Persistent Myalgia Following Whiplash

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Persistent myalgia following whiplash is commonly considered the result of poor psychosocial status, illness behavior, or failing coping skills. However, there is much evidence that persistent myalgia may be due to neurophysiologic mechanisms involving peripheral and central sensitization. Myofascial trigger points may play a crucial role in maintaining sensitization. Recent research suggests that the chemical environment of myofascial trigger points is an important factor. Several consequences are reviewed when central pain mechanisms and myofascial trigger points are included in the differential diagnosis and in the management of patients with persistent pain following whiplash.

Introduction

Every society in the world is faced with the consequences of road traffic injuries [1]. Following the first documented motor vehicle death in 1869, road traffic fatalities have become a public health epidemic and are anticipated to be the third most common cause of death in 2020 [1•,2,3]. However, global research on the prevention and management of traffic injuries is still in its infancy, as is the body of knowledge dealing with the possible mechanisms of persistent pain and dysfunction following whiplash injuries. Much progress has been made in understanding the potential role of spinal zygopophyseal joints [4]. However, persistent muscle pain following whiplash often is not considered in the medical literature or in courtrooms and its underlying mechanisms are still poorly understood. People presenting with chronic muscle pain following whiplash often are viewed as compensation-seeking, poorly motivated, or psychologically distressed, which may have serious medical, legal, and economic consequences for all of those involved [5••].

Background

Due to the complexity of motor vehicle accidents, many disciplines are involved, including public health, medicine, engineering, biomechanics and biomechanical trauma, epidemiology, psychology, economics, law and law enforcement, insurance, health policy, and ergonomics. Medical specialties involved in the treatment of survivors of motor vehicle accidents include neurology, orthopedic surgery and neurosurgery, rheumatology, internal medicine and family practice, ophthalmology, radiology, psychiatry, dentistry, psychology and clinical social work, chiropractic, and occupational and physical therapy. Despite the wide variety of disciplines and medical specialties involved, there is little consensus about the etiology, identification, and management of persistent pain following whiplash.

Fortunately, most traffic accidents do not result in fatalities. Most patients recover fully and do not require any long-term medical treatments. However, up to 40% of those who sustain a whiplash injury are reported to develop chronic whiplash-associated disorder, which eventually may lead to permanent disability in 6% to 18% of cases [6–10]. A Swedish study showed that 16% of employed individuals did not return to their previous employment following a motor vehicle accident [11].

Reported symptoms following whiplash injury generally fall within one of three categories: physical or somatic symptoms, cognitive symptoms, and affective symptoms.

Several authors have suggested that persistent symptoms are not the direct result of whiplash injuries, but due to an expectation of disability, cultural context, a family history of neck pain, the presence of pre-existing symptoms, litigation and compensation potential, or psychologic distress [12–14]. Although it is conceivable in principle that litigation and settlements would be linked to poor outcomes, several studies suggested little or no correlation [7,15–19]. In a recent study, 34% of the subjects involved in a rear-end collision experienced pain and dysfunction 24 months after a collision. Only 7.7% of the subjects sought financial compensation, while none received any compensation for pain and suffering [15]. Studies in Lithuania and other countries suggested that chronic pain problems do not necessarily have to develop following road traffic injuries [12]. However, in a detailed analysis, Freeman and Croft [17] described critical methodologic flaws and concluded that only 1% of the subjects were selected appropriately for inclusion in the Lithuanian study. The included subjects were identified from police records regardless of whether they had complained of symptoms. The number of subjects complaining of neck pain after the accident was very low and thus continued to be low several months later.

Several studies have confirmed that cognitive impairments and psychologic dysfunction are common following whiplash injuries. Neuropsychologic evaluations of cognitive functioning revealed significant and persistent age-adjusted cognitive deficits, primarily in the areas of executive functioning and working memory, that are not detectable by positron-emission tomography and single photon emission computed tomography scanning [20]. Duckworth and Iezzi [21] found a significant correlation between high levels of post-traumatic stress symptoms and greater physical injury and impairment, greater psychologic distress, and greater use of maladaptive pain-coping strategies. When pain symptoms do not decrease over time, patients may become increasingly concerned about the prospects of long-term suffering and disability, leading to depression, anxiety, and fear-avoidance behavior [22•]. Once the pain complaint is resolved, the psychologic distress commonly disappears [22•,23]. On the other hand, it is important to realize that psychologic distress seen following whiplash can not always be blamed on the injury and may be due to life stressors regardless of pain [24].

Biomedical Model

The arguments used to dismiss patients' chronic pain complaints in the absence of "objective" medical findings reflect the biomedical model and appear to negate current insights gained from the pain sciences [25]. The biomedical model is the predominant model used in court rooms and in the biomechanical, engineering, legal, and medical literature to explain pain following road traffic injuries. This model assumes that complaints of chronic pain following a collision would be conceivable only if there were objective evidence of persistent bodily damage after an acute injury. In the absence of such objective data collected with standardized tests and measures, patients with complaints of persistent pain are more likely to get dismissed with their pain being attributed to their poor psychosocial status, illness behavior, or failing coping skills.

Strict biomedical reasoning often is used erroneously by biomechanical engineers and physicians hired as defense experts in court. For example, a biomechanical engineer may present convincing biomechanical data that, based on the weight on the mandible and the assumed load during a car crash, the temporomandibular joint could not have been injured. The engineer may add that the forces most likely to have occurred during the car crash are comparable with forces exerted during normal chewing or biting. If the jaw were to be injured during a motor vehicle accident, it would have needed to be exposed to excessively high rotational acceleration forces, which the engineer would argue are mechanically impossible considering the calculated change in velocity, impact forces, and crash characteristics established with widely used and accepted accident reconstruction methods. The engineer may argue that most people do not injure their temporomandibular joint during whiplash injuries, thereby suggesting that a particular plaintiff's complaints of chronic temporomandibular pain would be highly unlikely.

Although the engineer may present convincing arguments to the judge and a lay jury that a plaintiff is faking injury, the expert testimony nevertheless is fraudulent with commonly used defense fallacies [26]. When a defense witness establishes that there is little damage to the vehicle, it cannot be implied that no one could have been injured. Freeman [26] refers to this kind of reasoning as a "fallacy of distraction" or a "fallacy of authority," whereby the witness expresses an epidemiologic opinion based on limited mechanical data. The engineer in this example also would be guilty of a "fallacy of generalization"; based on mechanical data, the engineer is not qualified to render a medical opinion regarding the plaintiff's condition [26]. The engineer offered a limited number of options and did not consider studies that have demonstrated that individuals do develop chronic temporomandibular pain following whiplash despite seemingly contradicting biomechanical data [27]. Furthermore, the engineer did not include results from the pain sciences with various lines of evidence that chronic temporomandibular pain involves central and peripheral sensitization, hyperexcitability of central neurons, and dysfunction of the descending inhibitory pain modulation systems, even when radiographic studies fail to demonstrate tissue damage [28,29•].

Myalgia and Sensitization

The biomedical model does not consider evidence from the pain sciences, which have demonstrated that apparent insignificant injury can result in persistent and disabling pain in the absence of tissue damage. There is a growing body of evidence that chronic muscle pain following whiplash can be explained and managed best from a pain sciences perspective rather than from a strict biomechanical point of view.

Several studies have established that people involved in whiplash injuries commonly experience prolonged muscle pain and dysfunction [30,31,32•,33,34]. Schuller *et al.* [31] found that 80% of 1096 subjects involved in low-velocity collisions demonstrated evidence of muscle pain. Motor system dysfunction has been shown to develop as early as 1 month after injury and actually may persist in those who develop chronic muscle pain and in individuals who have recovered or continue to have persistent mild symptoms [35•]. Scott *et al.* [36] determined that individuals with chronic whiplash pain develop more widespread hypersensitivity to mechanical pressure and thermal stimuli than those with chronic idiopathic neck pain. The differences were attributed to changes in central pain processing and were found to be independent of levels of anxiety. Reduced cold tolerance in patients with chronic whiplash was confirmed recently by Kasch *et al.* [37], whereas Johansen *et al.* [38] already had established a reduced mechanical

pressure threshold. Patients with chronic whiplash pain featured muscular hyperalgesia in the neck and shoulder regions and in distant areas, such as the anterior tibialis muscle, in which patients usually do not experience any pain.

There is no question that people with persistent pain following whiplash suffer from widespread central hyperexcitability, which can cause seemingly exaggerated pain responses, even with low-intensity nociceptive input [39,40••,41,42]. Persistent pain following whiplash may start with the so-called "wind-up" of dorsal horn neurons and activation of *N*-methyl-D-aspartate receptors. These phenomena can lead to central sensitization and its hallmark characteristics of allodynia and hypersensitivity, which, in animal models, can persist even after peripheral noxious input has been eliminated. Persistent pain following whiplash thus can be considered a dysfunctional pain disorder [5••].

Myofascial Trigger Points

A recent study established that central hypersensitivity is most likely dependent on local nociceptive input [43••]. Although generalized widespread hypersensitivity has been confirmed in chronic whiplash pain states [39,40••,41,42], the mechanisms that underlie hypersensitivity in different body regions may be quite different [43••]. The researchers found different pressure pain thresholds in the neck regions of whiplash patients compared with healthy adults. However, in the toe region, there were no significant changes, which suggests that there are different central mechanisms involved [43••]. Furthermore, the researchers concluded that in the presence of ongoing peripheral nociceptive input, central hypersensitivity most likely will be maintained. On the other hand, if such peripheral nociceptive input could be identified and subsequently be eliminated, it is conceivable that the central sensitivity also can be reduced or eliminated [43••]. Studies have shown that in motor vehicle accidents, neck sprains and strains are the most common type of injury, comprising nearly 28% of all injuries treated in emergency room departments in 2000 [44]. The question then is, what structures or mechanisms in the neck region continue to provide ongoing nociceptive input in patients with persistent pain following whiplash?

One such source of ongoing peripheral input are the cervical zygopophyseal joints [4]. Another source may consist of cervical myofascial trigger points (MTrP), even though the scientific proof of their relationship to persistent whiplash pain is still in its infancy [25]. Although lesions of the cervical zygopophyseal joints indicate a pathoanatomic source of pain and dysfunction, MTrPs are considered a consequence of dysfunctional motor endplates [4,45]. MTrPs can be identified by palpation only. The interrater reliability of the identification of MTrPs has been established [46,47]. There are no other diagnostic tests that can accurately identify an MTrP, although new methodologies using piezoelectric shockwave emitters are being explored [48]. The minimum essential features that need to be present are a taut or hardened band of muscle fibers, an exquisitely tender nodule in the taut band, and the patient's recognition of the pain complaint by pressure on the tender nodule [49].

In a retrospective review of 54 consecutive chronic whiplash patients, Gerwin and Dommerholt [50] reported that clinically relevant MTrPs were found in every patient, with the trapezius muscle involved most often. Assuming that MTrPs may constitute a form of ongoing peripheral nociceptive input, inactivation of the MTrPs therefore would result in dramatic improvements [43••]. Following treatment emphasizing the inactivation of MTrPs and restoration of normal muscle length, approximately 80% of patients experienced little or no pain, even though the average time following the initiating injury was 2.5 years at the beginning of the treatment regimen [50]. All of the patients had been seen previously by other physicians and physical therapists who apparently did not consider MTrPs in their thought process and clinical management. The identification of MTrPs requires extensive training, not unlike other medical diagnostic procedures [46]. In the study by Schuller *et al.* [31], the most common finding in 1096 subjects involved in low-speed collisions was the presence of myogeloses, which may describe the same phenomenon as MTrPs [51]. Baker [52] reported that the splenius capitis, semispinalis capitis, and sternocleidomastoid muscles developed symptomatic MTrPs in 77%, 62%, and 52% of 52 whiplash patients, respectively.

It is hypothesized that MTrPs are a common phenomenon in peripheral and central sensitization [25]. On the one hand, MTrPs may be expressions of secondary hyperalgesia due to central sensitization [53]. On the other hand, MTrPs may be a common source of ongoing peripheral nociceptive input and, as such, maintain central sensitization, allodynia, and hypersensitivity [25,54]. Inactivation of MTrPs usually leads to an immediate reduction of pain and a restoration of function. By removing a source of ongoing peripheral nociceptive input, the dysfunctional processing of the nervous system may be corrected, resulting in an immediate improvement. Treatment options for MTrPs include manual trigger point release, neuromuscular therapy, dry needling, trigger point injections, and other specific modalities [48,55].

There is new evidence that MTrPs indeed are a potential source of ongoing peripheral nociceptive input. Studies by Shah *et al.* [56] at the US National Institutes of Health indicate that a number of biochemical alterations are commonly found at the active MTrP site using microdialysis sampling techniques. Among the changes found are elevated bradykinin, substance P, and calcitonin gene-related peptide levels and lowered pH when compared with inactive or asymptomatic MTrPs and with healthy control subjects. Any of these chemicals are known

nociceptive substances. Increased levels of calcitonin gene-related peptide and a lowered pH reduce the efficiency of acetylcholinesterase, which in turn may maintain MTrPs and therefore the ongoing peripheral nociceptive input. Gerwin *et al.* [57] addressed the possible consequences of these new findings in a recent article. One of the unique features of MTrPs is the phenomenon of the local twitch response, which is an involuntary spinal cord reflex contraction of the contracted muscle fibers in a taut band following palpation or needling of the band or MTrP. Current research suggests that a local twitch response is essential in altering the chemical milieu of an MTrP [56].

There are important consequences when central pain mechanisms and MTrPs are included in the differential diagnosis and in the management of patients with persistent pain following whiplash. Once structural lesions have been ruled out with magnetic resonance imaging, computed tomography scans, and radiography, clinicians should consider that MTrPs can contribute to and maintain central sensitization phenomena. Eliminating the painful peripheral input is likely to break the pain cycle, discontinue dysfunctional pain patterns, and facilitate the return to a productive and pain-free life. Adding the identification and treatment of MTrPs to the clinical toolbox can provide patients with hope and optimism. Physical therapy or chiropractic treatments that emphasize conditioning programs without paying attention to MTrPs may be too intensive and maintain painful conditions. Identifying signs of dysfunctional pain, including MTrPs, likely will meet patients' needs more closely [5••,50]. Several recent clinical papers support using an integrated approach that includes inactivation of MTrPs [58–60].

Conclusions

Many of the biomechanical testimonies and legal arguments used in court rooms become less relevant and can be evaluated based on their limited perspective. Of course, the medical evaluation should always include a search for lesions; however, in the absence of such lesions, patients should not be dismissed. Defense strategies should not be based solely on biomechanical analyses. When MTrPs are not considered in the etiology of persistent pain following whiplash, physicians are more likely to conclude that there are no objective findings that substantiate subjective complaints and dismiss patients as malingering, compensation-seeking, or psychogenic. Psychologic and emotional findings usually are the result of unresolved pain and disability.

Persistent muscle pain following whiplash is not commonly considered in the medical, engineering, and legal literature. However, it is likely that chronic muscle pain occurs frequently and may be due to MTrPs. MTrPs are hypothesized to be sources of persistent nociceptive input, contributing to wind-up and central sensitization [5••, 25].

The role of MTrPs in peripheral and central sensitization needs to be explored further. There is a need for well-designed studies that define the contributions of MTrPs to persistent pain following whiplash and other trauma.

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