease [4,50,113]. Hollander et al.,[73] have demonstrated that along with the increased synthesis there is elevated degradation and turnover of type II collagen. The metabolism of the other collagens in OA articular cartilage has not been studied.

The synthesis and turnover of articular cartilage PGs in OA has been extensively studied. Some controversy exists as to whether PG synthesis is increased or decreased in OA. Numerous authors have demonstrated that the rate of PG synthesis by chondrocytes (as measured by total 35SO42- incorporation) was increased in animal models and human OA cartilage when compared to normal [18,108,109,158,163]. Other groups however, have found no difference in the rate of PG synthesis between OA and normal cartilage [25,31,57,58,121]. Direct comparison between the various studies is difficult because of the differences in species examined, model of disease used, stage of OA studied, the method of expressing the 35SO42- incorporation (relative to wet weight, dry weight, DNA, collagen, total GAG) and the culture conditions used for in-vitro studies. Three recent reports[92,93,183] have indicated that immediately after collection OA cartilage shows an increased rate of PG synthesis when compared to normal cartilage. Over the ensuing 3-4 days of culture the rate of PG synthesis in normal cartilage increases while that of OA tissue does not, such that the two cartilages eventually show no difference in synthetic rates.

The synthesis of small PGs, consistent in size with the DS-PGs was increased in a model of OA in dogs[33,183] and in human OA cartilage [59,170]. Identification of these newly synthesised small PGs as DS-PGs was not confirmed in these previous studies. We have identified an increase in synthesis of both DS-PG I and DS-PG II in articular cartilage from a model of OA in sheep99. Adams et al.,[3] have shown that the relative mRNA level for DS-PG I is significantly increased (4x) in a model of OA in dogs. These workers demonstrated an increase in DS-PG II mRNA (1.6x) but this increase was not significant. A correlation between these mRNA levels and protein synthesis was not evaluated but is consistent with the protein synthesis in our sheep studies.

Despite the contradictory results, it is generally accepted that in the early stages of OA, chondrocytes are hypermetabolic showing an overall increase in the rate of PG synthesis. This stage of OA has been termed the hypertrophic phase [2,22]. This reparative response declines however and eventually the rate of PG synthesis falls below that in normal cartilage. The reason for this "decompensation" is unclear but may be related to altered mechanical forces on the cartilage, or the synthesis of matrix components that are unable to withstand the normal weight bearing demands upon the tissue.

In contrast to PG synthesis, it has consistently been demonstrated that the rate of turnover of PGs in OA cartilage in-vitro is increased compared to normal [32,33,142]. These in-vitro results are consistent with data showing increased levels of matrix PG components in synovial fluids of OA joints[102,142,160,161]. Release of a number of the components of the aggrecan complex including LP, G1 and CS and KS bearing core proteins has been demonstrated[142,161]. The PGs released from the cartilage both in-vitro and in-vivo show evidence of proteolytic cleavage of a varying extent depending on the stage of OA destruction[161]. These authors have shown that in early OA there was release of LP and KS bearing peptides while in latter disease the G1 domain of aggrecan was also released.

It is possible that some release of matrix components is the result of synthesis of proteins that are not able to integrate appropriately within the tissue. However it seems more likely that an increased concentration and activity of a number of proteolytic enzymes is responsible for the cleavage and release of these tissue components. Increased concentrations of MMPs have been demonstrated in OA cartilage[44,134]. An imbalance between the levels of MMPs and their inhibitor TIMP have been reported in OA articular cartilage and synovial fluid[45,46,102,112]. Elevation and/or demonstration of the possible involvement in OA cartilage degeneration has been reported for cathepsin B[12,13,30] and plasmin and plasminogen activators[111]. As described earlier, the enzyme "aggrecanase" appears to be intimately involved in aggrecan turnover in OA but at present its identity is unknown. Evidence for the involvement of this enzyme comes from data identifying release from the cartilage matrix of aggrecan core protein cleaved in the E1 between amino acids 373 and 374 (human sequence enumeration) both in-vivo and in-vitro [75,96,101,159].

Pathogenesis of Osteoarthritis

Degeneration of articular cartilage in OA may occur through a combination of abnormal synthesis and catabolism of the various matrix components (See Figure 6). The factors which initiate, control and sustain the abnormal metabolism of articular chondrocytes in OA are still the subject of intense investigation. Indeed Lohmander[100] has stated that "OA is not a single disease entity but a common end-stage of cartilage and joint failure". Inherent within this statement is the idea that OA has a multifactorial pathogenesis with many risk

factors including age, sex, race and species and genetic susceptibility[56,100,194].

The use of animal models of OA has demonstrated that mechanical and humoral factors play a major role in the pathogenesis of this disease process[57,58,121,122,141,184]. A schematic representation of the postulated interaction between the various modulating factors involved in the pathogenesis of articular cartilage degradation in OA is shown in figure [6].

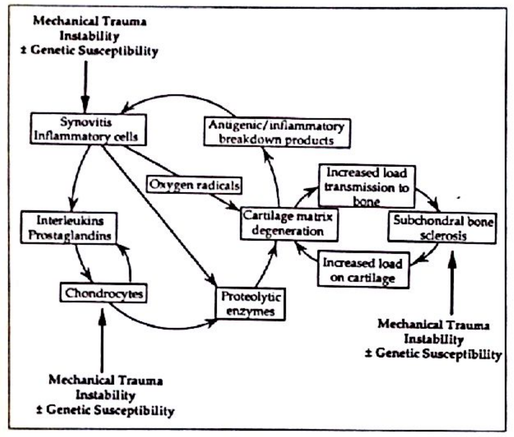


Figure 6.
Proposed pathogenic cycle responsible for cartilage degeneration in osteoarthritis.

Cytokines have a major influence on the regulation of matrix and proteinase synthesis by chondrocytes and it is believed that the synovium is the major source of these factors [24,133,144]. Increased levels of one or more of these cytokines (particularly IL-1 and TNF-a) may lead to decreased matrix synthesis and increased catabolism by the chondrocytes. The often catabolic influence of cytokines may be balanced by the anabolic effects of growth factors. However excessive levels of growth factors such as TGF-b may lead to the formation of osteophytes[179,180] and as such may be involved in the pathogenesis of OA.

Mechanical trauma, in the form of a single high impact or repetitive low impact load(s), has been demonstrated by numerous authors to lead to cartilage degeneration consistent with OA (reviewed by Radin et al.,[141]). These mechanical forces may result directly in disruption of the articular cartilage or they may cause damage to the subchondral bone. Altered stiffness of the subchondral bone decreases its compliance and increases the weight bearing stress absorbed by the articular cartilage and results in its subsequent failure. The "biochemical integrity" of the articular cartilage is vital for its resilience to compressive forces. Depletion of the matrix PG concentration or reduced cross-linking of the collagen fibres will result in disruption of the cartilage even under normal mechanical loads[168].

Degradation of the matrix components of articular cartilage and their release into the joint cavity and systemic circulation may induce an inflammatory response[20,43]. Furthermore the cartilage breakdown products are antigenic and can induce an immune reaction[35,42,87,138]. The cartilage breakdown products may therefore result in secretion of cytokines by synovial cells and chondrocytes themselves, thus perpetuating the degenerative process.

There is evidence that the chondrocytes from OA cartilage have an altered phenotype both in their response to mechanical stimuli and growth factors. Lafeber et al.,[91] demonstrated that OA cartilage was more responsive to cyclic compressive loading in-vitro than normal cartilage. Evidence for altered response to growth factors by OA chondrocytes was seen in their failure to increase PG synthesis when cultured in-vitro [92,93,183]. It has been demonstrated that OA chondrocytes are less responsive to growth factors such as IGF-1, EGF, FGF and PDGF than normal. Lafeber et al.,[94] have shown that chondrocytes derived from OA articular cartilage are more responsive to TGF-b than cells from normal tissue. It is unclear how this altered phenotype of OA chondrocytes is induced and whether it plays a role in the pathogenesis of disease.

OA is known to be a focal disease with the most severe cartilage degeneration observed in joint regions exposed to the highest mechanical loads in-vivo [108,174]. The variation in chondrocyte and matrix morphology and metabolism in topographically defined joint regions have been discussed in previous sections. It has not been determined whether these topographical differences are the result of variable mechanical loading or are inherent. This topic has important implications with regard to the susceptibility of different joint regions to OA degeneration. van Osch et al.,[182] have demonstrated that induction of joint disease in mice resulted in cartilage degeneration in different sites within the joint dependent on the inducing agent (intra-articular injection of iodoacetate or collagenase). These results support the view that chondrocytes from different joint regions may be variably susceptible to degeneration.

Conclusion

The preceeding discussion has highlighted the specialised biochemical composition of articular cartilage which enables it to perform its demanding mechanical role within the joint. It is apparent that the chondrocytes within the articular cartilage undergo metabolic changes with aging and mechanical loading. These cells produce a matrix which is able to best withstand the biomechanical stresses to which they are exposed, although this ability to respond may be compromised with aging and in some instances by genetic influences. Despite this remarkable adaptive capacity of the chondrocyte, mechanical trauma, either as a single supraphysiological event or with lower levels of repetitive stress, may lead to phenotypic changes which result in the production of an abnormal extracellular matrix. These events herald the onset of cartilage degeneration and osteoarthritis. The challenge before us is to identify those changes in chondrocyte metabolism and subsequent cartilage biochemistry, that preceed the deterioration of the biomechanical function of the tissue. Early identification of these "pre-osteoarthritic" abnormalities may enable strategies to be devised which would support chondrocyte metabolism and preserve cartilage integrity.

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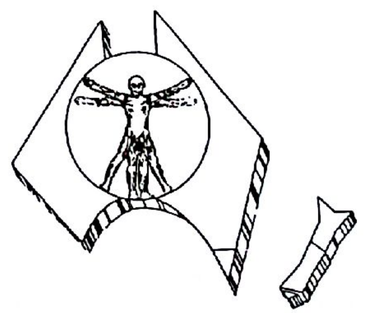

THORACIC SPINAL PAIN - ZYGAPOPHYSIAL JOINTS

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Introduction

Several factors probably account for the contemporary interest and emphasis on thoracic zygapophysial joints as the leading possible source of thoracic spinal pain. Foremost, this interest follows on the trail of considerable interest in the zygapophysial joints as sources of low back pain[10, 11, 13, 14] and neck pain.[1, 2, 6, 8] It seems only natural that investigators would adopt analogous hypotheses when confronted with the problem of thoracic spinal pain.

A second reason is technical. Most of the other potential sources of thoracic spinal pain are relatively inaccessible. The thoracic intervertebral discs lie deep in the thorax and are relatively inaccessible with needles for the practice of discography; the close proximity of the lungs and pleura poses the hazard of pneumothorax, which until recently has deterred discographers from pursuing thoracic discogenic pain.[12] Similarly, the costovertebral joints lie deep in the thorax, covered by lungs and pleura.

In contrast, the thoracic zygapophysial joints lie outside the chest cavity and are accessible without serious risk of pleural puncture. For this reason they are at least a convenient starting point in the exploration of thoracic spinal pain.

There is now sufficient data to implicate the thoracic zygapophysial joints as possible sources of thoracic spinal pain. Dreyfuss *et al.*[7] have shown that these joints can be rendered painful in normal volunteers, and Wilson[17] has described patients whose thoracic pain was relieved by anaesthetising these joints. These clinical data vindicate the existence of thoracic zygapophysial joint pain - an entity referred to in some clinical circles as "thoracic facet syndrome". Other investigators have embraced this notion and have ventured to treat thoracic facet syndrome.

Stolker *et al.*[15] reported their experience in diagnosing and treating thoracic zygapophysial joint pain. To diagnose the condition, they reportedly anaesthetised the nerve supply to these joints. To treat the pain, they performed percutaneous radiofrequency facet denervation - a procedure ostensibly designed to coagulate the nerves that innervate the target joint and thereby denervate it.

Paradoxically, these procedures were undertaken without the benefit of any antecedent studies of the anatomy of the target nerves to be blocked and coagulated. In the description of their technique, Stolker *et al.*[15] stated that they used a target point analogous to that described by Bogduk and Long[4] for lumbar medial branch blocks and for lumbar medial branch neurotomy, i.e., the junction between the superior articular process and the transverse process. The implication was that Stolker *et al.*[15] either expected or assumed that the anatomy of thoracic dorsal rami would be the same as that of the lumbar dorsal rami, and that this assumption was enough to justify their diagnostic and therapeutic techniques.

Existing textbook descriptions of the thoracic dorsal rami are limited in detail. Textbooks such as Gray's Anatomy[16] focus on the distribution of the lateral branches but provide no details on the course or articular distribution of the medial branches. Hovelacque[9] offers a more detailed description of the thoracic dorsal rami but again, explicit details as to radiological landmarks for the medial branch or its distribution of the zygapophysial joints are not provided. Thus, to prevent these surgical techniques from falling into disrepute because of an erroneous anatomical basis, a study was undertaken to determine the location and course of the medial branches of the thoracic dorsal rami which by analogy to the cervical and lumbar spine are the legitimate targets for thoracic medial branch blocks and thoracic medial branch neurotomy. In addition, the origin and course of the articular branches to the thoracic zygapophysial joints were explored.

Methods

Using a X40 dissecting microscope, microdissection was carried out on 96 thoracic medial branches from 8 sides of 4 embalmed human adult cadavers.

During the later stages of the dissection, a careful search was made for articular branches, in the region ventrolateral to each thoracic zygapophysial joint between the joint and the dorsal ramus, in the region caudal to each zygapophysial joint, and in the region above each joint.

Results

The course of the medial branches of the thoracic dorsal rami was essentially similar at all segmental levels, although certain topographic differences occurred at specific levels. Typically, the medial branch arose from the dorsal ramus within 5mm of the lateral margin of the intervertebral foramen. From its origin, the medial branch first passed dorsally, inferiorly but largely laterally within the intertransverse space (Fig. 1)Along this course, the nerve was embedded in areolar tissue and accompanied by arteries and veins. Opposite the tip of the transverse process, the medial branch curved dorsally through the intertransverse space, aiming for the superolateral corner of the subjacent transverse process. It then entered the posterior compartment of the back by crossing this corner and then running caudally along the posterior surface of the tip of the transverse process, lying in the cleavage plane between the origin of multifidus medially and that of semispinalis laterally (Figs. 2 and 3).

The archetypical course of the medial branches was consistently exhibited by the nerves at the upper thoracic levels (T1-T4) and lower levels (T9-T10). The course of the T11 and T12 medial branches differed because of the different osseous anatomy. The T12 transverse process was much shorter than typical transverse processes. Consequently, the T11 medial branch ran across the lateral surface of the root of the superior articular process of T12.

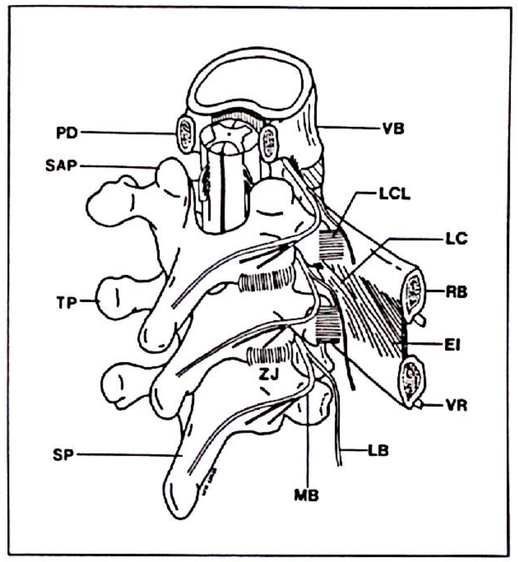

Figure 1
A sketch of the archetypical course and relations of the thoracic dorsal rami viewed from a right superior aspect. SP spinous process TP transverse process, SAP superior articular process, PD pedicle, ZJ zygapophysial joint, MB medial branch, LB lateral branch, VR ventral ramus, EI external intercostal muscle, RB rib, LC levator costae muscle, LCL lateral costotransverse ligament, VB vertebral body

The T12 medial branch assumed a course analogous to that of the lumbar medial branches, crossing the junction of the superior articular process and the base of the transverse process (Figs. 2 and 3).

At mid-thoracic levels (T5-T8), the medial branch did not always assume contact with the transverse process. The nerve sometimes appeared to be suspended in the intertransverse space as it passed dorsally. It assumed a course parallel to those at typical levels by was displaced somewhat superiorly. Instead of crossing the superolateral corner of the transverse process, the nerve entered the posterior compartment of the back by passing dorsally through the middle of the intertransverse space and wrapping medially around the fascicles of multifidus above the level of the transverse process (Figs. 2 and 3). It then curved medially but remain separated from the transverse process by the fascicles of the multifidus.

Two types of articular branches arose from the medial branches. Ascending branches arose from the medial branch as it passed caudal to the zygapophysial joint. These branches were short and they ramified in the inferior aspect of the zygapophysial joint capsule. A slender descending articular branch arose from the medial branch as it crossed the superolateral corner of the transverse process. It followed a sinuous course between the fascicles of multifidus to reach the superior aspect of the capsule of the zygapophysial joint below.

Discussion

The observation of the study are concordant with the descriptions of Hovelacque[9] but amplify the detail sufficient for clinical purposes. The key feature of the thoracic medial branches is that, by and large, they cross the superolateral corner of the subjacent transverse process. However, at mid-thoracic levels, the nerve may be displaced cephalad and therefore does not assume this relationship, while at lower thoracic levels the nerve assumes an osseous relationship ananlogous to that of the medial branches of the lumbar dorsal rami.

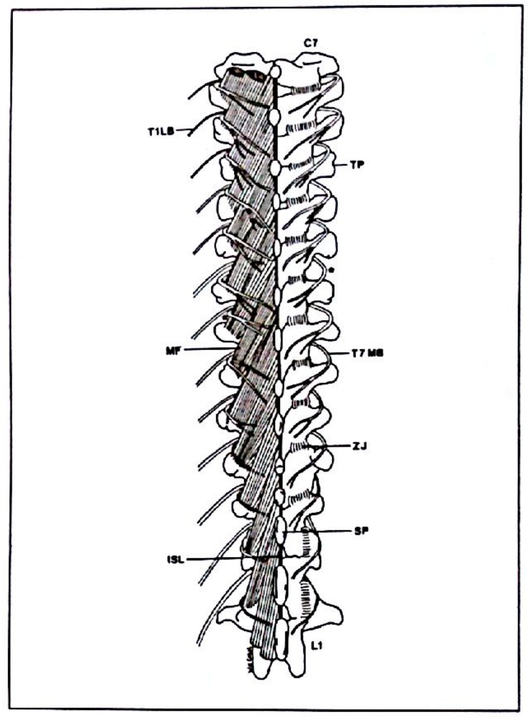

Figure 2
*A sketch of the medial branches of the thoracic dorsal rami viewed from behind. On the right side, the multifidus and lateral branches are not shown. TP transverse process, MB medial branch, ZJ zygapophysial joint, SP spinous process, LB lateral branch, MF multifidus muscle, ISL interspinous ligament, C cervical vertebra, L lumbar vertebra, * atypical medial branch*

At no time in the present study were nerves encountered crossing the superomedial corner of the transverse process which is the target point for thoracic medial branch neurotomy advocated by Stolker *et al.*[15] This means that the target points do not coincide with the course of the medial branch. Therefore, the procedure that they describe cannot consitute a thoracic medial branch neurotomy. When in contact with the transverse process, the medial branch lies at least 12mm lateral to the root of the transverse process. However, the maximum size of the lesions created by the thermocouple electrodes used in medial branch neurotomy measures only 1.1mm in radius.[5]

Stolker *et al.*[15] adopted the target point at the superomedial corner of the transverse process because they perceived this to be analogous with the site at which the medial branch of the lumbar dorsal ramus crosses bone. What they neglected was the homology between the lumbar and thoracic vertebrae. The so-called transverse process of the lumbar vertebra is formed largely by the costal element of the lumbar vertebra. The true transverse proces is represented by the mamillary process, the lateral surface of the superior articular process and the transverse process but only as far as the accessory process. That being the case, the lumbar medial

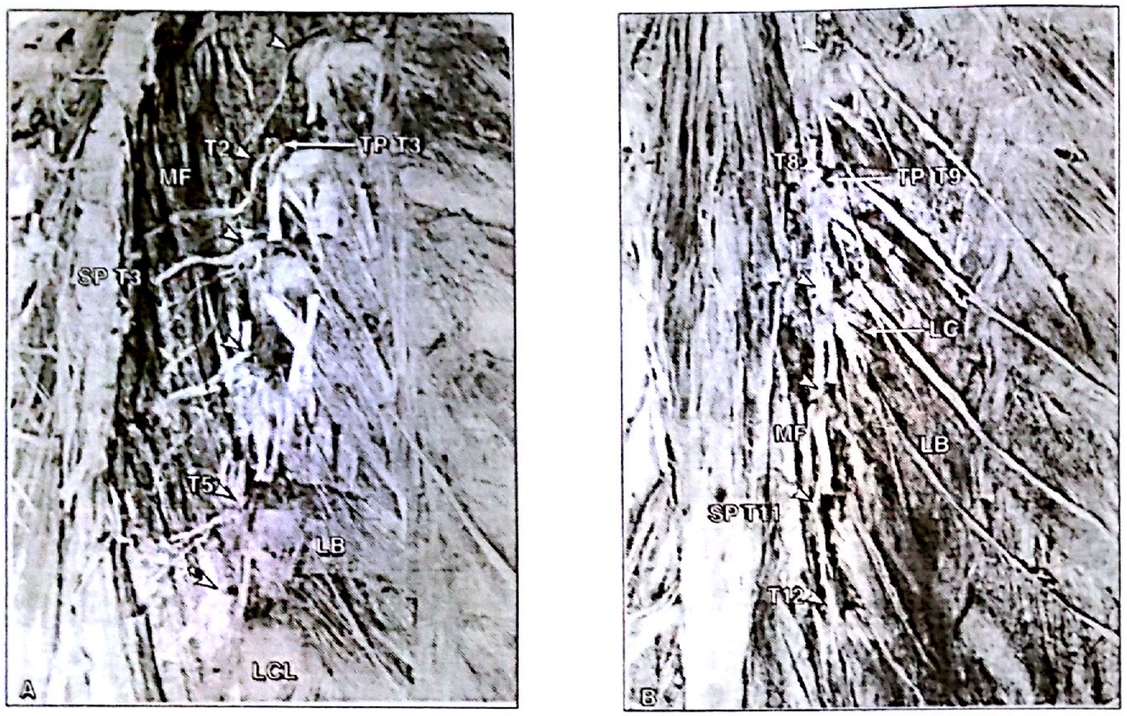

Figure 3 - A & B

*Photographs of a dissection of the branches of the right thoracic dorsal rami viewed from behind. (LEFT) Upper 6 thoracic levels; (RIGHT) lower 6 thoracic levels. The medial branches are indicated by black arrowheads. LB lateral branch, SP spinous process, TP transverse process, MF multifidus muscle, LCL lateral costotransverse ligament, LC levator costae muscle, * atypical medial branch.*

branches can be perceived as crossing the transverse element diagonally from its superolateral corner to its inferomedial corner. Thus, when addressing the thoracic medial branches, the course would be expected to run from the superolateral to the inferomedial corner of the transverse element which is the transverse process proper on the thoracic vertebra. This is where the medial branches were encountered in the present study. This homology is further endorsed by the myotendinous relations of the medial branch. At thoracic levels, the medial branch lies lateral to the multifidus but deep to the tendons of the semispinalis. In the lumbar spine, the nerve runs deep to the mamillo-accessory ligament which is homologous to the semispinalis muscle in the thoracic spine.[3]

Conclusion

If thoracic medial branch blocks and thoracic medial branch neurotomies are to be accepted as legitimate option for the management of thoracic zygapophysial joint pain, further validation of data is required. Either the technique of Stolker *et al.*[15] must be verified clinically in the form of double-blind controlled trial or the procedure needs to be modified so as to be concordant with the surgical anatomy of the thoracic medial branches.

This is the second paper on thoracic spinal pain. The above study was part of the author's Bachelor of Medical Science research project which was conducted at the University of Newcastle under the supervision of Professor Nikolai Bogduk.

Much of the research funding was kindly provided by the Australian Association of Musculoskeletal Medicine.

Consequent to the findings of this study, further studies were undertaken to establish the course of the thoracic medial branch in relation to the bony landmarks commonly observed on radiographs and also to assess the suitability of the superolateral corners of the transverse process as target points for thoracic medial branch blocks and neurotomy.

The above paper has been published in an international peer-reviewed journal and was presented at two overseas conferences.

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THE BIOMECHANICS AND TREATMENT OF THE MALALIGNMENT SYNDROME

Dr. Wolf Schamberger

CURRICULUM VITAE

Dr. Schamberger received his M.D. from the University of British Columbia, Vancouver, Canada, in 1970. After interning in Montreal, he spent 4 years in family practice in Duncan, B.C. He had always had interest in long distance running and throughout the 1970is was one of Canadaís top ten marathon runners. Dealing both with personal injuries and those of athletes from various sports sparked a keen interest in sports medicine.

At that time, a residency at U.B.C. in Physical Medicine and Rehabilitation seemed to come the closest to covering the neuromusculoskeltal problems one would encounter in sports. After a post-fellowship in sports medicine, cardiac rehabilitation, and electromyography in Seattle in 1980, he has continued to subspecialize in these areas. Since 1987 he has been especially interested in the effect of malalignment of the pelvis and spine in terms of altering the Biomechanics of the axial and appendicular skeleton and as a cause of local and referred pain submitted for publication.

His commitments as a clinical associate professor in the Division of PMR at UBC, consultant at the Allen McGavin Sports Medicine Clinic, and with a number of sports medicine and cardiac rehabilitation associations still allow time for running the dogs, hiking, climbing and kayaking in the never ending search for new injuries that, in the end, have always been the best teacher for learning to discover the effect of altered Biomechanics on the neuromusculoskeltal system.

ABSTRACT

'Malalignment syndrome' consists of a complex of muskuloskeletal symptoms and signs seen with anterior/posterior innominate rotation or upslip of the SI joint, with distortion of the pelvic ring malalignment of the axial and appendicular skeleton, and compensatory changes in attaching soft tissue structures. Diagnosis rests on finding a combination of typical symptoms, sites of tenderness and/or referred pain, and asymmetries of alignment, ranges of motion, muscle tension, bulk and strength, leg length, weightbearing. Clinical exam and investigations must rule out conditions that can present with overlapping findings and/or cause recurrence of malalignment. Realignment is best achieved with manual therapy combined with patient participation using self-assessment and self-treatment techniques, supplemented by use of orthotics, sacroiliac belts and prolotherapy/cortisone injections. Unrecognized, the syndrome represents a major cause of musculoskeletal symptoms, injury and impaired recovery from injury, as well as of error in research in this area.

Medicine to date has failed to recognize malalignment as one of the major causes of muskuloskeletal symptoms, injury, and impaired recovery from injury. Also, much of our research dealing with matters relating to weightbearing, ground reaction forces and muscle strength has failed to take into account the biomechanical effects of malalignment. Side-to-side differences in lower extremity ranges of motion or muscle strength, for example, lack meaning when we do not know whether the subjects enrolled in a particular study were in alignment or not.

Traditionally, malalignment has been thought of in terms of involving the pelvis and the spine. Two presentations of pelvic malalignment are particularly prevalent:

1. Rotational malalignment

   —resulting from excessive anterior or posterior rotation of an innominate relative to the sacrum; 'anterior' and 'posterior' refer to the direction of movement of the upper part of the innominate (e.g. Iliac crest, ASIS, PSIS) in the sagittal plane

2. An upslip of the sacroiliac joint

   — referring to a direct upward translation of the right or left innominate relative to the sacrum.

These two presentations of malalignment are now well recognized as a cause of back pain. Both result in:

1. Distortion of the ring of pelvic bones and the joints that are part of that ring: the symphysis pubis and the two SI joints
2. Pelvic obliquity and compensatory curvatures of the spine. In addition, there may be excessive rotation of one or more vertebrae. This can either result from the malalignment or can cause the malalignment to occur in the first place.

Malalignment places some of the bony elements and attaching soft tissue structures under increased stress. This can eventually result in pain around the pelvis and along the spine, with or without referral to other sites (e.g. lower extremities). However, malalignment of the pelvis and spine does not occur in isolation. The presentations described above are but part of a larger picture designated as the 'MALALIGNMENT SYNDROME', a clinical entity characterized by:

1. Distortion of the pelvic ring
2. Appendicular skeleton postural changes
3. Compensatory changes in the soft tissue structures, and
4. Sometimes visceral involvement, affecting the genito-urinary, gastro-intestinal and reproductive systems.

The DIAGNOSIS OF MALALIGNMENT rests on the findings of:

1. Asymmetrical alignment of the bones of the pelvis, trunk and extremities
2. Compensatory curvatures of the spine, with or without malrotation (excessive rotation) of one or more vertebrae
3. Asymmetrical ranges of motion of the head and neck, trunk and pelvis and the joints in the upper and lower extremities
4. Asymmetrical tension in muscle, tendons and ligaments
5. Asymmetrical muscle bulk and strength
6. An asymmetrical weightbearing pattern and
7. Pain localizing to the joints and soft tissues put under stress by these asymmetries and/or typical referred pain patterns originating from these structures, including visceral symptoms.

The biomechanical changes affecting the lower extremities literally turn the subject into a 'split personality' from the waist down. For example, one of the common findings associated with the asymmetry of weightbearing is that the athlete now pronates on one side and supinates on the other. In addition, hip, ankle and foot ranges of motion are typically asymmetrical and there is usually an asymmetry of lower extremity muscle strength, tone and bulk. Some of these asymmetries are predictable from the pattern of malalignment present. Also predictable are the restrictions that these asymmetries impose on activities and the most likely injuries that can occur as a result. Two main patterns of malalignment have emerged with typical associated findings:

1. Anterior rotation of the left innominate combined with dysfunction of movement of the left sacroiliac joint
   — external rotation of left, internal of right lower extremity; weightbearing shifts to right; wasting of left vastus medialis
2. All other presentations of anterior rotation, also right upslip
   — external rotation of right, internal of left lower extremity; weightbearing shifts to left; wasting of right vastus medialis

INVESTIGATIONS are of value to rule out:

1. Other conditions that can present with similar symptoms (e.g. nerve root compensation, sciatica, sacroiliitis)
2. Underlying conditions that can predispose to recurrence of malalignment following correction (e.g. disc protrusions, ovarian cysts, uterine fibroids).

TREATMENT consists primarily of correction of the malalignment using manipulation or mobilization techniques. Chances for recovery are improved by teaching the subject:

1. Self - assessment techniques, so he or she can determine whether malalignment is present or not, and, if so, what form
2. Self - treatment techniques, mainly muscle energy techniques which are successful in most for achieving and maintaining alignment in-between sessions with their therapist (who can be considered as doing the 'fine - tuning').

Pelvic stability may be increased by the addition of foot orthotics and a sacroiliac belt. If malalignment continues to recur and/or hypermobility of one of the SI joints or instability of a vertebral segment is evident, one can consider prolotherapy or injection of a chemical irritant (e.g. concentrated glucose) to induce fibroblasts to produce new collagen. The effect is to strengthen the ligaments. Cortisone injections may be helpful if ligament pain fails to settle even though realignment is being maintained. As long as malalignment keeps recurring, the emphasis is on symmetrical exercise unless the therapist specifically recommends some asymmetrical stretching or strengthening routines. Results with this approach have been excellent in patients who have often failed to respond to traditional therapeutic modalities and even regular manipulation or mobilization.

Recognition of the malalignment syndrome will hopefully lead to a greater awareness of the various kinetic chains and their interactions. The days of looking at an injury in isolation are over. For example, an athlete presenting with left lateral knee pain may well have pain localizing to the distal iliotibial band. Treating that area with standard physiotherapy, anti - inflammatory medication, ice and rest may get the athlete back on the road. However, if one ignores the fact that athlete is a supinator and that the right anterior innominate rotation has shifted weightbearing even more to the outside on the left, then the athlete is set up for a recurrence of the same injury. In fact inattention to these factors may prolong recovery from the initial injury or may result in failure to recover at all: the constant increase in tension exerted on the inflamed tissue as long as malalignment is present may interfere with the healing process.

Seminars, workshops and publications relating to malalignment are timely as this remains a poorly understood cause of dysfunction. In addition, there is not so much a lack of information as a failure to seek or exchange information. In Canada, there is also the unfavourable association of ìmalalignmentî with 'chiropractic'. The picture regarding malalignment is akin to the way that acupuncture was received in North America back in the 1960's. Hopefully it will find speedier acceptance.

CHRONIC GROIN PAIN - INCIPIENT INGUINAL HERNIA

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Chronic groin pain can be a difficult diagnostic dilemma for the physician. Review of 189 patients in my practice suggested that multiple pathology existed in 27% of cases. Setting specific clinical and investigational diagnostic criteria will aid in diagnosis and treatment of these patients. The site of pain on history is a good indicator for suggesting possible diagnoses.

Groin pain can be divided into areas with likely diagnoses such as iliac fossa, inguinal, pubic, anterior thigh or hip and upper inner thigh. Pain in the iliac fossa, tenderness over the ilio-inguinal nerve and improvement of pain after injection of local anaesthetic indicate ilio-inguinal nerve entrapment. Inguinal pain, cough impulse or posterior wall laxity, and positive herniography or ultra-sonography would suggest an incipient inguinal hernia or an inguinal hernia. Inguinal pain may also be referred from the hip or spine.

Central or pubic pain suggests osteitis pubis or rectus abdominis tendopathy. Local injection of anaesthetic may help differentiate a muscular lesion, or a technetium bone scan may be required to diagnose osteitis pubis or a pubic stress fracture.

Iliopsoas bursitis and tendinitis will usually present with upper anterior thigh pain and can be confirmed on clinical examination or by ultra-sonography. The hip joint needs to be carefully examined, particularly looking for pain at the end of internal range of motion. Injection of anaesthetic into the joint is sometimes useful as a diagnostic procedure.

Pain along the upper inner thigh with local tenderness and pain with resisted adduction would indicate an adductor tendon lesion. Recent anecdotal reports have suggested that chronic adductor pain not responding to treatment may be due to entrapment of the obturator nerve. Other causes of pain must also be considered such as uro-genital, rheumatological (eg spondylo-arthropathies), tumours and spinal/sacro-iliac referred pain.

Surgery to the inguinal canal as a treatment for chronic groin pain has been studied in recent years. Inguinal hernia was first described as a cause of groin pain in athletes by Smedberg in 1985, although Smodlaka had noted in 1980 anecdotal reports of athletes with undiagnosed chronic groin pain returning to sport after inguinal hernia repair. Smedberg et al investigated their group of athletes with herniography - intra-peritoneal injection of contrast medium with subsequent plain x-ray of the pelvis/abdomen. He found that 55% of the 78 athletes had inguinal hernia on herniography (usually undetected on clinical examination) and they found a 70% improvement (return to sport) after surgical repair to the inguinal floor. Gullmo has described herniographic findings of incipient inguinal hernia (inguinal wall weakness) in athletes, a finding not usually noted in a young population.

A tear of the conjoint tendon was then suggested as a possible cause of incipient inguinal hernia and groin pain in athletes, however biopsy of the tendon by myself and co-workers in 15 athletes revealed no tear, injury or chronic inflammation. Two Australian studies then reported finding incipient inguinal hernia in athletes with subsequent return to sport following repair of the posterior inguinal wall. In 1991 Polglase et al reported on 68 athletes with debilitating chronic groin pain and found 85% had a deranged posterior inguinal wall at surgery. Following repair 62.5% were ëcuredí and returned to competitive sport while 31% were improved and able to return to sport. In 1992 Peter Malycha and I reported on surgical exploration and inguinal wall repair of 50 athletes with chronic groin pain suggestive of incipient inguinal hernia. At operation forty athletes had a significant bulge of the posterior inguinal wall. Forty four athletes replied to a follow up questionnaire after 6 months with 41 returning to normal sporting activities - 33 (75%) rated the result as good and 10 (23%) as improved. Pain scores showed a significant improvement in groin pain following surgery. Hackney and Gilmore have noted similar findings in the United Kingdom.

The athlete with this injury will describe a dull aching pain in the inguinal area occasionally radiating to the upper inner thigh. Pain is often increased with coughing or sneezing, kicking and sprinting. The onset can usually be related to a game or training session but not to a specific incident. Pain is increased with activity and often continues the next day. Performance gradually deteriorates over a few weeks until the athlete is unable to train fully or play. Careful clinical examination is required to review other possible causes of groin pain; in this group the most common other causes of groin pain are adductor tendon lesions, osteitis pubis and pain referred from the lumbar spine.

Investigation by plain x-ray, technetium bone scan, ultra-sonography and herniography may be required to determine the cause of an athleteís groin pain. Herniography is an invasive procedure and should only be performed if there is doubt over the clinical diagnosis and surgery is still being considered. Ultra-sonography of the inguinal wall is now being performed at some centres and initial results suggest it may replace herniography as a non-invasive investigation of inguinal wall weakness and hernia.

As athletes do not improve with rest, the treatment of incipient inguinal hernia is surgical repair of the weakened posterior inguinal wall. Following surgery athletes can return to light activity after 3 weeks, running at 4-5 weeks, training at 6-8 weeks and competitive sport at 10-12 weeks. Over 90% of athletes are able to return to sport.

Chronic groin pain can be difficult to diagnose and treat. Careful attention to history, site of pain, clinical examination and investigation will lead to a diagnosis and appropriate treatment will then enable an athlete to return to sport.

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A SIMPLE ASSESSMENT OF THREE MUSCULOSKELETAL PALPATORY TESTS

Paul Quin and Mark Johnston

Abstract

Objective - To assess the sensitivity and specificity of three commonly used musculoskeletal tests, viz., comparative assessment of the height of the iliac crests, the springing test at the level D9, and the assessment of motion in the subtalar joint.

Design - On site study utilising attenders of a musculoskeletal conference.

Setting - Warwick Hotel Conference Centre, Korolevu, Fiji

Subjects - 3 'patients' with abnormal findings and 3 controls matched for age and sex were examined by 19 doctors.

Main outcome measurement - sensitivity and specificity of the above mentioned tests.

Introduction

It has been recognized by medical practitioners within the musculoskeletal field that its adherents have had diverse training, often from one or other of the many of the recognised doyens of Musculoskeletal Medicine scattered throughout the world, more especially from continental Europe but also from Britain, USA and more recently Australia and New Zealand. This exposure to a multiplicity of teachers has had the effect of creating disciplines so that a practitioner would tend to align himself to a particular philosophy and methodology in his/her musculoskeletal practice. As a result there has tended to be a lack of standardization in many aspects of examination, assessment and treatment of patients. This further confounded by practitioners declaring their use of specifically named tests and their results of those tests as though they were standardized. Accordingly it was decided to attempt a simple protocol using the 'two-by-two' test to assess validity of three tests done routinely in musculoskeletal practice.

Method

The three tests to be assessed were
(a) a comparison of the heights of the iliac crests,
(b) the springing test of mobility of D9 dorsal vertebra, and
(c) testing the subtalar joint for restricted range of motion.

At a musculoskeletal conference in September 1996 held at Warwick Hotel, Korolevu, Fiji, three of the attenders with known positive clinical signs were matched for age and sex with three controls and an attempt to set up a 'gold standard'was undertaken. The 'gold standard' for iliac crest height assessment was consensus agreement of the finding by five musculoskeletal teachers from New Zealand, Australia and Canada. For the springing test of mobility of the thoracic segment, there was a thoracic spine x-ray showing anterior and lateral bridging osteophytes of the thoracic spine from D7/8, D8/9, D9/10, D10/11. For the subtalar joint range of movement the previously mentioned teachers (with the exception of the Canadian teacher who did not examine the 'patient') confirmed the sign.

Prior to entering the programme a short tutorial on the springing test was offered to those who felt they were not familiar with the technique and a small group took advantage of this. The 'patients' with the target disorders were paired with their controls at one end of the conference room and the examining doctors were escorted by a recorder to each station in turn. At each station the appropriate question was read out and the answer recorded on a standard form after the 'patient' had been examined. Each question required a yes/no answer. On entering the test each candidate was asked if he/she was experienced - i.e. if they had had some practical experience in Musculoskeletal Medicine.

The clinical questions were as follows:-

Q1. Are the iliac crest heights level? Yes/No
Patient A
Patient B

Q2. Perform the springing test on the marked thoracic segment - is it mobile? Yes/No
Patient A
Patient B

Is the subtalar joint restricted in motion? Yes/No
Patient A
Patient B

Of the 19 doctors who undertook the exercise 14 declared themselves experienced and 5 declared they were not.

Results

The results are pictoralised in the 2 x 2 tables below

THE TOTAL GROUP

BOTH 'EXPERIENCED AND INEXPERIENCED'

Iliac Crest Levels
The initial table (Fig 1) shows the results for the entire group, both experienced and not experienced. The 'true positivity' or sensitivity of the test was 8/19 or 42%. The 'true negativity'or specificity of the test was 10/19 or 53%.

Fig 1.

	TARGET DISORDER ILIAC CREST LEVELS	
TEST	Present	Absent
Positive	8	9
Negative	11	10

Springing Test of the D9 Spinous Process
This table (Fig 2) shows the results for the entire group. The sensitivity of this test was 14/19 or 74% and the specificity was 17/19 or 90%

Fig 2.

	TARGET DISORDER SPRINGING TEST	
TEST	Present	Absent
Positive	14	2
Negative	5	17

Subtalar Joint Movement
The table shows the results for the entire group. The sensitivity was 11/19 or 58% and a specificity of 15/19 or 79%.

Fig 3.

	TARGET DISORDER SUBTALAR JOINT MOVEMENT	
TEST	Present	Absent
Positive	11	4
Negative	8	15

THE EXPERIENCED GROUP

Iliac Crest Levels
The sensitivity of the test for this group was 7/14 or 50%, the specificity for this group was 7/14 or 50%.

Fig 4.

	TARGET DISORDER ILIAC CREST LEVELS	
TEST	Present	Absent
Positive	7	7
Negative	7	7

Springing Test of the D9 Spinous Process
The sensitivity of the test for this group was 11/14 or 79% and the specificity was 13/14 or 93%

Fig 5.

	TARGET DISORDER SPRINGING TEST	
TEST	Present	Absent
Positive	11	1
Negative	3	13

Subtalar Joint Movement
The sensitivity for this test was 7/14 or 50% and the specificity was 10/14 or 71%

Fig 6.

	TARGET DISORDER SUBTALAR JOINT MOVEMENT	
TEST	Present	Absent
Positive	7	4
Negative	7	10

The sensitivities for the 'iliac crest levels' test were 42% and 50% for the total group and the experienced group respectively. The specilities were similarly respectively 53% and 50%. On the figures, this tends to suggest that the observed results were at best as good as tossing a coin to make the decision as whether one particular iliac crest was higher that the other within the experienced group and perhaps not as good as that when the whole group is taken into account. The same argument applies to the confirmatory hardness of the absence of the sign and hence its true negativity.

For the springing test of the thoracic spinous process the sensitivities for the total and experience groups respectively were 74% and 79%. The specificities were respectively 90% and 93%. This suggests that this sign quite well validated when it is present but even better when it is absent in both the total and experienced groups.

For the subtalar joint movement the sensitivities for the total and experience groups respectively were 58% and 50% and the specificities were 79% and 71%. This suggests that this test is better validated when the sign is not present than when it is present. Also by adding the inexperienced group to the examination, the sensitivity was improved.

Discussion

On the face of it, the results are somewhat disappointing. It initially appears that time-honored tests may not be very useful and that those who profess to have the greater experience in musculoskeletal techniques may not in fact have the greater skill and may be no better than a non-experienced practitioner in assessment of a sign. However, there may be some explanation for the apparent anomalies in the results of two of the tests viz. the iliac crest height and the subtalar joint movement.

Firstly, the participating doctors were not allowed to compare one side with the opposite side to assess dysfunction Comparison is an important practical diagnosis feature for musculoskeletal doctors, for comparison of one feature with its opposite or like feature is the usual recommended practice i.e. 'asymmetry of form or function' is part of the diagnostic triad. It may, therefore, be unrealistic to expect a high degree of reliability if the above mentioned signs are to be evaluated, as it were, cold without the practitioner utilising his whole diagnostic armamentarium. This criticism applies to the test evaluation of the subtalar joint movement especially.

Secondly, in the urgency to set up and conduct the test during the conference with the organizers trying to find 'pathology' within the attending group, it may be that the signs were too subtle for the nature of the evaluation. Certainly the difference in heights of the iliac crests were in the order of 0.5cm or less. This may have blurred the validity of the test.

Thirdly, it would have been interesting to have the group reassess the signs after putting the whole group through a tutorial standardizing the examination techniques. We suspect that inconsistent technique may significantly affect the test outcome but this remains to be proven.

Fourthly, having the group examine the signs on two separate occasions would have contributed to useful comment on observer reliability.

For all that, one might have expected a greater consistency in the results among such as group.

The difficulties experienced were recognised by Dr. Jiri Dvorak in Zurich who in 1984 evaluated the practical techniques of a large number of Swiss chiropractors, osteopaths and medical manipulators, and published a series of examination and treatment manual therapy techniques which he believed could rationally be standardized. This series of procedures was rigorously filtered and now they have been and published, and now constitute the 'Dvorak' school.

Historically, medical practice has developed from human's earliest desire to influence disease and pestilence, birth and death, and injury and sickness. With little science and a lot of imagination many of the problems were laid at the door of irritable gods and natural disasters. But man's development of observational science and an ingrained determination for truth took him beyond the confined beliefs and controls, as of the medieval church, and created an 'art' of medicine. With current medical knowledge expanding exponentially there is now the 'science' of medicine and a recruitment for critical reasoning to be applied to all branches of medicine and this applies equally to Musculoskeletal Medicine.

What we as clinicians subject our patients to in our diagnostic and therapeutic approaches in Musculoskeletal Medicine is a summation of a pre-history of the knowledge base. Often, whether what we do is of any use or has any validity is not routinely tested critically by any scientific method but lives on by common usage and anecdotal validity, often passed on by the 'famous' men in the speciality. Not that common usage is a bad thing but the concepts that arise from it need to be tested to examine their worth.

There are eight queries as described by Sackett and Haynes et al for deciding the clinical usefulness of a diagnostic test. These are: -

1. Has there been an independent, 'blind' comparison with a 'gold standard' of diagnosis ?
2. Has the diagnostic test been evaluated in a patient sample that included an appropriate spectrum of mild and severe, treated and untreated, disease, plus individuals with different but commonly confused disorders ?
3. Was the setting for this evaluation, as well as the filter through which study patients passed, adequately described ?
4. Have the reproducibility of the test result (precision) and its interpretation (observer variation) been determined ?
5. Has the term 'normal' been defined sensibly as it applies to this test ?
6. If the test is advocated as part of a cluster or sequence of tests, has its individual contribution to the overall validity of the cluster or sequence been determined ?
7. Have the tactics for carrying out the test been described in sufficient detail to permit their exact replication ?
8. Has the utility of the test been determined ?

Summary

We believe there is a need for the application of 'critical reasoning' in the area of musculoskeletal assessment and that further two-by-two testing is appropriate, relatively simple, and, if well set up, valid in its results. The above example has been a learning curve for those who set it up and an inspiration to set up better procedures for testing in the future. It also makes an argument for the standardization of teaching techniques within the musculoskeletal fraternity.

Authors:

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Dr. Mark Johnston is the present Treasurer of the NZ Association of Musculoskeletal Medicine, a teacher within that Association, and also Board member and Treasurer of the Australasian Faculty of Musculoskeletal Medicine. Address: 16 Moana Ave, Orewa, Hibiscus Coast, New Zealand.

References:

1. Sackett, DL., Haynes RB, et al. Clinical Epidemiology; A Basic Science for Clinical Medicine, Little, Brown & Coy, 2nd ed 1991

CONFERENCE REPORT

Australian and New Zealand Associations of Musculoskeletal Medicine

Combined Conference 30th September - 30th October 1996

This year's combined conference of the Australian and New Zealand Associations of Musculoskeletal Medicine was held offshore at the Hotel Warwick on the picturesque Coral Coast in Fiji. It ran from 30 September to 3 October and was structured in a way to soften the discipline of lecture and workshop programme with ample free time to experience Fijian culture, snorkel amongst the coral or simply laze by the pool. If nothing else was learnt by those attending, everyone at least came away knowing the ubiquitous Fijian greeting 'Bula!' and the captivating rhythms of Fijian music and dance.

The lecture and workshop programme focused on problems on the pelvic girdle and lower limbs. The three major invited speakers were Dr Wolf Schamberger from Canada, Dr Greg Lovell from Adelaide, Australia and Dr Charlie Baycroft from Christchurch, New Zealand.

Wolf Schamberger is a Physiatrist and a Clinical Associate Professor in Physical Medicine and Rehabilitation at the University of British Columbia. His lectures and workshops principally addressed his major research interest area, the malalignment syndrome, a complex of musculoskeletal symptoms and signs related to the pelvic ring and the associated skeletal and soft tissue structures. He also spoke on the theory and application of prolotherapy with a complementary presentation on this topic coming from Dr Gurmit Dhillon from Adelaide.

Greg Lovell, a Sports Physician, spoke on the assessment and treatment of groin pain, hip problems and knee injuries at sport at work. He has done some interesting work on groin pain caused by incipient inguinal hernias which is reported in this journal. Charlie Baycroft is a general practitioner with expertise in foot and leg problems who is probably best known for his work in making orthotics accessible and affordable to most people through his Formthotics products. He gave an excellent review and workshop about the biomechanics of the foot and how this influences the choice of sport shoes and orthotics.

Kenneth Orr from Auckland gave a lecture on the sciaticas, drawing from his many years of experience in Musculoskeletal Medicine. Other workshops included 'What the Radiologist did not say' (Ron Palmer), injection techniques (Norm Broadhurst), trigger points of the lower limb (Clements Franzmayr and Joe Brownlea), and back and neck pain (Paul Quin and Mark Johnston).

The workshops also provided a golden opportunity for some interobserver reliability studies into a range of musculoskeletal signs, particularly some of the more subtle signs of pelvic girdle dysfunction. There was a ready supply of self-confessed experts and willling subjects to test these signs in a systematic way. The results of one of these studies appears in this edition and sheds some light on the precarious reliability of many of the signs we use in everyday practice.

The conference provided a good balance of theory, critical review of current beliefs and instruction in practical skills in Musculoskeletal Medicine which often extended beyond the official programme into the less formal parts of the week. Even the most experienced registrants should have taken away some new information or skills, or at least have experienced a healthy challenge of their existing knowledge and practice. Thanks are due to the conference organisers, especially Norm Broadhurst from AAAM and Angus Johnston from NZAMSM, for all their efforts in getting the conference off the ground.

Summaries of many of the key presentations at the conference appear in the following pages and will be continued in the next edition of this journal.

SEMINAR REPORT

DR. C. CHAN GUNN WEEKEND SEMINAR

20-21 JULY 96

NEEDLE ACUPUNCTURE

This seminar was run by the Australian Medical Acupuncture Society over 2 days in Sydney. Most of the attendees were members of the Acupuncture society. There was one other A.A.M.M. member there. Dr. Chan Gunn has the title of Clinical Professor, Multidisciplinary Pain Centre, University of Washington School of Medicine in Seattle. The course began with a few lectures on the theory of acupuncture, based on his theory of 'denervation supersensitivity'. He published a paper in SPINE in 1990 in which he proposed that pain was due to receptor organs becoming hyperreactive or supersensitive following denervation (Cannonis Law). He felt that there may be insidious onset of neuropathy of nerves (or radiculopathy) secondary to spondylosis and in turn this leads to a denervation neuropathy in muscles causing them to become tightened and shortened resulting in contractures. He later modified his theories to incorporate the term neuropathic pain in 1989.

He then demonstrated his technique termed 'Intramuscular Stimulation' which in essence is the needling of tender motor points. A number of patients were brought in from Dr. Marlene Yee's practice with pain of a musculoskeletal origin and it was interesting to see Dr. Gunn's approach. Examination consisted of a quick screen of range of movement followed by palpation of tender points. For the lower back he would ask the patient to lean over the couch and he ran the palm of his hand down the patients back feeling for any prominent spinous processes. Invariably he found an abnormality at L2. There was no consideration of the biomechanics of injury or joint function. Treatment consisted of inserting a needle into the tender paravertebral muscles until the needle 'grabbed' followed by withdrawing a few seconds later when hopefully the muscle relaxed. If the needle did not relax quickly then he would use an electrical stimulator for a few seconds. Interestingly he chose to concentrate on the region less than one finger breadth lateral to the midline, feeling for tight transverse bands of muscle.

His dry needling technique appeared reasonably well tolerated by the patients attending with the notable exception

of myself. I foolishly volunteered as a subject who suffers from Achilles tendonitis. He found plenty of tight bands in my back in addition to my left sciatic and right tibial nerves. When I complained of the lancinating tingling pain radiating to my toes he seemed happy about the needle placement and left those in for a bit longer!! One treatment is not expected to cure, but in this instance the cure was certainly worse than the complaint! Having said all this I have used his technique since then with good effect. My feeling is that the most important thing is to search carefully for all the relevant the tender motor points.

Vic Wilk.

RON'S LAMENT

One night when dining with his friends,
Whilst at the Fiji meeting,
The hotel manager appeared
And delivered him this greeting -

"Bula ,Sir, the bus awaits,
Your plane - it leaves tonight."
Surprised, he answered calmly,
"I'm sure that isn't right.

There must be some mistake - you see,
Tonight my room is paid for.
I've one more breakfast voucher -
And another day's fine weather."

The manager departed,
We joked about it all,
Another red was ordered
But later came a call -

"Is there a a Dr Palmer here?
We've checked the list again ,Sir,
Please hurry to the waiting bus,
We can delay no further."

His red was left abandoned
He thought he was a gonner.
And so, henceforth, such parting
Is deemed as 'Done a Ronner'!

(Penned on a flight back to soggy
Melbourne -PAT)

INTERNET CORNER

New AAMM Website

Since the last edition of the journal, David McGrath has been working hard in Canberra developing a website for AAMM. This is an exciting development for those who have connected to the internet who wish to keep up to date with what is happening in Musculoskeletal Medicine. The website aims to provide the following information and facilities:

- Easy access to an information data base for members and doctors interested in what we do.
- Advertising site for diplomas.
- Member contact information .
- Open forum for discussion of relevant and topical issues in Musculoskeletal Medicine as it pertains to our practices.
- Debate about contentious issues.
- Means of communicating with the world at large.
- Data base source for related web sites, associations and allied interests.
- New ideas as imagination allows.

At the recent ANZAMM conference in Fiji, David McGrath was elected to the newly created position of Website Editor with the responsibilities of developing and maintaining the website. He is keen to hear from association members who would like to participate in the website. The website address is:

http://www.ozemail.com.au/~davmcg/Home.html

Please note the site is CASE sensitive. David can be contacted through this site or by email at davmcg@ozemail.com.au. If you are not yet on email he can be contacted about the website at work on: (Ph) 062 851833 or (Fax)06-2852280.

We look forward to a rich exchange of information and much healthy debate through the new website. Congratulations and thanks to David on his efforts to date.

UPCOMING MUSCULOSKELETAL MEDICINE EDUCATIONAL ACTIVITIES

QUEENSLAND

DATE	*TITLE/KEY RESOURCE PERSON*	*VENUE*	*PROVIDER*	*CONTACT*	*CME POINTS*
9/2/97 9am - 1pm	Assessment of Pelvic Dysfunction - Dr Christian de Vaux & Dr Michael Yelland	QEII Hospital, Nathan, Brisbane	AAMM	Michael Yelland. Ph. (07) 3275 5444	Application for 6
1-2/3/97	Basic 'Kenna/Murtagh' course on assessment and treatment of back and neck pain.	Laguna Quays near Mackay	Queensland Chapter of ACPM	Geoff Harding Ph. (07) 3269 1842	32
7-8/3/97	Upgrade course for doctors who have completed a basic 'Kenna/Murtagh' course	Seaworld Nara Resort, Gold Coast	Queensland Chapter of ACPM	Geoff Harding Ph. (07) 3269 1842	32
9/3/97 9am-1pm	Treatment of Pelvic Dysfunction - Dr Christian de Vaux & Dr Michael Yelland	QEII Hospital, Nathan, Brisbane	AAMM	Michael Yelland. Ph. (07) 3275 5444	Application for 6
5/97	Practicalities of the Physiological Barrier in Manual Medicine (including peripheral joints) - Terence Vardy	QEII Hospital, Nathan, Brisbane	Queensland Chapter of ACPM	Peter Jackson Ph. (07) 3371 4144	Application to be made
7/97	Muscle Energy : Spray and Stretch - Terence Vardy	QEII Hospital, Nathan, Brisbane	Queensland Chapter of ACPM	Peter Jackson Ph. (07) 3371 4144	Application to be made
25-28/9/97	Myofascial pain seminar - Prof David Simons	ANA Hotel, Gold Coast	Continuing Medical Pain Education	Marilyn Strauss Ph. (07) 55313810	102 plus 25 practice assessment points
14-17/4/98	FIMM Conference	Conrad Jupiters, Gold Coast	AAMM	Carillon Conference Management Ph 07-33682644	Application to be made

NEW SOUTH WALES

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
7 - 8am Wednesday mornings 23/10/96 - 11/12/96	Orthopaedic & Sports Medicine topics (e.g. 'Injections in Sports Injuries' on 23/10/96)	Orthopaedics Skills Laboratory, St George Hospital, Kogarah	CME GP's, Sutherland and St George Hospitals	Rachel Dean Ph. (02) 350 2830	Application made
1/12/96	Thoracolumbar Spine - Dr Collinson, Dr Gonora, Dr Vote and Dr Diwa	Education Centre, Sutherland Hospital. Caringbah	RACGP Education Program	David Collinson Ph. (02) 525 6666	10

VICTORIA

DATE	TITLE / KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
28/11/96 7.30-10.00pm	Introductory workshop on lumbar spine examination	Trawalla, Melbourne	A.A.M.M. Vic Branch	Vic Wilk Ph. (03) 9596 7211	5 pending
27/2/97 7.30-10.00pm	Workshop lumbar spine II	Trawalla, Melbourne	A.A.M.M. Vic Branch	Vic Wilk Ph. (03) 9596 7211	5 pending
24-28/4/97	Aust Pain Society 18*AGM	Ayers Rock	Aust Pain Society	APS conference secretariat Ph. (02) 439 6744	
4-7/7/97	A.A.M.M. 27* Annual meeting	Melbourne	AAMM	V ic Wilk Ph. (03) 9596 7211	Application to be made

SOUTH AUSTRALIA

Contact Person: Norm Broadhurst - Ph. (08) 204 4613

WESTERN AUSTRALIA

Contact Person: Arnold Jones - Ph. (09) 390 4444

NEW ZEALAND

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
1-5/4/97	Basic Course A - Examination and Treatment of the Musculoskeletal System	Quality Hotel, Logan Park, Auckland	NZAMM	Mark Johnston Ph 09-426 5437	
10/97	Basic Course B - Examination and Treatment of the Musculoskeletal System	Quality Hotel, Logan Park, Auckland	NZAMM	Mark Johnston Ph 09-426 5437	

DIPLOMA AND CERTIFICATE COURSES

GRADUATE DIPLOMA AND CERTIFICATE IN MUSCULOSKELETAL MEDICINE - FLINDERS UNIVERSITY

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
9/11/96 - 17/11/96	Anatomy, Physiology and Biomechanics of the Musculoskeletal System	Flinders, Medical Centre, Adelaide	Department of Orthopaedic Surgery, Flinders University	Norm Broadhurst - Ph. (08) 204 4613	140
8/2/97 - 16/2/97	Clinical Skills in Managing Non-surgical Musculoskeletal Dysfunction	Flinders, Medical Centre, Adelaide	Department of Orthopaedic Surgery, Flinders University	Norm Broadhurst - Ph. (08) 204 4613	140

Note that this unit can be taken as a stand alone unit.

POSTGRADUATE DIPLOMA IN PHYSICAL MEDICINE - UNIVERSITY OF SYDNEY

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
Commenc-ing16/2/97	Unit 1	University of Sydney	Department of Anatomy and Histology, University of Sydney	Adam Tierney, Ph. (02) 9351 7064	60

DIPLOMA IN MUSCULOSKELETAL MEDICINE - UNIVERSITY OF CHRISTCHURCH, OTAGO

DATE	TITLE/KEY RESOURCE PERSON	VENUE	PROVIDER	CONTACT	CME POINTS
20/2/97 - 5/6/97	First semester	Teleconference	Christchurch Clinical School of Medicine	Philip Watson - Ph: (W) (07) 3345 8999 (H) (07) 3344 4940 Fax (07) 3344 6688 OR Diploma Secretariat Ph. (07) 3844 1138 Fax (07) 3844 0909	50
17/7/97 - 23/10/97	Second semester	Teleconference	Christchurch Clinical School of Medicine	Philip Watson or Diploma Secretariat	50
14-23/3/97	First year paper (701) on campus	Brisbane	Christchurch Clinical School of Medicine	Philip Watson or Diploma Secretariat	50
8-12/9/97	First year paper (701) on campus	Brisbane	Christchurch Clinical School of Medicine	Philip Watson or Diploma Secretariat	50
TBA	Second year paper (709) on campus	Brisbane	Christchurch Clinical School of Medicine	Philip Watson or Diploma Secretariat	50

*DIPLOMA IN MUSCULOSKELETAL MEDICINE - UNIVERSITY OF NEWCASTLE**

DATE	*TITLE/KEY RESOURCE PERSON*	*VENUE*	*PROVIDER*	*CONTACT*	*CME POINTS*
11-20/1/97	Knee, Foot 1 - Prof Nik Bogduk	University of Newcastle	University of Newcastle	Prof Nik Bogduk - Ph. (049) 215 608	Nil
5-6/97	Chronic Pain	University of Newcastle	University of Newcastle	Prof Nik Bogduk - Ph. (049) 215 608	Nil
10-11/97	Medicolegal Issues	University of Newcastle	University of Newcastle	Prof Nik Bogduk - Ph. (049) 215 608	Nil

* Note that fresh enrolments in this diploma are not being taken, but irregular enrolments for people who want to do one module (for no credit) at a cost of $1,000 are being accepted.

ADVANCE NOTICE

THE 12TH INTERNATIONAL CONGRESS OF F.I.M.M.

Venue: **Brisbane, Australia**

Date: **14 - 17 April, 1998**

Contact: **Dr Philip Watson**

Australian Musculoskeletal Medicine November 1996

Flinders University of South Australia

Musculoskeletal Medicine Course

Graduate Certificate (3 modules) and Graduate Diploma (6 modules) programmes are available for medical practitioners.

The uniqueness of the Flinders programmme includes:

- *Live lecturers, open discussion, up-to-date information from the latest research.*
- *Prosected anatomy material and practical experiments.*
- *Various injection and dry needling techniques.*
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- *Four weeks of on-campus activity supplemented by Distant Learning Programs.*

Next Intake: 9th November to 17th November 1996

Topic: Anatomy, Physiology & Biomechanics

Enquiries: Mr. Michael McKay, Health Sciences
Flinders University, Bedford Park 5042

NB The program has been accredited for training by the Australian Faculty of Rehabilitation Medicine.

CME points available for RACGP.

Applications: **Closing Date: 30th September, 1997**

The Flinders Diploma begins in November each year and the on-campus modules take advantage semester breaks so that full access to all facilities is available.

This year's intake is fully subscribed.

The University of Sydney
Faculty of Medicine

Postgraduate Diploma in Physical Medicine (Musculoskeletal)

A Postgraduate Diploma in Physical Medicine (Musculoskeletal) will be conducted by course work on a part-time basis over two years. A total of six weeks full-time attendance on campus will be required to complete practical and clinical sessions. These sessions will be of one week's duration. The course is designed for general practitioners. A general training in all aspects of musculoskeletal medicine will be included. The main emphasis of the course will be to acquire knowledge of musculoskeletal anatomy, biomechanics and physical medicine diagnostic skills and treatment, including manual and injection techniques.

Enquiries: Adam Tierney
Administrative Officer
Department of Anatomy & Histology
The University of Sydney NSW 2006
Phone: (02) 9351 7064 Fax: 9351 6556
Applications close: 7 January 1997

Provisional CME approval - 30 points semester

PRESENTATION OF PAPERS FOR PUBLICATION

Most submissions for publication are now in electronic form and are preferred in that way.. To assist potential contributors we reprint the helpful hints from our printer which, if followed, save time and money in the preparation process.

1. Media - in order of preference:
   a) 3.5" computer disc
   b) 5.25" computer disc
   c) Typed double spaced - ensure good black type to allow fast scanning
   d) Handwritten - is a nightmare for non-medically experienced typists, far better for you to have typed then corrected by you. Typing bureaus can also prepare a disc for you.
2. Always provide hard copy - preferably double spaced for manual editing.
3. Software: MS Word or Wordperfect are preferred but most proprietary wordprocessing programmes may be translated. If using one of the more obscure programmes check to see if it can save or export to MS Word or Wordperfect formats.

NB. The types of software often provided free with some low cost computer packages usually do not translate well, if in doubt send us a sample disc well before the publication date for us to test.

When saving the file(s) to disc first check your programme to see if it has an 'Export' option and if so please use it. Look carefully to find the various options available in 'Save As' or 'Export' mode. In Windows programmes there is usually a box showing the format the file will be saved in, many people do not realise that this format can be changed by using the 'slider' to select a different format to that shown in the box.

If you have the very latest version of a programme then always save/export in an older format - again available for selection as described above.

4. Formatting: Ensure your typist CLEARS ALL TABS before starting and sets only the tabs needed for the article. If the typist uses only default tab settings and simply uses multiple tabs to position text or tables then these must all be removed manually and reset to fit our format - a long process in some papers.

   Similarly, do not use the space bar to position quantities of text or to tabulate. If numerous insets or headings are required - use the tabs.