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Whiplash-Associated Disorders: Comprehensive Insights into Pathology, Incidence, and Risk Factors for Long-Term Impact in Motor Vehicle Accidents

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Purpose Statement

This paper provides a clear overview of Whiplash-Associated Disorders (WAD), focusing on how these injuries happen, how common they are in car accidents, and how crash direction affects the type of injury. It also covers risk factors that can lead to long-term pain and disability. The goal is to stress the importance of early, well-rounded treatment and to guide professionals in managing and improving outcomes for patients with WAD.

Executive Summary

Whiplash-Associated Disorder (WAD) is a complex condition that can look different from person to person. The type of crash and the person's position during the accident can greatly affect how the injury develops. This paper explains the underlying anatomy involved in WAD and offers guidance on how to best care for patients after a motor vehicle accident.

Key Findings

- WAD often causes lasting pain and movement problems.
- Nerve-related pain can spread if not addressed early.
- Different crash directions cause different types of injuries.
- Muscle damage may lead to fatty buildup in neck muscles, which worsens pain and recovery.
- Concussions and brain injuries are more common in WAD than people realize, and often go undiagnosed.
- Early identification of risk factors can help guide better treatment decisions.
- Patients have better outcomes when treated early by a skilled team.

Implications

This paper highlights how complex whiplash injuries can be and explains why it's important to understand how the body moves during a crash and how symptoms can vary. It supports early, team-based care as the best way to help patients recover fully.

Introduction

Neck pain from a motor vehicle accident (MVA) is different from a typical neck strain. The term "whiplash" was first introduced in 1928 to describe this unique injury caused by the rapid acceleration and deceleration common in car crashes.[1] Since then, the term has evolved to include a wide range of symptoms such as pain in the neck, shoulders, back (upper, middle, and lower), arms and legs, as well as headaches.[2-7] Other symptoms may include depression, anxiety, PTSD, dizziness, tinnitus, memory issues, difficulty swallowing, voice changes, jaw pain, concussions, and even brain injuries.[2 8-15]

One of the biggest challenges in treating WAD is predicting which patients will recover and which will develop chronic, long-term symptoms. [16-18] Studies show that about half of patients recover within 3 to 6 months, but many continue to struggle for one to two years, or longer. Of those who don't recover, around 30–40% have ongoing mild to moderate pain, and 10–20% experience more severe symptoms. [18] Another study found that 69% of whiplash patients still had symptoms two years after the accident. [19]

WAD is also a global health concern. A 2009 study reported that whiplash injuries occur at rates ranging from 28 to 834 cases per 100,000 people per year. [20] In the European Union, an estimated 300,000 people suffer whiplash injuries annually. [17] Emergency room visits for WAD have increased tenfold over the past 20 years. [21]

The financial impact is substantial. In the UK, the cost of treating WAD and the related loss of productivity was estimated at £3.1 billion in 2002.[22] Other estimates include €10 billion across Europe and $9 billion in the United States.[22],[23] One U.S. study calculated the cost of WAD treatment in 2004 at $2.7 billion.[24] Another found that during the first four months after a whiplash injury, combined costs for care and lost productivity ranged from £99.55 to £668.53.[25]

Problem

As shown by the incidence and cost data above, WAD is a growing global health and economic issue. Its complexity goes beyond simple neck pain, patients often experience a wide range of symptoms that can be difficult to manage. This can be overwhelming for both patients and providers, even with recent advances in treatment. [26] Understanding how WAD develops and progresses is key to creating effective, targeted treatments that improve recovery outcomes.

Classification

To help clinicians and researchers better assess and treat whiplash injuries, the Quebec Task Force developed a widely used classification system. [27] This system ranks WAD from Grade 0 to IV based on severity and can help predict outcomes up to 24 months after injury.[27],[28]

- **Grade 0:** No neck complaints or physical signs.
- **Grade I:** Neck pain, stiffness, or tenderness only.
- **Grade II:** Neck pain plus musculoskeletal signs (e.g., limited motion, tenderness).
- **Grade III:** Neck pain plus neurological signs (e.g., reflex loss, weakness, sensory changes).
- **Grade IV:** Neck pain with fracture or dislocation.

Studies show this system is a reliable tool, especially when used by experienced WAD researchers. [29]

Symptoms

Whiplash injuries can lead to a wide range of symptoms. The most common is neck pain, which affects up to 84% of patients within the first week and continues in 38% of patients a year later. [30] This pain is more severe and longer-lasting than a typical neck strain. It often includes limited motion, muscle fatigue, and referred symptoms in the arms, hands, head, and upper back due to damage in the joints and soft tissues of the neck. [31-40]

Headaches are also common. [30], [41-43] One review found that 60% of patients reported headaches in the first week, and 38% still had them after 12 months.[30] These headaches may come from neck trauma (cervicogenic) or brain injury such as concussion.[11],[41],[42],[44],[45]

While this paper focuses on the neck, lower back pain is also frequently seen after motor vehicle accidents and can be part of WAD.[43],[46],[47] Concussions and traumatic brain injuries (TBIs) are additional possible outcomes.[11],[12] Gil and Decq's 2021 review highlights shared symptoms like dizziness, balance problems, and eye movement issues that may result from similar injury mechanisms.[12] Memory issues related to brain trauma are also common and can be missed in the emergency setting.[48],[49]

Dizziness is another frequent symptom linked to injury of the neck or inner ear.[50-52] Chronic tinnitus (ringing in the ears) may also develop after trauma and can be worse when combined with head or neck injury.[53],[54] One study found that up to 68% of patients had tinnitus tied to neck muscle issues.[55] Other symptoms like jaw pain, difficulty swallowing, and facial issues (craniofacial disorders) are also associated with WAD.[15],[56-58]

Biomechanics

To properly understand the wide range of symptoms caused by WAD, it's essential to examine the biomechanics of the head and body during an accident. This helps link specific injuries to symptoms. For a detailed explanation of how low-speed crashes affect the body, see the "Low Impact Motor Vehicle Accidents" white paper from this organization.

In a rear-end collision, the neck typically moves through three stages. [59] Chen et al.[59-60] described how, in the first stage, the neck straightens and retracts, transmitting shear forces from the lower to upper cervical spine. By the end of this phase, compressive forces shift into tensile forces. In the second stage, the lower vertebrae extend and the movement travels upward, causing the upper cervical spine to extend. Shear forces remain active. In the final stage, the entire cervical spine reaches full extension, with both shear and tensile forces acting at once. [59]

Wearing a seatbelt restrains the thoracic spine, which increases forward momentum of the head. This enhances eccentric muscle strain and axial compression. [61] The injury happens so quickly that the body doesn't have time to react, placing stress on passive (non-contractile) tissues. [61]

In a frontal impact, the vehicle decelerates while the torso moves forward due to inertia. [62] The head lags behind, causing the neck to bend into an inverse S-shape: upper cervical extension with lower cervical hyperflexion. This pattern stresses areas like C7-T1 and C5-C6 and may end with the head hitting the steering wheel or windshield. [62-64]

In a side-impact collision, the occupant may be on the side of the impact (near side) or opposite it (far side). [62] When hit, the torso shifts toward the impact while the head and neck bend and rotate in the opposite direction. This forms an S-shaped curve in the frontal plane, opposite lateral bending in the upper and lower cervical spine, with possible added rotation. [65]

Pathology with Injury and Direction of Forces

In each of these impact scenarios, various tissues are affected differently depending on the mechanics of the injury and the resulting stress distribution on the body. The following sections of this paper will explore rear-impact, frontal, and lateral impact collisions.

Rear-end impact collision

Rear-end motor vehicle accidents exert specific forces that make certain neck structures especially vulnerable to injury. Studies show that neck retraction during impact is often limited by pain, likely due to stress placed on the facet joints, particularly where the superior facet compresses into the inferior facet. [66]–[67] These joints are the most common site of injury in WAD and are rich in pain-sensitive receptors. [68]-[69]

When sitting in a neutral position (back supported, looking straight ahead), the lower cervical spine (C4–C7) is subjected to significant shear forces during a rear-end impact, even when neck movement stays within normal limits and headrests are barely engaged.[70-74] The degree of acceleration change influences how much damage occurs, with possible outcomes including facet capsule tears, ligament injuries, cartilage damage, and even micro- or microfractures.[75-78] These effects may be more severe in females due to anatomical differences.[76]

Beyond the facet joints, intervertebral discs are also at risk during rear impacts. The whiplash "S-curve" motion can cause anterior distraction and posterior compression, resulting in annular tears, disc nucleus damage, and vertebral endplate avulsions.[76-77] The posterior region of the disc often sustains the greatest strain.[62-79] These injuries most commonly affect the C5–T1 disc levels,[76-81] and disc pain can persist for over two years in some patients.[82]

Rear-end impacts also damage ligaments and soft tissues in the neck. A Swedish study of over 16,000 crash victims found that rear-end collisions posed the highest risk of soft tissue injury to the neck.[83] Injuries have been reported in the anterior and posterior longitudinal ligaments, ligamentum flavum, and capsular ligaments.[19,84] The upper cervical spine (C0–C2), which contributes to nearly half of head-neck motion and lacks disc support, is especially prone to injury.[85–86] Fice and Cronin identified the apical and alar ligaments as frequently injured in rear impacts, and Kaale et al. found alar ligament damage on MRI in 66.3% of their studied group.[87–88] Patients with their heads turned at the moment of impact were more likely to have severe ligament injuries.[88] Additionally, the posterior tissues of the upper cervical spine may be strained during retraction, contributing to headaches and dizziness after WAD.[89–95]

Finally, cervical muscles can be damaged during whiplash, especially from rapid lengthening (eccentric) contractions.[19,96] Muscles commonly injured include the sternocleidomastoid, multifidus, splenius, semispinalis, trapezius, masseter, and temporalis.[90,97-98] Extension of the neck during impact strains the anterior neck muscles and ligaments as they attempt to decelerate spinal motion, leading to injuries at both muscle bellies and their entheses (attachment points).[99–101] This muscle damage is linked to myofascial pain, headaches, and, due to the multifidus' role in the facet capsule, joint dysfunction.[20,90,102-103]

Frontal Impact Collision

Frontal-impact motor vehicle accidents can cause many of the same injuries as rear-end collisions, but often involve additional trauma to the lower extremities. These injuries may result from direct impact or force transmission through the ankles, femurs, or hips—with the hip frequently being the weakest link.[64] For example, a flexed knee striking the dashboard during a frontal crash is a common source of lower limb injury.[64] Frontal impacts also carry an increased risk of head and chest injuries due to contact with the steering wheel or windshield; however, seat belts and airbags significantly reduce this risk.[64,104-108]

One study showed that cervical spine motion is often impaired after frontal impacts. Specifically, cervical flexion is most limited in the early stages post-injury, while protraction and retraction limitations can persist up to 12 months.[66] The facet joint capsules, particularly at the C5–C6 and C6–C7 levels, are vulnerable to injury due to excessive strain during the forward motion of the neck.[19,73,109] Whiplash dynamics in a frontal impact cause the vertebrae to move around a higher-than-normal pivot point, resulting in multilevel facet compression.[19] Hyperextension rebound may also injure the C2–C3 levels.[78] Additionally, eccentric muscle loading during cervical spine deceleration can stress and injure the multifidus muscle and its capsular ligament attachment.[20]

Frontal collisions can also lead to intervertebral disc injuries, but often in different ways than rear-end impacts. The C5–C6 disc is most frequently affected, with the C2–C3 disc also showing strain. [19] These injuries can involve annular tears and disc herniations, especially in the posterior annular region. [110] Since such injuries are rare in asymptomatic individuals under age 40 (3–10%), [82,111] their presence may be highly relevant in post-accident evaluations. Moreover, disc injury may contribute to chronic pain by altering cervical load distribution, accelerating facet joint degeneration. [110]

Soft tissue injuries are also common in frontal-impact cases. One study documented damage across the posterior cervical ligaments from C2 to T1.[112] Another found the supraspinous, interspinous ligaments, and ligamentum flavum frequently injured, especially at the C3–C4 level.[113] Muscles such as the trapezius, sternocleidomastoid, splenius, semispinalis, and others undergo eccentric contraction, placing additional tension on the multifidus and its joint capsule interface.[19,114–115]

Lateral Impact Collision

Lateral-impact motor vehicle accidents (MVAs) can cause injuries like frontal and rear-end collisions but also present unique risks. Occupants in lateral impacts are at higher risk for severe injury than in other types of collisions. [62] These impacts may cause the torso to rotate toward the point of impact and slip out of the shoulder belt, creating abnormal S-curve motion and placing multidirectional strain on the cervical spine.[62,99]

A unique feature of lateral collisions is the side-specific injury pattern: the side closest to impact experiences direct trauma, while the opposite side may be injured from internal contact with the vehicle.[64] Lateral impacts also produce a coupled motion of rotation and lateral flexion away from the impact, often with added flexion or extension, creating complex, multi-planar stress on facets, discs, and soft tissues.[99],[116]

Compression forces are especially significant at C6–C7, where joint space deformation has been observed.[99],[117] On the opposite side of impact, ligaments restrict motion but are subjected to distraction forces, making facet capsular ligaments particularly vulnerable.[99] One study documented motion beyond normal physiological limits at C1–C2 and C3–C7, as well as injuries to the intervertebral discs, ligamentum flavum, anterior and posterior longitudinal ligaments, and capsular ligaments, particularly from C4–T1.[62],[117]

Muscular and soft tissue injuries were especially noted contralateral to the side of lateral flexion, particularly at C3–C5. [99] Muscles such as the sternocleidomastoid, trapezius, and splenius capitis are commonly affected. [118],[119]

Injury severity from lateral impacts can also vary based on several factors, including seat position, head rotation, occupant posture, braking at impact, and startle response.[109],[120]–[126] Bracing with the arms (such as against the steering wheel) may protect during frontal impacts but offers less benefit in rear impacts.[127] Age and sex also influence injury risk, and higher impact severity increases the potential for serious injury.[128]–[130]

Nerve Injury

In all types of motor vehicle impacts, neural tissues are at risk. A reduction in intervertebral foramen height during a collision may lead to nerve root compression, especially in areas of significant tissue deformation. [99] For example, C5–C7 nerve roots and ganglia are susceptible during rear-end impacts, potentially leading to chronic radicular pain.[131] Cervical spine rotation during impact can worsen narrowing and increase the risk of injury at C3–C7.[132],[133]

Beyond mechanical compression, traumatic inertial loading—from lateral bending or whiplash—has been linked to brainstem injury.[62],[134] Sudden fluid redistribution (e.g., CSF and internal vertebral venous plexus) creates internal pressure waves, causing hydraulic trauma to neural structures.[135],[136] This may result in central sensitization, a process involving changes to the spinal cord or brain that lead to abnormal pain perception.[137] These changes can include upregulated sensory receptors, axonal sprouting, loss of inhibitory neurons, and altered synaptic behavior.

Evidence also suggests that incomplete spinal cord injury may underlie persistent pain in chronic whiplash cases.[138]–[141] A large systematic review found neuropathic pain in 34% of WAD patients, with nerve pathology in 13–32%.[5] Another study showed T2 MRI signal changes in the nerve roots, brachial plexus, and median nerve, indicating peripheral nervous system inflammation.[142] In that study, 34–75% of patients reported neuropathic pain.

Once nerve injury occurs, fibrosis, blood–nerve barrier disruption, and microvascular dysfunction can affect downstream tissues. [143],[144] Facet joint injuries, common in WAD, can trigger neurogenic inflammation, contributing to widespread hypersensitivity.[145]–[147] Inflammation in the facet-nerve pathway can impair cartilage health, with elevated levels of Substance P seen in joint surfaces.[146]

Additionally, astrocytic TSP4 (Thrombospondin-4) expression in the spinal cord increases following facet injury, leading to excitatory synaptogenesis and a lower pain threshold.[148]–[150] A study by Greening et al. found patients with chronic whiplash had nerve hyperexcitability and median nerve deformation in the carpal tunnel, persisting from 3 to 171 months post-injury.[40]

Muscle Fatty Infiltration

When neural, articular, and soft tissue injuries occur—as seen in Whiplash-Associated Disorders (WAD)—they may lead to long-term, maladaptive changes. One key phenomenon is fatty infiltration of cervical muscles, which has been linked to chronic pain and movement dysfunction in WAD. Fatty infiltration is defined as the abnormal accumulation of fat cells within or between muscle fibers. [151], [152]

In the general population, fatty infiltration is associated with aging, disuse, injury, inflammation, or nerve damage. [153], [154] However, after a whiplash injury, these changes can occur rapidly, within two weeks, due to both direct tissue damage and disrupted neural signaling to the muscles.[151],[155]

This infiltration affects both deep and superficial cervical muscles and is correlated with:

- Poor functional recovery
- Postural instability
- High disability scores
- Ligamentous injury
- Increased pain levels[156–162]

These changes appear to be unique to traumatic neck injuries and may explain why some patients are resistant to standard treatments. [161–167] Consequently, targeted rehabilitation strategies may be needed.

As noted by Snodgrass et al., monitoring for muscle fatty infiltration may have value in diagnosis, treatment planning (theragnosis), and prognosis for patients with neck pain. [168]

Traumatic Brain injuries and Concussions:
Concussion and mild traumatic brain injury (mTBI) share many clinical features and are often used interchangeably in research. [169] After a whiplash injury, it is common for patients to experience concussive or mTBI-like symptoms, including:

- Sleep disturbances.
- Sensory sensitivities and dizziness.
- Cognitive difficulties (poor concentration, memory issues, mental slowness)[13]

Motor vehicle accidents (MVAs) involving these symptoms are associated with higher healthcare costs and increased hospitalization rates. [170] These symptoms often persist for 1–3 months, and when they continue beyond 6 months, they are classified as post-concussion syndrome. [13,169]

There is significant symptom overlap between WAD and mTBI, likely due to shared mechanisms such as rotational injury. [169, 171] This may contribute to the high rate of underreported concussions in whiplash cases.

Treatment requires specialized care, as management must address both vestibular dysfunction and cervical spine involvement. [172–175] Rehabilitation programs should be tailored to the unique needs of these patients, combining neurometabolic and orthopedic expertise.

Risk Factors Affecting Prognosis

Early identification of prognostic indicators is essential in guiding appropriate, multidisciplinary treatment for patients with WAD. Clinicians experienced in managing WAD can use these factors to stratify risk and optimize care pathways. The following have been consistently identified in the literature as predictors of poorer outcomes or delayed recovery:

- Severe initial symptoms including high pain intensity, headache, lack of post-secondary education, and high functional disability scores (e.g., elevated NDI scores) are associated with worse prognosis. [130, 176–179]
- Reduced cervical range of motion, presence of neuropathic symptoms, referred arm pain, and cold hyperalgesia are additional negative prognostic signs. [18, 180,181]
- Classification of Grades 2 or 3 WAD per the Quebec Task Force system indicates higher injury complexity. [28,69,182–185]
- Psychological factors such as kinesiophobia, low perceived recovery potential, anxiety, depression, and PTSD are strongly associated with prolonged disability. [186–193]
- Psychological trauma identified within the first 3 weeks post-injury correlates with elevated pain levels at 12 months. [194]
- Head rotation at the time of impact is linked to more complicated recovery. [161]
- Delayed initiation of care following the MVA is a risk factor for chronicity. [195–197]
- Socioeconomic factors, such as lower income or part-time/unemployment status, are associated with worse outcomes. [198]
- Older age at time of injury has a statistically significant correlation with slower or incomplete recovery. [195,198–200]
- A history of headaches or pre-existing cervical conditions, even if stable at the time of the MVA, increases the risk of persistent symptoms. [44, 201–205]
- Female sex has been consistently associated with a higher likelihood of poor prognosis. [9,46,99,141,182,195,198,199,206,207]

Interventions

A variety of interventions are beneficial for patients suffering from Whiplash-Associated Disorders (WAD), including active exercise therapies targeting the spine and extremities, vestibular rehabilitation for concussion and balance dysfunction, manual therapies such as mobilization and manipulation, anti-inflammatory medications, facet and epidural injections, and orthobiologic interventions including platelet-rich plasma (PRP), prolotherapy, stem cells derived from bone marrow or adipose tissue, and exosome therapies. Each of these treatments plays a role in the early and intentional management of WAD. A single practitioner may be familiar with these methods; however, optimal recovery outcomes are best achieved through a multidisciplinary healthcare team. Such a team should not only have expertise in each treatment modality but also be capable of recognizing the broad spectrum of WAD symptoms and identifying high-risk patients.

Research has demonstrated that early aggressive treatment involving only primary care physicians and chiropractic care may negatively impact patient recovery, whereas specialized care is associated with improved outcomes. 177,196 Although some early intervention strategies have been successful 196,208–210, studies have not clearly identified the optimal timing or approach for initiating care. Importantly, triage of acute WAD patients by pain management physicians, especially those exhibiting high pain levels, neuropathic symptoms, or cold hyperalgesia, has not been evaluated in large-scale trials and may represent a critical opportunity for preventing chronic symptoms. 211 According to Jull et al., “early implementation of effective pain management for those with early moderate to high pain levels is critical in attempts to lessen the transition to chronicity”. 18 A follow-up study by the same author emphasized that future research should focus on early and effective pain management, particularly in patients with poor prognostic indicators such as high pain levels.210

Facet joint injections have been shown to be effective not only in reducing pain but also as prognostic tools in patients suffering from whiplash-related pain. 212 Early reduction of pain through such interventions may enhance recovery expectations, reduce the likelihood of chronic symptoms, and improve long-term outcomes. 213–218 Furthermore, despite the known influence of concussion or mild traumatic brain injury (mTBI) on pain and disability, these conditions have often been excluded from research evaluating musculoskeletal outcomes in WAD. 209,210,219–221 Given that chronic concussion symptoms can negatively affect prognosis in musculoskeletal injuries. 190,222–225 comprehensive screening for concussion or mTBI should be integrated into WAD assessments.

When a collaborative team is capable of accurately diagnosing symptoms and pain generators, stratifying patients by risk, educating them on their condition, and applying appropriate interventions tailored to their symptoms and functional deficits, the likelihood of successful recovery significantly increases (226–232). Therefore, it is strongly recommended that patients, and their legal representatives, seek multidisciplinary care as early as possible. Timely referral for evaluation and treatment can assist in determining prognosis, initiating targeted interventions, and ultimately improving outcomes for those with WAD.

Conclusion

Whiplash-Associated Disorders (WAD) represent a significantly more complex clinical entity than a simple neck sprain. The condition involves a multifactorial interplay of biomechanical, neurological, psychological, and inflammatory components that often span across multiple bodily systems. As such, optimal patient outcomes require a multidisciplinary approach involving specialists from various healthcare domains. Achieving meaningful recovery and restoration of function depends on a comprehensive understanding of the underlying pathoanatomy, the specific mechanisms of injury, individualized prognostic indicators, and the timely implementation of appropriate, evidence-based interventions. This depth of insight is critical not only for alleviating pain but also for facilitating the patient's return to pre-injury levels of activity and quality of life.

References

1. Bragg KJ, Varacallo M. Cervical sprain. StatPearls [Internet]: StatPearls Publishing, 2023.
2. Ghorayeb JH. The nosological classification of whiplash-associated disorder: a narrative review. The Journal of the Canadian Chiropractic Association 2021;65:76.
3. Vállez García D, Dierckx RAJO, Otte A, et al. Whiplash, Real or Not Real? A Review and New Concept. PET and SPECT in Neurology, 2014;947-963.
4. Bannister G, Amirfeyz R, Kelley S, et al. Whiplash injury. The Journal of Bone & Joint Surgery British Volume 2009;91:845-850.
5. Fundaun J, Kolski M, Baskozos G, et al. Nerve pathology and neuropathic pain after whiplash injury: a systematic review and meta-analysis. Pain 2022;163:e789-e811.
6. Särkilahti N, Leino S, Takatalo J, et al. The symptom profile of people with whiplash-associated disorder–a mixed-method systematic review. Journal of Bodywork and Movement Therapies 2024.
7. Alderink GJ, Ashby BM. Cervical Spine: Lateral Whiplash, Neck Pain, and Nerve Palsy. Clinical Kinesiology and Biomechanics: A Problem-Based Learning Approach: Springer, 2023;145-177.
8. Al-Khazali HM, Ashina H, Iljazi A, et al. Psychiatric sequelae following whiplash injury: a systematic review. Frontiers in psychiatry 2022;13:814079.
9. Kuperman P, Granovsky Y, Fadel S, et al. Head- and neck-related symptoms post-motor vehicle collision (MVC): Separate entities or two-sides of the same coin? Injury 2021;52:1227-1233.
10. Sterling M, Jull G, Vicenzino B, et al. Physical and psychological factors predict outcome following whiplash injury. Pain 2005;114:141-148.
11. Rebbeck T, Evans K, Elliott JM. Concussion in Combination With Whiplash-Associated Disorder May Be Missed in Primary Care: Key Recommendations for Assessment and Management. J Orthop Sports Phys Ther 2019;49:819-828.
12. Gil C, Decq P. How similar are whiplash and mild traumatic brain injury? A systematic review. Neurochirurgie 2021;67:238-243.
13. Alexander MP. The evidence for brain injury in whiplash injuries. Pain Research and Management 2003;8:19-23.
14. He Y, Gao J, Liu Y, et al. Global trends and hotspots related to whiplash injury: A visualization study. Medicine 2024;103:e38777.
15. Stone D, Ward E, Knijnik S, et al. Whiplash-associated dysphagia and dysphonia: A scoping review. Dysphagia 2021;36:303-315.
16. Rodriquez AA, Barr KP, Burns SP. Whiplash: pathophysiology, diagnosis, treatment, and prognosis. Muscle Nerve 2004;29:768-781.
17. Haniffah A, Dawal S, Julaihi S. Whiplash injury mechanisms of car rear occupants: a review. Malaysian Journal of Public Health Medicine 2020;1:272-281.
18. Jull GA, Söderlund A, Stemper BD, et al. Toward optimal early management after whiplash injury to lessen the rate of transition to chronicity. Spine 2011;36:S335-S342.
19. Park D. The pathophysiology and mechanism of acute and chronic whiplash injury: a narrative review. International Journal of Pain 2022;13:20-24.
20. Siegmund GP, Winkelstein BA, Ivancic PC, et al. The anatomy and biomechanics of acute and chronic whiplash injury. Traffic Injury Prevention 2009;10:101-112.
21. Holm LW, Carroll LJ, Cassidy JD, et al. The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions: results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. Spine 2008;33:S52-S59.
22. Galasko CS, Murray P, Stephenson W. Incidence of whiplash-associated disorder. British Columbia Medical Journal 2002;44:237-240.
23. Kuppa S. Injury criteria and anthropomorphic test devices for whiplash injury assessment. NHTSA Docket 2004.
24. Hayashi K, Miki K, Ikemoto T, et al. Predictors of high-cost patients with acute whiplash-associated disorder in Japan. PLoS One 2023;18:e0287676.
25. Pink J, Petrou S, Williamson E, et al. Economic and health-related quality of life outcomes of whiplash associated disorders. Spine 2016;41:1378-1386.
26. Elliott JM, Walton DM. How do we meet the challenge of whiplash? JOSPT 2017;444-446.
27. Spitzer W, Skovron M, Salmi L, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining “whiplash” and its management. Spine 1995;20:1S-73S.
28. Hartling L, Brison RJ, Ardern C, et al. Prognostic value of the Quebec classification of whiplash-associated disorders. Spine 2001;26:36-41.
29. Shergill Y, Cote P, Shearer H, et al. Inter-rater reliability of the Quebec Task Force classification system for recent-onset whiplash-associated disorders. The Journal of the Canadian Chiropractic Association 2021;65:186.
30. Al-Khazali HM, Ashina H, Iljazi A, et al. Neck pain and headache after whiplash injury: a systematic review and meta-analysis. Pain 2020;161:880-888.
31. Stenneberg MS, Scholten-Peeters GG, den Uil CS, et al. Clinical characteristics differ between patients with non-traumatic neck pain, patients with whiplash-associated disorders, and pain-free individuals. Physiotherapy Theory and Practice 2022;38:2592-2602.
32. Stenneberg MS, Rood M, de Bie R, et al. To what degree does active cervical range of motion differ between patients with neck pain, patients with whiplash, and those without neck pain? A systematic review and meta-analysis. Archives of Physical Medicine and Rehabilitation 2017;98:1407-1434.
33. Anstey R, Kongsted A, Kamper S, et al. Are people with whiplash-associated neck pain different from people with nonspecific neck pain? Journal of Orthopaedic & Sports Physical Therapy 2016;46:894-901.
34. Kosek E, Januszewska A. Mechanisms of pain referral in patients with whiplash-associated disorder. European Journal of Pain 2008;12:650-660.
35. Fernández-Pérez AM, Villaverde-Gutiérrez C, Mora-Sánchez A, et al. Muscle trigger points, pressure pain threshold, and cervical range of motion in patients with high level of disability related to acute whiplash injury. Journal of Orthopaedic & Sports Physical Therapy 2012;42:634-641.
36. Parikh SS, Salazar T, Taborda R. Neck Pain: Whiplash and Cervicogenic Headache. Clinical Guide to Musculoskeletal Medicine: A Multidisciplinary Approach: Springer, 2022;51-57.
37. Anarte-Lazo E, Falla D, Devecchi V, et al. Differences in physical examination findings between those who present with or without headache soon after a whiplash injury: a cross-sectional study. Journal of Manual & Manipulative Therapy 2024;32:619-629.
38. Shanti BF, Shanti IF, Shanti ZIF. Whiplash Injuries: A Systemic Review. Acta Scientific Neurology 2023;6:51-67.

39. Watanabe K, Daimon K, Fujiwara H, et al. The long-term impact of whiplash injuries on patient symptoms and the associated degenerative changes detected using MRI: a prospective 20-year follow-up study comparing patients with whiplash-associated disorders with asymptomatic subjects. Spine 2021;46:710-716.
40. Greening J, Anantharaman K, Young R, et al. Evidence for increased magnetic resonance imaging signal intensity and morphological changes in the brachial plexus and median nerves of patients with chronic arm and neck pain following whiplash injury. Journal of Orthopaedic & Sports Physical Therapy 2018;48:523-532.
41. Ludvigsson ML, Peterson G, Widh S, et al. Exercise, headache, and factors associated with headache in chronic whiplash: analysis of a randomized clinical trial. Medicine 2019;98:e18130.
42. Anarte-Lazo E, Abichandani D, Rodriguez-Blanco C, et al. Headache features in people with whiplash associated disorders: a scoping review. Musculoskeletal Science and Practice 2023;66:102802.
43. Obermann M, Nebel K, Riegel A, et al. Incidence and predictors of chronic headache attributed to whiplash injury. Cephalalgia 2010;30:528-534.
44. Drottning M, Staff P, Sjaastad O. Cervicogenic headache (CEH) after whiplash injury. Cephalalgia 2002;22:165-171.
45. Lucas S. Headache management in concussion and mild traumatic brain injury. PM&R 2011;3:S406-S412.
46. Oka H, Matsudaira K, Fujii T, et al. Risk factors for prolonged treatment of whiplash-associated disorders. PLoS One 2015;10:e0132191.
47. Seroussi R, Singh V, Fry A. Chronic whiplash pain. Physical Medicine and Rehabilitation Clinics 2015;26:359-373.
48. Robinson JP, Burwinkle T, Turk DC. Perceived and actual memory, concentration, and attention problems after whiplash-associated disorders (grades I and II): prevalence and predictors. Archives of Physical Medicine and Rehabilitation 2007;88:774-779.
49. Peixoto C, Buchanan DM, Nahas R. Missed emergency department diagnosis of mild traumatic brain injury in patients with chronic pain after motor vehicle collision. Pain Physician 2023;26:101.
50. Moen U, Magnussen LH, Wilhelmsen KT, et al. Prevalence and distribution of musculoskeletal pain in patients with dizziness—a systematic review. Physiotherapy Research International 2022;27:e1941.
51. Humphreys BK, Bolton J, Peterson C, et al. A cross-sectional study of the association between pain and disability in neck pain patients with dizziness of suspected cervical origin. Journal of Whiplash & Related Disorders 2002;1:63-73.
52. Mallinson A, Maire R, Beyaert C, et al. Understanding and managing trauma-induced vestibular deficits. The Journal of International Advanced Otology 2021;17:559.
53. Tranter R, Graham JR. A review of the otological aspects of whiplash injury. Journal of Forensic and Legal Medicine 2009;16:53-55.
54. Folmer RL, Griest SE. Chronic tinnitus resulting from head or neck injuries. The Laryngoscope 2003;113:821-827.
55. Campagna CA, Anauate J, Vasconcœlos LGE, et al. Effectiveness of dry needling in bothersome chronic tinnitus in patients with myofascial trigger points. International Archives of Otorhinolaryngology 2022;26:e233-e242.
56. Salé H, Isberg A. Delayed temporomandibular joint pain and dysfunction induced by whiplash trauma: a controlled prospective study. The Journal of the American Dental Association 2007;138:1084-1091.
57. Häggman-Henrikson B, Lövgren A, Wu WYY, et al. Prevalence of temporomandibular disorder symptoms after whiplash trauma—a systematic review and meta-analysis. European Journal of Pain 2025;29:e4792.
58. Grönqvist J, Häggman-Henrikson B, Eriksson P-O. Impaired jaw function and eating difficulties in whiplash-associated disorders. Swed Dent J 2008;32:171-177.
59. Chen H-b, King HY, Wang Z-g. Biomechanics of whiplash injury. Chinese Journal of Traumatology 2009;12:305-314.
60. Grauer JN, Panjabi MM, Cholewicki J, et al. Whiplash produces an S-shaped curvature of the neck with hyperextension at lower levels. Spine 1997;22:2489-2494.
61. Kohles SS, McClaren JW. A stochastic model validated with human test data causally associating target vehicle Delta V, occupant cervicocranial biomechanics, and injury during rear-impact crashes. Journal of Forensic and Legal Medicine 2022;91:102431.
62. Ivancic PC. Mechanisms and mitigation of head and spinal injuries due to motor vehicle crashes. Journal of Orthopaedic & Sports Physical Therapy 2016;46:826-833.
63. Ivancic PC, Ito S, Panjabi MM, et al. Intervertebral neck injury criterion for simulated frontal impacts. Traffic Injury Prevention 2005;6:175-184.
64. Eid HO, Abu-Zidan FM. Biomechanics of road traffic collision injuries: a clinician's perspective. Singapore Medical Journal 2007;48:693.
65. Panjabi MM, Ivancic PC, Tominaga Y, et al. Intervertebral neck injury criterion for prediction of multiplanar cervical spine injury due to side impacts. Traffic Injury Prevention 2005;6:387-395.
66. Bunketorp OB, Elisson LK. Cervical status after neck sprains in frontal and rear-end car impacts. Injury 2012;43:423-430.
67. Lentell G, Kruse M, Chock B, et al. Dimensions of the cervical neural foramina in resting and retracted positions using magnetic resonance imaging. Journal of Orthopaedic & Sports Physical Therapy 2002;32:380-390.
68. Curatolo M, Bogduk N, Ivancic PC, et al. The role of tissue damage in whiplash-associated disorders: discussion paper 1. Spine 2011;36:S309-S315.
69. Eseonu K, Panchmatia J, Pang D, et al. A review of the clinical utility of therapeutic facet joint injections in whiplash-associated cervical spinal pain. Spine Surgery and Related Research 2022;6:189-196.
70. Kaneoka K, Ono K, Inami S, et al. The human cervical spine motion during rear-impact collisions. Journal of Whiplash & Related Disorders 2011;1:85-97.
71. Hellinga M, van Eerd M, Stojanovic M, et al. Cervical facet pain: Degenerative alterations and whiplash-associated disorder. Pain Practice 2025;25:e70005.
72. Smotrova E, Morris L, McNally D. Comparison of standard automotive industry injury predictors and actual injury sustained during significant whiplash events. European Spine Journal 2021;30:3043-3058.
73. Pearson AM, Ivancic PC, Ito S, et al. Facet joint kinematics and injury mechanisms during simulated whiplash. Spine 2004;29:390-397.

74. Deng B. Kinematics of human cadaver cervical spine during low-speed rear-end impacts. Wayne State University, 1999.
75. Tencer A, Huber P, Mirza S. A comparison of biomechanical mechanisms of whiplash injury from rear impacts. Annual Proceedings/Association for the Advancement of Automotive Medicine 2003;383.
76. Love BM. Finite Element Model-based Assessment of the Female Cervical Spine in a Rear-End Impact Simulation, 2022.
77. Taylor. Cervical Spinal Injuries: An Autopsy Study of 109 Blunt Injuries. 1996.
78. Dowdell J, Kim J, Overley S, et al. Biomechanics and common mechanisms of injury of the cervical spine. Handbook of Clinical Neurology 2018;158:337-344.
79. Yoganandan N, Cusick JF, Pintar FA, et al. Whiplash injury determination with conventional spine imaging and cryomicrotomy. Spine 2001;26:2443-2448.
80. Zhang J-G, Wang F, Zhou R, et al. A three-dimensional finite element model of the cervical spine: an investigation of whiplash injury. Medical & Biological Engineering & Computing 2011;49:193-201.
81. Panjabi MM, Ito S, Ivancic PC, et al. Evaluation of the intervertebral neck injury criterion using simulated rear impacts. Journal of Biomechanics 2005;38:1694-1701.
82. Pettersson K, Hildingsson C, Toolanen G, et al. Disc pathology after whiplash injury: a prospective magnetic resonance imaging and clinical investigation. Spine 1997;22:283-287.
83. Jakobsson L, Lundell B, Norin H, et al. WHIPS–Volvo's whiplash protection study. Accident Analysis & Prevention 2000;32:307-319.
84. Tominaga Y, Ndu AB, Coe MP, et al. Neck ligament strength is decreased following whiplash trauma. BMC Musculoskeletal Disorders 2006;7:1-9.
85. Wang Y, Jiang H, Teo EC, et al. Finite Element Analysis of Head–Neck Kinematics in Rear-End Impact Conditions with Headrest. Bioengineering 2023;10:1059.
86. Beyer B, Feipel V, Dugailly P-M. Biomechanics of the upper cervical spine ligaments in axial rotation and flexion-extension: considerations into the clinical framework. Journal of Craniovertebral Junction and Spine 2020;11:217-225.
87. Fice JB, Cronin DS. Investigation of whiplash injuries in the upper cervical spine using a detailed neck model. Journal of Biomechanics 2012;45:1098-1102.
88. Kaale BR, Krakenes J, Albrektsen G, et al. Head position and impact direction in whiplash injuries: associations with MRI-verified lesions of ligaments and membranes in the upper cervical spine. Journal of Neurotrauma 2005;22:1294-1302.
89. Ordway NR, Seymour RJ, Donelson RG, et al. Cervical flexion, extension, protrusion, and retraction: a radiographic segmental analysis. Spine 1999;24:240-247.
90. Anarte-Lazo E, Rodriguez-Blanco C, Bernal-Utrera C, et al. Headache production during physical examination in patients with and without headache attributed to a whiplash injury: a case-control study. Musculoskeletal Science and Practice 2023;66.
91. Malo-Urriés M, Tricás-Moreno JM, Estébanez-de-Miguel E, et al. Immediate effects of upper cervical translatoric mobilization on cervical mobility and pressure pain threshold in patients with cervicogenic headache: a randomized controlled trial. Journal of Manipulative and Physiological Therapeutics 2017;40:649-658.
92. Hall T, Briffa K, Hopper D, et al. Reliability of manual examination and frequency of symptomatic cervical motion segment dysfunction in cervicogenic headache. Manual Therapy 2010;15:542-546.
93. Reid D, Rebbeck T, McCarthy C. Clinical reasoning for complex cervical spine conditions. International Journal of Osteopathic Medicine 2018;27:45-51.
94. Escaloni J, Butts R, Dunning J. The use of dry needling as a diagnostic tool and clinical treatment for cervicogenic dizziness: a narrative review & case series. Journal of Bodywork and Movement Therapies 2018;22:947-955.
95. Treleaven J. Dizziness, unsteadiness, visual disturbances, and sensorimotor control in traumatic neck pain. Journal of Orthopaedic & Sports Physical Therapy 2017;47:492-502.
96. Han J, Kim HJ, Song L, et al. Characteristics of human responses in a braked stationary lead vehicle during low-speed, rear-end collisions. International Journal of Precision Engineering and Manufacturing 2019;20:1255-1264.
97. Kumar. The Effect of a 3-Point Harness Restraint and Car Seat in Whiplash-Type Lateral Impacts. Spine 2006.
98. Brolin K, Halldin P, Leijonhufvud I. The effect of muscle activation on neck response. Traffic Injury Prevention 2006;6:67-76.
99. Shen T-WD. Investigation of whiplash associated disorders using finite element neck models with active musculature in frontal, rear, and lateral impact. University of Waterloo, 2020.
100. Yoganandan. Biomechanical mechanisms of whiplash injury. Traffic Injury Prevention 2002.
101. Bismil Q, Bismil M. Myofascial-entheseal dysfunction in chronic whiplash injury: an observational study. JRSM Short Reports 2012;3:1-4.
102. Bodes-Pardo G, Pecos-Martín D, Gallego-Izquierdo T, et al. Manual treatment for cervicogenic headache and active trigger point in the sternocleidomastoid muscle: a pilot randomized clinical trial. Journal of Manipulative and Physiological Therapeutics 2013;36:403-411.
103. Castaldo M, Ge H-Y, Chiarotto A, et al. Myofascial trigger points in patients with whiplash-associated disorders and mechanical neck pain. Pain Medicine 2014;15:842-849.
104. Newgard CD, Lewis RJ, Kraus JF. Steering wheel deformity and serious thoracic or abdominal injury among drivers and passengers involved in motor vehicle crashes. Annals of Emergency Medicine 2005;45:43-50.
105. Chen R, Gabler HC. Risk of thoracic injury from direct steering wheel impact in frontal crashes. Journal of Trauma and Acute Care Surgery 2014;76:1441-1446.
106. Hitosugi M, Mizuno K, Nagai T, et al. Analysis of maxillofacial injuries of vehicle passengers involved in frontal collisions. Journal of Oral and Maxillofacial Surgery 2011;69:1146-1151.
107. Todorovic M, Vukcevic B, Cabarkapa M, et al. The assessment of airbag deployment and seatbelt use in preventing facial injuries. Forensic Science, Medicine and Pathology 2018;14:503-508.
108. Xiao S, You S, Tian T, et al. Investigation of lower limb injury under different contact stiffness for drivers during frontal crash. Acta of Bioengineering and Biomechanics 2022;24:83-93.

109. Shateri H, Cronin DS. Out-of-position rear impact tissue-level investigation using detailed finite element neck model. Traffic Injury Prevention 2015;16:698-708.
110. Ito S, Ivancic PC, Pearson A, et al. Cervical intervertebral disc injury during simulated frontal impact. European Spine Journal 2005;14:356-365.
111. D'Antoni AV, Croft AC. Prevalence of herniated intervertebral discs of the cervical spine in asymptomatic subjects using MRI scans: a qualitative systematic review. Journal of Whiplash & Related Disorders 2006;5:5-13.
112. Pearson AM, Panjabi MM, Ivancic PC, et al. Frontal impact causes ligamentous cervical spine injury. Spine 2005;30:1852-1858.
113. Panjabi MM, Pearson AM, Ito S, et al. Cervical spine ligament injury during simulated frontal impact. Spine 2004;29:2395-2403.
114. Fanta O, Hadraba D, Lopot F, et al. Pre-activation and muscle activity during frontal impact in relation to whiplash associated disorders. Neuroendocrinology Letters 2013;34:708-716.
115. Fice JB. Neuromechanics of Neck Muscles: Implications for Whiplash Injury. University of British Columbia (Vancouver), 2019.
116. Kumar S, Ferrari R, Narayan Y. Electromyographic and kinematic exploration of whiplash-type neck perturbations in left lateral collisions. Spine 2004;29:650-659.
117. Maak TG, Ivancic PC, Tominaga Y, et al. Side impact causes multiplanar cervical spine injuries. Journal of Trauma and Acute Care Surgery 2007;63:1296-1307.
118. Kumar S, Ferrari R, Narayan Y, et al. The effect of a 3-point harness restraint and car seat in whiplash-type lateral impacts. Spine 2006;31:E11-E18.
119. Sacher N, Frayne RJ, Dickey JP. Investigating cervical muscle response and head kinematics during right, left, frontal, and rear-seated perturbations. Traffic Injury Prevention 2012;13:529-536.
120. Yang KH, King AL. Neck kinematics in rear-end impacts. Pain Research and Management 2003;8:79-85.
121. Emori RI, Horiguchi J. Whiplash in low speed vehicle collisions. SAE Technical Paper, 1990.
122. Han J, Kim HJ, Song L, et al. Characteristics of human responses in a braked stationary lead vehicle during low-speed, rear-end collisions. International Journal of Precision Engineering and Manufacturing 2019;20:1255-1264.
123. Kang Y-S, Stammen J, Ramachandra R, et al. Biomechanical responses and injury assessment of post-mortem human subjects in various rear-facing seating configurations. SAE Technical Paper, 2021.
124. Omerovic S, Tomasch E, Gutsche AJ, et al. Comparative study of potential whiplash injuries for different occupant seated positions during rear-end accidents. Acta of Bioengineering and Biomechanics 2016;18.
125. Mang DWH, Siegmund GP, Inglis JT, et al. The startle response during whiplash: a protective or harmful response? Journal of Applied Physiology 2012;113:532-540.
126. Mang DW, Siegmund GP, Blouin J-S. Whiplash evokes descending muscle recruitment and sympathetic responses characteristic of startle. The Journal of the Canadian Chiropractic Association 2014;58:109.
127. Fice JB, Mang DW, Ólafsdóttir JM, et al. Neck muscle and head/neck kinematic responses while bracing against the steering wheel during front and rear impacts. Annals of Biomedical Engineering 2021;49:1069-1082.
128. Yoganandan N, Chirvi S, Voo L, et al. Role of age and injury mechanism on cervical spine injury tolerance from head contact loading. Traffic Injury Prevention 2018;19:165-172.
129. Carmo GP, Grigioni J, Fernandes FA, et al. Biomechanics of traumatic head and neck injuries on women: a state-of-the-art review and future directions. Biology 2023;12:83.
130. Callan B, Walton DM, Cleland J, et al. Exploring sex as a moderator of other prognostic variables in whiplash associated disorder: an observational study. PLoS One 2023;18:e0282640.
131. Panjabi MM, Maak TG, Ivancic PC, et al. Dynamic intervertebral foramen narrowing during simulated rear impact. Spine 2006;31:E128-E134.
132. Maak TG. Dynamic Intervertebral Foramen Narrowing During Whiplash. 2006.
133. Tominaga Y, Maak TG, Ivancic PC, et al. Head-turned rear impact causing dynamic cervical intervertebral foramen narrowing: implications for ganglion and nerve root injury. Journal of Neurosurgery: Spine 2006;4:380-387.
134. Gennarelli TA, Thibault LE, Tomei G, et al. Directional dependence of axonal brain injury due to centroidal and non-centroidal acceleration. SAE Transactions 1987:1355-1359.
135. Yao H-D, Svensson MY, Nilsson H. Transient pressure changes in the vertebral canal during whiplash motion–a hydrodynamic modeling approach. Journal of Biomechanics 2016;49:416-425.
136. Yao H-D, Svensson MY, Nilsson H. Deformation of dorsal root ganglion due to pressure transients of venous blood and cerebrospinal fluid in the cervical vertebral canal. Journal of Biomechanics 2018;76:16-26.
137. Davis CG. Mechanisms of chronic pain from whiplash injury. Journal of Forensic and Legal Medicine 2013;20:74-85.
138. Elliott JM, Dewald JPA, Hornby TG, et al. Mechanisms underlying chronic whiplash: contributions from an incomplete spinal cord injury? Pain Medicine 2014;15:1938-1944.
139. Smith AC, Parrish TB, Hoggarth MA, et al. Potential associations between chronic whiplash and incomplete spinal cord injury. Spinal Cord Series and Cases 2015;1:1-6.
140. Elliott JM, Hancock MJ, Crawford RJ, et al. Advancing imaging technologies for patients with spinal pain: with a focus on whiplash injury. The Spine Journal 2018;18:1489-1497.
141. Hoggarth MA, Elliott JM, Smith ZA, et al. Macromolecular changes in spinal cord white matter characterize whiplash outcome at 1-year post motor vehicle collision. Scientific Reports 2020;10:22221.
142. Ríos-León M, Taylor J, Segura-Fragoso A, et al. Usefulness of the DN4, S-LANSS, and painDETECT screening questionnaires to detect the neuropathic pain components in people with acute whiplash-associated disorders: a cross-sectional study. Pain Medicine 2024;25:344-351.

143. Lim TK, Shi XQ, Johnson JM, et al. Peripheral nerve injury induces persistent vascular dysfunction and endoneurial hypoxia, contributing to the genesis of neuropathic pain. Journal of Neuroscience 2015;35:3346-3359.
144. Lim TK, Shi XQ, Martin HC, et al. Blood-nerve barrier dysfunction contributes to the generation of neuropathic pain and allows targeting of injured nerves for pain relief. Pain 2014;155:954-967.
145. Duarte FC, Hurtig M, Clark A, et al. Experimentally induced spine osteoarthritis in rats leads to neurogenic inflammation within neurosegmentally linked myotomes. Experimental Gerontology 2021;149:111311.
146. Duarte FC, Zwambag DP, Brown SH, et al. Increased substance P immunoreactivity in ipsilateral knee cartilage of rats exposed to lumbar spine injury. Cartilage 2020;11:251-261.83-93.
147. MacDonald DI, Jayabalan M, Seaman JT, et al. Pain persists in mice lacking both Substance P and CGRPα signaling. eLife 2025;13:RP93754.
148. Crosby ND, Zaucke F, Kras JV, et al. Thrombospondin-4 and excitatory synaptogenesis promote spinal sensitization after painful mechanical joint injury. Experimental Neurology 2015;264:111-120.
149. Sterling M. Differential development of sensory hypersensitivity and a measure of spinal cord hyperexcitability following whiplash injury. Pain 2010;150:501-506.
150. Crosby ND, Gilliland TM, Winkelstein BA. Early afferent activity from the facet joint after painful trauma to its capsule potentiates neuronal excitability and glutamate signaling in the spinal cord. Pain 2014;155:1878-1887.
151. Zhu Y, Hu Y, Pan Y, et al. Fatty infiltration in the musculoskeletal system: pathological mechanisms and clinical implications. Frontiers in Endocrinology 2024;15:1406046.
152. Wang L, Valencak TG, Shan T. Fat infiltration in skeletal muscle: influential triggers and regulatory mechanism. iScience 2024;27.
153. Li Z, Liang Q, Li H, et al. Fatty infiltration of the cervical multifidus musculature and its clinical correlation to cervical spondylosis. BMC Musculoskeletal Disorders 2023;24:613.
154. Linnman C, Appel L, Fredrikson M, et al. Elevated [11C]-D-deprenyl uptake in chronic whiplash associated disorder suggests persistent musculoskeletal inflammation. PLoS One 2011;6:e19182.
155. Elliott JM, Dewald JP, Hornby TG, et al. Mechanisms underlying chronic whiplash: contributions from an incomplete spinal cord injury? Pain Medicine 2014;15:1938-1944.
156. Karlsson A, Leinhard OD, Åslund U, et al. An investigation of fat infiltration of the multifidus muscle in patients with severe neck symptoms associated with chronic whiplash-associated disorder. Journal of Orthopaedic & Sports Physical Therapy 2016;46:886-893.
157. Peolsson A, Karlsson A, West J, et al. Increased fatty infiltration of the multifidus muscle in individuals with severe disability due to chronic whiplash-associated disorders. Physiotherapy 2015;101:e1190.
158. Lund N, Dahlqvist Leinhard O, Elliott JM, et al. Fatty infiltrate and neck muscle volume in individuals with chronic whiplash-associated disorders compared to healthy controls–a cross-sectional case–control study. BMC Musculoskeletal Disorders 2023;24:181.
159. Elliott JM, O'Leary S, Sterling M, et al. Magnetic resonance imaging findings of fatty infiltrate in the cervical flexors in chronic whiplash. Spine 2010;35:948-954.
160. Elliott JM, Courtney DM, Rademaker A, et al. The rapid and progressive degeneration of the cervical multifidus in whiplash: an MRI study of fatty infiltration. Spine 2015;40:E694-E700.
161. Elliott JM, Noteboom JT, Flynn TW, et al. Characterization of acute and chronic whiplash-associated disorders. Journal of Orthopaedic & Sports Physical Therapy 2009;39:312-323.
162. Peolsson A, Bahat HS, German D, et al. Results of neck-specific exercise for altered postural sway in individuals with chronic whiplash-associated disorders: a longitudinal case–control study. Scientific Reports 2024;14:15235.
163. O'Leary S, Jull G, Van Wyk L, et al. Morphological changes in the cervical muscles of women with chronic whiplash can be modified with exercise—a pilot study. Muscle & Nerve 2015;52:772-779.
164. Elliott JM. Are there implications for morphological changes in neck muscles after whiplash injury? Spine 2011;36:S205-S210.
165. Elliott J, Galloway G, Jull G, et al. Magnetic resonance imaging analysis of the upper cervical spine extensor musculature in an asymptomatic cohort: an index of fat within muscle. Clinical Radiology 2005;60:355-363.
166. Peterson G, Nilsson D, Peterson S, et al. Changes in dorsal neck muscle function in individuals with chronic whiplash-associated disorders: a real-time ultrasound case–control study. Ultrasound in Medicine & Biology 2016;42:1090-1102.
167. Kwon H-J, Kim C-S, Kim S, et al. Association between fatty infiltration in the cervical multifidus and treatment response following cervical interlaminar epidural steroid injection. The Korean Journal of Pain 2023;36:358-368.
168. Snodgrass SJ, Weber KA, Wesselink EO, et al. Reduced cervical muscle fat infiltrate is associated with self-reported recovery from chronic idiopathic neck pain over six months: a magnetic resonance imaging longitudinal cohort study. Journal of Clinical Medicine 2024;13:4485.
169. Morin M, Langevin P, Fait P. Cervical spine involvement in mild traumatic brain injury: a review. Journal of Sports Medicine 2016;2016:1590161.
170. Vattipally VN, Weber-Levine C, Jiang K, et al. Motor vehicle collision characteristics and hospitalization outcomes associated with mild traumatic brain injury and concomitant whiplash injury. Neurosurgical Focus 2024;57:E14.
171. Zumsteg D, Wennberg R, Gütling E, et al. Whiplash and concussion: similar acute changes in middle-latency SEPs. Canadian Journal of Neurological Sciences 2006;33:379-386.
172. Rosker ZM, Kristjansson E, Vodicar M. How well can we detect cervical-driven sensorimotor dysfunction in concussion patients? An observational study comparing patients with idiopathic neck pain, whiplash-associated disorders, and concussion. Gait & Posture 2023;101:21-27.
173. Cheever K, McDevitt J, Phillips J, et al. The role of cervical symptoms in post-concussion management: a systematic review. Sports Medicine 2021;51:1875-1891.

174. Marshall CM, Vernon H, Leddy JJ, et al. The role of the cervical spine in post-concussion syndrome. The Physician and Sportsmedicine 2015;43:274-284.
175. Stenbro LW, Hellemose LA, Kothari SF, et al. Cervical range of motion and pericranial muscle tenderness in patients with persistent post-concussion symptoms: a cross-sectional study. The Journal of Head Trauma Rehabilitation 2025:10.1097.
176. Walton DM, Pretty J, MacDermid JC, et al. Risk factors for persistent problems following whiplash injury: results of a systematic review and meta-analysis. Journal of Orthopaedic & Sports Physical Therapy 2009;39:334-350.
177. Côté P, Hogg-Johnson S, Cassidy JD, et al. Early aggressive care and delayed recovery from whiplash: isolated finding or reproducible result? Arthritis Care & Research 2007;57:861-868.
178. Anarte-Lazo E, Barbero M, Bernal-Utrera C, et al. The association between neuropathic pain features and central sensitization with acute headache associated to a whiplash injury. Musculoskeletal Science and Practice 2024;74.
179. Ritchie C, Sterling M. Recovery pathways and prognosis after whiplash injury. Journal of Orthopaedic & Sports Physical Therapy 2016;46:851-861.
180. Jakobsson L, Norin H, Bunketorp O. Whiplash-associated disorders in frontal impacts: influencing factors and consequences. Traffic Injury Prevention 2003;4:153-161.
181. Farrell SF, Armfield NR, Kristjansson E, et al. Trajectories of cold but not mechanical sensitivity correspond with disability trajectories after whiplash injury. Pain 2022:10.1097.
182. Walton DM, MacDermid JC, Giorgianni AA, et al. Risk factors for persistent problems following acute whiplash injury: update of a systematic review and meta-analysis. Journal of Orthopaedic & Sports Physical Therapy 2013;43:31-43.
183. Tournier C, Hours M, Charnay P, et al. Five years after the accident, whiplash casualties still have poorer quality of life in the physical domain than other mildly injured casualties: analysis of the ESPARR cohort. BMC Public Health 2015;16:1-13.
184. Nedić D, Pilija V. Risk factors for developing chronic whiplash disorders. Journal of Back and Musculoskeletal Rehabilitation 2022;35:213-219.
185. Alalawi A, Devecchi V, Gallina A, et al. Assessment of neuromuscular and psychological function in people with recurrent neck pain during a period of remission: cross-sectional and longitudinal analyses. Journal of Clinical Medicine 2022;11.
186. Anarte-Lazo E, Liew BXW, Devecchi V, et al. Network analyses reveal the interaction between physical features, fear of movement and neck pain and disability in people with acute and chronic whiplash-associated disorders. European Journal of Pain 2023;28:322-334.
187. Ravn SL, Karstoft KI, Sterling M, et al. Trajectories of posttraumatic stress symptoms after whiplash: a prospective cohort study. European Journal of Pain 2019;23:515-525.
188. Buitenhuis J, de Jong PJ, Jaspers JP, et al. Relationship between posttraumatic stress disorder symptoms and the course of whiplash complaints. Journal of Psychosomatic Research 2006;61:681-689.
189. Campbell L, Smith A, McGregor L, et al. Psychological factors and the development of chronic whiplash–associated disorder(s): a systematic review. The Clinical Journal of Pain 2018;34:755-768.
190. Carroll LJ, Holm LW, Hogg-Johnson S, et al. Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. Spine 2008;33:S83-S92.
191. Pedrero-Martin Y, Falla D, Rodriguez-Brazzarola P, et al. Prognostic factors of perceived disability and perceived recovery after whiplash: a longitudinal, prospective study with one-year follow-up. The Clinical Journal of Pain 2024;40:165-173.
192. Szucs G. Emotionally concussed: the impact of pre-existing anxiety on concussion recovery. 2023.
193. Elliott J, Walton D, Albin S, et al. Biopsychosocial sequelae and recovery trajectories from whiplash injury following a motor vehicle collision. The Spine Journal 2023;23:1028-1036.
194. Modarresi S, MacDermid JC, Suh N, et al. How is the probability of reporting various levels of pain 12 months after noncatastrophic injuries associated with the level of peritraumatic distress? Clinical Orthopaedics and Related Research® 2022;480:226-234.
195. Dufton. Prognostic Factors Associated with Minimal Improvement in WAD. Spine 2006.
196. Rosenfeld M, Gunnarsson R, Borenstein P. Early intervention in whiplash-associated disorders: a comparison of two treatment protocols. Spine 2000;25:1782-1787.
197. Yadla S, Ratliff JK, Harrop JS. Whiplash: diagnosis, treatment, and associated injuries. Current Reviews in Musculoskeletal Medicine 2008;1:65-68.
198. Suissa S. Risk factors of poor prognosis after whiplash injury. Pain Research and Management 2003;8:69-75.
199. Stemper BD, Corner BD. Whiplash-associated disorders: occupant kinematics and neck morphology. Journal of Orthopaedic & Sports Physical Therapy 2016;46:834-844.
200. Sidhu KS, Fischgrund JS. Cervical whiplash injuries. In: Fractures of the Cervical, Thoracic, and Lumbar Spine. CRC Press, 2002;273-280.
201. Harinathan B, Jebaseelan D, Yoganandan N, et al. Effect of cervical stenosis and rate of impact on risk of spinal cord injury during whiplash injury. Spine 2023;48:1208-1215.
202. Malik K, Eseonu KC, Pang D, et al. Is preexisting cervical degeneration a risk factor for poor prognosis in whiplash-associated disorder? International Journal of Spine Surgery 2021;15:710-717.
203. Elliott JM, Parrish TB, Walton DM, et al. Does overall cervical spine pathology relate to the clinical heterogeneity of chronic whiplash? The American Journal of Emergency Medicine 2020;38:869-873.
204. Anarte-Lazo E, Falla D, Rodriguez-Blanco C, et al. Higher neck pain intensity and pain catastrophizing soon after a whiplash injury partially explain the presence of persistent headache: a prospective study. The Clinical Journal of Pain 2024;40:349-355.
205. Rydman E, Kasina P, Ponzer S, et al. Association between cervical degeneration and self-perceived nonrecovery after whiplash injury. The Spine Journal 2019;19:1986-1994.
206. John JD, Saravana Kumar G, Yoganandan N. Rear-impact neck whiplash: role of head inertial properties and spine morphological variations on segmental rotations. Journal of Biomechanical Engineering 2019;141:111008.

207. Falla D, Peolsson A, Peterson G, et al. Perceived pain extent is associated with disability, depression and self-efficacy in individuals with whiplash-associated disorders. European Journal of Pain 2016;20:1490-1501.
208. Côté P, Soklaridis S. Does early management of whiplash-associated disorders assist or impede recovery? Spine 2011;36:S275-S279.
209. Rebbeck T, Bandong AN, Leaver A, et al. Implementation of a risk-stratified, guideline-based clinical pathway of care to improve health outcomes following whiplash injury (Whiplash ImPaCT): a multicentre, randomized, controlled trial. Pain 2023;164:2216-2227.
210. Jull G, Kenardy J, Hendrikz J, et al. Management of acute whiplash: a randomized controlled trial of multidisciplinary stratified treatments. Pain 2013;154:1798-1806.
211. Nikles J, Keijzers G, Mitchell G, et al. Pregabalin vs placebo to prevent chronic pain after whiplash injury in at-risk individuals: results of a feasibility study for a large randomized controlled trial. Pain 2022;163:e274-e284.
212. Yang S, Boudier-Revéret M, Hsiao M-Y, et al. At least 5-year outcomes of whiplash-induced chronic neck pain following response to intra-articular facet joint corticosteroid injection. Journal of Pain Research 2022;15:2133-2138.
213. Griffin AR, Sterling M, Ritchie C, et al. Do expectations of recovery improve risk assessment for people with whiplash-associated disorders? Secondary analysis of a prospective cohort study. BMC Musculoskeletal Disorders 2022;23:395.
214. Shipton E, Tait B. Flagging the pain: preventing the burden of chronic pain by identifying and treating risk factors in acute pain. European Journal of Anaesthesiology 2005;22:405-412.
215. Ahmed A. Patient-centred approach to acute post-operative pain management. Sri Lankan Journal of Anaesthesiology 2012;19.
216. Slater R, Eccleston C, Williams A, et al. Reframing pain: the power of individual and societal factors to enhance pain treatment. Pain Reports 2024;9:e1161.
217. Cook CE, Showalter C, Kabbaz V, et al. Can a within/between-session change in pain during reassessment predict outcome using a manual therapy intervention in patients with mechanical low back pain? Manual Therapy 2012;17:325-329.
218. Cook C, Lawrence J, Michalak K, et al. Is there preliminary value to a within-and/or between-session change for determining short-term outcomes of manual therapy on mechanical neck pain? Journal of Manual & Manipulative Therapy 2014;22:173-180.
219. Kasch H, Kongsted A, Qerama E, et al. A new stratified risk assessment tool for whiplash injuries developed from a prospective observational study. BMJ open 2013;3:e002050.
220. Côté P, Hogg-Johnson S, Cassidy JD, et al. Initial patterns of clinical care and recovery from whiplash injuries: a population-based cohort study. Archives of internal medicine 2005;165:2257-2263.
221. Shearer HM, Carroll LJ, Côté P, et al. The course and factors associated with recovery of whiplash-associated disorders: an updated systematic review by the Ontario protocol for traffic injury management (OPTIMa) collaboration. European Journal of Physiotherapy 2021;23:279-294.
222. Snell DL, Martin R, Macleod A, et al. Untangling chronic pain and post-concussion symptoms: the significance of depression. Brain injury 2018;32:583-592.
223. Doroszkiewicz C, Gold D, Green R, et al. Anxiety, depression, and quality of life: a long-term follow-up study of patients with persisting concussion symptoms. Journal of neurotrauma 2021;38:493-505.
224. Mallen CD, Peat G, Thomas E, et al. Prognostic factors for musculoskeletal pain in primary care: a systematic review. British Journal of General Practice 2007;57:655-661.
225. Orenius T, Koskela T, Koho P, et al. Anxiety and depression are independent predictors of quality of life of patients with chronic musculoskeletal pain. Journal of health psychology 2013;18:167-175.
226. Söderlund A, Nordgren L, Sterling M, et al. Exploring patients' experiences of the whiplash injury-recovery process–a meta-synthesis. Journal of pain research 2018:1263-1271.
227. Kulin J, Reaston M. Musculoskeletal disorders early diagnosis: A retrospective study in the occupational medicine setting. Journal of occupational Medicine and Toxicology 2011;6:1-6.
228. Décary S, Longtin C, Naye F, et al. Driving the musculoskeletal diagnosis train on the high-value track. Journal of Orthopaedic & Sports Physical Therapy 2020;50:118-120.
229. Lin I, Wiles L, Waller R, et al. What does best practice care for musculoskeletal pain look like? Eleven consistent recommendations from high-quality clinical practice guidelines: systematic review. British journal of sports medicine 2020;54:79-86.
230. Chiarotto A, Fortunato S, Falla D. Predictors of outcome following a short multimodal rehabilitation program for patients with whiplash associated disorders. European journal of physical and rehabilitation medicine 2014;51:133-141.
231. Bergholm U, Johansson BH, Johansson H. New diagnostic tools can contribute to better treatment of patients with chronic whiplash disorders. Journal of Whiplash & Related Disorders 2004;3:5-19.
232. Yang S, Chang MC. The effectiveness of corticosteroid injection into cervical facet joint for managing whiplash-related neck pain. Annals of Palliative Medicine 2022;11:2569-2573.