Back and neck pain

Michael A. Adams
Professor of Biomechanics,
Centre for Comparative and Clinical Anatomy,
University of Bristol, Bristol, U.K

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

Facet joint stresses depend on posture and disc height


<table>
<thead>
<tr>
<th>POSTURE ANGLE</th>
<th>INITIAL DISC HEIGHT</th>
<th>1mm DISC HEIGHT LOSS</th>
<th>4mm DISC HEIGHT LOSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0° FLEXION</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3° INDUCTILE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0° EXTENSION</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6° EXTENSION</td>
<td></td>
<td></td>
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</table>

“Comfortable” postures reduce lumbar lordosis


Lordotic postures concentrate stresses in the neural arch and posterior annulus


Flexion removes these stress concentrations

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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)


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)


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).


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


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!


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


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.


Disc degeneration and back pain


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

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

<table>
<thead>
<tr>
<th>Severity of Annular Tears</th>
<th>No Back Pain</th>
<th>Sometimes</th>
<th>Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inner</td>
<td>0.75 (0.56-0.94)*</td>
<td>0.16 (0.06-0.26)</td>
<td>0.10 (0.0-0.19)</td>
</tr>
<tr>
<td>Outer</td>
<td>0.55 (0.25-0.85)</td>
<td>0.25 (0.15-0.35)</td>
<td>0.20 (0.0-0.40)</td>
</tr>
<tr>
<td>Leaking</td>
<td>0.30 (0.05-0.54)</td>
<td>0.28 (0.25-0.31)</td>
<td>0.23 (0.15-0.70)</td>
</tr>
</tbody>
</table>

* Mean probability in the class (95% confidence intervals).
Radial fissures allow blood vessel & nerve ingrowth

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


Matrix defects permit nerve & blood vessel ingrowth


Painful features of disc degeneration

2. Vertebral endplate lesions


<table>
<thead>
<tr>
<th>TABLE 1. Associations Between Endplate Lesions and Back Pain History*</th>
</tr>
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<tbody>
<tr>
<td>Endplate Lesions</td>
</tr>
<tr>
<td>Schmorl’s nodes</td>
</tr>
<tr>
<td>Fracture</td>
</tr>
<tr>
<td>Erosion</td>
</tr>
<tr>
<td>Calcification</td>
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</table>

Pain sensitisation in discs

(Olmarker (2008). Spine 33 8 850-9)

- 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)

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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)


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