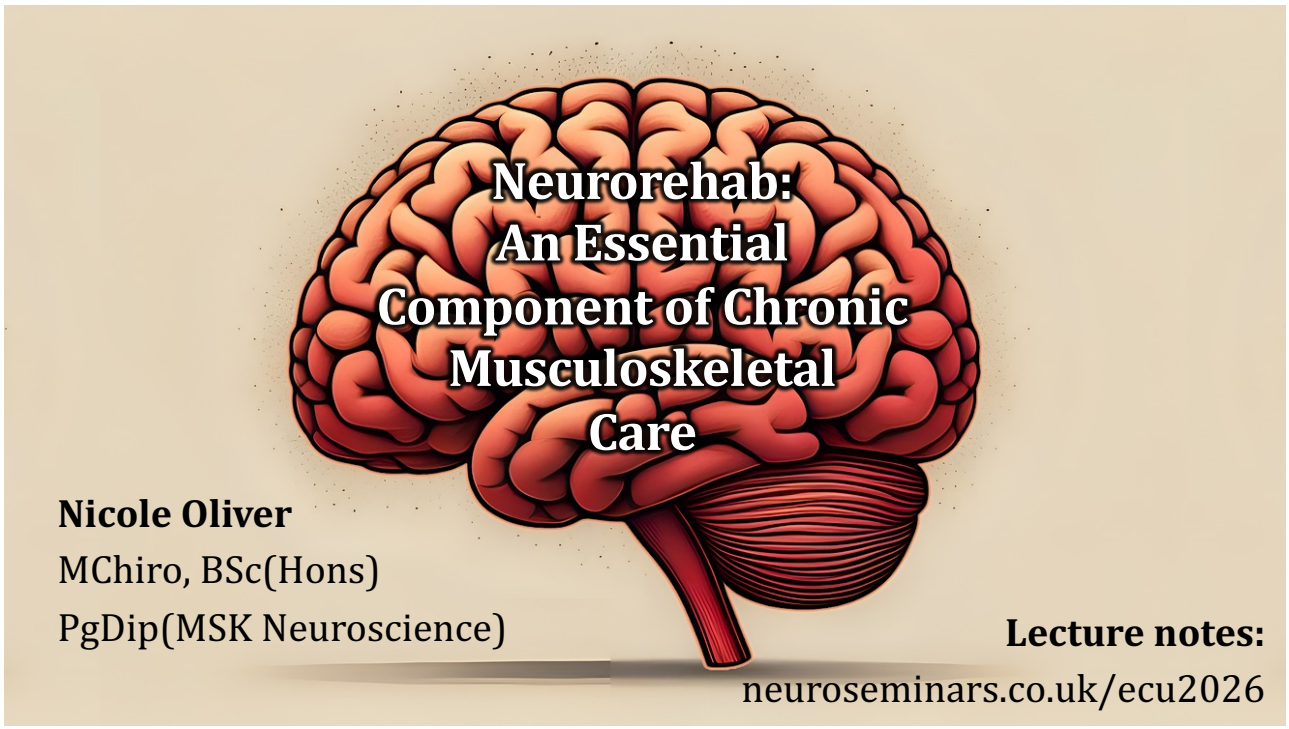
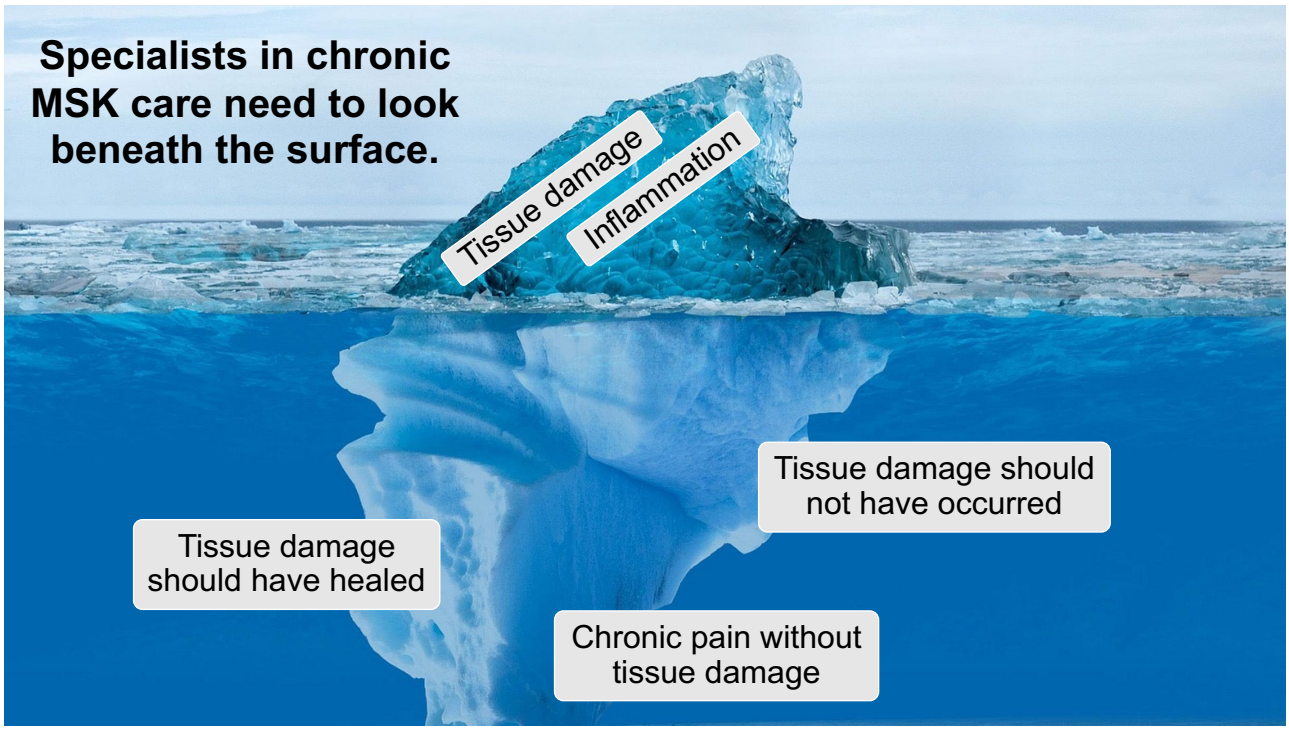
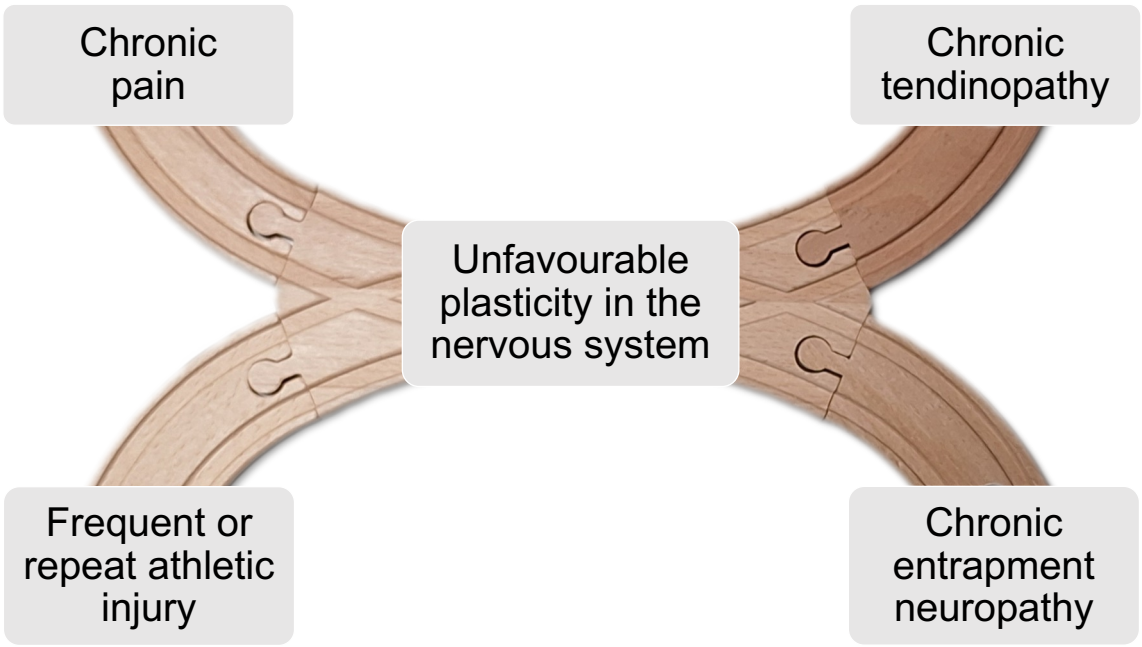
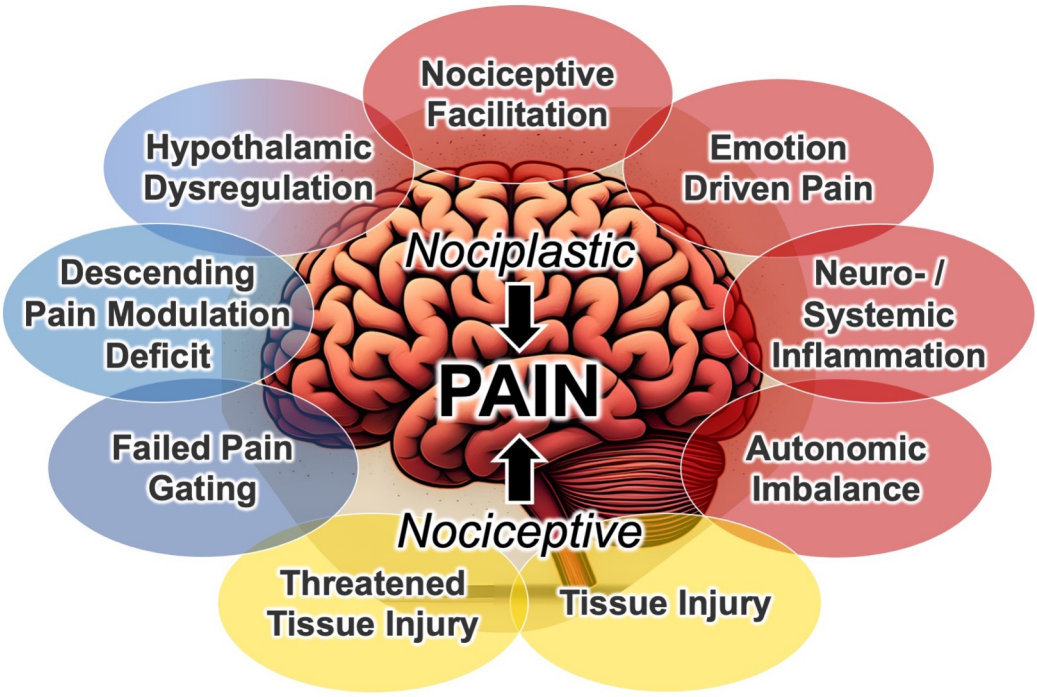

Neurorehab:
An Essential
Component of Chronic
Musculoskeletal
Care
Nicole Oliver
MChiro, BSc(Hons)
PgDip(MSK Neuroscience)
Lecture notes:
neuroseminars.co.uk/ecu2026


Specialists in chronic MSK care need to look beneath the surface.
Tissue damage
Inflammation
Tissue damage should not have occurred
Tissue damage should have healed
Chronic pain without tissue damage



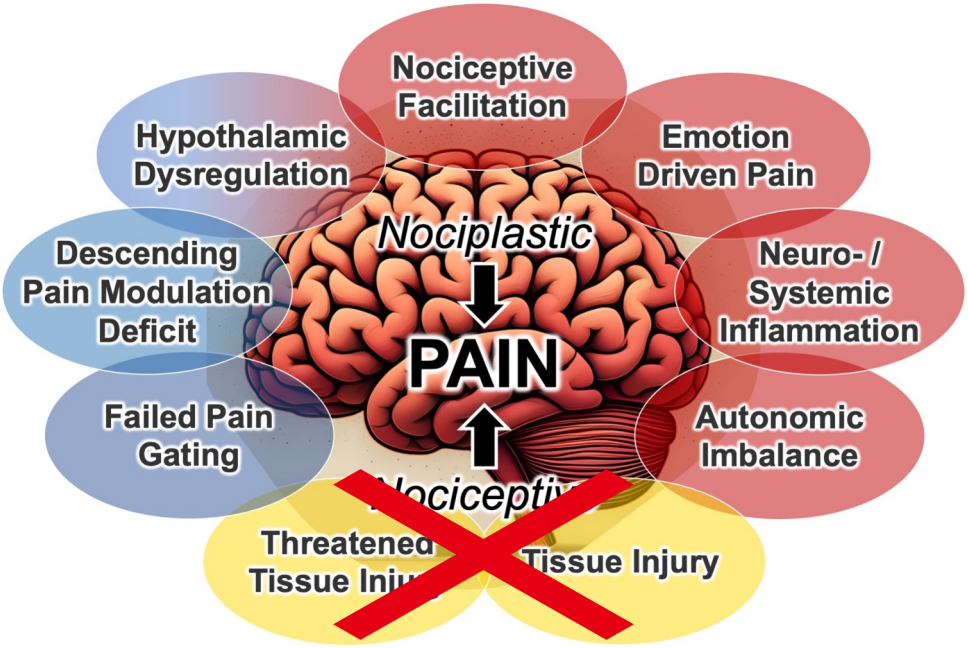
Lecture / Workshop Outline

- Chronic pain: neuro-pathophysiology & therapeutic approaches.
- How motor control abnormalities, insufficient postural stabilisation and neurologically-driven muscle imbalances can contribute to frequent or repeat injury, as well as negatively influence the systems involved in pain modulation.
- The effects of exercise on the central nervous system.
- Strategies to promote positive contextual effects.
- Basic examination and rehabilitation of parts of the nervous system involved in pain modulation, postural stabilisation and motor control.

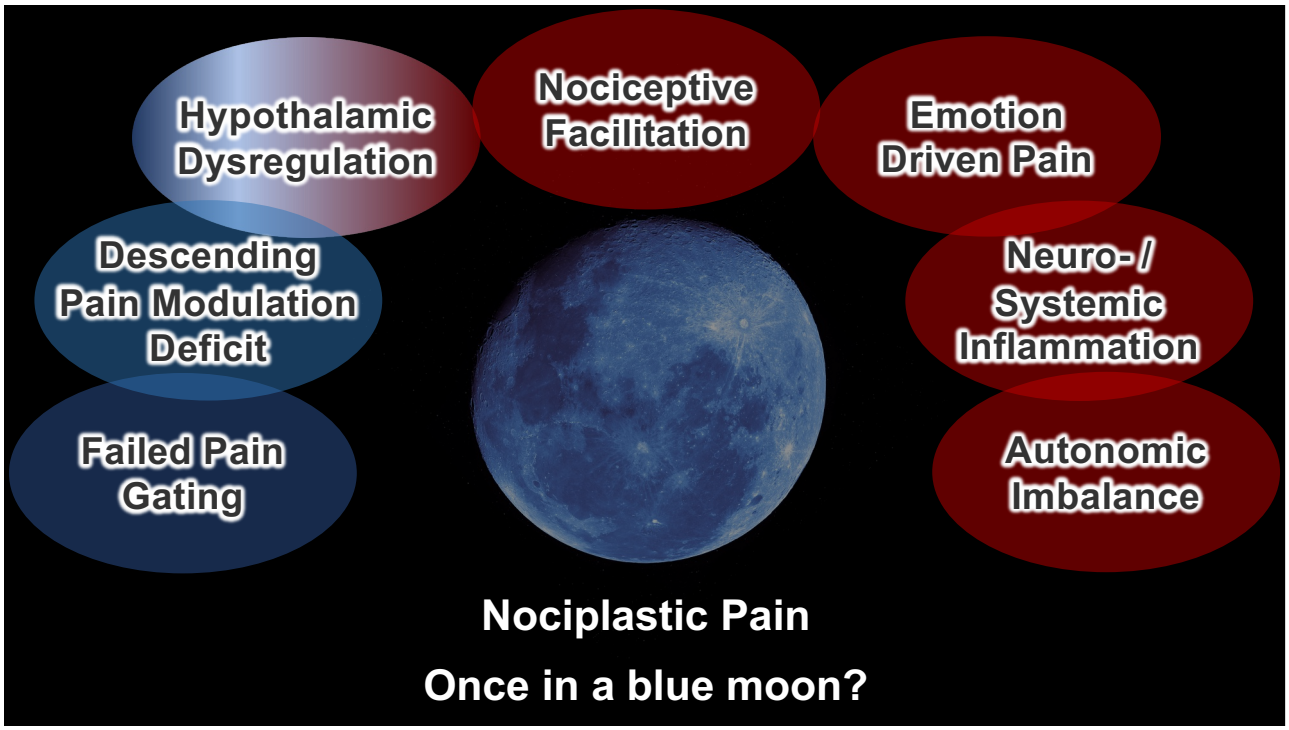


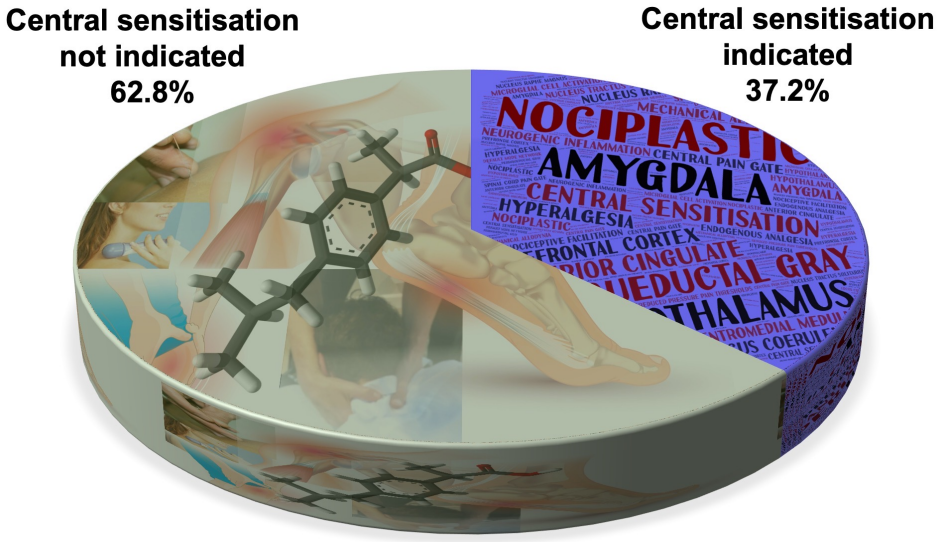
Nociceptive Pain	Nociplastic Pain
Unique features achieving greatest consensus for nociceptive pain: ✓ 'responsiveness to NSAIDs' ✓ 'signs of inflammation' ✓ 'predictable pain recovery based on expected time of tissue recovery'	For nociplastic pain, the most agreed unique features were: ✓ 'diffuse, widespread, or poorly localised pain' ✓ 'generalised hypersensitivity' ✓ 'multiple somatic symptoms (e.g., fatigue, memory / concentration / sleep disturbances)'
↓	↓
Features associated with tissue injury or inflammation ➢ nociceptive mechanisms	Central sensitisation

Shraim et al. (2022). Features and methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: a Delphi expert consensus study. https://doi.org/10.1097/j.pain.0000000000002577
Full text: https://vbn.aau.dk/ws/portalfiles/portal/460474258/00006396_900000000_97799.pdf

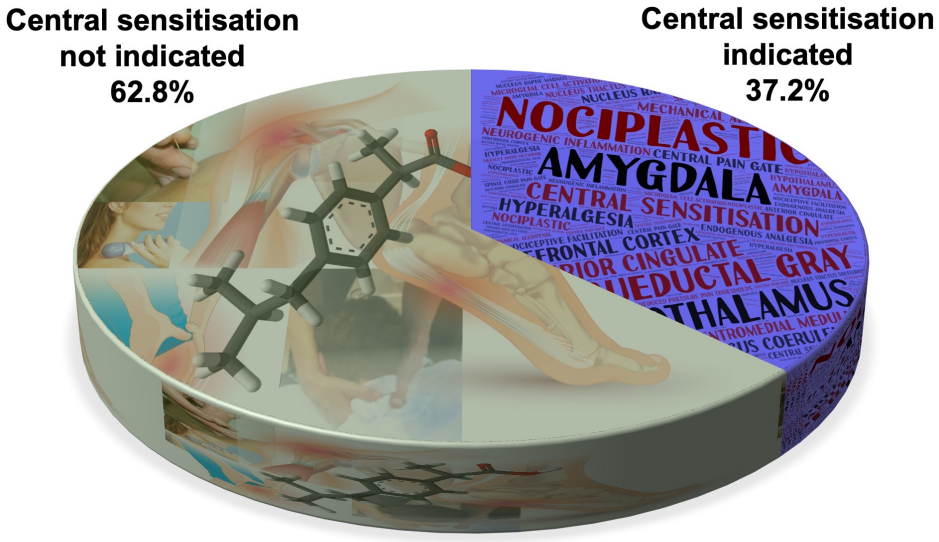


Pain can occur without tissue damage!

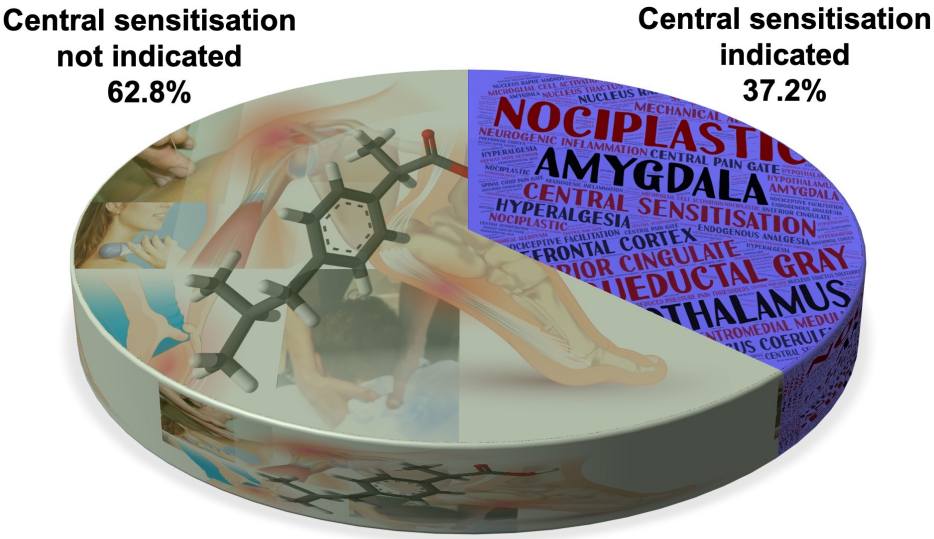




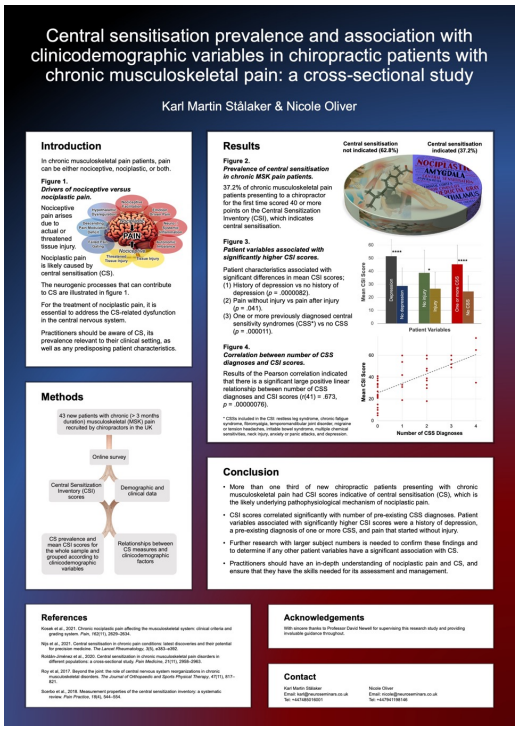
Central sensitisation is the likely underlying pathophysiological mechanism of nociplastic pain.



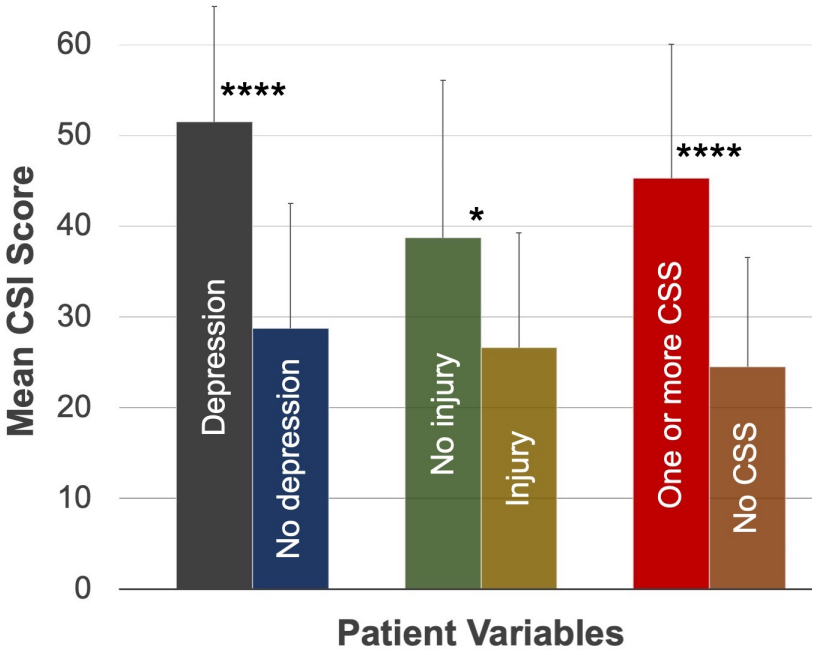
More than 1/3 of chiropractic new patients in the UK who had chronic pain (>3 months duration) were centrally sensitised.



More than 1/3 of chiropractic new patients with chronic pain will probably not respond well to manual therapy and/or NSAIDS alone.



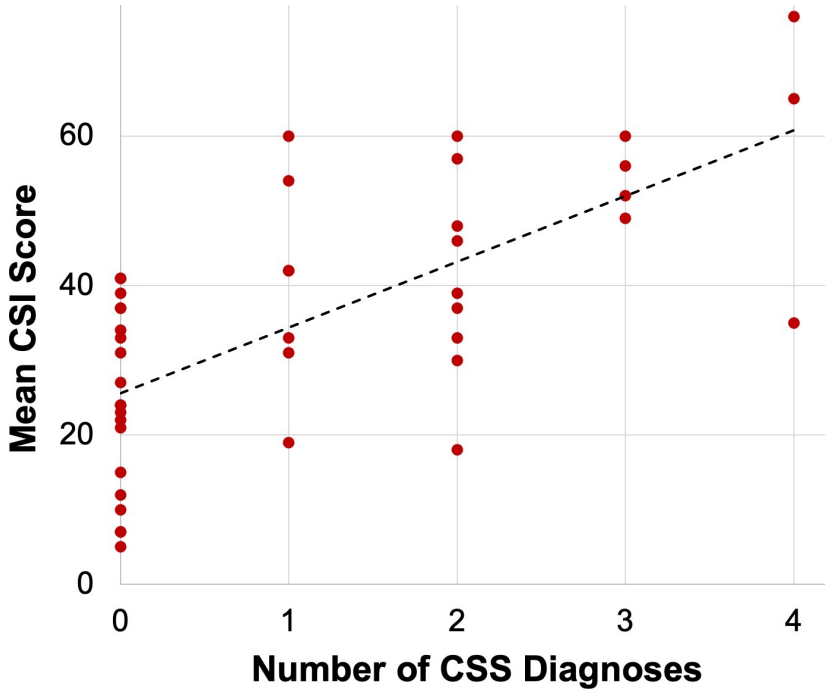
Stålaker, K.M. & Oliver, N. (2026, May 14-16). *Central sensitisation prevalence and association with clinicodemographic variables in chiropractic patients with chronic musculoskeletal pain: a cross-sectional study* [Poster presentation]. ECU Convention 2026, Zürich, Switzerland.



CSSs included in the CSI:

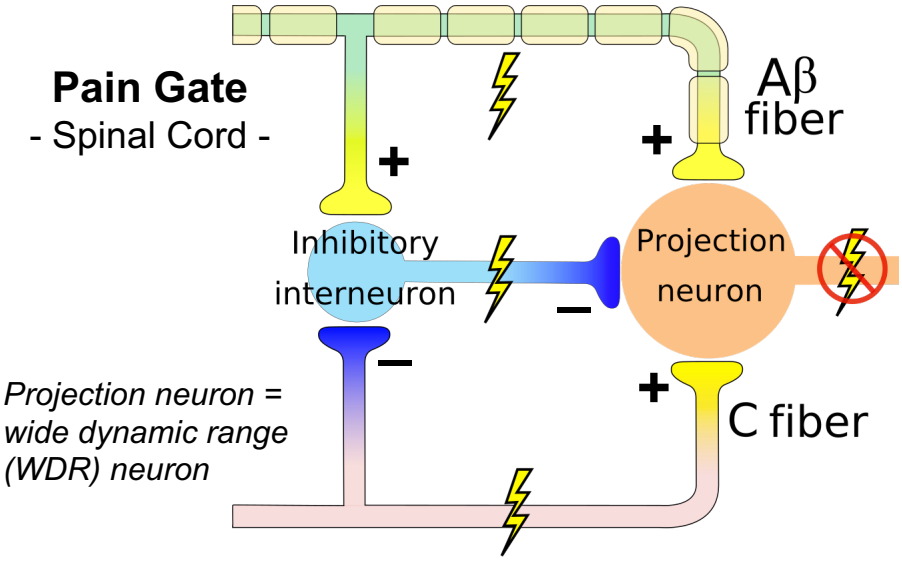
- Restless leg syndrome
- Chronic fatigue syndrome
- Fibromyalgia
- TMD
- Migraine or tension headaches
- Irritable bowel syndrome
- Multiple chemical sensitivities
- Neck injury
- Anxiety or panic attacks
- Depression

CSI = Central Sensitization Inventory *CSS = Central sensitivity syndrome*



Results of the Pearson correlation indicated that there is a significant large positive linear relationship between number of CSS diagnoses and CSI scores ($r(41) = .673$, $p = .00000076$).

Mechanoreceptor-Mediated Modulation of Pain

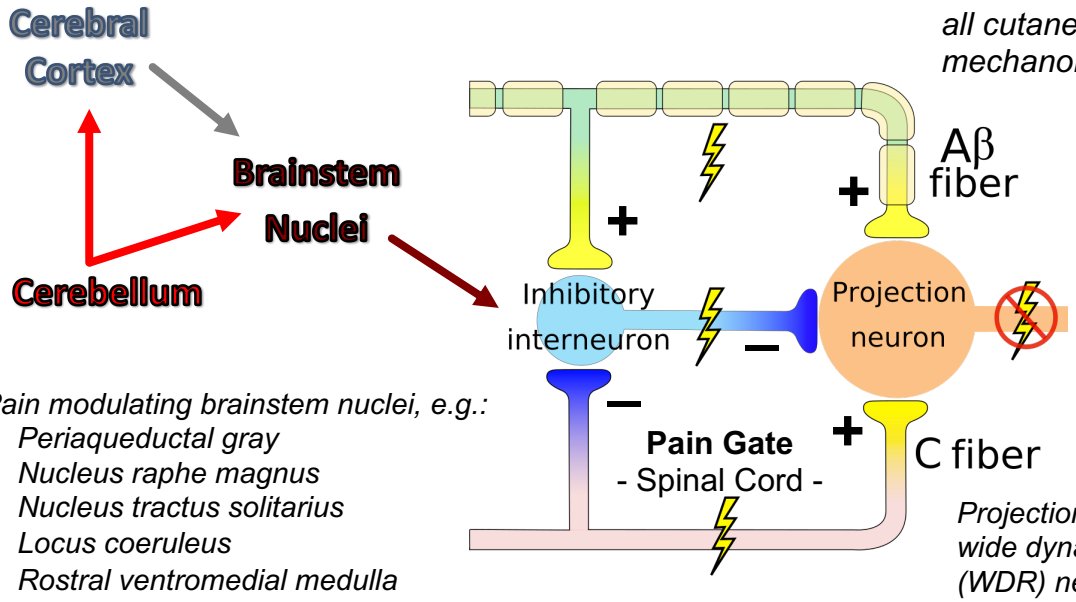


A beta (Aβ) fibres = secondary receptors of muscle spindles, all cutaneous mechanoreceptors and joint capsule mechanoreceptors

Projection neuron = wide dynamic range (WDR) neuron

Descending Pain Modulation

A beta (Aβ) fibres = secondary receptors of muscle spindles & all cutaneous mechanoreceptors



Pain modulating brainstem nuclei, e.g.:

- *Periaqueductal gray*
- *Nucleus raphe magnus*
- *Nucleus tractus solitarius*
- *Locus coeruleus*
- *Rostral ventromedial medulla*

Projection neuron = wide dynamic range (WDR) neuron

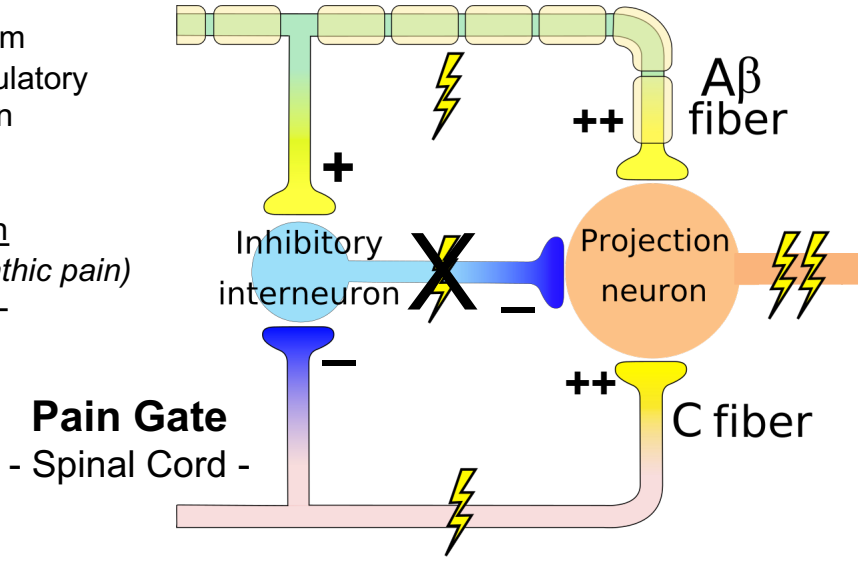
Mechanical Allodynia
- Failed Peripheral Pain Gating -

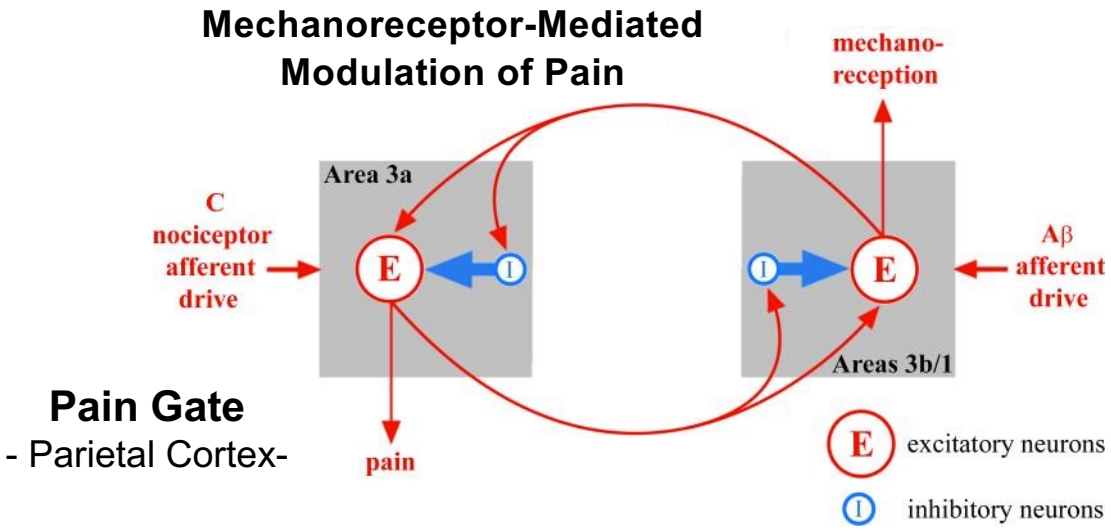
(1) <u>Inhibitory interneuron failure</u>

- Lack of stimulation from Aβ fibres *(e.g. immobilisation, sedentary lifestyle)*
- Lack of stimulation from descending pain modulatory pathways originating in brainstem nuclei.

(2) <u>Nociceptive sensitisation</u>

- Nerve injury *(neuropathic pain)*
- Upregulation of alpha-adrenoceptors *(more likely to occur with chronic stress or sedentary lifestyle)*

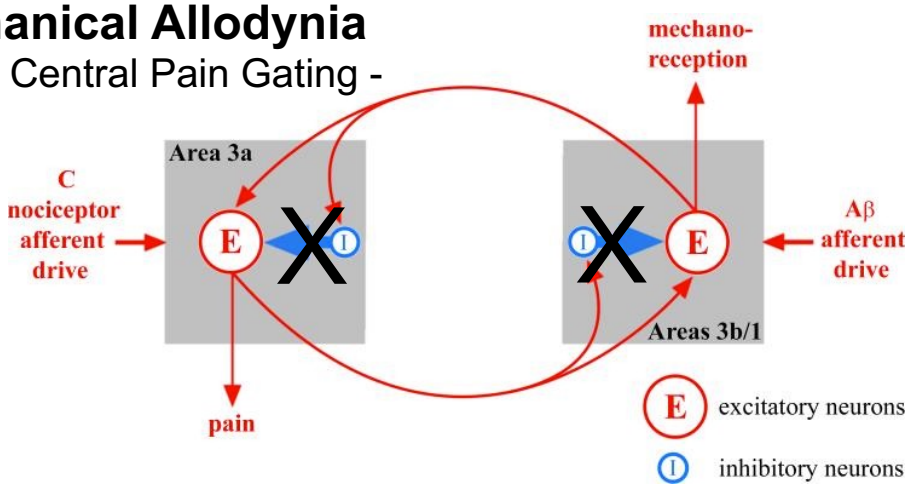




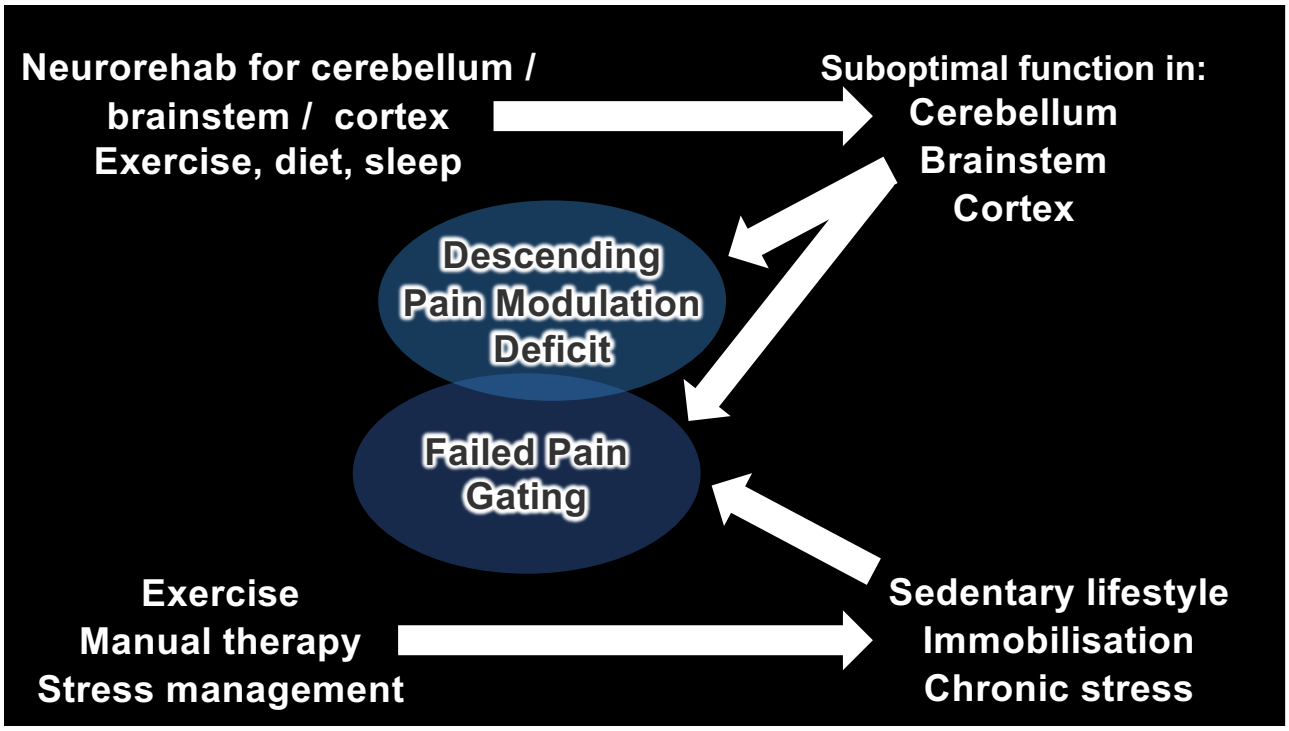
Pain Gate
- Parietal Cortex-

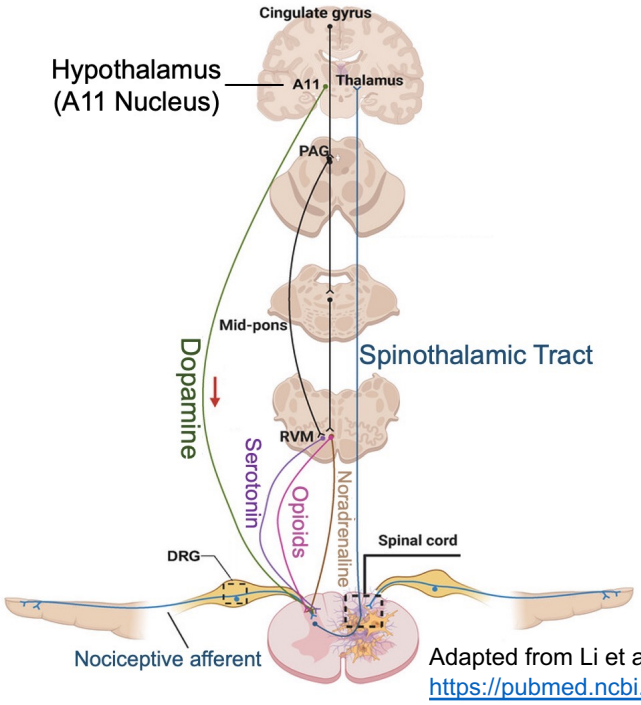
Somatosensory cortex areas 3a and 3b/1 are linked by both excitatory and inhibitory connections. Due to dominance of inhibitory interneurons relative to excitatory interneurons, the overall effect of the connections between areas 3a and 3b/1 is typically inhibitory. www.ncbi.nlm.nih.gov/pmc/articles/PMC4501501/

Mechanical Allodynia
- Failed Central Pain Gating -



In allodynia, the normally inhibitory interaction between areas becomes facilitatory. Now a tactile stimulus could be experienced as painful if vigorous activation of 3b/1 triggers activity in area 3a nociresponsive neurons proposed to underlie slow burning pain. www.ncbi.nlm.nih.gov/pmc/articles/PMC4501501/





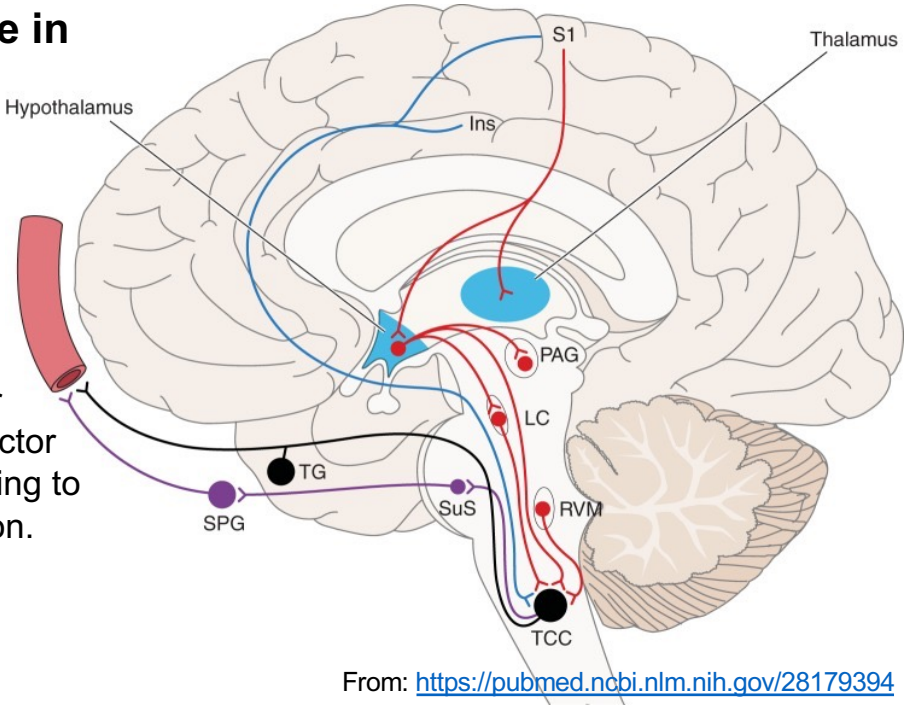
Adapted from Li et al. (2025)
https://pubmed.ncbi.nlm.nih.gov/40787071

Hypothalamic Role in Endogenous Pain Modulation

- Modulates pain through dopaminergic projections from its A11 nucleus.
- ➢ Contributes to descending pain modulation (dopaminergic via D2 receptors) by exciting the interneuron of the spinal cord pain gates.
- ➢ It appears that the A11 nucleus can switch to pain facilitation when dysregulated (via D1 receptors), contributing to chronic pain states.

Hypothalamic Role in Endogenous Pain Modulation

- Modulates trigeminal nociception.
  - A11 nucleus dysfunction is thought to be a major pathophysiological factor in migraine, contributing to trigeminal sensitisation.



From: https://pubmed.ncbi.nlm.nih.gov/28179394

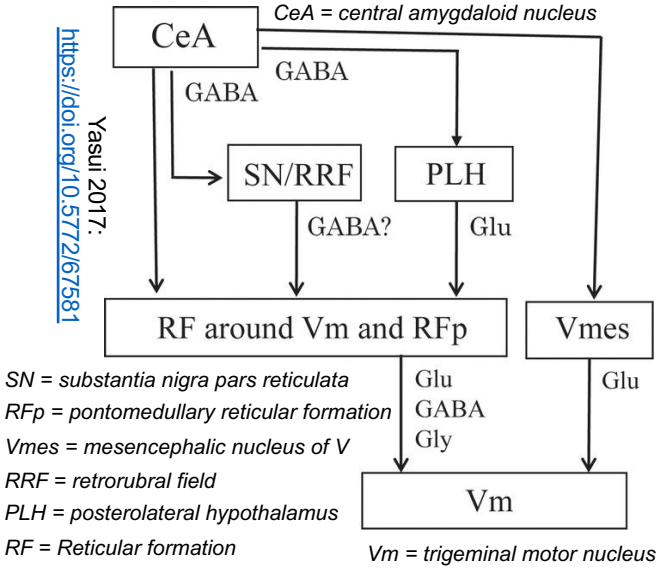
TMD & Limbic System Dysregulation

Temporomandibular disorder

- CGRP-mediated trigeminal sensitisation
- Neuroinflammation and subsequent dysregulation in:
  - Hypothalamus
  - Limbic system (emotions, fight-or-flight response)
  - Brain regions associated with endogenous pain modulation

Nascimento et al., 2021: https://doi.org/10.1002/jcp.30341

Emotional regulation of jaw muscle activity:



Yasui 2017: https://doi.org/10.5772/67581

Chronic stress
= jaw clenching & teeth grinding

The Jaw and the Limbic System

TMJ dysfunction can cause neuroinflammation and subsequent dysregulation in limbic structures.

Limbic overactivity can produce too much activation of jaw muscles.
➢ TMJ dysfunction

Consider treating the TMJ in chronic pain and headache patients.

Even if the TMJ dysfunction is subclinical / the patient isn't experiencing any jaw pain.

Emotion-Driven Pain

- Over-activation of the anterior cingulate cortex (ACC) is common in patients with chronic stress and anxiety.
- Some pain facilitatory projections from the anterior cingulate cortex (ACC) synapse directly onto neurons in the spinal cord dorsal horn!

  ➢ Emotion-driven pain amplification occurs even if the normal anti-nociceptive mechanisms are working well.

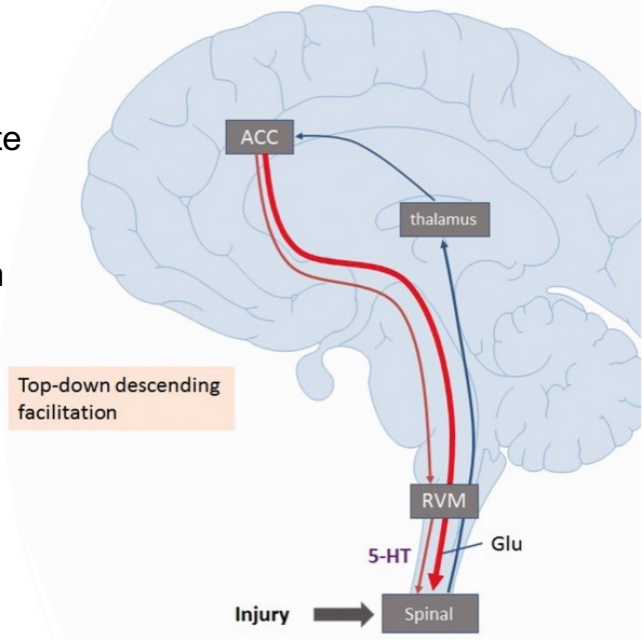


Image from: Zhuo M. (2017). Descending facilitation. Molecular pain, 13, 1744806917699212. https://doi.org/10.1177/1744806917699212

Emotion-Driven Pain

- Reduce their anxiety, reassure them.
- Be nice to them, make them feel like you are listening to them.
- Make visiting your practice a pleasant experience.

Embrace contextual effects as much as possible!

….. after all, it doesn't matter how we change brain function to be more anti-nociceptive!

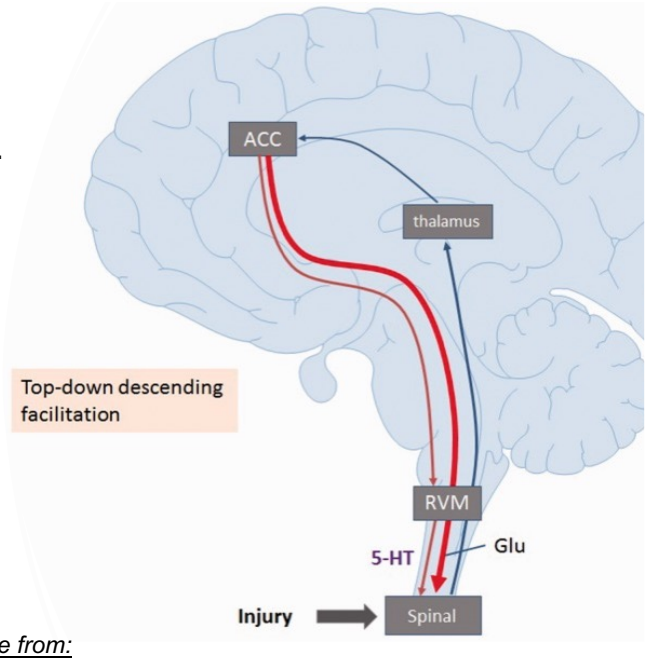


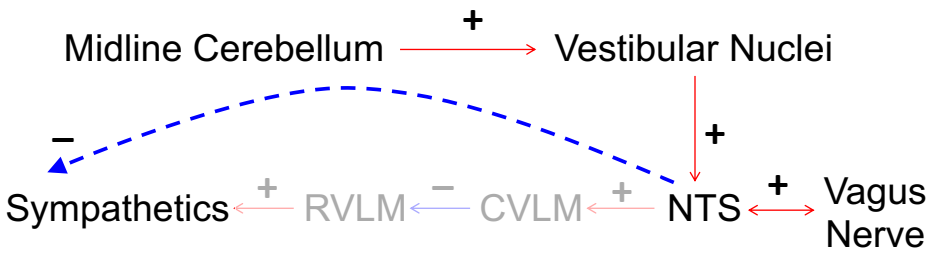
Image from:
Zhuo M. (2017). Descending facilitation. Molecular pain, 13, 1744806917699212. https://doi.org/10.1177/1744806917699212

Autonomic Dysregulation in Nociplastic Pain

- Many studies suggest dysregulation of the autonomic nervous system in chronic pain (e.g. PMID: 35420372, PMID: 28389378, PMID: 34286166).
- "Non-pharmacological therapeutics, such as vagus nerve stimulation, mindfulness-based meditation, and exercise, have shown promise in alleviating painful symptoms of joint diseases, and these interventions may be partially mediated through the autonomic nervous system."
  (Yeater et al., 2022: PMID: 35420372)

Autonomic Dysregulation in Nociplastic Pain

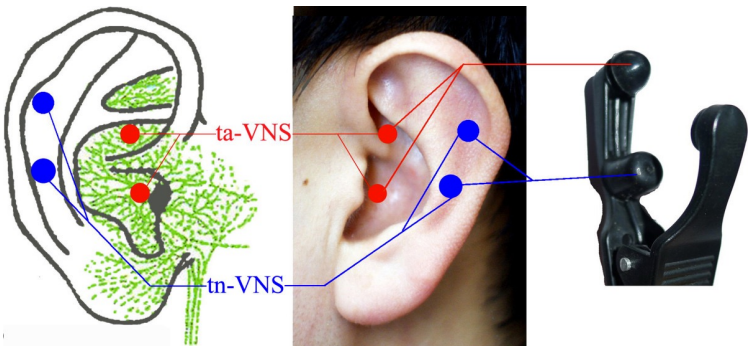
- Sympathetic modulation through vestibular & vagus nerve stimulation -



- Appropriate vestibular and/or vagus nerve stimulation can attenuate sympathetic activity.
  - ➢ Might help to reduce pain in chronic pain patients.

NTS = nucleus tractus solitarius

Vagus Nerve Stimulation



- Transcutaneous auricular vagus nerve stimulation
- Auricular acupuncture

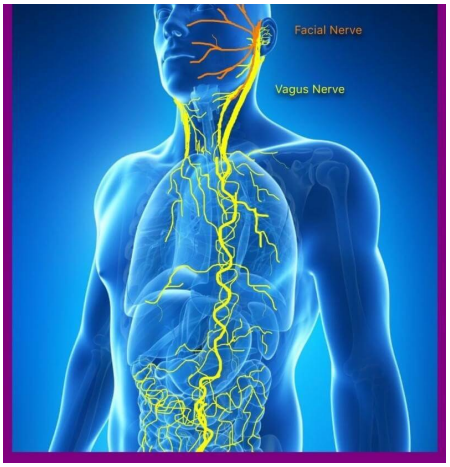
(blue points are site of sham/control stimulation)

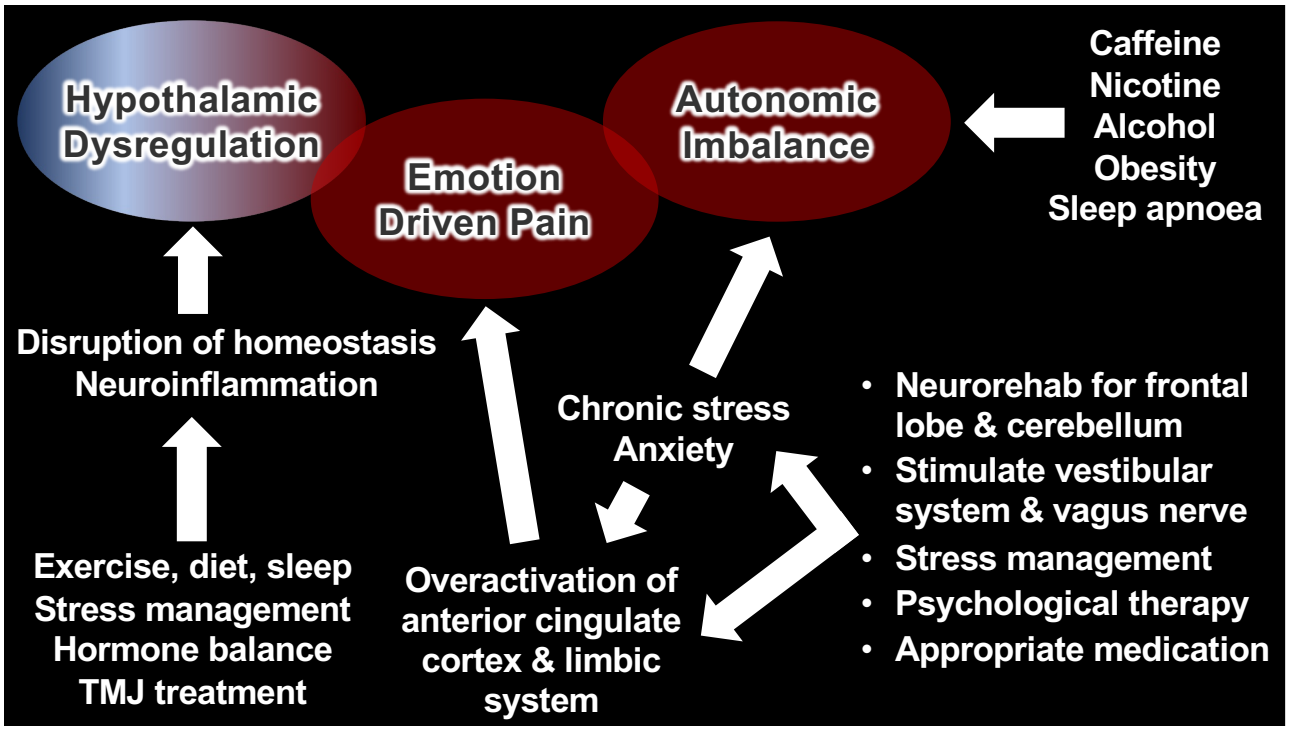
http://www.biomedcentral.com/1472-6882/12/255

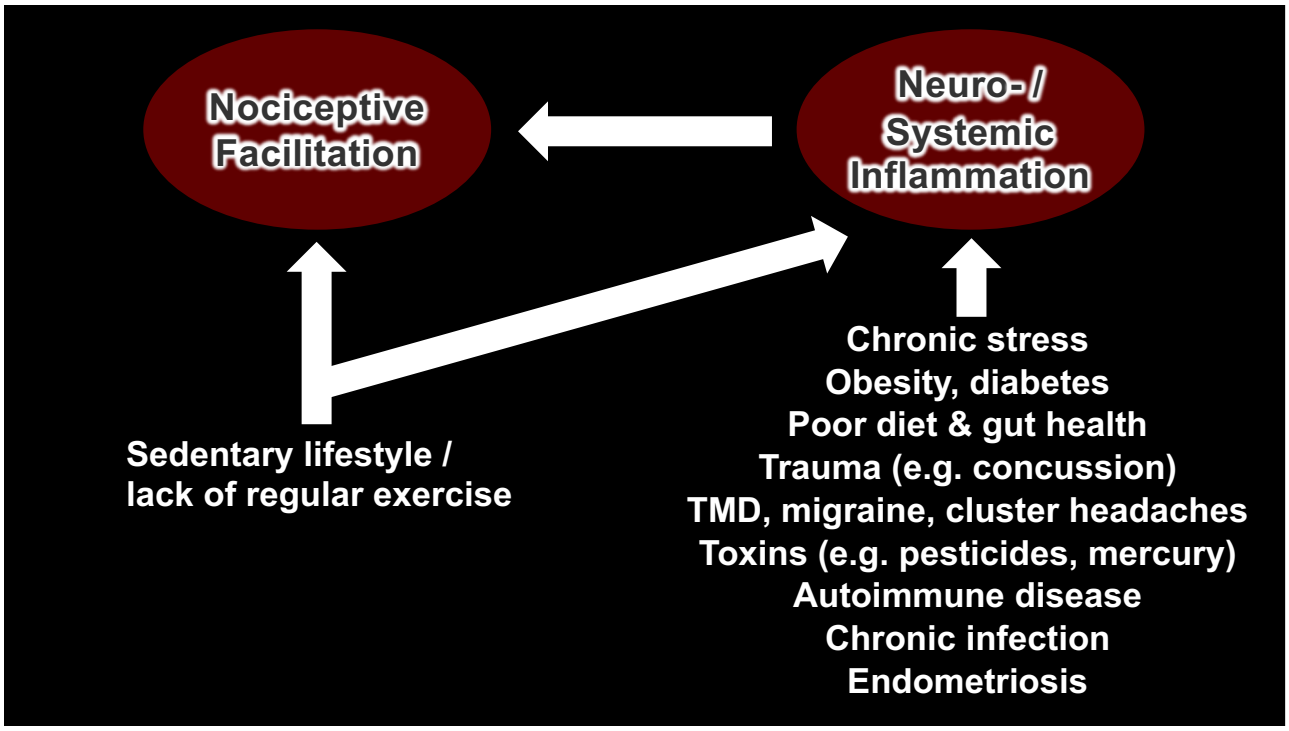
- Non-invasive electrical stimulation of the cervical vagus nerve.

Other Ways to Stimulate the Vagus Nerve

- Singing
- Laughing
- Breathing exercises
- Meditation
- Yoga, Tai Chi
- Gargling
- Chewing gum
- Probiotics & fish oil supplements
- Intermittent fasting







Exercise training:

- capable of reducing pain in chronic pain syndromes
- leads to increased pressure pain thresholds (PPTs)
- more effective than non-exercise interventions, such as pain education, massage and stress management, for reducing pain sensitivity
- **effect was greater when exercising the body part that hurts** compared with exercising remote regions

https://pubmed.ncbi.nlm.nih.gov/33253748

Motor control exercises may lead to greater reductions in pain and disability than strengthening exercises.

https://pubmed.ncbi.nlm.nih.gov/33609357

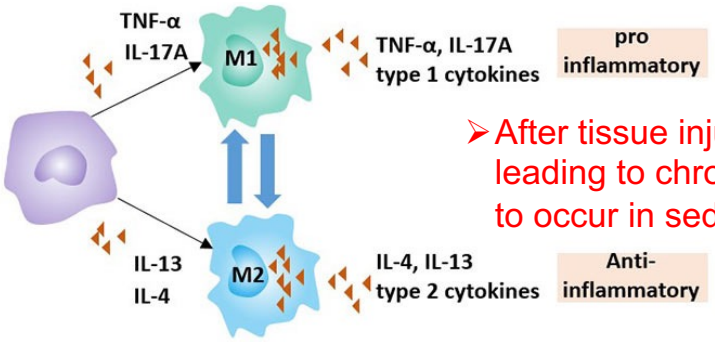
Sedentary cervicogenic headache patients had lower suboccipital PPTs than regularly active cervicogenic headache patients. https://pubmed.ncbi.nlm.nih.gov/36450870

Pain sensitivity is reduced by exercise training

Effect of regular exercise on local post-injury pro-inflammatory cytokine release

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6097240/

- Physical activity levels modulate phenotype of macrophages in muscle (M1 vs. M2 macrophage phenotype).
  - Regularly physically active = more M2 (release anti-inflammatory cytokines)
  - Sedentary = more M1 (release pro-inflammatory cytokines)



➢ After tissue injury, pain sensitisation leading to chronic pain is more likely to occur in sedentary individuals.

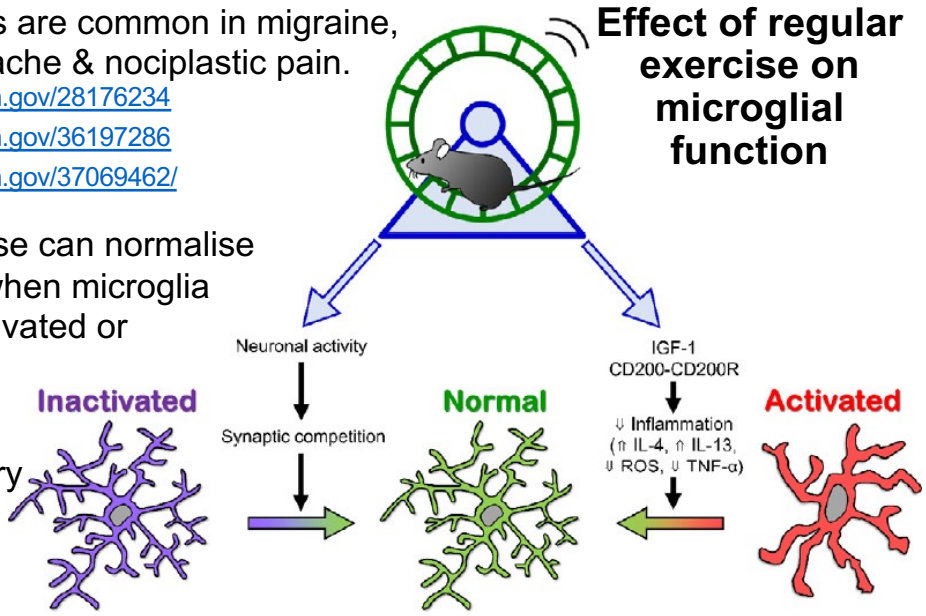
Effect of regular exercise on systemic pro-inflammatory cytokine levels

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6097240/

- Systemically circulating immune cells, i.e., peripheral blood mononuclear cells (PBMCs), are highly plastic and secrete inflammatory or anti-inflammatory cytokines based on their phenotype.
- Fibromyalgia patients have greater release and higher concentrations of circulating inflammatory cytokines.
- These levels reduce after several months (4-8 months) of exercise training – *these studies did aquatic exercise.*
- More studies need to be done, but it appears that in chronic pain patients and in healthy individuals exercise can alter systemic cytokines and reduce systemic inflammation.

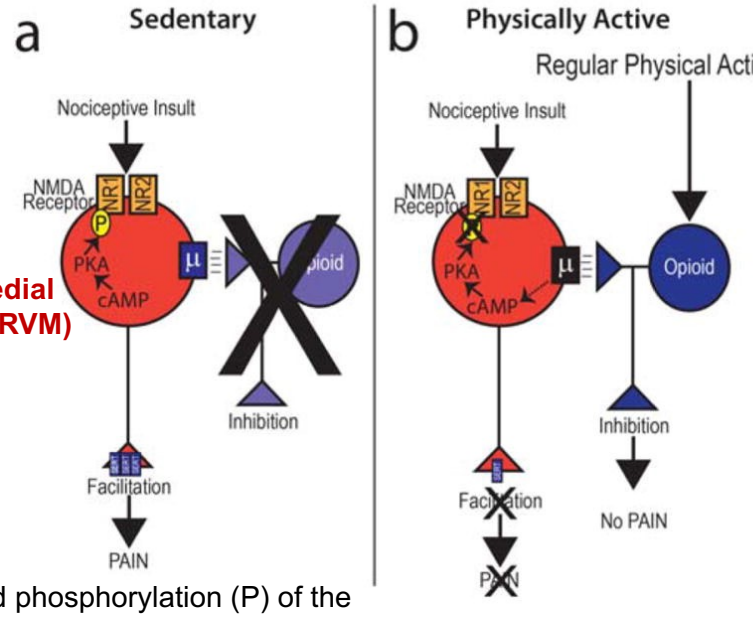
- Overactive glial cells are common in migraine, posttraumatic headache & nociplastic pain.
  https://pubmed.ncbi.nlm.nih.gov/28176234
  https://pubmed.ncbi.nlm.nih.gov/36197286
  https://pubmed.ncbi.nlm.nih.gov/37069462/
- It seems that exercise can normalise microglial function when microglia are either highly activated or highly inactivated.
- Regular exercise reduces inflammatory cytokines and increases anti-inflammatory cytokines.

Effect of regular exercise on microglial function



From: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7716033/

Rostral Ventromedial Medulla (RVM)



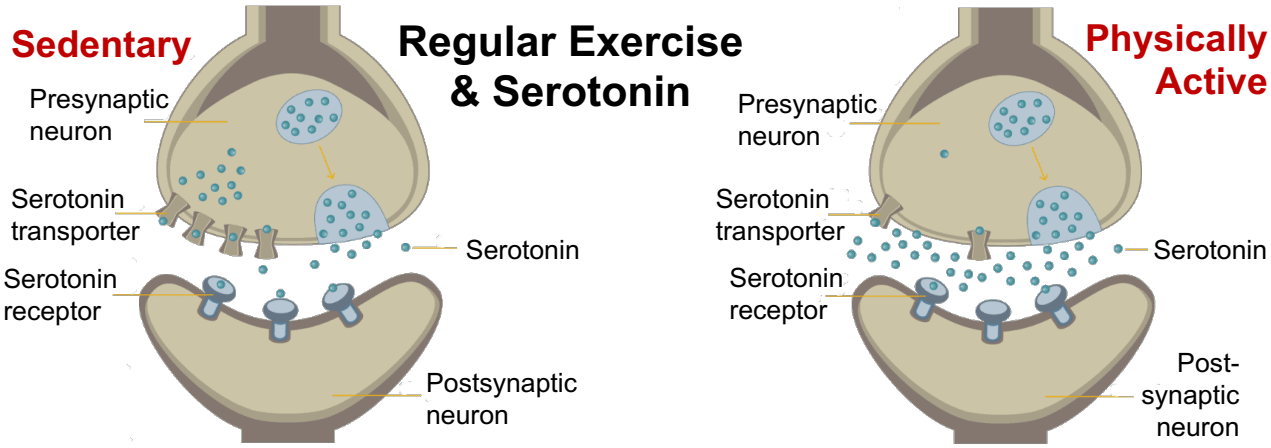
Increased phosphorylation (P) of the NR1 subunit of the NMDA receptor:
➢ ↑↑ excitability, hyperalgesia & pain

Sedentary individuals have less opioid tone in their nervous system

Regular exercise activates mu-opioid receptors on the pain facilitation neurons of the RVM
➢ ↓↓ cell excitability due to reduced NR1 subunit phosphorylation

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6097240/

Regular Exercise & Serotonin



Sedentary individuals have increased expression of the serotonin transporter (SERT).

SERT removes serotonin from synaptic cleft

- Excessive SERT expression = impaired serotonergic endogenous pain modulation

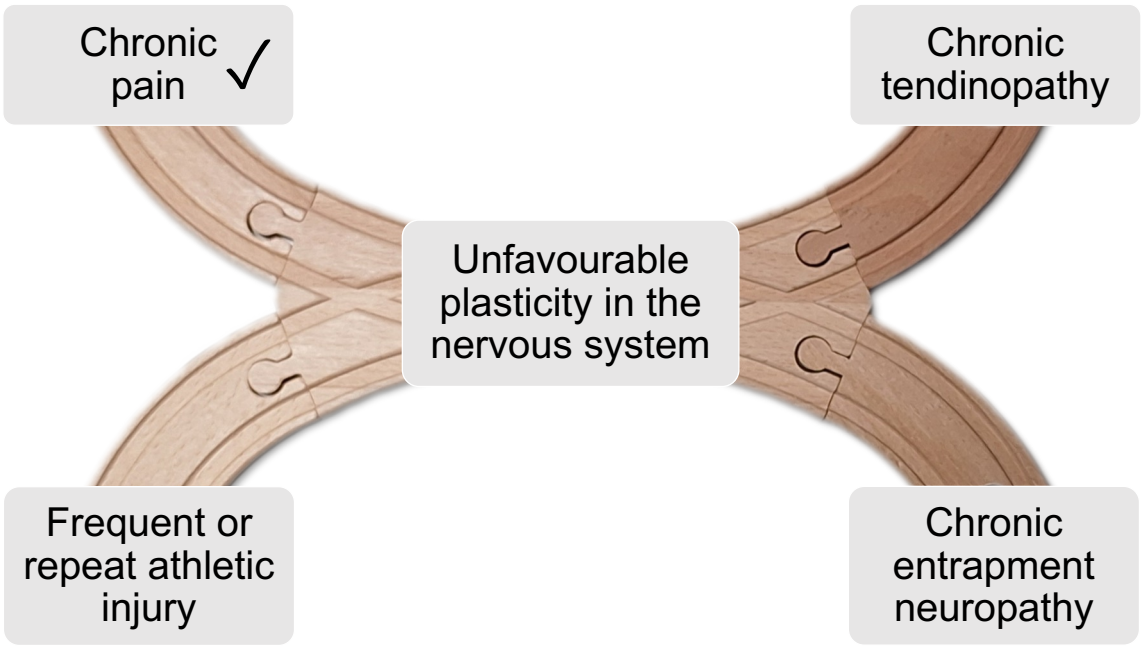
Images adapted from:
https://genomind.com/providers/slc6a4-and-ssris-spotlight

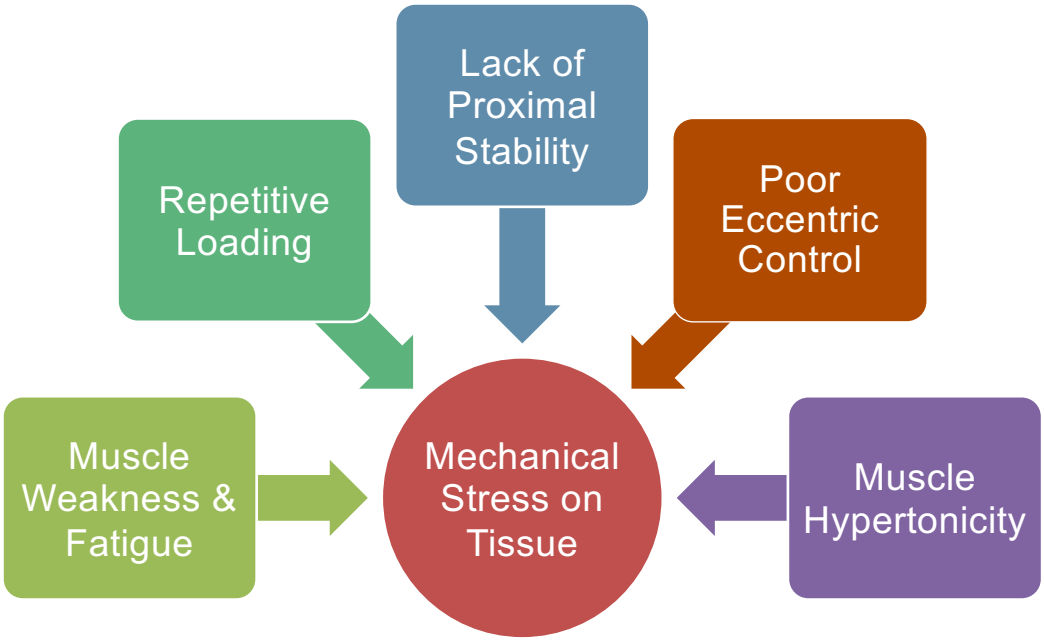
Communication Strategies for Chronic Pain & CS

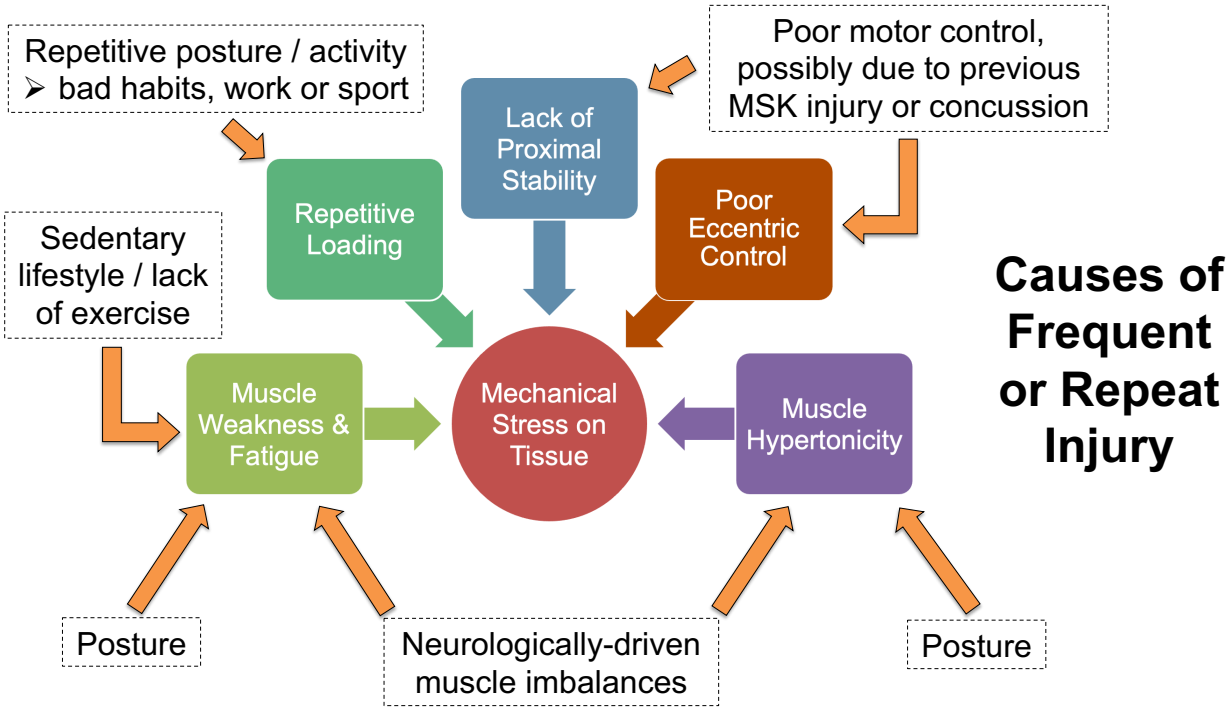
- Affective communication (Wiering et al., 2018; PMID: 30200955)
  - ✓ Empathy, reassurance, support
  - ✓ Attentive listening
  - ✓ Taking enough time
  - ✓ Building patient's trust in the physician's competence
  - ✓ Giving patients the feeling that the physician is doing all they can
- Facilitating effective coping mechanisms (Litt & Tennen, 2015; PMID: 26399377)
- Addressing false pain beliefs (Babadağ et al., 2015; PMID: 26320677)

Aim - reassure the patient that:

- Their pain is understood, validated and not a sign of continued damage
- Moving the painful body part is beneficial
- The practitioner is competent and doing all they can

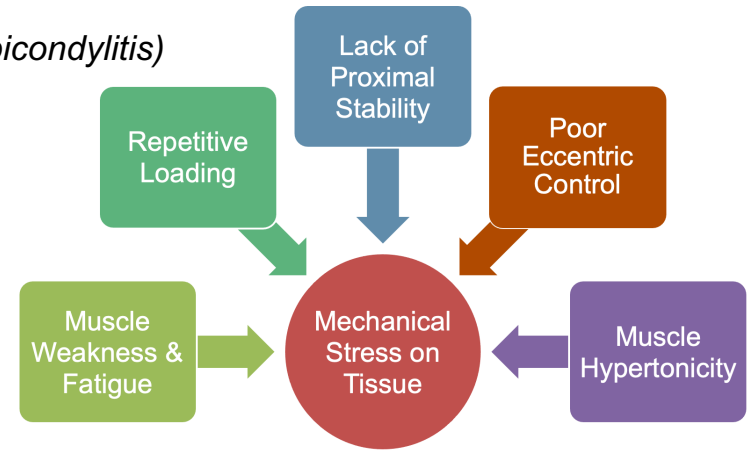

Chronic pain ✓
Chronic tendinopathy
Unfavourable plasticity in the nervous system
Frequent or repeat athletic injury
Chronic entrapment neuropathy


Lack of Proximal Stability
Repetitive Loading
Poor Eccentric Control
Muscle Weakness & Fatigue
Mechanical Stress on Tissue
Muscle Hypertonicity



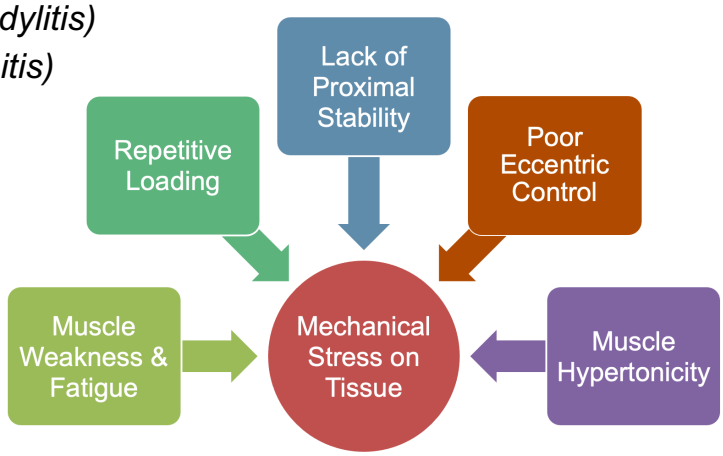
MSK Injuries
- Muscle Weakness & Fatigue -

- Predisposes to tendinopathy in the tendons of muscles that are naturally weaker than their antagonist, e.g.:
  - ➢ rotator cuff
  - ➢ wrist extensors *(lateral epicondylitis)*
  - ➢ gluteus medius / minimus *(+/- ITB syndrome)*
  - ➢ distal hamstring
  - ➢ tibialis posterior *(posterior shin splints)*
  - ➢ tibialis anterior *(anterior shin splints)*



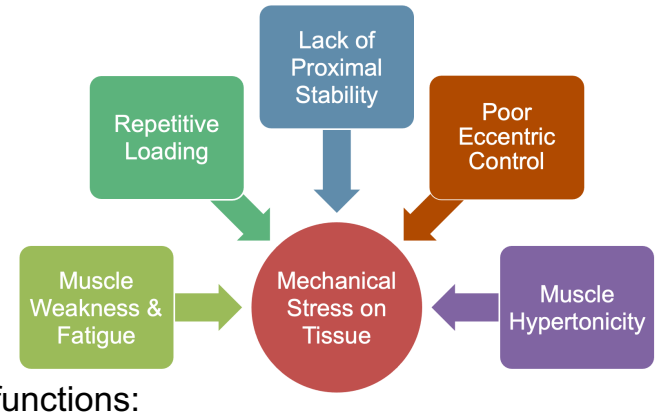
MSK Injuries
- Muscle Hypertonicity -

- Predisposes to tendinopathy in the tendons of muscles that are naturally stronger than their antagonist, e.g.:
  - wrist flexors *(medial epicondylitis)*
  - quadriceps *(patellar tendonitis)*
  - calf *(Achilles tendonitis)*
- Predisposes to entrapment neuropathies in muscles that are naturally stronger than their antagonist, e.g.:
  - brachial plexus
  - ulnar nerve
  - median nerve



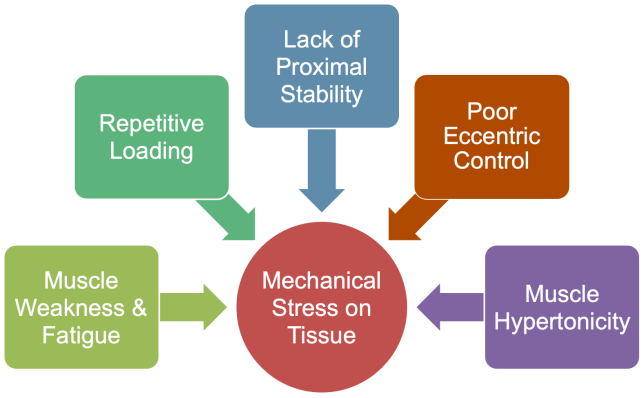
MSK Injuries
- Proximal Stabilisation -

- Optimal motor control requires feedforward (predictive) activation of core & proximal limb stabilisation.
- Lack of proximal stabilisation means that the more distal limb muscles have to take on stabilising functions:
  - Increased co-contraction or adaptive-reactive activation of local agonist and antagonist muscles
  - Too much stiffness / tone in the muscles
    - Predisposes to entrapment neuropathies
  - Chronically increased load on tendon
    - Predisposes to tendinopathy when muscles are repetitively loaded



MSK Injuries
- Eccentric Control -

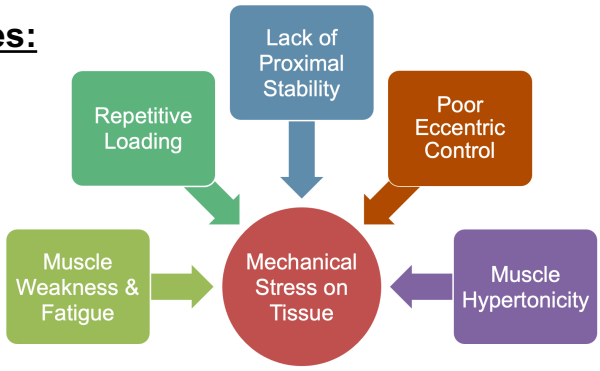
- Antagonist eccentric contractions are vital for control, deceleration, joint stabilisation and therefore injury prevention during movements.
- Eccentric contractions need more inhibitory and fine control.
  - ➢ Cerebellar Purkinje system & indirect pathway of the basal ganglia facilitate fine motor control through surround inhibition.
- The majority of MSK injuries occur during the eccentric phase of loading / due to insufficient eccentric control.



Chronic MSK Injury: Treatment

<u>Determine & remove underlying causes:</u>

- Repetitive posture / activity
- Poor posture
  - ➢ usually promotes flexor tone
- Lack of exercise
- Poor motor control & neurologically-driven muscle imbalances
  - ➢ **Neuromuscular rehab needed**



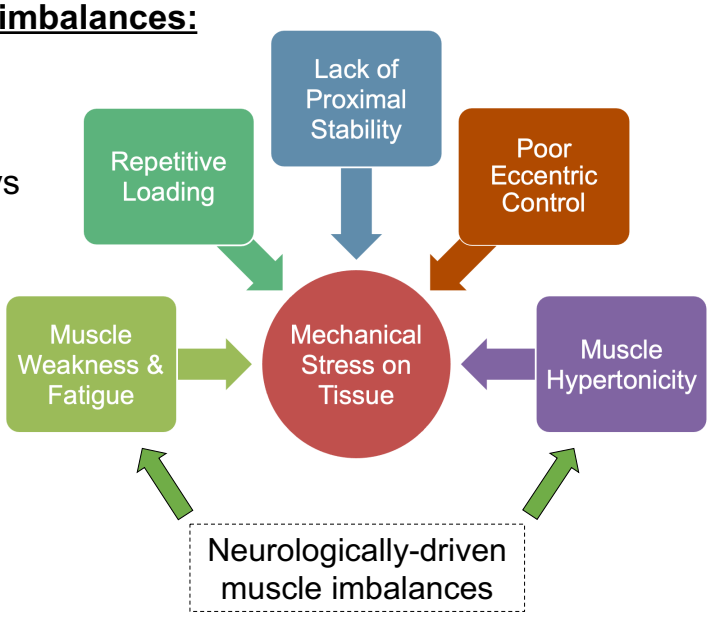
Chronic MSK Injury: Treatment

Neurologically-driven muscle imbalances:

Look for deficits in:

- Vestibulospinal pathways
- Cortico-reticulospinal pathways

Neurorehab of the cerebellum, cortex & brainstem may be needed.

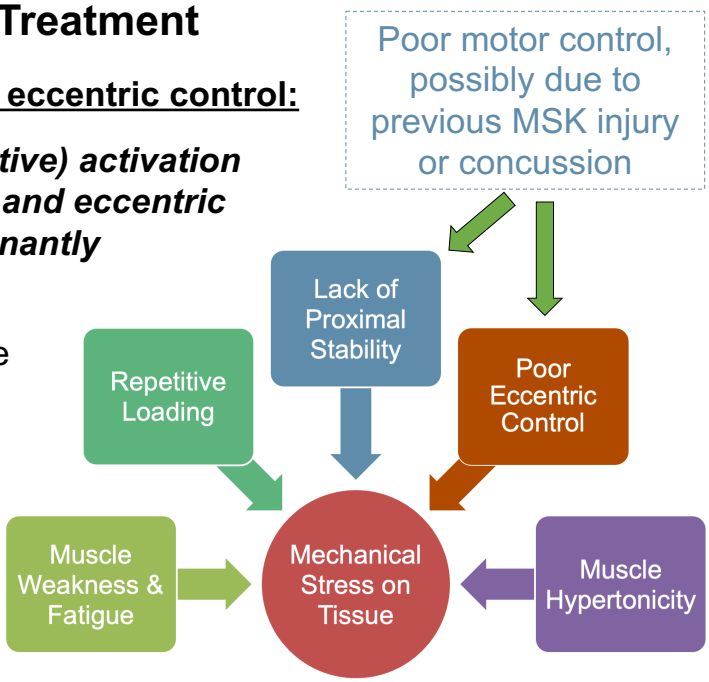


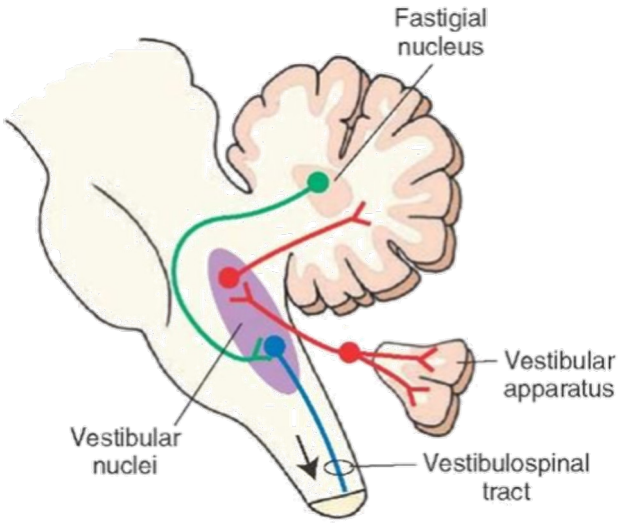
Chronic MSK Injury: Treatment

Lack of proximal stability / poor eccentric control:

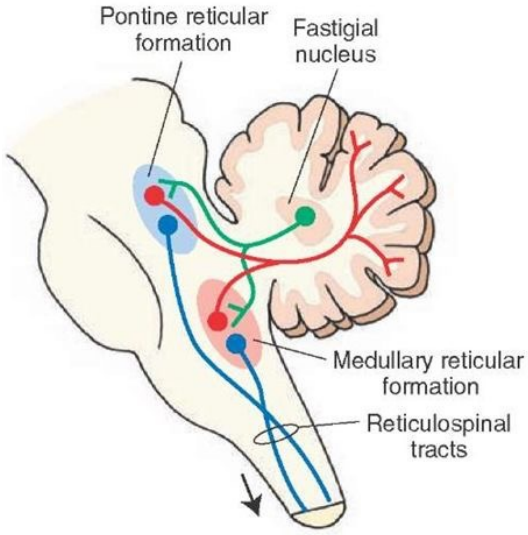
Automatic (feedforward / predictive) activation of proximal stabilising muscles and eccentric muscle contractions is predominantly the role of the cerebellum.

- It appears that all regions of the cerebellum contribute to this.
- Make sure all functions of the cerebellum are rehabilitated for optimal motor control.





Cerebellar motor control is better than cortical motor control!



The cerebellum is really good at activating all the necessary postural and limb stabilising functions at exactly the right time.

Reduced Cerebellar Activity After Musculoskeletal Injury

- Increased activation of the primary motor cortex and decreased activation of the cerebellum after anterior cruciate ligament (ACL) reconstruction and participation in a traditional concentric-based rehab programme.

  Lepley et al., 2017 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5496707

- Motor strategies that use an internal focus of attention and rely on conscious *(cortical)* rather than unconscious *(sub-cortical)* control of movement are often adopted after injury.
  - ➢ Less efficient & confer greater injury susceptibility.
  - ➢ More likely to break-down when fatigued or under stress.

  Grooms et al., 2015 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4641538

Increased Risk of MSK Injuries After Concussion

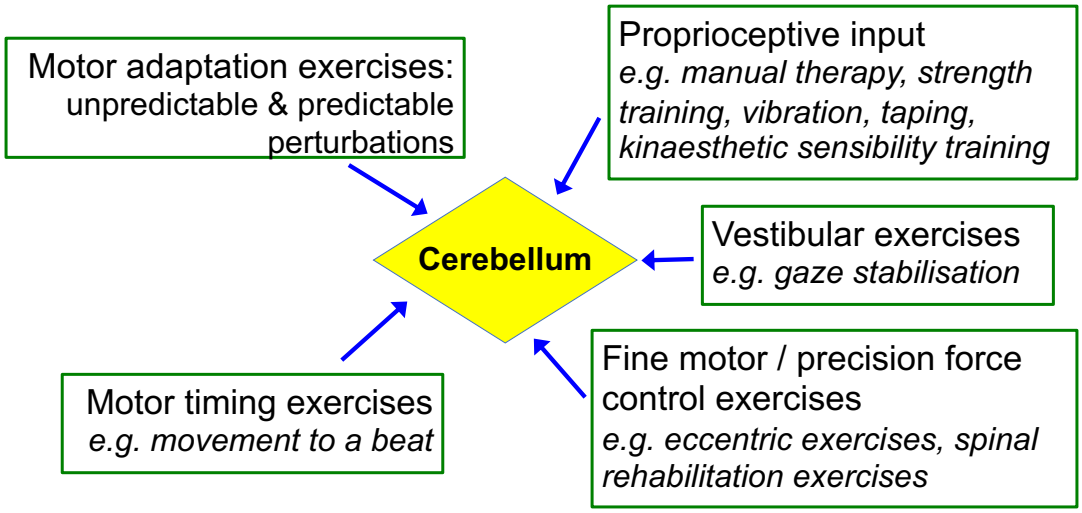
(see https://pmc.ncbi.nlm.nih.gov/articles/PMC7987572 for review of literature)

Cortical inefficiency and cerebellar/vestibular injury after concussion may mean that for the same motor task a concussed athlete has increased motor cortex activation and less subcortical (cerebellar) activation.

- More conscious and less unconscious/automatic control of movement.
- Likely to tie up cortical resources leading to inefficient processing of sensory inputs and concurrent motor demands in complex athletic environments.
- Motor programmes more susceptible to breakdown under dual-tasking conditions.

Examination & Neurorehab of the Cerebellum

Rehabilitate all functions of the cerebellum for optimum motor control, postural stabilisation and pain modulation:



Examination of the Cerebellum

Intermediate / lateral:

- Rapid alternating movements (hands & feet)
- Finger-to-nose

Midline:

- Romberg's
- Fukuda step test
- Tandem gait

Rapid Alternating Movements – Hands

- Less rapid or less well coordinated movement of R arm/hand indicates dysfunction in R Cb (this could be due to underactivity or overactivity)
- Test with elbows straight and elbows bent.
- Breakdown of rapid alternating movements = dysdiadichokinesia (DDK)

Rapid Alternating Movements – Feet

- Less rapid or less well coordinated movement of R foot indicates dysfunction in R cerebellum *(this could be due to underactivity or overactivity)*
- Listen to the tapping rhythm and observe the movement.
- Can be useful to ask the patient which side they thought was less good (applies to most neurological tests).

Finger to Nose Test

- Tests the ipsilateral cerebellum and contralateral cortex (especially parietal cortex).
- Stopping short of target, intention tremor, and then possibly still missing the nose on the ipsilateral side, indicates an underactive Cb.
- Overshooting the nose and mildly crashing the finger in the face indicated an overactive Cb.
- Observe for ‘cheating’ head movements towards the finger.

Cerebellar exercises – intermediate zone: rapid motor adaptations

- Arm movements using arm ipsi to ↓ cerebellum with adaptation to perturbation:

  Patient performs a figure 8 movement with their arm or leg around assistant's forearms; Assistant moves forearms unpredictably.

Cerebellar exercises – intermediate zone: rapid motor adaptations

- Arm movements using arm ipsi to ↓ cerebellum with adaptation to perturbation:

  Patient moves their arm or leg in circular motion while the assistant disrupts the motion unpredictably.

Cerebellar exercises – intermediate zone: rapid motor adaptations

- Make it fun and ideally relevant to a patient's normal recreational activities.

Cerebellar exercises – lateral zone: motor prediction

- Catching a ball with hand ipsi to ↓ cerebellum – ideally using only peripheral vision (visual signal relayed to contralateral parietal cortex).

Cerebellar exercises – lateral: motor timing

- Alternating hand tapping to metronome beat with hand ipsilateral to dysfunctional cerebellum.

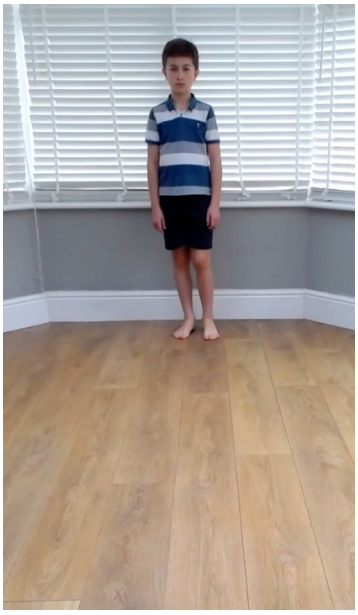
Romberg's Test

- Stand behind the patient and be ready to catch them if there is any possibility they might fall.
- Look for general unsteadiness and predominant direction of sway. R sway indicates:
  - underactivity of R cerebellum
  - or overactivity of L cerebellum
- Using a foam balance pad can make it easier to see direction of sway.

Fukuda Step Test

- Stand behind the patient if they are unsteady.
- Keep quiet during test to avoid auditory-spatial cues.
- Look for turning / sidewards movement over approx 30 steps. Towards R indicates:
  - underactivity of R cerebellum
  - or overactivity of L cerebellum
- If patients walks forward during the test, it is <u>not</u> usually indicative of a neurological problem; just a normal consequence of stepping.

Tandem Walking



- Patient attempts to walk heel to toe in a straight line.
- If they are good at walking forwards, ask them to walk backwards as well.
- Walk behind the patient and be ready to catch them if there is any possibility they might fall.
- Look for unsteadiness / deviation from straight ahead. Falling / deviating to R indicates:
  - underactivity of R cerebellum
  - or overactivity of L cerebellum

Spinning to the Right

- Activates right midline cerebellum & vestibular system.
- Be careful, some patients fatigue easily.
- Some patients find it easier to spin with eyes open, some find it easier with eyes closed.

R VOR Cancellation Spin

- Activates right midline cerebellum & vestibular system.
- Good for patients who fatigue easily on ‘normal’ spinning.
- Helps to recalibrate an overactive cerebellum (Purkinje neuron dysfunction).

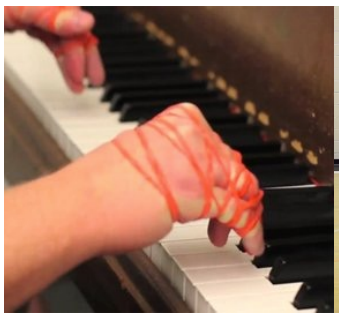
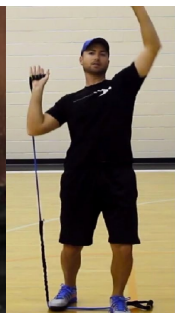
Gaze Stabilisation

- Recalibrates faulty vestibulo-ocular reflexes (VORs).
- Focus eyes straight ahead on dot/ letters.
- Gradually make the exercise harder by reducing stability.
- Rotation, flexion / extension & lateral flexion stimulates cerebellum bilaterally.
- Good for both underactive an overactive cerebellum.

Cerebellar Rehabilitation: Predictable Perturbations

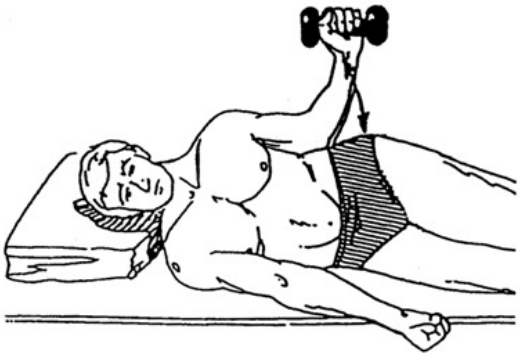
- Challenge feedforward systems by performing already learned movements with **predictable perturbations**.



Cerebellar Rehabilitation: Eccentric Exercises

- Eccentric muscle contractions can increase cerebellar activation and decrease cortical activation, thus potentially reversing a suboptimal cortical-subcortical state and conferring protection against MSK injury.
- Eccentric training has also been shown to improve flexibility.



Exercises to facilitate automaticity (cerebellar control) of motor programs:

- Challenge the system with dual-tasking (cognitive, visual-spatial or sensory challenges).
- Improve core stability.
- Restore reliance on proprioceptive rather than visual cues through eyes-closed or stroboscopic training.
  - ➢ Visual dependence for movement occurs both after MSK injury and concussion and is associated with increased motor response latency and a more 'cortical' control of movement.

  - ➢ *Less effective dynamic postural stabilisation and higher risk of MSK injury.*

Grooms et al., 2015
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4641538

Practicum: Examination & Rehab of the Cerebellum

Midline Zone	Intermediate / Lateral Zone
Romberg's	Rapid alternating movements: arm pronation / supination
Fukuda step test	Rapid alternating movements: foot tapping
Tandem Walking	Finger-to-nose

Try to rehabilitate your practicum partner using some of the following exercises, and re-test to measure their success.

Midline Zone	All Zones	Intermediate / Lateral Zone
Spinning (+/- VOR cancellation)	Proprioceptive stimulation	Arm / leg circles with adaptation to perturbation
Gaze stabilisation	Eccentric exercises	Ball catching in peripheral field of view
Tandem walking	Motor control exercises	Figure 8 rapid movement adaptations
Balance training	Spinal rehab exercises	Motor timing exercises
	Dual task training	Predictable perturbations