

Presidential Address, North American Spine Society: Failure of the Pathology Model to Predict Back Pain

SCOTT HALDEMAN, MD, PhD, FRCP (C)

IT IS ALWAYS a great honor and privilege to be able to serve as President of any learned society. This is particularly true of the North American Spine Society. I know of no other society as dynamic and progressive as NASS. The executive council is more dedicated than any I have served on and each committee is productive in developing reports and programs assigned to it. This is especially amazing as the Society is only 5 years old. There are numerous individuals who have worked hard for NASS. However, particular credit must be given to the leadership of the first four presidents. Drs. Wiltse and Kirkaldy-Willis must be considered the fathers of NASS. Their tremendous prestige, research experience, and understanding of the problems associated with the scientific and clinical investigation of the spine placed NASS immediately in the forefront of the spine societies. Drs. Nix and Mooney, with their extensive skills, organizational and political experience, and their dedication to a solid scientific interdisciplinary approach to problems related to the spine, took a fledgling society and elevated its meetings to a level equal to the most scientific of associations. At the same time, the various NASS committees have established programs to offer scholarships and awards, develop scientific studies, establish fellowship programs, and to begin discussion on terminology and practice guidelines. It is through programs like these that NASS has become a society in which its members may keep abreast of advancements in spinal research and the practice of specialist level management of spinal disorders.

One of the advantages of a presidential address is that one can raise issues and problems that are often expressed in vague terms and yet are fundamental to everything we do as spine clinicians and researchers. At a recent meeting, which included a number of past and present officers of the International Society for the Study of the Lumbar Spine and the North American Spine Society, a statement was placed on the blackboard and accepted by the group. It read, "We do not know the cause of back pain." This statement has recently been made at a number of meetings and presented in the scientific literature by some of the foremost leaders in the field. This raises the question, "Why don't we know the cause of back pain? Is there something wrong with our basic understanding of the spine, with our research, and with our current model of health and disease?"

THE EFFECTS OF RESEARCH ON BACK PAIN

The members of NASS and the other learned spine societies have shown tremendous dedication to research. A review of the papers presented at recent NASS meetings shows increasingly sophisticated and expensive methods of evaluating the structural and neurologic integrity of the spine. Such basic tools as myelography, computed tomography, and magnetic resonance imaging are being supplemented

by intravenous, intrathecal, intradiscal, and locally injected contrast media. Neurologic and functional testing has extended beyond electromyography and nerve conduction studies to include various forms of reflex, somatosensory, and cortical motor evoked responses and now includes multiple machines and tests to measure muscle strength, posture, and mobility. Our armamentarium of treatment protocols expands every year. Nonsurgical treatment methods including new medications, new manipulative techniques, new educational and exercise programs, and new physical modalities are presented at meetings. The sophistication of surgical approaches to spinal problems has increased with the development of multiple methods of performing discectomies and decompression as well as extensive, complicated, and complex methods of fusing vertebrae.

The question that must be asked is whether the proliferation of new technology and advanced clinical skills for the investigation and treatment of spinal pain has influenced the overall incidence, morbidity, cost, or disability related to spinal pain disorders. Unfortunately, the answer is not very encouraging. The overall lifetime prevalence of back pain remains at the 60–90% level, with an annual incidence of around 5%.^{20,54} There is no evidence that these figures are changing. Data from the National Center for Health Statistics show that the number of people disabled from back pain has increased by 168% in the United States between 1971 and 1986. This is 14 times faster than the population growth.^{49,50} Similarly, the cost of treatment and disability resulting from back pain is estimated to be reaching \$14–18 billion.⁹ The costs for occupational back pain alone has been estimated at \$6 billion.²¹

The importance of past advancements in technology and clinical skills cannot be ignored. Short-term, comparative, and sometimes double-blinded studies on the effectiveness of certain medications,^{1,57} manipulation,^{26,27} back school,^{29,38} and other conservative methods⁵² have demonstrated the ability of these techniques to relieve pain and reduce periods of disability in selected groups of patients. Certain intense rehabilitation programs may even have substantial ability to return specific groups of chronic debilitated patients to the workplace.^{41,42} None of these techniques, however, have been demonstrated to change the long-term course of patients with back pain. When conservative treatment approaches such as medication, physical therapy, or manipulation are discontinued, the natural process of this disease appears to progress, causing pain and disability.^{4,20,27} Long-term results from short periods of conservative treatment are inevitably no better than controls. In a similar manner, specific surgical procedures can achieve their anatomic and pathologic goal of discectomy or fusion and have been demonstrated to reduce pain and neurologic deficits in appropriately selected patients.^{35,46} However, as with conservative care, the long-term effects of surgery are not very encouraging. The only blinded controlled study of surgery⁵⁸ demonstrated the benefits of surgery in the first year. However, after 4 and 10 years, patients who had undergone surgery were no better than those who had not. Studies by Dvorak et al¹⁷ following postsurgical discectomy

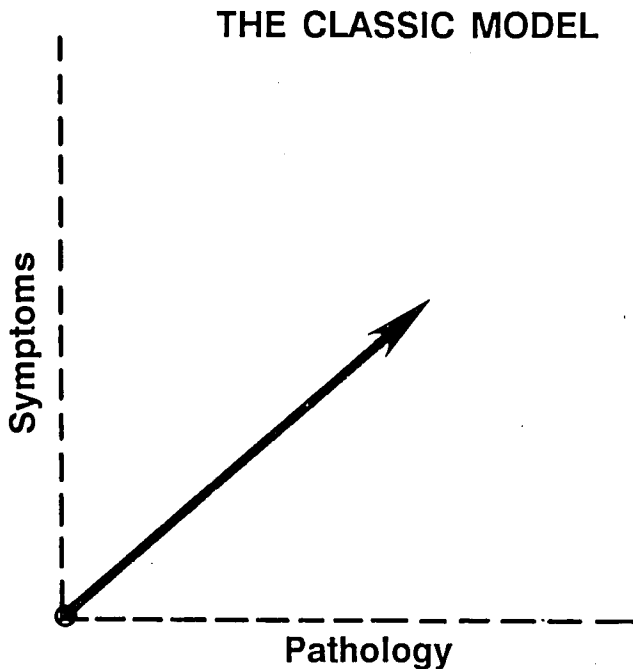


Fig 1. The classical pathology model relating increasing symptoms to increasing pathology.

patients for 4–17 years revealed that 70% still complained of back pain. Twenty-three percent complain of constant pain, and 35% were still under treatment. Lehmann et al⁴⁰ showed similar results with long-term follow-up of patients who had undergone fusion. In this case, 57% continued to have back pain in the last year, 53% were still using medication, and 15% required repeat surgery.

From these studies it is increasingly evident that a conservative or surgical cure for back pain is unlikely to be found. Although the capability of easing pain and suffering even temporarily and of reducing periods of disability cannot be diminished in importance, it is becoming essential that the larger picture of back pain in society be addressed. It is increasingly difficult for society to deal with a disease that absorbs up to 41% of the cost of Workers' Compensation, that incapacitates up to 20% of workers for periods of 3 weeks to 6 months, and that is estimated to cost society between \$14–18 billion per year.^{2,9,25}

THE CLASSICAL PATHOLOGY MODEL

A major problem in the development of diagnostic and treatment protocols for patients with back pain is an apparent difficulty in fitting this disorder into the classic model of health and disease. Classically, an organ free of demonstrable pathology is considered healthy and free of symptoms, while an organ with obvious pathology is supposed to be responsible for any reported symptoms. Figure 1 illustrates the linear relationship between pathology and symptomatology, which is predicted by this model of health and disease and is the basis for all the advanced imaging technology being used to detect pathology.

Unfortunately, for a large percentage of the population, this classical model breaks down. There is now ample evidence that virtually all but the most severe degenerative and discogenic changes in the cervical and lumbar spine can exist in asymptomatic individuals. Up to 95% of men and 70% of women show degenerative changes in the cervical spine by age 60–65.^{22,23} In addition, approximately 30% of asymptomatic individuals are reported to show abnormalities in the lumbar spine as measured by myelography, CT scanning, and MRI scanning.^{8,31,60} To complicate the matter further, there is a large percentage of symptom-

atic patients, sometimes with quite severe complaints of spinal pain, in whom testing fails to reveal a structural lesion.⁶¹ Nachemson⁴⁵ went so far as to state that only 20% of patients with acute back pain can be given a precise pathoanatomic diagnosis. In patients with chronic back pain, this breakdown in the relationship between symptomatology and demonstrable pathology becomes even more dramatic.²⁸ For example, Figure 2 presents the CT scan films of patients with virtually identical symptoms and clinical signs.

These facts require a modification of the classical pathology model. The patients described previously follow one of two different patterns. In the first, pathology can exist without symptoms (Figure 3). In the second, symptoms can occur without obvious pathology (Figure 4).

FACTORS INFLUENCING BACK PAIN

If one accepts the research described previously, two questions must be answered. "What makes a structurally normal spine symptomatic?" and "What makes a benign pathologically degenerated spine or disc herniation induce disability which may necessitate surgery?" If answers can be found to these questions it may be possible to prolong the positive results of conservative treatment and markedly improve the results of surgery. The possibility of reducing the incidence of back pain with its associated costs and disability makes the exercise worthwhile.

Fortunately, the last decade has seen a rapid increase in research and reported studies that address the natural history of spinal symptomatology and pathology. Biomechanical, physiological, and psychosocial factors that are associated with increased risk of back pain and disability have been the focus of such research. It is now possible to extract at least

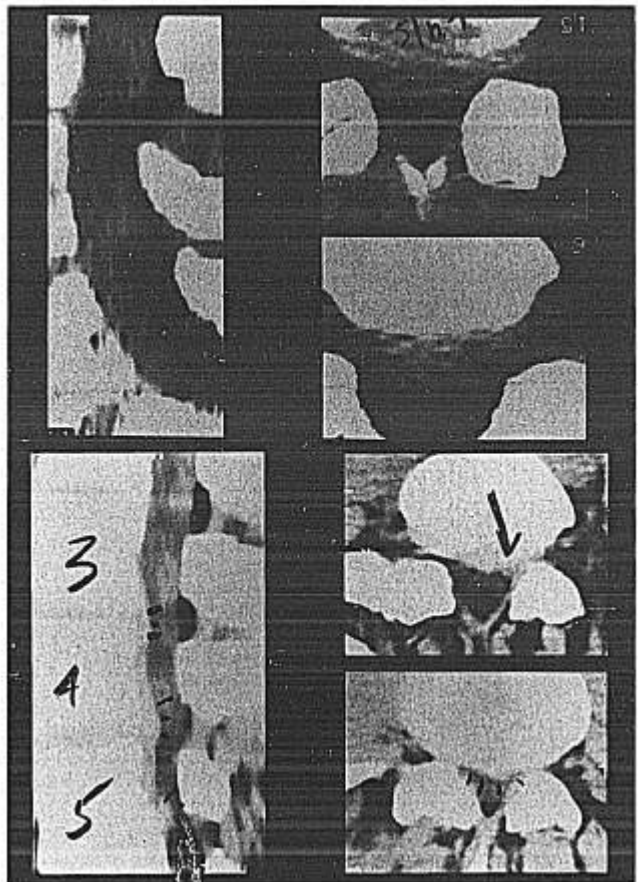


Fig 2. Examples of CT scans in two patients with chronic back pain and very similar symptoms and clinical examinations (From Halde-man et al, Spine 13:345, 1988).

MODEL OF PATHOLOGY WITHOUT SYMPTOMS

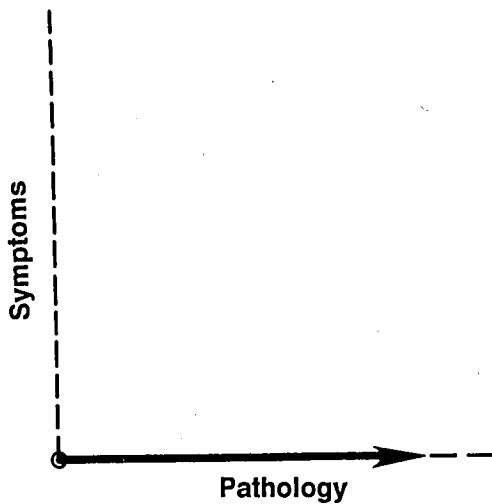


Fig 3. The model for asymptomatic pathology.

six major factors that appear to be important in the pathogenesis of back pain.

Aging

The natural effects of aging on the intervertebral discs has been well described.^{5,10,47} There is, first of all, a gradual decrease in the growth cartilage until maturity. The end-plate cartilage then undergoes calcification followed by resorption and replacement by bone. There is an increase in the collagen content and a decrease in the mucopolysaccharide content. These changes result in a reduction in the ability of the disc to receive nutrients that diffuse from the vascular structures in the bone. This process appears to occur in the entire population and in the absence of symptoms. Advanced degenerative changes have been noted in the asymptomatic population. Gore et al found dramatic changes with aging and the presence of even severe intervertebral narrowing, end-plate sclerosis, and osteophytic formation in asymptomatic patients over the

age of 40.^{22,23} However, it appears that there is some point when pathology becomes severe enough to cause stenosis beyond the capacity of the neural canals to adapt.^{19,44} The growth in sclerotic and hypertrophic bone in the end-plates and facets, in turn, leads to neural compression presumably at a level of decompensation of the degenerative aging process. Thus, the effect of natural aging can be represented as in Figure 5 where there is an initial increase in pathology without symptoms followed by a period when symptoms increase with pathology.

Acute Trauma

Acute trauma more than any factor should follow the classic pathology model. The effect of traumatic loads or impacts on the spine has been studied in some detail in a number of biomechanical laboratories. There is a growing understanding of the forces that are necessary to cause vertebral fractures as well as disc disruption and herniation with its well-documented clinical picture.^{20,39} There is also evidence that trauma can result in inflammatory changes in the disc and perhaps other tissues. This has led to the concept of intrinsic disc disruption as well as soft tissue sprains and strains.^{14,20} Such trauma potentially has the ability to cause pathology in a healthy spine and presumably can aggravate existing pathology. In addition, since the majority of patients with acute spine injuries recover spontaneously, the pathology caused by trauma has the ability to heal, perhaps with some remaining partially symptomatic or asymptomatic pathology or weakness. Figure 6 presents the three different potential effects of trauma on the relationship of trauma on pathology and symptoms.

Mechanical Occupational Stresses

An increasing amount of information is now available on the influence of certain occupational factors on spinal pathology and symptoms. Epidemiologic studies have revealed a number of mechanical occupational stressors that can increase the risk of developing back pain.^{18,36,62} The primary stressors appear to be the frequency and magnitude of lifting tasks, patterns of asymmetric postural demands, continuous sitting, and vibration. These factors all appear to increase the likelihood of developing pain. In addition, there is growing evidence that they can influence the structure of a disc and cause increased degeneration. These stressors have been demonstrated to

MODEL OF SYMPTOMS WITHOUT PATHOLOGY

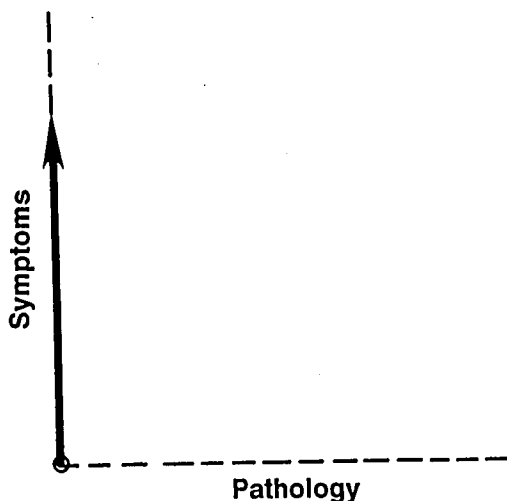


Fig 4. The model for symptomatic patients without documented pathology.

AGING

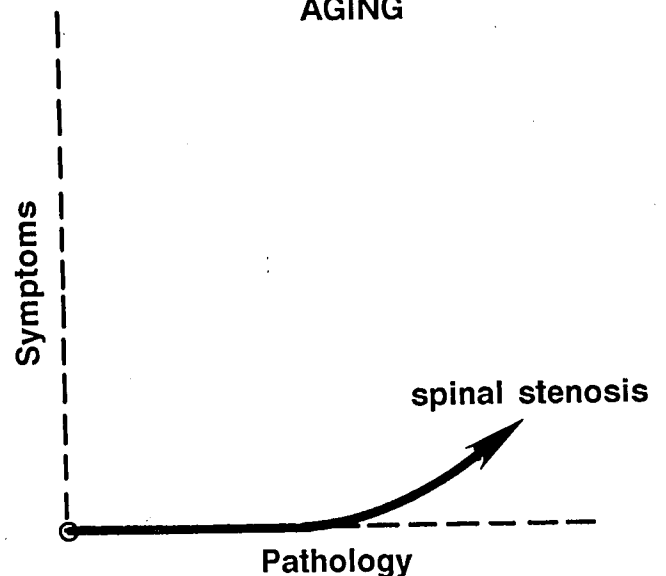


Fig 5. The model for the aging process.

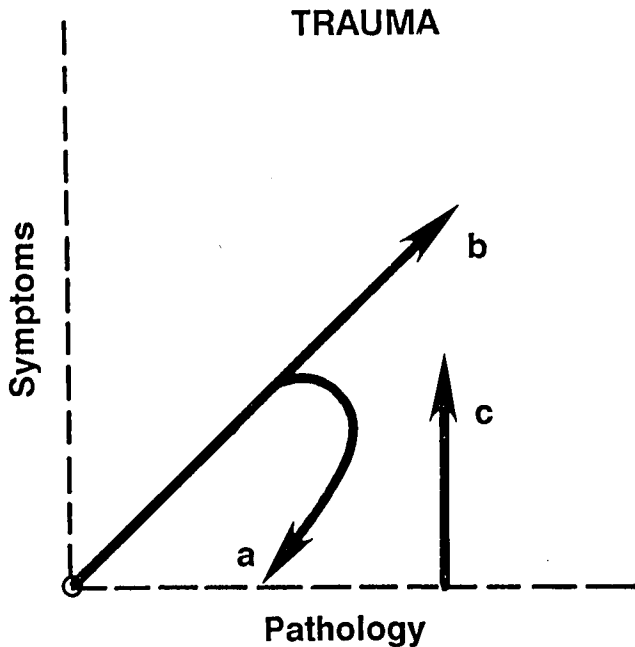


Fig 6. The model for acute trauma.

increase intradiscal pressure, to strain discal and possibly other fibers by torque or compression, and to cause mechanical softening and fatigue due to cyclic loading. This can occur in asymptomatic individuals¹⁸ and eventually result in symptomatic disc herniation.⁶² The influence of vibration on the nervous system, particularly the dorsal root ganglion, also may contribute to the increased likelihood of this group of patients developing pain.⁵⁹ Unlike acute trauma, the assumption is that this continuous trauma results in structural changes in the disc or other tissues and that it continues until these changes become symptomatic. Figure 7 presents the manner in which this process fits into the pathology model.

General Health

The relationship of general health to the incidence of back pain and disability is becoming increasingly clear. An important indicator for recurrence or persistence of back pain is the general health of the patient.^{3,6} Such factors as pain in the stomach, feelings of fatigue, previous hospitalizations or surgery, and daily smoking all are associated with increased incidence and severity of back pain. The relationship of cardiovascular diseases to low-back pain has been noted by Svenson et al,⁵⁵ whereas Holm has proposed an explanation for the mechanism whereby diabetes might induce degenerative changes in the intervertebral disc by influencing its nutrition. Deyo and Burn¹⁶ showed increased incidence of back pain in individuals who were obese. Of increasing importance is the relationship of smoking to back pain and disability. The effect of smoking on the diffusion of nutrients into the disc has been studied. The relationship between the prevalence of back pain producing injuries with subsequent disability and smoking has been confirmed by a number of investigators.^{6,16,37,45} Figure 8 illustrates how poor health can fit into the pathology model by increasing the nonsymptomatic degenerative changes associated with aging or by increasing the disability associated with trauma.

Exercise

Physical fitness and strength does not guarantee the absence of back pain or injury.¹¹⁻¹³ Nonetheless, there is increasing evidence that patients with back pain generally have reduced cardiovascular fitness,

MECHANICAL OCCUPATIONAL STRESSORS

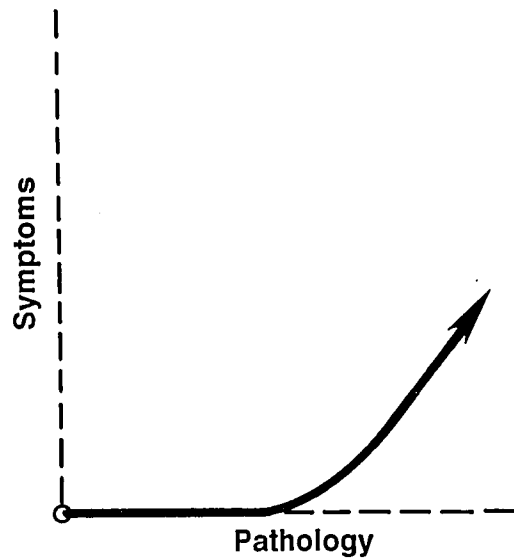


Fig 7. The model for continuous occupational stresses

muscle strength, and mobility.³³ This is particularly true in patients with chronic back pain and postsurgical patients.³⁴ There is also growing evidence that individuals with sedentary occupations have increased degenerative changes in the lumbar spine. Mellin⁴³ noted changes in thoracolumbar mobility that correlated with chronic on recurrent back pain. However, physical activity has been noted by Porter et al⁴⁸ to strengthen both discs and vertebrae. There is now a convincing body of research that demonstrates that strengthening exercise together with improvement of cardiovascular fitness and general functional restoration can reduce disability and possibly pain in patients with chronic low-back pain.^{30,41,42} Figure 9 illustrates the effect of exercise on the pathology model. The feeling of well-being after exercise appears to have the capacity to reduce pain. At this time, it appears to be the only factor that can increase strength and therefore

POOR HEALTH

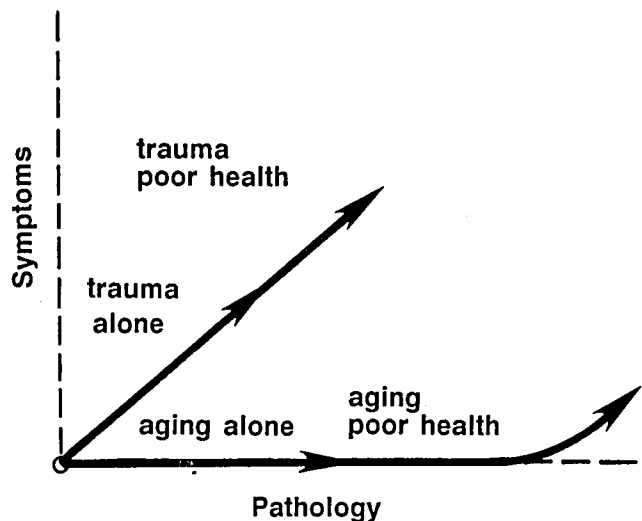


Fig 8. The model for the effect of general health on pathology and symptoms.

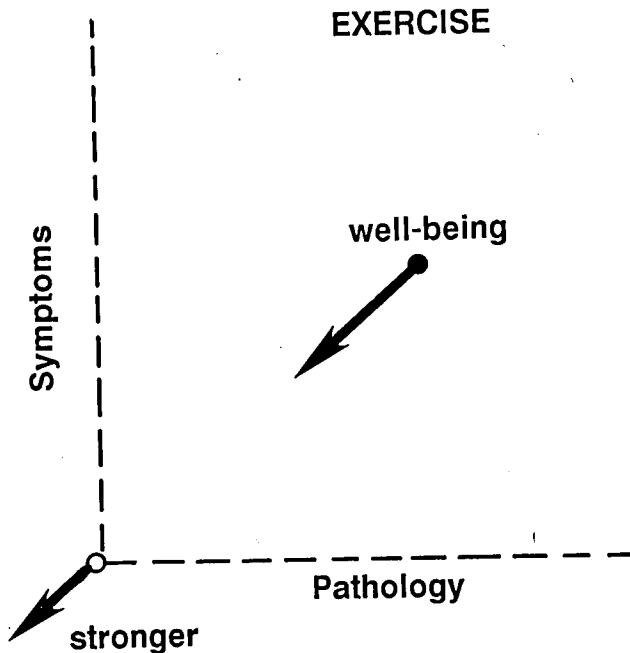


Fig 9. The model for the effect of exercise and fitness.

structural integrity of the spine. It also raises the interesting possibility that there may exist a state where a well-exercised healthy individual may be better able to resist the development of pathology and symptoms.

Psychosocial Factors

The close correlation between psychosocial factors and patients with chronic back pain is now conclusive, although the relative importance of various factors has yet to be worked out. An extensive multivariate analysis by Bigos et al⁷ demonstrated that physical measurements were

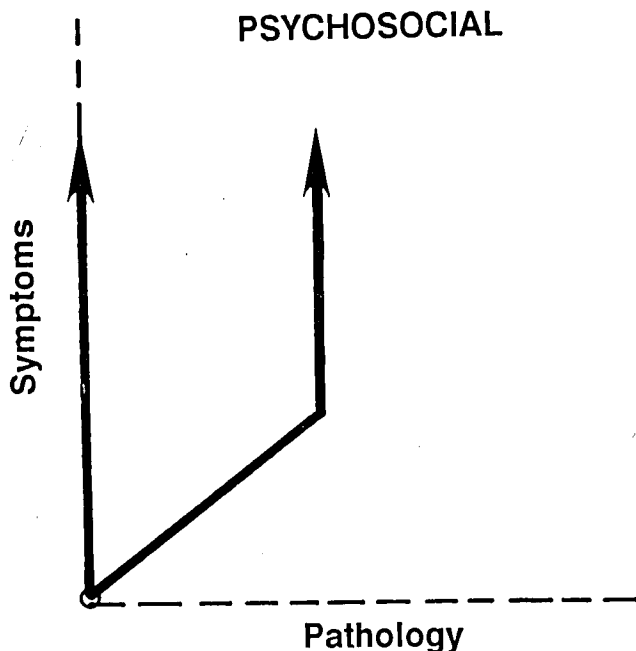


Fig 10. The model for the effect of psychosocial factors.

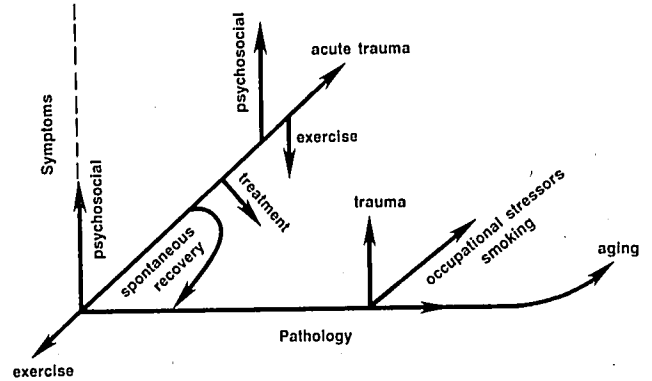


Fig 11. A composite of figures 3–10 for the pathology model. The relationship of symptomatology and pathology in an individual patient can follow any of the patterns noted in this figure.

much less important than psychosocial factors in predicting low-back injuries. Of particular interest was the observation that individuals who “hardly ever” enjoyed their work were at significantly greater risk of back complaints when compared with individuals who “almost always” enjoyed their work. Multiple other psychosocial factors such as litigation, Workers’ Compensation, loneliness, and coping skills have all been considered important.^{15,24,25,53} The observation that psychological testing before surgery can be used as a predictor of surgical outcome also illustrates that correction of pathology is not sufficient to reduce disability or symptomatology.^{51,56,63} Many individuals in detrimental psychosocial settings seem to develop long-term disabling symptoms in the absence of documented pathology. However, individuals with pathology in poor psychosocial settings appear to have enhanced pain and disability beyond that anticipated by the pathology. These processes are noted in Figure 10 on the pathology model.

THE PATHOLOGY MODEL AND BACK PAIN

Physicians and patients tend to feel most comfortable with a clear-cut relationship between pathology and symptomatology, between health and disease, and between cause and effect. Attempts are constantly being made in clinical practice to “fit” a particular patient’s symptoms to any demonstrated pathology seen on various tests.

Figure 11 illustrates how difficult this can be. This figure is a composite of the examples noted previously and illustrates the various ways in which pathology and symptomatology can interreact. The relationship of pathology to symptomatology is extremely complicated and the classic pathology model in its simplest form cannot explain back pain or disability. It is not possible to look at pathology and determine with any confidence the symptoms a patient may be suffering. It is also not possible, except in the most classic, unequivocal, and usually acute situations, to look at a patient with back pain but without neurologic deficits and determine the nature of the pathology that can be anticipated on testing.

The growing understanding of the intricate relationship of natural aging, acute injury, occupational stresses, general health, physical fitness, and psychosocial factors to spinal pathology and, more importantly, to spinal symptomatology and disability, holds the greatest promise of understanding disabling back pain.

We cannot stop developing more accurate diagnostic tools. We cannot let up on the search for more effective and less traumatic means of surgically treating vertebral pathology. We cannot give up on the further development and testing of conservative treatments such as medication, manipulation, exercise, and physical modalities that may reduce pain and suffering in our patients. We must, however, also

address the greater picture of how we can reduce the social and financial cost of this disease. Greater research effort must be made to look into the factors that influence the effectiveness of conservative and surgical treatment methods. Greater effort must be made to study the social, psychological, and industrial factors that are responsible for the most intractable and costly patients with back pain including methods for reversing disability in this group of patients. It will require a major effort on the part of industry, government, and the health care professions to impact the problem. Industry will have to modify the workplace to make it a less traumatic and more pleasant place to spend each day. Government will have to change tort and Workers' Compensation laws that encourage disability and expand its concept of health care payment to include all factors that contribute to spinal pain. The health care professions will have to maintain dedication to research, expand their conceptualization of back pain beyond the simple pathology model, and be willing to make changes in clinical skills and approaches to patients as the research develops. The North American Spine Society and the other learned spine societies will be expected to take the lead and ensure the improvement of spinal health in society. I have no doubt that NASS will live up to this expectation.

REFERENCES

- Amlie E, Weber H, Holme I: Treatment of acute low back pain with Piroxicam: Results of a double-blind placebo controlled trial. *Spine* 12:473-476, 1987
- Andersson GBJ: Epidemiologic aspects on low-back pain in industry. *Spine* 6:53-60, 1981
- Battie MC, Bigos SJ, Fisher LD, et al: A prospective study of the role of cardiovascular risk factors and fitness in industrial back pain complaints. *Spine* 14:141-147, 1989
- Bell GR, Rothman RH: The conservative treatment of sciatica. *Spine* 9:54-56, 1984
- Bernich S, Cailliet R: Vertebral end-plate changes with aging of human vertebrae. *Spine* 7:99-102, 1982
- Biering-Sorensen F, Thomsen C: Medical, social and occupational history as risk indicators for low-back trouble in a general population. *Spine* 11:720-725, 1986
- Bigos SJ, Battie M, Spengler D, et al: A longitudinal, prospective study of acute industrial back problems: The influence of physical and non-physical factors. Proceedings, International Society for the Study of the Lumbar Spine. Kyoto, Japan, 1989, p 19
- Boden SD, Davis DO, Vina TS, Patronas NJ, Wiesel SW: The incidence of abnormal lumbar spine MRI scans in asymptomatic patients. A prospective and blinded investigation. Proceedings, International Society for the Study of the Lumbar Spine. Kyoto, Japan, 1989, p 37
- Bonica JJ: Pain research and therapy: Past and current status and future needs. *Pain: Discomfort and Humanitarian Care*. Edited by L Ng, JJ Bonica. New York, Elsevier North, Holland, pp 1-46, 1980
- Brown MD, Tsaltas TT: Studies on the permeability of the intervertebral disc during skeletal maturation. *Spine* 1:240-244, 1976
- Cady LD, Bischoff DP, O'Connell ER, Thomas PC, Allan JH: Strength and fitness and subsequent back injuries in firefighters. *J Occup Med* 4:269-272, 1979
- Cady LD Jr., Thomas PC, Karwasky FJ: Program for increasing health and physical fitness of firefighters. *J Occup Med* 27:110-114, 1985
- Chaffin DB, Herrin GD, Keyserling WM: Preemployment strength testing: An updated position. *J Occup Med* 20:403-408, 1978
- Crock HV: Isolated disc disruption: A challenge to disc prolapse fifty years on. *Spine* 11:650-653, 1986
- Derebery VJ, Tullis WM: Delayed recovery in patients with a work compensable injury. *J Occup Med* 25:829-835, 1983
- Deyo RA, Bass JE: Lifestyle and low back pain. The influence of smoking and obesity. *Spine* 14:501-506, 1989
- Dvorak J, Velach L, Fuhrmann P, Heim E: The outcome of surgery for lumbar disc herniation. I. A 4-17 year's follow-up with emphasis on somatic aspects. *Spine* 13:1418-1422, 1988
- Evans W, Jobe W, Seibert C: A cross-sectional prevalence study of lumbar disc degeneration in a working population. *Spine* 14:60-64, 1989
- Friedenberg ZB, Miller WT: Degenerative disc disease of the cervical spine: A comparative study of asymptomatic and symptomatic patients. *J Bone Joint Surg* 45A:1171-1178, 1963
- Frymoyer JW: Back pain and sciatica. *N Engl J Med* 318:291-300, 1988
- Frymoyer JW, Mooney V: Current concepts review: Occupational orthopaedics. *J Bone Joint Surg* 68A:469-473, 1986
- Gore DR, Sepic SB, Gardner GM: Roentgenographic findings of the cervical spine in asymptomatic people. *Spine* 11:521-524, 1986
- Gore DR, Sepic SB, Gardner GM, Murray MP: Neck pain: A long-term follow-up of 205 patients. *Spine* 12:1-5, 1987
- Greenough CG, Fraser RD: The effects of compensation on recovery from low back injury. *Spine* 16:947-955, 1989
- Haddad GH: Analysis of 2932 Workers' Compensation back injury cases. The impact on the cost to the system. *Spine* 12:765-769, 1987
- Hadler NM, Curtis P, Gillings DB, Stinnett S: A benefit of spinal manipulation as adjunctive therapy for acute low back pain: a stratified controlled trial. *Spine* 12:703-706, 1987
- Haldeman S: Spinal manipulative therapy: a status report. *Clin Orthop* 179:62-70, 1983
- Haldeman S, Shouka M, Robboy S: Computed tomography, electrodiagnostic and clinical findings in chronic Workers Compensation patients with back and leg pain. *Spine* 13:345-350, 1988
- Hall H, Icton JA: Back school: an overview with specific reference to the Canadian Back Education Units. *Clin Orthop* 179:10-17, 1983
- Hazard RG, Fenwick TW, Kalisch SM, et al: Functional restoration with behavioral support. A one-year prospective study of patients with chronic low-back pain. *Spine* 14:157-161, 1989
- Hitselberger WE, Witten RM: Abnormal myelograms in asymptomatic patients. *J Neurosurg* 28:204-206, 1968
- Holm S: Does diabetes induce degenerative processes in the lumbar intervertebral disc? Proceedings, International Society for the Study of the Lumbar Spine. Kyoto, Japan, 1989, p 8
- Jackson CP, Brown MD: Is there a role for exercise in the treatment of patients with low back pain? *Clin Orthop* 179:39-45, 1983
- Kahanovitz N, Viola K, Gallagher M: Long-term strength assessment of postoperative discectomy patients. *Spine* 14:402-403, 1989
- Kahanovitz N, Viola K, McCulloch J: Limited surgical discectomy and microdiscectomy: A clinical comparison. *Spine* 14:79-81, 1989
- Kelsey JL, Hardy RJ: Driving of motor vehicles as a risk factor for acute herniated lumbar intervertebral disc. *Am J Epidemiol* 102:63-73, 1975
- Kelsey JL, Githens PB, O'Conner TO, et al: Acute prolapsed lumbar intervertebral disc: An epidemiologic study with special reference to driving automobiles and cigarette smoking. *Spine* 6:608-613, 1984
- Klaber-Moffett JA, Chase SM, Portek BS, Ennis JR: A controlled, prospective study to evaluate the effectiveness of a back school in the relief of chronic low back pain. *Spine* 11:120-122, 1986
- Knutson B: Comparative value of electromyographic, myelographic, and clinical neurological examination in diagnosis of lumbar root compression syndrome. *Acta Orthop Scand Suppl* 49:1-135, 1966
- Lehmann TR, Spratt KF, Tozzi JE, Weinstein JN, Reinarz SJ, El-Khoury GY, Colby H: Long-term follow-up of lower lumbar fusion patients. *Spine* 12:87-104, 1987
- Mayer TG, Gatchel RJ, Kishino N, et al: Objective assessment of spine function following industrial injury: A prospective study with comparison group and one-year follow up. *Spine* 10:482-493, 1985
- Mayer TG, Gatchel RJ, Mayer H, et al: A prospective two-year study of functional restoration in industrial low back injury: An objective assessment procedure. *JAMA* 258:1763-1767, 1987
- Mellin G: Correlations of spinal mobility with degree of chronic low back pain after correction for age and anthropometric factors. *Spine* 12:464-467, 1987
- Mooney V: Presidential address. International Society for the Study of the Lumbar Spine. Dallas, 1986: Where is the pain coming from? *Spine* 12:754-759, 1987
- Nachemson AL: Advances in low back pain. *Clin Orthop* 200:266-278, 1985
- Nasca RJ: Rationale for spinal fusion in lumbar spine stenosis. *Spine* 14:451-454, 1989

47. Oda J, Tanaka H, Tsuzuki N: Intervertebral disc changes with aging of human cervical vertebra. From the neonate to the eighties. *Spine* 13:1205-1211, 1988
48. Porter RW, Adams MA, Hutton WC: Physical activity and the strength of the lumbar spine. *Spine* 14:201-203, 1989
49. Prevalence of selected impairments, United States, 1971, Hyattsville, Maryland, National Center for Health Statistics, 1975. DHHS Publication No. (PHS) 75-1526 (Series 10, No. 99)
50. Prevalence of selected impairments, United States, 1981, Hyattsville, Maryland, National Center for Health Statistics, 1986. DHHS Publication No. (PHS) 87-1587 (Series 10, No. 159)
51. Rausford AO, Cairns D, Mooney V: The pain drawing as an aid to the psychological evaluation of patients with low back pain. *Spine* 1:127-134, 1976
52. Saal JA, Saal JS: Non-operative treatment of herniated lumbar intervertebral disc with radiculopathy: an outcome study. *Spine* 14:431-437, 1989
53. Sander RA, Meyers JE: The relationship of disability to compensation status in railroad workers. *Spine* 11:141-143, 1986
54. Svensson HO, Andersson GBJ: Low back pain in 40 to 47-year-old men: work history and work environmental factors. *Spine* 8:272-276, 1983
55. Svensson HO, Verdin A, Wilhelmsson C, Andersson GBJ: Low back pain in relation to other diseases and cardiovascular risk factors. *Spine* 8:277-285, 1983
56. Waddell G, Morris EW, FiPaola MP, Birdie M, Finlayson D: A concept of illness tested as an improved basis for surgical decisions in low-back disorders. *Spine* 11:712-719, 1986
57. Ward NG: Tricyclic antidepressants for chronic low back pain: Mechanisms of action and predictors of response. *Spine* 11:661-665, 1986
58. Weber H: Lumbar disc herniation: a controlled prospective study with ten years of observation. *Spine* 8:131-140, 1983
59. Weinstein J: Mechanisms of spinal pain: the dorsal root ganglion and its role as a mediator of low-back pain. *Spine* 11:999-1001, 1986
60. Wiesel SW, Tsourmas N, Feffer HI, Citrin CM, Patronas N: A study of computer-assisted tomography. 1. The incidence of positive CAT scans in asymptomatic group of patients. *Spine* 9:549-551, 1984
61. Wiesel SW, Feffer HL, Borenstein DG: Evaluation and outcome of low back pain of unknown etiology. *Spine* 13:679-680, 1988
62. Wilder DG, Pope MH, Frymoyer JW: The biomechanism of lumbar disc herniation and the effect of overload and instability. *J Spinal Disorders* 1:16-32, 1988
63. Wiltse LL, Rocchio PD: Preoperative psychological tests as predictors of success of chemonucleolysis in the treatment of the low back syndrome. *J Bone Joint Surg (AM)*, 57A:478-483, 1975