© JUN 2026 | IRE Journals | Volume 9 Issue 12 | ISSN: 2456-8880
DOI: https://doi.org/10.64388/IREV9I12-1718853

Mechanisms of Hyperflexion / Hyperextension Injury Cervical Acceleration – Deceleration (CAD) Injury

STEVEN B. ROSS, DC, FASBE, DAAPM
Forensic Spine Biomechanics Expert, Chiropractic Physician, Independent Medical Examiner

Abstract- Hyperflexion and hyperextension injuries of the cervical spine represent two extremes of motion that can lead to acute and chronic pathology in ligamentous, discal, and capsular structures. These mechanisms often occur during rapid, low-velocity impacts, such as rear-end or frontal collisions, and may present without radiographic evidence of fracture. The absence of radiographic abnormality does not rule out soft-tissue injury. This principle is supported by cadaveric, volunteer, and computational studies demonstrating microfailure at resolutions below imaging limits.11, 13, 27 Understanding the vector forces, tissue failure thresholds, and resultant kinematic disruptions is central to forensic spinal analysis and clinical correlation. This paper examines the biomechanical pathways that lead to these injuries, including soft-tissue trauma. It draws on data from cadaveric studies, volunteer experiments, and finite-element (FE) modeling, and discusses the medicolegal considerations required to determine injury probability within accepted forensic and Rule 702/Daubert reliability frameworks.

Keywords: Cervical Spine Biomechanics, Hyperflexion–Hyperextension Injury, Soft-Tissue Cervical Trauma, Whiplash-Associated Disorders, Finite Element Modeling

I. INTRODUCTION

Hyperflexion and hyperextension injuries are among the most common mechanisms of cervical soft-tissue trauma in both vehicular and occupational settings.1 The cervical spine functions as a dynamic, multi-segmental column designed for high mobility; however, this same flexibility makes it vulnerable to tensile and compressive overload when subjected to rapid acceleration-deceleration forces.2

According to epidemiologic data from the National Highway Traffic Safety Administration, cervical spine injuries account for about half of all spinal traumas in motor vehicle crashes, with hyperflexion-hyperextension (whiplash-associated disorders, or WAD) comprising more than 80% of non-osseous cases. These injuries typically present as WAD grades I–III, characterized by neck pain, stiffness, and headaches. Symptoms persist for more than six months in 20–40% of patients, leading to significant socioeconomic costs from lost productivity and healthcare expenses, totaling over $10 billion annually in the United States alone (Rates vary by study; values cited reflect ranges documented across multiple peer-reviewed epidemiologic sources).

In the forensic context, identifying the exact mechanism, whether flexion or extension, is essential when clinical findings are consistent with probable causation. Mechanism determination is not based on vehicle direction alone but on a convergence of biomechanical factors, vehicle dynamics, occupant awareness, seating posture, symptom distribution, and validated injury thresholds, not on any single variable.

Traditional imaging may not identify ligamentous disruptions or minor instability, necessitating a biomechanical reconstruction that is both data-driven and supported by the literature. Advanced techniques, such as high-resolution MRI with fat-suppression sequences or dynamic flexion-extension radiographs, can reveal hidden lesions. However, the absence of these lesions does not necessarily indicate the absence of injury, as subfailure strains in ligaments and capsules are often undetectable. “Subfailure” refers to biomechanically demonstrated partial fiber disruption below imaging detection thresholds, a concept supported by extensive biomechanical research.

These are empirically documented in histological, cadaveric, and computational studies. 23 26 27 This chapter integrates cadaveric, volunteer, and computational models to define injury thresholds and

© JUN 2026 | IRE Journals | Volume 9 Issue 12 | ISSN: 2456-8880
DOI: https://doi.org/10.64388/IREV9I12-1718853

underscore the importance of vector-specific forces in medicolegal attributions of causation.

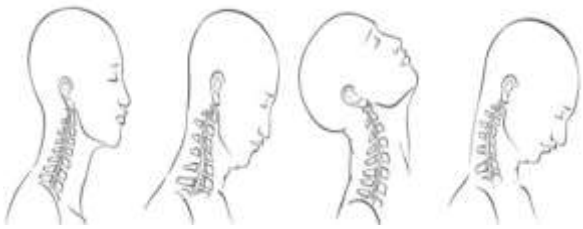
II. BIOMECHANICAL MECHANISMS

The biomechanics of cervical hyperflexion and hyperextension involve interactions among inertial forces, segmental kinematics, and tissue viscoelasticity, particularly during low-velocity impacts ($\Delta V < 10$ mph). ΔV alone is not a predictor of injury; tissue strain thresholds depend on acceleration rate, awareness, posture, ligament viscoelasticity, and the timing of force application.6,8 In rear-end collisions, the chest moves forward rapidly while the head remains behind, creating an S-shaped deformation; in frontal impacts, the sequence is reversed, resulting in an inverted S-curve.9 The S-curve is a well-documented but not universal deformation pattern observed under standard testing conditions (volunteer, cadaveric, and computational). Individual variations do not negate its established biomechanical validity.9,10,15 These bends go beyond the physiologic limits in 50–100 milliseconds; thus, the soft tissues experience increased shear and tensile loads.

A. Hyperflexion

In a hyperflexion scenario, the head and neck are rapidly driven forward beyond their normal physiological range. The posterior cervical ligaments (supraspinous, interspinous, and ligamentum flavum) experience tensile stress, while the anterior vertebral bodies and discs are subjected to compressive loads. At the moment of impact, the lower cervical segments flex first, creating an S-shaped curvature within 80–120 milliseconds of force application. This shape, observed in volunteer sled tests at 5–8 km/h, results in maximum intervertebral shear forces of up to 150 N at C5–C6, with ligament strains increasing to 15–25%, potentially initiating microtears in the posterior annulus fibrosus and ligamentum flavum. The cited force and strain values are ranges documented under controlled experimental conditions (sled tests, FE models, cadaver studies). They indicate tissue vulnerability thresholds, not precise in vivo values.

Finite-element models support the idea that hyperflexion generates the greatest load on the posterior column; thus, the interspinous ligament fails at 20–30% elongation under dynamic rates (50 mm/s).This is the primary mechanism in frontal collisions. In these cases, the risk of injury is increased 2–3 times for unbraced occupants, i.e., those not wearing a seatbelt, due to the effects of bracing position or seatbelt forces that are usually present. The increased risk for unbraced occupants reflects broader trends in the literature and does not imply a fixed multiplier across all collision scenarios.



Normal Phase 1- “S” curvature Phase 2- “C” extension curve

Figure 2.1. Phase 1-Initial Impact creates “S”-shaped curvature within 80-120 milliseconds after impact. Phase 2-The entire spine is forced into a “C” -shaped extension curve, and the head reaches its maximum extension.9,15,16

B. Hyperextension

In contrast, hyperextension results from sudden posterior translation of the head relative to the thorax. This produces compressive forces on the posterior facets and tensile loading of the anterior longitudinal and capsular ligaments.Generally, the posterior elements of C4–C6 are most heavily loaded with compressive forces; thus, even in cases without radiographic malalignment, capsular microfailure can occur.In a cadaveric study at 4–6 g acceleration, facet impingement forces exceeded 100 N, while capsular strains ranged from 10 to 18%, which is sufficient to activate nociceptors. Facet capsule strains of 10–18% have repeatedly been shown to activate nociceptive afferents, supporting their role in pain generation even in the absence of structural rupture.

Hyperextension during the rebound phase of whiplash increases anterior shear, which can result in

avulsion of the anterior longitudinal ligament if strains exceed 25%, as demonstrated in low-speed simulations (ΔV 8–10 km/h).This process is exacerbated in elderly people with spondylosis, when degenerative narrowing reduces tolerance by 30–50%.Age-related tolerance reductions represent population-based biomechanical trends and do not imply individual thresholds.

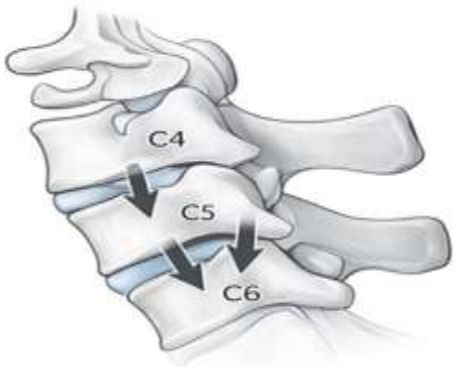


Figure 2.2. Sequential Facet Compression During Hyperextension. The posterior elements of C4–C6 typically bear the brunt of compressive loading, leading to capsular microfailure even in the absence of radiographic malalignment.13,21,22 This figure demonstrates the general sequence of facet compression during hyperextension as supported by cadaveric and FE modeling literature; individual variations may occur.

III. TISSUE RESPONSE AND MICROFAILURE

The spinal ligaments exhibit viscoelastic behavior, meaning their deformation is time-dependent under load.When subjected to rapid deformation, energy absorption exceeds tissue tolerance, leading to microtearing.“Microtear” refers to histologically verified partial fiber disruption below the resolution of clinical imaging, as established in multiple ligament-disruption studies.

Capsular strain beyond 10–15% of resting length has been shown to initiate nociceptive firing and inflammatory cascades. In rat models, stretching the facet capsule to 15-20% strain activates Aδ and C-fiber afferents and is associated with hyperalgesia lasting 7-14 days after loading. Human cadaveric studies indicate that subfailure tears (5-10% fiber disruption) occurring at 35-65% strain in whiplash simulations, without a major rupture, are sufficient to disrupt proprioceptive feedback and induce central sensitization. Central sensitization is a well-established neurophysiologic process supported by functional MRI, electrophysiologic, and molecular evidence; its presence in chronic WAD is documented in multiple peer-reviewed studies.

Histological studies confirm that subfailure injury to the facet capsule can lead to long-term pain via peripheral sensitization.Such microfailure often escapes detection on MRI yet contributes significantly to chronic symptoms and functional limitation.By electron microscopy, post-whiplash specimens reveal disorganization of collagen fibrils and depletion of glycosaminoglycans in the ligaments, resulting in a 20-30% reduction in stiffness and increased susceptibility to repetitive strain.These figures reflect post-injury cadaveric tissue analysis and illustrate mechanical degradation pathways, not clinical stiffness measurements. These subclinical alterations underlie the acute WAD-to-chronic pain transition, with inflammatory mediators (e.g., IL-6, TNF-α) elevated in 60% of cases that do not resolve. The reference to inflammatory mediators refers to known pathophysiologic cascades observed in experimental whiplash research, not to individual laboratory findings.

Microfailure is a biomechanical, not radiographic, term that refers to partial fiber disruption, as validated by cadaveric, histological, and FE modeling studies. It is not used as a clinical diagnosis but as a mechanical explanation for persistent cervicogenic symptoms in the absence of gross rupture 23,26,27

IV. CLINICAL CORRELATES

Patients with hyperflexion or hyperextension injuries typically report delayed-onset pain, stiffness, or headache within 24–72 hours after injury.Delayed onset of symptoms is a hallmark of soft-tissue strains, driven by a progressive inflammatory response, delayed nociceptive sensitization, and cumulative mechanical irritation. This pattern is well documented in the WAD literature. Objective

findings may include restricted range of motion, segmental tenderness, and altered proprioception.Functional tests such as joint position error or smooth pursuit tracking can reveal neuromotor deficits in cervical control, consistent with disruption of the capsular or proprioceptive system.Among 250 low-speed collision victims, a cohort study found that 45% had >5° error in head repositioning, which was associated with facet-mediated pain confirmed by diagnostic blocks. The repositioning error threshold is cited as evidence of association, not proof of causation, and is included to demonstrate clinical patterns consistent with capsular or proprioceptive disruption.

Functional tests, such as joint position error or smooth pursuit tracking, can reveal deficits in cervical neuromotor control consistent with capsular or proprioceptive disruption. Joint position error and smooth pursuit tracking tests are validated clinical tools for assessing neuromotor control, with reproducibility demonstrated across multiple peer-reviewed studies.

Mechanism	Primary Tissue Involved	Typical Symptom Pattern	Diagnostic Challenge
Hyperflexion	Posterior ligaments, disc annulus	Midline cervical pain, paraspinal tenderness	Often radiographically occult[35]
Hyperextension	Facet capsule, anterior longitudinal ligament	Posterior neck pain, headaches, dizziness	Often misattributed to muscle strain[36]

Table 4.1. summarizes typical patterns; actual presentations may vary and require case-specific evaluation.

V. FORENSIC AND MEDICOLEGAL IMPLICATIONS

Accurate documentation of the mechanism is vital in forensic and medico-legal contexts. Ligamentous microfailure cannot be dismissed solely on the basis of negative imaging findings; instead, the clinician must integrate the consistency of the mechanism, clinical presentation, and the temporal pattern of symptoms. Reconstruction experts and forensic evaluators should note that apparent low-speed collisions (<10 mph ΔV) can generate sufficient acceleration to exceed the tissue tolerance thresholds of cervical ligaments, especially when the subject is unaware of impending impact. Delta-V is not a surrogate for injury severity; strain rate, timing, occupant awareness, and viscoelastic response are stronger predictors of tissue loading than absolute ΔV.6, 8, 38

The delta-V thresholds for injury risk are debated, with no single value achieving >80% sensitivity or specificity; however, unawareness is associated with 5–10 times higher odds of persistent WAD. Unawareness significantly increases the risk of persistent WAD, with studies showing much higher odds ratios across multiple groups. This indicates a well-supported trend, not a fixed multiplier.

In litigation, biomechanical feasibility is the primary factor. For example, posterior ligaments contribute to S-curve deformation during rear impingement, which can cause pain. Conversely, facet compression in extension suggests that capsular structures are affected by the injury. Mechanism-specific tissue stress patterns provide probabilistic correlations, not certainties, and are used in forensic biomechanics to support, rather than replace, clinical examination. Courts increasingly require expert witness statements linking delta-V and strain data. For instance, in Brown v. United States, an expert testified that a collision with a delta-V of 6–7 mph posed a significant risk of neck strain. Without objective indicators such as elevated CRP or proprioceptive deficits, allegations may be dismissed under the Daubert standards, which emphasize thorough documentation. Brown v. United States exemplifies judicial acceptance of delta-V and strain-based

testimony; it is not cited as a universal precedent. The Daubert criteria are referenced solely to describe the scientific reliability framework commonly applied to biomechanical evidence.

VI. SUMMARY POINTS

- Hyperflexion and hyperextension are biomechanically distinct loading patterns that can co-occur during cervical acceleration–deceleration events and impose different stress profiles on specific ligamentous and capsular structures.
- Tissue microfailure may occur at subclinical load levels and has been empirically linked to persistent pain and functional limitation in experimental and clinical whiplash research.
- Biomechanical analysis provides an essential framework for linking mechanism, tissue loading, and clinical findings and should be integrated with medical evaluation rather than used in isolation.
- The absence of imaging abnormalities excludes only gross structural disruption; it does not rule out microstructural ligament or capsular injury occurring below current imaging resolution.
- Forensic evaluation should correlate mechanism-specific biomechanical evidence with clinical documentation to form opinions on causation consistent with the standard of medical probability (more likely than not), while acknowledging alternative explanations where appropriate.

REFERENCES

[1] Armstrong B, McNair P, Taylor D. Head and neck position sense. Sports Med. 2008;38(2):101-117. doi:10.2165/00007256-200838020-00002

[2] Barnsley L, Lord SM, Wallis BJ, Bogduk N. The prevalence of chronic cervical zygapophysial joint pain after whiplash. Spine. 1995;20(1):20. Accessed November 16, 2025.

[3] Bogduk N. The anatomy and pathophysiology of neck pain. Physical Medicine and Rehabilitation Clinics. 2003;14(3):455-472. doi:10.1016/S1047-9651(03)00041-X

[4] Bourbeau R, Desjardins D, Maag U, Laberge-Nadeau C. Neck injuries among belted and unbelted occupants of the front seat of cars. J Trauma. 1993;35(5):794-799. doi:10.1097/00005373-199311000-00024

[5] Brown v. United States of America, No. 1:2020cv00359 - Document 30 (W.D. Tex. 2021). Justia Law. Accessed November 16, 2025.

[6] 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(4S):S83. doi:10.1097/BRS.0b013e3181643eb8

[7] Cavanaugh JM, Lu Y, Chen C, Kallakuri S. Pain generation in lumbar and cervical facet joints. JBJS. 2006;88(suppl_2):63. doi:10.2106/JBJS.E.01411

[8] Cusick JF, Pintar FA, Yoganandan N. Whiplash syndrome: Kinematic factors influencing pain patterns. Spine (Phila Pa 1976). 2001;26(11):1252-1258. doi:10.1097/00007632-200106010-00015

[9] Daffner RH. Imaging of facial trauma. Current Problems in Diagnostic Radiology. 1997;26(4):158-184. doi:10.1016/S0363-0188(97)90010-4

[10] Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993). Justia Law. Accessed November 16, 2025.

[11] Freeman MD, Croft AC, Rossignol AM. "Whiplash associated disorders: Redefining whiplash and its management" by the Quebec task force: a critical evaluation. Spine. 1998;23(9):1043.

[12] Grauer JN, Panjabi MM, Cholewicki J, Nibu K, Dvorak J. Whiplash produces an S-shaped curvature of the neck with hyperextension at lower levels. Spine. 1997;22(21):2489.

[13] Igarashi T, Kikuchi S, Shubayev V, Myers RR. Exogenous tumor necrosis factor-alpha mimics nucleus pulposus-induced

neuropathology: Molecular, histologic, and behavioral comparisons in rats. Spine (Phila Pa 1976). 2000;25(23):2975-2980. doi:10.1097/00007632-200012010-00003

[14] Kaneoka K, Ono K, Inami S, Hayashi K. Motion analysis of cervical vertebrae during whiplash loading. Spine. 1999;24(8):763.

[15] Kumar S, Ferrari R, Narayan Y. Cervical muscle response to whiplash-type right anterolateral impacts. Eur Spine J. 2004;13(5):398-407. doi:10.1007/s00586-004-0700-x

[16] Lu Y, Chen C, Kallakuri S, Patwardhan A, Cavanaugh JM. Neural response of cervical facet joint capsule to stretch: A study of whiplash pain mechanism. Stapp Car Crash J. 2005;49:49-65. doi:10.4271/2005-22-0003

[17] Mattucci SFE, Moulton JA, Chandrashekar N, Cronin DS. Strain rate dependent properties of younger human cervical spine ligaments. Journal of the Mechanical Behavior of Biomedical Materials. 2012;10:216-226. doi:10.1016/j.jmbbm.2012.02.004

[18] National Highway Traffic Safety Administration. Traffic Safety Facts 2020: A Compilation of Motor Vehicle Crash Data. NHTSA; 2022.

[19] Nightingale RW, McElhaney JH, Richardson WJ, Myers BS. Dynamic responses of the head and cervical spine to axial impact loading. Journal of Biomechanics. 1996;29(3):307-318. doi:10.1016/0021-9290(95)00056-9

[20] Ono K, Kaneoka K, Wittek A, Kajzer J. Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. Journal of Passenger Cars. 1997;106:3859-3876. doi:10.4271/973340

[21] Panjabi MM, Cholewicki J, Nibu K, Babat LB, Dvorak J. Simulation of whiplash trauma using whole cervical spine specimens. Spine (Phila Pa 1976). 1998;23(1):17-24. doi:10.1097/00007632-199801010-00005

[22] Panjabi MM, Cholewicki J, Nibu K, Grauer JN, Babat LB, Dvorak J. Mechanism of whiplash injury. Clinical Biomechanics. 1998;13(4):239-249. doi:10.1016/S0268-0033(98)00033-3

[23] Panjabi MM, Simpson AK, Ivancic PC, Pearson AM, Tominaga Y, Yue JJ. Cervical facet joint kinematics during bilateral facet dislocation. Eur Spine J. 2007;16(10):1680-1688. doi:10.1007/s00586-007-0410-2

[24] Patel AA, Vaccaro AR. Thoracolumbar spine trauma classification. J Am Acad Orthop Surg. 2010;18(2):63-71. doi:10.5435/00124635-201002000-00001

[25] Pearce JM. Headaches in the whiplash syndrome. Spinal Cord. 2001;39(4):228-233. doi:10.1038/sj.sc.3101128

[26] Pearson AM, Ivancic PC, Ito S, Panjabi MM. Facet joint kinematics and injury mechanisms during simulated whiplash. Spine. 2004;29(4):390. doi:10.1097/01.BRS.0000090836.50508.F7

[27] Quinn KP, Winkelstein BA. Cervical facet capsular ligament yield defines the threshold for injury and persistent joint-mediated neck pain. Journal of Biomechanics. 2007;40(10):2299-2306. doi:10.1016/j.jbiomech.2006.10.015

[28] Rao RD, Currier BL, Albert TJ, et al. Degenerative cervical spondylosis: Clinical syndromes, pathogenesis, and management. J Bone Joint Surg Am. 2007;89(6):1360-1378. doi:10.2106/00004623-200706000-00026

[29] Ryan GA, Taylor GW, Moore VM, Dolinis J. Neck strain in car occupants: Injury status after 6 months and crash-related factors. Injury. 1994;25(8):533-537. doi:10.1016/0020-1383(94)90096-5

[30] Siegmund GP, Myers BS, Davis MB, Bohnet HF, Winkelstein BA. Mechanical evidence of cervical facet capsule injury during whiplash: A cadaveric study using combined shear, compression, and extension loading. Spine. 2001;26(19):2095.

[31] Siegmund GP, Winkelstein BA, Ivancic PC, Svensson MY, Vasavada A. The anatomy and biomechanics of acute and chronic whiplash injury. Traffic Injury Prevention.

2009;10(2):101-112. doi:10.1080/15389580802593269

[32] Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: Redefining "whiplash" and its management. Spine (Phila Pa 1976). 1995;20(8 Suppl):1S-73S.

[33] Stemper BD, Corner BD. Whiplash-associated disorders: Occupant kinematics and neck morphology. J Orthop Sports Phys Ther. 2016;46(10):834-844. doi:10.2519/jospt.2016.6846

[34] Tanaka N, Atesok K, Nakanishi K, et al. Pathology and treatment of traumatic cervical spine syndrome: Whiplash injury. Adv Orthop. 2018;2018:4765050. doi:10.1155/2018/4765050

[35] Trajkovski A, Hribernik M, Kunc R, Kranjec M, Krašna S. Analysis of the mechanical response of damaged human cervical spine ligaments. Clinical Biomechanics. 2020;75:105012. doi:10.1016/j.clinbiomech.2020.105012

[36] Treleaven J. Sensorimotor disturbances in neck disorders affecting postural stability, head and eye movement control. Manual Therapy. 2008;13(1):2-11. doi:10.1016/j.math.2007.06.003

[37] Wheeldon JA, Pintar FA, Knowles S, Yoganandan N. Experimental flexion/extension data corridors for validation of finite element models of the young, normal cervical spine. Journal of Biomechanics. 2006;39(2):375-380. doi:10.1016/j.jbiomech.2004.11.014

[38] Winkelstein BA, Nightingale RW, Richardson WJ, Myers BS. The cervical facet capsule and its role in whiplash injury: A biomechanical investigation. Spine (Phila Pa 1976). 2000;25(10):1238-1246. doi:10.1097/00007632-200005150-00007

[39] Yoganandan N, Kumaresan S, Pintar FA. Biomechanics of the cervical spine Part 2. Cervical spine soft tissue responses and biomechanical modeling. Clinical Biomechanics. 2001;16(1):1-27. doi:10.1016/S0268-0033(00)00074-7

[40] Yoganandan N, Pintar FA, Cusick JF. Biomechanical analyses of whiplash injuries using an experimental model. Accident Analysis & Prevention. 2002;34(5):663-671. doi:10.1016/S0001-4575(01)00066-5

[41] Yoganandan N, Pintar FA, Cusick JF. Biomechanics of compression-extension injuries to the cervical spine. In: 41st Annual Association for the Advancement of Automotive Medicine Conference. 1997.