International Orthopaedics (SICOT) (1985) 9: 1–10

International
Orthopaedics

Recent advances in the treatment of low back pain*

A. Nachemson

Department of Orthopaedic Surgery, Sahlgren Hospital, S-413 45 Göteborg, Sweden

Summary. *There is at the present time an epidemic of low back pain in the industrialized countries. Although the exact origin of such pain is still unknown, there is increasing awareness that the outcome is usually favourable. Only some 10% of those suffering an acute episode of back pain are incapacitated for more than 6 weeks. The causes of long standing back pain are being identified in an increasing number of patients. They include anatomical and pathological disturbances in the motion segment as well as psychological, social and political causes. There are numerous factors which influence the pathophysiology of the motion segment. The degree of loading has been successfully measured and delineated for various postures and exercises, including those at work. The nutritional pathways to the disc have been established and the effect of various external factors measured. Movement is good for the disc and the importance of continuous passive motion for the healing of diseased or injured connective tissues is now established. Activation of large muscle groups increases the production of the body's pain reducing encephalins. Early diagnosis and early mobilisation of the patient should be of benefit, and long term bed rest and inactivity must be prevented. Fewer cases will need operation in the future. Improved imaging techniques and better methods of operation and fixation will help those few who have a definite pathological lesion amenable to surgery.*

Résumé. *Il y a actuellement une véritable épidémie de lombalgies dans les pays industrialisés. Bien que l'origine exacte de ces douleurs demeure inconnue, il apparaît de plus en plus que le pronostic en est habituellement favorable. Dix pour cent seulement de ceux qui présentent un épisode lombalgique aigu ont une incapacité supérieure à 6 semaines. Les causes des lombalgies prolongées sont maintenant retrouvées dans un nombre croissant de cas. Elles englobent aussi bien des lésions anatomiques et pathologiques du segment mobile du rachis que des éléments psychologiques, sociaux et politiques. De nombreux facteurs sont en cause dans la physiopathologie de ce segment mobile. L'importance de la charge a été mesurée pour des positions et des mouvements divers, y compris ceux nécessités par le travail. Le mode de nutrition du disque a été précisé et l'effet de différents facteurs extérieurs a été mesuré. Les mouvements jouent un rôle favorable pour le disque et l'importance de la mobilisation passive continue dans la cicatrisation du tissu conjonctif malade ou traumatisé est maintenant bien établie. La mise en activité de groupes musculaires importants augmente la production d'encéphalines analgésiantes. Un diagnostic et une mobilisation précoces du malade sont bénéfiques, aussi le repos prolongé au lit et l'inactivité doivent-ils être évités. Dans l'avenir un petit nombre de cas nécessiteront un traitement chirurgical. Des techniques améliorées et de meilleures méthodes d'opération et de fixation aideront ceux chez qui une lésion pathologique définie justifie une intervention.*

Key words: *Low back pain, Pathogenesis, Treatment*

Introduction

Orthopaedic surgery has made great advances in the last decade in many areas like replacement of

* Current trends lecture SICOT 1984

total joints and the treatment of scoliosis. For the patient with low back pain progress has been somewhat slower. There are, however, indications of a breakthrough within the next decade, due to the collaboration of orthopaedic surgeons, specialists in rheumatology and rehabilitation, biochemists, biomechanical engineers and basic scientists in the pain field. At the present time population studies demonstrate an annual prevalence of low back pain ranging from 2%–5% with an incidence of 80% [9, 12, 40, 54, 63, 64, 75, 102, 125, 132, 142, 148].

The origin of low back pain

There are many structures in the motion segment which could theoretically cause pain [150]. The exact nature of the pain remains obscure, but the main interest has centred around the intervertebral disc, with its surrounding longitudinal ligaments, and the facet joints. Recent knowledge on both the muscles and the vertebraes will also be mentioned. Without doubt the mechanical integrity of the intervertebral disc proper influences all the surrounding structures. When the disc degenerates there is an increase in facet load [73, 152], altered distribution of stress concentrations in the end plates and the subchondral bone of the vertebrae [43], encroachment on the nerve root canal [14, 15, 82, 94] and an increased demand for the muscles, especially the intersegmental groups, to stabilize the motion segment [58, 111].

The intervertebral disc is the largest avascular structure in the body and its nutritional pathways have been established by several researchers, including our group in London and Gothenburg [50, 51, 53, 131]. The integrity of the nucleus pulposus depends on diffusion pathways through the end plates and the annulus fibrosus in order to maintain the proper balance of water, solutes, glycosaminoglycans, protein and collagen. Some solutes enter mainly through the end plates, while others pass through the annulus. The high level of anaerobic metabolism (95% of the disc tissue) has been established and is also illustrated by the finding that the oxygen concentration in the centre is only 1.3 kPa and near the end plates 6.5 kPa. Accordingly the lactic acid concentration is high in the nucleus, 8 μ Mol/g wet tissue, whilst near the end plate it is 2 μ Mol and in plasma 1 μ Mol. Lactic acid with its high hydrogen ion concentration can cause pain via chemical pain receptors. The most deficient nutrition is in the boundary zone between the nucleus and annulus [85]. This is also where fissuring starts to occur as the earliest sign of disc degeneration. These circumferential fissures weaken the lamellae of the annulus such that, theoretically at least, the existing tangential stresses could break the fibre bundles. There is thus a combined biomechanical and biochemical explanation for the occurrence of radiating fissures through the annulus to the external longitudinal ligament, particularly in the posterior part where the annulus is somewhat weaker.

In animal experiments several factors have been investigated and found to influence nutrition and metabolism in the disc. Spinal fusion has a negative influence on the discs included, while the discs adjacent to the fusion become metabolically hyperactive [51]. Motion increases the flow of nutrients into the disc in a positive way, and in dogs half an hour to an hour's exercise per day seems to be enough compared to just sitting or moving in the cages [52]. Smoking and vibration are other parameters with negative influences [53].

Hansson and his collaborators [44, 45] have established the frequent occurrence of healing microfractures of the trabeculae of the subchondral bone near the end plates. Biomechanical tests demonstrated the presence of such fractures in cadavers before any signs of change could be seen even on high quality x-rays. The importance of mechanical factors in abnormality of the motion segment is further demonstrated in recent Swedish statistics [114, 149], which show that workers who have a sedentary job are off work for about 10 days if they experience an attack of low back pain, but those whose job entails heavy lifting in uncomfortable positions, and who regard their work as very strenuous and monotonous, have an average of 40 days away from work with each attack. Chaffin and his collaborators [21, 65] have also demonstrated that a mismatch between the physical requirements of the job and the strength of the worker increases the frequency of injury to the low back.

Measurement of disc pressure

In order to evaluate the load on the spine I established a method of measuring disc pressure, first in cadaver material and then in living subjects, and have published several articles on this subject [84, 85, 87, 92]. Recent findings have emphasized

the importance of having a good lumbar support and armrest while performing sitting tasks, such as driving a car, for long periods of time. Kelsey et al. [61] recently and independently established from an epidemiological study that those driving a Volvo car, which has these mechanically advantageous features, had a significantly less risk of admission to hospital because of low back pain and sciatica. When rising from the sitting position with the help of an armrest 30% less load is experienced in the lumbar spine than when rising without [87].

Flexion and rotation also induce high loads on the lower motion segments. With 5 kg (50 N) in each hand the load exceeds 2100 N in only 20° of forward bending and rotation [107]. Again, Kelsey et al. [62] in a recent study found that those who were regularly subjected to this pattern of loading were six times more liable to be admitted to hospital because of a prolapsed intervertebral disc than those who were not. Twisting more than 20 times a day with a weight of more than 25 Lbs (10 kg) was the highest risk factor encountered.

Biomechanically, the use of corsets and braces has also been studied [93], demonstrating diminution of the load to the lumbar spine by about 30% when bending forward in the sagittal plane. We could find no significant differences between Camp corsets, the Rainey jacket or the Boston type brace for back pain.

It is necessary to define the limits beyond which mechanical loading of the back will produce pain [4, 76]. Intradiscal pressure measurements and the improved EMG techniques cannot be utilized widely at work to enable the establishment of these, but validated computer based models of the spine offer an opportunity. Some simple models have been established; Schultz et al. [93] confirmed the accuracy of this technique by EMG and disc pressure measurements. By using such models we will soon be able to produce scientifically backed guidelines for industry. However, well motivated guidelines are already in use [65, 83, 117].

Segmental instability is another mechanical factor which is important clinically but poorly defined and understood at the present time [67, 74, 91]. There are several indirect indications that this is one of the more important entities, the solution of which will mean a breakthrough in the understanding and treatment of low back pain. Increased mobility can be demonstrated with spondylolisthesis [95]. Recent studies have established some biomechanical criteria for two plane instability in cadaver material [99, 143]. Studies on the motion of the centres of axis for rotation, the centrodes, have been carried out on cadavers in Toronto by Seligman et al. [109], who have shown that in the moderately degenerated motion segments the location of the centrodes move eratically, but became more concentrated or near normal when the discs degenerated further. It has been observed clinically that improvement in symptoms in the more chronic back syndromes paralleled decrease in motion of the lumbar spine [71]; where definite instability was seen clinically, as in spondylolisthesis, a fusion helped in a fairly high percentage of cases, even in patients receiving Workmens Compensation Insurance [26, 36].

Very few radiological signs demonstrable on an ordinary x-ray are significant in low back pain [77]. However, spondylolisthesis, congenital or traumatic kyphosis, and Scheuermann's disease with involvement of two or more vertebral bodies in the lumbar spine all carry an increased risk for chronic back pain [118]. Ankylosing spondylitis, rheumatoid disease and osteoporosis are other causes of low back pain which may be diagnosed by plain radiographs.

The technique of ultra-sound scanning, first employed by Porter and his group in Doncaster [97], is a promising tool in epidemiological research. They have demonstrated that the average diameter of the spinal canal in patients with chronic low back pain is significantly less than in those who have never suffered back pain. Ramani [101] has shown this to be true in patients with symptoms from a disc hernia. However, the limitations of the method are considerable and it cannot be used in the individual case [34, 60].

The established method of investigating nerve root pain before contemplating surgery is a myelogram using water soluble contrast material such as metrizamide, and the literature is vast on its correct use and contraindications [55, 56]. Even better contrast materials, such as iohexol, are now becoming available.

The use of computerized tomography (CT) for establishing abnormalities of the motion segment such as disc herniation, spinal stenosis and facet arthritis has gained increasing popularity in this decade [16, 41], but recent studies [8, 146] have clearly demonstrated its limitations. Wiesel et al. [146] found in 52 volunteers without back pain an agreement of only 11% between 3 different interpreters of the scans; 35 scans were read as abnormal and 22% were diagnosed as having a disc herniation. In a continuation of this study [146] of the

prospective evaluation of patients with surgically proven herniation or stenosis, the metrizamide myelogram was significantly superior in correct diagnosis, even with spinal stenosis.

The future in this field may lie in nuclear magnetic resonance examination (NMR). The water content of the disc can be established, and this indicates both its mechanical competence and the degree of chemical degradation. Disc hernias have been demonstrated but not perhaps as clearly as with CT. Interestingly the postoperative picture shows swelling and changes in the muscles, which may perhaps also occur in some patients with acute back pain. This type of investigation should also enable studies of the importance of rupture of the muscle fibres in acute low back pain.

Pain

Knowledge of the chemical mediation of pain is increasing every year and we already know that there are several encephalins that are of importance [2, 57, 126, 127]. Rydevik and his collaborators [25, 105] have recently demonstrated that there is diffusion of substances including endorphines into the spinal nerve root. Thus, the persistence and competence of the nerve root sleeves offers a new dimension to understanding root pain. Proper function of the nerve root sleeve may well be of importance in supression of pain. Experiments both with rats [100] and men [127] have clearly demonstrated that the level of endorphine in the cerebrospinal fluid is of importance for pain supression, and other investigators [112] have shown that patients with chronic back pain have a diminished amount of the substance in the CSF. The importance of activating large muscle groups to increase the level of endorphine in the CSF in order to reduce pain sensitivity has also been established [17, 22, 37, 108, 153]. We, as orthopaedic surgeons, must use this information in the management of patients who are in pain, particularly where no definite pathological lesion can be established.

Treatment of low back pain

It is difficult to establish the effectiveness of treatment in prospective randomized trials in a disease in which the natural history is so extremely good. We again demonstrated recently in several thousand patients who were off work with back pain, that by 6 weeks 90% were back to work, 60% within 1 week [89]. Prospective randomised trials have demonstrated the effectiveness of pain relief and early return to work with both a few days of bed rest, and with an educational back programme such as the Swedish back school [10, 105]. However, many other methods of treatment which are currently used such as traction, William's flexion and extension exercises, x-ray therapy, shortwave therapy, ultrasound therapy, muscle relaxants, bio-feedback programmes, anti-inflammatory drugs and injections and manipulations of various types have failed to demonstrate any significant effect on the natural history or on return to work [86, 156]. Some, like manipulation, seem to offer shortlasting (1–3 h) relief of pain [29, 49, 113], an effect resembling certain drugs [6, 7, 47].

The scientifically proven methods for the treatment of acute back pain thus include bed rest, proper advice on back care and analgesics [10, 145]. It seems that in this field, as in many others, advice based on knowledge, particularly biomechanical knowledge, is superior to any type of treatment based on "armchair" thoughts on the pathology.

In the management of chronic low back pain the literature does not contain a single study demonstrating the positive effect of treatment, although several reports, not controlled, indicate some benefit from corsets, traction, transcutaneous nerve stimulation, acupuncture, facet injections and a comprehensive back care programme [1, 32, 81, 110, 123, 157].

Treatment of low back pain and sciatica

Again there is scientific evidence demonstrating that bed rest and certain drugs give relief of pain, and some studies favour epidural injections of steroid [27]. With a myelogram that is positive, indicating a disc herniation, other studies however, demonstrated no effect with epidural steroid injections [116, 144], but showed better results following surgical removal of the disc hernia than with continuous conservative treatment. Weber's landmark studies [138, 139, 140] have helped us orthopaedic surgeons tremendously. Both he and others have pointed out that surgery should be performed within 3 months of the onset of sciatica in order to get excellent results [119, 120, 121, 124]. Weber followed his patients for 1 year, when the difference between his groups was significant, and then for 4 and 10 years when the results were similar in the conservatively treated and the operated groups. He also established in the 10-year-follow-up [140], that although some 15% of his pa-

tients (an equal number in both groups) received a disability pension, they were all able to perform some tasks in their homes or on their farms, and did not suffer unduly from pain in their back or legs. There is increasing evidence that both back and leg pain have a time cycle of 4–5 years! [3, 129, 130].

In another study [141] patients with disc herniations which had been demonstrated by myelography were randomly treated by different methods of traction or without traction. Blind assessment of their progress was then undertaken. The methods of traction used were the True Trac apparatus, the Spina Trac apparatus, autotraction [72] and manual traction. The results showed no difference whether traction had been applied or not with any of the methods. However, with autotraction or manual traction, it was shown that the 25%–30% of patients who responded did so immediately. Thus, if the first trial of traction gives pain relief it is worth continuing with a few more sessions.

Chymopapain, a proteolytic enzyme which disrupts the protein mucopolysaccharide complexes in the nucleus pulposus, is widely used to treat disc herniation [69, 78, 115], and its superiority over saline injection has been proved [38]. However, in two randomised trials [23, 30] comparing chemonucleolysis with surgery, the latter was found to be better; there was a 50% failure rate in the chymopapain group against only 10% in those treated by operation. The overall rate of complications in a large number of uncontrolled studies seems to be the same in both methods, but the increased risk of allergic reactions and the rare occurrence of a severe transverse myelitis should restrict the use of chymopapain to the last resort in patients unsuitable for surgery. However, it is likely that improved chemical methods will evolve within the next decade.

The surgical removal of a disc hernia still remains the main treatment for these patients [48, 119, 120, 128]. Microsurgical techniques have been adopted and might be an improvement for the very young patients. The limited view makes a total evaluation of the nerve root and canal difficult, and this is often necessary in middle aged or older patients. The technique of proper positioning of the patient in the Mohammedan prayer position, and the use of a headlamp and magnifying loops are improvements in surgical methods which should be adopted by all surgeons operating on discs.

The value of further operations is questioned in many recent studies [35, 66, 134]. My own experience, with only 25% of patients improving after 3 years of follow-up, mirrors this gloomy outlook. Only if a new disc herniation is found are the results good enough to warrant another operation. A reminder for disc surgeons should read;

- You only got one chance!
- Before operating, remember the natural history.
- Before operating, don't forget the patients' social, psychological and work situation.
- Repeat surgery is most of the time unrewarding and should be avoided if at all possible.
- Dont't think you can find anything the previous surgeon overlooked. In 9 cases out of 10 you are wrong!!

When considering the management of chronic sciatica, there are no prospective studies in the literature demonstrating one method to be superior to another. We still use facet injections, denervations, epidural steroid injections, traction, TNS, plastic braces of various types and even sometimes resort to laminectomy with fusion [20, 33, 36, 39], usually with dismal results. The overall chance of successful rehabilitation to work of a patient suffering from low back pain and/or sciatica for longer than 6 months is only around 40% [13, 18, 19, 110, 122]. If the patient has had pain for more than 1 year this dwindles to about 15%. It seems that if the pain persists for more than 3 months the psychological make up of the patient changes, and Waddell [135, 136, 137] has illustrated these points very well. Everyone interested in the treatment of chronic low back pain should consult his findings.

It is obvious that, with the exception of the treatment of clearcut acute disc herniation, we need to stop, think and perhaps change our whole programme of management. This new way of thinking was recently published in Clinical Orthopaedics [90], "Work for all – for those with low back pain as well". Take into consideration our present knowledge of the natural history, the chance of recurrence which diminishes after a couple of years, and the influence of sick leave benefits [28, 80, 89, 129, 140].

The adverse psychological and somatic affects of inactivity are now well known to all physicians [13, 46, 122]. I have touched in this lecture on our own pain modulation and how it can be influenced by motion. We have undertaken studies on the function of the back in patients with lumbar pain and have shown that they have near-normal strength except in twisted positions [79, 88]. Many scientists have demonstrated that diseased tissues

of the type seen around the motion segment heal quicker with continuous passive motion [59, 104, 106, 133, 147]. Our own studies have demonstrated the improved nutrition to discs obtained by motion [52].

These observations indicate that we must tell our patients of the good prognosis following an attack of acute low back pain, we must instruct them in what to do, how to move and of the beneficial effects of mobilisation towards work. In the majority of cases, excluding those 15%–20% who have a demonstrable pathological lesion, mobilisation and a gradual return to work using proper mechanical care is the most effective treatment [19, 20]. Wynn Parry [151], studying patients with arm pain due to brachial plexus lesions, said that the most significant feature in improvement of pain was the patient's return to work.

In a controlled study of 70 patients, we have clearly demonstrated the effect of such an approach and a more scientifically controlled prospective study is under the way.

What can we expect surgically in the future? Considering the importance of instability I think that we urgently need to improve our diagnostic methods to delineate the normal and demonstrate abnormal movement in the lumbar motion segment. We must also improve our methods of diagnosis [42, 70] and fixation in patients with spinal stenosis and root canal stenosis. Our present techniques of operation often render the motion segment unstable [11, 88], or our fusions do not unite in a satisfactorily high percentage of patients [96, 103].

Of some importance today is the syndrome of spinal nerve entrapment [5, 24, 31]. However, our knowledge is too meagre to establish its proper place in our surgical armamentarium. Our methods of avoiding postoperative surgical scarring are not established [68, 154]. Recently Porter et al. [98] delineated the natural history of root entrapment syndromes and found them somewhat better than previously thought. Unfortunately his operated cases did not significantly differ from those treated conservatively at follow – up one to 4 years later.

Future improvement

Preventive methods have been used in various industries with some success and with our increasing knowledge of biomechanical factors we can expect even better results in the future [90]. Increased robotization will also help to improve the more strenuous work environments, which now account for many sufferers from chronic back pain. Improved prevention programmes, with help from politicians [28], industrial leaders and safety engineers, will diminish the problem of back pain in the future. Improved methods of diagnosis will delineate more factors of pathogenetic importance, and improved information and activation both to the general public and to those suffering their first attack of low back pain, will diminish chronicity.

I also predict that within the next decade we will do much less surgery, but have very much improved results, depending in particular on our specific laboratory and imaging methods for instability and nerve root pathology. We will also have improved methods of mechanical stabilization, and for the surgical relief of nerve root involvement. Thus, although we are now living in an epidemic of back pain we will, within the next decade, be able to control this. We will be able to do so, not by our surgical efforts, but by epidemiological, biomechanical and diagnostic research and by collaboration with other specialities including politicians and industrial leaders, across faculty borders.

References

1 Ahlgren SA, Hansen T (1978) The use of lumbosacral corsets prescribed for low back pain. Prosthet Orthop Int 2: 101–104
2 Almay BG, Foke J, von Knorring L, Terenius L, Wahlström A (1978) Endorphins in chronic pain. I. Differences in CSF endorphin levels between organic and psychogenic pain syndrome. Pain 5: 153–162
3 Andersson GBJ (1981) Epidemiologic aspects on low back pain in industry. Spine 6: 53–60
4 Andersson JAD (1980) Back pain and occupation. In: Jayson M (ed) The lumbar spine and back pain. Pitman medical, London, pp 57–82
5 Arnoldi CC, Brodsky AE, Cauchoix J, Crock HV, Dommisse GF, Edgar MA, Gargano FP, Jacobsson RE, Kirkaldy-Willis WH, Kurihara A, Langenskiöld A, Macnab I, McIvor GWD, Newman PH, Paine KWE, Russin LA, Sheldon J, Tile M, Urist MR, Wilson WE, Wiltse LL (1976) Lumbar spinal stenosis and nerve root entrapment syndromes. Definition and classification. Clin Orthop 115: 4–5
6 Baratta RR (1976) A double-blind comparative study of carisoprodol, propoxyphene, and placebo in the management of low back syndrome. Curr Ther Res 20: 233–240
7 Bell G, Rothman R (1984) The conservative treatment of sciatica. Spine 9–1: 54–56
8 Bell GR, Rothman RH, Booth RE, Cuckler JM, Garfin S, Herkowits H, Simeone FA, Dolinskas C, Han SS (1984) A study of computer-assisted tomography. II. Comparison of metrizamide myelography and computed tomography in the diagnosis of herniated lumbar disc and spinal stenosis. Spine 9: 552–556

9 Benn RT, Wood PHN (1975) Pain in the back; An attempt to estimate the size of the problem. Rheumatol Rehabil 14: 121–128
10 Bergquist-Ullman M, Larsson U (1977) Acute low back pain in industry. Acta Orthop Scand Suppl 170
11 Berkson M, Schultz A, Nachemson A, Andersson GBJ (1977) Voluntary strengths of male adults with acute low back syndrome. Clin Orthop 129: 84–95
12 Biering-Sörensen F (1984) Physical measurements as risk indicators for low-back trouble over a one-year period. Spine 9: 106–119
13 Blumer D, Heilbronn M (1982) Chronic back pain as a variant of depressive disease: The pain prone disorder. J Nerv Ment Dis 170: 381–406
14 Bose K, Balasubraminiam P (1984) Nerve root canals of the lumbar spine. Spine 9–1: 15–18
15 Breig A, Marions O (1963) Biomechanics of the lumbosacral nerve roots. Acta Radiol Diagn 1: 1141–1160
16 Burton CV, Heithoff KB, Kirkaldy-Willis W, Ray CD (1979) Computed tomographic scanning and the lumbar spine. Part II: Clinical considerations. Spine 4: 356–368
17 Cady LD, Bischoff DP, O'Connel ER, Thomas PC, Allan JH (1979) Strength fitness and subsequent back injuries in firefighters. J Occup Med 21: 269–272
18 Cairns D, Mooney V, Crane P (1984) Spinal pain rehabilitation: Inpatient and outpatient treatment results and development of predictors for outcome. Spine 9: 91–95
19 Catchlove R, Cohen K (1982) Effects of directive return to work approach in the treatment of workman's compensation patients with chronic pain. Pain 14: 181–191
20 Cauchoix J, Benoist M, Chassaing V (1976) Degenerative spondylolisthesis. Clin Orthop 115: 122–129
21 Chaffin DB (1979) Manual materials handling. J Environ Pathol Toxicol 2: 31–66
22 Colt EWD, Wardlaw SL, Frantz AG (1981) The effect of running on plasma β-endorphin. Life Sci 28: 1637–1640
23 Crawshaw C, Frazer AM, Merriam WF, Mulholland RC, Webb JK (1984) A comparison of surgery and chemonucleolysis in the treatment of sciatica. A prospective randomized trial. Spine 9: 195–198
24 Crock HV (1976) Isolated lumbar disk resorption as a cause of nerve root canal stenosis. Clin Orthop 115: 109–115
25 Dahlin LB, Rydevik B, Lundborg G (1984) Pathophysiology of nerve entrapments and nerve compression injuries. In: Hargens AR, (ed) Tissue nutrition and viability. Springer, Berlin Heidelberg New York (In press)
26 Davis IS, Bailey RW (1976) Spondylolisthesis. Indications for lumbar nerve root decompression and operative technique. Clin Orthop 117: 129–134
27 Dilke TFW, Burry HC and Grahame R (1973) Extradural corticosteroid injection in the management of lumbar nerve root compression. Br Med J 2: 635–637
28 Disability insurance program. Public hearings before the subcommittee on social security of the Committee on Ways and Means. House of Representatives, Ninety-fourth Congress, Second Session, May 17, 21: June 4, 11, 1976. Washington DC US Government Printing Office, 1976
29 Doran ML, Newell DJ (1975) Manipulation in treatment of low back pain: A multicentre study. Br Med J 2: 161–170
30 Ejeskar A, Nachemson A, Herberts P, Lysell E, Andersson G, Irstam L, Peterson L-E (1982) Surgery versus chemonucleolysis for herniated lumbar discs. Clin Orthop 171: 252–259
31 Epstein JA, Epstein B, Levine LS (1974) Surgical treatment of nerve root compression caused by scoliosis of the lumbar spine. J Neurosurg 41: 449–454
32 Fairbank JCT, Park WM, McCall IW, O'Brien JP (1981) Apophyseal injection of local anesthetic as a diagnostic aid in primary low back syndromes. Spine 6: 598–605
33 Farfan HF, Kirkaldy-Willis WH (1981) Present status of spinal fusion in the treatment of lumbar joint disorders. Clin Orthop 160: 198–214
34 Finlay D, Stockdale H (1981) Ultrasonic quantification of the lumbar spine: Some critical comments. J Bone Joint Surg 63-B: 639
35 Finnegan WJ, Fenlin JM, Marvel JP, Nardini RJ, Rothmann RH (1979) Results of surgical intervention in the symptomatic multiply-operated back patient. J Bone Joint Surg 61-A: 1077–1082
36 Flynn JC, Hoque MPA (1979) Anterior fusion of the lumbar spine. End result study with long-term follow-up. J Bone Joint Surg 61-A: 1143–1161
37 Fraioli F, Moretti C, Paolucci D, Alicicco E, Crescenzi F, Fortunio G (1980) Physical exercise stimulates marked concomintant release of β-endorphin and adrenocorticotropic hormone (ACTH) in peripheral blood in man. Experientia 36: 987–989
38 Fraser RD (1982) Chymopapain for the treatment of intervertebral disc herniation. Spine 7: 608–612
39 Frymoyer JW, Hanley E, Howe J, Kuhlmann D, Matteri R (1978) Disc excision and spine fusion in the management of lumbar disc disease. A minimum ten-year follow-up. Spine 3: 1–6
40 Frymoyer JW, Pope MH, Costanza MC, Rosen JC, Goggin JE, Wilder DG (1980) Epidemiologic studies of low back pain. Spine 5: 419–423
41 Genant HK, Wilson JS, Bovill EG, Brunelle FO, Murray WR, Rodrigo JJ (1980) Tomography, computed, of the musculoskeletal system. J Bone Joint Surg 62-A: 108–112
42 Haldeman S (1984) The electrodiagnostic evaluation of nerve root function. Spine 9: 42–48
43 Hansson T, Roos B (1981) The relation between bone mineral content, experimental compression fractures, and disc degeneration in lumbar vertebrae. Spine 6: 147–153
44 Hansson T, Roos B (1981) Microcalluses of the trabeculae in lumbar vertebrae and their relation to the bone mineral content. Spine 6: 375–380
45 Hansson T, Roos B, Nachemson A (1980) The bone mineral content and ultimate compressive strength of lumbar vertebrae. Spine 5: 46–55
46 Hansson T, Sandström J, Roos B, Jonson R, Andersson GBJ (1985) The bone mineral content of the lumbar spine in patients with chronic low back pain. (Accepted for publication, Spine)
47 Hindle TH (1972) Comparison of carisoprodol, butabarbital, and placebo in treatment of the low back syndrome. Calif Med 117: 7–11
48 Hirsch C, Nachemson A (1963) The reliability of lumbar disk surgery. Clin Orthop 29: 189–195
49 Hoehler FK, Tobis JS, Buerger AA (1981) Spinal manipulation for low back pain. JAMA 245: 1835–1838
50 Holm S, Maroudas A, Urban JPG, Selstam G, Nachemson A (1982) Nutrition of the intervertebral disc: Effect of fluid flow on solute transport. Clin Orthop 170: 296–302
51 Holm S, Nachemson A (1982) Nutritional changes in the canine intervertebral disc after spinal fusion. Clin Orthop 169: 243–258

52 Holm S, Nachemson A (1983) Variations in the nutrition of canine intervertebral disc induced by motion. Spine 8: 866–874
53 Holm S, Nachemson A (1984) Nutrition of the intervertribel disc: acute effects of cigarette smoking. An experimental animal study. Int J Micro 3: 406
54 Horal J (1969) The clinical appearance of low back pain disorders in the city of Gothenburg, Sweden. Thesis. Acta Orthop Scand Suppl 118
55 Irstam L (1974) Adverse effects of water-soluble contrast media in lumbar myelography. Thesis, University of Gothenburg
56 Irstam L (1978) Lumbar myelography with amipaque. Spine 3: 70–82
57 Johansson F, Almay BGL, von Knorring L, Terenius L (1980) Preditors for the outcome of treatment with high frequency transcutaneous electric nerve stimulation in patients with chronic pain. Pain 9: 55–61
58 Jokl J (1982) Muscle and low back pain. IN: White AA, Gordon SL (eds) Symposium on Idiopathic low back pain. CV Mosby, St Louis, pp 9–22
59 Järvinen M (1976) Healing of a crush injury in rat striated muscle. With special reference to treatment by early mobilization or immobilization (Thesis). Turku, Finland
60 Kadzioka R, Asztely M, Hanai K, Hansson T, Nachemson A (1981) Ultrasonic measurement of the lumbar spinal canal. The origin and precision of the recorded ehcoes. J Bone Joint Surg 63-B: 504–507
61 Kelsey J, Githens PB, O'Conner T, Weil U, Calogero JA, Holford TR, White A III, Walter SD, Ostfeld AM, Soutwich WO (1984) Acute prolapsed lumbar intervertebral disc: An epidemiologic study with special reference to driving automobiles and cigarette smoking. Spine 9: 608–613
62 Kelsey, JL, Githens PB, White AA III, Holford TR, Walter SD, O'Connor T, Ostfeld AM, Weil U, Southwick WO Calogero JA (1984) An epidemiologic study of lifting and twisting on the job and risk for acute prolapsed lumbar intervertebral disc. J Orthop Res 2: 61–67
63 Kelsey JL, Pastides H, Bisbee GE Jr, White AA (1978) Musculo-skeletal disorders: Their frequency of occurrence and their impact on the population of the United States. Prodist New York
64 Kelsey JL, White A III (1980) Epidemiology and impact of low-back pain. Spine 5: 133–142
65 Keyserling WM (1982) Strength testing as a method of evaluating ability to perform strenous work. In: Stanton-Hicks M, Boas R (eds) Chronic back pain. Raven Press, New York pp 149–155
66 Kirkaldy-Willis WH (1984) The relationship of structural pathology to the nerve root. Spine 9: 49–52
67 Kirkaldy-Willis WH, Farfan H (1982) Instability of the lumbar spine. Clin Orthop 165: 110–123
68 LaRocca H, MacNab I (1974) The laminectomy membrane: Studies in its evolution, characteristics effects and prophylaxis in dogs. J Bone Joint Surg 56-B: 545–550
69 Leavitt F, Garron DC, Whisler WW, D'Angelo CM (1980) A comparison of patients treated by chymopapain and laminectomy for low back pain using a multidimensional pain scale. Clin Orthop 146: 136–143
70 Leyshon A, Kirwan E, Wynn Parry CB (1981) Electrical studies in the diagnosis of compression of the lumbar root. J Bone Joint Surg 63-B: 71–75
71 Lidström A, Zachrisson M (1970) Physical therapy on low back pain and sciatica. An attempt at evaluation. Scand J Rehabil Med 2: 37–42
72 Lind GAM (1974) Auto-traction, treatment of low back pain and sciatica. Thesis. University of Linköping, Linköping, Sweden
73 Lorenz M, Patwardhan A, Vanderby R Jr (1983) Loard-Bearing characteristics of lumbar facets in normal and surgically altered spinal segments. Spine 8: 122–130
74 MacNab I (1971) The traction spur. An indicator of segmental instability. J Bone Joint Surg 53-A: 663–670
75 Magora A (1969) An investigation of the problem of sick-leave in the patient suffering from low back pain. Industr Med Surg 38: 398–408
76 Magora A (1973) Investigation of the relation between low back pain and occupation. 4. Physical requirements: Bending, rotation, reaching and sudden maximal efforts. Scand J Rehabil Med 5: 186–190
77 Magora A, Schwartz A (1980) Relation between low back pain and x-ray changes. 4. Lysis and olisthesis. Scand J Rehabil Med 12: 47–52
78 McCulloch JA (1980) Chemonucleolysis: Experience with 2000 cases. Clin Orthop 146: 128–135
79 McNeill T, Warwich D, Andersson GBJ, Schultz A (1980) Trunk strengths in attempted flexion, extension and lateral bending in healthy subjects and patients with low-back disorders. Spine 5: 529–538
80 Millon R, Hall W, Haavik Nilsen K, Baker RD, Jayson MIV (1982) Assessment of the progress of the back pain patient. Spine 7: 204–212
81 Mooney V, Robertson J (1976) The facet syndrome. Clin Orthop 115: 149–156
82 Murphy RW (1977) Nerve roots and spinal nerves in degenerative disc disease. Clin Orthop 129: 46–56
83 NIOSH Technical Report: Work Practices guide for manual lifting. DHHS (NIOSH) Publ. No 81–122. U.S. Department of Health and Human Services. U.S. Government Printing Office, Washington D.C. March 1981
84 Nachemson A (1966) The load on lumbar discs in different positions of the body. Clin Orthop 45: 107–122
85 Nachemson A (1976) The lumbar spine – An orthopaedic challenge. Spine 1: 59–71
86 Nachemson A (1979) A critical look at the treatment for low back pain. Scand J Rehabil Med 11: 143–147
87 Nachemson A (1981) Disc pressure measurements. Spine 6: 93–97
88 Nachemson A (1982) Low back pain including biomechanics of the intervertebral joint complex. In: Straub R and Wilson P Jr (eds) Clinical Trends in Orthopaedics. Thieme-Stratton New York, pp 296–303
89 Nachemson A (1982) The natural course of low back pain. In: White AA, Gordon SL (eds): Symposium on Idiopathic Low Back Pain. CV Mosby, St. Louis pp 46–51
90 Nachemson A (1983) Work for all. For those with low back pain as well. Clin Orthop 179: 77–85
91 Nachemson A (1984) Lumbar spine instability. A critical update and symposium summary. Spine (in press)
92 Nachemson A and Elfström G (1970) Intravital dynamic pressure measurements in lumbar discs. A study of common movements, manoeuvres and exercises. Scand J Rehabil Med Suppl 1 2: 1–40
93 Nachemson A, Schultz AB, Andersson GBJ (1983) Mechanical effectiveness studies of lumbar spine orthoses. Scand J Rehab Med Suppl 9: 139–149
94 Panjabi MM, Takata K, Goel VK (1983) Kinematics of lumbar intervertebral foramen. Spine 8, 4: 348–357
95 Penning L, Blickman JR (1980) Instability in lumbar spondylolisthesis: A radiologic study of several concepts. AJR 134: 293–301

96 Pheasant HC, Dyck P (1982) Failed lumbar disc surgery: Cause, assessment, treatment. Clin Orthop 164: 93–109
97 Porter W (1980) Measurement of the spinal canal by diagnostic ultrasound. In: Jayson MIV (ed) The lumbar spine and back pain, 2nd edn. Pitman Medical Limited, London, pp 231–245
98 Porter RW, Hibbert C, Evans C (1984) The natural history of root entrapment syndrome. Spine 9: 418–421
99 Posner I, White AA, Edwards WT, Hayes WC (1982) A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine. Spine 7: 374:389
100 Puig MM, Laorden ML, Miralles FS, Olaso MJ (1982) Endorphin levels in cerebrospinal fluid of patients with postoperative and chronic pain. Anesthesiology 57: 1–4
101 Ramani PS (1976) Variation in size of the bony lumbar canal in patients with prolapse of lumbar intervertebral discs. Clin Radiol 27: 301–307
102 Roland M, Morris R (1983) A study of the natural history of low back pain. Development of guidelines for trials of treatment in primary care. Spine 8: 145–150
103 Rothman RH, Booth R (1975) Failures of spinal fusion. Orthop Clin North Am 6: 299–304
104 Rubak JM, Poussa M, Ritsilä V (1982) Effects of joint motion on the repair of articular cartilage with free periosteal grafts. Acta Orthop Scand 53: 187–191
105 Rydevik B, Brown M, Lundborg G (1984) Pathoanatomy and pathophysiology of nerve root compression. Spine 9, 1: 7–15
106 Salter RB (1982) Regeneration of articular cartilage through continuous passive motion-Past, present and future. In: Straub R, Wilson P Jr (eds): Clinical Trends in Orthopaedics. Thieme-Stratton, New York, pp 101–107
107 Schultz A, Andersson GBJ, Örtengren R, Haderspeck K, Nachemson A (1982) Loads on the lumbar spine. J Bone Joint Surg 64A: 713–720
108 Scott V, Gijsbers K (1981) Pain perception in competitive swimmers. Br Med J 283: 91–93
109 Seligman JV, Gertzbein SD, Tile M, Kapasouri A (1984) Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 9: 566–573
110 Seres JL, Newman RI (1976) Results of treatment of chronic low back pain at the Portland Pain Centre. J Neurosurg 45: 32–36
111 Shirazi-Adl S, Shrivastava SC, Ahmed A (1984) Stress analysis of the lumbar disc-body unit in compression. A three-dimensional nonlinear finite element study. Spine 9, 2: 120–133
112 Shyu BC, Andersson SA, Thorén P (1982) Endorphin mediated increase in pain threshold induced by long-lasting exercise in rats. Life Sci 30: 833–840
113 Sims WH, Jayson MIV, Young SM, Baddeley H, Collins E (1978) Controlled trial of mibilisation and manipulation for patients with low back pain in general practice. Br Med J. 2: 1338–1340
114 Sjukfranvaron i Sverige (1981) HS 90-projektet-hälso- och sjukvård inför 90-talet. Socialstyrelsen redovisar 6
115 Smith L, Brown J (1967) Treatment of intervertebral disc lesions by direct injection of chymopapain. J Bone Joint Surg 49-B: 502–519
116 Snoek W, Weber H, Jörgensen B (1977) Double-blind evaluation of extra-dural methyl prednisolone for herniated lumbar disc. Acta Orthop Scand 48: 635–641
117 Snook SH (1982) Low back pain in industry. In: White AA, Gordon SL (eds) Symposium on idiopathic low back pain. CV Mosby, St Louis, pp 23–38
118 Sorenson KH (1964) Scheuermann's juvenile kyphosis. Clinical appearances, radiography, aetiology, and prognosis. Munksgaard, Copenhagen
119 Spangfort EV (1972) The lumbar disc herniation. A computeraided analysis of 2.504 operations. Acta Orthop Scand Suppl 142
120 Spengler DM (1982) Low back pain. Assessment and management. Grune & Stratton, New York
121 Spengler DM, Freeman C, Westbrook R, Miller J (1980) Lowback pain following lumbar spine procedures. Failure of initial selection? Spine 5: 356–360
122 Sternbach RA (1977) Psychological aspects of chronic pain. Clin Orthop 129: 150–155
123 Stovall King J (1977) Randomized trial of the Rees and Shealy methods for the treatment of low back pain. In: Buerger AA, Tobis JS (eds) Charles C Thomas, Springfield Ill, pp 70–83
124 Surin V (1977) Duration of disability following lumbar disc surgery. Acta Orthop Scand 48: 466–471
125 Svensson HO (1981) Low back pain in forty to fortyseven year old men: A retrospective cross-sectional study. Thesis, University of Göteborg, Sweden
126 Terenius L (1981) Endorphins and pain. Front Horm Res 8: 162–195
127 Terenius L, Tamsen A (1982) Endorphins and the modulation of acute pain. Acta Anaesthesiol Scand Suppl 74: 21–24
128 Tile M (1984) The role of surgery in nerve root compression. Spine 9, 1: 57–64
129 Troup JDG (1984) The prediction of recurrence following episodes of back and sciatic pain. Spine (in press)
130 Troup JDG, Martin JW, Lloyd DCEF (1981) Back pain in industry: A prospective survey. Spine 6: 61–69
131 Urban JPG, Holm S, Maroudas A, Nachemson A (1977) Nutrition of the intervertebral disc. An in vivo study of solute transport. Clin Orthop 129: 101–114
132 Valkenburg H, Haanen HCM (1982) The epidemiology of low back pain. In: White AA, Gordon SL (eds) Symposium on Idiopathic low back pain. CV Mosby, St Louis, pp 9–22
133 Videman T (1982) Experimental osteoarthritis in the rabbit: Comparison of different periods of repeated immobilization. Acta Orthop Scand 53: 339–347
134 Waddell G, Kummel EG McCulloch JA (1979) Failed lumbar disc surgery and repeat surgery following industrial injuries. J Bone Joint Surg 61A: 201–207
135 Waddel G, Main C (1984) Assessment of severity in low-back disorders. Spine 9: 204–208
136 Waddell G, Main C, Morris EW, Di Paola M, Gray ICM (1984) Chronic low-back pain, psychologic distress, and illnes behaviour. Spine 9: 209–213
137 Waddel G, McCulloch JA, Kummel E, Wenner RM (1980) Nonorganic physical signs in low back pain. 1979 Volvo Award in clinical science. Spine 5: 117–125
138 Weber H (1978) Lumbar disc herniation. Part I. J Oslo City Hosp 28: 33–64
139 Weber H (1978) Lumbar disc herniation. Part II. J Oslo City Hosp 28: 89–124
140 Weber H (1983) Lumbar disc herniation. A controlled, prospective study with ten years of observation. Spine 2: 131–140
141 Weber H, Ljunggren AE, Walker L (1984) Traction therapy in patients with herniated lumbar intervertebral discs. J Oslo City Hosp 34: 61–70

142 Westrin CG (1973) Low back sick-listing. A nosological and medical insurance investigation. Scand J Soc Med Suppl 7: 1–116
143 White AA, Panjabi MM (1978) Clinical biomechanics of the spine. JB Lippincott, Philadelphia
144 Wiesel SW, Bernini P, Rothman R, Feffer H (1982) Effectiveness of epidural steroids in the treatment of sciatica – A double blind clinical trial. The International society for the study of the lumbar spine, Toronto, Canada
145 Wiesel SW, Rothman RH (1980) Acute low back pain. An objective analysis of conservative therapy. Spine 5: 324–330
146 Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N (1984) A study of computer-assisted tomography. I. The incidence of positive cat scans in an asymptomatic group of patients. Spine 9: 549–551
147 Woo SL-Y, Gelberman RH, Cobb NG, Amiel D, Lothringer K, Akeson WH (1981) The importance of controlled passive mobilization on flexor tendon healing. A biomechanical study. Acta Orthop Scand 52: 615–622
148 Wood PHN, Badley M (1980) Epidemiology of back pain. In: Jayson M (ed) The lumbar spine and back pain. Pitman, London, pp 29–55
149 Working conditions and sickness absenteeism 1975/76. Official statistics of Sweden. Living conditions. Report No 15, 1979
150 Wyke B (1980) The neurology of low back pain. In: Jayson MIV (ed) The Lumbar Spine and Back Pain, 2nd edn. Pitman Medical, London, pp 265–339
151 Wynn Parry CB (1980) Pain in avulsion lesions of the brachial plexus. Pain 9: 41–53
152 Yang KH, King AI (1984) Mechanism of facet load transmission as a hypothesis for low-back pain. Spine 9: 557–565
153 Young RJ (1979) The effect of regular exercise on cognitive functioning and personality. Br J Sports Med 3: 110–117
154 Young-Hing K, Reilly J, de Korompay V, Kirkaldy-Willis WH (1980) Prevention of nerve root adhesions after laminectomy. Spine 5: 59–64
155 Zachrisson Forsell M (1981) The back school. Spine 6: 104–106
156 Zylbergold RS, Piper MC (1981) Comparative analysis of physical therapy treatments. Arch Phys Med Rehab 62: 176–179
157 Aberg J (1984) Evaluation of advanced back pain rehabilitation program. Spine 9: 317–318