

# Research paradigms in psychosomatic medicine with special emphasis on whiplash – Cervical Hyperextension Flexion Injury (CHFI)

Prof. Harold Merskey\*

---

## Abstract

There have been a number of attempts, particularly in the last five decades to understand the origins of pain in terms of psychological or psychosomatic patterns. These include psychoanalytic explanations relying on hysterical mechanisms, and psychophysiological proposals. The occurrence of pain in the course of psychiatric illness and its remission after the illness, has long been known and is not a controversial issue. However, the reported explanations of pain without

overt and obvious prior psychiatric illness have largely failed to convince a significant portion of the professional establishment. These explanations have very often coincided with the interests of insurance companies, whether those insurance companies were providing medical benefits, disability insurance or workers' or accident compensation. Critical examination of the evidence generated by insurance company related research indicates profound weaknesses in it.

---

## Introduction

The leading proponent of psychological medicine, FRANZ ALEXANDER, postulated the occurrence of "psychogenic organic disease" (1). Asthma in children was attributed to excessive dependency on the mother. A wish to be protected caused resentment at dependency and thus hostility. In rheumatoid arthritis ALEXANDER hypothesized unconscious conflict about anger and rebellion, with a desire to dominate, control and tyrannize others. It would only occur in individuals who harboured specific conflicts and were physiologically predisposed to the illness (1,2).

Views of this sort extended liberally from eczema to alopecia and from duodenal ulcers and ulcerative colitis to spastic bowel (3) but declined as standards of psychological proof increased. Most of the previous hypotheses are now ignored. The success of organic treatments – steroids, antileukotrienes, histamine blocking agents, proton pump inhibitors, antibiotics for *Helicobacter pylori*, methotrexate, etc reduces the need for psychiatry in treating asthma, peptic ulceration, eczema, and rheumatoid arthritis, etc. Stringent methodology has been applied to evaluating psychological hypotheses and while some psychological techniques can be demonstrated to be effective eg, cognitive therapy, their effect on organic markers seems limited or negligible. Further, major psychoanalytic ideas have been widely rejected (4), psychoanalysis is a therapeutic failure, and the work of FREUD scholars in history, biography, and textual analysis (4,5), the writings and testimony of Freud's former patients and the records of his own correspondence with ROBERT FLIESS, have all served to demolish his scientific claims.

## Theories of Pain: Psychoanalysis

FREUDIAN theories of pain were advanced by others who speculated that an unconscious defence against aggression would

result in guilt, resentment, hostility and pain; pain as a hysterical conversion symptom (6-8). ENGEL (6-8) claimed that the patients uniformly presented a masochistic character structure with varieties of self-punitive behaviour, unnecessary surgery, other somatic symptoms, tolerance of "organic pain" and remission of the pain at times of misfortune. He named this type of patient "pain prone" and asserted that pain would serve as a warning of bodily damage, had a communicative function and was linked with punishment, aggression, guilt, sexual feelings and the loss of a loved object.

Case histories supported ENGEL'S claim. Case-control studies in psychiatric patients with pain (9-11), failed to support his main hypotheses.

There have not been many studies of pain to examine ENGEL'S hypotheses since those times. A detailed investigation by ADLER conducted with great care still failed to demonstrate that there was adequate controlled evidence in support of ENGEL'S theories (12), having differences in the age and sex of the groups compared, and having problems in selection criteria.

## Behavioural Approaches

Psychologists next put forward behavioural theories of pain. Their impact can be conveniently dated from 1968 with the publication of a landmark paper by FORDYCE ET AL. (13) suggesting that health practitioners should treat pain behaviour, not pain. Talk of pain was to be avoided and patients were encouraged to act as if they were not in pain. This only applies if there is no significant physical problem and presumably not to individuals whose pain is the direct result of an obvious psychiatric illness such as a severe depressive state that could be treated with pharmacological techniques. FORDYCE elaborated his views in a widely cited monograph (14). Later in a report by an IASP Task Force it was suggested that "nonspecific back

---

\*DM FRCP FRCPC, FRCPsych., Professor Emeritus of Psychiatry, University of Western Ontario, London, Ontario, Canada  
Correspondence and reprints: Dr Harold Merskey, 1001 Adelaide Street North, Suite 205, London, Ontario N5Y 2M6. Telephone 519-434-8333, fax 519-434-8880, e-mail merskey@on.aibn.com

pain" should largely be treated by behavioural means alone (15). The authors defined nonspecific back pain as back pain which could not be accounted for by organic findings, particularly those based on imaging. In such patients the report recommended discarding the word pain and talking of "activity intolerance" after six weeks elapsed from the onset of the pain and adequate treatment had been provided. Adequate treatment would be conservative measures for low back pain. Medical benefits were to be denied at six weeks and also income replacement benefits, but rehabilitation opportunities were to be provided.

Strong objections were expressed to this approach (16-19). The executive of the Canadian Pain Society condemned the conclusions of the report and dissociated itself from it (19), a position that was ratified by the society in 1996, and the Council of the IASP determined that publications by IASP Press should not be regarded as Council policy unless so specified.

The preferred theoretical formulation now among psychologists in North America for patients with chronic pain is "cognitive behavioural treatment" rather than "behavioural treatment". This enables the practitioners who supported the behavioural approach to defuse some of the hostility to which it gave rise by adopting a less harsh attitude based more upon active rehabilitation and cognitive psychological measures. Cognitive treatment is close to traditional supportive psychotherapy which, in some form, is part of the practice of nearly all psychiatrists and psychologists. Substantial evidence exists for its usefulness but there is probably no evidence that it can cure whiplash and minimal evidence that behavioural treatment cures bad pain.

### Causal Sequences

Almost all of the pain patients sent to psychiatrists were said to lack evidence of physical illness causing pain. In the psychiatric setting and in the absence of organic illness and with the diagnostic criteria of the early 1960s, we had found psychiatric diagnoses for the patients, especially hysteria, in the more chronic patients. I would not be likely or able to make the same diagnoses again in the same patients using current criteria for psychiatric illness.

After 1967 at the National Hospital for Neurology and Neurosurgery in England the patients whom I saw for pain had much more physical disorder. Much of the psychiatric illness observed was secondary to the physical condition. Some psychiatric disorders were primary and independent and had given rise to issues of differential diagnosis, but once the neurological diagnosis had been made – with the best of current available techniques – there was still an area of uncertainty as to the origin of pain in quite a few patients.

WOODFORDE and I (20) showed that certain patients who had had supposed "minor head injury" did not recover from their symptoms after receiving compensation, or in circumstances where they were not eligible to receive compensation. Also, certain psychiatric medications that had an effect on chronic pain seemed to work for patients with undoubted physical illness, eg, amitriptyline or methotrimeprazine and in ways which were not always characteristic of patients responding to anti-depressants or phenothiazines for psychiatric pur-

poses (21). Studies of the personality traits of patients with chronic pain found that some of those with physical illness appeared to be more "neurotic" than those without evidence of lesions. This suggested that people who suffer from chronic pain perhaps become more anxious and obsessional as a result (22). The data provided support for the view that a significant proportion of the emotional disturbance associated with chronic pain is a secondary effect (23).

During the 1970s data also appeared suggesting that "tension headache" seemed less due to muscle tension and more due to other factors including, perhaps, emotional ones. Quantitative EMG studies showed only a small contribution to the overall variance when muscle contraction was taken to be a potential cause of headache or other sustained pain.

Further studies were conducted examining patients in different clinic settings in terms of their personality features, rising in responses to standard questionnaires. Dental patients then demonstrated an interesting finding which was contrary to expectation (24, 25, 27).

The patients came from an oral medicine clinic. The literature suggested that patients with temporomandibular pain and dysfunction syndrome (TMPDS) would represent a population whose pain resulted from their life experiences and their emotional state and that their scores for emotional disturbance would resemble those of psychiatric patients with anxiety and depression. It was further postulated that the patients with temporomandibular pain and dysfunction syndrome would show differences from those with known physical causes of pain in terms of their attitudes to parents and childhood experience. The actual comparison of TMPDS patients and patients with facial pain and lesions (or pathophysiological disorders) showed little evidence of neuroticism in either group; nor were parental bonding attitudes (i.e. adult views of childhood experience) abnormal. The features expected of psychological disorder were not found in our data.

Where pain is not associated with a definite dental lesion requiring a procedure, dentists quickly refer their patients to an oral medicine specialist who is "more comfortable" in dealing with dental or facial pain that does not require instrumental procedures. This could account for the large number of patients with pain but without emotional disturbance seen by the professor of oral medicine.

Other studies of patients with headache, back pain or other pain without important neurological lesions (28), and in four different chronic pain populations (29), all led to the same conclusions. The rates in an oral medicine facial pain clinic were as low as 30% with the General Health Questionnaire – 28 items (GHQ28) and in a psychiatrist's pain assessment and treatment service were 51%. Those in pain clinics were 37%. The psychiatric clinic patients were significantly more depressed according to the test subscales of the General Health Questionnaire and also tended to have more inward irritability. The extent of their somatic complaints and anxiety does not differentiate the majority of pain patients with primary psychological illness very well from others attending for treatment. However in the patients who had definite psychological symptoms, depression, social dysfunction and irritability provided a characteristic pattern (29).

## Selection

Selection effects can raise or reduce the amount of emotional disturbance found in patients under examination. ROMANO & TURNER (30) showed that the incidence of depression in clinics treating patients with chronic pain ranged from reports of a 10% frequency to 100%. Again, in a pain clinic compared with a community sample (31) patients in the clinic were more likely to have been injured, reported a greater intensity and constancy of pain and had more difficulties with the activities of daily living. They were more depressed and withdrawn socially and showed more long term consequences due to unemployment, litigation and alcohol and drug abuse. Whereas only 2% of patients in the family practice group were disabled or unemployed, 38% in a pain clinic were in that situation. The importance of not making generalizations when one is faced with a very special sample can hardly be more readily recognized than by looking at those two groups. Thus patients with chronic pain were not having their pain necessarily because of prior mental problems.

Meanwhile, an opportunity arose for my colleagues and myself to study the relationship between pain and depression. The United States Center for Health Statistics had gathered a great deal of data on a sample of over 3,000 patients who had been followed up eight to nine years later.

I will cite two items. At the beginning of the survey pain and depression were associated. Using the minimum criterion for chronic pain defined for the study, 14.4% of the United States population between the ages of 25-74 suffered from "definite chronic pain" related to the joints and musculoskeletal system. About 18% of the population with chronic pain had depression (using a high cut-off score for depression on the CESD of > 19). Among those without chronic pain depression affected only 8% of the population (32). On follow-up at eight to nine years those with "chronic pain" were 2.85 times as likely to be depressed as those who had not had pain initially. The reverse odds ratio i.e., depression initially, pain later, was 2.14 (33). Thus, prior pain predicts depression, and depression predicts pain, although a little less strongly, but the reasons for this could not be extracted from the existing data set.

A much shorter follow-up period might have shown a much clearer indication of the sequential relationships between pain and depression, since the longer the time apart the less opportunity there was for the initial depressive state to have an impact over many years, and similarly between pain initially and depression later.

## Compensation Issues

Psychological theories have been used to explain pain without lesions. It is often suggested also that pain has been primarily produced as a means to entitlement for compensation.

This issue dates at least from 1866 (34). In 1961 the late PROFESSOR HENRY MILLER, a prominent neurologist, citing an earlier English author, claimed that compensation neurosis is "a state of mind, born out of fear, kept alive by avarice, stimulated by lawyers and cured by a verdict". This claim was based upon a follow-up of a sample of individuals seen for headache without recognized physical illness in MILLER'S private practice, generally for insurance companies (35).

MENDELSON (36) observed that of ten studies since the Second World War none had confirmed them and all had found patients who continued to suffer from complaints of pain or disability subsequent to legal settlement of their claims. Many such patients with chronic pain were said to have "hysteria" and their pain was attributed to "secondary gain". However, there were considerable difficulties in determining the diagnosis of hysterical pain (37).

First, regional symptoms may resemble, or result from thoughts or ideas that the patient has about what a symptom should be like. Thus "non-anatomical" patterns of pain have been called hysterical or at least "psychogenic" complaints. This is a weak idea if not misleading. Pain patterns can correspond to single root distributions or the branches of particular nerves. At other times they do not. This is particularly true for non-skeletal soft tissue/muscle pain. We are all aware that if we overuse a limb, even briefly, and set up fatigue and aching in that limb, there will be a diffuse type of numbness or loss of sensation or loss of feeling of accurate movement or power at the end of the activity. We can make this much worse and much clearer if we go out into a garden or a forest, put a tourniquet on the dominant arm, take a saw, and start to cut up a thick piece of wood. There will soon be fatigue, aching and pain in the limb. The hand will be clumsy, gross tremor and other signs which will be taken to be "psychological" if we accept the argument in dispute.

Yet a very good physiological basis exists for that effect. First, the presence of pain reduces sensitivity in other modalities. Touch becomes less refined and so forth. Second, continued noxious stimulation to one area in animals tends to cause a regional pattern of sensitivity to additional stimuli whether they are light touch or pin prick. WALL (38) summarized the evidence that the receptive fields of afferent neurons in the dorsal horn can change and extend. In the rat three to four days after de-afferentation, cells that formerly responded to stimulation within the usual anatomical area begin to respond to stimuli from other areas. Intact afferents that were formerly ineffective now excite cells that have expanded their receptive fields to incorporate innervated peripheral structures. This fits with the fact that it has long been known that referred pain may produce exquisite sensitivity in normal skin. The receptive fields themselves also vary substantially after injury in a region (39,40). The receptive field of a cutaneous afferent neuron can be enlarged so that when formerly it would respond only to a firm mechanical stimulus or to pinch, later it will respond to light touch as if a more noxious stimulus had been employed.

Findings of plasticity in the operation of neurons in the spinal cord contradict and invalidate the claim that regional syndromes and regional hypo- or hypersensitivity are necessarily psychological in origin simply because they may correspond to a patient's idea. The organic situation is the same as the patient's view. If the issue is psychological causation then a psychological cause and pattern must be found, not merely a misleading or unimportant sign.

These considerations apply to various sorts of non-anatomical sensory patterns including those found with vibration, splitting sensation at the mid-line, and also other phenomena that were traditionally regarded as hysterical (41). A

number of the so-called "non-organic" signs of GORDON WADDELL (42) can be rationalized within the same considerations. They appear to be fairly typical of severe organic illness with few other signs, but with a good deal of pain, probably related to facet joint damage, or ligamentous sprains or strains, or other causes of chronic pain.

From 1991 onwards the group here in Berne produced the most important information to date. This impressive series of studies served to establish cervical sprain injury as a physical illness affecting the neck and resulting in a very variable outcome, often excellent but sometimes with a potential for prolonged physical and emotional difficulties (43). As well, it has long been established that there is an important potential physical basis for whiplash. The investigations of MACNAB (44) on monkeys subjected to cervical hyperextension flexion injuries showed a variety of serious and significant acute changes.

The same conclusion arises from the work of BOGDUK and his colleagues to be presented here. That work establishes that whiplash pain can frequently be accurately relieved by systematic study of the innervation of the facet joints of the cervical spine, and by anaesthetization or radiofrequency ablation of the nerve supply to facet joints. The psychological state then improves greatly. The work from Berne (43) and that from Newcastle, New South Wales has commanding authority in the field of pain, and provides a definitive scientific illumination of the causes and clinical phenomena of the whiplash syndrome.

In the Berne studies (43) a representative sample was obtained as far as possible of individuals acutely injured, with sufficient pain to seek treatment; the previous personality of the individuals was recorded or evaluated through the Freiburg Personality Test; symptoms of pain and other subjective phenomena were evaluated initially and later at six months and twelve months. It was found that: (1) initial pain predicted later pain; (2) initial pain predicted later difficulties in concentration and cognitive function; (3) initial pain predicted depression later, but not immediately; (4) prior injury predicted worse results later; and (5) personality differences at the initiation of the injury did not predict outcome of pain, depression or subjective cognitive function.

These findings speak for themselves. Neck pain disturbs the mind: it is not often due to the mind.

### Insurance-Funded Studies

Three studies achieved a good deal of notoriety and two of them were funded by insurance companies.

Despite MENDELSON'S trenchant comment on MILLER, the idea persists that no one with cervical sprain injury really has a chronic illness from the injury, as in the report of the Quebec Task Force (QTF) (46), a body constituted in 1990 by the Quebec Auto Insurance Company. This Task Force was headed by a respected epidemiologist PROFESSOR W. SPITZER but did not have many clinicians known with respect to contributions to the field of whiplash studies. The Task Force did a "best evidence" review of treatments available for cervical sprain injury and found very few for which adequate evidence existed. It deservedly gained credit for this contribution although one should note that "best evidence" may be lacking for very useful

treatment simply because it has not been sought or because there are some very valid obstacles to the collection of best evidence. The Task Force therefore provided a consensus report open to all the criticisms that it offered of prior work. Thirdly, the Task Force undertook a study of a cohort of patients, or claimants, who had made claims in 1985, equating prognosis with "claim closure". Claim closure by the insurance company, a monopoly provincial government insurance corporation, was used as the factor to determine whether or not people were better at one year after having suffered a whiplash injury. This does not inspire confidence in the extent to which symptoms will be examined at the end of the year, much less disability. All that we have is the judgement of the insurer.

The results of the QTF cohort study were represented as showing that only 1.9% of individuals who had whiplash without other injuries remained disabled at one year. This was a challenge to the standard well regarded follow-up studies of whiplash produced from medical sources which have indicated disability rates of 15-45% at one year. If individuals were included with whiplash who had also had some other injuries (such as a broken ankle) the figure rose to 2.9% (47).

This result still makes it seem as if whiplash injuries with persistent disability must be quite rare and that any other complaint about them may be confusing random neck pain in the population with the original injury, as some have alleged. The figures were widely presented in Canada and the United States, and elsewhere, to indicate that whiplash symptoms at one year should not be taken seriously. Another government insurance corporation in Canada, the Insurance Corporation of British Columbia (ICBC) set up an international meeting in Vancouver in early 1999 with the stated aim of "teaching the lessons" of the QTF to family doctors and others in British Columbia, and also of bringing in distinguished contributors to the field of whiplash injury to present their scientific information. The scientific basis of the curriculum for this meeting as prepared in the form of a volume was unfortunately poorly set out and marred by numerous scientific errors or false emphases with respect to the topic (47).

In the middle of the QTF report, wrapped around with numerous tables and figures, was a mild statement that there had been 204 "recurrences" of individuals with whiplash during the follow-up year. These cases of recurrences were not due to second injuries. We suppose that they must be individuals whose claims had been closed and who had renewed their claims after they found that attempting to work again or other activity increased their pain (47). Alternatively, patients drop their medication after a while, deteriorate and seek to reopen their claims. Sometimes pain also deteriorates after whiplash instead of improving because the patient has gone on to develop post-traumatic fibromyalgia. No information was given on those possibilities and the data was not included in the original study in the calculation of the survival rates at one year. If we add these 204 cases to the 2.9% who were still said to be ill at one year it would seem that as many as 9.5% of the original population were still sick and suffering at the end of one year. A proper presentation of the data from the QTF required that information to be highlighted. It should not have been left in a corner of the volume, neglected and barely mentioned.

The next study that appeared and sought to advocate for the "insurance" position – although I have no indication that it was insurance funded directly – is the Norwegian-Lithuanian Study by SCHRADER ET AL. (48). This study will not be described except to say that it confused the occurrence of a collision with an injury and lacked power. It was widely rejected.

The third study notably minimizing whiplash was introduced in Saskatchewan, a prairie province of just over one million people in 1994. The study was funded by Saskatchewan Government Insurance (SGI). The authors compared 3,046 individuals presenting with whiplash claims under a tort system with 4,416 individuals from a no-fault system that succeeded it (49). These 7,462 whiplash claimants were derived from an overall sample of 10,902 eligible individuals. The

investigators relied upon "claim closure" by the insurance company as the "proxy" for recovery, and are subject to the same criticisms as the QTF (47, 50). As well, the authors made a remarkable claim that the SGI was unable to determine the date when a claim had been closed once it had been reopened. This claim is implausible and has also been explicitly denied by a former adjuster at SGI (51) who is highly credible. However, on the basis of that claim the authors of the Saskatchewan study dropped 28% of their cases (49, 52).

Since the reopenings in this study amounted to 28% of the data the study must be highly suspect and the management of data by the authors has to be considered seriously unreliable. A detailed examination of this study is in preparation, but meanwhile the findings by the authors, as presented, greatly assist the position of the insurance company that paid for the study.

## REFERENCES

- ALEXANDER F. *Psychosomatic Medicine*. New York: Norton and Co., 1950.
- ALEXANDER F. Development of the fundamental concepts of psychoanalysis. In: *Dynamic Psychiatry*, (Eds. F ALEXANDER & H ROSS). Chicago: University of Chicago Press, 1952.
- DUNBAR HF. *Emotions and Bodily Changes*. 2nd Edition. New York: Columbia University Press, 1938.
- CREWS F. *Unauthorized Freud – Doubters Confront a Legend*. New York: Viking, 1998.
- ESTERSON A. *Seductive Mirage. An Exploration of the Work of Sigmund Freud*. Chicago: Open Court, 1993.
- MERSKEY H, SPEAR FG. *Pain: Psychological and Psychiatric Aspects*. London: Baillière Tindall, 1967.
- ENGEL G. Primary atypical facial neuralgia. A hysterical conversion symptom. *Psychosom Med*, 1861; 13:375-396.
- ENGEL G. "Psychogenic" pain and the pain prone patient. *Am J Med*, 1959; 26:899-918.
- MERSKEY H. The characteristics of persistent pain in psychological illness. *J Psychosom Res*, 1965a; 9:291-298.
- MERSKEY H. Psychiatric patients with persistent pain. *J Psychosom Med*, 1965b; 9:299-309.
- SPEAR FG. Pain in psychiatric patients. *J Psychosom Res*, 1967; 11: 187-193.
- ADLER RH, ZLOT S, HURNY C, MINDER C. Engel's "Psychogenic pain and the pain-prone patient": a retrospective, controlled clinical study. *Psychosom Med*, 1989; 51:87-101.
- FORDYCE WE, FOWLER RS, LEHMANN JE, DELATEUR BJ. Some implications of learning in problems of chronic pain. *J Chron Dis*, 1968; 21:179-190.
- FORDYCE WE. *Behavioral Methods in Chronic Pain and Illness*. St. Louis, MO: CV Mosby, 1976.
- FORDYCE WE. (Ed.) *Back Pain in the Workplace*. Seattle WA: IASP Press, 1995.
- THOMPSON E. Myofascial pain: asthma of the back and back pain in the workplace. *Pain*, 1996; 65:111-112.
- MERSKEY H. Re: Back pain in the workplace. (Ed. WE FORDYCE). *Pain*, 1996; 65:111-112.
- TEASELL RH. Re: Back pain in the workplace. (Ed. WE FORDYCE). *Pain*, 1996; 65:112.
- CANADIAN PAIN SOCIETY. Statement Approved by the Executive. *Pain*, 1996; 65:114.
- MERSKEY H, WOODFORDE JM. Psychiatric sequelae of minor head injury. *Brain*, 1972; Part III, 95:521-528.
- MERSKEY H, HESTER RN. The treatment of chronic pain with psychotropic drugs. *Postgrad Med J*, 1972; 48:594-598.
- WOODFORDE JM, MERSKEY H. Personality traits of patients with chronic pain. *J Psychosom Res*, 1972; 16:167-172.
- MERSKEY H, BOYD DB. Emotional adjustment and chronic pain. *Pain*, 1978; 5:173-178.
- PELZ M, MERSKEY H. A description of the psychological effects of chronic painful lesions. *Pain*, 1982; 14, 3:293-301.
- DEROGATIS L. *Symptom Check-List-90 (SCL-90): (Revised Version)*. Manual I. Baltimore MD: 1977.
- GOLDBERG DP. *Manual of the General Health Questionnaire*. Windsor: NFER Publications, 1978.
- SALTER M, BROOKE RI, MERSKEY H, FICHTER GF, KAPUSIANYK DH. Is the temporomandibular pain and dysfunction syndrome a disorder of the mind? *Pain*, 1982; 17, 2:151-166.
- MERSKEY H, BROWN A, BROWN J, MALHOTRA L, MORRISON D, RIPLEY C. Psychological normality and abnormality in persistent headache patients. *Pain*, 1985; 12,1:35-47.
- MERSKEY H, LAU CL, RUSSELL ES, BROOKE RI, JAMES M, LAPPANO S, NIELSEN J, TILSWORTH RH. Screening for psychiatric morbidity. The pattern of psychological illness and premorbid characteristics in four chronic pain populations. *Pain*, 1987; 30:141-157.
- ROMANO JM, TURNER JA. Chronic pain and depression: does the evidence support a relationship? *Psychol Bull*, 1985; 97:18-34.
- CROOK J, TUNKS E. An epidemiological follow-up survey of persistent pain sufferers in a group family practice and speciality pain clinic. *Pain*, 1989; 36:49-61.
- MAGNI G, CALDIERON C, RIGATTI-LUCHINI S, MERSKEY H. Chronic musculoskeletal pain and depressive symptoms in the general population. An analysis of the 1st National Health and Nutrition Examination Survey data. *Pain*, 1990; 43:299-307.
- MAGNI G, MARCHETTI M, MORESCHI C, MERSKEY H, RIGATTI-LUCHINI S. Chronic musculoskeletal pain and depressive symptoms in the National Health and Nutrition Examination. I. Epidemiologic follow-up study. *Pain*, 1993; 53:163-168.
- ERICHSEN, SIR JOHN E. *On Railway and Other Injuries of the Nervous System*. Philadelphia: Henry C. Lea, 1866.
- MILLER HG. Accident neurosis. *Brit Med J*, 1961; i: 919-925, 992-998.
- MENDELSON G. Not "cured by a verdict". Effect of legal settlement on compensation claimants. *Med J Austr*, 1982; 2:219-230.
- MERSKEY H. Regional pain is rarely hysterical. *Arch Neurol*, 1988; 45:915-918.
- WALL PD. The dorsal horn. In: *Textbook of Pain*. (Eds. PD WALL, R MELZACK). Edinburgh: Churchill Livingstone, 1989; pp. 80-87, 101-111.
- McMAHON SB, WALL PD. Receptive fields of rat lamina I projection cells move to incorporate a nearby region of injury. *Pain*, 1984; 19:235-247.
- COOK AJ, WOOLF CJ, WALL PD ET AL. Dynamic receptive field plasticity in rat spinal cord dorsal horn following C-primary afferent input. *Nature*, 1987; 325:151-153.
- GOULD R, MILLER BL, GOLDBERG MA ET AL. The validity of hysterical signs and symptoms. *J Nerv Ment Dis*, 1986; 174:593-598.
- WADDELL G, McCULLOCH JA, KUMMEL EG, VENNER RM. Non-organic physical signs in low back pain. *Spine*, 1980; 5:117-125.
- RADANOV BP, STURTZENEGGER MD, STEFANO G, SCHNIDRIG A. Relationship between early somatic, radiological, cognitive and psychosocial findings and outcome during a one-year follow-up in 117 patients suffering from common whiplash. *Brit J Rheumatol*, 1994; 33:442-448.
- MACNAB I. Acceleration injuries of the cervical spine. *J Bone & Joint Surg*, 1964; 46A:1797-1799.

45. WALLIS BJ, LORD SM, BOGDUK N. Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: a randomized, double-blind, placebo-controlled trial. *Pain*, 1997; 73:15-22.
46. THE QUEBEC TASK FORCE ON WHIPLASH-ASSOCIATED DISORDERS (WAD). Redefining "Whiplash" and Its Management. Société de l'Assurance Automobile du Québec. Québec City, 1995.
47. TEASELL RW, MERSKEY H. The Quebec Task Force on whiplash-associated disorders and the British Columbia Whiplash Initiative: A study of insurance industry initiatives. *Pain Res Manage*, 1999; 4, 3:141-149.
48. SCHRADER H, OBELIENIENE D, BOVIM G ET AL. Natural evolution of late whiplash syndrome outside the medical job context. *The Lancet*, 1996; 347:1207-1211.
49. CASSIDY JD, CARROLL LJ, COTÉ P, LEMSTRA M, BERGLUND A, NYGREN A. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims of whiplash injury. *N Engl J Med*, 2000; 342:1179-1186.
50. FREEMAN MD, ROSSIGNOL AM. Effects of eliminating compensation for pain and suffering on the outcome of insurance claims. *NEJM*, 2000; 343:1118.
51. KIVOL K. Re: Saskatchewan Government Insurance Study, *Pain Res Manage.*, 2000;5:129-130.
52. MERSKEY H, TEASELL RW. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims. *NEJM*, 2000; 343:1119 (letter).

---

This material was presented at the International Congress on Whiplash Associated Disorders, Berne, Switzerland, March 8 to 10, 2001. The paper appeared originally in the book "*Whiplash Associated Disorders*" – medical, biomechanical and legal aspects, published by Staempfli Publishers Ltd, Berne 2002. The paper is published in North America in *Pain Research & Management* with the permission of Staempfli Publishers Ltd.



**Hindawi**  
Submit your manuscripts at  
<http://www.hindawi.com>

