Newest Knowledge of Low Back Pain

A Critical Look

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Scientific scrutiny of the low back problem demonstrates its socioeconomic importance in most industrialized societies. Natural history studies reveal that the prognosis for the low back pain patient is excellent; for those with sciatica and painful spondylolisthesis it is good. It is even relatively good for those older patients with symptoms of spinal stenosis. Although today there is a better understanding of pain, the pathomechanism of low back pain is unknown. However, for patients with sciatica, spondylolisthesis, and spinal stenosis, physicians are beginning to get a better perception of what causes the pain. Psychosocial factors, including insurance benefits, have been demonstrated to be more important than biomechanical workload not only for acute but also for chronic low back pain patients who are unable to work. Orthopedic surgeons must recognize this fact when contemplating operations for patients with ill-defined back syndromes. Rarely are diagnoses scientifically valid, nor is the effectiveness of surgery proven by acceptable clinical trials.

Although, as evidenced by the excellent contributions to this Symposium, there is an increased amount of basic and clinical scientific efforts in this field, the enormous low back pain (LBP) problem still awaits a solution.

This critical overview applies strict scientific scrutiny to the most common disorders causing LBP.

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The following questions are crucial to an analysis of the LBP problem. (1) What is the extent and socioeconomic impact of the problem? (2) What causes the problem? (3) What is its natural history? (4) Are diagnoses well defined and diagnostic tests accurate? (5) Is treatment proven effective?

In the context of this Symposium, the author will apply this framework of thinking to some of the more common and more clearly (albeit not conclusively) defined low back syndromes: sciatica due to disk hernia or root canal stenosis, spondylolisthesis, and spinal stenosis. The present author will also try to delineate current knowledge in the area of greatest concern, the LBP syndromes of unknown etiology, *i.e.*, idiopathic or mechanical LBP.

THE EXTENT OF THE PROBLEM

Epidemiologic studies from all over the world have demonstrated the enormous societal impact of low back disorders that cause worker absenteeism.[1,11,32,51,82,111,113] It is clear that most people suffer from occasional low back trouble at some time during their life, even though disabling LBP is, fortunately, much less common.[52,53,60] Absenteeism has, however, in some parts of the world reached epidemic proportions.[82,140] An international comparison has been made (Table 1).

It has been long suspected that mechanical factors of various types affect disability caused by LBP, yet definite cause–effects have not been identified.

TABLE 1. International Comparison Yearly Incidence of Disabling Low Back Pain

Country	Inhabitants (millions)	Back-Pain-Related Days of Sickness (million days/year)	Back Diagnoses (% of workforce sicklisted/year)	Average Number of Days of Back-Related Absence Per Patient Per Year	Level of Insurance Benefit
USA[32,45,110]	240	20	2	9	0–80%
Canada[1,16,71]	23	10	2	20	40–90%
Great Britain[13,137]	55	33	2	30	0–80%
West Germany[58,68,69]	61	16	4	10	100% (0–4 weeks)* 80% (5–8 weeks)* 60* (9 weeks)*
The Netherlands[121,140]	14	4	4	25	80%
Sweden					
1980[20]	8	7	3	25	90%
1983[94]	8	13	5	30	90%
1987[82]	8.5	28	8	40	100%

* Parentheses indicate duration of LBP episode.

In this Symposium, Pope and Hanson[92] expand the existing knowledge of the effects of vibration on LBP. A completely new development of the last ten years, however, is the importance of various psychosocial factors. Bigos *et al.*[11] address this problem in one of the largest and longest prospective studies in this field. As a matter of fact, most case control studies of cross-sectional design that have addressed the mechanical and psychosocial factors influencing LBP, including job satisfaction, have concluded that the latter play a more important role than the extensively studied, mechanical factors.[7,9,31,47,72,116,117,120,124,126,136,138] This applies in particular to those 5–10% of patients who are disabled for more than three months and who account for 75–90% of the costs.[98,113,115]

For chronic LBP syndromes, certain insurance factors seem to have some influence.[3,6,17,19,27,36,95,101] Particularly disabling are those systems where the patient, to receive remuneration, has to prove in an adversarial fashion that his back pain was caused by work.[47,50,138] These evaluations usually take a long time to resolve, during which time the patient adapts to the sick role responding with pain behavior[38,124–126] and becoming increasingly difficult to rehabilitate to a productive and active life.

There have been various calculations of the total societal cost of back pain, and in one particular study in Sweden, the author found that it amounted to approximately 10 billion dollars.[82] Figures in the United States vary from 20 to as much as 50 billion dollars.[46,111]

WHAT CAUSES LOW BACK PAIN?

During the last decade, research into basic neurophysiologic pain mechanisms has intensified.[28,128] Now known is how various centers in the brain-stem can be modulated by various psychologic influences and can alter the production of pain mediating chemical substances such as enkephalines, serotonin, *etc.* This of course means that now explainable is why a person interprets any ailment or irritation as more painful when he or she is tired, discontented, or depressed. There has also been a recent breakthrough in genetic research where, at least in rats, the gene for a special type of pain sensitivity has been found.[129]

No evidence has been found of free naked nerve endings inside the vertebral disk, but

there is an abundance of new studies demonstrating their existence in the outer part of the annulus fibrosis, in the dorsal longitudinal ligament, and in the facet joint capsules.[15,82,139]

One of the more intriguing findings, one which may be genetically governed, is that the various pools of nerve cells in the dorsal columns can be hypersensitized and thus can signal a painful condition even though there is very little peripheral input. This explains why the response to any painful stimulus in the periphery is interpreted as extremely intense and also why researchers cannot rely on a positive pain response from diskographic analysis.[74,81,133] It also explains why patients treated with surgery for chronic LBP practically always have painful neuromas over the bone graft harvesting site at the iliac crest.[70] When bone graft is taken in the same manner in subjects with a nonpainful deformity like scoliosis or spondylolisthesis, no such painful scars ensue.[21,42,106]

The pathomechanism of pain in patients with spondylolisthesis is not really clear. Far from all patients with this condition have severe pain,[39,43,102] but instability, at least as demonstrated by traction–compression roentgenograms, seems to link the otherwise nebulous diagnosis of instability to a painful state.[44] In patients with sciatica caused by disk herniation, new knowledge about ganglion pain as well as other mechanisms, delineated by several contributors to this Symposium (Olmarker *et al.*[90] and Jayson[63]), has been acquired. These findings have been supported in experimental studies as well as clinical patient material.

In spinal stenosis, venous obstruction caused by mechanical compression seems to play a role, even though the direct pain mediator is unknown.[64,104]

Unfortunately, however, for the majority of patients, physicians do not know what specific structures in and around the motion segment are the source of pain. Table 2 summarizes the present knowledge of the causes of back pain, current research endeavors, natural history, and prognosis if left untreated.

The commonly used diagnosis of a facet syndrome for some low back patients must be questioned, as described by Jackson[61] in this issue. No one can say that the facets cannot be the source of pain, but diagnostic capabilities for testing the presence of a facet syndrome have been proven invalid.[18,61,88] The same thing goes for sacroiliac joint "dysfunction"[93,127] and other commonly used diagnostic labels. Degenerative disk disease is a nonvalidated diagnosis. The specificity, as judged from all available roentgenographic studies,[2,10,23,48,73,83] is extremely low. Even when strict radiologic criteria are adhered to, "disk degeneration" is demonstrated with equal incidence in subjects with or without pain.

The diagnosis of isolated disk resorption[22] is entertained by some surgeons, again without any tests or studies to validate the diagnosis, and unfortunately the same goes for segmental instability. Recent studies[65,108] using the Selvik stereophotogrammetric method and other newly developed means described in this Symposium by Pope *et al.*[91] may help us in the future. There is also some proof that high degrees of instability coincide with increased amounts of disabling pain.[44,67]

NATURAL HISTORY KNOWLEDGE

Spondylolisthesis

Researchers now have a fair knowledge of the natural history of at least young spondylolisthesis patients. There have been several studies published which demonstrate that, contrary to our previous beliefs, the risk for progression is small, even in the young girl (traditionally at greater risk) who come to us with the diagnosis.[39,40,41,43,102,103,107] The overall risk of progression only amounts to 10%. In some studies, this risk was more prevalent in boys.[39,40] Saraste[102] demonstrated that patients with spondylolisthesis have an increased incidence of LBP attacks, but the attacks are not necessarily chronically disabling. This has been confirmed by others.[43,96]

TABLE 2. Current Knowledge of Pathogenesis and Natural History of Some Painful Disorders of the Low Back

	Pathogenesis	*Current Research*	*Natural History*	*Prognosis Untreated*
Spondylolisthesis[41,43,96,102,103,107,135]	Inadequate	Genetics Biomechanics Stereophotogrammetric analysis	Relatively good	Excellent for progression Good for low back pain Fair for leg pain
Sciatica[5,54–56,63,79,132]	Relatively good	Neuropathology Neurophysical Ganglion pain	Good	Good
Spinal stenosis[65,80,90,97,104]	Good	Neuropathology Neurophysiology Nerve biomechanics CT, MR image area measurement	Fair	Relatively good
Low back pain, nonspecific[5,8,20,39,46,53,60]	Poor	Biochemistry, pathology, imaging biomechanics, psychology	Excellent	Good

CT, computed tomography; MR, magnetic resonance imaging.

The most significant finding of the long-term follow-up studies, however, is that the patient suffers significantly more sciatica than the population at large.

Sciatica

The natural history of sciatica caused by disk herniation has long been established, and the studies by Weber,[130,131] Hakelius[54] and others[100,132] demonstrate that the long-term results are the same whether the patient is treated with surgery. The recovery rate, however, is not as rapid as for patients with idiopathic LBP.[5,20,56]

Stenosis

Spinal stenosis has not been well investigated. However, the contribution by Johnson *et al.*[64] to this Symposium demonstrates that there does not seem to be a progression of symptoms with time; thus, again, the natural history seems to be somewhat more favorable than previously thought.

Idiopathic LBP

The natural history of idiopathic LBP is well known, and the prognosis is excellent; 90% of patients return to work within six weeks.[20,45] We have also in many studies[1,45,110,113] delineated that it is the last 4% or 5% of low back sufferers who are disabled for three months or more who account for 70–80% of the societal costs for the disease. For those patients with LBP and leg pain (not necessarily caused by a disk hernia), the recovery is less rapid.[5,45]

Longer follow-up periods for larger cohorts of patients have demonstrated that there is a very high recurrence rate 50–60%.[82,118,119] However, these tendencies for recurrence seem to taper off after three to five years.

ARE DIAGNOSES WELL DEFINED AND MEASUREMENTS ACCURATE?

A diagnosis is a clearly defined clinical state that is either present or absent in a given individual. To ascertain if patients have a certain diagnosis, physicians subject them to different tests that have been defined as a clinical intervention. Their primary purpose is to enhance a physician's knowledge of the patient's condition. The various tests we employ have different levels of *accuracy.* When performing these tests, the accuracy is calculated from the *sensitivity,* which is the proportion of individuals with the condition whose tests turn up positive, and the *specificity,* which is the proportion of individuals without the condition whose test turns up negative.[12] Take the example of disk hernia diagnosis. Various tests have been performed in patients with appropriate symptoms as well as in normal volunteers. Myelography,[59] computed tomography,[134] and magnetic resonance imaging[14] have all been used and have demonstrated that, in patients with appropriate symptoms, 90–98% show disk hernia. In normal volunteers without known symptoms, 28–35% show the same findings. This means that the sensitivity of these tests is good, whereas the specificity is only around 70%.

The specificity of these tests for spinal stenosis is even lower.[14,134] The diagnostic accuracy of lumbar diskography is unproven.[81,133] Actually, despite its frequent use in some parts of the world, there has been few attempts even to evaluate its efficacy. In the two published studies, the accuracy was found to be poor.[34,61]

The roentgenographic test used to diagnose spondylolisthesis is accurate at least for slips exceeding Meyerding's Grade 1 (>25%).[76] Unfortunately, the most likely cause of pain in patients with spondylolisthesis, segmental instability, is very difficult to diagnose. Recent studies clearly demonstrate that measurements from ordinary clinical roentgenograms are inaccurate up to ±20% of distances measured.[24,25,109] Such errors also apply to measurement of disk height, a frequently used sign of disk degeneration, where it has been demonstrated that errors are even higher (±50%).[4,23] The accuracy of

measurements of forward sliding as well as translation in flexion–extension roentgenograms are inherently poor, also discussed in depth by Pope *et al.*[91] in this Symposium.

IS TREATMENT PROVEN EFFECTIVE?

Even though in many instances physicians do not know the exact cause of the patient's LBP, for some categories there exist treatment modalities that are of proven effectiveness either in reducing pain or disability or both.[82,85]

The strongest and only conclusive evidence of effectiveness is the randomized, controlled trial,[114] which in the ordinary clinical situation is difficult to perform. All consecutive patients with a certain condition or defined pain in the same location and for the same length of time are randomly allocated to different types of treatment. These studies may be single blinded; that is, the patient does not know what treatment was received. It can be double blind, when neither the patient nor the outcome assessor knows, or it can even be triple blind where neither patient, therapist, nor assessor knows the treatment allocation.

Unfortunately, most of the follow-up studies that have been published in the medical literature on treatment of back pain are inherently poor[12] and probably have contributed to leading researchers erroneously to believe effectiveness when no such thing has been proven. When reading the clinical back literature, only those studies that are prospective and randomized should be read. Sadly, most of the studies are just case reports that describe a group of patients treated in a certain way. Such studies, however, only demonstrate that the phenomenon exists. The level of proof of effectiveness is zero.

There are case–control series that analyze the treatment received by patients who show different outcomes. They suggest conclusions but only if the study is good. The same is true for the cohort studies that compare outcomes, prospectively or retrospectively, of patients receiving different treatments but where no random allocation was performed. Such studies carry a low level of proof of effectiveness.

The quality of these latter studies depends greatly on whether they are prospective rather than retrospective. Patient selection must be clearly defined, with no mixture of various nonobjective diagnosis and a sufficient number of patients must be studied. Proper objective evaluations should also be performed before and after treatment. There is a vast literature on how to objectively evaluate LBP patients.[12,29,84,87]

In these case–control and cohort studies, the follow-up evaluation must be nonbiased, *i.e.*, never performed by the treating physician. It must be performed with the same evaluation forms as were used before the treatment. A sufficient follow-up period must elapse before evaluation; for surgical cases, the follow-up period must be no less than two years, and the number of patients observed must exceed 80%. Even so, a worst-case analysis must be performed on the patients not observed.[12] The bare minimum is to establish from the objective pretreatment evaluations that no significant differences exist between those not followed and those available for examination.

The level of proof of treatment effectiveness is high for good prospective case–control and cohort studies with a sufficient percentage of cases followed by a nonbiased observer. The only conclusive evidence of treatment effectiveness, again, is a good prospective randomized trial. During the last decade, this has repeatedly been mentioned in the orthopedic literature.[84,99]

TREATMENT

In the wake of clearly validated diagnostic labels for all the sufferers of LBP, it seems pertinent to use temporal delineation, *i.e.*, confine the diagnosis to LBP of unknown origin lasting for up to one week, from one week to six weeks, from seven weeks to three months, and more than three months. In this

TABLE 3. Level of Proof of Effectiveness of Various Common Treatment Modalities for Low Back Pain of Unspecified Origin

	Duration of LBP Episode			
	<7 days	*7 days–6 weeks*	*7 weeks–3 months*	*>3 months*
Bedrest < 2 days[29,49]	Conclusive	Conclusive	Low	Low
Bedrest > 7 days[82,85]	None	None	None	Negative
Medication: Paracetamol NSAID[87,87]	Conclusive	Conclusive	Conclusive	Low
Manipulation[75,82,87]	Low	High	Low	None
Back school[82,83,87]	Low	High	Low	Negative
Heat/cold[87,128]	Low	None	None	Low
General fitness exercise (supervised)[33,37,82,85]	None	Low	Conclusive	Low
Facet injection[61,74,88]	na	Negative	Negative	Negative
Stretching[33,82]	None	None	None	None
Traction[82,87]	None	None	None	None
Surgery/any type[32,35,36,82,87]	na	None	None	None

na, not applicable.

context, LBP can be defined as pain between the lower ribs and the gluteal folds, with minimal radiation to the thigh and never below the knee, whereas LBP and sciatica is defined when there is back pain together with radiating pain in the leg below the knee.

There have been a few serious attempts[82,87] in the literature during the last five years to evaluate what is known about treatment effectiveness. In the following tables, the level of proof is delineated with regard to the various LBP syndromes, divided temporally as mentioned above (Table 3), spondylolisthesis (Table 4), and sciatica caused by disk herniation and root canal stenosis and spinal stenosis (Table 5). The various supporting references have also been tabulated. For the subacute industrial LBP patient, general cardiovascular activation exercises, supervised and supported by behavioral psychology methods, are of proven benefit.[38,87]

Sciatica, caused by so-called root canal ste-

TABLE 4. Level of Proof of Effectiveness of Various Common Treatment Modalities for Spondylolisthesis With Disabling Symptoms Lasting Longer than Two Months

	Age			
	<25 years		*>25 years*	
	Slip			
Modality	*<50%*	*>50%*	*<50%*	*>50%*
Bedrest[40,41,102,105]	Low	Low	Low	Low
Rest from strenuous activities[40,102]	Low	Low	Low	None
Brace[40,105]	Low	Low	High	High
Posterolateral fusion *in situ*[42,105]	Low	Low	High	Low
Circumferential fusion *in situ*[42,106]	Low	High	Low	Low
Reduction and fusion	None	None	None	None

TABLE 5. Level of Proof of Effectiveness of Various Common Treatment Modalities for Low Back Pain and Leg Pain Radiating Below the Knee, Moderate Neurologic Signs, and Disabling Pain

	Disk Herniation Demonstrated			*Root Canal Stenosis Suspected Positive Nerve Root Block*	*Spinal Stenosis in Dural Sac Area (<60 mm)*
	<4 weeks	*5 weeks–3 months*	*>3 months*		
Bedrest for 1 week[82,87]	High	High	None	Low	None
Bedrest > 2 weeks[82]	None	None	Negative	None	Negative
Bicycling exercise[104]	None	None	Low	Low	High
Brace[82,104]	Low	Low	Low	High	High
Traction[82,87,132]	Low	Negative	Negative	None	None
Medication[82,87]	Conclusive	Conclusive	Low	Low	Low
Epidural steroidinj[82,87]	Low	Low	Low	Low	Low
*Chemonucleolysis[81,82,86,89]	Low	Conclusive	Low	Negative	Negative
Percentage discect[66]	Low	Low	Low	None	Negative
Laminotomy and removal of hernia or offending structure[65,122,130–132]	Low	Conclusive	High	High	High (if disk immobile)
Laminectomy removal and fusion[35,57,65,85]	None	Low	Low	Low	Conclusive (if disk mobile)

* Surgery superior to chemonucleolysis in several randomized studies.

nosis, has not been well delineated, and neither the natural history nor the measurements of its existence or treatment effectiveness has really been scientifically evaluated. It is hoped, however, that magnetic resonance imaging will allow better studies to be performed in these patients exhibiting sciatica without visible disk herniation, as well as differentiate between a recurrent disk herniation and scar.[77] Ross and Modic[97] bring researchers up to date in this Symposium. It has been shown[122] that the prospective use of a nerve root block helps in securing a good clinical result from a small laminotomy operation. If a nerve root block was positive, surgical decompression by small interlaminar approach showed satisfactory results in 80% of patients versus 60% when a larger procedure was performed without prior use of a diagnostic nerve block.

Lumbar spinal fusion is a common procedure for patients with poorly defined diagnostic labels.[35] In this Symposium, Esses and Huber[35] have critically scrutinized its use and found very few if any proper indications for it. However, a recent report by Herkowitz and Kurz[57] demonstrated (in patients with spinal stenosis in a prospective, randomized trial with a two-year follow-up period and an unbiased observer) that fusion added to a one level decompression was significantly superior to decompression alone. This is the first prospective, randomized trial in the history of fusion for any back condition. Mooney,[78] however, in a double-blind study, demonstrated that electrical stimulation via external induction could enhance the healing of an attempted fusion. No functional outcome evaluation was performed for these patients.

Orthopedic surgeons sometimes try to help chronic LBP patients by using rather large operative intervention, not without considerable risks. It must be stressed that in the last decade, an increasing amount of studies[31,37,38,123–126] have demonstrated the importance of pain or illness behavior in these patients. It is imperative that such psychologic signs are not interpreted as physical problems. As described in Frymoyer's contribution on disability,[46] it has been demonstrated that the illness behavior component of a patient's disability is more important than the underlying presumed physical trouble. Clinical studies with good controls and nonbiased follow-up examinations have demonstrated that psychologic factors need be addressed more thoroughly than hypothetical physical problems or biomechanical factors.[82,112,123–126]

Physicians must remember that symptoms and signs are functions both of pathology and illness behavior.

Finally, it is becoming clear in this field that ill-conceived diagnostic behavior on the physician's part can lead to abnormal illness behavior in patients, and this, in turn, may lead to abnormal treatment behavior.

REFERENCES

1. Abenhaim, L., and Suissa, S.: Importance and economic burden of occupational back pain: A study of 2500 cases representative of Quebec. J. Occup. Med. 29:670, 1987.
2. Ad Hoc Committee on low back x-rays: Low back x-rays. Criteria for their use in placement examinations in industry. J. Occup. Med. 6:373, 1964.
3. Allan, D. B., and Waddell, G.: An historical perspective on low back pain and disability. Acta Orthop. Scand. 234[Suppl.]:1, 1989.
4. Andersson, G. B. J., Schultz, A., Nathan, A., and Irstam, L.: Roentgenographic measurement of lumbar intervertebral disc height. Spine 6:154, 1981.
5. Andersson, G. B. J., Svensson, H. O., Oden, A.: The intensity of work recovery in low back pain. Spine 8:880, 1983.
6. Beals, R. K.: Compensation and recovery from injury. West. J. Med. 140:233, 1984.
7. Bergenudd, H., and Nilsson, B.: Back pain in middle age: Occupational workload and psychologic factors. An epidemiologic survey. Spine 13:58, 1988.
8. Biering-Sorensen, F.: Low back trouble in a general population of 30, 40, 50, 60 year old men and women. Study design representativeness and basic results. Dan. Med. Bull. 29:289, 1982.
9. Bigos, S. J., Spengler, D. M., Martin, N. A., Zeh, J., Fisher, L., and Nachemson, A.: Back injuries in industry: A retrospective study. III. Employee-related factors. Spine 11:252, 1986.
10. Bigos, S. J., Hansson, T. H., Castillo, R. N., Beecher, P. J., and Wortley, V. M.: The value of pre-employment radiographs for predicting acute back injury claims and chronic back pain disability. Clin. Orthop. (In press.)

11. Bigos, S. J., Battié, M. C., Spengler, D. M., Fisher, L. D., Fordyce, W. E., Hansson, T., Nachemson, A. L., and Zeh, J.: A longitudinal, prospective study of industrial back injury reporting. Clin. Orthop. 279:21, 1992.
12. Bloch, R.: Methodology in clinical back pain trials. Spine 12:430, 1987.
13. Blow, R. J., Jayson, M. I. V.: Back pain. *In* Edwards, F. C., McCallum, P. I., Taylor, P. J. (eds.): Fitness for Work. The Medical Aspects, vol. 9. Oxford University Press, 1988, p. 142.
14. Boden, S. D., Davis, D. O., Dina, T. S., Patronas, N. J., and Wiesel, S. W.: Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. J. Bone Joint Surg. 72A:403, 1990.
15. Bogduk, N.: The innervation of intervertebral discs. *In* Ghosh P. (ed.): The Biology of the Intervertebral Disc, vol. 1. Boca Raton, CRC Press, 1988, pp. 135–150.
16. Bombardier, C., Baldwin, J. L., and Crull, L.: The epidemiology of regional musculoskeletal disorders: Canada. *In* Hadler, N. M., and Gillings, D. D. (eds.): Butterworth's International Medical Reviews: Rheumatology and Arthritis in Society: The Impact of Musculoskeletal Diseases. Kent, Butterworth and Company, 1985, pp. 21–29.
17. Brena, S. F., and Chapman, S. L.: Pain and litigation. *In* Wall, P. D., and Melzack, R. (eds.): Textbook of Pain. London, Churchill Livingstone, 1984, pp. 1032–1042.
18. Butler, D., Trafimow, J. H., Andersson, G. B. J., McNeill, T. W., and Huckman, M. S.: Discs degenerate before facets. Spine 15:111, 1990.
19. Carron, H., DeGood, D. F., and Tait, R.: A comparison of low back pain patients in the United States and New Zealand: Psychological and economic factors affecting severity of disability. Pain 21:77, 1985.
20. Chöler, U., Larsson, R., Nachemson, A., and Peterson, L. E.: Ont i ryggen—Försök med vardprogram för patienter med lumbala smärttillstand. Sjukvandens planerings och rationali serrugs institut. Stockholm, Report 188, pp. 1–148.
21. Cochran, T., Irstam, L., and Nachemson, A.: Long-term anatomic and functional changes in patients with adolescent idiopathic scoliosis treatment by Harrington rod fusion. Spine 8:576, 1983.
22. Crock, H. V.: Internal disc disruption: A challenge to disc prolapse fifty years on. Spine 11:650, 1986.
23. Dabbs, V. M., and Dabbs, L. G.: Correlation between disk height narrowing and low-back pain. Spine 15:1366, 1990.
24. Danielson, B., Frennered, K., and Irstam, L.: Roentgenologic assessment of spondylolisthesis. I. A study of measurement variations. Acta Radiol. 29:345, 1988.
25. Danielson, B., Frennered, K., Selvik, G., and Irstam, L.: Roentgenologic assessment of spondylolisthesis. II. An evaluation of progression. Acta Radiol. 30:65, 1989.
26. Danielsson, B. J., Frennered, A. K., Irstam, L. K. H.: Radiological progression of isthmic lumbar spondylolisthesis in young patients. Spine. (In press.)
27. Derebery, V. J., Tullis, W. H.: Delayed recovery in the patient with a work compensable injury. J. Occup. Med. 25:829, 1983.
28. Devor, M.: The pathophysiology of damaged peripheral nerves. *In* Wall, P. D., Melzack, R. (eds.): Textbook of Pain, Edinburgh London Melbourne and New York, Churchill Livingstone, 1989, pp. 61–63.
29. Deyo, R. A.: Measuring the functional status of patients with low back pain. Arch. Phys. Med. Rehabil. 69:1044, 1988.
30. Deyo, R. A., Diehl, A. K., and Rosenthal, M.: How many days of bed rest for acute low back pain? A randomized clinical trial. N. Engl. J. Med. 315:1064, 1986.
31. Deyo, R. A., and Diehl, A. K.: Psychosocial predictors of disability in patients with low back pain. J. Rheumatol. 15:1557, 1988.
32. Deyo, R. A., and Tsui-Wu, Y. J.: Descriptive epidemiology of low back pain and its related medical care in the United States. Spine 12:264, 1987.
33. Deyo, R. A., Walsh, N. E., Martin, D., Schoenfeld, L., and Ramamurthy, S.: A controlled trial of transcutaneous electrical nerve stimulation and stretching exercises for chronic low back pain. N. Engl. J. Med. 322:1627, 1990.
34. Esses, S. I., Botsford, D. J., and Kostuik, J. P.: The role of external spinal skeletal fixation in the assessment of low back disorders. Spine 14:594, 1989.
35. Esses, S. I., and Huler, R. J.: Indications for lumbar spine fusion in the adult. Clin. Orthop. 279:87, 1992.
36. Fager, C. A., and Friedberg, S. R.: Analysis of failures and poor results of lumbar spine surgery. Spine 5:87, 1980.
37. Fordyce, W. E.: Pain and suffering: A reappraisal. Am. Psychol. 43:276, 1988.
38. Fordyce, W. E., Brockway, J. A., Bergman, J. A., and Spengler, D.: Acute back pain: a control group comparison of behavioral versus traditional management methods. J. Behav. Med. 9:127, 1986.
39. Fredrickson, B. E., Baker, D., Mcholick, W. J., Yan, H. A., and Lubicky, J. P.: The natural history of spondylolysis and spondylolisthesis. J. Bone Joint Surg. 66A:699, 1984.
40. Frennered, K.: Symptomatic lumbar spondylolisthesis in young patients: A clinical and radiological follow-up after non-operative and operative treatment. University of Götenborg, 1991. Thesis.
41. Frennered, K., Danielson, B., and Nachemson, A.: Natural history of symptomatic isthmic low-grade spondylolisthesis in children and adolescents. A seven-year follow-up study. J. Pediat. Orthop. (In press.)
42. Frennered, K., Danielson, B., Nachemson, A., and Nordwall, A.: Mid-term follow-up of young patients fused in situ for spondylolisthesis. Spine 16:409, 1991.
43. Frennered, K.: Spondylolisthesis among patients receiving disability pension due to chronic low back pain. Spine. Submitted, 1991.
44. Friberg, O.: Lumbar instability: A dynamic approach by traction—Compression radiography. Spine 12:119, 1987.

45. Frymoyer, J. W.: Back pain and sciatica. N. Engl. J. Med. 318:291, 1988.
46. Frymoyer, J. W.: Predicting disability from low back pain. Clin. Orthop. 279:101, 1992.
47. Gallon, R. L.: Perception of disability in chronic back pain patients: A long-term follow-up. Pain 37:67, 1989.
48. Gibson, E. S.: The value of replacement screening radiography of the low back. Occupational Medicine: State of the Art Reviews 3:91, 1988.
49. Gilbert, J. R., Taylor, D. W., Hildebrand, A., and Evans, C.: Clinical trial of common treatments for low back pain in family practice. Br. Med. J. 291:791, 1985.
50. Greenough, C. G., and Fraser, R. D.: The effects of compensation on recovery from low back injury. Spine 14:947, 1989.
51. Hadler, N. M.: Illness in the workplace: The challenge of muskulo-skeletal symptoms. J. Hand. Surg. 10A:451, 1985.
52. Hadler, N. M.: Regional musculoskeletal diseases of the low back. Cumulative trauma versus single incident. Clin. Orthop. 221:33, 1987.
53. Hadler, N. M.: The predicament of backache. J. Occup. Med. 30:449, 1988.
54. Hakelius, A.: Prognosis in sciatica: A clinical follow-up of surgical and non-surgical treatment. Acta Orthop. Scand. 129[Suppl.]:1, 1970.
55. Hanley, E. N., and Shapiro, D. E.: The development of low back pain after excision of a lumbar disc. J. Bone Joint Surg. 71A:719, 1989.
56. Heliövaara, M.: Epidemiology of sciatica and herniated lumbar intervertebral disc. Publications of the Social Insurance Institution, Helsinki, Finland, 60:76, 1988.
57. Herkowitz, H. N., and Kurz, L. T.: Degenerative spondylolisthesis: Prospective study comparing decompression versus decompression and fusion. J. Bone Joint Surg. 73A:802, 1991.
58. Hettinger, T.: Statistics on diseases in the Federal Republic of Germany with particular reference to diseases of the skeletal system: Ergonomics 28:1720, 1985.
59. Hitselberger, W. E., and Witten, R. M.: Abnormal myelograms in asymptomatic patients. J. Neurosurg. 28:204, 1968.
60. Horal, J.: The clinical appearance of low back disorders in the city of Gothenburg, Sweden: Comparisons of incapacitated probands with matched controls. Acta Orthop. Scand. 118[Suppl.]:1, 1969.
61. Jackson, R. P.: The facet syndrome—Myth or reality? Clin. Orthop. 279:110, 1992.
62. Jackson, R. P., Becker, G. J., Jacobs, R. R., et al: The neuroradiographic diagnosis of lumbar herniated nucleus pulposus (I): A comparison of computed tomography (CT), myelography, CT-myelography, discography and CT discography. Spine 14:1356, 1989.
63. Jayson, M. I. V.: The role of vascular damage and fibrosis in the pathogenesis of nerve root damage. Clin. Orthop. 279:40, 1992.
64. Johnsson, K.-E., Rosen, I., and Uden, A.: The natural course of lumbar spinal stenosis. Clin. Orthop. 279:82, 1992.
65. Johnsson, R., Selvik, G., Strömqvist, B., and Sundén, G.: Mobility of the lower lumbar spine after posterolateral fusion determined by roentgen stereophotogrammetric analysis. Spine 15:347, 1990.
66. Kahanovitz, N., Viola, K., Goldstein, T., and Dawson, E.: A multicenter analysis of percutaneous discectomy. Spine 15:713, 1990.
67. Kälebo, P., Kadziolka, R., and Swärd, L.: Compression—traction radiography of lumbar segmental instability. Spine 15:351, 1990.
68. Krämer, J. S.: Personal communication, 1989.
69. Kügelgen, B., and Hillemacher, A.: Die lumbale Bandscheibenerkrankung in der ärztlichen Sprechstunde. Springer, Berlin, Heidelberg, New York, Tokyo, 1985.
70. Kurz, L. T., Garfin, S. R., and Booth, R. E., Jr.: Harvesting autogenous iliac bone grafts. Spine 14:1324, 1989.
71. Lee, P., Helewa, A., Smythe, H. A., et al: Epidemiology of musculoskeletal disorders (complaints) and related disability in Canada. J. Rheumatol. 12:1169, 1985.
72. Magora, A.: Investigation of the relation between low back pain and occupation. V. Psychological aspects. Scand. J. Rehabil. Med. 5:191, 1973.
73. Magora, A., and Schwartz, A.: Relation between low back pain and x-ray changes. Scand. J. Rehab. Med. 12:47, 1980.
74. Marks, R.: Distribution of pain provoked from lumbar facet joints and related structures during diagnostic spinal infiltration. Pain 39:37, 1989.
75. Meade, T. W., Dyer, S., Browne, W., Townsend, J., and Frank, A. O.: Low back pain of mechanical origin: randomized comparison of chiropractic and hospital outpatient treatment. Br. Med. J. 300:1431, 1990.
76. Meyerding, H. W.: Spondylolisthesis. Surg. Gynecol. Obstet. 54:371, 1932.
77. Modic, M. T., Pavlicek, W., Weinstein, M. A., Boumphrey, F., Ngo, F., Hardy, R., and Duchesneau, P. M.: Magnetic resonance imaging of intervertebral disk disease. Clinical and pulse sequence considerations. Radiology 152:103, 1984.
78. Mooney, V.: A randomized double-blind prospective study of the efficacy of pulsed electromagnetic fields for interbody lumbar fusions. Spine 15:713, 1990.
79. Nachemson, A. L., White, A. A., and Gordon, S. L., eds.: The natural course of low-back pain. Symposium on idiopathic low back pain. St. Louis, C. V. Mosby, 1982, pp. 46–51.
80. Nachemson, A.: Advances in low back pain. Clin. Orthop. 200:266, 1985.
81. Nachemson, A.: Editorial comment: Lumbar discography-Where are we today? Spine 14:555, 1989.
82. Nachemson, A. L.: Low back pain. Causes, diagnosis and treatment (In Swedish). The Swedish Council of Technology Assessment in Health Care. Stockholm, 1991.
83. Nachemson, A., and Bigos, S. J.: The low back. Adult Orthopaedics. Vol 2. Edited by J. Cruess, W. R. J. Rennie. New York, Churchill Livingstone, pp. 842–937, 1984.

84. Nachemson, A. L., and LaRocca, H.: Editorial: Spine 1987. Spine 12:427, 1987.
85. Nachemson, A., Eek, C., Peterson, L. E., Wallin, L., Ohlund, C., and Lindström, I.: Chronic low back disability can largely be prevented: A prospective randomized trial in industry. AAOS 56th Annual Meeting, Las Vegas, Feb. 9–14, 1989.
86. Nachemson, A. L., and Rydevik, B.: Chemonucleolysis for sciatica. A critical review. Acta Orthop. Scand. 59:56, 1988.
87. Nachemson, A. with Spitzer, W. O., et al: Scientific approach to the assessment and management of activity related spinal disorders. A monograph for clinicians. Report of the Quebec task force on spinal disorders. Spine 12(7S) [Suppl. 1]:S1, 1987.
88. Nash, T. P.: Facet joints—Intra-articular steroids or nerve block? The Pain Clinic 3:77, 1990.
89. Norton, W. L.: Chemonucleolysis versus surgical discectomy: Comparison of costs and results in workers compensation claimants. Spine 11:440, 1986.
90. Olmarker, K., and Rydevik, B.: Single version double level nerve root compression: An experimental study on the porcine cauda equina with analyses of nerve impulse conduction properties. Clin. Orthop. 279:35, 1992.
91. Pope, M. H., Frymoyer, J. W., and Krag, M. H.: Diagnosing instability. Clin. Orthop. 279:60, 1992.
92. Pope, M. H., and Hansson, T. H.: Vibration of the spine and low back pain. Clin. Orthop. 279:49, 1992.
93. Potter, N. A., and Rothstein, J. M.: Intertester reliability for selected clinical tests of the sacroiliac joint. Phys. Ther. 65:1671, 1985.
94. Riksförsäkringsverket. Den ersatta sjukfranvarons diagnoser, Statistisk rapport. Riksjörskäkrrugs Verbets Rapporter IS-R 1987 5:1, 1989.
95. Robertson, L. S., and Keeve, J. P.: Worker injuries: The effects of workers compensation and OSHA inspections. J. Health Polit. Policy Law 8:581, 1983.
96. Ronnemaa, V. L., and Osterman, K.: Low back pain in middle aged and elderly spondylolisthetic population. Abstract. ISSLS Annual meeting Boston, Massachusetts, June 13–17, 1990, 30.
97. Ross, J. S., and Modic, M. T.: Current assessment of spinal degenerative disease with magnetic resonance imaging. Clin. Orthop. 279:68, 1992.
98. Rossignol, M., Suissa, S., and Abenhaim, I.: Working disability due to occupational back pain: Three-year follow-up of 2,300 compensated workers in Quebec. J. Occup. Med. 30:502, 1988.
99. Rudicel, S., and Esdaile, J.: The randomized clinical trial in orthopaedics: Obligation or option? J. Bone Joint Surg. 67A:1284, 1985.
100. Saal, J. A., Saal, J. S., and Herzog, R. J.: The natural history of lumbar intervertebral disc extrusions treated non-operatively. Spine 15:683, 1990.
101. Sander, R. A., and Meyers, J. E.: The relationship of disability to compensation states in railroad workers. Spine 11:141, 1986.
102. Saraste, H.: Long term clinical and radiological followup of spondylosis and spondylolisthesis. J. Pediatr. Orthop. 7:631, 1987.
103. Saraste, H., Broström, L. A., and Aparisi, T.: Prognostic radiographic aspects of spondylolisthesis. Acta Radiol. 25:427, 1984.
104. Schonstrom, N.: The narrow lumbar spinal canal and the size of the cauda equina in man. University of Göteborg; 1988. Thesis.
105. Seitsalo, S., Österman, K., Poussa, M., and Laurent, L. E.: Spondylolisthesis in children under 12 years of age: Long term results of 56 patients treated conservatively or operatively. J. Pediatr. Orthop. 8:516, 1988.
106. Seitsalo, S., Österman, K., Hyvärinen, H., Schelnzka, D., and Poussa, M.: Severe spondylolisthesis in children and adolescents. A long term review of fusion in situ. J. Bone Joint Surg. 72B:259, 1990.
107. Seitsalo, S., Österman, K., Hyvärinen, H., Tallroth, K., Schlenzka, D., and Poussa, M.: Progression of the spondylolisthesis in children and adolescents. A long-term follow-up of 272 patients. Spine (In press.)
108. Selvik, G.: Roentgen stereophotogrammetry: A method for the study of the kinematics of the skeletal system. Acta Orthop. Scand. 60[Suppl.]:1, 1989.
109. Shaffer, W. O., Spratt, K. F., Weinstein, J., Lehmann, T. R., and Goel, V.: The consistency and accuracy of roentgenograms for measuring sagittal translation in lumbar vertebral motion segment: An experimental model. Spine 15:741, 1990.
110. Snook, S. H.: The costs of back pain in industry. Spine: State of the Art Reviews 2:1, 1987.
111. Snook, S. H., and Webster, B. S.: The cost of disability. Clin. Orthop. 221:77, 1987.
112. Spratt, K. F., Lahman, T. R., Weinstein, J. N., and Sayre, H. A.: A new approach to the low back physical examination: Behavioral assessment of mechanical signs. Spine 15:96, 1990.
113. Spengler, D. M., Bigos, S. J., Martin, N. A., Zeh, J., Fisher, L., and Nachemson, A.: Back injuries in industry: a retrospective study. I. Overview and cost analysis. Spine 11:241, 1986.
114. Spitzer, W. O.: Selected non-experimental methods: An Orientation. *In* Troidl, H., Spitzer, W. O., McPeek, B., Mulder, D. S., and McKneally, M. F. (eds.): Principles and Practice of Research: Strategies for Surgical Investigators. Springer-Verlag, Berlin, Heidelberg, New York, London, Paris, Tokyo, pp. 222–229, 1986.
115. Svensson, H. O.: Low back pain in forty to forty-seven year old men. II Socio-economic factors and previous sickness absence. Scand. J. Rehab. Med. 14:55, 1982.
116. Svensson, H. O., and Andersson, G.: The relationship of low back pain, work history, work environment, and stress: A retrospective cross-sectional study of 38- to 64-year old women. Spine 14:517, 1989.
117. Taylor, P. J.: Personal factors associated with sickness absence. A study of 194 men with contrasting sickness absence experience in a refinery population. Brit. J. Industr. Med. 25:106, 1968.
118. Troup, J. D. G., Martin, J. W., and Lloyd, D. C. E. F.: Back pain in industry: A prospective study. Spine 6:61, 1981.

119. Troup, J. D. G.: Causes, prediction and prevention of back pain at work. Scand. J. Work Environ. Health 10:419, 1984.
120. Troup, J. D. G., Foreman, T. K., Baxter, C. E., et al: The perception of back pain and the role of psychophysical tests of lifting capacity. Spine 12:645, 1987.
121. Valkenburg, H. A., and Haanen, H. C. M.: The epidemiology of low back pain. *In* White, A. A., III, and Gordon, S. L. (eds.): Symposium on Idiopathic Low Back Pain. St. Louis, C. V. Mosby Company, 1982, pp. 9–22.
122. Van Akkerveeken, P. F.: Lateral stenosis of the lumbar spine. A new diagnostic test and its influence on management of patients with pain only. Rijksuniversiteit of Utrecht, The Netherlands, 1989. Thesis.
123. Waddell, G., Kummel, E. G., Lotto, W. N., et al: Failed lumbar disc surgery and repeat surgery following industrial injuries. J. Bone Joint Surg. 61A:201, 1979.
124. Waddell, G.: A new clinical model for the treatment of low back pain. Spine 12:632, 1987.
125. Waddell, G., Bircher, M., Finlayson, D., and Main, C. J.: Symptoms and signs: Physical disease or illness behaviour? Br. Med. J. 289:739, 1984.
126. Waddell, G., Main, C. J., and Morris, E. W., et al: Chronic low-back pain, psychologic distress, and illness behavior. Spine 9:209, 1984.
127. Walheim, G. G., and Selvik, G.: Mobility of the pubic symphysis. In vivo measurements with an electromechanic method and a roentgen stereophotogrammetric method. Clin. Orthop. 191:129, 1984.
128. Wall, P. D., and Melzack, R.: Textbook of pain. Edinburgh, Churchill Livingstone, 1989, pp. 1–1064.
129. Wall, P. D.: Editorial comments: A genetic factor in the reaction of rats to peripheral nerve injury. Pain 42:49, 1990.
130. Weber, H.: Lumbar disc herniation. A prospective study of prognostic factors including a controlled trial. J. Oslo City Hosp. 28:33, 1978.
131. Weber, H.: Lumbar disc herniation. Part II. J. Oslo City Hosp. 28:89, 1978.
132. Weber, H.: Lumbar disc herniation: a controlled prospective study with ten years of observation. Spine 8:131, 1983.
133. Weinstein, J., Claverie, W., and Gibson, S. S.: The pain of discography. Spine 13:1344, 1988.
134. Wiesel, S. W., Tsourmas, N., Feffer, H. L., Citrin, C. M., and Patronas, N.: A study of computer-assisted tomography. I. The incidence of positive CAT scans in asymptomatic group of patients. Spine 9:549, 1984.
135. Wiltse, L. L.: The etiology of spondylolisthesis. J. Bone Joint Surg. 44A:539, 1962.
136. Wolf, H. J., Greenwood, L., and Pearson, R. J. C.: Job satisfaction: A predictor of injury. NIOSH, USA. 1989.
137. Wood, P. H. N., and Bradley, E. M.: Epidemiology of back pain. *In* Jayson, M. I. V., III (ed.): The Lumbar Spine and Back Pain. London, Churchill Livingstone, 1987, pp. 1–15.
138. Woodyard, J. F.: Injury, compensation claims and prognosis. Part II. Journal of Social and Occupational Medicine 30:57, 1980.
139. Wyke, B. D.: The neurology of low back pain. *In* Jayson, M. I. V. (ed.): The Lumbar Spine and Back Pain, ed. 3. London, Churchill Livingstone, 1987, pp. 56–99.
140. Zuidema, H.: National statistics in the Netherlands. Ergonomics 28:3, 1985.