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Program Outline

10 Modules over 4 Hours

01

Whiplash vs WAD definition, dimensions, clinical burden and classification

02

The injury mechanism: reasoning from collision physics

03

The physical trauma: what are the tissue injuries?

2

Program Outline

04

From acute injury to chronic whiplash associated disorder

05

History, triage and differential diagnosis

06

The role of imaging – decision rules and advanced modalities

3

Program Outline

07

Clinical Examination: history guided and domain matched

08

Working Diagnosis: grading, pain mechanisms and domains

09

Prognosis: risk stratification and recovery pathways

10

Management: evidence informed, acute, subacute and complex chronic presentations

4

Learning Objectives

1. Describe the biomechanics of acceleration–deceleration (whiplash) injury affecting the cervical spine and brain
2. Recognise the clinical features of whiplash associated disorder (WAD) and early prognostic indicators for risk stratification.
3. Perform relevant orthopaedic and neurological examinations on the whiplash patient
4. Apply evidence-based clinical decision rules for imaging and referral.

5

Learning Objectives

5. Formulate a clear and relevant diagnosis incorporating QTF grade and contemporary pain neuroscience mechanisms
6. Apply validated clinical outcome measures and rating scales in WAD assessment.
7. Develop evidence-informed management strategies across acute, subacute and chronic phases of WAD.
8. Recognise indications for interdisciplinary referral and co-management.

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PROGRAM PURPOSE

Develop specialist-level clinical reasoning—not protocol memorization.

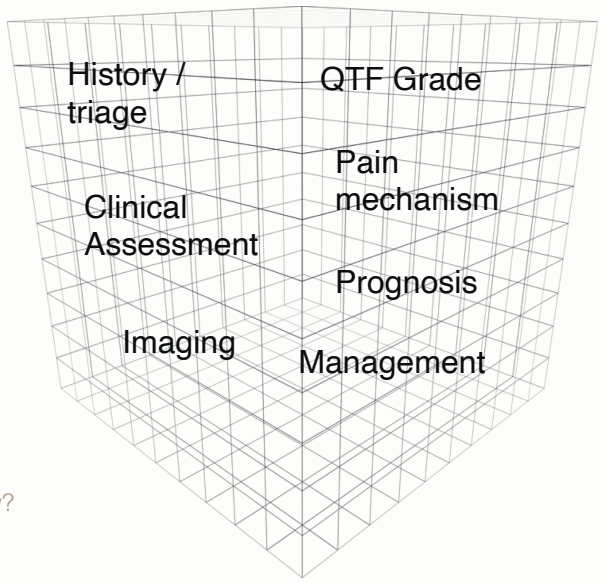
Graduates evaluate the traumatic event, screen for serious pathology, grade the severity of the injury, identify the peripheral drivers and dominant pain mechanisms, formulate a defensible diagnosis, match management strategy to clinical domains and recovery stage and professionally communicate their role.

7

Clinical problem-solving

Let's start by considering your current model for approaching the whiplash patient. Think about your mental process…

- What are the most likely injuries?
- What is important in the history?
- When do you image?
- What diagnosis do you make?
- What are the dominant pain mechanisms?
- What decides management and predicts chronicity?



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MODULE 1

WHIPLASH – THE CLININAL PROBLEM

The definition, clinical dimensions, burden and classification of whiplash and whiplash associated disorder (WAD)

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MODULE 1

Let's consider:

1. The definition of whiplash and WAD
2. The dimensions and clinical burden of WAD
3. The initial classification of whiplash and WAD
4. The neuroscience evolution of our models for WAD
5. The clinical and assessment domains of WAD

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DEFINITION OF WHIPLASH

Whiplash itself has been defined as a mechanism of energy transfer through the tissues of the neck that is often beyond their capacity to dissipate

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

11

The dimensions and clinical burden of WAD

Recovery rates for whiplash have remain unchanged for decades and generally accepted that:

50%

Of individuals experiencing whiplash from a MVC will recover within 12 weeks

Casey PP, Feyer AM, Cameron ID. Course of recovery for whiplash associated disorders in a compensation setting. Injury. 2015; 46:2118-2129.

50%

Will continue to experience symptoms to some degree and most of those continue to reports symptoms 12 months later

Carroll LJ, Hogg-Johnson S et al. Course and prognostic factors for neck pain in the general population: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders. Spine Phila Pa 1976). 2008;33: S75-S82.

3 months

If recovery occurs, it tends to occur within 3 months. Constellation of symptoms occurring after that is termed WAD

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

30 Years

Despite large research effort, rate of acute injury and transition to chronic pain has not changed much in 30 years

Sterling M, Hendrikz J, Kenardy J. Compensation claim lodgement and health outcome developmental trajectories following whiplash injury: a prospective study. Pain. 2010; 150:22-28.

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Whiplash compared to non-traumatic neck pain

Non-traumatic neck pain (NTNP) shares a constellation of complex symptoms, however:

The recovery trajectories of WAD are worse than non-traumatic neck pain (NTNP)

Patients with WAD are less responsive to treatment with trials of exercise interventions showing little effect in WAD compared to effects in NTNP

What is it about a whiplash injury that makes it behave so differently to NTNP?

Chen, J. et al. Differences in the clinical presentation of chronic whiplash-associated disorders and nontraumatic neck pain: a systematic review and meta-analysis. PAIN 166, 1738–1756 (2025).

13

An agreed classification of whiplash was needed

By1990s, clinicians and compensation systems faced an inconsistent and costly problem.

01

Clinical burden

Frequent symptoms after motor vehicle collisions and rising disability claims.

02

Diagnostic variation

No shared definition or consistent way to classify presentations.

03

Treatment uncertainty

Wide variation in care with little agreement on effective treatment.

04

Policy pressure

Insurers needed clearer prognosis, care pathways and cost control.

The Quebec Task Force was designed as both a clinical and policy response.

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How the 1995 Task Force approached the problem

The report combined population follow-up, evidence review and expert consensus.

01

Cohort study

Followed thousands of compensated patients to examine recovery, recurrence and disability costs.

02

Evidence synthesis

Reviewed the available literature and found limited high-quality evidence for most treatments.

03

Consensus recommendations

Used expert judgement to make clinical and policy recommendations where evidence was absent.

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QTF CONTRIBUTION

The Task Force created a common language for a previously inconsistent field.

It defined whiplash as an acceleration-deceleration energy transfer, introduced WAD as a clinical syndrome and formalized grades 0 to IV.

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The Quebec Task Force grading system

GRADE	CLASSIFICATION
0	No complaint about the neck. No physical sign(s).
I	Complaint of neck pain, stiffness or tenderness only. No physical sign(s).
II	Neck complaint AND musculoskeletal sign(s). Musculoskeletal signs include decreased range of movement and point tenderness.
III	Neck complaint AND neurological sign(s). Neurological signs include decreased or absent tendon reflexes, weakness and sensory deficits.
IV	Neck complaint AND fracture or dislocation.

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The Quebec Task Force grading system

0	I	II	III	IV
No neck complaint	**Subjective** complaint, no **objective** physical signs	Musculoskeletal signs, no neurological **signs**	Neurological signs	Fracture or dislocation

The grade identifies clinical signs. What does it leave unexplained?

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What the QTF changed in practice

Its influence extended well beyond classification.

01

Shared language

WAD grades entered clinical notes, research criteria and practice guidelines.

02

Active care

Reassurance and early return to comfortable movement became central.

03

Less immobilisation

Routine collars, prolonged rest and passive care lost support.

04

Imaging restraint

Most presentations lacked serious structural injury, and imaging often poorly matched symptoms.

These principles remain embedded in contemporary WAD care.

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The management philosophy changed

QTF helped move care away from protection through inactivity.



The durable QTF legacy is active recovery with proportionate investigation.

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What the QTF grades capture and what they miss

Classification organizes observable signs but can't explain the reason for pain or the trajectory.

THEY CAPTURE

- Complaint status
- Musculoskeletal signs
- Neurological signs or fracture

THEY DO NOT EXPLAIN

- Pain intensity and disability
- Symptoms beyond the neck
- The dominant mechanism

THEY DO NOT PREDICT

- Who develops persistent symptoms
- The pace of recovery
- The required care intensity

Two patients can share a grade but have very different conditions and trajectories.

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It depends upon what we mean by 'recovery'

The cohort findings were influential, but the method shaped what counted as recovery.

COHORT FRAME

- Insured and compensated patients
- Administrative records
- A major policy question

RECOVERY ENDPOINT

- End of compensation payments
- Not necessarily symptom resolution
- Not a full multidomain assessment

INTERPRET WITH CARE

- How recovery is defined
- Persistent symptoms may be underestimated
- Administrative and clinical recovery can diverge

Claim closure is an administrative outcome; it is not synonymous with full clinical recovery.

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THE EXPLANATORY GAP

The persistent subgroup challenged the simple recovery narrative.

The QTF described a generally favorable course, but could not fully explain widespread pain, dizziness, fatigue, cognitive symptoms or lasting disability in a substantial minority.

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Neuroscience expanded our explanatory models

The field progressively incorporated more of the person and the context of trauma.



01 Peripheral	02 QTF	03 Fear	04 Diathesis	05 Integrated
Pain linked mainly to tissue lesion	Clinical signs classified and active recovery promoted	Threat beliefs and avoidance linked to persistence	Pre-existing vulnerability or protection alters response	Tissue, CNS, stress, behaviour and context interact

Each model added explanatory capacity while preserving the need for clinical assessment.

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Persistent WAD does not follow a single pathway

Peripheral injury may initiate symptoms while recovery reflects interacting biological and contextual processes.

ACUTE NOCICEPTION

- Sprain or strain of cervical tissues
- Inflammation and nociception
- Protective pain and guarding

ADAPTIVE RECOVERY

- Tissue repair and safe movement
- Restored sleep and confidence
- Support for normal participation

CHRONIC PAIN & DISABILITY

- Ongoing nociception or neural amplification
- Sensorimotor or stress dysregulation
- Reduced activity and participation

Peripheral and central mechanisms can coexist, and their relative contribution can change over time.

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Pain reflects threat appraisal across multiple inputs

The nervous system integrates information before producing pain and protective behavior.

SIGNALS

- Nociception from cervical tissues
- Proprioceptive information
- Visual and vestibular input

MEANING

- Prior experience and expectation
- Beliefs about damage and recovery
- Emotional and social context

OUTPUT

- Pain sensitivity and muscle guarding
- Autonomic arousal
- Avoidance or adaptive action

Pain remains real even when its intensity no longer maps directly to tissue damage.

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Australian guidelines 2014



Guidelines for the management of acute whiplash-associated disorders for health professionals, third edition 2014.

This document is endorsed by:

APS Australian Psychological Society · CAANSW Chiropractors' Association of Australia (NSW) · Australian Physiotherapy Association

State Insurance Regulatory Authority: Guidelines for the management of acute whiplash-associated disorders – for health professionals. Sydney: third edition 2014.

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Further recommendations published in 2023

Technical Report Chapter 2: Assessment

Australian Clinical Guidelines for Health Professionals Managing People with Whiplash-Associated Disorders, Fourth Edition

Carvalho-e-Silva, A. P., Papic, C., Chia, L., Cameron, I. D., & Rebbeck, T. (2023). Australian Clinical Guidelines for Health Professionals Managing People with Whiplash-Associated Disorders, Fourth Edition: Technical Report Chapter 2 Assessment. State Insurance Regulatory Authority: Sydney, Australia.

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8 Clinical & assessment domains of WAD

1. Structural & tissue injury
2. Neurological injury / function
3. Vascular safety screening
4. Physical musculoskeletal impairment
5. Sensorimotor function
6. Pain sensitivity
7. Psychological and contextual recovery
8. mTBI overlap & additional symptoms

Assessment domain	Subcategories
Physical Musculoskeletal Impairment	Trigger points, Cervical Range of Motion (ROM) Muscle performance/postural changes Muscle function
Sensorimotor	Cervical joint position error Cervical movement sense Oculomotor disturbance Balance Coordination Test Others – Proprioception
Pain sensitivity	Pressure hyperalgesia Thermal hyperalgesia Dynamic pain sensitivity testing Quantitative Sensory Testing (QST) (vibration) Brachial Plexus Provocation Test (BPPT) Nociceptive Flexion Reflex (NFR) Others
Additional Psychological Factors	Fear avoidance Self-efficacy Mental disorder Psychological distress Cognitive deficits
Additional Symptoms	Additional symptoms Sleep disturbance Jaw symptoms Disability

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Recovery risk extends beyond the WAD grade

Initial pain and disability matter, while multidomain features refine prognosis.

01

Favorable pattern

Lower pain and disability, comfortable early movement and confidence in recovery.

02

Higher symptom load

High initial pain or disability and widespread sensory sensitivity.

03

Multisystem features

Dizziness, visual disturbance, cognitive symptoms or suspected mTBI.

04

Context and distress

Fear, traumatic stress, poor sleep, limited support or reduced agency.

The skilled physician must deeply conceptualize the full scale of WAD in all it's clinical domains

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Modern management builds on the QTF foundation

The grade sits inside a broader model of the person's current presentation.

01

Tissue

Local nociception, structural pathology, tissue health and healing stage.

02

CNS and sensorimotor

Hypersensitivity, proprioception, vestibular function, vision and cognition.

03

Stress and psychology

Threat appraisal, beliefs, autonomic arousal, sleep and coping.

04

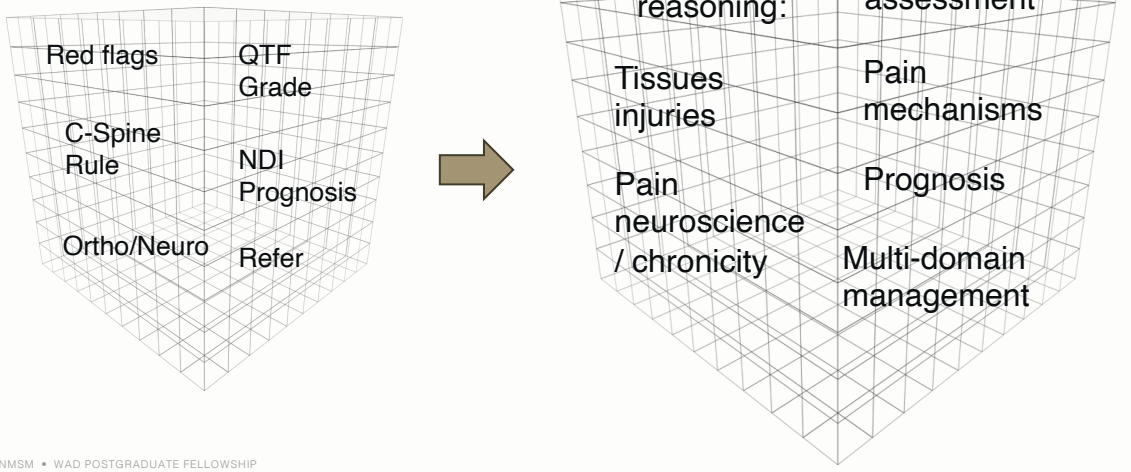
Context and function

Work demands, support, compensation, identity and participation.

Formulation identifies what is driving the presentation now and which finding would change the plan.

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Chiropractic physician to chiropractic specialist



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CLINICAL SYNTHESIS 1

The grade classifies signs. The specialist uses advanced reasoning to explains the person and their current condition.

QTF remains valuable for shared language and early management. Contemporary pain neuroscience explains heterogeneity, enables a more individualized diagnosis, more accurate prognosis and guides more targeted care.

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MODULE 2

Let's consider:

1. The effects of acceleration on the human body
2. The threshold for human injury
3. The kinematics of whiplash injury (velocity vs acceleration)
4. Patient specific factors influencing injury susceptibility

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High acceleration and the human body

High acceleration can occur at low velocity. What acceleration can humans sustain?

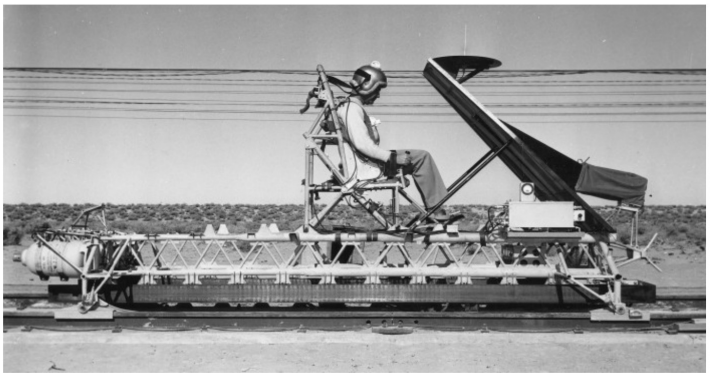


Figure 2: The 'Gee-whiz' rocket sled.

1946 - 1958
AAF Aero Lab

Col John Stapp
Investigated limits of voluntary tolerance to crash impacts and acceleration

High acceleration and the human body. M Voshell. 28th November, 2004. 32, 2004

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Acceleration is how quickly we change velocity

We don't 'feel' velocity, but we do 'feel' acceleration because it requires a FORCE



Figure 4: Col. Stapp Decelerating.

During his final run Col Stapp decelerated from 632 MPH to a complete stop in 1.4 seconds and experienced a record 46.2G of acceleration.

1g = 9.8 m/s/s

Force of 9.8N for every kg of mass

So why can MVC at 5MPH cause lasting injury?

High acceleration and the human body. M Voshell. 28th November, 2004. 32, 2004

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What is the threshold for human injury?

The change in velocity over the time it occurs (Delta-V) is the key factor

NATIONAL ACADEMY OF FORENSIC ENGINEERS

http://www.nafe.org

ISSN: 2379-3252

Vol. XII No. 2 December 1995

- Typical duration for low-speed rear end collision is 100 – 150 milliseconds
- The Threshold of Injury for Human Test Subjects is ~ 4 to 5 MPH Delta-V (6.4 to 8 km/hr)
- Tests at these levels resulted in peak G forces of 3.9 to 5.2G in lumbar spine, 4.5 to 7.4G in cervical spine and 10.1 to 13.7G at head position
- Cough ~ 3.5G, hopping of 8-inch step ~ 8.1G, plopping into desk chair ~ 10.4G blow to head of boxer ~ 70G

Baxter, Gene K. 1995. "G-Force Analysis For Low-Speed Rear-End Collisions". Journal of the National Academy of Forensic Engineers 12 (2).

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3. The kinematics of whiplash injury

[CLINICAL COMMENTARY]

BRIAN D. STEMPER, PhD[1,2] • BRIAN D. CORNER, PhD[3]

Whiplash-Associated Disorders: Occupant Kinematics and Neck Morphology

Whiplash injuries resulting from low-velocity automotive impacts continue to be a significant cause of neck pain and disability in the general population.[104] Despite considerable research effort, the incidence of acute injury[41] and rate of individuals transitioning to chronic pain symptoms have not remarkably changed over the past 30 years.[64,98]



to loading experienced during automotive impacts. These findings highlight the application of mechanical engineering principles to human anatomy, which forms the basis for the science of biomechanics. From a mechanical engineering stand-

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Rear impact is a coupled seat–torso–head event

The neck is loaded by relative motion—not by the head moving in isolation.

01

Vehicle and seat accelerate

The struck vehicle and seatback move forward with the collision pulse.

02

Torso is driven forward

Seatback contact accelerates the thorax; seat deformation absorbs part of the energy.

03

Head initially lags

Head inertia creates posterior translation relative to the accelerating torso.

04

Restraint and rebound

Head-restraint contact limits rearward motion before the occupant rebounds into flexion.

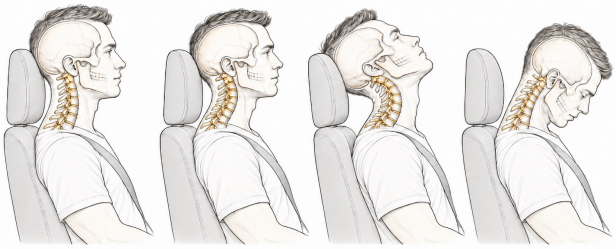
Stemper BD, Corner BD. Whiplash-Associated Disorders: Occupant Kinematics and Neck Morphology. J Orthop Sports Phys Ther. 2016;46(10):834–844

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Whiplash kinematics evolves within milliseconds

Early segmental S-curve precedes global extension and may be the critical tissue-loading phase.



NEUTRAL	S-SHAPED RETRACTION	C-SHAPED EXTENSION	REBOUND FLEXION
Pre-impact	50–75 ms	150–175 ms	After ~200 ms*

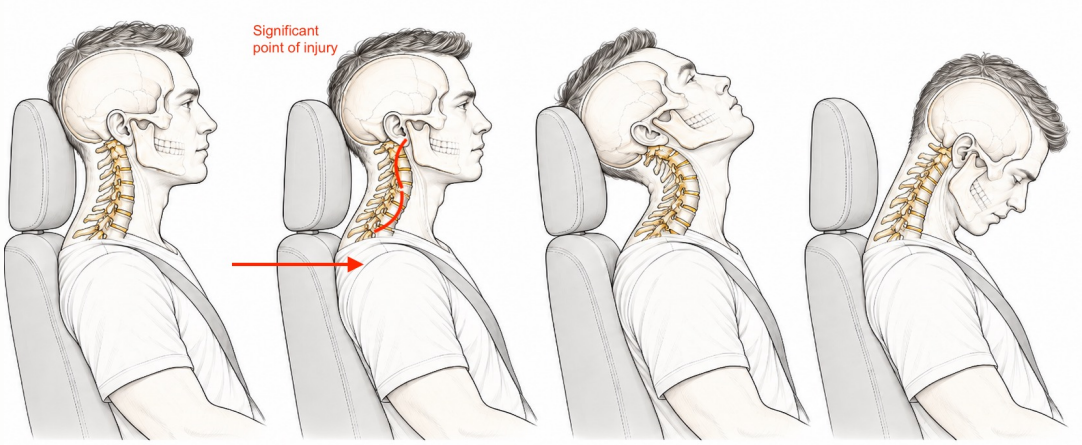
*Rebound begins after ~200 ms; timing varies with crash pulse and seat–restraint geometry.

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Non-physiological segmental motion

Cervical spine is compressed from below, causing lower cervical extension and upper cervical flexion



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The S-curve creates nonphysiologic segmental loading

At approximately 50–75 ms, the cervical column is not moving as one uniform arc.

UPPER CERVICAL

- Relative flexion
- Head translates posteriorly
- Motion differs from the lower neck

LOWER CERVICAL

- Extension develops early
- Segmental shear and bending increase
- Normal coupling is temporarily disrupted

TISSUE CONSEQUENCE

- Facet capsules and discs may be strained
- Subfailure injury is mechanically likely
- Pain need not require gross structural failure

The earliest potentially injurious phase may occur before the dramatic appearance of global extension.

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The C-curve is global extension—not the first event

Separating the phases prevents common assumption that whiplash is simply 'hyperextension'.

S-CURVE • 50–75 ms

- Upper flexion + lower extension
- High segmental deformation
- Most likely early injury window

C-CURVE • 150–175 ms

- All cervical levels extend
- Head rotates posteriorly
- Anterior tissues may be tensioned

REBOUND • AFTER ~200 ms

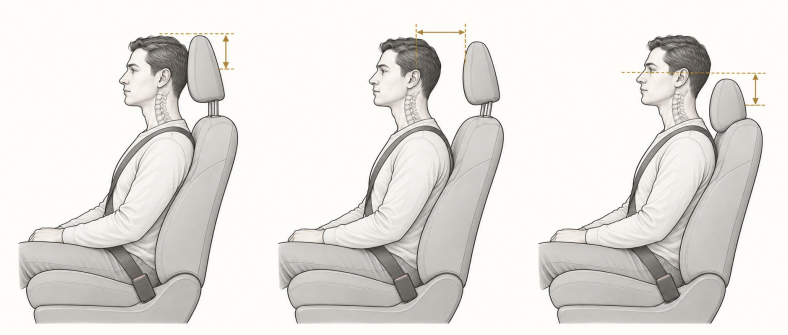
- Head-restraint and seat rebound
- Forward head-neck flexion
- Timing varies with system geometry

A complete account requires translation, segmental deformation, extension and rebound—not one motion alone.

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Head-restraint geometry changes the kinematic pathway

Height and horizontal backset determine when—and where—the head is arrested.



MINIMAL BACKSET
Earlier support

EXCESSIVE BACKSET
More rearward translation

RESTRAINT TOO LOW
Potential rotation over the restraint

Smaller backset generally limits rearward head translation earlier; excessive backset or poor height increases the opportunity for relative motion.

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What are the kinematic factors that *could* influence injury?

Epidemiological findings are mixed; the clinically useful question is which individual characteristics alter loading.

OBSERVATION

- Some studies report greater female risk
- Other reviews find no independent effect
- Female volunteers often show greater motion

PLAUSIBLE MEDIATORS

- Body and torso dimensions
- Head-restraint fit
- Neck slenderness and vertebral size
- Muscle capacity and moment arms

CLINICAL INTERPRETATION

- Do not infer risk from sex alone
- Measure the relevant **individual** features
- Separate susceptibility from prognosis

Morphology and seat interaction provide the mechanism; sex is an imperfect proxy for those variables.

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Cervical alignment changes how impact energy is distributed

The proposed mechanism is load transfer through the whole column—not the diagnostic meaning of a post-crash radiograph.

LORDOTIC COLUMN

- Bending can distribute and absorb energy
- Segmental coupling remains more physiologic
- Alignment is only one part of capacity

STRAIGHT OR KYPHOTIC MODEL

- Less curvature for energy absorption
- Greater localized segmental deformation
- Capsular distortion rose by up to 73% in one model

CLINICAL CAVEAT

- Curvature varies in asymptomatic people
- Post-injury straightening is not proof of lesion
- Mechanistic association ≠ outcome prediction

Use pre-existing alignment as contextual information; do not equate loss of lordosis with the cause of symptoms.

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Age and degeneration have a non-linear mechanical effect

Ageing tissues generally lose material strength, but advanced structural change may also reduce motion.



Lower ligament modulus and rupture stress	Disc height and annular integrity decline	Moderate degeneration may increase flexibility	Greater motion can increase local tissue strain	Severe osteophytes may partly restabilise a level

'More degeneration = more injury' is too simple: severity, distribution and segmental mobility matter.

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CLINICAL SYNTHESIS

Susceptibility emerges from the fit between occupant, seat and acceleration pulse.

Kinematics explain how tissues may be loaded. Individual morphology and capacity modify that loading. Neither alone provides a validated bedside prediction of injury severity or recovery.

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MODULE 3

THE TISSUE INJURIES

Consider the potential for whiplash injury to causes trauma to facet joints, capsules, synovial folds (meniscoids), discs, ligaments, muscles, peripheral nerves, spinal cord and brain

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MODULE 3

Let's consider the effect of whiplash on:

1. Facet joints and capsules
2. Synovial folds (meniscoids)
3. Ligaments
4. Cervical discs
5. Muscles
6. Peripheral nerves
7. Spinal cord and Brain

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THE CENTRAL PROPOSITION

A spectrum of subfailure injuries characterizes the most common whiplash presentations

Segmental kinematics predict high local strain. Controlled blocks have identified the most commonly painful joints. Experimental subfailure produces microscopic collagen disruption, sustained pain behavior and a distributed neural response—without gross rupture.

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1. Where is the neck vulnerable?
2. Where is disc degeneration most common?
3. Most likely level of instability?
4. Most common level for nerve root compression?



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1. Facet joints and capsules

Individual segments can exceed physiological motion – especially during S-curve phase.

C2–3

- Upper cervical flexion during the S-curve
- Clinically important source of post-whiplash headache
- Third occipital nerve provides a defined diagnostic target

C5–6 / C6–7

- Early lower-cervical extension
- Large segmental translation and capsular strain
- Common levels in experimental and clinical evidence

MECHANICAL LESSON

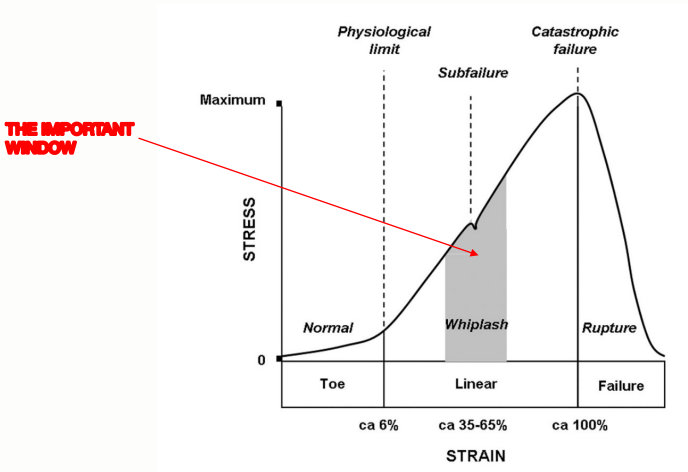
- Global range does not reveal local tissue strain
- Small segments can move nonphysiologically
- Gross failure is not required for injury

The relevant mechanical unit is the motion segment—not simply the total arc of head–neck extension.

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Stress–strain behavior explains the subfailure lesion

The capsule can pass its physiological range and sustain microscopic damage long before macroscopic rupture.



Damage before rupture

Whiplash strains fall well above the physiological range yet below gross failure. The yield-point inflexion represents altered collagen architecture: a subfailure lesion.

Adapted from Figure 1: Bogduk N, MacVicar J. Subfailure capsule strain as the cause of cervical zygapophysial joint pain after whiplash: a scoping review. Pain Med. 2026 Aug 1;27(8):883-892. doi: 10.1093/pm/pnag030. PMID: 41706694; PMCID: PMC13428909.

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Subfailure – between physiological motion and rupture

Pain-producing deformation begins far below catastrophic failure.

≈6%	23–27%	35–65%	≈100%
Physiological limit	**Pain behaviour**	**Whiplash strain**	**Gross rupture**
Slack collagen is recruited and the capsule enters its linear region.	Animal models show sustained pain behavior at strains in this range.	Experimental rear-impact studies commonly place capsular strain in this window.	Macroscopic failure occurs much later and varies widely among specimens.

These values describe experimental ranges as opposed to patient-specific injury thresholds.

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The 'lesion' is altered collagen architecture

Subfailure injury changes tissue structure and mechanics while preserving gross continuity.

MECHANICAL SIGNATURE

- Small yield-point inflexion
- At least ~10% reduction in slope
- Permanent change in stiffness

MICROSTRUCTURE

- Collagen fibers realign anomalously
- Crimp and fiber organization are disrupted
- Capsule remains macroscopically intact

CLINICAL VISIBILITY

- Conventional imaging may appear normal
- No gross rupture is required
- Pain generation remains biologically plausible

'No structural lesion seen' does not mean 'no tissue injury occurred'—the proposed lesion is microscopic.

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Facet joint pain – one of our best developed models

Our understanding of facet joint pain has evolved in stages.

1994–96

Controlled blocks

Comparative local-anaesthetic and placebo-controlled blocks established cervical zygapophysial joints as pain sources.

1996+

Radiofrequency

Medial-branch neurotomy showed that interrupting defined afferents can produce complete pain relief in selected patients.

2000s+

Subfailure models

Experimental capsule loading produced sustained pain behaviour without rupture and mapped neural responses.

2026

Integrated review

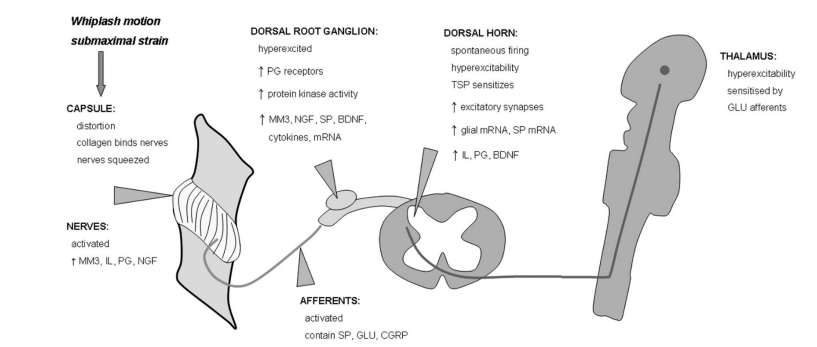
Forty-nine studies were assembled into a lesion-to-brain account of persistent post-whiplash joint pain.

The review by Bogduk links decades of diagnostic and therapeutic evidence with newer biomechanical and neurobiological findings.

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From capsule injury to central amplification

A brief local lesion may initiate a distributed response across the peripheral and central nervous systems.



CAPSULE	AFFERENTS	DRG	DORSAL HORN	THALAMUS
Subfailure collagen lesion	Terminal activation / injury	Neuronal stress + sensitization	Disinhibition + hyperexcitability	Amplified excitatory signaling

From Bogduk & MacVicar, 2026, Figure 3

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Five evidence streams converge on one mechanism

No single study proves the entire pathway; coherence emerges by linkage models.



01 Kinematics	02 Blocks	03 RF response	04 Subfailure	05 Neural response
Segmental S-curve and high local capsular strain	A painful joint can be localised clinically	Interrupting medial branches can abolish pain	Microscopic injury produces sustained pain behaviour	DRG, dorsal horn and thalamus become hyperexcitable

The synthesis is mechanistically persuasive—but living-human visualization of the microscopic lesion remains an unmet need.

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Facet joints are uniquely testable pain generators

Their defined afferent anatomy allows source-specific clinical experiments that are difficult elsewhere in the spine.

DEFINED INNERVATION

- Each joint is supplied by identifiable medial branches
- C2–3 is targeted via the third occipital nerve
- Afferent interruption is anatomically specific

CONTROLLED DIAGNOSIS

- Comparative local-anaesthetic blocks
- Placebo-controlled protocols reduce false positives
- About 60% in selected chronic post-whiplash cohorts

INTERVENTION TEST

- Thermal radiofrequency coagulation targets the same nerves
- Complete relief is possible in responders
- Recurrence with nerve recovery strengthens inference

Blocks identify the painful joint—not the microscopic lesion itself. Facet pain is common, not universal.

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But what explains the ongoing and persistent pain?

A nociceptive pain generator is activated by ongoing mechanical or chemical stimulation.

MECHANICAL INPUT

- Repeated loading or movement
- Ongoing tissue deformation
- Pain tracks the provoking stimulus

CHEMICAL INPUT

- Synovitis or inflammatory arthritis
- Persistent mediator release
- Continued nociceptor sensitization

THE MISMATCH

- Whiplash loading lasts milliseconds
- The capsule may remain grossly intact
- Pain can persist long after the stimulus ends

A transient event producing sustained pain requires a mechanism beyond continuing macroscopic joint deformation.

63

THE SUBFAILURE PARADOX

A millisecond mechanical insult can produce pain that outlives the tissue-loading event.

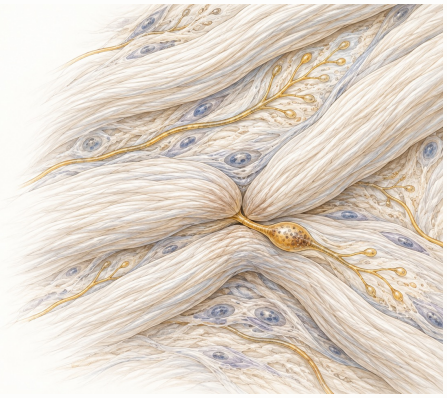
Animal models show sustained pain behavior, hyperalgesia and allodynia after subfailure capsule strain—features that are difficult to explain through a purely ongoing nociceptive joint model.

64

Microscopic nerve injury causing neuropathic pain?

Collagen can stretch, compress and pinch terminal nerve fibers embedded within the capsule.

- Capsular stretch alters collagen alignment
- Fibers squeeze or pinch terminal axons
- Focal axonal swelling has been observed with advanced MRI sequences
- Micro-neuroma-like spontaneous firing is a hypothesis



The proposed transition is from a connective-tissue lesion to focal axonal injury—and potentially spontaneous neural discharge.

65

Peripheral responses begin at the lesion

Capsular deformation can activate afferents, trigger local molecular signaling and sensitize primary sensory neurons.

CAPSULE

- Collagen fiber reorientation
- NGF and molecular inflammatory responses
- No macroscopic rupture required

TERMINAL AFFERENTS

- Group III and IV fibers respond to stretch
- Activity rises as strain increases
- pERK and substance P are increased

DORSAL ROOT GANGLION

- Neuronal stress markers
- BDNF / NGF and prostaglandin signalling
- Substance P, CGRP and glutamatergic involvement

The capsule is not merely passive collagen: its afferents and sensory neurons participate in a biologically active injury response.

66

Central responses can outlast the initiating event

Experimental subfailure changes excitability and synaptic organization beyond the injured joint.

DORSAL HORN

- Spontaneous firing and enhanced discharge
- More excitatory and fewer inhibitory synapses
- Astrocyte activation and inflammatory mediators

THALAMUS

- VPL neuronal hyperexcitability
- Increased excitatory amino acids
- Supported by only one study in the review

BEHAVIOUR

- Mechanical hyperalgesia
- Allodynia beyond the injured tissue
- Persistence reported to 7, 14 and 28 days

A local capsule injury can recruit a distributed pain system—but the strongest mechanistic evidence remains experimental.

67

Nociceptive, neuropathic and nociplastic

Subfailure broadens the mechanism beyond a simple category of ‘joint = nociceptive pain’.

NOCICEPTIVE INITIATION

Mechanical activation of joint afferents

Local molecular inflammatory response

Pain begins in injured capsular tissue

NEUROPATHIC COMPONENT

Terminal axons may be pinched or injured

Focal axonal swelling

Possible ectopic or spontaneous activity

NOCIPLASTIC-LIKE AMPLIFICATION

DRG and dorsal-horn sensitization

Reduced inhibition and increased excitation

Pain may extend beyond ongoing tissue input

This is a mechanistic synthesis—not a claim that every patient with chronic WAD has all three pain mechanisms.

68

Why might the pain system fail to reset?

The review suggests several mutually reinforcing mechanisms rather than one persistent macroscopic lesion.

01

Terminal firing

Microscopic axonal injury may generate continuing or spontaneous afferent activity.

02

Local signalling

NGF, substance P and other mediators can maintain peripheral sensitisation.

03

DRG amplification

Neuronal stress, glial activation and neurotrophic signalling raise sensory-neuron gain.

04

Central plasticity

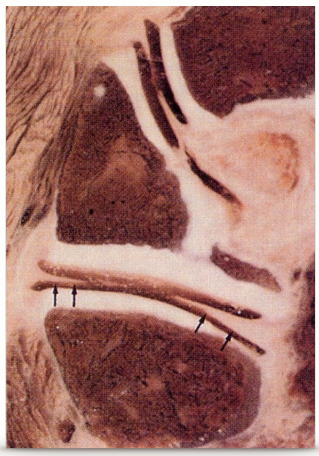
Dorsal-horn disinhibition, synaptogenesis and thalamic excitation may preserve amplification.

The unresolved question is whether one element maintains the system—or whether the cascade becomes self-sustaining.

69

2. Meniscoid (synovial fold) injuries

Meniscoid synovial folds suffer damage in whiplash, resulting in haemorrhage and pain





Capsular tearing and contusions of meniscoids account for up to 50% of chronic pain states, with the most commonly injured joints being C2-3 and C5-6

Webb, A. L., Collins, P., Rassoulian, H., & Mitchell, B. S. (2011). Synovial folds - a pain in the neck? Man Ther, 16(2), 118–124. doi:10.1016/j.math.2010.11.004
Farrell, S. F., Osmotherly, P. G., Cornwall, J., Sterling, M., & Rivett, D. A. (2017). Cervical spine meniscoids: an update on their morphological characteristics and potential clinical significance. European Spine Journal, 26(4), 939–947.

70

3. Ligament injuries following whiplash

Whiplash injury doesn't commonly cause 'ligamentous instability'

FACET CAPSULE

The ligament most consistently implicated experimentally is the zygapophyseal/facet joint capsule

This does not equate to 'instability'.

True instability requires substantial failure of the stabilising disco-ligamentous and/or osseous structures

Jaumard NV, Welch WC, Winkelstein BA. Spinal facet joint biomechanics and mechanotransduction in normal, injury and degenerative conditions. J Biomech Eng. 2011 Jul;133(7):071010.

ASSOCIATED ONLY WITH SEVERE TRAUMA

Disruption to ALL, PLL LF and interspinous ligaments require severe trauma.

Meta analysis has shown MRI signal abnormalities in the alar or transverse ligaments were not significantly associated with WAD and concluded that such MRI findings should not be used routinely to diagnose whiplash ligament injury

Li Q, Shen H, Li M. Magnetic resonance imaging signal changes of alar and transverse ligaments not correlated with whiplash-associated disorders: a meta-analysis of case-control studies. Eur Spine J. 2013 Jan;22(1):14-20.

71

4. Cervical disc injuries following whiplash

Similar collagen loading may occur in discs, but the investigation chain is less specific

FACET CAPSULE

- Defined medial-branch innervation
- Controlled diagnostic blocks
- Targeted radiofrequency intervention
- Experimental lesion-to-neural pathway

DISC / ANNULUS

- Extensively innervated
- Collagen fibers also experience complex strain
- Analogous subfailure lesions are plausible
- Innervation is less discretely targetable

Anatomical specificity is what makes the facet joint a powerful model for investigating microscopic post-whiplash pain generators.

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Journal of Pain Research

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PERSPECTIVES

Cervical disc degeneration and neck pain

This article was published in the following Dove Press journal:
Journal of Pain Research

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Abstract: Cervical intervertebral disc has long been considered a common source of neck pain. However, the pain caused by the disc itself has not been clearly defined so far, and its diagnosis and treatment has always been controversial. Degenerative cervical disc has a rich supply of nerve fibers, is prone to inflammatory reactions, and is susceptible to pain that can be provoked by disc stimulation or distention, and can be eliminated by block. Overwhelming clinical evidence demonstrates that neck pain in patients with degenerative cervical radiculopathy or myelopathy can be subsided rapidly by anterior cervical surgery, further indicating that this neck pain stems from the pathology of cervical disc itself. Cervical discography is advocated as the only test that connects disease to symptoms, but the procedure remains controversial. If

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Cervical discs account for a large proportion of non-traumatic chronic neck pain

"Discogenic neck pain alone without cervical disc herniation or cervical spondylosis accounts for a large proportion of chronic neck pain, and the reported prevalence is between 16% and 41%."

"This neck pain is usually accompanied by stiffness of the neck, headache, unilateral or bilateral shoulder pain, non-root arm pain, ocular and vestibular dysfunction, and pain in the anterior chest wall"

74

Cervical disc protrusion correlates with the severity of cervical disc degeneration

Proteoglycan fragmentation starts during childhood and increases with age.

These changes are enhanced by adverse genetic, biomechanical, and nutritional factors.

This largest cross-sectional study of cervical MR images in relatively healthy subjects showed that:

1. Mild cervical DD and protrusion started in the 20s
2. Cervical DD and protrusion progressed with age, especially from the 20s to the 50s, mainly at C5–C6 and C6–C7
3. The degree of cervical disc protrusion was significantly correlated with the severity of DD.

So why don't the guidelines suggest routinely image for cervical disc degeneration and pain in whiplash cases?

75

CLINICAL SYNTHESIS

A brief mechanical event can initiate a long-lived pain-system response without macroscopic rupture.

Subfailure capsule strain offers a biologically coherent bridge from segmental whiplash kinematics to a microscopic connective-tissue lesion, terminal nerve injury and distributed neural sensitization—while preserving clear boundaries between evidence, hypothesis and extrapolation.

76



5. MUSCLE RESPONSE TO INJURY

Rapid changes in muscle composition, their association with poor recovery and the imaging methods that may detect early changes in their physiology

77

THE CENTRAL OBSERVATION

The cervical multifidus can change within weeks in patients whose recovery trajectory is poor.

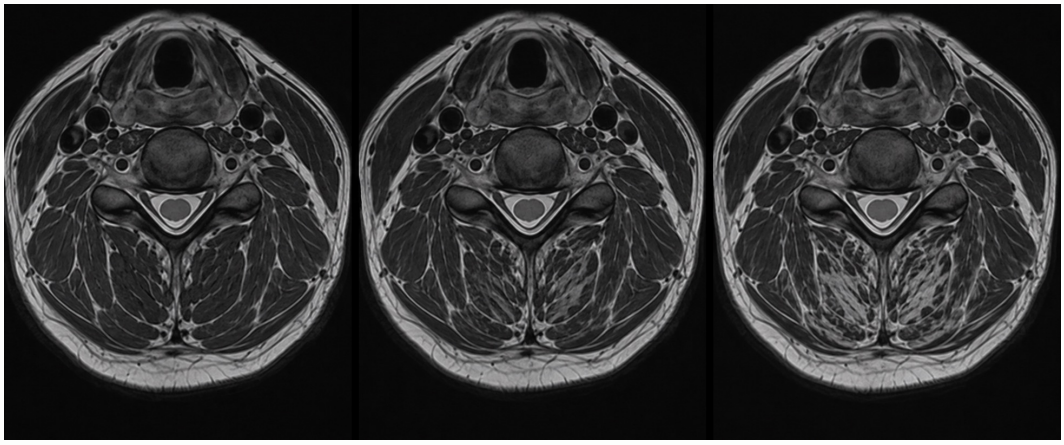
The longitudinal signal was selective. Participants who recovered or retained only mild disability showed little change, while the moderate to severe group developed rapidly increasing muscle fat fraction.

Elliott JM, Courtney DM, Rademaker A, Pinto D, Sterling MM, Parrish TB. The rapid and progressive degeneration of the cervical multifidus in whiplash: an MRI study of fatty infiltration. Spine. 2015;40(12):E694-E700

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Muscle fatty infiltration on quantitative MRI



LOW FAT FRACTION
Dark, relatively homogeneous multifidus

RISING FAT FRACTION
Increasing marbled high-signal tissue

HIGH FAT FRACTION
Marked bilateral compositional change

79

The 2015 prospective cohort

Serial MRI tracked the bilateral multifidus from C3 to C7 after acute WAD grade II.

36	28	8	C3-C7
Participants	**Recovered or mild**	**Moderate or severe**	**Imaging region**
Adults enrolled within one week of a motor vehicle collision and followed at one, two and twelve weeks.	Three-month NDI between 0% and 28%.	Three-month NDI of at least 30%.	Bilateral multifidus regions were traced on registered water-only and fat-only images.

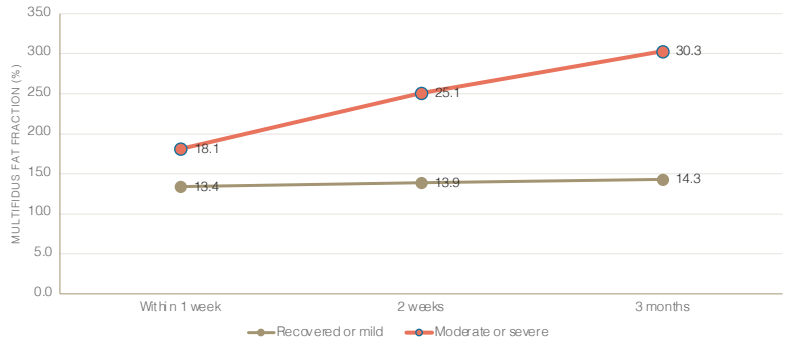
Outcome groups were defined at three months using the Neck Disability Index.

Elliott, J. M. et al. The Rapid and Progressive Degeneration of the Cervical Multifidus in Whiplash. Spine 40, E694–E700 (2015).

80

Multifidus fat fraction diverged by two weeks

The two groups had no statistically significant difference at enrolment, then followed markedly different trajectories.



The moderate to severe group rose from 18.1% to 25.1% between the first and second assessments. The recovered group changed by only 0.5 points.

Elliott et al, Spine, 2015

81

MUSCLE MARKER

“

The muscle response separated recovery trajectories between one and two weeks.

Rapid multifidus fatty infiltration is an early biological correlate of poor recovery, not yet an explanation or cause on its own.

82

6. Peripheral nerve injuries

- Although frank neurological signs are not a feature of WADII, studies show that many individuals exhibit subtle signs of peripheral nerve involvement – often assumed to be somatic referred pain
- There is evidence for peripheral neuroinflammation in grade II presentations
- 2/3 of individuals with WADII are considered to have neuropathic pain based upon features such as hyperalgesia and hypoesthesia being reported in upper limb dermatomes, as well as nerve mechanosensitivity on nerve tension tests

Ridehalgh, C. et al. Temporal development of peripheral neuroinflammation in whiplash-associated disorder grade II and its role in chronicity. PAIN 167, 356–371 (2026).

83

7. Spinal cord injuries

Routine WAD I–II → spinal cord injury is very unlikely

Major trauma with fracture/dislocation/disco-ligamentous instability → direct cord deformation becomes a serious concern.

Hyperextension + pre-existing cervical stenosis → important exception: significant cord injury/central cord syndrome can occur without fracture or obvious instability.

Divi SN, Schroeder GD, Mangan JJ, Tadley M, Ramey WL, Badhiwala JH, Fehlings MG, Oner FC, Kandziora F, Benneker LM, Vialle EN, Rajasekaran S, Chapman JR, Vaccaro AR. Management of Acute Traumatic Central Cord Syndrome: A Narrative Review. Global Spine J. 2019 May;9(1 Suppl):89S-97S.

84

8. BRAIN INJURY IN WHIPLASH



Brain loading, the neurometabolic response and the clinical findings that increase the likelihood of concussion symptoms overlapping with the whiplash presentation

85

CONCUSSION AS mTBI

A mechanical force produces an acute disturbance of brain function.

Rapid head motion can disturb neuronal membranes, axons, cerebral blood flow and network function without creating a lesion visible on routine imaging. Concussion and whiplash can occur together during the same event.

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The acute neurometabolic cascade

Mechanical deformation initiates a secondary physiological response that can outlast the obvious symptoms.

MEMBRANE STRAIN
Transient permeability and disturbed ionic gradients

EXCITATORY RESPONSE
Glutamate release and rapid depolarisation

ENERGY DEMAND
Ion pumps consume ATP to restore homeostasis

DELIVERY MISMATCH
Mitochondrial and blood flow responses may lag demand



The cascade explains neurophysiological vulnerability after concussion.

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[RESEARCH REPORT]

BENJAMIN S. ELKIN, PhD[1] • **JAMES M. ELLIOTT,** PT, PhD[2] • **GUNTER P. SIEGMUND,** PhD[3,4]

Whiplash Injury or Concussion? A Possible Biomechanical Explanation for Concussion Symptoms in Some Individuals Following a Rear-End Collision

The clinical presentations of whiplash injury and concussion have considerable overlap,[29] with both diagnoses generally based on presenting signs and symptoms, and a history of trauma. The earliest clinical reports of whiplash injury (called "railway spine" in the 19th century) implicated brain injury as a possible explanation for the type and severity of the initial and lasting symptoms described by individuals who lacked external signs of injury.[11] Since then, many others have noted similarities in the initial presentation, chronic symptoms, and even somatosensory evoked potentials following whiplash injury and mild brain injury.[1,3,12,15,18,62,63]

A common diagnostic theme of both conditions is the paucity of evidence of salient lesions in the cervical spine and brain using available imaging tech-

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mTBI is often missed in the ED after MVC

Pain Physician 2023; 26:101-110 • ISSN 1533-3159

Retrospective Study

Missed Emergency Department Diagnosis of Mild Traumatic Brain Injury in Patients with Chronic Pain After Motor Vehicle Collision

Cayden Peixoto, MSc[1], Derrick Matthew Buchanan, PhD[2], and Richard Nahas, MD[3]

"In particular, those presenting with loss of consciousness, an altered mental state, posttraumatic amnesia, or postinjury headache are at increased risk of post-concussion syndrome"

89

Shared symptoms across WAD and mTBI

The cervical spine, vestibular system, visual system and brain can generate overlapping post-traumatic symptoms.

HEADACHE
Cervicogenic, migraine-like or mixed

DIZZINESS
Cervical, peripheral vestibular, central or autonomic

VISUAL SYMPTOMS
Oculomotor, vestibulo-ocular or cervico-ocular

FATIGUE AND FOG
Brain physiology, pain, sleep, stress or medication



Timing of symptoms can be the most important clue. Often chronic issues in WAD.

90

Clinical signs that strongly increase mTBI likelihood

Acute observable signs carry more diagnostic weight than headache, dizziness or fatigue later.

AMS

Altered mental status

Dazed, confused, disoriented, unusually slow or unable to follow instructions.

PTA

Post-traumatic amnesia

A gap in memory for events after the injury or reliable retrograde amnesia.

LOC

Loss of consciousness

Any brief loss of consciousness linked to the event increases diagnostic probability.

NEURO

Acute neurological sign

Transient neurological dysfunction documented at or soon after the event.

New cognitive symptoms in chronic WAD could be predicted by nausea / vomiting in the acute period

** Samusyte G, Ciceliene J, Pajediene E, Stasaitis K, Petrikonis K, Bileviciute-Ljungar I. Is Chronic Whiplash-Associated Disorder Associated with Central Nervous System Impairments? A Controlled Observational Study in a Lithuanian Cohort. J Clin Med. 2025 Sep 3;14(17):6222.

91

Weak discriminators in a patient with WAD

These symptoms deserve assessment but do not establish a concomitant brain injury.

HEADACHE

Common to both

Cervical nociception and post-traumatic migraine phenotypes frequently overlap.

DIZZINESS

Multiple pathways

Cervical, vestibular, visual and autonomic mechanisms can produce similar complaints.

FATIGUE

Low specificity

Pain, sleep disruption, medication and stress can closely mimic post-concussion fatigue.

LATE FOG

Context matters

Concentration difficulty emerging later has weaker diagnostic value than an acute cognitive change.

Their diagnostic meaning depends on timing, pattern, provoking factors, examination findings and plausible alternatives.

92

Clinical reasoning spectrum

The question is whether brain involvement adds explanatory value beyond the cervical and vestibular findings.

WAD PREDOMINANT

- No acute alteration of brain function
- Symptoms closely track neck movement or cervical load
- Cervical and headache examination reproduces the main presentation

MIXED POSSIBILITY

- Event history incomplete
- Cervical findings coexist with cognitive or vestibulo-ocular abnormalities
- Several systems reproduce different parts of the presentation

mTBI MORE LIKELY

- Acute AMS, PTA, LOC or witnessed neurological sign
- Objective abnormalities across CNS domains
- Exertional or cognitive intolerance not adequately explained by the neck

A mixed diagnosis is often more accurate than forcing the presentation into a neck-only or brain-only category.

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MODULE 4

FROM ACUTE INJURY TO CHRONIC WAD

How tissue injury, neural plasticity, stress physiology and personal context interact over time

94

MODULE 4

Let's consider:

1. How an acute injury becomes a chronic pain syndrome
2. Chronic pain as a learned protective motor output behaviour
3. The three major pain mechanisms and their features
4. Primary sequelae of non-recovery
5. Secondary sequelae and altered life trajectory
6. The evolution from QTF grade to integrated neuroscience model

95

How does acute injury become chronic pain?

Chronic WAD develops through interacting processes rather than a single linear lesion pathway.



01 Lesion	02 Immune response	03 Neural gain	04 Meaning	05 Behaviour
Rapid loading strains local tissues and neural endings	Inflammatory mediators alter receptor thresholds	DRG and dorsal horn responses amplify signaling	Threat, expectations and context shape salience	Protection, withdrawal or graded recovery alter future input

Each stage can settle, persist or be modified. The sequence describes possibilities, not inevitability.

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Central sensitization

Combined with threat value, the pain experience is now de-coupled from nociceptive activation

PERIPHERY

Sensitized or injured terminals increase incoming signal

DORSAL ROOT

Ganglion and axonal responses alter excitability

DORSAL HORN

Wind-up, disinhibition and receptive-field expansion

SUPRASPINAL

Attention, salience and descending control shape the output



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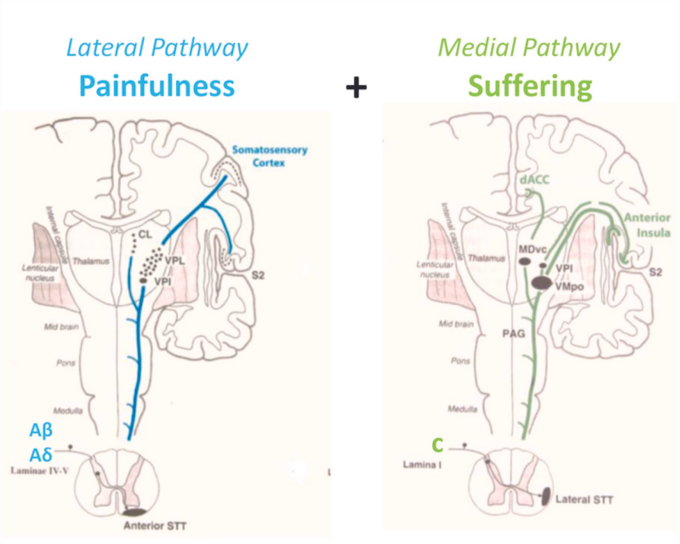
Lateral and medial pain pathways in the brain

- Supports location, intensity and sensory discrimination
- The VPL relays somatosensory information to somatosensory cortex

Lateral Pathway
Painfulness

+

Medial Pathway
Suffering



- Brainstem, medial thalamic and limbic connections
- Supports meaning, salience, threat, affect and suffering
- Context changes the value assigned to the signal

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99

Protective motor output

Motor control changes can reduce immediate threat while increasing effort, stiffness and sensory uncertainty over time.

EARLY STRATEGY

- Co-contraction and reduced movement
- Fewer movement options
- Visual fixation and reduced head motion

PERSISTENT COST

- Higher energy demand and fatigue
- Reduced variability and confidence
- Deconditioning and altered muscle composition

ALTERED CENTRAL REPRESENTATION

- Maladaptive brain plasticity
- Decreased brain volume
- Altered morphology of cortical representations of body parts
- Sensorimotor incongruence

What do we mean by altered central representation?

100

Cortical models and prediction

The nervous system continually updates internal models using sensory evidence, prior experience and the cost of being wrong.

PRIOR

Existing model

Previous injury, health beliefs and learned expectations establish a starting estimate.

INPUT

Current evidence

Symptoms, examination findings, movement and the social environment provide new information.

ERROR

Mismatch

Unexpected input drives learning, vigilance or revision of the internal model.

UPDATE

New prediction

Safe experience can recalibrate the model when the evidence is credible and repeated.

Every consultation supplies new evidence. The clinician can either reduce uncertainty or reinforce a danger model.

101

Sensorimotor incongruence

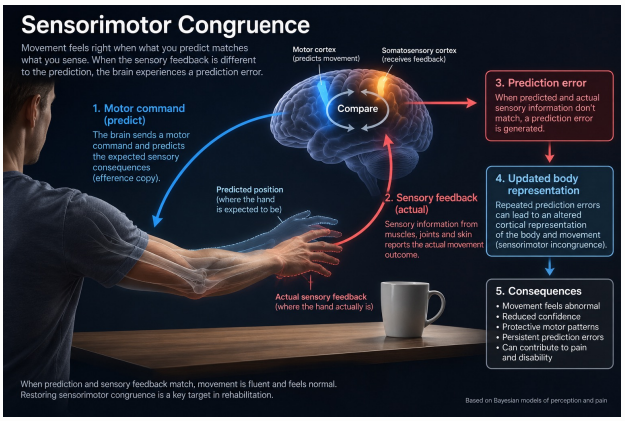
When predicted movement and incoming cervical, vestibular or visual information disagree.

PREDICTION
The brain estimates the sensory consequences of intended movement

INPUT
Cervical, visual and vestibular signals report what occurred

ERROR
Persistent mismatch increases uncertainty and corrective effort

OUTPUT
Dizziness, guarded movement, pain and reduced confidence can follow



Movement feels ‘safe’ and ‘right’ when what you predict matches what you sense

102

Vestibular and cerebellar adaptation

Proprioceptive 'poverty' in the neck impacts the control of gaze, balance and orientation

INPUT MISMATCH

- Altered cervical afference can conflict with vestibular and visual cues
- Pain and guarding change head movement experience

REFLEX ADAPTATION

- VOR, cervico-ocular and optokinetic systems interact
- Cerebellar calibration depends on repeated error signals

CLINICAL EXPRESSION

- Dizziness, unsteadiness and visual motion sensitivity
- Head-eye coordination and postural deficits
- Findings require differential diagnosis

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Neuroscience expanded our explanatory models

The field progressively incorporated more of the person and the context of trauma.



Pain linked mainly to tissue lesion

Clinical signs classified and active recovery promoted

Threat beliefs and avoidance linked to persistence

Pre-existing vulnerability or protection alters response

Tissue, CNS, stress, behaviour and context interact

Each model added explanatory capacity while preserving the need for clinical assessment.

104

Integrated model of WAD

Walton DM, Elliott JM. An integrated model of chronic whiplash-associated disorder. J Orthop Sports Phys Ther. 2017;47(7):462-471.

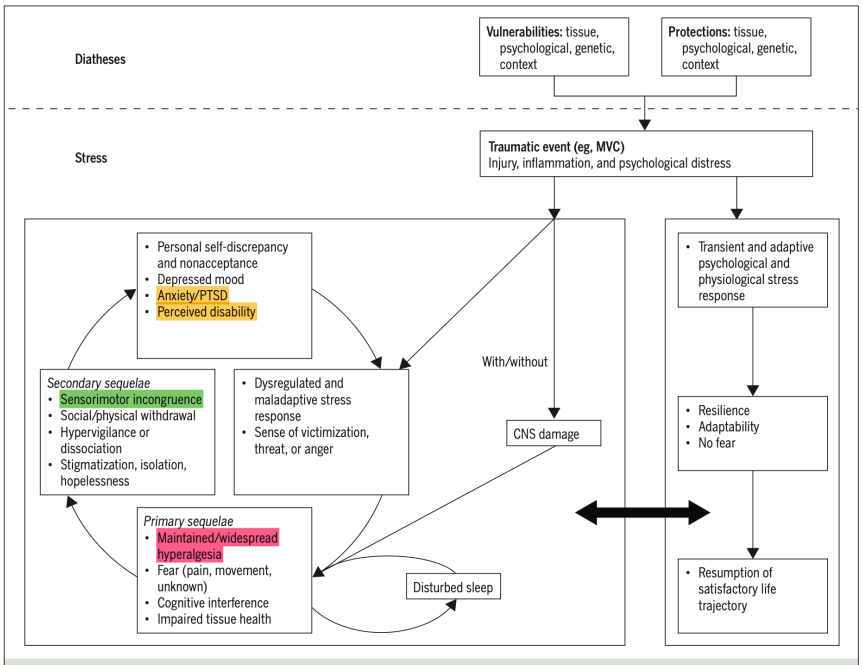
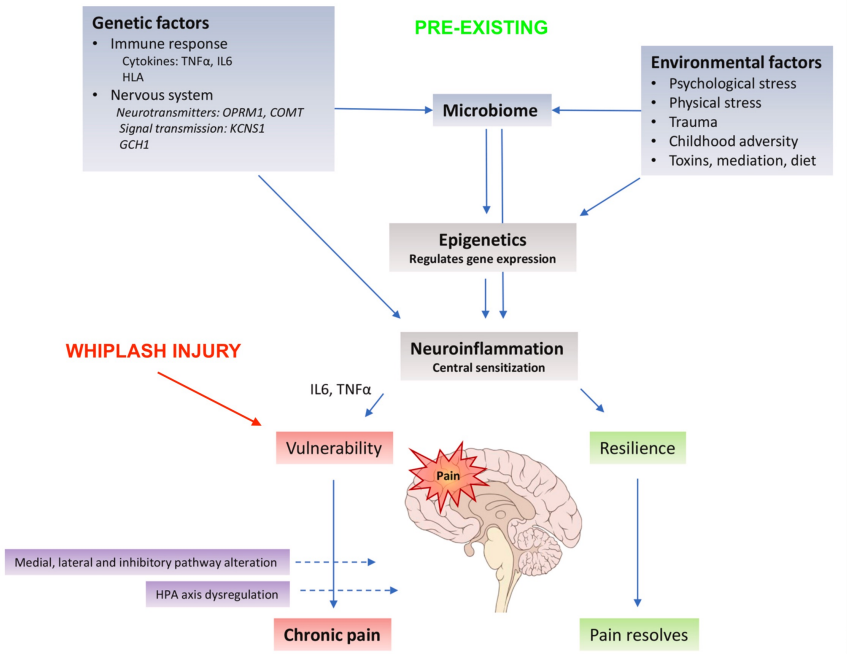


FIGURE. An integrated biopsychosocial model describing potential mechanisms for the onset and maintenance of persistent pain and disability following acute trauma. Abbreviations: CNS, central nervous system; MVC, motor vehicle collision; PTSD, posttraumatic stress disorder.

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Compare WAD to established chronic pain models



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MODULE 5

HISTORY, TRIAGE AND DIFFERENTIAL DIAGNOSIS

The history reconstructs the event, identifies urgent pathways and determines which examination domains deserve priority.

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MODULE 5

Let's consider the key points for:

1. The key objectives of the initial whiplash history
2. Classifying the WAD grade by QTF definition
3. Quantifying pain intensity and self-reported disability
4. Eliciting recovery expectations, beliefs and concerns
5. Identifying symptom and functional domains

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Key objectives of the initial whiplash history

What information should be prioritised in the history to support clinical triage, establish injury grade, stratify recovery risk, and direct a targeted examination?

1. CLASSIFY	2. QUANTIFY	3. QUANTIFY	4. ELICIT	5. IDENTIFY
WAD grade as indication of injury severity	Pain intensity using the VAS / NRS	Self-reported disability using the NDI	Recovery expectations, beliefs and concerns	Symptom and functional domains

Note the importance of self reported pain and disability. Research indicates they are better predictors of prognosis than the WAD grade

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Classifying the WAD grade by QTF definition

Tune your preferred history taking process based on the information prioritised by QTF grades

- Incorporate the criteria for the Canadian C-Spine rule into the initial history
- Establish collision characteristics and the immediate post-collision state relevant to cervical spine injury risk.
- Rule: dangerous mechanism versus simple rear-end MVC, whether the patient was ambulatory at any time, whether they were sitting in the ED/waiting area, delayed versus immediate onset of neck pain



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Clinical triage: from most serious back to most common

After WAD grade IV (clinically urgent cervical injury) is excluded, grade III is considered

- Characterise the pain — location, quality, behaviour, temporal pattern, aggravating and relieving factors.
- Paraesthesia and midline cervical tenderness. These are specifically high-risk elements in the validated CCR (reassuring when absent)
- By this stage, you have already begun excluding the likelihood of WAD grades 4 and 3
- The physical examination is then used to further differentiate WAD grades 1 and 2

111

Quantifying pain intensity and self-reported disability

The VAS and NDI convert a patient-reported construct into a numerical score that can be compared across time

Visual Analogue Scale
(actual analogue version)



https://www.mdcalc.com/calc/10532/neck-disability-index-ndi

Neck Disability Index
(Based upon Oswestry for low back)

I have no pain at the moment	0
The pain is very mild at the moment	+1
The pain is moderate at the moment	+2
The pain is fairly severe at the moment	+3
The pain is very severe at the moment	+4
The pain is the worst imaginable at the moment	+5

112

Eliciting recovery expectations, beliefs and concerns

There is a strong correlation between expectation of recovery and injury severity perception

"Do you think you are going to get better soon?"

"How bad to you think your neck is injured?"

"What do you think is causing your pain?"

"What makes you think that this may not get better?"

"Do you think there is anything that will prevent you returning to normal?"

Ferrari, R., D. Louw, Correlation between expectations of recovery and injury severity perception in whiplash-associated disorders. Journal of Zhejiang University Science B. 2011. 12(8): p. 683-686

113

Screen for a faulty model of the problem

Targeted questions reveal the beliefs that organize attention, movement and care-seeking behavior.

DAMAGE

- What do you believe happened physically?
- Which findings or explanations shaped that belief?
- What remains uncertain or frightening?

DANGER

- Which movements or symptoms signal harm?
- What do you predict will happen if you test them?
- What would make activity feel safe?

RECOVERY

- What does recovery mean to you?
- Which roles matter most?
- What obstacles, insurance or legal concerns affect the pathway?

Document beliefs neutrally. Compensation and legal context may influence stress and care pathways without explaining symptoms by themselves.

114

Screen for other vulnerabilities to chronic pain

A complete history establishes the individual patient's baseline rather than treating every post-event feature as new and working off statistical averages.

RESILIENCE	CAPACITY	STRESS	SUPPORT
Physical baseline	**Functional baseline**	**Psychological baseline**	**Recovery resources**
Migraine phenotype, previous concussion, vestibular disorder, neurological disease, advanced degeneration, sleep health, hypermobility	Work demands, physical conditioning, caregiving responsibilities and valued activities.	Previous trauma, anxiety, depression, stress sensitivity and current life burden.	Social support, workplace flexibility, access to care, health literacy and confidence in recovery.

Baseline information should contextualize prognosis without invalidating a genuine change after trauma.

115

Identifying symptom and functional domains

Characterise the broader effects of the injury and direct a targeted, domain-matched examination.

Does the patient report symptoms indicating:

- Sensorimotor function
- Pain sensitivity
- Additional psychological factors
- Additional symptoms such as jaw pain, lower back pain, coccyx pain etc
- mTBI / concussion – cognitive, memory, nausea

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CLINICAL SYNTHESIS

The history should change what you examine, what you defer and what you explain.

Mechanism, symptom timing, baseline vulnerability, urgent exclusions and the patient's model together define a defensible examination plan.

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MODULE 6

THE ROLE OF IMAGING

Decision rules identify who requires acute imaging. Imaging then answers a focused anatomical question rather than substituting for clinical reasoning.

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MODULE 6

Let's consider:

1. Clinical decision rules for imaging the neck post-trauma
2. The role of imaging in uncomplicated WAD I-II
3. The role of imaging in WAD III
4. The role of advanced imaging technology in WAD
5. The role of advanced medical tests in WAD

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Clinical Decision rules for acute cervical imaging

Canadian C-spine and NEXUS rules support clearance in defined blunt-trauma populations.

POPULATION

- Alert, stable patients after acute blunt trauma
- Use only within the rule's inclusion and exclusion criteria
- Do not apply casually to delayed or complex presentations

PURPOSE

- Identify clinically important cervical injury
- Safely reduce unnecessary imaging
- They do not grade WAD or diagnose soft-tissue lesions

RESULT

- A negative rule can support no imaging
- A positive rule means imaging is indicated
- The rule does not select every subsequent modality

A decision rule supports, rather than replaces, clinical judgement and local trauma pathways.

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The NEXUS criteria were introduced in 1992

Imaging can be delayed only when all five low-risk criteria are satisfied.

MIDLINE	NEURO	ALERT	CONTEXT
No tenderness	**No deficit**	**Normal alertness**	**No confounder**
No posterior midline cervical tenderness.	No focal neurological deficit on examination.	Normal level of alertness and reliable participation.	No intoxication and no painful distracting injury.

If any criterion fails, imaging is indicated according to the acute trauma pathway.

Hoffman JR, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. N Engl J Med. 2000;343:94-99.

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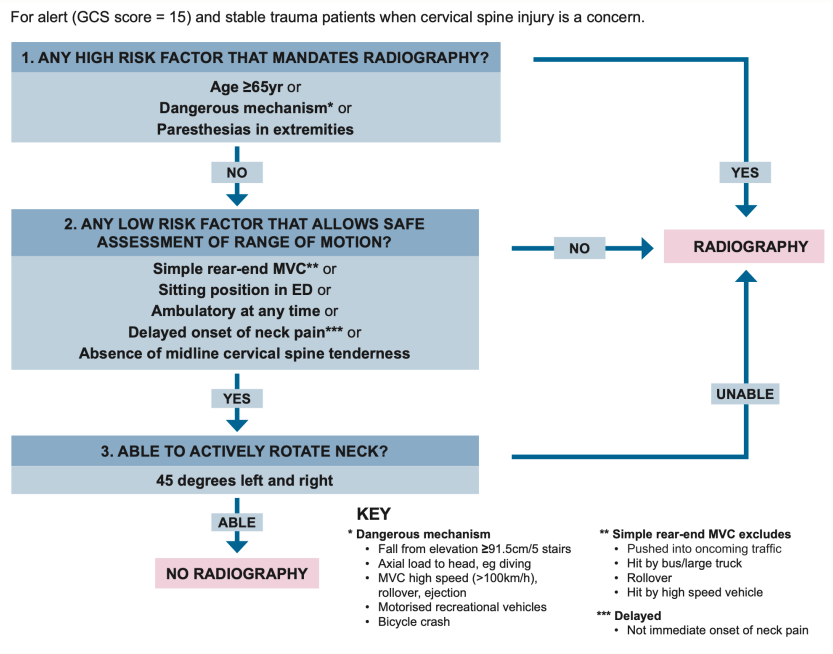
The Canadian C-Spine Rule

Sensitivity: 99.4%

Specificity: 42.5%

Following this rule assures that a fracture will not be missed (95% CI 98–100%)

Better accuracy than NEXUS criteria



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The role of imaging in uncomplicated WAD

DIAGNOSTIC IMAGING	
P2. Recommendation	Changed
Level of evidence	A
Reassure patients that changes (including degenerative or other minor pathological changes) on X-ray, MRI and CT are NOT associated with ongoing pain and disability following WAD.	

2007 Guidelines

There is strong evidence that degenerative changes on X-ray are not associated with ongoing pain symptoms following WAD.

New evidence since 2007 Guidelines

Five cohort studies (level II)[20-24] and a systematic review (level I)[14] assessed the association of radiological changes with prognosis of pain and disability following WAD. None of the studies reported an association between changes on diagnostic imaging and ongoing pain and disability.

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The role of imaging in WAD III

Specialised imaging may be appropriate in selected WAD III presentations

WAD III may pose a specific clinical question that requires and imaging answer and may change management

Imaging becomes indicated when a specific neurological lesion is suspected—for example nerve-root compression or spinal cord injury. Imaging is therefore hypothesis-driven rather than routine.

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The role of advanced imaging technology in WAD

A normal conventional scan does not exclude an evolving change in muscle composition or cellular physiology.

CONVENTIONAL MRI

- Looks for macroscopic structural lesions
- Common sequences often remain unrevealing in WAD
- Resolution and sequence selection constrain sensitivity

WATER-FAT MRI

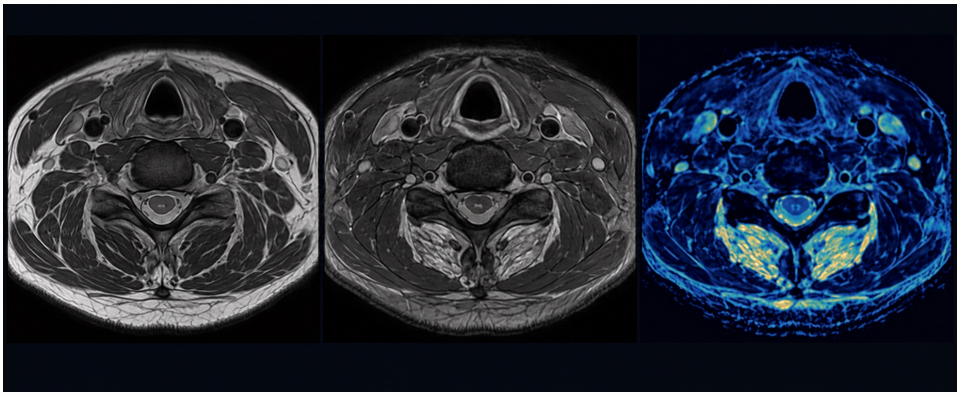
- Separates water-only and fat-only signal
- Quantifies proton-density fat fraction
- Detected group divergence by two weeks

PHYSIOLOGICAL METHODS

- Diffusion MRI measures water mobility
- Spectroscopy estimates metabolites
- Changes may precede visible fat deposition

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Quantitative MRI examines different layers of muscle biology



ANATOMICAL MRI
Macroscopic structure

DIXON FAT-ONLY
Tissue composition

DIFFUSION / ADC
Water mobility and early physiology

These methods remain research tools for WAD risk stratification and mechanism discovery.

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The advanced imaging toolkit

Each modality targets a different biological level and carries a different evidence boundary.

DIXON

Fat-water separation

Quantifies muscle fat fraction and provides the strongest direct longitudinal WAD evidence in these papers.

DW-MRI

Water diffusion

ADC may detect early denervation or physiological change before conventional imaging or electrophysiology.

MRS

Tissue metabolites

Could examine lipid peaks, energy metabolism and cellular biochemistry before visible fatty infiltration.

MT / fMRI

Neural context

May characterise spinal-cord pathways and functional pain processing alongside the muscle response.

These studies may contribute to understanding WAD mechanisms, but this does not yet make them useful routine diagnostic tests.

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The role of advanced medical tests in WAD

Biological differences do not equate to clinically meaningful tests

Research has examined stress hormones, inflammatory biomarkers, cerebral blood flow, skin/nerve-fibre measures and genetic markers. Some studies report group differences, but the guidelines do not support routine use because findings are limited/heterogeneous and generally do not establish diagnosis or treatment direction.

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Clinical decision-making summary

Clinical question	Investigation principle	Key message
Could there be fracture/dislocation?	Apply Canadian C-Spine Rule → radiography if indicated.	Image to exclude clinically important structural injury.
Is there neurological involvement?	WAD III + specific suspicion may justify targeted CT/MRI or specialist investigation.	Escalate only when the neurological presentation creates a specific diagnostic question.
Uncomplicated WAD I–II?	No routine CT/MRI.	More imaging is not better assessment.
Can advanced imaging explain persistent WAD?	Research findings exist, but routine morphological/advanced imaging is not recommended.	Research association is not diagnostic utility.
Should biomarkers/advanced medical tests be ordered?	Not routinely recommended.	Biological group differences do not automatically produce a clinically actionable test.
Should any test be ordered?	Ask whether it will change classification, referral or management.	Investigation should be clinically consequential.

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CLINICAL SYNTHESIS

“

Imaging is strongest when it answers a specific question that will change management.

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MODULE 7

CLINICAL EXAMINATION

A history-guided, domain-matched sequence that tests safety, localisation, mechanism and function

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MODULE 7

Let's consider:

1. What is the examination trying to accomplish?
2. The core focussed examination
3. The cervical ROM and musculoskeletal examination
4. Domain matched examination that is history-guided
5. Sensorimotor examination
6. Pain sensitivity examination
7. Differential exam for other associated domains
8. What not to do

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Recall our clinical & assessment domains of WAD

1. Structural & tissue injury
2. Neurological injury / function
3. Vascular safety screening
4. Physical musculoskeletal impairment
5. Sensorimotor function
6. Pain sensitivity
7. Psychological and contextual recovery
8. mTBI overlap & additional symptoms

Assessment domain	Subcategories
Physical Musculoskeletal Impairment	Trigger points, Cervical Range of Motion (ROM) Muscle performance/postural changes Muscle function
Sensorimotor	Cervical joint position error Cervical movement sense Oculomotor disturbance Balance Coordination Test Others – Proprioception
Pain sensitivity	Pressure hyperalgesia Thermal hyperalgesia Dynamic pain sensitivity testing Quantitative Sensory Testing (QST) (vibration) Brachial Plexus Provocation Test (BPPT) Nociceptive Flexion Reflex (NFR) Others
Additional Psychological Factors	Fear avoidance Self-efficacy Mental disorder Psychological distress Cognitive deficits
Additional Symptoms	Additional symptoms Sleep disturbance Jaw symptoms Disability

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What is the examination trying to accomplish?

The history identified the domains, the examination interrogates them

A good WAD examination is not a fixed battery of orthopaedic tests. It begins with a focused core examination that establishes safety, neurological status, WAD grade and key musculoskeletal impairment, then expands selectively into domain-matched testing according to the history, recovery risk and clinical presentation.

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For fellows (specialists):

For specialists (fellows), the higher-level skill is knowing which additional sensorimotor, oculomotor, proprioceptive and pain-sensitivity tests are indicated, how to perform them, and how the result changes treatment or referral.

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The focussed core examination

Your examination should be an organised and methodical progression

(i) Begin with safety and general observation

Before provocative testing, observe the patient's general presentation, head/neck posture, movement behaviour and signs that may suggest a need to modify or stop the examination. Reconsider red flags, associated injuries and general medical status where appropriate.

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(ii) Establish neurological status

Perform a focused neurological examination appropriate to the presentation.

Includes vascular screen (HINTS valuable for acute vertigo or dizziness)

Appropriate cranial nerve screen

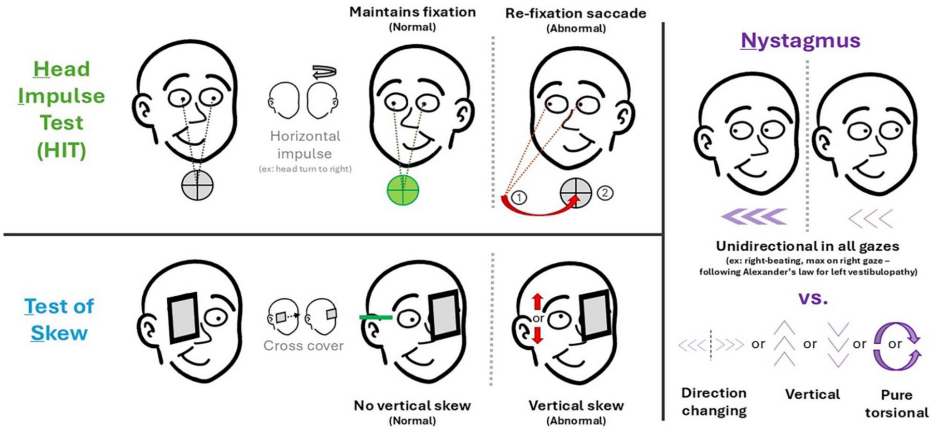
Assess relevant upper-limb myotomal power, sensation and reflexes and recognize features suggesting nerve-root or spinal cord involvement.

Neurological signs are central to distinguishing WAD II from WAD III and may trigger targeted imaging or medical referral.

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Hints to the H.I.N.T.S. Exam for Acute Vestibular Syndrome

(AVS = sudden-onset constant vertigo with nausea/vomiting, gait unsteadiness, and nystagmus)



Localization	Saccade on HIT*	Nystagmus	Skew deviation*
Peripheral (need all 3)	Present	Unidirectional	Absent
Central (need any 1)	Absent	Any other nystagmus	Present

*Perform maneuver on both sides

From: Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. Acad Emerg Med. 2013;20(10):986-996.

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Cervical ROM and M/S examination

Classification and baseline impairment

(iii) Assess cervical movement and M/S impairment

- Assess active cervical ROM in flexion, extension, rotation and lateral flexion, together with clinically relevant tenderness/palpation and movement behaviour.
- Cervical ROM is a core examination measure: it contributes to WAD grading, has prognostic relevance and can provide a baseline for reassessment.

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(iv) Assess muscle function when clinically indicated

- Muscle function/performance is not a mandatory test battery in every acute case.
- Consider endurance, motor control and performance testing when the history or observed function suggests impairment, or when the finding will direct rehabilitation.
- Examples include low-load cervical motor-control/endurance assessments such as the cranio-cervical flexion test.

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Domain matched examination: history guided

After core examination, the history guides the next examination domain

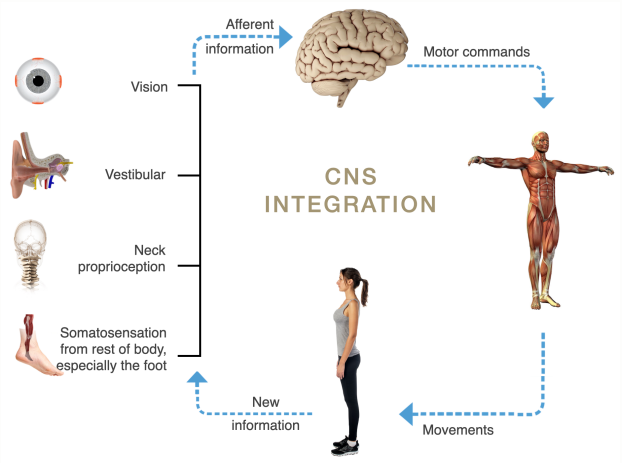
Ask yourself…

- Which symptom/functional domain was identified in the history
- What objective assessment would meaningfully interrogate it.
- This is the transition from a standard examination to a domain-matched examination.

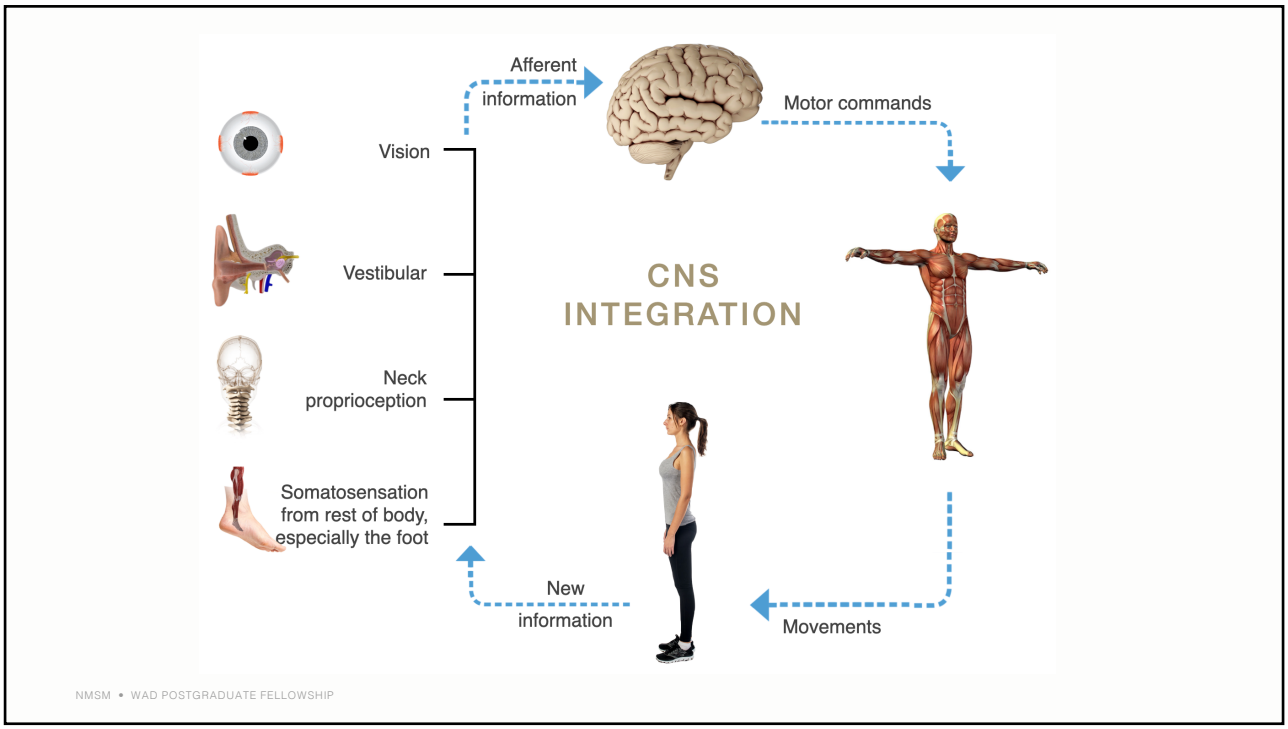
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Sensorimotor domain

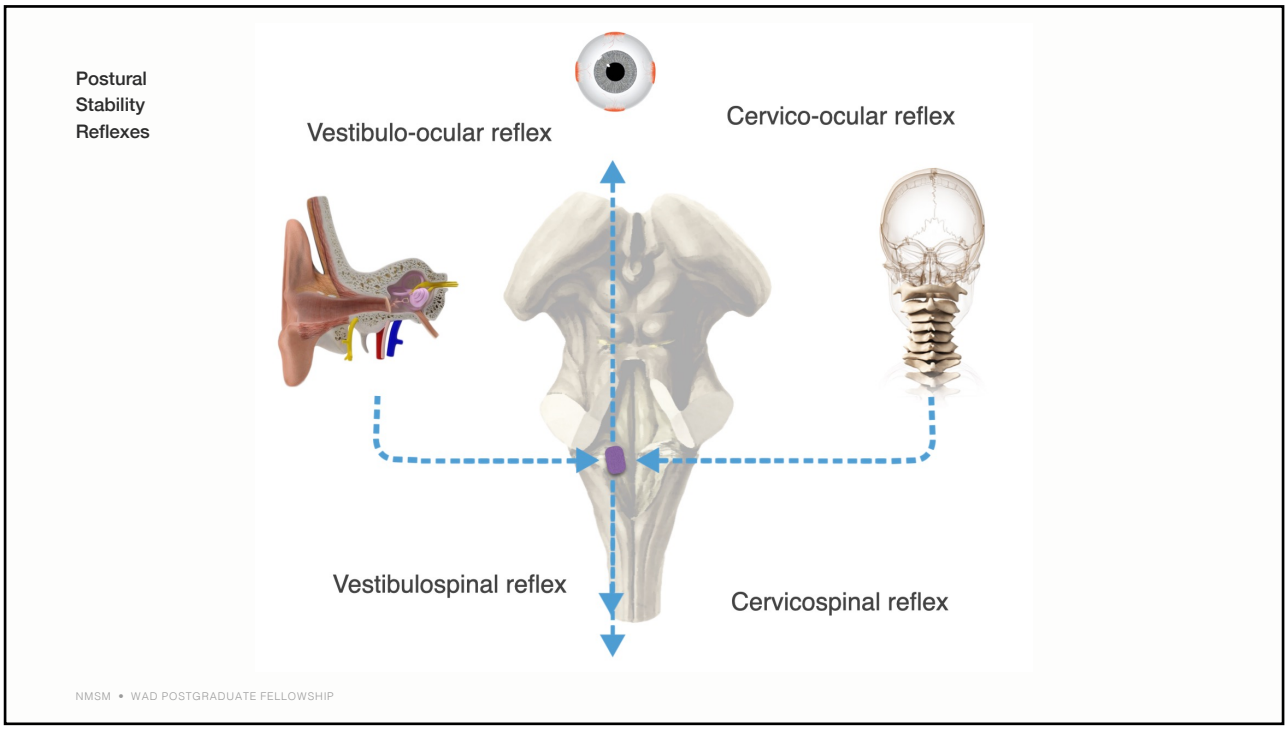
Requires a fundamental understanding of the reflex processes involved in sensorimotor control



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Afferent information
Motor commands
Vision
Vestibular
CNS INTEGRATION
Neck proprioception
Somatosensation from rest of body, especially the foot
New information
Movements
NMSM • WAD POSTGRADUATE FELLOWSHIP

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Postural Stability Reflexes
Vestibulo-ocular reflex
Cervico-ocular reflex
Vestibulospinal reflex
Cervicospinal reflex
NMSM • WAD POSTGRADUATE FELLOWSHIP

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Sensorimotor and oculomotor exam

Test symptomatic and or medium to high-risk acute cases

GAZE	VESTIBULAR	CERVICAL	POSTURE
Oculomotor	**Vestibulo-ocular**	**Sensorimotor**	**Balance**
Saccades, pursuit, convergence and visual motion tolerance, interpreted with vision and mTBI context.	Head impulse, dynamic visual acuity, positional testing and motion sensitivity when indicated.	Joint-position error, movement sense, head-eye coordination and neck torsion comparisons.	Static and dynamic tasks, altered visual or surface conditions, gait and dual-task challenge.

Becomes more relevant in chronic WAD (fellow-level specialist capability)

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Pain sensitivity examination

Most useful when the history or risk profile suggests altered pain processing

- History clues: widespread pain, disproportionate responses, pain with non-noxious stimuli, marked cold sensitivity or medium/high risk of poor recovery.
- The 2024 assessment guideline supports thermal hyperalgesia, pressure hyperalgesia, dynamic pain sensitivity/CPM and brachial plexus provocation testing in selected acute WAD presentations.

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How to assess for pain sensitivity in the clinic: neck and arm pain focus
Prepared by Trudy Rebbeck, Niamh Moloney and Darren Beales.

ABSTRACT: How to assess for pain sensitisation in the clinic: neck and arm pain focus

Rebbeck T [1], Moloney N[2], Beales D[3]

[1]Discipline of Physiotherapy, Faculty of Health Sciences, University of Sydney

[2]Dept. Health Professions, Macquarie University

[3] School of Physiotherapy and Exercise Science, Curtin University

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Practical clinical pain sensitivity tests (1-3)

Allodynia / light touch: Assess painful or exaggerated response to normally non-painful tactile stimulation. Compare a remote/baseline area before the symptomatic region.

Sharp hyperalgesia: Assess exaggerated pain response to a sharp stimulus, again using comparison with a remote/baseline region.

Pressure pain threshold (PPT): Use an algometer; repeat measures and average them. Local plus remote-site testing can help characterize the distribution of pressure hyperalgesia. Lower PPTs have shown prognostic associations in WAD.

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Practical clinical pain sensitivity tests (4-6)

- Cold hyperalgesia / ice-pain test: A practical clinical proxy for thermal pain sensitivity. Apply ice for the specified brief interval and obtain an NRS pain rating. >5/10 indicates a high likelihood of cold hyperalgesia on quantitative cold-pain testing.
- Conditioned pain modulation (CPM): Assesses endogenous pain modulation using a test stimulus before and after a conditioning noxious stimulus.
- Neural mechanosensitivity: Combine clinical history with neurodynamic testing (BPTT) and applicable nerve-trunk palpation.

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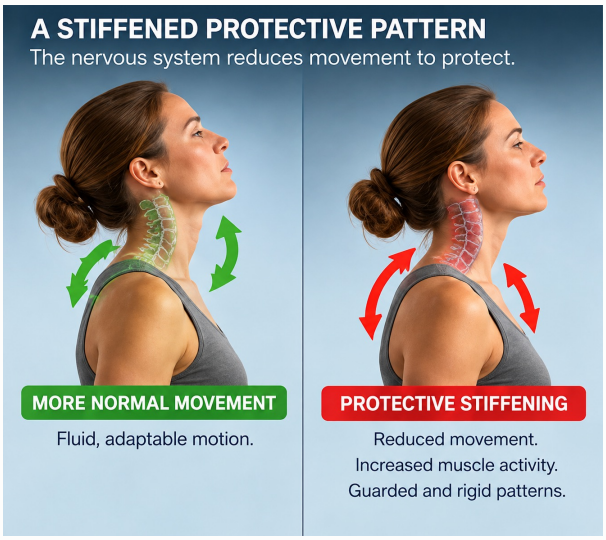
SPECIALISED EXAMINATIONS	
A6. Recommendation	**Unchanged**
Level of evidence	**Consensus ☑**

WAD grades I and II
Do not use specialised examination techniques (for example, EEG, EMG and specialised peripheral neurological tests) in patients with WAD grades I or II.

WAD grade III
Only use specialised examinations in selected patients with WAD grade III, for example, patients with suspected nerve root compression.

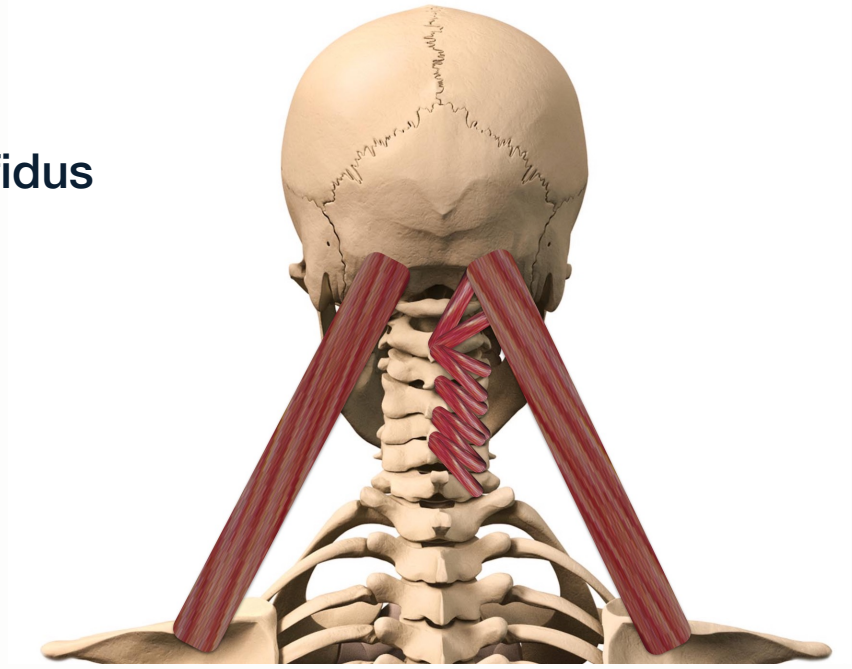
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The examination is educational and therapeutic



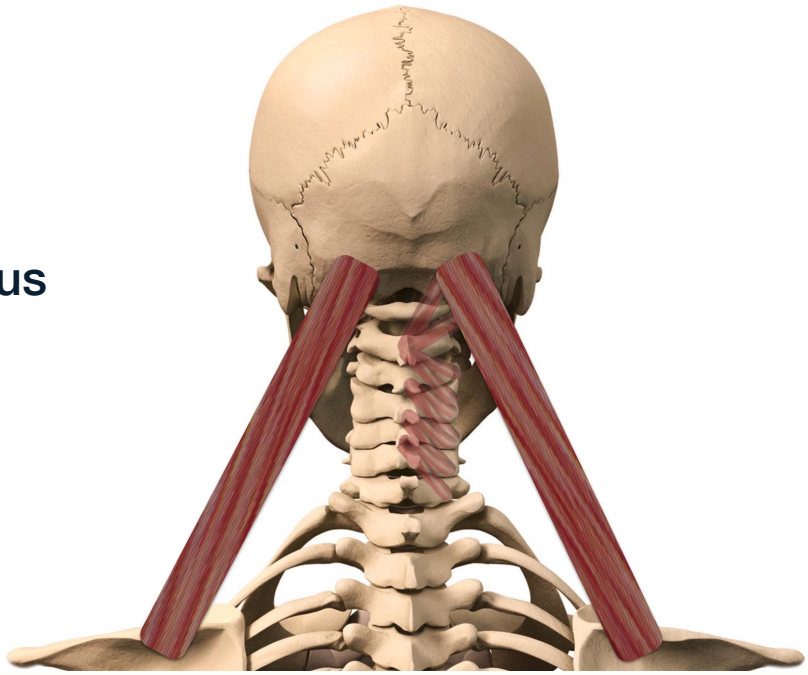
151

Multifidus



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Multifidus atrophy



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Other associated domains

Consider other overlapping and related symptoms and conditions

Sensorimotor and oculomotor findings should not automatically be labelled 'cervicogenic'. Use the examination to help characterise the impairment while considering competing explanations such as mTBI/concussion. Provocative tests may accumulate symptoms, so prioritise rather than performing multiple tests indiscriminately.

Also consider other related syndromes such as TMJ

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Traumatic brain injury screening

Screening estimates the likelihood of acute brain-function disturbance and identifies domains requiring further assessment.

EVENT EVIDENCE

- Plausible head or body force
- Acute altered mental status, amnesia or loss of consciousness
- Witness and contemporaneous records

CURRENT DOMAINS

- Cognitive, vestibular, oculomotor and balance findings
- Sleep, headache, mood and exertional tolerance
- Compare with cervical and pre-existing explanations

ESCALATION

- Progressive neurological or severe headache features
- Persistent multidomain impairment
- Refer for medical, neuropsychological or vestibular expertise when needed

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What NOT to do

- Do not perform an exhaustive battery of tests simply because they appear in a WAD assessment framework.
- Do not skip the basic neurological and cervical musculoskeletal examination in favor of sophisticated sensorimotor testing.
- Do not use a single positive sensory test to diagnose central sensitization.
- Do not interpret dizziness or oculomotor disturbance as automatically cervical in origin.
- Do not repeatedly provoke a highly irritable patient with multiple sensory or sensorimotor tests in one session.
- Do not measure impairments unless the result has a plausible role in classification, prognosis, treatment direction, referral or monitoring.

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Specialist level practical examination hierarchy

Level	Purpose	Examples	Who needs it?
1. Core safety / classification	Establish safety, neurological status and WAD grade	Observation; associated injury screen; neurological examination; cervical ROM; relevant tenderness/palpation	Essential focused examination; adapted to presentation
2. Musculoskeletal function	Characterise treatable physical impairment	ROM quantification; cervical muscle function/endurance; movement control; functional movement	When indicated by symptoms/function and likely to direct rehabilitation
3. Sensorimotor	Characterise dizziness, proprioceptive and balance-related impairment	Cervical joint position error; movement sense; oculomotor assessment; balance; selected coordination/proprioceptive tests	Acute: selected medium/high-risk or symptomatic patients. Chronic: greater guideline support, still presentation-matched
4. Pain sensitivity	Characterise hypersensitivity and pain-processing features	Ice-pain/cold hyperalgesia; PPT; allodynia; sharp hyperalgesia; BPPT/neural mechanosensitivity; selected CPM	Medium/high-risk or clinically suggestive presentations; not routine low-risk testing
5. Differential / associated domains	Test competing or concurrent explanations	Targeted vestibular, concussion/mTBI, TMJ, shoulder or other regional examination as indicated	Only when history/examination suggests the domain
6. Reassessment	Determine whether the targeted impairment is changing	Repeat key baseline measures rather than entire battery	Measures linked to treatment goals and clinical decisions

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MODULE 8

WORKING DIAGNOSIS

The working diagnosis should explain the syndrome, mechanisms, risk and functional consequence while remaining open to revision.

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MODULE 8

Let's consider:

1. Is the QTF grade a working diagnosis?
2. What constitutes a proper working diagnosis in WAD?
3. What question does the working diagnosis answer?
4. The elements of a WAD working diagnosis
5. What not to do?
6. Example WAD working diagnoses

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Is the QTF grade a working diagnosis?

The central distinction is classification versus working diagnosis

The QTF grade is essential, but it is best understood as a severity / classification label rather than a complete individualised working diagnosis.

A QTF grade answers:

"What broad WAD category does this presentation occupy?"

It efficiently communicates whether there are: no objective signs (Grade I), musculoskeletal signs (Grade II), neurological signs (Grade III), or fracture/dislocation (Grade IV).

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What constitutes a proper WAD diagnosis?

The QTF grade is a necessary part, but is not the 'whole'

The 2014 guideline requires QTF classification, but also separately requires history, focused examination, pain and disability assessment, recovery expectations, exclusion of fracture/dislocation and consideration of neurological or associated injuries.

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What question does the working diagnosis answer?

The working diagnosis should answer a broader clinical question:

"What is the patient presenting with, how severe is it, what important pathology has been excluded or identified, what associated clinical features are present, and which findings are relevant to management?"

The QTF grade is therefore part of that answer. It sits inside the working diagnosis but is not a substitute for the whole working diagnosis.

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The elements of a WAD working diagnosis

Component	What it contributes	Example wording
Condition + mechanism/context	Identifies the clinical entity and traumatic context	Acute whiplash-associated disorder following MVC
QTF grade	Standardized injury-severity classification	WAD Grade II
Dominant presentation	Describes the patient's main clinical problem	Predominantly mechanical cervical pain and stiffness with reduced ROM
Neurological status	Clarifies whether Grade III features exist and what they represent	No objective neurological deficit / neurological findings consistent with C6 radicular involvement
Serious injury status	Documents the safety/triage conclusion	No clinical indication of fracture/dislocation after CCR assessment; or imaging/referral indicated
Severity/function	Adds patient-reported burden	Pain 6/10; NDI 18/50
Associated symptom domains	Captures clinically relevant manifestations not represented by QTF grade	Headache and dizziness with sensorimotor features requiring targeted assessment
Prognostic modifiers	Adds recovery-risk information	Low recovery expectation / high initial pain-disability
Concurrent diagnoses	Separates other supported injuries/conditions	Associated concussion/mTBI, shoulder injury, TMJ disorder, etc., where clinically established

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What NOT to do (diagnostic pitfalls)

- Do not treat 'WAD Grade II' as if it identifies the injured anatomical structure. It is a clinical classification.
- Do not infer a specific disc, facet, ligament or muscle lesion unless the evidence genuinely supports that diagnosis.
- Do not make incidental or age-related imaging findings the primary diagnosis simply because they are visible.
- Do not label sensorimotor disturbance, pain hypersensitivity or psychological features as proven causal mechanisms when the assessment only demonstrates clinical features within those domains.
- Do not **overload the diagnosis** with every test abnormality. Include findings that materially describe the presentation, alter risk, or direct management.

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Example WAD working diagnoses

Simple uncomplicated presentation:

Acute WAD Grade II following MVC, characterised by cervical pain and stiffness with reduced cervical ROM and local tenderness; neurological examination intact; no indication for cervical imaging under the Canadian C-Spine Rule.

More multidimensional presentation:

Acute WAD Grade II following MVC, with moderate neck pain and self-reported disability, associated headache and dizziness/unsteadiness, and clinical features warranting targeted sensorimotor assessment; neurological examination intact.

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Example WAD working diagnoses

Neurological presentation:

Acute WAD Grade III following MVC, with neck and upper-limb pain and objective neurological findings consistent with cervical nerve-root involvement; targeted medical imaging/referral indicated according to the clinical findings.

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CLINICAL SYNTHESIS

“

A strong working diagnosis is specific enough to guide care and provisional enough to change with new evidence.

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MODULE 9



PROGNOSIS

Diagnosis tells us what the patient has. Prognosis estimates where the patient may be heading.

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MODULE 9

Let's consider:

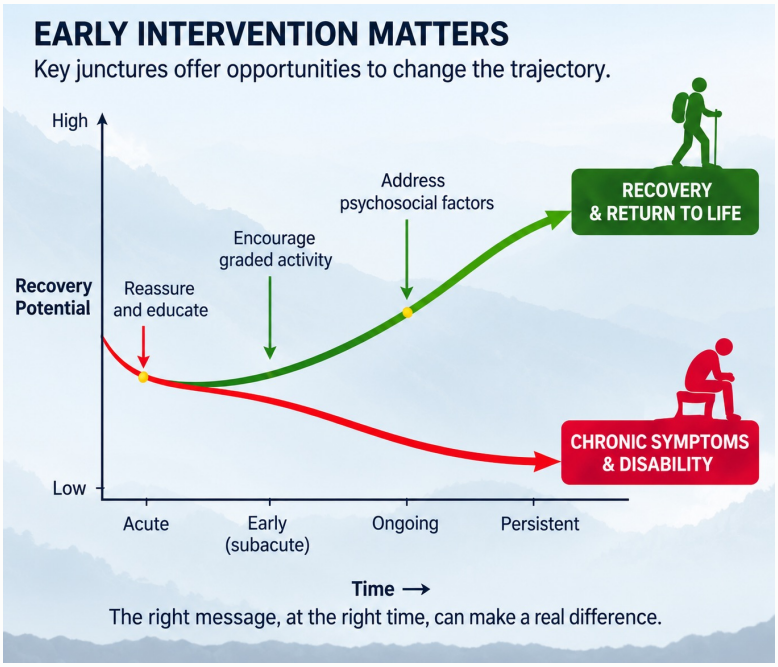
1. What is the clinical purpose of a prognosis?
2. What question does the prognosis answer?
3. What constitutes a useful WAD prognosis?
4. The strongest practical prognostic signals
5. What the prognosis is NOT
6. Risk stratification vs prognostic factor vs outcome measure
7. Example WAD prognoses
8. Fellow-level prognostic distinctions

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What is the clinical purpose of the prognosis?

- The clinical purpose is risk stratification: to identify who is likely to recover with simple care, who needs closer monitoring or more targeted assessment, and who requires earlier escalation.
- Prognosis should then be updated from the patient's actual recovery trajectory.

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EARLY INTERVENTION MATTERS
Key junctures offer opportunities to change the trajectory.
High
Low
Recovery Potential
Reassure and educate
Encourage graded activity
Address psychosocial factors
RECOVERY & RETURN TO LIFE
CHRONIC SYMPTOMS & DISABILITY
Acute
Early (subacute)
Ongoing
Persistent
Time ⟶
The right message, at the right time, can make a real difference.

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RIGHT INPUTS SWITCH THE TRACK
Timely guidance can redirect the course.
ONGOING DISABILITY
RECOVERY & FUNCTION

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What question does the prognosis answer?

Given what we know now, what is the likely recovery trajectory, what is the risk of persistent pain/disability, and how closely should this person be monitored?"

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What constitutes a useful WAD prognosis?

Should be more than simply 'favourable' or 'poor'

It should communicate:

1. The outcome of concern, such as persistent pain, disability, work/participation restriction or psychological symptoms
2. The patient's current risk profile
3. The principal factors contributing to that risk
4. The degree of uncertainty
5. How the prognosis changes management, monitoring and referral.

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The strongest practical prognostic signals

Domain	Factor	What it tells you	Practical use
Patient-reported intensity	High initial pain intensity	Higher risk of ongoing pain/disability	Core initial risk-stratification measure
Patient-reported disability	High initial NDI / disability	Strong signal for persistent disability	Core initial risk-stratification measure
Beliefs / expectations	Negative expectation of recovery	Associated with poorer pain/disability outcome	Elicit early; explore the reasons and monitor
Psychological	Post-traumatic stress symptoms	Associated with ongoing pain/disability	Screen when indicated, particularly if recovery is not progressing
Physical	Reduced initial cervical ROM	Predictive of ongoing disability in the 2014 guideline evidence	Adds prognostic information and provides a reassessment baseline
Pain sensitivity	Cold hyperalgesia	Predictive of ongoing disability; newer assessment guidance targets pain-sensitivity testing to medium/high-risk patients	Useful additional discriminator in selected higher-risk presentations
Clinical course	Failure of VAS/NDI to improve	Observed evidence that recovery is not progressing as expected	Review treatment, broaden assessment and consider escalation
Broader presentation	Multiple symptoms, dizziness, headache, back pain	Associated with poorer outcome, but generally secondary to stronger predictors	Adds context to the overall risk profile

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What prognosis is NOT

- Not simply the QTF grade.
- Not an exact estimate of how many days or weeks the patient will take to recover.
- Not a prediction based primarily on vehicle damage, impact speed or other crash mechanics.
- Not a conclusion drawn from one abnormal physical test.
- Not a list of every variable ever associated with persistent WAD.
- Not a deterministic label such as 'this patient will become chronic'.
- Not static—the prognosis should change as the patient's actual recovery trajectory becomes known.

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Risk stratification vs prognostic factor vs outcome measure

Term	Meaning in this module	Example
Prognostic factor	A characteristic measured at one point that is associated with a later outcome	Initial NDI, pain intensity, recovery expectation, PTSS, cold hyperalgesia
Outcome	The future state we are trying to estimate	Persistent disability, ongoing pain, work disability, psychological symptoms
Outcome measure	A tool used to quantify the patient's state and monitor change	NDI or VAS/NRS
Recovery trajectory	The pattern of change actually observed over time	Improving, plateaued, fluctuating or persistently high disability
Risk stratification	Combining prognostic information to guide the intensity and direction of care	Lower versus medium/high risk of poor recovery

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Factors PREDICTIVE of poor recovery

FACTOR	OUTCOME/S	STRENGTH OF EVIDENCE
SYMPTOMS		
Higher initial neck pain levels	Ongoing pain	A
	Ongoing disability	A
	Ongoing psychological symptoms	B
	Work disability	C
	Other (like muscle function)	D
Higher initial disability	Ongoing disability	A
Self-perceived injury severity	Ongoing pain/disability	B
Headache	Ongoing pain/disability	D
Higher number of symptoms	Ongoing pain/disability	C
WAD grade	Ongoing pain/disability	C
Back pain	Ongoing pain/disability	C
Dizziness	Ongoing pain/disability	C
PSYCHOLOGICAL		
Posttraumatic stress symptoms	Ongoing pain/disability	A
Negative expectation of recovery	Ongoing pain/disability	A
Somatisation	Ongoing pain/disability	B
Depression	Ongoing pain/disability	C
Pain catastrophising	Ongoing pain/disability	C
Coping strategies	Ongoing pain/disability	D
CRASH RELATED		
Self-rated collision severity	Ongoing pain/disability	C
DEMOGRAPHICS		
Age	Ongoing psychological symptoms	B
PHYSICAL		
Cervical range of motion	Ongoing disability	A
Cold hyperalgesia	Ongoing disability	A
PRE-COLLISION		
Pre-collision bodily pain	Ongoing pain/disability	B
Pre-collision psychological health	Ongoing pain/disability	C

Factors NOT PREDICTIVE of poor recovery

FACTOR	OUTCOME/S	STRENGTH OF EVIDENCE
SYMPTOMS		
Shoulder pain	Ongoing pain/disability	A
PSYCHOLOGICAL		
Kinesiophobia (fear of movement)	Ongoing pain/disability	C
Anxiety	Ongoing pain/disability	D
CRASH RELATED		
Seat belt use	Ongoing pain/disability	A
Awareness of collision	Ongoing pain/disability	A
Position in vehicle	Ongoing pain/disability	A
Speed of collision	Ongoing pain/disability	A
Head position at impact	Ongoing pain/disability	B
Use of head restraints	Ongoing pain/disability	B
Direction of impact	Ongoing pain/disability	B
Airbag deployment	Ongoing pain/disability	C
RADIOLOGICAL FINDINGS		
Radiological findings	Ongoing pain/disability	A
DEMOGRAPHICS		
Age	Ongoing pain	B
	Work disability	A
Living situation	Ongoing pain/disability	B
Work status	Ongoing pain/disability	C
Income	Ongoing pain/disability	C
PHYSICAL		
Lower pressure pain thresholds	Ongoing pain/disability	A
Motor/sensory-motor dysfunction	Ongoing pain/disability	A
BMI	Ongoing pain/disability	B
Cervical range of motion	Ongoing pain/disability	B
Sympathetic vasoconstriction	Ongoing pain/disability	B
PRE-COLLISION		
Pre-collision neck pain	Ongoing pain/disability	B
Pre-collision headache	Ongoing pain/disability	B
Pre-collision general health	Ongoing pain/disability	B
Pre-collision medication use	Ongoing pain/disability	B

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Notice a common theme that we've discussed?

Crash related factors that are NOT PREDICTIVE of poor recovery

Grade of evidence	Crash related factor
B	Head position at impact
B	Use of head restaints
B	Direction of impact
C	Airbag deployment

Crash related factors that are PREDICTIVE of poor recovery

Grade of evidence	Crash related factor
C	Self-rated collision severity

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Example WAD prognoses

Lower-risk acute presentation:

Current prognosis is favourable, with low pain and neck-related disability, positive recovery expectations and early functional improvement. Continue active management with routine outcome monitoring; complex assessment is not presently indicated.

Elevated-risk acute presentation:

The patient has an elevated risk of delayed recovery based on high initial pain and disability, negative recovery expectations and markedly restricted cervical ROM. Closer review and targeted assessment of relevant psychological, sensorimotor and pain-sensitivity domains are indicated.

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Example WAD prognoses

Poor trajectory despite initial care:

Prognosis is currently less favourable because pain and disability remain high with little measurable improvement and post-traumatic stress symptoms are present. Reconsider the clinical formulation and treatment direction and consider whiplash-expert and/or psychological referral.

Notice the consistency of the clinical domain structure and themes beginning at the history and continuing through the exam, working diagnosis, prognosis and now connecting through to the management

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Neck Disability Index

Instructions

This questionnaire has been designed to give your health professional information as to how your neck pain has affected your ability to manage in everyday life. Please answer every section and mark only the ONE box in each section which applies to you. We realise you may consider that two of the statements in any one section relate to you, but please just mark the box which most closely describes your problem.

Section 1 – Pain intensity	☐ I have no pain at the moment. ☐ The pain is very mild at the moment. ☐ The pain is moderate at the moment. ☐ The pain is fairly severe at the moment. ☐ The pain is very severe at the moment. ☐ The pain is the worst imaginable at the moment.
Section 2 – Personal care (washing, dressing etc.)	☐ I can look after myself normally without causing extra pain. ☐ I can look after myself normally but it causes extra pain. ☐ It is painful to look after myself and I am slow and careful. ☐ I need some help but manage most of my personal care. ☐ I need help every day in most aspects of my self care. ☐ I do not get dressed, I wash with difficulty and stay in bed.
Section 3 – Lifting	☐ I can lift heavy weights without extra pain. ☐ I can lift heavy weights but it gives extra pain. ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table. ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned. ☐ I can lift very light weights. ☐ I cannot lift or carry anything at all.
Section 4 – Reading	☐ I can read as much as I want to with no pain in my neck. ☐ I can read as much as I want to with slight pain in my neck. ☐ I can read as much as I want to with moderate pain in my neck. ☐ I cannot read as much as I want because of moderate pain in my neck. ☐ I can hardly read at all because of severe pain in my neck. ☐ I cannot read at all.
Section 5 – Headaches	☐ I have no headaches at all. ☐ I have slight headaches, which come infrequently. ☐ I have moderate headaches which come infrequently. ☐ I have moderate headaches which come frequently. ☐ I have severe headaches which come frequently. ☐ I have headaches almost all the time.
Section 6 – Concentration	☐ I can concentrate fully when I want to with no difficulty. ☐ I can concentrate fully when I want to with slight difficulty. ☐ I have a fair degree of difficulty in concentrating when I want to. ☐ I have a lot of difficulty in concentrating when I want to. ☐ I have a great deal of difficulty in concentrating when I want to. ☐ I cannot concentrate at all.
Section 7 – Work	☐ I can do as much work as I want to. ☐ I can only do my usual work, but no more. ☐ I can do most of my usual work, but no more. ☐ I cannot do my usual work. ☐ I can hardly do any work at all. ☐ I cannot do any work at all.
Section 8 – Driving	☐ I can drive my car without any neck pain. ☐ I can drive my car as long as I want with slight pain in my neck. ☐ I can drive my car as long as I want with moderate pain in my neck. ☐ I cannot drive my car as long as I want because of moderate pain in my neck. ☐ I can hardly drive at all because of severe pain in my neck. ☐ I cannot drive my car at all.
Section 9 – Sleeping	☐ I have no trouble sleeping. ☐ My sleep is slightly disturbed (less than 1 hr sleepless). ☐ My sleep is mildly disturbed (1-2 hrs sleepless). ☐ My sleep is moderately disturbed (2-3 hrs sleepless). ☐ My sleep is greatly disturbed (3-5 hrs sleepless). ☐ My sleep is completely disturbed (5-7 hrs sleepless).
Section 10 – Recreation	☐ I am able to engage in all my recreation activities with no neck pain at all. ☐ I am able to engage in all my recreation activities, with some pain in my neck. ☐ I am able to engage in most, but not all, of my usual recreational activities because of pain in my neck. ☐ I am able to engage in a few of my usual recreational activities because of pain in my neck. ☐ I can hardly do any recreation activities because of pain in my neck. ☐ I cannot do any recreation activities at all.

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Impact of Event Scale (IES)

On _____ you experienced a motor vehicle accident.
Below is a list of comments made by people after stressful life events. Please check each item, indicating how frequently these comments were true for you DURING THE PAST SEVEN DAYS. If they did not occur during that time please mark the 'NOT AT ALL' column.

	NOT AT ALL	RARELY	SOMETIMES	OTHER
1. I thought about it when I didn't mean to.				
2. I avoided letting myself get upset when I thought about it or was reminded of it.				
3. I tried to remove it from memory.				
4. I had trouble falling asleep or staying asleep because pictures or thoughts about it came into my mind.				
5. I had waves of strong feelings about it.				
6. I had dreams about it.				
7. I stayed away from reminders about it.				
8. I felt as if it hadn't happened or it wasn't real.				
9. I tried not to talk about it.				
10. Pictures about it popped into my mind.				
11. Other things kept making me think about it.				
12. I was aware that I still had a lot of feelings about it but I didn't deal with them.				
13. I tried not to think about it.				
14. Any reminder brought back feelings about it.				
15. My feelings were kind of numb.				

Impact of Event Scale (IES)

The IES (see page 44) was developed by Horowitz, Wilner and Alvarez to measure current subjective distress related to a specific event.[75] The IES is a self-report measure of posttraumatic disturbance and is very widely used. The scale is reproduced with permission of the author.

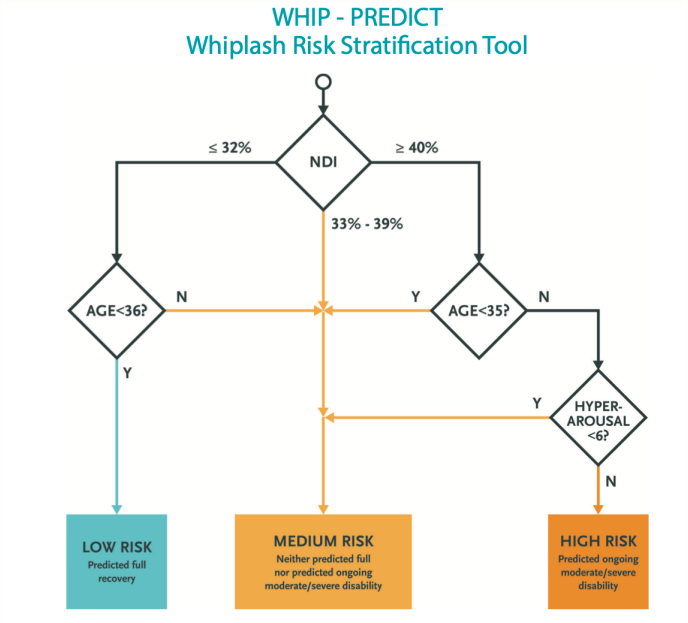
SCORING METHOD	Each item is scored:
Not at all	0
Rarely	1
Sometimes	3
Often	5

The item scores are summed. A total score of 25 or more, at three to six weeks after injury is in the 'moderate' range. A score of >43 is 'severe'.

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Ritchie C, Hendrikz J, Jull G, Elliott J, Sterling M. External validation of a clinical prediction rule following acute whiplash injury. J Orthop Sports Phys Ther. 2015;45:242-250.



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CLINICAL SYNTHESIS

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Initial disability, pain burden, recovery expectation and stress response matter more than the collision narrative alone.

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MODULE 10

MANAGEMENT

Active, domain and phase matched care that restores capacity, confidence and participation

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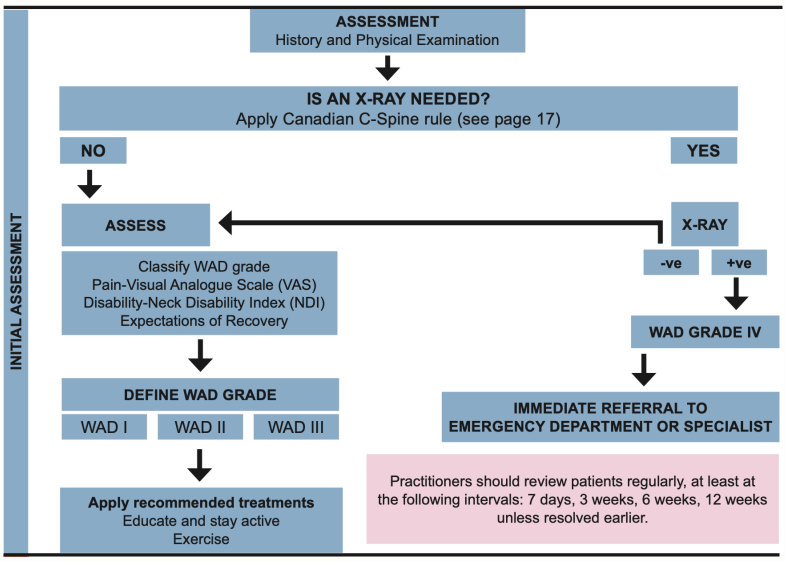
MODULE 10

Let's consider:

1. The therapeutic goals based upon the presentation
2. Management plans matched to dominant pain mechanism and domains of functional impairment
3. Bottom-up and top-down management approaches (Re-frame, re-map, re-learn)
4. Interdisciplinary care
5. The definition of maximal medical improvement (MMI)

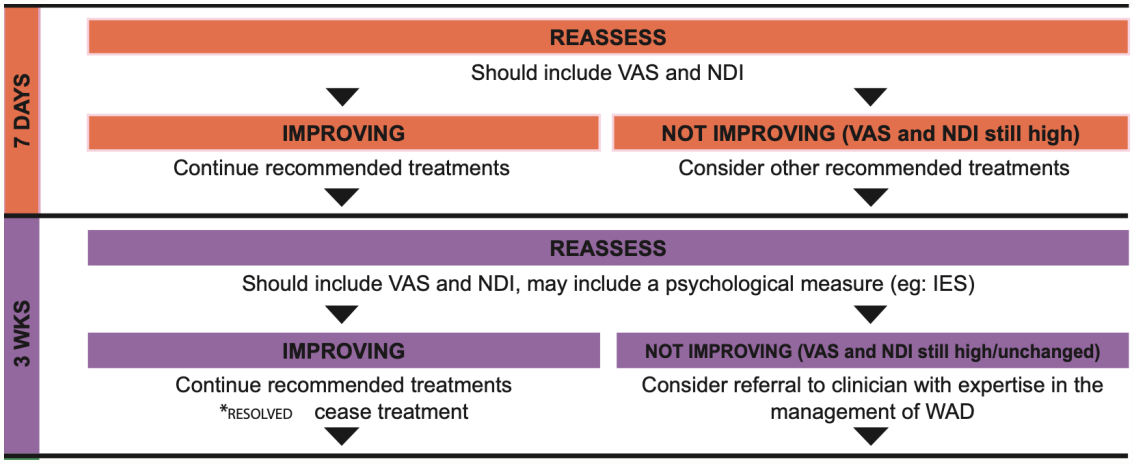
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Initial assessment



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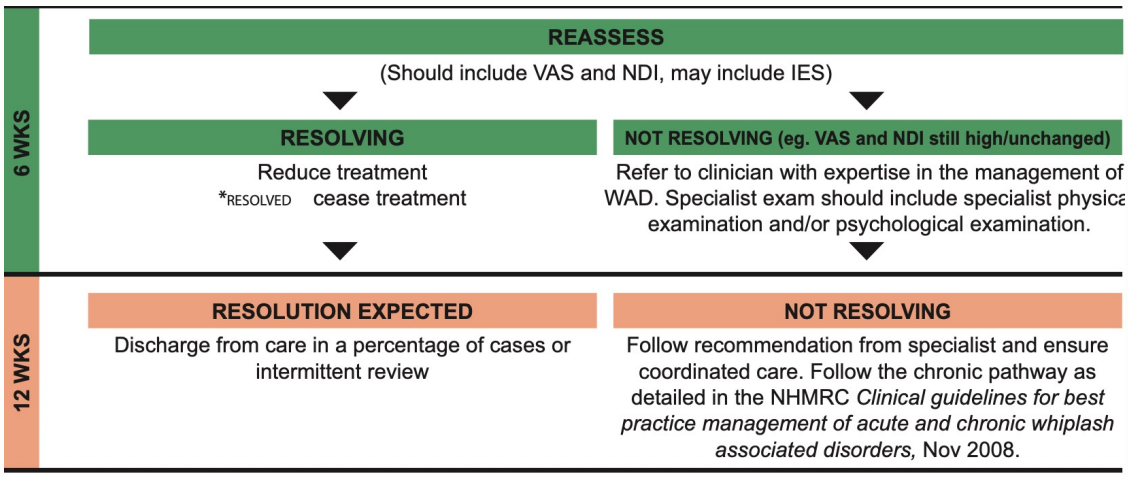
One week and three weeks



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Six weeks and 12 weeks



*Resolved is defined at VAS ≤3/10 and NDI <8/50

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Bottom-up and top-down strategies

Rehabilitation changes both the sensory input entering the system and the meaning assigned to that input.

BOTTOM-UP

- Full technique spectrum of manual treatment modalities (proprioceptive ‘re-mapping’)
- Graded tissue loading and movement
- Sensorimotor, vestibular and visual retraining

TOP-DOWN

- Reframing danger and uncertainty
- Attention, stress regulation and graded exposure
- Restoring agency and valued goals

INTEGRATION

- Reframe the problem
- Remap movement and sensory predictions
- Relearn capacity through repeated safe experience

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Domain-matched intervention

Treatment emphasis follows the dominant impairment rather than a standard package applied to every WAD grade.

CERVICAL

Movement and capacity

Mobility, motor control, endurance, strength and task-specific loading.

NEURAL

Nerve function

Protect progressive deficits, dose neurodynamic loading and coordinate medical pathways where required.

SENSOR

Sensorimotor

Joint position, head-eye coordination, balance, visual motion and vestibular tasks.

COGNITIVE

Stress and participation

Pain education, graded exposure, sleep, pacing, work participation and psychological care.

Mixed presentations usually need a sequence of priorities rather than simultaneous treatment of every abnormal finding.

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[CLINICAL COMMENTARY]

EYTHOR KRISTJANSSON, PhD, ManipTher, BSc[1] • JULIA TRELEAVEN, PhD, BPhty[2]

Sensorimotor Function and Dizziness in Neck Pain: Implications for Assessment and Management

Kristjansson E, Treleaven J. Sensorimotor function and dizziness in neck pain: implications for assessment and management. J Orthop Sports Phys Ther. 2009 May;39(5):364-77. doi: 10.2519/jospt.2009.2834. PMID: 19411769.

TABLE 1 EXAMPLE EXERCISE PROGRAM FOR CERVICOGENIC DIZZINESS/UNSTEADINESS[97]

1. Oculomotor exercises standing on an unstable surface (foam)
   - Eye movements with the head stationary, the patient moves the eyes between 2 black dots, 1 m apart, on the wall
   - The eyes follow a target moving side to side and up and down, with the head stationary
2. Eye-neck coordination exercises standing on an unstable surface (foam)
   - The patient moves the eyes first to a target and then the head, ensuring the eyes are kept focused on the target. The movements are rotations to the left and right, respectively
   - The patient fixes the gaze on a black dot on the wall, at 1 m distance, and moves the head in the transverse plane (rotation left and right) and in the sagittal plane (up and down)
   - Movements of the eyes to the left as the head is simultaneously moving to the opposite side (ie, right) and vice versa
3. Walking on uneven and on narrow surfaces (tandem)
   - Progression into performing various tasks while walking, such as throwing and catching a ball, and eye-neck coordination exercises (eg, following a stationary or moving object while walking)
4. Standing balance exercises: standing feet apart, feet together, and tandem, on an uneven surface, foam, balance board, and trampoline
   - The patient practices standing balance with their eyes open and closed, while performing various perturbations (eg, arm movements and exercises with a ball)
   - Eye-neck coordination exercises are also incorporated into these tasks
5. One of the latest sessions is performed outdoors, practicing walking on uneven surfaces like sand and stones, as well as uphill and downhill
   - Different tasks are performed while walking (eg, moving the head while following flying birds, watching flying birds or moving persons/cars with the head stationary)
6. Instructions for home exercises every day are given in accordance with the above

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Pain neuroscience education

Education should produce a more accurate and useful model, not a lecture that replaces treatment.

WORDS

- Use tissue language that validates injury without implying fragility
- Explain sensitization as changeable physiology
- Avoid telling the patient that pain is simply in the brain

CONCEPTUAL CHANGE

- Start with the patient's existing explanation
- Address contradictions with examination evidence
- Link the new model to a practical experiment

EVIDENCE LIMIT

- Education alone may not change disability
- Benefits depend on delivery and integration with activity
- Check what the patient understood rather than what was said

The power of education lies in credible language, shared reasoning and a successful behavioural test of the new model.

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FINAL THOUGHT..

Few things in clinical practice are more meaningful than helping someone who has lived with pain regain the confidence to move, work and live again. We may not always cure the condition, but relieving the burden of pain can profoundly change a life.

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