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Characterization of Acute and Chronic Whiplash-Associated Disorders

The whiplash neck injury, first described by Crowe in 1928, is the most common type of injury following motor vehicle crashes (MVC). Most individuals recover within 2 to 3 weeks following the injury. However, a significant proportion of those with whiplash injury (up to 42%) will transition from an acute to a chronic pain status,[6] and it is those in this group who disproportionately contribute to the significant economic burden related to managing this challenging condition. Individuals whose symptoms fail to resolve quickly often exhibit a myriad of symptoms soon after the injury event that include, but are not limited to, pain, dizziness, visual and auditory disturbances, temporomandibular joint dysfunction, photophobia, dysphonia, dysphagia, fatigue, cognitive difficulties such as concentration and memory loss, anxiety, insomnia, and depression.[2,10,83,92,116,120,123-125] The large variability of clinical symptoms has precluded the development of discrete pathoanatomical diagnoses and makes whiplash one of the more controversial, confusing, and costly musculoskeletal conditions to treat today.

In the state of Queensland, Australia, the total costs related to whiplash injury are substantial, exceeding $500 million Australian dollars (from 1994 to 2001).[76] Claims for personal injury after whiplash injury cost the United Kingdom more than £3 billion per year,[56] while data from the United States are even more staggering, with costs reaching $29 billion US dollars per annum.[7] Clearly, acute and chronic injuries related to whiplash injury result in significant economic, personal, and emotional burden for all those involved (eg, the injured, family members, practitioners treating the injured, medical-legal system, and third-party payors).

Much confusion could be avoided if strict diagnostic definitions were available, similar to those patients who have suffered an acute traumatic injury to the cervical nerve roots (radiculopathy) or spinal cord (myelopathy). These 2 diagnoses have their own distinctive clinical findings and accompanying abnormalities on standard radiological exams.[86] While radiological exams can be used to rule out serious pathology (eg, cervical fracture), routine radiological investigation in the evaluation of acute whiplash (eg, radiographs, magnetic resonance

• **SYNOPSIS:** The development of chronic pain and disability following whiplash injury is common and contributes substantially to personal and economic costs related with this condition. Emerging evidence demonstrates the clinical presence of alterations in the sensory and motor systems, including psychological distress in all individuals with a whiplash injury, regardless of recovery. However, individuals who transition to the chronic state present with a more complex clinical picture characterized by the presence of widespread sensory hypersensitivity, as well as significant posttraumatic stress reactions. Based on the diversity of the signs and symptoms experienced by individuals with a whiplash condition, clinicians must take into account the more readily observable/measurable differences in motor, sensory, and psychological dysfunction. The implications for the assessment and management of this condition are discussed. Further review into the pathomechanical, pathoanatomical, and pathophysiological features of the condition also will be discussed.

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imaging [MRI], and computed tomography [CT]) has not proven beneficial overall, often failing to identify salient pathoanatomical lesions.[8,90,96,131] However, recent investigations of individuals with chronic whiplash-associated disorders (WAD) have begun to document previously unidentified upper cervical ligamentous and cervical extensor muscular alterations.[28,29,61-63,70,71] Nevertheless, it is widely accepted that lesions may occur to any cervical structure (bone, intervertebral discs, facet joints, ligaments, muscles, and nerve tissues) as the result of a whiplash injury.[55,100,117] Therefore, attempts to identify a specific pathoanatomical source of symptoms provide little foundation for the development of more defined classification and subsequent management strategies. On the contrary, emerging evidence suggests that clinical focus should be directed towards treatment schemas that better address identified dysfunction and clinical examination findings that have been shown to characterize the acute and chronic conditions (eg, alterations in the sensory and motor systems, and signs and symptoms of psychological distress).[52,58,100,111,114,134]

This clinical commentary will outline recent advances made in understanding the pathomechanics, pathoanatomical, and pathophysiological mechanisms of whiplash and will provide a review of the physical and psychological impairments associated with both the acute and chronic stages of this enigmatic condition. Implications of these findings for developing management strategies will be discussed.

CLASSIFICATION OF WHIPLASH

The Quebec Task Force Review (QTF) defined whiplash injury as "an acceleration-deceleration mechanism of energy transferred to the neck,"[104] usually resulting from rear-end or side-impact MVC. It is not, however, dependent on the involvement of an automobile, as other injury mechanisms could also result in a whiplash-type injury (eg, skiing, diving). QTF's classification system broadly defines the condition into 4 groups: WAD I neck complaints, with stiffness or tenderness in the neck region and no physical signs of injury; WAD II neck complaints, with stiffness or tenderness, and some physical signs of injury, such as point tenderness or trouble turning the head; WAD III neck complaints, with stiffness or tenderness and neurological signs of injury, such as changes to reflexes or weakness in the arms; and WAD IV neck complaints, with a fracture or dislocation of the neck.[104] While this system provides some necessary information related to condition classification, a major systematic flaw exists, as the majority of patients with whiplash are grouped within 1 category (WAD II), which falsely assumes homogeneity of the most common complaints within this group.[107] Furthermore, the current management strategies for acute whiplash (WAD II) as a homogeneous condition do not appear to have lessened the frequency of this disorder's incidence of transition to a chronic state.[8,35,93,97,103] One reason for this may be that such treatments have not specifically addressed the physical and psychological impairments known to be predictive of poor recovery,[58,109] and this alone may help explain the questionable value of the QTF classification. Previously held opinions related to evaluation and management of the condition and their classification are now being overturned (or at least dramatically re-evaluated) due to the emergence of evidence into why pain and dysfunction in some individuals with WAD spontaneously improve, while in others they do not.[109-114]

PATHOMECHANICAL ASPECTS OF WHIPLASH

The proposed pathomechanics of whiplash injury have evolved from an injury model of rapid hyperextension of the cervical spine, creating large sagittal-plane angular displacements,[78] to the current model of injury resulting from the body's inertial response, causing the head-neck complex to undergo large amounts of displacement without being exposed to any direct impact.[5] For example, during a rear-end impact, the occupant's torso is rapidly carried forward as it is contacted by the forward-moving vehicle's seat. This movement is responsible for the development of an ephemeral "S-shaped" cervical curve, forcing the cervical spine into abnormal, nonphysiological motion of lower segmental extension and upper segmental flexion. As a result of this nonphysiological motion, energy is stored in the elastic components of the cervical spine, followed by an abrupt release of energy and subsequent forward thrust of the head and neck (eg, acceleration/deceleration of the head/neck and torso). The consequences of this energy release could potentially impact and injure any number of anatomical tissues in the cervical spine (eg, intervertebral discs, joint capsules, ligaments, facet joints, muscles, and nerve tissues).[55,65,100,117]

Numerous kinematic studies have demonstrated the in vivo and in vitro occurrence of this differential acceleration-deceleration of the head and torso during relatively low-velocity impacts.[38,65,84,85,136] This abnormal motion has been shown to produce elongation and subfailure strain of the facet capsular ligaments at the C6-7 level during the initial "S-shaped" phase (between 0 and 75 milliseconds). During this initial phase, the car seatback pushes the torso forward (0-50 milliseconds following impact), resulting in thoracic and cervical spine straightening. Following the initial 50 milliseconds (50-75 milliseconds), the car seat rapidly pushes forward the occupant's torso, while the head, secondary to inertia, remains stationary (**FIGURE 1**). Additionally, most of this involuntary motion occurs with little, if any, resistance from the supporting paraspinal musculature, as the latencies required for eliciting reflexive muscular activity are far longer in duration.[84]

Maximum head and neck displacement is observed during the second phase of this abnormal motion (>100 milliseconds), and all cervical segmental

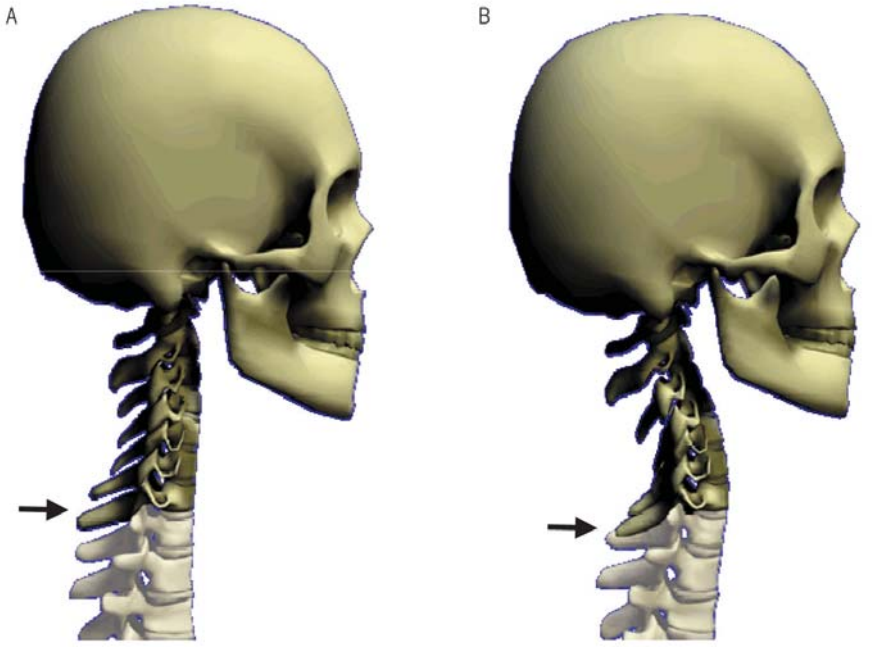

FIGURE 1. (A) Critical period from where the spine goes from a straightened position (0-50 milliseconds) to the abnormal "S-shaped" curve (50-75 milliseconds). (B) It is here (50-75 milliseconds) that the facet capsules can be stretched, pinched, and torn. Reproduced with permission from Body Mind Publications (www.injuryresources.com).

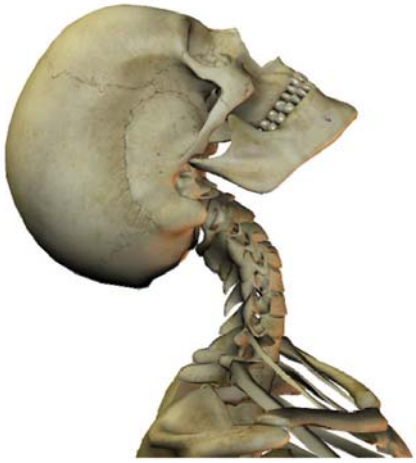

FIGURE 2. Illustration of the second phase of the "S-shaped curve" (>100 milliseconds); the torso has pulled so far forward on the lower neck that the head is forced backwards over the head restraint. Depending on the severity of the collision, the ligaments in the anterior portion of the spine can be injured during this phase of the collision. Reproduced with permission from Body Mind Publications (www.injuryresources.com).

levels are extended (**FIGURE 2**).[38] Kaneoka et al[65] corroborated these findings in vivo and further documented the occurrence of facet joint spearing of the superior articular facet onto the compromised inferior articular facet, as well as stretching of the anterior ligamentous tissues. The authors hypothesized that this movement contributes to the mechanism of injury and may explain the nature of painful symptoms following whiplash.

PATHOANATOMICAL LESIONS IN THE WHIPLASH INJURY

In a seminal study, Taylor and Twomey[118] investigated cadaveric cervical spines of individuals involved in fatal injuries compared with those who died of other natural causes. The authors were able to demonstrate a variety of unique injuries involving the cervical facet joints, spinal dorsal ganglia (DRG), ligamentous tissues, and intervertebral discs. While it must be noted that these studies investigated nonsurvivors of a MVC, the combined data from such investigations, bioengineering, and clinical studies provide reasonable argument for the presence of pathoanatomical lesions in some individuals sustaining a whiplash injury.

Facet Joints

Studies employing the cervical facet joint injury model have identified the occurrence of hemarthroses, capsular tears, articular cartilage damage, joint fractures, and capsular rupture.[55,117,135] Other studies have documented subfailure injuries to cervical facet capsular ligaments during whiplash trauma,[22,87,106,136] and recent evidence demonstrates the rapid development of nerve fiber morphology and axonal changes as a result of tensile stretching of the cervical facet joint capsule in an animal model.[64]

The facet joints are abundantly innervated with Aδ- and C-nerve fibers that operate at a high threshold and may become sensitized or excited by local pressure changes, capsular stretching, and naturally occurring proinflammatory agents (eg, substance P, phospholipase A, and interleukin 1ß).[3,47,48]

Clinical support for a facetogenic model of persistent pain generation in whiplash can be found in the literature.[74] The authors associated facet arthropathy with WAD by substantially lessening pain in a highly selected group of patients with persistent symptoms following a whiplash injury, using facet joint blocks. The C2-3 facet joint was found to have the highest prevalence (60%) of joint pain followed by the C5-6 facet joint.[74]

DRG and Nerve Roots

The anatomical locations of the DRG and nerve roots render them vulnerable to excessive stretching and injury during rapid acceleration/deceleration ("S-shaped" curve) or lateral bending of the neck, as has been demonstrated in rear-end and side-vector impact whiplash.

Taylor et al[119] investigated fatally injured persons involved in blunt trauma to the cervical spine and found a high prevalence of cervical DRG injury. Each segmental cervical DRG contains distinct nerve fibers responsible for relaying specific information to the spinal cord and brain (eg, proprioception, pain, and temperature).[9] DRG compression and soft-tissue changes, which remain

largely undetected with conventional radiography, may contribute to adaptation in the overall functioning of the cervical DRG[40,45,51] and may predispose an individual to abnormal, centrally mediated pain processing.[40,50]

Ligaments

Ligamentous injuries in the mid- and lower-cervical segments have also been suggested as contributing to the development of persistent symptoms following a whiplash injury. Tominaga et al[121] demonstrated reduced strength, subfailure injury, and altered mechanical properties of cervical spine ligaments secondary to simulated rear-end vector impact injuries. It was proposed that microscopic subfailure injuries of the segmental cervical ligaments may injure embedded mechanoreceptive and nociceptive nerve endings, and thereby lead to pain, inflammation, and chronic symptoms.[121]

Upper cervical ligament injuries have also been proposed as a factor in the development of chronic WAD symptoms, and support for such lesions can be found in studies using high-resolution proton-density-weighted MRI, demonstrating high signal intensity (indicative of damage) in both the alar and transverse ligaments, and in the tectorial membrane in some subjects with chronic WAD.[71] Later follow-up studies indicated a strong relationship between severity of alar ligament damage, head position (turned at time of impact), Neck Disability Index (NDI)[128] scores,[62,63] and reproduction of pain and excessive mobility with the manual examination techniques for the upper cervical ligaments.[61]

Disc Injuries

Despite controversy, some studies have documented the presence of cervical disc injury in 20% to 25% of subjects following whiplash injury, and these findings correlated with radicular symptoms.[55,89] The C5-6 segmental level was found to be the most common level of disc injury, and this may also align with biomechanical models showing greater risk of low-grade spinal cord injury during whiplash in individuals with pre-existing spinal canal narrowing at the C5-6 level.[49]

Muscle Injury

Previous work has shown potentially injurious musculotendinous strains of the sternocleidomastoid muscle during whiplash injury,[11] and recent evidence demonstrates the occurrence of larger strains in the superficial posterior neck muscles (semispinalis and splenius capitis and upper trapezius) during rear-end impacts.[127] Experimental studies have shown that lengthening (eccentric) contractions result in morphological alterations and loss of muscular force capabilities.[13,77] However, higher strain magnitudes may be required to injure the deeper cervical spine (muscle with higher type I fiber make-up) muscles when compared to the more superficial muscles (eg, those with a higher type II muscle fiber distribution).[75] Therefore, while evidence exists to suggest the occurrence of muscle strain during the eccentric phase of a rear-impact whiplash, it is currently unknown if differential responses are noted in the deep versus superficial extensor muscles. Future research is warranted to investigate this question.

Summary of Pathological Lesions

The lack of in vivo, gold-standard diagnostic tests aimed at specifically identifying these proposed lesions appears to be due to the poor sensitivity of current radiological imaging techniques. While evidence from in vitro studies indicates that the injury can damage any number of anatomical structures in the cervical spine at any segmental level, it is largely unknown if these lesions occur either in combination or are independent of one another. However, one thing remains clear: whiplash injury can, in some patients, trigger a cascade of events that promotes the development of chronic symptoms. It is, therefore, crucial that emphasis be directed towards understanding the underlying mechanisms and subsequent sequelae of the condition.

PHYSICAL AND PSYCHOLOGICAL FEATURES

Many studies have investigated various prognostic factors, such as sociodemographic status, crash-related variables, compensation/litigation, psychosocial, and physical factors in the development of chronic WAD.[17,66,94] However, prior to 2007, only 2 systematic reviews of prospective cohorts could agree on high initial pain intensity as being predictive of delayed recovery.[20,99] While initial pain intensity alone may provide some assistance in identifying patients at risk for developing persistent symptoms, new evidence implicates pain and functional disability levels to also be of value in the prediction of those at risk for transitioning from acute to chronic symptoms.[109]

Recent studies suggest that the combination of high pain and disability levels in tandem with physical and psychological factors, including the early presence of cervical movement loss, cold temperature hyperalgesia, and posttraumatic stress symptoms, are all strong predictors of poor outcome at 2 to 3 years post-MVC.[109] Thus, the robustness of these factors suggests the importance of their assessment in the acute phase. Thereby, the need for clinical-friendly assessment tools is high and this is currently under development in our lab.

CHARACTERISTICS OF THE WHIPLASH PRESENTATION

It is emerging that whiplash is a markedly heterogeneous and complex condition, with varied disturbances in motor, sensorimotor, and sensory function, as well as the presence of psychological distress. Deficits in the sensorimotor system have been reported to be features of patients with chronic WAD.[23,41,60,81,111] Some of these deficits are reflected by loss of active cervical range of movement, altered patterns of muscle recruitment, and alterations in postural control mechanisms such as kinesthetic sense, balance, and eye movement control.[1,59,110,123-125]

Motor Dysfunction

Assessment and documentation of active cervical range of motion is commonly used as a clinical tool and outcome measure in patients with neck complaints and, in particular, whiplash.[8,42,93] One of the most common clinical characteristics of patients with whiplash is that of restricted cervical spine range of motion. Numerous studies have indicated that those persons with chronic WAD (>3 months) continue to display long-term changes in the cervical motor system.[23,25,27,41,81,111] Measurement markers of range of motion have also been shown to accurately discriminate between those patients with persistent WAD compared to those with no history of neck complaint (sensitivity, 86.2%; specificity, 95.3%).[23] Sterling et al[111] documented motor system changes in all subjects with whiplash within 1 month following injury. Of note is that reduced range of motion persisted in only those with moderate/severe symptoms (NDI, >30/100) but returned to within normal limits in those who had recovered (NDI, <8/100) or reported persistent milder pain (NDI, 10-28/100) in the long term (2-3 years).[109] Results from this study identified the presence of cervical range-of-motion deficits within 1 month after whiplash in all subjects. However, it was only the group reporting moderate/severe symptoms that continued to manifest deficits in cervical range of motion at 2 to 3 years postinjury. The findings of reduced range of motion may reflect underlying disturbances in motor function as a consequence of the initial peripheral nociceptive input resulting from injured anatomical cervical structures. Further investigation of such potential mechanisms in WAD is required.

Altered patterns of muscle recruitment in both the cervical spine and shoulder girdle regions have been clearly shown to be a feature of chronic WAD.[57,80] Longitudinal data demonstrate that these changes are apparent from very soon after injury,[80] with greater deficits in those reporting higher levels of pain and disability.[109,111] Sterling et al[109,111] observed that the disturbed motor patterns persisted, not only in those with ongoing chronic symptoms but also in those with milder pain and disability and those who reported full recovery with this phenomena occurring at significant periods postinjury (up to 2 years). These persisting deficits in muscle control may leave recovered individuals more vulnerable to future episodes of neck pain; but this proposal needs to be substantiated with further investigation.[109] Nevertheless, these findings demonstrate the significant effect whiplash injury has on the motor function of the cervical spine and indicate that early and specific rehabilitation will likely be important in the management of all those with a whiplash injury, irrespective of reported symptoms.

Sensorimotor Dysfunction

Dysfunction of sensorimotor control is also a feature of both acute and chronic WAD. Greater joint repositioning errors have been found in patients with chronic WAD and also in those within weeks of their injury, particularly those reporting more severe pain and disability.[111,125] Loss of balance and disturbed neck-influenced eye movement control are present in chronic WAD[123,124]; but their presence in the acute stage of the injury are yet to be determined and is currently being investigated. Regardless, it is important to note that sensorimotor disturbances seem to be greater in patients who also report dizziness in association with their neck pain.[123,124]

Disturbances in Sensory Function

Whiplash presents with an intriguing sensory presentation. There is now consistent evidence from numerous cohorts demonstrating widespread sensory hypersensitivity (or decreased pain thresholds) to a variety of stimuli (pressure, thermal, electrocutaneous) in individuals with WAD (for review, see Sterling and Kenardy).[113] Such hypersensitivity has also been featured in patients undergoing testing that does not require a cognitive response. For example, Banic et al[4] demonstrated facilitated flexor withdrawal reflexes (using electromyographic signals of the biceps femoris muscle) in the lower limbs of subjects with chronic WAD following electrical stimulation of the sural nerve. In this test, reflex activity of the biceps femoris was measured and evidence of spinal cord hypersensitivity (central sensitization) was documented without relying on the subject's self-reported response to the stimuli, as is required with pain threshold testing. Sterling et al[108] also reported that the heightened reflex responses are not associated with psychological factors such as catastrophisation and distress. These hypersensitive responses, generally termed "sensory hypersensitivity," infer the presence of augmented central pain processing mechanisms.[21] These phenomena are a feature of many individuals with chronic whiplash pain[58] and, perhaps more importantly, were present early in the acute stage of injury in those individuals with poor functional recovery.[67,112]

The presence of sensory hypersensitivity is not unique to WAD. Individuals with cervical radiculopathy also present with widespread sensory hypersensitivity in the upper and lower limbs and contralateral to the side of nerve conduction loss.[18] In contrast, cervical spine pain of insidious onset shows a different sensory presentation. While this condition shows similar hyperalgesia locally over the cervical spine, no evidence could be found of more widespread sensory hypersensitivity.[101] It is argued that the local hyperalgesia may reflect peripheral sensitization processes occurring in neck structures, while the more widespread changes may be an indication of augmented central pain-processing mechanisms.[101] The reasons for the spread of sensory hypersensitivity to the upper and lower limbs in WAD and cervical radiculopathy, but only local hyperalgesia, in insidious-onset neck pain are not clear. There does appear to be some relationship to levels of pain and disability, with the latter condition showing much lower levels of pain and disability compared to WAD and

radiculopathy.[18,101] Similar relationships between pain and disability levels and sensory disturbance have also been demonstrated between subgroups of individuals with work-related neck pain.[54]

While it is generally acknowledged that the sensory hypersensitivity presentation of WAD reflects hyperexcitable central nervous system pain processes, some of the features, such as cold hyperalgesia and sympathetic nervous system changes, could be indicative of peripheral nerve injury/involvement. This proposal may have some merit. A recent investigation has shown the concomitant presence of hypoesthesia (elevated detection thresholds) to mechanical and thermal stimuli in the hands of individuals with chronic whiplash,[18] changes in motor unit action potentials indicative of neural injury in C6 and C7 innervated muscles of whiplash participants,[19] and increased mechanosensitivity with neural provocation tests.[46,115] Ongoing work should further the understanding of these mechanisms.

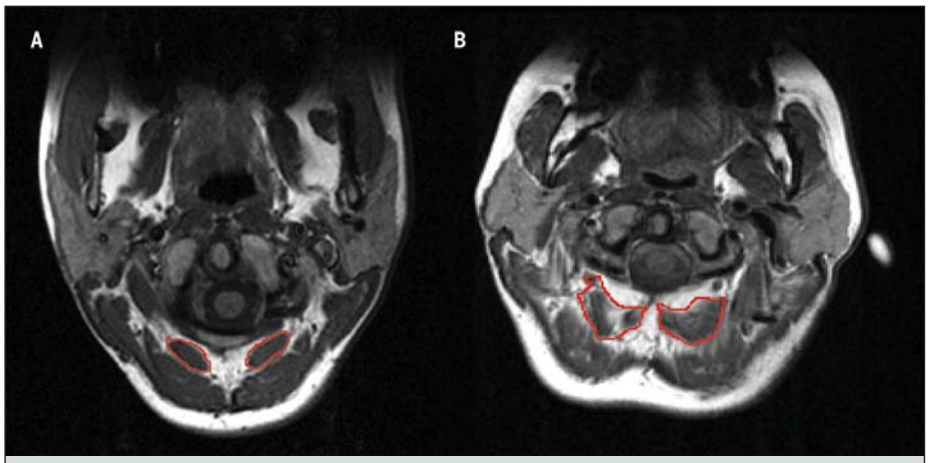

FIGURE 3. Bilateral axial magnetic resonance images of the rectus capitis posterior minor muscle (outlined in red) at Occiput-C1 (healthy control [A] compared to a subject with whiplash-associated disorder [WAD] [B]). Reproduced from Elliott J, Jull G, Noteboom JT, Darnell R, Galloway G, Gibbon WW. Fatty infiltration in the cervical extensor muscles in persistent whiplash-associated disorders: a magnetic resonance imaging analysis. *Spine*. 2006;31:E847-855. Reprinted with permission from Lippincott, Williams & Wilkins.

Psychological Factors in WAD

There is no doubt that chronic whiplash pain is associated with psychological distress, including affective disturbances, anxiety, depression, and behavioral abnormalities such as fear of movement.[82,88,133] Psychological distress is also present in the acute postinjury stage, with most people showing some distress regardless of symptom levels.[114] Data indicate that the ongoing psychological distress is associated with nonresolving pain and disability. A recent, large, cross-sectional study showed an association between anxiety, depression, and pain and disability in people whose accidents occurred over 2 years previously but not in those with acute injury, suggesting that symptom persistence is the trigger for psychological distress.[133] Longitudinal data indicate that initially elevated levels of distress decrease in those who recover, closely paralleling decreasing levels of pain and disability.[114]

Unique psychological factors may be involved in the etiology and development of chronic whiplash pain[114] when compared to other painful musculoskeletal conditions. For example, the role of fear-of-movement beliefs seems to be a less important factor in individuals with whiplash[112] than in those with low back pain.[129] The role of coping styles or strategies in individuals with whiplash injury is unclear. Some data indicate that a palliative reaction (eg, seeking palliative relief of symptoms such as distraction, smoking, or drinking) was associated with longer symptom duration.[15,16] In contrast, Kivioja et al[68] found no evidence that different coping styles in the early stage of injury influenced the outcome at 1 year postaccident. The different cohort inception times of these studies may account for the differences in findings, indicating that coping strategies may vary depending on the stage of the condition, and this requires further investigation.

One factor that is likely unique to WAD (when compared to other common musculoskeletal conditions), due to the mode of onset being a traumatic event, is that of posttraumatic stress. Symptoms of posttraumatic stress have been shown to be present in a proportion of people following a whiplash injury due to a MVC,[24,69,114] and these symptoms have shown prognostic capacity for poor functional recovery at 6 months and 2 years post-MVC.[14,109,112] These studies mostly utilized the Impact of Events Scale (IES).[44] The IES is an instrument that measures distress associated with a specific event (in the case of whiplash, a MVC). It should be noted that a diagnosis of posttraumatic stress disorder cannot be made from IES scores, and such a diagnosis is yet to be demonstrated in WAD. Nevertheless, the persistent presence of posttraumatic stress symptoms indicates the need for further psychological evaluation of these patients,[33] and clinicians should be aware of this factor in their assessment of all individuals with a whiplash injury.

Degeneration of the Cervical Extensor Musculature in Chronic WAD

Recent investigations have identified the presence of muscular degeneration (fatty infiltration) in the cervical extensor muscles of females (18-45 years of age) presenting with persistent pain and disability (>3 months and <3 years) following whiplash injury.[28] Larger amounts of fatty infiltrate were present in all cervical extensor muscles when compared to asymptomatic controls. While the findings of fatty infiltrate in the WAD group were widespread, it was present to a larger extent in the deep muscles in the suboccipital region (rectus capitus posterior major and minor) (**FIGURE 3**) and the multifidi

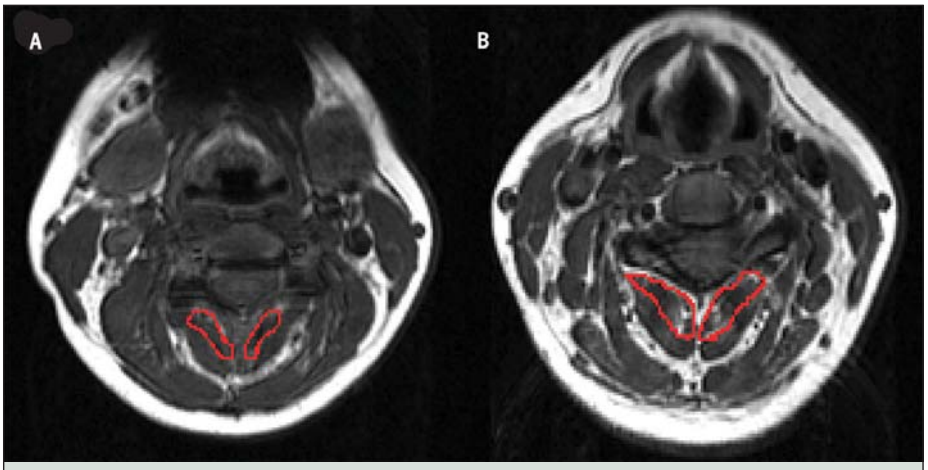

FIGURE 4. Bilateral axial magnetic resonance images of the segmental cervical multifidus muscles (outlined in red) at C3 (healthy control [A] compared to a subject with whiplash-associated disorder [WAD] [B]). Reproduced from Elliott J, Jull G, Noteboom JT, Darnell R, Galloway G, Gibbon WW. Fatty infiltration in the cervical extensor muscles in persistent whiplash-associated disorders: a magnetic resonance imaging analysis. *Spine*. 2006;31:E847-855. Reprinted with permission from Lippincott, Williams & Wilkins.

at the C3 segmental level (**FIGURE 4**). It is known that these muscles are rich in muscle spindle density,[72,73] and this may account for some of the functional impairments commonly observed in people with whiplash, including proprioceptive deficits, balance loss, and disturbed motor control of the neck. The muscle changes were independent of age, body mass index, compensation status, and duration of symptoms.

Currently, the significance of these muscular changes with respect to pain and disability and recovery rates amongst a more heterogeneous group of patients with whiplash are unknown. However, all of the sampled subjects displayed concomitant sensory (hyperalgesia) and psychological disturbances that are known to characterize and predict those patients with WAD who have a complex, nonresolving injury, suggesting that the fatty-muscle changes may be associated with nonrecovery. As the muscular changes were observed in the chronic state, it is not yet known whether they occur uniformly in all individuals, irrespective of recovery, or are unique to only those who develop chronic symptoms. Subgrouping these patients, based on the clinical presentation, would answer this question and is currently under investigation.

It is important to briefly consider the underlying mechanisms that could govern cervical extensor muscle degeneration following whiplash. These may include muscle injury, age, generalized muscle disuse, chronic denervation, or processes associated with the traumatic injury such as inflammation.

The mechanism of muscle degeneration is not likely to result from direct trauma to the paraspinal muscles, as our previous findings of fatty infiltrate were widespread and affected, to a greater extent, the deeper cervical muscles.[28] Aging is also unlikely to be the primary or only cause of our findings of fatty infiltration, as we found that the fat content was not influenced by the factor of age. This is not altogether surprising, as an age-related reduction in fat-free muscle mass is reported not to commence until much later in life[34,105,132] and our age group closely resembled young to middle-age adults (18-45 years).

Generalized disuse is a plausible explanation for the muscle degeneration in view of the widespread changes in all cervical extensor muscles. It is accepted that atrophic changes in muscle are not uniform and greater changes are found in muscles characterized by slow-oxidative fibers (eg, more type I fibers).[32,39,53,79] Type I fiber distribution is also linked with the larger distribution of muscle spindles,[26,95,98] which could account for the larger fat infiltration observed in the deep suboccipital muscles. It was reasoned that if disuse was the primary cause of the widespread fatty infiltrate in the cervical muscles in subjects with persistent whiplash, then it should also be a generic finding in patients with chronic, nontraumatic neck pain. However, we have now shown that subjects with insidious onset (nontraumatic) neck pain of longer duration did not feature fatty infiltrate in the cervical extensors, which refutes a disuse mechanism as the primary underlying cause of the muscle degenerative changes found in the chronic WAD group[29] and suggests that pain-related factors may play a more significant role.

There are several lines of research to support denervation and/or traumatic processes (eg, inflammatory response) as contributing to the development of muscular degeneration and higher pain and disability levels. First and foremost, as discussed earlier, the presence of fatty infiltrate was not featured in individuals with chronic insidious onset neck pain,[29] indicating that trauma-related processes, such as inflammation, may play a role. Subjects with insidious neck pain demonstrated fat index scores that mirrored those previously reported for asymptomatic subjects,[28] and they differed significantly on the sensory and psychological measures when compared to the group with chronic WAD. This is consistent with previous research showing that the physical characteristics of insidious neck pain are similar to those of the whiplash subgroup, with a relatively uncomplicated presentation,[97] suggesting that there may also be differential changes in the muscular alterations between whiplash subgroupings.

Secondly, Hodges et al[43] demonstrated rapid changes, including atrophy and fatty infiltration, in the segmental lumbar multifidus' cross-sectional area within 3 days following experimental lumbar disc and nerve lesion. Specifically, changes

were observed in the ipsilesional, segmental multifidus following disc lesion. However, a different distribution was observed following nerve lesion to the medial branch of the dorsal ramus, in which rapid atrophy and fatty infiltration occurred bilaterally at 3 segments caudal to the injury site. These findings provide evidence that rapid changes in multifidus occur following disc and nerve lesion but the resultant effects suggest differential mechanisms. Regardless, it could also be expected that a similar time frame for the development of muscular changes could occur in some subjects following traumatic whiplash injury. It may be that such changes could be greater in those with a more "complicated" presentation and poor recovery. This theory is currently being investigated.

Thirdly, mechanical tissue injury has been shown to create local and systemic inflammation, which can induce profound changes in muscle tissue.[36,37] Gradl and colleagues[36] found that exaggerated injury and greater inflammatory responses are associated with morphological muscle changes such as atrophy and apoptosis, and animals with a lesser injury showed no such muscle changes. This model fits well with that which has been developed for whiplash injury.[109,110] While we cannot accurately determine the injury severity in precise pathoanatomical structures, the presence of muscular degeneration, in tandem with the heterogeneity of the physical and psychological presentation in various whiplash subgroupings, suggests a more severe injury in those who become chronic.

Further work is warranted to investigate the potential mechanisms behind these objective markers of muscular degeneration and this would necessitate, for example, wideranging research on animal models where lesions to various cervical structures could be controlled, inflammatory markers and histological changes in nerve and muscle tissue could be documented in a longitudinal fashion, and activity levels relevant to the neck could be controlled.

CLINICAL IMPLICATIONS

The clinical evaluation of neck pain and whiplash symptoms has largely attempted to identify the primary anatomical structures considered to be responsible for the development of pain. However, such an approach provides little foundation for guiding treatment and providing information related to prognosis. On the contrary, new evidence suggests that the clinical evaluation should aim to identify the presence of physical and psychological impairments (which characterize the acute and chronic conditions) to provide more in-depth assessment and recognition of potential underlying mechanisms.[107,122,134]

Some commonly used clinical tests for the cervical spine (eg, manual palpation and visual inspection of cervical range of motion) lack reliability or validity.[102,126] However, recent evidence suggests that the use of more reliable and valid objective measures for cervical active range of motion could help guide the selection of interventions and possibly establish prognosis in patients with neck disorders.[91]

The assessment of motor dysfunction in the patient with WAD needs to be expanded to include identification of kinesthetic deficits (eg, joint position error) and cervical muscle recruitment patterns. There is ample evidence illustrating how this can be achieved with simple measures of joint position error and specific muscle tests (cranial-cervical flexion).[57,58,125] It is also vital to consider other measures of sensorimotor dysfunction, such as measurement of altered activity in the upper trapezius during functional tasks, poor control of balance, and impaired eye-movement control. Such impairments have been documented in individuals with chronic WAD,[30,123-125] but their presence and significance in the acute stage is currently unknown. This is being investigated.

A detailed neurological exam is also of importance, but is usually limited to basic screens (eg, manual muscle testing, deep tendon reflexes, and light touch sensation). Specific sensory assessment measures that investigate systemic hypersensitivity to temperature or touch have shown promise in their ability to classify patients in both the acute and chronic states. Furthermore, quantitative sensory testing (QST) can be utilized and should include measurements of mechanical pressure-pain thresholds with algometry (**FIGURE 5**) and thermal sensitivity (**FIGURE 6**). It is noteworthy that such sensory assessments, which, at present, require expensive laboratory equipment, provide useful prognostic information; however, there is no current consensus about the most appropriate testing method. The development of appropriate reliable and valid clinical measures for such sensory disturbances is currently in development. However, the clinician can gain an appreciation for the presence of central hyperexcitability from certain examination find-

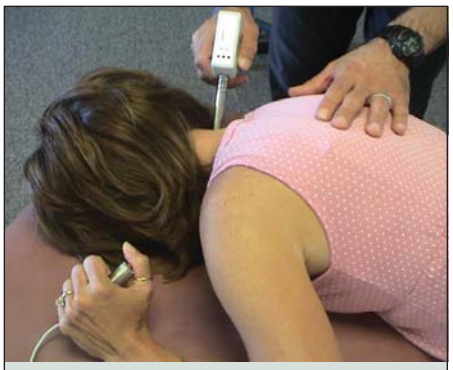

FIGURE 5. Measurement of pressure pain thresholds over C5-6 using pressure algometer (Somedic AB, Stockholm, Sweden).

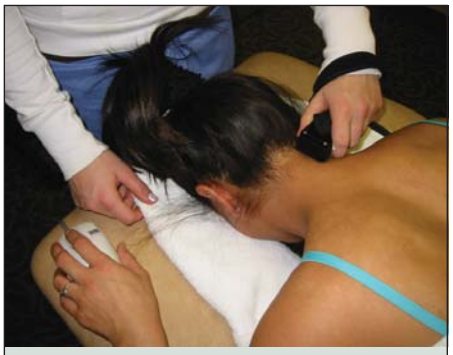

FIGURE 6. The thermotest system (Medoc, Israel) used to measure thermal pain thresholds.

ings, including the nature of the pain (for example a highly irritable condition implies mechanical hypersensitivity), the presence of hyperalgesia or allodynia with manual examination, and reports of pain exacerbation with cold.[113]

It is also crucial to recognize the psychological features that characterize this condition. The use of valid questionnaires, such as the IES,[44] which captures data related to the presence of posttraumatic stress reactions, would be of the utmost importance. Posttraumatic stress has been shown to play an important role in recovery.[109] Clinicians should therefore be cognizant of the presence of such a reaction in some patients and consider an appropriate psychological referral if warranted.

Summary of Clinical Implications

The observed changes in motor function, sensory disturbance, and psychological distress occur very soon following the whiplash event and remain unchanged (2-3 years post-MVC) only in those patients who report moderate/severe pain and disability. Certainly, this finding alone is particularly important for the clinician treating patients with whiplash and suggests that management strategies should be instituted early after injury. Unfortunately, specific management strategies aimed at preventing the development of chronic pain remain speculative and have been mainly restricted to behavioral approaches.[12,31,130] However, recent evidence points to more favorable outcomes (reduction in pain and disability) when employing a multimodal physiotherapy program addressing the specific impairments compared to a self-management behavioral approach for chronic WAD.[58] The effects, however, are mitigated in the presence of abnormal sensory features known to have prognostic value (eg, cold hyperalgesia and widespread allodynia).[58] Individuals with moderate/severe symptoms present with a more complex, debilitated pain state, and their clinical picture is complicated by the presence of widespread sensory hypersensitivity and psychological distress. It is argued that these patients may benefit more from early management strategies utilizing a multidisciplinary professional approach. Such a treatment approach may need to include physical rehabilitation, psychological support, and pharmaceutical pain management, and it is imperative to institute this program soon following injury rather than waiting to determine whether the symptoms are not resolving. In comparison, those with lesser symptoms are not likely to demonstrate such profound impairments and inference can be drawn that the clinical management of these patients should consist of strategies addressing the motor impairments (eg, range of motion and altered muscle recruitment patterns with active exercise).

CONCLUSION

Recent research into whiplash has added to the body of knowledge as it relates to mechanisms of injury, the possible symptomatology, and the physiological and psychological features that have been shown to influence outcomes. It is unknown whether conventional radiography could be used to better understand the pathoanatomical and pathophysiological features of acute and chronic WAD to provide a quantifiable measure of any processes. However, evidence now exists to show that whiplash can, in some patients, involve a complex chain of debilitating changes in muscle tissue and motor, sensory, and psychological functioning, regardless of pathoanatomical features. It would appear that the quest to better understand WAD has only just begun, and the results from recent research efforts have unlocked many further directions for investigation. As new knowledge emerges, the clinical assessment of the condition can only become more informed, and this can only equate to an improved treatment and prognostic picture for those experiencing a whiplash injury. ◉

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