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Back and neck pain

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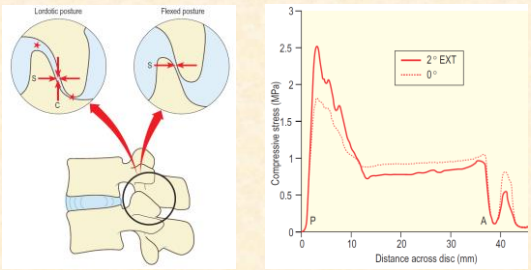
Back and neck pain

- Functional (postural) backache
- Non-specific back pain
- Specific back pain
  - Where are the nerves?
  - Pain provocation & blocking studies
- Discogenic back pain
- Nerve root pain
- Neck pain

'Functional' backache

- Postural habits can generate stress concentrations within innervated tissues (esp. discs and apophyseal joints)
- These could give rise to pain 'like a stone in your shoe', in the absence of any tissue changes

Lordotic postures concentrate stresses in the neural arch and posterior annulus

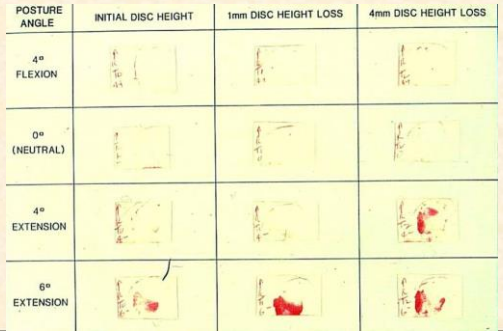


- Flexion removes these stress concentrations

Adams & Hutton (1980). JBJS (Br) 62: 358-62

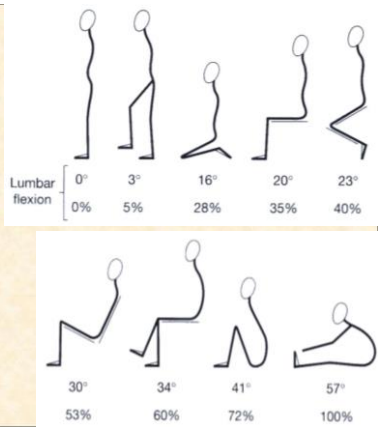
Facet joint stresses depend on posture and disc height

Dunlop et al. 1984. J Bone Joint Surg [Br] 66(5): 706-10)



"Comfortable" postures reduce lumbar lordosis

Dolan et al. Spine 1988;13:197-201.



Non-specific low back pain

- Low back pain "not attributable to a recognisable, known, specific pathology " (1)
- Concept leads to 'non-specific treatment' (1)
  - 'use of clinical imaging for diagnosis should be restricted'
  - 'mechanical factors .. probably do not have a major.. role'
  - 'mechanism of action of most treatments is low'
  - 'self management, with .. support, .. recommended'
  - review alternates between 'n-s' and 'all' back pain
- Fits within the 'Bio-psycho-social model' of back pain (2)

1. Balague et al. Lancet Oct 7th, 2011
2. Waddell: The Back Pain Revolution (2nd Ed). 2002. Churchill Livingstone

Hidden meaning in "non-specific low back pain"!

- 'Non-specific low back pain' really means that …
- the patient has back pain, .. AND …
- we do not know the cause, … AND ..
- we think it's not serious, and likely to get better (1)

1. Mulholland RC (2013). Eur Spine J 22(2): 250-259.

Uses of 'non-specific back pain'

- Convenient label for patient management/care (but not a diagnosis in the conventional sense)

Abuses of 'non-specific back pain'

- Using the term to refer to *all* back pain
- Using it as an outcome measure in epidemiology (boosts importance of risk factors for pain behaviour - final common pathway for all types of back pain)
- Using the term to de-medicalise *all* back pain

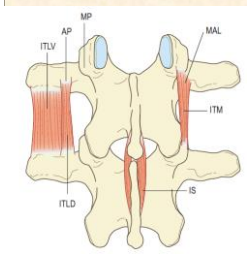
Psychosocial factors in low back pain (1)

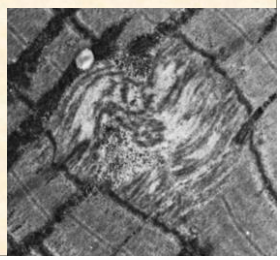
- The psyche (mind) influences all aspects of behavior, including reporting 'pain' and responding (or not) to treatment
- Fear, anxiety & depression are particularly important factors
- Social factors (such as a demanding workplace, or workers' compensation) also influence pain behaviour
- Psychosocial factors are good predictors of 'trivial' 1st-time back pain, but physical factors (e.g. mobility, long flat back) are better predictors of 'serious' 1st-time back pain (2).

1. *Waddell: The Back Pain Revolution (2nd Ed). 2002. Churchill Livingstone*
2. *Adams et al. (1999). Spine 24 2497-2505*

Back muscle injuries

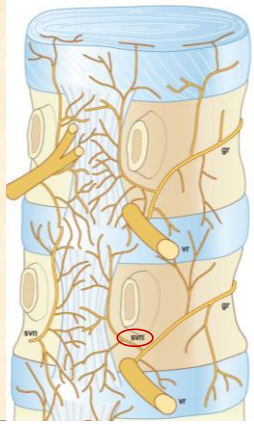
- Common (?), heal quickly, recur (?)
- Pain tends to be localised, and rarely chronic
- (Eccentric) injuries to back muscles most likely during flexion



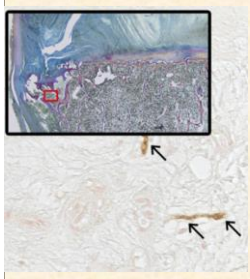


Disc innervation

- **sinuvertebral nerve (svn)** - capable of signalling 'somatic' and 'visceral' pain
- branches within the posterior longitudinal ligament
- penetrates the peripheral posterior annulus (1-3 mm?)
- further ingrowth in degenerated discs?



Nerves in the vertebral endplate



- Nerves are present in most end plate defects, at a higher density than in normal endplates

Fields et al. (2014). Spine J 14(3): 513-21.

Where does specific back pain come from?

- Pain provocation/blocking studies implicate the disc and facet joints (in surgical candidates) (1)
- Sciatica from nerve roots
- Muscle/tendon/ligament injuries may explain localised and transient pain
- 'Sensitisation': slight pressure often reproduces severe pain!

1. Kuslich et al., Orthop Clin N Amer 22 181-7, 1991

Other pain-provocation/blocking studies

- **39%** chronic LBP from internal disc disruption (1)
- **40%** chronic LBP from apophyseal joints (2)
- **13 - 30%** chronic LBP below L5-S1 from SI joints (3)

Difficult (unethical?) to perform these tests in routine practise, so must rely on averaged data from small studies

1. *Schwarzer et al. (1995). Spine;20:1878-83.*
2. *Schwarzer et al. (1995). Ann Rheum Dis;54:100-6.*
3. *Schwarzer et al. (1995). Spine;20:31-7.*

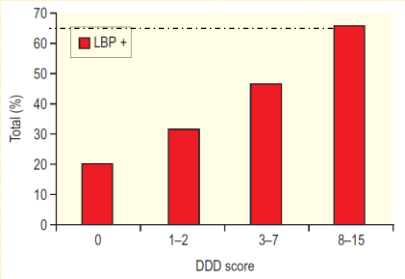
Back pain from the endplate

- Pain was reproduced when fluid was injected into the endplate lesion/defect
- Subsequent fusion surgery (with removal of affected bone) relieved most pain.

Peng et al. (2009). Eur Spine J 18(7):1035-40.

Disc degeneration and back pain

Cheung et al. (2009). Spine 34(9) 934-40.



Degenerative disc disease (DDD) was scored 0-3 and then summed over five lumbar discs

Painful features of disc degeneration

1. Radial fissures

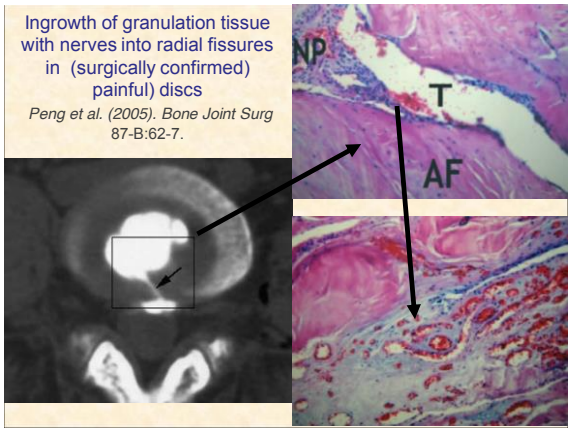
(Videman & Nurminen 2004 Spine 29:2668-76.)

Severity of Annular Tears	Probability Estimates of Frequency of Lifetime Back Pain		
	No Back Pain	Sometimes	Often
Inner	0.75 (0.56–0.94)*	0.16 (0.06–0.26)	0.10 (0.0–0.19)
Outer	0.55 (0.25–0.85)	0.25 (0.15–0.35)	0.20 (0.0–0.40)
Leaking	0.30 (0.05–0.54)	0.28 (0.25–0.31)	0.42 (0.15–0.70)

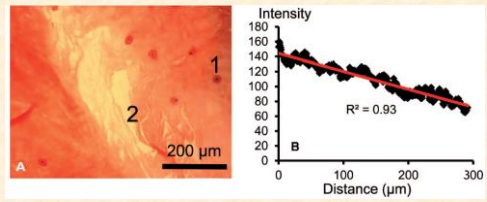
* Mean probability in the class (95% confidence intervals).

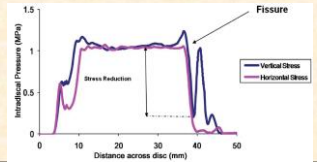
Ingrowth of granulation tissue with nerves into radial fissures in (surgically confirmed) painful) discs

Peng et al. (2005). Bone Joint Surg 87-B:62-7.



Matrix defects permit nerve & blood vessel ingrowth

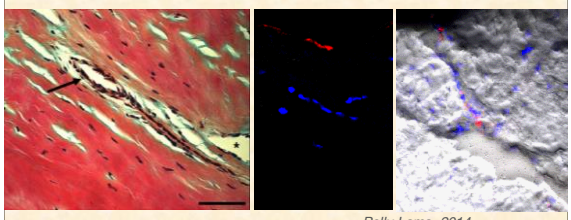




Stefanakis et al. (2012). Spine 37: 1883-91

Radial fissures allow blood vessel & nerve ingrowth

Nerves are then sensitised by inflammation, or infection (3)?



Polly Lama, 2014

1. Adams et al. (2010). Clinical Biomechanics 25 961-71.
2. Stefanakis et al. (2012). Annulus fissures are mechanically & chemically conducive to the ingrowth of nerves & blood vessels. Spine 37 1883-91.
3. Albert et al. (2013). Eur Spine J 22 697-707.

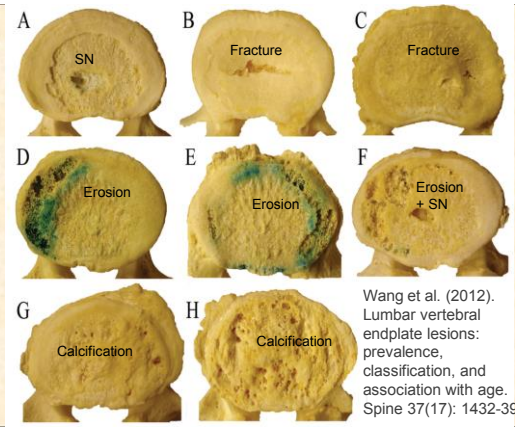
Painful features of disc degeneration

2. Vertebral endplate lesions

Wang et al. (2012). Spine 37 1490-6.

TABLE 1. Associations Between Endplate Lesions and Back Pain History*

Endplate Lesions	Occasional Back Pain		Frequent Back Pain	
	OR	95% CI	OR	95% CI
Schmorl's nodes	1.04	0.45–2.36	2.67	1.34–5.31†
Fracture	1.54	0.47–5.12	1.48	0.45–4.82
Erosion	2.36	0.93–5.97	2.72	1.05–7.06†
Calcification	0.53	0.05–5.55	5.50	1.15–26.23†



Wang et al. (2012). Lumbar vertebral endplate lesions: prevalence, classification, and association with age. Spine 37(17): 1432-39.

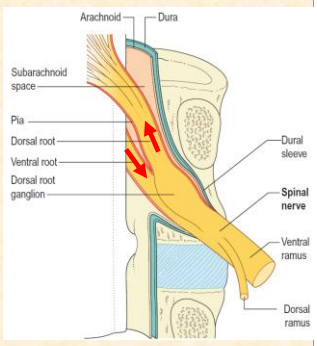
Pain sensitisation in discs

(Olmarker (2008). Spine 33 8 850-5)

- Nucleus pulposus (NP) causes morphological and physiological changes in adjacent neurons (and generates pain behaviour in animals)
- Degenerated NP has > effect than normal NP, and nerve compression/stretching amplifies the pain
- Cytokines involved, especially TNFα, but their role may vary with time
- Systemic use of TNFα blockers *can* help sciatica, but only in initial stages? And what about side-effects?
- NP within a radial fissure induces back pain from neurons in the outer annulus fibrosus.
- Pain sensitisation in humans confirmed by Kuslich etc

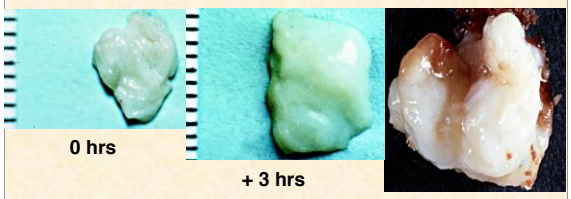
Nerve roots

- Spinal cord is 'cauda equina' (horse's tail) below L1-2.
- Spinal nerves (each with 2 'roots') exit bilaterally via the intervertebral foramen
- Dorsal root = afferent
- Ventral root = efferent
- Each nerve divides into ventral & dorsal rami

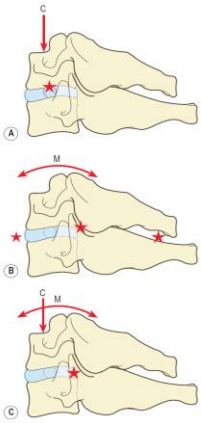


Disc herniation & sciatica

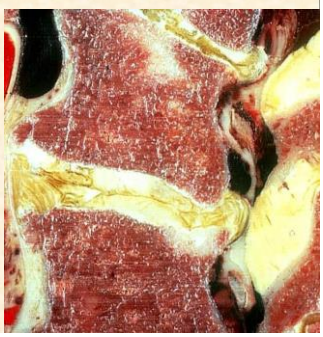
- Herniation generates radicular pain from a nerve root
- Displaced disc swells, loses proteoglycan, then shrinks (1)
- Pain from chemical *and* physical stimulation
- Herniated endplate (cartilage and/or bone) more stable (2)



1. *Dolan et al. (1987). J Bone Joint Surg [Br] 69(3): 422-428.*
2. *Lama et al. (2014). Euro Spine J 23: 1869-77.*



Structures injured in whiplash depend on the ratio of bending (M) to compression (C)



Injured cervical structure depends on:

Preparedness:
muscle activity determines ratio of bending to compression

Head turning:
torque and lateral bending determine apophyseal joint loading

Kapandji: 'Physiology of the joints" Churchill Livingstone 1974

Origins of Neck pain

- Often follows 'whiplash', but tissue origins and causes are usually unknown. Only 10% becomes chronic **(1).**
- From muscles? (Would be transient; eccentric damage?)
- From facets (55%) and discs (16%) **(2)**
  [consecutive patients in pain clinic, tolerant of invasive tests]
- From facets (23%), discs (20%), discs+facets (41%) **(3)**
  [post-trauma patients]
- **Treatments:** MDT marginally better than natural history **(4)**

1. *Bogduk (1999). Baillieres Best Pract Res Clin Rheumatol 13(2): 261-85*
2. *Yin & Bogduk (2008). Pain Med 9(2): 196-20*
3. *Bogduk & Aprill (1993). Pain 54(2): 213-217*
4. *Takasaki & May (2014). J Physiother 60(2): 78-84.*

Summary

- Posture-related stress concentrations can generate LBP 'like a stone in your shoe' in the absence of pathology
- 'Non-specific' low back pain (LBP) is *expected* to clear up, and is strongly influenced by psychosocial factors. It could often be caused by muscle injuries, which heal quickly
- Severe and chronic LBP most often arises from degenerated intervertebral discs & apophyseal joints
- *Certain aspects* of disc degeneration are often painful: radial fissures and endplate defects
- Nerves can be chemically 'sensitised', by inflammation & infection, so that pain is easily provoked