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Low velocity whiplash injury (LVWI)

Whiplash injury is surrounded by controversy in both the medical and legal world. The debate on whether it is either a potentially serious medical condition or a social problem is ongoing. This paper briefly examines a selection of studies on low velocity whiplash injury (LVWI) and whiplash associated disorder (WAD) and touches upon the pathophysiological and epidemiological considerations, cultural and geographical differences and the effect of litigation on chronicity. The study concludes that the evidence for significant physical injury after LVWI is poor, and if significant disability is present after such injury, it will have to be explained in terms of psychosocial factors.

Whiplash injury puts a significant financial burden on the UK economy. According to the Association of British Insurers the cost is over £2 billion per year in the UK. It causes substantial controversy in both the medical and legal world. Debate rages between it being a potentially serious medical condition at one end of the spectrum and a social problem at the other. The term whiplash refers to the mechanism of injury which is an acceleration/deceleration transfer of energy to the neck which may result, amongst other mechanisms, from motor vehicle accidents. The Quebec Taskforce definition of whiplash associated disorder (WAD) is widely recognised. It is believed that the differential acceleration of the head and upper torso in relation to the trunk as a result of the sudden change in velocity (Δv) is responsible for causing the whiplash injury. Paradoxically, claimants/patients involved in low speed accidents often report much higher levels of neck

pain than those involved in high velocity accidents and those who are victims of polytrauma.

QUALITY OF RESEARCH

Enthusiasts of whiplash injury as a distinct clinical condition argue that injury can occur irrespective of the magnitude of the collision. They believe that the immensity of the force has little to do with actual injury. Such arguments are usually based on the paper by Brault et al.¹ Brault's group looked at 42 subjects who were exposed to controlled low speed rear-end automobile impacts. Approximately 29% and 38% of those exposed to impacts of 4 kph and 8 kph, respectively, experienced WAD symptoms (not proven injury), with neck pain and headaches predominating. The authors (none of them doctors) therefore concluded that this established a causal relationship between low velocity rear-end collisions and clinical symptoms and signs. This research is still cited by a proportion of the legal and medical profession as proof that LVWI

may lead to months/years of symptoms/disability without any attempt at scientific analysis of the methodology and validity of the article. Subjective symptoms reported by volunteers have often been misinterpreted by experts who quote the article as evidence of objective injury. There is no proven injury in any of Brault's subjects. Since the high prevalence of spinal pain in the general population, even without injury, is widely known from epidemiological studies, other potential causes of symptoms were ignored by Brault's study. The subjects who reported symptoms did so for one to three days. The study does not provide any evidence of long-term symptoms or prolonged recovery times after LVWI. This is one example but it exemplifies the main problem associated with the literature on whiplash injury. Older studies with poor methodology have been repeated in later subjective reviews without a critical analysis of the earlier paper having been performed. The flawed research is then passed on as evidence.

A landmark paper in 2001 by Castro et al.² demonstrated effectively the production of symptoms that are commonly associated with acute whiplash injury in volunteers by fooling them into believing that they had been in a collision when in fact they had not. Approximately 20% of the subjects exposed to the placebo rear-end collision described symptoms consistent with WAD even though no biomechanical potential for injury existed. They found that certain psychological profiles placed an individual at higher risk for this phenomenon. This paper immediately challenged the validity of the conclusions made by Brault et al.¹ Many of the whiplash studies suffer from methodological flaws such as selection bias, retrospective nature, sample size, suboptimal statistical analysis, lack of control group and flawed assumptions. There are literature reviews supporting the concept of WAD as a distinct entity³ and, conversely, reviews supporting the concept that there is little evidence to support whiplash as a clinical entity.⁴

SYMPTOMS AND SIGNS OF INJURY

A wide spectrum of symptoms is reported by people involved in low velocity collisions. It is suggested that less than 50% of those who suffer WAD make a full recovery and 4.5% are permanently disabled. However, it is difficult to explain the reported long-term symptoms and disability on the basis of a persisting physical injury. There is very little that is objective when dealing with a so-called soft-tissue injury to the neck. In Quebec grades 1 and 2 WAD, the injury cannot be proven objectively and claimants report pain, tenderness and other subjective symptoms which are difficult to prove and difficult to refute. Most studies dealing with symptomatology and prognoses only look at claimants, thereby introducing an immediate bias. There is no control group. In medicine, an objective sign is one that does not require a response from the person being examined. In other words, it can be detected without the subject being asked a question or being required to do anything, for example it could be skin rash, a cut, swelling or deep tendon reflex. In the medico-legal setting tenderness or weakness require a response and, in the latter case, an effort from the patient, and therefore are non-objective signs. Restriction of cervical spine movement with tenderness may be found in someone who has slept awkwardly and woken with a stiff neck. Therefore, it is important in medico-legal practice to appreciate that similar symptoms and signs may arise from various

different causes. An abnormal physical finding does not automatically mean that it resulted from an injury and does not always give us a clear structural diagnosis for the cause of that abnormality. It is also important to ensure that our instructing legal parties understand this.

PATHOPHYSIOLOGICAL AND EPIDEMIOLOGICAL CONSIDERATIONS

Disc degeneration is very common in the general population and may be present without causing any symptoms. Twin studies have demonstrated a significant genetic contribution to disc degeneration. There is only a weak correlation between radiological evidence of disc degeneration and neck pain. There is also little correlation between the severity of disc degeneration and the severity of pain. A high incidence of radiological abnormalities has been demonstrated on cervical spine MRI scans in younger individuals. Studies on patients with whiplash injuries have demonstrated a similar incidence of MRI abnormalities to that in the general population. Studies of cervical spines in individuals who have no complaints referable to the neck and no history of injury have demonstrated various different abnormalities such as flattening of the normal cervical lordosis (curvature) and disc degeneration without symptoms or evidence of injury in asymptomatic individuals. Therefore, these radiological findings are not necessarily associated with symptoms or pain and cannot be used to establish causation in a case where there has been a whiplash injury. In clinical practice there can be severe neck pain with normal radiographs and MRI scans and, conversely, grossly abnormal radiographs and scans with no neck pain at all. Controlled studies of symptomatic and asymptomatic patients have shown no difference in the rate of disc degeneration on MRI, and abnormalities on MRI are not generally seen after whiplash.

CULTURAL AND GEOGRAPHICAL DIFFERENCES

Chronic whiplash injury does not exist in all countries that have cars and road traffic accidents. Clearly such accidents occur universally but the recovery rate from the so-called acute whiplash injury varies considerably from culture to culture. It appears that in countries such as Greece, Germany and Lithuania those injured in this manner recover quickly. This begs the question whether there is any structural difference in the cervical spines of people in these countries. The work of Schrader et al.⁵ in 1996 suggested

that chronic symptoms were not usually caused by the road traffic accident. Expectation of disability, family history and attribution of the trauma were probably more important determinants in the evolution of the late whiplash syndrome. In a country where there was no pre-conceived notion of chronic pain after rear-end collisions, and thus no fear of long-term disability, and no involvement of the medical profession, insurance companies or solicitors, symptoms from whiplash injury were short-lived and did not seem to evolve into the so-called late whiplash syndrome. However, studies like this have been vigorously criticised for their poor methodology. The authors repeated the study with improved methodology and made similar conclusions.⁶ Despite this, their work has still come in for criticism.⁷ It is likely that an acute injury does occur even in countries like Lithuania but there is a distinct lack of patients reporting symptoms beyond one to four weeks. This would suggest that the acute whiplash injury is similar to a neck sprain which would not be expected to progress to chronicity.

WHAT IS THE NATURE OF WHIPLASH INJURY?

Sophisticated imaging techniques such as MRI and bone scans have failed to detect a specific pathognomonic and reproducible lesion in the vast majority of whiplash patients and therefore there is nothing to suggest that in the majority the injury is nothing more than a minor sprain. The general assumption is that Quebec grades 1 and 2 WAD represent a sprain. Most volunteers in whiplash experiments behave as if they have sustained a minor sprain, the effects of which are exhausted in a few days or, at most, weeks. MRI scans and bone scans readily detect abnormalities of the bones and joints of the cervical spine if there is anything beyond a minor injury. Studies of patients with whiplash injuries have routinely failed to identify any significant radiological abnormalities. In general terms, muscle and ligamentous sprains occur elsewhere in the body, can be demonstrated with MRI scans and get better with time. It is therefore difficult to understand from a physiological or pathological point of view why a sprain of the muscles or ligaments in the neck should give rise to symptoms which would persist for months or years. Additionally, ligament sprains in other locations broadly demonstrate a correlation between the severity of the trauma, the clinical signs and imaging findings. Logically, therefore, if MRI scans do not

reveal any evidence of an injury in the neck as such, that injury (sprain) must be of lesser severity than that observed in other common sprains. Bone scans have not detected muscle or ligament injury in whiplash patients. Studies by Ronnen et al⁸ and Borchgrevink et al⁹ performed within three weeks of the collision and two to four days of the collision, respectively, did not detect any trauma-related abnormality on MRI scans in whiplash patients. This has been confirmed more recently by Ulbrich et al.¹⁰

WHIPLASH AND CERVICAL DEGENERATION

There is no robust, high quality evidence that suggests that degenerative changes in the cervical discs develop any more rapidly in a patient following a whiplash injury than they do in the general population. This is confirmed by the work of Matsumoto et al.¹¹ Equally, there is nothing to suggest that an acute whiplash injury makes pre-injury degenerative change any worse. It has been suggested that the facet (zygapophyseal) joints may be the source of pain after whiplash injuries.¹² However, there have again been methodological criticisms of the research used in support of this contention. MRI scans have failed to detect any facet joint injuries in whiplash patients. Facet joint pain can also occur in patients who have no history of trauma. Therefore, we feel that the evidence that facet joint injury causes neck pain after whiplash injury is relatively weak.

CHANGE IN VELOCITY

Where it is difficult to find an objective injury help has been taken from the engineering world using the concept of delta v or head acceleration. The popularity of the concept of delta-v is that because there is no demonstrable injury it is applied to estimate the likelihood of an injury occurring. When the delta-v is lower than a certain threshold the whiplash mechanism does not occur and therefore an explanation (causation) for the patient's injury/symptoms is improbable. The change in velocity defined by delta-v gives an indication of the severity of the impact and has been used by researchers as a key predictor of the probability of injury. Research has suggested that the probability of neck injury is low with a delta-v of less than 5 mph and even when symptoms are reported at this change in velocity, they are transient, short lasting and resolve rapidly without treatment. Proponents of whiplash injury and those

who oppose the concept of delta-v argue that it cannot be accurately measured and whiplash experiments do not replicate real-life impacts. Experiments have been conducted on human volunteers under controlled conditions. The work of Brault et al¹ and Castro et al² have been alluded to already. Several animal and dummy experiments have also been conducted to study this matter using different types of acceleration. The threshold for minor symptoms lasting from hours to one day appears to be a change of approximately 5 mph (8 kph) for the target vehicle struck in a rear-end collision. As far as we are aware no volunteer has ever reported chronic pain following the acute injury in these whiplash experiments. It has been shown that the hyperextension/hyperflexion mechanism of the cervical spine which is assumed to cause injury does not occur for impacts which result in a delta-v of less than 8 kph.¹³ In McConnell's study,¹⁴ the authors showed that there was no forcible hyperextension of the neck in rear-end collisions. Using analysis of elaborate photography, the cervical flexion and extension were found to fall within their physiological limits. Similar results have been found in other studies. The forces generated in impacts resulting in a delta-v of less than 5 kph are not sufficient to cause the head of the occupant to make contact with the head restraint. An earlier study from Castro et al¹⁵ included MRI scans taken before and after the collision. The scans showed no change. They reported that five subjects described transient symptoms when the delta v exceeded 11.4 kph. They felt that the stress sustained in rear-end collisions was similar to 'bumper car' collisions, which they also studied in this report. Although such experiments have been criticised, they probably represent the closest approximation to collisions that whiplash patients experience in real life. There seems to be a large number of claims of whiplash injury by bus occupants even when a significantly heavier bus has been struck by a car of much lower mass. Dubois¹⁶ found that when a vehicle strikes a bus in a rear-end collision, in order for the threshold for injury to be reached a delta-v must be at least 5 mph. For a car that has an eighth of the mass of a bus this would require the car to be travelling at over 60 mph and this would usually be fatal for the car driver. When the car was travelling at much lower speeds the volunteers were unaware that a collision had taken place. Therefore, it is difficult to rationalise how an accident mechanism of this nature could cause any physical injury to the occupants of the bus.

EFFECT OF LITIGATION ON CHRONICITY

Opinion remains divided on the role of litigation in perpetuating the chronicity of whiplash symptoms. The effect of elimination of the litigation process in recovery after whiplash injury is also keenly debated. A recent systematic review from the Australian Centre for Economic Research on Health considered 11 studies and concluded that "there is no clear evidence to support the idea that compensation and its related processes leads to worse health."^{17,18} The same group also examined the contentious issue of whether symptoms settled after the legal case completed. They used regression analysis comparing those people where the litigation had settled with those where it had not. Their conclusion was that "claimants do not appear to be cured by verdict." However, the matter remains controversial.

BIOPSYCHOSOCIAL MODELS OF CHRONICITY

Various factors have been blamed for increased vulnerability of certain patients/claimants following a whiplash injury. These factors have included age, sex, position of the occupant in the vehicle, position of the head, lack of awareness of the impact and pre-existing degenerative change, among others. Other studies have suggested that none of these factors are relevant and the principal factors relating to chronicity are psychological.¹⁹ The main predictive factors for long-term pain and disability appear to be higher initial pain levels and psychological factors such as catastrophising, perceived injustice, post-traumatic stress symptoms and depression. A number of biopsychosocial models have been proposed to explain why some people who have been injured in road traffic accidents involving a whiplash mechanism develop chronic symptoms while others do not. These models suggest that while it may be possible for physical sources and processes to cause pain, it is the psychosocial factors that act to generate chronic pain. These models also suggest that symptom expectation, amplification and attribution may be more important in the genesis and persistence of symptoms in some whiplash patients. The biopsychosocial model accepts a physical reason as a cause of the original pain but the severity and duration is governed by psychosocial factors which influence the behaviour of the patients/claimants.

CONCLUSION

It is clear that controversy still exists in many different areas surrounding whiplash injury and there is a requirement for high quality research

in this field in the future. However, the evidence for significant physical injury after LVWI is poor, and if significant disability occurs after such injury it will probably have to be explained in terms of psychosocial factors rather than in biological or structural terms.

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