An Advanced Learning, Interpretation and Application Course on Physical Therapist Management of Neck Pain

APTA - Combined Sections Meeting

San Antonio, Texas
February 14 & 15, 2017

James Elliott
PT, PhD
j-elliott@northwestern.edu
@elliottjim

Northwestern University
Dept of Physical Therapy &
Human Movement Sciences
Feinberg School of Medicine

Dave Walton
PT, PhD
dwalton5@uwo.ca
@uwo_dwalton

Western University
School of Physical Therapy

An Advanced Learning, Interpretation and Application Course on Physical Therapist Management of Neck Pain

Elliott JM, Walton DM

This two-day pre-conference course will provide participants the opportunity to discuss, apply, and interpret new research and clinical knowledge to optimize outcomes of traumatic and non-traumatic neck pain. The instructors will guide participants towards deeper understanding of key aspects of neck pain care, from assessment through prognosis to treatment decisions and outcomes measurement. These experienced clinician researchers will not only provide a balanced and accurate representation of the current state of evidence-informed practice for neck pain, but will use novel transformative teaching and learning tools to help participants make sense of complex topics and apply new knowledge in a way that leads to observable clinical impact.

The course will be broken into 3 relevant modules, each of which builds upon the previous: Assess, Predict, and Treat:

Assess: In this module, participants receive and discuss theoretical knowledge about, and practical experience applying, a number of novel assessment/evaluation tools for use in patients with acute or chronic neck pain. These include tools that tap each of the nociceptive/biomechanical, cognitive, affective, social, peripheral neuropathic and central neurogenic domains. A new framework that combines existing and easy to use measurement tools will be presented to help participants make sense of their patients' pain experiences and provide directions for more informed treatment planning to optimize patient outcomes.

Predict: In this module participants will learn about the nature of chronic neck pain and, more importantly, the transition from acute to chronic pain. Clinical questions that will be answered include, but are not limited to: 1) Who develops chronic pain and who doesn't? 2) Why does chronic pain develop in some people but not others? 3) What 'risk factors' can clinicians look for to help predict and prevent the development of chronic pain? Framed within a truly integrated biopsychosocial model of chronic pain development, participants will leave with a better understanding of how to confidently identify the 'at risk' patient, identify modifiable risk factors, discuss the nature of communicating risk and the influence of compensation/litigation on successful rehabilitation outcomes. Communication with

patients, funders, and other members of the healthcare circle will be key components.

Treat: In this module participants will build upon the knowledge gained from their *Assess* and *Predict* sessions to build informed treatment plans for patients with acute and chronic neck pain. New phrases such as 'plugging the biggest hole' will become common language for clinical reasoning as they learn about evidence-informed treatment approaches for addressing nociceptive/biomechanical, central neurogenic, peripheral neuropathic, cognitive, affective and social aspects of the pain experience that can be appropriately managed by rehabilitation professionals. Topics will include, but are not limited to, motor control, neuroplasticity, exercise-induced hypoalgesia, oculomotor retraining, use and benefit of manual therapies, targeted pain neurophysiology education, managing the depressed or anxious patient, and working as part of a multidisciplinary team including knowing when to refer for multimodal care. This session will include a mix of lecture-style sessions to advance knowledge supported by practical sessions to solidify new skills and behaviors. A focus on being 'critical consumers of knowledge' will give participants greater ability to appraise and interpret new evidence as it comes available even after completion of this course.

By the end of this course participants will be able to:

1. Describe and apply a new framework for pain assessment using a 'radar plot' as an approach to structure and interpret assessment findings

2. Conduct and interpret a comprehensive clinical assessment of patients with acute and chronic neck pain from pathomechanical, neural, and psychosocial perspectives.

3. Critically discuss the value and caveats of diagnostic imaging for patients with traumatic and non-traumatic neck pain.

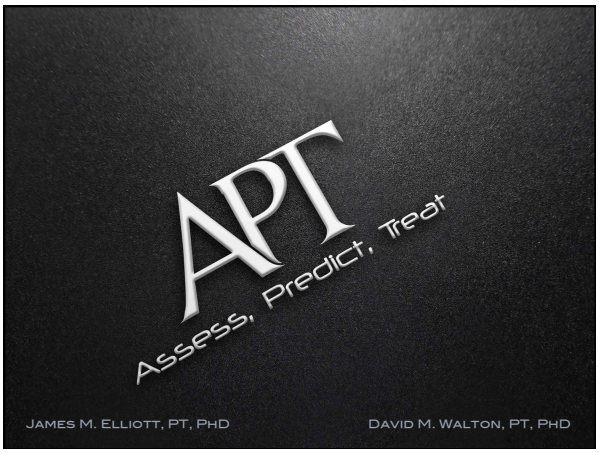
4. Identify, describe, and synthesize risk factors for chronicity in patients with acute traumatic neck pain, and create an 'Acute Injuries Prognostic Profile' that can assist with treatment planning and interdisciplinary communication.

5. Discuss and contrast the value of different intervention approaches for acute and chronic neck pain, as described in scientific literature and as indicated by the assessment and prediction frameworks.

Start & finish times (am/pm)	Topic or element (include lunch and tea breaks)	Presenters Names	Teaching strategies and learning activities

			(lecture, demonstration, discussion group, practical session)
Day 1			
8 - 8.30	**REGISTRATION**		
8.30 – 9.45	Whiplash and idiopathic neck pain – Can we predict and optimize recovery? • Differences b/w traumatic and non-traumatic neck pain (pathoanatomy, psychology, QST, prognosis etc.) • Expected trajectories of neck pain	Elliott	Lecture
9.45-10.00	**MORNING TEA**		
10:00 – 10:30	Introduction to the concept of triangulation in evaluation of neck pain – a useful tool for clinical decisions • Introduce the concepts of triangulation and the radar plot	Walton	Lecture
10:30-12:00	How to use validated (& meaningful) outcome measures without burdening you or the patient • Application and interpretation of key outcomes specific to neck pain • Include but not limited to o NDI o SRI o NPRS or other pain scale o SLANSS o BPI	Elliott/Walton	
12:00-1:00	**LUNCH**		
1:00 – 2:00	Outcome measures 1. Cont…with a focus on interpreting cut-scores and evaluating change. 2. Introduction of cases		Lecture
2:00 – 4:00	Where Mind meets Body: The Psychological Domain of Neck Trauma and How it Affects WAD Rehabilitation **Including AFTERNOON BREAK**	Walton/Elliott	Lecture & Discussion
4.00 – 4.15	**FINISH DAY 1 incl. formative feedback for next day**		
4:15	End		
Day 2			
8.30-10.00	Quantitative Sensory Testing in the clinic – practical advice to identify neck pain mechanisms 1. What does QST tell us? 2. Different types of QST for clinicians 3. Practical session: PPDT, how to apply and interpret	Walton	Lecture & Practical

	4. Discuss: what does QST mean for treatment planning?		
10.00-10.15	**Morning Tea**		
10.15 -11.15	Triangulation of the outcome measures – case examples • Finish triangulation exercise from yesterday.	Walton	Lecture
11.15-12.00	Burning questions – small group open discussions with the instructors		
12 noon – 1:00	**LUNCH**		
1.00-3:00	The role of physical rehabilitation in whiplash associated disorders- are we helping and how do we accurately quantify pain (our patient's pain experience)? Evaluation and training of the posterior/anterior neck muscles, oculomotor retraining, manual therapies, education	Elliott/Walton	Lecture & Practical
3:00 – 4:00	Case Management Examples with focus on assessment and treatment planning to address risk and facilitate recovery.	all	Discussion
4:30 – 5:00	**Wrap up and finish (incl. feedback)**		



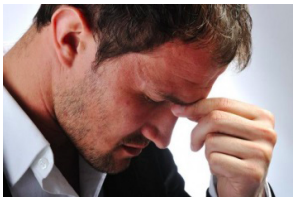
Objectives

By the end of this workshop, you will be able to:

1. Discuss the nature of neck pain, including the distinction between traumatic/non-traumatic and acute/chronic
2. Apply the Assess, Predict, Treat framework when dealing with neck pain
3. Critically discuss current knowledge and gaps regarding assessment, prognosis, and management of neck pain

Outline

This is what we are going to do this weekend…



FRANK MIELOWSKI

Frank is 38 years old, Executive Assistant.

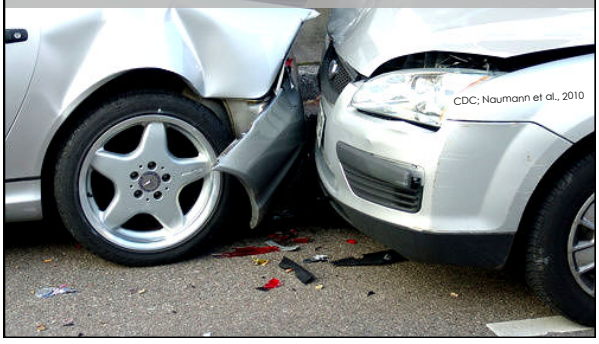
He was involved in a motor vehicle *accident* in which he was hit from behind while at a stop light 1 week ago.

His doctor diagnosed him with 'whiplash' and sent him to see you.

What are we going to do for him (and all the other stakeholders)?

PATHOMECHANICS

MOTOR VEHICLE-RELATED INJURIES SEND MORE THAN *4 MILLION PEOPLE* TO HOSPITAL EMERGENCY DEPARTMENTS EVERY YEAR



~ $100 Billion in Indirect costs

~$30 Billion in medical & rehabilitative costs

Naumann et al., 2010

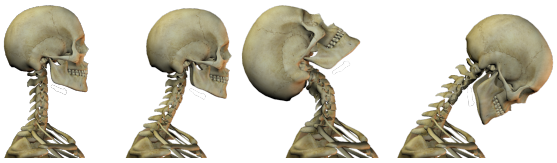
~$1.5 Billion from 1989-1998 in NSW

~$350 Million in Queensland (2011-2012)

£3 billion per year in the United Kingdom

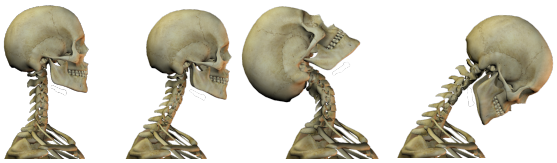
$4.5 billion in Ontario (2010)

Sterling M. 2014; Ontario Auto Insurance Anti-Fraud Task Force Interim Report. 2011



At the core of complex and wide-reaching matter is a *simple* question...

does injury following MVC exist?



THE SPECTRUM OF SIGNS AND SYMPTOMS OF WAD SUGGEST MULTIFACTORIAL ETIOLOGY

- NECK PAIN AND STIFFNESS
- HEADACHE
- RADICULAR SIGNS
- WIDESPREAD SENSORY HYPERSENSITIVITY
- COGNITIVE INTERFERENCE
- ANXIETY AND DEPRESSIVE SYMPTOMS
- A RANGE OF OTHER CONFUSING SYMPTOMS

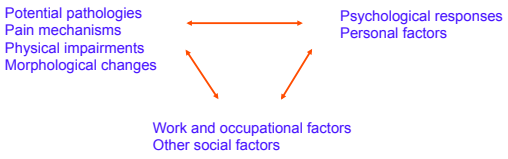
CONVERGING EVIDENCE AVAILABLE INDICATING THE PRESENCE OF A ***PERIPHERAL*** LESION IN SOME INDIVIDUALS FOLLOWING WHIPLASH INJURY (CURATOLO ET AL., 2011)

THERE IS ALSO EVIDENCE TO INDICATE THAT A 'LESION' MAY NOT BE A PREREQUISITE FOR SOME OF THE CLINICAL FEATURES OF PATIENTS WITH WHIPLASH ASSOCIATED DISORDERS (WAD). (STERLING ET AL., 2011)

Operating within a

Biopsychosocial Model

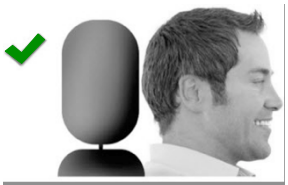
facilitates appreciation of all features in assessment and management



WHAT DO WE KNOW ABOUT THE PATHOMECHANICS OF WHIPLASH?

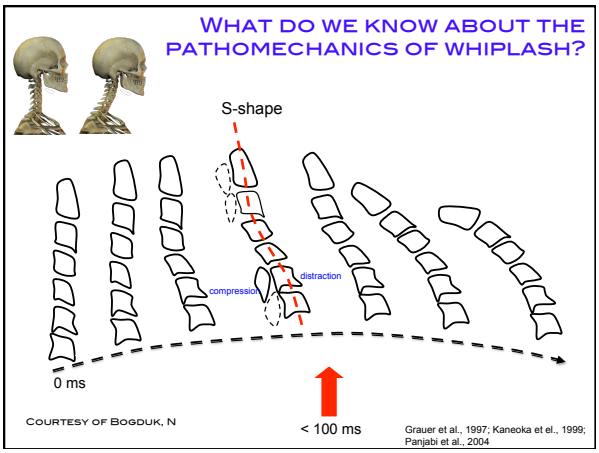
~0-200ms

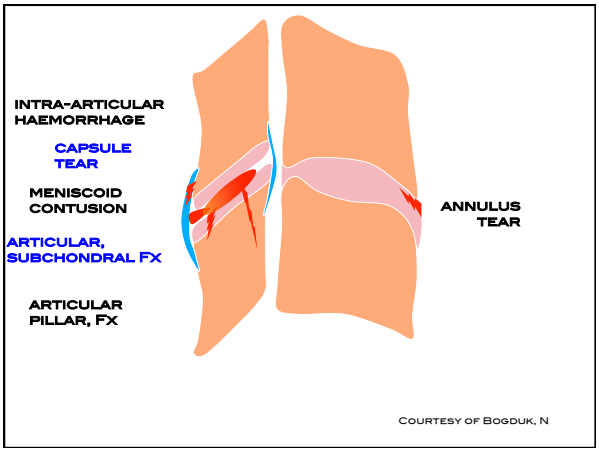
The Science of Safer Seats & Safer Cars

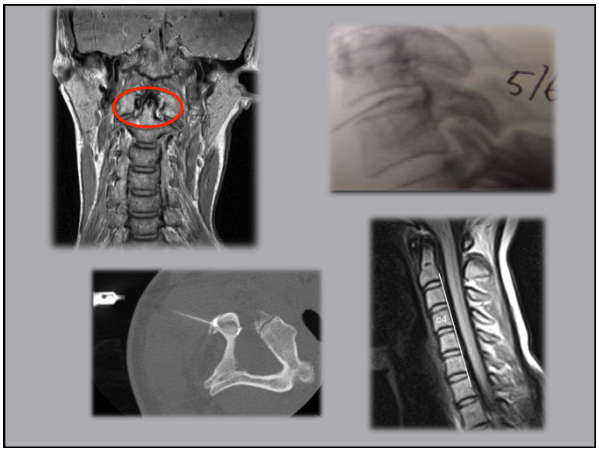


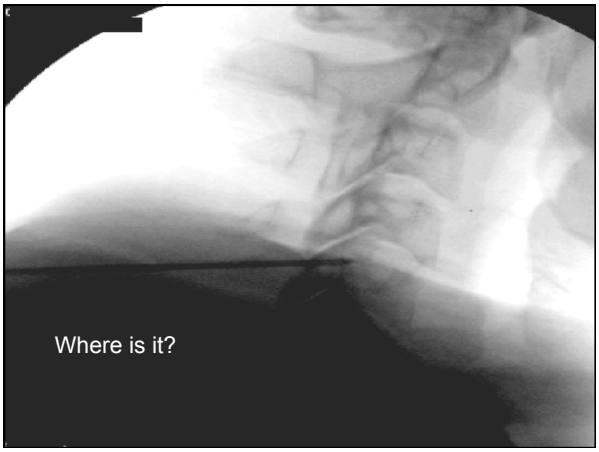
The question beyond 'smarter' seats and properly positioned head restraints becomes…

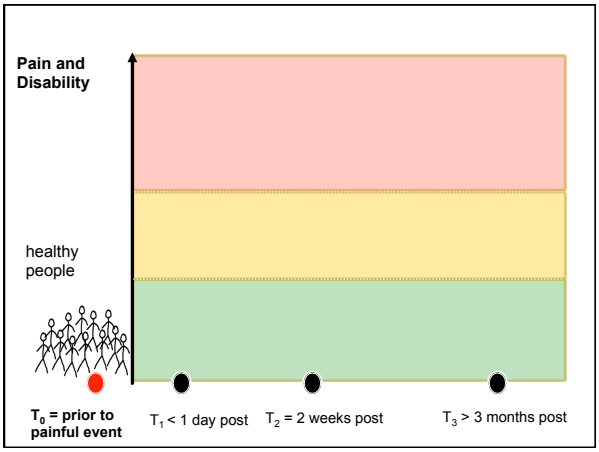
Why do some, but not others transition from acute to persistent pain-related disability?

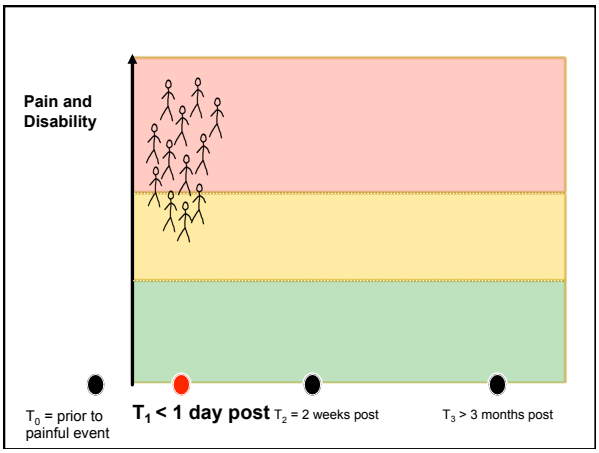

WHAT DO WE KNOW ABOUT THE PATHOMECHANICS OF WHIPLASH?
S-shape
compression
distraction
0 ms
< 100 ms
COURTESY OF BOGDUK, N
Grauer et al., 1997; Kaneoka et al., 1999; Panjabi et al., 2004

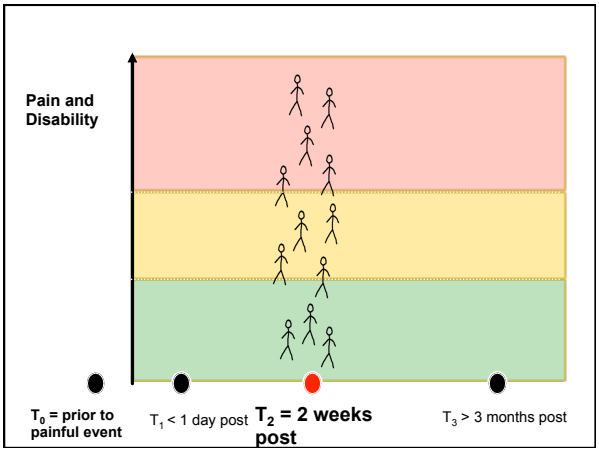

INTRA-ARTICULAR HAEMORRHAGE
CAPSULE TEAR
MENISCOID CONTUSION
ARTICULAR, SUBCHONDRAL FX
ARTICULAR PILLAR, FX
ANNULUS TEAR
COURTESY OF BOGDUK, N

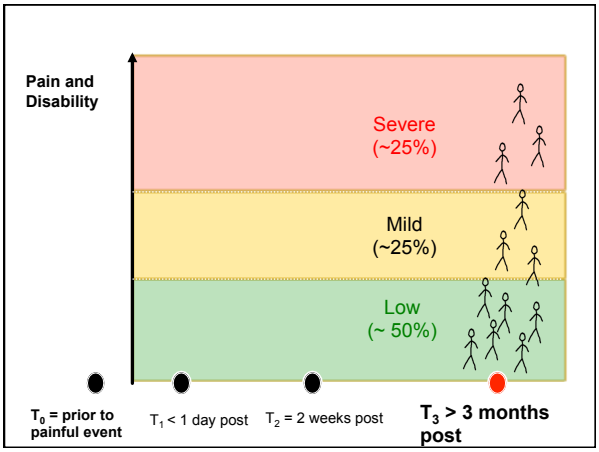



Where is it?


Pain and Disability
healthy people
T_0 = prior to painful event
T_1 < 1 day post
T_2 = 2 weeks post
T_3 > 3 months post


Pain and Disability
T0 = prior to painful event
T1 < 1 day post
T2 = 2 weeks post
T3 > 3 months post


Pain and Disability
T0 = prior to painful event
T1 < 1 day post
T2 = 2 weeks post
T3 > 3 months post


Pain and Disability
Severe (~25%)
Mild (~25%)
Low (~ 50%)
T0 = prior to painful event
T1 < 1 day post
T2 = 2 weeks post
T3 > 3 months post

What's unique about the 25%?

Walton et al., 2013

Neck pain, Stiffness, & Headache

What's unique about the 25%?

Walton et al., 2013

High Initial Pain Intensity (≥6/10) & Neck-Related Disability

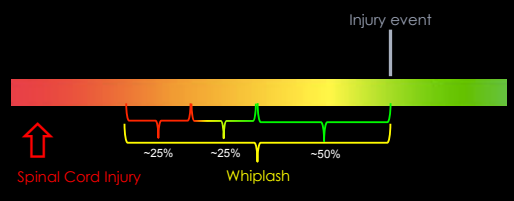
Widespread sensory hypersensitivity

Cognitive interference

Anxiety and Depressive symptoms

A range of other confusing symptoms
(cold/mechanical hypersensitivity/swallowing/voice)

A spectrum of neurobiological injury…



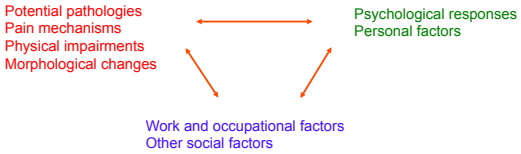
With BioPsychoSocial Factors…

WHAT ELSE DO WE KNOW?

Operating within a

Biopsychosocial Model

facilitates appreciation of all features in assessment and management

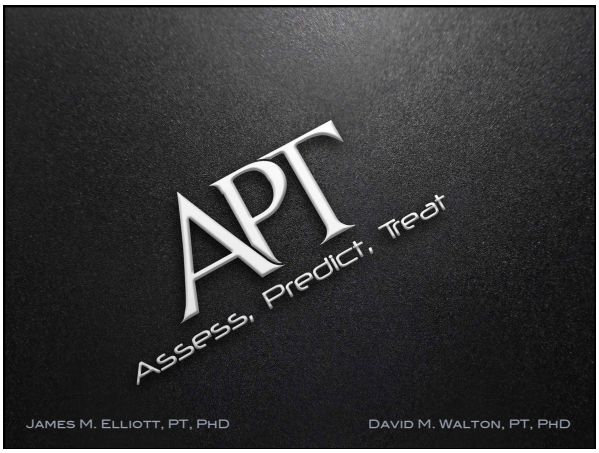


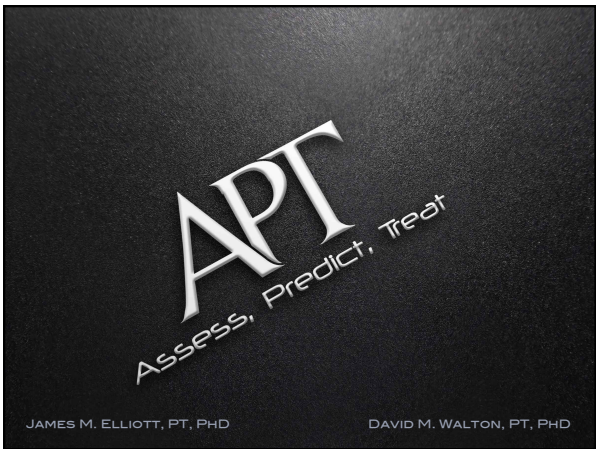
whiplash is not a homogenous condition

Most patients demonstrate a fairly uncomplicated clinical presentation of mild to moderate levels of pain and disability, local hyperalgesia over the neck, mild psychological distress and motor dysfunction.

At the other end of the spectrum, there is a group of whiplash patients (approx 25%) who demonstrate a complex clinical picture. It is this group that demonstrate poor functional recovery at both 6 months and 2 years post injury…

The APT workshop will help to Assess/Predict/Treat patients following whiplash injury

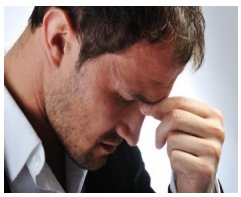

APT
Assess, Predict, Treat
James M. Elliott, PT, PhD
David M. Walton, PT, PhD



Objectives

- Describe frameworks applicable to acute and chronic, traumatic or non-traumatic neck pain
- Describe the use of a radar plot and triangulation for making clinical decisions
- Discuss the application and interpretation of patient-reported tools
- Discuss use of Quantitative Sensory Testing
- Discuss use of imaging modalities

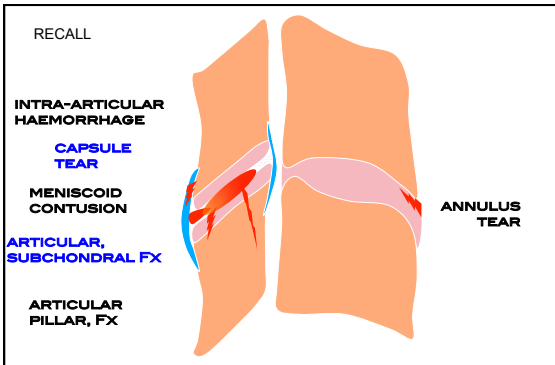
Frank Mielowski



A Proposed Framework

- Assessment of acute pain:
  - **Prognosis-Based Approach**
- Assessment of chronic pain:
  - **Comprehensive Mechanism-Based Approach**
- Traumatic vs. Non-traumatic:
  - Base approaches are similar for both
  - For traumatic pain, recognize the added influence of stress and likelihood of damage to additional tissues

RECALL

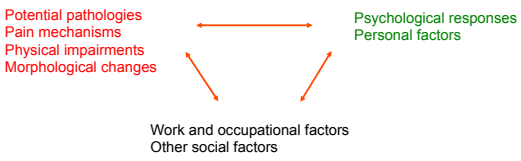


COURTESY OF BOGDUK, N

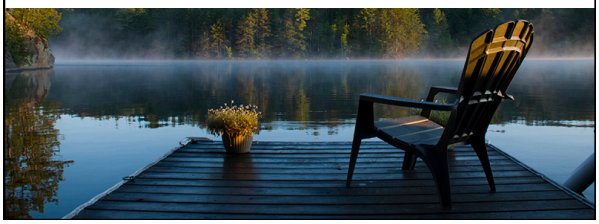
Operating within a

Biopsychosocial Model

facilitates appreciation of all features in assessment and management



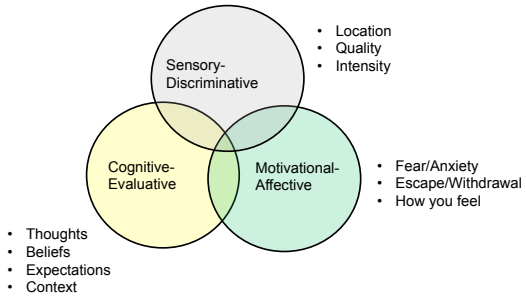
Using available measures to quantify the *cause/effect* of traumatic events



11 October, 2001

11 September, 2001

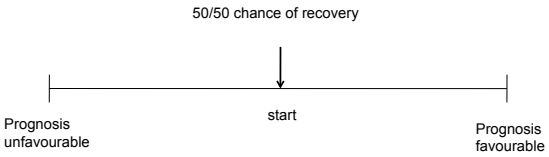
The Nature of Pain – Neuromatrix Model (R. Melzack)



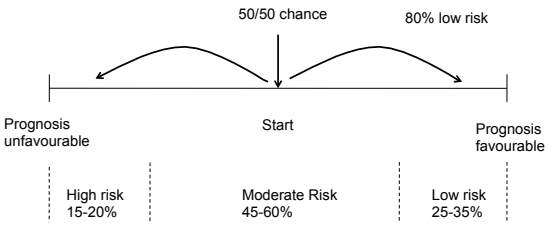
A Suggested Clinical Approach – Acute Trauma

1. **Quick Screening for Prognosis**
   - **CPR for WAD**
   - **BIPQ, TIDS or other**
2. **Low risk? Educate and follow-up**
   - **Estimated ~25-35%**
3. **Moderate or High Risk? Search for *modifiable mechanisms***

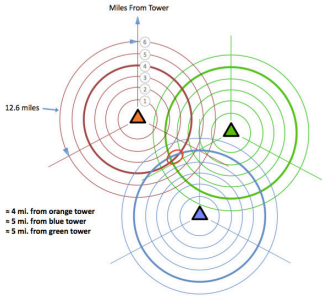
A conceptual model for clinical assessment of acute injuries



A conceptual model for clinical assessment

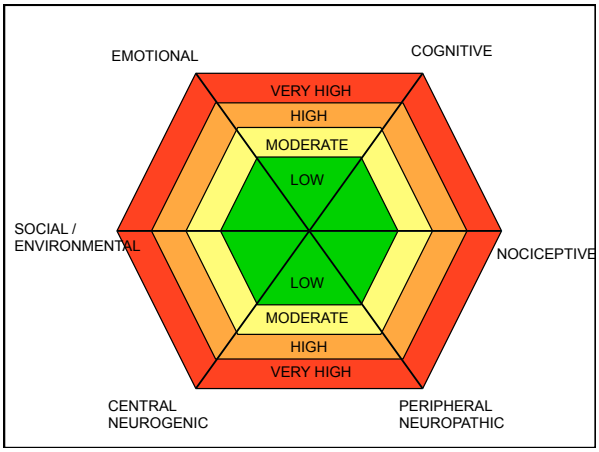


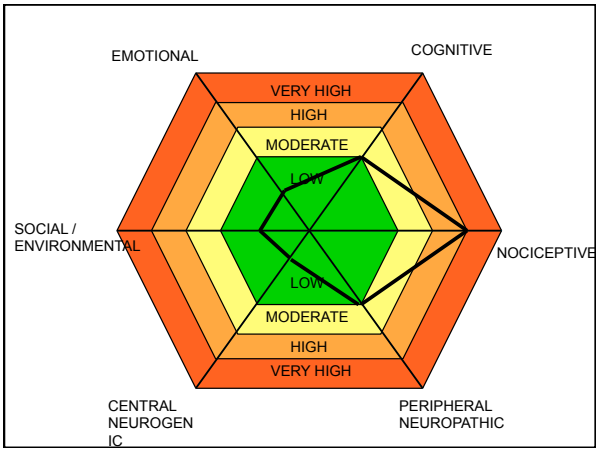
'Triangulating' Pain

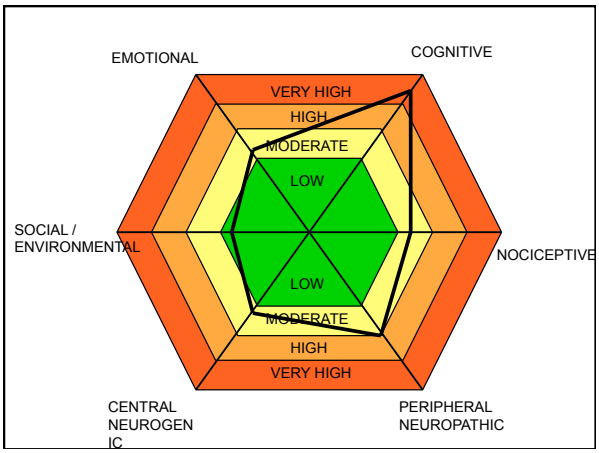


‘Triangulating’ Pain

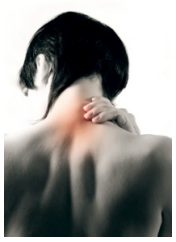
- **Use tools that tap distinct domains of the experience**
- **Decision regarding prognosis (incl. likely mechanism) should be based on results from *at least 3* tools**







Assessment of Acute WAD



Molly

An athletic 25-year old, was on her way home from the gym on Sunday afternoon. She was the restrained driver, stopped at a red light when she was rear-ended by a car that was braking from traveling at approximately 35 mph. The rear-end of her car was heavily damaged and had to be towed away. Molly reported severe pain (7-8/10) in her neck at the time of the crash. She was transferred to the emergency medical department (ED) via ambulance with no significant reductions in self-reported pain or radiographic evidence of structural damage. She has now come to you 2 days later for an initial assessment.

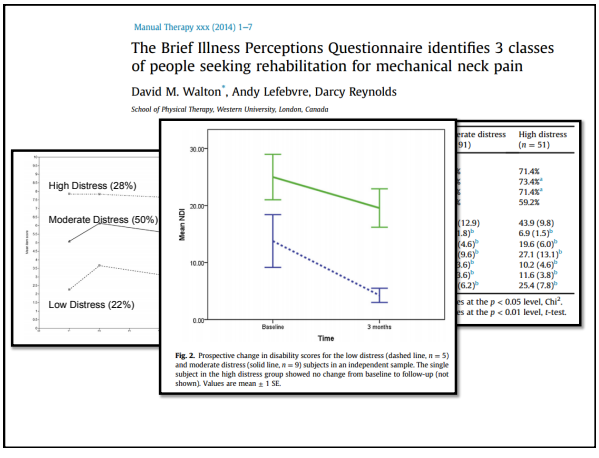
Describe your first-pass clinical tools

Have your 'go-to-' toolbox

- A set of 3-4 useful tools that every patient completes
- Example (total 5-8 minutes):
  - Body Diagram
  - Generic or Region-Specific Disability Scale
  - Cognitive tool (e.g. PCS or TIDS)
  - Brief Illness Perceptions Questionnaire

The Brief Illness Perceptions Questionnaire

- A quick (9-item) tool to quantify Leventhal's *Illness Representations*
- Quantifies the 6 domains:
  - Identity
  - Timeline
  - Consequences
  - Cause
  - Control/Cure
  - Illness Coherence



Manual Therapy xxx (2014) 1–7

The Brief Illness Perceptions Questionnaire identifies 3 classes of people seeking rehabilitation for mechanical neck pain

David M. Walton*, Andy Lefebvre, Darcy Reynolds

School of Physical Therapy, Western University, London, Canada

	erate distress (91)	High distress ($n = 51$)
		71.4%
		73.4%[a]
		71.4%[a]
		59.2%
	(12.9)	43.9 (9.8)
	(1.8)[a]	6.9 (1.5)[a]
	(4.6)[b]	19.6 (6.0)[b]
	(9.6)[b]	27.1 (13.1)[b]
	(3.6)[a]	10.2 (4.6)[a]
	(3.6)[a]	11.6 (3.8)[a]
	(6.2)[b]	25.4 (7.8)[b]

es at the $p < 0.05$ level, Chi^2.
es at the $p < 0.01$ level, t-test.

Fig. 2. Prospective change in disability scores for the low distress (dashed line, $n = 5$) and moderate distress (solid line, $n = 9$) subjects in an independent sample. The single subject in the high distress group showed no change from baseline to follow-up (not shown). Values are mean ± 1 SE.

Neck Disability Index

- Most widely used neck-specific functional scale in the world
- 10 domains, each rated 0-5
- Score < 5/50 (<10%) = No disability
- Score ≥25/50 (>50%) = Severe to complete disability

NDI-5 (Walton & MacDermid 2013)

- Rasch-based revision of the original NDI, sound measurement properties
- 5 items, score range 0-24
- Strong (r > 0.95) correlation with original
- Categories same as original when converted to percent

Digging Deeper

Domain	Test
Emotional	HADS
Cognitive	PCS
Nociceptive	PPT
Peripheral Neuropathic	CPT & WU & SLANSS
Central Neurogenic	Widespread PPT & CPM
Social/Environmental	Good interview questions

Emotional: Hospital Anxiety and Depression Scale (HADS)

- 14 items
- Starts with Anxiety domain, switches Anx/Dep every other item
- Items 2, 4, 6, 7, 12, 14 are reverse-scored
- Interpretation:
  - Normal (0-7), Mild (8-10), Moderate (11-14), Severe (15-21)

Notes on Emotional Screens

- HADS – more innocuous than other scales
- Structured quantification tool may not be necessary early, potentially even harmful – be judicious in its use
- What about PTSD?

Cognitive: Pain Catastrophizing Scale

- 13 items scored 0-4, measuring 'exaggerated negative orientation towards pain'
- Range 0-52
- Interpretation:
  - ≤20: Normal
  - 20-30: Clinically-relevant catastrophizing
  - >30: Severe catastrophizing

Don

Two-weeks following a motor vehicle collision, Don, a 28 year old construction worker informs you that he cannot tolerate the ice bag on the back of his neck after therapy stating it is irritating and 'stirs up' his symptoms of neck pain, headache, and dizziness.

What additional tests can you consider given this presentation? What mechanisms may be at play?

Nociceptive: Pressure Pain Threshold

- **'Psychophysical Quantitative Sensory Test'**
- **"I'm going to start slowly applying pressure to the skin over your [area]. Please tell me the moment the sensation changes from pressure to pain."**
- **5N/s (1kg/s) increase**
- **Use PPT app for no. of reps**

		1st	2nd	3rd
Male	Lbf			
	UFT	5.48	8.28	11.31
	TA	8.03	12.14	18.31
	Kgf			
	UFT	2.49	3.76	5.13
	TA	3.64	5.51	8.30
	N			
	UFT	24.36	36.81	50.26
	TA	35.67	53.97	81.37
Female	Lbf			
	UFT	3.63	5.48	8.13
	TA	5.06	8.31	11.32
	Kgf			
	UFT	1.64	2.49	3.69
	TA	2.29	3.77	5.13
	N			
	UFT	16.11	24.37	36.14
	TA	22.49	36.93	50.31

Normative values for mechanical (pressure) pain threshold from a sample of 227 females and 82 males with neck pain of varying cause and duration. The values are thresholds between quartiles. For example, the value in the '1st' column is the threshold between the 1st (lowest 25%) and 2nd (25-50%) quartiles.

Used with permission of Dr. David Walton, Western University Canada. Email dwalton5@uwo.ca

An Android-based smartphone app for interpreting PPT scores can be downloaded for free from www.pirlresearch.com/clinician-resources/

PPT Interpretation

- Classes:
  - Global Hypersensitivity (Low-Low) (48%)
  - Normosensitive (Mod-Mod) (38%)
  - Local Hypersensitivity (Mod – High) (6%)
  - Global Hyposensitivity (High – High) (9%)

Peripheral Neuropathic 1

- Cold hyperalgesia
  - Simple test: The 'cold nail'
  - 5 seconds application in local area and contralateral analogue
  - Cold Pain Rating Scale
    - Score > 13/20 OR side-to-side difference >5 points indicative of cold hyperalgesia

Peripheral Neuropathic 2

- 'Wind up Pain' (Temporal Summation)
- Pin prick, ~1Hz – mildly noxious level for 30 seconds
- Positive test:
  - Initially non-painful becomes painful
  - Initially painful increases in intensity

Peripheral Neuropathic 3

- SLANSS
  - Self-rated
  - 7 questions each rated 'Yes/No'
  - Each 'Yes' weighted by a different factor
  - Summed score >12 highly indicative of 'Pain of Primarily Neuropathic Origin' (POPNO)

Central Neurogenic

1. Widespread Hyperalgesia (PPT or CPT as per previous)
2. Central Sensitization Index (Mayer et al. 2012)

Central Neurogenic

Conditioned Pain Modulation

- Simple Method:

1. Test PPT in an appropriate area
2. Induce a painful but tolerable stimulus for at least 30 seconds
3. Re-test PPT in same area after 30-60 seconds
4. Normal: at least 10% increase in PPT

Environmental/Social

- Quantification tools do exist (e.g. the Spousal Response Inventory)
- What questions / domains should be explored?

IASP

PAIN® xxx (2014) xxx–xxx

PAIN®

www.elsevier.com/locate/pain

No man is an island: Living in a disadvantaged neighborhood influences chronic pain development after motor vehicle collision, and this effect is moderated by common genetic variation influencing HPA axis function

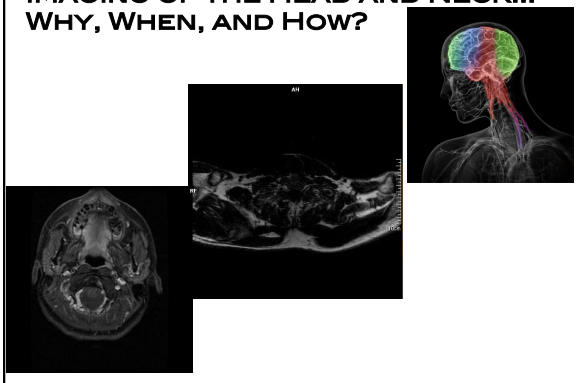
Jacob C. Ulirsch[a,b], Mark A. Weaver[c], Andrey V. Bortsov[a,b], April C. Soward[a,b], Robert A. Swor[d], David A. Peak[e], Jeffrey S. Jones[f], Niels K. Rathlev[g], David C. Lee[h], Robert M. Domeier[i], Phyllis L. Hendry[j], Samuel A. McLean[a,b,k,*]

(n = 948)

Follow-up evaluations were completed via telephone or Internet survey 6 weeks, 6 months, and 1 year after MVC on 91%, 89%, and 91% of participants

low neighborhood socioeconomic status increases the likelihood of worse MSK pain outcomes after traumatic stress exposures such as MVC, and that this influence is mediated in part via its influence on stress system function.

Imaging of the Head and Neck... Why, When, and How?



Julie

A 28 year-old female presents to your physical therapy clinic with direct access two days after a drivers-side collision. She was the restrained driver stopped at a stop sign when struck by another vehicle traveling approximately 40-mph in a school zone. Her car was pushed into the intersection and struck again on driver's side by a school bus exiting the school parking lot at approximately 5 mph. The driver of the vehicle that struck Julie's car was texting and driving oblivious to traffic in opposing lanes, according to witness reports. She reports her head was turned at the time of the first impact, but she cannot recall her position at the time of the 2nd impact.

More on Julie

This was not a simple rear-end MVC

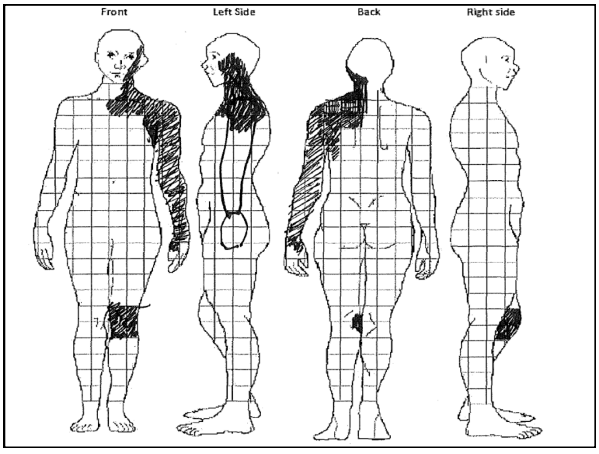
She's able to sit in and walk around your waiting room

She reports immediate onset of neck pain following the second crash

She is tender over all of the c-spine segments but notably at C5/6

What do you do?

Check ROM or go right to Radiography/CT?



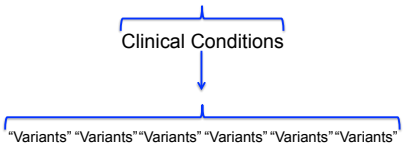
American College of Radiology Appropriateness Criteria

It is a set of consensus developed, evidence-derived guidelines for health care providers to assist in decision making for imaging based on apparent health condition or potential condition requiring investigation.

The stated goals of the ACR-AC are to enhance the quality of patient care and to contribute to the most efficacious use of radiology

Within the ACR-AC structure, patient presentations are categorized dependent upon

- **apparent prior patient history,**
- **probable etiology,**
- **signs and symptoms,**
- **and results of prior imaging.**



Example of clinical condition

Suspected Spine Trauma

There are 14 variants

8 of which directly apply to the cervical spine of adults
and
4 apply to those under the age of 14

Please reference the ACR handout

Suspected Spine Trauma

The criteria for determination of whether imaging for the cervical spine is indicated and the recommended modality are based upon the

- 1) Canadian Cervical Spine Rule (CCSR) and
- 2) the National Emergency X-Ray Utilization Study Low Risk Rule (NEXUS-LRR)
- along with suggestions of neurological or cervical vascular injury.

Rating Scale

The ACR-AC provides a Rating Scale for the Radiologic Procedure:

1,2,3 Usually not appropriate;

4,5,6 May be appropriate;

7,8,9 Usually appropriate

American College of Radiology
ACR Appropriateness Criteria®

Clinical Condition: Suspected Spine Trauma

Variant 1: Cervical spine imaging not indicated by NEXUS or CCR clinical criteria. Patient meets low-risk criteria.

Radiologic Procedure	Rating	Comments	RRL*
X-ray cervical spine	1		☢☢
CT cervical spine without contrast	1	With sagittal and coronal reformat.	☢☢☢
CT cervical spine with contrast	1		☢☢☢
CT cervical spine without and with contrast	1		☢☢☢
Myelography and post myelography CT cervical spine	1		☢☢☢☢
CTA head and neck with contrast	1		☢☢☢
MRI cervical spine without contrast	1		O
MRI cervical spine without and with contrast	1		O
MRA neck without and with contrast	1		O
MRA neck without contrast	1		O
Arteriography cervicocerebral	1		☢☢☢

Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate

*Relative Radiation Level

BUT WHAT ABOUT IMAGING INDICATED BY NEXUS OR CCR?

Variant 2: **Suspected acute cervical spine trauma. Imaging indicated by clinical criteria (NEXUS or CCR). Not otherwise specified.**

Radiologic Procedure	Rating	Comments	RRL*
CT cervical spine without contrast	9	With sagittal and coronal reformat.	☢☢☢
X-ray cervical spine	6	Lateral view only. Useful if CT reconstructions are not optimal.	☢☢
CT cervical spine with contrast	1		☢☢☢
CT cervical spine without and with contrast	1		☢☢☢
Myelography and post myelography CT cervical spine	1		☢☢☢☢
CTA head and neck with contrast	1	See variant 6.	☢☢☢
MRI cervical spine without contrast	1	See variant 3.	O
MRI cervical spine without and with contrast	1	See variant 3.	O
MRA neck without and with contrast	1	See variant 6.	O
MRA neck without contrast	1	See variant 6.	O
Arteriography cervicocerebral	1	See variant 6.	☢☢☢

Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate — *Relative Radiation Level

'CT is king'

In the presence of positive clinical assessment findings derived from the CCSR or the NEXUS-LRR, computed tomography (CT) is the initial imaging modality determined to be "usually appropriate" because of the primary concern for fracture or other destabilizing injury

What about Kids?

For children, these imaging criteria have yet to be thoroughly evaluated, and the position of the ACR-AC is that radiography is preferred in those under 14 years of age, although CT at optimized doses may be applicable

Clinical Condition: Suspected Spine Trauma

Variant 11: Child age <14 years, alert, no neck or back pain, neck supple, no distracting injury.

Radiologic Procedure	Rating	Comments	RRL*
X-ray cervical spine	1		☢☢
CT cervical spine without contrast	1	With sagittal and coronal reformat.	☢☢☢☢
CT cervical spine with contrast	1		☢☢☢☢
CT cervical spine without and with contrast	1		☢☢☢☢
CT thoracic and lumbar spine without contrast	1	Dedicated images with sagittal and coronal reformat or derived from TAP scan.	☢☢☢☢
CT thoracic and lumbar spine with contrast	1		☢☢☢☢
CT thoracic and lumbar spine without and with contrast	1		☢☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

Variant 12: Child age <14 years, alert, no neck or back pain, neck supple, fractured femur.

Radiologic Procedure	Rating	Comments	RRL*
X-ray cervical spine	5	AP, lateral, and open-mouth views. Distracting injury alone is not an indication for thoracolumbar imaging.	☢☢
CT cervical spine without contrast	3	With sagittal and coronal reformat. Should not be first-line evaluation.	☢☢☢☢
CT thoracic and lumbar spine without contrast	3	Dedicated images with sagittal and coronal reformat or derived from TAP scan. If TAP CT is performed for other reasons, then look at the spine.	☢☢☢☢
CT cervical spine with contrast	1		☢☢☢☢
CT cervical spine without and with contrast	1		☢☢☢☢
CT thoracic and lumbar spine with contrast	1		☢☢☢☢
CT thoracic and lumbar spine without and with contrast	1		☢☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

TAP = thorax, abdomen, pelvis body scans

Clinical Condition: Suspected Spine Trauma

Variant 13: Child age <14 years, with known cervical fracture.

Radiologic Procedure	Rating	Comments	RRL*
X-ray thoracic and lumbar spine	9	Not needed if fracture is visualized on TAP scan. Preferred modality.	☢☢☢
CT thoracic and lumbar spine without contrast	9	Dedicated images with sagittal and coronal reformat or derived from TAP scan.	☢☢☢☢
CT thoracic and lumbar spine with contrast	1		☢☢☢☢
CT thoracic and lumbar spine without and with contrast	1		☢☢☢☢
Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate			*Relative Radiation Level

TAP = thorax, abdomen, pelvis body scans

Why not MRI?

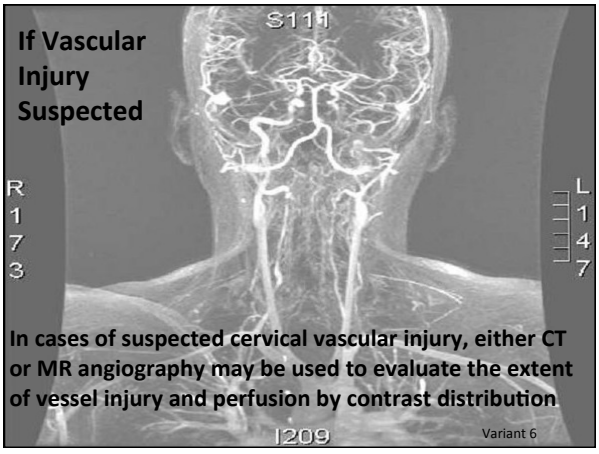
Magnetic resonance imaging (MRI) is recommended as "usually appropriate" in the ACR-AC Suspected Spine Trauma variants in which neurological involvement or overt ligamentous injury are suspected based on clinical examination, emergent CT results, or if the patient is un-evaluable for an extended period of time (e.g. unconscious or obtunded).

See variants 3, 4, 5, 6, 7

What about suspected vascular injury

Clinical suggestions of cervical vascular injury (carotid or vertebral arteries) can include non-specific symptoms such

- neck, occipital, or suboccipital pain or more overt indications of neurological involvement such as vertigo, ataxia, dysarthria, visual field deficit, diplopia, altered cognitive status and Horner's syndrome



In cases of suspected cervical vascular injury, either CT or MR angiography may be used to evaluate the extent of vessel injury and perfusion by contrast distribution

TEST: MRI CERVICAL SPINE WO CONTRAST
[redacted]

MRI CERVICAL SPINE WO CONTRAST

DATE OF EXAM: [redacted]

EXAMINATION: MRI CERVICAL SPINE WO CONTRAST

HISTORY: Neck pain; reported history of trauma, MVC [redacted] Patient reports left-sided neck and arm pain as well as left-sided arm numbness.

COMPARISON: None

TECHNIQUE: Multiplanar multi-sequence imaging of the cervical spine was performed without the use of intravenous gadolinium contrast

FINDINGS:
Straightening of the cervical spine is present. Spinal cord demonstrates a normal high signal intensity in cervical enlargement. No evidence of syrinx. Mild disk desiccation is present at C2-3 through C6-7. At C5-6, small focal central posterior disk protrusion is present which indents the thecal sac, but does not contact the cord. AP dimension of the canal at this location measures 9.5 mm. Mild vertebral disk space narrowing is present at this level. Throughout the cervical spine, no evidence of significant foraminal narrowing. The craniocervical relationship appears maintained. The atlantoaxial relationship appears maintained. There is no evident fracture. No spondylolisthesis. Evaluation of the paraspinal soft tissues shows lack of normal flow void appearance within the left vertebral artery. The right vertebral artery and carotid arteries show flow voids. No evident cervical adenopathy or mass. No abnormal fluid collections.

IMPRESSION:
Straightening of the cervical spine with small central disk protrusion at C5-6, without significant canal or foraminal stenosis.

Lack of normal flow void appearance within the left vertebral artery. A further evaluation with MRA of the neck is recommended.

TEST: MRI CERVICAL SPINE WO CONTRAST
[redacted]

MRI CERVICAL SPINE WO CONTRAST

DATE OF EXAM: [redacted]

EXAMINATION: MRI CERVICAL SPINE WO CONTRAST

HISTORY: Neck pain; reported history of trauma, MVC [redacted] Patient reports left-sided neck and arm pain as well as left-sided arm numbness.

COMPARISON: None

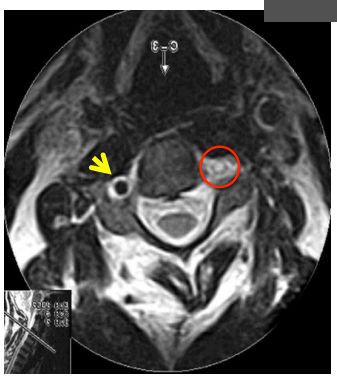
TECHNIQUE: Multiplanar multi-sequence imaging of the cervical spine was performed without the use of intravenous gadolinium contrast

FINDINGS:
Straightening of the cervical spine is present. Spinal cord demonstrates a normal high signal intensity in cervical enlargement. No evidence of syrinx. Mild disk desiccation is present at C2-3 through C6-7. At C5-6, small focal central posterior disk protrusion is present which indents the thecal sac, but does not contact the cord. AP dimension of the canal at this location measures 9.5 mm. Mild vertebral disk space narrowing is present at this level. Throughout the cervical spine, no evidence of significant foraminal narrowing. The craniocervical relationship appears maintained. The atlantoaxial relationship appears maintained. There is no evident fracture. No spondylolisthesis. Evaluation of the paraspinal soft tissues shows lack of normal flow void appearance within the left vertebral artery. The right vertebral artery and carotid arteries show flow voids. No evident cervical adenopathy or mass. No abnormal fluid collections.

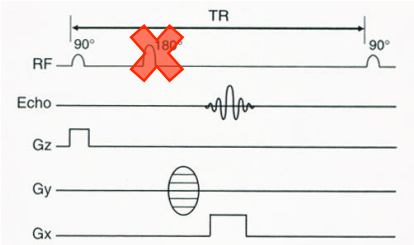
IMPRESSION:
Straightening of the cervical spine with small central disk protrusion at C5-6, without significant canal or foraminal stenosis.

Lack of normal flow void appearance within the left vertebral artery. A further evaluation with MRA of the neck is recommended.

What are Flow Voids? …are they normal?



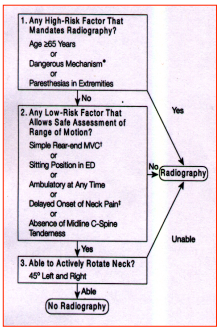
Flow voids on spin echo sequences are caused in part by a lack of refocusing of blood, which is excited by the 90° pulse but not by the 180° pulse.



Key Points

- Published imaging decision guidelines, primarily the ACR-AC, are valuable tools for clinicians assessing patients having experienced acute cervical trauma.
- For patients warranting imaging after cervical trauma, CT is the preferred initial modality.
- MRI may be warranted acutely with suggestions of neurological/arterial involvement or in longer standing cases based on individual patient circumstances in which the soft tissues require detailed assessment

Please refresh your memory on the Canadian C-spine Rule…



Do the same for the NEXUS Criteria

NEXUS criteria for cervical spine radiography.

According to NEXUS low-risk criteria, cervical spine radiography is indicated for trauma patients unless they exhibit *all* of the following criteria:
1. No posterior midline cervical spine tenderness
and
2. No evidence of intoxication
and
3. Normal level of alertness
and
4. No focal neurologic deficit
and
5. No painful distracting injuries.

Julie

You decided it was not appropriate to check ROM

CT was performed and negative for fracture.

However, the clinical presentation continues to unfold with Julie and she is concerned something must be wrong.

It is now 2 months and her signs/symptoms are not getting better…in fact, she reports they are worse

She asks you if a MRI would be appropriate. Is advanced imaging warranted?

N = 1211 ***healthy volunteers***
(20 – 70 years of age)
100 individuals per decade

- *Disc bulging*
- *Spinal cord compression (SCC)*
- *Increased signal intensity in spinal cord*

Nakashima et al., 2015

87% showed disc bulging (increased with age)

74 – 78% (males and females) in their 20's had disc bulges

Only 5.3% had SCC…but this ⇧ with age (> 50 yrs of age - ~60%)

Only 2.3% had ⇧ Signal Intensity of Spinal Cord

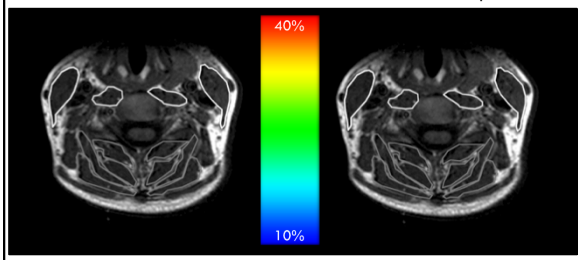
Nakashima et al., 2015

Clinical observations →

Larger magnitude of muscle fatty infiltration (MFI)…in **chronic whiplash**

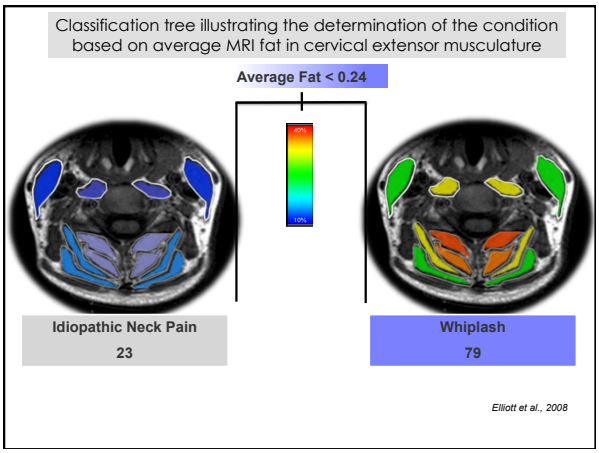
N = 255

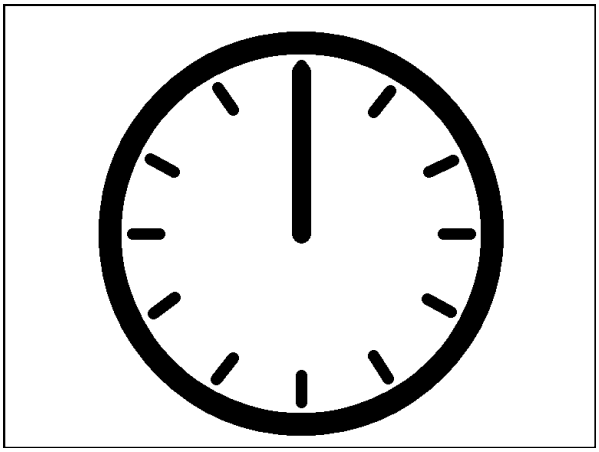
Control | Chronic Whiplash

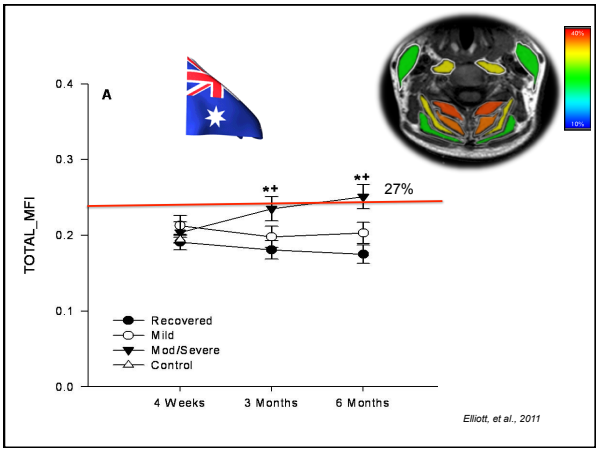


Fat

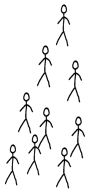
Elliott et al. 2006; 2008; 2009; 2010; 2011; 2014; 2015
Karlsson et al., 2016


Classification tree illustrating the determination of the condition based on average MRI fat in cervical extensor musculature
Average Fat < 0.24
Idiopathic Neck Pain
23
Whiplash
79
Elliott et al., 2008




A
TOTAL_MFI
0.4
0.3
0.2
0.1
0.0
27%
Recovered
Mild
Mod/Severe
Control
4 Weeks
3 Months
6 Months
Elliott, et al., 2011

Association with Initial Pain Intensity



&

Posttraumatic Stress Disorder

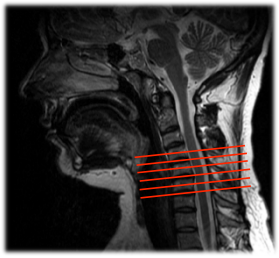
Poor recovery

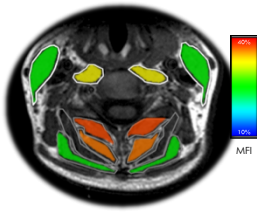
Favorable recovery

Elliott et al. ,2011

...appears unique to those that develop persistent WAD in tandem with known risk factors (older age, high initial pain, PTSD)

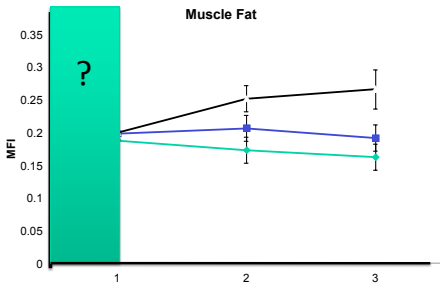
but questions remain…

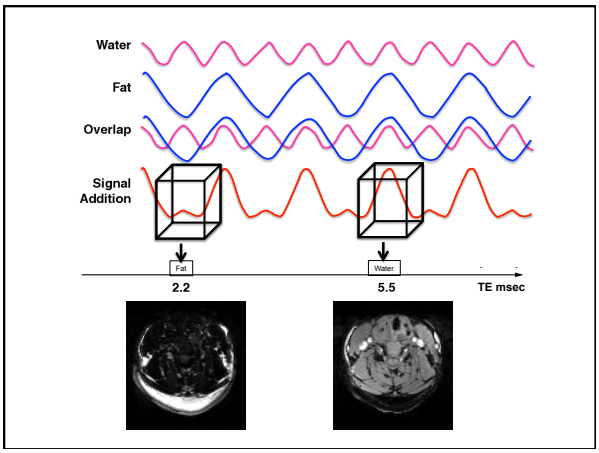




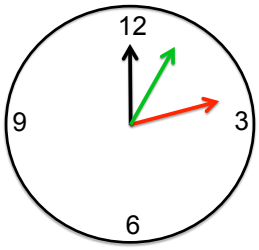
Elliott et al. 2011; 2015

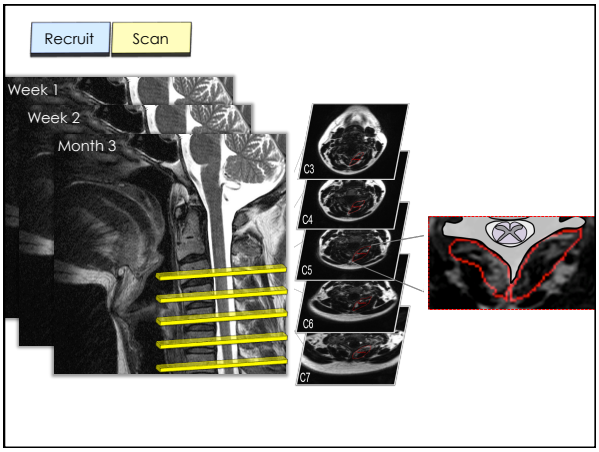
Using advanced (*but available*) imaging applications

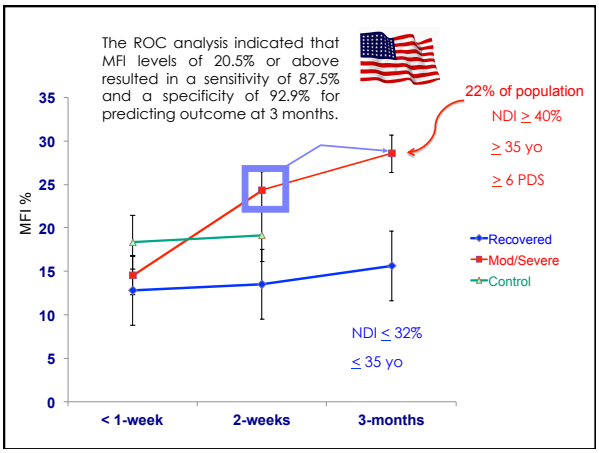


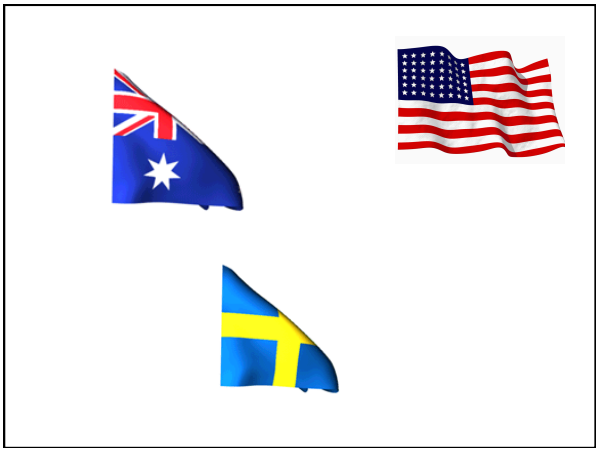


Fat/Water MRI









EJP
European Journal of Pain

ORIGINAL ARTICLE

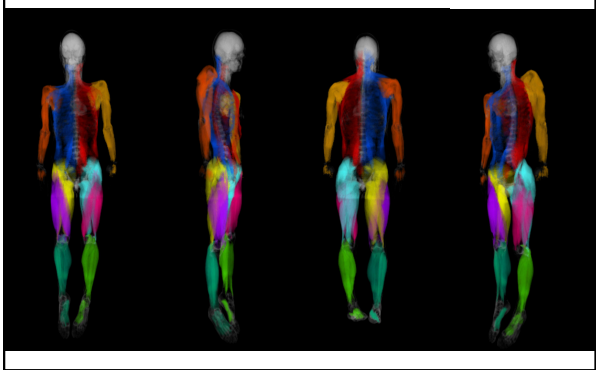
Decreased muscle concentrations of ATP and PCR in the quadriceps muscle of fibromyalgia patients – A ^{31}P-MRS study

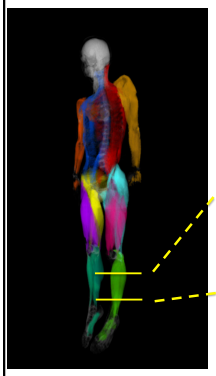
B. Gerdle[1,2], M.F. Forsgren[3,4,5], A. Bengtsson[6,7], O. Dahlqvist Leinhard[3,4,5], B. Sören[6,7], A. Karlsson[3,8], V. Brandejsky[3,4,5], E. Lund[4,5], P. Lundberg[3,4,5]

19 subjects with chronic fibromyalgia and 14 healthy controls

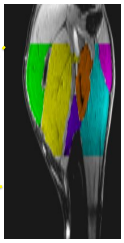
Conclusions: Alterations in intramuscular ATP, PCr and fat content in FMS probably reflect a combination of inactivity related to pain and dysfunction of muscle mitochondria...stress system dysregulation?

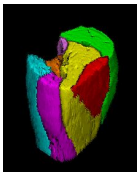
Whole Body Fat/Water Separation





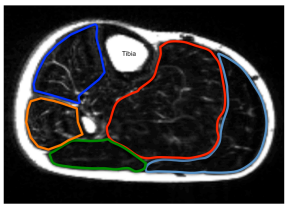
Do changes in extremity muscles also occur in chronic whiplash?

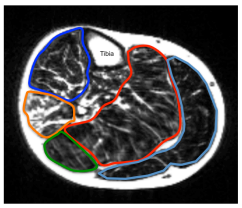




Recovered

Chronic WAD @ 3 months

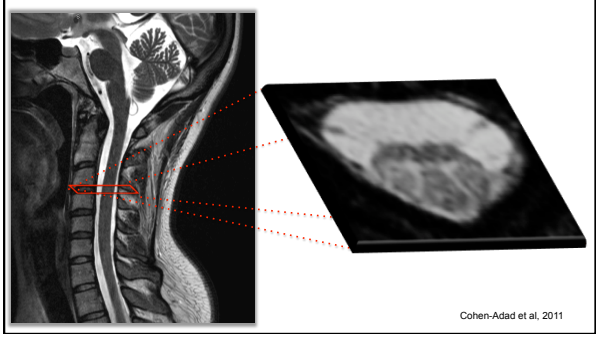




- Anterior Tibialis
- Lateral Compartment
- Gastrocnemius – Lateral Head
- Gastrocnemius – Medial Head
- Soleus

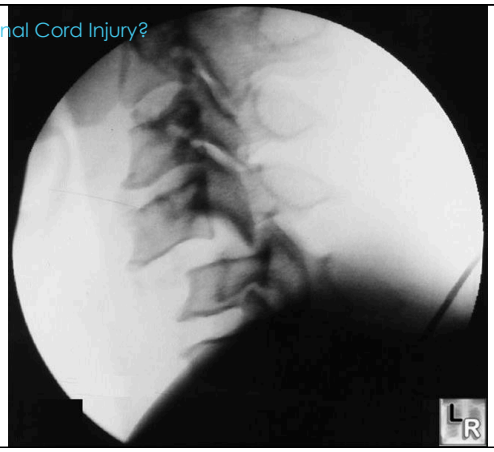
Elliott et al., 2014

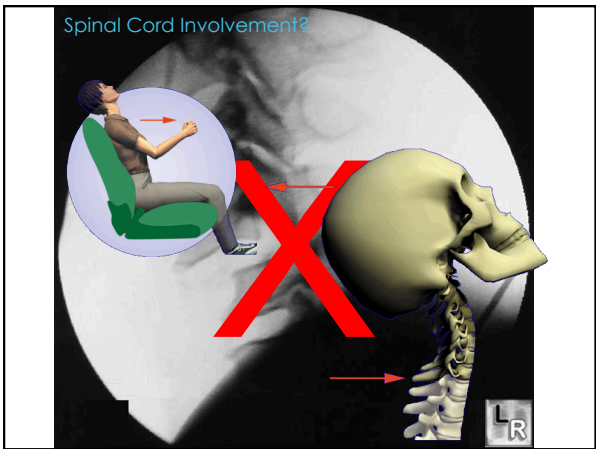
Spinal Cord Imaging

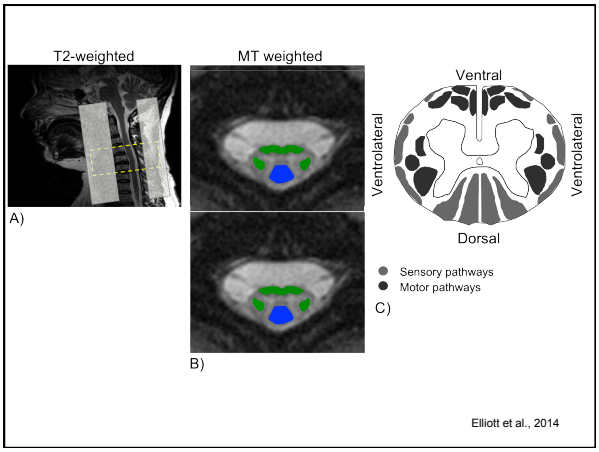


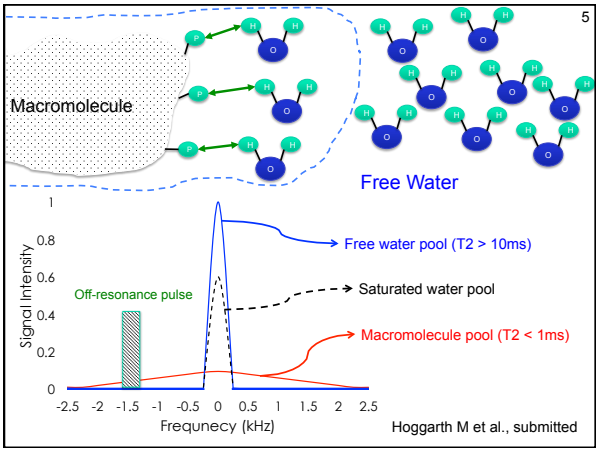
Cohen-Adad et al, 2011

Spinal Cord Injury?



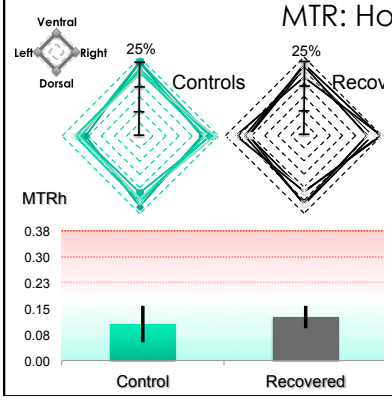

Spinal Cord Involvement?


T2-weighted
MT weighted
A)
B)
Ventral
Ventrolateral
Ventrolateral
Dorsal
Sensory pathways
Motor pathways
C)
Elliott et al., 2014


5
Macromolecule
Free Water
Free water pool (T2 > 10ms)
Saturated water pool
Macromolecule pool (T2 < 1ms)
Off-resonance pulse
Signal Intensity
Frequnecy (kHz)
Hoggarth M et al., submitted

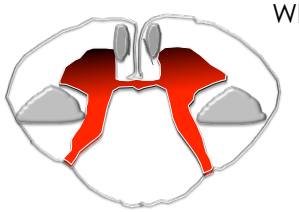
9

MTR: Homogeneity



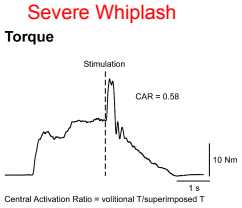
Hoggarth M et al., submitted

What do these changes represent?

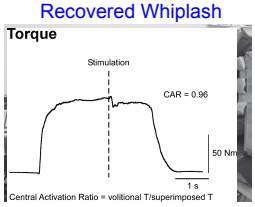


Peripheral or Central?
iSCI?
Disuse-induced atrophy?
Stress?

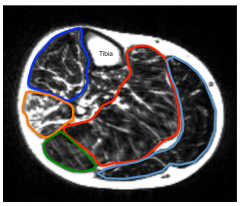
Severe Whiplash



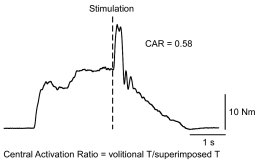
Recovered Whiplash



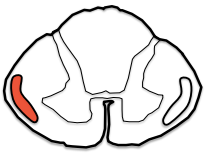
Elliott et al., 2014



Torque

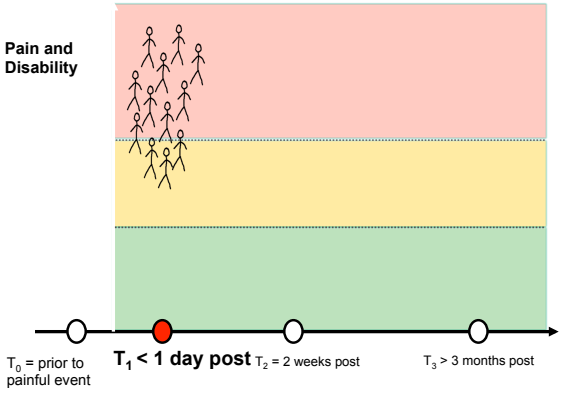


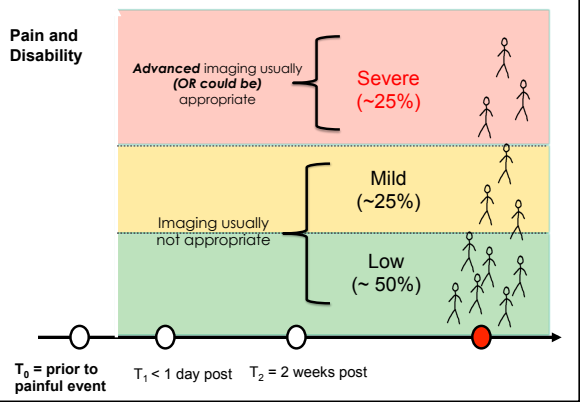
Central Activation Ratio = volitional T/superimposed T



Elliott et al., 2014

Acute Whiplash – imaging at the level of the emergent/primary care provider…
GUIDELINES exist and SHOULD be used…





Julie

You decide to look deeper

Julie demonstrates altered thresholds with PPT (local and distal).

CPM does not attenuate this response

She does not like cold temperatures

Her reflexes may be a bit brisk in her LE's

She has 3-4 beat clonus on the right ankle and demonstrates weakness and co-ordination problems with single-leg heel-raises

measures for helping to characterise the patient at risk for trajectory of chronic pain-related disability…

informing best clinical practice through informed Ax regimens that…

help inform good clinical decision making

(and avoid labelling/stigmatizing)

Bone and Joint Decade, 2008; Dufton et al., 2012

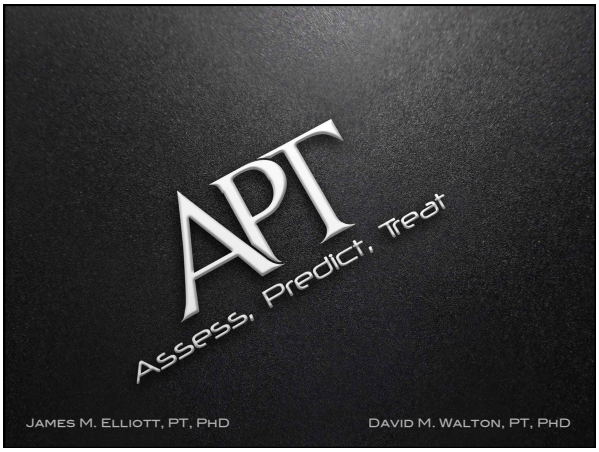
N.B. For Vast majority

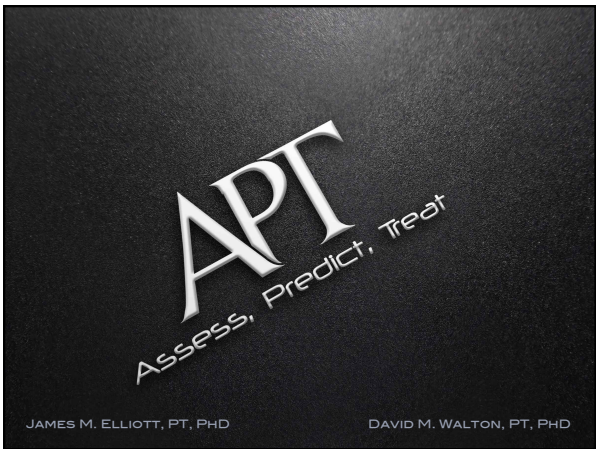
- advise them to allow natural recovery to occur

- circumventing delivery of unnecessary, and costly treatments (Lamb et al., 2012) that have been suggested to contribute to iatrogenic disability (Cote and Soklaridis, 2011)

Lamb et al., 2012; Cote et al., 2011

This work is supported by the National Institutes of Health: R01 - HD079076-01A1





What makes a 'good clinician'?

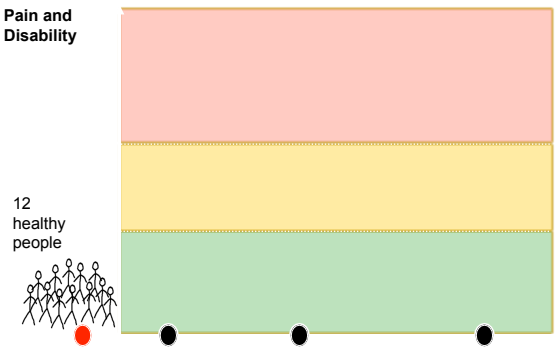
- 519-878-3746

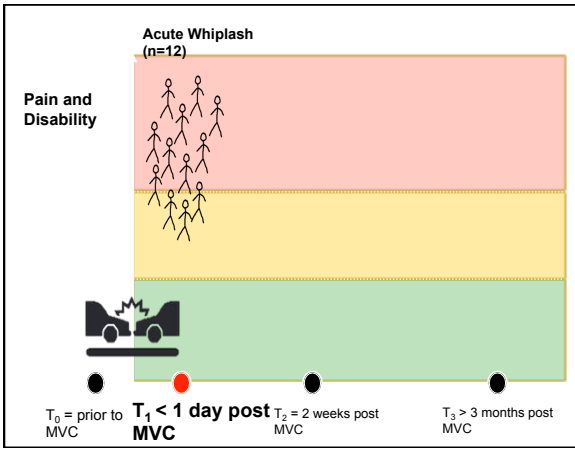
Objectives

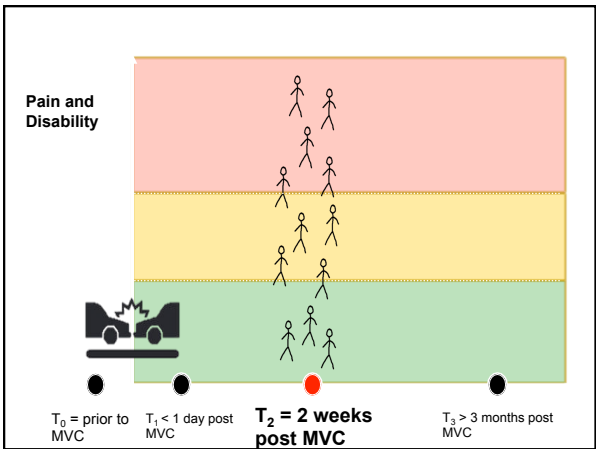
1. Describe the current evidence on consistent risk factors for chronic pain and disability in WAD and LBP
2. Describe common psychological phenomena arising as a result of neck trauma
3. Describe the mechanisms through which the psyche and soma interact to create the clinical picture of neck pain
4. Choose, apply, and interpret clinical measurement tools for prognostic purposes

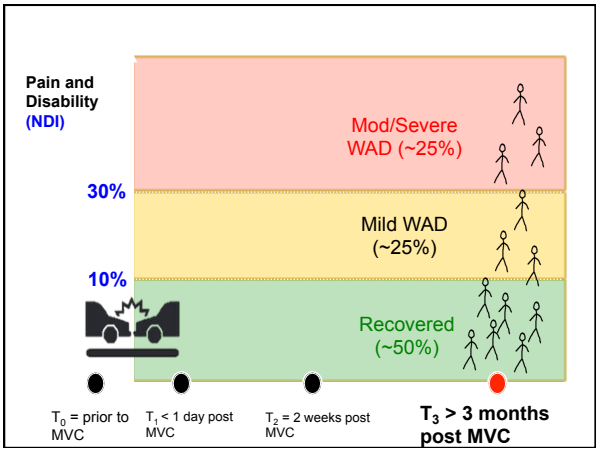
Karen

A 42 year-old female, made an appointment to see her primary care physician four days after an MVC, as her neck pain was not getting any better. She is worried that there might be something seriously wrong with her neck. She had a friend who had a similar injury whiplash last year and is no longer able to exercise for recreation. She is now seeing you on referral from her PCP, 1.5 weeks after the MVC.









Why Prognosis?

Course of Neck pain (WAD)

Sterling et al. *Pain* 2010

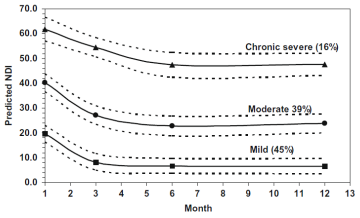


Fig. 1. Predicted NDI trajectories with 95% confidence limits and predicted probability of membership (%). Suggested cut-offs for the NDI are 0–8% (no pain and disability); 10–28% (mild pain and disability), 30–48% (moderate pain and disability), 50–68% (severe pain and disability) and >70% (complete disability) [13].

Course of neck pain (mixed)

Walton et al. *Arch Phys Med Rehabil* 2014

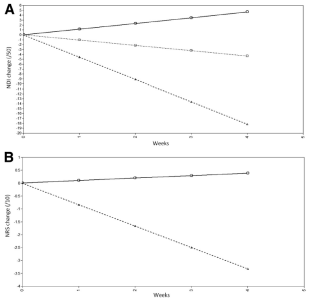


Fig 2 (A) Curves representing the 3 latent class trajectories in the NDI data. Solid line, worsening group (14.5%); dashed line, rapid improvement group (39.6%); dotted line, slow improvement group (65.8%). (B) Curves representing the 2 latent class trajectories in the NRS data. Solid line, stable group (47.9%); dashed line, improving group (52.1%). Note that in both cases, data have been centered around 0 (mean value − baseline mean) for descriptive purposes. See text for descriptions of slope.

Course of neck and back pain – general consensus

- **Majority of recovery occurs within first 3 months**
- **Characterizing as 'good' or 'bad' is probably overly simplistic**
  - **Most indicate at least 3 trajectories**
- ***Relative* minority (<25%) in worst trajectories**
- **Pain, Disability and Duration of symptoms consistent predictors**

The Walton method for preventing chronicity

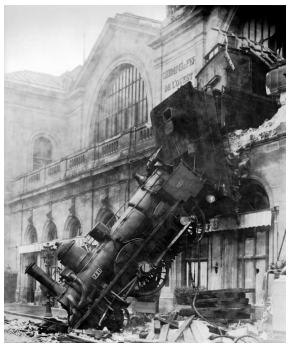
1. **See patients within hours of their event/injury**
2. **Teach them how to fill out disability questionnaires better**
3. **Give them high dose opioids**

What does the evidence say?

Current Evidence for whiplash (Walton et al. 2013)

High confidence of risk factors for chronicity	High confidence of no effect on outcome
High pain intensity (≥ 6/10)	Angular deformity of the neck
High neck-related disability	Impact direction
Post-traumatic stress symptoms	Seating position
Catastrophizing	Awareness of collision
Cold hypersensitivity	Head rest in place
*Mechanical hypersensitivity (distal > local)	Older age
	Vehicle speed

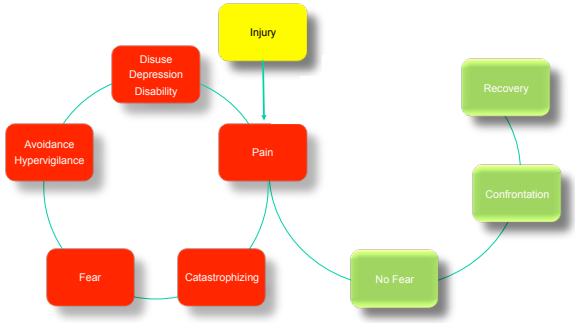
Where does it all go off the rails?



Common and emerging models

- Fear-avoidance model
- Biomechanical model
- Stress-dysregulation model
- Compensation hypothesis
- Neuronal Interference model

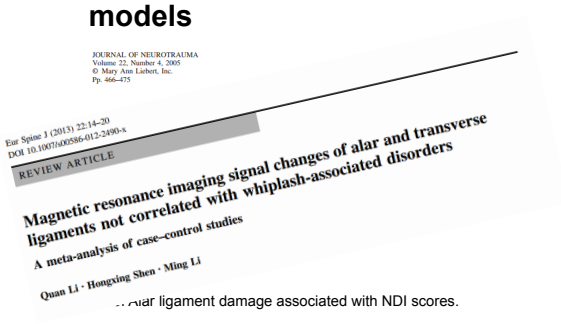
Fear-Avoidance model (Vlaeyen and Linton)



Biomechanical / Structural models

- Bogduk, Lord, Barnsley and Wallis (most of the 1990's)
  - Cervical facet joints are pain generators
  - Denervate the joint (RFN), remove the pain (1997a) and affective distress (1997b)
  - Cautions:
    - <45% of those with chronic WAD met IC
    - Only 12 subjects per arm
    - Pain is primary outcome
    - No one has yet to replicate

Biomechanical / Structural models



...Alar ligament damage associated with NDI scores.

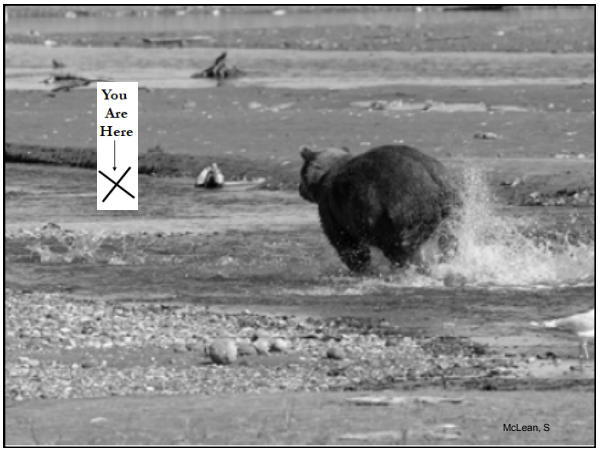
Stress-System Dysregulation

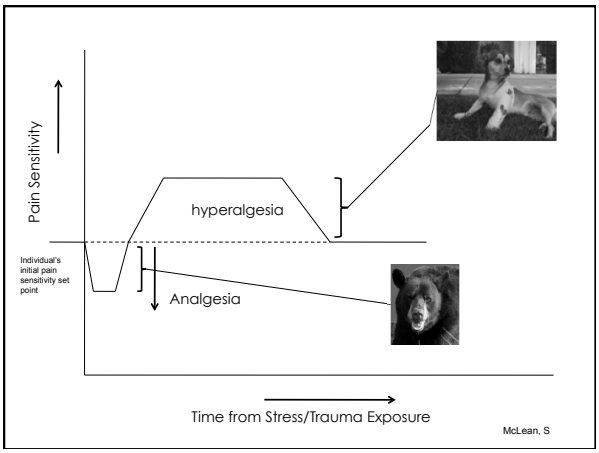
- Trauma is a *stressor* that represents a threat to homeostasis
- Chrousos on Defining Stress:
  - A state in which homeostasis is threatened or perceived to be so.
- Melzack and Loeser 1990:
  - "It is not the duration of pain that distinguishes acute from chronic pain, but rather the inability of the body to restore its physiological functions to normal homeostatic levels."

What 'Perceived Threats' are likely to influence distress?

- Personal safety (injury)
- Safety of others
- Pain and affective distress
- Threat to sense of self
- Financial security
- Social scrutiny
- Confrontation with others

Does the Stress Response Impact Outcomes?





Whiplash
Recovery vs Chronicity

- Higher initial pain and disability[1, 2]
- Posttraumatic stress reaction[1, 3, 4, 5]
- Cold hyperalgesia[1, 3]
- Older age[1,2]

1. Sterling, Jull, Vicenzio, Kenardy & Darnell, 2005
2. Buitenhuis, Spanjer, Fidler, 2003
3. Sterling et al, 2003
4. Buitenhuis et al, 2006
5. Jaspers, 1998

Post-Traumatic Stress

Exposure to traumatic event
involving actual or threatened death or serious injury

Fear, helplessness or horror

(American Psychiatric Association, 2000)

Post-Traumatic Stress

Symptoms from each of three domains:

A. Re-experiencing/Intrusion
- Distressing Thoughts or images of the event, dreams, feelings of event recurring, anxiety in related situations

B. Avoidance
- Avoidance of situations that are similar or connected to the traumatic event.
- Avoidance of thoughts of the event

C. *Physiological arousal*
- Sleep disturbance, hypervigilance, exaggerated startle, irritability

Duration > 1 month

(American Psychiatric Association, 2000)

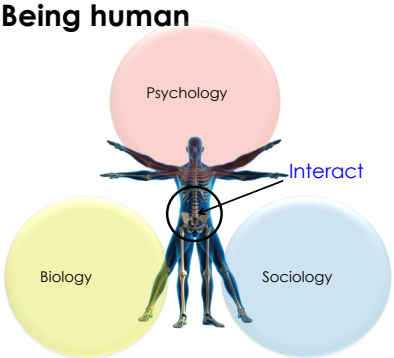
PTSD & Whiplash

Higher rates of PTSD in Whiplash patients[1,2,3].

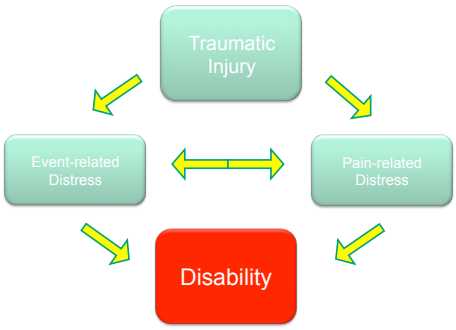
Overlapping epidemiologic and clinical features[1]

May involve stress system dysregulation[4]
- Cortisol abnormalities in both Whiplash[4,5] and PTSD[6]
- Sensory hypersensitivity (lower pain thresholds)[7]
- Impaired sensory nervous system functioning [7]

Being human

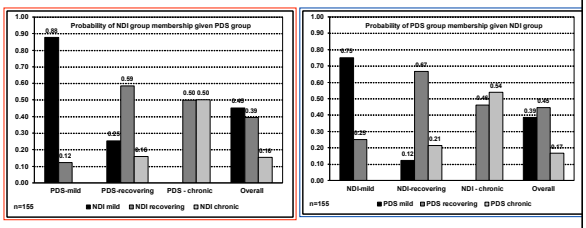


Psychological Reactions to Traumatic Injury -- Working Model



Kenardy & Sterling, 2008

PTSD & Pain: Mutual Maintenance



Sterling, Hendrikz, Kenardy 2010, Pain

Criteria for probable PTSD diagnosis (PDS)

1+ month post MVA

Re-experiencing the event

(eg, psychological distress on exposure to reminders)

Avoidance of reminders of the event

Hyperarousal

Stress-System Dysregulation

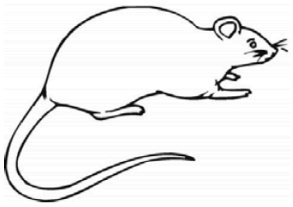
- ***If*** **Trauma is a stressor**
- ***and*** **Stress influences physiology**
- ***Then*** **Trauma should be viewed from more than purely biomechanical / pathoanatomical perspectives**

QUESTIONS…

Is tissue injury/damage the ONLY component of a MVC that can cause allodynia and hyperalgesia?

OR… can the stress system regulate psychological and neurosensory processing of allodynia and hyperalgesia?

McLean, S



McLean, S

Intermittent, repeated restrained stress models in rats

Restrained in cylinder 4x/week for 5 weeks

50% reduction in mechanical thresholds from baseline
&
200% increase in cold allodynia

Bardin et al., 2009

10-20 minutes of inescapable, non-painful, swim stress for 3 days

Subsequent development of hyperalgesia to both thermal (hot plate) and chemical (formalin) stimuli

Quintero et al., 2000, 2003

McLean, S

So far….

Stress system capable of causing allodynia and hyperalgesia

Adrenergic system may contribute to pain outcomes…

Question…?

McLean, S

American Pain Society
RESEARCH
EDUCATION
TREATMENT
ADVOCACY
PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 12, No 1 (January), 2011: pp 101-107
Available online at www.sciencedirect.com

Catechol *O*-Methyltransferase Haplotype Predicts Immediate Musculoskeletal Neck Pain and Psychological Symptoms After Motor Vehicle Collision

Samuel A. McLean,[*,†,‡] Luda Diatchenko,[†] Young M. Lee,[*,†] Robert A. Swor,[§] Robert M. Domeier,[¶] Jeffrey S. Jones,[#] Christopher W. Jones,[*,‡] Caroline Reed,[*] Richard E. Harris,[*] William Maixner,[†] Daniel J. Clauw,[*] and Israel Liberzon[‖]

McLean, S

Evidence – genetics?

- Bortsov et al. 2014a & b:
  - A specific SNP of the *catechyl-o-methyl-transferase* (COMT) gene predicts immediate and 6-week neck pain and psych symptoms after MVC.
  - COMT is an enzyme that metabolizes catecholamines (epinephrine, norepinephrine, dopamine)
  - Low activity COMT + high distress (++ catecholamines) = greater pain and psych distress

Evidence – genetics? (cont'd)

- Bortsov et al. 2013.
  - Another SNP in the *FK Binding Protein-5* (FKBP5) gene associated with persistent pain after MVC
  - FKBP5 encodes a key glucocorticoid (e.g. cortisol) receptor chaperone protein
  - Ineffective FKBP5 gene = more circulating glucocorticoids (cortisol)

Effects of hypercortisolism

- Muscle catabolism
- Widespread pain
- Cognitive interference
- Sleep disturbance
- Interrupted digestion
- Disrupted inflammation through effects on inflammatory cytokines
  - Disordered tissue repair and healing

Do Patients with Traumatic Neck Pain Exhibit Elevated Markers of Stress?

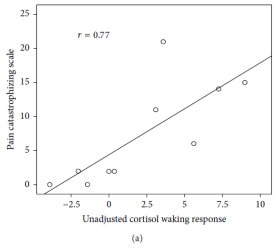
McLean, S

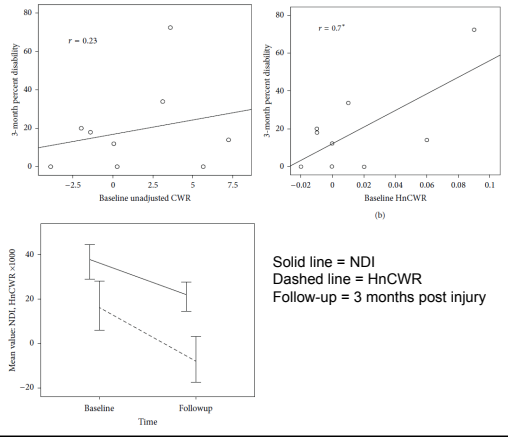
Research Article

Hair-Normalized Cortisol Waking Response as a Novel Biomarker of Hypothalamic-Pituitary-Adrenal Axis Activity following Acute Trauma: A Proof-of-Concept Study with Pilot Results

Hindawi Publishing Corporation
Pain Research and Treatment
Volume 2013, Article ID 876871, 9 pages
http://dx.doi.org/10.1155/2013/876871

David M. Walton,[1] Joy C. MacDermid,[2] Evan Russell,[3] Gideon Koren,[3,4,5] and Stan Van Uum[6]





Solid line = NDI
Dashed line = HnCWR
Follow-up = 3 months post injury

McLean et al. *Psychosomatic Medicine* 2005

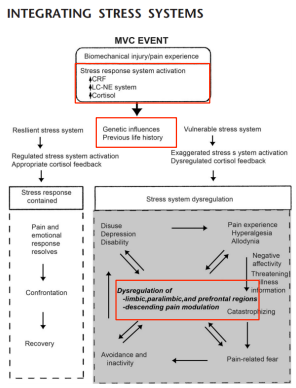
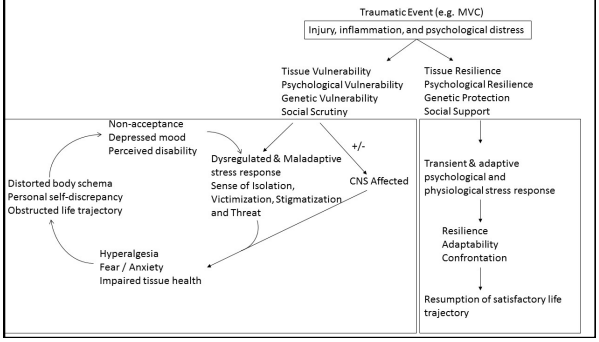


Figure 2. Amendment to Vlaeyen's cognitive-behavioral model of chronic pain pathogenesis, incorporating the hypothesized role of the stress response system in the development of aberrant pain and/or emotional processing after motor vehicle collision.



Stress-System Dysregulation

- **Summary**
  - **Trauma is a stressor**
  - **The magnitude of distress is influenced by several factors, personal and env.**
  - **Distress leads to activation of stress-regulatory pathways**
  - **High distress + genetic vulnerability = prime candidate for pain and disability**

The Compensation Hypothesis

- Discuss the implications of this statement:
  - "Patients in active compensation or litigation proceedings are more likely to report persistent pain and disability than are those not involved in compensation or litigation."

What *is* the relationship between disability and compensation?

- Ample evidence (empirical and anecdotal) of a relationship between the two
- Correlation does NOT imply causation
- Is compensation bad for health?

  OR
- Is bad health good for compensation?

Recent evidence

- Sterling et al. 2010
  - 155 subjects followed for 1 year
  - Comp. claim affected mild and moderate trajectories but not severe trajectories
  - Those with severe problems are likely to have severe problems regardless of compensation status
- Spearing et al. 2012
  - SR of 16 studies on the topic
  - 9 showed sig. neg. association b/w health and compensation, 7 showed none
  - None provided convincing evidence of cause-and-effect
  - Consistent problems with inconsistent outcomes

(sorry for the poor form)

NCBI Resources How To

PubMed.gov US National Library of Medicine National Institutes of Health — PubMed — Advanced

Display Settings: Abstract — Send to:

J Law Med. 2012 Sep;20(1):82-92.

Are people who claim compensation "cured by a verdict"? A longitudinal study of health outcomes after whiplash.

Spearing NM[1], Gyrd-Hansen D, Pobereskin LH, Rowell DS, Connelly LB.

Author information

Abstract

This study examines whether the lure of injury compensation prompts whiplash claimants to overstate their symptoms. Claim settlement is the intervention of interest, as it represents the point at which there is no further incentive to exaggerate symptoms, and neck pain at 24 months is the outcome of interest. Longitudinal data on neck pain scores and timing of claim settlement were regressed, controlling for the effect of time on recovery, to compare outcomes in claimants who had and had not settled their compensation claims. The results show clearly that removing the financial incentive to over-report symptoms has no effect on self-reported neck pain in a fault-based compensation scheme, and this finding concurs with other studies on this topic. Policy decisions to limit compensation in the belief that claimants systematically misrepresent their health status are not supported empirically Claimants do not appear to be "cured by a verdict".

IASP — PAIN® 155 (2014) 309–321 — PAIN®

www.elsevier.com/locate/pain

Incidence and predictors of neck and widespread pain after motor vehicle collision among US litigants and nonlitigants

CrossMark

Samuel A. McLean [a,b,c,*], Jacob C. Ulirsch [a,b], Gary D. Slade [d], April C. Soward [a,b], Robert A. Swor [e], David A. Peak [f], Jeffrey S. Jones [g], Niels K. Rathlev [h], David C. Lee [i], Robert M. Domeier [j], Phyllis L. Hendry [k], Andrey V. Bortsov [a,b], Eric Bair [d]

(n=948) presenting to the emergency department (ED) in the hours after MVC

Six weeks later, participants were interviewed regarding pain symptoms and asked about their participation in MVC-related litigation (83% were non-litigants).

Among individuals not engaged in litigation, persistent pain 6weeks after MVC was common:

28% had moderate or severe neck pain,

13% had widespread pain,

4% had fibromyalgia-like symptoms.

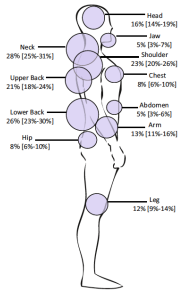
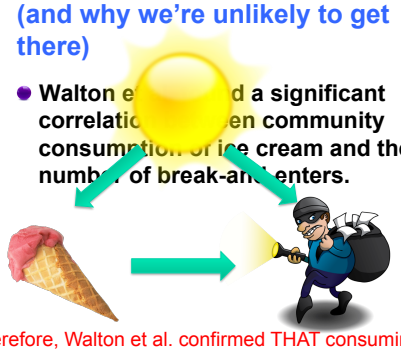


Fig. 2. Percentage of study participants with moderate or severe musculoskeletal pain (NRS ≥4) according to body region 6 weeks after motor vehicle collision (study participants not engaged in litigation only). NRS = numeric rating scale.

Proving Cause-and-Effect (and why we're unlikely to get there)

- Walton e[illegible]d a significant correlatio[illegible]en community consumption of ice cream and the numbe[illegible] of break-and-enters.



Therefore, Walton et al. confirmed THAT consuming ice cream leads to breaking and entering.

Bradford-Hill criteria for cause-and-effect

1. **Dose-response**
   - More compensation -> more disability
2. **Strength of association**
   - Needs to be strong enough to be convincing
3. **Temporality**
   - Compensation *always* occurs before disability
4. **Reversibility**
   - Remove compensation, remove disability
5. **Consistency**
   - Findings 1-4 are consistent across studies
6. **Biologic Plausibility**
   - Does it even make sense?

Is it all secondary gain?

Cortisol and adrenalin are both released in response to stress and are pronociceptive

Gut microflora dysbiosis is commonly seen in chronic stress – microflora can affect behaviour (at least in rodents)

Social rejection, loss of autonomy and personal self-doubt are strong predictors of depression

"If you have to prove you are ill, you can't get well." – Hadler 1996

The evidence as I see it…

Is there an association between compensation and reports of disability?

Yes.

Does that mean compensation *causes* disability, or are the more disabled more likely to seek compensation?
Don't know, in some ways probably both.

Is it all just malingering/exaggeration/ secondary gain/ faking / choose your own degrading term?
No.

So where does it go off the rails?

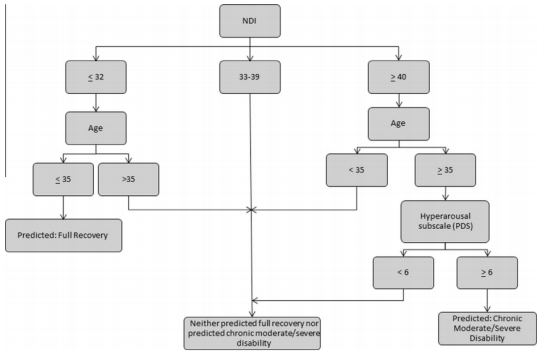
- **There are several things that *could* go awry, but causation still largely unproven**
- **Where's the chicken? Where's the egg?**
- **Favour an integrated biopsychosocial understanding**
- **The search for a single risk factor likely misleading**

Predisposed does not mean predestined

Clinical risk tools

- CPR for whiplash (Sterling et al.)
- StarTBACK tool
- Orebro Musculoskeletal Pain Questionnaire
- Quantitative Sensory Testing
- Emerging evidence:
  - Traumatic Injuries Distress Scale
  - Injustice Experience Questionnaire

Ritchie CPR (2013)



PAIN® 154 (2013) 2198–2206

PAIN®

www.elsevier.com/locate/pain

Derivation of a clinical prediction rule to identify both chronic moderate/ severe disability and full recovery following whiplash injury

Carrie Ritchie*, Joan Hendrikz, Justin Kenardy, Michele Sterling

Centre of National Research on Disability and Rehabilitation Medicine (CONROD), University of Queensland, Brisbane, Australia

An increased probability of developing chronic mod/sev disability was predicted in the presence of
1) older age (≥ 35);
2) initial higher levels of NDI (≥ 40%)
3) hyperarousal symptoms on PDS (≥ 6) – PPV 71%
- Sn

The probability of full recovery was increased in with initially lower levels of neck disability – PPV 71%
- Sn 45.3 & Sp - 84.5

[RESEARCH REPORT]

CARRIE RITCHIE, PhD[a] • JOAN HENDRIKZ, PGradStats[a] • GWENDOLEN JULL, PhD[a]
JAMES ELLIOTT, PhD[a] • MICHELE STERLING, PhD[b]

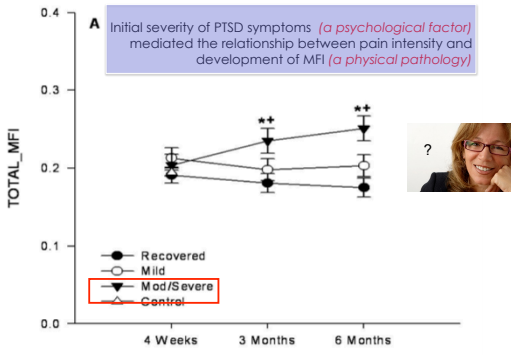
External Validation of a Clinical Prediction Rule to Predict Full Recovery and Ongoing Moderate/Severe Disability Following Acute Whiplash Injury

1) older age (≥ 35);
2) initial higher levels of NDI (≥ 40%)
3) hyperarousal symptoms on PDS (≥ 6) - PPV 91%
Sn – 43 & Sp 98

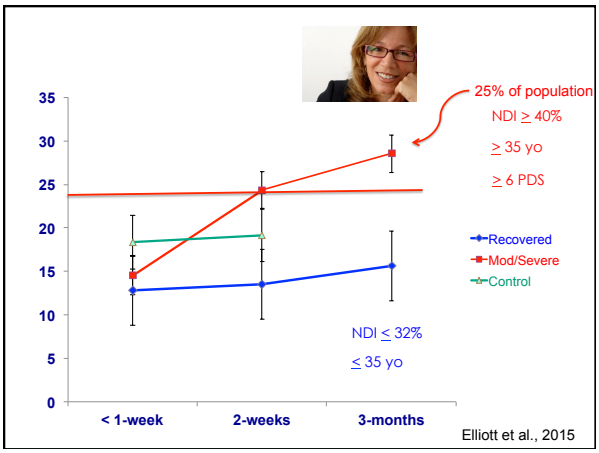
Furthermore, the probability of full recovery **increased to** 80% for younger individuals with initially lower levels of neck disability

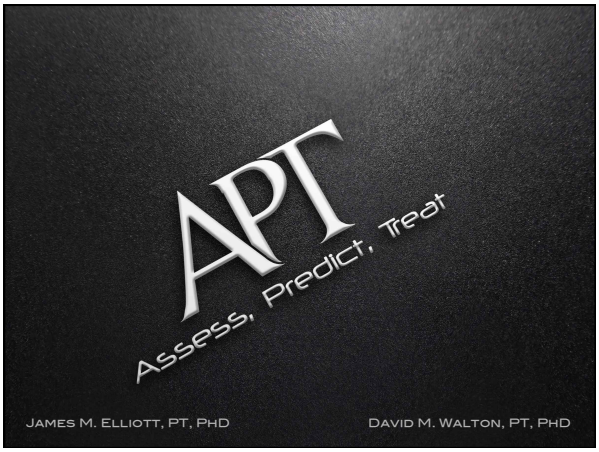
Sn – 54 & Sp 86

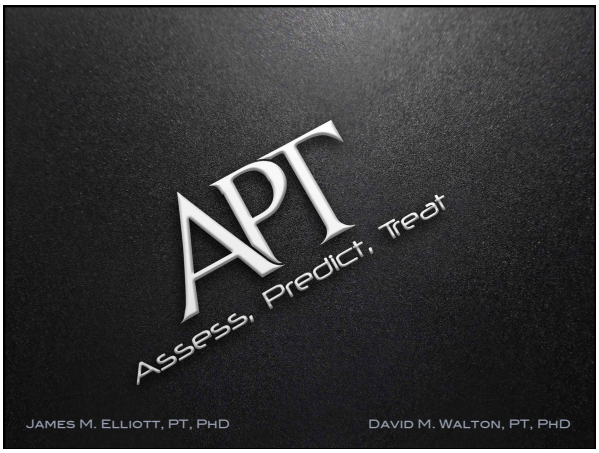
Ritchie et al., JOSPT 2015



Elliott et al., 2011


35
30
25
20
15
10
5
0
< 1-week
2-weeks
3-months
25% of population
NDI ≥ 40%
≥ 35 yo
≥ 6 PDS
Recovered
Mod/Severe
Control
NDI ≤ 32%
≤ 35 yo
Elliott et al., 2015


APT
Assess, Predict, Treat
JAMES M. ELLIOTT, PT, PHD
DAVID M. WALTON, PT, PHD



Objectives

1. Discuss the challenges of managing traumatic neck pain
2. Critically review the evidence supporting different management strategies
3. Practice, apply, and provide feedback on different management strategies

Some challenges of managing WAD

1. Depending on how it's measured, recovery rates range from 20-80% after 12 months, leaving a significant number with persistent problems (Walton 2013, Carroll 2013)
2. Little to no consistent evidence of 'bony or soft-tissue injuries' (Nordin 2008)
3. A number of psychosocial factors (e.g., coping, expectations, anxiety and depression) have been identified as prognostic of WAD recovery (Walton 2013)
4. But, little to no consistent evidence to support early active intervention over single sessions of advice and education (Lamb 2013, Faux 2015)

A General Approach

- Assume 3 consistent trajectories of acute WAD:
  1. Rapid, complete recovery ~20-30%
  2. Slow recovery ~55-65%
  3. Non-recovery, chronic disability ~10-15%
- Let Grp 1 recover with monitoring from a distance
- Early targeted intervention with multidisciplinary care more appropriate for Grp 3
- What to do with Grp 2?
- **In all cases the first 6-12 weeks are crucial**

Grp 1: Low Risk, rapid & complete recovery expected

- Probably most suited to the results of RCTs that endorse a single session of advice and education (e.g. Lamb 2013, Faux 2015)
- Avoid 'over-treatment' here – if recovery is expected, let it happen
- Follow-up after 1 month to ensure recovery is occurring as expected. **Remember that trajectories are not fixed.**

Grp 2: Unclear Risk, slow or incomplete recovery expected

- The hardest group for which to create an early intervention plan
- Requires additional assessment and evaluation – treatment should flow naturally based on evaluation findings
- Probably suitable for conservative rehab with low-level pharmaceuticals but should monitor closely to ensure recovery is occurring

Grp 3: High Risk, Non-recovery and chronic disability expected

- As the primary care provider, your job is to communicate effectively with rest of team
- Keep in mind: **Predisposed does not mean predestined**
- Treatment should be informed by assessment, but will usually include physical rehabilitation (addressing fear of movement as much as tissue damage), pharmaceutical or other symptom management, psychology, possibly social work or other assistance navigating life roles

Summary for Management of Acute WAD

- No 'one size fits all' approach
- Treatment should be informed by sound assessment and evaluation
- If low risk, let recovery occur naturally
- If high risk, document assessment findings well, identify key areas to target, and get the right team on board
- If moderate risk, evaluate further to identify treatment targets, monitor frequently, be prepared to adjust up/down as necessary
- First 6-12 weeks are crucial

Recent Evidence Summary

- Consider:
  - Advice and education to stay active
  - Up to 2 sessions of thoracic manipulation
  - Multimodal care (e.g. exercise, mobs, advice, therapeutic modalities)
  - TENS
  - OTC NSAIDs or simple analgesics
  - Semi-rigid collar for Gr. III problems (radicular)
- From: ICON, OPTIMA, APTA CPG, Cochrane COG

The Phenomenon of Chronic WAD

- Approx. 50% of people will continue to report some degree of problem 12 months post-WAD
- 20% will report severe disability or interference
- The mechanisms of chronicity are unclear but pictures are emerging:
  - Maladaptive beliefs and cognitions (Sullivan)
  - Stress system dysregulation (Walton, McLean)
  - Genetic vulnerability (Bortsov)
  - Injury to the central nervous system (Elliott)

What Does the Evidence Say?

- Similar to acute WAD, several reviews recently conducted (Teasell '10, ICON '13, OPTIMA '15, APTA CPG '16)
- More work done here, but still few consistent findings
- The evidence highlights the heterogeneity in WAD – almost all treatments offer benefit for some, no benefit for others

What Does the Evidence Say?

- Low-to-moderate confidence that:
  - Exercise programs offer some benefit, but the nature of those programs are unclear (all reviews)
  - Qigong or combined strength/ROM/flexibility programs are more beneficial than waitlist (OPTIMA)
  - Iyengar yoga is more effective than home exercise (OPTIMA)
  - 1 session of cervical manipulation is similar to kinesiotape for general neck pain (OPTIMA)

What Does the Evidence Say (cont'd)?

- Low-to-moderate confidence that:
  - Acupuncture / needling?
    - OPTIMA: No benefit vs. placebo
    - ICON: Moderate evidence of benefit vs. placebo
  - Intermittent traction short-term benefit for general neck pain (ICON)
  - Mind-body based interventions offer short-term benefit (ICON)
  - 10 weeks of 2 min/day scapula-thoracic endurance training offers some benefit (ICON)

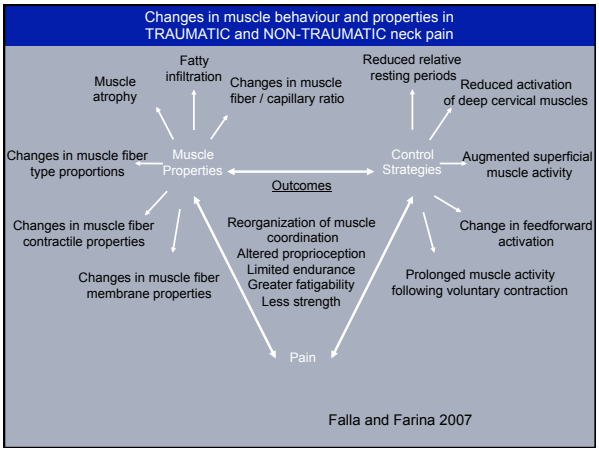
In the Face of Uncertainty, a Proposed Framework

- Must start with comprehensive assessment and evaluation of key contributors
  - Conduct a physical 'biomechanical' assessment, but be prepared for "inconsistent" findings
  - Explore context: support, doubt, scrutiny
  - Explore beliefs and cognitions
  - Explore signs/symptoms of central or peripheral nervous system dysfunction
- Construct a visual of the pain experience – where are the biggest contributors?
- Treatment should then flow naturally on a patient-by-patient basis

IMPLICATIONS FOR ACTIVITY/EXERCISE ?

Is Muscle Function/Coordination Disturbed in Chronic Neck Pain?

- Evidence indicates yes
- Elliott: consistent evidence of increased muscle fatty infiltration in non-recovered *traumatic* neck pain
- O'Leary: differential response in muscle structure across idiopathic and traumatic neck pain. Preliminary evidence suggests targeted exercise can influence structure/function of muscle (increased strength and meaningful reductions in pain-related disability)
- Treleaven: Evidence of increased joint repositioning error
- Jull/Falla: Consistent evidence of shift from deep to superficial muscle activation patterns



Eur J Appl Physiol (2006) 98:423–449
DOI 10.1007/s00421-006-0312-8

REVIEW ARTICLE

Influence of sympathetic nervous system on sensorimotor function: whiplash associated disorders (WAD) as a model

Magda Passatore · Silvestro Roatta

RECALL

Converging and diverging evidence available indicating the presence of a peripheral lesion following whiplash injury (Curatolo et al., 2011; Sterling et al., 2011)

Clinical Neurophysiology 123 (2012) 1403–1408

Contents lists available at SciVerse ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

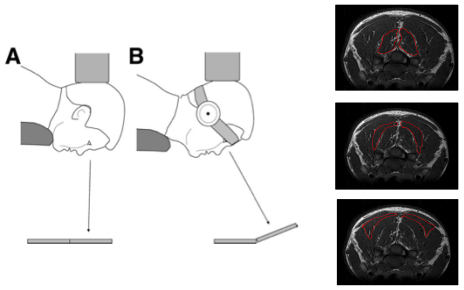
Chronic trauma-induced neck pain impairs the neural control of the deep semispinalis cervicis muscle

Jochen Schomacher [a], Dario Farina [b], René Lindstroem [a], Deborah Falla [b,c,*]

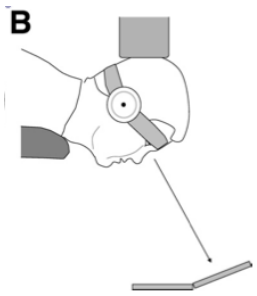
[a] Center for Sensory-Motor Interaction (SMI), Department of Health Science and Technology, Aalborg University, Denmark
[b] Department of Neurorehabilitation Engineering, Bernstein Focus Neurotechnology (BFNT) Göttingen, Bernstein Center for Computational Neuroscience, University Medical Center Göttingen, Georg-August University, Göttingen, Germany
[c] Pain Clinic, Center for Anesthesiology, Emergency and Intensive Care Medicine, University Hospital Göttingen, Göttingen, Germany

Reduced activation of the semispinalis cervicis may impact support of Cervical spine, influencing maintenance & *perpetuation* of symptoms

Best Exercise Targeting The Cervical Extensors?



Elliott et al. (2011) Archives of Physical Medicine and Rehabilitation



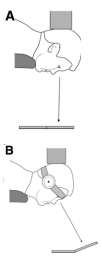
Heightened activity of Semispinalis Capitis muscle

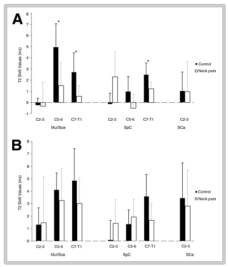
Supporting it's role as primary head/neck extender

Elliott, O'Leary, Cagnie, Durbridge, Danneells, Jull et al (2010) Arch Phys Med Rehab

Is Muscle Function Disturbed Chronic Neck Pain?

Cervical extensors

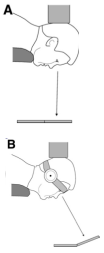


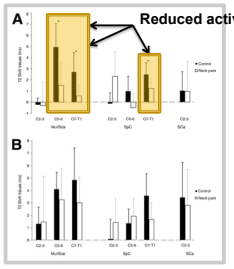


O'leary et al. (2011) Archives of Physical Medicine and Rehabilitation

Muscle Function *is* Disturbed in Chronic Neck Pain?

Cervical extensors





O'leary et al. (2011) Archives of Physical Medicine and Rehabilitation

In keeping with others…

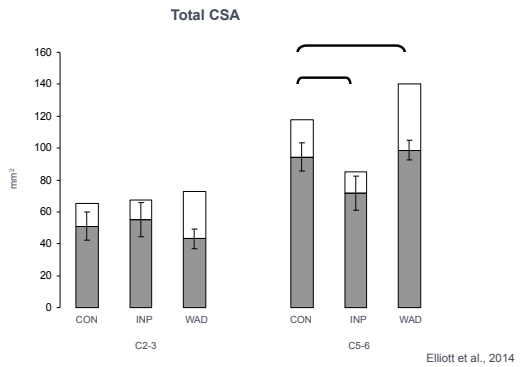
exercises targeting the neck extensors may be relevant to include in the management of patients with neck pain

-Improve health and function…

O'Leary et al.; Ylinen et al.; Schomacher et al.

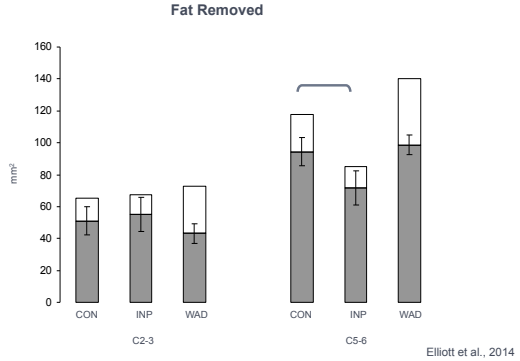
What about muscle structure?

Cervical Multifidus



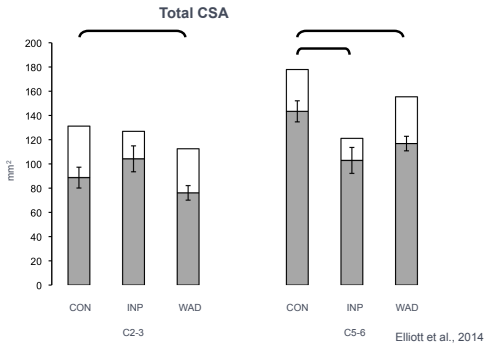
Elliott et al., 2014

Cervical Multifidus



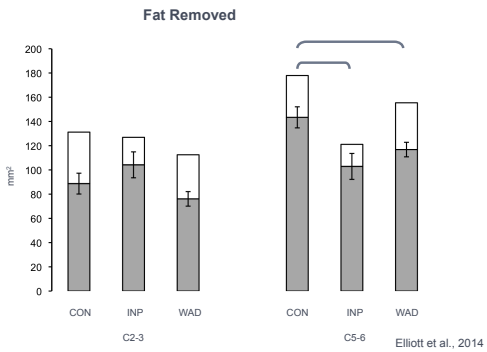
Elliott et al., 2014

Semispinalis Cervicis



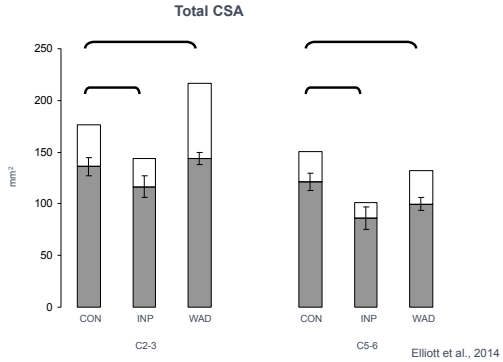
Elliott et al., 2014

Semispinalis Cervicis



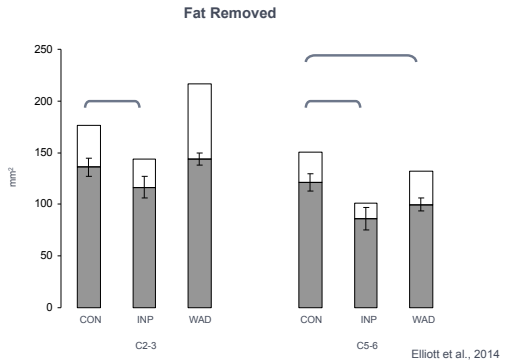
Elliott et al., 2014

Semispinalis Capitus



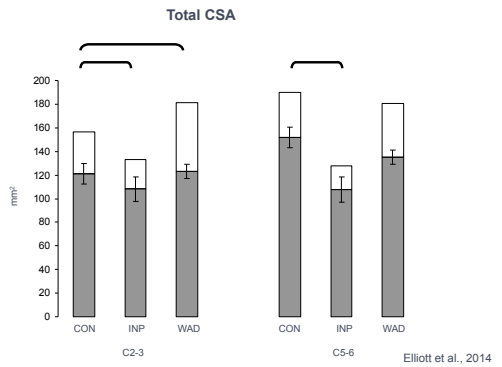
Elliott et al., 2014

Semispinalis Capitus



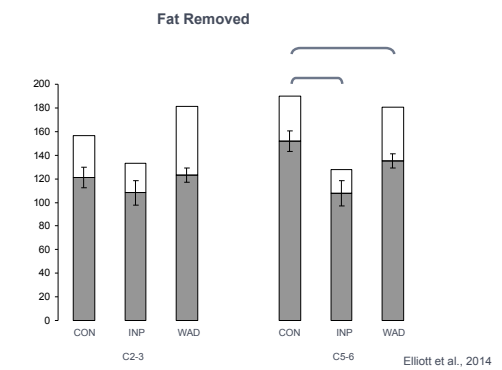
Elliott et al., 2014

Splenius Capitus



Elliott et al., 2014

Splenius Capitus



Elliott et al., 2014

Summary

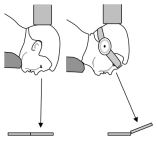
When fat removed 93% of the examined muscles in patients with WAD were similar to or significantly smaller than that those observed in healthy individuals.

In stark contrast when fat not removed 80% of the muscles in WAD participants were larger than or similar to the healthy individuals

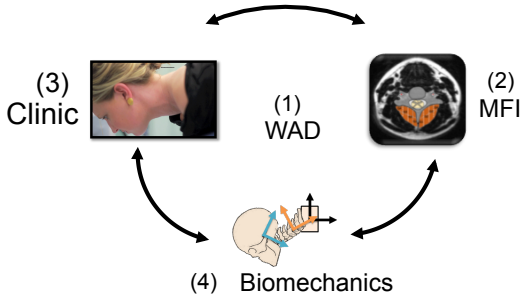
The removal of the fat from the CSA measurement did not alter the findings between idiopathic neck pain and healthy controls which was expected because of the similar levels of MFI in these 2 groups

Summary

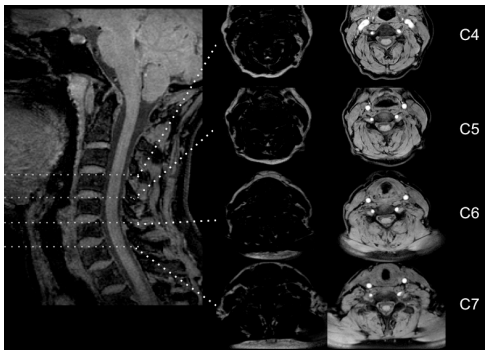
Morphological changes within the neck muscles of the patients with WAD (atrophy and fat infiltrates) and idiopathic neck pain atrophy only) expose a reduction in the contractile constituents that could diminish their capacity to generate and/or sustain force

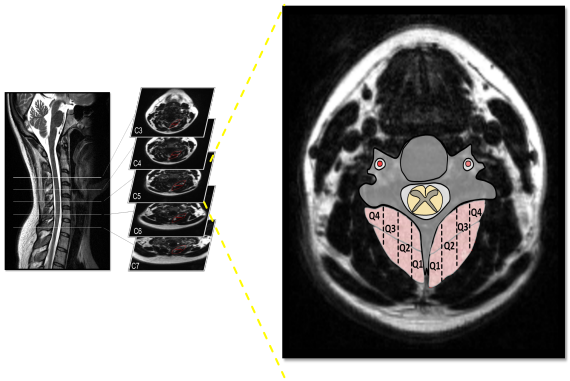


These findings indicate that, similar to idiopathic neck pain, targeted exercise for conditioning the cervical muscles of patients with chronic whiplash is a warranted component of the total management schema

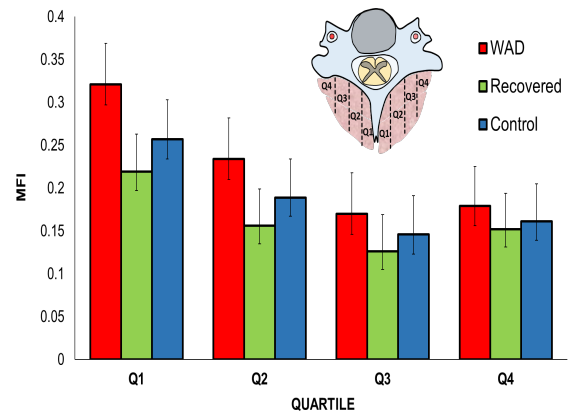


Abbott et al., 2015

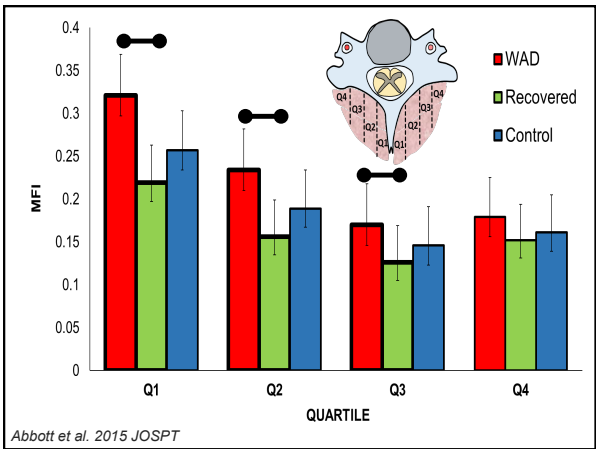




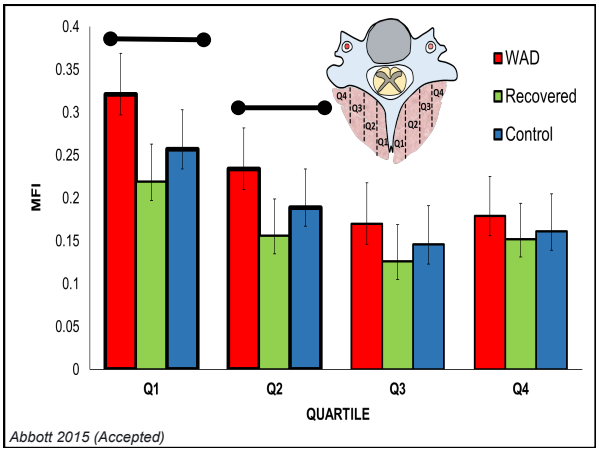
Abbott et al. 2015 JOSPT



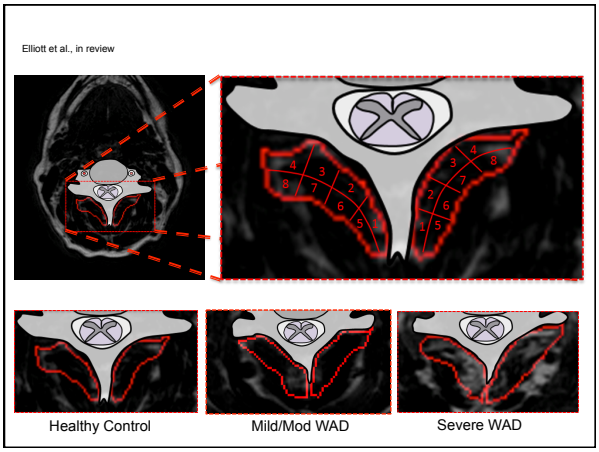
Abbott et al. 2015 JOSPT

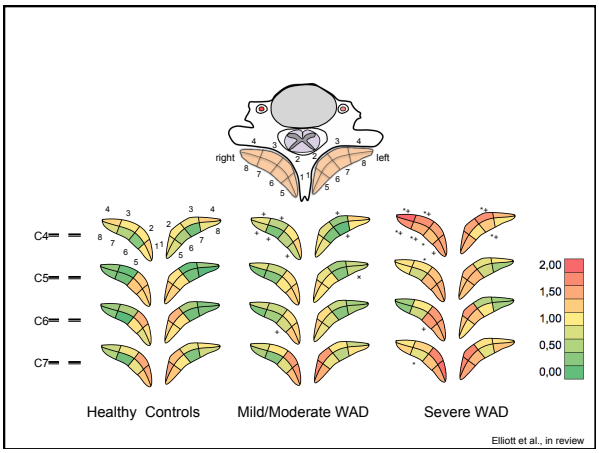


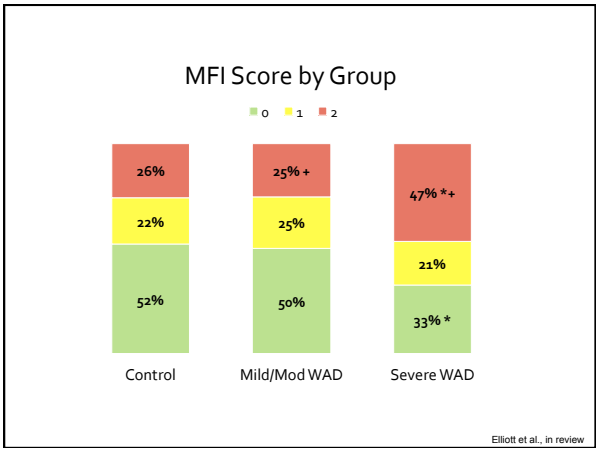
Abbott et al. 2015 JOSPT



Abbott 2015 (Accepted)







Can exercise help? If so what kind?

- Preliminary evidence suggests it depends, and that specificity of the exercise may be important (O'Leary et al. 2015)

Can we Reverse Fat in Whiplash

Participants: 5 females with WAD II

Measures:
Physical changes in extensors
- Muscle fatty infiltration (MFI)
- relative muscle Cross-sectional area (rmCSA)

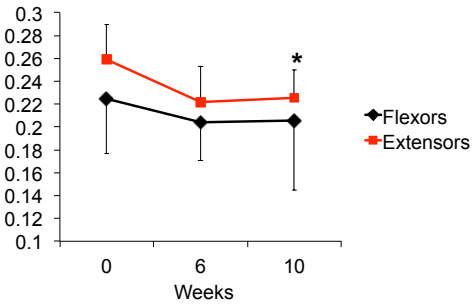
Clinical Outcomes
- Neck Disability Index

Program: 10 week progressive resistance with emphasis on cervical muscle hypertrophy.

Outcomes: Baseline, 6 weeks, 10 weeks.

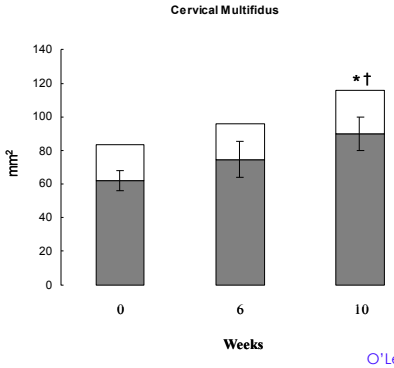
O'Leary 2015

Muscle Fatty Infiltration



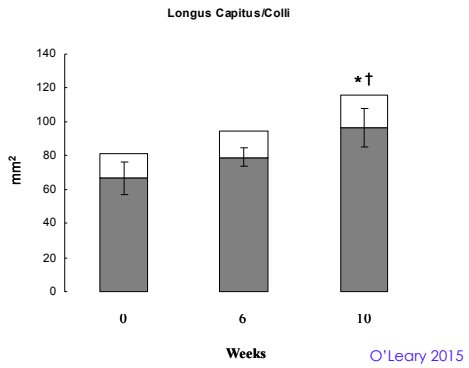
O'Leary 2015

Structural Change



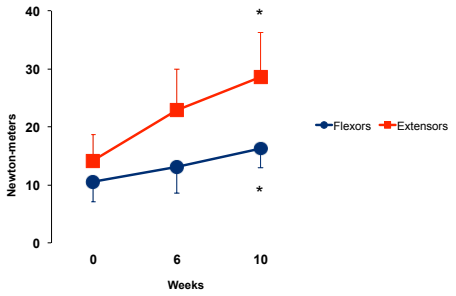
O'Leary 2015

Structural Change



O'Leary 2015

Strength Change

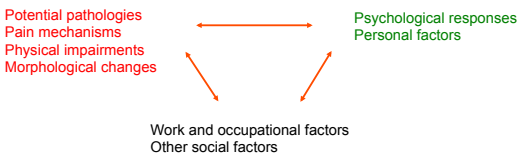


O'Leary 2015

Operating within a

Biopsychosocial Model

facilitates appreciation of all features in assessment and management



Recovery curve

Patients disabled with pain

Time

Judy – 46 y.o. account executive – Rear-End MVC 6 days ago
NPRS – 5/10
NDI – 42%
IES – 22%
BIPQ - ?
ROM – limited and painful

Roger – 33 y.o. Radiographer – Rear-End MVC 6 days ago
NPRS – 7/10
NDI – 52%
IES – 30%
BIPQ - ?
ROM – limited and painful

Beth – 39 y.o. attorney – Rear-End MVC 6 days ago
NPRS – 7-8/10
NDI – 68%
IES – 20%
BIPQ - ?
ROM – limited and painful

Judy – 46 y.o. account executive – Rear-End MVC 6 days ago
BIPQ –

1:	3
2:	1
3:	1
4:	2
5:	2
6:	3
7:	4
8:	2
9:	3
10:	2

Roger – 33 y.o. Radiographer – Rear-End MVC 6 days ago
BIPQ –

1:	3
2:	2
3:	1
4:	2
5:	4
6:	5
7:	3
8:	2
9:	4
10:	4

Beth – 39 y.o. attorney – Rear-End MVC 6 days ago
BIPQ –

1:	5
2:	4
3:	1
4:	2
5:	6
6:	3
7:	4
8:	2
9:	8
10:	7

Prediction of Milder Symptoms Following Whiplash Injury

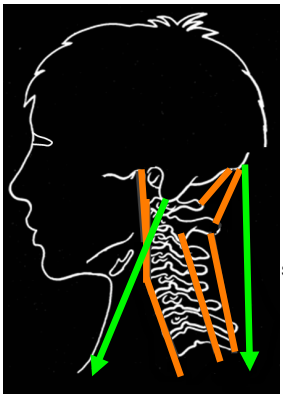
- High initial pain & disability
- Psychological distress
- Impaired muscle function

Implications for Rehabilitation

Treatment should be non-provocative, this can usually be tailored to be suitable immediately

Decrease in psychological distress parallels decreasing pain & disability

Flexor Impairments



Structural Changes
Fibre type changes (Uhlig 1995)
Fatty infiltration (Elliott 2010)

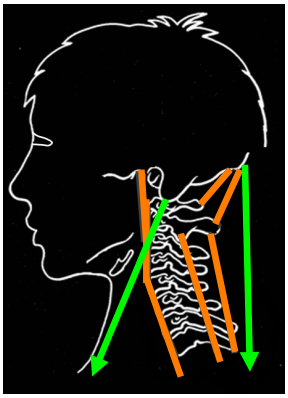
Behaviour Changes
Changes in coordination (Jull 2004, Falla 2004)
Changes in timing (Falla 2004)

Functional Deficits
Strength and endurance (Watson 1993, O'Leary 2007)

Cagnie, Dickx, Peeters, Tuytens, Achten, Cambier, Danneels (2008) JAP

O'Leary, Falla, Jull, Vicenzino (2007) JEK.

Extensor Impairments



Structural Changes
Fibre type changes (Uhlig 1995)
Cross-Section changes
(Kristjansson 2004; Elliott 2006)
Fatty infiltration (Elliott 2006)

Behaviour Changes
Changes in coordination
(Fernandez-de-las-Penas et al. 2008, O'Leary 2010)
Sensorimotor coordination (Bexander 2005)

Functional Deficits
Strength and endurance
(Jordan 1997, Lee 2005, Peolsson)
Sensorimotor disturbance
(Wenngren 2002; Treleaven 2005)

Don't forget extensor training

Summary

Gaining a greater understanding of the impairments associated with neck pain

Exercise design:
address the specific impairments

Judy – 46 y.o. account executive – Rear-End MVC 30 days ago

BIPQ –

1:	3
2:	1
3:	1
4:	2
5:	2
6:	3
7:	4
8:	2
9:	3
10:	2

Roger – 33 y.o. Radiographer – Rear-End MVC 30 days ago

BIPQ –

1:	3
2:	2
3:	1
4:	2
5:	4
6:	5
7:	7
8:	2
9:	5
10:	6

Beth – 39 y.o. attorney – Rear-End MVC 30 days ago

BIPQ –

1:	6
2:	7
3:	1
4:	2
5:	6
6:	8
7:	10
8:	2
9:	5
10:	9

WHAT ELSE CAN WE CONSIDER?

Jull et al., 2007, Pain

Issues in Rehabilitation

Reassurance
ROM
Basic Strengthening Exercises

Kinesthetic Training
Balance Training
Eye Movements
Gaze Stability Training
Co-ordination Training
Manual Therapy

What does the neck have to do with postural control?

H:
Cervical afferents important for:

knowledge of head in relation to body

Idiopathic

Whiplash

It occurs in all domains of neck disorders… but more so in patients with dizziness following whiplash

Revel 1991, Karlberg 1996, Tjell 2003

Neck mechanoreceptor dysfunction in neck pain

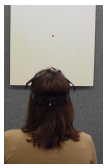
- direct damage trauma
- functional impairment muscles
- morphological changes muscles

- Inflammatory mediators - altered muscle activity
(Thunberg et al 2001, Ro and Capra 2001, Wenngren et al 1998)

- Pain

Recommendations for clinical assessment

Subjective
JPE
Balance
Oculomotor-



gaze stability

eye movement

eye/head co-ord

Joint position error

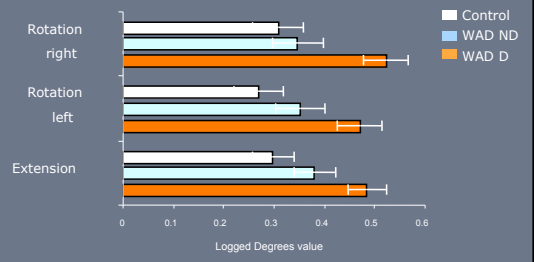
Sitting blindfolded
• repositioning to neutral
• repositioning to points in range

Laser pointer - target

• Jerky
• Searching
• Dizziness
• Overshooting
• Movement patterns

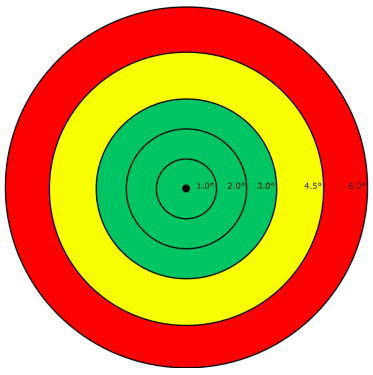
Neck pain patients exhibit altered kinaesthetic sense

Kristjannson, D'Allaiba and Jull 2003
Treleaven, Jull, Sterling 2003



Physical Therapy Intervention

- Proprioceptive and/or nocioceptive dysfunction of the cervical spine.
  - Altered Joint Position Error
    - Head mounted laser
    - 35" (~ 90cm) from target
    - Error >2.75" from target

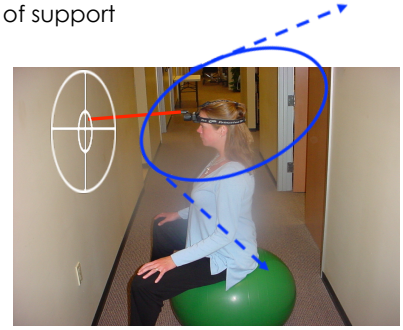


Rob Landel, PT, DPT, OCS

Joint Position Sense/Error. Pt starts with awareness of target object on wall. Then closes eyes and performs rotation R and L and/or flexion/extension all the while trying to relocate starting target. Have pt open eyes once they feel that they have succeeded. Measures of error can be made.

Jerky
Searching
Dizziness
Overshooting

JPE with progressing to challenging base of support



With and without active ROM

Balance

Comfortable
Narrow
Tandem
Single leg

Eyes open vs closed

Surface- firm vs soft vs unstable

Combining with Balance

Further challenge Pt with tandem stance and then unstable surface (barefoot) with JPE retraining

e.g. pillow

Examination

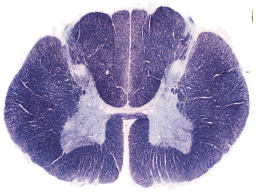
(Adapted from Leigh 2006, Kattah 2009)

- Observation – Abnormal head postures and lid abnormalities.
- ROM and alignment of visual axes.
- Oculomotor Examination
  - Fixation – primary position and eccentric gaze.
  - Saccades
  - Pursuit
  - Eye-head coordination
  - Vergence
- Smooth Pursuit Neck Rotation Test

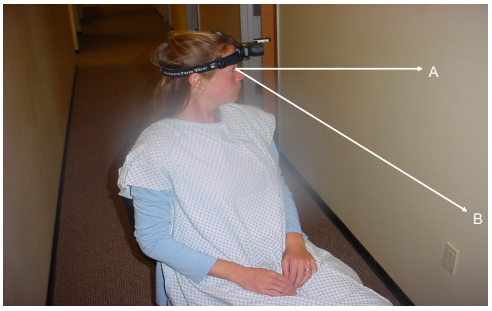
PRIMARY POSITION

Oculomotor Examination

Smooth pursuit neck torsion test (Tjell and Rosenhall, 1998). Static neck torsion results in pursuit gain depression.



Neck Torsion with Trunk Rotation



Neck Torsion Test-Now, trunk in sustained rotation ~45 deg R then L with head stationary with eye follow "A" then "B". Swivel chair can assist with dynamic trunk rotation R and L while head still and eyes follow from "A"-"B"—progress with eye goggles, sit/stand, etc, etc.

Posture of Head

Abnormal/compensatory head posture to alleviate diplopia caused by a vertical misalignment of the eyes due to a right CN IV palsy secondary to TBI.

Photo courtesy of Suzanne Wickum, OD & thanks to Dr. Janet Helminski, PT, PhD

Lid Abnormalities

Ptosis and abnormal eye alignment due to a right cranial nerve III palsy secondary to TBI.

Photo courtesy of Suzanne Wickum, OD & thanks to Dr. Janet Helminski, PT, PhD

Alignment of Visual Axes

- Tropia – Misalignment of the visual axes during binocular viewing of a single target.
- Phoria – Misalignment of the visual axes during monocular viewing of a single target.

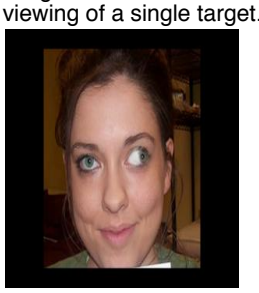
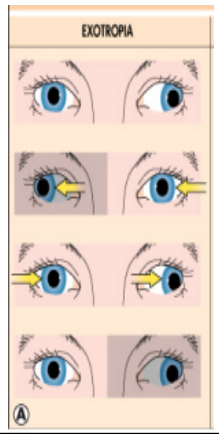


Photo courtesy of Suzanne Wickum, OD & thanks to Dr. Janet Helminski, PT, PhD

Cover – Uncover Test

Cover Test

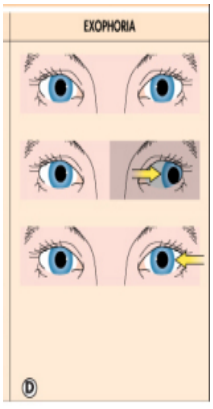
- While focusing on target, one eye is covered
- Look for "movement of redress" of uncovered eye
- Identifies tropia of uncovered eye (eso/exo/ hyper/hypo)



Cover – Uncover Test

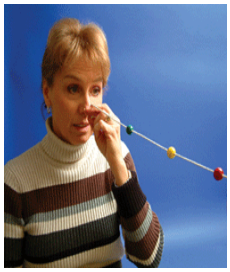
Uncover Test

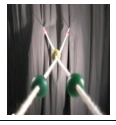
- While focusing on target, one eye is covered.
- Observe for movement of occluded eye when cover is removed
- Identifies phoria of covered eye (eso/exo/hyper/hypo)



Physical Therapy Differential Diagnosis

- Oculomotor Dysfunction
  - Vergence
    - Broc's string





CLINICAL POINTERS

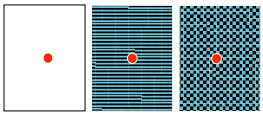
A simple pair of swim goggles can be helpful. Peripheral vision can be restricted by blackening out except for small area in center of each side.

Progression

position- supine, sitting, standing

active vs passive movement

pattern in background



focus point

vary speeds

neck torsion

THE UNIVERSITY OF QUEENSLAND

Physical Therapy and Human Movement Sciences

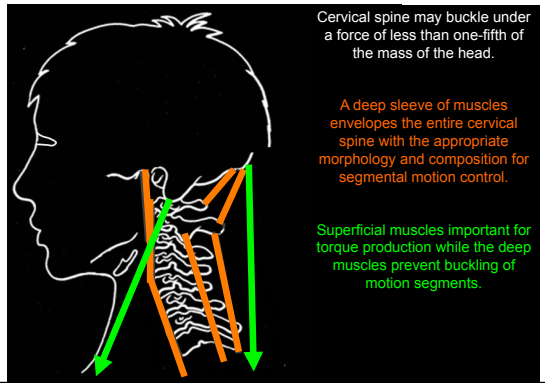
Exercise Prescription

Jim Elliott PT, PhD Shaun O'Leary PT, PhD

Queensland Government
Queensland Health

ccre spine

Importance of Muscles

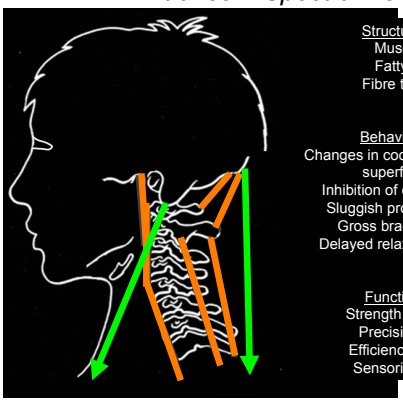


Cervical spine may buckle under a force of less than one-fifth of the mass of the head.

A deep sleeve of muscles envelopes the entire cervical spine with the appropriate morphology and composition for segmental motion control.

Superficial muscles important for torque production while the deep muscles prevent buckling of motion segments.

Evidence – Spectrum of Impairments

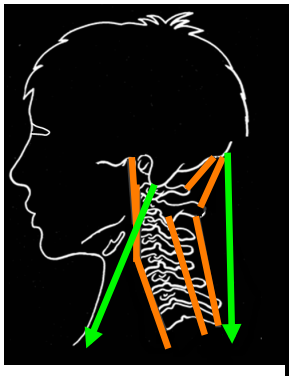


Structural Changes
Muscle wasting
Fatty infiltration
Fibre type changes

Behaviour Changes
Changes in coordination of deep and superficial muscles
Inhibition of deep neck muscles
Sluggish protective responses
Gross bracing mechanisms
Delayed relaxation following use

Functional Deficits
Strength and endurance
Precision and acuity
Efficiency of movement
Sensorimotor function

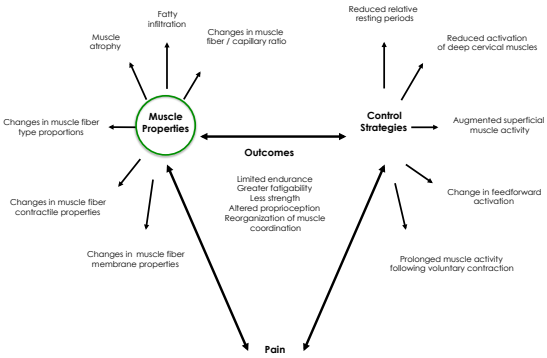
Training – Where do you start?



Structural Changes
Muscle wasting
Fatty infiltration
Fibre type changes

Behaviour Changes
Changes in coordination of deep and superficial muscles
Inhibition of deep neck muscles
Sluggish protective responses
Gross bracing mechanisms
Delayed relaxation following use

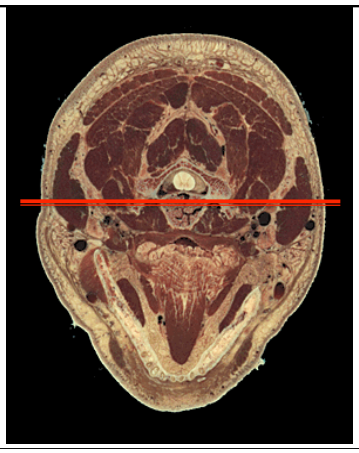
Functional Deficits
Strength and endurance
Precision and acuity
Efficiency of contraction
Sensorimotor function

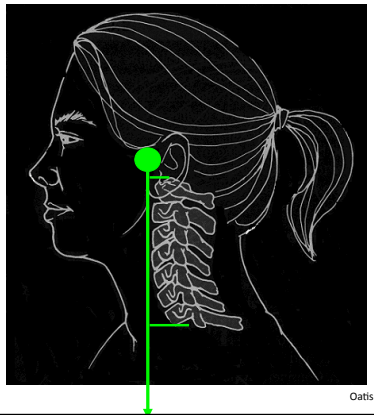


Falla and Farina (2009)

Muscle Properties

Are all neck muscles created equally?





Tendency for Flexion

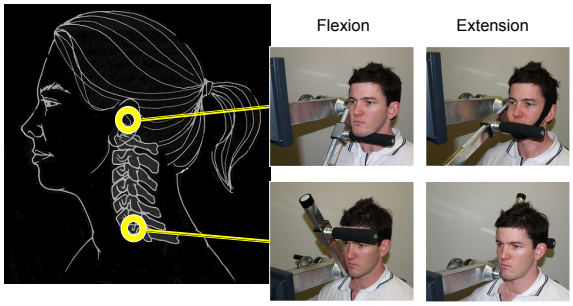
Oatis (2004). Kinesiology, Lippincott W&W

Extension : Flexion Strength Ratios

Extension to flexion ratios (CT axis) range from 1.4 to 2

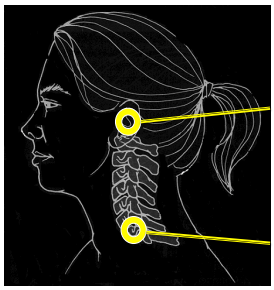
(Jordan 1999; Seng 2002; Vasavada 2001)

Performance Ratios



Van Wyk, Jull, Vicenzino, Greaves, O'Leary (2010) Applied Biomechanics

Strength Ratios (57 females, 49 males)



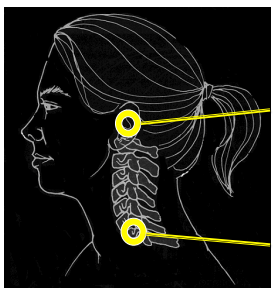
Flexion : Extension (95% CI)

1 : 1.3 (1.2 - 1.4)

1 : 1.9 (1.8 - 2)

Van Wyk, Jull, Vicenzino, Greaves, O'Leary (2010) Applied Biomechanics

Endurance Ratios (36 females, 29 males)

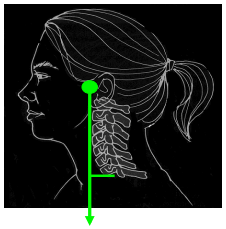


Flexion : Extension (95% CI)

1 : 1.9 (1.6 – 2.2)

1 : 2.3 (2.1 – 2.6)

O'Leary, Fagermoen, Hasegawa, Thorsen, Van Wyk, Jull (In Progress)



Oatis (2004). Kinesiology, Lippincott W&W

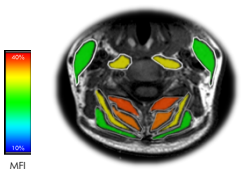
Muscle Properties

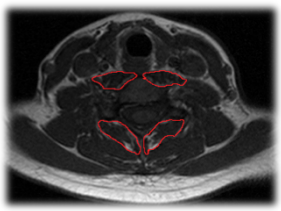
What does structural change in muscle mean for exercise prescription?

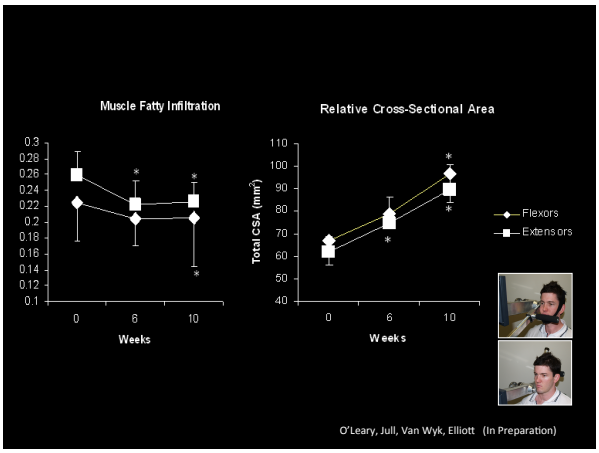
Fatty Infiltration in the Cervical Extensor Muscles in Persistent Whiplash-Associated Disorders

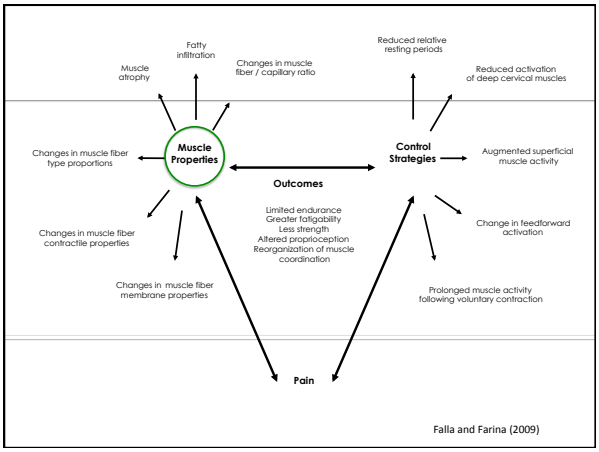
A Magnetic Resonance Imaging Analysis

James Elliott, PT, MS,*†‡ Gwendolen Jull, MPhty, PhD, FACP,*
Jon Timothy Noteboom, PT, PhD, SCS, ATC,† Ross Darnell, PhD,* Graham Galloway, PhD,‡
and Wayne W. Gibbon, MB BS, FRCS, FRCR, FRANZCR§



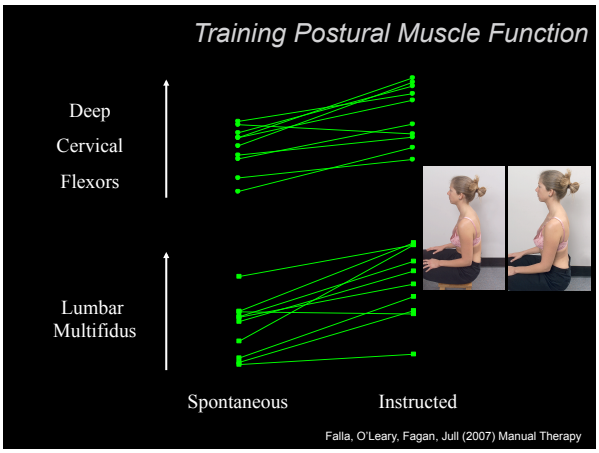






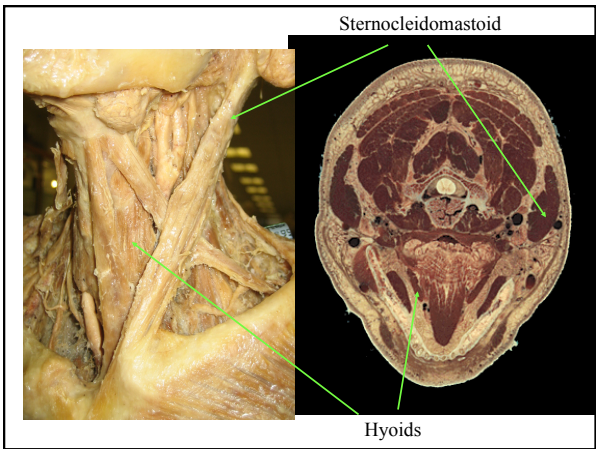
Control Strategies

Does specificity of the exercise manoeuvre matter?

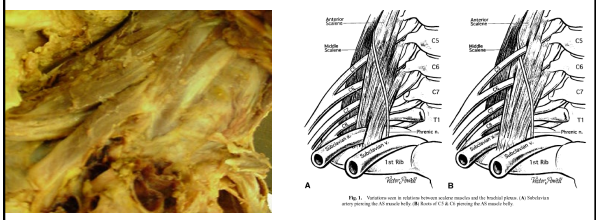


Anterior Muscles

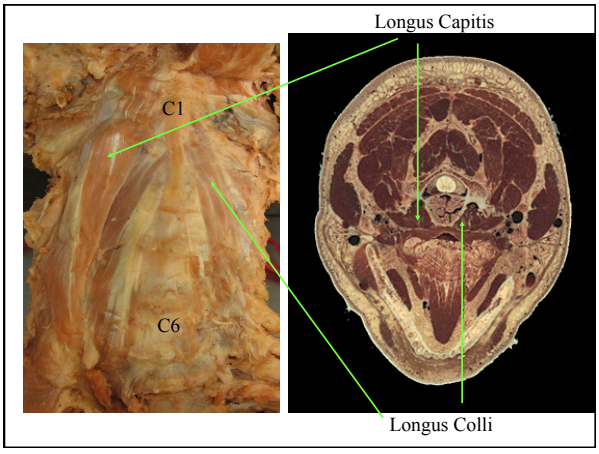
Anterior Neck Muscles

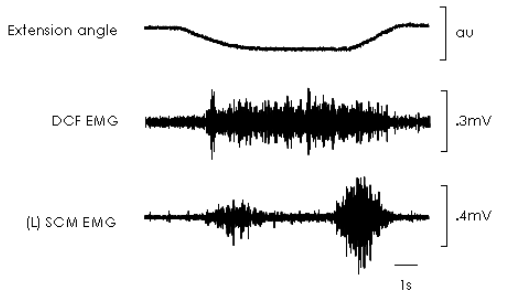


Scalene Muscles



Harry et al. (1997). Clin Anat, 10:250-252.



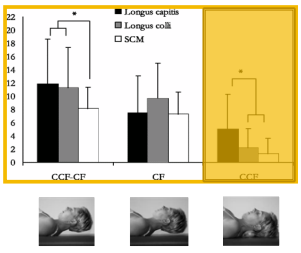


Falla et al Unpublished.

Cagnie, Dickx, Peeters, Tuytens, Achten, Cambier, Danneels (2008) JAP

O'Leary, Falla, Jull, Vicenzino (2007) JEK.

WHICH EXERCISE BEST TARGETS THE DEEP CERVICAL FLEXORS?



Cagnie et al (2008) J Appl Physiol

Indications for Cervical Flexor Training

Functional issues
- Poor active control of cervical extension in upright postures
- Forward head postures (craniocervical extension)
- Accentuated lordosis
- Difficulties lifting head off bed, during sit-ups

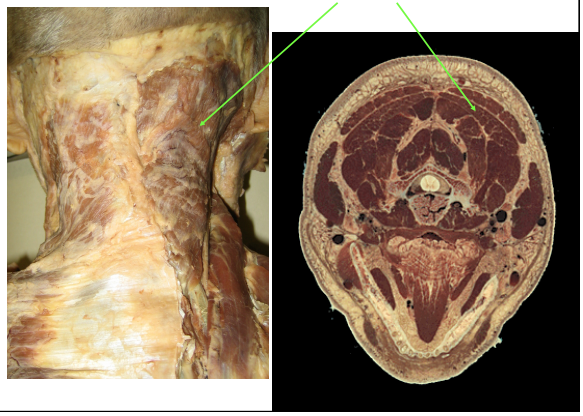
Patient performs poorly on formal tests of motor function
- Head lift test
- Craniocervical flexion test

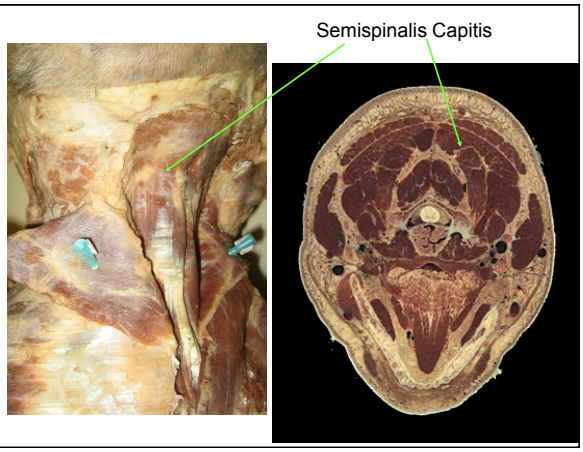
Patient immediately responds positively following the performance of cervical flexion exercise
- Cervical motion eg. extension, rotation
- Resting pain
- Palpation findings

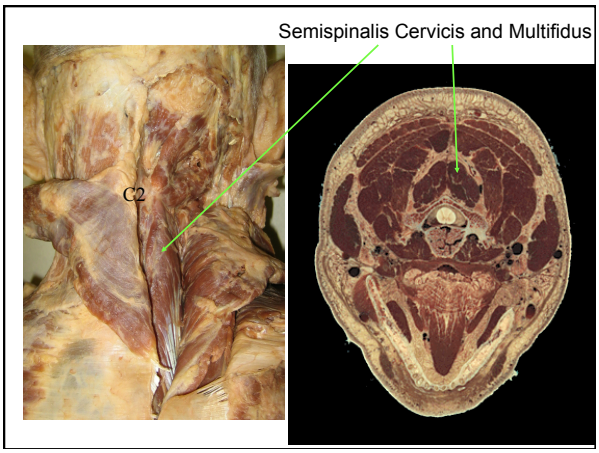
PRACTICE FLEXORS

Posterior Muscles

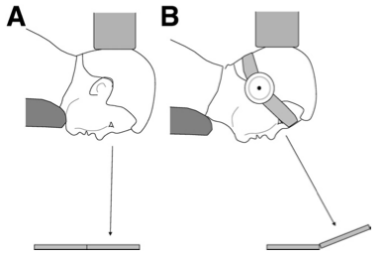
Posterior Neck Muscles







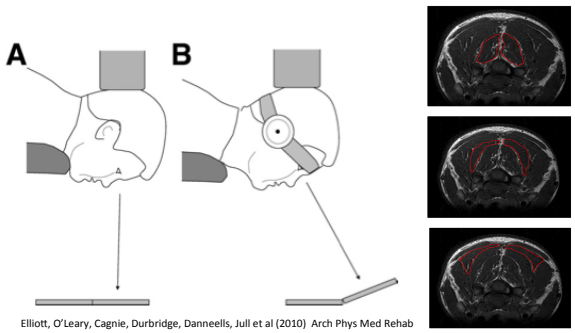
WHICH EXERCISE BEST TARGETS THE CERVICAL EXTENSORS?



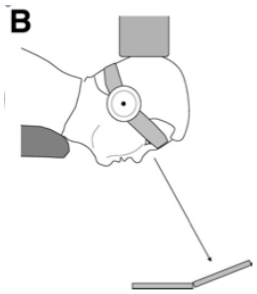
Elliott et al. (2011) Archives of Physical Medicine and Rehabilitation

Craniocervical Orientation Affects Muscle Activation When Exercising the Cervical Extensors in Healthy Subjects

James M. Elliott, PT, PhD, Shaun P. O'Leary, PT, PhD, Barbara Cagnie, PT, PhD, Gail Durbridge, MSc, Lieven Danneels, PT, PhD, Gwendolen Jull, PT, PhD

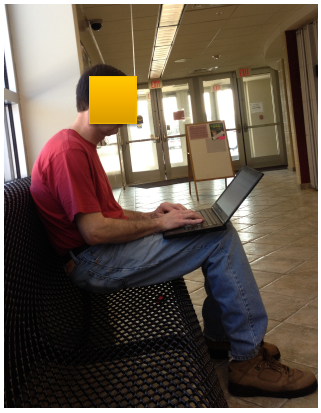


Elliott, O'Leary, Cagnie, Durbridge, Danneells, Jull et al (2010) Arch Phys Med Rehab



Heightened activity of Semispinalis Capitis muscle

Elliott, O'Leary, Cagnie, Durbridge, Danneells, Jull et al (2010) Arch Phys Med Rehab



Indications for Extensor Training

Functional issues
- Poor active control of upright cervical flexion or flexed cervical postures.
- Forward head postures (lower cervical flexion)
- Reduced lordosis
- Prominent reports of sensorimotor disturbances and positive sensorimotor tests

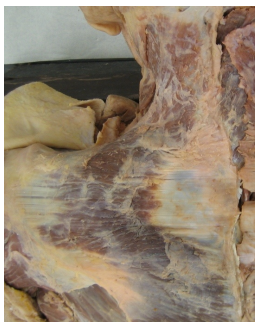
Patient performs poorly on formal tests of motor function
- Test for deep lower cervical extensors
- Test for deep craniocervical extensors

Patient immediately responds positively following the performance of cervical extension exercise
- Cervical motion
- Resting pain
- Palpation findings

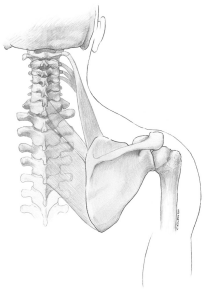
PRACTICE EXTENSORS

Axioscapular Muscles

Axioscapular Muscles



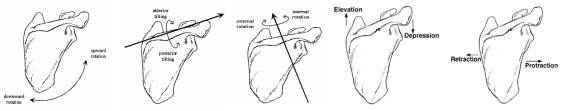
Axioscapular Muscles



Due to their superior attachments, axioscapular muscles such as levator scapulae have the capacity to induce motion and abnormally load cervical motion segments in the presence of impaired axio-scapular muscle function.

(Behrsin 1986)

Axioscapular Muscle Function



As the arm elevates through range in the first 30-40 degrees of elevation there is minimal scapular motion. As elevation progresses there is progressive upward rotation, posterior tilt, and external rotation. Posterior tilt and external rotation especially occur once the arm is beyond 90 degrees (McClure et al 2001- bone pin studies).

Axioscapular Impairments

Structural Changes
Morphological and histological changes in the upper trapezius (Kadi 1998, Larsson 1998)

Behaviour Changes
Changes in trapezius behaviour (Falla 2007; Johnston 2008; Nederhand 2000; Szeto 2005, Wegner 2010)
Altered Serratus Anterior (Helgadottir 2011)

Functional Deficits
Altered Scapular Kinematics (Helgadottir 2010)

Axioscapular Structural Changes

Morphological and histological changes in the upper trapezius muscle has been found in patients with chronic neck disorders (Kadi 1998, Larsson 1998)

Axioscapular Behaviour Changes

Changes in behaviour of the 3 portions of trapezius in chronic mechanical disorders of the shoulder girdle. (Cools et al., 2007; Lin et al., 2006; Lin et al., 2005)

Changes in the behaviour of the upper trapezius in chronic mechanical neck pain (Falla 2004; Johnston 2008; Nederhand 2000; Szeto 2005)

Indications for Axioscapular Training

Functional issues
- Patient complains of discomfort associated with upper limb activities
- Patient reports upper limb symptoms suggestive of chronic neural mechanosensitivity

Patient performs poorly on formal tests of motor function
- Poor scapular orientation at rest
- Poor scapular orientation during open or closed chain loaded tests, or the prone scapular holding test

Patient immediately responds positively to –
- Scapula repositioning
  - Cervical motion or upper limb function
  - Resting pain
  - Palpation of tender axioscapular muscles
- Joint palpation following performance of prone scapular hold test

Axioscapular Evaluation

Observation of scapular orientation
- At rest
- During upper limb tasks (functional, open/closed chain)

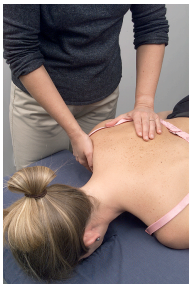
Common findings –
- *Good orientation at rest and during loading*
- *Good orientation at rest but poor during loading*
- *Poor orientation at rest and during loading*
- *Poor orientation at rest but corrects during loading*

What is the primary direction of control loss with regard to –

- *Scapular orientation eg. anterior tilt*
- *Shoulder movement eg. flexion*

Prone Tests of scapular synergy

Retest manual examination **



Observe

pattern of muscle control

Correct pattern

Incorrect

Incorrect

Test: holding capacity

Retest PAIVMs: Assess change in joint reactivity

Observe pattern of activity
- Functional task (eg typing)
- Arm elevation/abduction

Train scapular synergy

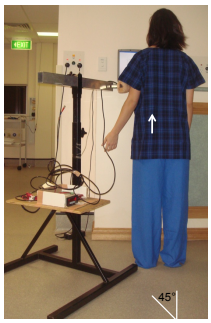
- **low load (side lying, prone)**
- **Emphasise precision and control of scapular rotation (tripartite trapezius)**
- **Train holding capacity at low loads used functionally in control of posture and arm movements**

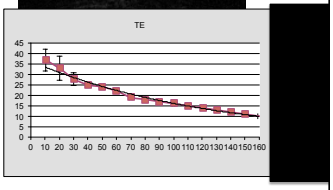
Behaviour Changes (cont):

Experimental muscle pain results in reorganization of coordination among trapezius muscle subdivisions during a repetitive shoulder task (Falla et al 2007)

Altered behaviour of the lower trapezius in neck pain during isometric shoulder girdle tasks (Zakharova-Luneva et al In review)

Altered behaviour of the lower and middle trapezius in neck pain during a typing task (Wegner et al In Press)





PRACTICE AXIOSCAPULAR

Postural deficits
(manual correction of scapular dyskinesis)

Primary direction of control LOSS

Joint Provocation

Summary - Exercise

- Most evidence syntheses indicate it is beneficial, but the type and parameters that are most beneficial are largely unknown
- Mechanisms could include: normalization of muscle morphology, reduced fear of movement, improved oxidative capacity (endurance), proprioception, exercise-induced hypoalgesia
- The total management of the patient with persistent neck pain should include specific exercise for the head/neck

Special Thanks to

- Dr. Shaun O'Leary, PT, PhD and Emeritus Professor Gwen Jull from the University of Queensland, Australia for their work and support of this presentation

Other Treatment Options

- **Education**
  - Type? Mode? Duration? Frequency?
- **Electrotherapeutic Modalities:**
  - Low-moderate evidence to support TENS and Laser for chronic WAD (APTA CPG)
  - No consistent evidence in acute WAD
- **Pillows & other supports**
  - Equivocal empirical evidence
  - Go with pt. preference
- **Ergonomics**
  - Highly individual effects

Summary: Management of WAD

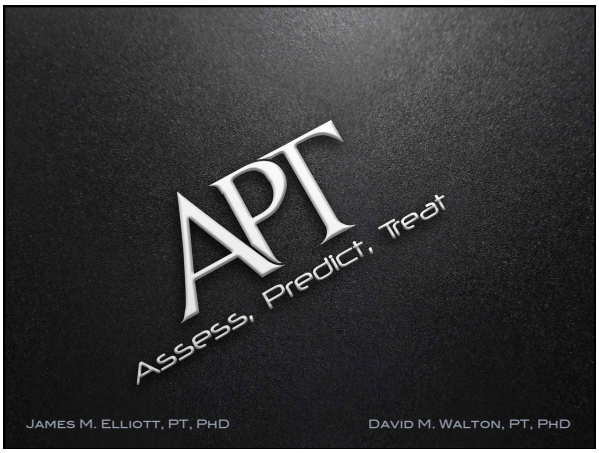
- Acute Neck Pain: Focus on prognosis-based assessment
  - Allow recovery to occur without interference in low risk
  - Conduct detailed evaluation and follow moderate risk closely
  - Consider early targeted intervention for high risk
- Chronic Neck Pain: Assess appropriately, make treatment decisions based on assessment findings
- Mechanisms of acute-to-chronic transition are unknown but models are emerging
- No 'one size fits all' approach for WAD, it is a very heterogeneous condition

Case Studies

1. Susan
2. Julie

For more information

- **Dr. Jim Elliott PT PhD**
  - j-elliott@northwestern.edu
  - www.nirl.nu
  - Twitter: @elliottjim
- **Dr. Dave Walton PT PhD**
  - dwalton5@uwo.ca
  - www.pirlresearch.com
  - Twitter: @uwo_dwalton
  - www.youtube.com/c/davewaltonPT


APT
Assess, Predict, Treat
James M. Elliott, PT, PhD
David M. Walton, PT, PhD