YOUR BRAIN AFTER IMPACT

DIAGNOSIS, OBJECTIVE ASSESSMENT, AND CONSEQUENCES OF THE INJURY THE SCAN DOES NOT SHOW

A PRIMER ON MILD TRAUMATIC BRAIN INJURY
AND POST-CONCUSSION SYNDROME



BY
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AND
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Your Brain After Impact

A primer on mild traumatic brain injury and post-concussion syndrome

Discover the main problems in concussion diagnosis and life-changing expert advice for a stronger recovery

Mike Pappolla, MD, PhD, and Remi Nader, MD

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Endorsements

“An essential read that demystifies concussion science and provides practical strategies for recovery. I highly recommend it to patients and professionals alike.”

Ruben Vidal, PhD, Professor of Neuroscience, Indiana University.

“A compelling, user-friendly guide that bridges clinical expertise with real-life applications. This work fills a critical gap in concussion education.”

Xiang Fang, MD, PhD, Professor of Neurology and John Sealy Distinguished Chair for Clinical Research in Alzheimer’s Disease, University of Texas Medical Branch.

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About this book

Every year, millions of people sustain a concussion, the injury physicians call mild traumatic brain injury, and most of them are sent home with little understanding of what has happened inside their heads. The scan is normal. The examination is normal. The symptoms are not. Headache, dizziness, foggy thinking, intolerance of noise and screens, a memory that no longer holds, a temper that has become unfamiliar: these are the marks of an injury that routine testing was never designed to see.

This book explains that injury from the inside. It sets out how a concussion is diagnosed and why the diagnosis is so often missed. It explains how severity is graded and what the word mild does and does not mean. It also shows how objective testing of eye movements, balance, brain electrical activity, and brain structure can measure what the ordinary examination cannot. It then follows the injury into the lives of patients: the persistent symptoms, the changes in thinking and mood, the pain that spreads beyond the place that was struck, and the treatments that help. Throughout, real clinical situations and plain explanations show how a brain that performs well in a quiet examination room can fail under the demands of work, school, and family life, and what can be done about it.

Written by a neurologist and a neurosurgeon who have spent their careers with these patients, this book serves several audiences. They include medical students, physicians and therapists outside neurology who see these patients first and most often, and attorneys and other professionals who must weigh the evidence when an invisible injury is in dispute. It shows how the injury is recognized, what the tests can and cannot establish, and what a sound evaluation and a sound plan of treatment look like.

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Introduction

This book brings together the science of traumatic brain injury, our own peer-reviewed research, and our experience caring for people affected by these injuries. The clinical judgments offered here grow out of that combination: questions encountered in practice, findings from our studies, and careful consideration of the work of other investigators. An appendix introduces six papers central to these discussions, with their published abstracts and links to the complete open-access articles. Readers can follow the evidence and reasoning behind our interpretations and judge their strengths and limits for themselves.

Concussion is a common brain injury and one that is often misunderstood by patients, families, and the clinicians who see them first. One source of misunderstanding is the gap between what routine testing shows and what the patient experiences. CT and conventional MRI are valuable for detecting bleeding and other structural injuries, but they may not reveal the changes underlying a concussion. A person can leave an emergency department with a normal scan, a discharge diagnosis of cervical strain, and a brain that no longer works the way it did that morning. The scan can be reassuring without explaining everything that has changed.

This book is about closing that gap. It is organized in four parts, followed by a closing chapter.

Part I is about diagnosis. It explains what traumatic brain injury is, how common it is, why official figures undercount it, and how mild traumatic brain injury is diagnosed under the 2023 criteria of the American Congress of Rehabilitation Medicine. Those criteria recognize different routes to diagnosis, which the chapters explain in detail. This part also examines how severity is graded, why the same injury can carry different labels in different records, why clinicians can miss mild injuries in acute care, and how clinicians can reconstruct the evidence weeks or months later. It closes with the clinical profiles that guide treatment.

Part II is about evaluation and measurement. The first chapter explains objective testing: what eye-movement recordings, balance testing, evoked potentials, quantitative electroencephalography, autonomic testing, computerized cognitive testing, and blood biomarkers measure; where they fit within diagnostic criteria; and how to interpret their results. The second is devoted to imaging, from the emergency department CT scan to diffusion tensor imaging of the brain's wiring. The third explains the cognitive examination: what each cognitive domain means in everyday life, how it is tested, and what the diagnostic labels of mild cognitive impairment and neurocognitive

disorder do and do not say. It also explains neurologists' training and capabilities in cognitive and neuropsychological assessment, along with the additional depth clinical neuropsychologists bring to specialized psychometrics and testing.

Part III is about consequences. It takes up persistent symptoms and the disagreement among the definitions used to describe them; the cognitive, emotional, and behavioral changes that patients and families live with; and pain, a frequent and often misunderstood source of lasting disability after brain injury.

Part IV is about treatment. It begins with the acute period, when some patients with mild or moderate injuries deteriorate and may need monitoring or surgery. It then turns to persistent symptoms: what helps, in what order, and what the evidence says about treatments that work, those that do not, and those whose benefits remain uncertain. A closing chapter follows one patient through the whole sequence, from the roadside to the treatment plan, and shows what a complete evaluation contains.

Two ideas run through every chapter. The first is that each test provides a keyhole view of the nervous system. A normal view through one keyhole does not establish that the rest of the house is in order, any more than an abnormal view, by itself, tells us what caused the damage. The second is the difference between rest and load. A patient with chronic lung disease may have a normal oxygen level on the couch and become breathless going upstairs. The couch reading is accurate, but it does not tell the whole story. Much testing of the injured brain takes place in similarly quiet conditions, one task at a time. Many difficulties emerge under divided attention, noise, time pressure, and fatigue. Much of what follows is about how to test the stairs.

The book is written for medical students, physicians outside neurology, attorneys, and other professionals who evaluate these injuries. It assumes no specialist training. The chapters can be read in order or consulted separately, and each ends with a short summary of its main points. References are numbered within each chapter.

Each paper in the appendix opens with a plain-language introduction that explains the question it addresses, its relevance to practice, and what readers should look for in the original. A panel then lists the title, authors, and the complete published abstract, followed by a link to the full article. The introductions clarify what each study or review contributes and what it cannot establish.

Appendix A introduces six research papers discussed throughout the book. Appendix B provides structured intake and diagnostic worksheets to accompany Chapter 5, with links to the official ACRM interview materials. Appendix C summarizes the ACRM criteria and the 2025 clarification of their retrospective and medicolegal use.

Part I. Diagnosis and classification

Chapter 1 An overview of traumatic brain injury

Chapter overview

Traumatic brain injury occurs when an external mechanical force disrupts brain function. It may result from a fall, a motor vehicle crash, an assault, a blast, a sports collision, or an impact from a moving object, and it may be closed or penetrating. The injury is graded as mild, moderate, or severe, but the term mild refers to the initial clinical presentation rather than the eventual burden. A patient who meets emergency department criteria for a mild injury may still develop disabling headache, dizziness, cognitive slowing, emotional dysregulation, sleep disturbance, visual intolerance, imbalance, and difficulty returning to work and family life [1].

This chapter introduces the injury, its scale and mechanisms, and the difficulty that occupies much of this book: a brain injury may be entirely real yet entirely invisible to routine testing. Standard neurological examination, computed tomography, and conventional magnetic resonance imaging may all be unremarkable in a patient whose brain no longer performs under the demands of ordinary life. Understanding why that happens and what to do about it is the work of the chapters that follow.

What traumatic brain injury is

Traumatic brain injury results from external biomechanical energy transferred to the head or body. The force may arrive as a direct blow, rapid acceleration and deceleration without direct impact, rotational shear, a pressure wave, or penetrating trauma. In penetrating brain injury, an object breaches the protective coverings and enters brain tissue. Nonpenetrating injury occurs when the brain is stretched, compressed, or rotated within the skull; a skull fracture alone does not indicate that the brain was penetrated.

The broader term head injury is less specific and less useful. It may refer to a scalp laceration, a skull fracture, or a brain injury. A person can have a head injury without brain dysfunction, and a brain injury can occur without any dramatic external sign. The distinction matters because a clean scan, an intact scalp, and the absence of description during a brief emergency department visit do not establish that the brain escaped injury.

Why the word mild is misleading

Clinicians assign severity categories of mild, moderate, and severe based on the early clinical presentation. These categories guide acute assessment and immediate

treatment considerations during the early post-injury period, but they do not accurately predict long-term consequences. They use the duration of loss of consciousness, the duration of altered mental status, the duration of post-traumatic amnesia, and the Glasgow Coma Scale score. Chapter 4 explains how these are applied and where the frameworks disagree.

What the categories do not describe is the long-term burden. A patient classified as mild may experience persistent headache, dizziness, visual motion sensitivity, sensitivity to light (photophobia), sensitivity to sound (phonophobia), sleep disruption, fatigue, slowed information processing, memory deficits, irritability, anxiety, depression, and reduced tolerance for cognitively demanding work. Expert groups have said repeatedly that traumatic brain injury is heterogeneous and needs more than a single label to be understood [1]. The severity grade is a starting point for care, not a prediction of outcome.

How common it is

Traumatic brain injury is among the most common serious neurological conditions in the United States. For the period 2002 to 2006, the Centers for Disease Control and Prevention estimated an average of about 1.7 million traumatic brain injury-related emergency department visits, hospitalizations, and deaths annually, comprising about 1,365,000 emergency department visits, 275,000 hospitalizations, and 52,000 deaths [4]. Later surveillance reported approximately 2.87 million such events in 2014 [5]. The CDC data summary available in October 2026 separately reported approximately 214,110 hospitalizations in 2020 and 68,663 deaths in 2023 [6].

Every one of these figures is an undercount, and the different reports are not directly comparable. Surveillance captures what is documented and coded, and mild injuries are often treated outside hospitals, incompletely documented, or not evaluated at all [2,3]. Chapter 2 explains why the numbers differ and what they leave out.

The cost is not measured only in visits and deaths. Direct medical costs and lost productivity represent a substantial economic burden [7]. Behind those costs stand long-term disability and the demands placed on caregivers. The injury can affect behavior, personality, social function, employment, education, relationships, sleep, mood, pain regulation, cognition, and independence. None of that personal burden appears in a surveillance table.

Mechanism matters

The mechanism of injury shapes the injury pattern, the associated extracranial trauma, and the rehabilitation needs.

Falls accounted for the greatest number of traumatic brain injury-related emergency department visits and hospitalizations in the 2002 to 2006 surveillance report, with a substantial burden among young children and older adults [4]. In that report, motor vehicle traffic was the leading reported cause of traumatic brain injury-related death, with the highest motor vehicle-related death rates among young adults [4]. Rankings vary by period and classification: by 2014, intentional self-harm accounted for the largest share of traumatic brain injury-related deaths, followed by unintentional falls and motor vehicle crashes [5]. Road traffic injury remains an important international public health problem [8]. Other major mechanisms include being struck by or against an object, assault, and self-inflicted injury.

Two points follow. Prevention must be mechanism-specific. Preventing fall-related injuries in older adults requires different measures than preventing firearm-related deaths. In clinical practice, the mechanism is the first element of the diagnostic assessment: every major diagnostic framework requires a biomechanically plausible mechanism before any clinical finding can be attributed to brain injury.

The injury that routine testing may not show

The most difficult aspect of this injury is that it can be obvious to the patient yet invisible to standard evaluation. Neurological examination, computed tomography, and conventional magnetic resonance imaging may all fail to identify functionally important disturbance, particularly after mild injury. This does not make the symptoms imaginary. It means the tools used in routine settings are often not sensitive to the relevant dysfunction.

Research imaging can reveal some of what routine tools miss. Diffusion tensor imaging studies have identified white matter microstructural differences following mild traumatic brain injury. The number of regions with abnormal diffusion measurements has been associated with impaired reaction time [9]. Attentional control and memory have been linked to distinct white matter pathways [10], and a small adolescent study detected acute diffusion changes despite negative computed tomography [11]. These are group findings, and an isolated diffusion abnormality in one patient is not specific to trauma; Chapter 8 discusses what diffusion imaging can and cannot establish. Clinical assessment must also consider the demands of ordinary life, including noise, stress, visual motion, multitasking, fatigue, pain, emotional load, and time pressure.

Clinicians naturally focus on the visible consequences of trauma: fractures, weakness, sensory loss, and bleeding. Subtle cognitive, vestibular, emotional, visual, sleep-related, and behavioral changes are then overlooked, and patients are sometimes told their symptoms are psychological, stress-related, age-related, or unrelated to the injury. Psychological symptoms may indeed be present. Depression, anxiety, irritability, post-traumatic stress, insomnia, and cognitive fatigue can coexist with a brain injury, be consequences of it, or result from the life disruption that follows it. None of them excludes the injury.

The diagnostic question this book asks

The diagnostic challenge is not limited to deciding whether a person is injured. It also involves determining whether the mechanism, symptoms, history, examination, neurophysiology, cognitive performance, and functional limitations form a coherent pattern.

The framework for answering that question changed in 2023, when the American Congress of Rehabilitation Medicine published revised diagnostic criteria. After a biomechanically plausible mechanism, the criteria allow diagnosis based on 1 or more qualifying clinical signs; at least 2 qualifying acute symptoms plus at least one appropriate laboratory finding; or unambiguous trauma-related intracranial abnormalities on computed tomography or structural magnetic resonance imaging [12]. The absence of a contemporaneous record does not end the inquiry. Chapter 3 sets the criteria out in full.

A useful way to keep the whole problem in mind is to recognize that a traumatic brain injury evaluation should not stop at the question of whether the scan is normal. The better question is whether this person's brain still functions normally under the demands of real life.

A clinical illustration: Ana

The following case is constructed from patterns that recur in clinical practice and is not an account of any individual patient.

Ana worked as a certified court interpreter, translating between Spanish and English in real time. The work is more demanding than it appears. An interpreter holds one sentence in working memory in one language while producing the preceding sentence in another, tracks legal terminology, monitors register and tone, and does all of this continuously for hours without pausing the proceedings. Ana had done this for eleven years and was known for being unflappable.

She was hanging curtains at home when the step stool shifted. She fell backward, struck the back of her head on the door frame, and sat on the floor for a minute or two, feeling, as she put it later, scrambled. She did not lose consciousness. Her husband asked whether she was all right, and she said she was. That evening, she had a headache and took acetaminophen. She did not seek care, and no medical record of the event exists.

She returned to work four days later. For the first hour, she was fine. By mid-morning, she noticed she was falling behind, holding the sentence in her head a fraction too long, then losing the end of it. She asked for a recess, something she had never done. Over the following weeks, she began declining the longer assignments. She could still translate a short exchange perfectly. What she could no longer do was sustain the simultaneous load, and simultaneity was the entire job.

Her physician examined her and found no abnormalities on the tests performed. Her cranial nerves were intact, her strength and reflexes were normal, her gait was normal, and she scored 30 of 30 on a bedside cognitive screen. She was told that the examination was reassuring and that stress and poor sleep were likely explanations. Both were present. Neither the examination nor the screen could say whether they explained her new difficulty.

Ana's case is the central problem of this book. She performed well on a brief, structured evaluation but failed during sustained, demanding work. A bedside cognitive screen is a cognitive task, but it is a ten-minute task in a quiet room, whereas her job required hours of simultaneous load. Her normal score did not resolve the discrepancy; it restated it. The next step would be a more detailed assessment of cognition, endurance, sleep, mood, pain, and the other contributors to her limitations.

Her case also shows why the diagnostic framework matters. No contemporaneous medical record documents the acute period, and no imaging was obtained. A careful interview would need to establish what she meant by scrambled and whether she experienced observable confusion, amnesia, or qualifying acute symptoms. The 2023 criteria allow the injury history to be reconstructed retrospectively [12], and that reconstruction, not the later testing, is what her diagnosis would rest on. The details given here warrant that assessment; they do not complete it.

Fast take: main points from chapter 1

- **What traumatic brain injury is.** An external mechanical force disrupts brain function. It may follow falls, motor vehicle crashes, assaults, sports injuries, blasts, or impacts from objects, and it may be closed or penetrating.

- **Direct head impact is not required.** Acceleration, deceleration, rotation, and blast overpressure can injure the brain without any blow to the head.

- **Mild describes the initial presentation, not the long term outcome.** A mild injury can produce persistent symptoms that disrupt work, relationships, mood, cognition, sleep, balance, vision, and daily function [1].

- **The injury is common and its true incidence is underestimated.** Approximately 1.7 million injuries annually in the 2002 to 2006 surveillance period [4], with later reporting substantially higher [5,6], and many cases treated outside hospital systems or never evaluated.

- **Mechanism matters.** Falls account for a large share of emergency department visits and hospitalizations; the leading fatal mechanism varies with the surveillance period [4,5]. Prevention and clinical assessment both start from the specific mechanism.

- **Standard testing may miss important dysfunction.** A normal computed tomography scan, conventional magnetic resonance imaging, or brief neurological examination does not exclude traumatic brain injury. Diffusion tensor imaging research has linked white matter measurements with reaction time [9], distinct attentional and memory functions [10], and acute changes in adolescents [11].

- **Psychological symptoms do not exclude brain injury.** Depression, anxiety, irritability, post-traumatic stress, insomnia, and emotional dysregulation may coexist with, worsen, or result from the injury.

- **Diagnosis has three routes.** The 2023 criteria permit diagnosis through qualifying clinical signs, at least two qualifying acute symptoms plus at least one appropriate acute examination or laboratory finding, or structural neuroimaging evidence [12]. Chapter 3 sets them out in full.

- **The right question is functional.** Beyond reading the scan, ask whether the brain still performs under the demands of the patient's actual life.

References

1. Saatman KE, Duhaime AC, Bullock R, Maas AI, Valadka A, Manley GT, et al. Classification of traumatic brain injury for targeted therapies. J Neurotrauma. 2008;25(7):719-738.
2. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375-378.
3. Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. J Head Trauma Rehabil. 2006;21(6):544-548.
4. Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002-2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.
5. Peterson AB, Xu L, Daugherty J, Breiding MJ. Surveillance Report of Traumatic Brain Injury-Related Emergency Department Visits, Hospitalizations, and Deaths, United States, 2014. Atlanta (GA): Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2019.
6. Centers for Disease Control and Prevention. TBI data [Internet]. Atlanta (GA): Centers for Disease Control and Prevention; 2026 Apr 27 [cited 2026 Oct 24]. Available from: https://www.cdc.gov/traumatic-brain-injury/data-research/index.html
7. Finkelstein EA, Corso PS, Miller TR. The Incidence and Economic Burden of Injuries in the United States. New York: Oxford University Press; 2006.
8. World Health Organization. Global Status Report on Road Safety: Time for Action. Geneva: World Health Organization; 2009.
9. Niogi SN, Mukherjee P, Ghajar J, Johnson C, Kolster R, Sarkar R, et al. Extent of microstructural white matter injury in postconcussive syndrome correlates with impaired cognitive reaction time: a 3T diffusion tensor imaging study of mild traumatic brain injury. AJNR Am J Neuroradiol. 2008;29(5):967-973.
10. Niogi SN, Mukherjee P, Ghajar J, Johnson C, Kolster R, Lee H, et al. Structural dissociation of attentional control and memory in adults with and without mild traumatic brain injury. Brain. 2008;131(Pt 12):3209-3221.
11. Wilde EA, McCauley SR, Hunter JV, Bigler ED, Chu Z, Wang ZJ, et al. Diffusion tensor imaging of acute mild traumatic brain injury in adolescents. Neurology. 2008;70(12):948-955.
12. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355.

Chapter 2 Causes and societal impact of traumatic brain injury

Chapter overview

Traumatic brain injury results from a wide range of events, including falls, motor vehicle crashes, blast exposure, industrial incidents, sports and recreational impacts, penetrating or firearm-related injuries, and assault. It is best understood as an alteration in brain function or other evidence of brain pathology caused by an external force. The mechanism may involve direct contact, rapid acceleration and deceleration, rotational forces, pressure waves, penetrating trauma, or a combination [1,2]. The biological consequences do not end at impact. Over the hours, days, and weeks that follow, secondary processes, including excitotoxicity, inflammation, oxidative stress, altered cerebral blood flow, edema, hypoxia, and metabolic dysfunction, can amplify the primary injury and contribute to persistent symptoms [3,4].

Although common and often described as a silent epidemic, traumatic brain injury remains underrecognized, particularly at the milder end. Many patients appear outwardly intact, have normal computed tomography or magnetic resonance imaging, and nonetheless experience headache, dizziness, visual or vestibular complaints, cognitive inefficiency, mood disturbance, fatigue, sleep disruption, and reduced tolerance for work and school demands. This chapter reviews how the injury occurs and why it is so often missed. It explains how surveillance limitations shape estimates of its frequency and why the personal, social, and economic burden is substantial. The 2023 revision of the diagnostic criteria, which addressed the same problems, is set out in full in Chapter 3 [5,6,7,8].

Understanding how traumatic brain injury happens

Injuries occur through many pathways. A person may fall from a ladder, slip on ice, strike the head on the ground, or sustain a sudden jolt without an external wound. Motor vehicle crashes may involve a direct blow, a whiplash-type acceleration-deceleration event, rotational shear, or a secondary collision with the vehicle interior. In military and civilian blast contexts, pressure waves can injure the brain even when the skull is not struck. Industrial work carries risks from falling debris, machinery impacts, and falls from elevation. Assaults, penetrating injuries, and sports collisions remain important contributors [2,9,10].

Traumatic brain injury is sometimes assumed to require a direct blow to the head or a clear loss of consciousness. That view is too narrow. The brain can be injured by rapid translation, rotation, or deformation within the skull. These forces can stretch or disrupt

axons, compromise white matter, and alter the function of entire networks. They can do so without leaving an external mark on the head. Blast exposure adds further complexity because the pressure wave may combine with blunt impact, acceleration, penetrating fragments, and systemic trauma [9,10].

The injury does not end at impact. Over the hours and days that follow, ionic shifts, glutamate-mediated excitotoxicity, altered glucose metabolism, mitochondrial dysfunction, inflammation, vascular dysregulation, blood-brain barrier disruption, edema, and hypoxic or ischemic stress can worsen the initial insult. This is why a patient who appears stable in the emergency department may report cognitive, vestibular, visual, affective, or sleep-related symptoms a day or two later [3,4].

Population-level patterns also shift over time. Historical mortality data showed progress in reducing deaths from motor vehicle-related injuries, alongside persistent difficulty in reducing deaths from penetrating mechanisms, including firearm-related injuries [11]. Prevention must therefore be mechanism-specific rather than delivered as a single public health message [2].

Table 2.1. Mechanisms of traumatic brain injury and their clinical implications.

Mechanism	How the brain is injured	Typical settings and populations	Clinical implications
Fall	Direct impact of the head against a surface, combined with acceleration and deceleration of the brain within the skull	Leading cause of TBI-related emergency department visits and hospitalizations in the 2002 to 2006 report. High burden in young children and older adults	Frequently unwitnessed, so the acute period is often undocumented. Syncope must be considered as a cause of the fall rather than only a consequence
Motor vehicles and other transport	Direct impact, rapid acceleration and deceleration without head contact, rotational shear, and secondary impact with the vehicle interior	Leading reported cause of TBI-related death in the 2002 to 2006 report, with the highest motor vehicle-related death rates in young adults. Later mortality rankings differ	Cervical injury coexists frequently and produces overlapping symptoms. Airbag non-deployment and minor vehicle damage do not exclude injury

Mechanism	How the brain is injured	Typical settings and populations	Clinical implications
Struck by or against an object	Direct contact force, usually focal, sometimes with a rotational component	Workplace, household, sport, and recreational settings	Commonly minimized by patient and clinician as trivial, particularly when there is no visible wound
Assault and interpersonal violence	Direct impact, sometimes repeated; strangulation may cause an additional hypoxic-ischemic brain injury	All settings. Intimate partner violence is substantially underrecognized	Documentation is often incomplete for reasons unrelated to medicine. Repetitive injury over time is common and rarely captured in a single encounter
Blast	Pressure wave injury, blunt impact from being thrown, penetrating fragments, and acceleration and deceleration, frequently in combination	Military deployment and civilian explosive events	Conventional imaging is often normal. Polytrauma and post-traumatic stress commonly coexist and complicate attribution
Sport and recreation	Repetitive acceleration and deceleration, rotational force, and direct impact	Contact and collision sport at all levels of play	Prior concussions and repeated exposure should be documented. Return-to-sport decisions require recognition of the injury, clinical assessment, and appropriate follow-up

Mechanism	How the brain is injured	Typical settings and populations	Clinical implications
Penetrating injury	An object penetrates the protective coverings and enters brain tissue	Firearm injury, industrial and occupational trauma, and high-velocity fragments	Requires urgent assessment of the wound trajectory and associated structural injury. Specific risks include infection, hemorrhage, seizure, vascular injury, and cerebrospinal fluid leak. Penetrating injury is outside the scope of the 2023 ACRM mild traumatic brain injury criteria

Table 2.1 legend. Mechanism shapes the injury pattern, associated extracranial trauma, and rehabilitation needs. Frequency statements refer to United States surveillance from 2002 to 2006 [5]; the leading causes of death have since changed [2,12].

Diagnosis: more than meets the eye

Clinicians rely on computed tomography and magnetic resonance imaging to identify hemorrhage, contusion, edema, skull fracture, and mass effect. These studies are essential in acute trauma care, where the question is whether the patient needs a neurosurgeon. They answer that question well. They do not settle the question of whether the brain was injured, least of all after a mild injury. Microscopic axonal injury, metabolic dysfunction, inflammatory change, and altered connectivity are not visible on routine clinical imaging, so a normal scan after a mild injury is sometimes interpreted as no injury, when the accurate conclusion is that no injury is visible on this test [7].

Mild traumatic brain injury is commonly called a concussion, particularly in sports medicine, and the 2023 diagnostic criteria confirm that the two labels may be used interchangeably when neuroimaging is normal or not clinically indicated [6]. The term mild refers to initial severity markers: Glasgow Coma Scale score, duration of loss or alteration of consciousness, post-traumatic amnesia, and the presence or absence of acute imaging abnormality. It does not mean clinically insignificant. Patients may experience persistent difficulty with attention, processing speed, memory, executive function, emotional regulation, headache, light sensitivity, dizziness, balance, visual

tracking, sleep, and fatigue, and some remain symptomatic for years despite normal imaging [6,8].

What the 2023 criteria changed

More accurate recognition was one stated reason for revising the diagnostic criteria. The 1993 definition rested on acute clinical signs and did not clearly separate what an observer sees from what a patient feels. The 2023 revision draws that line explicitly and provides three alternative routes to the diagnosis after a plausible mechanism: a qualifying clinical sign; at least two qualifying acute symptoms together with at least one appropriate acute examination or laboratory finding; or an unambiguous trauma-related abnormality on structural imaging, with a category of suspected mild traumatic brain injury for cases that fall short [6]. This matters when reviewing an individual record: the absence of an acute sign does not end the inquiry. Chapter 3 sets the criteria out in full, and Chapter 5 examines the diagnostic problems that remain.

Tools that may detect what routine imaging misses

Diffusion tensor imaging is examined in Chapter 8. Functional assessments include quantitative electroencephalography, evoked potentials, oculomotor testing, videonystagmography, and vestibular assessment. Each can characterize an aspect of brain or sensory-system function that structural imaging cannot [13,14,15,16]. None of these is a test for concussion, and the American Clinical Neurophysiology Society has concluded that the evidence does not support quantitative electroencephalography as a diagnostic test for mild traumatic brain injury [17]. What they can do is measure dysfunction that the examination and the scan did not show. They cannot, on their own, establish that the dysfunction came from the injury, since each test can be abnormal in other conditions as well. Chapter 7 explains what each of them measures and how the results should be read.

Incidence: a public health challenge

Incidence is the number of new cases in a defined population over a specified period. For traumatic brain injury, incidence is hard to measure because the condition ranges from fatal penetrating trauma to a concussion treated in primary care, urgent care, an athletic training room, a military aid station, or not treated at all. Surveillance systems count only what is documented, coded, and submitted; injuries outside that process are left out. This helps explain why the condition has long been recognized as a public health problem that extends beyond neurosurgical wards [18].

In the United States, population-based studies estimated approximately 1,565,000 traumatic brain injuries in 2003, including about 1,224,000 emergency department visits, 290,000 hospitalizations, and 51,000 deaths [19]. For 2002 to 2006, the CDC estimated an average annual incidence of approximately 1.7 million, comprising about 1,365,000 emergency department visits, 275,000 hospitalizations, and 52,000 deaths [5] (Figure 2.1).

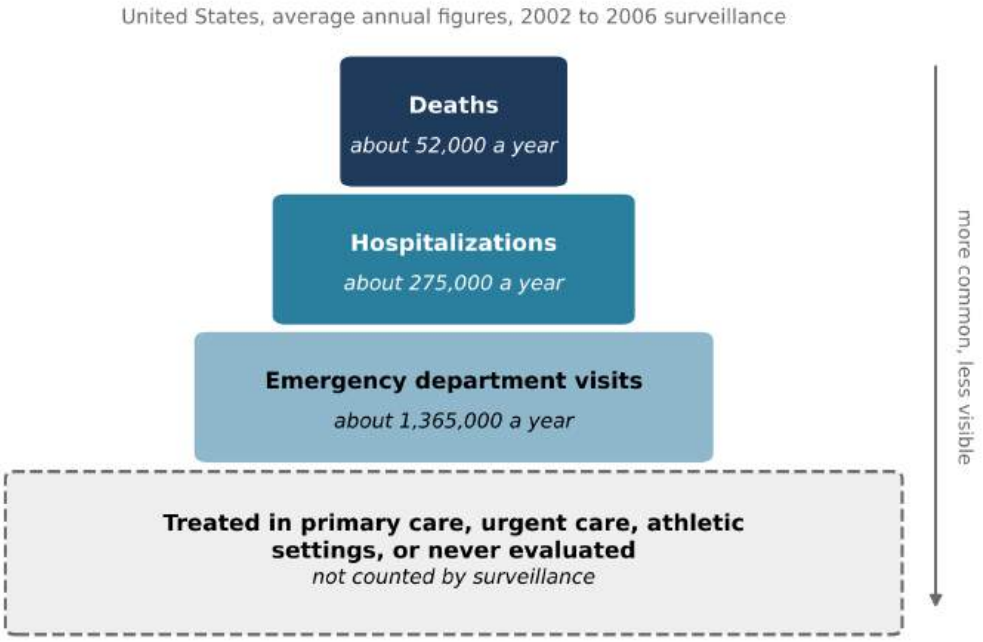


Figure 2.1. What surveillance counts and what it misses. Average annual United States figures for 2002 to 2006 [5]. Injuries treated in primary care, urgent care, or athletic settings, or never evaluated at all, are the most numerous and are not counted. Original schematic.

Later surveillance reported approximately 2.87 million traumatic brain injury-related emergency department visits, hospitalizations, and deaths in 2014, including more than 837,000 among children. This represents a 53% increase in the combined count from 2006, driven mainly by emergency department visits [12]. The CDC summary available in October 2026 separately listed approximately 214,110 hospitalizations in 2020 and 68,663 deaths in 2023; those figures exclude injuries managed only in emergency departments, primary care, or urgent care, and injuries for which no care was sought [20].

The reports use different methods and case definitions. Therefore, comparisons across them reflect changes in counting as well as changes in burden. Each total should be

read with its own definitions and settings. Within a single report using consistent methods, trends remain informative.

These numbers are why the condition is called a silent epidemic, and they are an undercount. The visible cases, including hospitalizations and deaths, are only part of the problem. The invisible cases include patients with normal scans, delayed symptoms, incomplete documentation, no medical evaluation, or symptoms attributed to stress, pain, anxiety, depression, poor sleep, or a musculoskeletal injury. Good surveillance depends on data quality, sensitivity, representativeness, timeliness, and usefulness [21]; traumatic brain injury surveillance struggles with all of them because the disorder is heterogeneous, often subtle, and managed across many care settings [20].

The human and economic toll

Many patients face long-term challenges. Difficulty with attention, memory, processing speed, executive function, mood, sleep, balance, pain, and sensory tolerance can substantially reduce quality of life. Survivors may lose the ability to work, study, drive, parent, manage finances, or perform previously routine tasks at the same level. The setting matters: a deficit that looks subtle in a clinic room can be disabling in a high-demand job.

The economic burden is substantial. A CDC Grand Rounds report estimated the United States burden at approximately 76.5 billion dollars in 2010, reflecting direct medical and indirect costs, including lost wages, lost productivity, and nonmedical expenditures [22]. This historical figure is consistent with the broader injury-cost framework of Finkelstein and colleagues, which extends well beyond the hospital bill [23]. Caregiving and the disruption of family life are not captured in that total.

The global burden is equally concerning. Tens of millions of people sustain a traumatic brain injury each year, and the burden is disproportionately shaped by road traffic injuries, falls, violence, occupational hazards, and uneven access to emergency and rehabilitation services [24,25].

Why traumatic brain injuries go unnoticed

Despite their frequency, these injuries are missed across emergency departments, primary care, specialty clinics, workplaces, schools, athletics, and military settings. The same factors recur.

Symptoms are not specific. Headache, dizziness, fatigue, nausea, irritability, poor sleep, slowed thinking, and light sensitivity also occur with stress, migraine, pain, viral illness, anxiety, depression, and medication use. Unless someone asks about the trauma, the diagnosis never enters the differential.

Onset may be subtle or recognized late. Acute qualifying symptoms generally emerge within the first hours, but patients often recognize their consequences only when they resume demanding activities. A symptom that is new weeks later is a different matter from one that was present from the start and noticed late. Chapter 3 separates delayed onset from delayed recognition and delayed diagnosis. That distinction matters considerably in practice [6].

Knowledge gaps. Non-specialists tend to look for skull fracture, bleeding, and loss of consciousness. They are less familiar with the visual, vestibular, cognitive, and affective symptoms, and the exertional intolerance, that make up most of the clinical picture after a mild injury.

No obvious external injury. Meaningful brain injury can occur without laceration, bruise, swelling, or fracture. An intact scalp does not establish an intact brain.

Reliance on normal imaging. Most mild injuries show nothing on computed tomography or conventional magnetic resonance imaging. A normal scan can falsely reassure clinicians, families, employers, insurers, and patients alike.

Patient underreporting. Memory problems, poor concentration, slowed processing, and reduced mental endurance are not obvious to the patient at first. In moderate and severe injury, impaired self-awareness reduces reporting further and makes collateral history essential [26].

Misinterpreting psychological symptoms. Irritability, anxiety, depression, emotional lability, and reduced frustration tolerance have many causes. Because brain injury and mental health disorders share fatigue, irritability, poor sleep, low motivation, slowed thinking, and reduced concentration, post-traumatic neurobehavioral change is easily filed as purely psychiatric. Careful temporal correlation separates the two, and often finds both.

Pre-existing conditions. In a patient with migraine, depression, anxiety, sleep problems, attention difficulties, chronic pain, or vestibular disease, new post-traumatic symptoms are read as more of the same rather than as a change from baseline. This is also where the exclusion of confounding factors becomes a formal diagnostic requirement, addressed in Chapter 5.

Absence of routine screening. Without standardized questions about head trauma, acceleration and deceleration events, falls, blast exposure, sports injuries, assaults, and post-traumatic symptoms, many injuries are never documented.

Time pressure. Busy emergency departments and short primary care appointments prioritize acute and life-threatening problems. That leaves no room for a detailed neurological history, review of mechanism, cognitive screening, vestibular and ocular-motor testing, collateral history, or a conversation about work and school demands. Subtle cognitive, vestibular, visual, and behavioral symptoms are deferred, incompletely documented, or never revisited.

Focus on visible injuries. In polytrauma, when several body regions are injured, orthopedic, thoracic, abdominal, and spinal injuries rightly command urgent attention and overshadow a concurrent brain injury, particularly when the patient is awake and the scan is negative.

Symptoms that emerge only under load. Many survivors function adequately in quiet, low-demand settings and fail under multitasking, noise, screens, rapid decisions, driving, complex calculation, emotional stress, or sustained work. A normal casual conversation is not evidence of normal cognitive endurance. That is why return-to-work and return-to-school follow-up is essential. It also explains why a normal office assessment can be actively misleading.

Expectations of rapid recovery. Patients, families, employers, insurers, and sometimes clinicians assume a concussion should resolve quickly. Many do. Some do not, and the word mild should not be used to dismiss persistent impairment.

Fast take: main points from chapter 2

- **Frequent causes.** Injuries commonly follow falls, motor vehicle crashes, blast exposure, industrial injury, sports and recreation, penetrating injury, and assault. Direct impact is not required; acceleration, deceleration, rotation, and blast can injure the brain on their own [2,9,10].

- **Secondary injury.** Damage continues after the initial trauma through swelling, inflammation, oxidative stress, altered cerebral blood flow, metabolic dysfunction, hypoxia, and excitotoxicity [3,4].

- **A normal scan is not lack of injury.** Imaging is usually normal in mild injury; it shows no visible lesion on that test, nothing more [7].

- **Mild is not always mild.** Concussion can produce persistent headache, dizziness, visual symptoms, fatigue, cognitive inefficiency, mood disturbance, sleep disruption, and reduced work tolerance [6,8].

- **The criteria changed in 2023.** Three alternative routes: a qualifying clinical sign; at least two qualifying acute symptoms plus at least one appropriate acute examination or laboratory finding; or structural neuroimaging evidence, each within the mechanism, confounder, and severity requirements [6]. Chapter 3 sets them out.

- **A silent epidemic.** Millions of injury-related emergency department visits, hospitalizations, and deaths occur annually in the United States, and many more cases are treated outside hospital systems, undocumented, or never evaluated. Surveillance figures must be read with the definitions of the report that produced them [5,12,19,20].

- **Economic strain.** The burden includes direct healthcare costs plus the indirect costs of lost productivity, disability, caregiving, educational disruption, and long-term rehabilitation [22,23].

- **Additional testing has specific roles and limits.** Neuropsychological assessment, vestibular and oculomotor testing, and electrophysiological methods can measure dysfunction that routine scans miss. Quantitative electroencephalography remains investigational as a diagnostic test [13,14,15,16,17]. Chapter 7 covers these methods; Chapter 8 examines diffusion tensor imaging.

- **Hidden deficits emerge under demand.** Longitudinal follow-up matters most for patients returning to work, school, driving, and athletics.

Traumatic brain injury deserves sustained attention from clinicians, public health systems, researchers, insurers, employers, schools, and the public. It is common, heterogeneous, frequently invisible on routine imaging, and often underestimated. Better prevention, more consistent screening, improved surveillance, and thoughtful use of appropriate diagnostic tools can identify it earlier, support recovery, and reduce its human and economic cost.

References

1. Menon DK, Schwab K, Wright DW, Maas AI; Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health. Position statement: definition of traumatic brain injury. Arch Phys Med Rehabil. 2010;91(11):1637-1640. doi:10.1016/j.apmr.2010.05.017

2. Centers for Disease Control and Prevention. Facts about TBI [Internet]. Atlanta (GA): Centers for Disease Control and Prevention; 2025. Available from: https://www.cdc.gov/traumatic-brain-injury/data-research/facts-stats/index.html

3. Saatman KE, Duhaime AC, Bullock R, Maas AIR, Valadka A, Manley GT, et al. Classification of traumatic brain injury for targeted therapies. J Neurotrauma. 2008;25(7):719-738. doi:10.1089/neu.2008.0586

4. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75(Suppl 4):S24-S33. doi:10.1227/NEU.0000000000000505

5. Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002-2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.

6. Silverberg ND, Iverson GL, Cogan A, Dams-O'Connor K, Delmonico R, Graf MJP, et al. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036

7. Shenton ME, Hamoda HM, Schneiderman JS, Bouix S, Pasternak O, Rathi Y, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. Brain Imaging Behav. 2012;6(2):137-192. doi:10.1007/s11682-012-9156-5

8. McInnes K, Friesen CL, MacKenzie DE, Westwood DA, Boe SG. Mild traumatic brain injury and chronic cognitive impairment: a scoping review. PLoS One. 2017;12(4):e0174847. doi:10.1371/journal.pone.0174847

9. Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. J Head Trauma Rehabil. 2003;18(4):307-316. doi:10.1097/00001199-200307000-00003

10. Cernak I, Noble-Haeusslein LJ. Traumatic brain injury: an overview of pathobiology with emphasis on military populations. J Cereb Blood Flow Metab. 2010;30(2):255-266. doi:10.1038/jcbfm.2009.203

11. Sosin DM, Sniezek JE, Waxweiler RJ. Trends in death associated with traumatic brain injury, 1979 through 1992: success and failure. JAMA. 1995;273(22):1778-1780. doi:10.1001/jama.1995.03520460060036

12. Peterson AB, Xu L, Daugherty J, Breiding MJ. Surveillance Report of Traumatic Brain Injury-Related Emergency Department Visits, Hospitalizations, and Deaths, United States, 2014. Atlanta (GA): Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2019.

13. Haneef Z, Levin HS, Frost JD Jr, Mizrahi EM. Electroencephalography and quantitative electroencephalography in mild traumatic brain injury. J Neurotrauma. 2013;30(8):653-656. doi:10.1089/neu.2012.2585

14. Rapp PE, Keyser DO, Albano A, Hernandez R, Gibson DB, Zambon RA, et al. Traumatic brain injury detection using electrophysiological methods. Front Hum Neurosci. 2015;9:11. doi:10.3389/fnhum.2015.00011
15. Washnik NJ, Anjum J, Lundgren K, Phillips S. A review of the role of auditory evoked potentials in mild traumatic brain injury assessment. Trends Hear. 2019;23:2331216519840094. doi:10.1177/2331216519840094
16. Balaban C, Hoffer ME, Szczupak M, Snapp H, Crawford J, Murphy S, et al. Oculomotor, vestibular, and reaction time tests in mild traumatic brain injury. PLoS One. 2016;11(9):e0162168. doi:10.1371/journal.pone.0162168
17. Tenney JR, Gloss D, Arya R, Kaplan PW, Lesser R, Sexton V, et al. Practice guideline: use of quantitative EEG for the diagnosis of mild traumatic brain injury: report of the Guideline Committee of the American Clinical Neurophysiology Society. J Clin Neurophysiol. 2021;38(4):287-292. doi:10.1097/WNP.0000000000000853
18. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375-378. doi:10.1097/00001199-200609000-00001
19. Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. J Head Trauma Rehabil. 2006;21(6):544-548.
20. Centers for Disease Control and Prevention. TBI data [Internet]. Atlanta (GA): Centers for Disease Control and Prevention; 2026 Apr 27 [cited 2026 Oct 24]. Available from: https://www.cdc.gov/traumatic-brain-injury/data-research/index.html
21. German RR, Lee LM, Horan JM, Milstein RL, Pertowski CA, Waller MN, et al. Updated guidelines for evaluating public health surveillance systems: recommendations from the Guidelines Working Group. MMWR Recomm Rep. 2001;50(RR-13):1-35.
22. Centers for Disease Control and Prevention. CDC Grand Rounds: reducing severe traumatic brain injury in the United States. MMWR Morb Mortal Wkly Rep. 2013;62(27):549-552.
23. Finkelstein EA, Corso PS, Miller TR. The Incidence and Economic Burden of Injuries in the United States. New York: Oxford University Press; 2006.
24. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung YC, Punchak M, et al. Estimating the global incidence of traumatic brain injury. J Neurosurg. 2019;130(4):1080-1097. doi:10.3171/2017.10.JNS17352
25. World Health Organization. Global Status Report on Road Safety: Time for Action. Geneva: World Health Organization; 2009.
26. Dromer E, Kheloufi L, Azouvi P. Impaired self-awareness after traumatic brain injury: a systematic review. Part 1: assessment, clinical aspects and recovery. Ann Phys Rehabil Med. 2021;64(5):101468. doi:10.1016/j.rehab.2020.101468

Chapter 3 Diagnosing traumatic brain injury

Chapter overview

Traumatic brain injury (TBI) is defined as an alteration in brain function or other evidence of brain pathology caused by an external mechanical force [1]. Although this unifying definition is widely accepted, operational criteria for confirming TBI at the bedside vary across organizations, with real consequences for clinical care, research, and medicolegal evaluation [2,3]. This chapter asks two questions: did the patient sustain a TBI, and on what basis?

This chapter begins with the conceptual definition and the mechanisms by which TBI occurs. It then addresses when clinical manifestations appear and how clinicians can make a diagnosis retrospectively. With that foundation, it examines the American Congress of Rehabilitation Medicine's 2023 diagnostic criteria in detail. These criteria provide a major multidisciplinary consensus framework. The 2023 criteria organize three alternative pathways after a plausible mechanism: qualifying clinical signs, at least two qualifying acute symptoms combined with at least one appropriate acute examination or laboratory finding, or unambiguous trauma-related structural neuroimaging evidence [2].

The criteria issued by the World Health Organization (WHO), the Centers for Disease Control and Prevention (CDC), the Department of Veterans Affairs and the Department of Defense (VA/DoD), the American Academy of Neurology (AAN), the Mayo Classification System, the Common Data Elements (CDE) working group, and the Concussion in Sport Group (CISG) are then compared with that standard. Agreement outweighs divergence. The common core is an external mechanical force with evidence of altered brain function or trauma-related brain pathology, interpreted in light of plausible alternative explanations; loss of consciousness is not required by any of them. The remaining differences concern the threshold for a sufficient alteration of mental status, the status of retrograde amnesia, the role of imaging, and whether symptoms alone can establish the diagnosis. Severity grading, which builds on a confirmed diagnosis, is the subject of Chapter 4.

Introduction

The first clinical question after a head injury, particularly a mild one, is not “how severe?” but “is this a TBI at all?” That question is more difficult than it appears. Symptoms are often nonspecific, routine imaging is frequently normal, and many patients present hours, days, or weeks after the injury, when the temporal link between

the mechanical event and current symptoms is no longer obvious. Misdiagnosis in either direction carries consequences. A missed injury means missed opportunities for early intervention, mislabeling as a primary psychiatric disorder, denied insurance claims, and inadequate documentation in legal proceedings. An injury diagnosed on insufficient evidence means treatment aimed at the wrong problem, symptoms with another cause left unexplained, and a diagnosis that will not withstand scrutiny.

The need for a clear, uniform diagnostic definition has long been recognized. In 2010, the Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements proposed a deliberately broad, unifying definition: TBI is an alteration in brain function or other evidence of brain pathology caused by an external force [1]. This definition is now widely cited and serves as the conceptual anchor for the more operationally specific criteria reviewed in this chapter. The 2023 ACRM diagnostic criteria explicitly build on it and operationalize the diagnosis in clinical settings [2].

The scale of the definitional problem is worth stating concretely. In one study, seventeen published definitions of mild TBI were applied to a prospectively collected sample of 11,907 children evaluated in emergency departments. The proportion meeting criteria for mild TBI ranged from 7% to 99%, depending solely on the definition used [2,26]. That heterogeneity is a recognized barrier to research and to the development of targeted treatment [2,4].

The stakes are set by the scale of the problem described in Chapter 2. U.S. surveillance documents a substantial burden of TBI-related emergency department visits, hospitalizations, disability, and death [5,6]. Older epidemiologic studies document the burden in mortality, healthcare utilization, and societal cost [7,8,9,10]. Current CDC materials emphasize that nonfatal injury and post-acute disability account for much of that burden [11]. A clear, consistent diagnostic framework is the foundation for surveillance, research, prevention, and rehabilitation.

Defining TBI: the core concept

The unifying definition has three essential components.

An external mechanical force. The injury must result from external biomechanical energy transferred to the head or body. This includes direct impact from a blow, fall, or assault; rapid acceleration-deceleration, including an acceleration-deceleration event without direct head impact; penetrating trauma; and blast overpressure [1,2]. This requirement distinguishes TBI from internal causes of

altered brain function, such as stroke, tumor, infection, hypoxia, or metabolic encephalopathy.

Altered brain function or other evidence of brain pathology. The mechanical force must produce evidence of brain involvement. Acceptable evidence includes loss of consciousness, post-traumatic amnesia, an alteration in mental state at the time of injury, focal neurological deficits, seizures, or imaging abnormalities consistent with trauma [1,2]. The 2023 ACRM criteria also recognize objectively measured cognitive, balance, oculomotor, and vestibular-oculomotor findings, and elevated blood biomarkers, as evidence of brain involvement when they accompany qualifying acute symptoms [2].

Exclusion of alternative explanations. Before confirming the diagnosis, symptoms attributable to intoxication, sedation, primary psychiatric reactions such as acute stress or dissociation, pre-existing neurological disease, or other medical conditions must be considered. All major frameworks make this exclusion an explicit or implicit requirement.

Normal CT or MRI does not rule out TBI. Mechanical forces can trigger ionic shifts, excitatory neurotransmitter release, altered glucose metabolism, mitochondrial dysfunction, and axonal disturbances that routine structural imaging does not reliably detect [12]. Therefore, dysfunction behind a normal scan is biologically plausible. Neither a normal scan nor a research imaging abnormality determines an individual patient's diagnosis; that is the work of the criteria set out below. Chapter 8 examines the imaging evidence in detail.

Mechanism-based classification

Beyond the severity grade discussed in Chapter 4, TBI is also characterized by the mechanism of injury. This characterization helps describe how energy was transferred and which associated injuries require consideration [1,2]. Road traffic injuries are an important setting for prevention [13]. Closed, penetrating, and blast-related injuries are useful descriptors, although they are not mutually exclusive: a blast can produce both blunt and penetrating trauma [14].

Closed-head injury

A closed-head injury is a nonpenetrating injury in which mechanical forces act on the brain without an object entering the intracranial compartment. A skull fracture may be present; an intact skull is not required. Falls, sports collisions, assaults, motor vehicle crashes, occupational accidents, and blast exposure are common causes. The

resulting damage may include focal contusions, hemorrhage, edema, axonal injury, and disruption of functional networks. In mild closed-head injury, routine imaging is usually normal [12].

Direct head impact is not required. Sufficient acceleration-deceleration or rotational loading can injure the brain without direct contact between the head and an object. The 2023 ACRM criteria explicitly recognize this mechanism [2]. A whiplash-type event is therefore a plausible mechanism. It is only a mechanism: the event alone does not establish brain injury, and the same forces can injure the neck and the inner ear, producing overlapping symptoms of their own.

Open-head (penetrating) injury

A penetrating brain injury occurs when an object breaches the skull and dura, entering the intracranial tissue; an open skull injury is not the same. Damage may be focal, multifocal, or diffuse, depending on trajectory, velocity, fragmentation, contamination, and associated vascular injury. The specific risks include infection, hemorrhage, seizures, cerebrospinal fluid leak, and focal deficits. The 2023 ACRM criteria lower the diagnostic threshold for nonpenetrating mild TBI; a penetrating injury requires its own anatomical and clinical assessment [2].

Explosive blast injury

Blast TBI may involve pressure-wave injury, blunt impact from being thrown, penetrating fragments, and acceleration-deceleration forces. In U.S. military personnel with clinically diagnosed blast-related TBI, diffusion tensor imaging demonstrated abnormalities consistent with traumatic axonal injury despite generally unremarkable conventional imaging [14]. However, none of the injured participants had isolated primary blast exposure, so that study could not separate the contribution of the pressure wave from accompanying injury mechanisms. The 2023 ACRM criteria explicitly include forces generated by a blast or explosion as a qualifying mechanism [2].

Timing: when manifestations appear, and when the diagnosis is made

When symptoms appear and when a diagnosis is made are distinct questions, but they are often confused. The distinctions set out here provide the framework for the discussions that follow.

Many TBI symptoms, including headache, nausea, confusion, dizziness, light or sound sensitivity, blurred vision, imbalance, irritability, sleep disruption, and memory problems, appear within seconds to minutes of the event. Others emerge over hours or

days. Still others become apparent only weeks or months later, usually when the patient returns to work, school, driving, or another demanding setting that exceeds the injured brain's capacity. Loss of consciousness, when present, usually lasts seconds to minutes. None of these features is universal, and none is required on its own.

The frameworks address this temporal spread differently. The CDC acknowledges that symptoms may not appear for hours or days and that emotional and sleep complaints may first be noticed a week or two after injury. The Concussion in Sport Group (CISG) similarly acknowledges that subjective symptoms such as headache or cognitive change may emerge minutes to hours after injury rather than at the moment of impact. The 2023 ACRM criteria are the most restrictive, for a specific reason: because acute symptoms can support a diagnosis without any observed clinical sign, the criteria limit them to a window in which attributing them to the injury remains justifiable. Under ACRM, the subjective sense of altered mental status must begin immediately after impact or upon regaining consciousness, and qualifying physical, cognitive, and emotional symptoms may be delayed by a few hours but nearly always appear within 72 hours [2].

Three situations are often confused: delayed onset, delayed recognition, and delayed diagnosis. Keeping them separate matters when deciding what the history can establish.

Delayed onset refers to a manifestation that was not present during the acute period and developed later. Late-onset complaints can be clinically important and may prompt a patient to seek evaluation, but under the ACRM criteria they do not, in themselves, satisfy a diagnostic criterion.

Delayed recognition describes a manifestation that was present acutely but went unnoticed, unreported, or undocumented, often because the patient was in an environment that placed no demands on the impaired function. A teacher who spends a week at home after a collision may not discover a divided-attention deficit until she returns to the classroom. On careful history, what appears to be a new symptom at six weeks is often a preexisting one that first became visible then. Distinguishing the two requires a detailed reconstruction of the first 72 hours rather than accepting the date on which the patient first complained.

Delayed diagnosis describes the common situation in which the diagnosis itself is made weeks, months, or years after the event. This is explicitly contemplated by the ACRM, VA/DoD, and Mayo frameworks and is entirely legitimate. What is reconstructed

retrospectively, however, is the acute picture: the mechanism, the acute signs, and the acute symptoms. The diagnosis is late; the qualifying features are not (Figure 3.1).

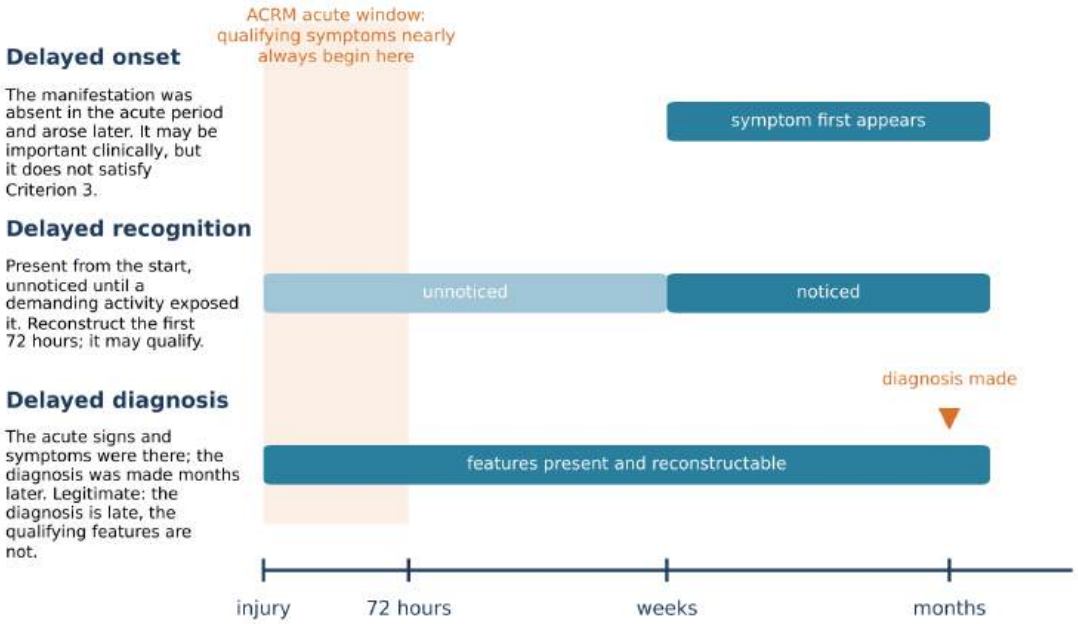


Figure 3.1. Three kinds of delay. In delayed onset, the manifestation was absent during the acute period and appeared later; it does not meet Criterion 3. In delayed recognition, it was present from the start and was noticed only when a demanding activity exposed it; reconstruction of the first 72 hours may qualify it. In delayed diagnosis, the acute features were present, and the diagnosis was simply made late. Pappolla ® Original schematic.

A fourth distinction, between identifying an injury and attributing symptoms to an already identified injury, is the source of a common citation error. Frameworks that address attribution use longer windows than the ACRM diagnostic criteria. The ICD-10 research criteria for postconcussional syndrome require that symptom onset occur no more than four weeks after head trauma [15]. That window governs when symptoms may be attributed to a concussion already established; it does not extend the window within which symptoms may establish that a brain injury occurred. The widely quoted figure that most mild TBI symptoms resolve within roughly 30 days invites the same confusion: it is a statement about expected recovery, not about onset or attribution.

The practical consequence is that late manifestations affect persistent impairment, outcome, rehabilitation needs, and clinical correlation, and they frequently prompt diagnostic evaluation, yet the diagnostic criteria themselves are anchored to the acute period. The ACRM acknowledges that its criteria address diagnosis, not clinical outcome, and that a person who sustains a mild TBI may recover within days or have

symptoms that persist for a prolonged period [2]. It also acknowledges that clinicians should use all available information and clinical judgment in cases that do not cleanly fit the criteria, and that a witnessed hard blow to the head creates high pre-test odds that should lower the strength of additional evidence required [2]. A patient whose deficits manifest late is therefore not undiagnosable; the diagnostic argument simply has to be built on a reconstruction of the acute period rather than on the late presentation itself.

Preserving these distinctions makes the evaluation of a late-presenting patient more accurate because it clarifies what the later findings do and do not show.

The American Congress of Rehabilitation Medicine criteria

The ACRM criteria are set out below in detail; the other frameworks are then compared with them.

The 1993 definition

The 1993 ACRM definition was the most widely cited clinical framework for mild TBI for three decades. It defines mild TBI as a traumatically induced physiological disruption of brain function, manifested by at least one of the following: loss of consciousness for 30 minutes or less; loss of memory for events immediately before or after the accident, with post-traumatic amnesia lasting no more than 24 hours; any alteration in mental state at the time of the accident, such as feeling dazed, disoriented, or confused; or focal neurological deficits that may or may not be transient. The Glasgow Coma Scale (GCS) score after 30 minutes is expected to be 13 to 15 [16].

The 2023 revision was undertaken because this definition had accumulated recognized limitations, most notably its failure to distinguish signs from symptoms, and because competing definitions had proliferated in the intervening thirty years [2].

The 2023 criteria: three pathways to diagnosis

The 2023 criteria represent an important expansion of the earlier sign-based definition. Following a biomechanically plausible mechanism of injury, mild TBI can be diagnosed through any of three pathways.

The first is the traditional pathway: one or more acute clinical signs attributable to brain injury, including loss of consciousness, an observed alteration of mental status, post-traumatic amnesia, or another acute neurological sign.

The second pathway applies when no such clinical sign is clearly documented: at least two new or worsened acute symptoms, together with at least one objective acute clinical examination or laboratory finding attributable to brain injury.

The third pathway is unambiguous trauma-related intracranial abnormality on computed tomography or structural magnetic resonance imaging.

These are alternatives rather than steps. Any one of the three establishes the diagnosis, and a patient who satisfies one need not satisfy the others.

The ACRM assigns a number to each element of the criteria, and those numbers are used throughout the literature and throughout the remainder of this chapter. They are as follows [2].

Criterion 1: mechanism of injury. A transfer of mechanical energy to the brain from an external force, arising from the head being struck by an object, the head striking a hard object or surface, the brain undergoing acceleration or deceleration without direct contact between the head and an object or surface, or forces generated by a blast or explosion. This criterion is required in all three pathways. It may be established by direct observation or video review, by collateral report, by review of acute care records, or by the patient's own account of the event.

Criterion 2: clinical signs. One or more of the following, observed or elicited: loss of consciousness immediately after injury; alteration of mental status immediately after injury or upon regaining consciousness, evidenced by reduced responsiveness or inappropriate responses to external stimuli, slowness to respond to questions or instructions, agitated behavior, inability to follow two-part commands, or disorientation to time, place, or situation; complete or partial amnesia for events following the injury; or another acute neurologic sign, such as observed motor incoordination on standing, a seizure, or tonic posturing. Anyone is sufficient.

Criterion 3: acute symptoms. Two or more new or worsened symptoms drawn from four categories: acute subjective alteration in mental status (feeling confused, disoriented, or dazed); physical symptoms (headache, nausea, dizziness, balance problems, vision problems, sensitivity to light, or sensitivity to noise); cognitive symptoms (feeling slowed down, mental fog, difficulty concentrating, or memory problems); and emotional symptoms (uncharacteristic emotional lability or irritability). The two symptoms need not come from different categories. Symptoms outside this list may be present but are not counted, and complaints with known poor specificity, notably fatigue and nervousness, were deliberately excluded to preserve specificity [2]. The timing requirements set out earlier in this chapter apply.

Criterion 4: clinical examination and laboratory findings. One or more of the following: cognitive impairment on acute clinical examination; balance impairment on acute clinical examination; oculomotor impairment or symptom provocation in response to vestibular-oculomotor challenge on acute clinical examination; or an elevated blood biomarker indicative of intracranial injury.

Criterion 5: neuroimaging. Trauma-related intracranial abnormalities on computed tomography or structural magnetic resonance imaging.

Criterion 6: not better accounted for by confounding factors. Pre-existing and co-occurring health conditions and other confounders have been considered and determined not to fully account for the findings relied upon. By its terms, this criterion addresses the clinical signs of Criterion 2, the acute symptoms of Criterion 3, and the examination and laboratory findings of Criterion 4 [2].

The three pathways are therefore combinations of these numbered elements:

Pathway 1 is Criterion 1 plus Criterion 2, subject to Criterion 6.

Pathway 2 is Criterion 1 plus Criterion 3 plus Criterion 4, subject to Criterion 6.

Pathway 3 is Criterion 1 plus Criterion 5.

Note that Criterion 6 is not intended to reach Criterion 5. The requirement that an intracranial abnormality be trauma-related is built into Criterion 5 itself, so the confounder analysis that governs signs, symptoms, and examination findings does not apply separately to the neuroimaging pathway.

Signs, symptoms, and objective findings: the distinction that organizes the criteria

The distinction between a sign and a symptom is familiar to neurologists, yet clinical records often blur the line. The 2023 revision makes that distinction explicit and places it at the center of the criteria.

A **sign** is something another person can observe, or that an examiner can elicit. A **symptom** is an internal experience that only the patient can report. Fever is a sign; feeling feverish is a symptom. In the head-injury setting, a patient who cannot say where she is when asked has an observable sign. A patient who later reports that she felt disoriented has described a symptom. An **objective clinical examination or laboratory finding** is a third category: a measured abnormality on a standardized test, interpreted against normative data or the patient's own preinjury performance.

The same neurological concept can appear as either a sign or a symptom. The term alone does not indicate which it is; you need to know how the evidence was obtained. The criteria deliberately pair the two forms. Slowness in responding to questions is a Criterion 2 sign; feeling slowed down is a Criterion 3 symptom. Disorientation to time, place, or situation is a sign; feeling disoriented or dazed is a symptom. A definite amnestic gap for events after the injury is a sign; subjective memory difficulty is a symptom. Observed motor incoordination on standing is a sign. Dizziness and balance problems are symptoms. An abnormal standardized balance examination is a Criterion 4 objective finding. The same clinical domain, balance, can yield evidence in all three categories, and which category applies depends entirely on how the evidence was obtained.

Two qualifications follow.

First, a Criterion 2 sign must be present during the acute injury period. The criteria require that altered mental status occur immediately after the injury or upon regaining consciousness. Disorientation observed at an office visit three weeks later is not a Criterion 2 sign at the time of injury; however, it is objectively documented. The same abnormality on standardized testing within the acute period is a Criterion 4 finding; documented later, it belongs to the assessment of persistent dysfunction, a separate and equally legitimate purpose.

Second, the ACRM permits Criterion 2 signs to be established retrospectively, using the sources of evidence specified in the criteria. The criteria state that Criterion 2 may be met by direct observation, including video review; by collateral witness report; by review of acute care records; or, when none of these are available, by the person's own recounting of the injury event [2]. The criteria go one step further, permitting the clinician to reconstruct whether a sign would have been observable, for example, by asking whether the patient would have been able to answer questions about where she was and what had happened immediately after the injury. In that circumstance, observable behaviors elicited through self-report may be counted as signs [2].

This departs from strict semiology. Consider the difference between two accounts from the same patient. "I felt confused" is a symptom. "For about five minutes I did not know where I was, I could not understand why I was on the road, and I kept asking my husband what had happened" is a retrospective account of observable altered mental status, which the criteria permit to satisfy Criterion 2 even though no clinician witnessed it. If the husband can corroborate it, the account rests on collateral witness report, one of the sources expressly permitted by the criteria. Document the source and the

specific observations on which a retrospectively established sign rests, because in contested settings that is precisely what will be examined.

Post-traumatic amnesia illustrates the same point. “My memory was bad afterward” is a symptom. “I remember the impact, and the next thing I remember is being in the emergency department two hours later” describes a definite amnestic interval, which the criteria treat as a Criterion 2 clinical sign, even though it is almost always established historically rather than observed. The operative meaning of “sign” in the ACRM framework is therefore not “personally witnessed by a clinician.” It is closer to an objectively characterizable manifestation of disrupted brain function, as distinct from subjective experiences such as headache, dizziness, fogginess, or confusion.

Why the second pathway was created

The rationale for the second pathway explains most of what is new in the 2023 criteria.

The Task Force recognized that many legitimate mild TBIs occur without a witness, without an immediate neurological examination, and without documented loss of consciousness, post-traumatic amnesia, or confusion. A patient who crashes alone on a rural road, walks away, and is first evaluated two days later may have an unambiguous brain injury yet no documented acute signs. Under a sign-only rule, that patient could not be diagnosed, and the old approach systematically missed such cases, with consequences for clinical care, insurance and disability determinations, and research cohorts.

At the same time, the panel did not want headache plus dizziness alone to establish a brain injury, because those complaints are nonspecific and common after trauma of any kind. The panel’s own survey rated symptoms as having variable and generally lower diagnostic importance than observable clinical signs [2]. **The solution was to require corroboration in proportion to the weakness of the evidence: when the diagnosis rests on symptoms rather than on an observable sign, an objective abnormality is also required.**

See Table 3.1 on the next page.

Table 3.1. The three diagnostic pathways of the 2023 ACRM criteria for mild TBI.

	Pathway 1	**Pathway 2**	**Pathway 3**
Basic evidence	Acute clinical sign (Criterion 2)	Acute subjective symptoms (Criterion 3)	Structural neuroimaging (Criterion 5)
Number required	One sign	Two or more symptoms	One unambiguous trauma-related abnormality
Objective test required	No	Yes, at least one Criterion 4 finding	The imaging is itself the objective evidence
Examples	Loss of consciousness, observed alteration of mental status, post-traumatic amnesia, other acute neurologic sign	Headache plus dizziness, together with objective balance, oculomotor, cognitive, or blood biomarker abnormality	Contusion or intracranial hemorrhage on CT or structural MRI
Timing	Sign occurs at the time of injury or on regaining consciousness	Subjective altered mental status is immediate; other symptoms nearly always begin within 72 hours; the supporting examination or laboratory finding is acute	When imaging is obtained, provided the abnormality is attributable to the injury being evaluated
Why sufficient	The sign is strong evidence of acute brain dysfunction	Symptoms are less specific, so objective corroboration is required	Structural injury is direct evidence of trauma to the brain

Table 3.1 legend. All three pathways require a biomechanically plausible mechanism of injury (Criterion 1). Pathways 1 and 2 are additionally subject to Criterion 6, which requires that confounding factors not fully account for the signs, symptoms, and examination findings relied upon. Criterion 6 is not written to reach Criterion 5, because the requirement that an abnormality be trauma-related is built into Criterion 5 itself.

Table 3.2. Summary of the six ACRM criteria

Element	Role in diagnosis
1 Mechanism	A plausible external mechanical event transferring energy to the brain is required.
2 Acute clinical signs	Evidence such as acute loss of consciousness, observable mental-state alteration, post-traumatic amnesia, or another qualifying acute neurological sign.
3 Acute symptoms	At least two qualifying new or worsened symptoms, with the prescribed acute timing.
4 Examination or laboratory findings	A qualifying acute cognitive, balance, oculomotor, vestibular-oculomotor, or blood-biomarker finding assessed using reliable methods.
5 Structural imaging	An intracranial CT or structural MRI abnormality attributable to the trauma.
6 Alternative explanations	Confounders must not fully explain the clinical signs, symptoms, or findings used.

Table 3.2 legend. Original summary based on the 2023 ACRM framework [2].

A worked example makes the second pathway concrete. A woman is rear-ended at speed. No one documents loss of consciousness. No one observes confusion. There is no demonstrable amnestic gap, no seizure, no tonic posturing, and no observed motor incoordination. Within hours, she reports dizziness, headache, photophobia, and mental fog. These are four qualifying symptoms, but they are symptoms rather than signs, so Pathway 1 is unavailable to her. If an appropriately performed acute vestibular and oculomotor assessment demonstrates objective oculomotor impairment, the structure of Criterion 3 plus Criterion 4 is satisfied, and mild TBI is diagnosed. If no such objective testing was performed acutely, the same patient meets criteria for suspected mild TBI rather than for the diagnosis itself.

The diagnostic reach of criterion 4, and what it means for specialized testing

Two limitations of the second pathway deserve emphasis.

First, objective findings alone are never sufficient. The ACRM states that available clinical examination and laboratory findings have imperfect sensitivity and specificity, so they cannot definitively rule in mild TBI but can increase diagnostic certainty in the context of a plausible mechanism and acute symptoms [2].

Second, Criterion 4 is intended to reflect an acute examination or laboratory finding. Most currently available cognitive, balance, oculomotor, and blood-based measures lose diagnostic sensitivity within the first 72 hours after injury, though the rate varies by test. The ACRM therefore states that Criterion 4 usually cannot be established in a post-acute assessment. At that point, diagnosis depends heavily on the accuracy of the patient's retrospective account of the event and the acute period [2].

The ACRM deliberately does not specify clinical instruments, neuroimaging sequences, or blood-based biomarkers to ensure the criteria remain current as technology advances. Clinical and laboratory tests should be considered only when they meet established standards for reliability and diagnostic accuracy. Impairment must be a clinically meaningful discrepancy between post-injury test performance and age-appropriate normative reference data, or, where available, the patient's pre-injury performance [2].

This bears directly on the specialized testing described in Chapter 7. An acute videonystagmography study showing oculomotor impairment is the kind of finding Criterion 4 contemplates, although the ACRM names no instrument; quantitative electroencephalography and evoked potentials are not among the domains it names. The same videonystagmography study performed months or years after the injury is different evidence. It documents persistent dysfunction, which can be correlated with the clinical picture, but it is not the acute finding that Criterion 4 was written to describe.

Suspected mild TBI

The 2023 criteria also define a category of suspected mild TBI. It applies when brain injury is a probable explanation for the clinical picture, but certainty is reduced by a subtle presentation, missing information, or prominent confounding factors [2]. Following a biomechanically plausible mechanism, a mild TBI is suspected when any one of three situations is present: at least two acute symptoms are present but no other criteria sufficient for diagnosis are met; at least two clinical examination or laboratory findings are present but no other criteria sufficient for diagnosis are met; or it is unclear whether the signs, symptoms, and findings are accounted for by confounding factors.

The category prevents both diagnostic extremes: dismissing a plausible case because acute documentation is incomplete and claiming that headache plus dizziness alone establishes a brain injury. The ACRM further recommends that a patient with suspected mild TBI identified in the first few days after injury should usually be managed as though a mild TBI had occurred, consistent with the principle of removing an athlete or service member from activity when the diagnosis is in doubt [2]. Later information, such as impaired cognitive testing at a clinic visit the day after injury, may increase diagnostic certainty without necessarily changing management, since management has already been initiated.

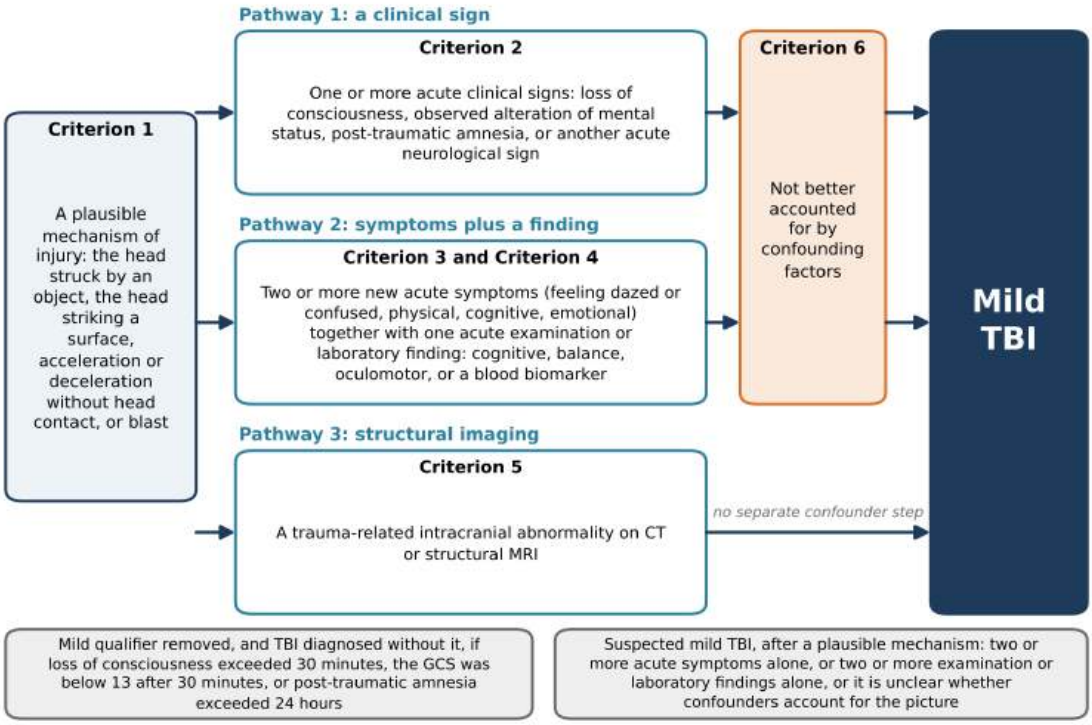


Figure 3.2. The three diagnostic pathways of the 2023 ACRM criteria for mild traumatic brain injury. Modified schematic drawn from the criteria of Silverberg et al. [2].

Concussion, the mild qualifier, and neuroimaging qualifiers

Three further features of the 2023 criteria complete the picture.

First, the panel addressed terminology directly: 30 of 32 expert panel members, or 93.8%, agreed that the diagnostic label concussion may be used interchangeably with mild TBI when neuroimaging is normal or not clinically indicated [2]. This is relevant whenever records treat the two terms as describing different injuries.

Second, the mild qualifier is dropped, and TBI is diagnosed without it if loss of consciousness exceeded 30 minutes, if the GCS remained below 13 after 30 minutes, or if post-traumatic amnesia exceeded 24 hours.

Third, when neuroimaging is performed and reveals a trauma-related intracranial abnormality, the qualifier "with neuroimaging evidence of structural intracranial injury" may be applied while the mild TBI classification is retained. When imaging is performed and is normal, the corresponding "without" qualifier may be used. When imaging is not performed, no qualifier is used [2].

The same imaging-positive injury may receive a different severity label under a different framework. This book follows the ACRM convention [2]; Chapter 4 compares the four approaches and explains how to interpret their labels in medical records.

Other diagnostic frameworks

The frameworks below share much with the ACRM criteria and with each other. Reading them side by side helps identify which differences matter for an individual diagnosis. Each is summarized here, followed by a comparison of their points of agreement and disagreement.

World Health Organization Collaborating Centre Task Force

The WHO Task Force defined mild TBI as an acute brain injury caused by mechanical energy to the head from external physical forces, with one or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, or other transient neurological abnormalities, such as focal signs, seizures, or intracranial lesions not requiring surgery. The GCS should be 13 to 15 at 30 minutes post-injury or on presentation to healthcare [19,20]. The WHO definition requires temporary confusion or disorientation as the indicator of altered mental status; it does not accept feeling dazed or sluggish thinking on their own as sufficient. The WHO framework was also the first to introduce the explicit requirement that clinical signs not be attributable to confounding factors such as acute pain, psychological trauma, and alcohol intoxication, a requirement retained as Criterion 6 of the 2023 ACRM criteria [2].

Centers for Disease Control and Prevention

The CDC addresses TBI through clinical guidance, public health information, and surveillance definitions, which serve different purposes. Its Report to Congress describes the clinical and public health burden of mild TBI and the cost of incomplete

recognition and documentation [6], and its guidance treats loss of consciousness as unnecessary for the diagnosis [2]. The surveillance framework uses coded diagnoses to estimate population burden; it is not a bedside diagnostic system [6].

Department of Veterans Affairs and Department of Defense

The VA/DoD framework defines TBI as resulting from an external force and an acute alteration of brain function. Its mild severity category includes loss of consciousness of 0 to 30 minutes, alteration of consciousness or mental state for up to 24 hours, post-traumatic amnesia for up to 1 day, and a GCS score of 13 to 15; structural imaging shows no clinically relevant abnormality [18]. These are upper severity limits, not requirements that every feature be present. The framework is most relevant to military and veteran populations, where blast exposure, polytrauma, repeated injuries, and comorbid post-traumatic stress disorder complicate diagnosis. Its key conceptual contribution is separating acute severity grading from post-acute symptom management: persistent vestibular, cognitive, sleep, headache, mood, or visual symptoms are assessed on their own terms, regardless of the initial severity category.

American Academy of Neurology

The AAN's 1997 practice parameter on the management of concussion in sports defined concussion broadly as a trauma-induced alteration in mental status that may or may not involve loss of consciousness [21]. This formulation is intentionally inclusive and explicit that loss of consciousness is not required, a position later adopted by the WHO Task Force, the CDC, and the VA/DoD. The AAN updated its sports concussion framework in 2013 [22]. Both documents have since been retired by the Academy, but the underlying diagnostic principle, altered mental status caused by trauma with or without loss of consciousness, remains influential.

Mayo Classification System

The Mayo Classification System grades TBI and rates its severity as definite moderate-severe, probable mild, or symptomatic, based on neuroimaging findings, GCS score, post-traumatic amnesia, loss of consciousness, and specified post-concussive symptoms [17]. It does not require loss of consciousness; confusion, disorientation, or memory impairment suffices. Its design goal was to make maximal use of whatever positive evidence appears in the record, which allows it to classify a larger proportion of cases than single-indicator systems [17]. Its definite, probable, and possible categories anticipated the graded certainty later adopted by the ACRM.

The complicated versus uncomplicated mild TBI distinction is separate from the Mayo system; Chapter 4 explains its origins and how the two approaches differ.

Common Data Elements working group

The Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements proposed the unifying definition cited at the start of this chapter: TBI is an alteration in brain function or other evidence of brain pathology caused by an external force [1]. This definition is widely adopted as a conceptual anchor across organizations and is explicitly referenced in the 2023 ACRM criteria [2].

Concussion in Sport Group

The CISG issues consensus statements primarily focused on sport-related concussion. The Amsterdam statement defines concussion as resulting from a direct blow to the head, neck, or body that transmits an impulsive force to the brain. Symptoms and signs may appear immediately or develop over minutes or hours; conventional structural imaging is typically normal [23]. The 2023 ACRM paper criticized the ambiguity of earlier sport-related definitions, under which an isolated nonspecific symptom could be interpreted as proof of concussion, and addressed this by requiring at least two qualifying acute symptoms plus at least one acute examination or laboratory finding when no clinical sign is present [2].

Comparative synthesis: where the frameworks agree and where they diverge

Historically, most definitions of TBI relied on four acute clinical features as evidence of disrupted brain function: loss of consciousness, altered mental status, post-traumatic amnesia, and acute neurological signs. Any one of these was considered sufficient. The 2023 ACRM criteria retain all four within Criterion 2, thereby preserving the traditional pathway, but they also add the symptom-plus-objective-finding pathway and the structural imaging pathway alongside it. The four historical features are no longer the only route to diagnosis.

Three points of agreement extend across all major frameworks.

All require that the evidence of injury be temporally related to the event, subject to the distinctions set out earlier in this chapter under timing.

All focus on the occurrence of a qualifying feature rather than on a minimum duration. A momentary alteration of mental status is sufficient for diagnostic purposes; duration is used for severity grading in Chapter 4, not for diagnosis.

All require that the qualifying feature not be better explained by another cause: intoxication, sedation, acute stress or dissociative reactions, pre-existing neurological disease, metabolic derangement, and other medical conditions. The 2023 ACRM criteria state this as a separate criterion and extend it across signs, symptoms, and examination findings, naming for each the confounders that must be considered [2]; Chapter 5 works through them. When it is unclear whether confounders explain the findings, the case is suspected mild TBI rather than confirmed or excluded.

The following remaining disagreements are narrow yet clinically meaningful:

Threshold for alteration of mental status. The WHO definition specifically names confusion or disorientation. Other frameworks describe a broader range of acute mental-state changes, but their wording and purpose differ. The 2023 ACRM criteria explicitly separate observed alteration of mental status, assigned to Criterion 2, from a subjectively experienced alteration, assigned to Criterion 3 [2,18,19,20]. Check the original wording before treating a brief report of feeling dazed as sufficient under a particular framework.

Retrograde amnesia. The 1993 ACRM definition and several later frameworks accepted loss of memory for events immediately before or after injury; the WHO definition specifically requires post-traumatic amnesia [2,16,19,20]. The 2023 ACRM criteria now also restrict its use, permitting retrograde amnesia to substitute for post-traumatic amnesia only when the latter cannot be reliably assessed, for example, because of polytrauma or sedating analgesia. The stated rationale is that retrograde amnesia rarely occurs without post-traumatic amnesia and that isolated retrograde amnesia may be more consistent with a non-TBI mechanism, such as syncope or psychological trauma [2]. The change matters when reviewing older records or applying older criteria retrospectively.

Acute neurological signs. Most frameworks accept objectively detected neurological deficits as sufficient evidence of altered brain function. The 2023 ACRM criteria removed the word “focal” and instead refer to other acute neurologic signs, giving observed motor incoordination on standing, seizure, and tonic posturing as examples, with an emphasis on what is observed rather than on what is reported. A focal neurological deficit also warrants assessment for a more serious or alternative lesion, regardless of how the event has been labeled.

Imaging. Frameworks differ in the consequences assigned to a trauma-related structural abnormality. Chapter 4 compares those rules with the ACRM convention used in this book.

Symptoms in the absence of signs. Whether post-concussion symptoms alone can establish mild TBI has been a major source of discrepancy, and the available research evidence does not clearly resolve it [2]. The 2023 ACRM criteria adopt an intermediate position: symptoms alone support suspected mild TBI, whereas two qualifying symptoms combined with an objective finding support the diagnosis itself.

These divergences explain why the same patient may receive a different diagnostic label depending on the framework used.

A note on the 2023 panel and the harmonization effort

The 2023 criteria emerged from a structured consensus process rather than a single authoring group. A Working Group of 17 members conducted rapid evidence reviews on twelve clinical questions, and an external interdisciplinary expert panel of 32 clinician-scientists voted across three Delphi rounds, achieving a 100% response rate in each. Consensus was prespecified as at least 80% agreement. The first round of voting on the diagnostic criteria yielded 75.8% agreement, below the threshold. After revision, the second and third rounds exceeded that threshold. The final criteria achieved 90.7% agreement. Public stakeholder feedback from 68 individuals and 23 organizations was analyzed and incorporated before the third vote [2]. The panel was deliberately assembled to include representatives of most of the organizations whose definitions had competed for the previous three decades, including the sport concussion, military, pediatric, common data elements, and rehabilitation communities. This is why the 2023 criteria are often described as a harmonization effort rather than simply an update of the 1993 definition.

One caution. An organization's representation on a panel is not the same as formal endorsement of the result. The 2023 criteria are an ACRM publication. Whether the WHO Collaborating Centre, the CISG, the VA/DoD, the CDC, and the major pediatric guideline groups will formally adopt them into their own guidelines is a separate process that will unfold over years. At present, most of those organizations still have their own active definitions in place, and clinicians may continue to encounter several frameworks side by side in the literature, research protocols, and medicolegal contexts. The ACRM itself notes that its published guidance is an educational service, is not a statement of the standard of care, and does not mandate the use of any particular diagnostic criteria [2]. The criteria are gaining traction because the panel was so broad, but the harmonization they aspire to remains a work in progress, and they acknowledge their need for validation and revision.

Why the correct diagnosis matters

A confirmed diagnosis changes what happens next in clinical care, research, and medicolegal evaluation. Clinically, it triggers follow-up, education on expected recovery, screening for vestibular, ocular-motor, sleep, and mood symptoms, and timely referral for rehabilitation. In research, the label determines eligibility for study participation and whether datasets can be combined across institutions. This was one reason for developing the CDE framework [24]. Medicolegally, it affects insurance coverage, disability determinations, return-to-work and return-to-play decisions, and litigation outcomes.

The cost of error runs in both directions. When an injury is missed and symptoms are attributed to stress, malingering, or pre-existing psychiatric conditions, patients do not receive appropriate treatment. Their functional decline is then misread as personal failure. Delay in receiving a diagnosis may itself be associated with prolonged recovery [2]. Patients given a diagnosis on insufficient evidence are treated for an injury they may not have, while the condition that actually explains their symptoms goes unaddressed.

A clinical perspective: Julia's story

The following case is fictional. It is constructed from patterns that recur in clinical practice and is not an account of any individual patient.

Julia was a high school mathematics teacher known for making complex equations intuitive. Her life changed during a staff-versus-students basketball game at a school assembly when a portable basketball stand toppled and struck her in the head. Initially, she experienced brief confusion and a persistent headache. Her emergency department evaluation, including a routine CT scan and a standard neurological examination, was documented as normal, and she was discharged with a diagnosis of mild concussion.

In the weeks that followed, skills Julia had taken for granted began to fail her. Equations she had taught for years grew difficult to recall. Lesson planning, once effortless, became exhausting. Her vibrant classroom presence dimmed; her students noticed her visible fatigue and frustration. Unexplained mood swings eroded the patience that had defined her teaching. She felt, as she later described it, like a pale imitation of herself, trapped in a fog she could not explain.

She became irritable and sad. Her self-confidence wavered as mounting medical bills and worsening performance reviews mounted, and she was eventually let go from her position at Oakwood High. Her family, colleagues, and the school administration

attributed her difficulties to stress, aging, pre-existing mental health concerns, or family history, anything but the injury itself.

Determined to find answers, Julia sought specialists who evaluated her persistent symptoms. Videonystagmography, evoked potentials, and quantitative electroencephalography showed abnormalities in the functions those tests sample. Neuropsychological assessment documented cognitive weaknesses, with performance and symptom validity measures within normal limits, making the cognitive results interpretable [25]. Taken together with her history and examination, the findings defined her functional limitations and rehabilitation needs.

Julia's case illustrates several points central to this chapter. Diagnostic frameworks permit a clinical diagnosis even in the absence of visible structural damage [2]. Specialized testing can characterize abnormalities when standard imaging is unremarkable, provided its purpose is stated. Dismissing patient-reported symptoms as psychological or pre-existing without investigation carries real clinical and medicolegal risk, as does attributing every later symptom to the injury without the same investigation.

Her case also illustrates the distinction drawn earlier. Julia's documented brief confusion at the time of injury satisfied the traditional sign-based pathway, so her diagnosis did not depend on the objective testing performed later. Had that acute documentation been absent, her qualifying acute symptoms would have supported a suspected mild TBI, and confirmation under the second pathway would have required an objective finding obtained during the acute period. The abnormalities documented months later served a different and equally important purpose: they provided objective evidence of persistent neurological dysfunction that correlated with her reported impairment. Keeping these two evidentiary roles separate makes the clinical and medicolegal reasoning more accurate, regardless of which side of a dispute is reading it.

Fast take: main points from chapter 3

- The unifying definition of TBI is an alteration in brain function or other evidence of brain pathology caused by an external force. A plausible mechanism and evidence of brain involvement must be established, and competing explanations for clinical findings must be considered [1,2].

- The 2023 ACRM criteria provide three alternative routes to diagnosis: an acute clinical sign; at least two qualifying acute symptoms plus at least one objective acute clinical or

laboratory finding; or unambiguous trauma-related structural neuroimaging evidence. Any one of these routes can establish the diagnosis after a plausible mechanism, with the applicable confounder and mild-severity requirements met [2].

- The criteria separate signs from symptoms. Observed altered mental status is a sign; the subjective feeling of being dazed or confused is a symptom that contributes to the second pathway. An abnormality demonstrated weeks later is not a sign at the time of injury [2].

- Loss of consciousness is not required by any major framework. Symptoms alone support suspected rather than confirmed mild TBI; the second pathway exists because many legitimate injuries occur unwitnessed and unexamined, and it requires objective corroboration because symptoms are less specific than signs [2].

- Objective findings alone never confirm the diagnosis. Criterion 4 describes an acute finding; most such measures lose diagnostic sensitivity over the first 72 hours, so Criterion 4 usually cannot be established post-acutely [2].

- Three timing questions must be kept apart: delayed onset, delayed recognition, and delayed diagnosis. Separately, attribution of symptoms to an already-established injury uses longer windows, such as the four weeks in the ICD-10 research criteria, and does not extend the window for establishing that an injury occurred [2,15].

- Direct head impact is not required. Acceleration-deceleration, rotational loading, and blast are plausible mechanisms; a mechanism alone does not establish the diagnosis [2].

- This book retains the ACRM mild label with a structural-injury qualifier when the clinical severity limits are met [2]; Chapter 4 compares the alternative classifications.

- The label concussion may be used interchangeably with mild TBI when neuroimaging is normal or not clinically indicated, by consensus of 93.8% of the 2023 expert panel [2].

- Definitional heterogeneity has hampered research and contributed to inconsistent care [4], and a missed diagnosis has real consequences for the patient, as Julia’s case illustrates. Severity grading is the subject of Chapter 4.

References

1. Menon DK, Schwab K, Wright DW, Maas AI; Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health. Position statement: definition of traumatic brain injury. Arch Phys Med Rehabil. 2010;91(11):1637-1640. doi:10.1016/j.apmr.2010.05.017.
2. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036.
3. Silverberg ND, Iverson GL; ACRM Mild TBI Definition Expert Consensus Group; ACRM Brain Injury Special Interest Group Mild TBI Task Force. Expert panel survey to update the American Congress of Rehabilitation Medicine definition of mild traumatic brain injury. Arch Phys Med Rehabil. 2021;102(1):76-86. doi:10.1016/j.apmr.2020.08.022.
4. Saatman KE, Duhaime AC, Bullock R, Maas AIR, Valadka A, Manley GT; Workshop Scientific Team and Advisory Panel Members. Classification of traumatic brain injury for targeted therapies. J Neurotrauma. 2008;25(7):719-738. doi:10.1089/neu.2008.0586.
5. Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002-2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.
6. Centers for Disease Control and Prevention. Report to Congress on Traumatic Brain Injury in the United States: Epidemiology and Rehabilitation. Atlanta (GA): National Center for Injury Prevention and Control, Division of Unintentional Injury Prevention; 2015.
7. Sosin DM, Sniezek JE, Waxweiler RJ. Trends in death associated with traumatic brain injury, 1979 through 1992: success and failure. JAMA. 1995;273(22):1778-1780. doi:10.1001/jama.1995.03520460060036.
8. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375-378.
9. Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. J Head Trauma Rehabil. 2006;21(6):544-548.
10. Finkelstein E, Corso PS, Miller TR. The Incidence and Economic Burden of Injuries in the United States. New York: Oxford University Press; 2006.

11. Centers for Disease Control and Prevention. Facts about TBI [Internet]. Atlanta (GA): CDC; 2025 Aug 4 [cited 2026 Oct 24]. Available from: https://www.cdc.gov/traumatic-brain-injury/data-research/facts-stats/index.html
12. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75 Suppl 4:S24-S33. doi:10.1227/NEU.0000000000000505.
13. World Health Organization. Global Status Report on Road Safety: Time for Action. Geneva: WHO; 2009.
14. Mac Donald CL, Johnson AM, Cooper D, Nelson EC, Werner NJ, Shimony JS, Snyder AZ, Raichle ME, Witherow JR, Fang R, Flaherty SF, Brody DL. Detection of blast-related traumatic brain injury in U.S. military personnel. N Engl J Med. 2011;364(22):2091-2100. doi:10.1056/NEJMoa1008069.
15. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research. Geneva: World Health Organization; 1993.
16. Kay T, Harrington DE, Adams R, Anderson T, Berrol S, Cicerone K, Dahlberg C, Gerber D, Goka R, Harley P, Hilt J, Horn L, Lehmkuhl D, Malec J. Definition of mild traumatic brain injury. J Head Trauma Rehabil. 1993;8(3):86-87.
17. Malec JF, Brown AW, Leibson CL, Flaada JT, Mandrekar JN, Diehl NN, Perkins PK. The Mayo classification system for traumatic brain injury severity. J Neurotrauma. 2007;24(9):1417-1424. doi:10.1089/neu.2006.0245.
18. Department of Veterans Affairs, Department of Defense. VA/DoD Clinical Practice Guideline for the Management and Rehabilitation of Post-Acute Mild Traumatic Brain Injury. Version 3.0. Washington (DC): VA/DoD; 2021. Available from: https://www.healthquality.va.gov/guidelines/Rehab/mtbi/
19. Carroll LJ, Cassidy JD, Holm L, Kraus J, Coronado VG; 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. 2004;(43 Suppl):113-125. doi:10.1080/16501960410023877.
20. Holm L, Cassidy JD, Carroll LJ, Borg J; Neurotrauma Task Force on Mild Traumatic Brain Injury of the WHO Collaborating Centre. Summary of the WHO Collaborating Centre for Neurotrauma Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2005;37(3):137-141. doi:10.1080/16501970510027321.
21. American Academy of Neurology, Quality Standards Subcommittee. Practice parameter: the management of concussion in sports (summary statement) [retired]. Neurology. 1997;48(3):581-585.

22. Giza CC, Kutcher JS, Ashwal S, Barth J, Getchius TS, Gioia GA, Gronseth GS, Guskiewicz K, Mandel S, Manley G, McKeag DB, Thurman DJ, Zafonte R. Summary of evidence-based guideline update: evaluation and management of concussion in sports [retired]: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology. 2013;80(24):2250-2257. doi:10.1212/WNL.0b013e31828d57dd.
23. Patricios JS, Schneider KJ, Dvorak J, Ahmed OH, Blauwet C, Cantu RC, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport, Amsterdam, October 2022. Br J Sports Med. 2023;57(11):695-711. doi:10.1136/bjsports-2023-106898.
24. Maas AIR, Harrison-Felix CL, Menon D, Adelson PD, Balkin T, Bullock R, et al. Common data elements for traumatic brain injury: recommendations from the interagency working group on demographics and clinical assessment. Arch Phys Med Rehabil. 2010;91(11):1641-1649. doi:10.1016/j.apmr.2010.07.232.
25. Sweet JJ, Heilbronner RL, Morgan JE, Larrabee GJ, Rohling ML, Boone KB, et al. American Academy of Clinical Neuropsychology (AACN) 2021 consensus statement on validity assessment: update of the 2009 AACN consensus conference statement on neuropsychological assessment of effort, response bias, and malingering. Clin Neuropsychol. 2021;35(6):1053-1106. doi:10.1080/13854046.2021.1896036.
26. Crowe LM, Hearps S, Anderson V, Borland M, Phillips N, Kochar A, et al. Investigating the variability in mild traumatic brain injury definitions: a prospective cohort study. Arch Phys Med Rehabil. 2018;99(7):1360-1369. doi:10.1016/j.apmr.2017.12.026.

Chapter 4 Classifying the severity of traumatic brain injury

Chapter overview

The severity of TBI is graded as mild, moderate, or severe. Four clinical features carry the grading itself: duration of loss of consciousness (LOC), duration of altered mental status, duration of post-traumatic amnesia (PTA), and the Glasgow Coma Scale (GCS) score after stabilization [1]. Structural imaging is a fifth feature. Its role in severity classification is the point of sharpest disagreement among the major frameworks, so this chapter considers it separately. These categories guide acute management, rehabilitation planning, prognosis, research enrollment, and public health surveillance, but they describe the early clinical expression of injury rather than the full biological complexity of what has happened to the brain. Symptoms overlap substantially across severity categories. A severity grade describes the probability of an outcome; it does not determine that outcome [2]. Mild TBI, the central focus of this book, is both the most common category and the most likely to be missed. Routine imaging is typically normal, and symptoms may be subtle, delayed, or attributed to other causes. Following the convention adopted in Chapter 3, this book applies the 2023 ACRM approach to imaging-positive injuries: the mild classification is retained and a structural-injury qualifier is added, rather than the injury being reclassified as moderate [3]. This chapter reviews the criteria used to grade severity, the major severity classification frameworks, the divergent handling of positive neuroimaging, the limitations of severity labels, and the special importance of recognizing that “mild” does not mean “benign.”

From diagnosis to severity

Chapter 3 addressed how the diagnosis of TBI is made. Severity grading is the next step. It uses some of the same clinical features: LOC, alteration of mental status, PTA, and imaging. Severity thresholds are then applied to assign a category of mild, moderate, or severe. The same patient may receive different severity labels depending on the framework used, which is a recurring source of confusion in clinical practice and litigation.

A severity label is not a complete description of the patient’s injury. A person may experience significant cognitive, vestibular, visual, affective, sleep, autonomic, or pain-related impairment whether the injury was labeled mild, moderate, or severe. The three categories are practical clinical divisions superimposed on a continuous spectrum of mechanical force, biological injury, symptom burden, recovery trajectory, and functional consequence [1].

The three severity categories

Mild TBI (concussion)

Mild TBI, often called concussion, usually involves brief or absent loss of consciousness, transient alteration of mental status, relatively subtle neurological findings, and typically normal routine neuroimaging on CT or conventional MRI. The 2023 ACRM criteria formalized the interchangeability of the two terms, with 93.8% of the expert panel agreeing that concussion may be used interchangeably with mild TBI when neuroimaging is normal or not clinically indicated [3]. Older epidemiologic reviews estimated that approximately 80% of TBIs are mild, and population-based studies suggest the proportion is even higher when outpatient, untreated, and unrecognized injuries are captured [4,5].

Despite the term “mild,” many patients experience persistent headaches, dizziness, visual motion sensitivity, photophobia, phonophobia, sleep disruption, fatigue, slowed information processing, memory inefficiency, irritability, anxiety, depression, or reduced tolerance for complex cognitive tasks. These changes are invisible on standard imaging. Chapter 8 examines the advanced imaging evidence and the limits of applying group findings to an individual. The limits of the mild label itself are the subject of the section “What a severity label does and does not tell you” below.

Moderate TBI

Moderate TBI typically involves a longer period of unconsciousness or amnesia, a lower GCS score than mild TBI, or more prominent cognitive and neurological impairments. Imaging may reveal contusions, traumatic subarachnoid hemorrhage, small subdural or epidural collections, edema, diffuse axonal injury, or other structural abnormalities. The early clinical presentation is generally clearer than in mild TBI, but many symptoms still overlap with those seen after mild injury, including headache, dizziness, memory loss, reduced concentration, fatigue, mood disturbance, and intolerance of cognitively demanding environments [1,2].

Severe TBI

Severe TBI is characterized by prolonged unconsciousness or coma and, in catastrophic cases, brain death. The GCS score is usually 8 or lower after initial stabilization and resuscitation. Patients may show widespread deficits including aphasia, dysarthria, motor weakness, spasticity, impaired coordination, profound cognitive deficits, seizures, dysautonomia, neuroendocrine dysfunction, or major behavioral changes.

Severe TBI carries a higher probability of diffuse and persistent disability, but it is a misconception that all severe injuries result in uniformly poor recovery. Individual outcomes after severe TBI vary widely and depend on injury pattern, secondary insults, age, comorbidities, access to acute neurosurgical care, rehabilitation intensity, family support, and other factors [6,7]. Angela's case at the end of this chapter is the illustration.

Criteria used to grade severity

Four clinical features carry the severity grading itself (Table 4.1 and Figure 4.1). Structural imaging is addressed separately in the section that follows, because the frameworks do not agree on what to do with it.

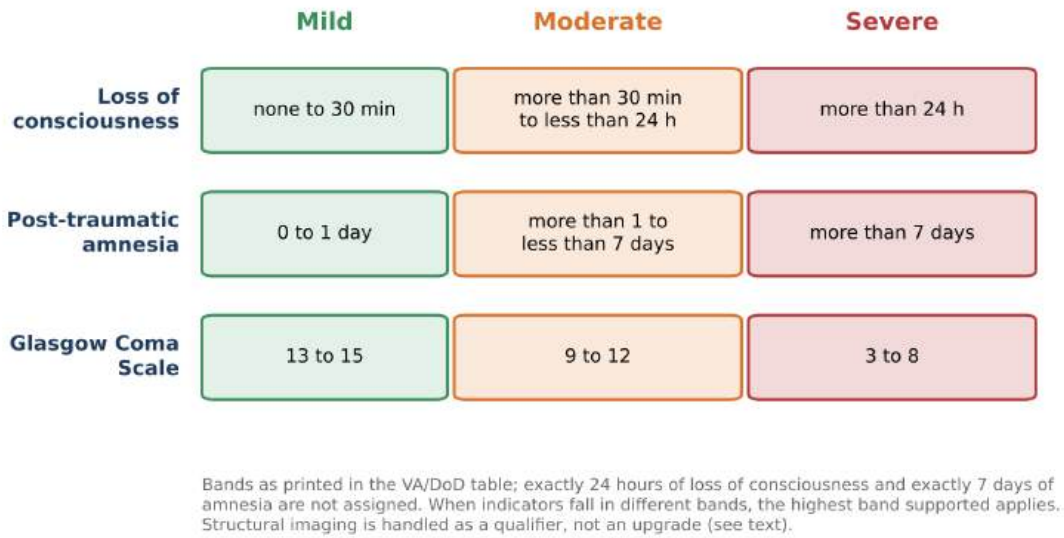


Figure 4.1. The three severity bands for loss of consciousness, post-traumatic amnesia, and the Glasgow Coma Scale, as printed in the VA/DoD table [11]. Exactly 24 hours of loss of consciousness and exactly 7 days of amnesia are not assigned by that table. Original schematic.

Glasgow Coma Scale

The Glasgow Coma Scale evaluates eye opening, verbal response, and motor response, producing a total score from 3 to 15 (Table 4.2) [8,9]. A GCS score of 13 to 15 after stabilization is generally considered mild, 9 to 12 moderate, and 3 to 8 severe.

The GCS is simple, widely taught, and useful for prognosis in many acute settings. Its familiarity can obscure limitations in reliability: Gill and colleagues found exact total-score agreement between examiners of only 32% in their emergency department sample, despite stronger rank correlations [10]. Intoxication, sedation, paralytics,

intubation, aphasia, facial trauma, ocular injury, preexisting neurological disability, young age, and shock all confound the score. It does not measure the cognitive and functional consequences of TBI. It is also not ideal for newborns, young children, or people with preinjury neurological impairments [1]. A GCS of 15 is compatible with a brain injury.

Loss of consciousness

LOC is measured during the immediate period after injury. Historically it was the primary severity marker. Mild TBI is associated with LOC of 30 minutes or less, or none; moderate TBI with LOC of more than 30 minutes but less than 24 hours; and severe TBI with LOC of more than 24 hours. The printed VA/DoD table does not explicitly assign an LOC duration of exactly 24 hours, so a boundary value should be documented and interpreted using the named framework rather than silently rounded into a category [11].

Longer LOC is associated with worse outcomes, particularly in moderate and severe injuries [6,12]. The relationship is not absolute. In a hospital-based mild head-injury sample, loss of consciousness did not reliably predict neuropsychological decrement after concussion, so LOC should not be treated as the sole determinant of severity or prognosis [13].

Post-traumatic amnesia

PTA describes a variable period of confusion after TBI during which the patient is disoriented and has difficulty forming continuous new memories. The Galveston Orientation and Amnesia Test (GOAT) assesses orientation to person, place, time, and events before and after the injury and is commonly used to evaluate PTA at the bedside [14].

The VA/DoD severity table lists PTA of 0 to 1 day for mild TBI, more than 1 and less than 7 days for moderate TBI, and more than 7 days for severe TBI [11]. Exactly 7 days is not explicitly assigned in that table, and the same rule applies: document the exact duration and the framework used. PTA duration is clinically useful in predicting functional and cognitive outcome because it captures the evolution of recovery after the initial event [15,16].

The 2023 ACRM criteria restrict the amnesia that counts for diagnosis to anterograde amnesia, that is, loss of memory for events following the injury; retrograde amnesia may substitute only when post-traumatic amnesia cannot be reliably assessed, for example because of polytrauma or sedating analgesia [3]. That restriction concerns the

diagnostic determination discussed in Chapter 3. The duration thresholds used for severity grading are unchanged.

Alteration of mental status

Alteration of mental status includes confusion, disorientation, feeling dazed, and other changes in mental state related to the injury. The VA/DoD mild band permits alteration lasting up to 24 hours. Beyond 24 hours, severity is determined using the other criteria; duration of altered mental status alone does not distinguish moderate from severe TBI [11].

For diagnostic purposes no minimum duration is specified for a qualifying acute sign, provided it is attributable to the injury and not better explained by a confounder. One qualification carries over from Chapter 3: the 2023 ACRM criteria treat an observed alteration of mental status as a clinical sign and the patient's own report of having felt dazed as a symptom, and the two are not interchangeable when documenting either the diagnosis or its severity [3].

Structural imaging and the imaging-positive mild injury

Mild TBI typically shows normal routine imaging. Moderate TBI may show contusion, hemorrhage, edema, or other traumatic findings. Severe TBI often shows mass lesions, hemorrhage, edema, diffuse injury, or signs of impending herniation. Severity cannot be determined by imaging alone: a patient with substantial functional impairment can have a normal scan, and a patient with imaging abnormalities can recover well.

The difficult case has mild clinical indicators: a GCS of 15, brief or absent LOC, and no significant PTA. Yet structural imaging shows a trauma-related abnormality. That patient receives four different labels across four widely used frameworks (Figure 4.2), because the classification rules differ, even though the patient's clinical status is the same.

The 2023 ACRM criteria retain the mild classification. When CT or structural MRI reveals a trauma-related intracranial abnormality, the qualifier "with neuroimaging evidence of structural intracranial injury" may be applied, and the mild designation is kept. When imaging is performed and is normal, the corresponding "without" qualifier may be used. When imaging is not performed, no qualifier is used. The ACRM favors a qualifier to describe the variation within the mild group, rather than reclassifying these injuries. Its rationale is that this approach matches the largest recent mild TBI cohort studies and will remain workable as MRI sensitivity improves and a growing proportion of injuries become imaging-positive [3].

The complicated versus uncomplicated distinction calls the same patient complicated mild TBI. This terminology originates with Williams, Levin and Eisenberg, who separated mild head injuries with intracranial lesions or depressed skull fracture from those without [17], and was developed further by Iverson, who found poorer performance on some acute neuropsychological tests in the complicated group, with small to medium effects and substantial overlap between groups [18]. The injury remains mild; the modifier carries the prognostic information. The distinction is frequently attributed to the Mayo Classification System. It does not originate there and is not part of it.

The Mayo Classification System reclassifies the patient as definite moderate-severe. Under Mayo, a case is classified as Moderate-Severe (Definite) if any one of the following is present: death due to the TBI, LOC of 30 minutes or more, post-traumatic anterograde amnesia of 24 hours or more, a worst GCS full score below 13 in the first 24 hours, or any of intracerebral hematoma, subdural hematoma, epidural hematoma, cerebral contusion, hemorrhagic contusion, penetrating TBI, subarachnoid hemorrhage, or brainstem injury [19]. A positive scan therefore moves the case out of the mild category outright, regardless of how benign the clinical indicators are.

The VA/DoD framework also reclassifies the injury as moderate or severe when conventional neuroimaging is positive [11].

This book follows the ACRM convention. An imaging-positive injury that otherwise meets mild criteria is reported as mild TBI with neuroimaging evidence of structural intracranial injury, with the term complicated mild TBI noted where it appears in records. This choice has two practical consequences for clinical and medicolegal work. First, interpreting a severity label in a medical record requires identifying the framework that produced it. The same patient may appear as “mild with structural injury” in a civilian neurology record and as “moderate TBI” in a VA record. Second, two of the four frameworks upgrade the injury and two do not. The same injury will therefore read as more severe in some records than in others for reasons that have nothing to do with the patient. Documenting which framework was applied prevents that artifact from being mistaken for either exaggeration or minimization. A worked example of this problem in a contested case appears in Chapter 5.

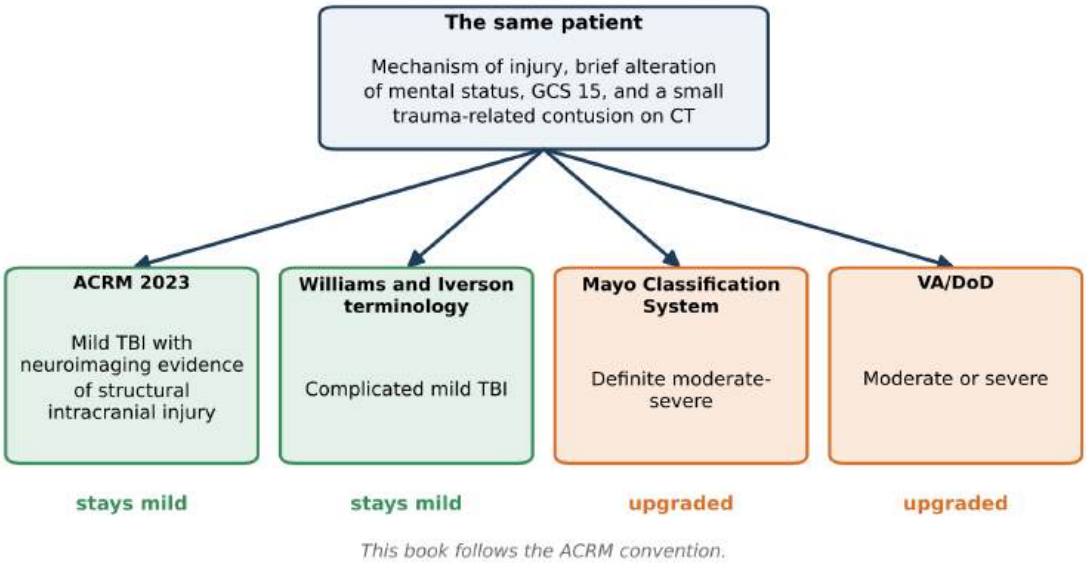


Figure 4.2. One patient, four labels. The same imaging-positive injury with mild clinical indicators is classified differently by four frameworks; two retain the mild label and two upgrade it [3,11,17,18,19]. Original schematic.

Table 4.1. Features used to grade TBI severity

Criterion	Mild	Moderate	Severe
Loss of consciousness	0 to 30 minutes, or none	More than 30 minutes and less than 24 hours	More than 24 hours
Alteration of mental status	Confusion, disorientation, feeling dazed, or other change up to 24 hours	More than 24 hours; distinguish moderate from severe using other criteria	More than 24 hours; distinguish moderate from severe using other criteria
Post-traumatic amnesia	0 to 24 hours	More than 24 hours and less than 7 days	More than 7 days
Glasgow Coma Scale	13 to 15 after stabilization	9 to 12 after stabilization	3 to 8 after stabilization

Criterion	Mild	Moderate	Severe
General rule	Classify as mild only when no moderate or severe criterion is met among the four features above	If any moderate criterion is met, classify as at least moderate	If any severe criterion is met, classify as severe
Structural imaging	Handled as a qualifier rather than an upgrade trigger under the convention adopted here; see the preceding section for the divergent approaches of the ACRM, Williams/Iverson, Mayo, and VA/DoD frameworks		

Table 4.1 legend. The LOC and PTA bands reproduce the VA/DoD table, whose printed inequalities leave exactly 24 hours of LOC and exactly 7 days of PTA unassigned; document such boundary values and the framework applied. When reliable indicators cross categories, use the highest category supported [1,8,11,20].

Table 4.2. The Glasgow Coma Scale

Component	Response	Score
Eye opening (E)	Spontaneous	4
	To sound or speech	3
	To pressure or pain	2
	None	1
Verbal response (V)	Oriented	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Motor response (M)	Obeys commands	6
	Localizes pain	5
	Withdraws from pain or normal flexion	4

Component	Response	Score
	Abnormal flexion	3
	Extension	2
	None	1
Total score	E + V + M; range 3 to 15	Mild 13-15; moderate 9-12; severe 3-8

Table 4.2 legend. Each component is scored as the best response the patient can give at the time of examination. The GCS is useful for acute communication and prognostication, not as a stand-alone measure of the presence, severity, or functional consequences of TBI [1,8,9,10].

Severity classification frameworks

Several organizations have published severity classification schemes. Their underlying logic is similar, but the details differ. Table 4.3 places the thresholds side by side so those differences are easier to follow.

ACRM

The 1993 ACRM definition assigned mild TBI to LOC of 30 minutes or less, PTA of 24 hours or less, and GCS of 13 to 15 after approximately 30 minutes [20]. The 2023 revision retained these upper thresholds for the mild qualifier, dropping the qualifier and diagnosing TBI without it when LOC exceeded 30 minutes, when the GCS remained below 13 after 30 minutes, or when PTA exceeded 24 hours [3]. The ACRM working group called this traditional upper threshold problematic but left it alone, because efforts to replace it with a multidimensional biomarker-based grading system are underway but not yet available [3]. The ACRM framework focuses on diagnosis rather than on severity grading as such, but the same thresholds inform severity bands in clinical practice.

WHO collaborating centre task force

The WHO uses similar mild TBI thresholds: LOC of 30 minutes or less, PTA of less than 24 hours, and GCS of 13 to 15 after 30 minutes or at presentation [21].

VA/DoD

The VA/DoD severity table supplies the LOC and PTA bands used in Table 4.1 and specifies GCS as the best available score in the first 24 hours, with mild 13 to 15, moderate 9 to 12, and severe below 9 [11]. It also excludes clinically relevant structural imaging abnormalities from its mild category.

Mayo Classification System

The Mayo system grades TBI as definite moderate-severe, probable mild, or symptomatic (possible), using imaging findings, GCS, PTA, LOC, and post-concussive symptoms [19]. Its distinctive contribution is that it maximizes the use of whatever positive evidence is available in the record, which allows a larger proportion of cases to be classified than single-indicator systems achieve, with a reported sensitivity of 89% and specificity of 98% for the moderate-severe category [19]. This makes it particularly useful for retrospective research and for record-based classification. As described above, any trauma-related intracranial abnormality places the case in the definite moderate-severe category under Mayo.

Marshall CT Classification

The Marshall CT Classification describes acute CT patterns and their prognostic relevance; it is not a severity scale for mild TBI [22]. Category I has no visible intracranial pathology. Category II permits visible pathology with preserved basal cisterns, midline shift no greater than 5 mm, and no high- or mixed-density lesion larger than 25 cm^3. Category III describes compressed or absent cisterns with shift no greater than 5 mm and no lesion larger than 25 cm^3; category IV describes shift greater than 5 mm without such a large lesion. Categories V and VI distinguish evacuated mass lesions from non-evacuated high- or mixed-density lesions larger than 25 cm^3. The categories stratify risk in the neurosurgical setting; they are not an ascending ladder of clinical severity.

NINDS Common Data Elements

The NINDS CDE framework standardizes how TBI variables are collected for research so that datasets can be pooled across institutions. These variables include LOC, PTA, GCS timing and method, neurological deficits, imaging findings, mechanism of injury, demographics, and outcome measures [7]. It is not a bedside grading system.

CDC surveillance

The CDC surveillance approach uses coded diagnoses and clinical records to estimate TBI incidence, prevalence, mortality, and rehabilitation burden at the population level, without specific severity thresholds [23]. Like the NINDS CDE, it is a public health framework rather than a bedside grading system.

Table 4.3. Severity thresholds across selected expert panels

Source	Loss of consciousness	Glasgow Coma Scale	Post-traumatic amnesia	Positive structural imaging
ACRM 2023 with retained 1993 clinical limits	Mild TBI: 30 minutes or less	Mild TBI: 13-15 after approximately 30 minutes	Mild TBI: 24 hours or less	Mild retained, structural-injury qualifier added
WHO Task Force	Mild TBI: 30 minutes or less	Mild TBI: 13-15 after 30 minutes or at presentation	Mild TBI: less than 24 hours	Intracranial lesion not requiring surgery is compatible with mild
VA/DoD	Mild: 0 to 30 min; moderate and severe: longer durations	Mild: 13 to 15; moderate: 9 to 12; severe: below 9; best available score in first 24 hours	Mild: 0 to 1 day; moderate: >1 and <7 days; severe: >7 days; exactly 7 days unspecified in source table	Reclassified as moderate or severe
Mayo Classification	Probable mild: <30 min; definite moderate-severe: ≥30 min	Probable mild generally 13-15; definite moderate-severe includes worst GCS <13 in first 24 h	Probable mild: <24 h; definite moderate-severe: ≥24 h	Definite moderate-severe
Williams / Iverson	Not a threshold system	Not a threshold system	Not a threshold system	Complicated mild TBI
CDC surveillance	No single severity threshold	No single severity threshold	No single severity threshold	Not used as a threshold

Source	Loss of consciousness	Glasgow Coma Scale	Post-traumatic amnesia	Positive structural imaging
NINDS CDE	Standardized variable, not a threshold system	Standardized variable, not a threshold system	Standardized variable with timing and method specified	Standardized variable

Table 4.3 legend. ACRM, WHO, VA/DoD, and Mayo provide explicit severity thresholds; the Williams and Iverson terminology is a within-mild descriptor rather than a grading system; CDC surveillance and NINDS CDE serve population-level and research-harmonization purposes. The final column is the principal reason the same patient may be assigned different severity labels depending on the framework used [2,3,7,11,17,18,19,20,21,23].

What a severity label does and does not tell you

The word “mild” describes the early clinical presentation. It does not describe the eventual burden, and the gap between the two is the single most consequential misunderstanding in this field.

Severity grades are useful when looking at groups of patients. Greater initial severity brings greater risk of persistent cognitive impairment, with the strength of the relationship varying by domain, follow-up interval, and study population [2,6,12]. Moderate and severe injury produce documented deficits in fluency, attention, and processing speed [24,25,26]; mild injury produces a more heterogeneous pattern, with most patients recovering well and subtle or persistent deficits in some cohorts [27,28]. Emotional symptoms remain substantial across severity groups [29], and subjective cognitive complaints and measured performance do not always agree [30]. For these reasons, severity grading has a useful role in risk stratification, research, and resource planning.

At the level of the individual patient it performs much less well, for three reasons.

The same deficit has different consequences in different lives. Subtle executive dysfunction may end the career of a physicist, surgeon, attorney, or accountant, and go largely unnoticed in a person whose work is repetitive and externally structured. Oculomotor and visual tracking impairment may make driving unsafe and matter little to someone who does not drive. Photophobia, motion sensitivity, and dizziness can make a supermarket intolerable while leaving the neurological examination essentially

normal. Severity grading measures the injury; disability arises from the interaction between the injury and the life it lands in.

Modest deficits accumulate. Clinicians find the obvious deficits of severe injury easier to identify than the subtler combination that characterizes mild and moderate injury. Yet the cumulative effect of cognitive, vestibular, visual, behavioral, sleep-related, autonomic, and pain-related impairment can be profoundly disabling even when no single component is severe. Behavioral change compounds this: depression, anxiety, irritability, emotional lability, impulsivity, apathy, and frustration intolerance interfere with daily function and may become the dominant source of disability regardless of the initial grade.

Many factors that influence recovery are not captured by the severity grade. Two patients with imaging-negative, GCS-15, brief-LOC injuries may follow entirely different trajectories depending on injury location, network vulnerability, premorbid cognitive demand, psychiatric history, sleep disturbance, pain, vestibular injury, visual dysfunction, autonomic disturbance, prior concussions, and access to rehabilitation. The complicated versus uncomplicated distinction captures part of this variability [17,18], but much remains outside that distinction. Any use of severity to predict outcome must also incorporate age, premorbid function, extracranial trauma, hypoxia, hypotension, seizures, intoxication, medications, psychiatric comorbidity, sleep disorder, pain, litigation context, social support, and rehabilitation access [7].

Two practical conclusions follow, and they point in opposite directions. A severity grade should never be used to dismiss a patient's symptoms or functional limitations, because a mild grade is compatible with severe and lasting disability. And a severe grade should not be read as a settled prognosis, because recovery after severe injury is more variable than is commonly assumed. Chapter 6 takes up what does and does not predict outcome, and why subclassification has performed better at directing treatment than at forecasting recovery.

Repeated injuries and neurodegenerative risk

Repeated head impacts and repeated TBIs warrant careful assessment because cumulative exposure may be associated with long-term neurological risk. Observational studies link a history of TBI with subsequent neurodegenerative diagnoses, including Parkinson disease, but association does not by itself establish causation or predict an individual's outcome [31,32,33]. Chronic traumatic encephalopathy is a neuropathological diagnosis associated with repetitive head-impact exposure; it should not be inferred from persistent symptoms or from a single concussion. Mechanisms,

dose-response relationships, confounding, and individual susceptibility remain under investigation.

Two clinical illustrations

The following cases are fictional. They are constructed from patterns that recur in clinical practice and are not accounts of individual patients.

David: when mild is anything but

David, a 35-year-old accountant, slipped on icy pavement and briefly lost consciousness. His computed tomography scan in the emergency department was clear, and he was given a diagnosis of mild concussion. He went on to develop chronic headache, severe light sensitivity, difficulty processing complex tasks, and mood swings that cost him his job. Only after specialized neurological testing, including evoked potentials and vestibular assessment, did he receive targeted rehabilitation. His therapists worked with him to identify his limitations and develop compensatory strategies, which allowed him to reclaim part of his working life, though he remained limited.

David's case illustrates the first conclusion above. His initial grade was accurate, but it did not capture the later functional burden, because the functions affected were central to his occupation.

Angela: defying the odds

Angela, a 27-year-old student, faced a grim prognosis after a motorcycle crash. Her initial GCS was 7 and computed tomography showed brain swelling. After aggressive intensive care and months of rehabilitation, she returned to university. She continues to have executive dysfunction, but otherwise she is doing well.

Angela's case illustrates the second conclusion. Her severe grade correctly identified a high-risk injury, but it did not determine how she would recover. At both the severe and mild ends of the spectrum, the classification describes the initial injury and leaves room for a range of outcomes.

Fast take: main points from chapter 4

• **Three severity levels.** TBI is classified as mild, moderate, or severe. Four features carry the grading: LOC, duration of alteration of mental status, PTA, and GCS.

• **Structural imaging is handled separately.** The frameworks disagree on it, and that disagreement is the main reason the same patient receives different severity labels.

• Integrate the criteria. No single feature describes the whole injury. When reliable indicators fall into different severity bands, use the highest band supported.

• **GCS thresholds.** Mild 13-15. Moderate 9-12. Severe 3-8.

• LOC and PTA thresholds in the VA/DoD table. LOC: mild 0 to 30 minutes, moderate more than 30 minutes and less than 24 hours, severe more than 24 hours. PTA: mild 0 to 1 day, moderate more than 1 and less than 7 days, severe more than 7 days. Exactly 24 hours of LOC and exactly 7 days of PTA are not explicitly assigned [11].

• **Imaging-positive mild injury, four labels.** ACRM retains mild and adds a structural-injury qualifier; the Williams and Iverson terminology calls it complicated mild TBI (a distinction that does not come from Mayo); Mayo classifies it as definite moderate-severe; VA/DoD reclassifies it as moderate or severe [3,11,17,18,19]. This book follows the ACRM convention.

• **Mild TBI is the most common and most missed.** Symptoms are nonspecific, routine imaging is often normal, and consequences may emerge only when patients return to demanding environments.

• **Severity is probabilistic, not deterministic.** Individual outcomes vary widely; severity classification should never be used to dismiss a patient's symptoms or functional limitations.

• **Repeated TBI matters.** Cumulative injury may amplify symptoms and is associated with increased risk of later-life neurodegenerative disease.

• **Loss of consciousness is not required by any framework,** and a normal scan does not exclude injury. Under the 2023 criteria a report of feeling dazed is a symptom, not a sign, and supports diagnosis through the second pathway described in Chapter 3 [3].

• **Severity describes the starting position, not the destination.** A mild grade is compatible with lasting disability, and a severe grade is not a settled prognosis.

References

1. Saatman KE, Duhaime AC, Bullock R, Maas AIR, Valadka A, Manley GT; Workshop Scientific Team and Advisory Panel Members. Classification of traumatic brain injury for targeted therapies. J Neurotrauma. 2008;25(7):719-738. doi:10.1089/neu.2008.0586.

2. Dikmen SS, Corrigan JD, Levin HS, Machamer J, Stiers W, Weisskopf MG. Cognitive outcome following traumatic brain injury. J Head Trauma Rehabil. 2009;24(6):430-438. doi:10.1097/HTR.0b013e3181c133e9.

3. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036.

4. Bruns J Jr, Hauser WA. The epidemiology of traumatic brain injury: a review. Epilepsia. 2003;44(Suppl 10):2-10. doi:10.1046/j.1528-1157.44.s10.3.x.

5. Feigin VL, Theadom A, Barker-Collo S, Starkey NJ, McPherson K, Kahan M, et al. Incidence of traumatic brain injury in New Zealand: a population-based study. Lancet Neurol. 2013;12(1):53-64. doi:10.1016/S1474-4422(12)70262-4.

6. Dikmen SS, Machamer JE, Powell JM, Temkin NR. Outcome 3 to 5 years after moderate to severe traumatic brain injury. Arch Phys Med Rehabil. 2003;84(10):1449-1457. doi:10.1016/S0003-9993(03)00287-9.

7. Maas AIR, Harrison-Felix CL, Menon D, Adelson PD, Balkin T, Bullock R, et al. Common data elements for traumatic brain injury: recommendations from the interagency working group on demographics and clinical assessment. Arch Phys Med Rehabil. 2010;91(11):1641-1649. doi:10.1016/j.apmr.2010.07.232.

8. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet. 1974;2(7872):81-84. doi:10.1016/S0140-6736(74)91639-0.

9. Teasdale G, Jennett B. Assessment and prognosis of coma after head injury. Acta Neurochir (Wien). 1976;34(1-4):45-55. doi:10.1007/BF01405862.

10. Gill MR, Reiley DG, Green SM. Interrater reliability of Glasgow Coma Scale scores in the emergency department. Ann Emerg Med. 2004;43(2):215-223. doi:10.1016/S0196-0644(03)00814-X.

11. Department of Veterans Affairs, Department of Defense. VA/DoD Clinical Practice Guideline for the Management and Rehabilitation of Post-Acute Mild Traumatic Brain Injury. Version 3.0. Washington (DC): VA/DoD; 2021.

12. Sherer M, Sander AM, Nick TG, High WM Jr, Malec JF, Rosenthal M. Early cognitive status and productivity outcome after traumatic brain injury: findings from the TBI Model Systems. Arch Phys Med Rehabil. 2002;83(2):183-192. doi:10.1053/apmr.2002.27497.

13. Lovell MR, Iverson GL, Collins MW, McKeag D, Maroon JC. Does loss of consciousness predict neuropsychological decrements after concussion? Clin J Sport Med. 1999;9(4):193-198. doi:10.1097/00042752-199910000-00002.

14. Levin HS, O'Donnell VM, Grossman RG. The Galveston Orientation and Amnesia Test: a practical scale to assess cognition after head injury. J Nerv Ment Dis. 1979;167(11):675-684. doi:10.1097/00005053-197911000-00004.
15. Zafonte RD, Mann NR, Millis SR, Black KL, Wood DL, Hammond FM. Posttraumatic amnesia: its relation to functional outcome. Arch Phys Med Rehabil. 1997;78(10):1103-1106. doi:10.1016/S0003-9993(97)90135-0.
16. Parker TD, Rees R, Rajagopal S, Griffin C, Goodliffe L, Dilley M, Jenkins PO. Post-traumatic amnesia. Pract Neurol. 2022;22(2):129-137. doi:10.1136/practneurol-2021-003056.
17. Williams DH, Levin HS, Eisenberg HM. Mild head injury classification. Neurosurgery. 1990;27(3):422-428. doi:10.1097/00006123-199009000-00014.
18. Iverson GL. Complicated vs uncomplicated mild traumatic brain injury: acute neuropsychological outcome. Brain Inj. 2006;20(13-14):1335-1344. doi:10.1080/02699050601082156.
19. Malec JF, Brown AW, Leibson CL, Flaada JT, Mandrekar JN, Diehl NN, Perkins PK. The Mayo classification system for traumatic brain injury severity. J Neurotrauma. 2007;24(9):1417-1424. doi:10.1089/neu.2006.0245.
20. Kay T, Harrington DE, Adams R, Anderson T, Berrol S, Cicerone K, Dahlberg C, Gerber D, Goka R, Harley P, Hilt J, Horn L, Lehmkuhl D, Malec J. Definition of mild traumatic brain injury. J Head Trauma Rehabil. 1993;8(3):86-87.
21. Carroll LJ, Cassidy JD, Holm L, Kraus J, Coronado VG; 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. 2004;(43 Suppl):113-125. doi:10.1080/16501960410023877.
22. Marshall LF, Marshall SB, Klauber MR, Van Berkum Clark M, Eisenberg HM, Jane JA, Luerssen TG, Marmarou A, Foulkes MA. The diagnosis of head injury requires a classification based on computed axial tomography. J Neurotrauma. 1992;9(Suppl 1):S287-S292.
23. Centers for Disease Control and Prevention. Report to Congress on Traumatic Brain Injury in the United States: Epidemiology and Rehabilitation. Atlanta (GA): National Center for Injury Prevention and Control, Division of Unintentional Injury Prevention; 2015.
24. Ruff RM, Evans R, Marshall LF. Impaired verbal and figural fluency after head injury. Arch Clin Neuropsychol. 1986;1(2):87-101.
25. Bate AJ, Mathias JL, Crawford JR. Performance on the Test of Everyday Attention and standard tests of attention following severe traumatic brain injury. Clin Neuropsychol. 2001;15(3):405-422. doi:10.1076/clin.15.3.405.10279.

26. Incoccia C, Formisano R, Muscato P, Reali G, Zoccolotti P. Reaction and movement times in individuals with chronic traumatic brain injury with good motor recovery. Cortex. 2004;40(1):111-115. doi:10.1016/S0010-9452(08)70924-9.
27. Vanderploeg RD, Curtiss G, Belanger HG. Long-term neuropsychological outcomes following mild traumatic brain injury. J Int Neuropsychol Soc. 2005;11(3):228-236. doi:10.1017/S1355617705050289.
28. Levin HS, Mattis S, Ruff RM, Eisenberg HM, Marshall LF, Tabaddor K, High WM Jr, Frankowski RF. Neurobehavioral outcome following minor head injury: a three-center study. J Neurosurg. 1987;66(2):234-243. doi:10.3171/jns.1987.66.2.0234.
29. Rapoport M, McCauley S, Levin H, Song J, Feinstein A. The role of injury severity in neurobehavioral outcome 3 months after traumatic brain injury. Neuropsychiatry Neuropsychol Behav Neurol. 2002;15(2):123-132.
30. Jamora CW, Young A, Ruff RM. Comparison of subjective cognitive complaints with neuropsychological tests in individuals with mild vs more severe traumatic brain injuries. Brain Inj. 2012;26(1):36-47. doi:10.3109/02699052.2011.635352.
31. Gardner RC, Byers AL, Barnes DE, Li Y, Boscardin J, Yaffe K. Mild TBI and risk of Parkinson disease: a Chronic Effects of Neurotrauma Consortium study. Neurology. 2018;90(20):e1771-e1779. doi:10.1212/WNL.0000000000005522.
32. Brett BL, Gardner RC, Godbout J, Dams-O'Connor K, Keene CD. Traumatic brain injury and risk of neurodegenerative disorder. Biol Psychiatry. 2022;91(5):498-507. doi:10.1016/j.biopsych.2021.05.025.
33. Barker S, Paul BD, Pieper AA. Increased risk of aging-related neurodegenerative disease after traumatic brain injury. Biomedicines. 2023;11(4):1154. doi:10.3390/biomedicines11041154.

Chapter 5 Diagnostic challenges of mild traumatic brain injury

Chapter overview

Mild traumatic brain injury, often called concussion, is one of the most frequently missed neurological diagnoses in acute care. This often reflects the demands of emergency care rather than careless practice. Emergency departments are designed to identify immediately life-threatening injuries. Their routines do not always capture brief confusion, transient disorientation, short-lived memory gaps, delayed headache, visual motion intolerance, subtle vestibular dysfunction, cognitive fatigue, or early personality change.

This chapter explains why mild traumatic brain injury is often missed at the first medical encounter and why the diagnosis is therefore often made retrospectively. It then outlines what a careful evaluation requires: a detailed history, collateral interviews, record review, disciplined attention to confounding conditions, and selected ancillary testing. It also examines the limits of rigid criteria, including the problem of delayed-onset post-traumatic headache. This chapter examines the 2025 medicolegal clarification in detail, including the meaning of observable signs, suspected mTBI, and the distinction between injury diagnosis and attribution of persistent symptoms [27].

Under-recognition of mild TBI in acute care was one problem the revised diagnostic criteria addressed. That revision provides the framework for this chapter. The 2023 ACRM criteria, set out in full in Chapter 3, allow a qualifying acute sign to be established retrospectively and provide additional pathways when no such sign can be established [1,27]. The task is to reconstruct the injury carefully, identify the pathway the evidence supports, and state any remaining uncertainty. A clinician or reviewer working from the current structure is better equipped than one working from the 1993 definition, regardless of which conclusion the evidence supports.

Under-recognition of mild traumatic brain injury in acute settings

Emergency departments and urgent care clinics appropriately prioritize injuries that can cause severe impairment or death. Open fractures, internal bleeding, spinal, chest, and abdominal trauma, and airway compromise demand immediate attention. In that setting, mild traumatic brain injury can fall through the cracks. A patient may be awake, speaking, and moving all extremities, while the underlying brain injury is masked by neck pain, back pain, lacerations, orthopedic injury, shock, or emotional distress.

The scale of the problem is documented. In a prospective study, Powell and colleagues found that 56% of emergency department patients who met Centers for Disease Control

and Prevention criteria for mild traumatic brain injury had no mild TBI diagnosis in their emergency department records [2]. In a later cohort of motor vehicle collision patients with chronic pain, Peixoto and colleagues found that many had likely sustained a mild traumatic brain injury that was not diagnosed at the acute visit [3]. Therefore, the absence of an emergency department concussion diagnosis is weak evidence that no concussion occurred.

Five factors contribute. First, the abnormalities may be brief. Confusion, disorientation, a dazed feeling, a memory gap, or slowed responses may resolve before the patient is examined. In contrast, they can also develop after the patient is released from care. A chart note reading "alert and oriented" at hour 3 says nothing about minute 3 or the next day. Second, routine CT is performed to exclude bleeding, fractures, and mass effect. Mild traumatic brain injury is usually a functional disturbance that CT cannot detect. Third, symptoms may be delayed. The CDC notes that some appear immediately, while others may not appear for hours or days [4]. A patient who leaves with neck and shoulder pain may develop headache, dizziness, light sensitivity, poor concentration, and irritability over the following days. (The distinctions among delayed onset, delayed recognition, and delayed diagnosis are set out in Chapter 3 and apply throughout this chapter.) Fourth, patients may not recognize their own symptoms. They may be frightened, medicated, distracted by pain, or eager to go home. Many do not know that feeling dazed or being unable to remember the event can indicate a brain injury. Fifth, the people who know most may never be asked: family members, coworkers, and witnesses who saw the patient repeat questions, act confused, respond slowly, or behave unlike himself.

A case in the emergency department

Daniel was a thirty-eight-year-old construction supervisor, husband, and father of two. On a Thursday afternoon, his pickup was rear-ended at moderate speed by a delivery van that failed to brake in time. His airbag deployed. His face hit the deflating bag, and his head snapped forward and back. He was not knocked unconscious. He climbed out of the truck on his own, spoke with the police officer, and called his wife. By the time paramedics arrived, he was sitting on the curb with a bleeding lip and complaining of neck pain. The emergency medical services run sheet listed him as alert, ambulatory, and oriented. In the mental status box, the medic wrote “GCS 15.”

In the emergency department, the focus was clear. Cervical collar. CT of the head and cervical spine. Sutures for a forehead laceration. Ketorolac for neck pain. The CT was read as normal. The discharge diagnosis was cervical strain and facial laceration. The

instruction sheet said “return for worsening headache.” There was no mention of concussion.

His wife arrived at the hospital after he had already been examined. She was the one who noticed something was wrong. He asked her three times in ten minutes how the children were getting home from school. He kept reaching for his phone in a pocket that no longer held one. When the discharge nurse explained the wound care instructions, Daniel nodded along and then asked her, almost word for word, the same questions she had just answered. His wife mentioned this to the nurse twice. The nurse said it was probably the pain medication.

Ketorolac is not a sedative. The repeat questioning, the lost phone, and the inability to retain new information for ten minutes were visible signs of the brain injury that the chart did not capture. Two months later, when his wife brought him to a neurologist because he had been written up at work for arithmetic errors he had never made before, was falling asleep at job sites, and was losing his temper with his crew, the emergency department record still read “GCS 15, no LOC, no concussion.” The history his wife gave that afternoon, recorded for the first time on paper, was what allowed the diagnosis to be made.

The injury had observable signs that were simply not documented in the chart. What the wife observed were Criterion 2 clinical signs, namely repeated questioning and failure to retain new information, as established by a collateral witness report. They were not symptoms. Daniel satisfied the traditional sign-based pathway on the day of injury, and the only thing missing was a clinician who asked the person standing next to him (Figure 5.1).

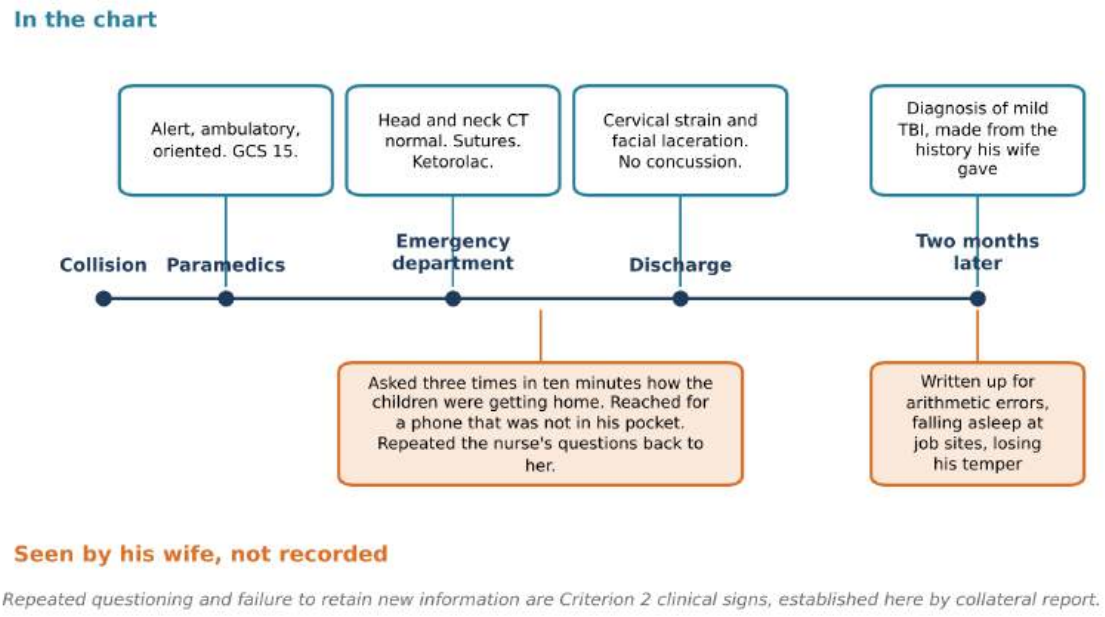


Figure 5.1. Daniel's injury on two timelines. What the chart recorded, above the line, and what his wife observed, below it. The repeated questioning and the failure to retain new information were Criterion 2 clinical signs, established by collateral report two months later. Pappolla ® original schematic.

Table 5.1. Why mild traumatic brain injury is missed during acute care

Diagnostic problem	**Why it happens**	**Clinical consequence**	**Practical correction**
Distracting injuries	Painful orthopedic, spine, facial, or soft tissue injuries dominate the encounter	Brain injury features are not elicited or documented	Ask directly about confusion, dazed feeling, amnesia, repeated questions, dizziness, headache, visual symptoms, and sensory intolerance
Normal CT scan	CT is used mainly to detect acute hemorrhage, fracture, or mass effect	Patient is told there is no brain injury	Explain that a normal CT does not exclude concussion

Diagnostic problem	Why it happens	Clinical consequence	Practical correction
No documented loss of consciousness	Older habits overemphasized loss of consciousness	Concussion is missed when the patient was dazed, confused, or amnestic but did not black out	Recognize a qualifying observable alteration of mental status or acute amnestic episode as Criterion 2 evidence, even if established later through a reliable account; apply the mechanism and confounding requirements [1,27].
Symptoms appear later	Headache, dizziness, fatigue, cognitive problems, mood changes, or sleep symptoms may develop after discharge	Patient does not connect symptoms to the injury	Provide written concussion precautions and follow-up instructions; document the first 72 hours carefully [1,4]
Patient underreports symptoms	Pain, stress, medication, shock, limited insight, or poor understanding reduce reporting	Important features remain undocumented	Use targeted questioning and collateral history
Incomplete records	EMS, nursing, triage, and witness observations may not be integrated	Later reviewers wrongly assume no concussion occurred	Review all contemporaneous records, not only the physician summary
Objective testing not performed acutely	Cognitive, balance, and oculomotor testing is rarely available in the emergency department	Acute abnormalities may be missed, reducing later diagnostic certainty	Where concussion is suspected, arrange objective assessment within the first days rather than weeks [1]

What the 2023 criteria changed about this problem

The 2023 revision addresses several problems that earlier writing on missed concussion approached through flexible clinical application. It provides additional diagnostic routes while expressly preserving clinical judgment when a case does not clearly fit the criteria [1, p.1353]. An incomplete record does not close the sign-based route: a reliable retrospective history may establish a qualifying acute sign that no clinician documented. When no such sign can be established, at least two qualifying acute symptoms, together with an appropriate acute examination or laboratory finding, may support the second pathway; unambiguous trauma-related structural imaging provides another. The suspected category addresses specified situations in which the full criteria are not met or confounding remains uncertain [1;27, p.1618]. For the clinician facing an incomplete record, the task is to examine every applicable pathway. Rejecting the diagnosis because the chart contains no sign overlooks the possibility of recovering one through history. Asserting that the full criteria are met without identifying the supporting evidence makes the opposite error. The suspected label and the clinician's separately reasoned degree of confidence should also be kept distinct.

Retrospective diagnosis and the neurologist's approach

Because mild traumatic brain injury is so often missed acutely, neurologists frequently make the diagnosis retrospectively. This is unremarkable in clinical neurology. Transient ischemic attack, syncope, seizure, migraine aura, neurodegenerative diseases, lacunar strokes, and transient confusional episodes are routinely diagnosed after the event has passed by reconstructing it from history, witness accounts, contemporaneous records, and the later course.

The ACRM directly addresses retrospective diagnosis. Mechanism, clinical signs, and acute symptoms (Criteria 1, 2, and 3) can be established retrospectively through detailed history taking, with attention to recall bias, response bias, and confounders. Newly administered post-acute tests usually cannot establish Criterion 4; the diagnostic performance of most available cognitive, balance, oculomotor, and blood-based measures declines after the acute period. An acute result recovered from earlier records remains acute evidence. Most patients presenting post-acutely do not require structural imaging, and when imaging is obtained, it is usually normal, limiting its contribution to Criterion 5 [1, p.1352]. The later section on ancillary testing examines the timing distinction alongside the authors' explicit guidance to use clinical judgment. This timing limitation does not make later neurological findings irrelevant. The ancillary-testing section below explains how they may corroborate a retrospective clinical opinion while preserving their actual evidentiary meaning.

A careful retrospective evaluation asks targeted questions. Was there any period of not knowing where you were, or not understanding what had happened? Did anyone have to repeat things to you? Is there a gap in your memory, and where does it begin and end? Did you feel dazed or confused, and for how long? Did anyone observe abnormal behavior? Was there vomiting, dizziness, imbalance, visual blurring, light or sound sensitivity, or unusual sleepiness in the first three days? Did symptoms worsen when you returned to work, school, driving, exercise, screen use, or visually complex environments?

The phrasing matters because a sign must have been observable, but need not actually have been witnessed. A subjective feeling of confusion differs from an inability to understand where one was or to follow a conversation. Begin with what the person remembers doing, saying, and understanding. The 2025 clarification also permits hypothetical prompts to clarify what someone could have observed; the account must still describe a qualifying disturbance during the actual event. The expanded discussion below explains how to use these sources without confusing remembered behavior with an assumed sign [1;27, pp.1617-1618].

Structured interviews improve the reliability of retrospective identification. Corrigan and Bogner developed the Ohio State University Traumatic Brain Injury Identification Method to elicit lifetime history of traumatic brain injury through structured questioning [6]. Asking only “were you ever knocked unconscious?” misses most mild traumatic brain injuries; the better approach asks about blows to the head, sudden acceleration or deceleration, falls, being dazed, memory gaps, confusion, and the symptoms that followed.

Retrospective diagnosis requires careful attention to the limits of memory. Stress, time, litigation, pain, and distress all shape what a patient recalls. The neurologist seeks and reviews available emergency medical services records, emergency department and triage notes, nursing notes, imaging reports, therapy records, and early primary care records. Entries such as “slow to respond,” “repeated questions,” or “does not remember accident” may help establish a sign even when no concussion diagnosis was entered. An isolated word such as “dazed” requires clarification. The absence of records does not make a retrospective diagnosis impossible [27, p.1618]. The ACRM acknowledges the risk of both false positive and false negative diagnoses in post-acute assessment [1]. Stating that risk plainly strengthens the credibility of the clinical opinion.

The clinical judgment has to bring the whole record together. A single phrase cannot carry that judgment. “No loss of consciousness” does not mean no brain injury.

“Glasgow Coma Scale 15” does not mean no concussion, and “normal CT” does not mean normal brain function. An isolated notation of “dazed” does not complete a diagnosis either. What is required is a coherent account of mechanism, acute manifestations and their evidentiary character, symptom evolution, and the exclusion of alternative explanations.

The 2025 medicolegal clarification

In 2025, Iverson and colleagues addressed the problem at the center of many contested evaluations: how to apply the ACRM criteria when the clinician first sees the patient long after the injury [27]. The article was written by a subgroup of the ACRM Mild TBI Task Force. It explains the medicolegal use of the 2023 criteria; it does not replace them with a new set. Its practical importance lies in separating the occurrence of an acute manifestation from the date on which the evidence for it is obtained.

Observable does not mean witnessed

A clinical sign must describe something that could have been observed, but someone need not actually have witnessed or recorded it. The 2025 paper expressly permits diagnosis weeks, months, or years after injury through a detailed interview and review of other information when available. Direct observation, recordings, witnesses, and acute records can establish a sign. When these sources are unavailable, the person's own account may establish it, provided the account describes a qualifying observable disturbance, and confounders are addressed [27, pp.1617-1618]. The sign-based pathway therefore remains available even when the emergency record contains no concussion diagnosis and no witness can be located.

The distinction becomes clear when memory is examined carefully. A person who recalls nothing between the collision and the hospital may describe the interval as being unconscious. Records showing that the person walked, spoke, and repeatedly asked the same questions would instead support post-traumatic amnesia. The person was awake but was not forming continuous memories. A later interview can identify that acute sign even if the original chart never named it [27, p.1618].

The paper also allows hypothetical prompts to clarify what another person might have observed when no witness account is available. These prompts should illuminate the actual event. A recollection of being unable to understand where one was has a different evidentiary character from feeling shaken, frightened, or mentally foggy. The clinician records the described behavior, whether remembered directly or learned from someone else, and explains why it meets the operational definition. A hypothetical

answer is not itself an eyewitness observation. Likewise, absent documentation is different from an explicit contemporaneous denial or a contradictory observation; disagreements in the record still require explanation [27, p.1618].

Symptoms must belong to the acute period

Retrospective diagnosis changes the date of assessment, not the required timing of the manifestations. The 2025 clarification places qualifying clinical signs in the immediate aftermath and requires qualifying symptoms to have been present to some degree within the first 3 days. Subjective mental-status symptoms belong to the immediate aftermath; other symptoms can evolve over hours, be masked by surgery, anesthesia, or analgesia, or become more obvious with physical or mental exertion. Those possibilities call for careful reconstruction; they do not establish earlier symptoms merely because later complaints exist. The authors distinguish genuinely new symptoms arising several days or weeks later and recognize that an evolving injury, such as an enlarging traumatic hematoma, can produce later manifestations [1;27, p.1618]. The symptom-timing rule should also be kept separate from the test-specific sensitivity discussion for Criterion 4 below.

Suspected mTBI and clinical probability

The suspected category applies to specified situations in which the full criteria are not met, or confounding remains unresolved. It does not assign a probability above or below 50%. Iverson and colleagues state that a clinician may consider an injury possible, probable, or very likely on the available evidence even though the full criteria are not met [27, p.1618]. A report should therefore identify the criteria classification and then explain the clinician's degree of confidence separately. Calling an injury suspected does not automatically defeat a more-likely-than-not opinion; neither does the label establish that opinion.

Consider a plausible injury followed by several qualifying acute symptoms, with no clinical sign established and no usable acute examination or imaging finding. The criteria may support suspected mTBI. Confidence in that conclusion depends on the quality of the history, its consistency with other evidence, and competing explanations. The same distinction applies when intoxication or acute stress makes the cause of a sign uncertain. Criterion 6 asks whether confounders fully account for the manifestations needed for diagnosis. Their mere presence does not answer that question [27, p.1618]. The paper states that suspected mTBI is managed clinically as mTBI; that approach to care does not establish a legal level of certainty [27, p.1619].

Diagnosing the injury and explaining its consequences

The criteria answer whether an injury occurred. They do not, by themselves, determine why a patient still has headache, dizziness, poor concentration, or sleep disturbance months later. Nor were they designed to predict recovery. The 2025 paper is explicit on both points: meeting the criteria does not establish that future health problems are caused by the injury, and it does not mean that the consequences must be slight or short-lived [27, p.1619].

For the clinician, this means making the reasoning visible in stages. First identify the evidence for the original TBI and the information supporting its severity classification. Then characterize the current disorders, their functional effects, and their likely relationship to that injury. Premorbid function, the chronology of change, the course of symptoms, later injuries, treatment responses, and competing neurological, cervical, vestibular, sleep, and psychiatric conditions all matter to the second inquiry. A well-supported injury diagnosis and a well-supported opinion about persistent impairment are related conclusions, but each needs its own evidence.

Scrutiny applies to both sides of the interview

The article discusses bias in the person being evaluated and in the examiner. Recall, language comprehension, health literacy, education, mental health, and motivations can shape an account. The examiner's experience, questioning style, expectations, and threshold for accepting an answer can shape the diagnosis [27, pp.1618-1619]. Useful safeguards include an unprompted account before focused clarification, documentation of the patient's actual words and their source, examination of conflicting evidence, and the same evidentiary standards whichever conclusion they support. A structured interview can make the process more consistent; it cannot eliminate these judgments.

Appendix C brings together the diagnostic pathways, signs, and acute symptoms illustrated in this article, with the supporting definitions from the 2023 publication. It also summarizes the retrospective and medicolegal qualifications. Appendix B supplies the practical intake and coding worksheets and links to the official structured interview package discussed next [1,27,28].

The ACRM structured interview and its companion forms

The next practical development aimed to make the diagnostic history more systematic. Lange, Gillow, and the ACRM Mild TBI Task Force developed the ACRM Structured TBI Interview to organize the information needed to apply the 2023 criteria [28]. The article

appeared online in November 2025 and in a 2026 journal issue. The package contains the full interview, an Administration Guide, a Diagnostic Coding Form, a Diagnostic Flow Diagram, and a Short Form. Each serves a different purpose: eliciting the history, supporting administration, organizing evidence by criterion, applying diagnostic pathways, or reducing time when a comprehensive interview is impractical.

What the full interview examines

The full interview contains 50 questions concerning one injury event. It is not a lifetime TBI screening questionnaire. Its central domains are the mechanism, loss of consciousness, memory surrounding the event, altered mental status, acute symptoms, other neurological signs, and possible confounding influences. Questions about acute care and other available evidence help place the account in context. The interviewer gathers information for Criteria 1, 2, 3, and 6. Criteria 4 and 5 require examination, laboratory, or imaging information obtained from records or other sources; a patient's account of having had an abnormal scan cannot replace the imaging evidence [28].

The distinction between clinical signs and subjective symptoms remains central. Feeling dazed or confused does not, by itself, demonstrate impaired orientation or responsiveness. The history should establish what the person understood and how the person behaved. Similarly, an interval without recall is not automatically an interval of unconsciousness. A person may have been awake and active while failing to form continuous memories. Retrograde amnesia may substitute for post-traumatic amnesia when the latter cannot be assessed reliably, but the duration of the retrograde gap does not determine injury severity [1,28]. These distinctions make a structured history more informative than a series of unchecked yes-or-no answers.

The interview was originally intended for postacute assessment based on retrospective report, including events weeks, months, or years earlier. It can also be used acutely when the person is no longer experiencing post-traumatic amnesia or altered mental status. If either remains present, the authors advise against administering this interview and recommend assessing the acute state with an appropriate validated instrument. The intended age range is 15 years and older; use in younger adolescents requires individual judgment and is not equivalent to having a validated pediatric version [28].

Highlights of the administration guide

The Administration Guide adds practical safeguards. Ask how a duration was estimated and distinguish a personally remembered event from information learned later. Separate isolated islands of recall from the return of continuous memory. Seek records,

witnesses, or recordings that can anchor the timeline. Preserve an undetermined response when the evidence cannot support a more definite answer. Confounders are evaluated for their actual influence: absent, present without influence, partially explanatory, fully explanatory, or uncertain. An influence on duration may limit severity classification even when injury occurrence is established. Questions about psychological trauma should explore its effect on thinking without requiring unnecessary retelling of distressing details. Pause when needed. The guide also makes clear that the coding form follows the interview and precedes the diagnostic flow diagram [28, supplementary Administration Guide].

These instructions should be applied within the original criteria. Acute subjective mental-status symptoms must relate to the immediate aftermath of impact or recovery of consciousness. Other qualifying symptoms must be new or worsened in the early postinjury period, operationalized as the first 72 hours in the interview. A symptom noticed later should still be documented, but its later recognition does not automatically establish its earlier presence. A remote interview can recover evidence about an acute event; it cannot change the date on which a clinical test was performed [1,28].

The development process deliberately reduced leading questions, increased opportunities for open-ended responses, simplified wording, and clarified prompts and skip instructions. Novice interviewers require training in TBI presentation, consciousness, amnesia, and confounders, followed by supervised practice. The paper recommends observation of initial patient interviews when feasible [28]. In clinical use, an initial free account should remain distinguishable from information elicited only after specific questioning. A polished account is not necessarily more accurate than a hesitant one, and a discrepancy calls for clarification rather than automatic acceptance of either version.

Using the coding form and diagnostic flow

The Diagnostic Coding Form converts collected information into explicit judgments for each criterion. It separates mechanism, clinical signs, acute symptoms, clinical or laboratory findings, neuroimaging, and confounding factors. The clinician should retain the supporting observation and its source, as well as the final designation. Missing examination or imaging information is recorded as unavailable rather than interpreted as a normal result. Confounding influences must be considered in relation to the specific sign, symptom, or test finding on which the diagnosis depends [1,28].

The Diagnostic Flow Diagram then applies the alternative diagnostic pathways. After a plausible mechanism, TBI can be supported by a qualifying clinical sign attributable to brain injury, by at least 2 qualifying acute symptoms together with at least 1 appropriate clinical or laboratory finding, or by unambiguous trauma-related intracranial structural imaging evidence. The first 2 pathways require that confounders do not fully account for the evidence used. A separate decision addresses suspected mild TBI when the evidence is incomplete or confounded. These are alternative routes, not a requirement that all 6 criteria be positive and not a numerical score obtained by adding checkmarks [1,28].

Occurrence and severity should be decided separately. The durations of loss of consciousness and post-traumatic amnesia, together with valid GCS information, guide whether the mild qualifier is appropriate. A clearly established brain injury may have uncertain severity if its duration indicators cannot be reconstructed. A structural imaging qualifier describes the imaging evidence; it does not by itself establish a more-than-mild injury. Finally, neither the interview nor its coding form settles whether every current complaint is caused by the historical injury. That requires a separate assessment of the subsequent clinical course and competing explanations [1,28].

The short form and the state of validation

The full interview was developed through internal review by 17 Task Force members, external review by 19 experts, and field testing by 11 interviewers who completed 25 interviews. Mean administration time was 28 minutes, with a range of 15 to 40 minutes. This was a development and feasibility study. At publication, the authors reported that external validation had not yet been completed and that validation studies were ongoing. Expert review and successful administration do not establish inter-rater reliability, sensitivity, specificity, or diagnostic accuracy [28].

A Short Form was subsequently developed for settings where the full interview is impractical. It removes some questions, condenses the acute-symptom inquiry, and permits selected skips when a criterion has already been established. It is intended for experienced interviewers who understand the ACRM criteria. The authors favor the full interview when feasible because abbreviated inquiry may miss information and produce diagnostic or severity-classification errors. The shortened form shares the need for further validation [28, supplementary Short Form].

Appendix B provides an independently written comprehensive intake form, an evidence-coding worksheet, and an original diagram of the diagnostic pathways for use alongside this discussion. They are educational adaptations of the diagnostic framework, not the

official ACRM interview, a validated substitute, or an ACRM-endorsed instrument. Their wording and organization differ from the published package. Readers who want the standardized questions, original coding instructions, or Short Form should obtain the official materials through the publisher links provided in Appendix B. This distinction preserves the difference between applying clinical concepts and claiming to have administered a particular published instrument. Appendix C provides a separate reference to the criteria and their retrospective and medicolegal interpretation.

Table 5.2. Evidence used in retrospective mild traumatic brain injury diagnosis

Evidence source	**What it may show**	**Which criterion it can support**
Direct observation or video review	Loss of consciousness, absence of protective action on falling, observed disorientation, motor incoordination	Criterion 2 when the observed behavior meets the definition
Witness or family report	Observed disorientation, repetitive questioning, or other qualifying acute behaviors; symptom history where appropriate	Criterion 2 when the report establishes a qualifying sign; may corroborate symptoms
Emergency medical services records	Scene confusion, vomiting, abnormal behavior, mechanism, GCS, transport observations	Criteria 1 and 2 when qualifying findings are documented; may also record acute symptoms
Emergency department and nursing notes	Triage complaints, observed behavior, medications given, imaging results	Criteria 1, 2, 3, and 5; often contains clues absent from the final diagnosis
Patient's own account	Dazed feeling, confusion, amnestic gap, headache, dizziness, cognitive fog	May establish Criterion 2 when the account describes a qualifying observable acute sign and other sources are unavailable; subjective acute symptoms support Criterion 3 [1,27].

Evidence source	What it may show	Which criterion it can support
Acute objective testing, if performed	Cognitive, balance, or oculomotor impairment; elevated blood biomarker	Criterion 4 when a qualifying acute result is established; an acute result found later in records remains acute evidence. Newly administered post-acute tests usually cannot establish it [1].
Structural neuroimaging	Trauma-related intracranial abnormality	Criterion 5 when the finding is unambiguously trauma-related after a plausible mechanism
Follow-up records	Persistent headache, dizziness, cognitive complaints, mood symptoms, work limitation	May document early manifestations, later trajectory, and functional consequences
Post-acute ancillary testing	Vestibular, oculomotor, electrophysiological, or cognitive abnormality	May document present dysfunction and corroborate a reasoned retrospective attribution. Newly administered late tests usually do not establish acute Criterion 4 [1,27,32-36].

Collateral information: the value of informant interviews

The patient's account may be incomplete. Patients may have poor recall of the acute event because of post-traumatic amnesia, may lack insight into early behavioral change, and may minimize symptoms or fail to notice cognitive slowing. The people around them saw what the patient could not: a spouse who reports that he was not himself for two days, a coworker who noticed unusual errors on his return, a friend who recalls that he kept asking what had happened, a family member who describes new irritability, sleepiness, forgetfulness, or intolerance of noise and light. These observations help distinguish the patient's post-injury behavior from the usual baseline. Only the details that meet the definition and timing of a qualifying sign establish Criterion 2; later behavioral changes require their own interpretation [1;27, pp.1617-1618].

The ACRM recognizes several sources for establishing a clinical sign: direct observation, including video review; collateral witness report; acute care records; and, when those are unavailable, the person's own recount of the event [1]. A qualifying patient account is not automatically limited to the suspected category simply because independent corroboration is unavailable; the issue is whether it reliably establishes an observable acute sign [27, p.1618]. It does not rank witnesses against medical records. The weight of each depends on proximity to the event, what was actually observed, specificity, consistency, and possible bias. The source of each finding should be documented. In forensic cases this is often where the diagnosis is decided: an emergency department note may read "no loss of consciousness" while the spouse describes a patient who was slow to respond and repeated the same question for ten minutes. If the account is reliable, it supplies an acute sign that the note never addressed; its reliability, timing, and consistency with the rest of the record are as open to examination as the note itself.

Patient and informant accounts may differ. Patients underrecognize some deficits and overestimate others; family members notice changes the patient cannot articulate. The discrepancy itself is informative, because it marks where further evaluation is needed, whether for mood disorder, poor insight, exaggeration, or misunderstanding. Even a brief question can be revealing: what did others notice about you after the injury, and did anyone say you were confused, repetitive, slow, or not acting like yourself?

Confounding factors and the differential diagnosis

No section of this chapter matters more in contested cases, and none is more often omitted. Every major framework requires that the features relied upon not be better explained by something else. The 2023 criteria state this as Criterion 6 and apply it to signs, symptoms, and objective findings alike [1]. A sign is discounted when it is better accounted for by acute musculoskeletal pain, psychological trauma, intoxication, pulmonary or circulatory disruption, or syncope preceding a fall. A symptom is discounted when it is better accounted for by drug, alcohol, or medication use; a co-occurring physical injury such as cervical or peripheral vestibular injury; a psychological condition such as an acute stress reaction; a pre-existing condition; or symptom exaggeration. An objective finding is discounted when any of these factors, or anything compromising test validity, explains it. A diagnosis that has not worked through this list is incomplete no matter how well the positive findings are documented.

Several confounders recur often enough to deserve individual comment.

Cervical injury. The same acceleration and deceleration may injure the neck, the brain, or both. Cervicogenic headache and altered cervical proprioceptive input overlap with

post-concussive symptoms, and altered smooth pursuit during neck torsion has been studied in whiplash-associated disorders [7]. This is not a choice between diagnoses. Both may be present, and the useful question is which findings each explains; a well-characterized acute amnestic episode, for example, is not explained by the neck.

Acute stress reaction and psychological trauma. A frightening collision can produce fear, dissociation, impaired attention, and a sense of unreality that overlap with the post-injury picture. Psychological trauma can also affect memory. The clinician therefore characterizes what was forgotten, whether new learning was impaired, what witnesses observed, and whether the chronology fits post-traumatic amnesia or something else. Stress-related illness and brain injury often coexist [1,8].

Intoxication, sedation, and medication. Alcohol, recreational drugs, opioids, benzodiazepines, and anticholinergics all impair mental status, and any of them may be present at the time of a collision or given shortly afterward. A notation of intoxication does not exclude a concurrent brain injury. Acute mental status findings must be assessed for whether intoxication fully accounts for them; its presence alone does not disqualify otherwise sufficient evidence of brain injury.

Pain and sleep disruption. Severe pain degrades attention, processing speed, and memory, and disrupted sleep does the same. Both are common after trauma, and neither selective cognitive deficits nor an abnormal oculomotor result can be assumed immune to them.

Pre-existing conditions. Migraine, prior concussion, attention deficit disorder, learning disability, depression, anxiety, obstructive sleep apnea, and vestibular disease all overlap with the post-concussive picture. Interpretation uses both population norms and the patient's documented or carefully estimated premorbid function; either comparison alone can mislead.

Symptom exaggeration. This possibility is considered without being assumed in either direction. Performance validity and symptom validity measures answer different questions and are selected and interpreted accordingly. Invalid performance limits interpretation of the affected results; it does not establish intentional deception or exclude a coexisting injury [9].

Two principles guide this assessment. First, the presence of a confounder does not by itself negate a brain injury; the two can coexist. Criterion 6 asks the more specific question of whether the confounder *fully accounts* for the findings relied upon. Second, when that remains unclear, the ACRM provides the category of suspected mild TBI [1].

Using that category states the uncertainty explicitly. It applies the criteria as intended and tells the reader why a firmer conclusion has not been reached.

Limits of strict diagnostic criteria

One persistent error is treating loss of consciousness as mandatory. It is not. A qualifying observable alteration of mental status or an acute amnestic episode can establish Criterion 2, including through a reliable retrospective account; neither requires a blackout or a contemporaneous witness. The mechanism and confounding requirements still apply [1,10,27]. Another error is treating a normal CT as proof that no brain injury occurred, when CT identifies structural emergencies and does not measure brain function.

Timing presents a subtler problem. As the 2025 clarification explains, qualifying symptoms must have been present to some degree during the first 3 days [27, p.1618]. A symptom first recorded at week 6 may satisfy that requirement if the history establishes its earlier presence; a genuinely new symptom beginning weeks later does not provide that acute evidence. The date of the first complaint alone cannot distinguish the two. The discussion above addresses masked symptoms, exertional provocation, and evolving intracranial injury.

Post-traumatic headache shows the tension between formal criteria and clinical reality, and it is frequently overstated. The main-body criteria of the International Classification of Headache Disorders, third edition, require headache onset within 7 days of injury, of regaining consciousness, or of recovering the ability to perceive and report headache [11]. That is a formal diagnostic criterion, not a research convention. It has, however, been challenged empirically: McGeary and colleagues found that about one third of patients with persistent post-traumatic headache after mild TBI reported onset outside the seven-day window [12], and the classification itself provides appendix criteria for delayed-onset headache beginning between 7 days and 3 months after injury. Later onset therefore calls for careful attribution and accurate labeling rather than automatic exclusion [11].

The 2023 criteria describe qualifying physical, cognitive, and emotional symptoms as nearly always appearing within 72 hours [1, p.1349]. The 2025 medicolegal clarification explicitly states that a symptom must have been present to some degree within the first three days to count toward the diagnosis, and the structured interview operationalizes the history within that interval [27, pp.1617-1618;28, supplementary Administration Guide]. This concerns evidence used to identify the original brain injury. The ICHD-3 onset interval concerns a different question: classification of headache attributed to

head trauma. A headache first beginning on day 5 may satisfy the ICHD-3 timing requirement but does not supply an acute symptom under the 2025 clarification. Neither interval alone settles causation.

Diagnostic criteria are instruments with defined purposes, not checklists and not obstacles. The question is never whether every feature fits perfectly, but whether the mechanism, the acute manifestations and their evidentiary character, the symptom trajectory, the examination, the collateral history, and the differential together form a coherent account.

When increased demands reveal a difficulty

Return to work, school, driving, or multitasking can expose a limitation that was inconspicuous during rest. There is experimental support for this possibility. Cicerone found that patients with mTBI differed from controls under added dual-task demands, with greater slowing of processing [29]. Gagné and colleagues studied 20 young adults with mTBI and matched controls approximately 71 days after injury. Despite no group differences in symptom or conventional neuropsychological measures, the injured group showed greater gait-speed cost during dual-task walking [30]. These findings support assessing the demands under which difficulty occurs. They do not establish that every later complaint reflects an acute deficit that went unnoticed.

CDC guidance likewise recognizes delayed onset or recognition and worsening or re-emergence of symptoms with exertion [31]. The history should identify the activity that first exposed the problem, whether the same demand had been attempted earlier, and what changed when the demand increased. Recognition on the first day back at work does not establish onset on that day. It also does not prove that the difficulty was present during the first 72 hours. Where that interval cannot be reconstructed, the uncertainty belongs in the opinion. Isolated normal values sample particular functions under particular conditions and do not exclude a prior concussion [1,29,30].

Table 5.3. Diagnostic assertions and their accurate counterparts

Assertion	More accurate interpretation
No loss of consciousness, therefore no concussion	Loss of consciousness is not required. A qualifying observable alteration of mental status or acute amnestic episode may establish Criterion 2, including through a reliable retrospective account [1,10,27].

Assertion	More accurate interpretation
Glasgow Coma Scale 15, therefore brain function was normal	A GCS of 15 does not exclude concussion and is compatible with all three diagnostic pathways
Normal CT, therefore no brain injury	CT excludes many acute structural emergencies but does not exclude functional brain injury; a normal scan leaves Pathways 1 and 2 fully available
No acute clinical sign was documented, therefore the criteria are not met	An acute sign may be established retrospectively, including from a reliable patient account when other sources are unavailable. A sign need not have been witnessed or charted. Other diagnostic pathways may also apply [1,27].
Headache began after 7 days, therefore not post-traumatic	Main-body ICHD-3 criteria use a 7-day onset interval; appendix criteria address delayed onset between 7 days and 3 months. Causal attribution still requires assessment [11,12]
No concussion diagnosis in the emergency department	Mild TBI is frequently missed in acute care, in one prospective series in 56% of qualifying patients [2,3]
Symptoms worsened after return to work, therefore unrelated	Increased cognitive, visual, vestibular, or emotional demand commonly reveals deficits not apparent at rest
A confounding condition is present, therefore no brain injury	Criterion 6 asks whether the confounder fully accounts for the findings, not whether one exists. Where this is unclear, the case is suspected mild TBI [1]
The patient felt dazed, therefore mild TBI	A reported dazed feeling is a Criterion 3 symptom, not a sign. Alone it does not meet the symptom threshold for suspected mild TBI; the diagnosis requires a second qualifying acute symptom and an acute objective finding, or another pathway [1]
Testing months later was abnormal, therefore TBI	Post-acute abnormalities may characterize current dysfunction and contribute to a broader clinical inference. They do not by themselves establish the occurrence or date of injury, and newly administered remote tests usually cannot establish Criterion 4 [1].

Assertion	More accurate interpretation
A collision occurred, therefore brain injury	Mechanism is Criterion 1 and is required by every pathway, but no pathway is satisfied by mechanism alone

Behavioral and developmental effects in pediatric traumatic brain injury

Children are not small adults. Their brains are developing, and the consequences of injury may become obvious only as developmental demands increase. A child may recover physically, return to school, and seem stable, until more complex academic, behavioral, social, or executive tasks are expected. Li and Liu reviewed behavioral outcomes after pediatric traumatic brain injury and found attention problems, conduct problems, aggression, emotional dysregulation, and social difficulties following childhood injury [13]. The phenomenon is sometimes called a developmental lag or neurocognitive stall. Early recovery looks reassuring, while the injury interferes with the later acquisition of complex skills. Parents and teachers eventually notice declining school performance, poor organization, reduced frustration tolerance, withdrawal, impulsivity, or outbursts. The ACRM criteria call for caution in young children. A child under five may be unable to report Criterion 3 symptoms, so proxy report or observed behavior may be required. Test interpretation also requires age-appropriate norms [1].

A case in the schoolyard

Marcus was a quiet, bright eight-year-old who fell from the top bar of a playground climbing structure during recess in May. He landed on the wood chips on the back of his head. He cried but stood up on his own. The recess monitor checked him, asked a few questions, and let him sit on the bench until the bell rang. He went back to class. His teacher noticed he seemed a little off for the rest of the afternoon but attributed it to the fall and a scraped elbow. The school nurse called his mother. He did not vomit. He answered questions. He was sent home, slept most of the evening, and woke the next morning saying he felt fine. No imaging was done. No follow-up was scheduled. The school year ended two weeks later.

Third grade started in August. Marcus's new teacher liked him immediately, but by October she was sending notes home. He was not finishing his math worksheets. He was getting upset over small frustrations. He was avoiding group projects. By December the school suggested an evaluation for attention-deficit hyperactivity disorder. The pediatrician started a stimulant trial. It helped a little, but not much. By fourth grade his reading scores had dropped from above grade level to below, and his mother was being

called twice a month because he had thrown a pencil, shoved another student, or refused to come in from recess.

It was the school psychologist who asked the question. She asked the mother whether anything had happened in the year before the changes started. The mother thought for a moment and remembered the playground fall. Until then, no one had connected the two. A pediatric neurologist saw Marcus shortly afterward. The neurologist treated the fall as an event worth investigating, not as a ready explanation for everything that followed. Increasing school demands can expose difficulties that were less obvious earlier, but the history still had to establish what happened at the time of injury and what else might explain the later course. The family now had a question to pursue and a plan for evaluating the problems.

The case also shows the limits of retrospective inference. The mechanism was documented, but the teacher's observation that Marcus seemed off was nonspecific, no acute signs or symptoms were systematically recorded, and no testing or imaging was obtained. Years later, that is not enough to establish a definite mild TBI or to attribute the whole developmental course to the fall; further history may support a suspected diagnosis, and educational, developmental, psychiatric, sleep, and family factors need evaluation alongside it. Monitoring and treatment do not wait for the attribution to be settled.

Neurological complications with delayed onset

Some neurological complications of head trauma may appear days, weeks, months, or years after injury. They are distinct from persistent post-concussion symptoms, but they make the same point: the initial encounter does not reveal the full clinical course.

Delayed intracranial hemorrhage

Delayed intracranial hemorrhage can occur after an initially negative CT, although it is uncommon. Alvarez-Sabin and colleagues described delayed post-traumatic hemorrhage in patients whose initial CT scans were normal, followed by later intracerebral hemorrhage after a symptom-free interval [14]. In a large nationwide population-based cohort of adults with minor head trauma and an initially negative emergency department CT, Kwon and colleagues reported a 0.4% incidence of delayed intracranial hemorrhage within fourteen days, and showed that delayed hemorrhage was associated with increased one-year mortality [15].

Most patients with mild traumatic brain injury never develop delayed hemorrhage, but the few who do can deteriorate seriously. New severe headache, repeated vomiting,

worsening drowsiness, confusion, focal deficit, seizure, or declining consciousness after head trauma calls for urgent evaluation, and older age, anticoagulant or antiplatelet use, and a significant mechanism raise the concern.

Post-traumatic seizures and post-traumatic epilepsy

Early post-traumatic seizures occur within the first seven days. Late post-traumatic seizures occur after seven days and may indicate post-traumatic epilepsy [16]. Risk increases with injury severity, intracranial bleeding, cortical injury, penetrating trauma, depressed skull fracture, and early seizures [16]. Petridis and colleagues reported pediatric data showing that late seizures are a clinically important concern after brain injury [17]. Lucke-Wold and colleagues reviewed mechanisms linking traumatic brain injury and epilepsy and noted that anticonvulsant prophylaxis may reduce early seizures but has not reliably prevented late post-traumatic epilepsy [18].

Patients and families should know the forms a seizure can take: convulsions, staring spells, sudden confusion, unexplained loss of awareness, automatisms, and episodic neurological symptoms. In a patient with episodes of altered awareness or unexplained spells, electroencephalography is appropriate.

Table 5.4. Delayed manifestations after traumatic brain injury

Domain	Delayed manifestation	Typical clinical concern	Refs.
Cognitive	Memory difficulty, slowed processing, executive dysfunction, reduced cognitive endurance	May become obvious only after return to work, school, driving, or multitasking	[19]
Affective and psychiatric	Depression, anxiety, irritability, post-traumatic stress symptoms, emotional dysregulation	May emerge or worsen during recovery and may coexist with neurological injury	[8]
Somatic	Headache, fatigue, dizziness, sensory intolerance	May peak early, persist, or appear after an initially reassuring period	[12,20]
Pediatric behavioral	Attention problems, conduct issues, emotional dysregulation, school problems	Deficits may emerge later as developmental demands increase	[13]

Domain	Delayed manifestation	Typical clinical concern	Refs.
Hemorrhagic	Delayed intracranial hemorrhage	Rare but potentially serious deterioration after initially negative CT	[14,15]
Epileptic	Late post-traumatic seizures or post-traumatic epilepsy	May appear after the first week and sometimes much later	[16, 17, 18]

Ancillary testing: what it can and cannot establish

Specialized testing helps when symptoms persist, when an injury was missed initially, or when routine imaging and bedside examination do not capture the difficulty. It can document neurological dysfunction, direct the differential diagnosis, and contribute to a retrospective clinical assessment. Mild TBI remains a clinical diagnosis; the 2025 clarification identifies no validated stand-alone test that settles it independently of the clinical evidence [27, pp.1618-1619]. In an appropriate acute assessment, objectively measured cognitive, balance, or oculomotor impairment can contribute to Criterion 4 and the symptoms-plus-findings pathway. The other requirements, including mechanism and consideration of confounders, still apply. The ACRM names no instrument: the measure must meet standards of reliability and accuracy, and the impairment must represent a clinically meaningful discrepancy from age-appropriate norms or preinjury performance [1].

Later findings also deserve a place in the diagnostic reasoning. A technically sound recording may confirm a present neurological abnormality and, when its pattern and clinical context support the connection, corroborate an attribution to earlier trauma. That is a substantive contribution, especially when the first assessment overlooked vestibular or ocular motor function. The clinician must explain the connection rather than assume it from an abnormal flag. Present dysfunction, its likely cause, and fulfillment of a time-defined diagnostic criterion are related questions with different evidentiary requirements. The following sections explain how to use later findings without discarding their value or assigning them an earlier date.

Interpreting the 72 hour window

Two timing questions need separate answers. Criterion 3 concerns when qualifying symptoms began. Criterion 4 concerns findings on acute clinical examination and relevant blood biomarkers. The latter includes clinical tests of cognition, balance, and vestibular-oculomotor function; laboratory testing is not mandatory for every diagnosis.

A qualifying clinical sign can establish the sign-based pathway without an abnormal laboratory result, and a qualifying traumatic structural lesion provides another route [1].

On page 1349, the 2023 paper describes cognitive, balance, and oculomotor findings on acute clinical examination. Its accompanying note states that the sensitivity of most clinical and laboratory tests declines during the first 72 hours, at rates that differ between tests [1, p.1349]. It subsequently says that Criterion 4 "usually cannot be established in a post-acute assessment" [1, p.1352]. This wording gives substantial weight to acute testing without describing a universal biological change at exactly 72 hours. Declining sensitivity means that a later normal result becomes less useful for excluding an earlier injury. An abnormal later result still requires evidence that the measure is reliable, that its diagnostic interpretation is valid at that interval, and that other conditions do not adequately explain it.

Retrospective review of an acute test must also be distinguished from performing a new test remotely. An abnormal balance examination performed during the initial assessment may be found in records years afterward and contribute to Criterion 4. The fact that no concussion diagnosis was entered at the time does not erase the examination. A balance test first performed years later measures function at that later time. Statements that Criterion 4 usually cannot be established post-acutely refer to the difficulty of recovering acute diagnostic information from a newly administered test, not to a prohibition against reviewing an existing acute result [1].

The same paper expressly directs clinicians to integrate all available information and clinical judgment in cases that do not clearly fit the criteria [1, p.1353]. That instruction permits reasoned assessment of later evidence. It does not establish a blanket exception for late testing. If a clinician considers a finding obtained outside the usual interval diagnostically informative, the opinion should explain the test-specific evidence, its relationship to the acute history, and the alternatives considered. The report should make clear whether the published diagnostic pathway is satisfied or whether the conclusion rests on a broader clinical inference.

The VOMS study discussed below illustrates why the timing question is test-specific: its patients were assessed an average of 5.5 days after concussion [21]. Its results cannot be generalized to every ocular motor test or to an examination years after an adult collision. Later studies may nevertheless identify a treatable vestibular, ocular motor, balance, or cognitive disorder. Their value depends on the question being asked. Demonstrating present dysfunction, supporting a history through clinical concordance, and establishing the occurrence and date of a brain injury require different evidentiary claims.

Recorded eye movements as neurological signs

An eye movement does not become a subjective symptom because a camera records it. Computerized eye-movement recording, including videonystagmography (VNG), can document movements during defined examination tasks. Digital nystagmography (DNG) is another term used in clinical practice; the evidence must be matched to the actual recording method and protocol. The Bárány Society classification by Eggers and colleagues treats direct observation and quantitative oculographic recording as ways of examining vestibular signs [32]. The clinical question is what the recording demonstrates. A reproducible pathological movement and a software-generated abnormal score require different explanations; the instrument's name alone supplies neither the interpretation nor the cause.

Kim and colleagues identify abnormal saccades and impaired smooth pursuit as central ocular motor signs and describe video-oculography as a means of documenting them [33]. Their consensus concerns vascular dizziness, so its relevance here is neurological localization, not proof of traumatic causation. Pursuit can also be affected by cognition, medications, and underlying nystagmus. The precise pattern must therefore be reviewed. Moreover, a neurological sign found today is not automatically a Criterion 2 acute sign: that criterion also specifies when the manifestation occurred [1,27].

Evidence months and years after injury

Kraus and colleagues provide a clinical example of later assessment [34]. Their 23 patients with predominant central vestibular dysfunction were evaluated a mean of 16 months after head injury, with a range of 3 to 64 months. Twenty-one had eye movement abnormalities. Diagnosis integrated the history, neurotological examination, vestibular testing, and imaging. This supports the clinical use of later findings in identifying post-traumatic central dysfunction. The cohort was selected and generally more severely injured: 20 of 23 had MRI abnormalities. It does not establish the accuracy of isolated VNG findings for diagnosing an undocumented remote uncomplicated concussion.

Taylor and colleagues studied 99 patients with persistent vestibular complaints, 95 previously classified as having mild TBI or concussion [35]. Testing occurred a median of 12 months after injury, ranging from 3 to 120 months. Eye-movement data were available for 95 participants, of whom 39 (41.1%) had central oculomotor abnormalities. The findings show that measurable dysfunction can remain long after the acute period. Because these patients already had TBI diagnoses, the study does not establish diagnostic accuracy for determining whether an uncertain historical injury occurred.

Using later findings in a retrospective opinion

The 2025 Action Collaborative guideline, endorsed by ACRM and the American Academy of Physical Medicine and Rehabilitation, includes validated objective tests of balance, cognition, and vestibular-oculomotor function among findings that may support diagnosis [36, p.557]. Its principal scope is the first six months, and it follows the 2023 ACRM criteria. This supports clinically integrated testing; it does not designate a particular commercial DNG platform or turn years-later testing into acute Criterion 4 evidence.

In practice, the later examination can add an important piece that was missing at the first encounter. Begin with the actual pattern, such as direction-changing gaze-evoked nystagmus with impaired vestibulo-ocular reflex suppression, rather than the phrase abnormal VNG. Review the recordings, calibration, task performance, and appropriate normative comparisons. Then explain what supports a central, peripheral, or mixed disorder and how that fits the patient's symptoms. A peripheral vestibular injury may follow head trauma without establishing an injury to the brain. Localization is one step in the reasoning; traumatic attribution requires the history and chronology as well [33-35].

The causal assessment should bring together the plausible mechanism, acute manifestations, onset and course of the relevant symptoms, premorbid function, intervening illness or injury, and competing explanations. Concordant findings may strengthen that assessment, but shared confounders and correlated measurements limit how much extra weight several abnormal tests add. The defensible conclusion identifies what was demonstrated, what is inferred, and why. When the evidence supports post-traumatic dysfunction, say so and explain the basis. If the formal criteria remain incomplete, retain that distinction while stating the separately reasoned clinical opinion [1,27]. Chapter 7 develops the examination and interpretation of these findings in greater detail.

Event-related potentials as adjunctive evidence

Event-related potentials (ERPs) measure the brain's electrical response during a task. Boshra and colleagues used ERP and wavelet features in a machine-learning model to classify past concussion in retired athletes, reporting 81% accuracy, 82% sensitivity, and 80% specificity [37]. This goes beyond a comparison of group averages and supports further investigation of remote-injury markers. Those estimates belong to the study's method and population. They cannot be assigned to a routine P300 measurement or a commercial BrainView examination.

Practice guidance is more cautious. The 2021 team-physician consensus found insufficient research to recommend EEG or evoked potentials for diagnosing or treating sport-related concussion [38]. For retrospective concussion attribution, ERP findings should therefore be presented as investigational adjunctive information, with attention to the task, signal quality, attention, sensory function, and relevant norms. They may help characterize a functional problem; they do not provide a generally endorsed stand-alone method for proving a remote concussion [24,37,38].

Evaluating the evidentiary value of modalities

The modalities answer different questions and do not carry equal weight for every purpose. Four considerations decide how much weight a given result deserves, and Chapter 7 applies them to each modality in detail.

The first is whether the diagnostic criteria name the domain the test measures. Criterion 4 names four: cognitive impairment, balance impairment, oculomotor impairment or symptom provocation on vestibular-oculomotor challenge, and elevated blood biomarkers [1]. Naming a domain validates neither every commercial instrument that measures it nor a ranking among them.

The second is vulnerability to measurement error and confounding. Recorded physiological signals add information beyond symptom report, but attention, task engagement, medications, sensory limitations, calibration, and artifact influence the result to a degree that differs from test to test.

The third is whether the evidence supports interpretation for an individual or only differences between groups. A technique may show robust, reproducible differences between injured and uninjured cohorts and still be unreliable for classifying the patient in front of you.

The fourth is whether a professional society has issued guidance on clinical use.

These four considerations help interpret the modalities currently in use. Oculomotor and vestibular assessment measures a Criterion 4 domain. The often-cited VOMS study by Mucha and colleagues included 64 young patients a mean of 5.5 days after sport-related concussion and 78 controls. Symptom provocation during vestibulo-ocular reflex and visual-motion-sensitivity tasks was associated with concussion, with odds ratios of 3.89 and 3.37. A near point of convergence of at least 5 cm also improved discrimination [21]. VOMS is a brief vestibular and ocular motor screening assessment; the accuracy of a full videonystagmography battery in chronic adult injury is a separate question. Computerized posturography measures another Criterion 4 domain and

quantifies postural performance and sensory organization. The pattern informs the assessment without localizing a lesion. Cognitive testing with validity measures assesses the third named domain. Even a thorough battery may not reproduce prolonged or complex everyday demands, so an isolated normal score does not exclude a meaningful functional limitation. Diffusion tensor imaging has a substantial research base. A meta-analysis confirmed reduced fractional anisotropy and increased diffusivity in mild traumatic brain injury, with the corpus callosum a prominent site [22]. Reviews of that literature are explicit that group differences do not translate into interpretation for a single patient [23]. The 2025 clarification makes a separate point about the criteria themselves: abnormalities on diffusion tensor imaging metrics or functional neuroimaging, such as SPECT, do not satisfy Criterion 5. Those experimental methods were not considered in deriving the structural-imaging pathway [27, p.1618]. The ERP evidence and its limits are discussed above. For quantitative electroencephalography, the American Clinical Neurophysiology Society concluded that the evidence did not support qEEG for diagnosing mild TBI acutely or remotely, or for distinguishing it from other diagnoses, and classified that use as investigational [25]; calling it supportive within a multimodal battery does not change that.

The distinction throughout is between measuring a function and establishing the cause of an abnormality. These tools are most useful when concordant with the clinical picture: abnormal ocular motor testing in the patient who complains of dizziness, visual motion sensitivity, and reading intolerance; delayed evoked potentials in the patient who complains of slowed processing and cognitive fatigue. None should be overstated, and none should be dismissed when it fits the patient's presentation.

Table 5.5. Ancillary studies in mild traumatic brain injury evaluation

Test	What it may add	Main limitation
Cognitive testing with validity measures	Domain-specific cognitive performance with assessment of performance validity; may contribute to Criterion 4 acutely	Normal testing does not exclude real-world dysfunction under divided-attention load

Test	What it may add	Main limitation
Digital nystagmography / videonystagmography	Records ocular motor and vestibular signs; the interpreted pattern may support localization and a retrospective clinical assessment [32-35]	Recording quality, task performance, medications, and competing disorders matter. A present sign alone does not establish traumatic cause or acute onset [26,32-35].
Computerized posturography	Quantifies balance dysfunction and sensory-integration dependence	Supportive; must be correlated with vestibular, visual, and proprioceptive function
Evoked potentials	Measures evoked responses; ERP research includes classification of past concussion [24,37]	Retrospective concussion attribution remains investigational; research classifier accuracy cannot be transferred to routine P300 testing [37,38]
Quantitative EEG (qEEG)	Quantitative features of recorded EEG activity	ACNS found insufficient evidence for clinical diagnosis of mild TBI; adjunctive labeling does not establish diagnostic validation [25]
Diffusion tensor imaging	Diffusion measurements that may reflect differences in white-matter microstructure	DTI abnormalities do not satisfy ACRM Criterion 5. Group-level evidence is stronger than individual interpretation; artifacts and normative methods matter [22,27].

Test	What it may add	Main limitation
Structural MRI with susceptibility-weighted sequences	Structural findings that may satisfy Criterion 5 if unambiguously trauma-related	An abnormality must be unambiguously trauma-related; imaging alone does not date the injury or establish mild severity. Most mild TBI imaging is negative [1,27].

A contested diagnosis: how the arguments actually run

The preceding sections describe the diagnostic problems in the abstract. The following composite case, drawn from clinical practice with identifying details altered, shows how they behave when the diagnosis is disputed.

A woman in her early thirties was stopped at an intersection when her car was struck from behind by a commercial truck, twice, as the driver braked and rolled forward again. She was restrained and the airbags did not deploy. She described whiplash and feeling, in her words, somewhat in a daze. After extricating herself she noted a new headache with dizziness, mental fogginess, and neck and back pain. She declined ambulance transport and was driven home.

She presented to an emergency facility the following day as a walk-in. The triage complaint was recorded as a minor motor vehicle collision. The examining physician documented that she was alert, not confused, oriented, with intact memory and a Glasgow Coma Scale of 15, and the review of systems was recorded as negative for headache, confusion, and amnesia. Head CT showed no acute intracranial process. She was discharged with a diagnosis of cervical sprain.

Over the following days the headache worsened and became daily. Within three weeks she reported photophobia, phonophobia, nausea, dizziness, sleep disturbance, forgetfulness, slowed thinking, and difficulty concentrating. She worked in sterile processing at a hospital, assembling and verifying surgical instrument trays. The job required prolonged standing, repetitive arm use, and frequent head and neck positioning. A miscounted tray could reach an operating room. About two weeks after the collision she requested a workplace accommodation. Every restriction granted was musculoskeletal: no repetitive arm work, no sitting, standing, or walking beyond fifteen minutes, no head turning. She was reassigned for four months to a clerical post. She did not ask to return to her regular duties; she completed physical therapy, was released by her physician, and the restrictions lapsed. She told the employee processing the

accommodation that she had increased frustration, fatigue, anxiety, and depression. She did not report the cognitive symptoms. Asked later why not, she said it never happened at work to a degree that interfered with her duties.

Two months after the collision she was referred for neurological evaluation. Her general neurological examination was normal. Cognitive screening, including a Mini-Mental State Examination, was normal at 30 of 30.

Why the case is difficult

Every element that makes mild TBI easy to diagnose was absent. There was no loss of consciousness. No one observed her in the first minutes. No clinician examined her until the following day, and that clinician documented a normal mental status and a review of systems that affirmatively denied the symptom she reported. Computed tomography was normal. She was working full time. Her bedside examination and cognitive screen were normal.

A reviewer who concludes that the criteria are not met can construct a coherent argument from this record. It is stated here in full, because it is a reasonable reading of the chart and because its points have to be answered rather than dismissed.

Applying the criteria, this patient did not sustain a mild traumatic brain injury. She had no loss of consciousness, no post-traumatic amnesia, and no transient neurological abnormality. She was examined the day after the collision and found alert, not confused, oriented, with intact memory and a Glasgow Coma Scale of 15, and the contemporaneous review of systems was negative for headache and confusion, contradicting the later account of headache onset at the scene. The word dazed was deliberately omitted from the WHO definition, and authoritative guidance instructs the clinician to distinguish a biomechanically induced alteration of awareness from the fright and emotional shock that follow any sudden collision. The post-concussive symptoms she reports are not specific to head injury and occur at similar rates in trauma patients without head injury. Her neuroimaging is nonspecific. The physiological studies were obtained months later by the treating physician, and the professional societies governing those modalities state that they should not be used to establish the diagnosis. She sought a workplace accommodation two weeks after the collision and every restriction she requested was musculoskeletal; she asked for nothing relating to attention, memory, or concentration, and she has testified that the cognitive symptoms never interfered with her duties. She returned to unrestricted work in a role where an error reaches a patient, and no error has been documented.

Much of that argument is correct and should be conceded: loss of consciousness or amnesia has not been established; the negative review of systems is contemporaneous evidence; the symptoms are nonspecific; and physiological studies obtained months later do not by themselves establish the acute diagnosis. But the lack of an observed sign is not the same as the lack of an observable sign that a careful history could establish. That distinction matters before the first diagnostic pathway is closed [27, p.1618]. The argument also contains other assertions that go beyond what the record supports.

Where the argument overreaches

It describes the diagnostic framework incompletely. The 2004 WHO Task Force definition was not limited to an observed clinical sign; it also allowed an intracranial lesion not requiring surgery [5]. The 2023 ACRM criteria make the pathways explicit: a sign-based route, a symptoms-plus-findings route, and a structural-imaging route, with attention to confounders and a suspected category [1]. In the account presented, a qualifying acute sign has not yet been established. A careful retrospective interview may clarify that question even without a witness; if it does not establish a sign, the other pathways still require consideration [27, p.1618]. The structural-imaging evidence is central to the analysis that follows.

It gives an incomplete record more certainty than it deserves. An explicit negative review of systems is evidence and must be weighed; it is not silence, and the later report of headache at the roadside conflicts with it and should be acknowledged as such. But whether the entry came from a template, a focused interview, a misunderstanding, or a true denial cannot be known from the entry itself. Powell's findings show that concussion diagnoses are often omitted; they do not show that this particular entry was wrong [2].

It asks the wrong things of the objective studies. Professional guidance must be matched to the particular modality and use; restrictions on qEEG or experimental ERP classification do not erase the examination value of a documented ocular motor sign. A study obtained at ten weeks does not by itself establish that an injury occurred on a particular date. It may document current dysfunction and contribute to a clinical attribution when the history, chronology, and differential diagnosis support that inference. A critique should address the actual findings and reasoning rather than treating all objective modalities as interchangeable [25,32-38].

It overreads the occupational record. The musculoskeletal restrictions establish which limitations were documented and accommodated. The absence of requested cognitive

restrictions is relevant but does not exclude cognitive symptoms. The reasons for nondisclosure require exploration. The four-month clerical reassignment deserves the same scrutiny. Clerical work is not inherently single-task, self-paced, or undemanding, and the useful comparison is between what the patient could do under those conditions and what her usual job required. Her reports of losing the thread of conversation, misplacing the car, and needing reminders identify complaints to investigate. They do not by themselves establish a divided-attention deficit. Her statement that cognitive symptoms did not interfere with her duties stays in the record and is weighed with the rest.

What establishes the diagnosis

Dedicated susceptibility-weighted imaging obtained approximately ten weeks after the collision demonstrated a solitary 4 mm hemosiderin deposition in the left frontal juxtacortical white matter. The interpreting neuroradiologist identified hemorrhagic contusion and diffuse axonal injury as the primary considerations and found no features suggesting cavernous malformation or developmental venous anomaly. The earlier noncontrast CT had shown no calcified lesion at that site.

Under the 2023 criteria, an unambiguous trauma-related intracranial abnormality on structural imaging satisfies the imaging pathway after a biomechanically plausible event, and the absence of acute clinical signs does not defeat that route [1]. The decisive issue is therefore whether the lesion is convincingly traumatic and attributable to this event. A frontal juxtacortical susceptibility focus is compatible with traumatic microhemorrhage, but location and size alone do not date it or settle its cause. The complete sequences, the differential, the history, and any prior imaging all bear on the answer. If expert neuroradiological review supports an unambiguous trauma-related lesion attributable to the collision, the case satisfies the structural-imaging pathway despite the uncertainty about the acute symptom account. Under the ACRM convention, this establishes TBI with neuroimaging evidence of structural intracranial injury. The mild qualifier requires separate support from the acute severity information; imaging alone does not supply it [1;27, p.1618]. Other frameworks handle positive imaging differently, as Chapters 3 and 4 explain. If the lesion remains indeterminate, that uncertainty stays in the opinion.

The lesson

In a contested case, the evidence that attracts the most attention often becomes the center of the diagnostic argument, even when stronger evidence is available elsewhere in the record. The patient's account of feeling dazed is vivid. It is what she remembers

and what the clinician heard first. It is also, under the current criteria, a symptom rather than a sign, and therefore the most contestable element in the file. The imaging finding, a single line in a radiology report, carries far more weight once its traumatic origin is established, and almost none until it is.

Before asserting a diagnosis, or disputing one, identify which pathway is claimed to carry it.

A commercial driver assessed years after a collision

A commercial driver in his 40s was involved in a substantial rear-end collision that pushed his vehicle into another vehicle. He denied losing consciousness. During a later neurological history, he recalled feeling dazed and confused immediately afterward and developing headache and dizziness within approximately an hour. The first emergency medical services assessment recorded a Glasgow Coma Scale score of 15 and no dizziness; a subsequent assessment the same day documented headache and dizziness. Head CT showed no acute intracranial abnormality, and he was discharged without a concussion diagnosis. Identifying details are omitted from this vignette.

Years later, he sought neurological assessment for headaches, headache-associated dizziness, forgetfulness, concentration and word-finding difficulty, fragmented sleep, mood symptoms, and blurred vision. He also had persistent cervical pain treated with spinal interventions. Although he described continuing headaches, intervening records included headache denials and reported improvement following cervical treatment. Prolonged driving and physical activity aggravated his symptoms. The available records contained no qualifying acute cognitive, balance, or ocular motor test result. Smooth-pursuit recordings from the later examination are shown in Figures 5.2 and 7.3. The examining clinician reported good effort despite marked irregular tracking.

Reconstructing the first minutes

The collision supplies a plausible mechanism, but the account of feeling dazed and confused needs clarification. Those words describe a subjective symptom unless the history establishes a qualifying disturbance of behavior or orientation. The clinician should begin with the driver's unprompted recollection and then ask what he understood, said, and did immediately after impact. Did he know where he was and what had happened? Could he follow a conversation? Was there a period for which he could not form continuous memories? Did anyone observe repetition, inappropriate responses, or disorientation? These are questions to investigate, not facts to add to the case [1].

The structured approach described by Lange and Gillow helps organize this inquiry [28]. A later normal GCS records his status when assessed; it cannot exclude a disturbance that had already resolved. The initial absence of dizziness and its subsequent same-day appearance can reflect symptom evolution. It does not require choosing one record and discarding the other. A detailed later account may establish an acute clinical sign, but only if the behavior described actually meets the definition and competing explanations have been considered [1].

Applying the available pathways

If reliable reconstruction establishes immediate disorientation, post-traumatic amnesia, or another qualifying clinical sign attributable to brain injury, the sign-based pathway may be satisfied despite the normal CT and absent acute diagnostic label. An abnormal late laboratory study would not be required. If the history remains limited to a subjective dazed feeling and acute headache and dizziness, at least two qualifying symptoms may be present, but the symptoms-plus-findings pathway still requires an appropriate Criterion 4 finding. The normal CT supplies no positive evidence for the structural-imaging pathway. A missing test is not a normal test, but it also cannot be counted as an abnormal finding [1].

At that point, suspected mild TBI is an appropriate consideration, provided the acute manifestations are not fully explained by other causes, or when the contribution of confounders remains uncertain. A firmer classification may become possible as collateral history or additional records are obtained. Iverson and colleagues explain why the suspected label alone cannot resolve a medicolegal probability question [27]. The clinician's degree of confidence must be justified separately, with the evidence that would strengthen or weaken it made explicit.

Interpreting later symptoms and testing

The continuing complaints deserve investigation even while the original injury is being reconstructed. Cervical pain, sleep fragmentation, headache, mood symptoms, medication effects, and brain injury may contribute in different proportions. Improvement in headache after cervical treatment supports a cervical contribution during that interval; it neither excludes a coexisting concussion nor establishes the cause of every subsequent headache. The later account of uninterrupted symptoms must be compared with the intervening denials and treatment responses. The clinician should ask what improved, for how long, and whether symptoms returned, rather than silently treating the records as continuous.

In this patient, the later recordings document irregular smooth pursuit and provide objective evidence relevant to the complaint of blurred vision (Figure 5.2). The recorded pattern must be interpreted with the examination, technical quality, applicable norms, and plausible alternatives. When that assessment supports central ocular motor dysfunction, the finding can add weight to a retrospective attribution supported by the injury history and subsequent course. The clinician should explain that inference and address discrepancies in the record. The test's date cannot be moved backward to fill an absent acute finding. Normal results require the same care. A normal cognitive score does not exclude vestibular dysfunction, headache, reduced endurance, or a difficulty that emerges under divided attention [1,29,30,32-35].

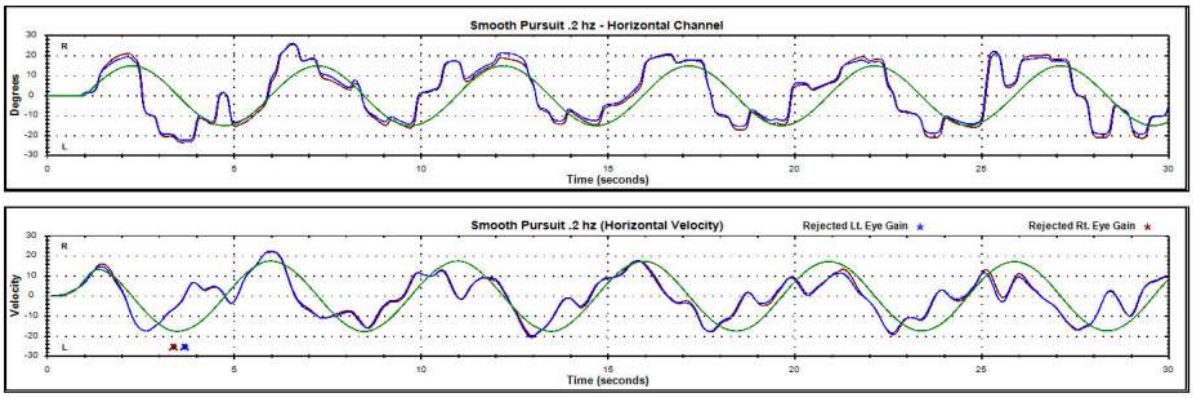


Figure 5.2. Irregular horizontal smooth pursuit at 0.2 Hz in the patient described in this vignette. The upper panel plots eye position and the lower panel plots eye velocity over 30 seconds. Eye traces repeatedly depart from the smooth target waveform, with abrupt corrections and irregular tracking. The examining clinician reported good effort and described the pattern as marked cogwheeling or saccadic pursuit. The patient reported blurred vision. The recording documents a finding relevant to that complaint and supports further ocular motor assessment; attribution to the earlier trauma requires the history and differential diagnosis. Asterisks identify gain samples rejected by the software. The faster recording from the same patient appears in Figure 7.3. Clinical recording provided by Dr. Pappolla; original traces preserved.

His report that driving aggravates symptoms does not by itself demonstrate impaired multitasking or identify the mechanism of the aggravation. Driving combines cervical posture, visual motion, sustained attention, and competing demands. The assessment should determine which of these provokes difficulty and, where appropriate, examine the relevant function under comparable demands. An objectively demonstrated limitation under load would help characterize present impairment. Whether it reflects a deficit present from the injury, a later consequence, or another disorder remains a separate clinical judgment.

Stating an opinion that can be revised

A useful opinion would identify the mechanism and the documented acute symptoms, explain whether a qualifying sign has been established, and state the diagnostic pathway supported at that stage. It would then address the current disorders and their attribution. The chronology is compatible with an injury, but it does not replace examination of alternatives. A missing emergency diagnosis does not foreclose a retrospective diagnosis; persistent symptoms do not automatically establish one. The case shows why further interviewing and testing can be appropriate without prejudging what they will demonstrate. The purpose of the criteria is served when new evidence can change the conclusion [1].

Clinical and forensic pitfalls

Applying a framework incompletely. A reviewer should identify the diagnostic definition used and examine every applicable route; under the 2023 criteria, absence of an acute sign leaves the symptoms-plus-findings and imaging pathways open [1]. Historical definitions should be represented accurately rather than dismissed as requiring loss of consciousness or a witnessed sign in every case.

Relying on normal imaging. Routine imaging identifies structural emergencies. Mild TBI affects function, physiology, connectivity, vestibular control, ocular motor systems, sleep, mood, and cognitive endurance in ways no routine scan shows.

Overemphasizing loss of consciousness. No framework requires it.

Failing to seek available collateral history. Someone who saw the patient at the scene or during the first days may supply details the patient cannot recall. Such evidence should be sought and weighed. When it is unavailable, a reliable patient account may still establish a qualifying observable sign; the absence of corroboration is a limitation to explain, not an automatic bar to diagnosis [1;27, p.1618].

Attributing symptoms prematurely to psychological causes. Depression, anxiety, post-traumatic stress, pain, and insomnia coexist with mild TBI and worsen its outcome. The task is not to choose between neurological and psychological explanations early but to determine what each accounts for.

Failing to address confounders at all. The mirror image of the preceding pitfall. A diagnosis that has not worked through Criterion 6 is incomplete, and a reviewer who says so is right.

Assuming malingering without evidence. Symptom exaggeration must be considered, but many genuine patients are doubted because their imaging is normal and their symptoms are subjective. Validity assessment, examination, collateral history, and properly interpreted physiological measures inform the question; an isolated abnormal validity result does not answer it [9].

Misstating the role of post-acute testing. Presenting a study obtained months later as though it were an acute examination misstates the evidence. Dismissing a well-characterized later neurological sign as irrelevant makes a different error: it can inform diagnosis of current dysfunction and contribute to a reasoned retrospective attribution when the history, chronology, and differential diagnosis support that connection.

Poor documentation. When the diagnosis is made retrospectively, its basis should be stated: the mechanism, the acute manifestations with the sources and reliability of the evidence, symptom onset and evolution, collateral history, examination findings, the confounders considered, and which pathway carries the diagnosis.

Returning the patient to full activity too quickly, or restricting too long. Patients with possible concussion need education, neurorehabilitation, symptom monitoring, and graded return. Prolonged unnecessary restriction is also harmful.

Failing to recognize the full spectrum of deficits. Mild TBI is not only a memory disorder. It may involve headache, vestibular dysfunction, visual intolerance, sleep disturbance, mood dysregulation, autonomic symptoms, fatigue, slowed processing, reduced multitasking capacity, and loss of cognitive endurance.

Conclusion

Mild traumatic brain injury is easy to miss when imaging is normal, manifestations are brief, documentation is incomplete, other injuries distract attention, complaints are delayed, or witnesses are absent. Those gaps in recognition do not make the injury less real. They require care in both making and excluding the diagnosis. The absence of loss of consciousness, a normal CT, a Glasgow Coma Scale of 15, and a missing emergency department diagnosis do not individually exclude concussion. An isolated report of feeling dazed or a post-acute abnormal study does not by itself establish a past TBI. A valid later abnormality can document dysfunction and corroborate the assessment, while its cause and competing explanations still require analysis.

A reliable evaluation begins with “is this a concussion?” and follows it with the more specific question “which pathway does the evidence support, and how good is the evidence?” These questions keep the reasoning tied to the evidence. The purpose

extends beyond assigning a label: to identify the injury, validate the patient's symptoms when medically justified, guide treatment, prevent avoidable deterioration, and reduce long-term disability.

Fast take: main points from chapter 5

• Normal routine imaging, a Glasgow Coma Scale score of 15, or absence of loss of consciousness does not by itself exclude mild traumatic brain injury.

• A retrospective diagnosis rests on the mechanism and evidence from the acute period. The account should identify its sources, their reliability, and the diagnostic pathway supported.

• A clinical sign must have been observable, but need not have been observed. A reliable patient account may establish an acute sign retrospectively when other sources are unavailable. Confounding explanations still require assessment.

• Later DNG/VNG can document neurological or vestibular signs and contribute to a retrospective clinical opinion. Interpret the specific pattern, recording quality, chronology, and competing causes. A new late test is not automatically an acute Criterion 4 finding; an acute result recovered later remains acute evidence.

• Suspected mild TBI does not express a numerical probability. State the criteria-based classification and the clinician's separately reasoned degree of confidence, and assess the cause of current symptoms separately from whether an injury occurred.

• Assessment should document the full range of symptoms and functional difficulties, then use the findings to guide treatment and follow-up.

References

1. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036.
2. Powell JM, Ferraro JV, Dikmen SS, Temkin NR, Bell KR. Accuracy of mild traumatic brain injury diagnosis. Arch Phys Med Rehabil. 2008;89(8):1550-1555.
3. Peixoto C, Buchanan DM, Nahas R. Missed emergency department diagnosis of mild traumatic brain injury in patients with chronic pain after motor vehicle collision. Pain Physician. 2023;26:101-110.

4. Centers for Disease Control and Prevention. Symptoms of mild TBI and concussion [Internet]. 2025 Sep 15 [cited 2026 Oct 24]. Available from: https://www.cdc.gov/traumatic-brain-injury/signs-symptoms/index.html
5. Carroll LJ, Cassidy JD, Holm L, Kraus J, Coronado VG. Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2004;(43 Suppl):113-125. doi:10.1080/16501960410023877.
6. Corrigan JD, Bogner J. Initial reliability and validity of the Ohio State University TBI Identification Method. J Head Trauma Rehabil. 2007;22(6):318-329.
7. Treleaven J, Jull G, LowChoy N. Smooth pursuit neck torsion test in whiplash-associated disorders: relationship to self-reports of neck pain and disability, dizziness and anxiety. J Rehabil Med. 2005;37(4):219-223. doi:10.1080/16501970410024299.
8. Schwarzbold M, Diaz A, Martins ET, Rufino A, Amante LN, Thais ME, et al. Psychiatric disorders and traumatic brain injury. Neuropsychiatr Dis Treat. 2008;4(4):797-816.
9. Sweet JJ, Heilbronner RL, Morgan JE, et al. American Academy of Clinical Neuropsychology (AACN) 2021 consensus statement on validity assessment: Update of the 2009 AACN consensus conference statement on neuropsychological assessment of effort, response bias, and malingering. Clin Neuropsychol. 2021;35(6):1053-1106. doi:10.1080/13854046.2021.1896036.
10. American Congress of Rehabilitation Medicine Mild Traumatic Brain Injury Committee. Definition of mild traumatic brain injury. J Head Trauma Rehabil. 1993;8(3):86-87.
11. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1-211.
12. McGeary DD, Resick PA, Penzien DB, et al. Reason to doubt the ICHD-3 7-day inclusion criterion for mild TBI-related posttraumatic headache: a nested cohort study. Cephalalgia. 2020;40(11):1155-1167.
13. Li L, Liu J. The effect of pediatric traumatic brain injury on behavioral outcomes: a systematic review. Dev Med Child Neurol. 2013;55(1):37-45.
14. Alvarez-Sabín J, Turon A, Lozano-Sánchez M, Vázquez J, Codina A. Delayed posttraumatic hemorrhage. Spät-apoplexie. Stroke. 1995;26(9):1531-1535.
15. Kwon H, Kim YJ, Lee JH, et al. Incidence and outcomes of delayed intracranial hemorrhage: a population-based cohort study. Sci Rep. 2024;14:19502.
16. Ding K, Gupta PK, Diaz-Arrastia R. Epilepsy after traumatic brain injury. In: Laskowitz D, Grant G, editors. Translational Research in Traumatic Brain Injury. Boca Raton (FL): CRC Press/Taylor and Francis Group; 2016.

17. Petridis AK, Doukas A, Maslehaty H, Mehdorn HM. Predictors and incidence of posttraumatic seizures in children and adolescents after brain injury. Clin Pract. 2012;2(3):e66.
18. Lucke-Wold BP, Nguyen L, Turner RC, et al. Traumatic brain injury and epilepsy: underlying mechanisms leading to seizure. Seizure. 2015;33:13-23.
19. Machamer J, Temkin N, Dikmen S, Nelson LD, Barber J, Hwang P, et al.; TRACK-TBI Investigators. Symptom frequency and persistence in the first year after traumatic brain injury: a TRACK-TBI study. J Neurotrauma. 2022;39(5-6):358-370.
20. De Koning ME, Scheenen ME, Van Der Horn HJ, Spikman JM, Van Der Naalt J. From “miserable minority” to the “fortunate few”: the other end of the mild traumatic brain injury spectrum. Brain Inj. 2018;32(5):540-543.
21. Mucha A, Collins MW, Elbin RJ, Furman JM, Troutman-Enseki C, DeWolf RM, Marchetti G, Kontos AP. A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: preliminary findings. Am J Sports Med. 2014;42(10):2479-2486. doi:10.1177/0363546514543775.
22. Aoki Y, Inokuchi R, Gunshin M, Yahagi N, Suwa H. Diffusion tensor imaging studies of mild traumatic brain injury: a meta-analysis. J Neurol Neurosurg Psychiatry. 2012;83(9):870-876. doi:10.1136/jnnp-2012-302742.
23. Shenton ME, Hamoda HM, Schneiderman JS, Bouix S, Pasternak O, Rathi Y, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. Brain Imaging Behav. 2012;6(2):137-192. doi:10.1007/s11682-012-9156-5
24. Nandrajog P, Idris Z, Azlen WN, Liyana A, Abdullah JM. The use of event-related potential (P300) and neuropsychological testing to evaluate cognitive impairment in mild traumatic brain injury patients. Asian J Neurosurg. 2017;12(3):447-453. doi:10.4103/1793-5482.180921.
25. Tenney JR, Gloss D, Arya R, Kaplan PW, Lesser R, Sexton V, Nuwer M. Practice guideline: use of quantitative EEG for the diagnosis of mild traumatic brain injury: report of the Guideline Committee of the American Clinical Neurophysiology Society. J Clin Neurophysiol. 2021;38(4):287-292.
26. Skora W, Stanczyk R, Pajor A, Jozefowicz-Korczynska M. Vestibular system dysfunction in patients after mild traumatic brain injury. Ann Agric Environ Med. 2018;25(4):665-668.
27. Iverson GL, Dams-O'Connor K, Panenka WJ, Stephens JA, Lockman SD, Al Lawati Z, Lequerica AH, McPherson JI, Kamins J, Silverberg ND. New diagnostic criteria for mild traumatic brain injury: medical-legal considerations. Arch Phys Med Rehabil. 2025;106(10):1615-1619. doi:10.1016/j.apmr.2025.06.015.

28. Lange RT, Gillow KC; ACRM Brain Injury Special Interest Group Mild TBI Task Force members. Development of a structured interview for the American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. J Neurotrauma. 2026;43(11-12):934-943. Published online November 24, 2025. doi:10.1177/08977151251396077. See also the supplementary Administration Guide, Structured TBI Interview, Diagnostic Coding Form, and Diagnostic Flow Diagram.
29. Cicerone KD. Attention deficits and dual task demands after mild traumatic brain injury. Brain Inj. 1996;10(2):79-89. doi:10.1080/026990596124566.
30. Gagné MÈ, McFadyen BJ, Ouellet MC. Performance during dual-task walking in a corridor after mild traumatic brain injury: a potential functional marker to assist return-to-function decisions. Brain Inj. 2021;35(2):173-179. doi:10.1080/02699052.2020.1863467.
31. Centers for Disease Control and Prevention. Managing return to activities [Internet]. 2025 Jul 28 [cited 2026 Oct 26]. Available from: https://www.cdc.gov/heads-up/hcp/clinical-guidance/
32. Eggers SDZ, Bisdorff A, von Brevern M, Zee DS, Kim JS, Perez-Fernandez N, Welgampola MS, Della Santina CC, Newman-Toker DE. Classification of vestibular signs and examination techniques: Nystagmus and nystagmus-like movements: Consensus document of the Committee for the International Classification of Vestibular Disorders of the Bárány Society. J Vestib Res. 2019;29(2-3):57-87. doi:10.3233/VES-190658.
33. Kim JS, Newman-Toker DE, Kerber KA, Jahn K, Bertholon P, Waterston J, Lee H, Bisdorff A, Strupp M. Vascular vertigo and dizziness: Diagnostic criteria: Consensus document of the committee for the classification of vestibular disorders of the Bárány society. J Vestib Res. 2022;32(3):205-222. doi:10.3233/VES-210169.
34. Kraus M, Hassannia F, Dabiri S, Vergara Olmos G, Rutka JA. Central vestibular dysfunction in head injury. J Otolaryngol Head Neck Surg. 2024;53:19160216241250354. doi:10.1177/19160216241250354.
35. Taylor RL, Wise KJ, Taylor D, Chaudhary S, Thorne PR. Patterns of vestibular dysfunction in chronic traumatic brain injury. Front Neurol. 2022;13:942349. doi:10.3389/fneur.2022.942349.
36. Silverberg ND, Lee K, Mikolić A, et al. Action Collaborative on Traumatic Brain Injury Care: Adapted Clinical Practice Guideline. Ann Fam Med. 2025;23(6):552-569. doi:10.1370/afm.250352.
37. Boshra R, Dhindsa K, Boursalie O, Ruiter KI, Sonnadara R, Samavi R, Doyle TE, Reilly JP, Connolly JF. From group-level statistics to single-subject prediction: machine learning detection of concussion in retired athletes. IEEE Trans Neural Syst Rehabil Eng. 2019;27(7):1492-1501. doi:10.1109/TNSRE.2019.2922553.

38. Herring S, Kibler WB, Putukian M, et al. Selected issues in sport-related concussion (SRC|mild traumatic brain injury) for the team physician: a consensus statement. Br J Sports Med. 2021;55(22):1251-1261. doi:10.1136/bjsports-2021-104235.

Chapter 6 Subtypes and clinical profiles of mild traumatic brain injury

Chapter overview

Two patients with mild traumatic brain injury may carry the same diagnosis and have entirely different symptoms, recovery trajectories, and outcomes. The diagnosis covers a wide range of presentations. For decades, clinicians have tried to make sense of that range by dividing the injury into subtypes, grades, or risk categories. The results have been uneven for a reason that is easy to miss. Subclassification has been asked to do two jobs: predict outcome and characterize the patient for treatment. Each job requires its own kind of validation.

At prediction, early grading systems and symptom scales have performed poorly. The Sports Concussion Assessment Tool [1], the Rivermead Post-Concussion Symptoms Questionnaire [2], and the retired American Academy of Neurology grading system [3] organize early findings and document symptom burden; none supports a confident individual long-term prognosis on its own. Prospective research, including the Transforming Research and Clinical Knowledge in Traumatic Brain Injury program [4], has identified what the risk factors are: age, sex, and pre-injury psychiatric and headache history [5,6,7].

For characterization, clinical profiles work. Lumba-Brown and colleagues defined five subtypes: headache and migraine, vestibular, cognitive, ocular-motor, and anxiety and mood. They also identified two associated conditions, sleep disturbance and cervical strain. Together, these tell the clinician what to evaluate and what to treat [8]. This chapter separates prediction from characterization. It reviews the subdivisions already present in the diagnostic criteria, the historical grading systems, and the instruments still in use. It then explains why prognosis has resisted subtyping, sets out the clinical profile approach, and closes on the multidimensional classification that may eventually replace the mild, moderate, and severe scheme.

Introduction

Mild traumatic brain injury produces a wide range of presentations. Some patients recover within days. Others develop persistent symptoms that interfere with work, school, family life, sleep, mood, cognition, balance, vision, and tolerance of sensory stimulation [6]. As Chapter 4 explains, the word mild describes the initial severity classification and not the eventual impact.

The impulse to subdivide the category is sound. Patients, employers, insurers, and courts want to know who will recover and when. The clinician treating a symptomatic

patient wants to know what to treat. These questions ask different things of a classification system. When they are treated as one, the literature can appear contradictory. A system that cannot forecast a six-month outcome may be excellent at telling a clinician that this patient's headaches are migrainous, her dizziness is vestibular rather than cervicogenic, and her reading intolerance is an oculomotor problem. This chapter keeps prediction and characterization separate and asks how well each approach does the job assigned to it.

Subclassification already embedded in the diagnostic criteria

The diagnostic distinctions described in Chapters 3 and 4 remain useful when considering subtypes: diagnosed versus suspected mild TBI, with versus without neuroimaging evidence of structural injury, and the interchangeable use of concussion and mild TBI when imaging is normal or not clinically indicated [9]. These describe diagnostic confidence, structural findings, and terminology rather than adding new severity grades. The complicated versus uncomplicated distinction has shown small to medium group differences in acute neuropsychological outcome, with insufficient separation to classify individuals [10,11].

Historical grading systems

The American Academy of Neurology published a sports concussion practice parameter in 1997 that graded concussion by symptom duration and loss of consciousness [3]. Grade 1 involved transient symptoms lasting less than fifteen minutes without loss of consciousness. Grade 2 involved symptoms lasting more than fifteen minutes without loss of consciousness. Grade 3 involved any loss of consciousness. Comparable systems in circulation at the time were built on the same two variables.

These systems gave clinicians a framework when concussion management was unstandardized. They are now abandoned, but their limitations still matter whenever a new grading scheme is proposed. They rested heavily on loss of consciousness. Yet loss of consciousness is not required for diagnosis and, when brief, does not consistently predict persistent cognitive impairment. At least one study found that it did not predict neuropsychological decrement after concussion [12]. The systems also graded the injury at a single time point, although its manifestations evolve. In doing so, they gave prognostic weight to variables that carry little of it. The Academy has since retired both the 1997 parameter and its 2013 successor, and current practice emphasizes individualized assessment, removal from risk, monitoring of evolving symptoms, graduated return to activity, and targeted treatment of specific symptom domains [1,13].

The general lesson is that a grading system built on one or two acute variables will not capture an injury whose course is determined by many. The ACRM reached the same conclusion about the mild, moderate, and severe scheme itself, whose upper severity thresholds it retained only while multidimensional alternatives were under development [9].

Assessment instruments and what they measure

Sports Concussion Assessment Tool

The Sport Concussion Assessment Tool, commonly known as the SCAT, is used for structured acute assessment of suspected sport-related concussion. SCAT6 is intended primarily for the first 72 hours and up to 7 days after injury, with office assessment tools used thereafter [13]. It combines symptom reporting, cognitive screening, neurological screening, balance testing, coordination assessment, and clinical decision support. The fifth edition derives from the Berlin consensus statement [1]; the sixth international consensus process has since produced an updated statement and instrument [13].

The SCAT gives clinicians a structured way to evaluate acute concussion and supports decisions about immediate removal from play, early monitoring, and return to sport. It is not a stand-alone test that confirms or excludes concussion; diagnosis remains clinical, through the pathways set out in Chapter 3. Its limitation for present purposes is prognostic. A SCAT profile obtained on the sideline is a snapshot of the acute period and was never designed to forecast which patients will still be symptomatic months later.

Rivermead Post-Concussion Symptoms Questionnaire

The Rivermead questionnaire measures symptoms commonly experienced after head injury, including headache, dizziness, nausea, noise sensitivity, sleep disturbance, fatigue, irritability, depressed mood, frustration, forgetfulness, poor concentration, slowed thinking, blurred vision, light sensitivity, and restlessness [2].

Its value is in quantifying symptom burden and tracking change over time. Serial administration is more informative than a single score, because the trajectory carries information the absolute value does not.

The limitation becomes clear when a patient scores dizziness as severe. That score may reflect vestibular, oculomotor, cervical, autonomic, psychological, or combined influences. It describes a problem that needs characterization, but leaves the

differential diagnosis unresolved. The same symptoms also occur at appreciable rates in people without head injury, so an elevated score is consistent with mild TBI without being specific to it. The clinical weight of a Rivermead score comes from its change over time and its concordance with objective findings, not from its magnitude alone.

Clinical profiles and treatment planning

A complementary approach asks which clinical domains are contributing to the patient's difficulties and which require treatment. Characterization and risk assessment can proceed together.

Lumba-Brown and colleagues, working through a multidisciplinary expert workgroup with systematic review and meta-analysis, defined five concussion subtypes and two associated conditions [8]. The subtypes are headache and migraine, vestibular, cognitive, ocular-motor, and anxiety and mood. The associated conditions are sleep disturbance and cervical strain, which frequently accompany the subtypes but do not stand alone as diagnostic features.

In the analysis of findings within three days of injury, prevalence differed by age. In pediatric patients the headache and migraine subtype was the most prevalent, at approximately 0.52, with the vestibular subtype also high at approximately 0.50. In adults the cognitive subtype was most prevalent, at approximately 0.40. Compared with uninjured or baseline controls, adult patients showed prevalence ratios of 4.4 for the cognitive subtype, 2.9 for the vestibular subtype, and 1.7 for the anxiety and mood subtype [8] (Figure 6.1).

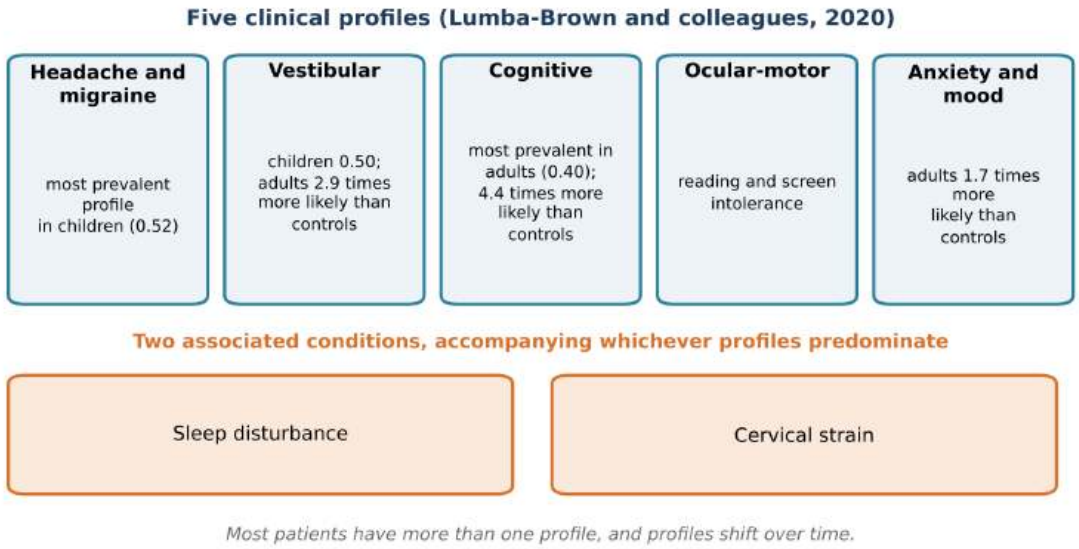


Figure 6.1. The five clinical profiles of mild traumatic brain injury and the two associated conditions defined by Lumba-Brown and colleagues, with the prevalence estimates and prevalence ratios reported in that analysis [8]. Original schematic.

Two caveats belong with these figures. First, the workgroup found insufficient information to analyze cervical strain, and sleep disturbance scores did not differ significantly from controls. These findings limit what the analysis can say about those two conditions; they do not show that the conditions are unimportant after concussion. Second, subtypes are not fixed. A patient may present predominantly with a headache profile and later develop features of the anxiety and mood profile [8]. A profile describes the current presentation, not a permanent category.

Each profile suggests targets for assessment and treatment. The intervention follows the specific diagnosis and the evidence for that intervention.

Headache and migraine. A migrainous post-traumatic headache phenotype can guide consideration of migraine-directed acute and preventive treatment, chosen with attention to comorbidities, adverse effects, and the thinner evidence base specific to post-traumatic headache. Medication-overuse headache should be looked for.

Vestibular. Vestibular rehabilitation may help appropriately selected patients with persistent dizziness or balance impairment. The evaluation should distinguish central, peripheral, cervical, and other contributors, because some disorders require a specific intervention, such as canalith repositioning for benign paroxysmal positional vertigo, rather than a generic rehabilitation prescription.

Ocular-motor. Convergence or accommodative dysfunction and other eye-movement abnormalities may contribute to reading or screen intolerance. Treatment should follow a defined examination finding and appropriate eye-care assessment; benefit from vergence therapy cannot be generalized to all pursuit, saccadic, or visual symptoms.

Cognitive. Persistent difficulties with attention, processing speed, or working memory may warrant cognitive rehabilitation, graded activity, and environmental accommodation once contributing sleep, pain, mood, and medication factors have been assessed. Brief bedside screening may miss these difficulties, for reasons discussed in Chapter 5.

Anxiety and mood. Anxiety, depression, and emotional lability after injury require behavioral health treatment in their own right. Treating them is not a concession that the injury was psychological; untreated mood symptoms degrade cognitive performance, sleep, and headache burden, and they are among the strongest modifiable contributors to persistent disability.

Sleep disturbance and cervical strain, the associated conditions, are addressed alongside whichever profiles predominate. Both worsen the overall symptom burden and both are treatable, which makes them disproportionately worth identifying.

Most patients present with more than one profile. The useful clinical exercise is to rank how much each contributes to the patient's difficulties, rather than select a single label. Consider the patient whose dominant profiles are vestibular and headache, with sleep disturbance as an associated condition. Those findings suggest a ranked set of priorities, potentially vestibular rehabilitation, headache management, and sleep intervention. A severity grade alone cannot supply that treatment order.

This is also where the objective testing described in Chapter 7 earns its place. Videonystagmography, computerized posturography, oculomotor assessment, and cognitive testing with validity measures characterize the impaired domains, refine the differential diagnosis, and select treatment targets; what they do not do, as Chapter 5 explains, is establish on their own that a past injury caused the current abnormalities.

Attempts to predict long-term outcome

There is still a good reason to pursue prognosis. If patients at high risk for persistent symptoms can be identified early, they could receive monitoring, education, rehabilitation, workplace accommodation, and targeted therapy before disability becomes established. That possibility continues to drive efforts to predict long-term outcome.

Prospective research has clarified what the risk factors are, even where prediction for the individual remains imprecise. TRACK-TBI analyses have shown that symptom burden persists in a meaningful subset of patients through the first year [6]. Age and sex influence symptom recovery [5]. Pre-injury psychiatric history and pre-injury headache or migraine history independently predict worse functional and post-concussive outcomes at three and six months [7]. Cnossen and colleagues developed a prediction model in which years of education, pre-injury psychiatric disorder, and prior traumatic brain injury were the strongest predictors of six-month post-concussive symptoms [14].

For the initial evaluation, these studies provide a short list of risk factors to document: prior traumatic brain injury, psychiatric history, migraine or headache history, and educational background [7,14]. Each should lower the threshold for early, active follow-up. Sleep disturbance, pain, social support, and occupational demands also inform care planning, with prognostic strength that depends on the population and outcome studied. A clinician who records these has done something more useful than assigning a grade.

Why prognostic subtyping has proven so difficult

Individual variability. Two patients with similar initial injuries may recover very differently. Age, sex, prior brain injury, migraine history, psychiatric history, sleep quality, pain sensitivity, autonomic function, vestibular and visual function, and cognitive reserve all contribute. A model built on acute variables can estimate risk; it cannot capture every influence or settle the outcome of one patient.

Dynamic manifestations. Symptoms are not static. Some appear immediately; others emerge when the patient attempts to return to work, school, driving, exercise, or visually demanding environments. An instrument administered shortly after injury captures one moment and may miss what appears under real-world load. The distinctions among delayed onset, delayed recognition, and delayed diagnosis set out in Chapter 3 apply directly here.

Instrument scope. The SCAT was designed for structured acute assessment in sport [1]. The Rivermead questionnaire measures symptom burden [2]. The retired Academy system classified by symptom duration and loss of consciousness [3]. None measures structural injury, the full range of vestibular and oculomotor disorders, autonomic function, sleep, pain, psychiatric vulnerability, or everyday environmental demands, all of which influence recovery and each of which needs a method suited to it.

Environment and circumstance. Recovery occurs inside real lives. Patients may be holding a job, caring for children, managing pain, attending legal appointments, tolerating noise and light, sleeping badly, and repeatedly explaining symptoms others cannot see. These factors do not make the injury non-neurological. They determine how an injured brain performs under load, which is precisely what the patient's functional outcome measures.

Multimodal prediction models

To move beyond single-variable systems, researchers have turned to multimodal models. These combine symptoms, demographics, premorbid history, imaging, biomarkers, and functional measures, increasingly through machine learning. Their strength is the ability to bring together risk factors that simple systems cannot hold together. Their limitations are familiar. They require external validation, and they perform differently in populations unlike the cohort from which they were developed. Even when a model supplies a probability estimate, the clinician still has to build a treatment plan.

A different problem: predicting acute deterioration

The prediction discussed above concerns long-term symptom persistence. Acute hospital prediction of early neurological deterioration is a separate question with a separate literature, and conflating the two produces confusion.

In the acute setting, the question is immediate: will a patient who appears stable now deteriorate over the next several hours? This matters most in older adults, patients with intracranial bleeding, those taking anticoagulants, and those with evolving neurological signs.

Abe and colleagues developed a machine-learning model to predict neurological deterioration after mild traumatic brain injury in older adults, using admission variables including D-dimer, fibrinogen, acute subdural hematoma thickness, cerebral contusion size, and systolic blood pressure [15]. The model achieved a testing-data area under the curve of 0.77, with sensitivity of 60% and specificity of 96% at the selected cutoff, compared with sensitivity of 20% and specificity of 94% for neurosurgeons assessing the same data [15]. The result is of interest for acute risk stratification, but it comes from a retrospective registry analysis with an internal test split. It needs external validation. At the reported cutoff, 40% of deterioration cases were missed, so the model cannot replace clinical monitoring. Its findings say nothing about persistent post-concussion symptoms.

Where the field is going

The ACRM working group directly acknowledged problems with the traditional mild, moderate, and severe scheme. The 2023 criteria retained it only because work on a multidimensional severity classification based on biomarkers was still underway and a replacement was not yet available [9]. The authors hoped the current three-tier scheme would eventually be replaced, and that the diagnostic criteria would remain for the lower threshold of TBI without the mild qualifier.

If that expectation is realized, the successor will combine structural imaging, fluid biomarkers, physiological measures, and clinical phenotype, and describe injury along several dimensions rather than on a single axis. The clinical profile approach is a step in that direction: it already describes a patient along five dimensions instead of one.

Conclusion

The value of subclassifying mild traumatic brain injury depends on the question being asked. A classification helps when it can answer that question; it misleads when it is asked to support a conclusion beyond its reach.

Historical grading systems, the SCAT, and the Rivermead questionnaire do not support a confident individual long-term prognosis on their own [1,2,3]. Symptom burden and other acute findings contribute to risk estimation, and prospective research supports attention to pre-injury psychiatric and headache history, prior injury, and educational background, with serial clinical assessment refining the prognosis over time [5,6,7,14].

Subclassification can, however, characterize what is wrong with a given patient in a way that directs treatment. The five clinical profiles and two associated conditions defined by Lumba-Brown and colleagues give a structure for doing so [8], and the diagnostic criteria themselves add three further divisions: diagnosed versus suspected, with versus without structural imaging evidence, and the terminological equivalence of concussion and mild TBI [9].

The best framework uses clinical profiles to characterize the individual patient and explicitly documents the factors that affect prognosis. It follows that patient over time, rather than judging the course from a single early score. This keeps the useful structure of subtyping without turning it into a rigid label or abandoning it altogether.

Fast take: main points from chapter 6

• Subclassification serves two purposes that need separating: risk estimation, where individual prognosis remains uncertain, and clinical characterization, where profiles organize evaluation and treatment.

• The diagnostic distinctions reviewed in Chapters 3 and 4 remain relevant to subtyping [9]; imaging-based group differences do not establish an individual prognosis [10,11].

• Historical concussion grades based on symptom duration and any loss of consciousness are no longer recommended. Loss of consciousness is not required for diagnosis, and brief loss of consciousness does not consistently predict persistent impairment, although its duration remains relevant to initial severity [3,9,12].

• The SCAT is for structured acute assessment and is not a stand-alone diagnostic or prognostic test [1]. The Rivermead questionnaire quantifies symptom burden but does not identify the mechanism behind any given symptom [2].

• Five clinical profiles organize contemporary management: headache and migraine, vestibular, cognitive, ocular-motor, and anxiety and mood, with sleep disturbance and cervical strain as associated conditions [8]. Most patients have more than one, profiles shift over time, and treatment follows the specific diagnosis within each profile.

• The prognostic studies support documenting prior traumatic brain injury, psychiatric history, migraine or headache history, and educational background at the first visit [5,6,7,14]. Sleep, pain, social support, and occupational demands also matter for care planning.

• Acute deterioration prediction is a separate problem with a separate literature and should not be conflated with post-concussion prognosis [15].

• The 2023 ACRM authors anticipated a future multidimensional classification while retaining the established severity thresholds pending an adequate replacement [9].

References

1. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport: the 5th International Conference on Concussion in Sport held in Berlin, October 2016. Br J Sports Med. 2017;51(11):838-847.
2. King NS, Crawford S, Wenden FJ, Moss NEG, Wade DT. The Rivermead Post-Concussion Symptoms Questionnaire: a measure of symptoms commonly experienced after head injury and its reliability. J Neurol. 1995;242(9):587-592.

3. American Academy of Neurology Quality Standards Subcommittee. Practice parameter: the management of concussion in sports (summary statement) [retired]. Neurology. 1997;48(3):581-585.

4. Yue JK, Vassar MJ, Lingsma HF, Cooper SR, Okonkwo DO, Valadka AB, et al.; TRACK-TBI Investigators. Transforming Research and Clinical Knowledge in Traumatic Brain Injury Pilot: multicenter implementation of the common data elements for traumatic brain injury. J Neurotrauma. 2013;30(22):1831-1844.

5. Levin HS, Temkin NR, Barber J, Nelson LD, Robertson C, Brennan J, et al.; TRACK-TBI Investigators. Association of sex and age with mild traumatic brain injury-related symptoms: a TRACK-TBI study. JAMA Netw Open. 2021;4(4):e213046.

6. Machamer J, Temkin N, Dikmen S, Nelson LD, Barber J, Hwang PH, et al.; TRACK-TBI Investigators. Symptom frequency and persistence in the first year after traumatic brain injury: a TRACK-TBI study. J Neurotrauma. 2022;39(5-6):358-370.

7. Yue JK, Cnossen MC, Winkler EA, Deng H, Phelps RRL, Coss NA, et al.; TRACK-TBI Investigators. Pre-injury comorbidities are associated with functional impairment and post-concussive symptoms at 3 and 6 months after mild traumatic brain injury: a TRACK-TBI study. Front Neurol. 2019;10:343.

8. Lumba-Brown A, Teramoto M, Bloom OJ, Brody D, Chesnutt J, Clugston JR, Collins M, Gioia G, Kontos A, Lal A, Sills A, Ghajar J. Concussion Guidelines Step 2: Evidence for Subtype Classification. Neurosurgery. 2020;86(1):2-13. doi:10.1093/neuros/nyz332.

9. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036.

10. Williams DH, Levin HS, Eisenberg HM. Mild head injury classification. Neurosurgery. 1990;27(3):422-428. doi:10.1097/00006123-199009000-00014.

11. Iverson GL. Complicated vs uncomplicated mild traumatic brain injury: acute neuropsychological outcome. Brain Inj. 2006;20(13-14):1335-1344. doi:10.1080/02699050601082156.

12. Lovell MR, Iverson GL, Collins MW, McKeag D, Maroon JC. Does loss of consciousness predict neuropsychological decrements after concussion? Clin J Sport Med. 1999;9(4):193-198.

13. Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport, Amsterdam, October 2022. Br J Sports Med. 2023;57(11):695-711.

14. Cnossen MC, Winkler EA, Yue JK, Okonkwo DO, Valadka AB, Steyerberg EW, et al. Development of a prediction model for post-concussive symptoms following mild traumatic brain injury: a TRACK-TBI pilot study. J Neurotrauma. 2017;34(16):2396-2409.
15. Abe D, Inaji M, Hase T, Suehiro E, Shiomi N, Yatsushige H, Hirota S, et al. A machine learning model to predict neurological deterioration after mild traumatic brain injury in older adults. Front Neurol. 2025;15:1502153. doi:10.3389/fneur.2024.1502153.

Part II. Objective testing

Chapter 7 An introduction to objective testing

Chapter overview

Part I established how the diagnosis of mild traumatic brain injury is made, how severity is graded, why the injury is so often missed, and how patients differ from one another within the diagnosis. This chapter turns to the instruments used to examine nervous system function. They include quantitative electroencephalography, evoked and event-related potentials, videonystagmography, computerized posturography, autonomic assessment, computerized cognitive testing, and blood biomarkers. Diffusion tensor imaging is introduced here and taken up in full in Chapter 8.

Four points organize it. Objective testing has a defined place within the diagnostic criteria, with timing central to that role [1]. Later testing can also contribute to a retrospective clinical assessment when the original injury was missed. The modalities carry different evidentiary weight, and their interpretation follows the same logic as testing elsewhere in medicine: a finding acquires meaning from the clinical context. The strength of an assessment rests on valid, convergent evidence, not simply on the number of abnormal results.

One caution runs through the chapter. Each of these techniques examines one narrow window into nervous system function. A normal result through one window does not establish that the rest of the house is in order, and an abnormal result through one window does not by itself establish what caused it.

From diagnosis to measurement

Part I asked how to classify the injury: did this patient sustain a traumatic brain injury, how severe was it, and what kind of injury was it? This chapter asks what we can measure. What can actually be demonstrated about how this patient's nervous system is functioning?

The two questions are connected but not identical. A clinician who treats an isolated abnormal test as proof of a past concussion overstates the evidence. A clinician who dismisses a recorded neurological sign because it was obtained later may discard useful evidence. The task is to explain what the test establishes now and what it contributes to the reconstruction of the earlier injury.

Where objective testing sits within the diagnostic criteria

The 2023 ACRM criteria give objective findings a defined role, and the role differs by criterion and by timing [1]. Chapter 3 sets out the full structure; what follows is the part that bears on testing.

Criterion 4 concerns clinical examination and laboratory findings. The criteria name cognitive impairment, balance impairment, oculomotor impairment or symptom provocation with vestibular-oculomotor challenge, and elevated blood biomarkers indicative of intracranial injury [1]. The examination findings are framed as acute findings. Together with at least two qualifying symptoms, an applicable finding can complete the second diagnostic pathway, provided the mechanism, confounder and mild-severity requirements are also met. A qualifying acute test is therefore a building block of that pathway.

Two limits matter here. First, the examination and laboratory findings in Criterion 4 have imperfect sensitivity and specificity and cannot establish mild traumatic brain injury alone [1]. Second, sensitivity declines during the first 72 hours for many acute measures, at different rates for different tests. The criteria accordingly state that Criterion 4 usually cannot be established in a post-acute assessment [1]. That wording does not create a universal 72-hour deadline for every instrument or blood assay, and it does not make all later findings clinically irrelevant.

Criterion 5 concerns structural imaging. A trauma-related intracranial abnormality on computed tomography or conventional structural magnetic resonance imaging can establish traumatic brain injury in the context of a plausible mechanism and appropriate exclusion of alternative explanations, even when acute symptoms or signs were not documented [1,32]. Assigning the mild qualifier still requires the relevant acute severity information. Diffusion tensor metrics and functional imaging do not satisfy this criterion. Chapter 8 explains the role of imaging in detail.

Later testing often serves several purposes at once. It can demonstrate current dysfunction, help localize it, clarify competing diagnoses, and contribute to a retrospective assessment of an injury that was initially missed. A finding first recorded months later does not automatically establish an acute Criterion 4 finding or date the onset of the abnormality. Its diagnostic contribution depends on the test, the pattern, and the rest of the evidence.

Chapter 5 develops the retrospective reasoning in detail. The clinician distinguishes whether the available evidence meets a diagnostic pathway from the broader judgment about what most likely happened and what explains the current findings. The 2023

criteria and the 2025 medicolegal clarification allow clinical judgment and consideration of all available information when a case does not fit neatly [1,32]. A later abnormality can strengthen that judgment when it is valid and concordant; it can also reveal an alternative explanation. Neither its timing nor its objective appearance settles the question by itself.

Later testing when the initial injury was missed

A missing emergency department diagnosis does not end the clinical inquiry. The later clinician may be the first person to examine eye movements carefully, challenge balance, or measure the function that fails during ordinary life. These findings deserve a place in the reconstruction. They can identify impairments that a symptom checklist alone could not characterize and make the source of continuing difficulty clearer. The useful question is what each finding adds to the evidence, not simply whether it was obtained late [1,32].

Computerized eye-movement recording includes videonystagmography (VNG); digital nystagmography (DNG) is another term used clinically. The particular protocol and recording method determine which evidence applies. A pathological movement documented by a camera can be an examination sign. The recording preserves what happened and permits closer inspection and measurement; it does not turn an observable abnormality into a subjective symptom. The Bárány Society consensus on vestibular signs explicitly includes assessment with oculographic equipment [25].

The distinction between central and peripheral findings matters. A sign of disordered brain control of eye movements is different from evidence of an injured inner-ear balance organ. Both may be important after trauma, but a peripheral vestibular abnormality alone does not establish brain injury. The clinician must identify the pattern and explain the anatomical interpretation rather than relying on an automated label saying that the study is abnormal.

A second Bárány Society document identifies abnormal saccades and impaired smooth pursuit as central ocular motor signs and discusses video documentation. It also notes that pursuit is influenced by medication, cognition and underlying nystagmus. Because that document concerns vascular disease, its contribution here is to examination and localization, not proof of traumatic causation [26].

Clinical series illustrate the value of later examination. Kraus and colleagues combined history, neurotological findings, vestibular tests and imaging to identify post-traumatic central vestibular dysfunction months to years after head injury. Their selected cohort

chiefly involved more substantial injuries, so it cannot validate an isolated VNG abnormality as proof of an undocumented uncomplicated concussion [27].

In Taylor and colleagues' chronic TBI series, central oculomotor abnormalities occurred in 39 of 95 patients with available data; assessment ranged from 3 to 120 months after injury. These were previously diagnosed patients. The study demonstrates measurable late dysfunction rather than diagnostic accuracy for deciding whether an uncertain historical concussion occurred [28].

The 2025 Action Collaborative guideline supports including validated objective vestibular-oculomotor tests in diagnostic assessment. ACRM and AAPM&R endorsed it. Its principal scope is the first six months, and it retains the 2023 ACRM criteria; it does not establish years-later testing as equivalent to acute Criterion 4 evidence [29].

For an individual report, the reasoning should be visible. Describe the actual abnormal movement, not only the device's score. Establish that the recording is interpretable and that any numerical comparison uses suitable reference data. Record the testing conditions and relevant medication exposure. Ask whether the pattern is reproducible, whether it agrees with the examination, and whether another disorder provides a better explanation. Several measurements from the same recording are not several independent confirmations. A persuasive report shows how the evidence fits together rather than accumulating abnormal flags.

Consider a patient whose early record described only neck pain, although the reconstructed history reveals immediate disorientation and new visual instability. Months later, a reproducible ocular motor abnormality may substantiate a continuing neurological problem and strengthen the overall assessment when the chronology and differential diagnosis agree. The historical disorientation and the present sign do different work: one helps establish what occurred at the injury; the other helps explain the patient examined today. If the history instead shows that the same problem preceded the collision, the causal inference changes. This is a clinical illustration, not a separate diagnostic rule.

The word confirmatory therefore needs a stated object. A technically sound recording may confirm that an abnormal eye movement is present. Establishing that a particular collision caused it requires the integrated assessment described in Chapter 5. A report can make a well-reasoned retrospective attribution while acknowledging that a late test does not supply a missing acute observation. Preserving that distinction gives objective findings their proper weight and makes the conclusion easier for clinicians and legal readers to examine [1,32].

The modalities do not carry equal weight

Chapter 5 sets out the ordering and the reasoning behind it. It is summarized here because it determines how the techniques in this chapter should be read against one another.

Four questions govern placement. Do the criteria name the domain the test measures? Is the measured function under voluntary control? Does the evidence support interpretation for an individual patient, or only differences between groups? Has a professional society issued guidance on clinical use?

For the purposes of this book, a useful provisional ordering begins with interpreted oculomotor and vestibulo-ocular findings, balance assessment, and cognitive testing with appropriate validity measures. These examine domains named in Criterion 4, although that recognition does not validate every device or every use of a test [1-3]. Diffusion tensor imaging and evoked potentials require separate consideration: their contributions depend on the particular measurement and clinical question, and evidence of group differences may not support diagnosis in one patient [4,5]. Quantitative electroencephalography has a specific limitation: the American Clinical Neurophysiology Society guideline found insufficient evidence for diagnosing mild traumatic brain injury and classified that use as investigational [6].

This is an interpretive framework for the book, not an official ACRM ranking. A technique's value for identifying a treatable disorder can be substantial even when it cannot establish traumatic causation. Its place in the assessment should follow the evidence for the particular use, rather than the instrument's name.

What objective testing is for

Diagnosis, in the sense set out above, is not the only purpose of testing, and in post-acute practice it is often not the most important. Four others matter.

Demonstrating that dysfunction exists. Many post-concussive symptoms are invisible during a short office visit. A patient may walk in normally, speak clearly, and have an entirely normal bedside neurological examination. That same patient may become dizzy in a supermarket, develop headache after screen work, lose balance in low light, or be cognitively exhausted after an hour. Objective testing can demonstrate measurable abnormality where observation cannot.

Identifying which systems are involved. Dizziness after a concussion is not one symptom with one cause. It may arise from the balance organ of the inner ear, from the brainstem or cerebellar pathways that process its signals, from the networks of the

cerebral cortex, from position sense in the neck, from migraine, from the automatic (autonomic) nervous system, or from sensitivity to visual motion. Each calls for different treatment, and testing separates them.

Directing treatment. This follows from the second and is the practical payoff. Abnormal visual tracking directs oculomotor rehabilitation. Central vestibular abnormality directs vestibular rehabilitation rather than rest [7]. Abnormal sensory organization on posturography directs balance therapy. Autonomic findings direct graded exercise, hydration, and pacing. Cognitive slowing directs cognitive rehabilitation and workplace accommodation. Chapter 6 sets out the clinical profiles this feeds, and Chapter 14 the treatments.

Documenting course. Serial testing shows whether dysfunction is resolving, persisting, or worsening, and a trajectory carries information that a single snapshot does not. Figure 7.1 shows the idea with a reaction time test, in which a button is pressed as fast as possible when a target appears. The patient pressed correctly every time. Reaction time at the first study was 455 thousandths of a second, within the normal range of 350 to 500. It was 836 at the second study and 672 at the third, nearly two years after the first. In plain terms, the brain reached the right answer each time and needed longer to get there: at the last study, about a sixth of a second longer than the upper limit of normal, which is the time a car at 60 miles per hour takes to cover 15 feet. Across the three studies, the pattern was worse and then partly better, but never back to normal. That pattern is less likely to reflect a single bad day than any one value would be. What caused the change is a separate question, answered from the whole record.

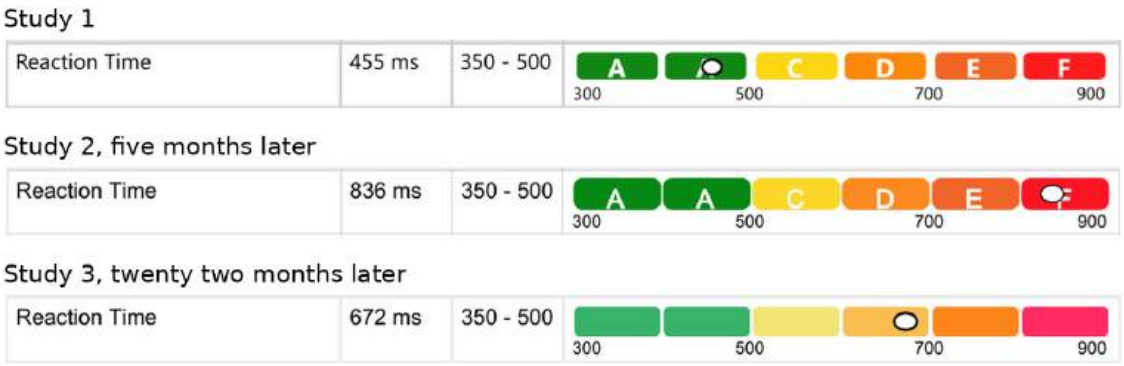


Figure 7.1. Serial testing documents course. Reaction time on a computerized button-press task from the same patient at three sessions over twenty-two months, as printed by the device. The colored bar is the device's scale from normal (green) to markedly abnormal (red), and the circle marks the patient's result. The times were 455 milliseconds (thousandths of a second) at the first study, within the reference range of 350 to 500, then 836 and 672 at the second and third. Accuracy was preserved at every session, with no wrong responses and about 1 percent or fewer missed, so the finding is slowness, not error. Device summary rows reproduced without modification; recording from a patient evaluated in Dr. Pappolla's outpatient neurology clinic, identifying information removed.

What each modality measures

What follows sets out what each technique measures, why it may be abnormal after a mild traumatic brain injury, what an abnormal result looks like, and its principal limitations.

Videonystagmography is a recording of eye movements. The patient wears goggles fitted with small infrared cameras. During the test, the patient follows a moving dot or moving stripes on a screen and holds the gaze in different directions. The examiner also places the patient in head and body positions that would set off an inner ear problem if one existed. The brain steers the eyes through well-mapped circuits in the frontal and parietal lobes, the brainstem, and the cerebellum (the structures at the base and back of the brain that coordinate movement). When the eyes cannot track a target smoothly, cannot jump accurately to it, or cannot keep up with moving stripes, the test is showing a fault in the brain's steering of the eyes, and the pattern tells the examiner whether the fault lies in the inner ear or in the brain. The components differ in how much they depend on the patient. The optokinetic response and nystagmus recorded without a fixation target are reflexive, whereas smooth pursuit and saccades require attention and cooperation. This is why the examiner reviews the quality of each recording. Eye movement and balance problems are common after concussion and track with how long recovery takes [3,8]. In daily life an abnormal result corresponds to the complaints

patients most often describe: reading that tires quickly or loses the line, difficulty following moving traffic, and dizziness in busy visual surroundings such as supermarket aisles, crowds, and scrolling screens. Figures 7.2 to 7.5 show the three eye movement subtests, what a normal result looks like, and what abnormality looks like on each.

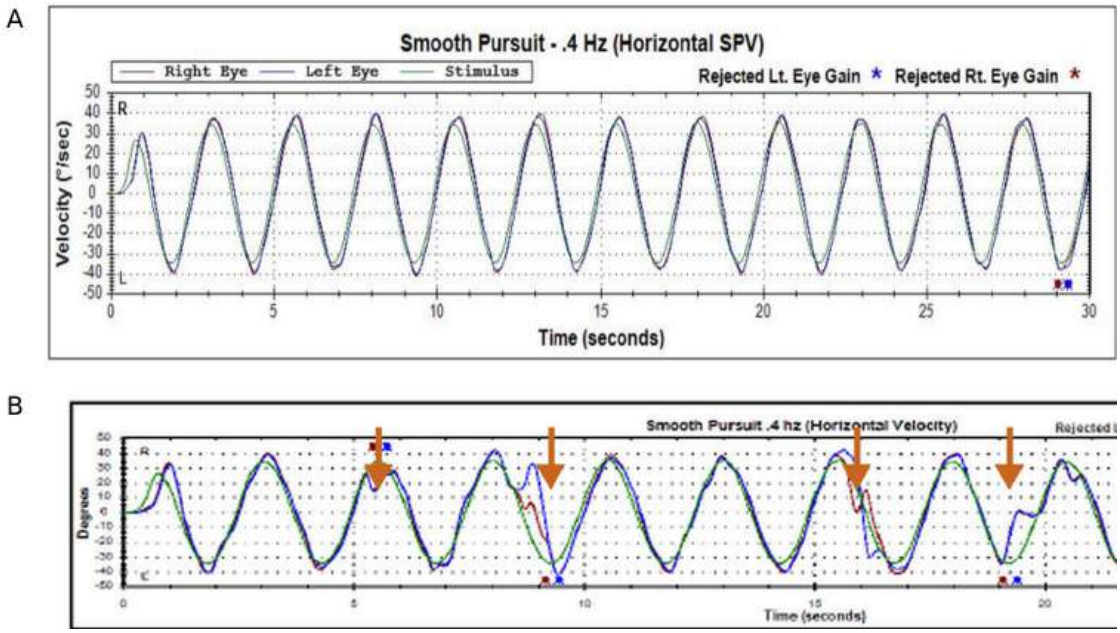


Figure 7.2. Smooth pursuit: following a moving dot. The patient followed a dot that swung from side to side across the screen once every two and a half seconds (0.4 Hz). The green line is the speed of the dot; the red and blue lines are the speed of the right and left eye. (A) Normal: the eyes glided with the dot, and the three lines lie on top of one another as a smooth wave. (B) Abnormal, in a different patient, first 21 seconds of the recording: the eyes repeatedly fell behind the dot and jumped to catch up (arrows). Each jump is a catch-up saccade (saccadic intrusion), and the device classified the pattern as consistent with involvement of the brain rather than the inner ear. In practical terms this is the patient whose eyes cannot glide along a line of print or follow a passing car without extra, tiring corrections. Smooth pursuit depends on attention, so the tracing is read together with the rest of the examination. Recordings from patients evaluated in Dr. Pappolla's outpatient neurology clinic; identifying information removed; arrows added.

The patient in the Chapter 5 vignette also had blurred vision and irregular pursuit at a faster target frequency. Figure 7.3 shows the 0.4 Hz recording from that examination; Figure 5.2 shows the 0.2 Hz recording. These findings help assess an ocular motor contribution to persistent post-concussion symptoms. They are complementary observations from the same examination, not independent confirmations of its cause.

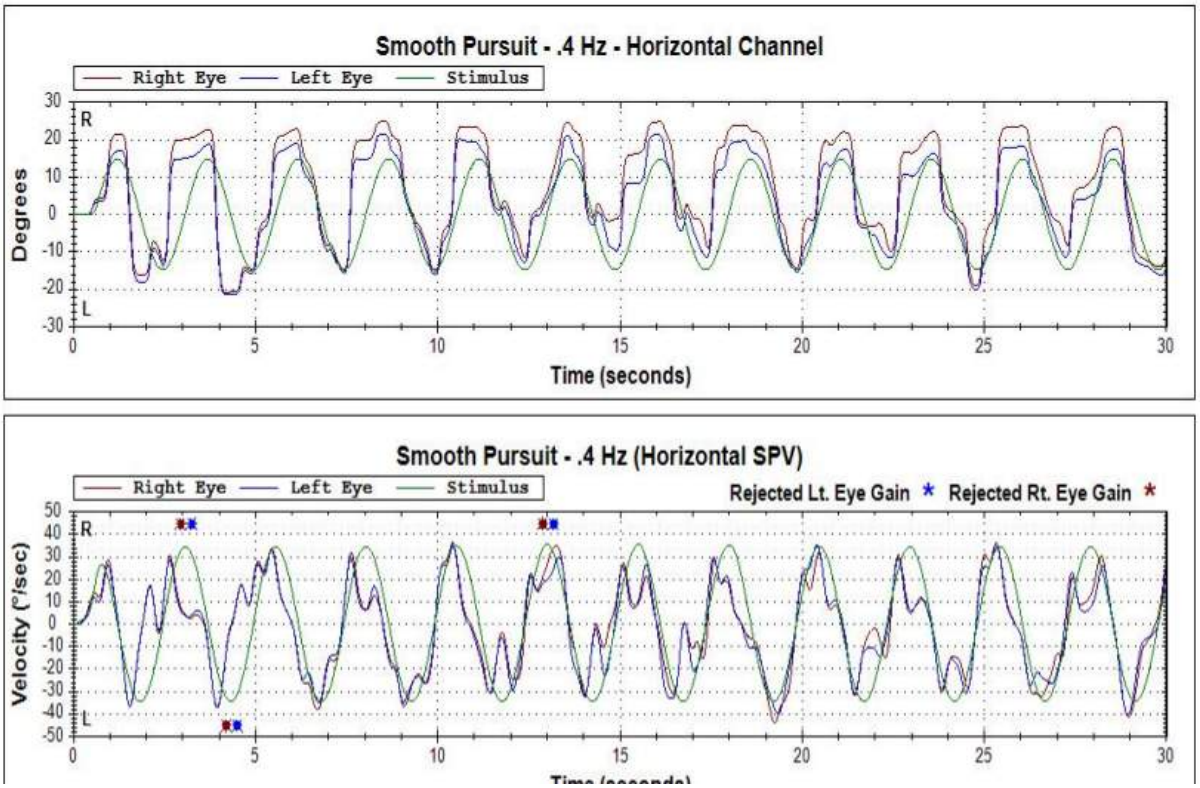


Figure 7.3. Horizontal smooth pursuit at the faster target frequency of 0.4 Hz in the same patient shown in Figure 5.2. The green line represents the stimulus; red and blue represent the right and left eyes. Eye position is shown above and horizontal slow-phase velocity (SPV) below. Repeated departures from smooth tracking and abrupt corrective movements are consistent with the examining clinician's description of frequent saccadic intrusions. Good effort was reported by the clinician; it cannot be established from the image alone. This finding provides an objective correlate to consider alongside the reported blurred vision. Asterisks mark rejected gain samples, so a validated summary gain cannot be inferred from these screenshots. The pattern requires clinical interpretation and does not independently date or establish traumatic causation. Original clinical recording provided by Dr. Pappolla.

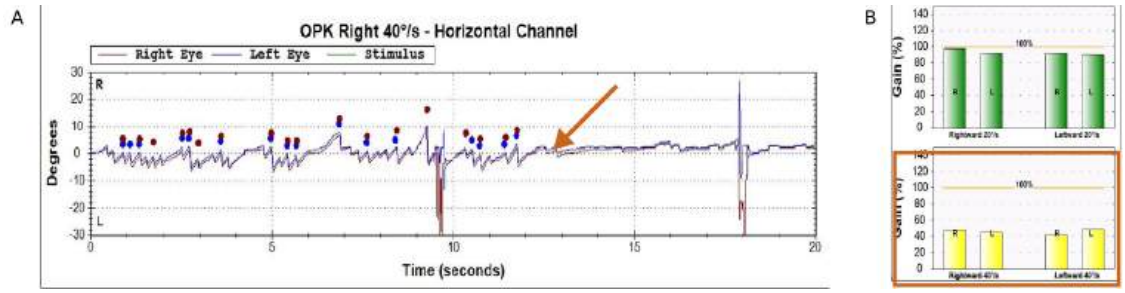


Figure 7.4. Optokinetic response: following moving stripes. Stripes moved across the screen and the eyes followed them automatically, the same reflex that makes a train passenger's eyes flick along with the telephone poles outside the window. (A) Eye position over 20 seconds with stripes moving at 40 degrees per second, a fast speed. For about 12 seconds the eyes kept flicking along with the stripes (the sawtooth pattern), then stopped following altogether and the line went flat (arrow). (B) Gain, which is how fast the eyes moved as a percentage of how fast the stripes moved; healthy eyes come close to 100 percent. At the slower stripe speed of 20 degrees per second this patient's gain was normal, 90 to 98 percent. At 40 degrees per second (boxed) it fell to 42 to 49 percent: the eyes moved at less than half the speed of the stripes before giving up. This is the patient who becomes dizzy or nauseated in traffic, in crowds, or while scrolling a screen, where the visual world moves faster than the brain's reflex can follow. At a repeat study twenty-two months later the gain at 40 degrees per second had recovered to 84 to 91 percent. Recording from a patient evaluated in Dr. Pappolla's outpatient neurology clinic; identifying information removed; arrow and box added.

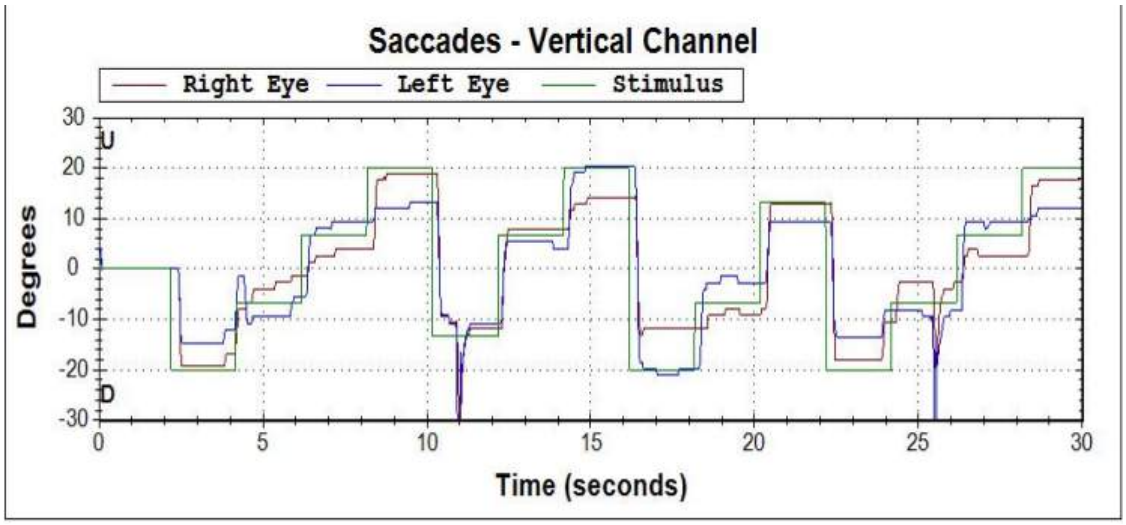


Figure 7.5. Saccades: jumping to a target. The dot (green) jumped between positions up and down the screen and the patient looked to each new position as fast as possible. The red and blue lines are the position of the right and left eye. Healthy eyes land on the target in a single jump. Here the eyes reached each target in two or more steps and often stopped short of it, a pattern the device reported as poor saccadic precision, which means poor aim. In reading, this is the eye that undershoots or overshoots the next word and needs a second try, which slows reading and makes it tiring. Recording from a patient evaluated in Dr. Pappolla's outpatient neurology clinic; identifying information removed.

Computerized posturography measures how steadily a person stands. Balance depends on three sources of information: the eyes, the balance organ of the inner ear, and the sense of position from the feet and joints (proprioception). The brain combines them continuously and corrects posture faster than the person is aware of. Posturography removes or degrades these sources one at a time and measures how much the body sways in each condition. A patient may appear steady on routine examination and become unstable when vision is removed or the support surface is altered, which is why patients report difficulty in the dark, on uneven ground, in crowds, and in visually busy environments.

Figure 7.6 shows a report from a fixed force plate, a flat platform that senses where the body's weight is pressing. The patient stood on it for 20 seconds in each of four conditions: eyes open on the firm plate, eyes closed on the firm plate, eyes open on a thick foam pad, and eyes closed on foam. Closing the eyes removes vision. Standing on foam blurs the information from the feet, which is what the moving platform of older systems was designed to do, so that the brain must rely on the eyes and the inner ear. The device tracks the point where the body's weight presses on the plate (the center of pressure) and adds up how far that point wandered during the 20 seconds; the longer

the path, the more the person swayed. Everyone sways more as the conditions get harder. What matters is how a patient's sway compares with healthy people of the same age and sex, which the device expresses as a percentile.

In this patient, the sway path was 13 cm with eyes open on the firm plate and 53 cm with eyes closed on foam. Three of the four conditions fell within the range the device treats as normal. The exception was eyes open on foam: a path of 41 cm, at the 14th percentile. That percentile means that 86 of 100 healthy people of the same age and sex swayed less than this patient did in this condition, when information from the feet was made unreliable and balance had to depend on the eyes and the inner ear. In daily life that is the person who is fine on a firm floor but unsteady on grass, sand, a thick carpet, a boat, or a moving bus. One condition in the bottom quarter does not by itself establish an injury. It is one measured abnormality, in a system the diagnostic criteria name, to be read alongside the eye movement findings and the history.

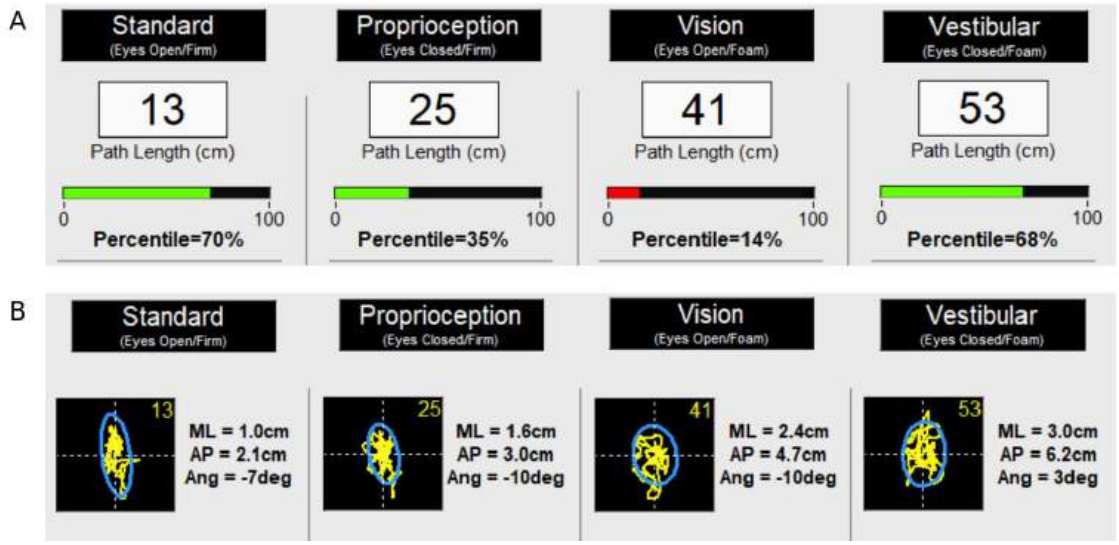


Figure 7.6. Computerized posturography on a fixed force plate (modified Clinical Test of Sensory Integration and Balance). (A) The four conditions, left to right: eyes open on the firm plate, eyes closed on the firm plate, eyes open on foam, and eyes closed on foam. The large number is the sway path in centimeters, that is, how far the body's center of pressure wandered in 20 seconds, and the bar beneath it shows the patient's percentile against healthy people of the same age and sex; green is within the device's normal range and red is the bottom quarter. Sway rose from 13 cm to 53 cm as the conditions became harder, as it does in everyone. The one flagged condition was eyes open on foam, 41 cm at the 14th percentile. (B) The actual sway path (yellow) for each of the same four trials, with an ellipse (blue) drawn around the area that contained 95 percent of it; the wider the ellipse, the more the patient moved. Recording from a patient evaluated in Dr. Pappolla's outpatient neurology clinic; identifying information removed.

Computerized cognitive testing presents a series of timed tasks on a computer: remembering words and shapes, pressing a key when a target appears, matching symbols, switching between rules. The program scores reaction time, attention, processing speed, memory, and executive function (the ability to plan, switch, and hold back a response), and compares each score with healthy people of the same age, again as a percentile. It also contains built-in validity checks, patterns of response that are implausible in someone making an honest effort. It is a screening instrument, not a full neuropsychological evaluation, and does not replace one [9]. Unlike reflexive eye movements or early evoked potentials, these scores depend on the patient: on effort, but also on sleep, pain, medication, mood, and the testing room. That is why the validity checks matter, and why a cognitive score carries more weight when it agrees with measurements that are less dependent on the patient's cooperation.

Blood biomarkers are the fourth domain named in Criterion 4, and the only one measured from a blood sample rather than from the patient's performance. When brain cells are injured, some of their structural proteins leak into the bloodstream, where they can be measured. The most studied proteins come from several parts of the nervous system. Glial fibrillary acidic protein (GFAP) and S100B come from the brain's supporting cells. Ubiquitin C-terminal hydrolase-L1 (UCH-L1) and total tau come from the nerve cells themselves. Neurofilament light (NfL) comes from the long fibers, or axons, that connect nerve cells [10].

Timing governs how a blood biomarker result should be interpreted (Figure 7.7). Each protein appears in the blood, peaks, and declines on its own schedule. The approximate patterns summarized in the review are an S100B peak within six hours, with a half-life of half an hour to two hours; a UCH-L1 peak at seven to nine hours; and a GFAP peak at twenty to twenty-four hours. Neurofilament light behaves differently, rising slowly and peaking between roughly ten days and six weeks, consistent with a more prolonged response to injury of the brain's fibers [10]. These are approximate patterns, not a clock that runs identically in every patient. NfL can remain elevated for months in some patients, so it is inaccurate to assume that every biomarker has cleared by the time persistent symptoms are assessed [21]. A later measurement cannot recreate a missed acute sample, however, and the collection window validated for a particular assay still matters. The absence of a biomarker result in the acute record is missing information, not a negative test.

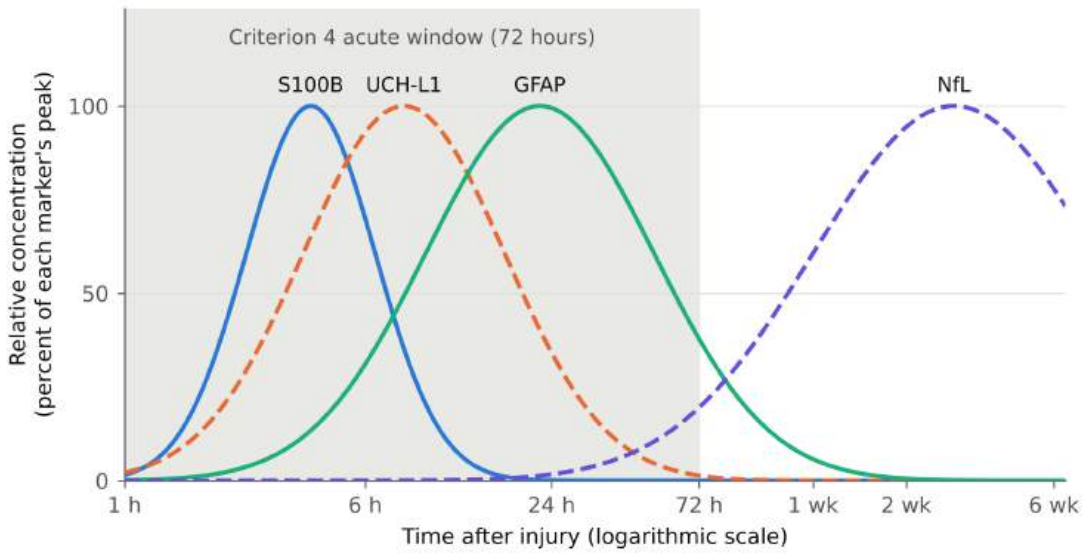


Figure 7.7. When each blood marker can be detected. Each curve shows the rise and fall of one protein in the blood after a brain injury, drawn to the peak times given in the text and scaled so that each marker's own peak is 100 percent: S100B within six hours, UCH-L1 at seven to nine hours, GFAP at twenty to twenty-four hours, and neurofilament light between roughly ten days and six weeks [10]. The shaded region marks the first 72 hours, a period during which sensitivity changes for many acute diagnostic measures [1]. The time axis is compressed (logarithmic) so that hours and weeks fit on one line. The curves are illustrative and are not drawn from measured data. The shading is not a universal Criterion 4 deadline, a common clearance time for all proteins, or the FDA-cleared collection window for every assay.

Two uses of blood biomarkers need to be kept distinct. The ACRM criteria recognize an elevated biomarker indicative of intracranial injury as one possible acute finding within a clinical diagnostic pathway [1]. FDA clearance answers a narrower question about a specified assay and use. For example, the whole-blood GFAP/UCH-L1 i-STAT assay is cleared to help assess the need for head CT in adults with suspected mild traumatic brain injury within twenty-four hours, alongside other clinical information [20]. In the earlier ALERT-TBI validation study, the combined assay showed sensitivity of 0.98 and specificity of 0.36 for CT-visible traumatic intracranial injury [11]. In plain terms, it identified 98 of every 100 patients with such a CT finding, but was also positive in 64 of every 100 patients whose CT was negative. Those figures describe that study and assay, not every biomarker platform. A low result can help rule out CT-visible injury in the population and time window for which the test was validated. An elevated result may support further assessment, but does not by itself diagnose concussion; a low result does not exclude all brain injury. The clinical diagnostic framework and an assay’s cleared CT-triage use are related, but they are not interchangeable.

Two further limitations should be stated. These proteins are not found only in the brain. S100B in particular rises with strenuous exercise and with injuries elsewhere in the body, and its level is influenced by the pigment cells of the skin and therefore by skin color [10]. In one large multicenter analysis, panels of several markers did no better than GFAP alone at predicting an abnormal CT scan [10]. There is also evidence beyond CT triage. In TRACK-TBI, GFAP measured within twenty-four hours was higher, on average, in patients with a negative CT whose later MRI showed traumatic abnormalities than in those whose MRI was also negative [12]. This was an association, not a diagnostic rule for every patient. Early NfL was associated with incomplete recovery in a cohort of 107 patients with mild traumatic brain injury [22]. In a larger study of CT-negative mild injury, however, adding GFAP, NfL, and S100B did not improve prediction beyond the best clinical model [23]. These findings do not establish a universal NfL cutoff that diagnoses concussion or predicts an individual's recovery. Prognostic performance depends on the population, sampling time, assay, and outcome being predicted; the American College of Emergency Physicians advises against using current diagnostic tools, including biomarkers, to reliably predict who will develop persistent post-concussion symptoms [13].

Evoked and event-related potentials measure how long the brain takes to respond to something it sees or hears. Electrodes on the scalp record the brain's electrical answer to a flash of light or a tone. The stimulus is repeated many times and the responses are averaged. The computer then measures the delay between the stimulus and the answer in thousandths of a second (milliseconds). Sensory evoked potentials time the visual, auditory, and touch pathways. Event-related potentials such as the P300 time the later steps in which the brain recognizes a stimulus and decides what to do about it, which makes them a measure of attention and processing. Their value is that timing matters: a patient may answer correctly but slowly, understand a task but need more effort to do it, and have normal strength and speech with delayed processing underneath. The response is locked to the stimulus. The early sensory components occur whether or not the patient tries and are largely outside voluntary control. The later cognitive components, such as the P300, depend on attention and task engagement. Both depend on the recording protocol and the patient's state [14]. Figure 7.8 shows what is recorded and how a delay (latency) is reported against the normal range.

Research also asks whether current electrophysiology contains evidence of past concussion. Boshra and colleagues studied a research classifier using ERP and wavelet features in retired athletes; its results cannot be transferred to routine P300 measurements on a different platform [30]. The 2021 team-physician consensus found insufficient evidence to recommend EEG or evoked potentials for sport-related

concussion diagnosis or treatment [31]. For retrospective concussion attribution, such findings should therefore be described as investigational adjunctive information and interpreted according to the particular protocol, rather than presented as an independently established test of remote traumatic causation.



Figure 7.8. An evoked potential and how it is reported. (A) The brain's averaged electrical answer, recorded over the back of the head (parietal region), to repeated flashes of light. Time runs left to right from 0.2 seconds before the flash to one second after it, with the flash at 0. The bumps and dips are the brain's response, and the small markers show the points whose timing the device measured. (B) The device's summary rows for one of those points, the visual N2, in the same patient at two studies twenty-two months apart. The brain took 256 milliseconds to reach it at the first study and 228 at the second; normal is under 220. The colored bar is the device's scale from normal (green) to markedly abnormal (red), and the circle marks the patient's result. In plain terms, this patient's brain registered what the eyes saw more slowly than normal on both occasions, with partial improvement over two years. The N2 is a later component and is read against the task protocol, the patient's attention, and the device's reference data. Recording from a patient evaluated in Dr. Pappolla's outpatient neurology clinic; identifying information removed.

Autonomic assessment examines the automatic (autonomic) nervous system, the part that regulates heart rate, blood pressure, sweating, body temperature, and the response to exertion without any conscious control. After a concussion some patients develop dizziness or a racing heart on standing, exhaustion after mild activity, heat intolerance, and worsening of all their symptoms after exertion. These complaints are frequently attributed to anxiety or to being out of shape. Testing measures what the body actually does. It records how heart rate varies from beat to beat (heart rate variability), and how heart rate and blood pressure respond to standing up, deep

breathing, and bearing down (the Valsalva maneuver). It also measures how the skin sweats in response to a stimulus (sudomotor testing). In some laboratories, it measures how the body copes with being tilted upright on a table [15,16]. An abnormal result means the body's automatic regulation is not responding normally. That is a measurable finding, but medication, hydration, breathing, pain, anxiety, cardiovascular disease, and deconditioning can all affect it. Physiological and psychological contributors can coexist. Heart rate variability in particular should be interpreted with care: the research shows an association between concussion history and reduced variability, but the methods used across studies are inconsistent [17].

Quantitative electroencephalography starts with an ordinary EEG, the recording of the brain's electrical rhythms from scalp electrodes, and applies computer analysis to it (Figure 7.9). The computer measures how strong each rhythm is in each region, whether the two sides of the brain match, and how closely different regions work together (coherence and connectivity), and compares each measurement with a database of healthy people of the same age. Candidate qEEG markers of mild traumatic brain injury continue to be reported [18]. The difficulty is that the slower rhythms increase whenever the brain is not working normally for any reason: injury, but also fatigue, sleep deprivation, medication, metabolic illness, aging, and prior injury, and no EEG pattern is unique to mild traumatic brain injury [19]. An abnormal qEEG therefore shows that a region's electrical activity is outside the normal range; it does not by itself say why.

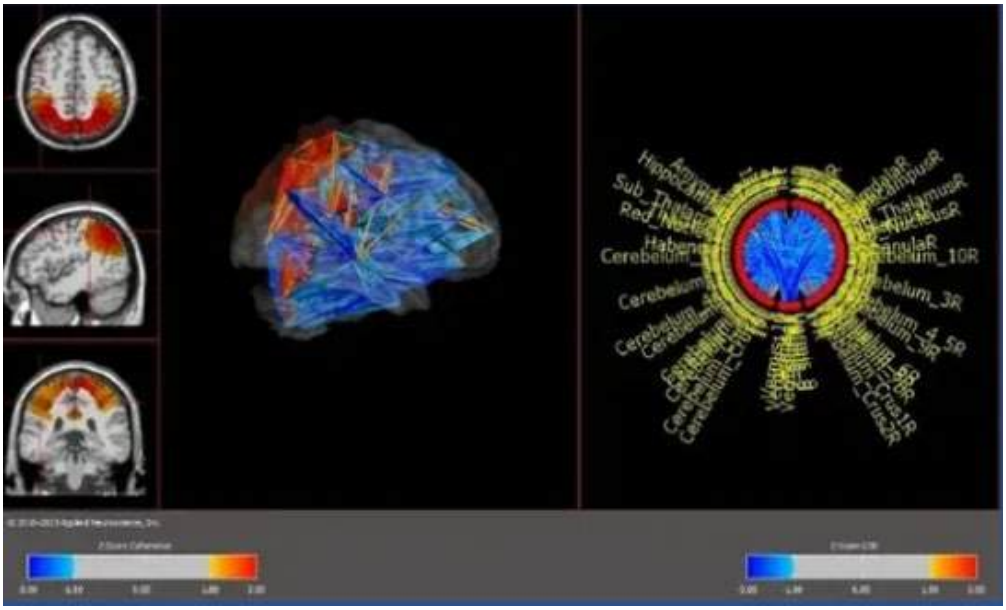


Figure 7.9. Computer-generated qEEG displays from a patient with traumatic brain injury: regional maps (left), a three-dimensional connection display (center), and a

circular network representation (right). Blue and red indicate negative and positive deviations on the displayed z-score scales. These patterns do not by themselves establish the cause of an abnormality.

Diffusion tensor imaging is an MRI technique that measures how water moves through brain tissue. In healthy white matter, the brain's wiring, water moves mainly along the fibers, like water along a bundle of straws; where fibers have been stretched or torn, the movement becomes less orderly, and the technique can detect that change even when the ordinary MRI pictures look normal. Because trauma stretches and shears these fibers, the technique has attracted great interest. The results, however, vary with the scanner, the acquisition settings, the processing method, the patient's age, prior injury, and vascular risk factors, and no universally accepted signature of mild traumatic brain injury exists [4,5]. It is considerably more reliable for showing that groups of injured patients differ from groups of healthy people than for deciding whether one particular individual was injured. Chapter 8 takes it up in detail, together with the rest of imaging.

A note on integrated testing platforms

Several office-based systems combine multiple measurements into a single session, typically some combination of electroencephalographic frequency analysis, quantitative electroencephalography, evoked and event-related potentials, computerized behavioral testing, and a heart rate variability segment (Figure 7.10).

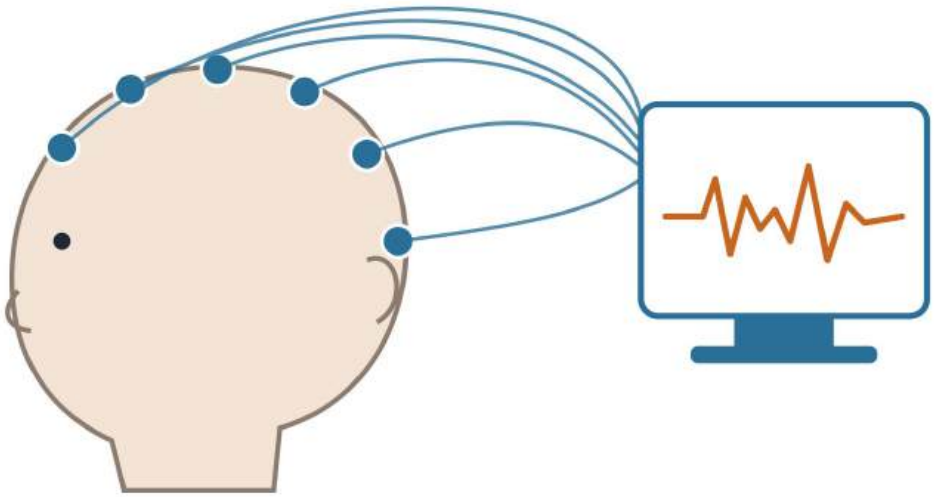


Figure 7.10. Acquisition for an office-based neurophysiology platform. Scalp electrodes record the brain's electrical activity while the patient rests, responds to flashes and tones, and performs a button-press task. One session yields several separate measurements: the conventional electroencephalogram, evoked and event-related potentials, quantitative spectral analysis, a behavioral reaction time test, and in some systems a heart rate variability segment. Each is a component with its own reference range and should be reported as such.

A session produces several measurements, each of which needs interpretation. The brand of equipment does not answer the clinical questions: which physiological systems were measured, were the data technically adequate, and do the abnormalities make sense given the history, examination, and symptoms?

This matters when writing a report or giving testimony. Saying that a neurophysiology study was abnormal leaves the reader with too little information. Specify which component was abnormal, which system it measures, and whether the abnormality fits the clinical picture. Identifying the components separately makes the report more informative and more defensible.

How medical tests are interpreted

The same principles guide diagnostic testing throughout medicine. They deserve a close look here because they are frequently misapplied to traumatic brain injury. Understanding that ordinary logic helps the reader follow the interpretation of each test in this chapter.

A useful finding can have more than one cause

Statistical specificity is the proportion of people without the target condition who test negative. It is an important measure of test performance, considered alongside sensitivity and the intended clinical use [24]. A different question is whether a finding occurs in only one disease. Few findings are that exclusive. Even apparently decisive results need context: a positive culture may represent contamination or harmless colonization, and a disease-causing genetic variant may have different effects in different people. A result can help answer a clinical question without uniquely identifying its cause.

Consider blood glucose. An elevated value supports a diagnosis of diabetes, but a single elevated value does not establish it. The patient may have eaten an hour earlier, may be acutely ill, may be recovering from surgery, may be under severe physiological stress after trauma, or may be taking corticosteroids. The range of possible causes does not make glucose measurement useless; it remains among the most useful tests in medicine. The clinician's task is to decide whether the result makes sense in this patient's situation, taking account of the reasons the glucose could be elevated.

The same applies to every technique in this chapter. Abnormal smooth pursuit occurs after traumatic brain injury and also with medication effects, fatigue, inattention, migraine, cerebellar disease, brainstem disease, and intoxication. Electroencephalographic slowing occurs with sleep deprivation, medication, metabolic disturbance, aging, and prior injury. Impaired balance occurs with vestibular disease, neuropathy, orthopedic injury, pain, visual impairment, medication, and fear of falling. None of this makes the findings meaningless. It means they must be interpreted rather than read.

Interpreting nonspecific findings

In contested settings, the fact that an abnormality can have several causes is sometimes treated as disqualifying. The argument takes the form: because this abnormality can occur in other conditions, it cannot support traumatic brain injury.

That reasoning would set aside most of clinical medicine. Fever occurs with infection, inflammation, malignancy, drug reaction, heat injury, and autoimmune disease. Chest pain occurs with coronary disease, reflux, anxiety, musculoskeletal strain, pulmonary embolism, and pneumonia. Headache occurs with migraine, trauma, hypertension, infection, tumor, medication overuse, and cervical injury. No physician discards thermometry, electrocardiography, or the neurological examination on the ground that findings may have more than one cause.

Nonspecific does not mean irrelevant. It means the finding has more than one possible cause, and that determining which cause applies to this patient is the clinician's task rather than the test's. The same reasoning requires attention to alternatives. An abnormality that fits the trauma does not settle the matter if a better explanation exists. An evaluator who raises that alternative is applying Criterion 6 and addressing the finding directly.

The evaluation begins with timing: did the symptom begin after the trauma? It then asks whether the abnormality is anatomically and physiologically plausible given the mechanism, whether it matches the patient's complaints, and whether it fits the examination. Do other objective findings point in the same direction? Does a better alternative explanation exist? Chapter 5 sets out the differential that last question requires.

An example makes the reasoning concrete. A patient with no prior dizziness develops immediate dizziness, visual motion intolerance, headache, and imbalance after a collision. Later testing demonstrates reproducible eye movement and balance abnormalities. These findings may support a clinical attribution to trauma when their pattern fits the history and examination and competing explanations have been examined. The report should also identify the evidence for the original brain injury, rather than assuming that post-collision dizziness establishes it. The same eye movement abnormality in a patient with longstanding cerebellar disease or substantial sedative exposure requires a different interpretation.

Convergence

The principle that gives objective testing much of its force is convergence. One abnormal test is a clue. Several abnormalities across independent systems, pointing in the same direction and matching the history, are a different order of evidence, because tests measuring different physiological systems are less likely to be abnormal in a consistent direction by chance. Two cautions are essential to that interpretation. First, different instruments do not guarantee independent errors. Eye movements and balance share pathways, and fatigue, medication, pain, and inattention can affect several measures at once. Second, the more endpoints a battery tests, the more abnormal values it will produce by chance alone. The weight of convergent evidence therefore depends on the validity and the correlation of its components, not simply on the number of abnormal results.

A useful distinction within convergent evidence concerns dependence on task performance. Cognitive scores, voluntary eye movement tasks and standing balance all

require cooperation. Some recorded responses, including particular forms of nystagmus and early sensory evoked potentials, are less dependent on deliberate performance. They still require adequate sensory input and recording quality. Reflex physiology does not make a measurement immune to artifacts or patient state, and posturography should not be treated as an involuntary test. Agreement between measurements with different vulnerabilities can strengthen the interpretation when shared confounders have been considered.

This should be stated carefully rather than absolutely. It is accurate that certain physiological responses are not under voluntary control. It is an overstatement to say that objective testing cannot be faked or that a battery rules out symptom amplification, and a claim in that form should not be made. The defensible statement has three parts. Measures differ in their vulnerability to deliberate manipulation. Agreement across measures of differing vulnerability strengthens the assessment. When validity is in question, performance and symptom validity measures interpreted by a qualified examiner are the tools for that question.

The keyhole principle

Every technique in this chapter examines one narrow window into nervous system function. This has consequences in both directions, and both must be respected.

A normal result does not exclude injury. A normal computed tomography scan does not exclude concussion, and neither does a normal magnetic resonance image, a normal videonystagmography study, a normal posturography study, a normal evoked potential study, a normal quantitative electroencephalogram, or a normal autonomic screen. A valid normal finding is evidence of preserved function within the scope of that measurement, and nothing more. The point matters most for standard cognitive testing, which is a resting measure, administered one task at a time in quiet conditions. The “couch and stairs” analogy from the Introduction applies here: test difficulty under load with a reliable method rather than presuming the result. Chapter 10 develops this point, and Chapter 11 shows the consequences domain by domain.

An abnormal result requires interpretation. It can document dysfunction and contribute to a diagnosis, but it does not by itself establish traumatic causation.

Holding both halves is what distinguishes competent interpretation from advocacy in either direction.

What follows

Chapter 8 follows these questions into imaging, from computed tomography to diffusion tensor imaging. For each technique, it asks what is measured, what the result contributes to the clinical evaluation, what it cannot establish, and how it should be reported. Chapters 10 to 12 turn to the consequences of the injury that most often bring patients to this kind of testing.

Fast take: main points from chapter 7

• Objective findings have defined roles in the ACRM pathways. A qualifying acute examination or laboratory finding may contribute through Criterion 4; trauma-related structural imaging may contribute through Criterion 5. The remaining requirements, including those for the mild qualifier, still apply [1,32].

• Objective findings alone never establish the diagnosis under Criterion 4, and that criterion usually cannot be established in a post-acute assessment [1].

• Later testing can contribute to a retrospective diagnosis and document present impairment. Its weight depends on the particular finding, technical validity, symptom chronology, examination and alternatives. It does not automatically substitute for an acute finding.

• Oculomotor, balance, and cognitive testing examine domains named in the criteria. DTI metrics and ERP methods require separate evidence for their intended use and are not designated diagnostic tests by those criteria. The ACNS guideline found insufficient evidence for qEEG in diagnosing mild TBI [1,6,30-32].

• Testing serves four purposes beyond diagnosis: demonstrating dysfunction, identifying the systems involved, directing treatment, and documenting course. In post-acute care the middle two often matter most.

• Blood biomarkers are the fourth domain in Criterion 4, and their interpretation depends strongly on timing. Cleared GFAP/UCH-L1 assays help assess the need for CT within their validated populations and collection windows. The sensitivity of 0.98 and specificity of 0.36 reported in ALERT-TBI concerned CT-visible injury, not all concussion. Biomarker evidence can contribute to a clinical assessment, but neither an elevated nor a low result settles the diagnosis on its own [1,10,11,20].

• An integrated testing platform is a collection of instruments, not a single test. Report which component was abnormal and what system it measures.

• A useful finding need not have only one possible cause. Statistical specificity remains important; the clinical meaning of an abnormal result also depends on the history, competing explanations, and the question being asked.

• Convergence across independent systems gives objective assessment its force. This is particularly so when measures that depend on cooperation agree with measures that largely do not depend on it. The interpretation must allow for shared confounders and the number of endpoints tested.

• Every technique is a narrow keyhole. A normal result does not exclude injury; an abnormal result can support the clinical assessment but does not establish traumatic causation alone.

References

1. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036
2. Mucha A, Collins MW, Elbin RJ, Furman JM, Troutman-Enseki C, DeWolf RM, Marchetti G, Kontos AP. A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: preliminary findings. Am J Sports Med. 2014;42(10):2479-2486. doi:10.1177/0363546514543775
3. Kontos AP, Deitrick JM, Collins MW, Mucha A. Review of vestibular and oculomotor screening and concussion rehabilitation. J Athl Train. 2017;52(3):256-261. doi:10.4085/1062-6050-51.11.05
4. Douglas DB, Iv M, Douglas PK, Anderson A, Vos SB, Bammer R, Zeineh M, Wintermark M. Diffusion tensor imaging of traumatic brain injury: potentials and challenges. Top Magn Reson Imaging. 2015;24(5):241-251. doi:10.1097/RMR.0000000000000062
5. Expert Panel on Neurological Imaging; Shih RY, Burns J, Ajam AA, et al. ACR Appropriateness Criteria: Head Trauma: 2021 update. J Am Coll Radiol. 2021;18(5S):S13-S36. doi:10.1016/j.jacr.2021.01.006
6. Tenney JR, Gloss D, Arya R, Kaplan PW, Lesser R, Sexton V, Nuwer M. Practice guideline: use of quantitative EEG for the diagnosis of mild traumatic brain injury: report of the Guideline Committee of the American Clinical Neurophysiology Society. J Clin Neurophysiol. 2021;38(4):287-292. doi:10.1097/WNP.0000000000000853
7. Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport, Amsterdam, October 2022. Br J Sports Med. 2023;57(11):695-711.

8. Defense Health Agency, Department of Defense. Clinical recommendation: assessment and management of dizziness and visual disturbances following concussion and mild traumatic brain injury. January 2026.
9. Arrieux JP, Cole WR, Ahrens AP. A review of the validity of computerized neurocognitive assessment tools in mild traumatic brain injury assessment. Concussion. 2017;2(1):CNC31. doi:10.2217/cnc-2016-0021
10. Hossain I, Marklund N, Czeiter E, Hutchinson P, Buki A. Blood biomarkers for traumatic brain injury: a narrative review of current evidence. Brain Spine. 2024;4:102735. doi:10.1016/j.bas.2023.102735
11. Bazarian JJ, Biberthaler P, Welch RD, Lewis LM, Barzo P, Bogner-Flatz V, et al. Serum GFAP and UCH-L1 for prediction of absence of intracranial injuries on head CT (ALERT-TBI): a multicentre observational study. Lancet Neurol. 2018;17(9):782-789.
12. Yue JK, Yuh EL, Korley FK, Winkler EA, Sun X, Puffer RC, et al. Association between plasma GFAP concentrations and MRI abnormalities in patients with CT-negative traumatic brain injury in the TRACK-TBI cohort: a prospective multicentre study. Lancet Neurol. 2019;18(10):953-961.
13. American College of Emergency Physicians Clinical Policies Subcommittee. Clinical policy: critical issues in the management of adult patients presenting to the emergency department with mild traumatic brain injury. Ann Emerg Med. 2023;81(5):e63-e105. doi:10.1016/j.annemergmed.2023.01.014
14. Li H, Li N, Xing Y, Zhang S, Liu C, Cai W, Hong W, Zhang Q. P300 as a potential indicator in the evaluation of neurocognitive disorders after traumatic brain injury. Front Neurol. 2021;12:690792. doi:10.3389/fneur.2021.690792
15. Esterov D, Greenwald BD. Autonomic dysfunction after mild traumatic brain injury. Brain Sci. 2017;7(8):100. doi:10.3390/brainsci7080100
16. Pertab JL, Merkley TL, Cramond AJ, Cramond K, Paxton H, Wu T. Concussion and the autonomic nervous system: an introduction to the field and the results of a systematic review. NeuroRehabilitation. 2018;42(4):397-427. doi:10.3233/NRE-172298
17. Wesolowski E, Ahmed Z, Di Pietro V. History of concussion and lowered heart rate variability at rest beyond symptom recovery: a systematic review and meta-analysis. Front Neurol. 2024;14:1285937. doi:10.3389/fneur.2023.1285937
18. Lewine JD, Plis S, Ulloa A, et al. Quantitative EEG biomarkers for mild traumatic brain injury. J Clin Neurophysiol. 2019;36(4):298-305. doi:10.1097/WNP.0000000000000588
19. Nuwer MR, Hovda DA, Schrader LM, Vespa PM. Routine and quantitative EEG in mild traumatic brain injury. Clin Neurophysiol. 2005;116(9):2001-2025. doi:10.1016/j.clinph.2005.05.008

20. US Food and Drug Administration. i-STAT TBI Cartridge with the i-STAT Alinity System: 510(k) substantial equivalence determination decision summary. K234143. 2024. Accessed October 25, 2026. https://www.accessdata.fda.gov/cdrh_docs/reviews/K234143.pdf
21. Bazarian JJ, Zetterberg H, Buki A, et al. Blood-based biomarkers for improved characterization of traumatic brain injury: recommendations from the 2024 National Institute for Neurological Disorders and Stroke Traumatic Brain Injury Classification and Nomenclature Initiative Blood-Based Biomarkers Working Group. J Neurotrauma. 2025;42(13-14):1065-1085. doi:10.1089/neu.2024.0581 https://doi.org/10.1089/neu.2024.0581
22. Hossain I, Mohammadian M, Takala RSK, et al. Early levels of glial fibrillary acidic protein and neurofilament light protein in predicting the outcome of mild traumatic brain injury. J Neurotrauma. 2019;36(10):1551-1560. doi:10.1089/neu.2018.5952 https://doi.org/10.1089/neu.2018.5952
23. Richter S, Winzeck S, Correia MM, et al. Predicting recovery in patients with mild traumatic brain injury and a normal CT using serum biomarkers and diffusion tensor imaging (CENTER-TBI): an observational cohort study. EClinicalMedicine. 2024;75:102751. doi:10.1016/j.eclinm.2024.102751 https://pmc.ncbi.nlm.nih.gov/articles/PMC11667275/
24. US Food and Drug Administration. Statistical guidance on reporting results from studies evaluating diagnostic tests: guidance for industry and FDA staff. March 2007. Accessed October 25, 2026. https://www.fda.gov/regulatory-information/search-fda-guidance-documents/statistical-guidance-reporting-results-studies-evaluating-diagnostic-tests-guidance-industry-and-fda
25. Eggers SDZ, Bisdorff A, von Brevern M, Zee DS, Kim JS, Perez-Fernandez N, Welgampola MS, Della Santina CC, Newman-Toker DE. Classification of vestibular signs and examination techniques: Nystagmus and nystagmus-like movements: Consensus document of the Committee for the International Classification of Vestibular Disorders of the Bárány Society. J Vestib Res. 2019;29(2-3):57-87. doi:10.3233/VES-190658.
26. Kim JS, Newman-Toker DE, Kerber KA, Jahn K, Bertholon P, Waterston J, Lee H, Bisdorff A, Strupp M. Vascular vertigo and dizziness: Diagnostic criteria: Consensus document of the committee for the classification of vestibular disorders of the Bárány society. J Vestib Res. 2022;32(3):205-222. doi:10.3233/VES-210169.
27. Kraus M, Hassannia F, Dabiri S, Vergara Olmos G, Rutka JA. Central vestibular dysfunction in head injury. J Otolaryngol Head Neck Surg. 2024;53:19160216241250354. doi:10.1177/19160216241250354.
28. Taylor RL, Wise KJ, Taylor D, Chaudhary S, Thorne PR. Patterns of vestibular dysfunction in chronic traumatic brain injury. Front Neurol. 2022;13:942349. doi:10.3389/fneur.2022.942349.

29. Silverberg ND, Lee K, Mikolić A, et al. Action Collaborative on Traumatic Brain Injury Care: Adapted Clinical Practice Guideline. Ann Fam Med. 2025;23(6):552-569. doi:10.1370/afm.250352.
30. Boshra R, Dhindsa K, Boursalie O, Ruiter KI, Sonnadara R, Samavi R, Doyle TE, Reilly JP, Connolly JF. From group-level statistics to single-subject prediction: machine learning detection of concussion in retired athletes. IEEE Trans Neural Syst Rehabil Eng. 2019;27(7):1492-1501. doi:10.1109/TNSRE.2019.2922553.
31. Herring S, Kibler WB, Putukian M, et al. Selected issues in sport-related concussion (SRC|mild traumatic brain injury) for the team physician: a consensus statement. Br J Sports Med. 2021;55(22):1251-1261. doi:10.1136/bjsports-2021-104235.
32. Iverson GL, Dams-O'Connor K, Panenka WJ, Stephens JA, Lockman SD, Al Lawati Z, Lequerica AH, McPherson JI, Kamins J, Silverberg ND. New diagnostic criteria for mild traumatic brain injury: medical-legal considerations. Arch Phys Med Rehabil. 2025;106(10):1615-1619. doi:10.1016/j.apmr.2025.06.015.

Chapter 8 Imaging in traumatic brain injury

Chapter overview

Brain images are easy to misread. A normal scan may be taken as proof that nothing happened; a colorful one may be treated as proof of injury and its extent. Understanding what each scan measures helps the reader assess both interpretations. This chapter explains, for a reader without medical training, what computed tomography and magnetic resonance imaging show after a head injury and where their limits lie. It examines why a conventional scan is usually normal after a concussion, what that result means, and how diffusion tensor imaging measures the condition of the brain's wiring in a way that conventional scans cannot.

The chapter uses pictures to explain bleeding and swelling on CT, the different views provided by MRI, and enlargement of the fluid spaces following loss of brain tissue. It then examines diffusion tensor imaging: what it measures, what the colors mean, how a tractography image is built, and how a report compares one patient's values with those of healthy people. The second half works through an actual diffusion tensor report from Dr. Pappolla's outpatient neurology clinic, step by step. Alongside the report, it considers the questions a careful reader should ask, the technique's limits, and the objections raised to it. The chapter closes with a method for reading an imaging report in a contested case.

What imaging can answer and what it cannot

Imaging answers different questions at different times. In the emergency department, the question is whether there is bleeding, swelling, a fracture, or a shift of the brain that needs immediate action, and the scan that answers it is a CT. Weeks or months later, when symptoms have not resolved, the questions change: is there an injury that the first scan was not designed to see, is there another explanation for the symptoms, and what does the pattern of injury mean for recovery? Those questions call for MRI, and sometimes for advanced MRI techniques. The right study is the one suited to the question and to the time since injury, not the one with the most impressive pictures [1].

Two principles govern everything that follows. The first is the keyhole principle introduced in Chapter 7: every imaging technique looks at the brain through a narrow opening, and sees only what it was built to see. CT sees blood, bone, and shifts of the brain at a scale of millimeters. Conventional MRI sees tissue water, blood products, and scarring at a somewhat finer scale. Neither sees the stretching and disconnection of individual nerve fibers that is the main injury of a concussion, which happens at a scale

a thousand times smaller than anything a clinical scanner can resolve. A normal CT or MRI therefore does not exclude a concussion, any more than a normal chest x-ray excludes asthma, and an abnormal image does not explain every symptom that followed an accident. In both directions the evaluator has to decide what the finding is, whether its location and timing fit the complaint, and whether something else explains it better. Diagnosis begins with the history and the clinical findings; imaging adds to that assessment and does not replace it [2].

The second principle is the distinction between seeing a lesion and measuring a difference. A bleed on a CT scan can be pointed to, measured, and followed over time. A diffusion tensor value outside a reference range is a number that differs from those obtained in healthy people. Its meaning depends on how the scan was acquired, how the numbers were calculated, how much healthy people vary, and whether the comparison group was appropriate. Both kinds of finding can be valuable, but they support different inferences. A report should make clear which kind it is presenting.

Reading a scan: a short orientation

A brain scan is a stack of slices cut in three named directions. Axial (or transverse) slices are horizontal, as if the head were cut like a loaf of bread from the top down. They are viewed from below, so the patient's right side appears on the viewer's left. Sagittal slices are vertical and run from front to back, dividing left from right. Coronal slices are vertical and run from side to side, dividing front from back. Most of the images in this chapter are axial. Three-dimensional renderings are common in diffusion tensor reports and can be viewed from any angle. Their viewing direction has to be stated separately.

On a CT scan, dense materials are bright and less dense materials are dark: bone is white, fresh blood is bright, brain tissue is grey, and the cerebrospinal fluid that fills the ventricles and surrounds the brain is dark. On an MRI, brightness depends on the sequence, as explained below, and the same structure can appear bright on one sequence and dark on another. In the color maps and tractography pictures of diffusion tensor imaging, the colors are a code for direction, not for health or injury, a point to which the chapter returns.

Computed tomography: the first examination

What it is and when it is used

A CT scan is an x-ray examination in which the beam rotates around the head and a computer reconstructs cross-sectional slices. It takes a few minutes, is available in every emergency department, and is the first imaging study in acute head trauma

because it answers the urgent questions: is there bleeding inside the skull, is the skull fractured, is the brain being pushed out of position, and are the fluid spaces blocked? Not every head injury needs a scan; validated decision rules based on the patient's age, the mechanism of injury, the symptoms, and the examination identify who should have one [1]. When a scan is needed, CT comes first, and an MRI should never delay the evaluation of a patient who may have an expanding bleed [1].

CT depicts differences in how much the tissues absorb x-rays. Reading it involves more than finding a bright patch of blood. The radiologist examines the surface of the brain, the deep structures, the ventricles (the fluid-filled chambers inside the brain), the cisterns (the fluid spaces around the base of the brain), and the symmetry of the two sides. Loss of the normal distinction between grey and white matter, compressed fluid spaces, or a shift of the midline indicate swelling that can be dangerous [3].

What bleeding looks like

The names of traumatic bleeds identify the compartment they occupy and convey information about mechanism and danger (Figure 8.1). An epidural hematoma lies between the skull and the tough outer membrane around the brain (the dura). It is usually caused by a torn artery under a skull fracture and has a lens shape because the dura is firmly attached to the skull and limits its spread. A subdural hematoma lies beneath the dura, on the surface of the brain. It is usually caused by torn veins stretched by movement of the brain within the skull and forms a crescent that follows the brain's curve. Subarachnoid blood lies in the thin space over the surface of the brain and fills the grooves between its folds. Intraventricular blood is inside the fluid chambers. A contusion is a bruise of the brain tissue itself, a mixture of blood and swelling. An intraparenchymal hematoma is a larger, more solid collection of blood within the brain. Several of these can coexist. Their importance depends on size, location, the swelling around them, and the patient's condition, not on the name alone.

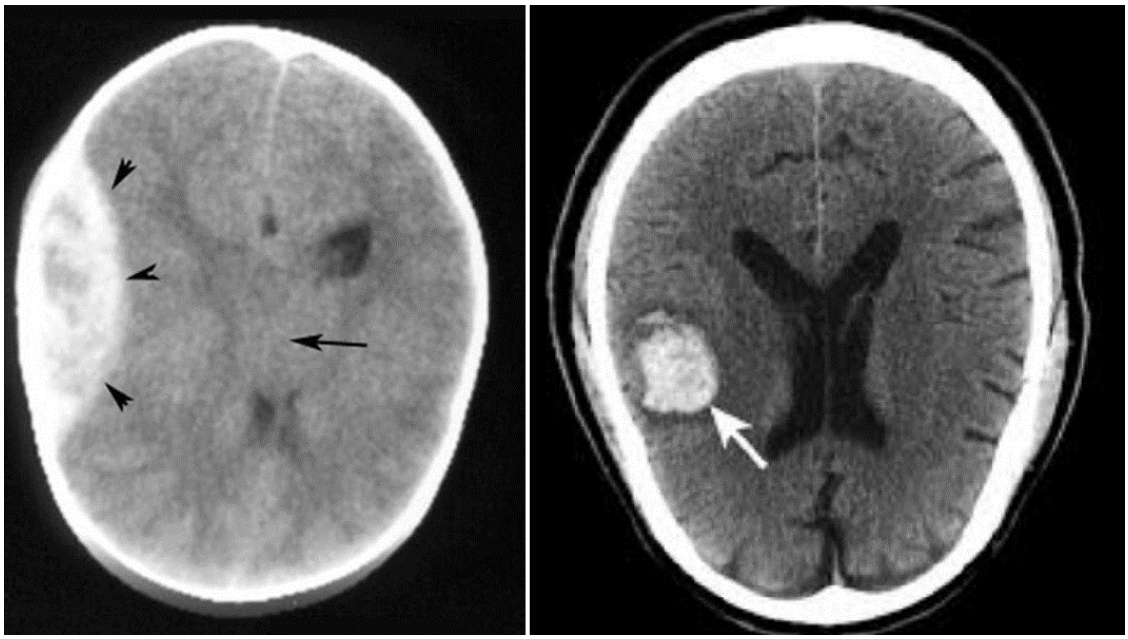


Figure 8.1. Two kinds of bleeding on axial CT scans. Left: a crescent-shaped bright collection along the surface of the brain, the appearance of a subdural hematoma, with the brain pushed across the midline toward the opposite side. Right: a focal bright collection within the brain tissue itself, an intraparenchymal hematoma. Fresh blood is bright on CT; the dark areas within the brain are the fluid-filled ventricles. These images illustrate the shapes; a complete examination and the clinical history are needed for interpretation.

Classifying the CT picture

Neurosurgeons describe the overall CT picture after a serious head injury with the Marshall classification, introduced in 1991 and still in use for research and communication [4]. It sorts scans by three features: whether the cisterns at the base of the brain are open or compressed, whether the midline is shifted and by how much, and whether there is a large mass of blood or bruising. Category I is a scan with no visible abnormality. Category II is a scan with the cisterns open, little or no midline shift, and no large lesion. Category III adds compression of the cisterns, the sign of generalized swelling. Category IV adds a midline shift of more than five millimeters. Categories V and VI describe a large mass lesion, evacuated by surgery or not. The categories were designed for moderate and severe injury and say nothing about concussion, and Category I means no visible abnormality on that scan, not the absence of brain injury [4]. The term “diffuse injury” in the names of the first four categories is a historical label for the CT appearance, not a histological diagnosis of diffuse axonal injury.

What a CT does not show

A CT scan is a snapshot of a particular moment. Bruises and bleeds evolve over the first days, and a lesion too small to see on arrival may be visible on a scan taken a day later [3]. More important for this book, CT is designed for the scale of acute surgical disease. It does not show the microscopic injury to nerve fibers that is the main injury of a concussion, it is insensitive to small old deposits of blood that MRI picks up readily, and it says nothing about how the brain is working. In mild traumatic brain injury the CT scan is normal in the large majority of patients; that is the expected result [1,2].

Magnetic resonance imaging

How it works, in plain terms

An MRI scanner uses a powerful magnet and radio waves to measure signals from the hydrogen atoms in the body's water and fat. By varying how the signals are collected, the scanner produces different "sequences," each sensitive to a different property of tissue, and an MRI examination is a collection of these. T1-weighted images show anatomy in sharp detail and are used to measure the size of structures. T2-weighted images are bright wherever there is extra water, which makes them sensitive to swelling and scarring. FLAIR (fluid-attenuated inversion recovery) is a T2 image with the signal from cerebrospinal fluid suppressed, so that abnormalities next to the fluid spaces stand out; it is the sequence on which the small white spots in the brain's wiring are usually noticed. Diffusion-weighted imaging measures how freely water moves and is used in the acute period to detect tissue in which water movement is restricted by swelling of the cells. Susceptibility-weighted imaging (SWI) and its older relative, the gradient-echo sequence, are exquisitely sensitive to iron, and therefore to the breakdown products of blood, so they reveal tiny old hemorrhages that nothing else shows. No single sequence tells the whole story, and a report should say which sequences were obtained.

What conventional MRI finds after a concussion

MRI is the appropriate study when symptoms persist, when the clinical course does not fit the CT, or when a deficit is unexplained [1]. It is more sensitive than CT to small bruises, to non-bleeding injuries, and to old blood. In a prospective study of 135 patients with mild traumatic brain injury, most of whom had normal CT scans, early MRI showed abnormalities in about a quarter of those with a normal CT, and the presence of bruising or of several small hemorrhages on MRI was associated with worse outcome three months later [5]. MRI adds information; it is not required to diagnose a

concussion, and the same study is a reminder that most patients with mild injury have normal conventional MRI as well.

Three MRI findings carry particular weight when present. Small hemorrhages at the junction of grey and white matter, in the corpus callosum (the bridge of fibers connecting the two hemispheres), or in the brainstem are the classic signature of traumatic axonal injury, the shearing of nerve fibers by rotation of the brain, and SWI reveals far more of them than older sequences do; in an early study of children with diffuse axonal injury, SWI showed several times as many lesions as the conventional sequence [6]. Restricted diffusion, confirmed by a low value on the apparent diffusion coefficient map that accompanies the diffusion-weighted image, has been described in traumatic axonal injury in the acute period [7]; it is not specific to trauma and has to be read with the distribution of the lesions and the clinical setting. And small bright spots in the white matter on FLAIR, when they appear in a young person without vascular risk factors and in a distribution that fits the injury, may represent small areas of scarring; the same spots in an older patient with hypertension more often represent small vessel disease. A careful report describes their location and pattern rather than labeling them traumatic or dismissing them as incidental.

Two technical cautions apply to susceptibility imaging. A dark spot on SWI is a signal effect, not a count of damaged fibers; its apparent size is exaggerated by the physics of the technique, and how many spots are seen depends on the strength of the magnet and the details of the sequence. And a normal SWI does not exclude axonal injury, because most sheared fibers do not bleed. A newer technique, quantitative susceptibility mapping, assigns a number to the magnetic property of each small volume of tissue and can help separate iron from calcium and myelin; a 2024 international consensus set out how it should be acquired and processed for research, and it remains a research tool in brain injury [8].

A normal MRI in a patient with persistent symptoms

Figure 8.2 is a FLAIR image from the conventional MRI of a patient whose diffusion tensor report is read later in this chapter. The brain is normal in appearance: the grey and white matter are symmetric, the ventricles are of normal size, and there are no bright spots, no old blood on the susceptibility sequence, and no bruising. The radiologist read the standard sequences as normal. This is the usual result after a concussion, including in patients with disabling symptoms, and the reason is scale: the injury is to fibers a hundredth of a millimeter across, and the picture is made of elements about a millimeter across. The image is included because it is the picture most often read as proof that nothing is wrong. It establishes that there is no bleeding,

no bruise, no scar, and no tumor or other disease that would explain the symptoms. It does not establish that the wiring is intact, and the rest of this chapter shows why.

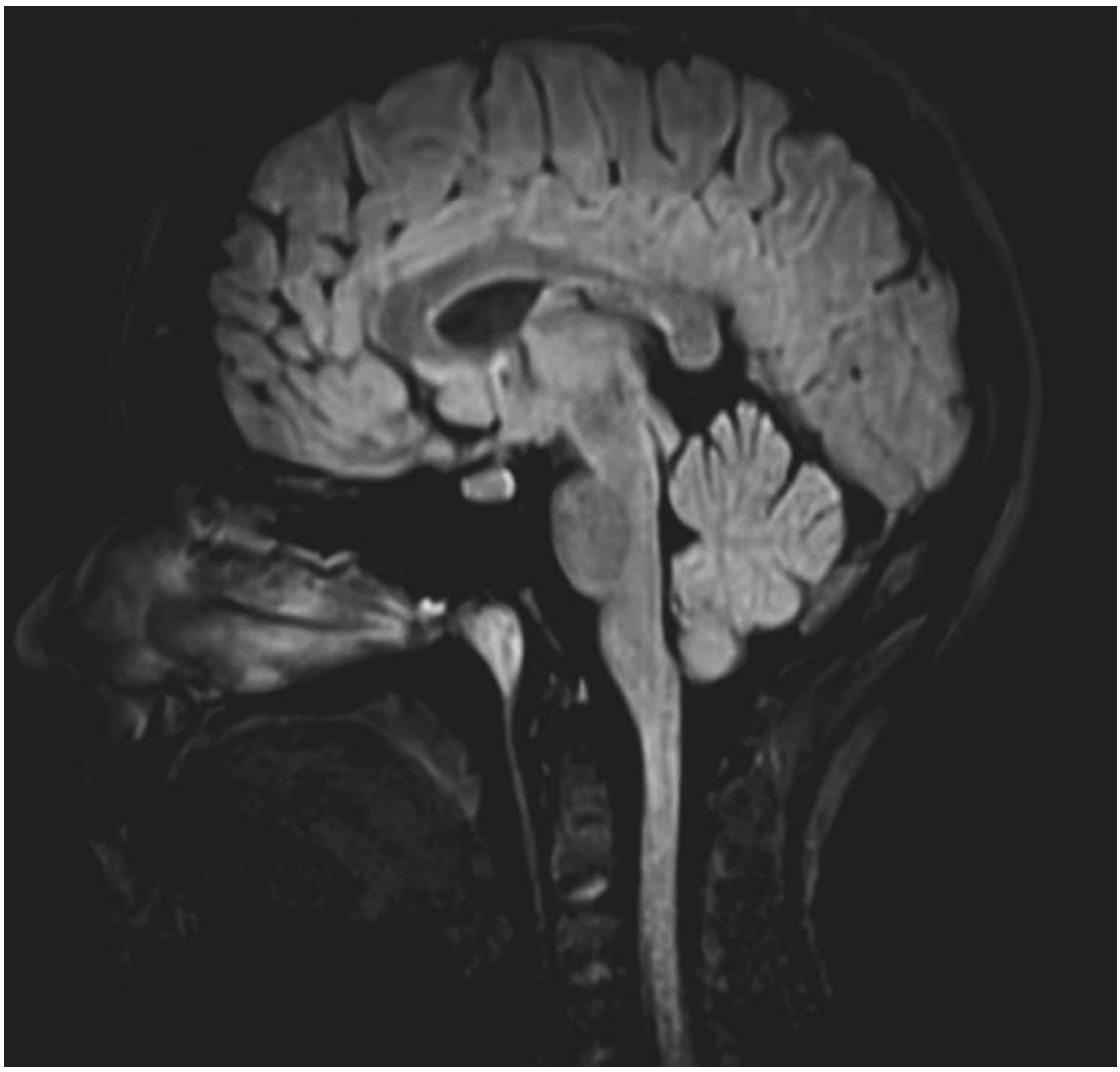


Figure 8.2. Normal conventional MRI in a patient with persistent symptoms after a concussion. A sagittal FLAIR image from the standard MRI examination of a patient in Dr. Pappolla's outpatient neurology clinic with persistent symptoms after a motor vehicle collision. The cerebral hemispheres, the corpus callosum, the ventricles (the dark fluid spaces in the center), the brainstem, the cerebellum, and the grooves on the surface of the brain are all normal, and the radiologist read every standard sequence as normal. The diffusion tensor analysis, shown later in this chapter, was not. Identifying information removed.

Practical limits

MRI involves no radiation, but it is a long examination in a loud, confined space, and a patient with headache, dizziness, and intolerance of noise may find it hard to stay still.

Movement degrades the images and can make a completed examination uninterpretable, a point that becomes important with diffusion imaging. Metal implants are not an absolute barrier, but each has to be identified and its safety conditions confirmed [9]. These practical matters explain why an advanced MRI protocol lasting an hour is not always obtainable in the patients who might benefit from it.

The ventricles: lost tissue or blocked fluid

The ventricles enlarge after brain injury for two different reasons, and telling them apart matters for treatment and for interpretation (Figure 8.3). When brain tissue is lost, the fluid spaces expand to fill the vacated room; this is called ex vacuo enlargement, and it is a sign of atrophy, not of a disorder of the fluid itself. When the circulation or absorption of cerebrospinal fluid is disturbed, pressure builds and the ventricles expand; this is post-traumatic hydrocephalus, and it may be treatable with a shunt [10]. The two can look alike on a single scan, and the distinction is made by comparing with earlier scans, by examining the pattern of enlargement of the ventricles relative to the grooves on the brain's surface, by the clinical course, and in selected cases by measurement of fluid dynamics [10,11]. A large ventricle is a finding; hydrocephalus is a diagnosis, and neither is assigned from ventricular size alone. Ventricular enlargement in a young patient after a significant injury, in the absence of a fluid disorder, is evidence of lost brain tissue, a structural consequence of the injury that can be measured.

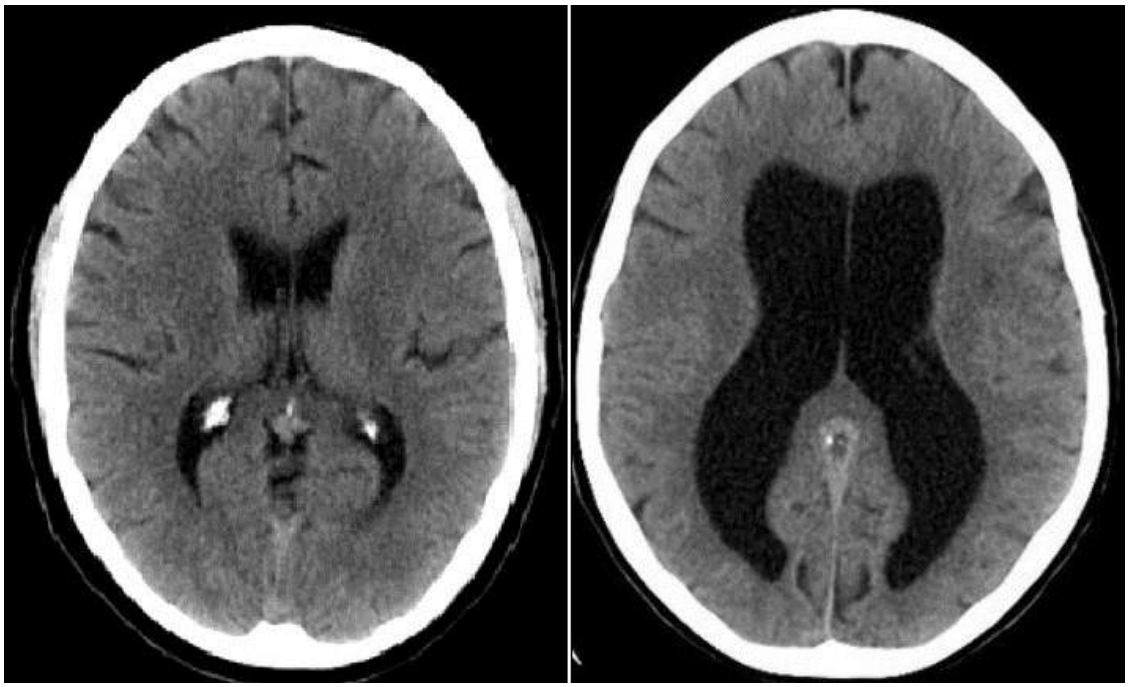


Figure 8.3. Two axial CT scans with ventricles of different size. The dark butterfly-shaped spaces in the center are the lateral ventricles, which are substantially larger in the right image. The pair alone does not establish whether the enlargement is from loss of brain tissue or from a disorder of cerebrospinal fluid circulation, or whether it is new; that determination requires the complete examination, earlier studies when available, and the clinical course.

Diffusion tensor imaging

The idea: water moves along the wires

Diffusion tensor imaging (DTI) is an MRI technique that measures the movement of water molecules in the brain, and it is the technique most likely to show the injury of a concussion because of a simple fact about the brain's wiring. The MRI is good at seeing water molecules. The white matter of the brain is made of nerve fibers (axons), each wrapped in insulation (myelin) and gathered into bundles, like the cables in a telephone trunk line. Water inside and around a healthy bundle moves easily along the fibers and with difficulty across them, in the way that water poured onto a bundle of drinking straws runs along the straws (Figure 8.4). When fibers stretch, break, or lose their insulation, the barriers break down, and water moves more freely in all directions. DTI measures that difference. In each small block of tissue, about two millimeters on a side (a voxel, the three-dimensional equivalent of a pixel), the scanner measures how far water moves in each of many directions over a few thousandths of a second, and a mathematical model (the tensor) summarizes the result [12]. The technique does not

photograph individual fibers; it infers their organization from the behavior of water, and it can detect disorganization in white matter that looks entirely normal on the conventional images [12,13].

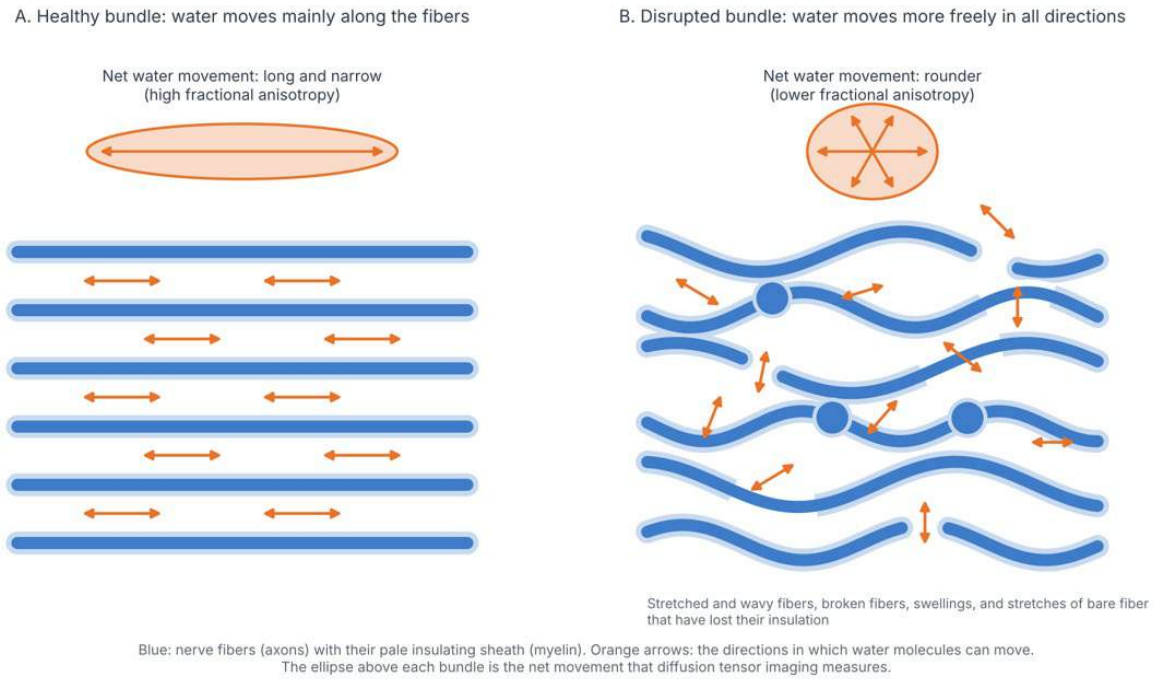


Figure 8.4. Why the movement of water reveals the condition of the brain's wiring. (A) In a healthy bundle of nerve fibers, water moves mainly along the fibers, because the fiber membranes and insulation block movement across them; the movement is strongly directional, which is what a high fractional anisotropy value means. (B) In a disrupted bundle, with fibers stretched, broken, or stripped of insulation, water moves more freely in every direction, and fractional anisotropy falls. Original schematic.

The numbers: FA, MD, axial diffusivity, and radial diffusivity

A DTI report expresses the behavior of water in each region as a few numbers, and a reader needs to know what each one means. Fractional anisotropy (FA) is the one most often quoted. It measures how directional the movement of water is, on a scale from 0, meaning water moves equally in all directions, as in a glass of water, to 1, meaning it moves in only one direction. In healthy, tightly packed white matter such as the corpus callosum, FA is high, typically 0.6 to 0.8; in grey matter it is low; and a reduction in FA within a tract is the most common DTI finding after brain injury [14,15]. Mean diffusivity (MD), also reported as the apparent diffusion coefficient (ADC), is the average amount of water movement in all directions; it rises when barriers break down and tissue becomes looser. Axial diffusivity is the movement along the main direction of the fibers, and radial diffusivity is the movement across them; in experimental work, a fall in axial

diffusivity has been associated with injury to the fibers themselves and a rise in radial diffusivity with loss of their insulation, though these correspondences are not exact in human disease [16]. Figure 8.5 illustrates how two regions can have the same average amount of water movement and very different directionality, which is why FA and MD measure different things and are reported together.

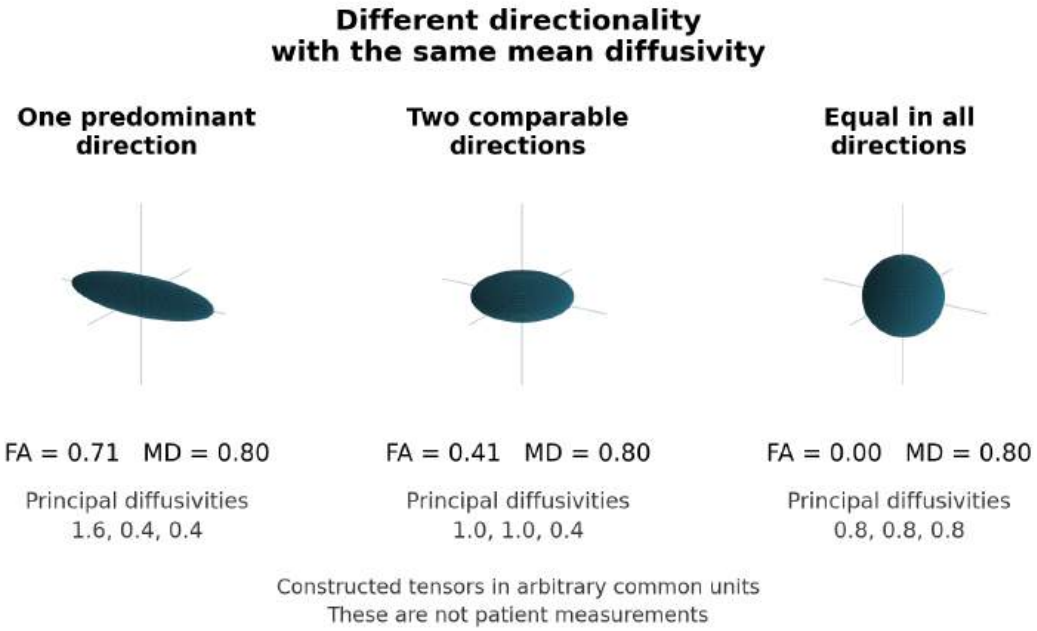
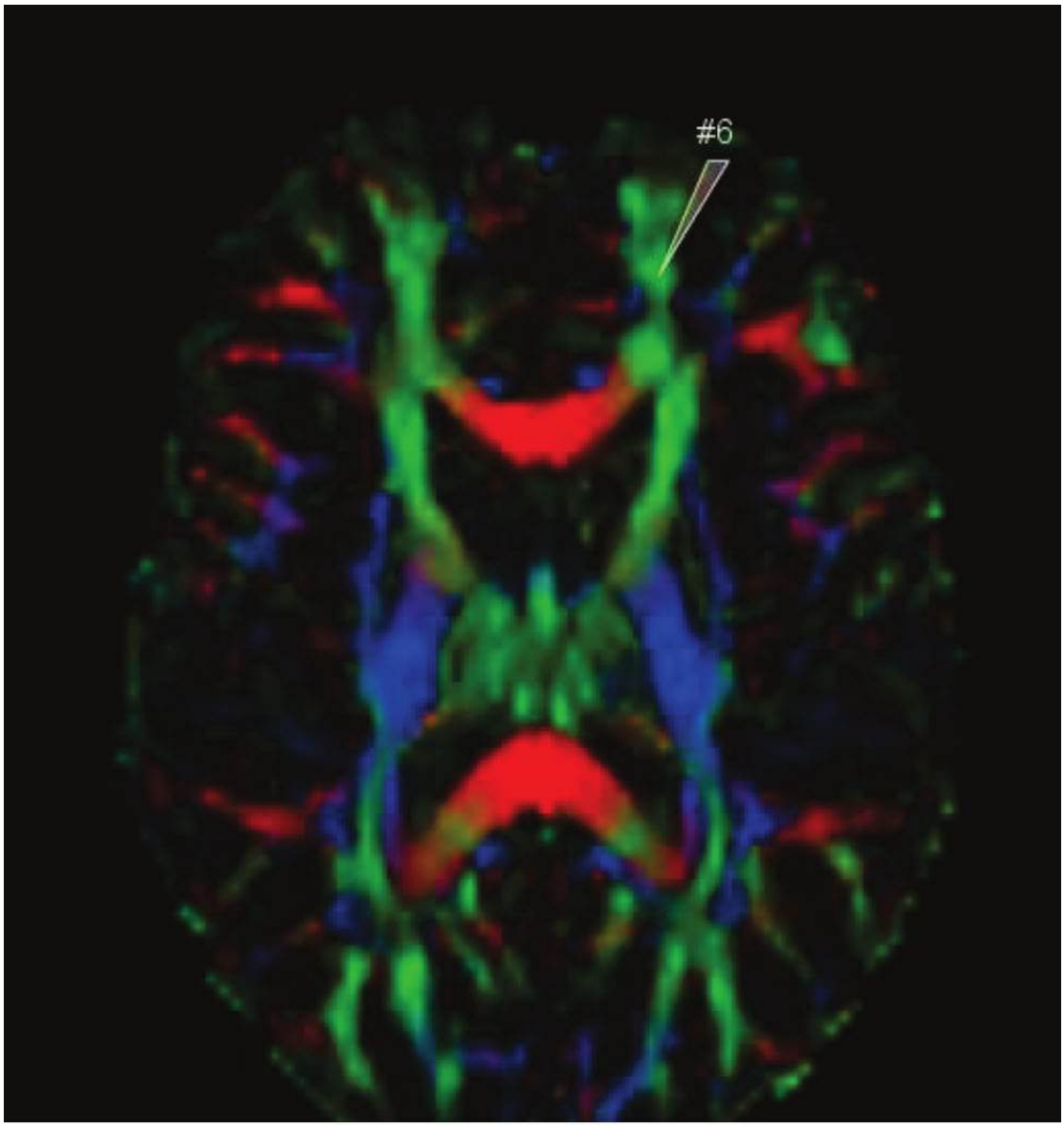


Figure 8.5. Three constructed diffusion tensors with the same mean diffusivity and different fractional anisotropy. Each glyph is drawn from three principal diffusivities, the amount of water movement along three perpendicular axes; an elongated glyph means strongly directional movement (high FA) and a round glyph means movement that is similar in every direction (low FA). The examples are mathematical illustrations with arbitrary units, not patient measurements, and show why the two numbers describe different properties of the same tissue. Original illustration for this edition.

Two cautions follow from how the numbers are made. FA is often called a measure of “white matter integrity,” a shorthand that conceals ambiguity: a low FA can mean injured fibers, but it can also mean two healthy bundles crossing within the same voxel, or a voxel that straddles the edge of a fluid space [16]. And the names axial and radial refer to the mathematical axes of the tensor, which coincide with the anatomical direction of the fibers only when a single bundle occupies the voxel. A careful report states which metric changed, in which structure, by how much, and relative to what comparison; it does not translate a number into a count of damaged fibers [16].

The colors: a code for direction

The most recognizable DTI picture is the color map (Figure 8.6), and the most common misreading of it is to take the colors as a measure of health or injury. They are not. In the standard convention, each voxel is colored by the main direction in which water moves, which is the direction in which the fibers run: red for fibers running left to right, green for fibers running front to back, and blue for fibers running up and down, with the brightness of the color scaled by the FA [17]. The large red band in the center of an axial map is the corpus callosum, whose fibers cross from one hemisphere to the other. The green bundles running front to back on either side are the long association tracts that connect the front of the brain to the back. The blue regions are the projection fibers descending from the cortex toward the brainstem and spinal cord. The map is an anatomical guide and a check that the data are sound, since the expected colors should appear in the expected places. It does not show where the injury is, and a map with vivid colors is not a map of damage.



Red: fibers running left to right
Green: fibers running front to back
Blue: fibers running up and down

Figure 8.6. A diffusion tensor color map (axial view) from the patient of Figure 8.2, with the standard color code added below. Each point is colored by the direction in which water moves most freely, which is the direction in which the nerve fibers run: red for left to right (the large red band is the corpus callosum, the bridge between the hemispheres), green for front to back (the long association tracts), blue for up and down (the fibers descending toward the brainstem). The arrow labeled 6 was placed by the interpreting neuroradiologist to mark the left superior longitudinal fasciculus, one of the tracts discussed later in the chapter. The colors encode direction, not injury. Identifying information removed.

Tractography: drawing the cables

Tractography is the computer reconstruction of the brain's fiber bundles from DTI data (Figure 8.7). Starting from a chosen point, the program follows the main direction of water movement from voxel to voxel. It traces a line (a streamline) until the direction becomes uncertain or a stopping rule is met, then repeats the process thousands of times to produce the familiar three-dimensional pictures of colored bundles [17]. The results are striking and have made the anatomy of the brain's connections visible in living people for the first time. Each line is a mathematical path through the data, not a photograph of a nerve fiber, and the result depends on the method, the starting points, and the rules chosen. Lines may stop where fibers cross or where the signal is poor, rather than where fibers end. In a large international test with simulated data of known anatomy, every method produced some connections that did not exist [18]. A thin or missing bundle in a tractography picture is therefore not proof of a missing tract; the quantitative analysis of a named tract rests on the measurements made within it, not on how the picture looks.

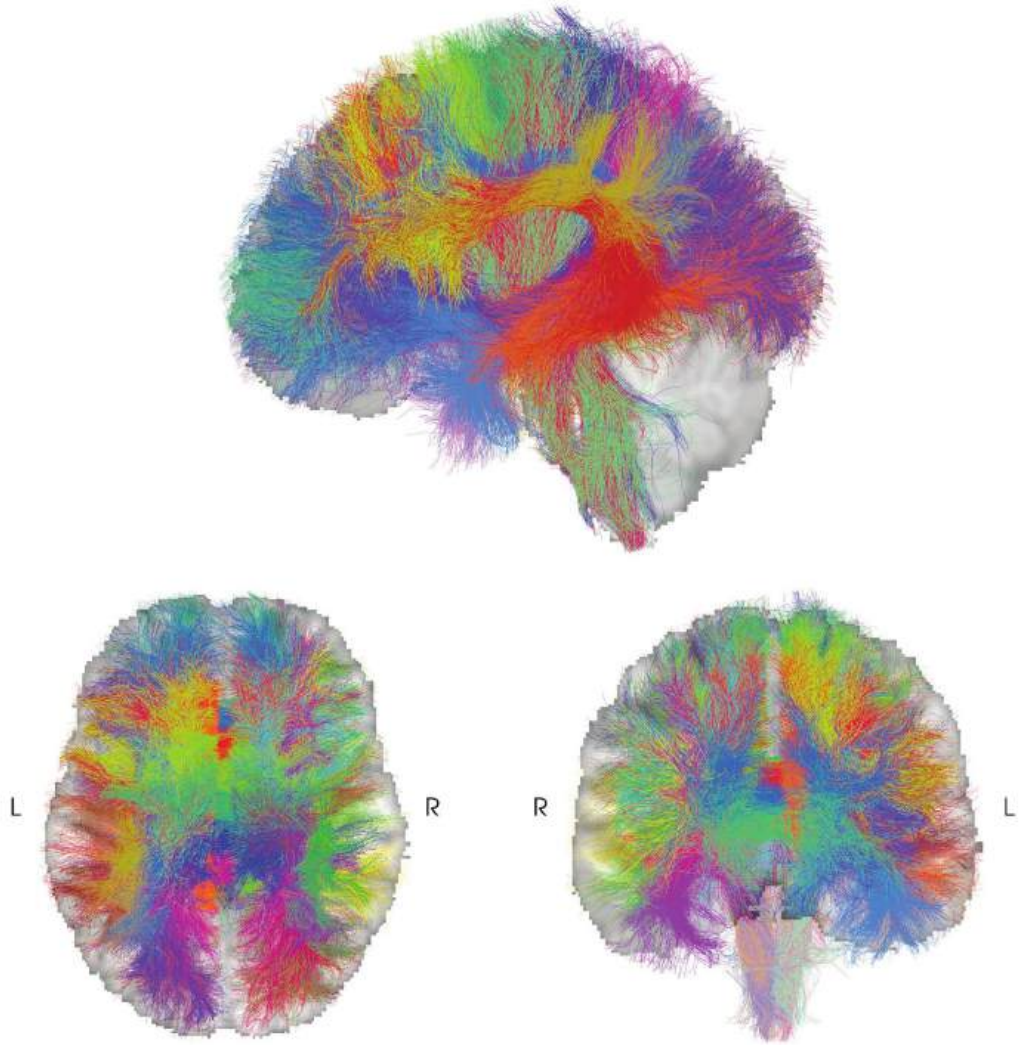


Figure 8.7. Whole-brain tractography from the same patient. Top: viewed from the left side, with the front of the brain to the left. Bottom left: viewed from above, with the front at the top (L and R mark the patient's left and right). Bottom right: viewed from the front. The computer has traced the paths along which water moves most freely, which follow the nerve fiber bundles, and colored each path by its direction using the code of Figure 8.6. Pictures of this kind are reconstructions from the measurements, not photographs of fibers; they display anatomy, and the detection of injury rests on the quantitative comparison described in the text. Identifying information removed.

What the research shows in mild traumatic brain injury

The DTI literature on brain injury is large and consistent in its main finding. A review of the first hundred studies found that 96 of them reported reduced FA in brain-injured patients, most often in the corpus callosum, the internal capsule, the cingulum, and the frontal white matter, the same regions that pathologists had identified decades earlier as the sites of axonal injury [15]. A meta-analysis of thirteen studies confined to mild

injury found significantly lower FA and higher MD in the corpus callosum, with the effect concentrated in its posterior part, the splenium, which the authors concluded is more vulnerable to mild injury than the front [19]. A comprehensive review of MRI and DTI in mild injury reached the same conclusion: group differences are reproducible, and DTI detects abnormalities in patients whose conventional scans are normal [20]. These group studies compare patients with controls either within named tracts or across the whole white matter at once, using methods such as tract-based spatial statistics that align every brain to a common skeleton of the major tracts [21]. In an early TRACK-TBI analysis, diffusion measures obtained within two weeks of a mild injury, by both approaches, predicted outcome at three and six months [22]. In the large American TRACK-TBI study, 391 patients with mild injury had lower FA and higher diffusivity than 148 controls [23]; in its European counterpart, CENTER-TBI, adding DTI to clinical information and blood biomarkers improved the prediction of incomplete recovery at three months in patients with normal CT scans, raising the accuracy of the model from 0.69 to 0.82 on a scale where 0.5 is chance and 1.0 is perfect [24]. A 2021 study developed a method of comparing an individual patient's tracts with a reference population and found abnormalities in individual patients with normal conventional MRI [13].

The literature also shows why timing and context matter. In the first days after injury, FA may rise rather than fall, apparently because swelling of the cells restricts water movement across the fibers. Ten adolescents scanned within a week of a concussion showed that pattern [25]. Diffusion abnormalities and symptoms do not necessarily resolve together; in college athletes followed through recovery, diffusion changes persisted after symptoms had resolved [26]. The relationship between a metric and outcome is not a simple "more abnormal means worse": in TRACK-TBI, higher axial diffusivity in the weeks after injury predicted better recovery [23]. With few exceptions, these are group findings. A difference between brain-injured and healthy groups does not establish that a particular patient's value is abnormal. Newer work links acute blood biomarkers with later diffusion abnormalities [27] and estimates a "brain age" from diffusion data that is modestly associated with outcome after injury [28]. These promising research tools are not yet established tests.

The professional bodies' position reflects both halves of this picture. The Radiological Society of North America's statement on brain injury imaging, updated in 2025, describes DTI and the other advanced techniques as investigational for the diagnosis and prognosis of individual patients, while acknowledging their value in research [2]; the American College of Radiology's reviews take the same view [29]. That position is compatible with the use of DTI as corroborative evidence within a converging clinical

picture, which is how Dr. Pappolla uses it and how the worked example below presents it. It is not compatible with a DTI report standing alone as proof of brain injury, and it is equally incompatible with a normal conventional MRI standing alone as proof that there is none.

How a report compares one patient with healthy people

A clinical DTI report answers the question the research cannot: is this patient's value unusual? It does so by comparing the patient's measurements, tract by tract, with a database of measurements from healthy people of similar age, and expressing the result as a percentile (Figure 8.8). A value at the 5th percentile means that 95 of 100 healthy people of the same age have a higher value; a value at the 50th percentile is average. The commercial platforms that perform this comparison divide the white matter into a few dozen named tracts, measure FA, MD, axial diffusivity, radial diffusivity, and volume in each, and flag the values that fall outside a chosen range, usually below the 5th or above the 95th percentile. FDA establishment registration and device listing do not themselves establish that a product has been cleared or approved, or that its safety and performance have been reviewed. Any regulatory authorization and its intended use must be verified separately; registration alone cannot validate a diagnostic inference from the output [36].

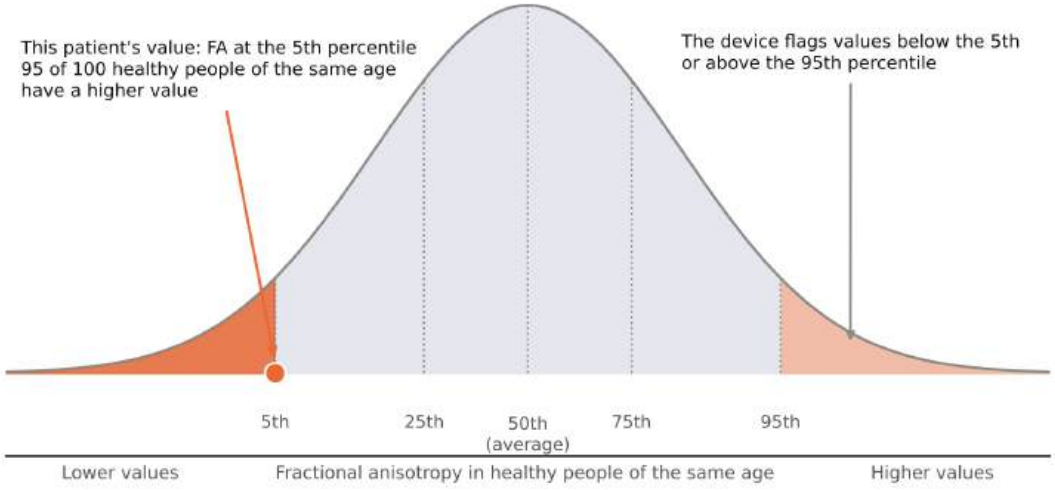


Figure 8.8. What a percentile means on a diffusion tensor report. The curve is the distribution of fractional anisotropy values in a particular tract among healthy people of the same age as the patient. A value at the 5th percentile, where the dot sits, is lower than the values of 95 of 100 healthy people. The platforms that produce these reports usually flag values below the 5th or above the 95th percentile, the shaded tails; values between the 5th and 20th percentiles are low but within the range the platform treats as normal, and a report that lists them as abnormal is applying a looser standard than the device does. Original schematic.

Several questions have to be asked of any such comparison, whichever party relies on it. Who were the healthy people? Diffusion values change throughout life, continuing to mature into the thirties and declining with age, so an adolescent cannot be compared with adults, and the database has to span the patient's age [30]. Were they scanned the same way? Different scanners, magnets, and sequence settings shift the values even when the tissue is identical, and statistical methods exist to harmonize data across sites, but a database collected on other scanners has to be shown to be comparable with the patient's scan [31]. Was the scan of good quality? Head movement during the scan systematically lowers FA and can produce apparent abnormalities in people who move more, and symptomatic patients move more than healthy volunteers [32]; the corrections for movement and distortion that modern processing applies reduce the problem but do not eliminate it, and a report should state whether the acquisition met quality standards [33]. How many comparisons were made? If a report examines thirty tracts and five metrics in each, it has made 150 comparisons, and some will fall below the 5th percentile by chance in a perfectly healthy person; the expected number of values below the 5th percentile in 31 tracts is between one and two, and below the 20th percentile about six. A report that counts every low value as a lesion turns normal

variation into an apparent burden of disease; the reading that survives scrutiny looks for a pattern rather than a tally.

A worked example: reading one patient's DTI report

The patient and the study

The patient is a young woman injured in a motor vehicle collision in her mid-teens and evaluated in Dr. Pappolla's outpatient neurology clinic. The images and values below come from her records, with identifying information removed. Her conventional CT and MRI were normal. Over the following years she had persistent difficulty with reading, studying, and concentration; intolerance of light, noise, and visual motion; dizziness in busy environments; fatigue; and, later, changes in mood. The objective testing described in Chapter 7 showed slowed reaction time with preserved accuracy, delayed visual and auditory evoked potentials, and abnormal eye tracking. Several findings were documented at more than one examination over two years. A little over two years after the injury she had an MRI with DTI on a 3-tesla scanner, with diffusion measured in 30 directions. A neuroradiologist analyzed the data using a commercial normative-comparison platform. Thirty-one of the platform's 33 named tracts met its quality standard for analysis; two did not and were excluded, which the report stated.

What the platform flagged and what the radiologist reported

The platform's own summary stated that all 31 analyzable tracts fell between the 5th and 95th percentiles for the microstructural measures (FA, MD, axial diffusivity, radial diffusivity), and that one tract was outside the normal range for volume: the splenium of the corpus callosum, at the 4th percentile. Applying a broader standard, the interpreting neuroradiologist listed six tracts whose FA fell between the 5th and 18th percentiles as outside the normal range: the cingulum on the left (5th percentile) and right (17th), the fornix on the left (10th), the inferior fronto-occipital fasciculus on the left (18th) and right (13th), and the third division of the superior longitudinal fasciculus on the left (16th). He described the pattern as highly suspicious for traumatic brain injury. He also listed six structures with volume at or below roughly the 10th percentile: the isthmus (10th) and splenium (4th) of the corpus callosum, the cingulum on both sides, the right inferior longitudinal fasciculus, and the right optic radiation. The report noted that there was no abnormality on the conventional images at any of these sites.

The first thing to notice is that the device and the radiologist applied different thresholds. By the device's standard, one value was abnormal; by the radiologist's, twelve were. Neither is wrong, but they are different claims, and a report should say

which standard it is applying. The arithmetic of the previous section applies with full force: among 31 tracts, about six would be expected to fall below the 20th percentile for any one metric by chance alone, so the count of low values is not evidence of anything. What has to be examined is the pattern.

The tracts, and what they do

Figure 8.9 shows how the platform presents a single tract, using the left cingulum, the most abnormal in this patient. The upper row shows the tract as reconstructed in her brain from three directions; the lower row shows her values for each measure as a dot against the shaded band of values from healthy people of her age. Her FA sits at the bottom edge of the band (5th percentile) and her volume in its lower part (9th percentile).

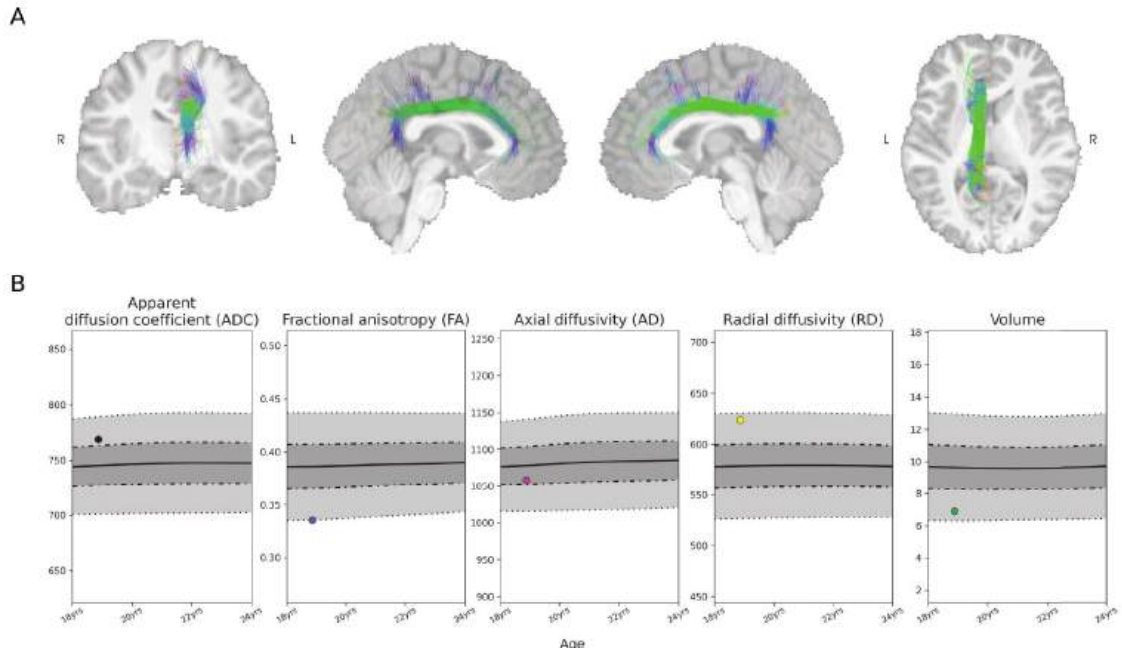


Figure 8.9. One tract as the analysis platform presents it: the left cingulum in the patient of Figure 8.2. (A) The tract reconstructed by tractography and displayed from the front, the left side, the right side, and above. (B) The patient's values (dots) for the apparent diffusion coefficient, fractional anisotropy, axial diffusivity, radial diffusivity, and volume, plotted against the range of values in healthy people of her age (the shaded bands, with the central line the average). Her fractional anisotropy lies at the 5th percentile, the lower edge of the normal range. Identifying information removed.

The six tracts the radiologist listed are shown together in Figure 8.10, and their anatomy is the heart of the matter, because they are not a random six. The cingulum is a bundle that runs front to back along the inner surface of each hemisphere, just above the corpus callosum, and connects the frontal lobe with the structures of memory and

emotion in the temporal lobe; it is a core component of the brain's attention and memory systems and of the circuits that regulate mood, and it is among the tracts most consistently found abnormal after brain injury [15]. The fornix is the main output cable of the hippocampus, the structure that forms new memories. The inferior fronto-occipital fasciculus runs the length of each hemisphere from the visual areas at the back to the frontal lobe, carrying visual information forward for interpretation and decision. The third division of the superior longitudinal fasciculus connects the parietal lobe with the frontal lobe and is involved in attention and, on the left, in the processing of language. In this patient, the cingulum was low on both sides and the inferior fronto-occipital fasciculus was low on both sides. Paired structures affected on both sides, in a patient with symptoms in exactly the functions those structures serve, is a pattern. Six scattered low values in unrelated tracts would not be.

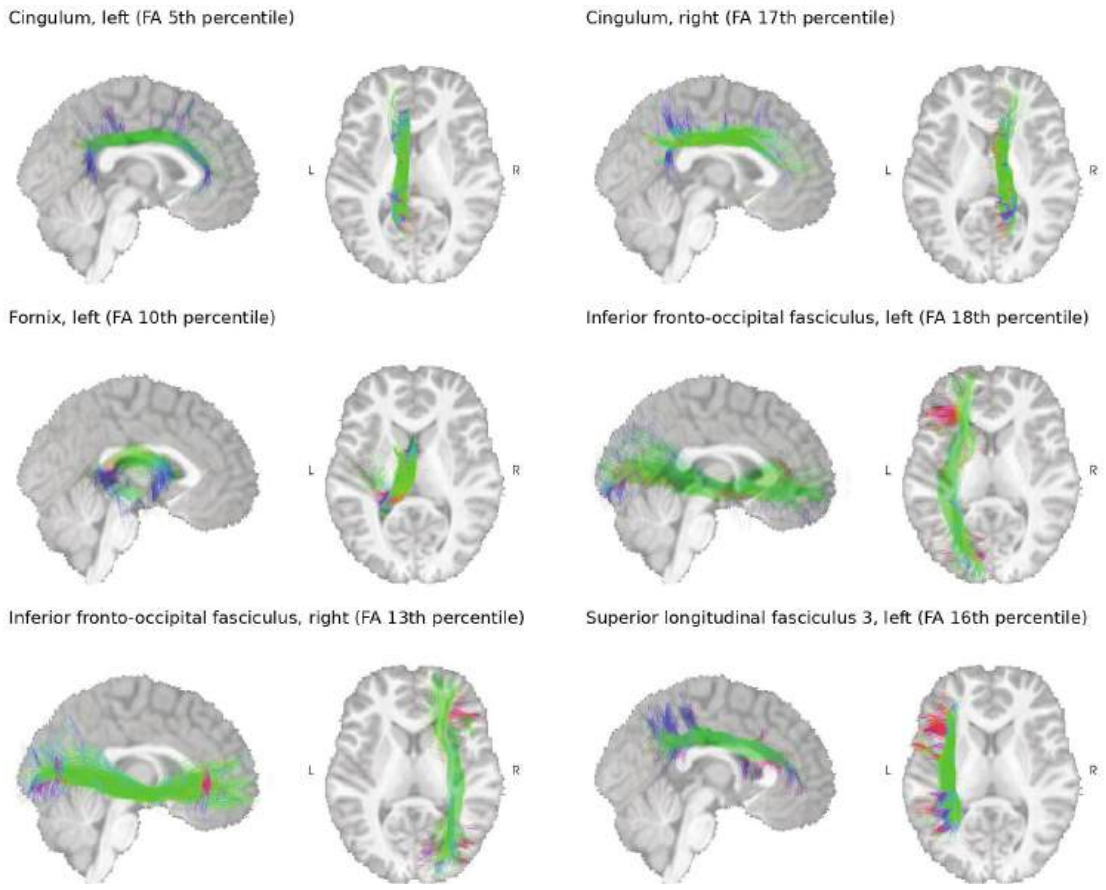


Figure 8.10. The six tracts the interpreting neuroradiologist listed as below the normal range for fractional anisotropy, each shown from the left side and from above, as reconstructed in the patient's brain. The cingulum (upper row) and the inferior fronto-occipital fasciculus (middle row, right, and lower row, left) were low on both sides. The cingulum connects the frontal lobe with the memory and emotion structures of the temporal lobe; the fornix is the output cable of the hippocampus, the memory structure; the inferior fronto-occipital fasciculus carries visual information from the back of the brain to the front; the superior longitudinal fasciculus connects the parietal and frontal lobes and serves attention and, on the left, language. Percentiles as reported. Identifying information removed.

Low measured volume in the corpus callosum

The one finding the platform itself flagged deserves separate attention (Figure 8.11). The corpus callosum is the thick bridge of some 200 million fibers that connects the two hemispheres, and its long fibers are vulnerable to traumatic stretching, particularly during rotational acceleration. Its rear portion, the splenium, connects the visual and parietal regions of the two sides. In the meta-analysis of mild injury cited above, the splenium was the part of the corpus callosum most clearly affected [19]. In this patient, two years after injury, the volume of the splenium was at the 4th percentile and the

volume of the adjacent isthmus at the 10th, while the FA of both was within the normal range. Volume estimates the size of the measured structure, which is a different question from the diffusion measures. These percentiles show that the splenium and isthmus were small relative to the reference group at that examination. They do not establish how much tissue, if any, was lost after the injury: a percentile is not a percentage of tissue loss, and a single scan does not measure change from the patient’s own earlier volume. Demonstrating acquired loss would require an appropriate longitudinal comparison or other evidence of change. The low splenial volume remains a finding to weigh alongside the history, clinical course, and other measurements, even though no corresponding lesion was apparent on the conventional images.

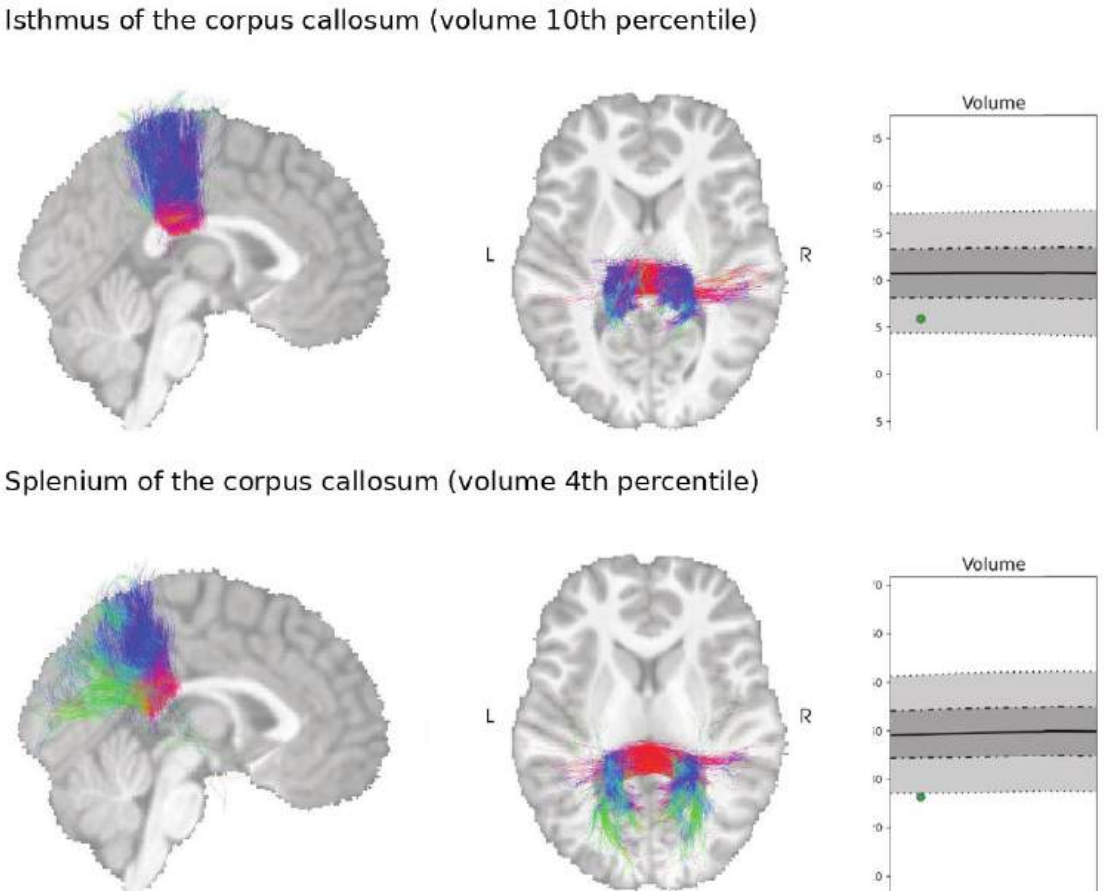


Figure 8.11. Low measured volume in the rear part of the corpus callosum, the bridge of fibers connecting the two hemispheres. Upper row: the isthmus, reconstructed from the left side and from above, with the patient's volume (green dot) plotted against the range in healthy people of her age (volume at the 10th percentile). Lower row: the splenium, the rearmost part, which connects the visual and parietal regions of the two sides; volume at the 4th percentile, the one value the analysis platform itself flagged as outside the normal range. The splenium is the part of the corpus callosum most consistently affected in mild traumatic brain injury in the published meta-analysis. The percentiles compare this examination with a reference group; they do not document a decrease from this patient's earlier volume. Identifying information removed.

Putting the DTI beside the other evidence

A DTI report in this book is never read alone, and Figure 8.12 sets this patient's DTI findings beside what the other tests and the patient herself showed. The tracts of memory and attention, the cingulum on both sides and the left fornix, were low; her reaction time on computerized testing, nearly four years after the injury, was slower than 98 of 100 healthy women of her age with accuracy fully preserved, and studying and reading exhausted her. The tracts carrying visual information forward, the inferior fronto-occipital fasciculus on both sides, were low, as was the volume of the splenium

that connects the visual regions of the two hemispheres; her visual evoked potential was delayed at two examinations two years apart, her eyes could not keep up with moving stripes or follow a moving target smoothly on eye tracking, and she could not tolerate light or visual motion. The left superior longitudinal fasciculus, serving attention and language, was low; her auditory evoked potential was delayed and worsened between examinations, she needed extra effort to follow speech, and she could not tolerate noise. None of these correspondences is exact, because no tract serves one function and no symptom arises from one tract. Together they are what the convergence principle of this book describes: independent measurements, made by different methods, several of them beyond the patient's voluntary control, pointing in the same direction.

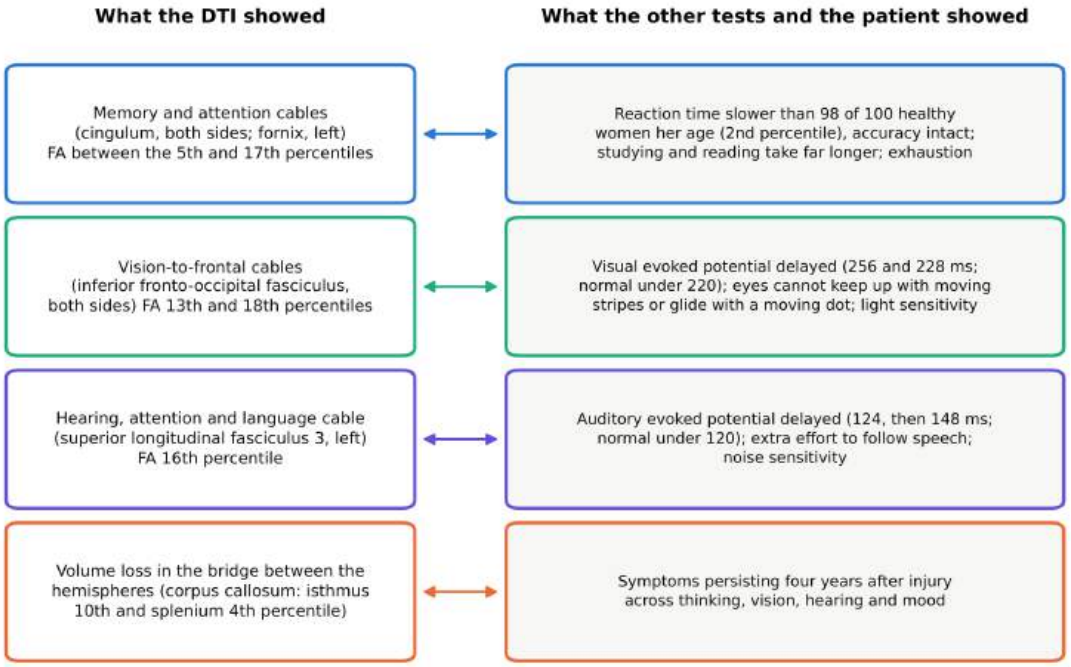


Figure 8.12. Convergence between the diffusion tensor findings and the rest of the evidence in the same patient. Left: the tracts and structures the report identified as low, grouped by the functions they serve. Right: the corresponding findings on objective testing performed between two and four years after the injury, and the patient's corresponding complaints. The correspondence is approximate, because tracts and symptoms do not map one to one; its force comes from several independent methods pointing the same way. The phrase "Volume loss" in the lower-left panel refers here to low measured volume relative to the reference group, not a demonstrated decrease since injury. Original schematic drawn from the patient's records; identifying information removed.

What this report can and cannot say

Read with discipline, the report supports the following statements. The patient's white matter shows a pattern of low FA in paired tracts serving memory, attention, and vision, with low measured volume in the posterior corpus callosum, in the absence of any visible lesion on conventional imaging and in the absence of any other disease. The pattern corresponds to the tracts most consistently affected in brain injury and to the functions in which this patient has objectively measured deficits. The findings are consistent with traumatic axonal injury and add corroborating weight to a diagnosis that rests on the history, the clinical course, and the objective physiological testing. The volume percentiles describe a cross-sectional difference from the reference group, not a measured change since the collision.

The report does not support the following. It does not date the injury: DTI cannot tell when a tract became abnormal, and the attribution to the collision rests on the timeline of symptoms and the absence of other causes, not on the scan. It does not, by itself, prove the injury: the values the radiologist listed lie within the range the device treats as normal, and the chance arithmetic is what it is. It does not predict the future: the relationship between DTI values and recovery is real at the group level and unvalidated for the individual. And it does not assign each symptom to a tract. The report's proper role is the one the professional statements allow and the convergence principle requires: corroboration within a coherent clinical picture.

The limits of DTI and the objections raised to it

Most objections raised to DTI in contested cases concern real limitations. Their significance depends on how the particular study was performed: an objection that is decisive for one report may be immaterial to another. The following sections explain each limitation and the questions needed to assess its weight.

Crossing fibers. Much of the white matter contains fibers running in more than one direction within a single voxel, and two healthy crossing bundles produce a lower FA than one, so a low FA in such a region may not mean injury [16]. The limitation is real and is one reason the analysis of named tracts, whose anatomy is known, is more interpretable than a search of the whole brain for low values; it is also addressed by newer models that resolve more than one fiber direction. It does not explain a bilateral reduction in a tract compared with the same tract in healthy people scanned and analyzed the same way, since the crossing fibers are present in the healthy people too.

Partial volume and free water. A voxel at the edge of a ventricle contains both tissue and fluid, and the fluid lowers FA and raises MD; swelling has a similar effect. Methods

that separate the “free water” component from the tissue signal improve the measurement [34]. A report should state how the tracts were defined and whether fluid contamination was addressed.

Motion. Symptomatic patients move more than healthy volunteers, and motion lowers FA [32]. The answer is quality control: inspected raw data, a reported degree of motion, and the corrections applied [33]. A report that does not document quality has left a central question unanswered.

The reference database. The healthy comparison group must match the patient’s age and must have been scanned in a comparable way. Harmonization across scanners must be demonstrated rather than assumed [30,31]. Without an appropriate reference database, the meaning of a low percentile is unclear.

Group versus individual. Almost all of the research is group research, and the professional bodies describe individual diagnosis by DTI as investigational [2,29]. This is the strongest argument and it has a precise answer: DTI in an individual patient is corroborative, not diagnostic; it is one keyhole among several; and its weight comes from convergence with independent evidence, not from standing alone.

Multiple comparisons and the tally of low values. A report that counts every value below a threshold invites the response that such values are expected by chance, and the response is correct. The protection is the pattern: anatomically paired structures, tracts known to be vulnerable to the injury, correspondence with measured deficits, and the values the device itself flags.

Tractography is not anatomy. The colored bundles are reconstructions and can show paths that do not exist or omit paths that do [18]. The quantitative comparison does not depend on the appearance of the picture, and an argument from how thin or thick a bundle looks, in either direction, misreads the method.

“Not FDA approved.” The platform in the example above is registered with the agency as a medical device, and the MRI scanner and sequences are cleared for clinical use. What the agency has not done is validate any diagnostic conclusion. The regulatory status of software is not the question; the scientific validity of the inference is.

A normal conventional MRI. The purpose of DTI is to measure what conventional MRI cannot see, so a normal conventional scan in a patient with abnormal DTI is the expected finding in axonal injury, not a contradiction [13,20]. The normal scan still matters: it excludes the bleeding, bruising, scarring, and other disease that would change the interpretation.

Reading an imaging report in a contested case

The following questions can help a reader without medical training examine a conventional or advanced imaging report, whether it is offered by one side or the other in a contested case.

First, identify what kind of claim the report makes: a lesion that was seen, or a measurement that was unusual. The first can be checked by looking at the images; the second has to be checked by asking about the method and the comparison.

Second, establish the timing: how long after the injury was the study obtained, and what was the clinical state at that moment? A scan on the day of injury and a scan two years later answer different questions. The research used to interpret a finding has to match that interval.

Third, read the whole report, including the technical sections. Which sequences were obtained? Were the images of adequate quality? For DTI, how many directions were measured, what was the magnet strength, and which reference database was used? How many tracts were analyzed or excluded, and what threshold defined an abnormal value? Was the device's threshold the same as the reader's?

Fourth, look for the pattern rather than the count. Are the abnormal structures anatomically related? Are paired structures affected on both sides? Are they the structures known to be vulnerable to this injury? Do they serve the functions in which the patient has deficits?

Fifth, set the imaging beside the independent evidence: the history, the examination, the objective physiological tests of Chapter 7, and the cognitive findings. Imaging that converges with these carries weight. Imaging that stands alone, in either direction, carries little.

Sixth, insist that the report's conclusions match its findings. A DTI report that claims to prove, date, or quantify an injury has overreached; a conventional report that is read as excluding one has been misread. The multidisciplinary consensus on the ethical use of brain images in testimony asks for exactly this discipline: qualified interpretation, transparent methods, and a plain account of what the image can and cannot establish [35]. A vivid picture is not a substitute for a valid inference.

Other advanced techniques

Functional MRI maps changes in blood oxygenation as an indirect sign of brain activity during a task or at rest; magnetic resonance spectroscopy measures the concentration

of a few chemicals in a region of brain; perfusion imaging measures blood flow; and quantitative electroencephalography and magnetoencephalography, discussed in Chapter 7, measure electrical and magnetic activity directly. Each has shown group differences after brain injury, and each depends on acquisition, analysis, and factors other than trauma. The American College of Radiology's review of the advanced techniques reached the same conclusion for all of them: promising in research, not yet validated for routine individual diagnosis [29]. A report that treats any of them as a confirmation test should be read with the same questions as a DTI report.

Fast take: main points from chapter 8

• CT is the first study in acute head injury and detects bleeding, fracture, swelling, and shift; it is normal in most concussions, and that is the expected result, not evidence against injury [1,2].

• Conventional MRI is more sensitive than CT to small bruises and old blood, and about a quarter of mild injuries with normal CT show abnormalities on early MRI; most show none, because the injury of a concussion is at a scale a thousand times smaller than the picture [5,6].

• Ventricular enlargement after injury usually means lost tissue; distinguishing it from a fluid disorder requires earlier scans and the clinical course [10].

• Diffusion tensor imaging measures the organization of the brain's wiring through the behavior of water; fractional anisotropy falls when fibers are disrupted, and the colors of the maps encode direction, not injury [12,17].

• Nearly every study of brain injury has found reduced FA, most consistently in the corpus callosum, cingulum, and frontal white matter; in mild injury the splenium is the most affected part of the corpus callosum [15,19].

• Clinical reports express a patient's values as percentiles relative to those of healthy people; the questions to ask are who the healthy people were, how they were scanned, whether the scan was of good quality, how many comparisons were made, and what threshold the device and the reader each applied [30,31,32].

• About six of 31 tracts will fall below the 20th percentile by chance; the evidence lies in the pattern, bilateral involvement of related tracts known to be vulnerable, with correspondence to measured deficits, not in the count.

• In the worked example, paired low values in the tracts serving memory, attention, and vision, with low measured volume in the splenium, corresponded to slowed reaction

time, delayed visual and auditory evoked potentials, and abnormal eye tracking in the same patient; that convergence, not the DTI alone, is the evidence. The scan's normative percentiles do not themselves demonstrate loss since injury.

• A DTI report can corroborate a diagnosis within a converging picture; it cannot date, prove, or quantify an injury by itself, and professional bodies describe its individual use as investigational [2,29].

• Imaging presented as evidence, by either side, should be interpreted by qualified readers with transparent methods and a plain statement of its limits [35].

References

1. Expert Panel on Neurological Imaging; Shih RY, Burns J, Ajam AA, et al. ACR Appropriateness Criteria Head Trauma: 2021 update. J Am Coll Radiol. 2021;18(5S):S13-S36. doi:10.1016/j.jacr.2021.01.006
2. Radiological Society of North America. RSNA statement on traumatic brain injury (TBI) imaging. Updated June 24, 2025. Available at: https://www.rsna.org/-/media/Files/RSNA/Media/Traumatic-Brain-Injury-TBI-Imaging. Accessed October 2026.
3. Wintermark M, Sanelli PC, Anzai Y, Tsiouris AJ, Whitlow CT; ACR Head Injury Institute. Imaging evidence and recommendations for traumatic brain injury: conventional neuroimaging techniques. J Am Coll Radiol. 2015;12(2):e1-e14. doi:10.1016/j.jacr.2014.10.014
4. Marshall LF, Marshall SB, Klauber MR, et al. A new classification of head injury based on computerized tomography. J Neurosurg. 1991;75(Suppl):S14-S20. doi:10.3171/sup.1991.75.1s.0s14
5. Yuh EL, Mukherjee P, Lingsma HF, et al. Magnetic resonance imaging improves 3-month outcome prediction in mild traumatic brain injury. Ann Neurol. 2013;73(2):224-235. doi:10.1002/ana.23783
6. Tong KA, Ashwal S, Holshouser BA, et al. Hemorrhagic shearing lesions in children and adolescents with posttraumatic diffuse axonal injury: improved detection and initial results. Radiology. 2003;227(2):332-339. doi:10.1148/radiol.2272020176
7. Liu AY, Maldjian JA, Bagley LJ, Sinson GP, Grossman RI. Traumatic brain injury: diffusion-weighted MR imaging findings. AJNR Am J Neuroradiol. 1999;20(9):1636-1641.
8. QSM Consensus Organization Committee; Bilgic B, Costagli M, Chan KS, et al. Recommended implementation of quantitative susceptibility mapping for clinical research in the brain: a consensus of the ISMRM electro-magnetic tissue properties study group. Magn Reson Med. 2024;91(5):1834-1862. doi:10.1002/mrm.30006

9. US Food and Drug Administration. MRI (magnetic resonance imaging): benefits and risks. Available at: https://www.fda.gov/radiation-emitting-products/mri-magnetic-resonance-imaging/benefits-and-risks. Accessed October 2026.
10. Marmarou A, Foda MA, Bandoh K, et al. Posttraumatic ventriculomegaly: hydrocephalus or atrophy? A new approach for diagnosis using CSF dynamics. J Neurosurg. 1996;85(6):1026-1035. doi:10.3171/jns.1996.85.6.1026
11. Iaccarino C, Kolias A, Adelson PD, et al. Consensus statement from the international consensus meeting on post-traumatic cranioplasty. Acta Neurochir (Wien). 2021;163(2):423-440. doi:10.1007/s00701-020-04663-5
12. Basser PJ, Mattiello J, LeBihan D. MR diffusion tensor spectroscopy and imaging. Biophys J. 1994;66(1):259-267. doi:10.1016/S0006-3495(94)80775-1
13. Jolly AE, Bălăeţ M, Azor A, et al. Detecting axonal injury in individual patients after traumatic brain injury. Brain. 2021;144(1):92-113. doi:10.1093/brain/awaa372
14. Pierpaoli C, Basser PJ. Toward a quantitative assessment of diffusion anisotropy. Magn Reson Med. 1996;36(6):893-906. doi:10.1002/mrm.1910360612
15. Hulkower MB, Poliak DB, Rosenbaum SB, Zimmerman ME, Lipton ML. A decade of DTI in traumatic brain injury: 10 years and 100 articles later. AJNR Am J Neuroradiol. 2013;34(11):2064-2074. doi:10.3174/ajnr.A3395
16. Jones DK, Knösche TR, Turner R. White matter integrity, fiber count, and other fallacies: the do's and don'ts of diffusion MRI. Neuroimage. 2013;73:239-254. doi:10.1016/j.neuroimage.2012.06.081
17. Catani M, Howard RJ, Pajevic S, Jones DK. Virtual in vivo interactive dissection of white matter fasciculi in the human brain. Neuroimage. 2002;17(1):77-94. doi:10.1006/nimg.2002.1136
18. Maier-Hein KH, Neher PF, Houde JC, et al. The challenge of mapping the human connectome based on diffusion tractography. Nat Commun. 2017;8:1349. doi:10.1038/s41467-017-01285-x
19. Aoki Y, Inokuchi R, Gunshin M, Yahagi N, Suwa H. Diffusion tensor imaging studies of mild traumatic brain injury: a meta-analysis. J Neurol Neurosurg Psychiatry. 2012;83(9):870-876. doi:10.1136/jnnp-2012-302742
20. Shenton ME, Hamoda HM, Schneiderman JS, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. Brain Imaging Behav. 2012;6(2):137-192. doi:10.1007/s11682-012-9156-5
21. Smith SM, Jenkinson M, Johansen-Berg H, et al. Tract-based spatial statistics: voxelwise analysis of multi-subject diffusion data. Neuroimage. 2006;31(4):1487-1505. doi:10.1016/j.neuroimage.2006.02.024

22. Yuh EL, Cooper SR, Mukherjee P, et al. Diffusion tensor imaging for outcome prediction in mild traumatic brain injury: a TRACK-TBI study. J Neurotrauma. 2014;31(17):1457-1477. doi:10.1089/neu.2013.3171
23. Palacios EM, Yuh EL, Mac Donald CL, et al. Diffusion tensor imaging reveals elevated diffusivity of white matter microstructure that is independently associated with long-term outcome after mild traumatic brain injury: a TRACK-TBI study. J Neurotrauma. 2022;39(19-20):1318-1328. doi:10.1089/neu.2021.0408
24. Richter S, Winzeck S, Correia MM, et al. Predicting recovery in patients with mild traumatic brain injury and a normal CT using serum biomarkers and diffusion tensor imaging (CENTER-TBI): an observational cohort study. EClinicalMedicine. 2024;75:102751. doi:10.1016/j.eclinm.2024.102751
25. Wilde EA, McCauley SR, Hunter JV, et al. Diffusion tensor imaging of acute mild traumatic brain injury in adolescents. Neurology. 2008;70(12):948-955. doi:10.1212/01.wnl.0000305961.68029.54
26. Wu YC, Harezlak J, Elsaid NMH, et al. Longitudinal white-matter abnormalities in sports-related concussion: a diffusion MRI study. Neurology. 2020;95(7):e781-e792. doi:10.1212/WNL.0000000000009930
27. Mononen M, Mohammadian M, Hossain I, et al. Association of acute blood biomarkers with diffusion tensor imaging and outcome in patients with traumatic brain injury presenting with GCS of 13-15. Neuroimage Clin. 2026;49:103934. doi:10.1016/j.nicl.2025.103934
28. Rodrigues L, Parker D, Broomand Lomer N, et al. Brain age gap as a diffusion MRI-based marker of traumatic brain injury-related brain changes and associated outcomes. Brain Commun. 2026;8(4):fcag254. doi:10.1093/braincomms/fcag254
29. Wintermark M, Sanelli PC, Anzai Y, Tsiouris AJ, Whitlow CT; American College of Radiology Head Injury Institute. Imaging evidence and recommendations for traumatic brain injury: advanced neuro- and neurovascular imaging techniques. AJNR Am J Neuroradiol. 2015;36(2):E1-E11. doi:10.3174/ajnr.A4181
30. Lebel C, Beaulieu C. Longitudinal development of human brain wiring continues from childhood into adulthood. J Neurosci. 2011;31(30):10937-10947. doi:10.1523/JNEUROSCI.5302-10.2011
31. Fortin JP, Parker D, Tunç B, et al. Harmonization of multi-site diffusion tensor imaging data. Neuroimage. 2017;161:149-170. doi:10.1016/j.neuroimage.2017.08.047
32. Yendiki A, Koldewyn K, Kakunoori S, Kanwisher N, Fischl B. Spurious group differences due to head motion in a diffusion MRI study. Neuroimage. 2014;88:79-90. doi:10.1016/j.neuroimage.2013.11.027

33. Andersson JLR, Sotiropoulos SN. An integrated approach to correction for off-resonance effects and subject movement in diffusion MR imaging. Neuroimage. 2016;125:1063-1078. doi:10.1016/j.neuroimage.2015.10.019
34. Pasternak O, Sochen N, Gur Y, Intrator N, Assaf Y. Free water elimination and mapping from diffusion MRI. Magn Reson Med. 2009;62(3):717-730. doi:10.1002/mrm.22055
35. Meltzer CC, Sze G, Rommelfanger KS, Kinlaw K, Banja JD, Wolpe PR. Guidelines for the ethical use of neuroimages in medical testimony: report of a multidisciplinary consensus conference. AJNR Am J Neuroradiol. 2014;35(4):632-637. doi:10.3174/ajnr.A3711
36. US Food and Drug Administration. Important reminders about registration and listing. Accessed October 25, 2026. https://www.fda.gov/medical-devices/device-registration-and-listing/important-reminders-about-registration-and-listing

Chapter 9 The cognitive examination and neuropsychological testing

Chapter overview

Cognitive complaints bring more patients with a brain injury to a specialist than any other symptom, and the cognitive examination is the part of the evaluation that attorneys, insurers, and courts scrutinize most closely. This chapter explains what a cognitive examination is, from the bedside mental status examination through screening instruments and the office-based neurobehavioral status examination to the comprehensive neuropsychological battery, and what each level can and cannot do. It describes the six cognitive domains of the DSM-5 and their subdomains in plain language, with an everyday example of failure in each and the kind of test that measures it. Neurologists are trained to evaluate cognition and can perform cognitive and neuropsychological assessments within their competence. Clinical neuropsychologists bring greater depth in specialized psychometrics. Drawing on the authors' published position, the chapter explains why competence rather than professional title determines who can perform a particular assessment, and how both disciplines contribute to diagnosing neurocognitive disorders. Attribution to traumatic brain injury or neurodegenerative disease requires medical evaluation [1]. It defines mild cognitive impairment, mild and major neurocognitive disorder, and neurocognitive disorder due to traumatic brain injury, explains what these labels do and do not say about cause, states what a normal result means, and closes with the questions a careful reader should ask of a neuropsychological report.

What a cognitive examination is

A cognitive examination uses structured tasks to assess how a person's thinking works. The tasks have a known difficulty, and performance is compared with that of healthy people of the same age and usually the same education. The examination ranges from a few bedside questions to a day of standardized testing. These levels differ in what they measure, how precisely they measure it, and which questions they can answer. Keeping the levels distinct matters in clinical practice and litigation alike [1,2].

The bedside mental status examination

Every neurological examination includes a mental status examination. The neurologist notes whether the patient is alert and attentive, oriented to the date and the place, able to repeat a short series of numbers forward and backward, to learn three words and recall them a few minutes later, to name common objects, follow a multi-step command, copy a drawing, and explain a proverb, and watches whether the patient

loses the thread, searches for words, or is unaware of errors. It takes ten minutes, needs no equipment, and can be repeated at every visit. It has no formal scoring and no normative data; it detects obvious impairment and misses subtle impairment, particularly in people of high ability.

Screening instruments

Screening instruments bring scoring and cut-offs to the bedside. The Mini-Mental State Examination (MMSE) is a 30-point test of orientation, registration, attention, recall, language, and copying, published in 1975 and still the most widely used [3]. The Montreal Cognitive Assessment (MoCA) is also scored out of 30 but samples executive function, abstraction, and delayed recall more heavily, and was designed to detect milder impairment [4]. The Mini-Cog combines three-word recall with a clock drawing and takes three minutes [5].

The systematic review prepared for the United States Preventive Services Task Force found that brief instruments perform adequately for dementia and poorly for mild cognitive impairment, and that a brief score corresponds only loosely to how the person actually functions [6]. The National Academy of Neuropsychology has stated that screening tests help identify people who need comprehensive assessment and can track treatment, but should not substitute for it [7]. After mild traumatic brain injury the MMSE has a sensitivity in the range of 0.43 to 0.58, which means that it misses roughly half of the impaired patients [8].

Two properties limit all screening instruments, in both directions. A ceiling effect means that a person of high premorbid ability can lose a great deal and still score in the normal range, because the test was never hard enough to measure where they started. A floor effect means that a person with little schooling, a learning disability, limited literacy, or limited command of the language of testing can fail items for reasons that have nothing to do with brain disease [1,7]. Education, language, and culture shape every cognitive score, and the norms against which a score is read were collected in a particular population; a patient tested through an interpreter, or outside the language in which the norms were developed, is at risk of misclassification [1].

The neurobehavioral status examination

Neurologists assess cognition beyond the brief screening score. One established approach is the office-based examination that the American Academy of Neurology’s Behavioral Neurology Section Workgroup described in 2015 [9]. The workgroup surveyed 200 behavioral neurologists and identified five to fifteen standardized tests for each cognitive domain, selected for having normative data and validation in neurological

disorders, that fit an office visit [9]. The result, the neurobehavioral status examination, is a domain-by-domain examination with scored tests, more thorough than a screen and shorter than a battery. It suffices for most diagnostic and management questions in neurological practice and falls short when the question calls for psychometric methods it does not provide, for example when illiteracy or a sensory or motor deficit prevents the patient from performing the standard tasks [1,9].

Computerized cognitive testing

Computerized batteries present timed tasks on a screen and score reaction time, attention, memory, and executive function against age norms, with built-in validity checks. Chapter 7 describes them; they are a screening and tracking instrument, not a substitute for a neuropsychological evaluation.

The comprehensive neuropsychological battery

A comprehensive neuropsychological assessment is distinguished from everything above by its measurement model rather than by the number of tests [1,2]. The examiner selects standardized instruments across the cognitive domains and across mood, personality, and behavior, then administers and scores them under fixed rules. Each score is compared with norms chosen for the patient's age, education, sex, and, where possible, language and culture. The examiner estimates the person's ability before the injury or illness, studies strengths, weaknesses, and error patterns, and uses validity measures to assess whether performance can be interpreted. These findings are translated into an account of what the person can and cannot do and what would help [7,10]. Clinical neuropsychologists bring extensive specialized training to this process. Neurologists can also perform neuropsychological assessments within their training and competence; describing an assessment as comprehensive carries the same methodological requirements whoever performs it [1]. A full battery takes several hours, sometimes spread over two days, and produces a report of ten to thirty pages. Table 9.1 compares the three levels by method and purpose without assigning ownership of cognitive evaluation to any profession [1].

Table 9.1. Three levels of cognitive evaluation

Feature	Cognitive screening	Integrated cognitive assessment	Comprehensive neuropsychological assessment

Feature	Cognitive screening	Integrated cognitive assessment	Comprehensive neuropsychological assessment
Primary purpose	Detect possible impairment and decide whether more evaluation is needed	Answer a diagnostic or management question by placing cognition within the medical and neurological context	Characterize cognitive, behavioral, emotional, and functional status in psychometric detail
Typical methods	MMSE, MoCA, Mini-Cog, or a single-domain screen	History, informant account, neurological examination, functional review, targeted standardized tests, and the relevant medical data	Standardized multi-domain batteries, norm-referenced interpretation, validity measures, error-pattern analysis, functional recommendations
Interpretive emphasis	Sensitivity to global impairment; little information about cause	Localization, differential diagnosis, comorbidity, functional consequences, treatment	Cognitive profile, severity, strengths and weaknesses, validity, functional implications
Question it answers best	Should this person be evaluated further?	What medical or neurological process best explains this presentation?	What is the detailed cognitive phenotype, and how valid, severe, and functionally relevant are the findings?
Principal limits	Ceiling and floor effects; insensitive to subtle impairment and to high premorbid ability; shaped by education and language	Additional psychometric methods, referral, or collaboration may be needed for complex norms, formal validity assessment, or fine-grained analysis, depending on the examiner's training and available methods	Does not by itself establish a medical cause; requires integration with neurological, systemic, imaging, and biomarker data when cause is at issue

Table 9.1 legend. Adapted from the authors' published framework [1]. MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment.

The cognitive domains

Cognition is divided into domains much as the body is divided into organ systems: different brain networks do different jobs, can fail separately, and are tested separately. The division is not fixed by nature. Textbooks have drawn the boundaries in several ways [10]. For classification, the DSM-5 Neurocognitive Disorders Work Group settled on six principal domains, each with subdomains: complex attention, executive function, learning and memory, language, perceptual-motor function, and social cognition [11,12]. The DSM-5 gives examples of symptoms and of ways to test each domain but endorses no proprietary test [12]. This chapter follows that scheme because it is the one under which diagnoses are written. Figure 9.1 shows the six domains and their subdomains. The following sections explain each domain in plain language, with examples of difficulty in daily life and the tests used to measure it. Chapter 11 describes how each domain is affected by a brain injury; here the subject is what the domain is and how it is examined.

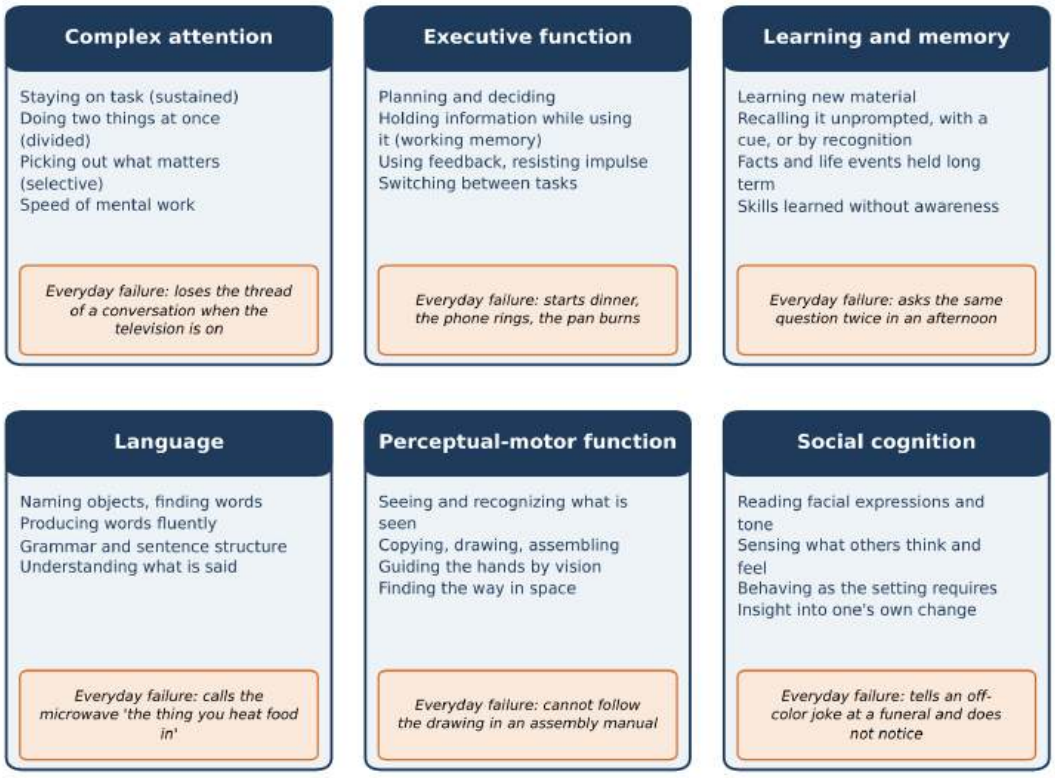


Figure 9.1. The six cognitive domains of the DSM-5 and their subdomains, in everyday terms, each with an example of what failure looks like in daily life. Original schematic; the domain and subdomain list follows the DSM-5 and the account of its Neurocognitive Disorders Work Group [11,12].

Complex attention

Attention is the capacity to select what matters, hold it in mind, and keep it there against competition. The DSM-5 lists four subdomains. Sustained attention is staying with a task over time: following a lecture to the end, reading a page without drifting. Selective attention is picking out the relevant signal and ignoring the rest: hearing one voice at a noisy table. Divided attention is doing two things at once: driving while following directions, cooking while supervising a child. Processing speed is how fast the mental work gets done, and it sits in this domain because speed and attention are measured together and fail together [11,12].

In daily life, attention difficulties can mean losing the thread of a conversation when the television is on, rereading the same page, being unable to talk and drive, or becoming exhausted after an hour of mental work that used to feel effortless. Attention is tested by asking the patient to repeat strings of digits forward and backward, to press a key whenever a target appears on a screen over several minutes while letting other stimuli pass, to say the color of the ink in which a color word is printed when the word and the ink disagree, to connect numbered circles in order as fast as possible, and to substitute symbols for numbers against a key under a time limit [10]. The last two are timed and show slowing most reliably: the answers are correct but take too long. Divided attention is tested by asking for two tasks at once, for example connecting circles while alternating between numbers and letters, or walking while counting backward. It is the subdomain that standard testing samples least, a point Chapter 10 develops.

Executive function

Executive functions are the management functions of the mind: deciding what to do, planning how, starting, keeping the plan in mind, resisting the impulse to do something else, noticing when the plan is failing, and changing it. The DSM-5 subdomains are planning, decision making, working memory, responding to feedback and error correction, overriding habits and inhibition, and mental flexibility [11,12]. Working memory is the capacity to hold a small amount of information in mind while doing something with it: keeping a telephone number while finding a pen, carrying a figure while adding a column, holding the first half of a sentence while the second half arrives. It provides a temporary mental workspace and fails with attention.

Executive difficulties appear in familiar situations: starting dinner, answering the telephone, and burning the pan; struggling to organize a once-routine trip; starting several tasks and finishing none; persisting with something that is not working; or saying the first thing that comes to mind. Executive function is tested by asking the patient to sort cards by a rule that changes without warning and to notice from the examiner’s

feedback that it has changed; to plan a sequence of moves that will rearrange colored discs in the fewest steps; to produce as many words as possible beginning with a given letter in one minute; to repeat a list of digits in reverse order; to alternate between two sequences; and to inhibit a well-practiced response, as in the color-word test above [10]. These tests are structured and supervised, so they understate the difficulties of a patient who struggles only when nobody sets the task. The family's account of everyday behavior belongs alongside the test findings.

Learning and memory

Memory is several abilities that can fail separately. The first step is learning, or encoding, taking information in. The second is storage, keeping it. The third is retrieval, getting it back. The DSM-5 subdomains run through that sequence: immediate memory span, recent memory with free recall, cued recall, and recognition, very-long-term memory of facts (semantic) and of one's own life (autobiographical), and implicit learning, the acquisition of skills and habits without awareness [11,12]. The three forms of recall matter for interpretation. Free recall is producing the list unprompted. Cued recall is producing it when given a category or a first letter. Recognition is picking the words out from a longer list. A patient who cannot recall freely but recognizes well has stored the information and cannot find it, a retrieval problem that points to the front of the brain and its connections; a patient who recognizes no better than chance never stored it, a problem that points to the memory structures of the temporal lobe [10].

Memory difficulties may become apparent when someone asks the same question twice in an afternoon, cannot recall yesterday's meeting, loses track of whether a bill was paid, or relies on a spouse to remember for the household. Memory is tested by reading a list of words several times and asking for it back immediately, after a delay of twenty or thirty minutes, with cues, and by recognition; by reading a short story and asking for it back; by showing designs or faces and asking the patient to draw or pick them out later; and by asking about well-known public events and the patient's own history [10].

Language

Language is the ability to understand and produce words and sentences, spoken and written. The DSM-5 subdomains are expressive language, which includes naming objects, finding words, fluency, and grammar and syntax, and receptive language, which is comprehension [11,12]. After a stroke, language can fail outright; after a closed head injury that is rare, and what is common is the milder failure of word finding and of the social use of language, which Chapter 11 describes.

Language difficulties can surface as calling the microwave “the thing you heat food in,” substituting a general word for a specific one, losing the point halfway through a sentence, or struggling to follow a conversation among several people. Naming is tested by showing line drawings of objects, from common to rare, and asking for the name [10]. Fluency is tested by asking for as many animals, or as many words beginning with a letter, as possible in one minute. Comprehension is tested by commands of increasing grammatical complexity, by pointing to the picture that matches a sentence, and by questions whose answers depend on word order. Repetition, reading, and writing complete the examination.

Perceptual-motor function

This domain covers seeing and recognizing what is seen, understanding where things are in space, and guiding the hands by vision. The DSM-5 subdomains are visual perception; visuoconstructional reasoning, including copying, drawing, and assembling; and perceptual-motor coordination, which integrates vision with movement. Related abilities include praxis, carrying out a learned movement on request, and gnosis, recognizing objects and faces [11,12].

In daily life, perceptual-motor difficulties may appear as an inability to follow an assembly diagram, getting lost in a familiar parking garage, misjudging distances when parking, difficulty arranging places at a table, or failure to recognize faces. The domain is tested by asking the patient to copy a complex geometric figure and later draw it from memory, to assemble colored blocks to match a printed pattern under a time limit, to judge the angle of lines, to identify objects drawn as overlapping outlines or seen from unusual angles, to draw a clock set to a given time, and to show how a tool would be used [10]. Several of these tests require good vision and steady hands. After a head injury, double vision, an eye movement disorder, or a tremor can cause failure for reasons that are not perceptual. The neurological examination helps interpret those scores.

Social cognition

Social cognition is the ability to read other people and to conduct oneself accordingly. The DSM-5 added it as a domain because some brain disorders primarily alter social behavior while other domains remain preserved. Earlier manuals classified these changes as personality change [12]. The subdomains are recognition of emotions, from faces and tone of voice; theory of mind, the capacity to work out what another person knows, intends, or feels; and insight, including awareness of one’s own change [11,12].

Changes in social cognition may be striking: an off-color joke at a funeral without noticing the silence, failure to sense that a listener has lost interest, taking sarcasm literally, blunt personal remarks to strangers, lack of concern for a distressed family member, or insistence that nothing has changed while the family describes a different person. Social cognition is tested by asking the patient to name the emotion shown in photographs of faces or of eyes alone, to say what a character in a short story believes or intends when the character's knowledge differs from the listener's, to detect a social blunder in a described scene, and to rate their own difficulties on a questionnaire that a family member fills out independently. The gap between the two ratings provides a measure of insight [10]. This is the domain where the family interview carries the most weight, because the patient with impaired insight is the least reliable witness to it.

What the DSM-5 scheme does not list separately

Three functions examined routinely are assigned to other domains in the DSM-5 scheme. Orientation means knowing the date, place, and situation. It is examined first because disorientation changes the meaning of everything that follows. The scheme treats it as an element of attention and memory rather than a separate domain. It is the function most disturbed in delirium, which must be excluded before any neurocognitive diagnosis is made [12]. Praxis, carrying out a learned purposeful movement on request, such as showing how to brush the teeth, is placed under perceptual-motor function. Calculation, tested by serial subtraction and simple written arithmetic, draws on attention, working memory, and language, and a failure has to be traced back to one of them. In a report these three appear within the mental status examination rather than in the domain summary.

Who does what

Clinicians use the terms cognitive testing, cognitive assessment, and neuropsychological assessment interchangeably, and this habit creates confusion when results are interpreted outside the scope for which they were obtained [1,2]. The authors set out, in a 2026 review written with a clinical neuropsychologist and colleagues in neurosurgery and rehabilitation medicine, a framework that distinguishes what is being done from who is doing it; its positions are stated here as the paper states them [1].

Two related activities

Cognitive assessment is a structured medical evaluation of cognitive function, integrated with the neurological and systemic context. Its defining feature is not any

particular instrument but the synthesis of cognitive observations with the history, an informant's account, functional status, the neurological examination, and relevant medical data, including imaging, electrophysiology, vestibular testing, sleep studies, and laboratory results, to answer a diagnostic or management question [1]. It is not inherently brief, and it may incorporate standardized neuropsychological instruments.

Neuropsychological assessment is a specialized form of cognitive evaluation distinguished by its measurement model: comprehensive, standardized, norm-referenced testing across multiple domains, with attention to reliability and validity, profile analysis, performance validity assessment when interpretability is a concern, and translation of findings into functional and rehabilitative recommendations [1,7]. It can support a DSM-based diagnosis of a neurocognitive disorder, characterize a cognitive phenotype, describe function, and generate diagnostic hypotheses. Test performance alone does not establish a specific medical or neurological cause [1].

Competence rather than professional title

Cognitive evaluation is part of neurological practice. Neurologists are trained to examine cognition and can perform cognitive and neuropsychological assessments using methods within their training and competence [1,9]. Their work can extend beyond screening to standardized testing and interpretation integrated with the history, neurological examination, and other medical findings. The standard for any particular procedure is appropriate education and training, experience with the instruments, standardized administration and scoring, understanding of psychometric limits, and clinical integration [1]. That standard applies to every examiner. A physician’s title alone does not establish competence with a complex psychometric battery, just as a battery alone, whoever administers it, cannot establish medical cause without the relevant neurological evaluation. The report should make clear what was done, the examiner’s competence for that work, and what the findings establish [1].

The training expectations are explicit. ACGME neurology residency requirements include clinical and didactic experience in behavioral neurology and the neurology of aging, as well as demonstrated knowledge in those fields [27]. ABPN continuing-certification specifications include neuropsychological and cognitive testing among neurological diagnostic procedures [28]. These expectations support the place of cognitive evaluation within neurology; they do not imply identical training across professions or individual examiners. As Pappolla et al. emphasize, clinical neuropsychologists receive more extensive training in psychometrics, test development, validity methodology, and normative interpretation than neurologists generally do [1]. That additional expertise matters when a question requires specialized

methods or unusually complex interpretation. The AAN's 2018 mild cognitive impairment guideline recommends more detailed evaluation after a positive brief assessment, such as neuropsychological testing interpreted against appropriate norms, without restricting it to one professional title [13]. The American Psychological Association guidelines likewise require psychologists to distinguish screening, cognitive testing, and neuropsychological testing and to judge their competence for each [1]. Cognitive testing occurs in many disciplines. Without attention to its psychometric limits and neurological correlates, findings can be attributed to psychiatric or developmental causes while a neurological cause goes unexamined [1].

What each discipline contributes

Table 9.2 describes complementary contributions, with areas of overlap [1]. Neurologists examine and measure cognition within a medical evaluation; they do not simply receive scores produced elsewhere. Clinical neuropsychologists contribute detailed psychometric interpretation, including instrument selection, estimates of premorbid ability, reliability and validity assessment, error-pattern analysis, demographic and cultural norms, and a cognitive phenotype that guides the differential diagnosis [1,7]. The useful question is which expertise the patient's particular problem requires and whether the examiner has it.

Table 9.2. Complementary contributions of the neurologist and the clinical neuropsychologist

Clinical question	Neurologist	Clinical neuropsychologist	Value of combining them
Medical diagnosis and cause	Links symptoms to the neurological examination, systemic illness, imaging, biomarkers, medications, sleep, and disease mechanism	Provides the cognitive phenotype and behavioral data that support or challenge a diagnostic hypothesis	Less reliance on test scores alone or on clinical impression alone

Clinical question	Neurologist	Clinical neuropsychologist	Value of combining them
Psychometrics and norms	Administers and interprets standardized cognitive and neuropsychological instruments within training and competence, integrating the scores with medical findings	Selects and interprets comprehensive batteries with demographic, cultural, and premorbid adjustment	Greater precision and less misclassification
Performance validity	Selects and interprets appropriate validity measures within competence and considers pain, fatigue, medication, and neurological findings	Applies and interprets embedded and stand-alone validity measures, especially in forensic settings	Better interpretability and evidentiary reliability
Language, culture, and education	Identifies when language, literacy, education, sensory loss, or socioeconomic context may affect results	Adapts the strategy, the norms, and the interpretation when specialized methods are required	Fairer interpretation across populations
Functional and occupational questions	Measures cognitive and other neurological impairments and relates them to medical restrictions, function, prognosis, and treatment	Quantifies cognitive strengths, weaknesses, and functional consequences when formal measurement is required	Role-specific recommendations without confusing diagnosis with legal capacity
Management and follow-up	Tracks cognitive and neurological change, treats medical contributors, and integrates findings with the disease course	Tracks change and provides rehabilitation, compensatory, and caregiver recommendations	Patient-centered diagnosis and care planning

Table 9.2 legend. Adapted from the authors' published framework [1]. The columns describe overlapping contributions and differences in training depth, not exclusive ownership of particular tasks. Each clinician must work within competence.

Why the neurological examination cannot be skipped

Many diagnoses in cognitive neurology depend on findings that no cognitive test can produce, and the paper gives two examples [1]. An older patient whose testing shows attention and executive deficits may, from the scores alone, be read as having early Alzheimer's disease; the caregiver's account of visual hallucinations and day-to-day fluctuation, and the examiner's finding of asymmetric rigidity and slowness, change the diagnosis to dementia with Lewy bodies. A middle-aged patient with a history of brain injury whose testing shows mild reductions in processing speed and attention, which are nonspecific, has impaired smooth pursuit and convergence insufficiency on examination and describes difficulty with visually demanding tasks and in busy sensory environments; integrated, the cognitive complaint is better understood as part of the injury's effect on the visual and attentional systems than as unrelated decline [1]. Subtle neurological signs such as primitive reflexes and abnormal saccades and pursuit predict the severity of cognitive and functional impairment after brain injury, and only a neurological examination finds them [14]. The history remains the single most important diagnostic tool in the evaluation of a cognitive complaint [15].

Performance validity testing

Cognitive tests measure performance, and performance depends on the person taking the test. A patient who is exhausted, in pain, sedated, depressed, frightened of the result, or not trying will score below their ability, and the examiner cannot tell from the scores alone which it was. Performance validity tests address this. Stand-alone validity tests look like memory tests but are so easy that people with severe brain disease pass them. Embedded validity indicators are patterns within the regular tests that are implausible in someone performing to capacity, for example recognizing fewer words than were freely recalled. Symptom validity tests, which are different, examine whether the symptoms reported on questionnaires are internally consistent and plausible. The American Academy of Clinical Neuropsychology's 2021 consensus statement expects multiple validity measures spread through the battery, interpreted together, with the examiner stating what was used and what was concluded [16]. The authors' paper emphasizes the need for training in neuropsychological validity methods, particularly in medicolegal work. This is a requirement for the methods being used, not a claim that a neurologist cannot use them when appropriately trained. The American Psychological

Association's position is that behavioral observation and clinical judgment alone are not sufficient to determine validity [1].

Two facts govern how a validity result is read. First, failure is common across clinical populations in which there is no apparent incentive to underperform. McWhirter and colleagues reviewed 69 studies covering 45 validity tests in patients with intellectual disability, degenerative brain disease, brain injury, psychiatric disorders, functional disorders, and epilepsy, excluding litigating, forensic, and military samples, and found failure in every group, exceeding one in four for some groups and tests [17]. A failed validity test therefore tells the reader that the cognitive scores from that session cannot be taken at face value. It does not by itself say why, and it is not a finding of dishonesty; the reasons include fatigue, pain, medication, severe depression, a functional disorder, misunderstanding, and in some cases deliberate underperformance, and distinguishing among them is a clinical judgment made from the whole record [1,16,17]. Second, passing a validity test supports the interpretability of performance but does not guarantee that every score is valid. Its weight depends on the measure's sensitivity, the other validity findings, and the testing circumstances [16]. Neither a passed nor a failed validity measure, by itself, establishes whether the injury caused the complaints. A flag is a reason for caution and closer reading, not a disqualification.

The three-tier model

The paper proposes a tiered, method-matched approach for aging populations and patients with traumatic brain injury (Figure 9.2) [1]. Tier 1 is a neurologist-led cognitive assessment using validated screening and single-domain tests, modeled after the neurobehavioral status examination, for diagnostic and management questions, integrated with the neurological examination, imaging, and biomarkers. Tier 2 is referral for comprehensive neuropsychological assessment when developmental or educational factors complicate interpretation, sensory or motor limitations constrain testing, performance validity is a concern, fine-grained phenotyping is needed, or a formal functional determination is required. Tier 3 is collaborative integration, in which neuropsychological findings inform the neurological diagnosis and management, with the recognition that attribution to a cause requires medical evaluation [1]. The paper presents the model as an arrangement of existing guidance into a practical workflow, not as a replacement for any guideline. It notes that in many settings the tiers run concurrently in a memory clinic or a rehabilitation program rather than in sequence, and adds that collaboration means direct communication between the disciplines to align concepts and interpretive assumptions, not parallel reports that never meet [1].

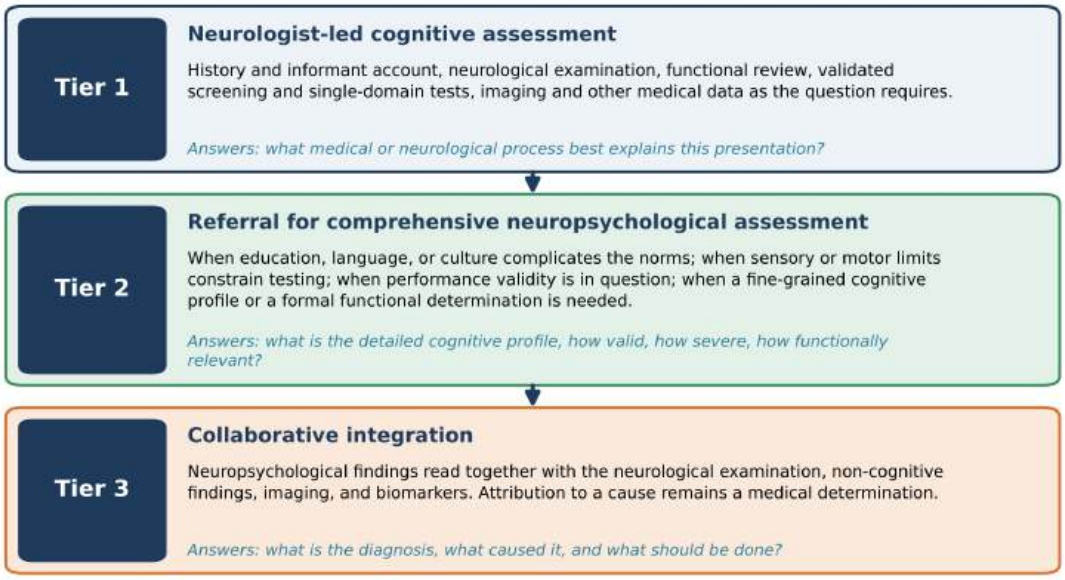


Figure 9.2. The three-tier, method-matched model of cognitive evaluation. Tier 1 is neurologist-led cognitive assessment; Tier 2 is referral for comprehensive neuropsychological assessment when the listed conditions apply; Tier 3 is collaborative integration, in which attribution to a cause remains a medical determination. Original schematic redrawn from the authors' published model [1].

The diagnostic labels

The diagnostic labels used after a cognitive examination have precise meanings, but those meanings can become blurred between the report and the courtroom. Four are especially relevant here: mild cognitive impairment, mild neurocognitive disorder, major neurocognitive disorder, and neurocognitive disorder due to traumatic brain injury.

Mild cognitive impairment

Mild cognitive impairment (MCI) was defined by Petersen and colleagues at the Mayo Clinic in 1999 to describe the state between normal aging and dementia: a memory complaint, preferably confirmed by an informant; memory impairment on testing that is abnormal for age; general cognitive function otherwise essentially preserved; daily activities largely intact; and no dementia [18]. The concept was broadened over the following decade to include impairment in domains other than memory. The 2011 criteria of the National Institute on Aging and the Alzheimer's Association, written for MCI due to Alzheimer's disease but containing a general definition, require a concern about a change in cognition from the patient, an informant, or a clinician; impairment in one or more cognitive domains, typically one to one and a half standard deviations

below the mean for age and education; preserved independence in daily function, with allowance for mild difficulty and greater effort in complex tasks; and no dementia [19].

The American Academy of Neurology's 2018 practice guideline update on MCI summarized its epidemiology and provided recommendations [13]. Prevalence rises steeply with age: 6.7 percent at 60 to 64 years, 8.4 percent at 65 to 69, 10.1 percent at 70 to 74, 14.8 percent at 75 to 79, and 25.2 percent at 80 to 84. Among people with MCI older than 65 who were followed for two years, 14.9 percent developed dementia, meaning most did not. The guideline recommended assessing for MCI with validated tools in appropriate scenarios; referring to a specialist experienced in cognition when the clinician lacks that experience; conducting more detailed evaluation, such as neuropsychological testing, interpreted against appropriate norms when a screen is positive; searching for modifiable risk factors; recommending regular exercise twice a week; offering cognitive training as an option; withholding cholinesterase inhibitors or, if they are offered, discussing the absence of evidence; and discussing diagnosis, prognosis, and long-term planning with the patient and family [13]. The AAN lists this guideline as retired as of February 2024 without naming a replacement, so it should be cited as the Academy's 2018 statement rather than as current guidance; its definition and prevalence figures remain in general use.

MCI, under every definition, describes a cognitive state, measurable impairment with preserved independence, and says nothing about cause.

Mild and major neurocognitive disorder

The DSM-5, published in 2013 and revised as the DSM-5-TR in 2022, replaced the older terms dementia and cognitive disorder not otherwise specified with two parallel syndromes, mild neurocognitive disorder and major neurocognitive disorder, which share the same structure but differ in thresholds [11,12]. Mild neurocognitive disorder is the DSM-5 framework for what clinicians call MCI; major neurocognitive disorder is largely synonymous with dementia, a term the manual retains in parentheses [12].

Both diagnoses rest on the same logic: a decline from a previously attained level of function, in one or more of the six domains described above, established by two kinds of evidence together. The first is concern, expressed by the patient, by a knowledgeable informant, or by the clinician, that there has been a decline. The second is objective impairment on testing, preferably standardized neuropsychological testing or, where that is unavailable, another quantified clinical assessment [11,12]. The deficits must not occur only during delirium and must not be better explained by another mental disorder such as major depression or schizophrenia. The decline is the essential idea. A

neurocognitive disorder is acquired; a person who has always scored at the tenth percentile has not declined by scoring there now, which is why the estimate of premorbid ability, and any school or employment record that bears on it, matters so much.

The two diagnoses differ in severity and in function. In mild neurocognitive disorder the decline is modest. As a guide, the DSM-5 places test performance between one and two standard deviations below the normative mean, roughly the third to the sixteenth percentile. The deficits do not interfere with the capacity for independence in everyday activities, though the person may need greater effort, compensatory strategies, or accommodation to pay bills, manage medications, and run a household [11,12]. In major neurocognitive disorder the decline is significant. Performance typically falls two or more standard deviations below the mean, below the third percentile. The deficits interfere with independence, at a minimum requiring help with complex activities such as finances or medications [11,12]. Independence in everyday activities is the dividing line, and it is judged from the account of the patient and the family together with the clinician’s judgment, not from the scores alone [12].

Three features of the scheme deserve attention. First, decline in a single domain is sufficient for either diagnosis; memory impairment is not required. Second, the diagnosis carries no requirement of further decline: the course may be progressive, as in a neurodegenerative disease, or static, as after a traumatic brain injury, and mild neurocognitive disorder is not necessarily a precursor of major. Third, the DSM-5 does not specify which tests, or how many per domain, must be given. A bedside assessment may supply the objective evidence when formal testing is not feasible. The work group nevertheless noted that formal neuropsychological assessment matters more in mild than in major neurocognitive disorder, because the mild syndrome has to be distinguished both from normal aging and from major neurocognitive disorder [12].

The diagnosis is made in two steps. The syndrome, mild or major, is established first. The cause is then assigned as a subtype: Alzheimer’s disease, frontotemporal lobar degeneration, Lewy body disease, vascular disease, traumatic brain injury, substance or medication use, HIV infection, prion disease, Parkinson’s disease, Huntington’s disease, another medical condition, multiple etiologies, or unspecified [11,12]. For most subtypes the clinician must also exclude the other causes, because the cognitive profile by itself is not specific enough to name the disease [12]. The 2024 revision of the Alzheimer’s Association criteria carries that logic to its conclusion for one disease: Alzheimer’s disease is now defined by biomarkers of its pathology, in cerebrospinal fluid, on amyloid positron emission tomography, or in blood, with the clinical syndrome

staged separately [20]. In an older patient with a brain injury and a cognitive complaint, whether a second, degenerative process is present is answered by medical evaluation and, where indicated, biomarkers, not by the pattern of scores.

Neurocognitive disorder due to traumatic brain injury

The DSM-5 criteria for major or mild neurocognitive disorder due to traumatic brain injury have three parts [11]. First, the criteria for the syndrome must be met. Second, there must be evidence of a traumatic brain injury, defined as an impact to the head or another mechanism of rapid movement or displacement of the brain within the skull, with one or more of the following: loss of consciousness, post-traumatic amnesia, disorientation and confusion, or neurological signs, of which the manual's examples are neuroimaging showing injury, new-onset seizures or marked worsening of a seizure disorder, visual field cuts, loss of smell, and hemiparesis. Third, the neurocognitive disorder must present immediately after the injury, or immediately after recovery of consciousness, and persist past the acute post-injury period [11]. The manual adds that the diagnosis depends on performance across domain-specific cognitive assessments interpreted in the light of the person's prior functioning, together with an assessment of functional status, and that neuroimaging and other clinical findings may support but cannot independently establish it [1,11].

Two consequences follow. The injury evidence the DSM-5 requires overlaps with but is not the same as the 2023 ACRM diagnostic criteria for mild traumatic brain injury that Chapter 3 sets out; a clinician who makes this diagnosis has asserted that a brain injury occurred, and that assertion rests on the injury evidence, not on the cognitive scores [21]. And a cognitive decline beginning months after an injury, with no cognitive symptoms in between, does not fit the criterion of onset immediately after the injury, so a report should show that the chronology was examined. Cognitive disturbance after a mild injury usually resolves; persistent impairment past the expected period is what the diagnosis describes, and the fact that most patients recover is not evidence that a particular patient did.

How these labels relate to persistent post-concussion syndrome

Chapter 10 describes persistent post-concussion syndrome and the gap left when the DSM-5 and ICD-11 withdrew their stand-alone post-concussional categories. The two labels are independent. Neurocognitive disorder requires cognitive decline and nothing else; a patient with disabling headache, dizziness, visual intolerance, and sleep disturbance after a concussion, whose cognition tests normal, has no neurocognitive disorder and may have persistent post-concussion syndrome. A patient with

documented cognitive decline after an injury and no other symptoms has a neurocognitive disorder due to traumatic brain injury and may not meet a post-concussion definition that requires several symptoms. Many patients have both. Neither diagnosis is required for the other, and a report that treats the absence of one as evidence against the other has conflated two questions [8].

What a label says about cause

For an attorney, the distinction between syndrome and cause is one of this chapter's most useful ideas. Mild cognitive impairment and mild or major neurocognitive disorder, without an etiological subtype, describe how much cognition has declined and whether the person remains independent. Testing interpreted against prior ability and an account of daily function establish the syndrome. Both neurologists and clinical neuropsychologists can diagnose neurocognitive disorders under standardized criteria within their competence [1]. A subtype such as due to traumatic brain injury or due to Alzheimer's disease makes an additional claim about cause. It requires evidence beyond the scores: the injury record, neurological examination, chronology, imaging, biomarkers, and consideration of other explanations. Any report assigning a medical cause should show that evidence. A neurological report asserting cognitive decline should also show how the decline was quantified. The syndrome and its cause each need an explicit evidentiary basis.

What a normal result means

Each cognitive test, like each test in Chapter 7, is a keyhole, and a normal view through it shows that the function it measures was preserved on the day it was measured, and nothing more. Two guidelines and one critique say this about brain injury in particular. The 2025 adapted clinical practice guideline of the Action Collaborative on Traumatic Brain Injury Care states that neuroimaging and neuropsychological testing are neither necessary nor routinely performed to confirm or exclude a diagnosis of traumatic brain injury in the post-acute stage, advises against comprehensive neuropsychological assessment during active recovery, and reserves referral for patients with persisting cognitive symptoms beyond one month, to guide treatment or return to school, work, or sport [22]. The 2021 Veterans Affairs and Department of Defense guideline suggests against comprehensive neuropsychological or cognitive testing during the first 30 days after a mild injury and found no evidence that routine testing in the post-acute period diagnoses the injury, guides treatment, or improves outcomes [23]. Bigler and colleagues argue in a 2024 critique that the traditional neuropsychological battery was not developed to detect the subtle changes of mild traumatic brain injury and was never validated as a stand-alone method for doing so; this is a critique by experienced

investigators rather than a validation study [24]. The position the authors have taken in print follows: normal cognitive or neuropsychological testing does not exclude a traumatic brain injury, because many disabling consequences of the injury are not cognitive, because deficits may appear only under load or fatigue, and because high premorbid ability can hide a real decline inside the normal range [1]. The further limitation, that standard testing is given one task at a time in a quiet room and samples poorly the divided attention that daily life demands, belongs to Chapter 10, where the evidence for it is weighed.

The principle has a second half, and the same reader must hold both. An abnormal score does not establish an injury. Binder, Iverson, and Brooks showed that in normative samples given batteries of 20 or more measures, with abnormality defined as a score more than one standard deviation below the mean, the great majority of healthy adults had at least one abnormal score, that large gaps between a person's best and worst scores were usual, and that restricting the sample to people of higher intelligence or education, or moving the cut-off to two standard deviations, did not eliminate the abnormal scores [25]. A pattern of low scores that cluster in the domains the injury would be expected to affect, that match the complaints and the neurological findings, that persist on retesting, and that have no better explanation is evidence of a different order. And an abnormal score that is real still has to be assigned a cause; depression, pain, poor sleep, medication, sleep apnea, and alcohol produce the same kinds of low scores that an injury does. A valid, abnormal profile tells the reader that this person performed below expectation in these domains on this day. What caused it is answered from the whole record.

Reading a neuropsychological report in a contested case

People reading a neuropsychological report in litigation may not have ordered the assessment or been present during it. The following questions help a reader on either side understand how the examiner reached the conclusions.

What was the estimate of the patient's ability before the injury, and on what did it rest? A report should say whether premorbid ability was estimated from a word-reading test, a formula using education and occupation, school or employment records, or a combination. Decline is a difference between then and now, and the "then" is always an estimate.

Which norms were used? The age, education, sex, and where relevant language and ethnicity of the comparison group determine the percentile, and the same raw score

can be average against one set of norms and impaired against another. A report that does not name its norms cannot be checked.

What validity measures were given, what did they show, and how did the examiner reason from them? The reader should look for whether failures were weighed against the clinical context, and whether a passed validity profile was weighed as evidence supporting score interpretation, with attention to the measures' limitations, rather than as a guarantee of validity or a finding about the cause of the complaints [16].

Which domains were impaired, by what cut-off, and how were the scores labeled? The American Academy of Clinical Neuropsychology's 2020 consensus statement recommends a uniform descriptive vocabulary: scores below the second percentile are exceptionally low; the second to eighth percentile, below average; the ninth to twenty-fourth, low average; the twenty-fifth to seventy-fourth, average; and so upward. These labels describe scores rather than people. Whether an ability is impaired is a separate clinical judgment that takes premorbid ability into account [26]. Examiners use different cut-offs for impairment, and a report should state its own; the convention applied in Dr. Pappolla's outpatient neurology clinic, with a score at or below the tenth percentile used as a flag for possible impairment that requires clinical interpretation, is described in Chapter 11. How many tests were given also matters, because of the chance findings described above [25].

Were the non-cognitive neurological findings considered? The report should show that the examiner knew the results of the neurological examination, the eye movement recording, the computerized posturography, the imaging, and any other objective testing, and either integrated them or stated that they were outside the scope of the evaluation. A report that concludes "no evidence of brain injury" from cognitive scores alone has answered a question it did not examine; a report that concludes "cognitive testing shows no impairment in the domains assessed, with valid performance" has stated exactly what it found, and that statement is useful to everyone [8].

Does the conclusion about cause rest on the test scores alone? If the report attributes the deficits to the injury, it should show the injury evidence, the chronology, and the alternatives considered. If it attributes them to depression, pain, or pre-existing conditions, it should show the same, and explain why the alternative accounts for the particular pattern found. A conclusion about cause in either direction that cites only the profile has exceeded what a profile can show.

Finally, what does the report say about function? Whether the person can manage money, medications, work, and a household determines both the practical significance

of a finding and the distinction between mild and major neurocognitive disorder. A report that gives scores without describing function omits a part of the diagnosis that matters greatly to the patient.

Fast take: main points from chapter 9

• A cognitive examination ranges from the bedside mental status examination, through scored screening instruments and the office-based neurobehavioral status examination, to the comprehensive neuropsychological battery. The levels answer different questions and should not be confused [1,2,9].

• Screening instruments detect dementia adequately and mild impairment poorly; after mild traumatic brain injury the MMSE misses about half of impaired patients. Ceiling effects hide decline in people of high ability and floor effects produce failure for reasons unrelated to brain disease [6,7,8].

• The DSM-5 recognizes six cognitive domains, each with subdomains: complex attention, executive function, learning and memory, language, perceptual-motor function, and social cognition. Each is tested by specific tasks, and each failure has a recognizable everyday form [11,12].

• Cognitive assessment is a medically integrated process; neuropsychological assessment is a specialized psychometric methodology. Neurologists are trained in cognitive evaluation and can perform cognitive and neuropsychological assessments within their training and competence. Clinical neuropsychologists bring greater depth in specialized psychometrics, validity methods, and complex normative interpretation. The assessment should match the question and the examiner's demonstrated skills [1,9,27,28].

• Neurologists and clinical neuropsychologists are qualified to diagnose neurocognitive disorders under standardized criteria within their competence. Attribution to a specific cause, a traumatic brain injury or a neurodegenerative disease, requires medical evaluation that extends beyond cognitive testing [1].

• Performance validity failure occurs across clinical populations without external incentive. A failed measure means the scores cannot be taken at face value, not that the patient was dishonest; a passed measure supports interpretation but does not guarantee valid scores or establish that the injury caused the complaints [16,17].

• The three-tier model matches the method to the question: neurologist-led cognitive assessment, referral for comprehensive neuropsychological assessment when specified conditions apply, and collaborative integration [1].

• Mild cognitive impairment and mild neurocognitive disorder describe measurable decline with preserved independence; major neurocognitive disorder requires loss of independence. Both rest on concern plus objective evidence of decline from a previous level, and neither names a cause until a subtype is assigned [11,12,13,18,19].

• Neurocognitive disorder due to traumatic brain injury requires the syndrome, evidence of the injury, and onset immediately after the injury with persistence past the acute period. It and persistent post-concussion syndrome are independent diagnoses; a patient can have either without the other [8,11].

• Current guidelines state that neuropsychological testing is neither necessary nor routinely performed to confirm or exclude a traumatic brain injury. A normal result shows preserved performance in the domains and conditions tested; an abnormal result shows impaired performance and does not by itself say why [22,23,25].

• In a contested case, read a neuropsychological report for the premorbid estimate, the norms, the validity measures and their interpretation, the domains impaired and the cut-off used, whether non-cognitive findings were considered, and whether the conclusion about cause rests on the scores alone.

References

1. Pappolla MA, Pappolla SL, Nader R, Hamza MK, Fang F, Fang X. Evaluating cognition across aging and traumatic brain injury: integrating neurological and neuropsychological approaches. J Clin Med. 2026;15(10):3822. doi:10.3390/jcm15103822
2. Block CK, Johnson-Greene D, Pliskin N, Boake C. Discriminating cognitive screening and cognitive testing from neuropsychological assessment: implications for professional practice. Clin Neuropsychol. 2017;31(3):487-500. doi:10.1080/13854046.2016.1267803
3. Folstein MF, Folstein SE, McHugh PR. “Mini-mental state”: a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975;12(3):189-198. doi:10.1016/0022-3956(75)90026-6
4. Nasreddine ZS, Phillips NA, Bédirian V, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53(4):695-699. doi:10.1111/j.1532-5415.2005.53221.x

5. Borson S, Scanlan J, Brush M, Vitaliano P, Dokmak A. The Mini-Cog: a cognitive “vital signs” measure for dementia screening in multi-lingual elderly. Int J Geriatr Psychiatry. 2000;15(11):1021-1027. doi:10.1002/1099-1166(200011)15:11<1021::AID-GPS234>3.0.CO;2-6

6. Patnode CD, Perdue LA, Rossom RC, et al. Screening for cognitive impairment in older adults: updated evidence report and systematic review for the US Preventive Services Task Force. JAMA. 2020;323(8):764-785. doi:10.1001/jama.2019.22258

7. Roebuck-Spencer TM, Glen T, Puente AE, et al. Cognitive screening tests versus comprehensive neuropsychological test batteries: a National Academy of Neuropsychology education paper. Arch Clin Neuropsychol. 2017;32(4):491-498. doi:10.1093/arclin/acx021

8. Pappolla MA, Nader R, Fang F, Fang X. Persistent post-concussion syndrome after its withdrawal from DSM-5 and ICD-11: definitional disagreement and the neurocognitive gap. J Clin Med. 2026;15(20):8031. doi:10.3390/jcm15208031

9. Daffner KR, Gale SA, Barrett AM, et al. Improving clinical cognitive testing: report of the AAN Behavioral Neurology Section Workgroup. Neurology. 2015;85(10):910-918. doi:10.1212/WNL.0000000000001763

10. Lezak MD, Howieson DB, Bigler ED, Tranel D. Neuropsychological Assessment. 5th ed. New York (NY): Oxford University Press; 2012.

11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed, text revision (DSM-5-TR). Washington (DC): American Psychiatric Association Publishing; 2022.

12. Sachdev PS, Blacker D, Blazer DG, et al. Classifying neurocognitive disorders: the DSM-5 approach. Nat Rev Neurol. 2014;10(11):634-642. doi:10.1038/nrneurol.2014.181

13. Petersen RC, Lopez O, Armstrong MJ, et al. Practice guideline update summary: mild cognitive impairment: report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology. Neurology. 2018;90(3):126-135. doi:10.1212/WNL.0000000000004826

14. Wortzel HS, Frey KL, Anderson CA, Arciniegas DB. Subtle neurological signs predict the severity of subacute cognitive and functional impairments after traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2009;21(4):463-466. doi:10.1176/jnp.2009.21.4.463

15. Arvanitakis Z, Shah RC, Bennett DA. Diagnosis and management of dementia: review. JAMA. 2019;322(16):1589-1599. doi:10.1001/jama.2019.4782

16. Sweet JJ, Heilbronner RL, Morgan JE, et al. American Academy of Clinical Neuropsychology (AACN) 2021 consensus statement on validity assessment: update of the 2009 AACN consensus conference statement on neuropsychological assessment of effort, response bias, and malingering. Clin Neuropsychol. 2021;35(6):1053-1106. doi:10.1080/13854046.2021.1896036
17. McWhirter L, Ritchie CW, Stone J, Carson A. Performance validity test failure in clinical populations: a systematic review. J Neurol Neurosurg Psychiatry. 2020;91(9):945-952. doi:10.1136/jnnp-2020-323776
18. Petersen RC, Smith GE, Waring SC, Ivnik RJ, Tangalos EG, Kokmen E. Mild cognitive impairment: clinical characterization and outcome. Arch Neurol. 1999;56(3):303-308. doi:10.1001/archneur.56.3.303
19. Albert MS, DeKosky ST, Dickson D, et al. The diagnosis of mild cognitive impairment due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimers Dement. 2011;7(3):270-279. doi:10.1016/j.jalz.2011.03.008
20. Jack CR Jr, Andrews JS, Beach TG, et al. Revised criteria for diagnosis and staging of Alzheimer's disease: Alzheimer's Association Workgroup. Alzheimers Dement. 2024;20(8):5143-5169. doi:10.1002/alz.13859
21. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036
22. Silverberg ND, Lee K, Mikolić A, et al. Action Collaborative on Traumatic Brain Injury Care: adapted clinical practice guideline. Ann Fam Med. 2025;23(6):552-569. doi:10.1370/afm.250352
23. U.S. Department of Veterans Affairs, Department of Defense. VA/DoD Clinical Practice Guideline for the Management and Rehabilitation of Post-Acute Mild Traumatic Brain Injury. Version 3.0. Washington (DC): Department of Veterans Affairs; 2021.
24. Bigler ED, Allder S, Victoroff J. What traditional neuropsychological assessment got wrong about mild traumatic brain injury. II: limitations in test development, research design, statistical and psychometric issues. Brain Inj. 2024;38(13):1053-1074. doi:10.1080/02699052.2024.2376261
25. Binder LM, Iverson GL, Brooks BL. To err is human: "abnormal" neuropsychological scores and variability are common in healthy adults. Arch Clin Neuropsychol. 2009;24(1):31-46. doi:10.1093/arclin/acn001

26. Guilmette TJ, Sweet JJ, Hebben N, et al. American Academy of Clinical Neuropsychology consensus conference statement on uniform labeling of performance test scores. Clin Neuropsychol. 2020;34(3):437-453. doi:10.1080/13854046.2020.1722244
27. Accreditation Council for Graduate Medical Education. ACGME Program Requirements for Graduate Medical Education in Neurology including FAQs. Effective July 1, 2026. Sections 4.6.b.1 and 4.11.e. Available from: https://www.acgme.org/globalassets/pfassets/programrequirements/2026-prs/180_neurology_2026.pdf. Accessed October 25, 2026.
28. American Board of Psychiatry and Neurology. Continuing Certification Examination in Neurology: Content Specifications. Posted July 29, 2025. Dimension 2, Diagnostic procedures. Available from: https://abpn.org/wp-content/uploads/Neurology_CC_Content_Specifications.pdf. Accessed October 25, 2026.

Part III. Consequences

Chapter 10 Persistent post-concussion symptoms and the neurocognitive gap

Chapter overview

Most people recover from a mild traumatic brain injury within days or weeks. A substantial minority do not, and for them the diagnostic landscape is unsettled. DSM-5 discontinued the provisional postconcussional disorder category, and ICD-11 does not retain the standalone ICD-10 postconcussional syndrome. The neurocognitive categories that replaced them require cognitive decline. That leaves the patient whose disability is vestibular, visual, headache-related, autonomic, or pain-related without a label that describes her. The published criteria that remain differ in symptom count, duration, functional requirements, and whether objective cognitive impairment is necessary. Applied to a single cohort, they produced prevalence estimates from 11.4% to 38.7%.

This chapter reviews the disagreement and its implications. It sets out a provisional clinical framework that separates characterizing the syndrome from attributing it to an injury, and explains why the cognitive and non-cognitive consequences must be evaluated together. The central argument is that a cognitive test score measures performance under specified conditions. By itself, it cannot establish the cause of an abnormal result or exclude deficits in systems it never tested.

Introduction

Most people recover from a mild traumatic brain injury (mTBI). Within days or weeks the headache resolves, the fog lifts, sleep returns, and life resumes. For a substantial minority it does not happen that way. Months or years after an injury that a hospital record may describe as minor, they continue to have daily headache, dizziness in a supermarket aisle, intolerance of screens and of noise, a memory that no longer holds what it used to, sleep that does not restore, pain in the neck and shoulders that no imaging explains, and a temper that has become unfamiliar to their family. Many have lost work they were good at.

That such patients exist is well documented across civilian, athletic, and military populations. How many there are depends on the population, the follow-up interval, and the definition used. A scoping review of 45 studies reported that approximately half of individuals with a single mTBI demonstrated long-term cognitive impairment, with 1963 of 3593 participants classified as impaired by its method [1]. A separate review estimated persisting post-concussion symptoms in 10 to 25% of patients [2]. These are different outcomes and should not be treated as interchangeable. A published rebuttal

challenged the McInnes review's definition of impairment: statistically significant group differences on individual tests had been used to classify entire injured samples as impaired, making the method inappropriate for estimating individual prevalence [3]. The disagreement illustrates why group-level effects, individual impairment, symptom persistence, and functional disability must be distinguished. It does not justify picking a prevalence midway between the published estimates.

The diagnostic approach is equally unsettled. Formal criteria differ in symptom count, duration, functional requirements, and whether objectively demonstrated cognitive impairment is necessary. Applied to the same patients, they identify substantially different groups. DSM-5 and ICD-11 no longer retain the earlier standalone postconcussional categories, and their neurocognitive categories address cognitive decline rather than the entire range of post-injury manifestations. A reader can mistake that change in classification for evidence that persisting disability does not occur. The inference is not warranted. Disagreement about a syndrome that lacks a confirmatory test does not establish that affected patients are well. It does not establish that the construct is sound, either: disagreement can reflect real limitations in the proposed syndrome, in symptom specificity, or in measurement. Three practical consequences follow. Any statement that a patient meets criteria should identify the criteria used. Removal of a label from a manual does not establish recovery. And characterizing the syndrome and attributing it to a particular injury require separate reasoning.

The scale of the definitional disagreement

A clinician looking for the diagnostic criteria for persistent post-concussion syndrome will find several conflicting answers. The differences are greater than most assume.

Rose, Fischer, and Heyer surveyed 597 physicians about the definition of post-concussion syndrome [4]. Respondents reported minimum symptom durations of less than 2 weeks (26.6%), 2 weeks to 1 month (20.4%), 1 to 3 months (33%), and more than 3 months (11.1%); these categories accounted for 91.1% of respondents. Physicians, in other words, did not agree even on how long symptoms must last before the syndrome exists.

Voormolen and colleagues quantified what that disagreement does to prevalence. In 731 patients assessed 6 months after mTBI, divergent methods applied to the same Rivermead Post-Concussion Symptoms Questionnaire data produced prevalence estimates from 11.4% to 38.7% [5]. The approaches included mappings to ICD-10 and DSM-IV criteria, a Rivermead total-score method, the RPQ-3 subscale, and a three-

factor model, with different severity cutoffs. The patients and their answers were unchanged; only the classification varied.

What the formal criteria require

ICD-10 clinical criteria

The ICD-10 description of postconcussional syndrome (F07.2) identifies a history of head trauma and a cluster including headache, dizziness, fatigue, irritability, impaired concentration or memory, insomnia, and reduced tolerance to stress, emotional excitement, or alcohol [6]. A commonly used research operationalization requires 3 or more of 8 symptoms [7]. That symptom-count rule should be distinguished from the descriptive WHO category and from the separate ICD-10 Diagnostic Criteria for Research. The clinical description specifies no duration at which symptoms become persistent, which is one source of the variation in operational definitions [8].

DSM-IV post-concussional disorder

DSM-IV proposed post-concussional disorder as a provisional diagnosis for further study. Its criteria required a history of head trauma causing significant cerebral concussion and evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention or memory. Three or more of eight symptoms had to begin shortly after trauma and persist at least three months: fatigability, disordered sleep, headache, vertigo or dizziness, irritability or aggression, anxiety or depression or affective lability, personality change, and apathy. The criteria also required onset following trauma or substantial worsening of pre-existing symptoms; significant social or occupational impairment; and exclusion of dementia due to head trauma and other better explanations [7]. The distinctive requirement is the second. DSM-IV made objectively demonstrated cognitive impairment mandatory. A patient with disabling post-traumatic headache, vestibular dysfunction, visual or gait impairment, and mood disturbance, but intact cognitive testing, therefore did not meet the criteria. Whether that requirement was well conceived is the question taken up in the section on the limits of cognitive testing.

The Lagacé-Legendre expert consensus definition

The clearest modern explicit definition comes from a Delphi consensus reported by Lagacé-Legendre and colleagues [8]. It requires an established mTBI, since this is a post-injury diagnosis, and at least one new post-concussion symptom; the panel declined to require a minimum number beyond that. Symptoms must begin within hours of the injury, cannot be attributed to a pre-existing condition, and must not be better

accounted for by another mental disorder. At least one symptom must, in the panel's language, still be present every day 3 months after the trauma and affect at least one sphere of the person's life [8].

This is an expert consensus definition rather than a validated diagnostic instrument or a professional-society standard [8]. The panel set no mandatory symptom count and no diagnostic Rivermead cutoff. That is not a verdict against symptom measurement. The Rivermead questionnaire documents symptom burden and change well, but a score alone does not establish the injury or replace clinical assessment.

The four-week threshold

The 2023 Amsterdam consensus statement on concussion in sport uses a shorter threshold, conventionally more than four weeks, under the terminology of persisting symptoms after concussion [9]. It is a current and relevant consensus document, but the threshold is a management designation rather than a causal diagnostic definition: it identifies when continuing symptoms warrant detailed multimodal evaluation and allows that such symptoms may be pre-existing, concussion-related, or both. A separate four-week figure comes from the historical ICD-10 research criteria. There it referred to the permissible interval between injury and symptom onset, rather than to persistence. Those criteria also required loss of consciousness, inconsistently with contemporary practice.

Table 10.1. Principal definitions of persistent post-concussion syndrome compared.

Framework	Symptom count	Duration required	Objective cognitive impairment required	Status
ICD-10 F07.2 and common operationalization	Common rule: 3 or more of 8	Not specified	No	Historical WHO category; coding use varies by jurisdiction [6,7]
DSM-IV postconcussional disorder	Three or more of eight	At least three months	Yes, mandatory	Provisional when published; superseded [7]
Lagacé-Legendre expert consensus	At least one; affecting function	Daily at three months	No	Expert Delphi consensus, not a society standard [8]

Framework	Symptom count	Duration required	Objective cognitive impairment required	Status
Amsterdam 2022, persisting symptoms after concussion	Not specified as a count	More than four weeks	No	Management designation rather than causal definition [9]
ICD-11	No standalone PCS category	Not applicable	Required for mild neurocognitive disorder	Mild neurocognitive disorder may describe a qualifying cognitive presentation associated with trauma [10,11]
DSM-5	Provisional DSM-IV category discontinued	Not applicable	Required for major or mild neurocognitive disorder	Major or mild neurocognitive disorder due to TBI addresses qualifying cognitive decline [12,13]

The columns that differ most are symptom count and duration, which is why prevalence estimates in a single cohort vary more than threefold [5].

The withdrawal from DSM-5 and ICD-11

DSM-5 discontinued the DSM-IV provisional postconcussional disorder category and organized persistent cognitive sequelae of TBI within major or mild neurocognitive disorder due to traumatic brain injury [12,13]. ICD-11 does not retain the standalone ICD-10 F07.2 category; mild neurocognitive disorder can describe a qualifying cognitive presentation associated with trauma [10,11]. These were decisions of two separate classification systems, not a single coordinated one. Both neurocognitive frameworks require cognitive decline and therefore cannot describe the full range of post-injury

disability. Other neurological, psychiatric, and symptom diagnoses remain available when their requirements are met.

Poor concordance and limited specificity are the relevant background. Boake, McCauley, and colleagues assessed 178 adults with mild to moderate TBI and 104 with extracranial trauma 3 months after injury. PCS prevalence was 64% using ICD-10 criteria and 11% using DSM-IV criteria, and both sets were also satisfied by some patients without head injury [7]. Clark and colleagues suggested that such lack of specificity may have influenced the removal of the categories [10]; that is an interpretation, not the recorded rationale of either committee. What the evidence does establish is that apparently similar diagnostic labels can identify substantially different populations.

Three consequences follow. First, a classification change does not establish that patients with persisting symptoms are no longer ill. Where cognitive decline meets the relevant requirements, major or mild neurocognitive disorder due to TBI is a useful formulation. Where post-concussional terminology is used clinically, the clinician should identify its definition and document the individual disorders and functional limitations. Second, neurocognitive classification does not resolve every diagnostic question. Clark and colleagues argue that a multidimensional formulation is needed because an umbrella label can obscure contributors to persisting symptoms [10]. Third, a patient can have disabling non-cognitive sequelae without meeting any neurocognitive diagnosis. A person whose principal difficulties are vestibular, oculomotor, headache-related, autonomic, sleep-related, or pain-related may require several diagnoses even when formal cognition is preserved. Auxéméry and Gayraud criticized the removal of post-concussion syndrome and argued for multidisciplinary recognition of its clinical reality [14]. Iverson emphasized that the DSM-5 neurocognitive category is based on cognitive decline rather than a post-concussion symptom checklist [15]. The gap is the absence of a standalone category that describes the multidomain presentation. Other diagnoses and codes remain available. How many patients fall into that gap cannot be estimated from the cited evidence.

Clinical use of the terminology has continued despite its withdrawal from the manuals. The terms have evolved, from post-concussion syndrome to persistent post-concussion symptoms to persisting symptoms after concussion, and the entity has continued to be recognized, treated, and studied under each name. ICD-10 also remains in clinical use in much of the world as jurisdictions move through the transition at different rates.

A body of work frames persisting symptoms in biopsychosocial terms, emphasizing pre-injury, psychological, social, and environmental contributors [10]. Attributing every

continuing complaint exclusively to concussion is not defensible, and treatment should address modifiable maintaining factors. None of that excludes an initiating or contributing role for the injury. In an individual case, that role must be supported by the chronology, the injury evidence, premorbid function, and the competing explanations. An integrated formulation can describe injury-related and non-injury-related contributors without assuming that one invalidates the other.

A proposed operational approach

The approach proposed here draws on established criteria and Dr. Pappolla's clinical judgment. The distinction is stated for each element.

Established mild traumatic brain injury

The diagnosis presupposes an established injury. The 2023 American Congress of Rehabilitation Medicine criteria, set out in full in Chapter 3, require a plausible mechanism, evidence of acute brain dysfunction through one of three pathways, and consideration of confounding explanations [16]. Establishing the injury does not require that a diagnostic label was entered in the initial record, but it does require evidence sufficient to satisfy the relevant criteria.

Patients referred for persistent symptoms often undergo their first focused brain injury assessment weeks or months after the event, and many acute records lack a concussion diagnosis. In a prospective emergency department series, 56% of patients identified by study personnel as having mTBI had no corresponding diagnosis documented in the emergency record [17]. Competing injuries, medication effects, intoxication, and incomplete symptom elicitation all get in the way. In a level I trauma center series of 3042 patients exposed to a possible mTBI mechanism, 65.1% had at least one complicating factor; emergency department narcotic administration was the most frequent, affecting 43.7% [18]. These studies justify caution about a missing label. They do not establish that every undocumented concussion occurred; the significance of absent documentation depends on what was actually assessed, recorded, and potentially confounded.

Retrospective assessment is an accepted way to evaluate a suspected prior mTBI. Walker and colleagues described the clinical interview as the existing gold standard in this setting [19]. Structured approaches to lifetime TBI identification have evidence of reliability and validity [20]. The ACRM framework also allows historical information to establish the relevant injury features, with attention to recall bias, response bias, and confounding explanations [16]. Contemporaneous observations, records, collateral

accounts, and the patient's report should be compared rather than treated as interchangeable; agreement among independent sources increases confidence, and gaps should be stated. Chapter 5 describes this reconstruction in detail.

Retrospective diagnosis reconstructs the injury: its mechanism, acute signs, and acute symptoms, with exclusion of alternatives. The evidence must concern the injury itself; present symptoms alone cannot establish it. A patient whose record shows a high-energy collision, whose collateral history describes confusion at the scene, whose own account places headache and dizziness within hours, and in whom no other condition accounts for the picture, has an injury established by evidence about the injury. A patient whose only evidence is that headache and poor concentration are present now has not, because those symptoms are nonspecific and their presence at three months does not by itself locate an injury at time zero.

Temporal onset, persistence, functional consequence, and exclusion

The proposed approach gives particular weight to acute onset, persistence, functional consequence, and differential diagnosis. The Lagacé-Legendre definition requires symptom onset within hours and daily persistence at 3 months [8]. The ACRM framework uses an acute window, generally less than 72 hours for qualifying symptoms, to help establish the initial injury [16]. These serve related but different purposes. The Amsterdam consensus uses more than 4 weeks as a threshold for persisting symptoms and recommends multimodal assessment [9]. This chapter uses 3 months for the provisional syndrome framework, a convention rather than a proven boundary. Symptoms must affect functioning, and pre-existing conditions, new disorders, and interacting contributors must be considered.

Symptom count

In Dr. Pappolla's outpatient neurology clinic, 4 Rivermead symptoms have generally served as a prompt to consider the syndrome. This is a local clinical convention with unknown sensitivity and specificity, not a validated threshold, and raising the count cannot be assumed to improve accuracy when so many of the symptoms are nonspecific. The Lagacé-Legendre consensus adopted no mandatory minimum [8]. A 3-symptom convention has been used in ICD-10 and DSM-IV operationalizations [7]. Ingebrigtsen and colleagues applied a 3-symptom definition in a consecutive minor-head-injury cohort: at 3 months, 62% reported at least 1 symptom and 40% met their syndrome definition [21]. That approach used the full 16-item Rivermead inventory. The RPQ-3 and RPQ-13 distinction derives from psychometric analysis and does not itself

establish a diagnostic threshold [22]. Any count should be reported together with symptom severity, functional impact, the injury evidence, and the differential diagnosis.

One or two manifestations may suffice when severe enough to produce significant functional impairment. Symptom count stands in for burden; where burden can be assessed directly, it should guide the judgment.

Causation

Characterizing the syndrome and attributing it to a particular injury are related but distinct judgments. This chapter proposes a 3-part clinical reasoning framework adapted from a causation discussion concerning symptomatic spinal disk injury after motor vehicle trauma [23]: a compatible temporal relationship; a plausible injury mechanism and supporting evidence of brain injury; and assessment of competing or interacting explanations. It is a structured reasoning aid, not a validated diagnostic algorithm or a legal standard. Each part should be documented and the remaining uncertainty made explicit.

The temporal question concerns both onset and subsequent course. A complaint first appearing months after injury raises a different question from one present during the initial days. Genuine delayed onset requires a plausible explanation for the interval. Delayed recognition is also possible: a difficulty may become apparent only when a patient resumes a demanding activity. Neither should be assumed from the date of the first recorded complaint; detailed history and corroborating evidence should distinguish the two, symptom by symptom, acknowledging uncertainty when the early course cannot be reconstructed.

The mechanism must be capable of transmitting mechanical energy to the brain [16]. Direct head impact is not required, but the occurrence of a collision alone does not establish brain injury. Vehicle damage, airbag deployment, and other crash descriptors are not patient-specific measurements of brain loading, and no single mechanical threshold determines whether an individual sustained an mTBI. Mechanism informs the probability of injury and must be integrated with the acute clinical evidence and the confounders rather than used to bypass them.

The third step extends the differential-diagnostic reasoning required when establishing the initial injury [16]. At later assessment, additional contributors may include medication-overuse headache, deconditioning, disordered sleep, mood disturbance, and chronic pain. Coexistence does not exclude an injury contribution, but neither does a preceding injury make every later symptom its consequence. The clinician should compare the course with premorbid function and explain how alternatives were

assessed. The most defensible formulation identifies supported initiating factors, maintaining factors, comorbid disorders, and unresolved uncertainty separately.

The clinical syndrome: cognitive and non-cognitive consequences

Symptom lists are the conventional way to describe the syndrome. For assessment, it is more useful to organize the manifestations into cognitive and non-cognitive impairment, then to consider the non-cognitive systems separately. These distinctions map to different anatomy, assessment methods, and treatments. They also organize the two sections that follow.

Cognitive impairment after mTBI may affect attention, memory, executive function, or processing speed in different combinations. Conventional testing provides standardized information about the domains and conditions it samples, but it may not reproduce the demands of daily life, and selected dual-task studies have detected difficulties under added load despite relatively preserved conventional performance [24,25,26]. A normal bedside screen, including an MMSE score of 30, does not exclude mTBI, persisting symptoms, or subtle domain-specific impairment; it remains informative about the functions it sampled.

Non-cognitive manifestations may be highly disabling. Post-traumatic headache often has a migrainous phenotype, and medication overuse can complicate its course. Dizziness may reflect peripheral or central vestibular dysfunction, migraine, cervical contributions, autonomic disturbances, or more than one process. Convergence, accommodation, pursuit, or saccadic abnormalities can contribute to reading and screen intolerance despite preserved visual acuity [27]. Orthostatic or exercise intolerance warrants consideration of autonomic dysfunction, alongside medication effects, cardiovascular causes, deconditioning, and anxiety. Sleep disturbance amplifies everything else. Difficulty understanding speech in noise merits auditory assessment even when pure-tone thresholds are normal. Mood and behavioral changes may be related to the injury, its consequences, pre-existing vulnerability, or interacting factors [2,9,10].

Chronic pain deserves specific attention because its mechanisms are often mixed; Chapter 12 is devoted to it. Persistent cervical, shoulder, and back pain can follow the same event that produces concussion. Nociceptive pain reflects activation of nociceptors by tissue injury or threat; neuropathic pain requires a disease or lesion of the somatosensory system; nociplastic pain describes altered nociception when nociceptive or neuropathic pathology does not adequately explain the pain [28]. Our research group studied 67 selected patients with PPCS and chronic pain in a

retrospective clinic cohort and found prominent widespread pain and symptom-severity features interpreted as evidence of central sensitization [29]. Those are self-reported measures, so they support altered pain processing without establishing sensitization at the neuronal level. Treatment should address demonstrable peripheral contributors as well as altered central processing when the clinical assessment supports it.

Two patients may carry the same diagnostic label while having substantially different limitations. A research technician may struggle with cognitive slowing and reduced attentional endurance, whereas a roofer may have preserved cognition but disabling imbalance and pain during rapid head movement. Cognitive testing may characterize an important component of the first patient's presentation while leaving the second patient's principal impairments outside its measurement scope. A comprehensive neuropsychological evaluation can still recognize those limitations through history and functional assessment; the restriction applies to the test scores, not to the entire evaluation.

The interaction between cognitive and non-cognitive impairment

The cognitive and non-cognitive distinction is useful for organizing assessment but should not imply that the domains operate independently. They interact through shared networks, sensory input, compensatory effort, pain, arousal, and attention. An abnormal cognitive score may therefore have several contributors, and treatment of a non-cognitive disorder may improve cognitive performance. Whether the combined burden is additive or greater than additive must be demonstrated rather than assumed.

Oculomotor control and cognition provide the clearest example: they share anatomy. Saccades, smooth pursuit, fixation stability, and gaze holding depend on frontal eye fields, dorsolateral prefrontal cortex, posterior parietal cortex, superior colliculus, brainstem, and cerebellum [30]. Those same frontoparietal networks subserve attention, working memory, and inhibitory control. In the antisaccade task, a subject must suppress a reflexive glance toward a target and look away from it. The task measures both eye movement and inhibitory control, and is used for both purposes.

Visual information is acquired through coordinated eye movements, so impaired fixation, vergence, accommodation, or saccadic control can complicate any visual task. Poor visual-memory performance may reflect impaired encoding, deficient visual acquisition, or both; a score alone cannot distinguish them. Reading requires accurate saccades, stable fixation, and sustained near alignment, and abnormalities in these functions contribute to reading intolerance and loss of place [27]. Faulkner and colleagues analyzed Rivermead symptom networks in 2 cohorts, one assessed about 6

weeks after injury and one 10 years after injury. Blurred vision, frustration, and concentration difficulty were central in both networks [31]. This association among self-reported symptoms does not prove that an oculomotor deficit caused impaired cognition, but it is the same pairing the anatomy predicts.

Postural control and movement impose attentional demands of their own, measurable as dual-task costs [24]. Pain, poor sleep, mood symptoms, auditory processing difficulty, and sensory overload all draw on the same attentional resources. The pain study described above found correlations between pain-related and non-painful symptoms [29]. Several explanations are possible: shared mechanisms, reciprocal effects, or common reporting influences. A cognitive test score therefore measures performance without uniquely identifying its mechanism. Poor sustained attention may be driven by pain, sleep, medication, mood, sensory difficulty, or primary attentional dysfunction, and telling these apart requires the test pattern, the history, the examination, and the relevant additional assessments together. Treating a documented visual, vestibular, headache, sleep, or pain disorder may improve overall function and sometimes cognition; the size of the benefit should be measured rather than presumed.

The limits of cognitive testing considered alone

A recurring diagnostic error is to draw a conclusion broader than the cognitive testing can support. The limits need to be stated precisely.

Persistent post-concussion syndrome has cognitive and non-cognitive consequences, and the non-cognitive systems are assessed by methods entirely distinct from those used to assess cognition. Two limits follow. When an evaluation confined to one system is negative, it cannot exclude the syndrome, which may reside entirely in what was not assessed. When it is positive, the finding may be attributed to the injury if the causation analysis above is satisfied. Its mechanism cannot be established from that evaluation alone, however: the systems share substrates, and a deficit measured in one may originate in another. A single-system evaluation may therefore miss what it did not assess and misattribute the mechanism of what it did.

The error most often encountered takes this form. A neuropsychological evaluation is performed, cognitive testing is within normal limits or mildly abnormal, and the conclusion drawn is that the patient does not have a TBI, or does not have post-concussion syndrome, or has no residual injury-related impairment. The first two conclusions do not follow from the data. The third does not either, unless expressly limited to cognition.

The issue is scope and integration. Neurologists assess cognition and can use cognitive and neuropsychological methods within their training and competence as part of the neurological evaluation. Clinical neuropsychologists contribute additional specialized depth in psychometrics, normative interpretation, and performance and symptom validity. Validity findings require clinical context in both directions: a failed measure does not, by itself, establish intentional deception, and a passed one does not settle the cause of the symptoms reported. We and our colleagues have discussed elsewhere the distinction between cognitive testing and comprehensive assessment, and the need to integrate cognitive results with neurological findings [32]. Bigler and colleagues argue in an opinion-review series that conventional neuropsychological measures, used alone, cannot characterize the full neuropathological and functional complexity of mTBI [33,34]; these are expert critiques rather than validation studies. Specialized vestibular, oculomotor, headache, pain, autonomic, and audiological assessments require the appropriate clinical expertise and cannot be replaced by cognitive scores. Training and scope vary among individuals, so the limitation should be stated in terms of what was actually assessed rather than as a claim about a profession.

Consider a patient with disabling migrainous headache, abnormal smooth pursuit or optokinetic responses, convergence insufficiency, orthostatic intolerance, and cervical pain, whose cognitive testing is normal. If the injury history, chronology, technical validity, and differential diagnosis support attribution, that patient may have a disabling post-concussional presentation despite preserved tested cognition. A conclusion that no residual disorder exists, based solely on the cognitive scores, would exceed the scope of those measurements.

Three qualifications are necessary. First, the error concerns the scope of the conclusion, not the profession of the examiner. A neurologist who declares a patient cognitively intact on a bedside screen, without formal testing or validity measures, has made the same error in the opposite direction. Second, a properly framed neuropsychological conclusion is legitimate and valuable. A finding of no impairment in the cognitive domains assessed, with valid performance, is true and useful. The difficulty arises only when that domain-limited finding becomes a global conclusion. Third, objectively abnormal vestibular or oculomotor findings do not establish cognitive impairment.

A second limitation concerns the demands sampled. Most standardized neuropsychological tasks are administered one at a time, in a quiet room, by an examiner who waits for the answer. Patients describe failing somewhere else: tracking a conversation in a noisy restaurant, reading in a visually busy setting, completing a task

while monitoring competing demands. This is the “couch and stairs” distinction introduced earlier and illustrated in Chapter 11. When a battery does not sample the conditions under which the patient fails, normal performance cannot settle that question, and selected dual-task studies show that added load reveals deficits the resting battery did not. That does not mean every normal battery conceals an unmeasured deficit. The appropriate response is to assess the difficulty under load with a reliable method, not to assume an abnormality whenever the resting result is normal.

The supporting studies differ in population and design. McDowell and colleagues demonstrated disproportionate dual-task difficulty in 25 people recovering from severe TBI [35], which supports the principle without being mTBI evidence. Cicerone found that patients with mTBI differed from controls under added dual-task demands [25]. Cox and Fernandes reported a larger divided-attention cost for associative memory in young adults with remote mTBI, with associations between fatigue and performance [26]. Gagné and colleagues found greater dual-task gait-speed cost in a small mTBI sample despite no group differences in symptom or conventional neuropsychological measures [24]. Sinopoli and colleagues reported altered task-related activation in young male athletes with mTBI during a dual-task imaging experiment [36]. Conventional scores also predict function imperfectly. In the study by Jeffay and colleagues, neither the Behavioural Assessment of Dysexecutive Syndrome nor conventional measures differentiated employment status after correction for multiple testing [37]. In a cohort of 1554 patients across mild to severe TBI, Wilson and colleagues found that cognition related differently to function at different outcome levels, with weaker discrimination at higher levels of recovery [38]. The diagnostic performance of particular dual-task protocols remains to be established.

A valid normal cognitive profile is evidence of preserved performance in the domains and conditions tested, and nothing more. Conversely, a suspected difficulty under load should be demonstrated through appropriate evaluation rather than inferred from the limitations of conventional testing. The practical implication is that a complete evaluation addresses cognition, headache, vestibular and oculomotor function, pain including its central mechanisms, autonomic regulation, sleep, hearing, and mood, and that such an evaluation requires more than one discipline.

Implications for assessment and management

The Rivermead questionnaire documents reported symptom burden and change but does not identify the underlying mechanism [22]. An identical dizziness rating may accompany peripheral vestibular disease, migraine, cervical factors, autonomic symptoms, or overlapping conditions. The history and examination should guide the

selection of vestibular and oculomotor assessment, balance testing, autonomic evaluation, or sleep investigation. These are targeted assessments, not a universal battery. When cognitive concerns warrant neuropsychological assessment with appropriate validity measures, they complement it. Chapter 7 describes what each of these measurements shows and how it is read.

Management should follow the individual clinical profile. Headache treatment is guided by phenotype and medication use. Documented vestibular or cervical dysfunction may warrant targeted rehabilitation, and visual complaints require assessment of refraction, ocular disease, accommodation, vergence, and other relevant functions before treatment is selected [9,27]. Cognitive rehabilitation and graded resumption of activity may be appropriate for demonstrated functional difficulties. Pain management should address the supported nociceptive, neuropathic, and nociplastic components without assuming that a normal scan proves a central mechanism [28,29]. Individually prescribed aerobic exercise can form part of concussion rehabilitation, but orthostatic or exercise intolerance also requires assessment of its cause and contraindications. Sleep and mood disorders should be treated directly. Evidence strength varies by intervention and population, and improvement in one domain should not be assumed to normalize another.

Conclusion

Persisting symptoms and disability after concussion are well documented, but the boundaries and mechanisms of the syndrome remain debated. Definitions differ in symptom count, duration, functional requirements, and the necessity of objective cognitive impairment. Applied to the same patients, they produce prevalence estimates differing more than threefold. DSM-5 and ICD-11 no longer retain the earlier standalone postconcussional categories, while their neurocognitive categories require cognitive decline. This leaves patients whose main difficulties are non-cognitive without a label that describes them. They may still have other valid diagnoses, and the classification does not establish the cause of their symptoms.

The provisional clinical framework proposed here begins with adequate acute or retrospective evidence of the injury. It then characterizes symptom timing and functional consequences, evaluates alternatives, and distinguishes attribution from description. The framework and the 4-symptom convention used in Dr. Pappolla's outpatient neurology clinic have not been validated and should not be treated as established diagnostic standards. Cognitive and non-cognitive systems interact, so no isolated score or ancillary abnormality should substitute for clinical integration. A normal result remains informative within its scope but does not exclude a prior

concussion or impairments that the test did not assess. Where difficulties arise under load, their presence and functional significance should be evaluated with appropriate methods, and multidisciplinary assessment should be matched to the patient's clinical questions.

Fast take: main points from chapter 10

• Persisting symptoms after mild TBI are well documented, but published estimates measure different things: about half of patients were classified as cognitively impaired by one scoping review's method [1], while persisting symptoms are estimated at 10 to 25% by another [2]. The figures are not interchangeable.

• The definitions disagree. Physicians surveyed could not agree on the minimum duration [4], and applying different classification methods to one cohort of 731 patients produced prevalence estimates from 11.4% to 38.7% [5]. Any statement that a patient meets criteria should say which criteria.

• DSM-IV made objective cognitive impairment mandatory; the Lagacé-Legendre consensus requires one daily symptom at 3 months with functional impact and no cognitive testing; the Amsterdam statement uses more than four weeks as a management threshold [7,8,9].

• DSM-5 and ICD-11 dropped the standalone postconcussional categories. Their neurocognitive disorders require cognitive decline and do not describe the patient whose disability is vestibular, visual, headache-related, autonomic, or pain-related [10,11,12,13]. Removing a label from a manual does not establish recovery.

• The syndrome presupposes an established injury under the 2023 ACRM criteria, which may be reconstructed retrospectively; an injury is established by evidence about the injury, not by the persistence of nonspecific symptoms now [16,17,19,20].

• Attribution to a particular injury is a separate judgment with three parts: a compatible temporal relationship, a plausible mechanism with supporting evidence of brain injury, and assessment of competing or interacting explanations [23].

• Cognitive and non-cognitive systems share anatomy and interact. Oculomotor control and attention run on the same frontoparietal networks, so a cognitive score does not by itself identify its mechanism [30,31].

• A normal cognitive evaluation establishes preserved performance in the domains and conditions tested. It does not exclude the syndrome, which may reside entirely in systems that were not assessed, and it may not sample the divided-attention demands

under which some patients report failing; a suspected deficit under load is demonstrated with an appropriate method, not presumed [24,25,26,35].

• Pain after mild TBI is often nociplastic; in our series of 67 patients, widespread pain and symptom severity pointed to central sensitization [29]. Chapter 12 takes this up.

• Evaluation should be multidisciplinary and matched to the patient's complaints, and management should follow the individual profile rather than a universal battery or a single label.

References

1. McInnes K, Friesen CL, MacKenzie DE, Westwood DA, Boe SG. Mild traumatic brain injury (mTBI) and chronic cognitive impairment: a scoping review. PLoS One. 2017;12:e0174847. doi:10.1371/journal.pone.0174847.
2. Polinder S, Cnossen MC, Real RGL, Covic A, Gorbunova A, Voormolen DC, et al. A multidimensional approach to post-concussion symptoms in mild traumatic brain injury. Front Neurol. 2018;9:1113. doi:10.3389/fneur.2018.01113.
3. Iverson GL, Karr JE, Gardner AJ, Silverberg ND, Terry DP. Results of scoping review do not support mild traumatic brain injury being associated with a high incidence of chronic cognitive impairment: commentary on McInnes et al. 2017. PLoS One. 2019;14:e0218997. doi:10.1371/journal.pone.0218997.
4. Rose SC, Fischer AN, Heyer GL. How long is too long? The lack of consensus regarding the post-concussion syndrome diagnosis. Brain Inj. 2015;29:798-803. doi:10.3109/02699052.2015.1004756.
5. Voormolen DC, Cnossen MC, Polinder S, von Steinbuechel N, Vos PE, Haagsma JA. Divergent classification methods of post-concussion syndrome after mild traumatic brain injury: prevalence rates, risk factors, and functional outcome. J Neurotrauma. 2018;35:1233-1241. doi:10.1089/neu.2017.5257.
6. World Health Organization. ICD-10 Version 2019. F07.2 Postconcussional syndrome. Available at: https://icd.who.int/browse10/2019/en/GetConcept?ConceptId=F00-F09 (accessed 24 October 2026).
7. Boake C, McCauley SR, Levin HS, Pedroza C, Contant CF, Song JX, et al. Diagnostic criteria for postconcussional syndrome after mild to moderate traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2005;17:350-356. doi:10.1176/jnp.17.3.350.
8. Lagacé-Legendre C, Boucher V, Robert S, Tardif PA, Ouellet MC, de Guise E, et al. Persistent postconcussion symptoms: an expert consensus-based definition using the Delphi method. J Head Trauma Rehabil. 2021;36:96-102. doi:10.1097/HTR.0000000000000613.

9. Patricios JS, Schneider KJ, Dvorak J, Ahmed OH, Blauwet C, Cantu RC, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport, Amsterdam, October 2022. Br J Sports Med. 2023;57:695-711. doi:10.1136/bjsports-2023-106898.
10. Clark CN, Edwards MJ, Ong BE, Goodliffe L, Ahmad H, Dilley MD, et al. Reframing postconcussional syndrome as an interface disorder of neurology, psychiatry and psychology. Brain. 2022;145:1906-1915. doi:10.1093/brain/awac149.
11. World Health Organization. Clinical descriptions and diagnostic requirements for ICD-11 mental, behavioural and neurodevelopmental disorders. Geneva: World Health Organization; 2024. Section 6D71, Mild neurocognitive disorder. Available at: https://iris.who.int/server/api/core/bitstreams/dcf73611-9e0f-4d5c-85f6-85a58b0da6de/content (accessed 24 October 2026).
12. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013.
13. Wortzel HS, Arciniegas DB. The DSM-5 approach to the evaluation of traumatic brain injury and its neuropsychiatric sequelae. NeuroRehabilitation. 2014;34:613-623. doi:10.3233/NRE-141086.
14. Auxéméry Y, Gayraud F. Post-concussion syndrome: still forgotten? From the disappearance of the DSM-5 to a multidisciplinary consultation. Encephale. 2021;47:491-494. doi:10.1016/j.encep.2020.06.017.
15. Iverson GL. Network analysis and precision rehabilitation for the post-concussion syndrome. Front Neurol. 2019;10:489. doi:10.3389/fneur.2019.00489.
16. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104:1343-1355. doi:10.1016/j.apmr.2023.03.036.
17. Powell JM, Ferraro JV, Dikmen SS, Temkin NR, Bell KR. Accuracy of mild traumatic brain injury diagnosis. Arch Phys Med Rehabil. 2008;89:1550-1555. doi:10.1016/j.apmr.2007.12.035.
18. Furger RE, Nelson LD, Lerner EB, McCrea MA. Frequency of factors that complicate the identification of mild traumatic brain injury in level I trauma center patients. Concussion. 2016;1:CNC11. doi:10.2217/cnc.15.11.
19. Walker WC, Cifu DX, Hudak AM, Goldberg G, Kunz RD, Sima AP. Structured interview for mild traumatic brain injury after military blast: inter-rater agreement and development of diagnostic algorithm. J Neurotrauma. 2015;32:464-473. doi:10.1089/neu.2014.3433.

20. Corrigan JD, Bogner J. Initial reliability and validity of the Ohio State University TBI Identification Method. J Head Trauma Rehabil. 2007;22:318-329. doi:10.1097/01.HTR.0000300227.67748.77.
21. Ingebrigtsen T, Waterloo K, Marup-Jensen S, Attner E, Romner B. Quantification of post-concussion symptoms 3 months after minor head injury in 100 consecutive patients. J Neurol. 1998;245:609-612. doi:10.1007/s004150050254.
22. Eyres S, Carey A, Gilworth G, Neumann V, Tennant A. Construct validity and reliability of the Rivermead Post-Concussion Symptoms Questionnaire. Clin Rehabil. 2005;19:878-887. doi:10.1191/0269215505cr905oa.
23. Freeman MD, Centeno CJ, Kohles SS. A systematic approach to clinical determinations of causation in symptomatic spinal disk injury following motor vehicle crash trauma. PM R. 2009;1:951-956. doi:10.1016/j.pmrj.2009.07.009.
24. Gagné MÈ, McFadyen BJ, Ouellet MC. Performance during dual-task walking in a corridor after mild traumatic brain injury: a potential functional marker to assist return-to-function decisions. Brain Inj. 2021;35:173-179. doi:10.1080/02699052.2020.1863467.
25. Cicerone KD. Attention deficits and dual task demands after mild traumatic brain injury. Brain Inj. 1996;10:79-89. doi:10.1080/026990596124566.
26. Cox AW, Fernandes MA. Long-term cognitive and affective consequences of mild traumatic brain injury: comparison with older adults. Brain Inj. 2024;38:1133-1146. doi:10.1080/02699052.2024.2376769.
27. Ciuffreda KJ, Ludlam DP, Yadav NK, Thiagarajan P. Traumatic brain injury: visual consequences, diagnosis, and treatment. Adv Ophthalmol Optom. 2016;1:307-333. doi:10.1016/j.yaoo.2016.03.013.
28. International Association for the Study of Pain. Terminology. Nociplastic pain, neuropathic pain, nociceptive pain, and central sensitization. Available at: https://www.iasp-pain.org/resources/terminology/ (accessed 24 October 2026).
29. File C, Nader R, Villasante-Tezanos A, Ahmed R, Pappolla S, Ahmed F, et al. Pain management in mild traumatic brain injury: central sensitization as a multispecialty challenge. Pain Physician. 2025;28:231-240.
30. Leigh RJ, Zee DS. The neurology of eye movements. 5th ed. New York: Oxford University Press; 2015.
31. Faulkner JW, Theadom A, Snell DL, Williams MN. Network analysis applied to post-concussion symptoms in two mild traumatic brain injury samples. Front Neurol. 2023;14:1226367. doi:10.3389/fneur.2023.1226367.
32. Pappolla MA, Pappolla SL, Nader R, Hamza MK, Fang F, Fang X. Evaluating cognition across aging and traumatic brain injury: integrating neurological and neuropsychological approaches. J Clin Med. 2026;15:3822. doi:10.3390/jcm15103822.

33. Bigler ED, Allder S, Dunkley BT, Victoroff J. What traditional neuropsychological assessment got wrong about mild traumatic brain injury. I: historical perspective, contemporary neuroimaging overview and neuropathology update. Brain Inj. 2025;39:1161-1183. doi:10.1080/02699052.2025.2551162.
34. Bigler ED, Allder S, Victoroff J. What traditional neuropsychological assessment got wrong about mild traumatic brain injury. II: limitations in test development, research design, statistical and psychometric issues. Brain Inj. 2024;38:1053-1074. doi:10.1080/02699052.2024.2376261.
35. McDowell S, Whyte J, D'Esposito M. Working memory impairments in traumatic brain injury: evidence from a dual-task paradigm. Neuropsychologia. 1997;35:1341-1353. doi:10.1016/S0028-3932(97)00082-1.
36. Sinopoli KJ, Chen JK, Wells G, Fait P, Ptito A, Taha T, et al. Imaging "brain strain" in youth athletes with mild traumatic brain injury during dual-task performance. J Neurotrauma. 2014;31:1843-1859. doi:10.1089/neu.2014.3326.
37. Jeffay E, Ramani S, Zakzanis KK. On the ecological validity of the behavioural assessment of dysexecutive syndrome measure in mild traumatic brain injury. Brain Inj. 2025;39:1283-1294. doi:10.1080/02699052.2025.2554248.
38. Wilson L, Horton L, Kunzmann K, Sahakian BJ, Newcombe VFJ, Stamatakis EA, et al. Understanding the relationship between cognitive performance and function in daily life after traumatic brain injury. J Neurol Neurosurg Psychiatry. 2021;92:407-417. doi:10.1136/jnnp-2020-324492.

Chapter 11 Cognitive and non-cognitive consequences of traumatic brain injury

Chapter overview

The consequences of a brain injury that matter most to patients and their families are rarely the ones that can be seen. Years after an injury, the wounds have healed and the scans have long been read as normal. Yet the person cannot hold attention in a noisy room, loses the thread of a conversation, forgets what was agreed yesterday, flares at small frustrations, and no longer starts anything without being prompted. The person may not fully recognize that any of this has changed. This chapter describes those consequences, from the cognitive deficits sampled by standardized tests to changes in mood, motivation, and personality that require clinical interviews, behavioral assessment, and an account of life outside the testing room.

The chapter has three sections. The first describes the cognitive domains: attention, processing speed, memory, executive function, language, and social cognition. It explains what injury does to each, how deficits are measured, and why standard measures do not always capture the problems that matter most in daily life. The second, the longer one, covers the non-cognitive consequences that have no chapter of their own: depression, anxiety, post-traumatic stress disorder, disorders of motivation, disorders of awareness including anosognosia, personality change, irritability and aggression, acquired attention deficit, sleep disturbance and fatigue, psychosis, and the risk of suicide. For each, the chapter describes the injured brain systems, the frequency and features of the condition, how it differs from similar conditions, and its effect on the patient's function. The third explains how to evaluate these consequences when the findings will be contested and answers the questions most often raised about them.

Why these consequences matter most

A reader coming to this chapter from the earlier ones might expect the physical injuries to dominate the long-term picture. Cognitive and behavioral changes can be just as consequential. In a CENTER-TBI study of 1,554 people assessed six months after injuries ranging from mild to severe, cognitive performance was related to substantial disability, while mental health differences remained important among those with better overall recovery [39]. Patients in this book can walk, speak, and drive; what they cannot do is sustain attention under load, remember reliably, regulate their emotions as they once did, or, in some cases, recognize that they have changed. These deficits can end careers and marriages.

These deficits are also the ones most often disputed. They are invisible, depend in part on the patient's own report, and overlap with symptoms of depression, anxiety, stress, and pain. They can be present even when scans and a brief examination are normal. None of that makes them less real. It makes them harder to evaluate, and it is why the evaluation has to be done with discipline.

Cognitive consequences

What cognition is

Cognition is the collective name for the mental operations by which a person takes in information, holds it, works with it, and acts on it. Neuropsychology divides it into domains, each served by a recognizable set of brain regions and the wiring between them (Figure 11.1), and each measured by its own tests.

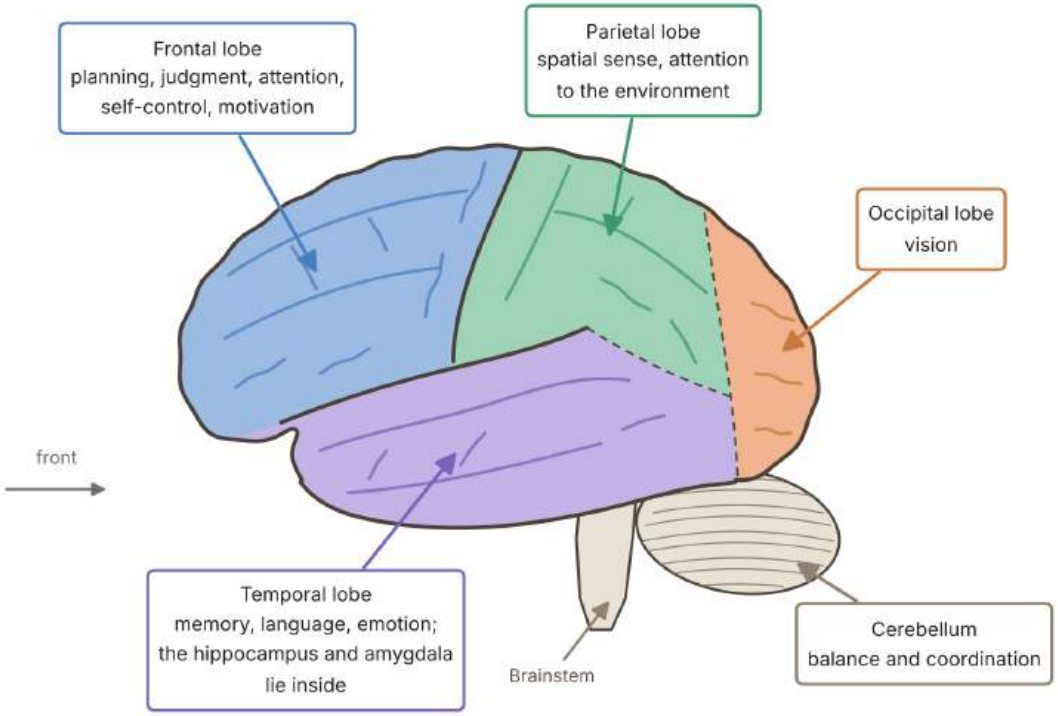


Figure 11.1. The main cognitive domains and some of the brain regions that support them, seen from the left side with the front of the brain to the left. Frontal systems contribute to planning, judgment, attention, self-control, and motivation. Temporal regions contribute to memory and language; the hippocampus and amygdala help link memory and emotion. Parietal regions contribute to spatial processing and attention, and occipital regions to vision. These abilities depend on interacting networks rather than isolated compartments. White matter connects their components and is vulnerable to traumatic stretching. Original schematic.

Attention is the ability to select what matters and hold it in mind against distraction; its components, selective, sustained, and divided attention and the speed of processing that underlies them, can fail separately. Memory is the ability to take in new information, store it, and retrieve it, from the brief holding of a telephone number to the lasting record of an event. Executive function is the collective name for the abilities that direct the others: setting a goal, planning the steps, starting, sequencing, inhibiting the wrong response, monitoring performance, and changing course when the plan fails. Language covers understanding and producing words and their social use in conversation. Visuospatial function is the sense of where things are in space. Social cognition is the ability to read other people's intentions and emotions and to behave appropriately with them.

Injury affects these domains unevenly. Frontal and anterior temporal contusions and damage to connecting white matter can disrupt several abilities at once, but there is no single cognitive profile shared by every patient. In TRACK-TBI, six-month testing showed more frequent impairment after moderate or severe injury; processing speed was especially affected in that group, whereas mild-injury profiles were more varied across memory, speed, and executive measures [1]. Basic language and perception may remain useful strengths even when these other abilities are compromised.

Attention and speed of processing

Slowed processing is an important cognitive consequence of brain injury, particularly after moderate or severe injury [1,27]. The patient takes longer to react, understand, decide, and answer. The delay may grow as the task becomes more complex. A person who manages a simple reaction task may struggle when choosing among several responses or keeping two things going at once. Patients describe being unable to keep up with conversations, losing their train of thought, being unable to follow a meeting, or being exhausted after an hour of mental work that used to take no effort. Damage to the networks and connections described in Chapter 2 can contribute to this slowing. Evaluation also considers hearing, vision, sleep, pain, mood, fatigue, and medication, because these can add to the attentional burden [27].

Divided attention is the ability to manage competing demands. It matters when a patient must follow a conversation while navigating a noisy room, or carry out a familiar task while answering questions. Some deficits become clearer when two tasks are combined: in Cicerone's mild-TBI study, patients differed from controls under dual-task conditions although the simpler conditions did not distinguish them [31]. Chapter 10 sets out this argument and the dual-task evidence in detail. Figure 11.2 shows the analogy we use with non-medical audiences, a patient with chronic lung disease who is

normal on the couch and short of breath on the stairs. Standard cognitive tests can themselves be demanding; the question is whether their combination, duration, and setting reproduce the demands under which this particular patient struggles.

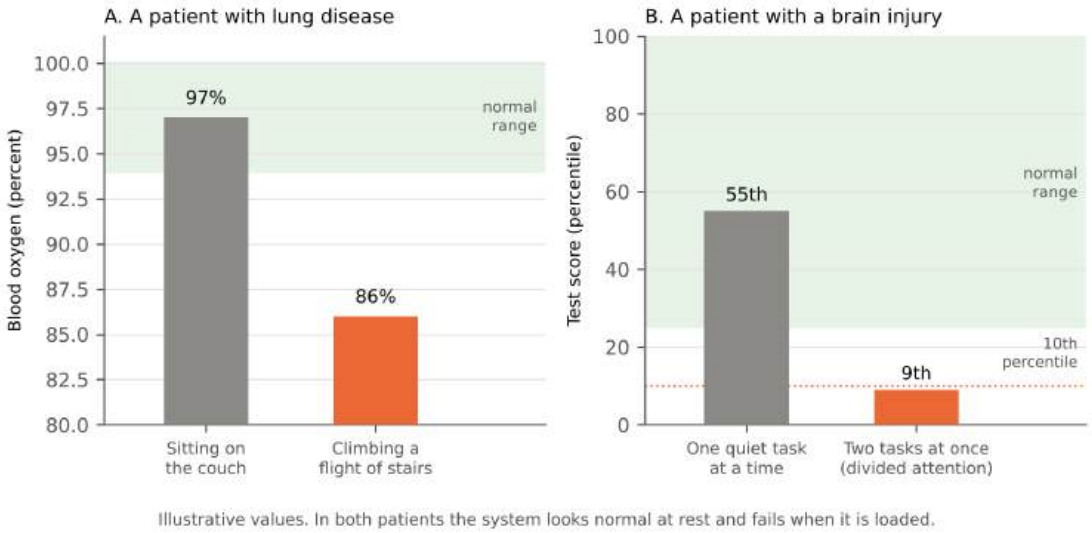


Figure 11.2. At rest and under load. (A) A patient with chronic lung disease has normal blood oxygen sitting on the couch and low oxygen climbing a flight of stairs; the lungs are the same, the demand is not. (B) A patient with a brain injury may perform within the expected range on one cognitive task and have greater difficulty when two tasks are combined. This is an analogy about demand, not a claim that cognitive testing is physiologically at rest. The values, shading, and dotted line are hypothetical illustrations, not a diagnostic cutoff or a pattern expected in every brain injury. Original schematic.

Memory

Memory complaints have several parts that can be affected differently. Anterograde memory concerns the formation of new memories; retrograde memory concerns events learned before the injury. A gap around the event may narrow during recovery, although some memories never return. Memory for events and experiences is episodic memory; remembering facts and retaining learned skills are different capacities. A patient may forget what happened at yesterday's meeting while remembering how to do the job. Working memory holds information in mind while it is being used, as when keeping a telephone number in mind while dialing or following the steps of a recipe. It is closely tied to attention. Prospective memory is remembering to carry out an intention later, such as keeping an appointment or taking medication. Contemporary memory rehabilitation addresses both episodic and prospective difficulties and their effects on daily life [28].

Immediately after an injury, the period during which the patient cannot form continuous new memories is called post-traumatic amnesia, and its duration is one of the measures of severity set out in Chapter 4. After the acute period, memory problems can remain consequential, especially after moderate or severe injury [28]. Many people with mild injury improve, but a normal memory score and a continuing complaint need to be understood together. The score describes what the person achieved in that task. The history describes reliability across the day, the effort involved, and what happens amid distraction, fatigue, or several competing demands. A large prospective mild-TBI study found a subgroup with poor cognitive outcomes at one year, assessed across learning, memory, speed, and executive measures [30].

Memory is also the domain in which other impairments masquerade. A patient who cannot attend cannot encode, and a patient who cannot organize cannot retrieve; what looks like a memory failure is often an attention or executive failure upstream. Depression, pain, and poor sleep each impair memory in their own right. The evaluation has to sort these out, which is one reason a memory complaint cannot be read from a single test score.

Executive function and the front of the brain

Executive functions are the abilities that run the rest: deciding what to do, planning how to do it, starting, keeping the plan in mind, resisting distraction and impulse, checking the result, and changing course when it is not working. They depend on prefrontal regions and their connections with the rest of the brain. A useful anatomical framework distinguishes lateral frontal contributions to task setting and monitoring; superior medial contributions to initiating and sustaining activity; ventromedial and orbital contributions to emotional and behavioral regulation; and frontopolar contributions to integrating information and reflecting on one's own thinking [32]. These are interacting processes, not four independent boxes. Contemporary rehabilitation therefore addresses executive performance and self-awareness in relation to the person's goals and daily activities [14].

Two facts about executive deficits matter here. First, executive control depends on connected systems, so the evaluation cannot be reduced to whether a scan shows a focal frontal lesion [32]. The acute record, the course of recovery, test performance, and everyday behavior each contribute information. A scan describes anatomy at the resolution of that examination; it does not directly measure whether a person can plan, initiate, or regulate behavior.

The second is the dissociation between the test and life. Patients with injury to ventromedial or orbitofrontal regions can perform well on conventional intellectual tests yet have striking difficulty making decisions in everyday situations. The classic laboratory demonstration is a gambling task: patients with ventromedial prefrontal damage continued to favor immediate rewards despite unfavorable future consequences, whereas controls learned to choose more advantageously [33]. The task made an everyday problem measurable. The historical example is Phineas Gage, the railroad foreman who survived an iron rod passing through his frontal region in 1848 and whose subsequent behavior became central to the study of personality and frontal function. In the clinic, loss of smell after head injury also deserves attention. It may reflect injury to the nose, olfactory fibers, or central pathways, so it does not by itself locate a ventromedial frontal lesion [34]. A complete evaluation includes a structured family interview and, where appropriate, behavioral measures such as the Frontal Systems Behavior Scale, which asks about apathy, disinhibition, and executive difficulty in daily life.

Language and communication

A patient may retain vocabulary and grammar yet struggle to communicate. Word-finding problems can produce the familiar sense that a word is on the tip of the tongue; fluency tasks examine how efficiently words can be retrieved under a rule. Communication also requires much more: following a conversation with several participants, keeping to the topic, reading the listener's reaction, understanding tone, humor, and implication, organizing a complex account, and processing speech in noise. These failures can isolate patients socially and cost them at work. The INCOG 2.0 communication guideline, developed principally for moderate to severe TBI, emphasizes assessment of cognitive communication and social cognition in personally relevant settings, with attention to communication partners as well as the patient [29]. A speech-language pathologist's assessment can examine these demands beyond what a basic language score shows.

How cognitive deficits are measured, and what the numbers mean

Cognitive deficits are measured by comparing performance on standardized tests with appropriate normative groups, often adjusted for age and education, and expressing the result as a percentile or a standard score. A score at the 10th percentile means that about 90 percent of the reference group scored higher. Dr. Pappolla uses the 10th percentile as a flag for closer clinical assessment, not as a stand-alone diagnosis of impairment. Low scores also occur in healthy people, particularly when many tests are administered, so their pattern, the person's prior ability, functional history, and validity

findings matter [36]. Change between sessions should be judged with test-specific reliable-change methods that account for measurement variability and practice effects; a fixed difference of 10 percentile points is not a reliable rule [37]. These principles apply to cognitive testing. The physiological tests of Chapter 7 have their own reference ranges.

Two features of cognitive testing govern how its results should be read. First, performance is influenced by attention, sleep, pain, mood, medication, and engagement on the day, and a sound evaluation includes appropriate validity measures. A validity flag calls for caution and closer reading rather than a verdict. Performance validity failure occurs in clinical populations without an identified incentive to exaggerate and is not by itself evidence of dishonesty [2]. Second, a cognitive score measures performance in the domains and conditions tested. It may not reproduce the combined or prolonged demands of daily life; a normal result does not establish that every brain function is intact, and an abnormal result does not identify its cause. A single-domain evaluation cannot by itself establish or exclude the clinical diagnosis of brain injury, a point we have argued in print with colleagues [3]. Chapter 10 examines the limits of cognitive testing considered alone. Chapter 9 explains the distinction between screening, cognitive assessment, and full neuropsychological evaluation.

What happens over time

The course of cognitive deficits depends in part on injury severity. After moderate and severe injuries, difficulties with attention, memory, and executive function can persist for years, although recovery varies considerably [4]. After mild injury, most patients improve substantially during the early months [5]. That average course does not describe everyone. In TRACK-TBI, 13.5 percent of 656 participants with mild TBI met a study definition of poor cognitive outcome at one year, compared with 4.5 percent of 156 controls. The definition required multiple low scores, reliable decline, or both; it was not a diagnosis based on a single complaint or test [30]. Persistent cognitive difficulties can occur within the broader persistent post-concussion presentation described in Chapter 10, which also includes patients whose main limitations are non-cognitive. The disciplines for determining what has persisted in an individual patient are the subject of the last section of this chapter and of Chapters 7 and 8.

Non-cognitive consequences: mood, behavior, and psychiatric disorders

Where these fit

This book divides the consequences of brain injury into two categories: cognitive impairment, the subject of the first half of this chapter, and non-cognitive impairment, which covers everything else the injury does to the brain's operation. Several of the non-cognitive systems are covered elsewhere. The vestibular, oculomotor, visual, and autonomic systems, and the objective tests that measure them, are the subject of Chapter 7; headache and pain have Chapter 12; and Chapter 14 covers sleep and the endocrine disturbances that follow pituitary injury. The rest of this chapter covers the largest and most disputed group of non-cognitive consequences, addressed nowhere else in the book: the changes in mood, motivation, awareness, personality, and behavior that follow injury to the brain.

Why a brain injury causes psychiatric illness

Readers without medical training sometimes assume that a psychiatric diagnosis after an injury is only a reaction to pain, loss, and circumstances. That leaves out an important part of the picture. Brain injury can disturb the systems that support mood and behavior, while prior vulnerability, the psychological trauma of the event, and its practical consequences also shape the outcome.

The frontal, temporal, and connected subcortical systems discussed above contribute to mood, motivation, emotional control, and social behavior [32]. In a prospective TBI study, major depression was associated with reduced left prefrontal gray-matter volume and poorer executive and social functioning [7]; another study linked aggression with frontal lesions, depression, and premorbid social and substance-use problems [22]. These associations support a neurological contribution without assigning one lesion to every psychiatric presentation. Recent genetic evidence adds another part of the explanation: in CENTER-TBI and TRACK-TBI, inherited susceptibility to PTSD and depression was associated with adverse mental health outcomes after mild injury [6]. The individual outcome reflects both injury and vulnerability.

The epidemiology also matters. In a health maintenance organization population of 939 patients with brain injury and 2,817 matched controls, psychiatric illness was recorded during the first year in 49 percent of those with moderate or severe injury, 34 percent with mild injury, and 18 percent of controls. Among people without psychiatric illness in the preceding year, the adjusted relative risks during the first six months were 4.0 and 2.8, respectively [10]. In TRACK-TBI, 1,155 civilians with mild TBI were compared with 230 patients with orthopedic injuries: screening identified probable PTSD or major

depression in 20 percent versus 9 percent at three months and 21 percent versus 12 percent at six months [11] (Figure 11.3). The orthopedic group helps account for the burden of trauma outside the brain. The remaining difference is important evidence of increased risk after TBI, although an observational comparison cannot separate every contribution of injury, prior vulnerability, and circumstances.

Mood and anxiety disorders therefore belong in the clinical assessment after brain injury. The diagnostic manuals provide categories for depressive, anxiety, and personality disorders judged to be the physiological consequence of another medical condition [12]. Naming the brain injury as that condition makes a causal claim. The timing, clinical findings, prior history, and alternative explanations must support it; a psychiatric disorder that begins after the injury does not automatically meet that definition.

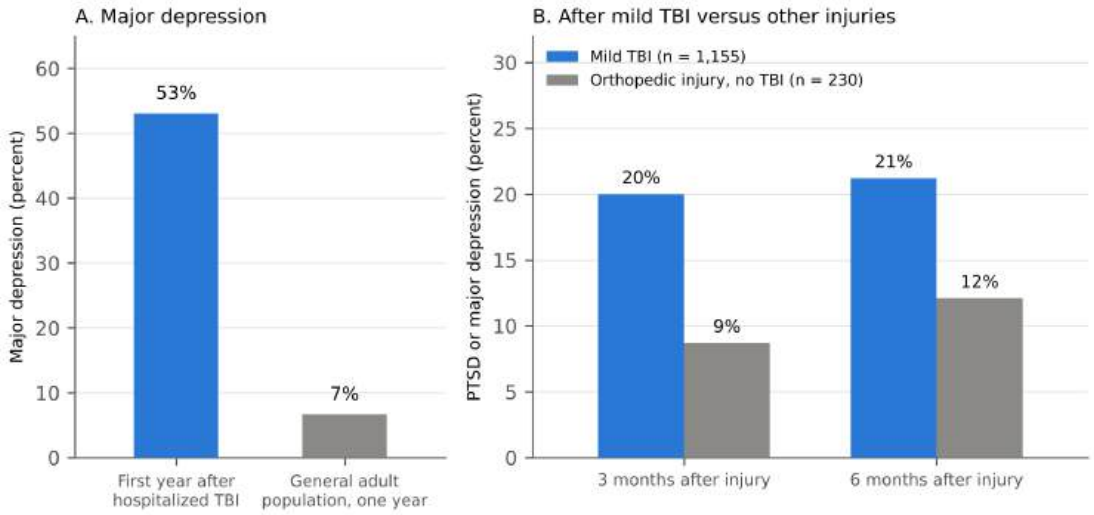


Figure 11.3. Mood and trauma-related disorders after brain injury. (A) In 559 adults followed for a year after hospitalization for complicated mild to severe TBI, 53 percent met study criteria for major depression at least once; fewer than half received antidepressant treatment or counseling [13]. The comparison with the general population is contextual, not a matched-control estimate. (B) TRACK-TBI screening identified probable PTSD or major depression in 20 percent of patients with mild TBI versus 9 percent of orthopedic controls at three months, and 21 percent versus 12 percent at six months [11]. Panel B reports the combined screening outcome, not PTSD alone. Original chart drawn from the published figures.

Depression

Depression is a common psychiatric consequence of brain injury and one of the most consequential. In a prospective study of 559 adults hospitalized with complicated mild

to severe injury and followed for a year, 53 percent met criteria for major depressive disorder at least once. Injury severity did not predict depression within that cohort, and fewer than half of the depressed patients received antidepressants or counseling [13]. In the Iowa prospective series, a third of 91 patients developed major depression in the first year; among those depressed patients, about three quarters also had anxiety and more than half had aggressive behavior [7]. These are findings from particular clinical populations, not a single prevalence that applies to every concussion.

The features are those of depression generally: low mood, loss of interest and pleasure, hopelessness, guilt, worthlessness, disturbed sleep and appetite, and in some patients thoughts of death. What matters in the brain-injured patient is the company it keeps and the way it is missed. In the hospitalized cohort, depression was associated with substantially more anxiety and poorer health-related quality of life at one year [13]. Cognitive problems, pain, sleep disturbance, and fatigue must be assessed alongside it. Poor concentration, sleep disturbance, and irritability can belong to more than one condition, and patients may attribute their low mood to circumstances rather than reporting it as an illness. Current rehabilitation guidance calls for repeated mental health assessment and treatment that combines appropriate psychological, social, and medical approaches [40].

Chapter 14 sets out the treatment. The point here is that depression after brain injury deserves its own diagnosis and care. Its presence does not explain away every cognitive or physical consequence, and evidence of brain injury does not remove the need to consider the other contributors to depression [6,16]. Treating the depression is part of treating the patient.

Anxiety

Anxiety disorders after brain injury take several forms, including generalized worry, panic, and trauma-related fear. Anxiety can also develop around the injury's physical consequences. A patient with a vestibular disorder who becomes dizzy in supermarkets and crowds learns to avoid them. A patient with visual motion intolerance avoids driving; a patient with noise sensitivity avoids restaurants. These examples call for an examination of the sensory disturbance as well as the anxiety, because the two can coexist [16]. Anxiety may accompany the realization, as awareness returns during recovery, of how much has changed, and it often accompanies depression [7,13]. Sleep deserves attention too: in a large prospective study, psychiatric history was associated with a more persistent severe-insomnia trajectory after TBI [15].

Post-traumatic stress disorder

Post-traumatic stress disorder is the psychiatric condition most often raised in these cases, sometimes to explain the patient's symptoms without a brain injury and sometimes to explain them without stress. Neither position survives the evidence.

Post-traumatic stress disorder is the syndrome that follows exposure to a frightening, life-threatening event and is defined by four groups of symptoms: reliving the event through intrusive memories, nightmares, and flashbacks; avoiding reminders of it; persistent negative changes in mood and thinking; and a state of heightened arousal with sleep disturbance, irritability, hypervigilance, exaggerated startle, and poor concentration, lasting more than a month and impairing function [12]. Trauma-linked re-experiencing and avoidance are particularly useful diagnostic clues; many other symptoms overlap with depression, anxiety, and persistent post-concussion symptoms [41].

PTSD is common after some of the events that cause brain injury, but its frequency depends on the event, population, and assessment method. In TRACK-TBI, the combined screening rate of probable PTSD or major depression after mild injury was 20 percent at three months and 21 percent at six months. Prior mental health problems and injury by assault or violence were among the factors associated with greater PTSD risk [11]. In soldiers returning from Iraq, PTSD screening criteria were met by 44 percent of those reporting a brain injury with loss of consciousness, 27 percent with altered mental status only, 16 percent with other injuries, and 9 percent with none [17]. These estimates describe different populations and outcomes and should not be treated as interchangeable.

That military study is often cited in contested cases, and it is worth being precise about what it showed. Hoge and colleagues found that, once PTSD and depression were taken into account, mild brain injury was no longer associated with most of the reported physical health outcomes, except headache [17]. The study used questionnaires in soldiers returning from deployment. It did not establish that persistent symptoms after civilian brain injury are all caused by PTSD. The two conditions can interact through impaired emotional regulation, sleep disruption, and shared vulnerabilities; the frontal and limbic systems involved in fear and regulation provide plausible mechanisms, rather than a lesion map that proves the explanation in an individual patient [6,18].

The overlap of symptoms is the practical difficulty (Figure 11.4). Poor concentration, memory complaints, irritability, sleep disturbance, fatigue, anxiety, and low mood occur in both presentations. A structured interview examines the traumatic event, re-experiencing, avoidance, and the timing of symptoms; neurological assessment

examines complaints such as dizziness, imbalance, headache, and visual disturbance. No symptom list alone separates the conditions. A patient who cannot clearly recall the collision can still develop PTSD, and a history of TBI does not prevent effective PTSD treatment [41]. The course provides useful evidence: improvement in some functions alongside increasing trauma-linked avoidance suggests that more than one process needs attention. Persistent vestibular or oculomotor findings also require their own differential diagnosis; their presence does not automatically identify the cause of the psychiatric symptoms [16].

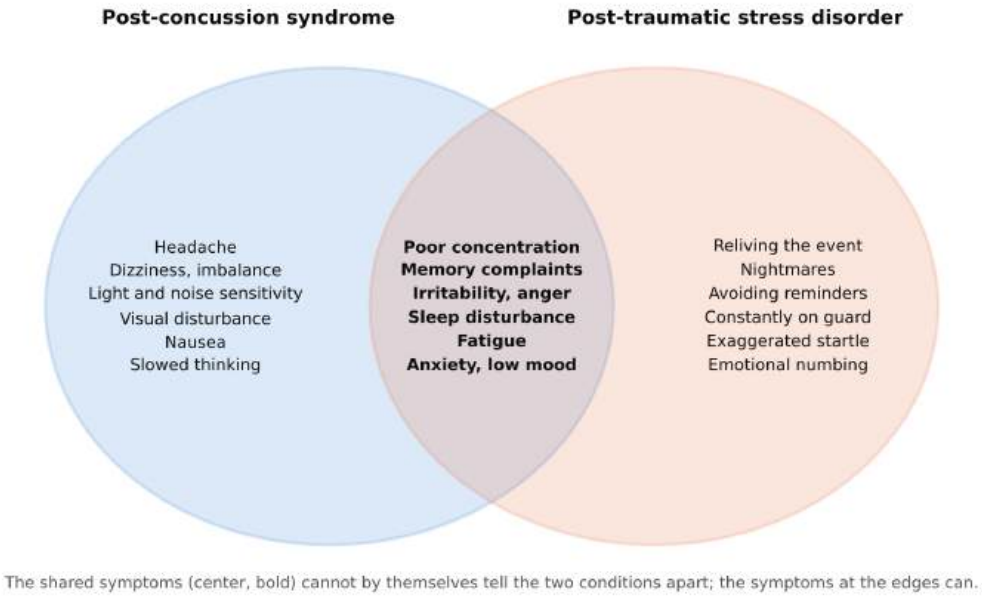


Figure 11.4. Persistent post-concussion symptoms and PTSD overlap. The center lists shared complaints that cannot distinguish the conditions on their own. The outer groups illustrate features that direct further assessment: trauma-linked reliving and avoidance prompt PTSD assessment; headache, dizziness, imbalance, light and noise sensitivity, and visual disturbance prompt neurological and sensory assessment. Placement at an edge does not make a symptom exclusive to that condition, and the physiological tests in Chapter 7 do not by themselves distinguish every cause. The two conditions can coexist. Original schematic.

PTSD after a collision is a real consequence of the event and should be treated. Its presence does not exclude brain injury. A careful history, structured psychiatric assessment, and examination of neurological and sensory function establish what evidence supports each diagnosis and where uncertainty remains [16,41].

Disorders of motivation: apathy, abulia, and akinetic mutism

Motivation gives behavior its direction, vigor, and persistence, and it can be impaired after brain injury. Apathy is a reduction in goal-directed behavior, thought, or emotion. Abulia describes a more profound loss of initiative, with sparse or slowed speech and activity; akinetic mutism describes the extreme clinical state in which a wakeful patient shows little spontaneous movement or speech. These terms describe a spectrum of motivational and initiation disorders, rather than conditions diagnosed from inactivity alone. Apathy is the main concern here. In an older series of 83 patients attending a neuropsychiatric clinic, about 71 percent had apathy, often with depression [19]. That selected-clinic estimate should not be read as the prevalence among all people with TBI. A more recent controlled study confirmed increased informant-rated apathy across executive, emotional, and initiation dimensions after moderate to severe TBI [9].

The apathetic patient does not start things. When asked, the patient may describe goals and act on them when prompted, yet do so less often, less intensely, or for less time than before. The family describes a person who sits for hours, no longer initiates conversation, projects, or outings, and needs prompting to shower, eat, or make a telephone call. The patient may be less troubled by the change or less aware of it. In the controlled study, patients and informants differed in their ratings of executive and initiation apathy [9]. Frontal and connected motivational systems contribute to these behaviors, but the pattern also has to be understood alongside depression, cognition, fatigue, medication, and the patient's environment [8,32].

Apathy must be distinguished from depression, and the distinction is useful in practice (Figure 11.5). Depression centers on disturbed mood, loss of pleasure, and often distress, guilt, or hopelessness; apathy centers on reduced initiation and goal-directed activity. A patient can have both [19]. The difference matters because treatment aimed only at low mood may leave the initiation problem untouched. An employer, family member, or examiner who has not considered apathy may call it laziness, lack of motivation, or malingering. Missed appointments, an unfinished treatment plan, or failure to seek work should prompt assessment of what prevents action, including motivation, awareness, memory, practical barriers, and mood. They do not establish a character flaw, or a specific neurological explanation, on their own [8].

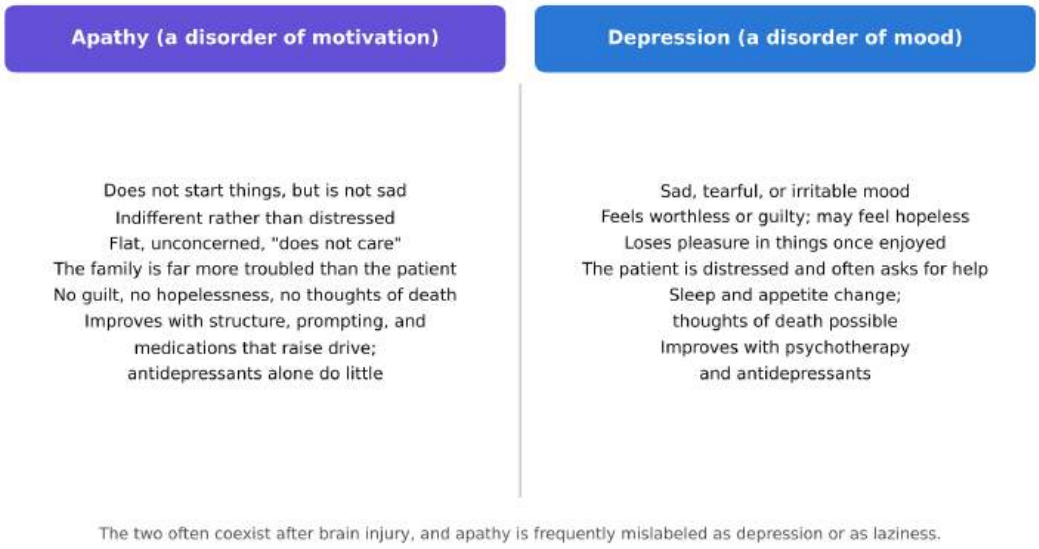


Figure 11.5. Apathy concerns motivation and initiation; depression concerns mood and related symptoms. The illustration contrasts reduced initiation with depressive distress, but individual patients vary: a depressed person may not seek help, and an apathetic person may experience distress or have depression as well. The two conditions can coexist after brain injury. Original schematic.

The functional cost is large. A patient who cannot initiate action may need support to take medication, attend therapy, or begin a search for work. Care therefore examines treatable contributors and builds practical structure around the tasks that matter. Prompts, manageable goals, feedback, and family participation can make a treatment plan usable. Current behavioral rehabilitation guidance covers apathy as well as aggression, while executive rehabilitation supports goal management and self-monitoring [8,14]. Medication decisions require an individual assessment; a label of apathy alone does not select an established drug treatment.

Disorders of awareness and anosognosia

Among the strangest and most consequential effects of a brain injury is reduced ability to recognize one's own difficulties. The clinical term anosognosia refers to impaired awareness of a deficit. In TBI it may involve cognitive or behavioral changes as well as physical problems. Psychological denial is a defensive response to an unwelcome truth; impaired self-monitoring is a different process, although the two may coexist and be hard to separate. The evaluation asks what the person recognizes, what happens during actual tasks, and how that account compares with observations over time [14,20].

Awareness can change during recovery. In a longitudinal study of 123 people with moderate to severe TBI, agreement between patients' and relatives' ratings improved from about 45 days after injury to one year. The largest changes were in behavioral and emotional awareness; motor and sensory ratings had agreed more closely at the outset. Even at one year, patients on average rated their functioning more favorably than relatives did [20]. This pattern supports repeated assessment and realistic rehabilitation goals. It does not justify assuming that every discrepancy is permanent.

Awareness has several aspects (Figure 11.6). A longstanding rehabilitation model distinguishes intellectual awareness, knowing in the abstract that one has a problem; emergent awareness, noticing the problem while it is happening; and anticipatory awareness, predicting that the problem will arise and planning around it [21]. A patient may acknowledge that memory is not what it was yet fail to notice a missed appointment or think to write it down. The hierarchy is a useful model for assessment and treatment, not a fixed sequence in which every injury removes abilities. Current executive rehabilitation guidance emphasizes feedback and metacognitive strategies to strengthen self-monitoring in daily tasks [14].

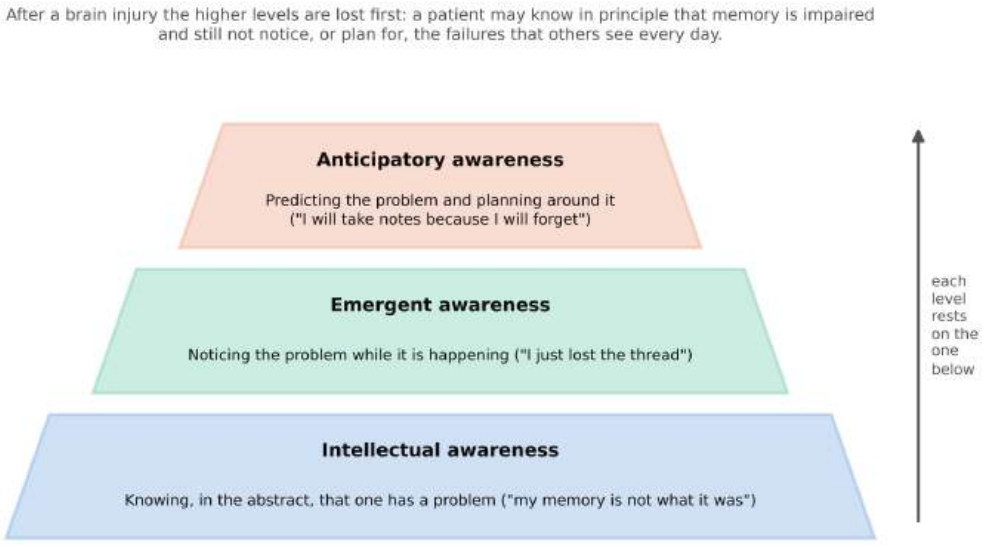


Figure 11.6. Levels of awareness in a hierarchical rehabilitation model. Intellectual awareness is knowing that one has a deficit; emergent awareness is noticing it while it operates; anticipatory awareness is predicting it and planning around it. A patient may acknowledge a memory problem yet fail to notice or plan for everyday failures. The hierarchy illustrates increasing demands on awareness, not a universal order of loss after injury. Original schematic.

Unawareness can be assessed through discrepancy: the patient's ratings are compared with family or clinician ratings and with observed performance, using instruments such as the Patient Competency Rating Scale and the Awareness Questionnaire [20]. A discrepancy is a finding to explain. The evaluator considers cognitive ability, opportunities to observe the behavior, mood, relationships, and performance during real tasks before deciding whether impaired awareness is the best account. Neither the patient nor the informant is automatically the more accurate witness in every domain.

The medicolegal significance of impaired awareness runs in a direction the reader may not expect. Self-report instruments assume that the person can recognize and describe the problem. A patient with limited awareness may minimize difficulties at interview or report few symptoms while relatives describe substantial change. The patient who insists on returning to a job they can no longer manage, and then struggles at it, is a familiar clinical problem. That discrepancy calls for assessment of awareness; it is not, by itself, proof for or against exaggeration. Collateral history from a spouse, parent, or coworker and direct observation of functioning are therefore important parts of the evaluation. Rehabilitation should address self-monitoring as well as the underlying difficulty [14,20].

Personality change

The complaint that a brain-injured patient ‘is not the same person’ is one that families bring to the clinic, and it deserves a careful account of what has changed. Personality change due to another medical condition is a recognized diagnosis: a persistent disturbance from the person's previous pattern, attributable to the physiological effects of the condition, with subtypes named for features such as lability, disinhibition, aggression, apathy, or paranoia [12]. The clinical task is to identify the new behavior and its effect on relationships, work, and independence. Executive, social-communication, and behavioral assessments provide complementary ways to examine those changes [8,14,29].

The features include impaired social perceptiveness, reduced self-control, behavior driven by the immediate situation, emotional change, and difficulty learning from social experience. A person may have trouble starting and stopping, shifting approach, monitoring mistakes, or understanding implication and nuance. Two familiar patterns help organize the examination. Disinhibition may appear as impulsivity, tactlessness, inappropriate familiarity, euphoria, or poor judgment; diminished initiation may appear as apathy, indifference, and loss of drive. Orbital and ventromedial systems contribute to emotional and behavioral regulation, while superior medial systems contribute to initiating and sustaining activity, but these functions depend on interacting networks

[32]. The behavior and its change from baseline deserve assessment whether or not routine imaging shows a focal frontal lesion.

Personality change can be mistaken for other conditions. Disinhibition may resemble an established personality disorder; apathy may resemble depression; a flat voice may be interpreted as blunted affect; avoidance of crowds may reflect visual or vestibular discomfort as well as anxiety; difficulty processing speech in noise may be described simply as inattention. These are differential diagnoses to examine, not shortcuts to either a psychological or neurological conclusion [16,29]. The correction begins with a history of the person before the injury, obtained from records and people who knew them, followed by assessment of the relevant functions. The work context matters too. Small changes in planning, judgment, or communication may have substantial consequences for a lawyer, health professional, entrepreneur, or anyone whose job relies heavily on those abilities.

A patient seen in Dr. Pappolla's outpatient neurology clinic supplies an illustration; the names have been changed. Marianne was a college student, the eldest daughter of a close family, described by everyone who knew her as curious, warm, and even-tempered. Returning from college one winter she was in a severe collision and sustained a traumatic brain injury. Her imaging was unremarkable, but she had lost her sense of smell completely, and quantitative electroencephalography was reported as showing dysregulation over the frontal lobes. Those findings warranted attention but did not, by themselves, locate a lesion on the under-surface of the frontal lobes: olfactory loss has several possible sites of injury, and qEEG is not an established method for diagnosing mild TBI [34,35]. As her physical injuries healed, the change in her personality became apparent. She became impulsive, unpredictable, and emotionally volatile; the laughter her family remembered was replaced by outbursts that none of them could anticipate. Her younger brother, who had relied on her as a confidante, found that ordinary shared activities turned into confrontations and that his sister had become a stranger. Her parents grieved for a daughter who was alive and present and no longer the person they had raised. She performed within normal limits on much of her cognitive testing. Her family's account described changes that those scores alone could not explain.

Irritability and aggression

Irritability and aggression are recognized problems after brain injury. In a prospective series of 89 patients with TBI and 26 patients with trauma elsewhere in the body, aggressive behavior was recorded during the first six months in about a third of the TBI group and 12 percent of the comparison group. Depression, frontal lesions, premorbid

social difficulties, and a history of alcohol or substance misuse were associated with aggression [22]. Current behavioral rehabilitation guidance treats aggression as a problem requiring assessment of the person, the setting, and possible contributors [8].

Some post-injury outbursts are reactive and disproportionate to the trigger: a small frustration is followed by an abrupt verbal or physical response, then embarrassment or regret. Others have a different pattern. Reactivity, explosiveness, and remorse help describe what happened, but they are not diagnostic signs that prove brain injury caused it. The assessment records triggers, frequency, severity, consequences, and what helps the person regain control. Depression and substance-use problems deserve particular attention because they were associated with aggression in the prospective study [22]. The aim is to understand the behavior well enough to improve safety and choose a useful intervention [8].

The evaluator must determine whether the aggression preceded the injury, began after it, or became worse afterward. Pre-existing temper, alcohol and drug use, medications, pain, sleep disruption, seizures, and other medical or psychiatric conditions have to be considered. A normal cognitive score does not answer that behavioral question. The pattern may be much clearer at home or work than in a structured appointment, so the assessment needs specific observations from those settings as well as the patient's account. Current guidelines emphasize assessment and individualized management rather than assigning causation from the behavior alone [8]. The consequences, for the family that withdraws, the employer that dismisses, and the patient who is arrested, can be among the most damaging after brain injury.

Attention deficit and acquired ADHD

Attention-deficit/hyperactivity disorder is a developmental condition that begins in childhood. Brain injury can produce overlapping attention and behavioral problems, and in children the acquired syndrome is often called secondary ADHD. In a study following children for five to ten years, severe TBI was associated with a higher risk of new attention-deficit/hyperactivity symptoms or diagnosis than orthopedic injury [23]. Adults may also develop inattention, distractibility, difficulty sustaining mental effort, disorganization, and impulsivity after TBI. The distinction from developmental ADHD depends on the developmental history, the timing of change, school and employment records, and other possible contributors. Records can be incomplete, so their silence is not proof that a prior problem was absent. Treatment can overlap: the INCOG attention guideline recommends methylphenidate to improve processing speed in appropriate patients, while also addressing sleep, pain, mood, sensory problems, and medication effects [27]. Treating attention alone does not address every consequence of the injury.

Sleep disturbance and fatigue

Sleep disturbance is common after brain injury and can remain a problem well beyond the acute period. A meta-analysis found sleep difficulties in about half of patients, including insomnia and excessive daytime sleepiness [24]. More recent TRACK-TBI data show why follow-up matters: among 2,022 participants, most with mild injury, 61.6 percent followed a trajectory of persistent mild insomnia symptoms during the first year, 31.0 percent improved from initially mild symptoms, and a smaller group had persistent or emerging severe symptoms [15]. These modeled symptom trajectories are not the same as the prevalence of a diagnosed sleep disorder. Assessment also considers sleep apnea, circadian disruption, pain, medication, and psychiatric symptoms. The acute severity label alone cannot describe the patient's sleep problem.

Fatigue is another common and disabling complaint. In CENTER-TBI, the proportion reporting fatigue was 47 percent at baseline, 48 percent at three months, and 46 percent at six months, using the fatigue item on the Rivermead questionnaire [38]. Patients describe feeling drained, losing concentration, and finding that ordinary activity takes more out of them than it should. Fatigue has several contributors: that study found associations with injury characteristics, pre-existing health problems, disturbed sleep, and depressive symptoms. The pituitary hormone deficiencies described in Chapter 14 are another clinical consideration. The assessment should examine what brings fatigue on, how long recovery takes, and which contributors can be treated. Calling it laziness substitutes a judgment for that work.

Psychosis

Psychotic illness after brain injury deserves attention, although a temporal sequence alone does not establish its cause. A 2024 Swedish register study identified 4,184 people with schizophrenia and 18,681 with bipolar disorder and compared them with matched controls and, separately, unaffected siblings. A history of TBI was associated with higher subsequent risk of schizophrenia and bipolar disorder, with incidence rate ratios of 1.33 and 1.78; the associations persisted in sibling comparisons and were stronger after moderate or severe than mild injury [25]. When psychosis appears after TBI, the evaluation considers the timing, prior and family history, medications, substances, seizures, and other medical causes before deciding how the conditions are related.

Suicide

The gravest consequence of the psychiatric sequelae of brain injury is the risk of suicide, and it is well documented. In a nationwide Danish cohort of more than seven

million people followed over three decades, those with a history of traumatic brain injury died by suicide at about twice the rate of those without: 41 against 20 per 100,000 person-years. Risk was highest in the first six months after injury and remained elevated for years. It was present after mild injury as well as severe, and rose with severity, longer hospital stays, and more medical contacts [26]. Potential contributors include depression, impulsivity, loss of role and relationships, pain, insomnia, and alcohol use; the registry study does not determine the mechanism in an individual death. The psychiatric consequences of brain injury are not a soft category of complaint; they carry mortality. A patient with persistent symptoms who expresses hopelessness is a patient whose treating physicians should be asked what they are doing about it.

How these consequences interact

These conditions often overlap. Depression can accompany anxiety and aggression; apathy can coexist with depression; sleep disturbance can add to problems with pain, mood, attention, and fatigue [7,9,15,19]. Impaired awareness may make the whole pattern harder to recognize. Chapter 14 describes these interactions and where treatment can interrupt them. For the evaluator, a cluster of cognitive, emotional, behavioral, sleep, and pain complaints calls for an organized assessment of the relationships among them. It should not be dismissed as an implausible catalog, or assumed to arise from one mechanism simply because the symptoms appeared together.

Evaluating these consequences when the evaluation will be contested

Evaluation of the cognitive and psychiatric consequences of brain injury draws on the history, examination, standardized instruments, and objective physiological testing described in Chapter 7. Several elements may be questioned, from either direction. The following disciplines make the evaluation more defensible.

The history must come from more than the patient when awareness, memory, or organization is impaired. A spouse, parent, adult child, close friend, or coworker who knew the patient before the injury can describe changes that a brief appointment misses. A competent evaluation seeks specific examples and, where appropriate, uses structured informant measures. Differences between accounts should be examined by domain and against records or observed performance; family reports are valuable evidence, not automatically the correct account [20].

The baseline must be established from records, not assumed. The question in every contested case is what the patient was like before. School records, employment

records, performance reviews, military records, prior medical and psychiatric records, and the accounts of people who knew the patient establish the baseline against which the change is measured. A patient with a documented history of depression, attention deficit, or temper before the injury presents a different problem from one with none, and the distinction between a condition the injury caused and a condition it aggravated is made here, from the record, not from the diagnosis.

The diagnosis should state the evaluator's conclusion precisely. The diagnostic manuals provide labels such as mild or major neurocognitive disorder due to traumatic brain injury and depressive, anxiety, or personality disorder due to another medical condition [12]. These labels assert a causal relationship, so the clinical evidence must support that relationship rather than timing alone. Prior history, the injury record, the subsequent course, examination, relevant tests, and alternative explanations all matter. A psychiatric disorder may also coexist with TBI without meeting the criteria for direct physiological attribution.

Self-report instruments should be read as self-report. Questionnaires of depression, anxiety, post-traumatic stress, and post-concussive symptoms provide useful standardized information. Their scores are read alongside the clinical interview, informant accounts, records, and objective findings. A discrepancy is information to investigate: low symptom endorsement with observed difficulty may reflect reduced awareness, while severe symptoms with preserved performance may reflect demands or conditions the tests did not sample. Neither pattern establishes a diagnosis or a conclusion about validity on its own [14,20,41].

Validity is assessed, and its limits are respected. Cognitive testing includes performance validity measures, and the evaluator should use and report them. Failure occurs in clinical populations without an identified incentive to exaggerate, so it calls for careful interpretation of the affected scores rather than an automatic judgment of dishonesty [2]. Physiological measures can add information about the functions tested, but their technical quality, relevant confounders, and requirements for cooperation still matter. Agreement among tests, history, records, and observed function strengthens an interpretation; disagreement calls for closer examination of what each measure can establish. Neither an abnormal physiological result nor a normal one settles every question about symptoms or validity.

Physiological testing can add to the psychiatric assessment, but it does not settle causation by itself. A patient may have depression or anxiety together with abnormal oculomotor recordings, balance findings, evoked potentials, or cognitive scores. Each finding should be interpreted according to what the test measures and the other

conditions that can affect it. Convergence can support a broader account of post-injury dysfunction; it does not automatically show that all abnormalities arise from one frontal or white-matter lesion or that the psychiatric disorder is its direct physiological result. The causal diagnostic formulation still rests on the complete clinical assessment [12,16].

Questions commonly raised about these findings, and what the evidence says

The questions below are raised by careful evaluators and by the parties in contested cases. Each has an answer in the literature, and the answer sometimes supports the diagnosis and sometimes does not; the evaluator's obligation is to say which.

The symptoms are psychiatric, so they are not neurological. That distinction is too simple. TBI is associated with increased psychiatric morbidity, and the effects of injury can interact with prior vulnerability and the consequences of the event [6,10,11]. A psychiatric diagnosis does not exclude a neurological contribution. Calling the disorder a direct physiological consequence of TBI is a further clinical judgment that must meet the relevant diagnostic criteria [12].

The patient was already depressed, anxious, or difficult. Sometimes true, and the record decides it. A documented prior condition is a baseline against which the change is measured, not an explanation that removes the injury, and not one that can be dismissed either. The relevant findings are whether the condition worsened, whether its character changed, and whether the post-injury pattern includes new problems with impulse control, initiation, or awareness. These findings deserve assessment but are not individually specific to TBI. A prior psychiatric history is also, as the TRACK-TBI data show, a risk factor that makes a brain-injured patient more likely to develop a post-injury disorder, not less [11].

It is post-traumatic stress disorder, not brain injury. The two can coexist and share symptoms. Trauma-linked re-experiencing and avoidance help direct PTSD assessment; neurological and sensory findings need their own interpretation. There is no single symptom checklist or physiological test that separates every case [16,41]. The military questionnaire study often cited in this argument did not establish that civilian brain injury produces no persistent symptoms [17].

The cognitive testing was normal. A normal result is evidence of preserved performance in the domains and conditions tested. It should be weighed alongside the demands of daily life. The classic prefrontal decision-making experiment showed why a patient could retain conventional intellectual abilities yet struggle with consequential

choices [33]. Conversely, consistently preserved performance, function, and a lack of corroborating evidence weigh against a claim of substantial impairment. Chapter 10 sets out both readings in detail.

The patient does not report many symptoms, so the symptoms are not severe. Limited self-report may miss important problems when awareness is impaired. Low endorsement alongside observed difficulty and a contrasting collateral history should prompt assessment of awareness, including how the accounts differ across domains [20]. It is neither proof of recovery nor an independent test for exaggeration.

The patient is exaggerating. Exaggeration is one possibility an evaluator may need to consider, but performance validity failure is not by itself evidence of intentional deception [2]. The affected scores must be interpreted in context, including the test's requirements and limitations. A defensible conclusion draws on records, collateral history, observed function, appropriate validity measures, and physiological findings assessed for technical quality and alternative explanations. No single part of that evaluation provides automatic protection against error.

The patient did not seek psychiatric treatment. In the hospitalized cohort, more than half of those meeting study criteria for major depression received neither antidepressants nor counseling during follow-up [13]. Symptoms are attributed to the injury or to circumstances, the patient does not recognize them, apathy prevents help-seeking, and the health system fails to look. An untreated condition is common and does not by itself mean the condition was absent. A record in which the patient was seen repeatedly for other reasons and never mentioned a symptom is a different matter and weighs the other way.

Fast take: main points from chapter 11

• Cognitive and behavioral changes can substantially limit work, relationships, and independence after brain injury, even when physical recovery appears good [14,29,39].

• Slowed processing and difficulty managing competing demands can affect daily life. Their pattern varies with injury severity and the individual, and combined-task assessment may reveal difficulty that simpler tasks do not (Chapter 10) [1,31].

• Executive function depends on connected systems. The presence or absence of a visible focal frontal lesion does not substitute for assessment of planning, initiation, judgment, and self-monitoring [14,32].

• Psychiatric morbidity is increased after TBI. Injury, prior vulnerability, and the consequences of trauma can contribute together; a diagnosis ‘due to another medical condition’ requires an individual causal assessment [6,10,11,12].

• In one prospective cohort hospitalized for complicated mild to severe TBI, 53 percent met study criteria for major depression during the first year, and only 44 percent of those patients received antidepressants or counseling [13].

• TRACK-TBI found probable PTSD or major depression in about one fifth of the mild-TBI group at three and six months. That is a combined screening outcome, not the rate of PTSD alone [11].

• Apathy concerns reduced initiation and goal-directed activity. It can coexist with depression and interfere with rehabilitation; assessment should examine both the patient's and informant's accounts [8,9,19].

• Awareness of cognitive and behavioral difficulties may be impaired and can improve during recovery. Patient-informant discrepancies help guide assessment but do not automatically establish which account is correct [14,20].

• Changes in inhibition, initiative, emotional regulation, and communication can profoundly affect personality and relationships. Their assessment requires a baseline and examples of everyday functioning [14,29,32].

• Aggression occurred in about a third of TBI patients during the first six months in one prospective study. Its causes and contributors must be assessed; reactive or regretted behavior does not by itself prove an injury mechanism [8,22].

• Sleep problems and fatigue are common and can persist. Their course varies, and evaluation should consider injury characteristics alongside mood, pain, medication, sleep disorders, and other medical contributors [15,24,38].

• The risk of suicide was roughly doubled overall after TBI, was also elevated after mild injury, and was highest in the first six months [26].

• A contested evaluation rests on collateral history, pre-injury records, diagnoses that name the mechanism, self-report read as self-report, validity measures read probabilistically, and convergence with the objective physiological testing of Chapter 7.

References

1. Bryant AM, Rose NB, Temkin NR, et al. Profiles of cognitive functioning at 6 months after traumatic brain injury among patients in level I trauma centers: a TRACK-TBI study. JAMA Netw Open. 2023;6(12):e2349118. doi:10.1001/jamanetworkopen.2023.49118
2. McWhirter L, Ritchie CW, Stone J, Carson A. Performance validity test failure in clinical populations: a systematic review. J Neurol Neurosurg Psychiatry. 2020;91(9):945-952. doi:10.1136/jnnp-2020-323776
3. Pappolla MA, Pappolla SL, Nader R, Hamza MK, Fang F, Fang X. Evaluating cognition across aging and traumatic brain injury: integrating neurological and neuropsychological approaches. J Clin Med. 2026;15(10):3822. doi:10.3390/jcm15103822
4. Dikmen SS, Corrigan JD, Levin HS, Machamer J, Stiers W, Weisskopf MG. Cognitive outcome following traumatic brain injury. J Head Trauma Rehabil. 2009;24(6):430-438. doi:10.1097/HTR.0b013e3181c133e9
5. Carroll LJ, Cassidy JD, Peloso PM, et al. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2004;(43 Suppl):84-105. doi:10.1080/16501960410023859
6. Kals M, Wilson L, Levey DF, et al. Genetic vulnerability and adverse mental health outcomes following mild traumatic brain injury: a meta-analysis of CENTER-TBI and TRACK-TBI cohorts. EClinicalMedicine. 2024;78:102956. doi:10.1016/j.eclinm.2024.102956
7. Jorge RE, Robinson RG, Moser D, Tateno A, Crespo-Facorro B, Arndt S. Major depression following traumatic brain injury. Arch Gen Psychiatry. 2004;61(1):42-50. doi:10.1001/archpsyc.61.1.42
8. MacKenzie HM, Velikonja D, Devito J, et al. Updated Canadian Clinical Practice Guideline for the Rehabilitation of Adults With Moderate to Severe Traumatic Brain Injury: behavioral recommendations. J Head Trauma Rehabil. 2024;39(5):382-394. doi:10.1097/HTR.0000000000000973
9. Quang H, McDonald S, Huynh-Le P, et al. Apathy in a high prevalence population of moderate to severe traumatic brain injury: an investigation in Vietnam. Neuropsychology. 2022;36(1):94-102. doi:10.1037/neu0000781
10. Fann JR, Burington B, Leonetti A, Jaffe K, Katon WJ, Thompson RS. Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. Arch Gen Psychiatry. 2004;61(1):53-61. doi:10.1001/archpsyc.61.1.53
11. Stein MB, Jain S, Giacino JT, et al. Risk of posttraumatic stress disorder and major depression in civilian patients after mild traumatic brain injury: a TRACK-TBI study. JAMA Psychiatry. 2019;76(3):249-258. doi:10.1001/jamapsychiatry.2018.4288

12. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed, text revision (DSM-5-TR). Washington (DC): American Psychiatric Association Publishing; 2022.
13. Bombardier CH, Fann JR, Temkin NR, Esselman PC, Barber J, Dikmen SS. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. JAMA. 2010;303(19):1938-1945. doi:10.1001/jama.2010.599
14. Jeffay E, Ponsford J, Harnett A, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, part III: executive functions. J Head Trauma Rehabil. 2023;38(1):52-64. doi:10.1097/HTR.0000000000000834
15. Wickwire EM, Albrecht JS, Capaldi VF, et al. Trajectories of insomnia in adults after traumatic brain injury. JAMA Netw Open. 2022;5(1):e2145310. doi:10.1001/jamanetworkopen.2021.45310
16. Department of Veterans Affairs; Department of Defense. VA/DoD clinical practice guideline for the management and rehabilitation of post-acute mild traumatic brain injury. Version 3.0. Washington (DC): Department of Veterans Affairs and Department of Defense; 2021. Available from: https://www.healthquality.va.gov/guidelines/Rehab/mtbi/VADODmTBICPGFinal508.pdf
17. Hoge CW, McGurk D, Thomas JL, Cox AL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med. 2008;358(5):453-463. doi:10.1056/NEJMoa072972
18. Stein MB, McAllister TW. Exploring the convergence of posttraumatic stress disorder and mild traumatic brain injury. Am J Psychiatry. 2009;166(7):768-776. doi:10.1176/appi.ajp.2009.08101604
19. Kant R, Duffy JD, Pivovarnik A. Prevalence of apathy following head injury. Brain Inj. 1998;12(1):87-92. doi:10.1080/026990598122908
20. Hart T, Seignourel PJ, Sherer M. A longitudinal study of awareness of deficit after moderate to severe traumatic brain injury. Neuropsychol Rehabil. 2009;19(2):161-176. doi:10.1080/09602010802188393
21. Crosson B, Barco PP, Velozo CA, et al. Awareness and compensation in postacute head injury rehabilitation. J Head Trauma Rehabil. 1989;4(3):46-54.
22. Tateno A, Jorge RE, Robinson RG. Clinical correlates of aggressive behavior after traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2003;15(2):155-160. doi:10.1176/jnp.15.2.155
23. Narad ME, Kennelly M, Zhang N, et al. Secondary attention-deficit/hyperactivity disorder in children and adolescents 5 to 10 years after traumatic brain injury. JAMA Pediatr. 2018;172(5):437-443. doi:10.1001/jamapediatrics.2017.5746

24. Mathias JL, Alvaro PK. Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: a meta-analysis. Sleep Med. 2012;13(7):898-905. doi:10.1016/j.sleep.2012.04.006

25. Cheng KY, Robinson N, Ploner A, et al. Impact of traumatic brain injury on risk for schizophrenia and bipolar disorder. Psychiatry Res. 2024;339:115990. doi:10.1016/j.psychres.2024.115990

26. Madsen T, Erlangsen A, Orlovska S, Mofaddy R, Nordentoft M, Benros ME. Association between traumatic brain injury and risk of suicide. JAMA. 2018;320(6):580-588. doi:10.1001/jama.2018.10211

27. Ponsford J, Velikonja D, Janzen S, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, part II: attention and information processing speed. J Head Trauma Rehabil. 2023;38(1):38-51. doi:10.1097/HTR.0000000000000839 https://pubmed.ncbi.nlm.nih.gov/36594858/

28. Velikonja D, Ponsford J, Janzen S, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, part V: memory. J Head Trauma Rehabil. 2023;38(1):83-102. doi:10.1097/HTR.0000000000000837 https://pubmed.ncbi.nlm.nih.gov/36594861/

29. Togher L, Douglas J, Turkstra LS, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, part IV: cognitive-communication and social cognition disorders. J Head Trauma Rehabil. 2023;38(1):65-82. doi:10.1097/HTR.0000000000000835 https://pubmed.ncbi.nlm.nih.gov/36594860/

30. Schneider ALC, Huie JR, Boscardin WJ, et al. Cognitive outcome 1 year after mild traumatic brain injury: results from the TRACK-TBI study. Neurology. 2022;98(12):e1248-e1261. doi:10.1212/WNL.0000000000200041 https://pubmed.ncbi.nlm.nih.gov/35173018/

31. Cicerone KD. Attention deficits and dual task demands after mild traumatic brain injury. Brain Inj. 1996;10(2):79-89. doi:10.1080/026990596124566 https://pubmed.ncbi.nlm.nih.gov/8696318/

32. Stuss DT. Functions of the frontal lobes: relation to executive functions. J Int Neuropsychol Soc. 2011;17(5):759-765. doi:10.1017/S1355617711000695 https://pubmed.ncbi.nlm.nih.gov/21729406/

33. Bechara A, Damasio AR, Damasio H, Anderson SW. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition. 1994;50(1-3):7-15. doi:10.1016/0010-0277(94)90018-3 https://pubmed.ncbi.nlm.nih.gov/8039375/

34. Howell J, Costanzo RM, Reiter ER. Head trauma and olfactory function. World J Otorhinolaryngol Head Neck Surg. 2018;4(1):39-45. doi:10.1016/j.wjorl.2018.02.001 https://pmc.ncbi.nlm.nih.gov/articles/PMC6051255/

35. Tenney JR, Gloss D, Arya R, et al. Practice guideline: use of quantitative EEG for the diagnosis of mild traumatic brain injury: report of the Guideline Committee of the American Clinical Neurophysiology Society. J Clin Neurophysiol. 2021;38(4):287-292. doi:10.1097/WNP.0000000000000853 https://pubmed.ncbi.nlm.nih.gov/34038930/
36. Brooks BL, Iverson GL. Comparing actual to estimated base rates of "abnormal" scores on neuropsychological test batteries: implications for interpretation. Arch Clin Neuropsychol. 2010;25(1):14-21. doi:10.1093/arclin/acp100 https://pubmed.ncbi.nlm.nih.gov/20015965/
37. Brooks BL, Holdnack JA, Iverson GL. To change is human: "abnormal" reliable change memory scores are common in healthy adults and older adults. Arch Clin Neuropsychol. 2016;31(8):1026-1036. doi:10.1093/arclin/acw079 https://pubmed.ncbi.nlm.nih.gov/27680088/
38. Andelic N, Røe C, Brunborg C, et al. Frequency of fatigue and its changes in the first 6 months after traumatic brain injury: results from the CENTER-TBI study. J Neurol. 2021;268(1):61-73. doi:10.1007/s00415-020-10022-2 https://pmc.ncbi.nlm.nih.gov/articles/PMC7815577/
39. Wilson L, Horton L, Kunzmann K, et al. Understanding the relationship between cognitive performance and function in daily life after traumatic brain injury. J Neurol Neurosurg Psychiatry. 2021;92(4):407-417. doi:10.1136/jnnp-2020-324492 https://jnnp.bmj.com/content/92/4/407
40. Mehta S, Devito L, Patsakos EM, et al. Updated Canadian Clinical Practice Guideline for the Rehabilitation of Adults With Moderate to Severe Traumatic Brain Injury: mental health recommendations. J Head Trauma Rehabil. 2024;39(5):359-368. doi:10.1097/HTR.0000000000000961 https://pubmed.ncbi.nlm.nih.gov/39256157/
41. Larsen SE, Vasterling JJ. Traumatic brain injury and PTSD. National Center for PTSD, US Department of Veterans Affairs [Internet]. [cited 2026 Oct 25]. Available from: https://www.ptsd.va.gov/professional/treat/cooccurring/tbi_ptsd_vets.asp

Chapter 12 Pain after traumatic brain injury

Chapter overview

Pain is the most common lasting consequence of a traumatic brain injury, and the least understood by the people who have to evaluate it. About half of all patients with a brain injury develop chronic pain, and the proportion is higher, not lower, after a mild injury than after a severe one. Headache leads, but neck, back, and widespread body pain follow closely, and the different kinds of pain usually coexist in the same patient.

This chapter explains what is known about that pain and what it means for the reader who must weigh it. Three ideas organize it. Pain comes in three mechanistic types: nociceptive, neuropathic, and nociplastic. The third, in which the nervous system's own pain processing has been altered, can contribute to pain after brain injury. Central sensitization provides a biological explanation for pain that is widespread, disproportionate to visible damage, and resistant to treatments aimed at the body part that hurts. The clinical symptom patterns discussed here are compatible with that mechanism, but do not directly measure it or establish that it predominates. Pain also tracks with the cognitive, sleep, and mood consequences of the injury and makes each of them worse. It belongs within the post-concussive picture.

The chapter closes by explaining how pain is evaluated in a medicolegal setting and what the evidence says about the questions commonly raised.

Why pain belongs in a book about brain injury

A reader coming to this subject from the outside might expect pain to be the least neurological of the consequences of a brain injury. The brain itself has no pain receptors. Headache after a blow to the head seems to need no explanation. Neck and back pain after a collision seem to belong to the orthopedist. On that view, pain is a side issue that happens to accompany a brain injury and should be evaluated and compensated separately.

The evidence does not support that view. Pain is among the most common and most persistent consequences of a brain injury. In the largest cohort studied, patients with current chronic pain did worse on every measure of function than patients without it [1]. Treating physicians consistently identify pain as one of the main obstacles to rehabilitation and return to work. It also behaves in ways that an orthopedic explanation cannot account for: it is more common after mild injuries than after severe ones, it spreads to parts of the body that were never injured, and it responds poorly to

treatments directed at the painful part. Those three facts point to the brain, and the rest of this chapter explains why.

For the reader whose task is to evaluate such a case, the practical consequence is this. A patient with a mild traumatic brain injury who reports headache, neck pain, back pain, and diffuse aching that has lasted for years, and whose imaging and orthopedic examinations are unremarkable, is not presenting an implausible story. That patient is presenting the most common story there is.

How common pain is after a brain injury

The most frequently cited figures come from a systematic review published in the Journal of the American Medical Association in 2008, which pooled 23 studies and more than 4,000 patients [2]. Chronic pain was present in 51.5 percent of civilians with a traumatic brain injury and in 43.1 percent of veterans. Chronic headache alone affected 57.8 percent of the patients in the studies that measured it. Among civilians, the prevalence of chronic pain was 75.3 percent after mild injury and 32.1 percent after moderate or severe injury (Figure 12.1). The review also found that the association between brain injury and chronic pain held independently of post-traumatic stress disorder and depression, so the pain is not accounted for by the psychological reaction to the event alone.

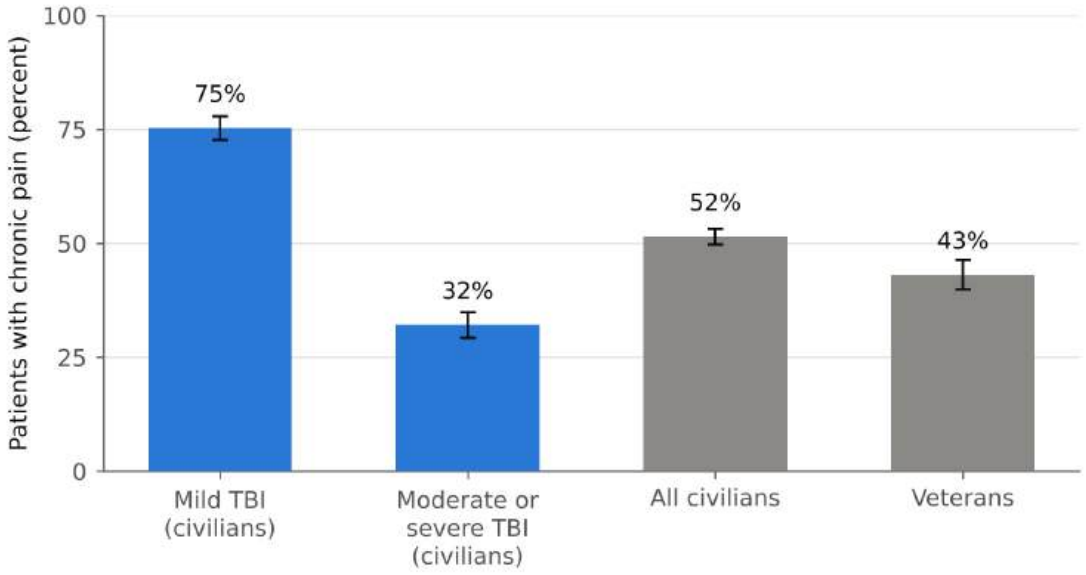


Figure 12.1. How common chronic pain is after traumatic brain injury. Pooled figures from a systematic review of 23 studies and 4,206 patients [2]. The bars show the percentage of patients with chronic pain; the short vertical lines are the 95 percent confidence intervals, that is, the range within which the true figure almost certainly lies. Chronic pain was more than twice as common after mild injury as after moderate or severe injury.

The mild-to-severe reversal surprised many readers of that review, and it has been confirmed repeatedly since. An earlier Australian study found chronic pain in 58 percent of patients with mild injury and 52 percent with moderate or severe injury [3]. The Traumatic Brain Injury Model Systems is a federally funded network that follows patients with moderate and severe injuries after inpatient rehabilitation. Its 2024 report found current chronic pain in 46 percent of participants and resolved past pain in another 14 percent. About 60 percent had therefore lived with chronic pain at some point after the injury [1]. Even among severe injuries, pain is the rule rather than the exception; after mild injuries it affects three patients in four.

Why should a lighter injury produce more pain? Several explanations are plausible and they are not mutually exclusive. Patients with severe injuries may have reduced awareness of pain or reduced ability to report it. Patients with mild injuries are more often injured in collisions and falls that also strain the neck and spine. The diffuse, microscopic injury that characterizes a concussion can also disturb the brain networks that regulate pain without destroying them. The system remains intact enough to generate pain but is no longer able to dampen it. That last mechanism is developed below.

Headache is the single most common pain complaint. In a prospective study of 212 patients enrolled within a week of a mild traumatic brain injury, 54 percent reported new or worse headache in the first week, 62 percent at three months, 69 percent at six months, and 58 percent at one year. Over the whole year, 91 percent had new or worse headache at some point, against a pre-injury headache history in 18 percent [4] (Figure 12.2). The headache did not fade with time in the way the public, and many physicians, expect. In the companion study of 378 patients with moderate and severe injuries, migraine or probable migraine was again the most common pattern [5]. A 2023 meta-analysis of civilian studies from the preceding 14 years found persistent post-traumatic headache, meaning headache still present three months or more after the injury, in a pooled 47 percent of patients, with little difference between mild and more severe injuries and little change between three months and three years [6].

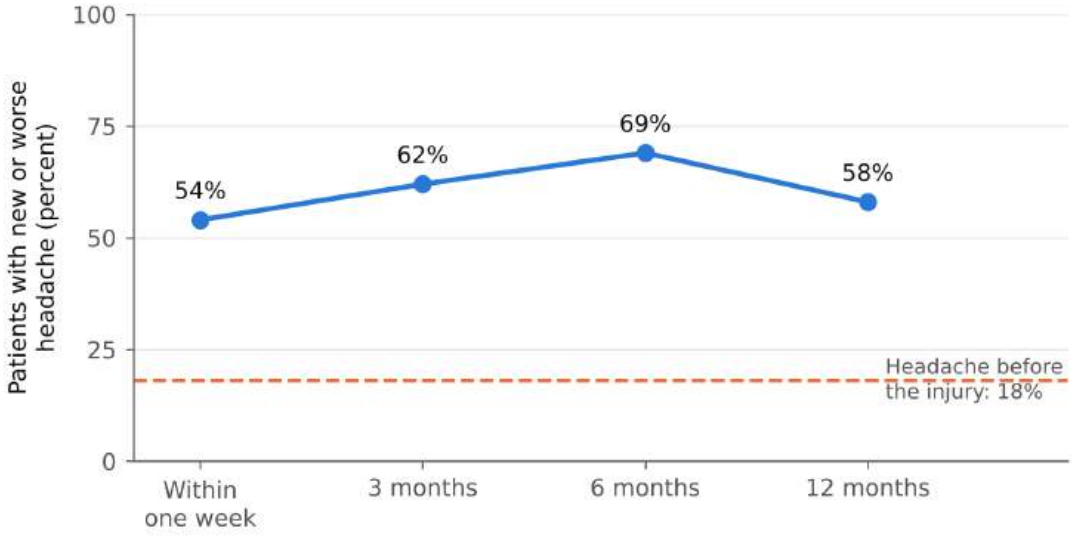


Figure 12.2. Headache after mild traumatic brain injury during the first year. Percentage of 212 patients with new or worse headache at each follow-up point, from a prospective study at a level 1 trauma center [4]. The dashed line is the 18 percent who had a headache problem before the injury. Over the whole year, 91 percent had new or worse headache at some point. The headache did not fade over the first year; it was about as common at twelve months as in the first week.

Pain in the rest of the body is also common. Neck pain is nearly universal after collisions, because the forces that accelerate the head also strain the neck. Back and shoulder pain follow. And a substantial fraction of patients develop pain in regions that were never struck, a pattern the next sections explain.

Three kinds of pain

Pain is one word for three different things, and the distinction decides both what the pain means and what will treat it. The International Association for the Study of Pain recognizes three mechanisms, and the third was formally added only in 2017 [7] (Figure 12.3).

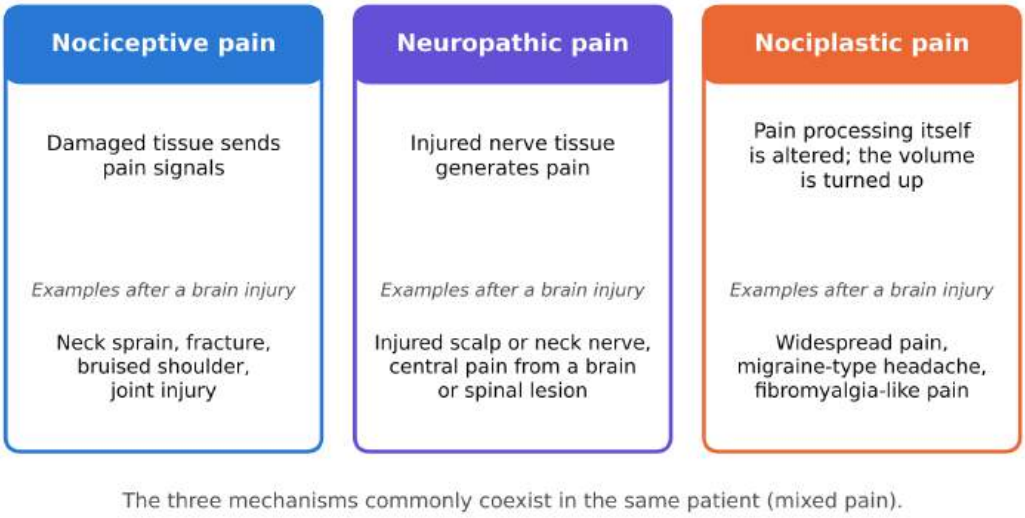


Figure 12.3. The three mechanisms of pain recognized by the International Association for the Study of Pain, with examples of each after a brain injury. Most patients with chronic pain after a brain injury have more than one mechanism at work at the same time. Original schematic.

Nociceptive pain is the pain of damaged tissue. A sprained neck, a bruised shoulder, a fractured rib, an inflamed joint: the injured tissue sends signals along pain nerves to the spinal cord and brain, and the pain is felt where the damage is. This is the pain that anti-inflammatory drugs, rest, physical therapy, and local injections are designed for, and when the tissue heals the pain is expected to stop.

Neuropathic pain is pain caused by injury to the nervous system itself, in a nerve, the spinal cord, or the brain. It is often burning, electric, or shooting, and it can be felt in an area that is numb to touch. After a brain injury it is less common than the other two kinds, but it occurs: a scalp nerve crushed in the impact, a neck nerve stretched in the collision, or, rarely, a true central pain syndrome from a lesion in the brain's own pain pathways. A careful study of such patients found that their pain began late, on average more than six months after the injury, and was almost always one-sided. Patients described it as pricking, throbbing, and burning. In the painful regions, testing showed

measurable loss of temperature sensation together with exaggerated responses to touch and repeated stimulation [8].

Nociplastic pain is the third kind, and the one that matters most here. The word was coined to describe pain that arises from altered processing of pain signals within the nervous system, without the ongoing tissue damage that would explain nociceptive pain and without the nerve lesion that would explain neuropathic pain [7,9]. The pain is real and the mechanism is biological; what has changed is the way the brain and spinal cord handle pain signals. Nociplastic pain tends to be felt in more than one region, to be out of proportion to any identifiable damage, and to travel with a recognizable set of companions: fatigue, poor sleep, trouble with memory and concentration, and low mood [9]. Fibromyalgia is the prototype. Migraine, tension-type headache, irritable bowel syndrome, and much chronic low back pain belong to the same family. Formal clinical criteria for grading nociplastic pain as possible or probable were published in 2021 [10].

The three mechanisms are not alternatives. A patient injured in a collision may have a nociceptive neck sprain, a neuropathic injury to an occipital nerve, and a nociplastic amplification of both, all at once, and the balance among them shifts over time. The World Health Organization's eleventh revision of the International Classification of Diseases took effect in 2022. It created, for the first time, a diagnosis of chronic post-traumatic pain: pain that develops or intensifies after a tissue injury and persists for more than three months, beyond the usual course of healing [11,12]. Within that framework the mechanism, or mixture of mechanisms, is what the clinician is asked to identify, because the mechanism determines the treatment [9].

Central sensitization: the brain turns up the volume

The process that underlies nociplastic pain, and that contributes to the other two kinds when they become chronic, is called central sensitization. The International Association for the Study of Pain defines it as an increased responsiveness of the pain-signaling neurons of the central nervous system to normal or below-threshold input. The idea was worked out over three decades, principally by Clifford Woolf, and is now part of the standard account of how chronic pain develops [13].

Pain signals from the body pass through relay stations in the spinal cord and the brainstem on their way to the parts of the brain that register pain. Those relay stations are not passive wires. They can be tuned up or down, and the brain itself sends signals down to them that normally hold the volume in check. After an injury, particularly after repeated or sustained pain signals, the relay neurons become hyperexcitable. They fire

more readily and for longer, and begin to respond to signals that are not pain signals at all. At the same time, the brain's descending control, the system that normally turns the volume down, becomes less effective. The result is a nervous system that produces more pain from a given input than it did before, and that can produce pain from inputs that would not have hurt at all.

The patient's experience can be described in specific terms (Figure 12.4). Allodynia is pain from a stimulus that should not be painful: a light touch, the weight of a shirt, a hairbrush, normal pressure on the neck muscles. Hyperalgesia is more pain than expected from a stimulus that is mildly painful: a firm press that most people would rate as uncomfortable becomes severe. A third phenomenon, called temporal summation or wind-up, is the progressive increase in pain when the same mild stimulus is repeated; a few taps that start as pressure end as pain. All three can be measured in a laboratory with calibrated stimuli, a set of methods called quantitative sensory testing, and all three have been documented in patients with pain after a brain injury [8,14].

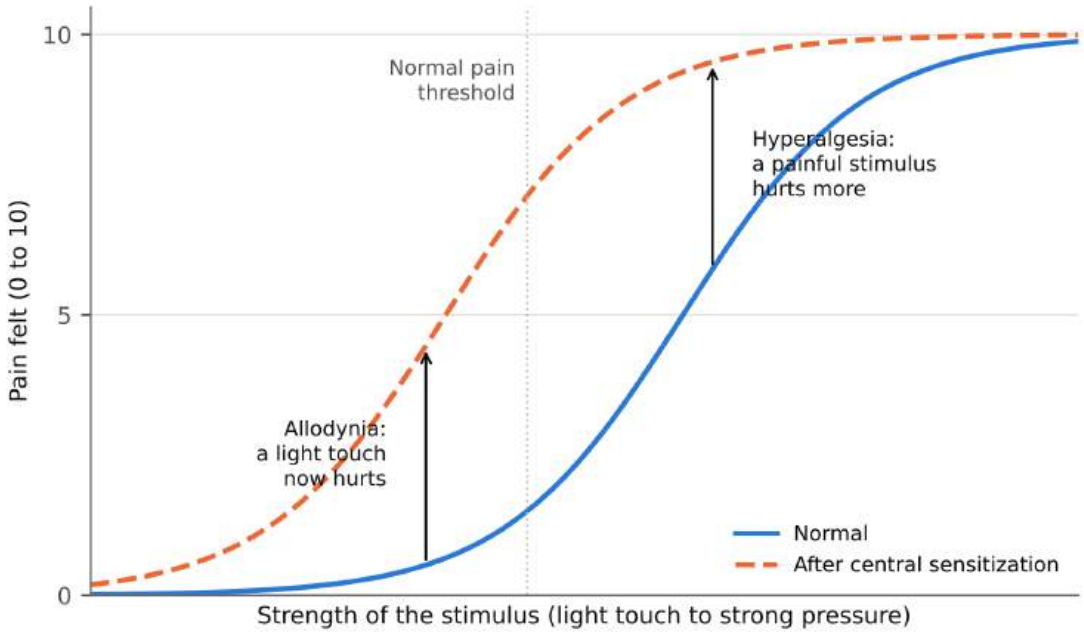


Figure 12.4. What central sensitization does to pain. The horizontal axis is the strength of a stimulus, from a light touch to strong pressure; the vertical axis is the pain the person feels. In a normal nervous system (solid line), light stimuli produce no pain and pain rises only past a threshold. After central sensitization (dashed line) the whole curve shifts: stimuli that were below the threshold now hurt (allodynia), and stimuli that were already painful hurt more (hyperalgesia). Original schematic; the curves are illustrative.

Why would a brain injury do this? Two lines of evidence converge. The first is anatomical. The brainstem centers that regulate pain include the periaqueductal gray

and the rostral ventromedial medulla. These centers, and the networks connecting them to the frontal cortex, are among the structures most vulnerable to the stretching and shearing forces of a concussion. Laboratory studies of brain injury show lasting loss of descending pain inhibition and heightened spinal pain signaling [15,16]. The second is clinical. Patients with mild traumatic brain injury and chronic post-traumatic headache were tested with quantitative sensory methods. Their descending pain inhibition, measured by a technique called conditioned pain modulation, was deficient compared with both healthy controls and brain-injured patients without headache. The weaker the inhibition, the more severe the headache [14]. In plain terms, the injured brain had lost part of its ability to turn pain down, and the patients with the least remaining ability had the most pain.

This is the biological content of a phrase patients and their families often use: it is as if the whole body hurts. For a substantial fraction of them it does, and the reason is in the nervous system rather than in the body parts that hurt.

Assessing widespread pain and associated symptoms

Quantitative sensory testing is a research tool and is not available in most clinics. What is available is a validated questionnaire approach that was developed for fibromyalgia and has since been applied across the range of chronic pain conditions.

The 2010 and 2016 criteria of the American College of Rheumatology for fibromyalgia abandoned the old tender point examination in favor of two scores [17]. The Widespread Pain Index counts the number of body regions, out of 19, in which the patient has had pain in the past week. It is usually completed on a body map; the Michigan Body Map is a validated version [18]. The Symptom Severity Scale rates the companions of nociplastic pain, fatigue, waking unrefreshed, and cognitive difficulty, each from 0 to 3, and adds points for headache, abdominal pain, and depression over the preceding six months, for a total of 0 to 12. Fibromyalgia is diagnosed when the Widespread Pain Index is 7 or more with a Symptom Severity Scale of 5 or more, or when the Widespread Pain Index is 4 to 6 with a Symptom Severity Scale of 9 or more, provided the pain has been present at a similar level for at least three months. The two scores can also be summed into a single 0 to 31 scale, sometimes called the fibromyalgia survey score or polysymptomatic distress scale. A total of 13 or more is used in survey research as corresponding to the diagnosis [17].

Applied to patients with other pain conditions, the same instrument characterizes widespread pain and associated symptoms, whether or not they carry a fibromyalgia label. A high score can suggest a contribution from altered pain processing, but does

not directly establish that mechanism. Patients with knee arthritis and high scores can have different treatment outcomes from those with the same arthritis and low scores. The clearest demonstration comes from orthopedic surgery. In a prospective study of patients undergoing knee or hip replacement, each one-point increase in the survey score predicted more opioid use after surgery, independently of the condition of the joint [19]. At six months, each one-point increase independently predicted less pain relief from the operation [20]. In a review for the Journal of the American Medical Association, Daniel Clauw, whose group did much of this work, made the general argument that the degree of centralization, not the degree of tissue damage, predicts how a patient will respond to treatments aimed at the periphery [21].

What our study showed

We and our colleagues applied this instrument to patients with mild traumatic brain injury and persistent post-concussion syndrome, and published the results in Pain Physician in 2025 [22]. The question was simple. Of the three kinds of pain, which one predominates in these patients?

The study was a retrospective review of the records of 223 patients with mild traumatic brain injury evaluated in Dr. Pappolla's outpatient neurology clinic between 2020 and 2023. The review excluded patients with chronic pain before the injury, those still within 90 days of it, those whose imaging showed a structural injury that would have placed them in a higher severity category, and those with another neurological condition that could have confounded the findings. Sixty-seven patients remained: 39 men and 28 women, average age 46, ethnically diverse. All had objective abnormalities on neurophysiological testing of the kind described in Chapter 7, providing evidence of neurological dysfunction alongside the clinical history. Post-concussive symptom burden was measured with the Rivermead Post-Concussion Symptoms Questionnaire. The Widespread Pain Index and Symptom Severity Scale characterized the distribution of pain and accompanying symptoms; they did not directly measure neuronal sensitization.

Three findings matter here. First, the group reported a substantial burden of widespread pain and associated symptoms. The mean combined score was 12.8, and 32 of the 67 patients, 48 percent, scored at or above 13, the fibromyalgia survey cutoff used in the paper. That cutoff alone does not establish an individual clinical diagnosis. Anyone with chronic pain before the injury had been excluded from this group. Second, the Widespread Pain Index correlated modestly with the total Rivermead score and both of its symptom groupings: patients reporting more widespread pain tended to report a greater post-concussive symptom burden. Third, neither age, nor sex, nor ethnicity, nor

the time elapsed since the injury showed a statistically significant association with the scores in this sample. The analysis did not follow changes in each patient's pain over time, so it cannot show whether the pain was fading.

The study has the limitations of its design, which the paper states: it was retrospective, the pain and symptom measures were self-reported, and many of the patients were involved in litigation. The neurophysiological abnormalities provide evidence of neurological dysfunction, but do not validate the pain ratings or remove response and recall bias. The data show an association between widespread pain and post-concussive symptom burden in this selected cohort. That pattern is compatible with a contribution from altered pain processing, but does not establish that nociplastic pain predominates, that central sensitization caused the symptoms, or that pain and the other symptoms share a single mechanism. Dr. Pappolla sees the practical concern repeatedly in clinical practice. A patient describes intense pain in one discrete place: the low back, the neck, or a shoulder. Treatment directed only at that place may be insufficient when pain involves a broader disturbance of pain processing. The clinician must assess that possibility alongside local sources of pain.

Post-traumatic headache

Headache deserves its own treatment because it is the most common pain after a brain injury and because it is governed by a specific classification. The International Classification of Headache Disorders, third edition, recognizes headache attributed to traumatic injury to the head as a secondary headache. It divides this into acute and persistent forms, with persistent headache defined as headache that continues more than three months after the injury [23]. The classification requires that the headache begin, or be reported to have begun, within seven days of the injury, of regaining consciousness, or of regaining the ability to sense and report pain. That seven-day interval is a formal classification criterion, and it has been challenged empirically. The classification's own appendix provides criteria for delayed-onset headache beginning between seven days and three months. The prospective studies cited above also show that headache prevalence rose, not fell, between the first week and six months [4]. Chapter 5 discusses the rule and the evidence against reading it mechanically. A headache first documented at week three therefore calls for careful attribution, not automatic exclusion.

The classification deliberately says nothing about what the headache feels like, and the clinical studies show why: post-traumatic headache does not have a single appearance. In the Lucas studies, when the headaches were classified by their features, migraine or probable migraine was the most common type at every time point,

accounting for up to 49 percent after mild injury, with tension-type headache next at up to 40 percent and cervicogenic headache, meaning headache arising from the neck, in a smaller fraction [4,5]. A 2021 review in Lancet Neurology described post-traumatic headache as a heterogeneous disorder with subphenotypes that respond differently to treatment. In the absence of trials specific to post-traumatic headache, clinicians generally treat it according to the primary headache it most resembles [24]. In practice that means treating a migraine-like post-traumatic headache as migraine, with migraine preventives and migraine-specific acute medications, and treating a tension-type pattern accordingly.

Two further points bear on evaluation. The first is medication overuse. A patient with daily headache who takes an over-the-counter analgesic every day can develop a second headache disorder, medication-overuse headache, that perpetuates the first. The classification sets the threshold at 15 or more days of simple analgesic use per month, or 10 or more days for combination analgesics, triptans, or opioids [23]. This is a common and reversible contributor to persistent post-traumatic headache and should be looked for in the medication history. The second is that migraine-type post-traumatic headache is itself a nociplastic condition. Migraine is a disorder of brain excitability, and its appearance for the first time after a concussion, in a patient who never had migraines, is one of the clearest clinical signs that the injury altered the brain's pain processing. For the reader it is also one of the clearest examples of a finding that is both real and invisible on imaging.

Pain that comes from the neck and the body

Not all pain after a head injury comes from the brain, and an evaluator who attributes everything to the brain will be as wrong as one who attributes nothing to it. The forces that produce a concussion in a collision are the same forces that strain the cervical spine, and neck injury is almost universal in those patients. The neck can produce headache of its own, through the convergence of neck nerves and head nerves in the upper spinal cord; it can produce dizziness, through disturbed position sense from the neck muscles; and it can produce the fogginess and fatigue that accompany any chronic pain. A well-known study of patients with persistent symptoms after sport concussion found that symptoms alone could not distinguish those whose problem was in the brain from those whose problem was in the neck or inner ear. The examination could [25]. For that reason, evaluation of every patient with persistent pain after a head injury should include examination of the neck and the vestibular and oculomotor testing described in Chapter 7. These assessments sort the mechanisms.

The sorting has a second purpose. Neck injury is itself a potent trigger of central sensitization. A controlled study of 161 patients with traumatic injuries found that fibromyalgia developed in 21.6 percent of those with a neck injury, compared with 1.7 percent of those with a leg fracture, a thirteen-fold difference. Fibromyalgia appeared on average about three months after the trauma. All patients were still working, and insurance claims were not more frequent among those who developed it [26]. A patient with both a concussion and a neck injury has two independent reasons to develop centralized pain, and that is the typical patient after a motor vehicle collision.

Pain and the other consequences of the injury

Pain does not sit beside the cognitive, sleep, and emotional consequences of a brain injury. It interacts with each of them, in both directions, and the interactions are large enough that they must be considered in any assessment of a patient's function.

Pain impairs cognition. Chronic pain consumes the resources on which attention, working memory, and processing speed depend. A large body of clinical and laboratory evidence shows that chronic pain by itself produces measurable deficits in those functions [27]. In a patient with a brain injury the two sources of impairment add, and separating them on a cognitive test is often impossible. This cuts in both directions in a contested case: cognitive findings may be attributed entirely to pain when the injury contributes, or entirely to the injury when pain contributes. The accurate statement is usually that both contribute and that the pain is itself a consequence of the injury.

Pain disturbs sleep, and poor sleep lowers the pain threshold, which is one of the most robust findings in pain research. In the Traumatic Brain Injury Model Systems cohort, current chronic pain was associated with worse sleep quality and with higher levels of depression, anxiety, and post-traumatic stress symptoms, with effect sizes in the medium to large range after adjustment for demographics and injury severity [28]. In an earlier prospective study of patients followed for a year after moderate and severe injury, pain and depression frequently co-occurred. A patient with pain at one year was nearly eight times more likely to be depressed than a patient without it [29].

Depression and anxiety amplify pain, and patients with chronic pain are frequently depressed and anxious. The pain is therefore sometimes read as a manifestation of the mood disorder. The 2008 systematic review addressed this directly and found that the association between brain injury and chronic pain was independent of both post-traumatic stress disorder and depression [2]. Our study also found an association between widespread pain and post-concussive symptom burden, but did not establish that this relationship was independent of mood symptoms [22]. Mood disorders after a

brain injury can accompany and amplify pain. The adjusted findings in the systematic review indicate that they do not account for the entire association between brain injury and chronic pain.

Why treatment so often fails

The clinical history of a patient with chronic pain after a mild traumatic brain injury frequently contains a long list of treatments directed at the painful place, none of which worked: anti-inflammatory drugs, muscle relaxants, physical therapy for the neck, epidural steroid injections, facet joint injections, radiofrequency ablation, sometimes surgery. An evaluator reading that history may conclude that the pain is absent or exaggerated, or that the treatments were aimed at the wrong target.

The last is usually correct, and the reason has already been given. Nociplastic pain, by definition, is not sustained by the tissue that hurts. Treatments that act on that tissue, whether drugs, needles, or surgery, address a mechanism that is not the main one, and nociplastic pain responds poorly to all of them [9]. The orthopedic data make the point concrete: the more centralized a patient's pain, the less relief the patient gets from an operation on the joint that hurts, even when the joint is unquestionably damaged [20]. A record full of failed local treatments is therefore not evidence against the pain. It is evidence about its mechanism.

Opioids deserve a specific comment. They are poorly effective for nociplastic pain, they impair cognition and sleep that are already impaired, and sustained use can itself increase pain sensitivity. Their presence in the record usually reflects the absence of a better option rather than a judgment that the pain is nociceptive.

What does help is treatment aimed at the nervous system. The evidence base in brain injury specifically is thin, and no consensus guideline exists for post-traumatic pain [16]. Clinicians therefore borrow from fibromyalgia and migraine, where the evidence is substantial. Treatments include centrally acting medications such as the serotonin-norepinephrine reuptake inhibitors, tricyclic antidepressants, and gabapentinoids; treatment of the headache according to its phenotype, including migraine preventives and, where indicated, the newer agents that block calcitonin gene-related peptide; treatment of sleep; graded aerobic exercise; and cognitive and behavioral approaches to pain [16,21,24]. Dr. Pappolla's clinical position, developed at length elsewhere, is to treat a large central component first. Procedures directed at a specific painful structure should be reserved for pain that remains, and remains concordant with that structure, after the central component has been addressed. The same position was argued in the Pain Physician paper and reflects the general direction of the pain literature [21,22].

How pain is evaluated in a medicolegal setting

There is no objective test for pain. Pain is an experience, and all measures of it ultimately rest on the patient's report. This places pain in a different category from the eye movement, balance, and evoked potential abnormalities described in Chapter 7. Those are recorded by instrument and depend far less on the patient's report. It does not place pain outside the reach of evaluation. Clinicians evaluate pain every day, and there are recognized ways to do it well.

The history comes first, and in a medicolegal setting its most important feature is the timeline. Pain that began with the injury or in the weeks after it, that has been documented consistently in the records since, and that has not been attributed by any treating physician to another cause, satisfies the first requirement of causation, which is temporal. Pre-injury records matter as much as post-injury records. A patient with no pain complaints in five years of prior records who develops widespread pain after a collision presents a different picture from one with a long prior history. The comparison should be explicit.

The body map is the second element. A patient who marks pain in a few regions related to the injury on one visit, and in many regions on another, needs a review of what changed. The body map and Symptom Severity Scale help document that change, but cannot by themselves distinguish evolving pain mechanisms from inconsistent reporting. Widespread pain that is stable across visits and across examiners, and that is accompanied by the recognized companions of nociplastic pain, is a coherent clinical picture. Widespread pain that appears for the first time at a medicolegal examination, in a patient whose treating records describe localized pain, requires an explanation of that difference.

The examination contributes in two ways. It sorts the mechanisms, as described above, identifying the nociceptive and neuropathic contributions in the neck, the shoulders, the scalp nerves, and the joints, and documenting allodynia and hyperalgesia where they are present. It also establishes the context for the pain report. A patient with abnormal eye movement recordings, abnormal balance testing, prolonged evoked potential latencies, and a documented new migraine pattern has objective evidence of neurological dysfunction. Widespread pain in that patient may involve altered pain processing, but those accompanying findings do not identify its mechanism by themselves. This is the convergence principle of Chapter 7 applied to pain. The pain report cannot be verified by instrument, but its company can.

Finally, the evaluator should resist two temptations. The first is to call pain psychogenic because its mechanism is central. Central sensitization is a physiological process with

a defined neurobiology. Calling it psychological is a category error; the fact that mood disorders amplify it does not make it one of them. The second is to call pain feigned because it is widespread, disproportionate, or unresponsive to treatment. Each of those features is the expected behavior of nociplastic pain, and each has been documented in populations studied outside any litigation context. Equally, none of those features verifies the pain; verification, to the extent it is possible, comes from the structure of the evaluation described above.

Questions commonly raised about pain after brain injury, and what the evidence says

The same questions recur about pain complaints after brain injury, and each has a legitimate core. The evidence sometimes supports the complaint and sometimes does not. The evaluator's task is to say which.

The injury was mild, so the pain cannot be severe. The assumption is reasonable on its face, and the evidence runs the other way. Chronic pain is more common after mild brain injury than after severe brain injury in every large civilian series [2,3]. Severity of injury, as graded by the acute measures described in Chapter 4, is not a predictor of the severity of chronic pain.

The pain comes from the neck, not the brain. Often both. Neck injury accompanies most concussions sustained in collisions and is itself a cause of headache, dizziness, and central sensitization [25,26]. The presence of a neck injury does not exclude a brain injury, and the evaluator's task is to sort the contributions rather than choose one and discard the other. Whichever source predominates, the mechanism of the centralized pain is the same.

Imaging is normal, so there is nothing to cause pain. For nociceptive pain the question is a fair one, because imaging often shows the source. Imaging shows structure. Central sensitization is a change in function, and no structural scan detects it. Normal imaging is the expected finding in mild traumatic brain injury, as Chapter 3 explains, and it is the expected finding in fibromyalgia, migraine, and every other nociplastic pain condition. The keyhole principle of Chapter 7 applies.

Widespread pain means fibromyalgia, and fibromyalgia is psychological. The first half is often correct and the second half is not. Fibromyalgia is the prototype of nociplastic pain, has a defined neurobiology, and is classified in the World Health Organization's ICD-11 as a chronic primary pain condition, a disease in its own right in the classification's framing [9,11]. Its development after physical trauma has support from the controlled study of neck injury described above [26]. Whether trauma causes

fibromyalgia is still debated; whether fibromyalgia is a real condition has not been, in the scientific literature, for many years.

The patient is depressed, and depression causes pain. The point is legitimate as far as it goes. Depression amplifies pain and pain causes depression; the two travel together after brain injury, as the Model Systems data show [28,29]. But the association between brain injury and chronic pain survives adjustment for depression and post-traumatic stress disorder [2]. Both conditions are commonly consequences of the injury, and the presence of one does not account for the other.

Nothing has helped, so the pain is not real. A record of failed treatment is a fair thing to notice, and what it shows depends on what was tried. Failed treatment directed at the painful body part is the expected course of nociplastic pain and is evidence of its mechanism, not of its absence [9,20]. The relevant question is whether treatment directed at the nervous system was ever tried.

The patient is in litigation. The point is legitimate and should be met directly rather than dismissed. Pain is self-reported, and self-report can be influenced by incentive. The evaluation described above helps assess the report in context. It brings together a documented timeline, consistency across visits and examiners, a body map read alongside symptom severity measures, and a clinical examination that sorts the possible mechanisms. Objective neurophysiological abnormalities can document accompanying neurological dysfunction, but do not independently validate pain severity or establish that incentive has had little influence. Litigation alone is not evidence that the pain is feigned. The evaluator should explain the support for the report, any inconsistencies, and the uncertainty that remains.

What this chapter does not claim

Pain after a brain injury is common, can involve several mechanisms including altered central pain processing, and often accompanies the other consequences of the injury. None of that means that every pain complaint after a head injury is caused by the brain injury, or that pain cannot be exaggerated, or that the evaluation can dispense with the ordinary disciplines of history, examination, and record review. The common objections to a pain complaint are that it is too widespread, too severe for the injury, too resistant to treatment, or too invisible on imaging to be real. Those features can occur with nociplastic pain after brain injury. Taken by themselves, they do not count against the complaint; treating them that way mistakes a possible presentation of nociplastic pain for evidence of its absence.

Fast take: main points from chapter 12

• About half of all patients with a traumatic brain injury develop chronic pain, and the prevalence is higher after mild injury (75 percent in pooled civilian data) than after moderate or severe injury (32 percent) [2].

• Headache is the most common pain. After mild injury, new or worse headache affects more than half of patients throughout the first year and 91 percent at some point in it; migraine is the most common pattern [4].

• Pain has three mechanisms, nociceptive (tissue damage), neuropathic (nerve injury), and nociplastic (altered pain processing), and they can coexist. Nociplastic pain can contribute after brain injury, but the symptom-score study does not establish that it predominates.

• Central sensitization is the process behind nociplastic pain. It produces allodynia, hyperalgesia, and wind-up, it is measurable, and brain-injured patients with chronic headache have demonstrable loss of the brain's own pain inhibition [13,14].

• The Widespread Pain Index and Symptom Severity Scale measure pain distribution and associated symptoms, not neuronal sensitization directly. In our series of 67 patients with persistent post-concussion syndrome, 48 percent scored at or above the fibromyalgia survey cutoff used in the paper, and widespread pain correlated with post-concussive symptom burden. These associations do not establish the underlying mechanism [22].

• Neck injury accompanies most concussions, produces headache and dizziness of its own, and is an independent trigger of central sensitization; the examination, not the symptoms, sorts the mechanisms [25,26].

• Pain, cognition, sleep, and mood interact after brain injury, but the association between brain injury and chronic pain is independent of depression and post-traumatic stress disorder [2,28].

• Nociplastic pain responds poorly to treatments aimed at the painful body part. A record of failed injections and procedures is evidence about the mechanism of the pain, not against its existence [9,20].

• There is no objective test for pain, but there is a disciplined way to evaluate it: timeline, pre-injury records, body map over time, an examination that sorts mechanisms, and convergence with objective neurophysiological findings.

• The questions commonly raised about pain complaints (mild injury, neck not brain, normal imaging, fibromyalgia is psychological, depression, failed treatment, litigation) each have a legitimate core and an answer in the evidence, and the strongest answer is the structure of the evaluation itself.

References

1. Harrison-Felix C, Sevigny M, Beaulieu CL, et al. Characterization and treatment of chronic pain after traumatic brain injury: comparison of characteristics between individuals with current pain, past pain, and no pain: a NIDILRR and VA TBI Model Systems collaborative project. J Head Trauma Rehabil. 2024;39(1):5-17. doi:10.1097/HTR.0000000000000910
2. Nampiaparampil DE. Prevalence of chronic pain after traumatic brain injury: a systematic review. JAMA. 2008;300(6):711-719. doi:10.1001/jama.300.6.711
3. Lahz S, Bryant RA. Incidence of chronic pain following traumatic brain injury. Arch Phys Med Rehabil. 1996;77(9):889-891.
4. Lucas S, Hoffman JM, Bell KR, Dikmen S. A prospective study of prevalence and characterization of headache following mild traumatic brain injury. Cephalalgia. 2014;34(2):93-102. doi:10.1177/0333102413499645
5. Lucas S, Hoffman JM, Bell KR, Walker W, Dikmen S. Characterization of headache after traumatic brain injury. Cephalalgia. 2012;32:600-606.
6. Herrero Babiloni A, Bouferguene Y, Exposto FG, et al. The prevalence of persistent post-traumatic headache in adult civilian traumatic brain injury: a systematic review and meta-analysis on the past 14 years. Pain. 2023;164(12):2627-2641. doi:10.1097/j.pain.0000000000002949
7. Kosek E, Cohen M, Baron R, et al. Do we need a third mechanistic descriptor for chronic pain states? Pain. 2016;157(7):1382-1386. doi:10.1097/j.pain.0000000000000507
8. Ofek H, Defrin R. The characteristics of chronic central pain after traumatic brain injury. Pain. 2007;131(3):330-340. doi:10.1016/j.pain.2007.06.015
9. Fitzcharles MA, Cohen SP, Clauw DJ, Littlejohn G, Usui C, Häuser W. Nociplastic pain: towards an understanding of prevalent pain conditions. Lancet. 2021;397(10289):2098-2110. doi:10.1016/S0140-6736(21)00392-5
10. Kosek E, Clauw D, Nijs J, et al. Chronic nociplastic pain affecting the musculoskeletal system: clinical criteria and grading system. Pain. 2021;162(11):2629-2634. doi:10.1097/j.pain.0000000000002324
11. Treede RD, Rief W, Barke A, et al. Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the International Classification of Diseases (ICD-11). Pain. 2019;160(1):19-27. doi:10.1097/j.pain.0000000000001384

12. Schug SA, Lavand'homme P, Barke A, Korwisi B, Rief W, Treede RD; IASP Taskforce for the Classification of Chronic Pain. The IASP classification of chronic pain for ICD-11: chronic postsurgical or posttraumatic pain. Pain. 2019;160(1):45-52. doi:10.1097/j.pain.0000000000001413
13. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152(3 Suppl):S2-S15. doi:10.1016/j.pain.2010.09.030
14. Defrin R, Riabinin M, Feingold Y, Schreiber S, Pick CG. Deficient pain modulatory systems in patients with mild traumatic brain and chronic post-traumatic headache: implications for its mechanism. J Neurotrauma. 2015;32(1):28-37. doi:10.1089/neu.2014.3359
15. Irvine KA, Clark JD. Chronic pain after traumatic brain injury: pathophysiology and pain mechanisms. Pain Med. 2018;19(7):1315-1333. doi:10.1093/pm/pnx153
16. Chen Q, Bharadwaj V, Irvine KA, Clark JD. Mechanisms and treatments of chronic pain after traumatic brain injury. Neurochem Int. 2023;171:105630. doi:10.1016/j.neuint.2023.105630
17. Wolfe F, Clauw DJ, Fitzcharles MA, et al. 2016 revisions to the 2010/2011 fibromyalgia diagnostic criteria. Semin Arthritis Rheum. 2016;46(3):319-329. doi:10.1016/j.semarthrit.2016.08.012
18. Brummett CM, Bakshi RR, Goesling J, et al. Preliminary validation of the Michigan Body Map. Pain. 2016;157(6):1205-1212. doi:10.1097/j.pain.0000000000000506
19. Brummett CM, Janda AM, Schueller CM, et al. Survey criteria for fibromyalgia independently predict increased postoperative opioid consumption after lower-extremity joint arthroplasty: a prospective, observational cohort study. Anesthesiology. 2013;119(6):1434-1443.
20. Brummett CM, Urquhart AG, Hassett AL, et al. Characteristics of fibromyalgia independently predict poorer long-term analgesic outcomes following total knee and hip arthroplasty. Arthritis Rheumatol. 2015;67(5):1386-1394. doi:10.1002/art.39051
21. Clauw DJ. Fibromyalgia: a clinical review. JAMA. 2014;311(15):1547-1555. doi:10.1001/jama.2014.3266
22. File C, Nader R, Villasante-Tezanos A, et al. Pain management in mild traumatic brain injury: central sensitization as a multispecialty challenge. Pain Physician. 2025;28:231-240.
23. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1-211. doi:10.1177/0333102417738202
24. Ashina H, Eigenbrodt AK, Seifert T, et al. Post-traumatic headache attributed to traumatic brain injury: classification, clinical characteristics, and treatment. Lancet Neurol. 2021;20(6):460-469. doi:10.1016/S1474-4422(21)00094-6

25. Leddy JJ, Baker JG, Merchant A, et al. Brain or strain? Symptoms alone do not distinguish physiologic concussion from cervical/vestibular injury. Clin J Sport Med. 2015;25(3):237-242. doi:10.1097/JSM.0000000000000128
26. Buskila D, Neumann L, Vaisberg G, Alkalay D, Wolfe F. Increased rates of fibromyalgia following cervical spine injury: a controlled study of 161 cases of traumatic injury. Arthritis Rheum. 1997;40(3):446-452. doi:10.1002/art.1780400310
27. Moriarty O, McGuire BE, Finn DP. The effect of pain on cognitive function: a review of clinical and preclinical research. Prog Neurobiol. 2011;93(3):385-404. doi:10.1016/j.pneurobio.2011.01.002
28. Hanks R, Ketchum JM, Peckham M, et al. Associations of chronic pain with psychosocial outcomes after traumatic brain injury: a NIDILRR and VA TBI Model Systems collaborative project. J Head Trauma Rehabil. 2024;39(1):18-30. doi:10.1097/HTR.0000000000000921
29. Sullivan-Singh SJ, Sawyer K, Ehde DM, et al. Comorbidity of pain and depression among persons with traumatic brain injury. Arch Phys Med Rehabil. 2014;95(6):1100-1105.

Part IV. Acute care and treatment

Chapter 13 Acute care: monitoring the injured brain and when surgery is needed

Chapter overview

Most of this book concerns injuries that leave no mark on a scan. This chapter concerns the minority in which a scan shows an injury, either at first or as the injury evolves. A patient who is awake and talking in the emergency department can deteriorate over the following hours or days from a hematoma that enlarges, a contusion that bleeds into itself, or a brain that swells, and a small number of patients classified as mild on arrival require neurosurgery. The chapter sets out how often that happens and who is at risk, the physiology that makes a rising pressure inside the skull dangerous, how intracranial pressure is measured and what the thresholds mean, the tiers of medical treatment that follow, and the evidence on when surgery is required, including evacuation of hematomas and decompressive craniectomy. It closes with what this means for the reader of a book about mild injury: the warning signs that send a discharged patient back to the hospital, and the fact that a normal CT and a same-day discharge do not exclude a later surgical complication.

Why a book about mild injury needs a chapter on acute care

The patient at the center of this book is awake, oriented, and talking. The diagnostic problem, described in Chapters 3 and 5, is that the injury is invisible to the examination and the scan. Acute care poses a different problem. A small number of patients who look well in the first hours have an injury that is still evolving. Some will need an operation.

The numbers are small but not negligible. In the derivation cohort of the Canadian CT Head Rule, 3,121 adults with a Glasgow Coma Scale score of 13 to 15 were studied prospectively; 254 (8 percent) had a clinically important brain injury on CT and 44 (1 percent) required a neurological intervention, four of whom died of the head injury [1]. In the New Orleans study that produced a parallel set of CT criteria, 1,429 patients with minor head injury and a GCS of 15 were enrolled; 93 (6.5 percent) had a positive CT and six (0.4 percent) required neurosurgery [2]. Among the patients whose first CT already shows an intracranial injury, the proportion who go on to deteriorate is higher. A systematic review and meta-analysis of 49 studies of patients with a GCS of 13 to 15 and a CT-identified injury found pooled rates of 11.7 percent for clinical deterioration, 3.5 percent for neurosurgical intervention, and 1.4 percent for death [3].

Under the classification in Chapter 4, these patients are mild on arrival. The classification does not change when the complication appears: the severity grade

describes the initial presentation, and the later course is recorded separately. That is one reason this chapter belongs in the book. The other is that every reader of a medical record will eventually meet a patient whose chart says “normal CT, discharged” on the first day and “craniotomy” on the third, and should know how that happens.

What can go wrong in the first hours and days

Three processes account for most deterioration after an injury that appeared mild.

Delayed intracranial hemorrhage. A patient whose first CT is negative can bleed later. In a nationwide population-based cohort of adults with minor head trauma and an initially negative emergency department CT, delayed intracranial hemorrhage occurred in 0.4 percent within fourteen days, and the patients in whom it occurred had higher mortality at one year [4]. Anticoagulant and antiplatelet drugs raise the risk of immediate bleeding substantially and of delayed bleeding modestly. In a prospective multicenter emergency department study, 37 of 724 patients taking warfarin (5.1 percent) had intracranial hemorrhage on the first CT and 4 of 687 with a negative first CT (0.6 percent) had a delayed hemorrhage; among patients taking clopidogrel, 12 percent had immediate hemorrhage and none of 243 had delayed hemorrhage [5]. A negative CT still requires discharge instructions about delayed bleeding and a plan for reassessment if symptoms worsen. It does not automatically require admission or another scan: ACEP advises against routine repeat imaging or admission solely because of anticoagulant or antiplatelet use when the initial CT shows no hemorrhage, the patient is at neurological baseline, and there is no other reason for extended monitoring [24].

Direct oral anticoagulants (DOACs), including apixaban, rivaroxaban, and dabigatran, require drug-specific management. A head injury alone is not a reason to give a reversal agent. When life-threatening bleeding, confirmed intracranial hemorrhage, or emergency surgery makes reversal necessary, treatment is urgent and depends on the drug, its last dose, renal function, and the likelihood that clinically important anticoagulation remains [25]. Idarucizumab reverses dabigatran; four-factor prothrombin complex concentrate (4F-PCC) is an off-label option for factor Xa inhibitors such as apixaban and rivaroxaban [25,27]. The history of andexanet alfa shows how this field can change. The FDA granted accelerated approval in 2018 for life-threatening or uncontrolled bleeding associated with apixaban or rivaroxaban. Later safety findings, including serious thrombotic events, changed the balance: the FDA concluded that the risks outweighed the benefits, and AstraZeneca ended US sales and manufacture for the US market on December 22, 2025 [26]. Reversal decisions therefore follow the current local emergency protocol and balance bleeding against thrombosis risk [25].

Hemorrhagic progression of a contusion. A contusion, a bruise of the brain surface, is not a fixed lesion. In a substantial fraction of patients, repeat imaging shows it enlarging over the first day or two. Small vessels in the injured zone fail, and new bleeding accumulates around the original contusion. This is hemorrhagic progression of contusion. The enlargement can convert a lesion that needed only observation into one that needs evacuation, and it is the usual reason a stable patient with a small contusion is admitted and rescanned rather than discharged [6].

Cerebral swelling. The brain can swell diffusely after an injury that produced no hematoma at all. In the classic series of Lobato and colleagues, 211 head-injured patients who talked after the injury and then deteriorated into coma were studied with CT; four of every five had a focal mass lesion, most often a contusion or intracerebral hematoma (42 percent), an epidural hematoma (22 percent), or a subdural hematoma (17 percent), and one in three died [7]. The remaining fifth had no focal lesion to evacuate; their deterioration came from diffuse swelling or ischemia. Diffuse swelling is a particular danger in children and young adults. Bruce and colleagues described a syndrome of malignant brain edema in children in whom a seemingly minor injury was followed within hours by diffuse cerebral swelling, loss of consciousness, and in some cases death, with CT showing small ventricles and compressed cisterns rather than a hematoma [8].

The patient who talks and then deteriorates is the acute-care counterpart of the patient this book describes elsewhere. In Chapter 5, Daniel walked out of an emergency department with a normal CT and a concussion that nobody recorded. The patients in this chapter walk out of the same department and come back, or do not get the chance to. Both are failures of the first encounter, and the second kind is the one that kills.

Pressure inside a closed box

A hematoma or a swollen brain becomes dangerous for a mechanical reason, described in the nineteenth century. The Monro-Kellie doctrine states that the skull is a rigid container of fixed volume holding three things: brain tissue, about 80 percent of the volume; blood within the cerebral vessels, about 10 percent; and cerebrospinal fluid, about 10 percent. Any increase in one must be matched by a decrease in another, or the pressure inside the container rises (Figure 13.1).

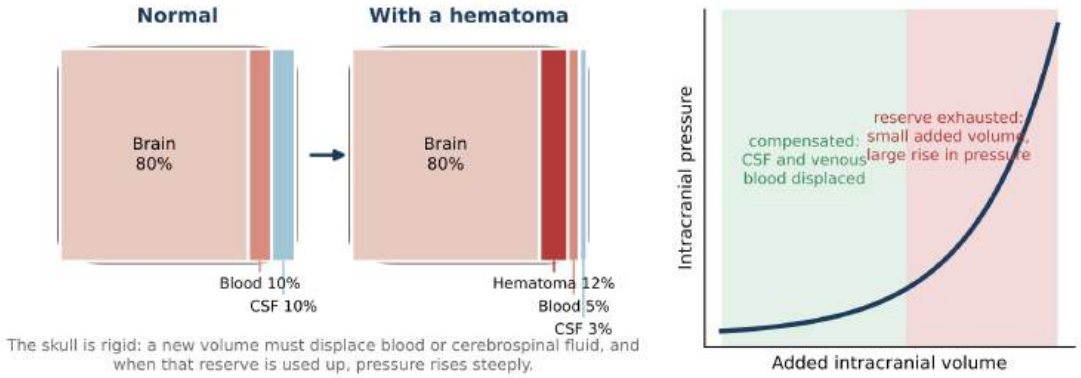


Figure 13.1. The Monro-Kellie doctrine and the pressure-volume curve. Left: the skull holds brain, blood, and cerebrospinal fluid in a fixed volume; a growing hematoma can at first be accommodated by displacing cerebrospinal fluid into the spinal canal and venous blood out of the head. Right: once those reserves are spent, each further increment of volume produces a steeper rise in pressure, which is why a patient can be stable for hours and then deteriorate in minutes. Original schematic.

The injured brain has two compensatory reserves. Cerebrospinal fluid can be displaced from the cranial vault into the spinal subarachnoid space, and venous blood can be squeezed out of the head. Together they allow a developing hematoma or focal edema of modest size to be accommodated with little rise in intracranial pressure. The reserves are finite. Once they are exhausted, the relation between added volume and pressure becomes exponential, and a small further increase in volume produces a large rise in pressure. That curve explains why a patient can talk and then deteriorate. The lesion was growing all along; pressure rose little until the reserve was gone.

Two consequences follow when the pressure climbs. Cerebral perfusion pressure is the pressure driving blood into the brain: mean arterial pressure minus intracranial pressure. As intracranial pressure rises, perfusion falls and the brain becomes ischemic. When regional pressure differences become large enough, brain tissue is displaced across the rigid dural folds or through the foramen magnum. These herniation syndromes compress the brainstem and can kill within minutes.

Normal intracranial pressure in a supine adult is 7 to 15 mmHg, lower in children (3 to 7 mmHg) and infants (1.5 to 6 mmHg), and it fluctuates with respiration, the pulse, and posture. The current Brain Trauma Foundation guidelines recommend treating intracranial pressure above 22 mmHg, revised from the earlier threshold of 20 mmHg on

the basis of outcome data, and maintaining cerebral perfusion pressure between 60 and 70 mmHg; values below 60 mmHg are associated with ischemia, and forcing perfusion pressure above 70 mmHg with fluids and vasopressors carries a risk of acute respiratory distress syndrome [9]. The threshold is a guideline rather than a rule: patients differ in cerebral compliance and autoregulation, the duration of an elevation matters as much as its height, and sustained elevation is more harmful than a brief spike.

Measuring intracranial pressure

Intracranial pressure cannot be read from the examination or the scan with any precision, and in a sedated, ventilated patient the examination is nearly blind. The decision to measure it directly rests on clinical assessment, imaging, and the likelihood that the measurement will change management.

The Brain Trauma Foundation guidelines recommend that patients with severe TBI (GCS 3 to 8) be managed with information from intracranial pressure monitoring, a recommendation supported by level IIB evidence of reduced in-hospital and two-week mortality [9]. The criteria used in practice come from earlier editions of the guidelines: monitoring for severe TBI with an abnormal CT (hematoma, contusion, swelling, herniation, or compressed basal cisterns), and for severe TBI with a normal CT when two of three features are present, age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure under 90 mmHg. The only randomized trial of monitoring itself, conducted in Bolivia and Ecuador, found no outcome difference between management guided by a monitor and management guided by serial imaging and examination [10]. That result has been read as showing the limits of a single number rather than the uselessness of measuring it. Large observational cohorts have found lower mortality with monitoring, particularly in the most severe injuries [11,12,13]. The international SYNAPSE-ICU cohort documented wide variation in practice between centers and countries and an association between invasive monitoring, more aggressive treatment, and lower six-month mortality in the more severely injured [11].

Monitoring is not indicated in mild TBI, and not routinely in moderate TBI (GCS 9 to 12). It is considered when a moderate injury deteriorates to severe, when imaging shows progressive mass effect or effaced cisterns, and when the patient cannot be examined serially because of sedation or prolonged surgery for other injuries. Severe coagulopathy is a relative contraindication to placing any device, and in a patient with a devastating injury for whom aggressive treatment is not planned, monitoring serves no purpose.

Two devices are in common use (Figure 13.2). The external ventricular drain, a catheter passed through a burr hole into the lateral ventricle and connected to a transducer and a drainage system, is the reference standard. It measures pressure and it also treats it: cerebrospinal fluid can be drained the moment pressure rises, which can be life-saving in an acute crisis, and the transducer can be re-zeroed at any time, so the measurement does not drift. Its disadvantages are real. Placement can be difficult when the ventricles are compressed or shifted, and malplacement rates of 10 to 40 percent are reported; the catheter passes through brain, with a small risk of tract hemorrhage; and infection, including ventriculitis, has been reported in 2 to 22 percent of patients depending on the series and the definition, although standardized insertion and maintenance protocols have brought rates below 2 percent at many centers [12]. The intraparenchymal monitor is a fiberoptic, strain-gauge, or pneumatic sensor placed about 2 cm into the brain through a small bolt in the skull. It is simpler to place regardless of ventricular anatomy and reads reliably in any position. It carries a lower infection risk and a hemorrhage risk of about 2.5 percent, with clinically significant bleeding in under 1 percent. It cannot drain fluid and reads local rather than global pressure. Most models cannot be recalibrated once placed and may drift. Comparisons of the two devices have been mixed: one prospective observational study found higher survival with ventricular drains [14], but selection bias is likely, since sicker patients are chosen for drains, and a large multicenter TRACK-TBI analysis found no difference in six-month functional outcome [13]. In practice a drain is chosen when drainage is likely to be needed, as with intraventricular hemorrhage, hydrocephalus, or significant swelling, and a parenchymal monitor when it is not.

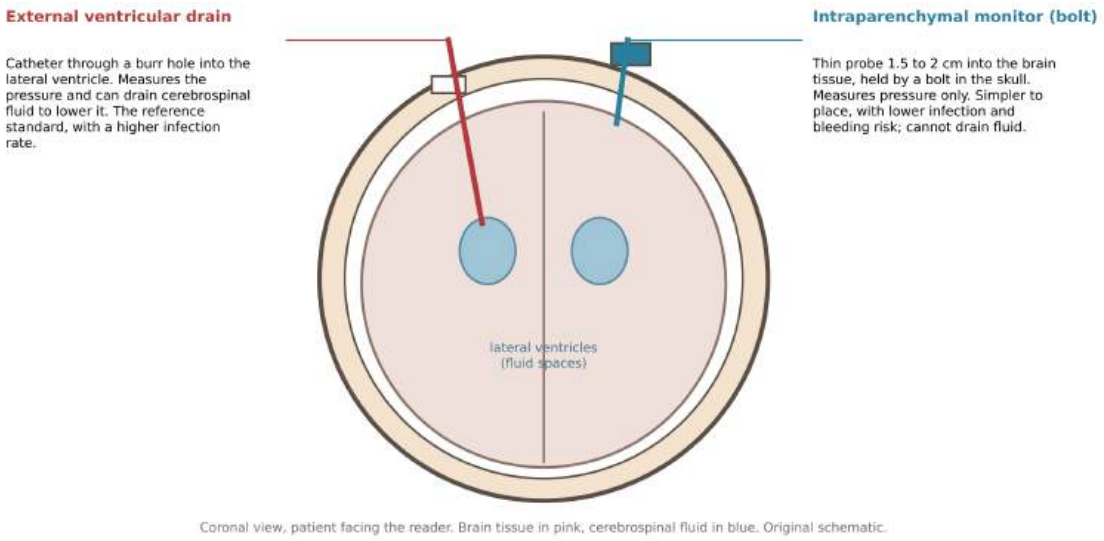


Figure 13.2. The two common ways of measuring intracranial pressure, shown on a coronal section. An external ventricular drain is passed through a burr hole into the lateral ventricle; it measures pressure and can drain cerebrospinal fluid to lower it. An intraparenchymal monitor is placed about 2 cm into the brain through a bolt in the skull; it measures pressure only. Original schematic.

Whichever device is used, its reading has to be trusted before it is acted on. A ventricular transducer must be zeroed at the level of the foramen of Monro, referenced to the tragus or the external auditory meatus, and re-leveled whenever the head moves; an unleveled transducer is the commonest source of a false reading. The fluid column of a working drain oscillates with the pulse and with breathing, and a flat trace means an occluded or malpositioned catheter, not a calm brain. The waveform itself carries information. Of its three components, the first (P1, arterial pulsation) is normally the tallest. When the second (P2, which reflects compliance) overtakes it, the brain is signaling that its reserve is nearly spent and that pressure may climb steeply with little further provocation. Plateau waves, sustained elevations to 50 to 100 mmHg lasting 5 to 20 minutes, mean that compensatory reserve is gone and that herniation is close [9,15].

A pressure reading alone describes one variable. Multimodal monitoring adds others. A probe placed beside the pressure monitor measures brain tissue oxygen tension, normally 25 to 35 mmHg. Values below 20 mmHg indicate cerebral hypoxia, and values below 15 mmHg are associated with worse outcome. Such episodes predict poor outcome independently of pressure and perfusion pressure [16]. Cerebral microdialysis samples the extracellular fluid for glucose, lactate, pyruvate, glutamate, and glycerol; a lactate-to-pyruvate ratio above 40 with low glucose defines a metabolic crisis that can occur with normal pressures. Continuous electroencephalography detects seizures in a sedated or comatose patient: in the study that established the point, seizures occurred

in 22 percent of monitored patients with moderate to severe injury, more than half of them with no visible convulsion, and status epilepticus was uniformly fatal [17]; seizures and periodic discharges after trauma are accompanied by metabolic crisis on microdialysis [18]. These modalities are not available everywhere, and microdialysis remains largely a research tool, but together they explain why a patient can be in trouble with a normal pressure and why a pressure elevation does not always demand escalation [15].

Treating the pressure, in tiers

Treatment of raised intracranial pressure proceeds in tiers, from measures that cost almost nothing to measures that carry their own risk (Figure 13.3).

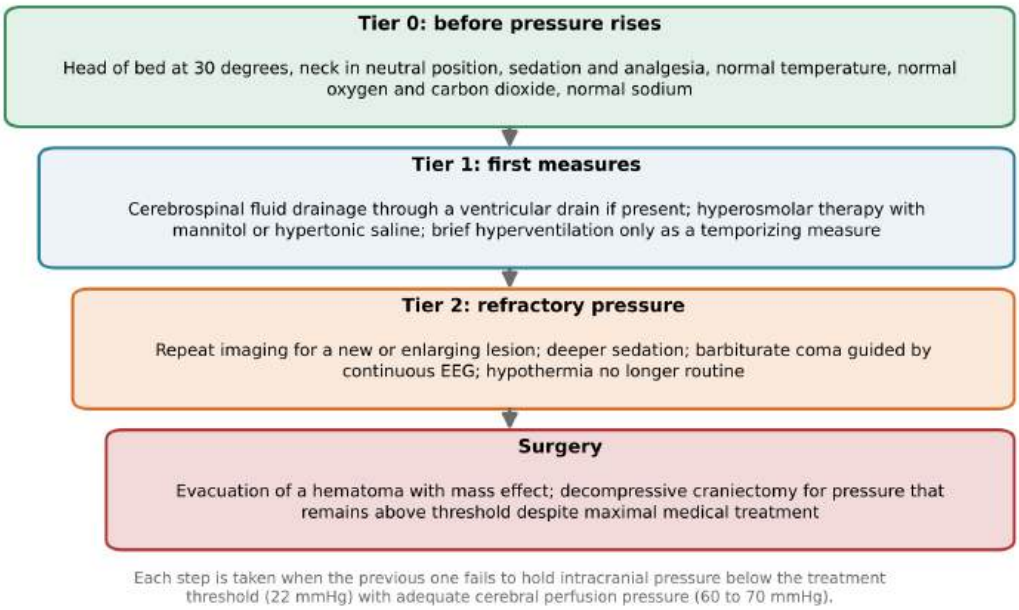


Figure 13.3. The tiers of treatment for raised intracranial pressure. Each tier is tried before the next, and a persistent rise despite the measures of a given tier is the signal to escalate, to re-image, and, at the top of the ladder, to operate. Original schematic drawn from the management sequence in the text.

The first tier is physiological housekeeping. The head of the bed is raised to 30 degrees and the neck kept in neutral alignment so that venous blood can leave the head. Pain and agitation are controlled with analgesia and sedation, because both raise pressure. Fever is treated, since cerebral metabolic rate rises by roughly 10 percent for each degree Celsius. If a ventricular drain is in place, cerebrospinal fluid is drained, either continuously against a set height or intermittently in response to elevations; continuous

drainage is generally preferred in trauma. Hyperosmolar therapy draws water out of the brain into the circulation: mannitol in boluses of 0.25 to 1 g per kilogram with serum osmolality kept below 320 mOsm per liter, or hypertonic saline, 23.4 percent in 30 mL boluses through a central line or 3 percent by infusion, which has advantages in a volume-depleted patient who will not tolerate mannitol's diuresis [9].

The second tier is reserved for pressure that does not respond. Hyperventilation to an arterial carbon dioxide tension below 30 mmHg constricts cerebral vessels and lowers pressure within minutes, at the cost of reducing blood flow; it is a temporizing measure for impending herniation, not a sustained therapy. High-dose barbiturates, usually pentobarbital titrated to burst suppression on continuous EEG, suppress cerebral metabolism and lower refractory pressure. They also cause hemodynamic instability, immunosuppression, and long intensive care stays. A Cochrane review found no evidence that they improve survival [19]. Therapeutic hypothermia to 32 to 34 degrees Celsius lowers pressure as well, but randomized trials have not shown better outcomes and rewarming can provoke rebound, so it is no longer routinely recommended [9].

The signal that medical management is failing is pressure that stays up despite escalation, a new or enlarging lesion on repeat imaging, or clinical signs of herniation despite treatment. The commonest error in pressure-guided care is to treat the number rather than the patient: a brief, self-limited elevation may need nothing, a patient with a large bone defect can herniate at a normal reading, and a persistently high reading usually means an evolving surgical lesion that no amount of mannitol will fix. The response to persistent elevation is a repeat CT, not another bolus.

When surgery is needed

Surgery after traumatic brain injury takes two forms: removal of a mass, and removal of bone to make room.

Hematomas are evacuated according to size, mass effect, and the patient's condition, and the thresholds were set out in the Brain Trauma Foundation's surgical guidelines. An epidural hematoma larger than 30 cm^3 is evacuated regardless of the GCS; a smaller one, under 15 mm thick, with less than 5 mm of midline shift, in a patient with a GCS above 8 and no focal deficit, can be observed with serial CT in a neurosurgical center, and a comatose patient with unequal pupils goes to surgery as soon as possible [20]. An acute subdural hematoma thicker than 10 mm or producing more than 5 mm of midline shift is evacuated regardless of the GCS; in a comatose patient with a smaller subdural hematoma, surgery is indicated if the GCS has fallen by 2 or more points since the injury, the pupils are asymmetric or fixed, or the intracranial pressure exceeds 20

mmHg, and every comatose patient with a subdural hematoma should have pressure monitored [21]. The same logic, size and mass effect weighed against the patient's state, governs the evacuation of contusions and intracerebral hematomas. These thresholds also apply to patients who arrived awake. An epidural hematoma of 40 cm^3 in a patient with a GCS of 15 is still removed. That is how a patient classified as mild comes to have a craniotomy.

Decompressive craniectomy removes a large piece of skull and opens the dura so that the swollen brain has somewhere to go. It is the last step on the ladder for diffuse swelling. Its two randomized trials reached different conclusions because they asked different questions. DECRA randomized 155 patients with diffuse injury and early, moderately raised pressure (above 20 mmHg for 15 minutes within an hour despite first-tier treatment) to early bifrontal craniectomy or continued medical care. Craniectomy lowered pressure and shortened intensive care, but functional outcome at six months was worse in the surgical group and mortality was the same, about 19 percent in each arm [22]. RESCUEicp randomized 408 patients whose pressure remained above 25 mmHg for 1 to 12 hours despite the first two tiers of medical treatment to craniectomy as a last resort or to continued medical care. At six months, mortality was 26.9 percent with surgery and 48.9 percent without, a difference that persisted at twelve months. The survivors added by surgery were distributed across the outcome scale. The surgical group had more patients in a vegetative state (8.5 versus 2.1 percent) and with severe disability; the proportion with moderate disability or good recovery did not differ significantly [23]. Read together, the trials say that craniectomy does not help as an early substitute for medical management of moderate pressure elevation and does save lives as a last resort for refractory elevation, at the price that some of the lives saved are lived with severe disability. That trade is the substance of the conversation a neurosurgeon has with a family at the top of the ladder.

What this means for the reader of this book

Three points carry over to the rest of the book.

The first is practical. A patient discharged from an emergency department after a head injury is given a sheet of warning signs for a reason. A new or worsening headache, repeated vomiting, increasing drowsiness or difficulty waking, new confusion, weakness or numbness of a limb, unequal pupils, a seizure, or a decline in consciousness in the days after injury requires immediate emergency reassessment, with repeat CT and neurosurgical assessment as clinically indicated, and the threshold is lower in older patients, in anyone taking an anticoagulant or antiplatelet drug, and after a high-energy mechanism. The record of a patient who returned is read differently from the record of a

patient who did not. These warning signs prompt reassessment for evolving hemorrhage or another complication, rather than an assumption that worsening symptoms are simply part of concussion.

The second concerns the record. A normal CT and a same-day discharge document the state of the brain at one moment. They do not exclude a hemorrhage that declared itself on day three, and they do not exclude the functional injury that is the subject of the rest of this book. A record can show “CT negative, discharged” and, days later, a craniotomy without contradicting itself. The lesion may have been below the resolution of the first scan or may not yet have bled.

The third concerns classification. The severity grade of Chapter 4 is assigned from the initial presentation: duration of loss of consciousness, post-traumatic amnesia, and the GCS after stabilization. A surgical complication that develops later does not retroactively convert a mild injury into a severe one, any more than good recovery converts a severe injury into a mild one. What changes is the course, and the course is documented on its own terms. Under the ACRM convention this book follows, the patient who arrived with a GCS of 15, was found to have a contusion, and had it evacuated carries a diagnosis of mild TBI with neuroimaging evidence of structural intracranial injury, together with the record of what was done about it. Chapter 14 takes up what treatment looks like once the acute period is over.

Fast take: main points from chapter 13

• A small fraction of patients who are awake and talking after a head injury deteriorate: in prospective emergency department cohorts, about 1 percent of patients with a GCS of 13 to 15 needed a neurological intervention [1], and among those whose first CT already showed an injury, pooled rates were 11.7 percent for deterioration and 3.5 percent for neurosurgery [3].

• The mechanisms are delayed hemorrhage after a negative CT (0.4 percent within two weeks in a population cohort [4], higher with anticoagulants [5]), hemorrhagic progression of a contusion [6], and diffuse swelling, which is the particular danger in children and young adults [7,8].

• The skull is a closed box. Once the reserves of displaceable cerebrospinal fluid and venous blood are spent, small increases in volume produce large rises in pressure, perfusion falls, and herniation follows [9].

• Normal intracranial pressure in adults is 7 to 15 mmHg; treatment is recommended above 22 mmHg, with cerebral perfusion pressure kept at 60 to 70 mmHg [9].

• Pressure is monitored in severe TBI, not in mild, and only exceptionally in moderate injury that deteriorates. A ventricular drain measures and treats; a parenchymal monitor measures only, with fewer complications. Outcomes with the two are similar in the largest comparison [13].

• A number is not a patient. Transducer leveling, catheter patency, and the waveform all have to be checked, and a persistent elevation means a repeat CT, because it usually means an evolving surgical lesion.

• Treatment proceeds in tiers: head position, sedation, normothermia, drainage, and hyperosmolar therapy first; hyperventilation, barbiturates, and hypothermia as temporizing or last-resort measures with real costs and no proven survival benefit [9,19].

• Hematomas are evacuated by size and mass effect regardless of the GCS: an epidural hematoma over 30 cm^3, a subdural hematoma over 10 mm or with more than 5 mm of shift [20,21]. That is how a patient classified as mild comes to surgery.

• Decompressive craniectomy failed as an early treatment of moderate pressure elevation in DECRA [22] and halved mortality as a last resort for refractory elevation in RESCUEicp, with more survivors left severely disabled [23].

• For the reader of records: a normal CT and a discharge describe one moment; a later surgical complication neither contradicts them nor changes the initial severity classification.

References

1. Stiell IG, Wells GA, Vandemheen K, Clement C, Lesiuk H, Laupacis A, et al. The Canadian CT Head Rule for patients with minor head injury. Lancet. 2001;357(9266):1391-1396.
2. Haydel MJ, Preston CA, Mills TJ, Luber S, Blaudeau E, DeBlieux PM. Indications for computed tomography in patients with minor head injury. N Engl J Med. 2000;343(2):100-105.
3. Marincowitz C, Lecky FE, Townend W, Borakati A, Fabbri A, Sheldon TA. The risk of deterioration in GCS13-15 patients with traumatic brain injury identified by computed tomography imaging: a systematic review and meta-analysis. J Neurotrauma. 2018;35(5):703-718. doi:10.1089/neu.2017.5259
4. Kwon H, Kim YJ, Lee JH, et al. Incidence and outcomes of delayed intracranial hemorrhage: a population-based cohort study. Sci Rep. 2024;14:19502.

5. Nishijima DK, Offerman SR, Ballard DW, Vinson DR, Chettipally UK, Rauchwerger AS, et al. Immediate and delayed traumatic intracranial hemorrhage in patients with head trauma and preinjury warfarin or clopidogrel use. Ann Emerg Med. 2012;59(6):460-468.
6. Kurland D, Hong C, Aarabi B, Gerzanich V, Simard JM. Hemorrhagic progression of a contusion after traumatic brain injury: a review. J Neurotrauma. 2012;29(1):19-31. doi:10.1089/neu.2011.2122
7. Lobato RD, Rivas JJ, Gomez PA, Castañeda M, Cañizal JM, Sarabia R, et al. Head-injured patients who talk and deteriorate into coma: analysis of 211 cases studied with computerized tomography. J Neurosurg. 1991;75(2):256-261. doi:10.3171/jns.1991.75.2.0256
8. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. Diffuse cerebral swelling following head injuries in children: the syndrome of “malignant brain edema”. J Neurosurg. 1981;54(2):170-178.
9. Carney N, Totten AM, O’Reilly C, Ullman JS, Hawryluk GW, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery. 2017;80(1):6-15.
10. Chesnut RM, Temkin N, Carney N, Dikmen S, Rondina C, Videtta W, et al. A trial of intracranial-pressure monitoring in traumatic brain injury. N Engl J Med. 2012;367(26):2471-2481.
11. Robba C, Graziano F, Rebora P, Elli F, Giussani C, Oddo M, et al. Intracranial pressure monitoring in patients with acute brain injury in the intensive care unit (SYNAPSE-ICU): an international, prospective observational cohort study. Lancet Neurol. 2021;20(7):548-558.
12. Fried HI, Nathan BR, Rowe AS, Zabramski JM, Andaluz N, Bhimraj A, et al. The insertion and management of external ventricular drains: an evidence-based consensus statement. Neurocrit Care. 2016;24(1):61-81.
13. Greil ME, Barber JK, Temkin NR, et al. External ventricular drain versus intraparenchymal pressure monitor in severe traumatic brain injury: a TRACK-TBI study. Neurosurgery. 2025. Epub ahead of print.
14. Liu H, Wang W, Cheng F, Yuan Q, Yang J, Hu J, et al. External ventricular drains versus intraparenchymal intracranial pressure monitors in traumatic brain injury: a prospective observational study. World Neurosurg. 2015;83(5):794-800.
15. Le Roux P, Menon DK, Citerio G, Vespa P, Bader MK, Brophy GM, et al. Consensus summary statement of the International Multidisciplinary Consensus Conference on Multimodality Monitoring in Neurocritical Care. Neurocrit Care. 2014;21 Suppl 2:S1-S26.

16. Oddo M, Levine JM, Mackenzie L, Frangos S, Feihl F, Kasner SE, et al. Brain hypoxia is associated with short-term outcome after severe traumatic brain injury independently of intracranial hypertension and low cerebral perfusion pressure. Neurosurgery. 2011;69(5):1037-1045.
17. Vespa PM, Nuwer MR, Nenov V, Ronne-Engstrom E, Hovda DA, Bergsneider M, et al. Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring. J Neurosurg. 1999;91(5):750-760.
18. Vespa P, Tubi M, Claassen J, Buitrago-Blanco M, McArthur D, Velazquez AG, et al. Metabolic crisis occurs with seizures and periodic discharges after brain trauma. Ann Neurol. 2016;79(4):579-590.
19. Roberts I, Sydenham E. Barbiturates for acute traumatic brain injury. Cochrane Database Syst Rev. 2012;12:CD000033.
20. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of acute epidural hematomas. Neurosurgery. 2006;58(3 Suppl):S7-S15.
21. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of acute subdural hematomas. Neurosurgery. 2006;58(3 Suppl):S16-S24.
22. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med. 2011;364(16):1493-1502.
23. Hutchinson PJ, Kolias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. N Engl J Med. 2016;375(12):1119-1130.
24. American College of Emergency Physicians Clinical Policies Subcommittee. Clinical policy: critical issues in the management of adult patients presenting to the emergency department with mild traumatic brain injury. Ann Emerg Med. 2023;81(5):e63-e105. doi:10.1016/j.annemergmed.2023.01.014 https://www.acepnow.com/article/acep-clinical-policy-on-mild-traumatic-brain-injury/?singlepage=1
25. American College of Surgeons. Best Practices Guidelines: The Management of Traumatic Brain Injury. Chicago: American College of Surgeons; 2024. Anticoagulant management and Table 7, pp. 55-56. https://www.facs.org/media/vgfgjpfk/best-practices-guidelines-traumatic-brain-injury.pdf
26. US Food and Drug Administration. Update on the safety of Andexxa. FDA Safety Communication. December 18, 2025. Accessed October 25, 2026. https://www.fda.gov/vaccines-blood-biologics/safety-availability-biologics/update-safety-andexxa

27. US Food and Drug Administration. Kcentra: product information and approved indication. Accessed October 25, 2026. https://www.fda.gov/vaccines-blood-biologics/approved-blood-products/kcentra

Chapter 14 Treatment after mild traumatic brain injury

Chapter overview

Most people who sustain a mild traumatic brain injury recover within weeks with little more than good advice. The patients who appear in this book are the others: those whose symptoms persist for months or years. For them, treatment is not a single prescription but a program assembled from several disciplines, each aimed at one of the systems that the evaluation showed to be malfunctioning. This chapter describes how Dr. Pappolla's outpatient neurology clinic assembles that program and the support it draws from current guidelines and clinical trials.

The chapter is organized around a simple principle: treat what the evaluation found, not the diagnostic label. Headache is treated according to the kind of headache it is. Abnormal eye movements and visual processing are treated by neuro-optometric and oculomotor rehabilitation. Dizziness and imbalance are treated by vestibular and cervical physiotherapy. Slowed thinking and memory failure are treated by cognitive rehabilitation and speech-language therapy. Exercise intolerance and autonomic dysfunction are treated with graded aerobic exercise. Sleep, mood, and pain are treated in their own right, because each one, left alone, undoes the rest. Before and alongside these therapies, the patient's general medical conditions are found and treated: hypertension, diabetes and insulin resistance, and the hormone deficiencies that the injury itself can cause. The injured brain recovers in a body, and a body that cannot support repair slows recovery. The chapter closes with the treatments at the edge of the evidence, including hyperbaric oxygen, and with the questions about treatment that arise in contested cases: whether it was reasonable, whether it was necessary, what it will cost in the future, and what it means when it has not worked.

Why treatment belongs in this book

Even a reader concerned mainly with evaluating a case needs to understand its treatment. Three reasons explain why.

First, the treatment record is evidence. What was prescribed, when, by whom, and with what result tells the reader how the clinicians who saw the patient understood the injury and whether the course of the condition behaved as a brain injury should. A patient who was referred for vestibular therapy at week six because the examination showed abnormal eye movements has a record that corroborates the complaint; a patient who received nothing but a prescription for rest has a record that says little either way.

Second, the reasonableness and necessity of treatment are themselves disputed. The therapies described here are not exotic. Several, however, are unfamiliar to general physicians and to those who review their bills, and are sometimes challenged as unproven. The reader needs to know which treatments rest on controlled trials, which rest on guideline consensus, and which remain investigational.

Third, the future matters. In a case involving persistent symptoms, the cost of future care is usually the largest element, and it cannot be estimated without knowing what care is appropriate, how long it continues, and what it is expected to achieve.

The principles that organize treatment

Treat the profile, not the label

The most important development in the management of concussion over the past decade is the recognition that it is not one condition. Patients with persistent symptoms sort into recognizable clinical profiles, and the profiles call for different treatments. The most widely used scheme, derived from an expert review of the evidence in 2020, identifies five: a headache or migraine profile, a vestibular profile, an ocular or visual profile, a cognitive profile, and an anxiety or mood profile, with sleep disturbance and cervical injury as modifying factors that cut across all of them [1]. Chapter 6 describes these profiles. Most patients with persistent symptoms have more than one profile. The treatment plan matches each to its therapy (Figure 14.1).

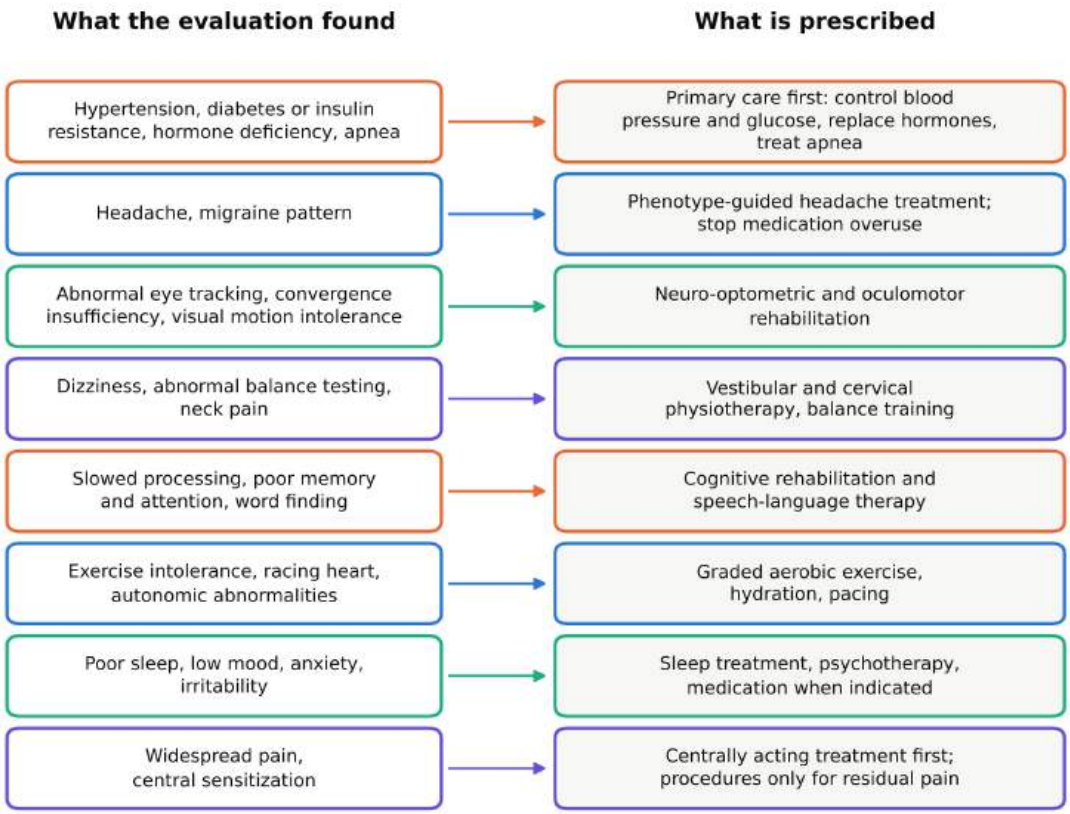


Figure 14.1. From finding to treatment. The left column lists the kinds of findings an evaluation produces; the right column lists the treatment each one calls for. Most patients with persistent symptoms have several findings and therefore several treatments running at once. Original schematic.

This is why the objective testing described in Chapter 7 is not only diagnostic. A videonystagmography study that shows abnormal smooth pursuit is a prescription for oculomotor rehabilitation. A posturography study that shows instability when the information from the feet is degraded is a prescription for balance training. A cognitive test that shows slowed processing speed with preserved accuracy is a prescription for cognitive rehabilitation aimed at speed and for workplace accommodation. Dr. Pappolla's outpatient neurology clinic, which is consultative, uses the test results in exactly this way: to map the deficits, to write a treatment plan organized by system, and to hand that plan to the physicians and therapists who will carry it out.

Activity, not rest

For most of the twentieth century patients with concussion were told to rest in a dark room until the symptoms resolved. The evidence has reversed that advice. Prolonged

rest beyond the first day or two prolongs recovery; early, gradual, symptom-limited return to physical and cognitive activity shortens it. The 2022 international consensus statement on concussion in sport recommends relative rest for no more than 24 to 48 hours, followed by light activity and a stepwise return, and it explicitly recommends against strict rest [2]. The Veterans Affairs and Department of Defense guideline for post-acute mild traumatic brain injury and the Ontario living guideline for adults with prolonged symptoms point the same way [3,4].

Education first

The simplest intervention has one of the best evidence bases. A controlled study published in 2002 examined adults seen within a week of a mild head injury. Those who received a single session of information about expected symptoms, their usual course, and how to manage them had fewer symptoms and less distress at three months than those who received routine care [5]. Every guideline since has made early education a first-line recommendation [3,4]. The content is not complicated: the symptoms are real, they are expected, most of them resolve, and a gradual return to activity helps rather than harms. The patients in this book are usually those who did not receive this message, or in whom the symptoms persisted despite it.

The sequence of care

Treatment proceeds in phases (Figure 14.2). In the first days, the tasks are diagnosis, education, brief relative rest, and a plan for returning to school or work. From the second week through the third month, the medical conditions that affect recovery are identified and treated, and treatment targets the profiles that have become apparent: vestibular and oculomotor rehabilitation, cervical physiotherapy, graded exercise, a headache plan, attention to sleep, and early counselling where mood or anxiety is prominent. Most patients recover in these two phases. Beyond three months, when symptoms are by definition persistent, the approach changes. Objective testing maps the systems involved. The plan becomes multidisciplinary. Cognitive rehabilitation, behavioral health treatment, and treatment of centralized pain take a larger place. Progress is measured by serial reassessment rather than assumed.

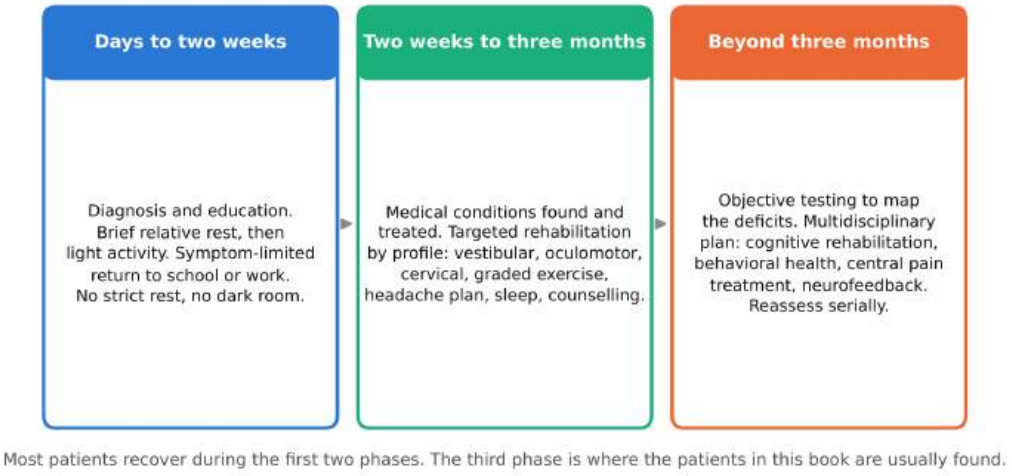


Figure 14.2. The sequence of care after mild traumatic brain injury. Most patients recover during the first two phases. Persistent symptoms, the subject of this book, are managed in the third. Original schematic drawn from current guideline recommendations [2,3,4].

Dr. Pappolla's consultative model

Dr. Pappolla's outpatient neurology clinic is a diagnostic and consultative center. It evaluates the patient, performs or orders the objective studies, and produces a written plan of treatment that is given to the patient and to the physicians who will follow the patient over time, usually the primary care physician working with a general neurologist. The practice does not schedule or coordinate outside appointments, and it does not follow patients long term; it remains engaged on a consultative basis and re-evaluates when asked. In the plan given to the patient, the recommendations are listed one by one, each with the consequence of not following it, and the patient is advised to take on two or three items every two weeks rather than all of them at once, because a patient with impaired attention, headache, and fatigue cannot start eight therapies in the same month. Where a medication is recommended, the prescription is deferred to the physician who will monitor it. This division of labor is stated here because it recurs in the records of Dr. Pappolla's patients and is sometimes misread as a failure to treat.

Primary care first: the medical conditions that govern recovery

Before any of the therapies described below, and alongside all of them, the patient's general medical condition has to be brought under control. This is the step most often missing from the records of patients with persistent symptoms. It belongs high in the treatment plan because the brain does not heal in isolation. Recovery from a

concussion depends on biological processes that take place over months: the formation of new connections between nerve cells (synaptogenesis), the repair of the insulation along nerve fibers, the restoration of normal blood flow regulation, and the settling of the inflammatory response that the injury set off. Every one of those processes requires a steady supply of oxygen and glucose through healthy small blood vessels, normal insulin signaling, normal levels of the hormones that regulate growth and repair, and the absence of continuing inflammation from elsewhere in the body. A patient with uncontrolled hypertension, uncontrolled diabetes, or an untreated hormone deficiency is trying to heal a brain injury in a body that cannot support the repair, in the same way that a wound heals slowly in a patient whose diabetes is out of control. The analogy is exact rather than loose: the same vascular and metabolic failures are responsible in both cases.

Hypertension. High blood pressure damages the small arteries that supply the deep white matter, the brain's wiring, and over years produces the scattered white matter injury seen on the scans of hypertensive patients. It also impairs the brain's ability to hold its own blood flow steady when blood pressure changes, a function called autoregulation that concussion itself disturbs. The two insults add. The evidence that controlling blood pressure protects cognition is strong: in a randomized trial of more than 9,000 adults, intensive control of systolic pressure to below 120 reduced the rate of new mild cognitive impairment by about a fifth compared with standard control, with 14.6 against 18.3 cases per 1,000 person-years [6]. Blood pressure control also matters for headache, which hypertension aggravates, and several blood pressure medications double as migraine preventives, so the two problems can sometimes be treated with one drug.

Diabetes, insulin resistance, and the metabolic syndrome. Insulin is not only the hormone that moves glucose into muscle and fat. It acts in the brain as a signal that supports the formation and strengthening of synapses, particularly in the hippocampus, the structure most responsible for forming new memories. When the body becomes resistant to insulin, the brain does too, and brain insulin resistance is now recognized as a contributor to cognitive decline and to the brain changes of Alzheimer's disease [7]. High blood glucose is directly toxic to small vessels and nerve fibers, drives inflammation, and worsens the outcome of brain injury of every severity. Obesity and the metabolic syndrome add a state of low-grade chronic inflammation that the injured brain does not need. The metabolic syndrome is the cluster of abdominal weight, high blood pressure, abnormal lipids, and insulin resistance. Obesity and the metabolic syndrome are also the main cause of obstructive sleep apnea, which fragments the sleep that recovery requires. Dr. Pappolla and colleagues have observed a higher than

expected frequency of abnormal glycemic indices in patients with persistent post-concussion symptoms, an observation being prepared for publication, and Dr. Pappolla's clinical evaluation routinely includes screening for insulin resistance and diabetes with fasting glucose, hemoglobin A1c, and a lipid profile, with referral to primary care for confirmation and management when the screen is abnormal. The dietary and exercise measures described later in this chapter are the first treatment for insulin resistance; medication follows when they are not enough.

Endocrine disturbances, including those caused by the injury itself. The pituitary gland, which hangs from the base of the brain on a thin stalk and controls most of the body's hormone production, is vulnerable to the same forces that cause concussion. Post-traumatic hypopituitarism, a deficiency of one or more pituitary hormones after a head injury, was for many years thought to follow only severe injuries. A systematic review published in the Journal of the American Medical Association in 2007 found a pooled prevalence of 27.5 percent in the chronic phase after traumatic brain injury of all severities, higher after severe injury but present after mild injury as well [8]. When patients are retested over time, some deficiencies recover and others appear; the proportion with persistent hypopituitarism is estimated at about 12 percent [9]. Growth hormone deficiency is the most common, followed by deficiency of the hormone that drives cortisol production, the sex hormones, and thyroid hormone [9].

This matters here because the symptoms of these deficiencies are the symptoms of persistent post-concussion syndrome: fatigue, poor concentration and memory, low mood, irritability, reduced exercise capacity, weight gain, loss of libido, and disturbed sleep. A patient can carry an untreated growth hormone or testosterone deficiency for years while every one of those complaints is attributed to the concussion, and the leading review of the subject states plainly that untreated hypopituitarism may slow or limit functional and cognitive recovery [9]. The screening is simple: a morning cortisol, thyroid function tests, insulin-like growth factor 1 as a marker of growth hormone, testosterone or estradiol with the pituitary hormones that drive them, and prolactin, obtained in any patient with persistent symptoms at three to six months and repeated at a year if symptoms continue, with referral to an endocrinologist for confirmatory testing and replacement when a result is abnormal. Replacement of a deficient hormone is among the few treatments in this field that can produce a large and rapid improvement.

Thyroid disease unrelated to the injury, vitamin D and vitamin B12 deficiency, anemia, untreated sleep apnea, and excessive alcohol use belong on the same list. Each produces fatigue, slowed thinking, or low mood; each is common in the general population; each is found by routine laboratory testing; and each is treatable. The

differential diagnosis that Criterion 6 of the diagnostic criteria requires, as Chapter 5 explains, is not only a list of alternative explanations to be excluded. It is also a list of treatable conditions that, when present alongside the injury, are slowing the recovery from it.

The discovery that a patient with persistent symptoms also has diabetes, hypothyroidism, or a pituitary deficiency is sometimes read as showing that the symptoms were never due to the injury. The finding has to be weighed in both directions. A comorbid condition can account for part, and occasionally all, of a symptom picture. Criterion 6 requires the evaluator to ask how much the condition explains. It does not by itself make a documented brain injury disappear, and in the case of hypopituitarism it may be a condition the injury caused. What the finding most reliably shows is a treatable factor that has been impairing recovery. The proper response is to treat it and to reassess, which is what Dr. Pappolla's treatment plans direct the primary care physician to do.

Graded aerobic exercise and the autonomic system

Exercise has moved from contraindicated to prescribed. The mechanism is partly autonomic: concussion disturbs the brain's regulation of heart rate, blood pressure, and cerebral blood flow, and many patients find that exertion provokes headache, dizziness, and a surge of all their symptoms. The instinctive response is to avoid exertion. That produces deconditioning, which lowers the threshold further.

The treatment is to exercise below the threshold at which symptoms appear and to raise the threshold gradually. The threshold is measured with a graded treadmill or cycle test in which the patient exercises at increasing intensity until symptoms worsen, and the heart rate at that point is recorded [10]. The patient is then prescribed daily aerobic exercise at roughly 80 to 90 percent of that heart rate, 20 minutes a day, and retested at intervals as the threshold rises (Figure 14.3). Two randomized trials in adolescents with sport-related concussion have shown that this approach, begun within the first ten days, shortens recovery compared with a placebo stretching program. The first trial found a median of 13 days against 17, with a lower rate of delayed recovery [11]. The second found a similar advantage, with a lower rate of persistent symptoms [12]. The 2022 international consensus adopted sub-symptom threshold aerobic exercise as a recommended treatment within the first days after injury [2]. The trials were conducted in adolescents and in sport; the extension to adults injured in collisions rests on the shared physiology rather than on separate trials.

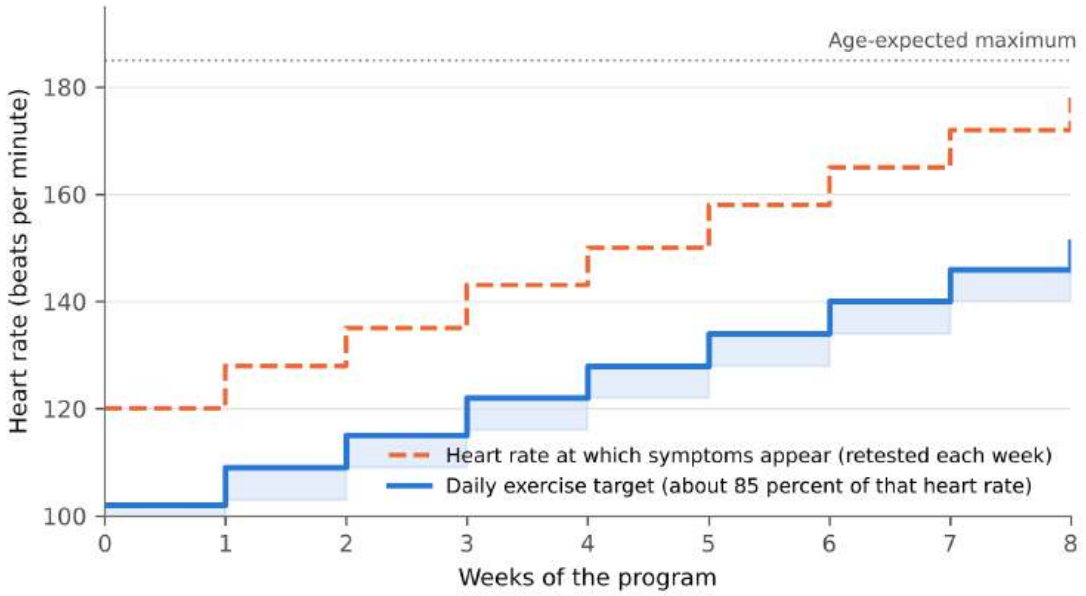


Figure 14.3. Graded aerobic exercise. The patient is tested to find the heart rate at which symptoms appear (dashed line), exercises daily at about 85 percent of that rate (solid line), and is retested at intervals. As the threshold rises the prescription rises with it. The values are illustrative; the program is individualized.

For patients whose autonomic testing shows abnormal heart rate or blood pressure regulation, the exercise program is combined with the measures used in other autonomic disorders: adequate hydration and salt unless contraindicated, pacing of activity through the day, avoidance of prolonged standing and of heat, and in some cases medication to support blood pressure or slow the heart [13]. These are standard treatments for autonomic dysfunction; what is specific to brain injury is recognizing that the symptoms have an autonomic cause rather than attributing them to anxiety or lack of fitness.

Headache

Headache is treated according to the kind of headache it is, because post-traumatic headache has no treatment of its own. The clinical studies described in Chapter 12 show that most post-traumatic headaches look like migraine or probable migraine, with tension-type and neck-related headaches making up most of the rest, and the current approach, endorsed in a 2021 review in Lancet Neurology, is to treat each according to the primary headache it most resembles [14].

In practice there are three tasks. The first is to treat the headache pattern. For the migraine pattern, a preventive medication is started when headaches occur more than a

few days a month: the usual candidates are the tricyclic antidepressants, which also help sleep and pain; certain blood pressure medications and anti-seizure medications long used for migraine; and, for patients who do not respond, onabotulinumtoxinA injections or the newer antibodies that block calcitonin gene-related peptide or its receptor, part of a signaling pathway central to migraine. An open-label study of one such antibody, erenumab, in persistent post-traumatic headache reported benefit [15], although placebo-controlled data for this indication remain limited. Acute attacks are treated with migraine-specific medications. For the tension-type pattern, the emphasis is on the neck, posture, sleep, and stress, with physiotherapy and sometimes a tricyclic. For headache arising from the neck, cervical physiotherapy and, in selected cases, nerve blocks or injections directed at the upper cervical joints are used.

Gepants are smaller molecules that also block the CGRP receptor. In adults, rimegepant is approved for acute migraine treatment and prevention of episodic migraine, ubrogepant for acute treatment, and atogepant for prevention [44,45,46]. Using them specifically for post-traumatic headache is off label. Direct evidence in that population remains limited, including a published atogepant case report, so migraine trial results should not be presented as established efficacy for post-traumatic headache [47,49]. The choice is individualized to the headache pattern, other conditions, and previous treatment response.

The second task is to find and stop medication overuse. A patient with daily headache who takes an over-the-counter pain reliever every day can develop medication-overuse headache, a second headache disorder that sustains the first. The international classification requires headache on at least 15 days a month and medication overuse for more than three months; the medication threshold is 15 or more days a month for simple analgesics, or 10 or more days for combination analgesics, triptans, or opioids [16]. The treatment is withdrawal of the overused medication under supervision, with a preventive started at the same time. This is one of the most common and most correctable reasons for a headache that has not improved, and the medication history should be read for it. Gepants appear less prone to this problem: an open-label study of 1,800 adults with migraine found no medication-overuse headache signal during rimegepant treatment for up to a year [48]. That finding does not prove zero risk for every gepant or patient, and the prescribed schedule and monitoring still matter [44,48].

The third task is to address the contributors: sleep, neck injury, visual strain from convergence insufficiency, and mood. A headache that is being driven by poor sleep and an untreated neck will not respond to a migraine preventive alone.

Neuro-optometric and oculomotor rehabilitation

The eye movement abnormalities described in Chapter 7 are treatable. The brain's control of eye movements can be retrained, in the same way that the brain's control of balance can, and the retraining is the province of two disciplines: neuro-optometry, which addresses how the two eyes work together and how they focus, and oculomotor rehabilitation, which addresses tracking, saccades, and gaze stability and is delivered by optometrists, occupational therapists, and vestibular physiotherapists.

The most common treatable visual problem after concussion is convergence insufficiency, an inability to turn the eyes inward to focus on near objects, which produces eye strain, blurred or double vision, and headache with reading and screen work. Accommodative insufficiency, difficulty adjusting focus between distances, is nearly as common. In a series of 218 patients with concussion seen in two optometric practices, 82 percent had an oculomotor problem: convergence insufficiency in 47 percent and accommodative insufficiency in 42 percent. Among those who completed vision therapy, convergence insufficiency was resolved in 85 percent and improved in the remainder [17]. An earlier series from a university clinic reported improvement in 90 percent of patients with acquired brain injury treated for oculomotor dysfunction [18]. These are case series rather than controlled trials, which limits what they can show about the treatment in this population; the underlying treatment, office-based vergence therapy, has been validated in randomized trials for convergence insufficiency not caused by injury, and the exercises are the same.

The treatment consists of structured exercises, in the office and at home, that progressively demand convergence, accommodation, smooth tracking, and accurate saccades; of prism lenses that reduce the work the eyes must do while they recover; and of tinted or filtered lenses for light sensitivity. A course typically runs twelve to twenty-four weekly sessions. The abnormalities found on videonystagmography are therefore not only evidence of dysfunction but targets of treatment, and a record showing that vision therapy was prescribed and that the patient improved is corroboration that the abnormality was real.

Vestibular, cervical, and balance therapy

Dizziness and imbalance after concussion arise from the inner ear, from the brain's processing of balance information, from the neck, or, most often, from some combination. Vestibular rehabilitation is the physiotherapy of this system. It uses three kinds of exercise. In gaze stabilization, the patient keeps the eyes fixed on a target while moving the head to retrain the reflex that steadies vision during movement. In

habituation, the movements and visual environments that provoke dizziness are rehearsed in graded doses until the brain stops over-responding to them. Balance training uses progressively less stable surfaces, with and without vision, to retrain the brain to weight information from the eyes, the inner ear, and the feet correctly. The posturography findings described in Chapter 7 tell the therapist which conditions to train.

The evidence is good. A case series from a university vestibular clinic reported improvement in dizziness and balance in most patients with persistent symptoms after concussion [19]. In a randomized trial of 31 young athletes with persistent dizziness, neck pain, or headache after concussion, those who received cervical spine and vestibular physiotherapy were nearly four times as likely to be medically cleared within eight weeks as those who received standard care, 73 percent against 7 percent [20]. The 2022 international consensus recommends cervicovestibular rehabilitation for patients whose dizziness, neck pain, or headache persists beyond ten days [2].

The cervical component is often omitted. The forces that produce a concussion in a collision strain the neck, and the neck contributes headache, dizziness, and impaired position sense of its own. Manual therapy, exercise, and postural retraining directed at the neck are part of the program, and a patient treated for the vestibular component alone, with the neck ignored, often fails to improve.

Cognitive rehabilitation and speech-language therapy

Cognitive rehabilitation is the structured retraining of attention, memory, processing speed, and executive function, and it is delivered by neuropsychologists, occupational therapists, and speech-language pathologists. It has two arms. Restorative training exercises the impaired function directly, through graded tasks that demand increasing attention or speed. Compensatory training teaches strategies that work around the deficit: external memory aids, structured routines, environmental modification, and the explicit planning of tasks that used to be automatic. The evidence, assembled in the INCOG guidelines and updated in 2023, favors a combination, with strategy training that is tied to the patient’s real daily demands and taught in a way that minimizes errors during learning [21]. For memory, the strongest recommendations are for teaching internal strategies and for external aids, with smartphone-based reminders preferred and computerized drill used only with a therapist’s guidance; medications and brain stimulation for memory have limited support [22].

Speech-language pathology after brain injury is often misunderstood as treatment for speech. Its main contribution after concussion is to cognitive-communication: word

finding, following conversation in noise, organizing what one wants to say, reading comprehension, and the social use of language, all of which depend on attention and processing speed and all of which are commonly impaired [23]. The speech-language pathologist also treats the swallowing and voice problems that follow more severe injuries, which are rare after concussion.

Cognitive rehabilitation can improve function in daily life substantially, and it is the treatment most directly aimed at the complaint that most often ends careers after concussion, the inability to sustain attention under load. It does not restore processing speed to its pre-injury value in patients whose slowing is physiological, and the recorded test scores may improve less than the patient's function does, because the compensatory strategies that make the difference at work are not tested in a quiet room one task at a time. Chapters 10 and 11 develop that point. A patient who has completed cognitive rehabilitation and still shows slowed processing on testing has not failed treatment; the treatment was never going to change that measurement.

Sleep

Sleep disturbance is nearly universal after concussion and is one of the most important things to treat, because poor sleep worsens every other symptom: pain threshold falls, headache frequency rises, attention and memory deteriorate, mood sinks, and the autonomic system becomes less stable. The patients in this book are usually caught in a cycle in which sleep, pain, mood, fatigue, and cognition each worsen the next, and sleep is often the most accessible point at which to break it (Figure 14.4).

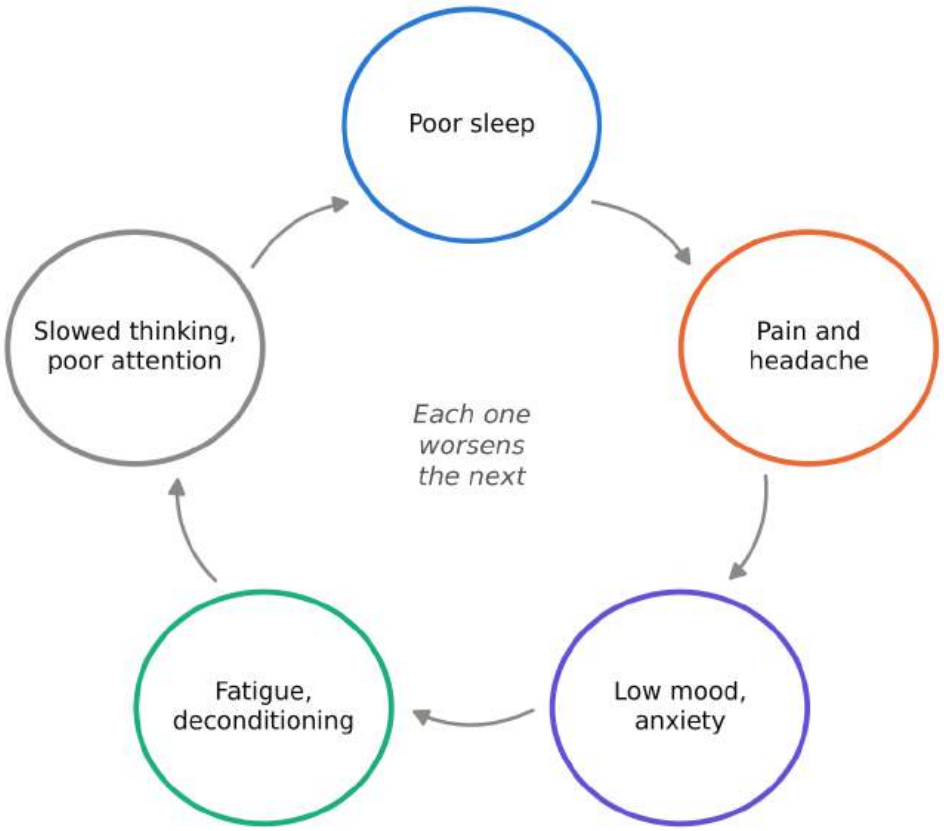


Figure 14.4. The cycle that sustains persistent symptoms. Poor sleep lowers the pain threshold and worsens headache; pain and headache disturb mood; low mood and anxiety produce fatigue and avoidance of activity; deconditioning and fatigue slow thinking; impaired attention disturbs sleep. Treatment can enter the cycle at any point, and sleep is often the most accessible. Original schematic.

Treatment starts with finding the cause. Insomnia after concussion responds to the same structured behavioral treatment that works for insomnia generally: cognitive behavioral therapy for insomnia. The guidelines recommend it as first-line [3,4]. Melatonin has been tested in a randomized, placebo-controlled trial in patients with insomnia after brain injury and improved sleep quality and the proportion of time in bed spent asleep, with less anxiety and fatigue [24]. Sedative hypnotics and benzodiazepines are avoided or limited, because they impair the cognition and balance that are already impaired and because dependence develops quickly. Obstructive sleep apnea is common in this population and is frequently unrecognized; Dr. Pappolla's outpatient neurology clinic screens for it, often with a home sleep study, because

untreated apnea produces exactly the fatigue, headache, and cognitive slowing that are otherwise attributed to the injury, and because it is treatable. Pain and nocturia, depression, and the medications prescribed for other symptoms are reviewed as contributors.

Mood, anxiety, and post-traumatic stress

Depression and anxiety after concussion are common and are treated in their own right. They are usually consequences of the injury and its disruption of the patient's life rather than explanations of it; Chapter 11 sets out the evidence that the association between brain injury and chronic pain is independent of depression, and the same holds for the other persistent symptoms. Treating the mood disorder improves the patient's capacity to engage with every other treatment and should not be deferred on the theory that it will resolve when the other symptoms do.

Psychotherapy is the first line. Cognitive behavioral therapy adapted to the post-concussive syndrome addresses the cycle described above directly: the catastrophic interpretation of symptoms, the avoidance that produces deconditioning, the sleep habits that sustain insomnia, and the demoralization that follows months of unexplained illness. In a randomized trial of 46 adults with persistent post-concussional symptoms after predominantly mild to moderate injury, a twelve-session individualized program improved quality of life and, after adjustment, reduced post-concussional symptoms, anxiety, and fatigue compared with a waiting list. The outcome did not depend on whether the patient was in litigation [25]. Where the injury occurred in frightening circumstances, as it usually did in a collision, post-traumatic stress symptoms are assessed and treated with the trauma-focused therapies that have the best evidence for that condition.

Medication is added when symptoms are moderate or severe or when psychotherapy alone is insufficient. The selective serotonin and serotonin-norepinephrine reuptake inhibitors are used as in depression generally, with attention to the sensitivity of the injured brain to side effects and with a preference for the agents that also treat pain or headache when those are present. The trial evidence for antidepressants specifically after brain injury is thinner than for depression generally and is mixed, so the guidelines recommend treating according to the usual standards for the mood disorder while monitoring closely [3]. Behavioral health follow-up is a standing recommendation in Dr. Pappolla's treatment plans.

Pain and central sensitization

Chapter 12 sets out why most chronic pain after mild traumatic brain injury is nociplastic, driven by central sensitization, and why treatment aimed at the painful body part so often fails. The treatment implications are stated there and repeated here in brief. When the degree of centralization is high, the central component is treated first, with the medications that act on pain processing (the serotonin-norepinephrine reuptake inhibitors, tricyclics, and gabapentinoids), with graded exercise, with treatment of sleep, and with the behavioral approaches to pain; procedures directed at a specific painful structure are reserved for pain that remains, and remains concordant with that structure, after the central component has been addressed [26,27,28]. Opioids are avoided. The practical test of whether a treatment plan for pain after concussion was appropriate is whether it recognized the mechanism; a plan consisting only of injections and anti-inflammatory drugs for a patient with widespread pain did not [29].

Pharmacological management: the principles

No medication treats concussion. Medications treat the individual symptoms, and the pharmacology of persistent post-concussive symptoms is therefore a matter of choosing, for each symptom, the agent with the best evidence and the fewest costs to the other symptoms. Several principles govern the choice.

Use agents that serve more than one purpose. A low-dose tricyclic antidepressant at night may improve sleep, reduce headache frequency, and dampen centralized pain at once. A serotonin-norepinephrine reuptake inhibitor may treat depression, anxiety, and pain. Choosing these over three separate medications reduces the burden on an injured brain.

Avoid agents that worsen cognition or balance. Benzodiazepines, sedating antihistamines, muscle relaxants, and opioids all impair attention, memory, and postural stability, and all are commonly found in the records of patients with persistent symptoms. Their presence often explains part of the cognitive complaint, and their withdrawal is sometimes the most effective cognitive intervention available.

Start low and go slow. The injured brain is more sensitive to side effects, and a patient who stops a medication after a bad first week has lost a treatment that might have helped.

Review the whole list regularly. Polypharmacy accumulates as different physicians treat symptoms one by one. Interactions among six or eight medications can produce the fatigue, fogginess, and dizziness then attributed to the injury.

Treat what can be measured. Where a medication is given for headache, headache days are counted. Where it is given for sleep, sleep is measured. Where it is given for mood, the standard questionnaires are repeated. A medication that has not changed the measure after an adequate trial is stopped.

Stimulant medications are sometimes used for the fatigue and slowed processing that follow brain injury. The evidence is limited and mostly comes from more severe injuries. The drugs can worsen headache, anxiety, and sleep. In Dr. Pappolla's clinical experience, they are a second-line consideration after sleep, mood, and pain have been addressed.

Brain health: diet, metabolic risk, and conditioning

The brain recovers in a body, and the state of the body affects the recovery. The medical conditions that govern recovery, hypertension, diabetes and insulin resistance, and endocrine deficiency, were set out earlier in the chapter. Two further elements of general health belong in every treatment plan, and both act partly through the same pathways.

The first is diet. Nutrients affect the brain directly: the molecular signals that regulate the formation and strengthening of synapses, above all brain-derived neurotrophic factor, respond to what is eaten, and diets high in refined sugar and saturated fat reduce that signaling while diets rich in omega-3 fatty acids, antioxidants, and unrefined foods support it [30]. The Mediterranean dietary pattern, built on vegetables, fruit, legumes, whole grains, fish, olive oil, and nuts, with little red or processed meat and little refined sugar, has the best evidence of any diet for the brain and is also the first treatment for insulin resistance. In a randomized trial of more than 400 older adults, those assigned to a Mediterranean diet supplemented with olive oil or nuts had better cognitive performance after four years than those assigned to a low-fat control diet [31]. A related pattern designed specifically for brain health, the MIND diet, has been associated with substantially lower rates of dementia in observational studies [32]. There are no trials of diet as a treatment for concussion. The rationale for recommending it after brain injury is indirect: it improves the metabolic factors described earlier, it is anti-inflammatory, and it supports the brain under all other conditions studied. It is recommended in Dr. Pappolla's treatment plans on that basis.

The second is physical conditioning, which has already been discussed as graded aerobic exercise. Beyond its specific effect on autonomic symptoms, regular aerobic exercise improves sleep, mood, pain threshold, and cognition in every population in which it has been studied, and the deconditioning that follows months of avoidance is itself a cause of fatigue and exercise intolerance. The reconditioning program is therefore continued after the symptom threshold has normalized, as a permanent part of the patient's routine.

Neurofeedback and neuromodulation

Neurofeedback displays a processed version of the patient's own electroencephalogram. Through reward, the patient learns to shift the brain's rhythms toward a target pattern. It is a form of biofeedback. It has been used after brain injury for decades. The published evidence consists mostly of small uncontrolled studies and case series reporting improvement in attention, headache, and mood, and the reviews that have assembled it conclude that it is promising but that the quality of the evidence is low [33]. Dr. Pappolla's outpatient neurology clinic uses it selectively, after neurorehabilitation has been completed and only when quantitative electroencephalography shows that abnormalities persist, on the reasoning that the patient should first receive the treatments with the stronger evidence and that neurofeedback is aimed at what remains. It is not recommended before rehabilitation.

Other forms of neuromodulation, including transcranial magnetic stimulation and transcranial direct current stimulation, have been studied for post-traumatic headache, depression after brain injury, and cognitive symptoms. Transcranial magnetic stimulation is an established treatment for depression that has not responded to medication, and that use is appropriate in patients with brain injury whose depression meets the same criteria. For the other indications, the trials are small and the results mixed, and the INCOG guidelines specifically do not recommend transcranial direct current stimulation for memory rehabilitation [22]. These remain investigational for post-concussive symptoms.

Hyperbaric oxygen and other alternative treatments

In hyperbaric oxygen therapy, the patient breathes pure oxygen in a chamber pressurized above atmospheric pressure for about an hour, typically in a course of 40 sessions. It is the most prominent treatment proposed for persistent post-concussive symptoms outside mainstream practice, and the one about which the reader is most likely to be asked. The evidence deserves a careful statement, because both its advocates and its critics tend to cite only the trials that favor their view.

The case for it rests on a plausible mechanism, on animal data, and on clinical trials of two kinds. A randomized crossover trial published in 2013 in PLOS ONE treated 56 patients one to five years after mild traumatic brain injury with 40 sessions at 1.5 atmospheres and found improvement in cognitive testing and quality of life, with changes in brain perfusion imaging that paralleled the clinical improvement; the comparison was with delayed treatment, so there was no sham arm [34]. The case against it rested, for a decade, on a series of trials sponsored by the United States Department of Defense in service members with persistent symptoms, which included a sham arm in which the chamber was pressurized with air to a level that produced the sensation of treatment without a therapeutic dose of oxygen. In the largest of the early trials, 72 service members were randomized to hyperbaric oxygen, sham, or routine care. Both chamber groups improved more than routine care, but the oxygen group did not improve more than the sham group. The investigators attributed the benefit to the experience of treatment rather than to oxygen [35]. A subsequent sham-controlled military trial found short-term improvement in post-concussive and post-traumatic stress symptoms at 13 weeks that regressed by six months [36].

The picture has since shifted. In 2025 the same group that led the military trials published a double-blind, sham-controlled trial of 40 sessions in 47 adults with persistent symptoms, most after traumatic brain injury. At 13 weeks, the oxygen group improved by 10.6 points on the Neurobehavioral Symptom Inventory against 3.6 for sham, a statistically significant difference. Specific gains were reported in sleep, anxiety, vestibular complaints, and sense of smell [37] (Figure 14.5). The trial is small, its durability data are limited, and it is one trial; it is also a sham-controlled trial with a positive result from investigators who had previously reported negative ones. On the present evidence, hyperbaric oxygen belongs in the category of unsettled rather than disproven, and equally not in the category of established.

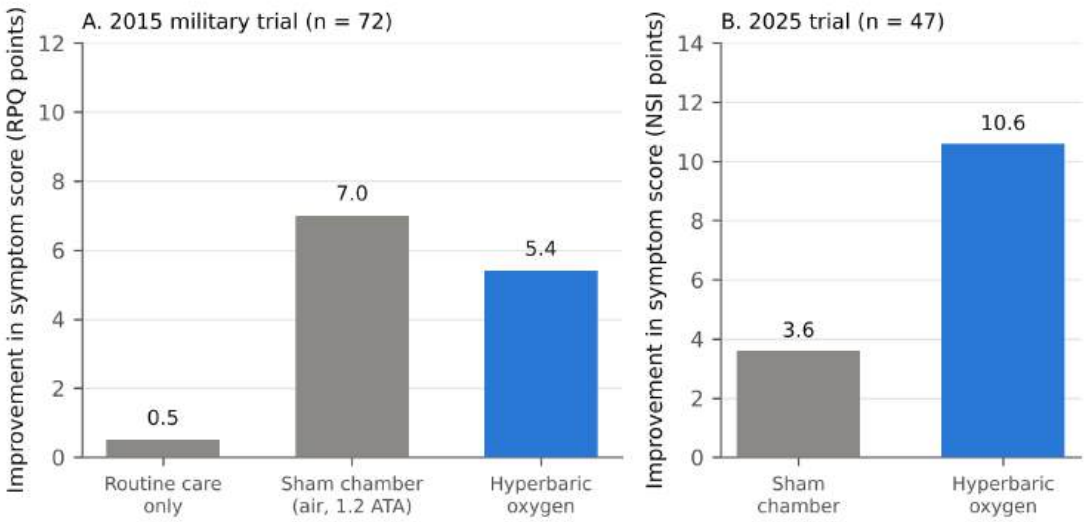


Figure 14.5. Hyperbaric oxygen in sham-controlled trials. (A) In a 2015 trial of 72 service members, both the sham chamber and the oxygen chamber produced more improvement in post-concussion symptoms (Rivermead questionnaire total) than routine care, but oxygen was not better than sham [35]. (B) In a 2025 double-blind trial of 47 adults, mostly with traumatic brain injury, the oxygen group improved more than the sham group on the Neurobehavioral Symptom Inventory at 13 weeks, a difference of 7.0 points with a 95 percent confidence interval of 1.7 to 12.3 [37]. The two trials used different symptom scales, so the panels are not on a common axis. Values are mean changes as reported by each trial.

Dr. Pappolla's position is that hyperbaric oxygen is a reasonable option for a patient with persistent symptoms who has completed the treatments with established evidence and remains impaired, provided the patient understands that the evidence is mixed, that the treatment is not approved by the Food and Drug Administration for this indication and is not covered by most insurers, and that the benefit, if any, may not be lasting. It is not a first-line treatment and it is not a substitute for rehabilitation. When its cost is questioned as experimental, the accurate answer is that its evidentiary status is contested in the literature, with sham-controlled data now on both sides, and that a reviewer who calls it unproven and a clinician who calls it reasonable in a selected patient are both describing the same evidence.

Other alternative treatments are offered to these patients in great variety. Omega-3 fatty acids, creatine, magnesium, riboflavin, and other supplements have mechanistic rationales and, in some cases, evidence in migraine or in severe brain injury, but no controlled evidence of benefit for persistent symptoms after concussion; they are low in risk and low in demonstrated value. Acupuncture has some support for headache generally. Chiropractic manipulation of the neck carries a small but real risk of vertebral

artery injury and is approached with caution in a patient who has already sustained a neck injury. Cannabinoids are widely used by patients and have no controlled evidence of benefit in this condition, with the disadvantage that they impair the cognition, balance, and motivation under treatment. Photobiomodulation, the application of near-infrared light to the scalp, is under study and remains investigational. The reader evaluating a treatment record should distinguish treatments with trial evidence, treatments with guideline support, and treatments with neither, and should not assume that a treatment's presence in the record implies that a physician endorsed it.

Return to work, school, and sport

Returning to work, to school, or to sport is part of treatment, not only its outcome. Prolonged absence worsens mood, sleep, and conditioning, and every current guideline recommends a graded return that begins as soon as the patient can tolerate part of the demand [2,3,4]. The three settings share a sequence. The patient takes on an increasing load in steps. Each step is held until it is tolerated; a step that provokes more than a mild and brief worsening of symptoms is repeated rather than advanced. What differs is the load, the stakes of a failed step, and who makes the decision.

Return to work

Most people who sustain a mild traumatic brain injury return to work within days or weeks, and the systematic review of the International Collaboration on Mild Traumatic Brain Injury Prognosis found that most of those who are off work for longer return within three to six months [38]. The minority who do not are the patients this chapter is about. The same review identified what predicts a delayed return: fewer years of education, extracranial injuries, severe head or bodily pain early after the injury, and a job with little independence or latitude in how the work is done [38]. The last of these matters for planning. A worker who controls the pace and order of tasks can accommodate a deficit; a worker on a line, a schedule, or a phone queue cannot.

A graded return starts from the patient's profile, not from the job description. The objective testing of Chapter 7 shows which systems are impaired, and the plan is built around them. Slowed processing and divided-attention failure call for shorter days, one task at a time, written instructions, and scheduled breaks; a vestibular or visual deficit calls for limits on screen time, driving, ladders, and machinery; noise and light intolerance call for a quieter workplace or a changed shift; headache and sleep disturbance call for a later start and a protected rest period. The first step is usually a few hours a day of the least demanding tasks, two or three days a week, with the hours and the task load advanced every one to two weeks as tolerated. Safety-critical duties,

which include commercial driving, operating machinery, working at height, and clinical responsibility for patients, are returned last and only after the relevant systems have been shown to be normal, because the cost of an error in those settings is borne by others.

The ordinary office is the flight of stairs described in Chapter 10: a patient who performs adequately on a single task in a quiet room can fail in an open-plan office with a ringing phone, two deadlines, and a colleague at the desk. A plan that ignores this returns the patient to full duty, watches the symptoms return, and records the result as a failed attempt. The failure belongs to the plan. The return is monitored by symptom scores, by the patient's and the employer's account of how the work is going, and, when the response is unclear, by repeat objective testing.

Driving deserves its own decision. Driving is a continuous divided-attention task, and a simulator study of concussed young adults tested within forty-eight hours of becoming symptom-free found more lane excursions and more erratic lateral position on curves than in matched controls, with the deficits tracking performance on tests of processing speed, memory, and motor speed [39]. Resolution of symptoms is not by itself clearance to drive. A patient with abnormal eye movements, abnormal balance testing, or slowed processing should not drive until those have improved, and the record should show that the question was asked and answered.

Two points follow for the reader of a record. A patient who is working is not by that fact recovered. In a community cohort followed for four years after mild traumatic brain injury, 17 percent had left the workforce or reduced their hours and 16 percent reported work limitations attributable to the injury. Difficulty with thinking at one month predicted productivity loss at four years [40]. In a clinic sample interviewed six to eight months after injury, 61 percent of those who had returned fully to their previous job reported getting less work done and 42 percent reported making more mistakes [41]. Many patients work at reduced capacity and at considerable cost, which a clinic note that records "returned to work" does not capture. And a patient who has not returned to work has not necessarily failed to try. The record should show whether a graded return was attempted, what accommodations were offered, whether the employer could provide them, and why the attempt did not succeed, because an unaccommodated return that failed and a refusal to attempt one are different facts.

Return to school

The student's job is to learn, and learning is the activity most directly affected by the injury. The Amsterdam consensus sets out a four-step return to learn: daily activities at

home, schoolwork outside the classroom, part-time attendance with support, and full-time attendance as tolerance improves [2,51]. Cognitive load increases gradually after the first 24 to 48 hours of relative rest, without more than mild and brief symptom worsening. Return to learning and the early stages of return to sport can proceed together. Before unrestricted sport, however, the student should have resumed preinjury learning without new concussion-related accommodations. Supports that were needed before the injury do not have to be withdrawn.

The accommodations are practical: a shortened day, a reduced course load, extended time for tests and assignments, a quiet place to work, breaks from screens, notes provided rather than taken, and postponed examinations. A systematic review of return to school after sport-related concussion in children found that between 17 and 73 percent of students needed accommodations or had difficulty returning, and that students were more likely to receive accommodations when the school had a concussion policy, when the clinician supplied a letter, and when medical follow-up was regular [42]. The letter matters. A school that receives a specific, dated set of recommendations acts on them; a school that receives a note saying “concussion, please accommodate” usually does not. In the adult student the same principles apply, and the cognitive profile from Chapter 7 shows which accommodations are needed.

Return to sport

Sport differs from work and school in one respect: the activity itself carries a risk of another injury, and a second concussion in the period before the first has resolved is both more likely and more consequential. In the largest prospective study of collegiate football players, athletes with a history of three or more concussions were three times as likely to sustain another, and 92 percent of the repeat concussions sustained in the same season occurred within ten days of the first [43]. The rare catastrophic brain swelling that follows a second impact in an athlete who has not recovered from the first, described mostly in adolescents, is a further reason the decision is not left to the athlete.

The Concussion in Sport Group’s current return-to-sport materials put the Amsterdam consensus, published in 2023, into a practical sequence [2,50]. The first steps restore daily activity and exercise tolerance. The later steps test readiness for the demands and risks of the sport. Permission to walk or use a stationary bicycle is therefore a different decision from permission to return to a crowded playing field.

Suspected concussion calls for immediate removal from play and assessment. An athlete diagnosed with concussion does not return to play that day. During the first 24 to

48 hours, relative rest allows ordinary activities and gentle walking as tolerated; prolonged isolation and complete inactivity are discouraged. The six steps below summarize the consensus and the CISG professional and public guides [2,50,53].

The staged progression

1. Resume daily activity. Begin with ordinary tasks and short walks that are tolerated. This step can begin within the first 24 hours. The aim is to reintroduce everyday activity while avoiding another head injury.

2. Add aerobic exercise. After the initial period of relative rest, start light exercise, such as stationary cycling or walking, at up to approximately 55 percent of predicted maximum heart rate. Progress to moderate exercise at up to approximately 70 percent. The consensus estimates maximum heart rate as 220 minus age. Light resistance exercise may also begin if symptoms increase only mildly and briefly.

3. Add individual sport skills. Introduce running, changes of direction, or individual drills away from the team. Choose activities without a risk of head impact. If the proposed exercise carries an inadvertent risk of head impact, obtain medical clearance before this step.

4. Resume demanding non-contact drills. After the recovery and clearance requirements below are met, increase exercise intensity and add more complex drills, including team activities. These challenge coordination and thinking as well as conditioning. A drill called non-contact may still carry a collision risk.

5. Resume normal practice. Return to unrestricted training, including full-contact practice when that is part of the sport and has been medically authorized. This gives the athlete and coaching staff an opportunity to assess functional skills and confidence before competition.

6. Return to competition. Resume normal game play after completing the preceding steps and receiving the required determination of readiness.

Each step typically takes at least 24 hours, and progression is individualized. The earliest possible completion of the sequence is about a week; that minimum is not a promised recovery time or an automatic clearance date [2,50,53].

When symptoms change the plan

During Steps 1 to 3, a mild and brief increase means no more than 2 points on a 0-to-10 symptom scale, lasting less than one hour, compared with the level immediately before activity. Greater or longer worsening calls for stopping the activity and reassessment of

the exercise dose, with another attempt the next day. This allowance supports early rehabilitation; it does not extend to the later stages [2,50].

Steps 4 to 6 require resolution of symptoms, cognitive abnormalities, and other clinical findings attributable to the current concussion, including symptoms during and after exertion. If concussion-related symptoms recur in these stages, the athlete returns to Step 3 and establishes symptom-free exertion before resuming activities that carry a risk of injury. Medical authorization precedes any activity with a risk of head impact through contact, collision, or falling; it should not be postponed until full-contact practice [2]. The CISG guides also require tolerance of usual cognitive activities without additional concussion-related accommodations before Steps 4 to 6 [50,53].

The clearance examination

Feeling well at rest is only part of recovery. The clinician also examines cognition, balance, vestibular and oculomotor function, and the response to increasing physical and cognitive demands. Complete symptom resolution includes remaining symptom-free during and after maximal physical and cognitive exertion, assessed when clinically appropriate [2]. In Dr. Pappolla's outpatient neurology clinic, this includes review of the eye movement and balance findings described in Chapter 7. Findings are interpreted against preinjury function, the examination, and exertional tolerance. No single symptom score or cognitive test settles the clearance decision [2,52].

The CISG tools help organize these assessments. The Sport Concussion Assessment Tool 6 (SCAT6) is chiefly an acute assessment tool, most useful within 72 hours and usable up to seven days. The Sport Concussion Office Assessment Tool 6 (SCOAT6) supports multimodal office assessment after 72 hours and serial follow-up. Child versions address ages 8 to 12; the standard versions address ages 13 and older. They guide clinical evaluation rather than provide an automatic pass or fail for return to sport [2,52].

Worsening symptoms call for reassessment. Failure to improve progressively over two to four weeks warrants renewed multimodal assessment and rehabilitation matched to the findings. Continuing difficulty is a reason to look again at the headache, cervical, vestibular, visual, sleep, mood, and exercise-tolerance problems discussed earlier in this chapter. Simply repeating a failed stage is not a treatment plan [2].

The record should identify the stage reached, the symptoms and examination findings, the response to exertion, and the activities authorized or still restricted. For students it should also record progress in learning. Written clearance should meet the applicable

sporting and local requirements. The consensus informs the clinical reasoning; its authors expressly state that it is not itself a legal standard of care [2].

Retirement from contact or collision sport requires an individualized discussion. There is no fixed number of concussions that settles the decision. The clinician considers the injury history, recovery after each event, persistent symptoms or deficits, the sport's risks, and the athlete's preferences; a multidisciplinary assessment may help. For a child or adolescent, parents or guardians participate. The discussion should acknowledge uncertainty, document the reasons for the recommendation, and preserve opportunities for safe physical activity [2].

What recovery looks like, and what it costs

Most patients with mild traumatic brain injury recover fully within weeks. The patients with persistent symptoms do not, as a group, return to baseline, but most of them improve substantially with the program described here, and the improvement follows a recognizable course: the vestibular and oculomotor symptoms respond first and most completely, headache improves as its contributors are treated and overuse is removed, sleep and mood follow, and the cognitive symptoms improve more slowly and less completely, with compensatory strategies doing much of the work. The pain described in Chapter 12 is the most persistent element in many patients.

Maximum medical improvement, the point at which further treatment is not expected to produce further recovery, is usually reached between eighteen months and three years after injury in patients with persistent symptoms, although it depends on when treatment began; a patient who first received appropriate treatment at two years has not reached it at two years. After that point, care does not end. It changes to maintenance: continuation of the exercise program and the sleep and pain regimens, periodic neurological review, repeat objective testing when symptoms change, management of the headache disorder over the long term, and renewed therapy when function declines, as it does in a proportion of patients over the years. These are the elements of a life care plan for persistent post-concussion syndrome, and they can be estimated with reasonable precision from the treatment record once the profile and its response to treatment are known.

Questions about treatment that arise in contested cases, and what the record should show

The same questions about treatment recur in contested cases. A careful reading of the record usually answers them. Sometimes the answer favors the treatment; sometimes it does not.

The patient did not seek treatment, so the symptoms were not serious. The question is reasonable, and the answer depends on the gap. Delay in treatment after mild traumatic brain injury is common: the injury is often missed in emergency departments focused on life-threatening conditions, patients are told they will recover, the symptoms are attributed to stress or to the neck, and the referral to someone who recognizes the pattern may come months later. A gap of that kind is evidence about the health care system's handling of concussion, which is well documented, before it is evidence about the patient. A gap during which the patient was seen for other reasons and recorded no symptoms is a different matter and weighs the other way, and the record should be read for which kind of gap it was.

The treatment was excessive or unnecessary. The test is whether each treatment was matched to a documented finding. Vestibular therapy for a patient with abnormal balance testing, vision therapy for a patient with convergence insufficiency, and cognitive rehabilitation for a patient with slowed processing are indicated. The reader should look for the finding that justified each prescription. Where it is present, the treatment was reasonable; where it is absent, the question is fair.

The treatment was experimental. This applies to some treatments and not to others, and the distinction should be made with care. Graded exercise, vestibular and cervical rehabilitation, cognitive rehabilitation, cognitive behavioral therapy, phenotype-guided headache treatment, and treatment of sleep and mood all rest on randomized trials or guideline consensus or both. Vision therapy rests on case series in concussion and on randomized trials in the same disorder from other causes. Neurofeedback and hyperbaric oxygen are contested, and Dr. Pappolla says so to patients. A blanket characterization of concussion treatment as experimental is wrong; a specific challenge to a specific investigational treatment may be right.

The patient did not improve, so the treatment was wrong or the symptoms are not real. Persistent post-concussion syndrome is persistent by definition. Treatment improves function, often substantially, and rarely restores it fully; a patient who has completed appropriate treatment and remains impaired is a common outcome in this population. The useful question is whether the treatment produced the improvements it can produce, in the domains where it works, and serial objective testing of the kind

described in Chapter 7 is how that is shown. Where it was not, the question of why deserves an answer: treatment that was never matched to a finding, treatment that was not carried out, or a condition that did not respond are different cases.

The patient improved, so the injury was minor. Improvement with treatment is evidence that the treated deficit was real. A patient whose abnormal eye tracking normalized after vision therapy, or whose balance testing normalized after vestibular rehabilitation, had an abnormality that responded to the treatment designed for it. Improvement says nothing about the severity label, which Chapter 4 explains is assigned from the acute presentation; it does say that there was something to treat.

Fast take: main points from chapter 14

• Treatment is organized by clinical profile, not by the diagnostic label. Headache, vestibular, visual, cognitive, and mood profiles, with sleep and the neck as modifiers, each call for a specific therapy, and most patients with persistent symptoms need several at once [1].

• Primary care comes first and runs throughout: hypertension, diabetes and insulin resistance, and hormone deficiencies impair the vascular, metabolic, and hormonal conditions the brain needs to repair itself. Post-traumatic hypopituitarism affects about one patient in four in the chronic phase after brain injury of all severities and mimics the post-concussion syndrome; it is found by simple blood tests and treated by replacement [8,9].

• Rest beyond the first day or two prolongs recovery. Early education and a graded return to activity are first-line treatments with controlled evidence [2,5].

• Graded aerobic exercise below the symptom threshold shortens recovery in randomized trials and is the main treatment for the autonomic symptoms [11,12].

• Post-traumatic headache is treated according to the primary headache it resembles, usually migraine; medication overuse is sought and corrected [14,16].

• Abnormal eye movements and convergence insufficiency are treated by neuro-optometric and oculomotor rehabilitation, with high rates of improvement in case series [17].

• Cervical and vestibular physiotherapy produced medical clearance within eight weeks in 73 percent of patients against 7 percent with standard care in a randomized trial [20].

• Cognitive rehabilitation and speech-language therapy improve daily function through strategy training and external aids; they improve test scores less than they improve function [21,22].

• Sleep, mood, and pain are treated in their own right because each sustains the others. Cognitive behavioral therapy improved quality of life and symptoms in a randomized trial, independently of litigation status [25].

• Medications treat symptoms, not concussion. Agents that serve several purposes are preferred, and those that impair cognition and balance are avoided.

• Diet, metabolic health, and conditioning are addressed because they affect recovery; the evidence for the Mediterranean diet is strong for brain health generally and indirect for concussion [31].

• Hyperbaric oxygen has sham-controlled trials on both sides, the most recent positive; it is unsettled rather than established or disproven, and in Dr. Pappolla's outpatient neurology clinic it is an option after established treatments, with full disclosure of its status, and not a first-line treatment [35,37].

• Return to work, school, and sport is graded, planned from the patient's profile, and monitored; symptom resolution alone is not clearance to drive or to resume contact sport. A patient who is working is not by that fact recovered, and a patient who has not returned has not necessarily failed to try [2,38,39,43].

• Early exercise may produce a mild, brief symptom increase. Later sport stages require resolution of current-concussion symptoms and clinical findings, including with exertion, with medical authorization before exposure to head-impact risk. Students complete full return to learning before unrestricted sport [2,50,51].

• In a contested case, each treatment should be matched to the finding that justified it: where the finding is present the treatment was reasonable, and where it is absent the question is fair. A gap in treatment should be read for its cause before it is read as evidence about the patient; improvement with treatment corroborates the deficit it treated.

References

1. Lumba-Brown A, Teramoto M, Bloom OJ, et al. Concussion guidelines step 2: evidence for subtype classification. Neurosurgery. 2020;86(1):2-13. doi:10.1093/neuros/nyz332

2. Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport, Amsterdam, October 2022. Br J Sports Med. 2023;57(11):695-711. doi:10.1136/bjsports-2023-106898

3. Department of Veterans Affairs, Department of Defense. VA/DoD Clinical Practice Guideline for the Management and Rehabilitation of Post-Acute Mild Traumatic Brain Injury. Version 3.0. Washington (DC): VA/DoD; 2021.

4. Ontario Neurotrauma Foundation. Living Concussion Guidelines: Guideline for Concussion and Prolonged Symptoms for Adults 18 Years of Age or Older. Toronto (ON): Ontario Neurotrauma Foundation; updated 2024. Available at: https://concussionsontario.org. Accessed October 2026.

5. Ponsford J, Willmott C, Rothwell A, et al. Impact of early intervention on outcome following mild head injury in adults. J Neurol Neurosurg Psychiatry. 2002;73(3):330-332. doi:10.1136/jnnp.73.3.330

6. SPRINT MIND Investigators for the SPRINT Research Group; Williamson JD, Pajewski NM, Auchus AP, et al. Effect of intensive vs standard blood pressure control on probable dementia: a randomized clinical trial. JAMA. 2019;321(6):553-561. doi:10.1001/jama.2018.21442

7. Arnold SE, Arvanitakis Z, Macauley-Rambach SL, et al. Brain insulin resistance in type 2 diabetes and Alzheimer disease: concepts and conundrums. Nat Rev Neurol. 2018;14(3):168-181. doi:10.1038/nrneurol.2017.185

8. Schneider HJ, Kreitschmann-Andermahr I, Ghigo E, Stalla GK, Agha A. Hypothalamopituitary dysfunction following traumatic brain injury and aneurysmal subarachnoid hemorrhage: a systematic review. JAMA. 2007;298(12):1429-1438. doi:10.1001/jama.298.12.1429

9. Tanriverdi F, Schneider HJ, Aimaretti G, Masel BE, Casanueva FF, Kelestimur F. Pituitary dysfunction after traumatic brain injury: a clinical and pathophysiological approach. Endocr Rev. 2015;36(3):305-342. doi:10.1210/er.2014-1065

10. Leddy JJ, Willer B. Use of graded exercise testing in concussion and return-to-activity management. Curr Sports Med Rep. 2013;12(6):370-376. doi:10.1249/JSR.0000000000000008

11. Leddy JJ, Haider MN, Ellis MJ, et al. Early subthreshold aerobic exercise for sport-related concussion: a randomized clinical trial. JAMA Pediatr. 2019;173(4):319-325. doi:10.1001/jamapediatrics.2018.4397

12. Leddy JJ, Master CL, Mannix R, et al. Early targeted heart rate aerobic exercise versus placebo stretching for sport-related concussion in adolescents: a randomised controlled trial. Lancet Child Adolesc Health. 2021;5(11):792-799. doi:10.1016/S2352-4642(21)00267-4

13. Pertab JL, Merkley TL, Cramond AJ, Cramond K, Paxton H, Wu T. Concussion and the autonomic nervous system: an introduction to the field and the results of a systematic review. NeuroRehabilitation. 2018;42(4):397-427. doi:10.3233/NRE-172298
14. Ashina H, Eigenbrodt AK, Seifert T, et al. Post-traumatic headache attributed to traumatic brain injury: classification, clinical characteristics, and treatment. Lancet Neurol. 2021;20(6):460-469. doi:10.1016/S1474-4422(21)00094-6
15. Ashina H, Iljazi A, Al-Khazali HM, et al. Efficacy, tolerability, and safety of erenumab for the preventive treatment of persistent post-traumatic headache attributed to mild traumatic brain injury: an open-label study. J Headache Pain. 2020;21(1):62. doi:10.1186/s10194-020-01136-z
16. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1-211. doi:10.1177/0333102417738202
17. Gallaway M, Scheiman M, Mitchell GL. Vision therapy for post-concussion vision disorders. Optom Vis Sci. 2017;94(1):68-73. doi:10.1097/OPX.0000000000000935
18. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry. 2008;79(1):18-22. doi:10.1016/j.optm.2007.10.004
19. Alsalaheen BA, Mucha A, Morris LO, et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. J Neurol Phys Ther. 2010;34(2):87-93. doi:10.1097/NPT.0b013e3181dde568
20. Schneider KJ, Meeuwisse WH, Nettel-Aguirre A, et al. Cervicovestibular rehabilitation in sport-related concussion: a randomised controlled trial. Br J Sports Med. 2014;48(17):1294-1298. doi:10.1136/bjsports-2013-093267
21. Bayley MT, Janzen S, Harnett A, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury: methods, overview, and principles. J Head Trauma Rehabil. 2023;38(1):7-23. doi:10.1097/HTR.0000000000000838
22. Velikonja D, Ponsford J, Janzen S, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part V: memory. J Head Trauma Rehabil. 2023;38(1):83-102. doi:10.1097/HTR.0000000000000837
23. Togher L, Douglas J, Turkstra LS, et al. INCOG 2.0 guidelines for cognitive rehabilitation following traumatic brain injury, Part IV: cognitive-communication and social cognition disorders. J Head Trauma Rehabil. 2023;38(1):65-82. doi:10.1097/HTR.0000000000000835
24. Grima NA, Rajaratnam SMW, Mansfield D, Sletten TL, Spitz G, Ponsford JL. Efficacy of melatonin for sleep disturbance following traumatic brain injury: a randomised controlled trial. BMC Med. 2018;16(1):8. doi:10.1186/s12916-017-0995-1

25. Potter SDS, Brown RG, Fleminger S. Randomised, waiting list controlled trial of cognitive-behavioural therapy for persistent postconcussional symptoms after predominantly mild-moderate traumatic brain injury. J Neurol Neurosurg Psychiatry. 2016;87(10):1075-1083. doi:10.1136/jnnp-2015-312838
26. File C, Nader R, Villasante-Tezanos A, et al. Pain management in mild traumatic brain injury: central sensitization as a multispecialty challenge. Pain Physician. 2025;28:231-240.
27. Clauw DJ. Fibromyalgia: a clinical review. JAMA. 2014;311(15):1547-1555. doi:10.1001/jama.2014.3266
28. Fitzcharles MA, Cohen SP, Clauw DJ, Littlejohn G, Usui C, Häuser W. Nociplastic pain: towards an understanding of prevalent pain conditions. Lancet. 2021;397(10289):2098-2110. doi:10.1016/S0140-6736(21)00392-5
29. Brummett CM, Urquhart AG, Hassett AL, et al. Characteristics of fibromyalgia independently predict poorer long-term analgesic outcomes following total knee and hip arthroplasty. Arthritis Rheumatol. 2015;67(5):1386-1394. doi:10.1002/art.39051
30. Gomez-Pinilla F. Brain foods: the effects of nutrients on brain function. Nat Rev Neurosci. 2008;9(7):568-578. doi:10.1038/nrn2421
31. Valls-Pedret C, Sala-Vila A, Serra-Mir M, et al. Mediterranean diet and age-related cognitive decline: a randomized clinical trial. JAMA Intern Med. 2015;175(7):1094-1103. doi:10.1001/jamainternmed.2015.1668
32. Morris MC, Tangney CC, Wang Y, Sacks FM, Bennett DA, Aggarwal NT. MIND diet associated with reduced incidence of Alzheimer's disease. Alzheimers Dement. 2015;11(9):1007-1014. doi:10.1016/j.jalz.2014.11.009
33. May G, Benson R, Balon R, Boutros N. Neurofeedback and traumatic brain injury: a literature review. Ann Clin Psychiatry. 2013;25(4):289-296.
34. Boussi-Gross R, Golan H, Fishlev G, et al. Hyperbaric oxygen therapy can improve post concussion syndrome years after mild traumatic brain injury: randomized prospective trial. PLoS One. 2013;8(11):e79995. doi:10.1371/journal.pone.0079995
35. Miller RS, Weaver LK, Bahraini N, et al. Effects of hyperbaric oxygen on symptoms and quality of life among service members with persistent postconcussion symptoms: a randomized clinical trial. JAMA Intern Med. 2015;175(1):43-52. doi:10.1001/jamainternmed.2014.5479
36. Weaver LK, Wilson SH, Lindblad AS, et al. Hyperbaric oxygen for post-concussive symptoms in United States military service members: a randomized clinical trial. Undersea Hyperb Med. 2018;45(2):129-156.
37. Weaver LK, Ziemnik R, Deru K, Russo AA. A double-blind randomized trial of hyperbaric oxygen for persistent symptoms after brain injury. Sci Rep. 2025;15(1):6885. doi:10.1038/s41598-025-86631-6

38. Cancelliere C, Kristman VL, Cassidy JD, et al. Systematic review of return to work after mild traumatic brain injury: results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95(3 Suppl):S201-S209. doi:10.1016/j.apmr.2013.10.010
39. Schmidt JD, Hoffman NL, Ranchet M, et al. Driving after concussion: is it safe to drive after symptoms resolve? J Neurotrauma. 2017;34(8):1571-1578. doi:10.1089/neu.2016.4668
40. Theadom A, Barker-Collo S, Jones K, et al. Work limitations 4 years after mild traumatic brain injury: a cohort study. Arch Phys Med Rehabil. 2017;98(8):1560-1566. doi:10.1016/j.apmr.2017.01.010
41. Silverberg ND, Panenka WJ, Iverson GL. Work productivity loss after mild traumatic brain injury. Arch Phys Med Rehabil. 2018;99(2):250-256. doi:10.1016/j.apmr.2017.07.006
42. Purcell LK, Davis GA, Gioia GA. What factors must be considered in “return to school” following concussion and what strategies or accommodations should be followed? A systematic review. Br J Sports Med. 2019;53(4):250. doi:10.1136/bjsports-2017-097853
43. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2549-2555. doi:10.1001/jama.290.19.2549
44. Pfizer. NURTEC ODT (rimegepant): US prescribing information. Revised March 2026. Accessed October 25, 2026. https://labeling.pfizer.com/ShowLabeling.aspx?id=19036
45. AbbVie. UBRELVY (ubrogepant): US prescribing information. Revised March 2025. Accessed October 25, 2026. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/211765s013lbl.pdf
46. AbbVie. QULIPTA (atogepant): US prescribing information. Revised September 2025. Accessed October 25, 2026. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/215206s013lbl.pdf
47. Ashina H, Diener HC, Tassorelli C, et al. Guidelines of the International Headache Society for controlled trials of pharmacological preventive treatment for persistent post-traumatic headache attributed to mild traumatic brain injury. Cephalalgia. 2024;44(3):03331024241234068. doi:10.1177/03331024241234068 https://doi.org/10.1177/03331024241234068
48. Croop R, Berman G, Kudrow D, et al. A multicenter, open-label long-term safety study of rimegepant for the acute treatment of migraine. Cephalalgia. 2024;44(4):03331024241232944. doi:10.1177/03331024241232944 https://doi.org/10.1177/03331024241232944

49. Witt MA, Katyal N. Tackling post-traumatic headache with atogepant: a case report [conference abstract]. BMJ Open Sport Exerc Med. 2025;11(Suppl 3):A18. doi:10.1136/bmjsem-2025-concussion.35 https://bmjopensem.bmj.com/content/11/Suppl_3/A18.1
50. Concussion in Sport Group. Return to Sport Strategy (Professional) [infographic]. Available at: https://www.concussioninsportgroup.com/wp-content/uploads/2025/05/RTS-for-professionals.png. Accessed October 27, 2026.
51. Concussion in Sport Group. Return-to-Learn (RTL) Strategy [infographic]. Available at: https://www.concussioninsportgroup.com/wp-content/uploads/2025/05/CISG-RTL-Professional.jpg. Accessed October 27, 2026.
52. Concussion in Sport Group. CISG Tools: SCAT6, Child SCAT6, SCOAT6, and Child SCOAT6. Available at: https://www.concussioninsportgroup.com/scat-tools/. Accessed October 27, 2026.
53. Concussion in Sport Group. Return to Sport Strategy (Public) [infographic]. Available at: https://www.concussioninsportgroup.com/wp-content/uploads/2025/05/RTS-for-public.png. Accessed October 27, 2026.

Chapter 15 Putting it together: the complete evaluation

Chapter overview

The preceding chapters took the injury apart: how it is diagnosed, how it is graded, how it is measured, what it does to the people who have it, and what can be done about it. This chapter puts it back together. It follows one patient from the roadside to the treatment plan, showing how the pieces form an evaluation that a reader without specialist training can follow and check. At each step, it identifies the question, the chapter that supplies the method, and what the answer does and does not establish. It closes with what a complete evaluation contains, so that a reader who has to judge one, whether as a treating physician, a student, or an attorney, knows what to look for and what its absence means.

One patient

A 44-year-old warehouse supervisor was stopped at a light when his pickup was struck from behind by a delivery truck. He did not lose consciousness. He got out of the vehicle, exchanged information, and drove home. His passenger, a coworker, later said that for several minutes after the impact he asked twice where they had been going and could not say which street they were on. That evening he had a headache and felt, in his word, foggy. He went to an emergency department the next morning because of neck pain. He was alert and oriented. The CT scan of his head and neck was normal. He was discharged with a diagnosis of cervical strain.

Over the following three weeks, the headache became daily. He could no longer tolerate the warehouse scanner screens or the noise on the loading dock. He lost his place in inventory counts he had done for twelve years, and he slept badly. He returned to work after four days because he was the supervisor, and he stayed at work by handing the counts to a subordinate. Ten weeks after the collision his family physician referred him to a neurologist with the question that organizes this book: did this man sustain a brain injury, and if so what is wrong with him now?

Each section that follows answers one part of that question and points back to the chapter that supplies the method. Figure 15.1 shows the sequence.

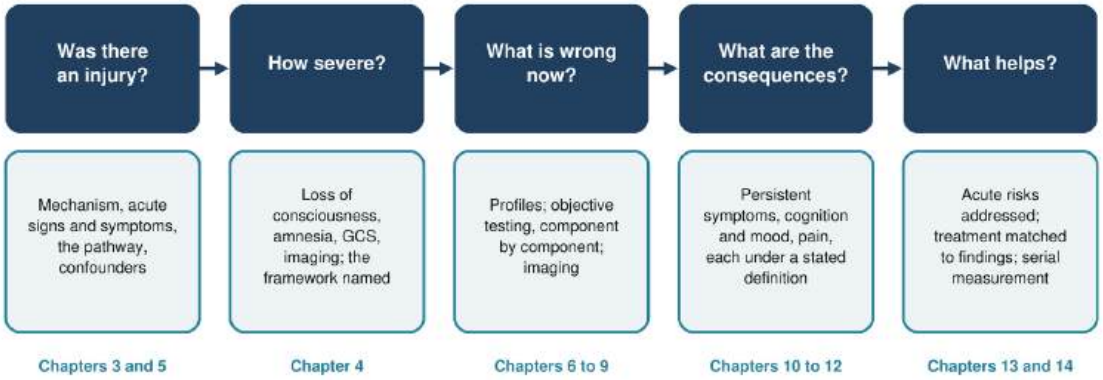


Figure 15.1. The five questions of a complete evaluation, in the order they are answered, with the chapters that supply the method for each. Original schematic.

Was there an injury?

The first question is whether he sustained a brain injury. The answer rests on the 2023 criteria of the American Congress of Rehabilitation Medicine, set out in Chapter 3 [1]. The criteria require a biomechanically plausible mechanism, which a rear-end collision with acceleration of the head is, and then one of three routes.

The first route is a clinical sign. The coworker's account describes a man who, immediately after the impact, could not say where he was or where he had been going and asked the same question twice. Under the criteria, that is an observed alteration of mental status, a Criterion 2 sign. It is established by collateral witness report, which the criteria accept as a source for a sign alongside direct observation, the acute record, and the patient's own account. The emergency department record does not contradict it: the record documents a normal mental status the following morning, and, as Chapter 5 explained with Daniel's case, a normal mental status at hour eighteen says nothing about minute three. The coworker's account supplies a qualifying sign for the first pathway. The diagnosis also requires the confounder and severity checks that follow.

Had there been no witness, the evaluation would have turned to the second route: two or more qualifying acute symptoms together with an acute examination or laboratory finding. The headache and the subjective fog on the evening of the injury are qualifying symptoms within the 72-hour window. But no cognitive, balance, or oculomotor examination was performed in the acute period, and no blood biomarker was drawn, so Criterion 4 cannot be satisfied. The criteria give that situation a name, suspected mild traumatic brain injury, and the name is the correct answer rather than a failure of the

evaluation. A test performed at ten weeks cannot be moved back to the acute period to fill the gap; Chapter 7 explains why.

The third route, a trauma-related abnormality on CT or MRI, is not available to this patient. His CT showed no acute traumatic abnormality. That result is reassuring about the complications visible on CT; it does not exclude microscopic injury or every lesion that MRI might detect. Chapters 2 and 8 explain the limits of each method.

Criterion 6 asks whether something other than brain injury fully accounts for the sign relied on. The confounders the criteria name are considered one by one: he was not intoxicated, he had no syncope before the collision, his neck pain does not explain confusion about where he was, and an acute stress reaction can affect attention and recall, but the history does not identify it as a sufficient explanation for the immediate confusion described. None fully accounts for the sign. The diagnosis stands, and the evaluation records the pathway it rests on and the source of the evidence for it, because that is what will be examined if the diagnosis is questioned.

How severe, and what the label leaves out

Severity is graded from the acute features under the frameworks compared in Chapter 4. There was no loss of consciousness, the alteration of mental status lasted minutes, there was no documented post-traumatic amnesia beyond that interval, and the Glasgow Coma Scale was 15 when first recorded. Every framework calls this mild, and the imaging was negative, so the question of what to do with a positive scan, on which the frameworks disagree, does not arise.

The label is accurate and, as Chapter 4 said, it describes the starting position rather than the destination. The grade says nothing about the headache, the intolerance of screens and noise, the lost inventory counts, or the sleep, and it predicts none of them. The evaluation records the grade and the framework that produced it, then moves on to what the grade cannot say.

What is wrong now

At ten weeks the patient is well past the acute period, and the purpose of testing has changed. It is no longer to establish that an injury occurred; that was done from the history. It is to find out which systems are not working, to measure how badly, and to direct treatment. Chapter 7 set out the instruments and the rules for reading them, and Chapter 9 the cognitive examination. The neurologist can perform the cognitive assessment and use standardized neuropsychological methods within competence,

seeking specialized neuropsychological input when the question requires additional psychometric depth.

The examination begins with the clinical profiles of Chapter 6, because the complaints already point to them: a headache that has become daily, visual and noise intolerance, cognitive inefficiency under load, and disturbed sleep. The testing then measures the systems those profiles implicate. Eye movement recording shows reduced optokinetic gain at the faster stimulus speed and catch-up saccades during smooth pursuit, which fits a man who cannot tolerate the scanner screens and the moving traffic of a loading dock. Computerized posturography shows increased sway in the conditions that remove vision or degrade the information from the feet, which fits his report of unsteadiness in the dark warehouse aisles. Computerized cognitive testing shows reaction time and processing speed in the impaired range with accuracy preserved: the brain reaches the right answer slowly. The validity indicators are within normal limits. The visual evoked potential shows a prolonged latency. Structural MRI is normal; diffusion tensor imaging is not obtained, because, as Chapter 8 explained, its evidence base is at the level of groups and it would not change what is done for this patient.

Each of these findings is a keyhole. Each shows one narrow view of the nervous system, each has causes other than trauma, and none of them, alone, establishes anything about what happened at the intersection. Read together with the history, they do what post-acute testing is for: they demonstrate that the dysfunction the patient describes is measurable, they identify the systems involved, they show that the abnormalities fit the complaints and the mechanism, and they tell the treating physicians what to treat. The evaluation reports each component separately, identifies the system it measures, and explains what the finding supports. It does not report that “the neurophysiology study was abnormal,” because that sentence contains no information.

The normal findings are reported as preserved function within the scope of each test. The patient’s Mini-Mental State Examination score of 30 documents performance on that screen; it does not by itself exclude dementia or another neurocognitive disorder. Nor does it answer the question of inventory counts under time pressure on a noisy dock, a demand the screen does not sample. Chapter 9 explains what screening measures and what fuller assessment adds. The couch-and-stairs analogy in Chapter 11 applies here: when the complaint is failure under load, the evaluation tests that demand with appropriate divided-attention and speed measures, rather than inferring the result from a quiet-room screen.

Naming the consequences

The patient now has a diagnosis, a grade, and a set of measured abnormalities. The next step is to name the consequences of the injury. Each is treated by name and has its own evidence.

He meets the definition of persistent post-concussion symptoms under every current criteria set reviewed in Chapter 10: an established injury, symptoms that began within hours, that have lasted beyond three months by the time the evaluation is written, that affect his work, and that are not better explained by another condition. Chapter 10 also explains why the evaluation says which definition it is applying, since applying different published definitions to the same patients identifies very different groups [2], and why the withdrawal of a standalone category from the diagnostic manuals is a change in nomenclature and not a finding that these patients are well. His cognitive consequences are characterized domain by domain, as in Chapter 11: slowed processing and reduced divided attention with intact memory storage. These are distinguished from his mood consequences, irritability and a low mood that his wife dates to the second month. Chapter 11 explains how these may follow the injury and the disruption of daily life, while also influencing cognitive performance and recovery. His headache is classified by its features as migrainous post-traumatic headache, and his neck pain is examined and found to have a cervical component, which Chapter 12 showed is the usual combination after a collision and which carries its own risk of central sensitization; a body map and symptom severity score are recorded so that any later spread of the pain can be measured against a baseline.

Nothing in this step is inferred from the test results alone, and nothing is dismissed because a test was normal. Each consequence is anchored to the history, the examination, and the measured finding that supports it. This patient does report unsteadiness in dark warehouse aisles, and the posturography finding helps characterize that balance difficulty. Further clinical assessment determines the contribution of vestibular, visual, sensory, and musculoskeletal factors; posturography alone does not assign the cause.

What could have gone wrong, and what helps

The acute period is reconstructed for one more reason. Chapter 13 showed that a small proportion of patients with a mild injury and a normal first scan deteriorate from delayed bleeding or swelling, and that the risk is concentrated in the first days and in patients who are older or anticoagulated. This patient was 44, on no anticoagulant, and ten weeks out with no new headache, vomiting, or drowsiness; the risk has passed. The

evaluation says so, because the attorney or the physician who later reads it should not have to wonder.

Treatment follows the findings, in the order Chapter 14 sets out. The medical conditions that govern recovery are checked first: his blood pressure, his sleep apnea screen, his metabolic markers. The headache is treated as the migraine it resembles, and his daily use of over-the-counter analgesics is addressed because it can sustain the headache it is taken for. The oculomotor findings go to neuro-optometric rehabilitation and the balance findings to vestibular and cervical therapy. The cognitive findings go to a graded program of return to the inventory work under conditions of increasing load, with the accommodations that the testing justifies: no safety-critical driving of warehouse equipment until the reaction time improves, screen time broken up, and counts done away from the dock noise until his tolerance returns. Sleep and mood are treated directly. Graded aerobic exercise, prescribed below the symptom threshold that his exercise test identifies, is started, with the caution Chapter 14 states that its trials were in adolescents with sport-related injury. He is retested at intervals, because serial measurement is how recovery is documented, and the plan is handed to the physician who will follow him.

What a complete evaluation contains

A reader judging an evaluation of mild traumatic brain injury can use the same sequence as a checklist. A complete evaluation contains the following elements; omissions show where it is incomplete.

It identifies the mechanism and says why it is biomechanically plausible. It identifies the acute signs and symptoms, says where each came from (direct observation, a witness, the acute record, or the patient's recollection), and places them in time. It names the diagnostic framework and the pathway on which the diagnosis rests, and if the evidence supports only suspected mild traumatic brain injury it says so. It considers the confounders the criteria name and states which have been excluded and why. It grades severity and names the framework used, because the same scan produces different labels under different frameworks. It reports objective testing component by component, states what system each component measures and what each finding supports, and reports normal findings as preserved function within the scope of each test. It distinguishes what the testing demonstrates about the present from what the history establishes about the injury, and does not present a post-acute study as the acute finding the criteria describe. It names each consequence under a stated definition and anchors each to history, examination, and measurement. It addresses the acute risks that have passed. And it matches each element of the treatment plan to a finding.

An evaluation that has these elements can be checked line by line against the chapters of this book, and it will read the same to a treating physician, to a student, and to the attorneys on either side of a dispute, because every statement in it says what it rests on. An evaluation that lacks them can still be correct, but no one can tell.

Two ideas, once more

Everything in this book comes back to two ideas. They are worth stating plainly one last time.

The first is the keyhole. Every test, from the CT scan in the emergency department to the diffusion tensor image, looks at the nervous system through one narrow opening. A normal view through one keyhole does not mean the rest of the house is in order, and an abnormal view through one keyhole does not say what caused it. The evaluation that respects both halves of that sentence is the one that holds up.

The second is the couch and the stairs. Most of medicine's testing of the injured brain is done at rest, one task at a time in a quiet room, and most of what matters to the patient happens under load. The reading on the couch is accurate, and it says nothing about the stairs. The remedy is not to assume what the stairs would show. It is to test them.

Fast take: main points from chapter 15

• The evaluation answers five questions in order: was there an injury, how severe, what is wrong now, what are its consequences, and what helps. Each has its own method and its own chapter.

• The diagnosis rests on the reconstruction of the acute period under the 2023 ACRM criteria, with the pathway and the source of each sign recorded; a normal mental status the next morning does not contradict a witnessed alteration of mental status at the scene [1].

• Severity describes the starting position. It is recorded with the framework that produced it and predicts none of the consequences.

• Post-acute testing demonstrates and localizes dysfunction and directs treatment. It is reported component by component, with normal findings reported as preserved function within the scope of each test. It does not substitute for the missing acute finding.

• Consequences are named under stated definitions and anchored to history, examination, and measurement: persistent post-concussion symptoms, the cognitive and mood consequences, headache and pain.

• Acute risks are addressed and, when passed, said to have passed. Treatment is matched element by element to the findings and documented by serial measurement.

• A complete evaluation can be checked line by line. The two ideas that make it possible are the keyhole and the couch and the stairs.

References

1. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036
2. Voormolen DC, Cnossen MC, Polinder S, et al. Divergent classification methods of post-concussion syndrome after mild traumatic brain injury: prevalence rates, risk factors, and functional outcome. J Neurotrauma. 2018;35(11):1233-1241. doi:10.1089/neu.2017.5257

Appendix A. Research behind the book

These six papers develop several of the clinical questions discussed in the book. Each entry explains the question in plain language, summarizes the contribution, and identifies the limits of the evidence. The accompanying panel reproduces the title, author line, and complete published abstract. A link leads to the full open-access article, including its methods, figures, tables, references, and publication notices.

The abstracts retain the published wording. The summaries provide context rather than revise the original papers. Review articles, observational studies, and case reports answer different kinds of questions; their findings should be read with those differences in mind.

Cognitive testing and neurological diagnosis

Recognizing persistent problems beyond cognition

Widespread pain after concussion

Nerve blocks for persistent posttraumatic headache

Posttraumatic headaches with tearing and nasal symptoms

Building a coordinated plan for pain after brain injury

Cognitive testing and neurological diagnosis

Evaluating Cognition Across Aging and Traumatic Brain Injury: Integrating Neurological and Neuropsychological Approaches

Pappolla MA, Pappolla SL, Nader R, Hamza MK, Fang F, Fang X. Journal of Clinical Medicine. 2026;15:3822. https://doi.org/10.3390/jcm15103822

The central point of this paper is that cognitive assessment is one part of a complete neurological evaluation. Memory, attention, and processing-speed scores describe important aspects of performance, but they do not describe the whole injured brain. A person may perform normally on cognitive tests and still have disabling disturbances of balance, eye movements, autonomic regulation, or headache. Normal cognitive findings therefore cannot exclude a concussion or its non-cognitive neurological consequences.

Neuropsychological assessment should clearly report the psychometric findings, their validity, their functional implications, and the limits of interpretation. Clinical neuropsychologists can diagnose neurocognitive disorders within their competence. Establishing a specific neurological cause, however, requires evidence beyond the test scores. Psychometric findings alone cannot establish that a traumatic brain injury occurred, attribute the findings to a particular injury, or exclude injury when scores are normal. The medical evaluation must integrate the injury history, the acute signs and symptoms, the neurological examination, relevant investigations, and competing explanations. The paper emphasizes that non-cognitive neurological findings belong in that evaluation and cannot be replaced by a cognitive battery.

The second major point is that cognitive assessment is part of neurology, and neurologists are qualified to perform cognitive and neuropsychological testing when they have the appropriate training and competence for the procedures used. Their role can extend well beyond a brief screening examination. Qualification depends on education, experience with the instruments, standardized administration and scoring, understanding of the normative data and test limitations, and clinical interpretation. A professional title alone does not establish competence with every test, and neuropsychological testing is not reserved exclusively to one profession.

Clinical neuropsychologists contribute specialized depth in psychometrics, validity assessment, and complex cognitive interpretation. Neurologists integrate cognitive and non-cognitive findings within the medical diagnosis and treatment plan. This review explains why both contributions matter while keeping its main distinction clear: measuring cognitive performance and determining its neurological cause are related, but different, clinical tasks.

Read the complete open-access paper

Evaluating Cognition Across Aging and Traumatic Brain Injury: Integrating Neurological and Neuropsychological Approaches

Miguel A. Pappolla, Sean L. Pappolla, Remi Nader, Mohammad K. Hamza, Felix Fang and Xiang Fang

Journal of Clinical Medicine. 2026;15:3822. https://doi.org/10.3390/jcm15103822

ABSTRACT

Background/Objectives: The evaluation of cognition is central to many neurological conditions, including traumatic brain injury, Alzheimer's disease, Lewy body disease, frontotemporal degeneration, and vascular disorders. In clinical practice, particularly in aging populations, cognitive complaints often arise in the context of mixed neurological processes, requiring careful integration of cognitive and non-cognitive findings. Despite this, there remains limited clarity regarding the respective roles of neurologists and clinical neuropsychologists and the distinction between cognitive and neuropsychological assessments, terms that are often used interchangeably despite important differences in methodology and scope. This lack of a shared framework has practical consequences. Cognitive test results, when interpreted in isolation for diagnosis, may be misconstrued as comprehensive measures of brain function, particularly when non-cognitive neurological features such as motor, cerebellar, or vestibular abnormalities should have been considered (but were not).

Methods: In this narrative review, we synthesize clinical guidelines, consensus statements, regulatory sources, and representative empirical literature to articulate a competence-based framework in which cognitive assessment is a medically integrated process incorporating history, functional evaluation, neurological examination, and the targeted use of standardized neuropsychological instruments.

Results: Neurologists are trained to establish medical diagnoses and integrate cognitive findings into the context of neurological disease, while neuropsychologists contribute detailed psychometric characterization, culturally and demographically informed interpretation, cognitive phenotyping, functional characterization, and validity assessment in complex clinical and medicolegal contexts. Although neuropsychologists are qualified to diagnose neurocognitive disorders using standardized diagnostic criteria, attribution to specific neurological etiologies requires a comprehensive medical evaluation that extends beyond cognitive testing alone.

Conclusions: We outline a tiered approach to evaluation that aligns assessment methods with clinical questions and supports accurate diagnosis, interdisciplinary collaboration, and patient-centered care.

Keywords: aging; behavioral neurology; clinical neuropsychology; cognition; cognitive assessment; medicolegal evaluation; neurocognitive disorders; neurological examination; neuropsychological assessment; traumatic brain injury

Read the complete article online 1 / 1

Recognizing persistent problems beyond cognition

Persistent Post-Concussion Syndrome After Its Withdrawal from DSM-5 and ICD-11: Definitional Disagreement and the Neurocognitive Gap

Pappolla MA, Nader R, Fang F, Fang X. Journal of Clinical Medicine. 2026;15:8031. https://doi.org/10.3390/jcm15208031

Persistent difficulties after concussion do not follow one pattern. For some people, memory and concentration are the main concerns. For others, headache, dizziness, disturbed sleep, or difficulty tolerating visual activity causes the greatest disruption. These problems may coexist and influence one another, yet the findings in one area can differ substantially from those in another. An assessment needs to follow the person's difficulties across those areas.

This paper examines how the language of diagnosis can help or hinder that work. We review the differing definitions of persistent post-concussion syndrome and the changes in DSM-5 and ICD-11. Our concern is the absence of a single category that adequately brings together the syndrome's cognitive and noncognitive consequences. Individual conditions, such as headache or vestibular disorders, can still be diagnosed. The gap we discuss concerns their recognition as part of a broader post-injury picture. Different symptom and duration requirements also make it difficult to compare research findings or explain why prevalence estimates vary so widely.

We propose separating the description of a current disorder from the judgment about what caused it. First, the clinician establishes the injury history and characterizes the present problems. Attribution then requires attention to when each symptom began, how it developed, its plausible relationship to the injury, and other explanations or contributing conditions. Sleep, pain, mood, medications, and preexisting illness may all matter. This approach recognizes that an injury can initiate difficulties whose persistence later involves several interacting factors. It also gives cognitive and noncognitive findings their own clinical weight.

The article is a narrative review and a clinical proposal, not a new patient study or a validated set of diagnostic criteria. Its suggested operational framework, including its symptom-count convention, reflects the authors' interpretation of the literature and needs independent testing. Readers should keep that distinction in mind when using it. Its central practical message is to assess the functions that are troubling the patient and affecting daily life. Cognitive testing can contribute substantially to that assessment. Evaluations of balance, eye movements, headache, sleep, and other relevant problems provide different information, helping clinicians develop a more complete explanation and a more useful plan of care.

Read the complete open-access paper

Persistent Post-Concussion Syndrome After Its Withdrawal from DSM-5 and ICD-11: Definitional Disagreement and the Neurocognitive Gap

Miguel A. Pappolla, Remi Nader, Felix Fang and Xiang Fang

Journal of Clinical Medicine. 2026;15:8031. https://doi.org/10.3390/jcm15208031

ABSTRACT

A substantial minority of patients do not recover fully after mild traumatic brain injury. Persistent post-concussion syndrome includes both cognitive and non-cognitive manifestations, yet DSM-5 and ICD-11 withdrew the syndrome as a distinct diagnosis and reorganized its persistent sequelae exclusively within neurocognitive categories. While this reorganization addresses the cognitive component of the syndrome, it provides no single category for its non-cognitive consequences, including vestibular, oculomotor, headache-related, autonomic, sleep, and pain-related manifestations. In some patients, these may be the most disabling consequences of the injury. This narrative review examines the resulting diagnostic gap and other continuing disagreements. Published criteria differ in symptom count, duration, and other requirements, yielding an unacceptably wide range of prevalence estimates, from 11.4% to 38.7%. We review the current status of this syndrome and propose an approach that separates diagnosis from attribution. Finally, we discuss the interactions between cognitive and non-cognitive consequences through shared neural substrates, so abnormalities in one domain may influence function in another. Because cognitive and non-cognitive manifestations may dissociate, a patient may have normal or minimal findings in one domain and functionally significant abnormalities in the other; therefore, evaluation confined to one domain alone (as reflected in the new classifications) cannot exclude the syndrome.

Keywords: mild traumatic brain injury; concussion; post-concussion syndrome; persistent post-concussion symptoms; diagnostic criteria; DSM-5; ICD-11; neurocognitive disorder; vestibular; oculomotor; central sensitization

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Widespread pain after concussion

Pain Management in Mild Traumatic Brain Injury: Central Sensitization as a Multispecialty Challenge

File C, Nader R, Villasante-Tezanos A, Ahmed R, Pappolla S, Ahmed F, Miranda-Morales EG, Zhao Y, Fang X, Kaye AD, Pappolla MA. Pain Physician. 2025;28(3):231-240.

Pain after concussion may reach well beyond the head. When pain affects several body regions alongside fatigue, poor sleep, and difficulty thinking, treating each painful site separately may miss part of the problem. This paper examines that broader pattern and asks whether the extent of pain is related to the burden of persistent post-concussion symptoms.

We reviewed records from an outpatient neurology clinic covering January 2020 through December 2023. Of 223 patients initially considered, 67 met the study criteria for mild traumatic brain injury, persistent post-concussion symptoms, and chronic pain. Patients with chronic pain before the injury were excluded. The Widespread Pain Index recorded how many body regions were painful, while a symptom severity questionnaire captured associated complaints. The Rivermead questionnaire measured post-concussion symptom burden.

Patients reporting pain in more body regions tended to report more severe post-concussion symptoms. The association appeared with the total Rivermead score and both of its symptom groupings. It was statistically significant but modest: pain distribution accounted for about 9% of the variation in the total Rivermead score. We analyzed the pain-location score separately because the two symptom questionnaires shared several items. That helped reduce the risk of finding an association simply by counting the same complaints twice.

The paper interprets this pattern in relation to central sensitization, increased responsiveness in the nervous system's pain pathways. The distinction between a clinical pattern and its mechanism matters here. The questionnaires describe widespread pain and accompanying symptoms; they do not directly measure sensitization of neurons. This retrospective study also had no comparison group, depended substantially on patient reports, and drew from a selected specialty-clinic population. It cannot establish that central sensitization caused the symptoms or that the same pattern occurs in everyone with a concussion.

The practical lesson is to assess the full distribution of pain and its relationship to sleep, fatigue, and cognition. Local injuries still deserve examination and treatment, and several pain mechanisms can coexist. A broader assessment can help clinicians recognize when a painful shoulder, neck, or back is part of a more extensive symptom pattern. The study provides a reason to investigate that pattern further, including which patients benefit from treatments directed at altered pain processing.

Read the complete open-access paper

Pain Management in Mild Traumatic Brain Injury: Central Sensitization as a Multispecialty Challenge

Christopher File, BSA, Remi Nader, MD, Alejandro Villasante-Tezanos, PhD, Rowaid Ahmed, MD, Sean Pappolla, Fauwad Ahmed, MD, Ernesto G. Miranda-Morales, PhD, Yingxin Zhao, PhD, Xiang Fang, MD, PhD, Alan D. Kaye, MD, PhD, and Miguel A. Pappolla, MD, PhD

Pain Physician. 2025;28(3):231-240.

ABSTRACT

Background: Several studies indicate that approximately two-thirds of individuals with mild traumatic brain injury (mTBI) will develop chronic pain, which is often debilitating and a primary factor in long-term disability. Patients with mTBI can suffer concurrently from multiple pain types, such as chronic neuropathic (central or peripheral), nociceptive, or nociplastic pain; however, the prevailing pain types in mTBI patients remain undetermined. This knowledge void limits the formulation of effective therapies for mTBI-related pain.

Objective: We aimed to identify the predominant pain mechanism in patients who had developed persistent post-concussive syndrome (PPCS) after the onset of their mTBI.

Study Design: We conducted a retrospective observational study following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Our study focused on a cohort of mTBI patients with PPCS and chronic pain.

Setting: This study was conducted at an outpatient neurology clinic from January 2020 to December 2023.

Methods: The study included patients who met the criteria for post-mTBI PPCS. Exclusion criteria consisted of a history of chronic pain before the injury, being in the acute/subacute stage (fewer than 90 days after receiving the injury), or the presence of any other neurological comorbidities. We employed a range of diagnostic instruments, including a clinical research tool to measure the degree of central sensitization. Since patients with mTBI often show normal structural imaging, we used several neurophysiological techniques, including evoked potentials, videonystagmography, and quantitative electroencephalography, to confirm the presence of brain pathology objectively. The severity of the post-concussive symptoms was measured using the Rivermead Post-Concussion Symptoms Questionnaire. Central sensitization was assessed using the Widespread Pain Index and the Symptom Severity Index. The correlation between concussion severity and widespread pain was analyzed statistically.

Read the complete article online 1 / 2

Pain Management in Mild Traumatic Brain Injury: Central Sensitization as a Multispecialty Challenge

ABSTRACT, CONTINUED

Results: Out of 223 initial mTBI patients, 67 met the study criteria. The main reasons for exclusions included pre-existing chronic pain or other neurological diagnoses. Among the patients, 39 (58%) were male, averaging 45.7 years of age (range: 20-72). Ethnicity distribution was as follows: 26 (39%) Hispanic, 22 (33%) White, 12 (18%) Black or African American, and 7 (10%) Asian or Pacific Islander. We found that patients with PPCS exhibited high levels of central sensitization, highlighting its critical role in the pathophysiology of chronic pain post-mTBI. We observed a significant correlation between the extent of central sensitization and the presence of non-painful symptoms, suggesting shared neuropathological processes between chronic pain and other PPCS manifestations.

Limitations: This project was a retrospective study, which made it subject to limitations. Also, the measures used to assess some variables were self-reported, subjecting the data to recall bias.

Conclusion: We showed that high levels of central sensitization were universally present in the cohort studied and should be considered the primary therapeutic target in managing chronic post-mTBI pain. Therefore, chronic pain in this population is likely driven by central nervous system pathology that contributes to both the pain experience and other post-concussive symptomatology. A significant clinical implication of our study is that patients displaying high levels of central sensitization often report severe pain in discrete body parts, leading clinicians to mistakenly focus on treating the issue with antinociceptive therapies or interventional procedures that are frequently ineffective.

Key words: Mild traumatic brain injury, concussion, chronic pain, central sensitization, nociplastic pain, persistent post-concussion syndrome, fibromyalgia, pain management

Read the complete article online 2 / 2

Nerve blocks for persistent posttraumatic headache

Efficacy of Nerve Blocks for Managing Refractory Posttraumatic Headaches

File C, Fang X, Ahmad R, Harazeen A, Jung J, Ahmed F, Ahmad N, Pappolla S, Nader R, Pappolla MA. Pain Physician. 2025;28(2):137-145. doi:10.36076/ppj.2025.28.137.

Persistent headache can make it difficult to work, sleep, or take part in rehabilitation after a head injury. When medication has provided inadequate relief, the next treatment decision matters. This paper describes what happened after a group of patients with frequent, disabling posttraumatic headaches received nerve blocks in a neurology clinic.

The study was a retrospective review of 34 adults treated between January 2022 and July 2023. Their headaches had begun within the first week after injury, persisted for more than three months, and occurred at least three times a week with moderate or severe pain. All had a headache when the procedure was performed. Treatment involved combinations of occipital nerves at the back of the head and, when indicated, supraorbital and supratrochlear nerves near the forehead. Twenty-three patients received the full bilateral combination; 11 received a more limited combination based on where they hurt.

The immediate change was substantial. Across all 34 patients, the reported average reduction in headache pain was 88% within the first hour. Among the 31 patients reached the next day, the average reported improvement was 73%. At three months, 27 patients remained available and reported an average improvement of 73%; at six months, 19 patients reported an average improvement of 78%. These percentages describe average pain improvement among those assessed at each visit. They are not percentages of the original group who recovered.

That distinction is essential when reading the longer-term results. Only 19 of the original 34 patients contributed a six-month assessment, and patients receiving repeat injections were no longer followed in the same response analysis. Some also received preventive headache medications. There was no untreated or placebo group, and the choice of blocks was not randomized. The study therefore cannot separate the effects of the blocks from other treatment, natural improvement, or differences between patients who returned and those who did not.

For clinicians, the useful finding is the observed reduction in pain in patients who had remained symptomatic despite medication. It supports further study of nerve blocks as one possible component of care. The records reported no procedural adverse effects, but this small series cannot establish the frequency of uncommon complications. Controlled studies with more complete follow-up are needed to determine who benefits, which nerves to target, and how long relief attributable to the procedure lasts.

Read the complete open-access paper

Efficacy of Nerve Blocks for Managing Refractory Posttraumatic Headaches

Christopher File, BSA, Xiang Fang, MD, PhD, Rowaid Ahmad, MBBS, Ahmed Harazeen, MD, John Jung, BS, Fauwad Ahmed, BS, Naveed Ahmad, BS, Sean Pappolla, Remi Nader, MD, and Miguel A. Pappolla, MD, PhD

Pain Physician. 2025;28(2):137-145. doi:10.36076/ppj.2025.28.137.

ABSTRACT

Background: Nerve blocks (greater occipital, lesser occipital, others) are commonly used, singly or in combination, to treat various forms of refractory headaches, including migraine and cervicogenic headaches. Their efficacy in treating posttraumatic headaches, however, particularly those unresponsive to medications or severely disabling, is not well documented.

Objectives: To characterize the efficacy of nerve blocks in the treatment of posttraumatic headaches.

Study Design: Retrospective chart review.

Setting: A single-specialty outpatient neurology clinic.

Methods: Patients from January 2022 through July 2023 who fulfilled International Headache Society criteria for posttraumatic headache (i.e., new onset headache developing within the first week following head trauma) were included. A rigorous, comprehensive, and unbiased selection process was followed via Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Headaches were refractory to various treatments, including analgesic agents or headache prophylactic medications. The treatments the study patients received were a combination of nerve blocks, including greater, lesser, and third occipital nerve blocks, and supraorbital and supratrochlear nerve blocks. We used the percentage of pain improvement in order to assess the response to the blocks: minimal improvement (< 50%), moderate improvement (50–75%), and significant improvement (> 75%) pain relief.

Results: Thirty-four patients met the inclusion criteria; 15 were women (44%). The mean (SD) age was 43.11 ± 14 years. Of these 34, 28 stated a significant improvement in headache pain immediately following their injection. Twenty-one patients (75%) rated their response to nerve blocks as a ≥ 90% improvement in headaches. Six patients reported complete resolution of their headache pain. Expressed as percentage headache pain improvement, average pain improvement was 88%. Six patients reported moderate improvement of their headaches.

Thirty-one patients reported an average headache improvement of 73% on Postprocedure Day One. Nineteen of these 31 patients had significant pain improvement from baseline, with 12 of them reaching a ≥ 90% pain improvement. Eight patients reported moderate improvement, while 4 had minimal headache pain improvement.

Twenty-seven patients were available for a 3-month follow-up; they reported an average headache improvement of 73%. Thirteen of these patients reported significant improvement in their headache pain, with 12 of them having a ≥ 90% improvement in their headaches. Twelve patients reported moderate pain improvement, and 2 had minimal or no pain improvement.

Read the complete article online 1 / 2

Efficacy of Nerve Blocks for Managing Refractory Posttraumatic Headaches

Published abstract, continued

ABSTRACT, CONTINUED

Nineteen patients returned for a 6-month follow-up; they reported an average pain improvement of 78%. Twelve patients reported significant pain improvement, with 11 having an improvement of ≥ 90%. Four patients reported moderate pain improvement, and 3 reported minimal or no pain improvement. Some patients experienced a biphasic response with partial headache recurrence at 3 months, followed by complete headache resolution at 6 months.

Limitations: Several patients who received multiple nerve blocks were concurrently prescribed prophylactic medications for headache management. Later score improvements cannot be determined to be solely caused by the nerve blocks.

Conclusion: This retrospective review offers preliminary but compelling evidence that nerve blocks are a highly effective option for patients with posttraumatic headaches who have not benefited from medication or who suffer from severe, incapacitating symptoms.

Key words: Headache, nerve block, posttraumatic headache, concussion, postconcussive syndrome, greater occipital nerve block, lesser occipital nerve block, third occipital nerve block, supraorbital nerve block, supratrochlear nerve block

Read the complete article online 2 / 2

Posttraumatic headaches with tearing and nasal symptoms

Efficacy of Nerve Blocks for Posttraumatic Trigeminal Autonomic Cephalalgias: A Case Series

Ahmad R, File C, Forero JC, Nader R, Fang X, Kaye AD, Abd-Elsayed A, Pappolla MA. Pain Med Case Rep. 2026;10(2):101-106.

A severe headache on one side of the head, accompanied by tearing, a red eye, or nasal congestion, deserves a careful description. Those associated symptoms can help distinguish the pattern from a more typical posttraumatic headache. This paper concerns four patients whose headaches after head injury had features of trigeminal autonomic cephalalgias, often shortened to TACs. These are headache patterns in which pain occurs with involuntary changes around the eye or nose.

We reviewed four patients treated at one clinic between January 2021 and December 2023. They were 37, 50, 57, and 77 years old. Each had developed headaches after trauma, with severe unilateral pain and associated symptoms such as tearing or nasal congestion. Some also had a daily migrainous headache or cervical pain. Medication trials had not adequately controlled the troublesome attacks. The cases illustrate how more than one headache pattern can be present in the same patient.

The patients received combinations of nerve blocks selected according to their symptoms. These included occipital nerves at the back of the head and, in some cases, supraorbital and supratrochlear nerves near the forehead. One patient also received cervical medial branch blocks for neck pain. Outcomes were recorded through headache diaries and follow-up assessments. At two months, two patients described near-complete resolution of their TAC-like headaches. The other two reported approximately 50% and 70% improvement, respectively. Associated eye or nasal symptoms also improved, and no significant adverse effects were reported.

These observations are encouraging, but four cases cannot establish a treatment's effectiveness or safety. There was no comparison group, the procedures varied, and follow-up lasted only two months. Medication changes and other treatment could have contributed. The report also concerns headaches with TAC-like features; readers should attend to the individual descriptions rather than assume that every patient had the same narrowly defined headache disorder.

The clinical value lies in recognizing the pattern and documenting its response to treatment. Asking where the pain occurs, how long attacks last, and whether the eye tears or the nose becomes congested can reveal details that a general headache label misses. This case series offers a starting point for testing whether selected nerve blocks help this group of patients, with larger studies needed to establish durable benefit and an appropriate treatment approach.

Read the complete open-access paper

Efficacy of Nerve Blocks for Posttraumatic Trigeminal Autonomic Cephalalgias: A Case Series

Rowaid Ahmad, MD, MBBS, Christopher File, BSA, Juan Camilo Forero, BS, Remi Nader, MD, Xiang Fang, MD, PhD, Alan D. Kaye, MD, PhD, Alaa Abd-Elsayed, MD, and Miguel A. Pappolla, MD, PhD

Pain Med Case Rep. 2026;10(2):101-106.

ABSTRACT

Background: Posttraumatic headaches (PTHs) frequently occur following mild traumatic brain injury and may occasionally present with features of trigeminal autonomic cephalalgias (TACs). PTH-TAC is characterized by unilateral craniofacial pain and autonomic symptoms and can be significantly debilitating. Management of this headache subtype can be particularly challenging as it can be refractory to medical therapies. Currently, there is a paucity of evidence on the optimum medical management for this condition.

Objectives: This case series evaluated the effectiveness of nerve blocks in managing PTH with TAC features, highlighting their therapeutic potential for this debilitating headache phenotype.

Setting: In this retrospective study, we identified 4 patients seen at St. Michael's clinic from January 2021 to December 2023.

Methods: We identified 4 patients meeting the International Classification of Headache Disorders diagnostic criteria for PTH, presenting with TAC features. They initially failed medical management and later underwent anesthetic nerve block procedures, including occipital, supraorbital, and supratrochlear nerve blocks. Clinical outcomes, including changes in headache frequency, pain intensity using a standard 11-point Numeric Rating Scale (NRS-11), where 0 indicates no pain, and 10 indicates the worst pain imaginable., and autonomic symptoms, were documented over a 2 month follow-up period.

Results: All patients demonstrated significant improvement, with reductions in headache intensity and frequency > 50% in 2 cases and near-complete resolution in 2 cases recorded at the 2 month follow-up. Associated autonomic symptoms, including nasal congestion, tearing, and periorbital pain, also showed marked improvement. No significant adverse effects were reported during or after the procedure.

Limitations: This is a small case series with a limited follow-up period (2 months).

Conclusions: Nerve blocks represent a potentially safe and effective intervention for PTH with TAC-like features, providing both rapid and sustained relief for patients with refractory symptoms. However, we need larger prospective cohort studies with extended follow-up periods to confirm the findings of this small case series. Such studies will help standardize evidence-based treatment protocols for this challenging subset of headaches.

Key words: mTBI, mild traumatic brain injury, TAC, trigeminal autonomic cephalalgia, PTH, posttraumatic headache, PT-TAC, posttraumatic trigeminal autonomic cephalalgia, ICHD-3, International Classification of Headache Disorders 3 criteria

Read the complete article online 1 / 1

Building a coordinated plan for pain after brain injury

Pharmacologic and Nonpharmacologic Pain Management in Patients with Traumatic Brain Injury: A Multidisciplinary Approach

Esneault BS, Maddox MB, Loewe EM, Pappolla MA, Parker-Actlis TQ, Shekoohi S, Kaye AD. Journal of Clinical Medicine. 2025;14:8713. https://doi.org/10.3390/jcm14248713

Pain can make the work of recovery harder. It can interrupt sleep, limit movement, compete with attention, and reduce a person's ability to participate in rehabilitation. After a brain injury, treating pain also requires attention to balance, fatigue, cognition, and the effects of medication. A treatment that reduces pain but leaves someone too drowsy to function may need to be reconsidered. The goal is relief that helps the person live and recover more effectively.

This review brings together pharmacologic and nonpharmacologic approaches to that problem. It explains three broad mechanisms: pain arising from injured or inflamed tissues, pain resulting from damage or disease affecting the nervous system's sensory pathways, and pain associated with altered pain processing. These are called nociceptive, neuropathic, and nociplastic pain. More than one mechanism may contribute in the same person. Understanding the likely contributors helps clinicians choose treatments and evaluate why a particular approach is helping, failing, or causing unwanted effects.

The review considers medications alongside physical and occupational therapy, psychological approaches to pain management, selected procedures, and other interventions. Attention to sleep, mood, activity, and family support runs through the discussion. Psychological treatment can help people manage pain and its consequences; its inclusion does not diminish the physical reality of the problem. The multidisciplinary approach described here depends on coordination, with clinicians sharing findings and adjusting care around the patient's needs. Medication tolerability deserves particular care when symptoms already include slowed thinking, dizziness, or fatigue.

This is a narrative review of existing literature, not a trial comparing treatments in a new group of patients. The evidence is uneven: some recommendations draw on research in other pain conditions, and some mechanisms or proposed therapies come from experimental work. Inclusion in the review should therefore not be taken as proof of effectiveness for every form of post-TBI pain. Its discussion of artificial intelligence, biomarkers, and regenerative approaches describes developing research whose clinical role remains to be established. Read the paper as an overview of options and the reasoning behind individualized care. The useful question throughout is whether a treatment's expected benefit, evidence, and risks fit the particular person's pain problem and recovery goals.

Read the complete open-access paper

PUBLISHED ABSTRACT PAPER 6

Pharmacologic and Nonpharmacologic Pain Management in Patients with Traumatic Brain Injury: A Multidisciplinary Approach

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ABSTRACT

Traumatic brain injury (TBI) is a major global health problem and a leading cause of long-term neurological disability. TBI produces a spectrum of persistent symptoms, including cognitive impairment, mood and behavioral disturbances, sleep disruption, fatigue, and autonomic dysregulation. Chronic pain is among the most debilitating sequelae, affecting physical, emotional, and social functioning. The etiology of post-TBI pain is multifactorial, arising from initial structural and biochemical injury to the nervous system, maladaptive neuroplastic changes, neuroinflammation, and psychological comorbidities that amplify pain perception and chronicity. This review explores the complex pathophysiology of post-TBI pain and outlines a multidisciplinary framework for management. Pain syndromes are classified according to the International Association for the Study of Pain's mechanistic framework as nociceptive pain (resulting from tissue injury and inflammation), neuropathic pain (due to lesion or disease of the somatosensory nervous system), and nociplastic pain (arising from altered nociceptive processing without clear evidence of tissue or nerve damage). Many patients exhibit mixed pain phenotypes driven by neuroinflammation and central sensitization. Pharmacologic approaches, including anti-inflammatory agents, anticonvulsants, and antidepressants, require cautious titration due to TBI-related comorbidities. Equally essential are non-pharmacologic strategies such as physical and occupational therapy, cognitive behavioral therapy, and neuromodulation techniques, which target both biomechanical and psychosocial contributors. Emerging innovations, machine learning for prognostication, blood biomarkers for structural injury, and neuro-reparative agents, represent the next frontier in personalized management. Effective care for post-TBI pain requires an integrated strategy that combines mechanistic classification, multidisciplinary treatments, and advancing diagnostic technologies.

Keywords: traumatic brain injury; nociplastic pain; neuropathic pain; nociceptive pain; neuroinflammation; multidisciplinary management

Read the complete article online 1 / 1

Prescribing clarification. The full review's statement that nebivolol does not worsen bronchospasm should not be read as a safety assurance. Current US prescribing information advises that patients with bronchospastic disease generally should not receive beta blockers. Individual treatment requires the relevant precautions.

Source: Nebivolol tablets, US prescribing information, section 5.3, revised January 2026. DailyMed prescribing information

Appendix B

Structured intake for retrospective assessment of traumatic brain injury

Comprehensive interview form with evidence coding and diagnostic flow

This appendix provides an independently written clinical intake and documentation framework for one potentially traumatic brain injury event. It draws on the 2023 ACRM diagnostic criteria and the development paper by Lange, Gillow, and colleagues [1,2]. It is intended to help the clinician document the evidence, its source, and its limitations before making a diagnostic judgment.

This is an author-developed adaptation. It is not the official ACRM Structured TBI Interview, does not reproduce its 50-item wording or response codes, and has not been validated or endorsed by ACRM. The official full interview, guide, coding form, diagnostic diagram, and Short Form are linked at the end of this appendix. Use those documents when standardized administration of the published instrument is intended.

How to complete this form

Interview the patient about one event at a time. Invite an uninterrupted account before introducing specific prompts. Record the patient's own descriptions and identify which details were remembered personally, supplied by another person, or obtained from records. A family member may contribute, but preserve each person's account separately. Use an interpreter when needed and document any communication limitation.

Use the response spaces for the history, then complete the coding worksheets after reviewing corroborating evidence and confounders. Unknown, not asked, not available, and absent are different findings. A missing result is not a normal result. Attach additional pages when necessary; do not shorten a substantive answer to fit a blank. The published interview is primarily intended for ages 15 and older; use at ages 12 to 14 requires clinical judgment. Inexperienced interviewers need training and supervised practice. This appendix is not a validated pediatric instrument [2].

Do not use retrospective interviewing to replace assessment of an ongoing acute confusional state or post-traumatic amnesia. At publication, external validation of the published interview had not been completed [2]. This independently written form likewise organizes clinical reasoning and does not establish diagnostic accuracy.

Identification and sources

Patient name or ID _____ Date of birth _____

Interview date _____ Injury date _____ Interval since injury _____

Clinician _____ Interview mode _____

Language and interpreter _____ Informants _____

Records reviewed and dates _____

Records or collateral information still needed _____

Event reconstruction and prior functioning

Patient ID _____ Event date _____

1. Describe the event from just before it began through the first period afterward that you can recall clearly.

Begin with free recall. Establish date, approximate time, location, activity, and whether more than one impact occurred.

Response: _____

_____

_____

2. What happened to your head and body during the event?

Clarify contact, sudden acceleration or deceleration, rotation, fall height or blast exposure. Record the basis for any estimate of force or speed.

Response: _____

_____

3. Who was present, and what information could help us check the sequence?

Identify direct witnesses, emergency responders, photographs, video, dispatch times, messages, or other contemporaneous records.

Response: _____

_____

4. What care did you receive during the first day?

Record facilities, times, transport, major bodily injuries, admission or discharge, and any emergency procedures.

Response: _____

_____

5. How were your thinking, balance, headaches, vision, mood, and sleep before this event?

Record relevant baseline disorders, medications, prior injuries, and whether prior symptoms had resolved.

Response: _____

_____

6. Have there been other head injuries or major medical events before or after this one?

Identify dates separately. Do not combine manifestations from different events into a single injury history.

Response: _____

Awareness and behavior immediately after impact

Patient ID _____ Event date _____

7. What do you remember understanding about your location and what had happened in the first moments afterward?

Record actual examples of orientation, comprehension, and actions. Do not infer a sign solely from the word confused.

Response: _____

_____

8. Describe your first interactions with anyone after the event.

What was said or asked? How did you respond? Explore delayed, inappropriate, absent, or repeated responses without assuming they occurred.

Response: _____

_____

9. What did another person directly observe about your responsiveness or behavior?

Identify the observer and exact behavior. Separate direct collateral information from what the patient recalls being told.

Response: _____

_____

10. Was there an interval when you were motionless or did not respond to people around you?

☐ Yes ☐ No ☐ Uncertain. Identify who observed it and whether the observation preceded sedating treatment.

Response: _____

_____

11. If consciousness may have been lost, how was its duration established?

Record the shortest and longest plausible duration, timing anchors, and a waking or recovery experience. A memory gap alone does not establish unconsciousness.

Response: _____

_____

12. When you describe being dazed, confused, or disoriented, what did that experience involve?

Distinguish a subjective feeling from demonstrable impairment of orientation or responsiveness. Clarify onset immediately after impact or return of consciousness.

Response: _____

_____

Memory before and after the event

Patient ID _____ Event date _____

13. Describe the last event before impact that you remember yourself.

Separate personal recall from a reconstruction based on photographs, records, or other people's accounts.

Response: _____

_____

14. Is there a gap between that memory and the injury? What establishes its duration?

Record whether the patient was awake before impact and any credible clock, travel, witness, or other timing anchor.

Response: _____

_____

15. Describe the earliest event after impact that you remember yourself.

Record its place and context. Establish whether the patient was already speaking, walking, or receiving treatment.

Response: _____

_____

16. From what point can you remember the subsequent events continuously?

Identify isolated memories before continuous recall returned. One remembered moment does not necessarily mark the end of post-traumatic amnesia.

Response: _____

_____

17. What happened during the interval you cannot recall, according to other people or records?

Document repeated questions, actions without later recall, sedation, sleep, or other explanations. Identify the source of each detail.

Response: _____

_____

18. What evidence supports the estimated memory gap, and what remains uncertain?

Record the patient's estimate separately from the clinician's estimate. Use retrograde amnesia as a substitute only if post-traumatic amnesia cannot be reliably assessed; do not use its duration to assign severity.

Response: _____

_____

Acute symptoms and their timing

Patient ID _____ Event date _____

19. What new problems did you notice in the first hours and days?

Obtain spontaneous symptoms first. Then clarify relevant domains without suggesting that a symptom must have occurred.

Response: _____

_____

20. For each symptom, when did it begin or become worse, and how did it differ from before the event?

Distinguish onset from first recognition. Record the source and any uncertainty in the time estimate.

Response: _____

For the inventory below, enter baseline status, earliest onset or worsening, source, and whether the symptom is potentially eligible for Criterion 3. Final eligibility requires assessment of confounders. Use Y, N, or U for yes, no, or uncertain.

Symptom	Before injury	Onset or worsening / source	Within 72 h Y / N / U
Headache			
Nausea			
Dizziness			
Balance difficulty			
Vision problems			
Light sensitivity			
Noise sensitivity			
Slowed thinking			
Mental fog			
Concentration difficulty			
Memory difficulty			
Uncharacteristic emotional lability			
Irritability			

Immediate subjective mental-status symptoms: confused ____ disoriented ____ dazed ____

Timing relative to impact or recovery of consciousness _____

These immediate symptoms have a different timing requirement from the other acute symptoms. Record distinct symptoms and avoid counting repeated descriptions of the same experience as separate evidence [1].

Other symptoms, including those beginning later:

_____

Other acute neurological signs and competing explanations

Patient ID _____ Event date _____

21. What happened when you first tried to stand or walk?

Identify any witnessed incoordination, who observed it, and whether leg injury, pain, faintness, intoxication, or another condition explains it.

Response: _____

_____

22. Did anyone describe a seizure, sustained stiff posture, or another unusual neurological event immediately afterward?

Record the observed sequence, timing, duration, source, and alternative explanations. A reported seizure requires clinical characterization.

Response: _____

_____

23. What was your state immediately before the event?

Clarify sleep, fainting, presyncope, seizure, altered awareness, or illness that preceded the impact.

Response: _____

_____

24. What bodily pain or injuries affected your attention, movement, or ability to answer questions?

Record location, severity at the relevant time, and the specific function affected. A painful injury can coexist with a brain injury.

Response: _____

_____

25. Were breathing problems, major bleeding, low blood pressure, or resuscitation part of the event?

Document timing from records where possible. Separate physiological compromise from effects attributable to mechanical brain injury.

Response: _____

_____

26. How did fear, shock, or distress affect your thinking and recollection at the time?

Explore effects without requiring a detailed retelling of traumatic experiences. Record uncertainty and pause if the interview causes distress.

Response: _____

_____

Medication and substance effects

Patient ID _____ Event date _____

27. What prescribed or nonprescribed medicines had you taken before the event?

Record names, doses when known, timing, usual use, and effects on alertness, thinking, or balance.

Response: _____

_____

28. What medicines were given afterward, and when did their effects begin and end?

Include pain medication, sedatives, anesthesia, and other agents that could alter responsiveness or memory.

Response: _____

_____

29. Were you intubated, anesthetized, or medically sedated?

☐ Yes ☐ No ☐ Unknown. Record the last reliable observations before treatment and the first reliable observations after it.

Response: _____

_____

30. What alcohol or other substances had you used, and what effects were apparent?

Record substance, amount, timing, observed behavior, and available toxicology. The amount reported alone does not establish its effect.

Response: _____

_____

31. What other condition could explain some or all of the changes around the event?

Consider relevant pre-existing neurological, vestibular, psychiatric, cognitive, sleep, or medical conditions. State the specific effect being considered.

Response: _____

_____

Clinician note on timing

Do not assign the duration of medically induced unresponsiveness to traumatic loss of consciousness. If a confounder prevents reliable duration estimates, document that limitation even when an acute brain injury is otherwise established [1,2].

Unresolved medication or substance questions:

_____

_____

Examination imaging and other medical evidence

Patient ID _____ Event date _____

Complete from actual records, reports, or examination data. Distinguish the date of the original assessment from the date on which it was reviewed. Patient recollection can identify a record to obtain but does not substitute for the finding itself [1,2].

Source / provider	Assessment date and time	Key finding / document location
EMS or scene assessment		
Emergency department		
Hospital or specialist examination		
Other contemporaneous source		

Clinical and laboratory findings relevant to Criterion 4

Domain	Test and timing	Result / norms / validity / confounders
Cognition		
Balance		
Oculomotor or vestibular-oculomotor challenge		
Blood biomarker		

Later review of an acute record is different from a newly performed remote test. Criterion 4 usually cannot be established post-acutely. Explain any test-specific exception, and avoid counting correlated outputs as independent evidence [1].

Structural imaging relevant to Criterion 5

CT / structural MRI _____ Date _____

Interpreting radiologist and report location:

_____

Intracranial finding and evidence for traumatic origin:

_____

_____

Imaging evidence ☐ Trauma-related lesion ☐ Normal ☐ Indeterminate ☐ Not obtained ☐ Unavailable

Patient report only? ☐ Yes ☐ No Actual report reviewed? ☐ Yes ☐ No

Lowest valid GCS more than 30 minutes after injury _____ Assessment time _____ Source _____

Factors affecting GCS validity or interpretation:

_____

Subsequent course corroboration and discrepancies

Patient ID _____ Event date _____

This section characterizes the course and present function. It does not turn later symptoms into evidence that the same symptoms were present acutely.

32. How did the symptoms change over the following days, weeks, and months?

Identify improvement, remission, recurrence, treatment response, and any later injury or illness.

Response: _____

_____

33. What activities first made a problem noticeable, and when were those activities resumed?

Clarify work, school, reading, driving, physical exertion, interruptions, or simultaneous tasks. Distinguish a reported difficulty from a demonstrated deficit.

Response: _____

_____

34. What can you do now, and under what conditions does performance become difficult?

Record concrete examples, assistance, compensatory strategies, endurance, and comparison with preinjury functioning.

Response: _____

_____

35. Do any records or other accounts differ from your recollection?

Discuss specific denials, symptom-free intervals, timing differences, or treatment responses neutrally. Record the explanation and what remains unresolved.

Response: _____

_____

36. Who can provide independent information about the acute event or subsequent change?

List the person, relationship, period directly observed, information sought, and whether contact or records are available.

Response: _____

_____

Records or observations that would most change the assessment:

_____

Interview limitations affecting interpretation (recall, communication, inconsistent accounts, third-party influence): _____

Diagnostic coding worksheet for history

Patient ID _____ Event date _____

Complete after the interview and source review. This is an original evidence worksheet, not the published ACRM coding form. The C labels refer to the diagnostic criteria, not to a point score [1,2].

C1 Mechanism ☐ Supported ☐ Equivocal ☐ Not supported

Mechanical event and supporting source:

_____

_____

If the mechanism remains equivocal, continue reviewing the acute evidence. The official coding form includes equivocal mechanisms under C1 met. The Administration Guide recommends a TBI diagnosis in such cases when C2 or C3 plus C4, together with C6, provides clear and compelling evidence of acute brain dysfunction. Document the uncertainty and the reasoning supporting the final judgment [2].

C2 Acute clinical signs

Candidate sign	Present / absent / uncertain	Timing or duration and source
Immediate loss of consciousness		
Immediate altered mental status		
Post-traumatic amnesia		
Retrograde amnesia if PTA cannot be assessed		
Other acute neurological sign		

Examples of an altered mental-status sign include disorientation, inappropriate or delayed responses, and impaired ability to follow instructions. A subjective dazed feeling alone belongs in C3. Complete the confounder assessment before determining whether a candidate sign qualifies [1].

C2 after consideration of C6 ☐ Met ☐ Not met ☐ Uncertain

Qualifying sign and reason for attribution:

_____

C3 Acute symptoms

Number of distinct qualifying acute symptoms _____

Symptoms counted with onset and source:

_____

_____

C3 after consideration of C6 ☐ Met with 2 or more ☐ Not met ☐ Uncertain

Immediate subjective mental-status symptoms must occur immediately after impact or recovery of consciousness. Other counted symptoms must be new or worsened within 72 hours. Document other complaints separately [1,2].

Diagnostic coding worksheet for objective evidence and confounders

Patient ID _____ Event date _____

C4 Clinical examination and laboratory findings

Finding status ☐ At least 1 qualifying finding ☐ None established ☐ Unavailable ☐ Uncertain

Number of qualifying findings _____ Dates _____

Finding and basis for eligibility including timing and validity:

_____

_____

Eligible domains are acute cognitive impairment, acute balance impairment, acute oculomotor impairment or vestibular-oculomotor symptom provocation, and elevated blood biomarkers indicative of intracranial injury. Reliability, diagnostic accuracy, reference standards, and alternative explanations must be considered. Normal values in one domain do not exclude an injury or dysfunction elsewhere [1].

C5 Structural neuroimaging

Status ☐ Met ☐ Not met ☐ Indeterminate ☐ Unavailable or not obtained

Unambiguous trauma-related intracranial abnormality and actual report:

_____

C6 Influence of competing explanations

For each manifestation relied upon, document whether an alternative explanation is absent, present without material influence, partially explanatory, fully explanatory, or of uncertain influence. Identify the reasoning, not just the existence of a condition.

Manifestation or finding	Alternative explanation and evidence	Effect on attribution or duration
Loss of consciousness		
Post-traumatic / retrograde amnesia		
Altered mental status / other sign		
Acute symptoms being counted		
Clinical or laboratory findings		

C6 for the proposed sign-based route: ☐ Met ☐ Not met ☐ Unclear

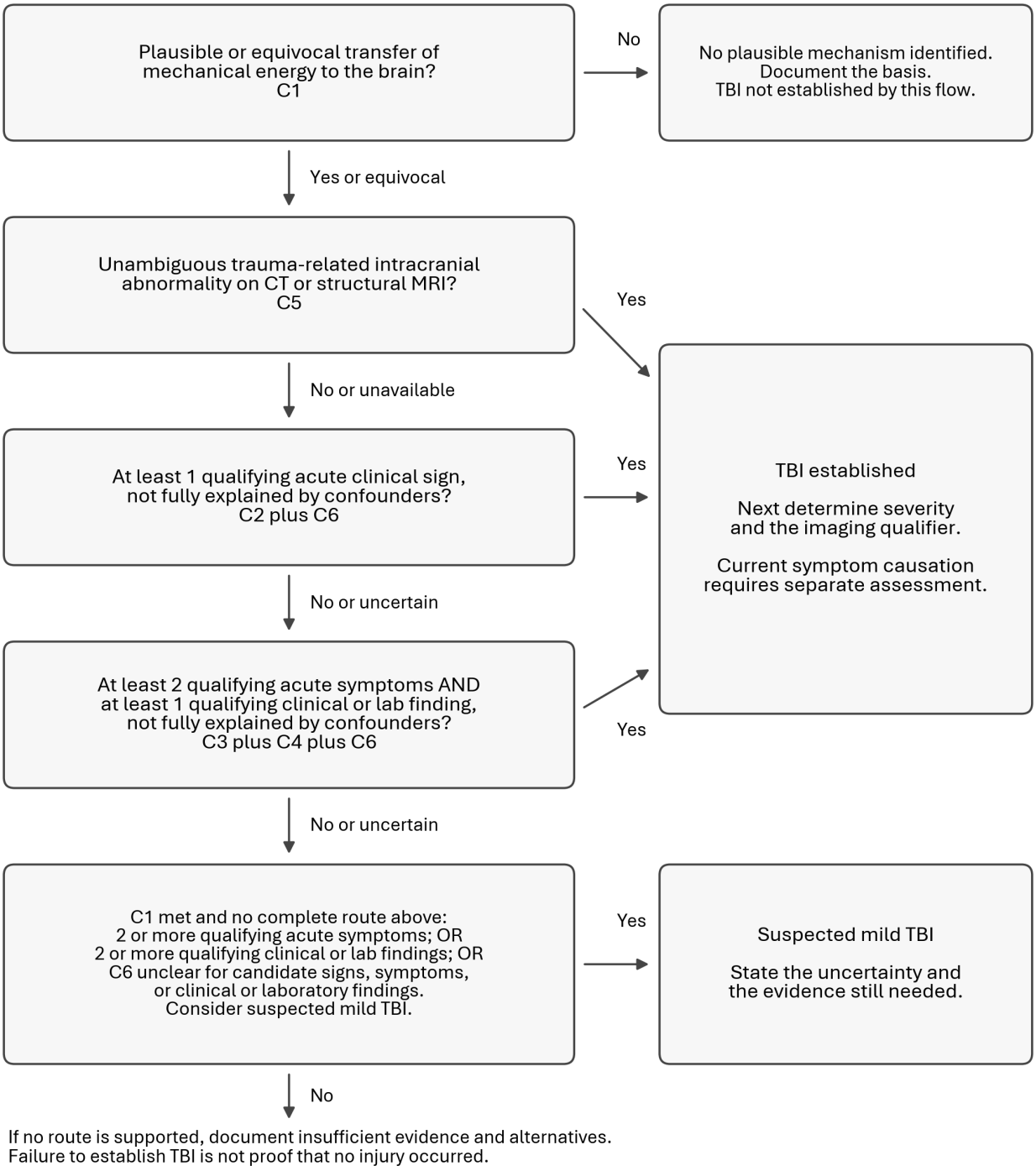
C6 for the proposed symptoms-plus-findings route: ☐ Met ☐ Not met ☐ Unclear

Partial contribution does not necessarily exclude TBI. If alternatives fully explain the evidence required for a route, that route is not established. If their contribution is unresolved, retain the uncertainty. Do not add the criterion designations into a total score [1].

Diagnostic flow from evidence to classification

Patient ID _____ Event date _____

Original diagram of the ACRM diagnostic pathways [1,2]. Apply after the coding worksheets. An uncertain branch means that the next route should be examined, not that injury has been disproved.



C1 mechanism; C2 signs; C3 symptoms; C4 examination or laboratory findings; C5 imaging; C6 confounders. Count only evidence that satisfies the relevant definitions. A fully explanatory alternative does not support a suspected diagnosis on the same evidence [1]. An equivocal mechanism warrants further assessment; the guide calls for clear and compelling acute evidence before diagnosing TBI in such cases [2].

Severity imaging qualifier and clinical opinion

Patient ID _____ Event date _____

First decide whether TBI is established. Then assign severity from reliable acute indicators. When indicators differ, the higher supported severity governs. Current symptom burden alone does not retrospectively set acute injury severity [1,2].

Indicator	Mild range	Beyond the mild range
Loss of consciousness	Absent or no more than 30 minutes	More than 30 minutes
Valid GCS after 30 minutes	13 to 15 when available	Less than 13
Post-traumatic amnesia	Absent or no more than 24 hours	More than 24 hours

A missing GCS is not a low GCS. If traumatic LOC or PTA is established but duration cannot be determined, severity may remain unknown. If a confounder could have prolonged an estimate but even the total duration remains within the mild range, that fact can still support mild classification. Retrograde amnesia duration is not a severity threshold [1,2].

Diagnostic conclusion ☐ Evidence supports no TBI ☐ Insufficient evidence ☐ Suspected mild TBI ☐ TBI established

If established ☐ Mild ☐ Greater than mild ☐ Severity undetermined

Imaging qualifier
☐ With neuroimaging evidence of structural intracranial injury
☐ Without neuroimaging evidence of structural intracranial injury
☐ No imaging qualifier

Use the negative imaging qualifier only when structural imaging was obtained and normal. If imaging was not performed, no imaging qualifier is used. An indeterminate finding requires explanation. The term concussion may be used for mild TBI when imaging is normal or not clinically indicated [1].

Diagnostic route and decisive evidence: _____

_____

Missing information or uncertainty that limits the conclusion:

_____

_____

Current disorders and separate assessment of their attribution:

_____

_____

Further information needed and plan for review:

_____

Clinician _____ Signature _____

Date _____

A diagnosis not established from available evidence is different from an adequately supported conclusion that no TBI occurred. State which conclusion is intended and why.

Official materials short form and references

Patient ID _____ Event date _____

The official package is supplied with the Lange, Gillow, and colleagues publication [2]. The links below lead to the publisher's materials. They allow readers to obtain the original questions, administration instructions, coding form, and flow diagram rather than treating this independently written appendix as the standardized instrument.

Publisher article and Supplementary Material section

Stable website: https://doi.org/10.1177/08977151251396077

Full ACRM Structured TBI Interview

Administration Guide

Diagnostic Coding Form

Diagnostic Flow Diagram

ACRM Structured TBI Interview Short Form

In print, open the stable DOI website above and select Supplementary Material. If direct links change or access fails, the authors list ACRM.TBI.Interview@gmail.com for requesting the package. Links checked October 26, 2026.

The short form

The official Short Form offers a briefer history when the comprehensive interview is impractical. It is intended for experienced interviewers who already understand the ACRM criteria. It condenses or omits selected questions and allows optional skips after sufficient evidence for a criterion is obtained. The authors favor the full interview when feasible and caution that abbreviation can miss diagnostic or severity information [2, supplementary Short Form].

The shorter format does not remove the need to assess confounding factors or obtain the records needed for examination, imaging, and severity judgments. Its convenience does not establish psychometric validation. At the time of publication, the authors reported that validation of the interview package was still needed [2].

References

1. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. Arch Phys Med Rehabil. 2023;104(8):1343-1355. doi:10.1016/j.apmr.2023.03.036.

2. Lange RT, Gillow KC; ACRM Brain Injury Special Interest Group Mild TBI Task Force members. Development of a structured interview for the American Congress of Rehabilitation Medicine diagnostic criteria for mild traumatic brain injury. J Neurotrauma. 2026;43(11-12):934-943. Published online November 24, 2025. doi:10.1177/08977151251396077. See accompanying supplementary materials.

Attribution: The diagnostic concepts originate in the cited ACRM work. The question wording, form organization, evidence worksheets, and diagram in this appendix were independently prepared for this book. This appendix does not claim ACRM authorship, endorsement, standardized equivalence, or validation.

Appendix C. ACRM criteria and retrospective diagnosis

This appendix summarizes the 2023 American Congress of Rehabilitation Medicine (ACRM) criteria as explained by Iverson and colleagues in their 2025 article on medicolegal considerations. The diagnostic pathways and sign/symptom lists follow that article's Figures 1-3; the fuller definitions and qualifications are checked against Boxes 1-2 of the 2023 publication. This is an original summary, not new criteria or an official ACRM form. Appendix B organizes the evidence; this reference explains what it can establish. [1,2]

The three routes to diagnosis

Begin with Criterion 1: a plausible transfer of mechanical energy to the brain. Examples include an object striking the head, the head striking a surface, acceleration or deceleration without a direct head impact, and blast forces. An impact alone does not establish brain injury. [1]

Route	Required evidence
Clinical-sign route	At least one qualifying acute clinical sign (C2), with competing explanations assessed (C6).
Symptoms-plus-findings route	At least two qualifying acute symptoms (C3) and at least one qualifying clinical-examination or laboratory finding (C4), with competing explanations assessed (C6).
Structural-imaging route	An unambiguous trauma-related intracranial abnormality on CT or structural MRI (C5).

Any one route can establish TBI after a plausible mechanism. Establishing that the injury is mild requires a separate appraisal of acute severity; an abnormal scan alone does not settle severity. For the first two routes, the findings relied on must be attributable to brain injury and not fully explained by confounders. [1,2]

Mild severity and the imaging qualifier

Do not use the mild qualifier if a reliable acute indicator shows loss of consciousness longer than 30 minutes, a Glasgow Coma Scale score below 13 after 30 minutes, or post-traumatic amnesia longer than 24 hours. Missing information is not a normal finding; document when injury severity remains uncertain. Current symptom burden does not retrospectively determine initial severity. [1,2]

For an injury already classified as mild, qualifying structural imaging supports the wording mild TBI with neuroimaging evidence of structural intracranial injury. When imaging was obtained and normal, without neuroimaging evidence of structural intracranial injury may be used. If imaging was not performed, use no imaging qualifier. The term concussion may be used interchangeably with mild TBI when imaging is normal or not clinically indicated. [1]

Criterion 2 acute clinical signs

- Immediate loss of consciousness. Examples include failing to protect oneself while falling after impact or being motionless and unresponsive.
- An observable change in mental status immediately after injury or after consciousness returns: reduced or inappropriate responsiveness, unusually slow answers or responses to instructions, agitation, inability to follow a two-part command, or disorientation to time, place, or situation.
- Partial or complete loss of memory for events immediately after injury or after consciousness returns. If post-traumatic amnesia cannot be assessed reliably, a gap in memory immediately before injury may substitute; the reason for using that substitution should be clear.
- Another acute neurological sign, such as incoordination when first standing, a seizure, or sustained abnormal posturing immediately after injury.

A sign need not have been witnessed at the time; it must describe a disturbance that could have been observed. Evidence may come from observation or video, a witness, acute records, or, when these are unavailable, the person's detailed account. Feeling dazed or confused without an observable disturbance belongs under symptoms, not automatically under clinical signs. A memory gap does not by itself prove unconsciousness. [1,2]

Criterion 3 acute symptoms

Count at least two distinct symptoms that were new or worse after injury. They may come from the same category. [1]

Category	Symptoms included
Subjective change in mental status	Feeling confused, disoriented, or dazed.
Physical	Headache, nausea, dizziness, balance difficulty, vision problems, sensitivity to light, sensitivity to sound.
Cognitive	Feeling mentally slowed, mental fog, difficulty concentrating, memory problems.
Emotional	Uncharacteristic emotional lability or irritability.

The subjective mental-status change must occur immediately after impact or when consciousness returns. The other symptoms may emerge over hours but must have been present to some degree within the first 72 hours to count toward this criterion. Later recognition, including when exertion exposes a problem, is different from a symptom that first begins much later. Other important complaints, such as fatigue or sleep disturbance, should be evaluated but do not count toward C3. [1,2]

Criterion 4 examination and laboratory findings

- Cognitive impairment on an acute examination.

- Balance impairment on an acute examination.
- Impaired eye-movement function, or symptoms provoked by an acute vestibular-oculomotor challenge.
- Elevated blood biomarkers indicating intracranial injury.

Use tests with adequate reliability and diagnostic accuracy. Impairment should represent a clinically meaningful difference from age-appropriate norms or, when available, valid preinjury performance. Timing matters: diagnostic sensitivity generally falls during the first 72 hours, at different rates for different tests. A newly performed post-acute examination usually cannot establish C4. Reviewing an authentic acute test later is different from treating a late abnormal test as if it documented the acute state. [1]

Criteria 5 and 6 imaging and competing explanations

C5 concerns a trauma-related intracranial abnormality on CT or structural MRI, such as a contusion or hemorrhage. Imaging is not required for an mTBI diagnosis, and normal structural imaging does not exclude it. An abnormal diffusion tensor imaging (DTI) measurement or a functional imaging result, including SPECT, does not satisfy C5 in this framework. [1,2]

For C6, assess whether another explanation fully accounts for the signs, symptoms, or examination/laboratory findings needed for the proposed route. Relevant possibilities include medication or substance effects, acute pain or other injuries, acute psychological distress, pre-existing conditions, syncope, impaired circulation or breathing, and problems affecting the validity of symptom reports or tests. A competing condition can coexist with TBI. Document what it explains and what remains attributable to the injury; if attribution cannot be resolved, state that uncertainty. [1,2]

When the classification is suspected mTBI

After a plausible mechanism, suspected mTBI can be used when no complete diagnostic route is established but there are at least two qualifying acute symptoms, or at least two qualifying examination/laboratory findings. It can also apply when it remains unclear whether confounders account for the relevant signs, symptoms, or available examination/laboratory evidence. [1]

Suspected is a diagnostic category, not a percentage. It does not place an opinion above or below 50% certainty. The clinician should separately explain the degree of confidence, the evidence supporting it, and what remains unresolved. The 2025 article states that suspected mTBI is clinically managed as mTBI; the category itself does not settle a medical-legal burden of proof. [2]

Using the criteria months or years later

Retrospective diagnosis is permitted. Reconstruct what happened and what followed through a detailed interview and available records and collateral accounts. An acute evaluation usually provides the strongest opportunity for diagnostic certainty, but the absence of a documented sign is not proof that no sign occurred. Distinguish the person's memories from information

learned later, and distinguish missing documentation from an explicit negative observation. Consider recall, comprehension, language, interview methods, and examiner interpretation. [1,2]

Sedation, analgesia, or competing injuries may mask early symptoms. A problem first recognized under exertion may also have been present earlier. These situations require evidence about timing, not an assumption that all later symptoms were acute. The 2025 article separately recognizes that documented progression of an intracranial injury can produce later symptoms. [2]

Account for cultural and linguistic differences, developmental stage, and preinjury cognitive or communication limitations. Appropriate proxy observations may help when a person cannot report symptoms reliably, and test standards must suit the person's age. [1]

Finally, these criteria identify an injury. They do not predict recovery or establish that every later health problem was caused by that injury. Nor does a mild acute classification promise minor symptoms or a brief recovery. Diagnosis, present impairment, prognosis, and causation of current problems each require their own reasoning. [1,2]

References

1. Silverberg ND, Iverson GL; ACRM Brain Injury Special Interest Group Mild TBI Task Force; ACRM Mild TBI Diagnostic Criteria Expert Consensus Group. The American Congress of Rehabilitation Medicine Diagnostic Criteria for Mild Traumatic Brain Injury. Archives of Physical Medicine and Rehabilitation. 2023;104:1343-1355. doi:10.1016/j.apmr.2023.03.036.

https://doi.org/10.1016/j.apmr.2023.03.036

2. Iverson GL, Dams-O'Connor K, Panenka WJ, et al. New Diagnostic Criteria for Mild Traumatic Brain Injury: Medical-Legal Considerations. Archives of Physical Medicine and Rehabilitation. 2025;106:1615-1619. doi:10.1016/j.apmr.2025.06.015.

https://doi.org/10.1016/j.apmr.2025.06.015