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The myth of chronic whiplash syndrome

JMS Pearce*,[1]

[1]*304 Beverley Road, Anlaby, East Yorkshire*

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Introduction

Nobody doubts that if the neck is injured in a mechanical sprain that it will cause local pain, soreness, tenderness and stiffness. The term neck sprain is more accurate and therefore preferable to the term whiplash injury with its emotive associations that prejudge the issues.[1]

The term, introduced by Harold Crowe[2] in 1928 described the effects of sudden acceleration-deceleration forces on the neck and upper trunk due to external forces exerting a 'lashlike effect'. It referred to a mechanism, not to the pathological or clinical sequelae. Crowe claimed that prolonged distress and disabilities often lasted for several years. Bosworth took the opposing view; 'The neck is not a whip... The diagnosis is vague and thoroughly unscientific... to the honest, a bulwark behind which ignorance skulks; to the dishonest a mirage with which to confuse and deluge...'.[3] The term became popular after Gay and Abbott's paper centred on car accidents with rear end shunts in which they mistakenly concluded that the initial impetus pushed the victim's head forwards.[4]

Definition

If correctly defined as a soft-tissue injury of the muscular-ligamentous structures supporting the spine, whiplash injury is no different from other simple sprains, where there are ligaments, muscles and tendons adjacent to bones or joints. Neck sprains or uncomplicated whiplash injury are defined by grades I and II of the Quebec classification (Table 1). It is known that such soft tissue injuries heal quickly and that with the brief rest that nature compels by the restrictive role of pain, symptoms quickly disappear. If for example a sprained ankle has not largely resolved within 3–6 weeks,[5] it is likely that complications have arisen, eg, a disastasis of the joint, or an unrecognised fracture. There is no *a priori* reason to believe that the neck behaves differently.

Yet it has become accepted that a small but significant proportion of patients (variously stated as 2–10%) with whiplash injuries have continued pain and stiffness, and often a large number of other symptoms for 1–2 years. Further, there is evidence that if a patient is symptomatic after 1–2 years he or she will remain so indefinitely. This paper is confined to the 'chronic whiplash syndrome' conventionally defined by symptoms persisting more than 6 months after the injury.[6,7]

Complaints without objective damage?

In a medicolegal context, it is inappropriate for experts to accept all complaints at face value; each symptom requires a scrupulous appraisal of its cause, and attribution. To accept chronic whiplash symptoms in the absence of demonstrable causation is to be uncritical. This is because: (a) acceptance rests on purely subjective statements by the patient, and (b) such patients are rarely encountered in out-patient clinics or in settings where there is no possibility of compensation through litigation.

Despite this, certain expert's acceptance of complaints at face value have filtered into the Courts, which in turn have become accustomed to the incondite notion of chronic pain and disability, which then reinforces these expert opinions in a vicious circle. The expert's professional standing is then open to suspicion. Unable to demonstrate an objective mechanism they sometimes presume to categorise other opinions as extreme, callous, or unsympathetic to the claimant, whereas the Courts demand strict impartiality based on a corpus of established medical knowledge, not on speculation or conjecture. Increasingly, Courts insist on objectivity from experts. Those submitting explanations based on mere conjecture or bias may be asked to justify them.

The controversy is whether such trauma, with little or no demonstrable injury, can cause persistent symptoms (so-called 'late whiplash syndrome'). In

*Correspondence: 304 Beverley Road, Anlaby, East Yorkshire, HU10 7BG

Table 1 Proposed clinical classification of whiplash-associated disorders. The Quebec classification[7]

Grade
0 = No complaint about the neck. No physical sign(s)
I = Neck complaint of pain, stiffness or tenderness only. No physical sign(s)
II = Neck complaint AND Musculoskeletal sign(s)[a]
III = Neck complaint AND Neurological sign(s)[b]
IV = Neck complaint AND Fracture or dislocation

[a]Musculoskeletal signs include decreased range of motion and point tenderness. [b]Neurological signs include decreased or absent deep tendon reflexes.

the face of recent, scientifically established evidence, I shall attempt to examine fairly the view that chronic whiplash syndrome exists, is responsible for chronic pain and disability, and is just grounds for financial compensation in personal injury claims.

Imprecise definitions and absence of non-litigant controls, and uninjured controls seriously flaw much of the early literature. Recent papers, not marred by these major defects have shown that when there is no system for compensation to colour or prejudice the patient, the duration of neck symptoms is not longer than 3 weeks, and on average is about 3 days.[8] Similar results are emerging from studies in Germany[9] and Greece.[10] In clinical practice, we rightly start the history by accepting that the symptoms reported by patients are the truth. It is an unwritten contract that the patient says: 'I am ill, and in order to help you to diagnose and treat me effectively, I will tell you my complaints as accurately and as truthfully as I can'. In return the doctor accepts this at face value, unless there are gross inconsistencies or symptoms that on investigation have no explanation based on physical or psychological illness.

However, in medicolegal practice, which is the principal setting for claimants of chronic whiplash syndrome, such a contract does not exist. The doctor does not, or should not, act in his normal role, but as an independent and impartial assessor of the claimant, with duties solely to the court, not to the patient. Indeed, the claimant is not a patient. Many physicians and surgeons have grave difficulties in adopting this impartial role, so alien to our normal job of doing everything possible to sympathetically help the patient.

When dealing with personal injury cases, many doctors seem to ignore the fact, quite obvious to the man in the street, namely that if successful, the claimant will receive a large sum of money, a sum often much larger than anything he has previously handled. It is overweeningly naïve to ignore this. It is more naïve to neglect that the plaintiff is backed, advised and prompted by his solicitor or trade union representative whose rightful job it is to enhance his claim as much as possible.

Can uncomplicated neck sprain cause chronic incapacity?

What is the evidence that an uncomplicated neck sprain can cause chronic symptoms and incapacity? Critical to accurate diagnosis is accurate definition of what is and what is not a whiplash injury.[6,8,11–13]

In a common usage, the term whiplash applies to uncomplicated soft tissue injury, and not as originally proposed by Crowe to the hypothetical mechanism of the injury. There are neck injuries that result in fractures of vertebral bodies, transverse or spinous processes; there are injuries resulting in fracture-dislocation and paraplegia or tetraplegia. None of these are whiplash injuries by definition. It is now accepted that when a major mechanical force is abruptly applied to the spine the vertebral bone will break or the joints will dislocate before the fibro-elastic disk will rupture or herniate.

But, many older published series include such cases and paint a different picture with a gloomy prognosis. Norris's series, updated by Gargan and Bannister[14] for example, showed only 56% pain free at 1 year, and after 2 years symptoms did not alter. However, 45% had paraesthesiae, 42% back pain, and 14% auditory symptoms: not a representative series of uncomplicated whiplash. Similarly, Hohl[15] reported 55% symptom free at 1 year; but Deans[16] show: 49% symptom free at 3 months and 96% free or with occasional pain at 1 year. There are many similar papers. But selection of patients and methodology are faulty. As the Quebec Task Force report[7] concluded: 'Almost all studies are flawed because they include patients with complicating radiculopathy, disk lesions, facet joint injuries, and psychological illness'. Bogduk and Lord (who support the notion of the chronic whiplash syndrome), state: 'Systematic reviews paint a poor picture of the quality of literature upon which many conventional, conservative therapies are based. Conclusive scientific data are lacking'.[12] Stovner's thorough review reaches the same conclusions. Such patients have to be excluded from this category to avoid hopeless confusion over several different pathological entities that have in common only a whiplash mechanism. In the Quebec schema: 'symptoms and disorders that can be manifest in all grades include deafness, dizziness, tinnitus, headache, memory loss, dysphagia and temporomandibular joint pain, all remote from the cervical spine and defying rational explanation founded in the known pathology. The evidence that chronic symptoms may follow such injuries (Types I and II) is contaminated by a literature that includes a large percentage of cases falling into grades III and IV, which by general agreement fall outside the whiplash syndrome, and therefore confound analysis.

In Western society where access to compensation is almost universal, though within differing legal frameworks, there remain those with complaints after 6

months, which tend to persist indefinitely, even after compensation has been settled.

Explanations for chronic whiplash symptoms

Only four explanations are possible:

- Organic pathology caused by injury
- Pre-accident symptoms which have continued
- Psychological illness in response to injury
- Exaggeration and malingering

Organic factors
Modern clinical methods enhanced by sophisticated and sensitive imaging by MRI,[17,18] have shown no persistent pain-producing lesion attributable to the injury.

MRIs obtained shortly after and at 6 months after the crash were assessed independently by two radiologists for evidence of fracture or other injury; loss of lordosis and spondylosis were also recorded. Initial MRI was performed on 29 patients, of whom 19 had repeat studies at 6 months; 48 examinations were thus examined. Apart from spondylosis and loss of lordosis, only one abnormality was detected: an incidental intramedullary lesion consistent with a small cyst or syrinx. There were no statistically significant associations between the outcome of injury and spondylosis and loss of lordosis. No significant changes were found when comparing the initial and follow-up MRI.[18] There are now over 400 published examples of MRI of the brain and neck[17,19] as well as Brainstem Auditory Evoked Potentials studies. None shows unequivocal pathology caused by the injury.

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. But, Yarnell and Rossie in patients with severe debility at 12 months, concluded: 'In the subacute period, neurological examination, imaging and clinical electrophysiological studies were unable to localise, structurally or functionally, the source of the [cognitive] dysfunctions.[20] Similarly, of 68 patients with symptomatic cervical whiplash injuries, plain X-rays, EEG, computerised tomography and radionuclide brain scans failed to demonstrate associated structural abnormalities.[21] Sophisticated 18fluorodeoxyglucose positron emisson tomography (PET), similarly did not allow reliable diagnosis of physiological or metabolic disturbances in the brain for individual patients.[22]

Cervical spondylosis It is essential to correlate the relevance of investigations with clinical findings, since 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, osteophytes or spinal stenosis by the age of 40; this increases to 50% at the age of 60.[23] Thus the demonstration of pre-accident spondylosis is common, and in certain subjects it may explain some of the apparently chronic symptoms. There is much evidence that the spondylotic process is not accelerated by soft tissue sprain, though the notion is controversial.[24] Spondylosis may increase the liability to transient neck pain. Jarring of an already entrapped root can cause radicular pain, but unless there is evidence of neural damage, ie, new root or cord signs or of bony damage, the effect is transient.

Lord *et al*. have claimed 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.[25] Percutaneous radiofrequency neurotomy has therefore been used by the same authors in the treatment of pain from the cervical zygapophysial joints, but the results, they say, have been variable,[25] modest and not compelling.[27] Similarly, they acknowledged their failure with steroid injections into zygapophysial joints, third occipital neurotomy, 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? Their small numbers of selected case may have been inadvertently confounded by cases carrying factors unrelated to the whiplash injury in question.

We know that symptoms due to pre-existing spondylosis commonly continue, often intermittently, and may worsen irrespective of the trauma. This accounts for some of the apparently chronic neck pain.[13]

Chronic pain syndrome This is often declaimed as the explanation for pain in the absence of organic aetiology. The term is descriptive, but it neither explains nor validates the mechanism. Chronic pain syndrome is predicated on 'maladaptive pain typically the result of damage to the nervous system'[28] peripheral or central, and is known as neuropathic pain. It embraces a combination of negative symptoms or sensory deficits and positive symptoms including abnormal sensations paraesthesiae and dysaesthesiae, which are resistant to non-steroidal anti-inflammatory drugs (NSAIDs) and to opiates. Two pain patterns are recognised. Stimulus-evoked pain (mechanical, thermal or chemical) is typical of peripheral nerve damage and presents with *hyperalgesia*, increased pain response to a suprathreshold noxious stimulus, and *allodynia*, pain induced by non-noxious stimuli. It is mediated principally by nociceptor C fibres and large myelinated Aβ fibres. More commonly, stimulus-independent pain, a persistent lancinating, burning, can occur resulting from spontaneous firing of C fibres and possibly of sympathetic nerves. Central pain is

characterised by spread of pain beyond the territory of the damaged nerve, an increased pain response to suprathreshold input, and lowering of the previous pain threshold, ie, central sensitisation. Unfortunately, and importantly, there is no evidence for peripheral nerve or dorsal horn damage, nor of neuropathic pain in whiplash patients with soft tissue injury.[28] Therefore, such interesting theoretical considerations are inapplicable. This may seem like an attempt to explain *obscurum per obscurius*. Therapeutic neurosurgical lesions have a negligible role.[28]

Pre-accident symptoms

Under-reporting of complaints before an accident is frequent. One Norwegian study 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[26,29] may result from recall-bias or by denial in a medico-legal context. In either case, continuing symptoms may erroneously be attributed to the accident.

Pre-accident symptoms are common. A random study of 10 000 adult Norwegians showed that 34.4% had experienced neck pain within the last 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'.[30]

Psychological sequelae

These are common. Anxiety, phobic states and depression can magnify the intensity of complaints, but most patients are reassured that they have not sustained serious damage, that they are not in danger of physical handicap of spinal cord injury, and the initial anxieties generally settle in days or weeks. Inappropriate medical caution or the threat that 'some people go on for years in pain, and can't work again,' serve only to falsely increase fears and depression; they constitute iatrogenic psychogenic illness, not psychopathology directly stemming from the accident. Bogduk and Lord[12] report: 'chronic neck pain after whiplash is not psychogenic, and psychologic distress is secondary to the pain'. Smed showed[31] that one month after the accident, 85% of the patients had resumed work. Subjective cognitive disturbances, however, were frequent but unrelated to test performances, which were within the normal range. Patients reporting stressful life events unrelated to the injury had more symptoms and elevated levels of distress. At follow-up their distress was unchanged, but subjective cognitive function had deteriorated. He concluded: 'stressful life events unrelated to the accident and a high level of distress 1 month postinjury may augment the risk of late whiplash syndrome'.

In another study,[32] 34 consecutive cases of whiplash injury were examined clinically within 14 days, after 1 month and finally 7 months postinjury. MRI of the brain and the cervical spine, neuropsychological tests and motor evoked potentials (MEP) were done 1 month postinjury and repeated after 6 months, if abnormalities were found. 'The total recovery rate (asymptomatic patients) was 29% after 7 months. MRI was repeated in six patients. The correlation between MRI and the clinical findings was poor. Cognitive dysfunction as a symptom of brain injury was not found. Stress at the same time predicted more symptoms at follow-up. All MEP examinations were normal. In this study, long-lasting distress and poor outcome were more related to the occurrence of stressful life events than to clinical and paraclinical findings'.

Psychological distress can serve to lower the threshold to pain; conversely, in others, psychological distress is a consequence of chronic pain.[33] Seldom does one encounter examples of uncomplicated post-whiplash pain alleviated by invasive procedures, but if chronic pain occasionally subsides, neurotic and depressive complaints tend to disappear. There are genuine instances of psychogenic illnesses. But, since Radanov *et al*.[34] found that psychosocial factors at injury do not predict the outcome, though '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 to 126 accident victims with multiple injuries without serious head injury.[35]

Thus, in whiplash, there is no general basis for regarding chronic pain as a somatic manifestation of psychological injury derived from the accident. But psychosocial, rather than psychogenic issues determine the behaviour of the accident victim, his attribution of symptoms after the injury and his expectation of after effects. Such factors are assessed and manipulated by both lawyers and doctors. In clinical practice some doctors may act as the person who legitimises the symptoms with an arbitrary diagnosis (eg, chronic pain behaviour) and thereby permits the claimant entry into the chronic sick role as a chronic pain sufferer.[36] The diagnosis of chronic pain syndrome or chronic pain behaviour is often accompanied by a poor prognosis and by many fruitless, complicated treatments: cognitive-behavioural therapy, counselling by social workers and psychologists, psychotropic drugs, acupuncture, transcutaneous nerve stimulation (TENS), and not infrequently surgery of dubious value.[28] Symptoms sometimes fleetingly improve for a week or two after dramatic measures, but almost invariably quickly return, to the disappointment and resentment of the claimant.

Pre-accident neurosis or depression often colour the description of complaints, but that is not attributable

unless we can demonstrate a relevant psychological deterioration. As one authority 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'. . . [37]

It is part of normal behaviour and experience to feel irritable, frustrated and fed-up in relation to the daily problems of life; but it is foolish to regard such feelings as signs of clinical depression.

Psychologists' assessments are often appended to claims. Many rely more on a succession of standardised scales, eg, for depression, general health, post-traumatic stress disorder, than on clinical features individual to the patient. Tests that rely on questionnaires, provide a large number of leading questions, which suggest positive responses. The General Health Questionnaire (GHQ), for example, produces abnormal scores in 6.5% 'healthy' males and 19.6% 'healthy' females[38] another study showed 33% have an abnormal score, yet without psychological morbidity.[39]

Many patients quickly learn the expected response, and sadly, this adds to their distressing symptomatology. Since they are not trained to judge medical, physical and radiological signs, psychologists' appraisals can lack objectivity. Faust, a forensic psychologist believes that, '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'.[40]

Exaggeration

This is probably present to some extent in many personal injury claims. It is part of a lawyer's job to do the best for his client, and in this way symptoms may be suggested, enhanced or moulded to legal expectations. Malingering is an emotive term, and is rarely capable of proof. Without unequivocal evidence, it is better not used. But, spurious 'inappropriate' signs, or marked discrepancy between accidental injury and subsequent complaints and incapacity makes some degree of exaggeration probable. It should, when present, be highlighted by physicians, to assist the Courts in their judgements. Conscious simulation of illness or attempted deception are not interpreted as psychological illness.

The expert witness should regard exaggeration as probable,[8,36,37] if the following features are present:

- When symptoms are discordant with the injury;
- When restricted spinal movement is discrepant with the evident tissue damage;
- When there are 'spurious, false, or inappropriate' physical signs;
- When analgesics, collars and a wide range of physical (pain clinic) therapies fail to produce substantial, lasting relief: and
- When observed physical activities (by witnesses or video observation) are variable and inconsistent with clinical signs and behaviour during examination.

Deliberate exaggeration can be motivated by financial reward, increased attention and sympathy from family and friends who are often unwittingly entwined in a complex social disorder of assumed invalidity. This is often called 'sick role', 'chronic pain syndrome', or 'illness behaviour'. But although the social pressures and probable financial rewards are persuasive forces, the final choice to adopt the sick role is one that can only be made by the patient. To be successful, and to achieve the goals of illness behaviour, it has to become a way of life. It is thus understandable that claimants wear this mantle of chronic pain and disability that sadly, so rarely makes for happiness or contentment. But the decision to do so is theirs, and it is a decision made deliberately. The family and friends come to accept the sick role behaviour, and knowingly or unknowingly become part of the social pattern determined by the new adopted lifestyle.

Plaintiffs and experts may mislead not only their families and practitioners, but also Judges. They find it difficult to imagine that a claimant will submit to ill-judged surgery. Surgeons are commonly persuaded to operate on such patients in the altruistic endeavour of doing something to try to help them, but benefit seldom results. Patients often submit to surgery and other physical therapies if the perceived rewards are sufficient. They may abandon worthwhile and remunerative work without adequate medical cause to do so; Courts are then asked to enhance payment for future loss of earnings. The undoubted fact that there is a large number of middle aged people suffering from frequent neck pain and often headaches who are able to continue their normal job is easily overlooked. Huge payments for loss of earnings may be apportioned by a sympathetic Judge to a pleasant and plausible plaintiff who declares her unending devotion to the work she loves and the frustration of being incapable of resuming such work. Sometimes what starts as deliberate exaggeration becomes an adopted way of life that persists after legal settlement.

A recent study by Schmand *et al*. has indicated that the prevalence of malingering or cognitive underperformance in late post-whiplash patients is substantial, particularly in a litigation context.[41]

Attribution

It is scientifically sound to require that a causal relationship between the trauma mechanism and the symptoms and signs is firmly established.

Construct validity refers to the degree to which theories about causation of a disease is supported by empirical evidence. The basis for a cause-effect relationship[42] (that two factors injury and chronic

pain seem to be associated), has been based for the most part on purely descriptive evidence in patients from different countries who seek to relate their symptoms to a whiplash injury. Statisticians accept that descriptive studies may be used to *formulate* hypotheses concerning causal relationships, but they are not suited to *test* the adequacy of these hypotheses.[6] The following criteria for assessing whether association with an environmental factor is the cause of a disease have been proposed by Bradford Hill and others.[43,44]

Strength means that a strong statistical association is better evidence for causality than a weak one with a retrospective cohort study design no statistically significant positive correlation between rear-end car collisions and chronic neckache or headache has been found.[6,8]

Dose-response relationship means that large exposure to the cause should be related to a large effect and vice versa. In the literature this relationship has been investigated but not demonstrated in whiplash. Radanov *et al*.[45] did not find any correlation between pain duration and the patients' perception of the severity of the car damage or accident. Several studies have shown no clear correlation between duration of pain and the use of headrest, which one would expect, since the forces acting on the neck are reduced by the headrest.

Reversibility means that fewer incidents of exposure to a cause should lower rates of the disease. There seems to be a steady increase in the incidence of the whiplash syndrome in a period when there is a decrease in the incidence of other injuries resulting from car accidents.[46] This argues against a causal relationship. Similarly, the use of seatbelts[47] prevents fractures of the odontoid process in the neck but increases the frequency of whiplash symptoms.[6]

Temporality described the obvious principle that cause must precede effect. Subsequence does not imply consequence. It is often argued that all symptoms started after the accident, and that the victim of injury never had such symptoms before. It is also common to hear claims of symptoms beginning over 48 h, often weeks after injury, which refutes causation according to the criterion of temporality. It is well known that medical records show that about 15% of patients denying any previous symptoms have entries for pre-accident, similar symptoms in their medical records.[46,48] The occurrence of previous whiplash injuries is present in a considerable number of claimants. Such evidence would at least indicate that isolated trauma might not be sufficient to cause of chronic symptoms.

Consistency means that several investigations in various groups of patients produce the same conclusion. Although chronic whiplash syndrome has been described in large numbers in various countries, there are big differences in the prevalence and incidence of whiplash in different countries. This suggests it is related to social and legal expectations and less likely to cultural variation.[6,49] There is a marked lack of consistency in associated symptoms published in various series, particularly the inclusion of: dizziness, vertigo, temporomandibular pain, arm pain, paraesthesiae, fatigue, backache and other non-specific symptoms.

Biological plausibility Symptoms after a potential cause must arise in a fashion consistent with the known effects of the causal agent in producing a relevant lesion. The occurrence of chronic pain and disability in the absence of demonstrable pain-producing pathology is intrinsically implausible. Herniated cervical discs occur in *asymptomatic* patients, and thus are not necessarily caused by injury when found in patients with whiplash syndrome. Valid, temporal relation is a necessary condition, but is in itself only a weak indication of causation. The temporal relation may be coincidental, or the injury may act only as a transient aggravating or precipitating factor for an underlying disorder that would have become symptomatic in any event.

Experimentally induced whiplash in animals and cadavers is relevant only if lesions or difficulties observed can be shown in human patients with whiplash, for then the biological plausibility of the syndrome would be enhanced. A convincing animal model is impossible because patients with whiplash have mostly subjective symptoms not replicable in animals.

Assessments of the velocity of impact relate to the mechanical forces applied to both vehicles and passengers. How rare it is to see the occupants or driver of the impacting 'offending vehicle' with any neck symptoms, yet they too have sustained a reciprocal force, acting in the opposite direction. It has been calculated that for the *possibility* of all but mild and short-lived symptoms, the change in velocity (delta V) for the struck vehicle should exceed 17 km/h, equivalent to a striking speed of 26 km/h if the two vehicles are of similar weight in a rear-end collision. For a sideways or front-on impact delta V should exceed 34 km/h.[50] A crucial observation is that despite thousands of experimental collisions there is not one documented example of chronic whiplash symptoms.[8]

The role of litigation has caused much controversy. No connection has been found between the timing of compensation and resolution of symptoms.[11,51] Seeking economic compensation has been found to be a bad prognostic sign, but this does not establish that most litigants are malingerers.[47] Time spent waiting for a hearing and delays in the medical and legal assessments can prolong and intensify concern. However the overwhelming prevalence of 'chronic cases' in litigants is highly significant, and to ignore the effects of financial rewards afforded by that process is a biased, uncritical approach leading inevitably to serious errors of judgement.

The common opposing arguments can be simplified as shown in Table 2.

Table 2

Chronic symptoms are attributable	*Chronic symptoms are not attributable*
Chronic symptoms lasting more than 6 months are the result of an uncomplicated whiplash injury.	Chronic symptoms lasting more than 6 months are not the result of an uncomplicated whiplash injury.
The complaints and disabilities are consistent with the injury.	The complaints and disabilities are discrepant with the injury.
Although there are no signs of lasting tissue damage, clinical or radiological, to explain the symptoms, the patient is genuine, the pain and disabilities are real.	There are no signs of clinical or radiological lasting tissue damage to explain the symptoms.
They derive from an ill-understood chronic pain syndrome with chronic pain behaviour that accounts for the clinical features.	Suggestions of chronic pain syndrome with chronic pain behaviour only describe this picture without providing a mechanism or validation.
Psychological factors, if present, are the consequences of the accident.	Any psychological features apparent are not sufficient cause of the clinical features and disability.
	The extent of exaggeration and the possibility of malingering are matters for the Court to judge.

Conclusion

After nearly 50 years of acceptance of *chronic* symptoms after a simple mechanical neck sprain, the validity of this syndrome has been seriously questioned. Careful assessments have shown no evidence of persisting physical injury as sufficient cause of continuing chronic symptoms. A consensus of disbelief is evolving. There remains a lack of agreement about which of the multitude of symptoms attributed by certain authors to whiplash are in fact caused by the injury and not by other mechanisms. Chronic complaints can be genuine, but there is almost always a valid explanation that is not related to the injury.

1. It has been shown that many of these symptoms are quite common in the random population who have not suffered a neck sprain.
2. Continuation, without change, of pre-accident symptoms, and of cervical spondylosis accounts for some. The effects of the whiplash injury are superimposed, but disappear within days or weeks; after that period the pre-accident symptoms continue and are confused with the effects of the injury.
3. In exceptional cases, the emotional impact of a horrendous accident whereby the victim has suffered other major injuries, or someone has been killed, explain an attributable, severe neurotic or depressive illness.
4. More often there is deliberate exaggeration of symptoms. That is entirely understandable in the context of potentially large financial rewards, yet is rarely considered by professionals enjoying an elevated status and income in comparison to many of these plaintiffs.

Chronic whiplash now emerges as an over-diagnosed 'pseudosyndrome' similar to 'Myalgic encephalomyelitis', chronic abdominal pain, coccydynia, temporomandibular joint dysfunction, and fibromyalgia, for which exhaustive medical investigations from many disciplines have failed to show a scientifically valid basis. This group of labels recalls the nineteenth century medical nomenclature such as miasma, chills, or nervous debility that now have no useful place in rational medicine. There appears no objective basis for the concept and no coherent or meaningful pathophysiology has been demonstrated as its cause. Many treatments including physical therapies have not withstood the scrutiny of scientifically controlled trials, and indeed the Quebec taskforce consensus[7] suggests that the more treatment given, the longer symptoms take to recover. The Norwegian and Lithuanian data in over 820 patients and controls in which compensation was not a possible factor provides sound evidence of the short-lived nature of this common injury, and the lack of lasting disability.

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