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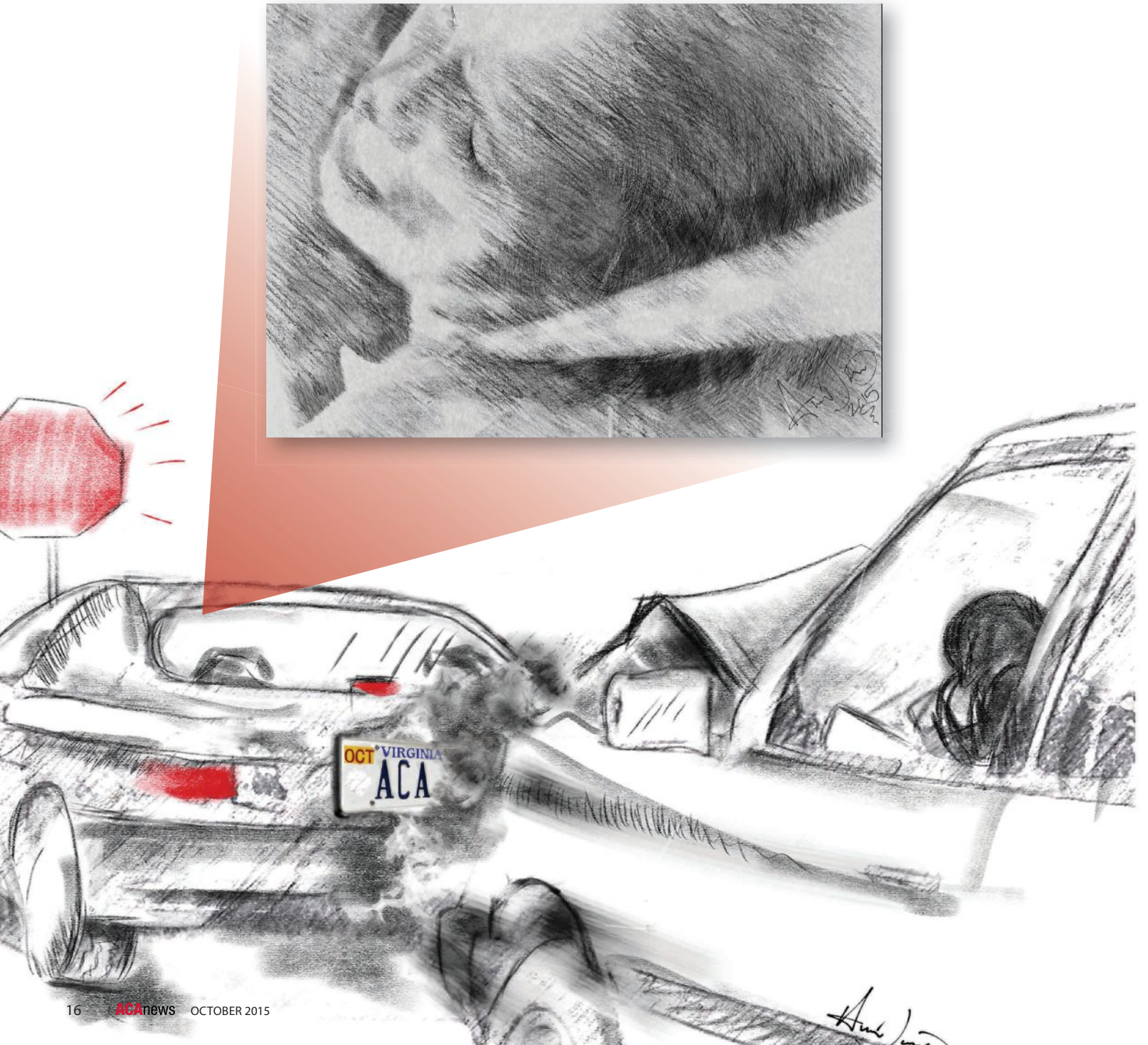
Whiplash Associated Disorders

The pathway from acute pain to chronic pain syndrome, a biopsychosocial dilemma.

By James J. Lehman, DC, and Anthony D. Nicholson, MChiro



SINCE CROWE FIRST COINED THE TERM "WHIPLASH" IN 1928,[1] IT HAS BEEN ABUSED BY SPINE CLINICIANS, MISUNDERSTOOD BY THE INSURANCE INDUSTRY, APPRECIATED BY PLAINTIFF ATTORNEYS AND CLARIFIED TO SOME DEGREE WITH RECENT RESEARCH. UNFORTUNATELY, MORE THAN 40 PERCENT OF INDIVIDUALS INJURED IN MOTOR VEHICLE COLLISIONS SUFFER FROM POSTTRAUMATIC, CHRONIC PAIN.[2]

The ICD-9-CM diagnostic code 847.0 was used for whiplash (it has changed under the ICD-10 coding system effective Oct. 1, 2015). I (Lehman) was introduced to the whiplash-type injury at a Parker seminar by Frigard and Belli. It is my understanding that L. Ted Frigard, DC, with his book *847.0, the Whiplash Injury*, published by Richmond Hall Inc. in 1970, introduced many chiropractic physicians and attorneys to the term whiplash.

Dr. Frigard was invited to address California law students at the first annual Practical Law Institute in Riverside, Calif., by his close friend, the renowned attorney Melvin Belli, chairman of the institute. Dr. Frigard's charge was to familiarize attendees with the developments in the field of chiropractic and the chiropractic management of the whiplash injury. The list of speakers at this inaugural meeting included F. Lee Bailey, Naval Judge Advocate Joseph McDevitt, former Governor of California Pat Brown and Melvin Belli.[3] It appears that Dr. Frigard was the first DC to introduce California lawyers to chiropractic management for patients suffering with pain as a result of the whiplash injury.

Another book, *The Cervical Syndrome*, written by Dr. Ruth Jackson and published in 1966, described pertinent information regarding the evaluation and management of the cervical syndrome and whiplash injuries.[4] Fortunately, I still have my original copy of the final edition. To author a book describing the conservative care for patients suffering with a cervical syndrome or whiplash associated disorders (WAD) was quite a bold endeavor for a female orthopedic surgeon at that time. [Also realize that in 1966, a very popular movie, *The Fortune Cookie* [alternative UK title: *Meet Whiplash Willie*] starring Walter Matthau and Jack Lemmon, described a whiplash patient as a fraud, seeking financial reward through the court system.]

The whiplash-type of injury, as described by Davis, is responsible for the greatest percentage of cervical nerve root irritations. This type of injury is caused by a sudden forceful movement of the neck in any direction with a sudden recoil in the opposite direction. Such injuries cause typical sprains of varying degrees with subluxation of the articular processes and stretching, tearing or avulsion of, and varying amounts of hemorrhage into the ligamentous and capsular structures. Automobile accidents are responsible for the greatest number of such injuries.[5]

After meeting Dr. James A. Mertz in 1976 at a Logan College of Chiropractic homecoming, my perspectives regarding the evaluation and management of whiplash patients changed for the better. He introduced me to the use of videofluoroscopy and the diagnosis of cervical sprain injuries with resultant ligament laxity and biomechanical joint dysfunction. Dr. Mertz described to me the long-term, spinal soft-tissue effects of whiplash injuries and the probability of spinal degenerative joint and disc disease. Unfortunately, he did not publish his data because of the restrictive position of the American Chiropractic College of Radiology (ACCR). Dr. Arthur Croft (Spine Research Institute of San Diego), who has researched and published many articles and books regarding whiplash associated disorders, confirmed the previous and current ACCR positions.

Videofluoroscopy (VF), previously known as cineradiography, has long been a source of polemics in our profession. Researchers in the 1970s concluded that

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VF was somewhat unreliable due primarily to technical difficulties related to patient positioning and geometric distortion. Due to these criticisms and a concern about inadequate training, the lack of normative data on intersegmental motion, the lack of standardized technique and patient selection protocol, as well as a concern for patient exposure to ionizing radiation, the American Chiropractic College of Radiology (ACCR) adopted a rather restrictive position on VF. More recently, the ACCR has modified and relaxed its position in regard to VF, but not all practitioners are aware of these guidelines, and few refer patients for VF evaluation. At this institute, we have looked into the issue of inter-interpreter reliability, and we have candidly surveyed the attitudes of several of our most prominent radiologists. In this paper, we shall discuss our findings and review salient parts of the most recent (1991) ACCR position on VF.[6]

Economic Impact

The United Nations estimated during 2005 the economic costs of the rear-impact type of whiplash injuries in the United States to be approximately $2.7 billion per year, including quality of life impacts.[7] It was estimated in 2008 that motor vehicle crashes cost the United States government an estimated $35 billion annually.[8] The *British Columbia Medical Journal* published an article that estimated the costs for whiplash associated disorders to be $6.8 billion in the United Kingdom and $13.4 billion for Europe.[9] Unfortunately, I cannot find any peer-reviewed evidence that reports the actual costs for the treatment of whiplash associated disorders in the United States.

The lack of available U.S. data is particularly startling when considering that the National Highway Traffic Safety Administration (NHTSA) estimates that approximately 806,000 occupants sustain "whiplash" injuries in motor vehicle crashes producing modern-day economic and quality of life costs of more than $9 billion yearly.

Although we could not locate the medical costs for the treatment of whiplash associated disorders in the United States, the NHTSA reported the total costs of motor vehicle crashes in 2000 at $230.6 billion, with medical expenses of $32.6 billion.

This report presents the results of an analysis of motor vehicle crash costs in the United States in the year 2000. The total economic cost of motor vehicle crashes in 2000 was $230.6 billion. This represents the present value of lifetime costs for 41,821 fatalities, 5.3 million nonfatal injuries and 28 million damaged vehicles, in both police-reported and unreported crashes. Lost market productivity accounted for $61 billion of this total, while property damage accounted for nearly as much as $59 billion. Medical expenses totaled $32.6 billion, and travel delay accounted for $25.6 billion. Each fatality resulted in an average discounted lifetime cost of $977,000. Public revenues paid for roughly 9 percent of all motor vehicle crash costs, costing taxpayers $21 billion in 2000, the equivalent of more than $200 in added taxes for every household in the U.S.[10]

Signs and Symptoms of the Whiplash-Type Injury

The majority of whiplash patients suffers mild muscle strains and recovers within three weeks without medical care. Unfortunately, up to 42 percent of these traumatized patients begin a journey down a path from acute pain to chronic pain syndrome,[11] a frequently misdiagnosed disease in itself.[12] It is essential that the attending physician recognize the signs and symptoms of a whiplash-type injury early in the evaluation phase. Some of the most common signs and symptoms include pain in the neck, visual and auditory disturbances, temporomandibular joint dysfunction, photophobia, dysphonia, dysphagia, fatigue and cognitive difficulties such as concentration, memory loss, anxiety, insomnia and depression.[13, 14, 15, 16, 17, 18, 19, 20, 21] Yet, aberrant active cervical range of motion, sensory hyperalgesia, post-traumatic stress disorder, history of previous neck pain and the patients' perceptions of their pain and disabilities due to a whiplash injury are significant findings that point toward a higher probability of chronic pain as a result of the whiplash-type injury.[22, 23]

Howell concluded that chronic neck pain following a whiplash-type injury could be predicted with the use of the Neck Disability Index (NDI). She found correlations with pain, disability and driving task scores with whiplash associated disorder (WAD) patients. In addition, she reported that active cervical range-of-motion (CROM) reductions were common and important clinical outcome measures related to clinical prognosis of neck disability.[24]

Pathomechanics of the Whiplash Injury

Cervical Facet Joint Injuries (Zygapophyseal Joints)

Neuroscientific studies have demonstrated since 1976 that whiplash patients suffer with chronic headaches and neck pain because of cervical zygapophyseal joint pain. Approximately 50 percent of these patients experience upper cervical spine joint pain and headaches, while 60 percent experience lower cervical spine joint pain and chronic neck pain following whiplash-type injuries.[25] Post-whiplash-type injuries to the cervical facet capsules are a major cause of acute and chronic neck pain. The biomechanical rationale suggests that sprain injury or overstretching of the zygapophyseal joint capsules stimulates the firing of pain receptors.[26] It has been demonstrated that spinal manual therapy has been effective with the treatment of chronic neck pain due to whiplash-type injuries.[27]

Dorsal Root Ganglion and Nerve Root Injuries

Whiplash patients often do not receive an accurate diagnosis or effective treatment. A patient with neck pain following a whiplash-type injury should receive a clinical examination that identifies the type of tissue injured and the pain generators. The differential diagnosis must determine if the patient has incurred a whiplash injury with nerve root entrapment.[28]

Dorsal root ganglia and cervical

nerve roots compressed by herniated disc material and/or osteophytes should lead to a diagnosis of cervical radiculopathy, which may be managed with conservative care, including medications and cervical spinal manipulation.[29] Undiagnosed and maltreated compression of dorsal root ganglia and/or nerve roots is common and may contribute to the adaptation in the overall functioning of the neural tissue, which may predispose the whiplash-injured patient to abnormal, centrally mediated pain processing and chronic pain.[30] Cervical nerve root entrapments may cause entrapment of the upper cervical nerve roots with resultant progressive, occipital lancinating pain and dysesthesias and sensory deficits that do not respond to conservative chiropractic care. These non-responsive patients suffering with entrapment of the cervical nerve roots may respond to surgical decompression.[31, 32]

Cervical Ligament Sprains

Kinematic studies show three distinct periods with the potential to injure the cervical ligaments and facets. The initial stage involves functional flexion deformity of the cervical spine with a loss of the cervical lordotic curve. Next, the cervical spine is subjected to an S-shaped curve caused by extension of the lower and then upper cervical vertebrae. Finally, the entire cervical spine is extended during the third stage of the whiplash mechanism,[33] which may sprain cervical ligaments due to overstretching and possibly tearing of ligaments. A sprained cervical ligament demonstrates decreased strength following a whiplash-type injury,[34] which may permit hypermobility or even instability. The "O'Donoghue maneuver" requires performance of a series of range-of-motion tests that have been used during clinical examinations to determine cervical sprain of ligaments following whiplash-type injuries. A positive test for sprain normally presents ligamentous pain with both active and passive cervical range of motion. The passive range-of-motion-testing is provocative and demonstrates an increased range of motion, which may be confirmed with specialized imaging.[35]

O'Donoghue maneuver procedure – While the patient is sitting, the cervical spine is actively moved through resisted range of motion then through passive range of motion. Pain during resisted range of motion, or isometric contraction, signifies muscle strain. Pain during passive range of motion signifies ligamentous sprain.[36]

Spinal rehabilitation specialists in the Chicago area claim that when capsular ligaments are sprained, they become elongated and exhibit laxity, which causes excessive movement of the cervical vertebrae (hypermobility or instability) and a variety of neurological signs and symptoms or whiplash associated disorders. These specialists propose that based upon their clinical studies and experience, patients suffering with chronic pain due to post-whiplash capsular laxity and cervical instability might be offered a curative treatment option rather than surgical intervention.[37] Chiropractic physicians performing motion palpation examinations might determine these hypermobile joints to be restricted because of muscle guarding, a common finding with cervical spine instability, and perform spinal manipulation.[38] Chiropractic cervical manipulation of patients with posttraumatic cervical spine instability is considered a contraindication[39] and warrants additional evaluation with cervical spine motion imaging study to determine appropriate management. [40, 41]

Intervertebral Disc Injuries

Patients often present with injuries to the intervertebral discs and ligaments following whiplash-type injuries.[42] Large disc protrusions demonstrated on MRI may cause severe and persistent radiating pain. Without early detection and successful conservative care, surgical interventions may be necessary to alleviate the severe radiating pain. Unfortunately, patients with severe injuries to the skull and cervical spine are often misdiagnosed initially. Jonsson et al. reported that 90 percent of whiplash-injured patients with severe sprains to the cervical spine and rupture of ligamentous soft tissues were missed after radiographic examination. Even more alarming, 98 percent of whiplash-injured patients with discoligamentous sprains were missed with the initial radiographic examination. It is common that whiplash patients with multiple-level soft-tissue injuries such as ruptured intervertebral discs are misdiagnosed upon initial examination within medical facilities, most especially the emergency rooms.[43]

Unlike the frequency of intervertebral disc injury, spinal cord injuries are uncommon in whiplash patients. A spinal cord injury is more likely to occur with a whiplash-type of injury in patients with narrow spinal canals.

Unlike the frequency of intervertebral disc injury, spinal cord injuries are uncommon in whiplash patients. A spinal cord injury is more likely to occur with a whiplash-type of injury in patients with narrow spinal canals. The C5-6 segmental level is the most common level of disc injury and the most common site of narrow cervical canals. Hence, there is a greater risk of a low-grade spinal cord injury at this level due

to preexisting spinal canal narrowing, the dynamic whiplash narrowing effect and the subsequent increasing cerebral spinal fluid pressure.[44]

It is suggested that the initial evaluation with MRI of patients with whiplash-type injuries to be unnecessary in the acute phase because it is difficult to correlate the initial symptoms and signs to the MRI findings because of the high proportion of false-positive findings. Yet, MRI imaging is indicated later in the course of treatment if the patient continues to experience persistent arm pain with the presence of clinical signs (neurologic deficits) of nerve root compression.[45] If conservative chiropractic care does not alleviate the pain and the neurologic deficits persist, it is reasonable to order an MRI and refer the patient for a neurosurgical consultation.

Cervical Muscle Strains

The mild whiplash-type injury to the cervical spine involving an overstretching of the cervical muscles (Grade 1 strain) without change in active cervical range of motion or neurological deficits would be categorized as a major injury category 1 (MIC 1).[46] It is probable that the majority of these patients do not present to the emergency room or the chiropractic physician's office for evaluation and they heal without permanent disability or chronic pain, whereas patients with a more significant injury to the cervical muscles (Grade 2 and 3 strains) caused by a whiplash injury may grade their pain and disability as more severe, which may become problematic and lead to myofascial pain syndromes, muscle fatty infiltration and chronic pain.

The study by Brault, Siegmund and Wheeler demonstrated that muscles contract rapidly in response to rear-end motor vehicle collisions, and the potential for muscle injury exists due to lengthening contractions. During the acute phase of evaluation, the physical examination procedures requiring provocative maneuvers must be performed carefully in order to prevent additional insult to the traumatized soft tissues. It is important that the clinician perform an appropriate physical examination and differentiate the soft-tissue whiplash-type injury. As previously explained, the use of the O'Donoghue maneuver does enable the clinician to clinically differentiate strained muscles from sprained ligaments. Patients with a moderate (Grade 2) cervical muscle strain will demonstrate reduced and painful active cervical range of motion, while isometric range of motion will produce obvious pain in the strained musculature. Initially, therapeutic interventions should focus on healing of the injured tissues and relief of pain. Long-term care should attempt to mobilize and strengthen the strained musculature. The chiropractic physician should educate these patients in order to prevent additional whiplash injuries. Patients should be advised to reduce the distance between the head and head restraint while traveling in motor vehicles.[47]

> Possibly, future studies will confirm fatty infiltration of the cervical musculature occurs with patients who develop chronic pain following whiplash-type injuries.

The demonstration of soft-tissue injuries following whiplash injuries, especially strained muscles, with imaging studies, has been elusive. A 2011 study demonstrated temporal development of fatty infiltrates in the cervical musculature (multifidi) following whiplash injury and an association with chronic pain and post-traumatic stress disorder.[48] A follow-up MRI study, published in 2015, provided further evidence of rapid and progressive degeneration of the cervical musculature following whiplash injuries with fatty infiltration.[49] Possibly, future studies will confirm fatty infiltration of the cervical musculature occurs with patients who develop chronic pain following whiplash-type injuries. Such findings would further support a biopsychosocial basis underlying poor functional recovery and development of posttraumatic, chronic pain syndrome.

Biopsychosocial Causes of Chronic Pain Syndrome Following WAD

The early notion of whiplash focused heavily on its peripheral dimensions. However, whether or not an individual descends into chronic pain after a whiplash injury seems to hinge more upon how his or her central nervous system *responds* to the insult rather than the degree of tissue damage.[50] Nociceptive inputs that report actual tissue damage are interpreted by the central nervous system in order to establish a perceived threat level, and the spinal pain pathways are then upregulated accordingly. Indeed, the concept of central sensitization and its role in amplifying and maintaining a patient's pain experience, even beyond the resolution of the original tissue injury, is now well-known.[51]

The so-called *fear-avoidance model* for WAD has gained considerable traction in the literature over recent years.[52] This conceptual model aims to explain the role of psychosocial factors in promoting central sensitization and therefore developing and maintaining the chronic pain of WAD. Incorporating newer concepts of pain science into daily clinical reasoning will help the clinician better understand, recognize and address these important drivers of central sensitization earlier in the process. The following brief review may therefore be helpful.

The initial injury will involve an intense array of noxious stimuli from

the structures mentioned earlier. A noxious stimulus is one that is capable of exacting tissue damage and may be mechanical, chemical (such as inflammation) or thermal. The nociceptive system consists of primary afferent neurons that respond to noxious stimuli and transmit this information onto second-order pain neurons in the spinal cord. If sufficiently intense, these projection neurons relay nociceptive inputs upward to a variety of processing areas in the brainstem and cerebrum that are involved in generating the pain experience. Collectively, these areas have been described as forming a central pain neuromatrix, which performs the role of protection from an evolutionary perspective.[53] In this context, pain is not considered to be a sensation, but is rather more akin to an emotion. We might describe an emotion as an individual's behavioral response to the environment that has adaptive value. This response would therefore be dependent upon his or her beliefs, the social context and circumstances. In this regard, pain is now being viewed as an *output* of the brain based upon perceived threats and the need for protective behavior. There is also known to be an internal evaluative component to pain. That is to say that the pain neuromatrix can be activated by both ascending nociceptive inputs (i.e., reporting real or potential tissue damage) and/or the perceived likelihood of damage occurring based upon the individual's beliefs and expectations. The resulting output behavior is capable of altering motor control (to avoid the use of vulnerable tissues), autonomic control and the descending modulation of the spinal cord neurons that receive primary sensory inputs from tissues. Fundamentally then, the brain seems to determine the way in which we perceive the environment via descending influences. This might explain why expectation acts to modify pain perception[54] (due to upregulated response properties of spinal cord neurons), or the perceptual bias that occurs when a switch that will relieve pain is perceived to be closer by a patient who is experiencing pain versus someone who is asymptomatic.[55]

Gifford's Mature Organism Model nicely conceptualizes the impact of abnormal pain behavior.[56] The process begins with sensory input to the CNS, which is then scrutinized before an appropriate output behavior is generated. Using the example of whiplash, the brain is constantly sampling neck inputs to assess their integrity, as well as the surrounding environment for perceived threats. This input is then scrutinized based upon current beliefs, diagnostic explanations provided by clinicians, past experience and traumas, social constructs (i.e., views of other people as well as factors such as legal or compensatory processes) and cultural influences, etc. It is easy to see how this critical step of central interpretation could produce an output that promotes either recovery or further illness behavior. For example, an overarching belief that the neck is weak and damaged is likely to generate a behavior of overprotection, which is no longer adaptive but rather maladaptive. This would lead to further tissue deconditioning, amplification of the pain response and the ability of non-noxious stimuli, such as joint movement to access the central pain pathways (allodynia). It is therefore not surprising that psychological factors significantly influence the self-ratings of disability on the NDI in chronic WAD sufferers.[57] However, one can also see how the effect of unhelpful beliefs could extend well beyond the perception of pain and play a central role in the development of many of the functional impairments that have been associated with chronic neck pain. These functional impairments include delayed onset of deep neck flexors, increased activation of superficial neck flexors, decreased flexor muscle endurance, decreased cervical muscle strength, multifidus muscle atrophy, lower movement velocity, jerky movement patterns, reduced trajectory movement control and irregular and stiffer movement patterns.[58]

There is also another consequence of altered sensory input to the central nervous system from a whiplash injury that is important to consider. Faulty proprioceptive inputs from an injured cervical spine are known to produce postural control deficits.[59] Indeed, the concept of impaired sensorimotor control has added a new dimension to our understanding of the central mechanisms of whiplash disorder.[60] Cervical joints and muscles are richly endowed with receptors that report kinesthetic information directly to the balance control areas of the brainstem. When this reporting is distorted, a conflict arises among visual, vestibular and cervical sensory streams. The result can be a variety of functional impairments in postural stability and eye movement control that lead to symptoms such as dizziness, unsteadiness and visual disturbances.[61] Furthermore, when present in the early stages following injury, these features have been associated with a greater likelihood of transition to chronic pain.[62]

> Pain is now being viewed as an output of the brain based upon perceived threats and the need for protective behavior.

To be maximally effective in managing the whiplash patient, clinicians should recognize clinical features that are predictive of a transition to chronic pain, identify key points for effective intervention and understand how to gain maximum therapeutic leverage from their explanations, recommendations and manual applications. In general, it would appear that clinicians

The astute clinician should realize the importance of uncovering any unhelpful beliefs and negative interpretations that patients may have regarding the nature and severity of their condition.

probably underestimate both the role of a patient's beliefs (meaning perspective) and the influence of their own words and explanations in determining the level of pain and disability that the patient will or will not experience. The presence of catastrophizing or negative associations between pain and tissue damage and high self-ratings of disability on the NDI should highlight the important need to reframe the patient's interpretation of his or her condition. As well as recognizing symptoms and signs of impaired sensorimotor control, a series of cervical nonorganic signs during the examination have also been established to help identify those individuals with predominantly centrally augmented pain.[63]

Based upon our current understanding, an overall management strategy that addresses both the peripheral and central dimensions of whiplash injury might therefore be summarized as "reframe, remap and relearn." "Reframe" refers to addressing negative beliefs and interpretations. "Remap" refers to restoring a more accurate proprioceptive representation of the cervical spine in the central nervous system. "Relearn" refers to restoring normal patterns of motor control and healthier associations between movement and pain.

Conclusions

It is essential that chiropractic physicians take a patient history, perform an appropriate physical examination and recognize the signs and symptoms of a whiplash associated disorder. The examination must identify the injured tissues, pain generators, pathomechanics and differential diagnoses of whiplash associated disorders. Prior to treatment, a competent, evidence-based and patient-centered clinician should recognize the signs and symptoms that might lead to a chronic pain syndrome. We strongly recommend the use of the Neck Disability Index, which might predict chronic neck pain, especially when there are high self-ratings of disability. Indeed, the astute clinician should realize the importance of uncovering any unhelpful beliefs and negative interpretations that patients may have regarding the nature and severity of their condition. The ability of the doctor to supplant a negative association such as *pain equals damage* with a more recovery-oriented association such as *pain equals tissue de-conditioning and the need for reconditioning* may mean the pivotal difference between pain persistence and recovery. It is also important to recognize the presence of sensorimotor control impairments and nonorganic cervical signs during the examination as being more predictive of pain chronicity. Ultimately, a prognosis of chronic pain based upon factors such as previous neck pain, posttraumatic aberrant active cervical range of motion, postural control deficits and prominent psychosocial factors should signal the need to refer the patient to a behavioral medicine provider with expertise in the area of chronic pain syndrome.

Diagnosis is the key to successful treatment. (Richard C. Ackerman, DC, FACO) ■

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