

EDITORIAL

A critical appraisal of the chronic whiplash syndrome

“It is incident to physicians, I am afraid, beyond all other men, to mistake subsequence for consequence.”

Samuel Johnson from a review of Dr Lucas’s Essay on Waters (1734)

Patients with protracted complaints after neck sprains (whiplash) continue to pose difficulties for physicians, expert witnesses, and the Courts.

Strict definition is essential for clear and objective thinking. Courts are often misled by experts’ reference to publications that do not adhere to critical criteria. The term “whiplash” is confusingly used both as a shorthand for a description of the injury mechanism—a flexion-extension, or torsional movement—and more correctly for symptoms better designated neck sprains.¹

Much confusion can be avoided if patients suffering from an acute traumatic lesion of the cervical nerve roots (radiculopathy) or spinal cord (myelopathy) are excluded by definition.²⁻³ So too should patients with an acute annular disk tear, fracture, or dislocation of the facet joints and bony spine. Such cases have their own distinctive clinical features, and accompanying abnormalities on x ray films and MRI.

It is those patients without these pathologies who have suffered injury to the muscles, ligaments, and soft tissues, who are correctly labelled acute neck sprains (whiplash). The lesion is akin to a sprain with or without contusions in other areas of the body—the pulled hamstring, calf muscle, or groin sprain of football or athletics.

Sprains

A sprain is a mechanical stretching of muscular and ligamentous soft tissues with or without local bruising (contusion). It causes pain, stiffness, oedema, and variable local tenderness and muscle spasm. Such injuries and their symptoms, resolve within about 2–6 weeks.⁴ The duration is prolonged only if definable complicating factors come into play.

Prognosis

Symptoms of sports injuries, when treated with rest and analgesia followed by increasing exercise, subside within

days or a few weeks, generally without complications. In acute whiplash too, this is the rule (table 1). But, many published series appear to show a worse prognosis in whiplash injury. Norris’s series, for example, showed only 56% pain free at 1 year, and after 2 years symptoms did not alter.⁵ However, 45% had paraesthesiae, 42% back pain, and 14% auditory symptoms: not a representative series of uncomplicated whiplash. Similarly, Hohl⁶ reported 55% symptom free at 1 year; and Deans *et al*⁷ 49% at 3 months and 96% free or with occasional pain at 1 year. Importantly, the massive Quebec Task Force report⁸ concludes that almost all studies are flawed because they include patients with complicating radiculopathy, disk lesions, facet joint injuries, and psychological illnesses.

Table 1 summarises selected variable published data that includes patients who by strict criteria should be excluded. It offers a simplified schema⁹ of probable mechanisms in whiplash injuries. There is an evident overall tendency to improvement within a few months, and early return to work for most victims.

Chronic whiplash?

The problem is those with symptoms and disabilities after 6 months. To label such cases as “chronic whiplash” strongly implies continuing disease caused by their injury. Logically, only four explanations are possible:

(1) ORGANIC DAMAGE

—evident, or overlooked, in the spine or brain. The notion has arisen that “subtle” or subclinical brain damage sustained during an accident causes altered perception of pain, or prolongation of the period of pain. Yarnell and Rossie in patients with severe debility at 12 months, concluded that: “in the subacute period, neurological examination, imaging and clinical electrophysiological studies were unable to localise, structurally or functionally, the source of the (cognitive) dysfunctions.”¹¹ Similarly, of 68 patients with symptomatic cervical whiplash injuries, plain x ray films, EEG, CT, and radionuclide brain scans failed to demonstrate associated structural abnormalities.¹² There are now over 350 published examples of MRI of the

Table 1 Duration of symptoms and inability to work: postulated mechanisms

| Clinical features* | 0–3 months | 3–6 months | >6 months |
|---------------------|---|--|--|
| Pain free | 40–66% | 58–82% | 55–86% |
| RTW | 76–86% | 73–92% | 75–95% |
| Possible mechanisms | Sprained ligaments, muscles, and facet joints → inflammatory oedema. No fracture or subluxation | Painful inflammation and oedema subsided. Preceding spondylosis ± neurosis or exaggeration | Preceding spondylosis ± neurosis or exaggeration |

*Data from references^{5-7 10 14 19} and a personal series of 223 consecutive whiplash patients in medicolegal practice. RTW=return to work.

brain and neck,^{13 14} as well as studies of brainstem auditory evoked potentials. None shows unequivocal attributable pathology.

Pre-accident spondylosis

Cervical spondylosis may increase the vulnerability to a neck sprain. Jarring of an already entrapped root can cause radicular pain. Unless there is evidence of neural damage—that is, new root signs, or traumatic disc lesions (rare)—the effect is transient. Lord *et al* have found that 49% of “chronic whiplash pain” originated in the zygapophysial joints, as judged by a diagnostic test of placebo controlled anaesthetic blocks of the medial branches of the nerve supplying those joints.¹⁵ However, they acknowledged their failure with steroid injections into zygapophysial joints, and of radiofrequency lesions used for long term treatment. These meticulous studies inculpate the joints only by indirect means. If the zygapophysial joints account for chronic pain, what is the mechanism in the 51% with negative tests, and why is it that MRI studies have after months or years failed to show any such attributable pathology? We know that symptoms due to pre-existing spondylosis commonly continue and may worsen, irrespective of the trauma.¹⁶

It is essential to correlate the relevance of investigations with clinical findings, as quite gross radiological abnormalities are present in asymptomatic subjects and can be irrelevant to the symptoms presented. At least one third of asymptomatic volunteers at MRI have disc herniation, degeneration, or spinal stenosis by the age of 40; this increases to 50% at the age of 60.¹⁷ Evidence for acceleration of the spondylotic process is controversial but not convincing.¹⁸

(2) PSYCHOLOGICAL ILLNESS

Such illness includes: anxiety, phobic state, hysterical conversion, and depression. There are instances where the psychological distress precedes and causes the chronic pain; conversely, in others, psychological distress is a consequence of chronic pain.¹⁹ Seldom have I seen examples of uncomplicated post-whiplash pain alleviated by invasive procedures, but on such rare occasions when pain subsides, neurotic and depressive complaints disappear. There are genuine instances of psychogenic illnesses. But, since Radanov *et al*²⁰ found that psychosocial factors at injury do not predict the outcome, although “neuroticism correlated with the initial pain intensity”, they are an acceptable explanation in only occasional complainants. In a series of 74 whiplash patients, there was no significant difference in continuing emotional distress, phobic travel anxiety, or post-traumatic stress disorder after 3 and 12 months when compared with 126 accident victims with multiple injuries without serious head injury.²¹

Pre-accident neurosis or depression often continues after injury and colours the description of complaints, but that is not attributable unless relevant psychological deterioration is demonstrable. As a psychiatrist comments: “Once settlement is achieved, those who have a deep psychological need to be in the sick role, stay sick, or perhaps even become worse, having had the legitimacy of their behaviour endorsed by the court. Whiplash is a “man-made disease”...”²²

Being irritable, frustrated and fed-up are common reactions, but do not themselves constitute clinical depression.

Psychologists’ assessments are often appended to claims. Many rely more on a succession of standardised scales—for example, for depression, general health, post-traumatic stress disorder—than on clinical features individual to the patient. Tests that rely on questionnaires, which provide a large number of leading questions, suggest positive

responses. Many patients quickly learn the expected response, and sadly, this adds to their distressing symptomatology. Schmand *et al* have recently indicated that the prevalence of malingering or cognitive underperformance in late post-whiplash patients is substantial, particularly in a litigation context.²³ Because they are not trained to judge clinical and radiological signs, psychologists’ appraisals often lack objectivity. Despite its promise, “neuropsychological evidence generally lacks scientifically demonstrated value for resolving legal issues, and thus, if admitted into court, should be accorded little or no weight.”²⁴

(3) UNREPORTED PRE-ACCIDENT SYMPTOMS

Underreporting of symptoms preceding minor neck and head injuries is common.^{25 26} In one Norwegian study²⁵ of 27 consecutive and unselected litigation cases for “chronic whiplash”, 14 claimants had had similar significant symptoms before the injury, as shown by medical records. In eight of these they were not mentioned or were denied. These observations may be accounted for by recall bias, or by denial in a medicolegal context. In either case, continuing symptoms may erroneously be attributed to the accident.

(4) CONSCIOUS EXAGGERATION

When financial rewards are at stake, it is not surprising that exaggeration frequently occurs. Malingering is a dangerous term, and is unacceptable without good evidence. Simulation of illness and deception are of course patterns of behaviour consciously chosen, and should not be interpreted as psychological illness. Physicians and expert witnesses should suspect exaggeration as probable if one or more of the following features are present:

- When symptoms are discordant with the injury
- When restricted spinal movement is discrepant with the pathology in the spinal canal within a short time of examination
- When there are “spurious”, or “inappropriate” physical signs
 - when analgesics, collars, and a wide range of physical therapies fail to produce reasonable relief
 - When physical activities (observed by witnesses or video observation) are variable and inconsistent with clinical signs and behaviour during an examination at about the same time after injury.

Deliberate exaggeration^{22 27} can be motivated by financial reward and by increased attention and sympathy. Family and friends are often unwittingly entwined in a complex social disorder of assumed invalidity that may be masked by the euphemisms: “sick role”, “illness behaviour”, and “chronic pain syndrome”. These terms are commonly used in reports, but they afford neither an explanation nor validation for symptoms: they restate the problem in jargon. They fail to verify it.

Occasionally, plaintiffs and experts may mislead judges. Understandably, judges find it difficult to imagine that a plaintiff will submit to (ill-judged) surgery. But, surgeons are commonly persuaded to operate on such patients in the altruistic endeavour of doing something to try to aid recovery; but benefit seldom accrues.²⁸ Patients often subject themselves to surgery and other physical therapies if the perceived rewards are sufficient. They may abandon worthwhile and remunerative work without adequate medical cause: Courts may then be asked to recommend payment for future loss of earnings.

It is easy to overlook the undoubted fact (see below) that there is a large number of people suffering from frequent neck pain and often headaches, who are able to continue their normal jobs. Inappropriate loss of earnings may be

Table 2 Controlled study of whiplash injury (adapted from Schrader *et al*³¹)

| | Any neck pain in previous year | Chronic neck pain (>7 days/ month) | Chronic headache (>7 days/ month) | Chronic neck pain or headache before accident |
|------------------|--------------------------------|------------------------------------|-----------------------------------|---|
| Accident (n=202) | 71 (35%) | 17 (8.4%) | 19 (9.4%) | 7/17 neckache 10/12 headache |
| Controls (n=202) | 67 (37%) | 14 (6.9%) | 12 (5.9%) | "identical to above" |

apportioned by a sympathetic judge to a pleasant and plausible plaintiff who declares both unending devotion to the work he or she loves, and frustration at being incapable of resuming such work.

Sometimes what starts as deliberate exaggeration becomes an adopted way of life. The unfortunate patients may come to believe that they are ill; symptoms then persist after settlement.

Controlled data

The layman's knowledge about the sequelae of whiplash can determine both expectations^{29 30} and symptoms. Until recently there was a serious dearth of data in whiplash controls—that is, those with no compensation claims.

In Lithuania, few car drivers and passengers are covered by insurance and there is little awareness among the general public about the consequences of whiplash injury. In a controlled retrospective cohort study³¹ there were 202 individuals (157 men mean age 43; 45 women mean age 44) identified from the traffic police department in Kaunas, Lithuania. They had been involved in a car collision with rear-end impact 1–3 years earlier. Acute neck pain occurred in 31 accident victims; it started within 1–3 days, was transitory in all, and in most (22/31) it lasted less than 1 week. There was a high incidence of neck pain and headache before the accident, in both control and injured subjects (table 2). None had persistent or disabling complaints that could be linked to the accident. No significant differences were found between the accident victims and 202 matched controls in respect of prevalence of symptoms including neck pain, headache, and subjective cognitive dysfunction at the time of the interview.

This parallels a random study of 10 000 adult Norwegians of whom 34.4% had experienced neck pain within the past year, and 13.8% reported neck pain that lasted for more than 6 months... "The reported prevalence of persisting pain after whiplash injuries is of the same magnitude as the prevalence of chronic neck pain in the general population."³²

Obelieniene *et al* (this issue, pp 000–000) report a prospective, but new controlled inception cohort study. Two hundred and ten rear-end collision victims were consecutively identified from the daily records of the Kaunas traffic police. Neck pain, neck mobility, and headache were evaluated by questionnaires shortly after the accident, and at 2 months, and 1 year. As controls, 210 sex and age matched individuals were randomly taken from the population register of the same area and evaluated for the same symptoms immediately after their identification and after 1 year.

Initial pain was reported by 47% of accident victims; 10% had neck pain alone, 18% had neck pain together with headache, and 19 % had headache alone. The median duration of the initial neck pain was 3 days and maximal duration 17 days. Headaches lasted on average 4.5 hours and at most, 20 days. After 1 year, there were no significant differences between the accident and control groups concerning frequency and intensity of these symptoms.

Gender, sitting position in the car, head position at the moment of the accident, use of seat belts, and presence and adjustment of the headrest did not influence the incidence of acute neck pain.

Although a questionnaire method is not wholly reliable, in this study any errors would be without bias in both con-

trols and accident victims; so the results are acceptable. Prevalence of neck pain and headache was the same as is found in western countries,³³ confirming that the questionnaires were sufficiently sensitive. The results show no evidence that Lithuanians are more or less prone to pain after injury than people in western societies.

ATTRIBUTION

The apparent association of neck pain and injury does not automatically mean a causal link. Weaknesses in published work reflect many factors; most are in methodology and erroneous deductions. We can apply these potential sources of error (in italics) pointed out by Bradford Hill and others,^{34 35} to the chronic whiplash syndrome.

(1) Inadequate numbers or poor matching may give an *apparent chance association*; most whiplash series are small.

(2) *Bias* in selection of patients; most arise from compensation claims in medicolegal practice.

(3) *Lack of controls*; series of non-litigant whiplash cases are rare.

(4) *Confounding* by other factors/illness/drugs; common in whiplash cases.

(5) Early or presymptomatic illness may cause abnormalities which are mistaken for the cause of the disease—that is, *reverse cause*; both coincidental neck pain and spondylosis are common in uninjured subjects; psychoneurotic illness frequently precedes and continues after injury.

(6) A *consistent association* shown in different types and timed studies is unlikely to be due to chance; the occurrence of chronic pain is an occasional—not a consistent—sequel to whiplash injury

(7) A known mechanism relating cause to effect is helpful, but *biological plausibility* may be logical, compelling, and wrong. No consistent mechanism for chronic whiplash complaints has been shown³⁶; different mechanisms are claimed but not proved, for different patients.

Conclusion

It is well known that neck sprains resulting from a fall, drunken assault, or accident, are not followed by chronic pain if litigation is not involved. Similarly, patients subjected to the extensive soft tissue damage of surgery on the cervical spine are not prone to a stiff painful neck lasting more than a week or two. In a country where there is no expectation of symptoms or disability, and where a minority of car drivers are insured for personal injury, acute pain after whiplash is frequent but short lasting and self limiting. These recent studies cast grave doubt on the clinical validity of the "chronic whiplash syndrome".³⁴ For doctors to provide patients and Courts with a mechanism or validation of chronic pain they must consistently show a morbid process—anatomical, physiological, biochemical, or psychological—that is sufficient cause. If such evidence is not available, the balance of probability weighs heavily against causation of protracted symptoms by injury. If the pathogenesis or mechanism of what is termed "chronic whiplash syndrome" cannot be shown, then its existence is doubtful. Further opinion is pure conjecture, best avoided in science, best decided by the Courts.

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