ScienceDirect

Journal of Forensic and Legal Medicine

Available online 13 August 2026, 103230

In Press, Journal Pre-proof What's this?

Addressing Scientific Limitations of Common Causation Opinions in Spinal and Traumatic Brain Injury Litigation

Oregon K. Hunter Jr. MD [1], Oscar B. Depaz MD [2]

Show more

https://doi.org/10.1016/j.jflm.2026.103230 ↗

ABSTRACT

Objective

To critically examine ten commonly asserted defense medicolegal opinions encountered in crash-related spinal and traumatic brain injury litigation, evaluate their scientific validity against the peer-reviewed literature, and provide an evidence-based framework for countering unsubstantiated forensic opinions.

Design

Narrative evidence-based review informed by systematic analysis of defense medical evaluation reports. The authors, two physiatrists with extensive clinical and forensic experience, reviewed more than 800 comprehensive medical evaluations (CMEs) prepared by defense-retained physicians in spinal and brain injury litigation over three decades (1987–2025). The ten most clinically significant and frequently recurring defense opinions (five pertaining to spinal injury and five to traumatic brain injury) were selected for

analysis. A comprehensive literature search was conducted to identify any literature supporting the defense opinions under review.

Results

No peer-reviewed scientific literature was identified in support of any of the ten defense opinions examined. For spinal injuries, the reviewed literature refutes the assertions that low-velocity motor vehicle crashes (MVCs) cannot cause disc injury; that post-crash disc pathology is invariably pre-existing and degenerative; that spinal injury requires MRI confirmation; that radiculopathy requires visible nerve root compression; and that MVC-related spinal injuries uniformly resolve within 6–12 weeks. For traumatic brain injury (TBI), the literature refutes the assertions that TBI requires a direct blow to the head, loss of consciousness (LOC), or an abnormal Glasgow Coma Scale (GCS) score; that TBI must be diagnosed at the scene or in the emergency department; that TBI diagnosis requires hemorrhage or gross structural abnormality on conventional neuroimaging; that a normal focal neurological examination excludes TBI; and that TBI prognosis is uniformly favorable with no increased risk of dementia.

Conclusions

Ten forensic defense opinions commonly advanced in spinal and brain injury litigation are each contradicted by robust peer-reviewed evidence and lack any identifiable scientific foundation. These unsubstantiated maxims grounded in anecdotal clinical experience rather than the medical literature distort causation analysis and undermine legitimate injury claims. Accurate forensic evaluation of crash-related spinal and brain injuries requires integration of biological plausibility, individual vulnerability, clinical evolution, advanced neuroimaging, and long-term outcome data, consistent with current scientific and diagnostic standards.

Keywords list

Motor vehicle collision; Spinal injury; Intervertebral disc injury; Facet joint injury; Traumatic brain injury; Litigation; Medico-legal opinion; Expert witness testimony

Abbreviations

AANS
: American Association of Neurological Surgeons

AAOS
: American Academy of Orthopedic Surgeons

ACRM
: American Congress of Rehabilitation Medicine

ADL
: activities of daily living

AMA
: American Medical Association

CDC
: Centers for Disease Control and Prevention

CHIEF
: Craig Hospital Inventory of Environmental Factors

CMB
: cerebral microbleed

CME
: compulsory medical evaluation

CT
: computed tomography

DAI
: diffuse axonal injury

DTI
: diffusion tensor imaging

ED
: emergency department

EMS
: emergency medical services

ER
: emergency room

GCS

Glasgow Coma Scale

HIZ
: high-intensity zone

ICD-10
: International Classification of Diseases, 10th Revision

LOC
: loss of consciousness

MRI
: magnetic resonance imaging

MVC
: motor vehicle crash

mTBI
: mild traumatic brain injury

NASS-CDS
: National Automotive Sampling System – Crashworthiness Data System

NEDS
: Nationwide Emergency Department Sample

NHL
: non-hemorrhagic lesion

PDTBI
: psychotic disorder due to traumatic brain injury

PET/CT
: positron emission tomography–computed tomography

SRC
: sports-related concussion

TAI
: traumatic axonal injury

TBI
: traumatic brain injury

WAD
: whiplash-associated disorder

WMH
: white matter hyperintensity

INTRODUCTION

Treating physicians typically employ a common-sense, clinically grounded approach to the causation of spinal and brain injury, which encompasses three steps:

1. **Plausibility:** Is it reasonable, based on clinical experience and medical literature, that the traffic crash could have caused the diagnosed injury in a manner consistent with the patient's reported history?
2. **Temporal Relationship:** Did the onset of symptoms associated with the diagnosed injury occur proximate to the crash?
3. **Alternative Explanations:** Given the patient's pre-crash history, was the development of the same diagnoses at the same point in time likely to occur absent the crash?

In crash injury litigation regarding spinal and brain injuries, there are consistently opposing causation opinions presented by medical or engineering experts who are experienced in countering treating physician opinions of causation.

These commonly asserted medicolegal opinions asserted by the defense are presented as scientifically established but are typically based on "my 30 years of experience," with misinterpretations, ignorance, or selective readings of the relevant medical literature.

It is the position of the authors that misuse and misinterpretation of scientific literature by defense experts have resulted in the widespread adoption of opinions that lack a sound scientific foundation. These opinions have become prevalent maxims (or commonly cited maxims) in litigation involving spinal and brain injuries. Such unscientific medical opinions undermine claims analysis and contradict established medical evidence.

This critical narrative review aims to:

1. Document the authors' experience with defense medical evaluations in spinal and brain injury litigation.

2. Identify and describe the most common causation maxims encountered.

3. Provide an evidence-based review of scientific literature that refutes these maxims.

4. Restore scientific integrity to forensic evaluation processes by clarifying misconceptions that are pervasive in forensic medical practice.

METHODS

The authors' combined forensic and clinical practices ~~entail the systematic~~ involved review of defense medical reports and deposition testimony in spinal and brain injury litigation cases, spanning more than 800 evaluations over a period of three decades. ~~Most reviews~~ The majority were completed from 2015 to the present date. Each ~~case~~ reviewed case contained at least one of the maxims analyzed here, and most contained several ~~had multiple maxims~~. The maxims~~chosen~~ selected for review in this paper were those appearing most frequently across reports and depositions and judged by the authors to be the most significant for inclusion in this narrative, evidence-based review. ~~mostlikely to be found in the reviewed reports and depositions.~~

A ~~review~~ tabulation of the maxims found in ~~of the~~ 26 consecutive spine and brain injury cases reviewed from 10.1.25 to 6.1.26 is presented in Table 1, showing the frequency of the opinions by specialty in these cases reviewed.

Though dozens of unscientific opinions have been noted in the forensic reports, the clinical importance and frequent reoccurrence of these ten opinions directed the choice of which opinions to review. Five of the most common spine and five of the most common brain injury opinions offered by the defense were chosen for analysis.

The forensic opinions offered by the defense are typically based on the foundation of "30 years of experience" rather than being supported by citations from scientific literature.

The references used to support the scientific research that counters the defense forensic opinions have been accumulated over decades utilizing typical search engine resources including: Index Medicus, MEDLARS, MEDLINE, PubMed, Google Scholar, and utilizing AI deep research tools to search ~~looking at~~ PubMed-indexed articles, journal publisher sites, trial registries, and guideline documents.

Defense forensic opinions are characteristically absolute rather than nuanced and are offered without citation of supporting medical literature. A representative medicolegal

opinion asserts that a diagnosis of TBI requires documented loss of consciousness (LOC); no peer-reviewed literature is offered to support this position.

To evaluate whether there is any scientific basis for such medicolegal opinions offered by the defense, a systematic literature search was conducted for each of the ten commonly asserted medicolegal opinions examined in this paper. Using AI-assisted deep research platforms querying more than 220 million indexed articles across Semantic Scholar, PubMed, and OpenAlex, searches were structured to identify any publications that would affirmatively support the defense position. For example, searching for any literature establishing that a TBI diagnosis cannot be made in the absence of documented LOC, no citations supporting this opinion were identified. Example research prompt: “provide any medical literature supporting the assertion that a TBI diagnosis requires a documented loss of consciousness.” The same search methodology was applied to each of the remaining nine medicolegal opinions that were the subject of this paper. Identical negative results were obtained.

This paper will focus on the medical literature that counters the unscientific opinions offered by the defense. This medical literature will be reviewed, summarized and cited. If ~~it~~ such literature exists, literature supporting the defense opinions will be cited.

SECTION 1 - SPINAL INJURIES

Background

The relationship between traffic crashes and spinal disc injuries has become increasingly controversial in medical and engineering literature. This controversy is driven largely by economic factors related to litigation of spinal disc injury claims attributed to traffic crashes, particularly those occurring at low speeds or low damage claims.

Freeman et al., in a seminal 1999 Spine article, "A Review and Methodologic Critique of the Literature Refuting Whiplash Syndrome," performed a detailed critique of studies that attempted to deny the validity of whiplash injuries[1]. They concluded that all such refutational articles contained significant methodological flaws, including inadequate sample sizes, nonrepresentative crash tests, biased sampling, and inappropriate study designs, and that there was no scientific basis for claims that rear-impact rear impact forces/motions experienced during the collision~~s~~ event without visible vehicle damage do not cause injury or that whiplash trauma is biomechanically comparable to activities of daily living (ADLs).

Two opposing perspectives have emerged:

Clinical/medical perspective: Clinicians and biomedical experts who support injury claims recognize that virtually any severity of traffic crashes can result in symptomatic spinal disc injuries.

Defense perspective

- Biomechanical and engineering experts often use force or acceleration calculations, epidemiologic databases, cadaver, crash dummy testing, and comparisons to ADLs to claim that certain collisions cannot cause disc injuries and do not consider subfailure.
- Orthopedic surgeons retained for the defense frequently adopt these engineering-based interpretations and offer unscientific opinions in forensic evaluations.
- Radiologists retained by the defense may ignore established principles of medical causation and the broader clinical context when formulating opinions.

Faulty or incomplete science regarding disc injury from trauma is a major driver of these opinions, and several related maxims amplify the problem. The present paper addresses these pervasive maxims in forensic medical evaluations, particularly those opinions that rely on flawed engineering literature to dispute legitimate injury claims.

Spine injury analysis is summarized in Table 2.

Opinion 1: Traumatic events such as a low-velocity MVC cannot cause disc injury What research supports this opinion? None

Causation analysis is critical

When defense experts argue that a motor vehicle crash (MVC) did not cause a spinal injury, they frequently omit any formal causation analysis. Typical opinions assert that a crash cannot cause spinal injury of any consequence beyond a simple sprain/strain, disregarding the scientific evidence on spinal injury mechanisms and individual susceptibility.

The claim that crash forces are too low to cause tissue damage ignores the fact that each injured person has unique vulnerability, influenced by age, pre-existing degeneration, and

overall health. Defense arguments equating low-velocity crashes to ADLs and concluding that such crashes therefore cannot injure the spine are not supported by the literature.

Though MRI imaging studies are often considered to be the gold standard when assessing spine trauma, Zhuge[2]1 found that using MRI to evaluate pre-surgery to post-surgery patients lacked sensitivity and specificity. The accuracy for detecting intervertebral disc injury had a sensitivity at 62.5% and the specificity at 88.2%.

Causation can be established through systematic analysis that includes biological plausibility, temporality, and the absence of more probable alternative explanations. Freeman describes a 3-step causation framework, requiring evaluation of plausibility, temporality, and competing explanations; this process conforms to medical standards of practice, and its omission is a central flaw in typical defense opinions. In addition, a comprehensive causation analysis should include other factors that support medical causation. These include: symptom evolution, prior medical history, physical examination findings, diagnostic studies to include imaging, electrodiagnostic, and vestibular evaluation. Individual vulnerabilities and comorbidities can contribute to the causation due to a specific injury causing event. Perhaps the most important factor to consider in causation analysis is the functional impact. A comprehensive analysis of the impact of the injury on the individual's ability to perform work activities, activities of daily living, and overall cognitive and emotional function changed by the injury.

Rondinelli, in the AMA Guides to the Evaluation of Permanent Impairment, 6th Edition, outlines a similar process for determining causation. The method requires: (1) a clearly defined causal event, (2) objective evidence of impairment, (3) evidence that the event can cause the condition, and (4) a determination that the event did cause or materially contributed to the condition[3].

How much trauma is needed to injure a disc?

Beginning in 2013, several engineering publications used the National Highway Traffic Safety Administration's NASS-CDS crash injury database to claim that disc injuries either do not result from crashes or are exceedingly rare[4]. Freeman and Leith (2020) identified critical limitations of these analyses, including the fact that NASS-CDS only captures diagnoses from the first few days after a crash, primarily from emergency department visits, whereas symptomatic disc injuries are most often identified later by MRI after conservative care fails[5]. Also excluded were those injured patients who were not transported to or seen in Emergency Rooms for evaluation.

Freeman and Leith reported an average of 2,791 cervical disc injuries per year attributed to crashes in a national emergency department database (NEDS), compared with only 332 such injuries per year in NASS-CDS. They concluded that NASS-CDS likely surveys less than

1% of all cervical crash-related disc injuries—estimated at 33,000–34,000 annually in the U.S.—and that it has no utility for surveillance of crash-related disc injuries in any spinal region.

Three years later, Kent et al., a group of nonmedical engineers, published a new analysis of national crash and emergency department data and reported only 26 crash-related cervical disc injuries in NEDS for 2018[6]. Using a denominator of 20,850,013 crash-exposed individuals, they calculated a cervical disc injury risk of 1 per 800,000 crash exposures, a figure far lower than that found by Freeman and Leith.

The Kent analysis suffers from fundamental methodological problems, including misuse and generalization of ICD-10 codes. Kent et al. included only the code S13.0XXA ("traumatic disc injury" with dislocation), thereby excluding the vast majority of cervical disc diagnoses. They omitted 7,595 other cervical disc cases coded as M50.xx, as well as codes for cervical disorders with myelopathy or radiculopathy, disc displacement, and disc disorders associated with neck pain, which they labelled "degenerative" and pre-existing despite lacking medical training.

This exclusion removed more than 99% of crash-related cervical disc injuries from their analysis.

Kent et al. also perpetuated the misleading comparison between ADLs and minimal-damage crashes. Nolet et al. analysed 408 crash tests and found that even minimal-damage rear impacts (3–11 km/h deltaV) produced peak head accelerations averaging 5.9 g and up to 17 g, several times greater than peak ADL accelerations, which are typically below 4 g[7] ~~[7]~~. They concluded that real-world minimal-damage rear-impact crashes carry an injury risk at least 2,000 times greater than any ADL, and that using occupant acceleration as a simple proxy for injury risk is scientifically invalid.

Freeman has further explained that collision force alone is not useful in determining the probability of disc injury because the tissue-level injury threshold has never been established. Thus, causation analysis does not require a precise correlation between collision force and disc injury risk[8].

Trauma as a recognized cause of disc injury, Aly[9] found that the presence of a spinal fracture was not correlated to disc herniation unless there was a significantly increased kyphotic

angle.

Exposure to significant trauma, including sports such as soccer, rugby, and equestrian activities, is cited by Kelly as a cause of disc disease[10]. Dydyk identifies trauma as the second most common cause of disc herniation[11]. Furman describes disc protrusion following a single traumatic event such as a whiplash injury[12].

Hashish reported that crash-related forces influence whether the cervical or lumbar region is more likely to be injured, with cervical discs more often affected in side-swipe (T-bone) collisions and lumbar discs more often injured in head-on collisions[13]. Hoffberg, however, found that the extent of property damage was a poor predictor of the type or extent of disc disruption[14].

Pettersson suggested that intervertebral discs are potential pain generators in both acute and chronic whiplash syndromes[15]. Li et al. demonstrated that whiplash trauma can injure spinal ligaments, facets, and discs in experimental models, MRI, surgical cases, and autopsy studies, with deeper structural damage occurring through compression, stretch, and shear forces rather than mere superficial muscle strain[16].

Moheimani notes that MVCs can cause back pathology, including disc herniation, and that a disc need not be degenerative before it herniates; whiplash symptoms can occur at crash speeds as low as 2.5 mph, and the risk of disc herniation is increased by automobile accidents[17]. Pencle identifies cervical disc injuries as a potential consequence of MVC[18]. The Mayo Clinic lists trauma as a cause of disc herniation[19].

Aging discs and vulnerability to traumatic injury

Disc injuries can result from both major and minor trauma, and degenerative change increases susceptibility to herniation. Adams supports the conclusion that disc injury can follow relatively minor trauma[20]. The University of Rochester notes that injury may cause an abnormal disc to herniate or worsen an existing herniation[21].

The American Association of Neurological Surgeons (AANS) states that herniated discs can be caused by trauma and that age-related degeneration makes discs more vulnerable to damage from relatively minor strains or twisting movements—forces easily generated in MVCs[22]. The American Academy of Orthopedic Surgeons (AAOS) and Paliaoutas likewise conclude that aging predisposes discs to injury[23],[24].

Opinion 1 — Summary

Evidence from clinical, epidemiologic, biomechanical, and guideline-based sources clearly supports the conclusion that low-velocity MVCs can cause disc injuries, particularly in individuals with pre-existing degeneration that increases vulnerability. Attempts to dismiss these injuries by relying on flawed databases, misused diagnostic codes, or simplistic ADL comparisons are methodologically unsound and conflict with mainstream medical literature. Crash-related forces, even in low-speed, minimal-damage events, substantially exceed those encountered in routine ADLs and are associated with markedly higher injury risk. Comparison of the forces generated in crashes to those created by daily activities fails to capture the complex differential movement of the spinal segments. Therefore, comparing injuries due to MVCs to those caused by benign daily activities to deny causation is a scientifically unsupported strategy that ignores well-established biomechanical data.

Opinion 2: After an MVC, disc and facet pathology is always pre-existing and degenerative, never traumatic, and cannot be worsened by trauma

What research supports this opinion? None.

It is expected that spinal imaging will show increasing degenerative changes with age; however, the presence of degeneration does not prove that a disc lesion is solely age-related or clinically insignificant. Degenerative findings may remain asymptomatic for years and become clinically significant only after a traumatic event such as an MVC.

The defense evaluation provides the common medicolegal argument that, because degenerative findings are present on imaging, the patient's post-traumatic symptoms or impairment must be unrelated to the crash. This is an incomplete analysis.

Evaluating physicians have a duty to distinguish between pre-existing imaging findings, pre-existing symptoms, pre-existing impairment, and traumatic aggravation of a vulnerable but previously asymptomatic or minimally symptomatic condition. An individual's pre-injury functioning versus post-injury functioning must also be taken into account when analyzing the significance of degenerative changes of the spine which are brought to light after injury in an MVC.

In summary, the presence of pre-existing degenerative spinal findings does not, by itself, exclude traumatic causation, traumatic exacerbation, or traumatic aggravation. A more scientifically sound analysis requires distinguishing pre-existing imaging abnormalities from pre-existing symptoms, impairment, and functional limitation, while also considering whether the traumatic event materially altered the patient's clinical course and need for treatment and for loss of function (e.g., ability to work).

Boden demonstrated that cervical disc degeneration increases with age but disc bulging or herniation is relatively uncommon, especially in younger individuals[25]. In those subjects under the age of 40 only 10% were found to have disc herniation. In subjects over 40, herniation was found in 5% and bulges were found in 3%. For the lumbar spine, Boden reported disc herniation in 21% of asymptomatic individuals under 40 and 36% in those over 60, again indicating that not all older adults have herniated discs[26].

Brinjikji, in a large review of 3,110 asymptomatic individuals, found that lumbar disc bulges and protrusions do increase with age but do not approach universality: bulges were present in 30% of 20-year-olds and 84% of 80-year-olds, and protrusions increased from 29% at age 20 to 43% by age 30[27]. These data show that while degenerative findings are common, they are not inevitable for every disc at every level and do not exclude the superimposition of traumatic pathology.

Colwell reported that geriatric patients are more likely to sustain injuries of all types due to age-related anatomic and physiologic changes, and that frailty and overall health status are more important determinants of outcome than chronological age alone[28]. Felson found that vulnerable joints, including spinal facet joints, in older athletes are more likely to develop osteoarthritis and that this process accelerates following injury[29].

Freeman explains that degenerative discs are more prone to injury from relatively minor trauma, such as low-speed traffic crashes[30]. Kos similarly notes that early degenerative changes weaken the annulus fibrosus, permitting the nucleus pulposus to herniate more readily[31]. The Mayo Clinic emphasizes that age-related disc desiccation and loss of flexibility make discs more susceptible to tearing, even with minor strain or twisting movements[32]. Rondinelli, in the AMA Guides, clearly states that pre-existing degenerative spinal conditions can be permanently aggravated by trauma[33].

Siccoli offers a somewhat different perspective, arguing that disc herniation in younger patients can occur in the absence of pre-existing degeneration because intact discs can generate sufficient internal pressure to herniate[34]. Importantly, this view does not refute the possibility of traumatic aggravation of degenerative discs; rather, it underscores that traumatic herniation is possible in both degenerated and non-degenerated discs.

Facet joints (zygapophyseal joints) of the back part of the spinal column are critical for spinal movement and stability and are prone to injury from whiplash and other trauma. Studies confirm that facet joint pain is a common outcome of whiplash-associated disorder (WAD) and that facet joint injuries, even minor ones, can lead to post-traumatic osteoarthritis, causing pain and loss of function.

A paper by Yoganandan discusses that when pathological findings of whiplash injuries are not found on initial imaging studies, clinicians then frequently dismiss the validity of a patient's claims. But pathologic changes have been found on cryomicrotomy evaluations of injured facet joints, confirming structural abnormalities due to facet joint injury[35].

Aarnio reported positive imaging study findings showing injury to the cervical spine facet joints; these objective studies use PET/CT imaging and detect organic lesions that correspond to whiplash injuries causing persistent pain and disability[36].

The zygapophysial joint (facet joint) was considered to be the source of neck pain in WAD. Curatolo predicted by biomechanical and animal studies, with diagnostic and therapeutic interventions[37].

Petersohn finds that cervical facet joints are frequently the cause of neck pain post-trauma in up to 50–60% of cases[38].

Not all crash victims will develop facet joint problems; Freeman argues that individual susceptibility (an unpredictable variable) to injury is the reason that joint injury due to trauma cannot be excluded from causation[39].

Injury of the facet capsule during whiplash activating nociceptors of the joint, identified by histologic and anatomic studies, is correlated with pain syndromes that develop due to WAD[40]. Neuro-inflammatory cascades in the spinal cord following facet joint loading show the relationship of injury to the development of pain from whiplash injury.

Siegmund describes the mechanical basis for injury during whiplash events which includes shearing, bending, and compression of the facet joint capsular ligaments[41],[42]. The facet joint capsule is injured during whiplash events and can localize chronic neck pain from whiplash to the cervical facet joint, with injury to the facet capsular ligament shown in human and cadaveric subjects, and with individual variability, low speed rear-end collisions could injure the facet joint capsular ligaments[43].

Tidy describes whiplash injury occurring in low speed rear-end collisions, most commonly at speeds less than 14 mph[44].

Injury to the articular cartilage of joints can result in post-traumatic degenerative changes and osteoarthritis[45]. Facet joints are lined with cartilage and, like other limb joints, are susceptible to this process.

Per Punzi, inflammatory chemicals released after injury predispose joints to the development of post-traumatic arthritis[46].

The Mayo Clinic reports that joint injury can increase the risk of osteoarthritis even in old, seemingly healed injuries[47].

Opinion 2 — Summary

Degenerative disc changes are common with aging but are not universal and do not preclude traumatic injury or aggravation. The literature supports that both previously normal and pre-existing degenerated discs can herniate or worsen following MVCs, and authoritative sources recognize that pre-existing spinal degeneration can be permanently aggravated by trauma. Facet joint pathology is not exclusively degenerative; there is substantial evidence that whiplash and other crash-related trauma can injure facet capsules and articular cartilage, leading to chronic pain and post-traumatic osteoarthritis. Denying traumatic facet pathology in MVC victims overlooks extensive experimental, imaging, and clinical data.

Opinion 3: Spine imaging must show acute changes to prove injury, and there cannot be a spine injury without MRI abnormalities

What research supports this opinion? None.

Determining the precise timing of disc injuries on MRI is inherently difficult, and imaging alone cannot reliably date a herniation. Radiology reports routinely recommend correlation with the clinical history and examination, and the treating clinician—who knows the patient's pre-crash status, symptom onset, and functional impact—is best positioned to assess causation.

Divi concluded that the timing of disc herniation cannot be accurately predicted based solely on MRI signal characteristics[48]. In the Nomenclature 2.0 paper, Fardon emphasized that assigning a specific date of origin to a disc herniation requires clinical correlation and, ideally, comparison with prior imaging[49]. Herzog similarly advised that prior studies are essential for dating a disc lesion and noted that a disc herniation can represent an acute lesion superimposed on chronic degenerative change[50].

Jensen found that while disc bulges and protrusions can be incidental, asymptomatic findings, disc extrusions are much less likely to be coincidental, underscoring the importance of clinical context[51]. Munter cautioned against using disc hyperintensity and high-intensity zones (HIZ) as sole indicators of acuity, noting that slice thickness and other technical factors can lead to missed or misleading findings; serial imaging may be required to document lesion evolution[52].

Ross pointed out that an apparent disc margin extension in the cervical spine may represent bulge, herniation, osteophyte, cartilage, or calcification, making it difficult to determine the exact tissue involved without careful interpretation[53].

Haris evaluated MRI sensitivity for intervertebral disc and ligamentous injuries and found that MRI detected only 75% of surgically confirmed disc injuries[54]. Detection rates were even lower for ligamentum flavum (62%) and interspinous ligament injuries (63%), highlighting that a "normal" MRI does not exclude clinically significant trauma. Acosta reported that soft-tissue abnormalities such as facet joint effusion are frequently missed in radiology reports, with sensitivities as low as 6–22%[55]. Although Konieczny proposed a classification system using signal intensity ratios to estimate the age of lumbar disc herniations, this system has not been adopted in routine clinical practice[56].

Annular fissures have been suggested as markers of disc injury age, but Rafiee emphasized that the age of an annular fissure cannot be inferred from a single study; only the appearance of a new fissure on a current study, absent on a prior study, supports recent onset[57].

ZX Wang reported that HIZs are of uncertain clinical significance, with wide variability in symptoms; HIZs were present in 58% of symptomatic low back pain patients but also in nearly half of asymptomatic subjects[58]. H. Wang further showed that an HIZ is not a reliable indicator of the symptomatic disc and argued that discography remains necessary for definitive diagnosis of discogenic pain[59].

Haris's work also demonstrated that a negative MRI does not rule out trauma-related disc or ligament injury: disc abnormalities were missed in 25% of cases, and ligamentous injuries were missed in about one-third[60].

Moheimani described biochemical mediators of discogenic pain, including nitric oxide, prostaglandin E2, and interleukin-6, which are not visible on imaging; elevated prostaglandin E2 levels correlate with a positive straight leg raise test[61].

Sehgal showed that internal disc disruption may present with normal CT and myelography but be identified as pain-generating on discography[62]. Patients with internal disc disruption often have diffuse low back pain, limited lumbar motion, decreased sitting tolerance, and non-specific or absent neurologic signs, leading to mislabeling as functional or malingering. Discography can confirm discogenic pain in 30–50% of patients with chronic low back pain.

Theodore described how nerve fibers in the annulus fibrosus and nucleus pulposus become sensitized by inflammatory cytokines in degenerative discs, producing discogenic

pain even in the absence of gross structural compression on imaging[63].

Opinion 3 — Summary

MRI and other imaging modalities have important limitations in detecting and dating spinal injuries, and a normal or nonspecific study does not exclude clinically significant trauma. Discogenic pain and radiculopathy can arise from biochemical and microstructural pathology that is invisible or under-recognized on routine imaging, making clinical correlation essential for accurate causation analysis.

Opinion 4: Radiculopathy occurs only when a disc protrusion directly compresses a nerve root

What research supports this opinion? None.

For decades, the prevailing paradigm held that disc herniation causes radiculopathy solely via mechanical compression of the nerve root, and that without visible compression there can be no true radicular pain. Clinical experience and experimental data have long challenged this view, demonstrating severe pain in some patients with minimal or no radiographic compression and, conversely, asymptomatic patients with substantial herniations.

Inflammatory mediators released from injured discs can induce nerve root irritation and radiculopathy in the absence of significant mechanical deformation. Carette described multiple inflammatory chemicals, including prostaglandins, released by herniated disc material, providing a rationale for steroid-based anti-inflammatory therapy[64].

Cosamalón-Gan emphasized that herniated disc tissue is biologically active rather than inert, expressing a range of inflammatory mediators demonstrated by immunologic and molecular methods[65].

Furman noted that cervical disc herniation can cause radiculopathy either by direct nerve root compression or via inflammatory chemicals leaking from the disc[66]. Hsu reported that rupture of the annulus fibrosus with extrusion of disc material can cause nerve root injury and abnormal EMG findings without visible mechanical compression, and that up to 50% of patients with radiculopathy identify an inciting event such as an MVC[67].

Lin showed that annular tears release inflammatory mediators that provoke an inflammatory response around the dorsal nerve roots and dorsal root ganglion, with pain severity not necessarily correlating with herniation size or degree of thecal sac

deformation; experimental models confirm that exposure of live nerve roots to disc material causes pain and structural nerve changes lasting up to six weeks[68].

Slipman provided strong clinical evidence for chemical radiculopathy, demonstrating patients with clear clinical radiculopathy and annular tears but no mechanical nerve root compression[69]. Tenny described radiculopathy arising from annular tears and irritated root sleeves, manifesting as pain, paresthesia, or weakness[70].

Kobayashi documented ultrastructural changes in nerve roots adjacent to herniated disc material, implicating inflammatory neovascularization and macrophage activity in nerve root damage[71].

Opinion 4 — Summary

Radiculopathy can arise from biochemical inflammation of nerve roots and dorsal root ganglia, with or without visible mechanical compression on imaging. Restricting causation to direct compressive herniations ignores decades of basic science and clinical data on chemical radiculitis and leads to erroneous conclusions in forensic evaluations.

Opinion 5: Spinal injury in an MVC is a sprain/strain of soft tissue and will resolve in 6–12 weeks

What research supports this opinion? None.

While some uncomplicated soft tissue injuries can improve within 6–12 weeks, this timeframe does not apply uniformly to all spinal injuries, particularly when discs, vertebrae, or facet joints are involved. Chronic symptoms and persistent impairment after WAD and other spinal trauma are common.

Hoffberg distinguished between healing timelines for superficial soft tissue injuries and deeper structures, noting that discs and facet joints require longer recovery and may never fully heal[72]. Li (F.) reported that up to 50% of WAD patients develop chronic symptoms, including injuries to facet joints, spinal ligaments, nerve roots, and intervertebral discs[73]. Petersohn similarly found that up to 40% of patients may experience chronic symptoms, with 12% developing moderate to severe pain and 10% exhibiting some degree of disability[74].

Even for ligaments, experimental data contradict the notion of complete healing within weeks. Hefti showed in a rabbit model that transected ligaments did not regain normal strength and flexibility at three months or even one year post-injury; more severe lesions

led to post-traumatic osteoarthritis in adjacent joints[75]. These findings indicate that "healed" ligaments may remain structurally and functionally compromised over the long term.

Opinion 5 — Summary

The assertion that all MVC-related spinal sprain/strain injuries resolve within 6–12 weeks is inconsistent with clinical and experimental evidence. Disc, facet, and ligament injuries frequently lead to chronic pain and functional limitation, and a substantial proportion of WAD patients develop long-term symptoms and disability.

Conclusion (Spinal Injuries)

Forensic evaluation of spinal injuries requires a rigorous, evidence-based approach rather than reliance on unsubstantiated maxims. The literature reviewed here shows that low-velocity crashes can cause disc and facet injuries, that imaging has important limitations, that radiculopathy can arise through inflammatory as well as mechanical mechanisms, and that symptoms may be delayed and chronic rather than uniformly self-limited.

Accurate causation analysis must incorporate biological plausibility, temporality, individual susceptibility, clinical examination, and the known limitations of imaging and neurodiagnostic testing. Applying these scientifically grounded principles improves the credibility of medico-legal opinions and promotes fairer outcomes in litigation involving spinal trauma.

SECTION 2 - TRAUMATIC BRAIN INJURY

Background

Defense medical experts in litigation commonly advance outdated and scientifically unsupported positions regarding traumatic brain injury (TBI), often relying on narrow, obsolete criteria that ignore current consensus definitions and the broader clinical spectrum of TBI. These opinions typically emphasize the absence of direct head impact, loss of consciousness (LOC), or abnormal early imaging (or lack of imaging) as proof that no brain injury occurred, despite decades of literature demonstrating that such requirements are neither necessary nor sufficient for diagnosis. Modern diagnostic frameworks, including those of the American Congress of Rehabilitation Medicine (ACRM), the Centers for Disease Control and Prevention (CDC), and the Department of Veterans Affairs, recognize TBI as a clinical diagnosis based on external force to the brain and resulting disturbances in brain

function, which may be subtle, delayed, or predominantly cognitive, emotional, or behavioral rather than focal on neurologic examination.

These outdated commonly asserted medicolegal opinions have substantial consequences in forensic contexts, where they are used to deny causation, minimize symptom severity, or dismiss persistent disability following mild traumatic brain injury (mTBI). The following section addresses five prevalent maxims concerning TBI diagnosis, imaging, clinical examination, and prognosis, and contrasts these opinions with contemporary evidence and consensus guidelines.

TBI analysis is summarized in Table 3.

Opinion 1: A TBI requires a blow to the head, LOC, or abnormal GCS score

What research papers were found to support this opinion? None.

The literature overwhelmingly supports that LOC is not required for the diagnosis of TBI. Modern diagnostic frameworks emphasize a broader set of clinical features, including alteration of mental state and amnesia, to capture the full spectrum of TBI presentations. A diagnosis of TBI does not require a blow to the head; indirect forces such as rapid acceleration-deceleration or blast waves can also cause TBI. Diagnosis is based on clinical evidence of brain dysfunction following an external force, not solely on the presence of head impact. A normal GCS score does not exclude the diagnosis of TBI. A GCS score may have improved when assessed by EMS rescue services who arrive several minutes after an injury occurs. The eye opening, verbal and motor responses are typically assessed very quickly and in a manner that does not consider nuances. Small contusions, DAI, or extra-axial bleeds may not immediately affect eye, verbal, or basic motor responses, so GCS stays 15 even though pathology is evolving on CT/MRI. Deterioration may occur later with expansion of hematoma, edema, or herniation. Mild TBI, concussion, and diffuse metabolic injury often present with normal GCS but later show cognitive, behavioral, vestibular, or sleep disturbances, which GCS does not measure at all. Symptom onset can be delayed (e.g., headache, confusion, irritability emerging hours to days later).

Comprehensive assessment, including imaging and clinical context, is necessary for accurate diagnosis and management. Follow-up evaluations post-injury for weeks or months or longer are necessary to monitor for the development of TBI sequelae.

Defense medical experts frequently rely on outdated concepts including data from animal and crash dummy testing that fail to incorporate current scientific understanding of TBI mechanisms in humans. When these experts emphasize the absence of direct head trauma

as evidence against brain injury, they demonstrate a fundamental misunderstanding of established medical principles that have been recognized by leading medical organizations for decades. At a minimum, a defense expert providing a forensic opinion regarding TBI would be expected to request to see the plaintiff seated in a similar or same vehicle to analyze potential head impact from surfaces adjacent to the plaintiff's head.

The ACRM (American Congress of Rehabilitation Medicine) definition of mTBI (mild traumatic brain injury) specifically includes scenarios where the brain undergoes an acceleration-deceleration movement without direct external trauma to the head[76]. The brain is a soft gelatinous tissue that is housed inside the skull, which is a rigid bony structure. The mobility of the brain tissue within the skull structure allows the brain to move in a forward and backward direction, allowing fragile brain tissue to strike against the bony rigid structure of the skull, causing bruising and bleeding within brain tissue.

Rotatory forces allow the brain tissue to twist within the skull, causing stretching and tearing of axonal tissue.

De Luigi discusses how a direct blow to the head is not required to cause a brain injury, and that force transmitted from other parts of the body may reach the head and cause brain injury, explaining how athletes may suffer TBI in contact sports causing sports-related concussions[77]. The published CDC Criteria also support brain injury from acceleration-deceleration forces as well as blunt or penetrating trauma[78].

LOC can be difficult to assess accurately, especially retrospectively. Patients may not recall being unconscious, or they may confuse amnesia with unconsciousness. Witness accounts are often unavailable or unreliable. Medical records can lack adequate documentation or may include conflicting and contradictory reports regarding LOC. The defense doctor will argue that there cannot be a TBI because there was no documentation of loss of consciousness. The requirement for LOC at the scene, en route to ED, or at the ED as a mandatory criterion for diagnosis of TBI ignores other important alterations in mental status that TBI patients may experience, including confusion, disorientation, being dazed, dizziness, or amnesia.

The Mayo Classification System incorporates multiple indicators including symptoms such as confusion, amnesia, and focal neurological deficits for classification of TBI severity, precisely because relying on a single indicator like LOC is inadequate, resulting in the exclusion of many true cases of TBI[79]. Sports-related concussion (SRC), which is mTBI, can result in a range of clinical signs and symptoms that do not require a loss of consciousness[80].

The ACRM criteria (1993) for definition of mild traumatic brain injury lists a physiological disruption of brain function that includes any one of the following[81]:

1. Any period of LOC.
2. Any memory loss.
3. Any alteration of mental status proximate to the injury.
4. Focal neurological deficit that may be transient.

The revised ACRM criteria (2023)[82] provide pathways that support establishing the diagnosis of mTBI following a plausible mechanism of injury:

"Mild traumatic brain injury (TBI) is diagnosed when, following a biomechanically plausible mechanism of injury (Criterion 1) one or more of the criteria (i-iii) listed below are met.

i. One or more clinical signs (Criterion 2) attributable to brain injury.
ii. At least 2 acute symptoms (Criterion 3) and at least one clinical or laboratory finding (Criterion 4) attributable to brain injury.
iii. Neuroimaging evidence of TBI, such as unambiguous trauma-related intracranial abnormalities on computed tomography or structural magnetic resonance imaging (Criterion 5)."

Clinical signs include loss of consciousness, alteration of mental status, amnesia, other neurologic signs. Acute symptoms include confusion, disorientation, dazed, headache, nausea, dizziness, vision problems, sensitivity to light or noise, emotional lability, irritability.

With the background of extensive experience in managing the TBI population, the Department of Veterans Affairs concludes that the victim may remain conscious or have an LOC for any duration[83]. Mullaly, citing American Academy of Neurology, defines concussion as a trauma-induced mental status that may or may not be accompanied by LOC[84].

When a TBI event is not witnessed, when there is a delay in evaluation by EMS, or when the LOC is not recognized or recalled by the injured person, there will be no record of LOC[85]~~[82]~~. A brief LOC may be present prior to the arrival of first responders. The Glasgow Coma Scale was designed for rapid triage and not as a nuanced diagnostic tool. It is insufficiently sensitive for detecting subtle or evolving brain injury. A GCS of 15 does not rule out diffuse

axonal injury (DAI) or microbleeds that may only be discovered on advanced brain imaging studies. Secondary injuries, post-concussion syndrome, vestibular, visual, endocrine, cognitive, emotional, and behavioral problems, and neurodegenerative disorders may only be identified over time and can develop due to a TBI despite a normal GCS.

Per Ruff, an initial GCS score may not be obtainable because there was no one present to evaluate for it[86]. Per Madhok, there were patients with a GCS of 15 and negative head CT who were later found to have mTBI on subsequent evaluation[87]. These TBI patients had incomplete recovery and continued to have TBI symptoms even at 6 months post-injury. The author suggested that emergency department physicians should recommend a 2-week follow-up visit to identify those patients who did not have a complete recovery after TBI.

Opinion 1 — Summary

The evidence shows that a traumatic brain injury does not require a direct blow to the head, documented loss of consciousness, or an abnormal GCS score. TBI is a clinical diagnosis that can result from acceleration-deceleration forces without head impact, may present with alteration of mental state or amnesia alone, and may occur in patients whose GCS remains 15 throughout acute care.

Opinion 2: TBI symptoms must begin immediately after injury and TBI cannot be diagnosed unless the diagnosis was made in the ER or at the scene of injury

What research papers were found to support this opinion? None.

The assertion that traumatic brain injury (TBI) must begin immediately after injury, or that TBI cannot be diagnosed unless the diagnosis is made in the emergency room or at the scene, is not supported by current research or clinical guidelines. At the scene, who is going to be able to make an assessment of the injured party? What is their training or experience? Do they know how to perform a GCS evaluation?

Current evidence and clinical guidelines do not require that all TBI symptoms appear immediately after injury, nor that the diagnosis be established at the scene or in the emergency department. TBI is better conceptualized as a disease process that may evolve over hours, days, weeks, or even years, with manifestations that can be physical, cognitive, emotional, behavioral, vestibular, visual, or endocrine in nature.

The clinical signs and symptoms of TBI can include acute, subacute, late, and chronic findings and complications. These findings may evolve over time. Several factors contribute to delayed recognition. Anosognosia (impaired awareness of one's own deficits) is a well-

recognized consequence of brain injury and can prevent patients from accurately perceiving or reporting cognitive, behavioral, or functional changes[88]. Individuals with anosognosia may deny memory problems, attentional deficits, mood changes, or executive dysfunction even when these are obvious to observers, leading to underreporting and delayed care. Unawareness of deficits such as poor balance, slowed thinking, or impaired judgment can put the person at risk for accidents or making unsafe decisions.

De Luigi and colleagues describe the evolution of sports-related concussion symptoms over minutes to hours, emphasizing that early manifestations may be subtle and not immediately reported[89]. For some patients with mTBI per ACRM, there may be a lack of awareness of symptoms or the extent of the symptoms, which may only become clear following an attempt to return to normal activities. In these cases, the evidence for mTBI allows for the diagnosis by reconstruction of the clinical history.

The symptoms of TBI may evolve over time, and because they may involve multiple organ systems, it may require an astute and knowledgeable clinician to connect the dots and make the TBI diagnosis per Bazarian, who notes that some TBI-related problems—such as neuropsychiatric syndromes, seizures, and neurodegenerative changes—may only become apparent months to years after the index injury[90]. Early symptoms can include visual-motor, vestibular, or endocrine disorders that will be identified only if they are screened for by a knowledgeable and experienced brain injury specialist. Seizures, for example, may not develop for months to years after a TBI. Neurodegenerative disorders, e.g., dementia, may occur even years after injury yet may be considerably disabling.

Patient-facing guidance from the CDC and Cleveland Clinic similarly emphasizes that mild TBI can present with a broad array of physical, cognitive, emotional, and sleep-related symptoms that may vary in timing and expression among individuals[91],[92]. An mTBI is a disease process that will be expressed uniquely in each individual, per the CDC, affecting how an individual feels, thinks, sleeps, and acts. An mTBI victim may not recognize or understand how daily activities are affected by the injury, thus the problems may be overlooked and underreported to the healthcare provider.

Carney describes the typical symptoms or some component of these that are present after a concussion (mTBI)[93]:

1. Disorientation or confusion immediately.
2. Impaired balance.
3. Slower reaction time.

4. Impaired verbal learning and memory.

Fuji discusses how TBI can present as a psychiatric disorder, Psychotic Disorder Due to Traumatic Brain Injury (PDTBI), with delusions and hallucinations that need to be differentiated from schizophrenia. These are typically males with psychiatric symptoms occurring within 2 years after moderate or severe TBI, the majority showing abnormalities on MRI/CT and EEG[94].

At the time of injury, a person may not be present to competently observe the neurologic status of an injured person. The EMS system requires time to arrive at the scene. There is a delay with a variable period during which no trained medical personnel can assess the injured person. In emergency rooms, the goal of evaluation and treatment is to establish whether there is a life-threatening injury. If not, the ER moves quickly to assess and discharge the injured person from the hospital. Details and nuances are overlooked in the evaluation process, thus a TBI may be overlooked, particularly if there are other orthopedic or major injuries to attend to.

Per Ruff, the ER is not necessarily offering a deliberate approach that would be needed to thoroughly evaluate for TBI[95]. An initial GCS of 15 is insufficient to exclude an mTBI, and a retrospective analysis to establish whether there was an altered consciousness, gaps in memory or amnesia, or other neurologic signs caused by TBI is necessary. A mild TBI can be overlooked if there is a more dramatic physical injury[96].

Peixoto[97] reports that there was a high prevalence of presumed mTBI in this studied sample involving MVCs, 69.7% of patients, but an acute care diagnosis was made in only 39.1% of cases. Patients whose diagnosis of mTBI was missed in the ER ended up with significantly more severe postconcussion symptoms.

Marco discusses how delays in medical care post-injury may be multifactorial: transportation barriers, financial constraints, homelessness, subjective interpretation of symptoms that are dismissed by patients with little insight, or even fear of emergency rooms and hospitals[98]. TBI symptoms can include visual symptoms, nausea, vomiting, generalized fatigue and weakness, and headaches. These symptoms may appear less severe and may be temporary but are the consequence of TBI. Pozzato discusses how documentation of mTBI is problematic, resulting in underreporting, finding that only 23% of patients with mTBI had an mTBI diagnosis clearly recorded in the medical notes[99].

Opinion 2 —Summary

The literature refutes the claim that TBI must be diagnosed immediately in the field or emergency department and that symptoms must begin at once. Clinical, cognitive, psychiatric, vestibular, and endocrine manifestations of TBI can emerge or be recognized days to years later, are often underdiagnosed in acute settings, and may be obscured by anosognosia, co-injuries, or documentation gaps.

Opinion 3: Structural changes must be seen on neuroimaging studies with bleeding in the brain to diagnose a TBI and DTI and volumetric abnormalities cannot be used to diagnose TBI

What research papers were found to support this opinion? None.

The requirement that structural changes with bleeding must be seen on neuroimaging to diagnose TBI is not supported by current evidence; advanced imaging findings such as DTI and volumetric abnormalities can indicate TBI even in the absence of visible bleeding or gross structural lesions.

There is a critical misconception in traumatic brain injury (TBI) diagnosis: the belief that brain imaging must show blood or hemorrhage to confirm a TBI. This assumption can lead to missed diagnoses and inadequate treatment for patients who have sustained significant brain trauma. The medical reality is that TBI is a clinical diagnosis based on the mechanism of injury, symptom presentation, and neurological examination findings—not solely on imaging results. Many patients with legitimate brain injuries who present with no visible bleeding on standard brain scans still experience the full spectrum of TBI symptoms including confusion, memory loss, and cognitive and emotional impairments.

Koenigsberg emphasizes that pathologic findings in brain tissue are often worse than what imaging studies can show, with microscopic injury to the brain exceeding what conventional imaging can detect[100]. Even when specialized imaging techniques like gradient-echo MRI are used, they may fail to capture the full extent of injury, as hemorrhage markers can fade over time or be too subtle for visualization.

The clinical significance of non-hemorrhagic lesions (NHLs) in the brain in relationship to diffuse axonal injury (DAI) is discussed by Chung[101]. The location of NHLs is similar to the distribution of DAI lesions from trauma, including the splenium of the corpus callosum, thalamus, and mesial temporal lobe. Substantial white matter damage can occur without visible blood products, and moderate to severe TBI patients show diffusion abnormalities indicative of axonal injury that are missed by standard imaging protocols[102].

Dahl notes that cerebral microbleeds (CMBs) have a limited ability to serve as a surrogate for white matter damage because substantial white matter damage can occur without CMBs[103]. Messori discusses how hemosiderin can disappear over time and fail to be visualized by even advanced MRI imaging techniques; foci consistent with isolated intracerebral hemosiderin deposits on images at 4–6 months were not seen at 24-month assessment, and even with the use of gradient-echo (GRE) MRI, time affects the visibility of hemorrhagic intracerebral lesions[104]. The caveat being that normal GE images may not exclude old hemorrhage. Jolly reports that the absence of bleeding in the brain does not mean that brain injury does not exist, based on research finding that 52% of moderate-severe TBI patients have diffusion abnormalities indicative of DAI, abnormalities often missed using T1, FLAIR, or SWI imaging[105].

Bruggeman discusses the non-hemorrhagic traumatic axonal injury (TAI) pathophysiology and imaging findings, defined as multiple, scattered, small hemorrhagic and/or non-haemorrhagic lesions[106]. Also described are axonal swelling, impaired axoplasmic transport, and axonal tearing and disruption due to TBI, typically more confined to white matter distribution. Standard MRI studies of the brain may not detect hemosiderin (old blood product deposits) that result from bleeding caused by trauma, per Wardlaw, who recommends gradient-echo sequences (GRE) to supplement the TBI imaging evaluation[107].

Crucially, advanced MRI techniques such as diffusion tensor imaging (DTI) and volumetric analyses provide objective biomarkers of TBI-related white and gray matter injury.

Kinnunen and colleagues reported widespread reductions in fractional anisotropy in multiple white matter tracts—including the corpus callosum, cingulum, fornix, internal and external capsules, and major association pathways—in patients with mTBI, changes that correlated with impairments in memory and executive function[108]. DTI is highly sensitive to microstructural white matter disruption and can detect axonal injury within hours of trauma, with abnormalities that may persist over time even when conventional MRI appears normal.

Hellström et al. demonstrated robust associations between DTI measures of white matter integrity and cognitive, somatic, and emotional symptoms 12 months after mTBI, particularly in frontal regions such as the genu of the corpus callosum[109].

Volumetric MRI studies similarly show that mTBI can result in measurable brain atrophy. MacKenzie and others observed whole-brain volume loss in patients with mild or moderate TBI at an average of 11 months post-injury, with greater atrophy in those with LOC[110].

Patel et al. found that patients with mTBI had a higher burden of white matter hyperintensities and regionally specific parenchymal volume loss compared with controls, features associated with injury-related cognitive dysfunction[111]. Reber and colleagues demonstrated that damage to densely connected white matter hubs is more strongly associated with cognitive impairment than comparable lesions in gray matter[112], underscoring the importance of microstructural and network-level disruption rather than gross hemorrhage alone.

Gale discussed the clinical significance of gray matter damage caused by TBI; brain-injured patients had decreased gray matter concentration in multiple brain regions including frontal and temporal cortices, cingulate gyrus, subcortical gray matter, and the cerebellum, which correlated with lower scores on tests of attention[113]. Hellström, in 2017, nearly a decade ago, discussed the importance of DTI in evaluation of DAI; diffuse axonal injury (DAI) may be the primary pathologic feature of mTBI but is normally not detectable by conventional imaging technology[114]. DTI is sensitive to microstructural properties of brain tissue and has been suggested to be a promising candidate for the detection of DAI in vivo. This study reported strong associations between brain white matter DTI and self-reported cognitive, somatic, and emotional symptoms at 12 months post-injury in 134 mTBI patients.

Patel discusses the significance of white matter hyperintensities (WMH) in TBI, noting that white matter hyperintensities were seen in 81% of the brain-injured group versus 60% of healthy controls, with a frontal lobe-only distribution of white matter hyperintensities more commonly seen in the mild traumatic brain injury group[115]. The odds of one white matter hyperintensity in the brain-injured group were about 3.5 times the odds for the healthy control's cohort, with the mTBI injury cohort showing regional parenchymal volume loss in the areas of damage.

The location of the lesions found on brain MRI images is a determining factor in prognosis per Reber, noting that lesions of white matter regions with dense structural connections have the strongest association with cognitive impairment, while gray matter functional centrality measures have weaker associations[116].

There is no shortage of evidence that contradicts the persistent maxim that brain imaging studies showing DTI and volumetric abnormalities do not support a diagnosis of TBI. Extensive literature demonstrates that advanced neuroimaging techniques not only support TBI diagnosis but are essential for comprehensive patient evaluation and management. The clinical application of these techniques is no longer confined to research settings but has become an integral component of current-day TBI assessment. It is remarkable that defense

doctors have not been keeping up to date with the TBI medical literature that supports the clinical (not research) application of advanced MRI imaging studies.

Opinion 3 — Summary

The notion that TBI requires visible bleeding or gross structural abnormalities on standard CT/MRI, and that DTI or volumetric changes do not support the diagnosis, is not scientifically defensible. Many patients with clinically significant TBI have normal conventional imaging yet show microstructural white matter damage, non-hemorrhagic axonal injury, and progressive atrophy on advanced MRI that correlate with persistent cognitive and functional deficits. The brain is a non-homogeneous living structure, and the components of the brain and individual brains are subject to responding differently to traumatic events.

Opinion 4: There were no focal neurological findings on the exam, therefore there is no TBI

What research papers were found to support this opinion? None.

Many of the defense doctors we have reviewed were found to have performed neurological examinations that are flawed, non-comprehensive and incomplete. They used these incomplete examinations to argue that there can be no TBI because mental status, cranial nerves, strength, sensation, coordination, reflexes, and gait are reported as normal based on their incomplete examinations. This means that the opinions that they rendered are based upon incomplete and flawed data. An analysis and review of these defense medical examinations was prepared by Hunter.[117]

The absence of focal neurological findings on exam does not rule out traumatic brain injury (TBI), as especially mild forms can occur without focal deficits and may be detectable only through imaging, biomarkers, cognitive, behavioral or emotional changes, or subtle clinical changes. The defense position that a neurological examination must be abnormal is strongly contradicted by the evidence that the clinical picture of traumatic brain injury can come in many forms and is not limited to the neurological examination alone. To limit the scope of brain injury to abnormalities of strength, sensation, reflexes, and coordination glaringly ignores the spectrum of the consequences of TBI, which include cognitive, behavioral, emotional, and social consequences of brain injury.

The ACRM notes that patients with mTBI may experience persistent emotional, cognitive, behavioral, and physical symptoms—alone or in combination—resulting in substantial functional disability[118]. Physical symptoms include headache, dizziness, nausea, sleep

disturbance, visual changes, and fatigue; cognitive deficits encompass impairments in attention, concentration, memory, speech-language, and executive functions; behavioral changes include irritability, disinhibition, emotional lability, and reduced frustration tolerance. None of these domains is adequately captured by a terse statement such as "cranial nerves II–XII grossly intact" or a brief screening neurological exam performed in a busy emergency department.

Ruff and colleagues emphasize that comprehensive neurologic and neuropsychological evaluation is rarely undertaken in acute care settings focused on triage and life-threatening conditions[119]. Detailed assessment of cranial nerve function, including olfaction and subtle ocular motor deficits, balance, gait, coordination, language, and higher cortical functions requires time and expertise that are often unavailable in emergency environments. Moreover, vestibular, visual, and oculomotor abnormalities—common after TBI—may be missed unless specifically evaluated.

Rauchman and coauthors describe how TBI-related visual and perceptual disturbances can significantly impair reading, driving, navigation, and other daily activities despite a largely normal standard neurologic examination[120]. Focal neurological signs that result from TBI could be found in a comprehensive neurological examination of vision, hearing, language, sensory-perception, motor function, coordination, gait, balance, and cranial nerves including sense of smell. But how often are all 12 cranial nerves tested in detail?

There are more than 40 potential cranial nerve tests that could be performed in the emergency room when an acute TBI is being evaluated. This means that the typical statement from the emergency department doctor evaluation "cranial nerves II–XII grossly intact" does not hold much weight.

Opinion 4 — Summary

The absence of focal neurological findings on examination does not exclude TBI, particularly in mild injuries. Modern criteria recognize that TBI commonly produces predominantly nonfocal consequences—cognitive, emotional, behavioral, sensory, and balance disturbances—that may be missed by a brief, gross neurological exam focused only on strength, reflexes, and coordination. Consequently, the claim that a normal focal neurological examination proves the absence of TBI is contradicted by both consensus criteria and clinical experience and represents an unjustified narrowing of the diagnostic lens in forensic settings.

Opinion 5: TBI prognosis is always good and TBI does not increase the risk of dementia

What research papers were found to support this opinion? None.

The prognosis of TBI—especially mTBI—is far more heterogeneous than suggested by the simplistic assertion that patients fully recover within weeks to months. A substantial proportion of individuals with mTBI experience persistent symptoms, functional limitations, and increased long-term risks of neuropsychiatric and neurodegenerative conditions, including dementia. Multiple longitudinal studies demonstrate that mTBI can result in long-standing problems with cognition, emotional regulation, sleep, and daily functioning.

Mutch described the prognosis for mTBI, defined by a GCS of 13, noting that many patients in this category often experience long-term debilitating symptoms that interfere with normal daily activities[121]. Carroll reports that mTBI continued to show significant impairment at 2 years post-injury with problems with emotional well-being, alcohol use, and cognition, as well as symptoms such as headaches and sleep disturbance, which are common after mTBI and often long-standing[122]. Brain atrophy is found in moderate to severe TBI, discussed by Harris, with brain tissue loss on the order of 5% per year for moderate-to-severe traumatic brain injury, with generalized atrophy across the entire brain or focal atrophy in specific brain regions: thalamus, hippocampus, and cerebellum in gray matter and the corpus callosum, corona radiata, and brainstem in white matter[123].

McInnes et al. reported chronic cognitive impairment in up to 50% of mTBI patients, challenging the prevailing notion that most concussive symptoms resolve within 3 months[124]. Nelson and the TRACKTBI investigators found that only 47% of patients with mTBI treated at level I trauma centers had returned to pre-injury functional levels at 1 year, with increased rates of headaches, fatigue, depression, forgetfulness, and poorer performance on cognitive testing compared with controls[125].

TBI is also associated with progressive structural brain changes and elevated suicide risk. Agrawal et al. found that a history of TBI—even without LOC—is associated with adverse neuropathologic outcomes, including neurodegenerative and vascular changes[126]. Lu and coauthors reported that adults with TBI had more than double the risk of suicide attempts compared with non-injured controls[127]

Bazarian discusses the multiple long-term problems found in TBI patients, with a strong association with several neurologic disorders 6 months or more after injury[128]; with an increased risk for parkinsonism in patients with mild TBI and LOC and with more severe TBI, with impairment in ocular and visual-motor function after a mild TBI that may persist up to 1 year post-injury, and saccadic eye movement disorders resulting from TBI of all severities.

They further report that a broad range of cognitive, physical, and affective symptoms are more common in those with mild and more severe TBI than in those with other injuries that exclude the head.

Singh describes TBI functional disability at 1-year post-trauma, with outcome after TBI being worse than many other studies have documented even in a largely mild injury group[129]. Sendroy-Terrill reports post-TBI increasing loss of function in each subsequent decade, resulting in increased restrictions of function; noting that increasing decades post-injury predicted declines in physical and cognitive functioning and declines in societal participation[130]. Also finding increases in fatigue, declines in societal participation, and declines in perceived environmental barriers, with the likelihood of substantial overall restrictions in participation increased by 62% for each increase of 1-decade post-injury.

The instrument used to assess the impact of environmental factors on participation was the Craig Hospital Inventory of Environmental Factors (CHIEF), a measure of the frequency and magnitude of environmental barriers to participation reported by respondents.

Visual problems after TBI can significantly affect function per Rauchman, with manifestations of TBI that disturb vision, perception, and ability to perform essential everyday tasks such as reading, typing, driving, and navigating within the environment[131]. If brain imaging studies reveal structural abnormalities in mTBI, the prognosis is worsened. Williams finds that disability persisting for 6 months post-injury was more common after mild injuries complicated by either an extra parenchymal or intracerebral lesion than after uncomplicated mild injuries, resulting in more severe behavioral sequelae and disability[132].

Despite the reluctance of some medical professionals to acknowledge this connection, the evidence presents overwhelming scientific findings demonstrating the link between TBI and increased dementia risk. Most concerning is the evidence showing that even a single traumatic brain injury is associated with increased dementia risk. The defense position is not merely an academic disagreement but a systematic denial of well-established medical evidence.

There is a significant increase in the risk of dementia in veterans reported by Barnes in this study of more than 350,000 veterans; mild TBI without LOC was associated with more than a 2-fold increase in the risk of dementia diagnosis[133]. LoBue discusses the increased risk of neurodegenerative conditions in TBI even if an individual risk level cannot be determined[134][130]. Finding that there is evidence to support a link between the history of TBI and the later development of neurodegenerative conditions, and though individual risk

cannot be determined, TBI appears to be associated with earlier onset of some neurodegenerative disorders.

Johnson found an increased risk of dementia associated with a single TBI event, presenting evidence of persistent inflammation and ongoing white matter degeneration for many years after just a single traumatic brain injury[135]. Schneider reports a dose-dependent relationship of TBI and dementia in this community-based cohort with 25-year follow-up[136]. Fakhran reports a disturbing pattern of white matter lesions in symptomatic mTBI similar to early Alzheimer's dementia[137].

Functional outcomes over the lifespan also reflect the chronic impact of TBI. Singh and coauthors found that functional disability at 1 year after TBI was worse than previously appreciated, even in largely mild injury groups. Sendroy-Terrill et al. observed that each additional decade after TBI was associated with declining physical and cognitive function, reduced participation in society, and increased fatigue, highlighting TBI as a long-term health condition rather than a transient event[138]. When structural abnormalities are present on imaging after mTBI, the likelihood of persistent disability is further increased.

Accordingly, the defense position that TBI prognosis is uniformly good and unrelated to dementia risk is starkly at odds with a large body of clinical, epidemiologic, and neuropathologic evidence and fails to meet contemporary standards for scientifically grounded medical opinion.

Opinion 5 — Summary

The assertion that TBI prognosis is always good and does not increase dementia risk is contradicted by longitudinal clinical, imaging, and neuropathologic data. Even mild TBI is associated in many patients with long-term symptoms, progressive brain atrophy, elevated risks of suicide and neurodegenerative disease, and a dose-responsive increase in later-life dementia, demonstrating that TBI is often a chronic, evolving condition rather than a transient event. The presence of a robust and caring family support system and financial and geographical resources can dramatically improve both the coping ability and the outcome of a TBI patient.

Conclusion (Traumatic Brain Injury)

The analysis of these traumatic brain injury maxims demonstrates a systematic disconnect between commonly asserted forensic~~defense~~ opinions and current scientific understanding of TBI mechanisms, diagnosis, and prognosis. The literature confirms that TBI can occur without direct head impact or documented LOC, that early emergency evaluations

frequently miss or under-document mTBI, that clinically significant axonal and volumetric injury is often invisible on routine structural imaging, and that clinically important TBI may exist in the absence of focal neurological signs. Furthermore, longitudinal cohort, imaging, and neuropathologic studies establish that TBI is frequently a chronic disease process, with substantial risks of persistent symptoms, progressive atrophy, and increased incidence of later-life neurodegenerative disorders, including dementia, even after injuries classified as mild. Forensic opinions that rely on these maxims therefore lack a scientific foundation and should be replaced by evidence-based assessments that integrate mechanism of injury, clinical evolution, advanced imaging, and long-term outcome data when evaluating claimed brain injury in litigation contexts.

SECTION 3 - INTEGRATED SUMMARY AND CONCLUSIONS

This review article identifies and analyzes a series of persistent forensic maxims concerning the causation, diagnosis, and prognosis of spinal and traumatic brain injuries in motor vehicle and other trauma litigation. For the spine, commonly asserted maxims (that low-velocity crashes or any calculated delta-V cannot cause disc injury, that all disc and facet pathology is purely degenerative and not permanently aggravated by trauma, that normal or nonspecific MRI findings exclude clinically important injury, that radiculopathy requires visible nerve root compression, and that all spinal injuries are self-limited sprain-strain conditions resolving within weeks) are contradicted by epidemiologic, biomechanical, imaging, and clinical outcome studies. The literature instead demonstrates that intervertebral discs and facet joints are highly vulnerable to trauma, that pre-existing degeneration often increases susceptibility to injury, that standard imaging underestimates both the presence and timing of structural damage, and that a substantial subset of patients develop chronic pain, radiculopathy, and functional impairment after seemingly minor collisions.

Similarly, in the domain of traumatic brain injury, the recurrent forensic opinions (that mild TBI requires a direct head blow with documented loss of consciousness and abnormal GCS, that delayed recognition or documentation precludes the diagnosis, that only hemorrhage or overt structural lesions on conventional CT/MRI can substantiate TBI, that a normal focal neurological examination rules out brain injury, and that prognosis is uniformly good with no increased risk of dementia) are inconsistent with modern diagnostic frameworks and long-term outcome data. Contemporary consensus criteria, advanced MRI techniques including DTI and volumetrics, and large cohort studies show that acceleration-deceleration mechanisms without head impact can cause TBI, that symptoms may evolve over hours to years, that microstructural and volumetric changes can be present despite normal

conventional imaging, and that even a single mild TBI confers an elevated risk of persistent symptoms, progressive atrophy, and later neurodegenerative disease.

Together, these findings support a scientifically grounded forensic approach that incorporates causation analysis, individual vulnerability, and the full breadth of clinical and imaging evidence, rather than reliance on unsubstantiated maxims and anecdotal experience. Both spinal and brain injuries merit serious consideration as potentially chronic conditions with substantial impact on long-term function and quality of life, and forensic medical opinions should reflect current scientific understanding rather than outdated or economically motivated perspectives.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the authors used Consensus AI, Perplexity Enterprise Pro Deep Research, and Claude Opus and Sonnet to perform medical literature searches and to assist in editing and summarizing content. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Funding source

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of Competing Interest

None

Appendix A. Supplementary data

Download all supplementary files included with this article What's this? ↗

The following are the Supplementary data to this article:

Download: Download Word document (16KB)

Download: Download Word document (18KB)

Download: Download Word document (18KB)

Recommended articles

REFERENCES

1 Freeman M.D., Croft A.C., Rossignol A.M., Weaver D.S., Reiser M.
A Review and Methodologic Critique of the Literature Refuting Whiplash Syndrome
Spine, 24 (1) (1999), pp. 86-96
View in Scopus ↗ Google Scholar ↗

2 Zhuge W., Ben-Galim P., Hipp J.A., Reitman C.A.
Efficacy of MRI for assessment of spinal trauma: correlation with intraoperative findings
J Spinal Disord Tech, 28 (4) (2015), pp. 147-151, 10.1097/BSD.0b013e31827734bc ↗
View in Scopus ↗ Google Scholar ↗

3 Rondinelli RD, Genovese E, Katz RT, et al. AMA Guides to the Evaluation of Permanent Impairment. 6th ed. Chicago, IL: American Medical Association; 2022. doi.org/10.1001/978-1-64016-282-2
Google Scholar ↗

4 Nolet P.S., Nordhoff L., Kristman V.L., Croft A.C., Zeegers M.P., Freeman M.D.
Is acceleration a valid proxy for injury risk in minimal damage traffic crashes? A comparative review of volunteer, ADL, and real-world studies
Int J Environ Res Public Health, 18 (2021), p. 2901, 10.3390/ijerph18062901 ↗
Google Scholar ↗

5 Freeman M.D., Leith W.M.
Estimating the number of traffic crash–related cervical spine injuries in the United States: an analysis and comparison of national crash and hospital data
Accid Anal Prev, 142 (2020), Article 105571, 10.1016/j.aap.2020.105571 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

6 Kent R., Cormier J., McMurry T.L., Ivarsson B.J., Funk J., Hartka T., Sochor M.
Spinal injury rates and specific causation in motor vehicle collisions
Accid Anal Prev., 186 (2023 Jun), Article 107047, 10.1016/j.aap.2023.107047 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

7 Nolet P.S., Nordhoff L., Kristman V.L., Croft A.C., Zeegers M.P., Freeman M.D.
Is acceleration a valid proxy for injury risk in minimal damage traffic crashes? A comparative review of volunteer, ADL, and real-world studies
Int J Environ Res Public Health, 18 (2021), p. 2901, 10.3390/ijerph18062901 ↗
Google Scholar ↗

8 Freeman Michael D., Centeno Christopher J., Kohles Sean S.
A Systematic Approach to Clinical Determinations of Causation in Symptomatic Spinal Disk Injury Following Motor Vehicle Crash Trauma
PM&R, 1 (10) (2009), pp. 951-963
Crossref ↗ View in Scopus ↗ Google Scholar ↗

9 Aly T.
Correlation between presence of traumatic disco-ligamentous injuries as an MRI finding with the results of management of thoracolumbar and lumbar injuries
Journal of Orthopaedics, Trauma and Rehabilitation, 30 (2023), pp. 1-8, 10.1177/22104917221147690 ↗
Google Scholar ↗

10 Kelly J.C., Groarke P.J., Butler J.S., Poynton A.R., O'Byrne J.M.
The natural history and clinical syndromes of degenerative cervical spondylosis
Adv Orthop, 2012 (2012), Article 393642, 10.1155/2012/393642 ↗
Google Scholar ↗

11 Dydyk AM, Ngnitewe Massa R, Mesfin FB. Disc herniation. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2023. Updated January 16, 2023. Accessed February/19/2026.
Google Scholar ↗

12 Furman M.B.
Cervical disc disease clinical presentation
Medscape (February 8, 2017)
Google Scholar ↗

13 Hashish R., Badday H.
Frequency of acute cervical and lumbar pathology in common types of motor vehicle collisions: a retrospective record review

BMC Musculoskelet Disord, 18 (1) (2017), p. 437, 10.1186/s12891-017-1797-5 ↗

View in Scopus ↗ Google Scholar ↗

14 Hoffberg H.
Whiplash injuries in motor vehicle accidents
Pract Pain Manag, 2 (5) (2002)
Google Scholar ↗

15 Pettersson K., Hildingsson C., Toolanen G., Fagerlund M., Björnebrink J.
Disc pathology after whiplash injury. A prospective magnetic resonance imaging and clinical investigation
Spine (Phila Pa 1976), 22 (3) (1997 Feb 1), pp. 283-287
Google Scholar ↗

16 Li F., Liu N., Liu H., Zhang B., Tian S., Tan M., Sandoz B.
A review of neck injury and protection in vehicle accidents
Transp Saf Environ, 1 (2) (2019), pp. 89-105, 10.1093/tse/tdz012 ↗
View in Scopus ↗ Google Scholar ↗

17 Moheimani R.S., Arastoo S., Moheimani A.M.
Narrative review: disc herniation after motor vehicular trauma
BackBone, 3 (1) (2020)
https://escholarship.org/uc/item/32j4415t ↗
Google Scholar ↗

18 Pencle FJ, Mesfin FB. Cervical disc injuries. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2023.
Google Scholar ↗

19 MayoClinic.Herniateddisc—symptomsandcauses.PublishedOctober24,2023.
https://www.mayoclinic.org/diseases-conditions/herniated-disc/symptoms-causes/syc-20354095 ↗
Google Scholar ↗

20 Adams M.A., Roughley P.J.
What is intervertebral disc degeneration, and what causes it?
Spine, 31 (2006), pp. 2151-2161
Crossref ↗ View in Scopus ↗ Google Scholar ↗

21 University of Rochester Medicine. What is a Herniated Disc? https://www.urmc.rochester.edu/conditions-and-treatments/herniated-disc ↗. Accessed February/19/2026.

Google Scholar ↗

22 American Academy of Neurological Surgeons.https://www.aans.org/patients/conditions-treatments/herniated-disc/ ↗. Accessed February/19/2026.

Google Scholar ↗

23 American Academy of Orthopaedic Surgeons. Herniated disc in the lower back. Accessed Month Day, Year. https://orthoinfo.aaos.org/en/diseases--conditions/herniated-disc-in-the-lower-back/ ↗. Accessed February/19/2026.

Google Scholar ↗

24 Paliaoutas O.V., Evangelopoulos D., Vasiliadis E., *et al.*
Role of platelet-rich plasma (PRP) in the management of stage III and IV degenerative disc disease
Cureus, 17 (2) (2025), Article e79504, 10.7759/cureus.79504 ↗

Google Scholar ↗

25 Boden S.D., McCowin P.R., Davis D.O., Dina T.S., Mark A.S., Wiesel S.
Abnormal magnetic resonance scans of the cervical spine in asymptomatic subjects: a prospective investigation
J Bone Joint Surg Am, 72 (8) (1990), pp. 1178-1184

Crossref ↗ View in Scopus ↗ Google Scholar ↗

26 Boden S.D., Davis D.O., Dina T.S., Patronas N.J., Wiesel S.W.
Abnormal magnetic resonance scans of the lumbar spine in people without back pain
J Bone Joint Surg Am, 72 (3) (1990), pp. 403-408

Crossref ↗ View in Scopus ↗ Google Scholar ↗

27 Brinjikji W., Luetmer P.H., Comstock B., Bresnahan B.W., Chen L.E., Deyo R.A., Halabi S., Turner J.A., Avins A.L., James K., Wald J.T., Kallmes D.F., Jarvik J.G.
Systematic literature review of imaging features of spinal degeneration in asymptomatic populations
AJNR Am J Neuroradiol, 36 (4) (2015), pp. 811-816, 10.3174/ajnr.A4173 ↗

View in Scopus ↗ Google Scholar ↗

28 Colwell C. Geriatric trauma: initial evaluation and management. UpToDate. Literature review current through December 2023. https://bsgdtphcm.vn/thamkhaotam/d/topic.htm?path=geriatric-trauma-initial-evaluation-and-management ↗ Accessed February/19/2026.

Google Scholar ↗

29 Felson D.T.
Risk factors for osteoarthritis: understanding joint vulnerability
Clin Orthop Relat Res, 427 (Suppl) (2004), pp. S16-S21, 10.1097/01.blo.0000144971.12731.a2 ↗

View in Scopus ↗ Google Scholar ↗

30 Freeman M.D., Cahn P.J., Franklin F.A.
A practicable and systematic approach to medicolegal causation
Orthopedics, 41 (2018), pp. 70-72, 10.3928/01477447-20180227-02 ↗

View in Scopus ↗ Google Scholar ↗

31 Kos N., Gradisnik L., Velnar T.
A brief review of the degenerative intervertebral disc disease
Med Arch, 73 (6) (2019), pp. 421-424, 10.5455/medarh.2019.73.421-424 ↗

View in Scopus ↗ Google Scholar ↗

32 Mayo Clinic. Herniated disc—symptoms and causes. Accessed October 24, 2023. https://www.mayoclinic.org/diseases-conditions/herniated-disc/symptoms-causes/syc-20354095 ↗

Google Scholar ↗

33 Rondinelli RD, Genovese E, Katz RT, et al. AMA Guides to the Evaluation of Permanent Impairment. 6th ed. Chicago, IL: American Medical Association; 2022.

Google Scholar ↗

34 Siccoli A., Staartjes V.E., de Wispelaere M.P., Schröder M.L.
Tandem disc herniation of the lumbar and cervical spine: case series and review of the epidemiological, pathophysiological, and genetic literature
Cureus, 11 (2) (2019), Article e4081, 10.7759/cureus.4081 ↗

Google Scholar ↗

35 Yoganandan N., Cusick J.F., Pintar F.A., Rao R.D.
Whiplash injury determination with conventional spine imaging and cryomicrotomy
Spine (Phila Pa 1976), 26 (22) (2001), pp. 2443-2448, 10.1097/00007632-200111150-00010 ↗

View in Scopus ↗ Google Scholar ↗

36 Aarnio M., Fredrikson M., Lampa E., Sörensen J., Gordh T., Linnman C.
Whiplash injuries associated with experienced pain and disability can be visualized with [11C]-D-deprenyl positron emission tomography and computed tomography
Pain, 163 (3) (2022), pp. 489-495, 10.1097/j.pain.0000000000002381 ↗
View in Scopus ↗ Google Scholar ↗

37 Curatolo M., Bogduk N., Ivancic P.C., McLean S.A., Siegmund G.P., Winkelstein B.A.
The role of tissue damage in whiplash-associated disorders: discussion paper 1
Spine, 36 (2011), pp. S309-S315, 10.1097/BRS.0b013e318238842a ↗
View in Scopus ↗ Google Scholar ↗

38 Petersohn JD, Padmanabhan G, Desai MJ. Whiplash associated disorder and cervical facet pain. In: Diwan S, Deer TR, eds. Advanced Procedures for Pain Management: A Step-by-Step Atlas. New York, NY: Springer; 2015:247-257. doi:10.1093/med/9780199931484.003.0015
Google Scholar ↗

39 Freeman M.D.
Cervical sprain and strain
Medscape (October 17, 2023)
Google Scholar ↗

40 Siegmund G.P., *et al.*
The anatomy and biomechanics of acute and chronic whiplash injury
Traffic Inj Prev., 10 (2) (2009), pp. 101-112, 10.1080/15389580802593269 ↗
View in Scopus ↗ Google Scholar ↗

41 Siegmund G.P., Myers B.S., Davis M.B., Bohnet H.F., Winkelstein B.A.
Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading
Spine, 26 (19) (2001), pp. 2095-2101, 10.1097/00007632-200110010-00010 ↗
View in Scopus ↗ Google Scholar ↗

42 Siegmund G.P.
The biomechanics of whiplash injury
BC Med J, 44 (5) (2002), pp. 243-247

View in Scopus ↗ Google Scholar ↗

43 Siegmund G.P.
The biomechanics of whiplash injury
BC Med J, 44 (5) (2002), pp. 243-247
View in Scopus ↗ Google Scholar ↗

44 Tidy C.
Whiplash and cervical spine injury
Patient (February 17, 2021)
info. Reviewed by Knott L.
https://patient.info/doctor/whiplash-and-cervical-spine-injury ↗, Accessed 17th Feb 2021
Google Scholar ↗

45 Kramer W.C., Hendricks K.J., Wang J.
Pathogenetic mechanisms of posttraumatic osteoarthritis: opportunities for early intervention
Int J Clin Exp Med, 4 (4) (2011), pp. 285-298
View in Scopus ↗ Google Scholar ↗

46 Punzi L., Galozzi P., Luisetto R., Favero M., Ramonda R., Oliviero F., Scanu A.
Post-traumatic arthritis: overview on pathogenic mechanisms and role of inflammation
RMD Open, 2 (2) (2016), Article e000279, 10.1136/rmdopen-2016-000279 ↗
View in Scopus ↗ Google Scholar ↗

47 Mayo Clinic. Osteoarthritis—symptoms and causes. Accessed June 16, 2021.
https://www.mayoclinic.org/diseases-conditions/osteoarthritis/symptoms-causes/syc-20351925 ↗
Google Scholar ↗

48 Divi S.N., *et al.*
Can imaging characteristics on magnetic resonance imaging predict the acuity of a lumbar disc herniation?
Int J Spine Surg, 15 (3) (2021), pp. 458-465, 10.14444/8032 ↗
View in Scopus ↗ Google Scholar ↗

49 Williams A.L., Murtagh F.R., Rothman S.L., Sze G.K.
Lumbar disc nomenclature: version 2.0
AJNR Am J Neuroradiol, 35 (11) (2014), p. 2029, 10.3174/ajnr.A4108 ↗

View in Scopus ↗ Google Scholar ↗

50 Herzog R.J.
The radiologic assessment for a lumbar disc herniation
Spine, 21 (1996), pp. 19S-38S, 10.1097/00007632-199612151-00004 ↗
Google Scholar ↗

51 Jensen M.C., Brant-Zawadzki M.N., Obuchowski N., *et al.*
Magnetic resonance imaging of the lumbar spine in people without back pain
N Engl J Med, 331 (1994), pp. 69-73, 10.1056/NEJM199407143310201 ↗
View in Scopus ↗ Google Scholar ↗

52 Munter F.M., Wasserman B.A., Wu H.M., Yousem D.M.
Serial MR imaging of annular tears in lumbar intervertebral discs
AJNR Am J Neuroradiol, 23 (7) (2002), pp. 1105-1109
View in Scopus ↗ Google Scholar ↗

53 Ross J.S.
Counterpoint: appropriately ambiguous—the disc osteophyte complex
AJNR Am J Neuroradiol (2016), 10.3174/ajnr.A4928 ↗
Google Scholar ↗

54 Haris A.M., Vasu C., Kanthila M., Ravichandra G., Acharya K.D., Hussain M.M.
Assessment of MRI as a modality for evaluation of soft tissue injuries of the spine as compared to intraoperative assessment
J Clin Diagn Res, 10 (3) (2016), pp. TC01-TC05, 10.7860/JCDR/2016/17427.7377 ↗
Google Scholar ↗

55 Acosta Julbe J.I., Gottreich J.R., Ermann J., *et al.*
Reporting of facet joint inflammation in lumbar spine MRI studies in patients with low back pain
Spine, 49 (10) (2024), pp. 733-740, 10.1097/BRS.0000000000004923 ↗
View in Scopus ↗ Google Scholar ↗

56 Konieczny M.R., Reinhardt J., Prost M., *et al.*
Signal intensity of lumbar disc herniations: correlation with age of herniation for extrusion, protrusion, and sequestration
Int J Spine Surg, 14 (1) (2020), pp. 102-107, 10.14444/701 ↗
View in Scopus ↗ Google Scholar ↗

57 Rafiee F., Asadollahi S., Luna R., Motaghi M., Hoseinyazdi M., Gong G., Yousem D.M.
Long-term follow-up of lumbar spine annular fissures
J Orthop Sports Med, 5 (2023), pp. 241-248, 10.26502/josm.511500107 ↗
Google Scholar ↗

58 Wang Z.X., Hu Y.G.
High-intensity zone (HIZ) of lumbar intervertebral disc on T2-weighted magnetic resonance images: spatial distribution and correlation of distribution with low back pain (LBP)
Eur Spine J, 21 (7) (2012), pp. 1311-1315, 10.1007/s00586-012-2240-0 ↗
View in Scopus ↗ Google Scholar ↗

59 Wang H., Li Z., Zhang C., Zhang W., Li L., Guo J., Wu W., Hou S.
Correlation between high-intensity zone on MRI and discography in patients with low back pain
Medicine (Baltimore), 96 (30) (2017), Article e7222, 10.1097/MD.0000000000007222 ↗
View in Scopus ↗ Google Scholar ↗

60 Haris A.M., Vasu C., Kanthila M., Ravichandra G., Acharya K.D., Hussain M.M.
Assessment of MRI as a modality for evaluation of soft tissue injuries of the spine as compared to intraoperative assessment
J Clin Diagn Res, 10 (3) (2016), pp. TC01-TC05, 10.7860/JCDR/2016/17427.7377 ↗
Google Scholar ↗

61 Moheimani R.S., Arastoo S., Moheimani A.M.
Narrative review: disc herniation after motor vehicular trauma
BackBone, 3 (1) (2020)
https://escholarship.org/uc/item/32j4415t ↗
Google Scholar ↗

62 Sehgal N., Fortin J.D.
Internal disc disruption and low back pain
Pain Physician, 3 (2) (2000), pp. 143-157
View in Scopus ↗ Google Scholar ↗

63 Theodore N.
Degenerative cervical spondylosis
N Engl J Med, 383 (2) (2020), pp. 159-168, 10.1056/NEJMra2003558 ↗
PMID: 32640134

View in Scopus ↗ Google Scholar ↗

64 Carette S., Fehlings M.G.
Clinical practice: cervical radiculopathy
N Engl J Med., 353 (4) (2005), pp. 392-399, 10.1056/NEJMcp043887 ↗
View in Scopus ↗ Google Scholar ↗

65 Cosamalón-Gan I., Cosamalón-Gan T., Mattos-Piaggio G., Villar-Suárez V., García-Cosamalón J., Vega-Álvarez J.A.
Inflammation in intervertebral disc herniation
Neurocirugia (Engl Ed), 32 (1) (2021), pp. 21-35, 10.1016/j.neucir.2020.01.001 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

66 Furman M.B.
Cervical disc disease clinical presentation
Medscape (February 8, 2017)
Published
Accessed 8th Feb 2017
Google Scholar ↗

67 Hsu PS, Armon C, Levin K. Acute lumbosacral radiculopathy: pathophysiology, clinical features, and diagnosis. UpToDate. Updated August 27, 2021.
Google Scholar ↗

68 Lin J.H., Chiang Y.H., Chen C.C.
Lumbar radiculopathy and its neurobiological basis
World J Anesthesiol, 3 (2) (2014), pp. 162-173, 10.5313/wja.v3.i2.162 ↗
Google Scholar ↗

69 Slipman C.W., Isaac Z., Lenrow D.A., Chou L.H.
Clinical evidence of chemical radiculopathy
Pain Physician, 5 (3) (2002), pp. 260-265
Crossref ↗ View in Scopus ↗ Google Scholar ↗

70 Tenny S, Gillis CC. Annular disc tear. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2024. Updated August 7, 2023.
Google Scholar ↗

71 Kobayashi S., Meir A., Kokubo Y., Uchida K., Takeno K., Miyazaki T., *et al.*

Ultrastructural changes in lumbar disc herniation using surgical specimens: role of neovascularization and macrophages in hernias

Spine (Phila Pa 1976), 34 (7) (2009), pp. 655-662, 10.1097/BRS.0b013e31819c9d5 ↗

View in Scopus ↗ Google Scholar ↗

72 Hoffberg H.

Whiplash injuries in motor vehicle accidents

Pract Pain Manag, 2 (5) (2002)

https://www.medcentral.com/pain/chronic/whiplash-injuries-motor-vehicle-accidents ↗, Accessed 19th Feb 2026

Google Scholar ↗

73 Li F., Liu N., Liu H., Zhang B., Tian S., Tan M., Sandoz B.

A review of neck injury and protection in vehicle accidents

Transp Saf Environ, 1 (2) (2019), pp. 89-105, 10.1093/tse/tdz012 ↗

View in Scopus ↗ Google Scholar ↗

74 Petersohn JD, Padmanabhan G, Desai MJ. Whiplash associated disorder and cervical facet pain. In: Diwan S, Deer TR, eds. Advanced Procedures for Pain Management: A Step-by-Step Atlas. New York, NY: Springer; 2015:247-257.

Google Scholar ↗

75 Hefti F.L., Kress A., Fasel J., Morscher E.W.

Healing of the transected anterior cruciate ligament in the rabbit

J Bone Joint Surg Am, 73 (3) (1991), pp. 373-383

Crossref ↗ View in Scopus ↗ Google Scholar ↗

76 Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine

Definition of mild traumatic brain injury

J Head Trauma Rehabil, 8 (3) (1993), pp. 86-87

Google Scholar ↗

77 De Luigi A.J., Bell K.R., Bramhall J.P., Choe M., Dec K., Finnoff J.T., Halstead M., Herring S.A., Matuszak J., Raksin P.B., Swanson J., Millett C.

Consensus statement: an evidence-based review of exercise, rehabilitation, rest, and return to activity protocols for the treatment of concussion and mild traumatic brain injury

PM R (2023), 10.1002/pmrj.13070 ↗

Google Scholar ↗

78 National Center for Injury Prevention and Control, Division of Unintentional Injury Prevention. Report to Congress on Traumatic Brain Injury in the United States: Epidemiology and Rehabilitation. Atlanta, GA: Centers for Disease Control and Prevention; 2015. Accessed February/19.2026. https://stacks.cdc.gov/view/cdc/29215 ↗

Google Scholar ↗

79 Malec J.F., Brown A.W., Leibson C.L., Flaada J.T., Mandrekar J.N., Diehl N.N., Perkins P.K.
The Mayo Classification System for traumatic brain injury severity
J Neurotrauma, 24 (9) (2007), pp. 1417-1424, 10.1089/neu.2006.0245 ↗

View in Scopus ↗ Google Scholar ↗

80 De Luigi A.J., Bell K.R., Bramhall J.P., Choe M., Dec K., Finnoff J.T., Halstead M., Herring S.A., Matuszak J., Raksin P.B., Swanson J., Millett C.
Consensus statement: an evidence-based review of exercise, rehabilitation, rest, and return to activity protocols for the treatment of concussion and mild traumatic brain injury
PM R (2023), 10.1002/pmrj.13070 ↗

Google Scholar ↗

81 Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine
Definition of mild traumatic brain injury
J Head Trauma Rehabil, 8 (3) (1993), pp. 86-87

Google Scholar ↗

82 Silverberg N.D., Iverson G.L., ACRM Brain Injury Special Interest Group
The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury
Arch Phys Med Rehabil, 104 (8) (2023), pp. 1343-1355, 10.1016/j.apmr.2023.03.036 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

83 National Academies of Sciences, Engineering, and Medicine, Health and Medicine Division, Board on Health Care Services, Committee on the Review of the Department of Veterans Affairs Examinations for Traumatic Brain Injury. Evaluation of the Disability Determination Process for Traumatic Brain Injury in Veterans. Washington, DC: National Academies Press; 2019.

Google Scholar ↗

84 Mullally W.J.
Concussion
Am J Med., 130 (8) (2017), pp. 885-892, 10.1016/j.amjmed.2017.04.016 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

85 Ruff R.M., Iverson G.L., Barth J.T., Bush S.S., Broshek D.K., NAN Policy and Planning Committee
Recommendations for diagnosing a mild traumatic brain injury: a National Academy of Neuropsychology education paper
Arch Clin Neuropsychol, 24 (1) (2009), pp. 3-10, 10.1093/arclin/acp006 ↗
View in Scopus ↗ Google Scholar ↗

86 Ruff R.M., Iverson G.L., Barth J.T., Bush S.S., Broshek D.K., NAN Policy and Planning Committee
Recommendations for diagnosing a mild traumatic brain injury: a National Academy of Neuropsychology education paper
Arch Clin Neuropsychol, 24 (1) (2009), pp. 3-10, 10.1093/arclin/acp006 ↗
View in Scopus ↗ Google Scholar ↗

87 Madhok D.Y., Rodriguez R.M., Barber J., *et al.*
Outcomes in Patients With Mild Traumatic Brain Injury Without Acute Intracranial Traumatic Injury
JAMA Netw Open, 5 (8) (2022), Article e2223245, 10.1001/jamanetworkopen.2022.23245 ↗
Google Scholar ↗

88 Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine
Definition of mild traumatic brain injury
J Head Trauma Rehabil, 8 (3) (1993), pp. 86-87
Google Scholar ↗

89 De Luigi A.J., Bell K.R., Bramhall J.P., Choe M., Dec K., Finnoff J.T., Halstead M., Herring S.A., Matuszak J., Raksin P.B., Swanson J., Millett C.
Consensus statement: an evidence-based review of exercise, rehabilitation, rest, and return to activity protocols for the treatment of concussion and mild traumatic brain injury
PM R (2023), 10.1002/pmrj.13070 ↗
Google Scholar ↗

90 Bazarian J.J., Cernak I., Noble-Haeusslein L., Potolicchio S., Temkin N.

Long-term neurologic outcomes after traumatic brain injury
J Head Trauma Rehabil, 24 (6) (2009), pp. 439-451, 10.1097/HTR.0b013e3181c15600 ↗
View in Scopus ↗ Google Scholar ↗

91 Centers for Disease Control and Prevention. Report to Congress on Traumatic Brain Injury in the United States: Epidemiology and Rehabilitation. Atlanta, GA: Centers for Disease Control and Prevention; 2015.
Google Scholar ↗

92 Cleveland Clinic. Concussion and mild traumatic brain injury (mTBI). Accessed February/19/2026. https://my.clevelandclinic.org/health/diseases/15038-concussion ↗.
Google Scholar ↗

93 Carney N., Chesnut R.M., Maynard H., Mann N.C., Patterson P., Helfand M.
Effect of cognitive rehabilitation on outcomes for persons with traumatic brain injury: a systematic review
J Head Trauma Rehabil, 14 (3) (1999), pp. 277-307, 10.1097/00001199-199906000-00008 ↗
View in Scopus ↗ Google Scholar ↗

94 Fuji M., Ahmed A.O., Compton M.T.
Psychosis secondary to traumatic brain injury
Psychiatry Res, 215 (1) (2014), pp. 95-98
h ↗
t ↗
tps://doi.org/10.1176/ ↗
jnp.13.1.61 ↗
Google Scholar ↗

95 Ruff R.M., Iverson G.L., Barth J.T., Bush S.S., Broshek D.K., NAN Policy and Planning Committee
Recommendations for diagnosing a mild traumatic brain injury: a National Academy of Neuropsychology education paper
Arch Clin Neuropsychol, 24 (1) (2009), pp. 3-10, 10.1093/arclin/acp006 ↗
View in Scopus ↗ Google Scholar ↗

96 Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine
Definition of mild traumatic brain injury
J Head Trauma Rehabil, 8 (3) (1993), pp. 86-87
Google Scholar ↗

97 Peixoto H., Müller F., Werling S., *et al.*
Mild traumatic brain injury is significantly underdiagnosed in patients with severe multiple trauma
Brain Inj, 30 (4) (2016), pp. 416-423
Google Scholar ↗

98 Marco C.A., Snoad TBL, Poisson C., Flamm A.
Delayed Diagnosis of Intracranial Trauma
Cureus, 15 (10) (2023 Oct 26), Article e47738, 10.7759/cureus.47738 ↗
Google Scholar ↗

99 Pozzato I., Meares S., Kifley A., Craig A., Gillett M., Van Vu K., Liang R., Osborn M., Marsh J., Glozier N.
Challenges in the acute identification of mild traumatic brain injuries: results from an emergency department surveillance study
BMJ Open, 10 (2) (2020 Feb 3), Article e034494, 10.1136/bmjopen-2019-034494 ↗
PMID: 32019818; PMCID: PMC7045002
View in Scopus ↗ Google Scholar ↗

100 Koenigsberg R.A.
Imaging of traumatic brain injury
Neuroimaging Clin N Am, 28 (3) (2018), pp. 407-426
Google Scholar ↗

101 Chung S., Cohen A.D., Parker D., *et al.*
Non-hemorrhagic traumatic axonal injury: conventional imaging and advanced diffusion-weighted imaging
J Neurotrauma, 30 (10) (2013), pp. 846-854
Google Scholar ↗

102 Jolly A.E., Bălăeţ M., Azor A., Friedland D., Sandrone S., Graham NSN, Zimmerman K., Sharp D.J.
Detecting axonal injury in individual patients after traumatic brain injury
Brain, 144 (1) (2021), pp. 92-113, 10.1093/brain/awaa372 ↗
View in Scopus ↗ Google Scholar ↗

103 Dahl J., Tenovuo O., Posti J.P., Hirvonen J., Katila A.J., Frantzén J., Maanpää H.R., Takala R., Löyttyniemi E., Tallus J., Newcombe V., Menon D.K., Hutchinson P.J., Mohammadian M.

Cerebral microbleeds and structural white matter integrity in patients with traumatic brain injury: a diffusion tensor imaging study

Front Neurol, 13 (2022), Article 888815, 10.3389/fneur.2022.888815 ↗

View in Scopus ↗ Google Scholar ↗

104 Messori A., Polonara G., Mabiglia C., Salvolini U.

Is haemosiderin visible indefinitely on gradient-echo MRI following traumatic intracerebral haemorrhage?

Neuroradiology, 45 (12) (2003 Dec), pp. 881-886, 10.1007/s00234-003-1048-3 ↗

View in Scopus ↗ Google Scholar ↗

105 Jolly A.E., Bălăeţ M., Azor A., Friedland D., Sandrone S., Graham NSN, Zimmerman K., Sharp D.J.

Detecting axonal injury in individual patients after traumatic brain injury

Brain, 144 (1) (2021 Feb 12), pp. 92-113, 10.1093/brain/awaa372 ↗

View in Scopus ↗ Google Scholar ↗

106 Bruggeman R., Saleh A., Roberts D.

Non-hemorrhagic traumatic axonal injury

Radiology, 292 (2) (2019), pp. 371-382

Google Scholar ↗

107 Wardlaw J.M., Statham P.F.

How often is haemosiderin not visible on routine MRI following traumatic intracerebral haemorrhage?

Neuroradiology, 42 (2) (2000 Feb), pp. 81-84

View in Scopus ↗ Google Scholar ↗

108 Kinnunen K.M., Greenwood R., Powell J.H., Leech R., Hawkins P.C., Bonnelle V., Patel M.C., Counsell S.J., Sharp D.J.

White matter damage and cognitive impairment after traumatic brain injury

Brain, 134 (Pt 2) (2011), pp. 449-463, 10.1093/brain/awq347 ↗

View in Scopus ↗ Google Scholar ↗

109 Hellström T., Westlye L.T., Kaufmann T., Trung Doan N., Søberg H.L., Sigurdardottir S., Nordhøy W., Helseth E., Andreassen O.A., Andelic N.

White matter microstructure is associated with functional, cognitive and emotional symptoms 12 months after mild traumatic brain injury

Sci Rep, 7 (1) (2017), Article 13795, 10.1038/s41598-017-13628-1 ↗

View in Scopus ↗ Google Scholar ↗

110 MacKenzie J.D., Siddiqi F., Babb J.S., Bagley L.J., Mannon L.J., Sinson G.P., Grossman R.I.
Brain atrophy in mild or moderate traumatic brain injury: a longitudinal quantitative analysis
AJNR Am J Neuroradiol, 23 (9) (2002), pp. 1509-1515

View in Scopus ↗ Google Scholar ↗

111 Patel J.B., Wilson S.H., Oakes T.R., Santhanam P., Weaver L.K.
Structural and volumetric brain MRI findings in mild traumatic brain injury
AJNR Am J Neuroradiol, 41 (1) (2020), pp. 92-99, 10.3174/ajnr.A6346 ↗

View in Scopus ↗ Google Scholar ↗

112 Reber J., Hwang K., Bowren M., Bruss J., Mukherjee P., Tranel D., Boes A.D.
Cognitive impairment after focal brain lesions is better predicted by damage to structural than functional network hubs
Proc Natl Acad Sci U S A., 118 (19) (2021), Article e2018784118, 10.1073/pnas.2018784118 ↗

View in Scopus ↗ Google Scholar ↗

113 Gale S.D., Baxter L., Roundy N., Johnson S.C.
Traumatic brain injury and grey matter concentration: a preliminary voxel-based morphometry study
J Neurol Neurosurg Psychiatry, 76 (7) (2005), pp. 984-988, 10.1136/jnnp.2004.036210 ↗

View in Scopus ↗ Google Scholar ↗

114 Hellström T., Westlye L.T., Kaufmann T., Trung Doan N., Søberg H.L., Sigurdardottir S., Nordhøy W., Helseth E., Andreassen O.A., Andelic N.
White matter microstructure is associated with functional, cognitive and emotional symptoms 12 months after mild traumatic brain injury
Sci Rep, 7 (1) (2017), Article 13795, 10.1038/s41598-017-13628-1 ↗

View in Scopus ↗ Google Scholar ↗

115 Patel J.B., Wilson S.H., Oakes T.R., Santhanam P., Weaver L.K.
Structural and volumetric brain MRI findings in mild traumatic brain injury
AJNR Am J Neuroradiol, 41 (1) (2020), pp. 92-99, 10.3174/ajnr.A6346 ↗

View in Scopus ↗ Google Scholar ↗

116 Reber J., Hwang K., Bowren M., Bruss J., Mukherjee P., Tranel D., Boes A.D.

Cognitive impairment after focal brain lesions is better predicted by damage to structural than functional network hubs

Proc Natl Acad Sci U S A., 118 (19) (2021), Article e2018784118, 10.1073/pnas.2018784118 ↗

View in Scopus ↗ Google Scholar ↗

117 Hunter Oregon K., M.D.

Evaluation of the Scientific Validity of Forensic Medical Evaluations

American Journal of Physical Medicine & Rehabilitation, 99 (3) (March 2020), Article a304 (Supplement), Abstracts of Scientific Papers and Posters Presented at the ISPRM World Congress and Annual Meeting of the Association of Academic Physiatrists, Orlando, Florida, March 4–9, 2020

Google Scholar ↗

118 Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine

Definition of mild traumatic brain injury

J Head Trauma Rehabil, 8 (3) (1993), pp. 6-87

Google Scholar ↗

119 Ruff R.M., Iverson G.L., Barth J.T., Bush S.S., Broshek D.K., NAN Policy and Planning Committee

Recommendations for diagnosing a mild traumatic brain injury: a National Academy of Neuropsychology education paper

Arch Clin Neuropsychol, 24 (1) (2009), pp. 3-10, 10.1093/arclin/acp006 ↗

View in Scopus ↗ Google Scholar ↗

120 Rauchman S.H., Zubair A., Jacob B., Rauchman D., Pinkhasov A., Placantonakis D.G., Reiss A.B.

Traumatic brain injury: mechanisms, manifestations, and visual sequelae

Front Neurosci, 17 (2023), Article 1090672, 10.3389/fnins.2023.1090672 ↗

View in Scopus ↗ Google Scholar ↗

121 Mutch C.A., Tecklenburg K., Fallen T., *et al.*

Defining moderate traumatic brain injury: a systematic review and decision tree

J Neurotrauma, 33 (5) (2016), pp. 416-423

Google Scholar ↗

122 Carroll L.J., Cassidy J.D., Holm L., Kraus J., Coronado V.G.

WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. Methodological issues and research recommendations for mild traumatic

brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury

J Rehabil Med (43 Suppl) (2004), pp. 113-125, 10.1080/16501960410023877 ↗

View in Scopus ↗ Google Scholar ↗

123 Harris T.C., de Rooij R., Kuhl E.

The Shrinking Brain: Cerebral Atrophy Following Traumatic Brain Injury

Ann Biomed Eng, 47 (9) (2019 Sep), pp. 1941-1959

Crossref ↗ View in Scopus ↗ Google Scholar ↗

124 McInnes K., Friesen C.L., MacKenzie D.E., Westwood D.A., Boe S.G.

Mild traumatic brain injury (mTBI) and chronic cognitive impairment: a scoping review

PLoS One, 12 (4) (2017), Article e0174847, 10.1371/journal.pone.0174847 ↗

View in Scopus ↗ Google Scholar ↗

125 Nelson L.D., Temkin N.R., Dikmen S., *et al.*

Recovery After Mild Traumatic Brain Injury in Patients Presenting to US Level I Trauma Centers: A Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) Study

JAMA Neurol, 76 (9) (2019), pp. 1049-1059

Crossref ↗ View in Scopus ↗ Google Scholar ↗

126 Agrawal A., Timothy J., Pandit L., Manju M.

Post-traumatic epilepsy: an overview

Clin Neurol Neurosurg, 108 (5) (2006), pp. 433-439, 10.1016/j.clineuro.2005.09.001 ↗

View PDF View article View in Scopus ↗ Google Scholar ↗

127 Lu Y.C., Wu M.K., Zhang L., Zhang C.L., Lu Y.Y., Wu C.H.

Association between suicide risk and traumatic brain injury in adults: a population based cohort study

Postgrad Med J, 96 (1142) (2021 Feb), pp. 747-752

Crossref ↗ Google Scholar ↗

128 Bazarian J.J., Cernak I., Noble-Haeusslein L., Potolicchio S., Temkin N.

Long-term neurologic outcomes after traumatic brain injury

J Head Trauma Rehabil, 24 (6) (2009 Nov-Dec), pp. 439-451, 10.1097/HTR.0b013e3181c15600 ↗

PMID: 19940677

View in Scopus ↗ Google Scholar ↗

129 Singh R., Dawson J., Mason P.S., Lecky F.
What are the functional consequences after TBI? The SHEFBIT cohort experience
Brain Inj, 35 (12-13) (2021), pp. 1630-1636, 10.1080/02699052.2021.1978549 ↗
View in Scopus ↗ Google Scholar ↗

130 Sendroy-Terrill M., Whiteneck G.G., Brooks C.A.
Aging with traumatic brain injury: cross-sectional follow-up of people receiving inpatient rehabilitation over more than 3 decades
Arch Phys Med Rehabil, 91 (3) (2010), pp. 489-497, 10.1016/j.apmr.2009.11.011 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

131 Rauchman S.H., Zubair A., Jacob B., Rauchman D., Pinkhasov A., Placantonakis D.G., Reiss A.B.
Traumatic brain injury: mechanisms, manifestations, and visual sequelae
Front Neurosci, 17 (2023), Article 1090672, 10.3389/fnins.2023.1090672 ↗
View in Scopus ↗ Google Scholar ↗

132 Williams D.H., Levin H.S., Eisenberg H.M.
Mild head injury classification
Neurosurgery, 27 (3) (1990), pp. 422-428, 10.1097/00006123-199009000-00014 ↗
View in Scopus ↗ Google Scholar ↗

133 Barnes D.E., Byers A.L., Gardner R.C., Seal K.H., Boscardin W.J., Yaffe K.
Association of mild traumatic brain injury with and without loss of consciousness with dementia in US military veterans
JAMA Neurol, 75 (9) (2018), pp. 1055-1061, 10.1001/jamaneurol.2018.0815 ↗
View in Scopus ↗ Google Scholar ↗

134 LoBue C., Wilmoth K., Cullum C.M., Rossetti H.C., Lacritz L.H., Hynan L.S., Hart J.Jr, Womack K.B.
Traumatic brain injury history is associated with earlier age of onset in frontotemporal dementia
J Neurol Neurosurg Psychiatry, 87 (8) (2016), pp. 817-820, 10.1136/jnnp-2015-311438 ↗
View in Scopus ↗ Google Scholar ↗

135 Johnson V.E., Stewart W., Smith D.H.
Traumatic brain injury and amyloid-β pathology: a link to Alzheimer's disease?
Nat Rev Neurosci, 11 (5) (2010), pp. 361-370, 10.1038/nrn2808 ↗

View in Scopus ↗ Google Scholar ↗

136 Schneider E.B., Daugherty J., Simmons S.O., *et al.*
Traumatic brain injury management and research: is there a standard of care?
Clin Neurosurg, 79 (2016), pp. S207-S211
Google Scholar ↗

137 Fakhran S., Yaeger K., Collins J., *et al.*
Acute white matter abnormalities provide missed opportunities for traumatic axonal injury diagnosis in mild traumatic brain injury
AJNR Am J Neuroradiol, 35 (12) (2014 Dec), pp. 2331-2337
Google Scholar ↗

138 Sendroy-Terrill M., Whiteneck G.G., Brooks C.A.
Aging with traumatic brain injury: cross-sectional follow-up of people receiving inpatient rehabilitation over more than 3 decades
Arch Phys Med Rehabil, 91 (3) (2010), pp. 489-497, 10.1016/j.apmr.2009.11.011 ↗
View PDF View article View in Scopus ↗ Google Scholar ↗

Cited by (0)