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Scientific Review

Aspects of the failed back syndrome: role of litigation

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Objective: A review that attempts to identify the mechanism and causation of persistent or recurring low back pain.
Design: A personal assessment of clinical features with a selective review of the literature.
Results: Thirty to forty per cent of our population aged 10–65 years report that back trouble occurs on a monthly basis and in 1% to 8% this interferes with work. A definite patho-anatomical cause for the pain is demonstrable in only a minority. It can be deduced that psychosocial factors, including insurance benefits are of importance for this variation.
Conclusions: Neither non-operative nor surgical procedures have a major impact on the capacity for work in this substantial minority of backache sufferers. The main risk factors identified are: Wrong diagnosis, repeated medical certificates for sickness benefits, failed surgery, symptoms incongruous with signs or imaging, multiple spinal procedures, poor social support and poor motivation, psychological illness, clinical depression before or after injury or operation. Pending compensation and delays in settlement are important additional features in claimants for compensation. For patients with unproven diagnostic labels such as 'pain-behaviour', no evidence exists that any type of surgery is cost effective.
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Keywords: low back pain; backache; sciatica; lumbar disk; failed back; chronic pain syndrome

Introduction

Thirty to 40% of our population aged 10–65 years report that back trouble occurs on a monthly basis and in 1% to 8% this interferes with work. A definite patho-anatomical cause for the pain is demonstrable in only a minority. It can be deduced that psychosocial factors, including insurance benefits are of importance for this variation. Sweden, with 100% sickness benefits, has the highest disability rate.[1]

Patients with acute back pain, spontaneous or traumatic, complain of back pain, tenderness and restricted movements. Much less frequent are more severe injuries with fractures of the lumbar vertebrae, subluxations, and in a minority, a paraplegia may result from compression of the conus or cauda equina. Even in those with major injuries, the natural course is towards recovery with abatement of severe pain within a few days or weeks. Population studies indicate that of all patients with acute back and leg pain only 1% to 2% actually suffers from disk herniation and require surgery.[2] But, after surgery it is estimated that some degree of Failed Back Syndrome (FBS) is found in approximately 15% of patients.[3] By comparison, patients who undergo major spinal surgery for other reasons, eg for a tumour, start to walk within a week and are usually free of severe or disabling spinal pain within 1 month.

This paper addresses the issues of mechanism and causation in a substantial minority of patients, who display persistent or recurring low back problems of a severity that significantly impairs the quality of life and/or ability to work. Many have unsettled compensation claims, many follow accidents, neural-destructive or surgical techniques: a situation comparable to the chronic whiplash syndrome.[4,5]

Definition

'Disabling back pain and/or sciatica of apparently disabling severity despite extensive therapy'[6] is a reasonable but descriptive definition. Pain for more than 3 months is an arbitrary criterion used in some studies.

Non-operative treatment

After an acute episode, patients first are treated non-operatively, and only if this fails by various invasive or

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surgical procedures. In FBS, patients have apparently disabling symptoms despite long periods of rest, analgesia, tractions and physiotherapy.[7] Many have sought acupuncture, pain clinic therapies, osteopathy and 'alternative-medicine' treatments. Many have had more than one operation.

There are about 10% to 20% failures in non-litigation, hospital practice,[8–10] but the many patients in general practice who quickly recover spontaneously and, often without seeking continued treatment suggests that the overall failure rate in the community is probably less than 5%. It is the failures of medical treatment who are subjected to more aggressive treatments, including surgery. Thus, series of operated patients generally reflect either more severe pathology or intractable complainants, or both.

Operative treatment

There are sound indications for disk surgery, notably: (1) persistent back pain or sciatica that has failed to improve with skilled and adequate conservative treatment if (2) accompanied by definite tension signs (straight leg raising) and/or segmental nerve root deficit, usually (3) with imaging showing a significant disk herniation corresponding anatomically to the symptoms and signs.

The success rate is high (80%+) in uncomplicated patients without psychosocial issues and with good premorbid personalities. However, disappointing results occur. A well studied group consisted of failures and poor results in 160 patients after lumbar spinal surgery between 1980 and 1984; they were analyzed retrospectively.[11] A self-rated questionnaire carried out 12 months after operation revealed 20 (12.5%) poor results. These occurred most commonly after multiple operations, or decompression and fusion as compared to disk excision. The commonest cause was 'failure to recognise abnormal pain behaviour before operation'. Another large surgical series[12] showed a rate of 6% recurrences of 984 operated patients of which one-third developed during the first year after operation. An unsatisfactory outcome was found in 11%. 'The majority of patients with the 'failed-back syndrome' had pending legal or workers' compensation claims, or were at psychological risk for surgery.' This statement again focuses attention on the importance of litigation.

The most frequent causes of FBS[7,13] are bad selection of patients, disregard of personality and psychosocial factors, surgery at the wrong level, inadequate surgical techniques, recurrent disk herniation, and epidural fibrosis.

The wide range of treatments carried out testifies to the intractability of these chronic complaints and, sadly, to the futility of the treatments employed in many cases. Therapeutic enthusiasm should not be mistaken for proof of efficacy, nor for a pathological diagnosis.

Invasive treatments include: (1) epidural spinal cord stimulation,[14,15] (2) laminectomy, foraminotomy, fusion, and other approaches to removal or decompression of protruding disk fragments,[16,17]; (3) chirotherapy completed by rehabilitation of the 'active motion apparatus', chemonucleolysis or percutaneous nucleotomy[18] (4) percutaneous rhizolysis[19] (5) intraspinal therapy: methylprednisolone[14,20] or intrathecal morphine,[21] (6) facet joint injection and facet nerve block,[22] (7) lumbosacral distraction spondylodesis,[23] and (8) osteopathy, chiropractic manipulation, acupuncture, transcutaneous nerve stimulators etc.

The cost of some of these procedures can be salutary. For example, with adverse events and costs, estimates for intrathecal morphine given for 60 months are $82 893 (an average of $1382 per month).[21]

Clinical features and mechanisms

There are many types of back injury, with a spectrum varying from transient back sprains to severe anatomical disruption of the bony spine and its muscular-ligamentous supporting tissues.

The medical expert has to try to distinguish these several syndromes (see below) and a variety of differing mechanisms of the injury and symptomatology. Defining criteria[22] are important (Table 1).

Back sprain

A blow or a fall onto the back generally causes bruising of skin and soft tissues without injury to the disks. Pulling against a large load that gives way or slips from the grasp can cause the subject to fall backwards with a local soft tissue injury or low back sprain. Many instances of low back pain are due not to disk tears or extrusion but to the muscular, ligamentous, or facet joint stretching, bruising, or tearing that constitute a back sprain. Clinically the back is painful, tender and stiff for a few days, but spontaneous recovery is the rule within 1–3 weeks.

Table 1 Disk lesions: criteria[31]

1	Disk protrusion = local or asymmetric extension of the disk beyond the interspace, the base against the disk broader than the protruded material.
2	Extrusion (syn. prolapse or hernia) = more severe extension or sequestration of the disk beyond the inter space, the base against the disk narrower than the extruded material.
3	Minor radiological signs seldom related to symptoms:
	Bulge = circumferential symmetric extension of the disk beyond the interspace
	Schmorl's nodes = herniation of the disk into the vertebral-body end plate
	Annular defects = disruption of the outer fibrous ring of the disk
	Facet arthropathy = degenerative disease of the posterior articular processes of vertebrae.

There is generally no sciatica, and movement, but not usually coughing or sneezing, aggravates the pain. Older patients and those with previous backache take longer to recover; any pre-accident lumbar spondylotic complaints can be expected to continue, and frequently are minimised or suppressed by claimants, despite medical documentation.

Disk prolapse (syn extrusion or hernia)

A major mechanical strain or a direct blow on the spine will cause a fracture or subluxation before a disk ruptures. This is a result of the physical properties of the disk; made of pliable cartilage, encased in the elastic and fibrous tissues of the annulus, it is highly resistant. Thus, it is *prima facie* improbable that a back injury will cause disk rupture, and indeed most authorities go further in saying that a healthy disk does not rupture in response to injury.

We should consider the physical effects of a mechanical force applied to the spine. The spine is built like a crane with strong and large vertebral bodies, which act as the jib of the crane; the ligaments, and the muscles of the back act as the cables or tension members. The function of the ligaments anywhere in the body is to resist tension forces and to absorb strain energy thus protecting the adjacent joints from injury. If then a force is applied to the spine, energy is dissipated. And, if the compressive force is absorbed by the jib (the vertebral bodies), the tensile force is absorbed by the ligaments and back muscles. In the case of a massive flexion or extension injury it has been shown that ligaments will rupture or vertebral bodies will fracture causing an unstable spinal fracture disolocation. Only then is the disk vulnerable to injury, deprived of its usual protective mechanisms. But, with anything less than these severe forces, the disk is inviolate (Personal communication from Prof. RA Dickson).

If the cadaveric freshly excised lumbar spine is loaded in standard engineering testing machines, then subjected to compression loading to the point of 'failure', it produces vertebral bony fractures, irrespective of the rate of loading and the amount of applied load but never produces disk 'failure'.[24,25] If a spine is twisted to breaking point, an almost inconceivable physiological situation, it can produce damage to the annulus surrounding the disk but does not produce a disk protrusion or prolapse.[26,27] Indeed if a disk is incised with a scalpel right through the annulus into the soft nucleus, so that it could well be envisaged that the nuclear material would protrude (liked a slipped disk) and the spine is again loaded to 'failure', no nuclear protrusion or extrusion occurs. Instead the spine fractures again at bone level. Should a spine be flexed forward to 'failure' it does so by tearing the posterior ligamentous support of the spine or by fracturing the laminae, and again does not produce a slipped disk.

Clinical and biomechanical evidence therefore indicates quite clearly that disk protrusions or prolapse occur gradually, with the disk failing under cyclical loading with millions of activities of daily living contributing to this mechanical failure. In addition, disk protrusions or prolapses only occur in disks that are undergoing their natural, constitutional process of degeneration which occurs in everyone. Smeathers[28] has demonstrated that as normal people move about, more than 80% of the load is taken by the legs and only about 16% by the spine, and this is dealt with by the ligaments, muscles and vertebrae and not by the disks. The disks are therefore not shock absorbers but rather are a part of the spinal joint (mobile link), which in moving allows the ligaments and muscles to stretch and absorb the energy.

It is established in non-litigation practice that 75% to 80% of disk extrusions occur spontaneously or with minimal strains that are of daily occurrence in everyday, even sedentary activities. The cause is primarily a longstanding dehydration and vertical narrowing of the disk with a fragmentation or microscopical tear in the postero-lateral, less often the central, part of the annulus. A trivial or heavy lift may prove to be the last straw in precipitating a prolapse, but it is not the cause. A heavy weight is difficult to define, depending on the age, gender and physical fitness of the individual, the mechanisms of a lift may be important, eg, the backstrain is magnified if a lift is performed with knees straight. The picture characteristic of an acute disk herniation is of low back pain and often sciatica within 0–24 h after an acute mechanical strain. In acute disk herniation, the back and leg pain and positive straight leg-raising test usually appear within hours of the onset of symptoms.[29]

Car injury

The driver or passenger of a car struck usually from behind may sustain a jolt with an extension/flexion mechanism to his back. This can produce a short lived episode of backache for days, or up to 2 or 3 weeks, but only in severe cases where the damage to the car and car seat often hints at the force imparted, will there result a compression fracture of a vertebra. Major fractures and displacements of the lumbar spine are nearly always caused by a vertical force acting through the long axis of the spine. This may be applied from above when a heavy weight falls from a roof, or from below where there is a fall from a height onto the feet or buttocks. A flexion injury is the result. Minor fractures of the transverse process, or spinous process generally causes localised pain, worse on movement for 2–6 weeks. Ultimate restoration of normal function is the rule. Many such injuries cause little pain after the first fortnight and are discovered accidentally by a later X-ray, commonly performed for other reasons. A few patients continued to complain of backache after

abnormally heavy lifting, but most recover without further complaints.

Direct back injury

An example is the miner with a fall of stone onto his back. Only rarely will it precipitate a disk hernia, but it does produce severe local skin and soft tissue bruising. In more severe cases, fractures of the vertebral body, transverse process or spinous processes occur. Root compression does not usually complicate this injury since the nerve roots are shielded by the bony intervertebral foraminal canal and are anatomically distant.

A heavy weight falling on a man with back flexed, can produce a compression fracture of the vertebral body, most often T11, 12 or L1. Again, nerve root compression is not a complication unless there is gross displacement (slip) of the vertebral body, or an associated subluxation of facet joints, both of which break the smooth ring of the spinal canal and render the nerve roots liable to compression. With a slip (spondylolisthesis) of more than 5 mm, compression of one or more nerve roots occurs in some patients with consequent root pain and weakness of the relevant muscle(s) supplied. In the serious, large central compression that disrupts the cauda equina there is paraplegia with weakness and sensory loss in the perineum and both legs, loss of control of bladder, bowel and sexual functions.

Most patients involved in litigation have a fall, a jolt in a car collision, a sprain whilst pulling a weighty object, or are struck by a weight. Clinical symptoms are almost immediate, and may progress over 72 h. They do not develop *de novo* after 48 h, as is sometimes claimed. The ability to walk from the site of accident, the continuation of work, and the ability to drive home are telling features that reflect the degree and nature of the injury. Such functions are well nigh impossible in the presence of a major fracture, subluxation or acute disk hernia.

An important clue to the presence of exaggeration is that pain is very rarely continuously moderate or severe. Non-litigation patients with acute back symptoms will relate that within a day or two of the onset, they can find a comfortable position almost free of pain; but if they do move, pain is instantaneous and severe for seconds or a few minutes. Those that exaggerate tell of constant unceasing pain, day and night.

Physical signs

In the presence of such major lesions, examination in the emergency room will usually show limited movements of spinal flexion, a tilt due to sciatic scoliosis, marked restriction of straight leg raising (SLR) often of $20-50^{\circ}$ on the worse side; local tenderness to palpation and tapping, and bruising are variable and unreliable. Motor, sensory and reflex deficits can occur only if the nerve roots are damaged. The gait is hesistant, and spinal posture sometimes visibly bent and tilted to one side. Such findings are non-diagnostic, but neurological signs and severely impaired SLR are not seen in uncomplicated lumbar sprains and soft tissue injuries. X-rays will show fractures and subluxations, but in the acute stages will not disclose a disk extrusion.

Radiological findings

It is often overlooked that considerable radiological 'abnormalities' commonly exist in people devoid of symptoms and complaints. Hence the relation between abnormalities in the lumbar spine and low back pain is often controversial. Boden showed that at least one third of symptomless volunteers have Magnetic resonance imaging (MRI) signs of herniated disks and/or stenosis of the spinal canal. The incidence of abnormal MRI scans in such asymptomatic individuals increases with age to nearly 50% in the over 60s.[2,30] MRI examinations on 98 asymptomatic subjects[31] were also examined independently by two neuroradiologists blind to the clinical status of the subjects. To reduce bias, abnormal MRI scans from 27 people with back pain were mixed at random with the asymptomatic subjects. The five lumbosacral intervertebral disks were classified as: normal, bulge, protrusion, and extrusion (see Table 1). Sixty-four per cent of the 98 asymptomatic subjects had abnormal disks. With the results of the two readings averaged, 52% of subjects had a bulge at least one level, 27% had a protrusion, and 1% had an extrusion. Thirty-eight per cent had an abnormality of more than one intervertebral disk. The prevalence of bulges, but not of protrusions, increased with age. The most common non-intervertebral disk abnormalities were Schmorl's nodes in 19% of the subjects; annular defects in 14%; and facet arthropathy in 8%. The findings were similar in men and women.

Thus on MRI examination there is a high prevalence of radiological 'abnormality' in symptomless subjects. The discovery by MRI of bulges or protrusions in people with low back pain is usually coincidental.

Aspects of litigation

The effects of litigation have to be considered as potential factors than can enhance claims for both severity and duration of symptoms, irrespective of their medical or surgical treatment. The detailed appraisal by an experienced clinician can disclose telling differences that hint strongly at the role of such factors. Table 2 constrasts the clinical features that typify those with and without pending insurance benefits claims. The differences in claimants are striking, and suggest causal factors which differ from the normal clinical context between patient and doctor in which compensation does not apply.

In the litigant, only in the event of a missed tumour, inflammatory spinal lesion, or if there is an unrelieved

Table 2 Clinical features in non-litigants and litigants

Clinical features	*Acute back sprain or injury. No litigation*	*Acute back sprain or injury. Litigation*
Course of symptoms	Partial or complete recovery	Worsens or fails to improve
Leg radiation if present	L5, S1 back and side of buttock, thigh to foot	Often L2, 3, 4 - diffuse or front of thigh, not below knee (non-anatomical)
Onset	Immediate or within 24 h	May be delayed after 48 h
Pain	Intermittent not constant; worse for seconds after inappropriate movement, sneezing or coughing	Constant every minute of every day
Relief by physical therapies	Partial or complete	Partial for hours or days only, or worse after treatment
Effect of posture on pain	Relief by lying horizontal or by walking; walking better than sitting	Worse with all positions; walking worse than sitting
Inappropriate signs on examination	Absent	Often present

large disk fragment sequestered in the spinal canal, is surgery likely to succeed. Unless these conditions are satisfied, a surgeon or pain clinic[32] may, at best, be capable of ill-judged invasive interference or at worst of causing harm to the claimant. Whilst litigation is pending, it is exceptional for surgical interventions to secure lasting, significant improvement in symptoms and disabilities for the plaintiff. Thus, the medical expert should advise accordingly against the commonly performed procedures listed above, for which incidentally, defendants are often asked to pay.

Lawyers may misconstrue the willing consent of the patient to undergo surgery or repeated invasive procedures as an indication of the genuine nature of the complaints. In other pain syndromes such behaviour is common, serving to attract sympathy and attention to an unhappy person dissatisfied with his/her life, or as a respectable means of avoidance of unpleasant work or a bad employer. Add to that the possibility of financial recompense, then the willingness for surgical procedures is understandable.

'Inappropriate signs' and 'illness or chronic pain behaviour' are often used as devices to render legitimate and attributable a variety of bogus signs that plaintiffs evince during a clinical examination. Their real purpose however, is to deceive the examiner into believing there is more severe disease or disorder than actually exists.

Waddell and colleagues described 'Non-organic physical signs in low back pain described and standardised in 350 North American and British patients,... distinguishable from the standard clinical signs of physical pathology...'[33] Known as the Waddel signs (Table 3), they were held significant if three or more were positive; their reliability was shown by observer agreement in 86%. Waddell commented: ''Nonorganic signs present in medicolegal cases, compensation patients and in other problem patients. They are correlated with the 'neurotic triad' scores of Minnesota Personality Inventory – a non-specific general measure of psychological distress. They are correlated with prolonged pain and disability, failed treatment, previous surgery, and psychosocial reactions and problems.'' Pain drawings, with excellent inter-evaluator reliability of 73% to 78% were obtained from a group of 651 patients who had chronic low-back pain and demonstrated that a large proportion of patients with high Waddell scores had nonorganic pain drawings.[34]

Table 3 Waddel *et al*.[33] non-organic physical signs in low-back pain

Tenderness	Superficial, non anatomical – deep, wide area
Simulated pain	On axial loading Rotation of pelvis with shoulders
Distraction	Reduced straight leg raising → normal when sitting = 'flip test'
Regional	Weakness: cogwheel or giving way Sensory loss divergent from accepted neuroanatomy
Over-reaction	Disproportionate verbalisation, facial expression, muscle tension and tremor, collapsing and sweating

On clinical examination any one of these signs is sufficient in establishing an exaggeration of the physiological state, so that Waddell's insistence on three factors is not generally accepted. Each sign requires both a preconception of a pattern of signs that the patient thinks supports or proves his contention that he has been seriously injured, and necessitates a voluntary attempt by the patient to produce the apparent physical signs.

False or 'inappropriate' physical signs Grimacing, grunting, and inappropriate gross restriction of movements, non-anatomical sensory loss or weakness during examination are common accompaniments, which are plainly deliberate, and not the result of dissociation or hysterical illness. Spurious restriction of hip flexion is common, as is apparent back pain when the supine subject dorsiflexes the toes. They are clearly a

conscious reaction to the examination: an attempt to present the doctor with a picture of illness or pain as conceived by the claimant. Some subjects give the strong impression of a well rehearsed and even tutored performance.

'Illness behaviour' Not surprisingly, this term often puzzles the Judiciary. Psychiatrists and pain clinic physicians relate it to the experience of chronic pain and suffering, but usually fail to tell us how, or by what mechanism. The terms: illness behaviour, chronic pain syndrome, maladaptive disorder, only describe, but do not explain the fact that the patient alleges chronic and severe pain, and may exhibit withdrawal from normal daily activity in a negative fashion. It often serves to attract attention, and may prove a face-saving device, which excuses long absences from work. It does not merit the respectability of a validated diagnosis, since it specifies neither causative mechanisms nor abnormal causal pathology.

Psychiatric illness Does a psychological mechanism determine the abnormal behaviour? Is it anxiety, a phobic state, depression, a hysterical conversion syndrome or somatoform disorder? Such psychiatric diagnoses, acceptable in a clinical context, are seldom proffered as comprehensive explanations by psychiatrists in these plaintiffs. They take shelter under meaningless phrases such as – illness behaviour, maladaptive reaction – as if they constituted psychiatric illness.[35]

Diagnoses are neither diseases nor illnesses.[36] The distinguished psychiatric authorities Mindham Scadding and Cawley observed: 'The psychiatric community seems determined to ground its medical legitimacy on principles that confuse diagnoses with disease. If mental illnesses are diseases of the Central Nervous System, they are diseases of the brain, not the mind. If mental illnesses are the names of (mis)behaviour, they are forms of behaviour, not diseases.'[37]

In other cases, there are serious difficulties in accepting the validity of deliberately vague, global terms: 'Post-traumatic syndrome' and 'Post-traumatic stress disorder'. Like chronic pain syndrome, these are the product of arbitrary criteria of committee consensus, devoid of reproducible objective abnormality. Crucially, they are scientifically untestable and therefore as working diagnoses are untenable.

The psychosocial model This represents the injury victim seeking to maintain the sick role: the patient's attempts to convert a pre-existing life of discontent into a socially acceptable form of disability that leads to secondary gain. Without the accident, they cannot secure the sick role because psychological disability or 'failure to cope' are sources of blame for lack of will power or flaws of character. Thus the sick role is granted only when the individual's behaviour is 'not their fault' and 'beyond their control'. This must be conveyed to doctors and others, as symptoms of organic disease that is someone else's fault. The claimant must then emphasise pain from 'injury', and must seek out others who will allow him through the no-fault gate. Many physicians and other health care professionals are more than willing to act as the enabling no-fault gatekeepers, often because of the associated tertiary gains.[4]

Can we predicte the failed back syndrome?

In unsettled litigation claims, there is a number of identifiable risk factors. Prominent amongst them is the possibility of compensation, and importantly, the associated prolonged period when lawyers obtain reports and examinations by several medical experts. A full inspection of past medical records is essential to disclose longstanding or recurrent back pain or sciatica; symptoms are commonly forgotten or suppressed in medico-legal examinations.

Predisposing conditions may also be identified in previous records, or on X-rays taken at the time of injury. They materially affect the question of causation, and can predispose to later chronic back problems without inculpating the injury. Spondylolysis is a congenital defect in the interarticular part of the neural arch, allowing a slip, usually of L5 on S1. It causes a spondylolisthesis. Often symptomless it can be associated with persistent back pain and minor root irritation. Similarly, a narrow spinal canal, (spinal stenosis) often congenital or spondylotic, can cause pre-accident symptoms of 'claudiation of the cauda equina', consisting of bilateral spreading pain and paraesthesiae in the buttocks and legs with variable weakness. Symptoms are provoked by walking and relieved slowly after 10 – 20 min. by rest. Patients tend to stoop in a flexed simian posture because flexion widens the narrow canal, and thus lessens symptoms. A further intrusion into the stenosed canal by injury, or more often by chronic hard central protrusion(s) will precipitate symptoms.

After a wrong diagnosis has been excluded, certain risk factors crop up repeatedly to an extent less often seen in non-litigation practice. They include repeated medical certificates for sickness benefits given by the primary care physician; failed surgery – especially if operation is executed for the wrong indications; multiple spinal procedures; a past history of prolonged periods off work with illnesses and operations; inadequate social support or poor motivation for rehabilitation and return to work; psychological illness or clinical depression before or after injury; and positive inducements that reward continuing symptoms and disabilities (Table 4).

Physicians can improve the outlook and prevent some failed back syndromes by careful attention to the need for an initial period of strict bedrest and analgesia, and after a few days graded active mobilisation and encouragement, with positive advice

Table 4 Risk factors for failed back syndrome

1.	Wrong diagnosis – missed spinal tumour, diskitis, osteitis, other
2.	Repeated medical certificates for sickness benefits
3.	Failed surgery – first operation wrong indication eg no root signs; symptoms do not match signs or MRI; too soon; too late
4.	Multiple spinal procedures
5.	Prolonged absence from work in past illnesses and operations
6.	Poor social support and/or motivation for rehabilitation and return to work
7.	Psychological illness, 'pain-behaviour', or clinical depression before or after injury or operation.
8.	Pending compensation.
9.	Factors that reward continuing symptoms and disability: retreat into illness.

that the patient returns early to work. Spinal supports, rest, passive modalities, and encouraging a behaviour where one 'does not push one's limits', or 'takes it slowly and gradually until fully healed' are not helpful approaches, and may indeed be very harmful. The results is that the paraspinal muscles weaken because of disuse; this virtually guarantees prolonged disability.

Patients should sympathetically be persuaded of the importance of putting up with some discomfort, for by adapting to it their recovery will be accelerated. Early return to work is encouraged, with a brief period (weeks not months) of part-time light duties, then early, graduated return to normal activities.

Conclusion

Nachemson, a distinguished authority, states:[1] 'Few non-surgical methods have proven effective in rendering the patient better for him to return to work. Even fewer studies demonstrate any benefit from surgery, simple open removal of a proven disk hernia being the only exception. For patients with unproven diagnostic labels such as facet arthritis, degenerative disk disease, internal disk resorption and instability, no evidence exists that any type of surgery is cost-effective. More attention must be paid to illness behaviour by anyone treating chronic low back pain syndromes (>3 months). Such psychological reactions to an originally nociceptive pain stimulus somewhere in the motion segment, must be elucidated and addressed, before embarking on risky and expensive treatment modalities including surgery.'

The prime clinical feature of the failed back syndrome subsequent to trauma is often the discrepancy between the injury sustained and the apparent severity of the symptoms and disabilities. Some elements are iatrogenic, depending on generally well intentioned, but overzealous treatments and investigation by physicians and surgeons. These sadly encourage disability, abnormal illness behaviour, and provide tertiary gains for the therapist. In the remainder, the labels of 'chronic pain syndrome' and such pseudo-diagnoses are myofasciitis, fibromyalgia, and post-traumatic stress disorder as explanations, are, scientifically unacceptable in the absence of objective and verifiable criteria. Though inherently unpalatable to medical experts and judges alike, conscious exaggeration is in some litigants the mechanism for the disparity between the injury sustained and the apparent intractability of symptoms. There is now good evidence that prolonged, skilled conservative care will sometimes prevent the failed back that so often complicates ill-judged invasive procedures.

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References

1 Nachemson A. Chronic pain – the end of the welfare state? *Qual Life Res* 1994; **3 (Suppl 1)** S11 – S17.
2 Long DM. Low back pain and sciatica. In: Johnson RT (ed). *Current therapy in neurologic disease*. BC Decker Inc CV Mosby: Philadelphia, Toronto, 1985, pp. 69 – 72.
3 Robertson JT. Role of peridural fibrosis in the failed back: a review. *Eur Spine J* 1996; **5 (Suppl 1)** S2 – S6.
4 Ferrari R *et al*. The best approach to the problem of whiplash? One ticket to Lithuania, please. *Clinical and Experimental Rheumatology* 1999; **17:** 321 – 326.
5 Pearce JMS. A critical appraisal of the chronic whiplash syndrome. *Journal of Neurology, Neurosurgery and Psychiatry* 1999; **66:** 273 – 276.
6 Long DM. Failed back syndrome. In: Johnson RT (ed). *Current therapy in neurologic disease*. BC Decker Inc CV Mosby: Philadelphia, Toronto. 1985, pp. 51 – 53.
7 Wynn Parry CB, Girgis F, Moffat B, Bhalla AK. The failed back: a review. *J R Soc Med* 1988; **81:** 348 – 351.
8 Boden SD. Indications for low back surgery for a presumed herniated disk with nerve root impingement. *Hippocrates' Lantern 1*994; **2:** 1 – 5.
9 Pearce JMS, Moll JMH. Conservative treatment and natural history of acute lumbar disk lesions. *J Neurol Neurosurg Psychiatry* 1967; **30:** 13 – 17.
10 Pearce JMS. The lumbar disk syndrome. *Postgrad Med Journal* 1969; **45:** 278 – 284.
11 Dhar S, Porter RW. Failed lumbar spinal surgery. *Int Orthop* 1992; **16:** 152 – 156.
12 Davis RA. A long term analysis of 984 surgically treated herniated lumbar disks. *J Neurosurg* 1994; **80:** 415 – 421.
13 Fiume D *et al*. Treatment of the failed back surgery syndrome due to lumbo-sacral epidural fibrosis. *Acta Neurochir Suppl Wien* 1995; **64:** 116 – 118.
14 Kumar K, Nath R, Wyant GM. Treatment of chronic pain by epidural spinal cord stimulation. A 10 year experience. *J Neurosurg* 1991; **75:** 402 – 407.
15 Devulder J, De Laat M, Van Bastelaere M, Rolly G. Spinal cord stimulation: a valuable treatment for chronic failed back surgery patients. *J Pain Syndrome Manage* 1997; **13:** 296 – 301.
16 Davis RA. A long term outcome analysis of 984 surgically treated herniated lumbar disks. *J Neurosurg* 1994; **80:** 415 – 421.

17 Jonsson B, Stromqvist B. Repeat decompression of lumbar nerve roots. A prospective two year evaluation. *J Bone Jt Surg B* 1993; **75:** 894–897.
18 Lack W. The spine in adults. Die Wirbelsaule Im Erwachsenenalter. Wien Med Wochenschr 1993; **1430:** 259–267.
19 Stuckey RH. Percutaneous rhyzolysis: Active treatment of chronic back pain. *J Neurol Orthop Med Surg* 1991; **12:** 116–119.
20 Nelson DA. Intraspinal therapy using methylprednisolone acetate: Twenty three years of clinical controversy. *Spine* 1993; **18:** 278–286.
21 de Lissovoy G *et al*. Cost-effectiveness of long-term intrathecal morphine therapy for pain associated with failed back surgery syndrome. *Clin Ther* 1997; **19:** 96–112 (Discussion 84–85).
22 Marks RC, Houston T, Thulbourne T. Facet joint injection and facet nerve block. A randomised comparison in 86 patients with chronic low back pain. *Pain* 1992; **49:** 325–328.
23 Moller J *et al*. Results of lumbosacral distraction spondylodesis for the treatment of spondylolisthesis, failed back syndrome, and lumbar instability. *Eur Spine J* 1992; **1:** 117–124.
24 White AA, Panjabi M. In (eds) *Clinical Biomechanics of the Spine*. 2nd Edn. JB Lippincott: Philadelphia, 1990.
25 Pratt ES, Green DA, Spengler DM. Herniated intervertebral disks associated with unstable spinal injuries. *Spine* 1990; **16:** 662–666.
26 Farfan HF. Mechanical disorders of the low back. Lea & Febiger, Philadelphia, 1973.
27 Roaf R. A study of the mechanics of spinal injuries. *J Bone Joint Surgery* **42B:** 810, 1960.
28 Smeathers JE. Shocking news adout disks. *Current Orthopaedics* 1994; **8:** 45–48.
29 Rybock JD. Acute back pain and disk herniation. In: Jonsson RT (ed) *Current therapy in neurologic disease*. 2nd edn. BC Decker Inc CV Mosby: Philadelphia, Toronto. 1987, pp. 48–50.
30 Boden SD, David DO, Dina TS. Abnormal lumbar spine MRI scans in asymptomatic subjects: A prospective investigation. *J Bone Joint Surg* 1990; **72A:** 403–408.
31 Jensen MC *et al*. Magnetic reasonance imaging of the lumbar spine in people without back pain. *New Engl J Med* 1994; **331:** 69–73.
32 Kay NRM, Morris Jones H. Pain clinic management of medicolegal litigants. *Injury* 1998; **29:** 305–308.
33 Waddell G, McCulloch JA, Kummel E, Venner RM. Nonorganic physical signs in low-back pain. *Spine* 1980; **5:** 117–125.
34 Chan CW *et al*. The pain drawing and Waddell's nonorganic physical signs in chronic low back pain. *Spine* 1993; **18:** 1717–1722.
35 Pearce JMS. Post traumatic syndrome and whiplash injuries or neck sprains. In: Kennard C (ed). *Recent advances in clinical neurology*, 8 edn, Chapter 7, Churchill Livingstone: Edinburgh, 1995, pp. 133–149.
36 Pearce JMS. New diagnoses for old diseases: dangers and distractions. *Quarterly J Med* 1994; **87:** 253–258.
37 Mindham RHS, Scadding JG, Cawley RH. Diagnoses are not diseases. *Br J Psychiatry* 1992; **161:** 686–691.