What is a ‘typical’ posterolateral disc protrusion?

Where does discogenic pain originate?

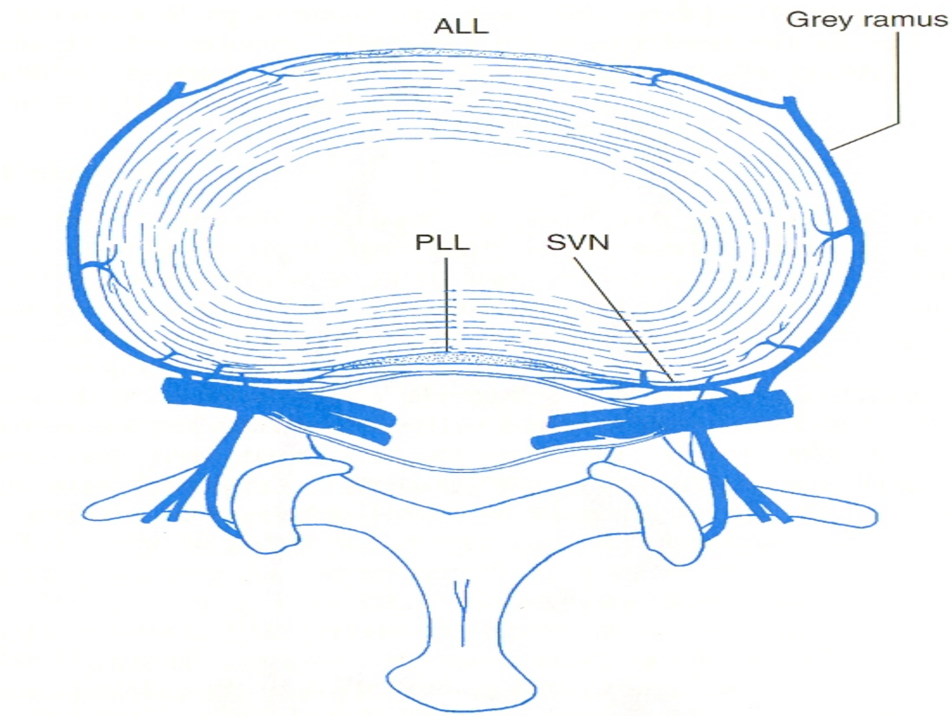
An evidence based review

How is intervertebral disc pain generated?

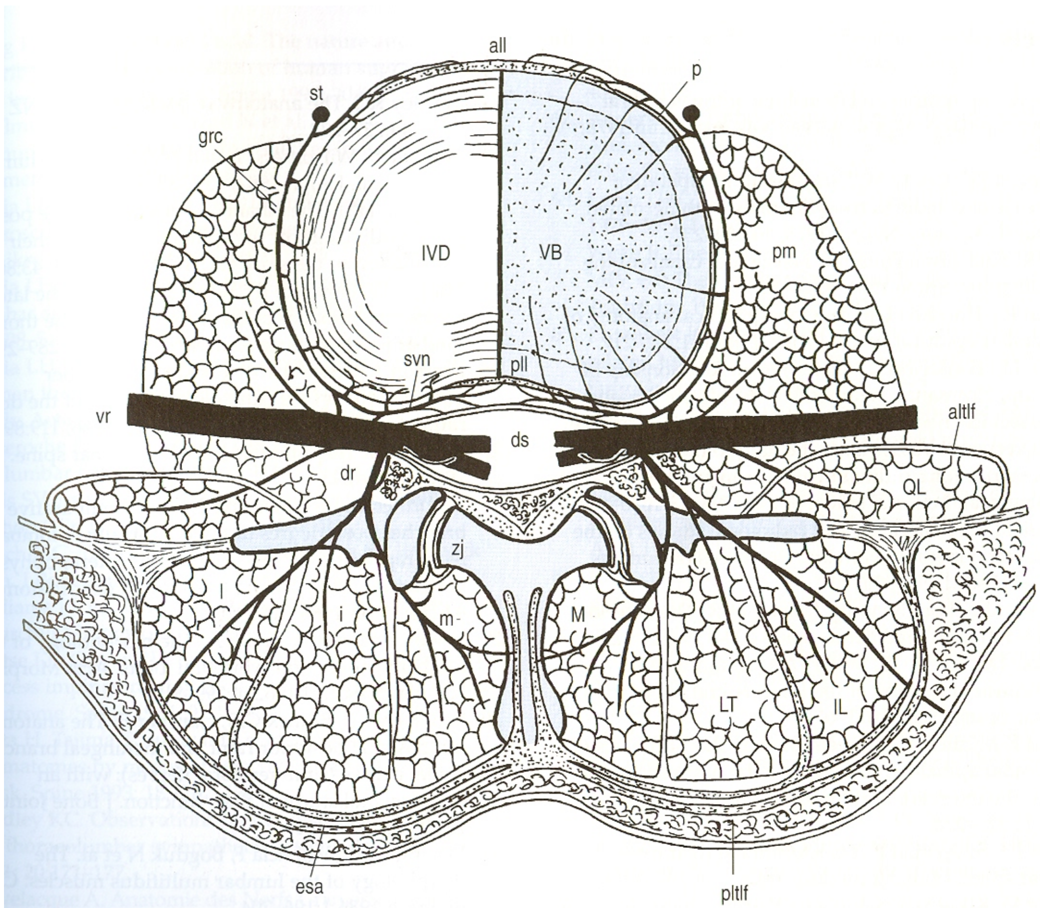
- Initial view is the nerve endings in the outer one third of the annulus (Inman VT et al1947)

- Innervation substantiated bv several researchers:
- Groen G et al 1990
- Hirsch C et al 1963
- Jackson HC et al 1966
- Bogduk N et al 1981
- Bogduk N et al 1988

etcetera, etcetera



Annular or endplate?



It has NOT been proven that the annular innervation is nocciceptive.

Malinsky and Farfan

Proprioception vs nocciception

Farfan demonstrates that electrical discharges given off by disc distortion

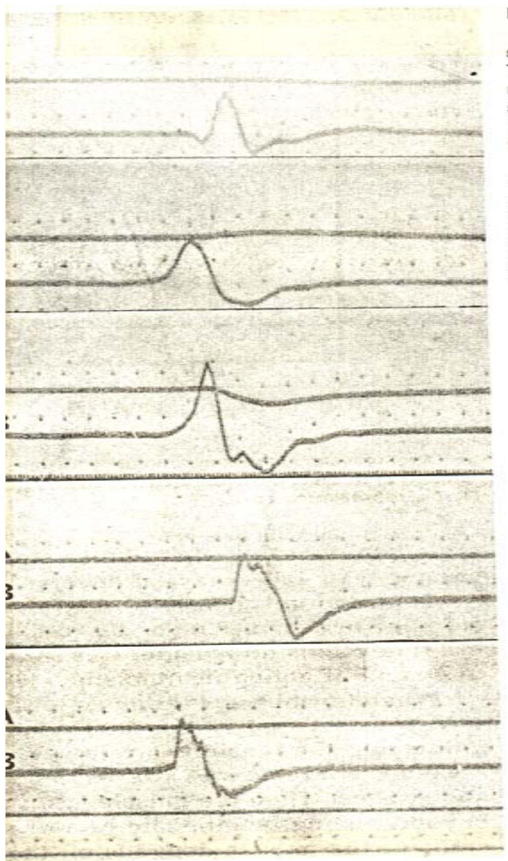


Fig. 8-5

(A) Electrical responses to various mechanical stresses. Stress was rapidly applied and removed. Tracings (A) from corresponding nucleus (B) from corresponding anulus. Note that the potential spike is the same sign regardless of the direction of load applied. 1, torsion; 2, compression; 3, tension; 4, extension; 5, flexion.

(B) Electrical response to repeated torsion: A, rotation clockwise and released; B, rotation anticlockwise and released; C, rotation clockwise and held; D, a second additional clockwise rotation. The potential in C decays, but before it decays to zero a second torque gives a positive spike of higher magnitude. (Courtesy of J. C. Sutton, Jr.)

In addition, claimed that electrical stimulation of the outer annulus led to a reflex contraction of the multifidus.

Proprioception vs nocciception

- Does electrical discharge given off by distortion of the annulus stimulate the peripheral annular nerve endings?

- Does this lead to reflex contraction of multifidus to help coordinate and stabilize vertebral segments during motion?

Proprioception vs nocciception

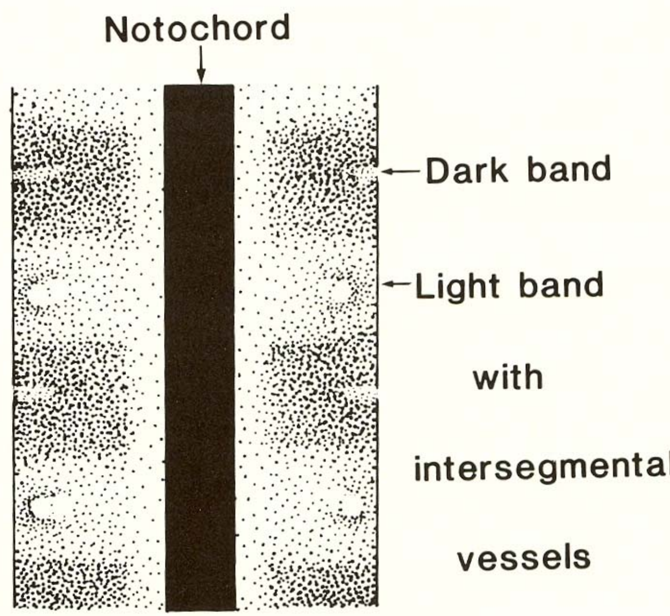
- Following disc damage does the loss of this reflex, biofeedback mechanism explain the inevitable weakness and wasting of the multifidus muscle?

- Does this explain the inevitable deterioration (without intervention) from disc trauma to lumbar segmental instability?

Endplate vs annulus

- Assume the peripheral annular innervation is proprioceptive
- Assume the vertebral body/endplate junction IS the main source of nocciceptors
- The pathomechanical factors leading to endplate trauma needs to be reviewed.

Embryology – 7mm (3wks)



Future disc/endplate

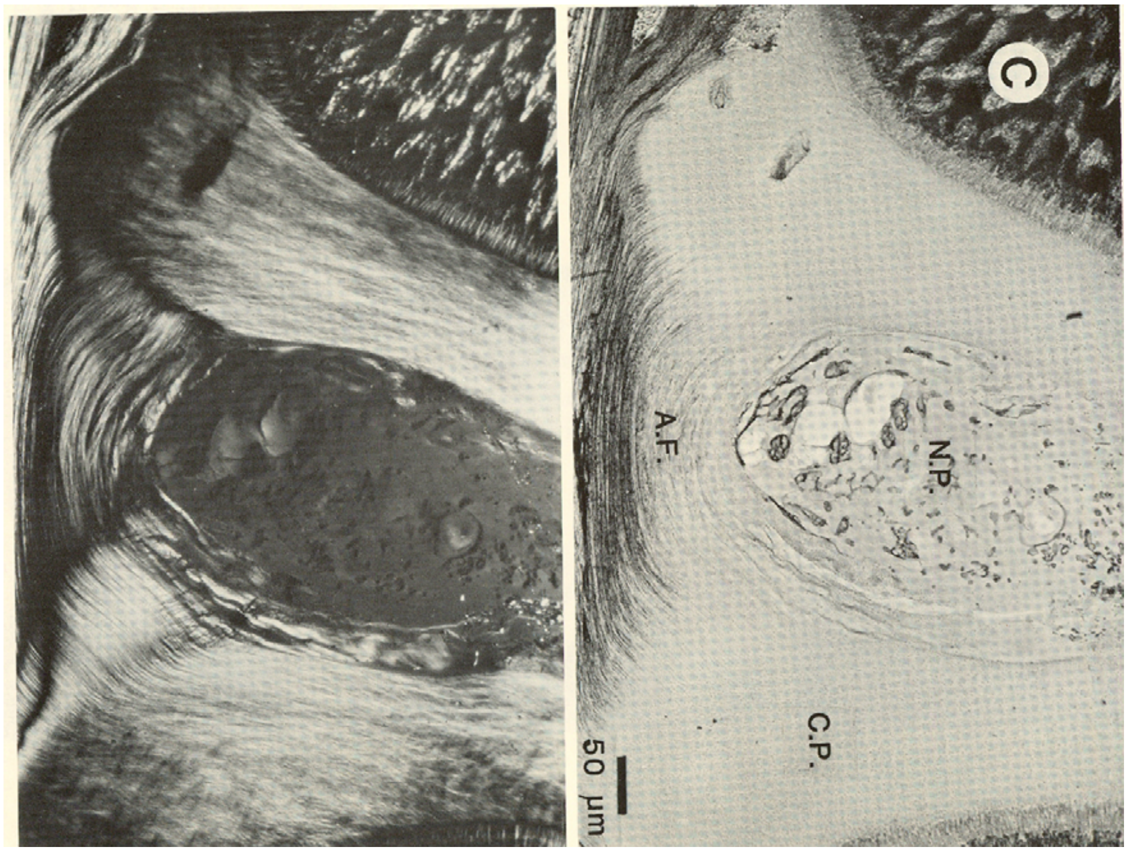
Future vertebral body

FIGURE 2. Diagram of coronal section of 7-mm human embryo, showin segmentation of the perichordal blastemal vertebral column into successiv light and dark bands. The dark band is the perichordal disc, whose centra part retains a similar appearance to the ''precartilage'' of the light band whicl is the primordium of the vertebra. (From Taylor, J. and Twomey, L., *Moder Manual Therapy of the Vertebral Column*, Grieve, G., Ed., Churchil Livingstone, Edinburgh, 1986. With permission.)

Embryology – 12-40mm

- At 12mm (6 weeks) spine undergoes chondrification
- At 40mm ossification begins in ‘light bands’
- At 40mm ‘dark band’ invaded by fibroblasts – future annulus fibrosis

Embryology – full-term disc



Annular fixation to the vertebral body

- Outer few layers become integrated/absorbed into the ring epiphysis
- The majority remain *surrounding* the nucleus and are *embedded* into the vertebral cartilagenous endplate
- NP is a gel NOT a liquid

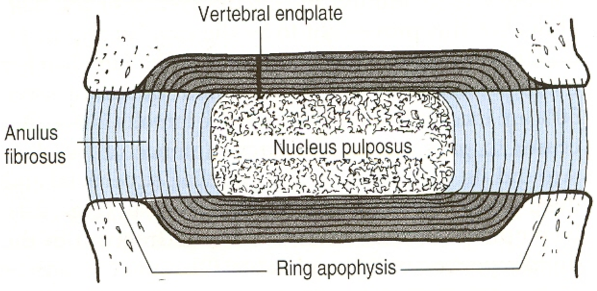
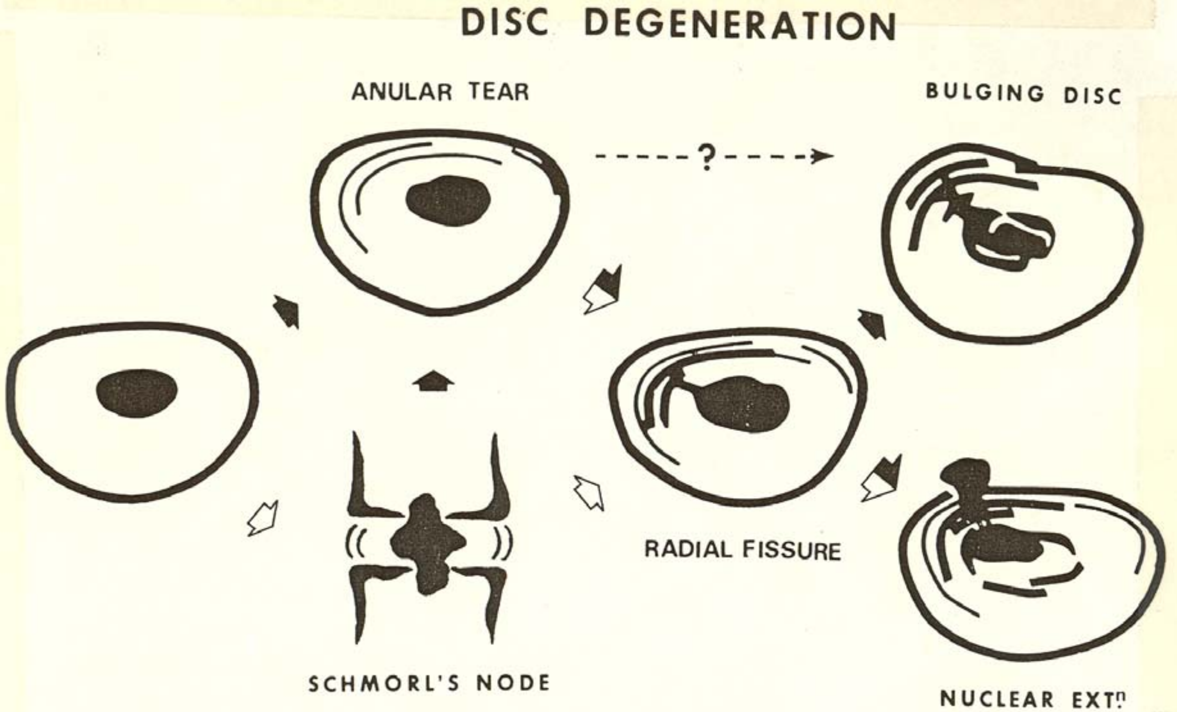


Figure 2.5 Detailed structure of the vertebral endplate. The collagen fibres of the inner two-thirds of the anulus fibrosus sweep around into the vertebral endplate, forming its fibrocartilaginous component. The peripheral fibres of the anulus are anchored into the bone of the ring apophysis.

The ‘traditional view’ (Farfan)



The progress of degenerative process towards the two surgical "end points." Solid and open arrows denote respectively torsion and compression strains.

Nuclear extrusions (disc herniations)

- Well documented

- Histological analysis of extruded material:

  a) endplate material

  b) blood vessels and nerves

  c) excessive amounts of amyloid (40-90%)

  (Melrose and Ghosh)

- Tends to endorse *biochemical* disruption rather than *biomechanical*

Annular protrusions (disc bulges)

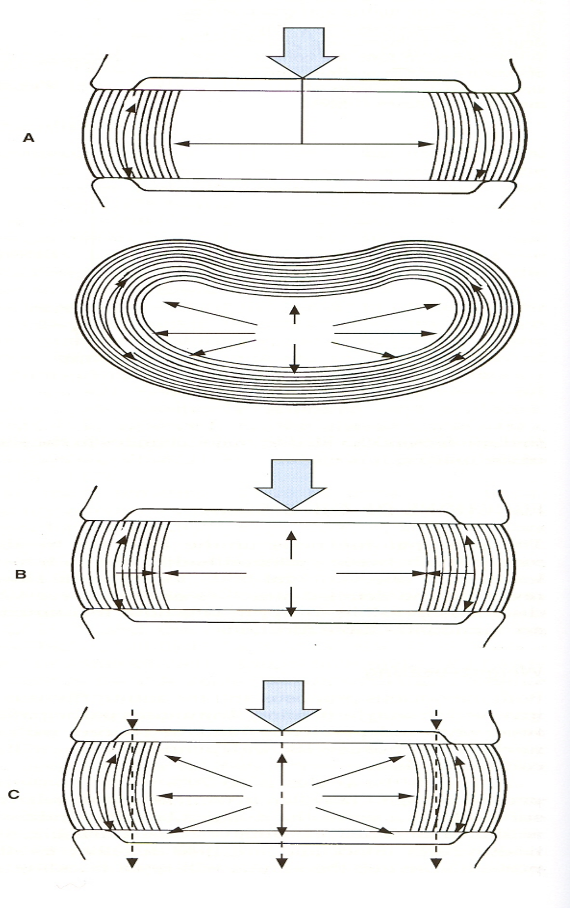
- Likened to a ‘horizontal flat tire’
- NOT a part of normal aging (Taylor, Twomey)
- More likely an insidious onset of Internal Disc Disruption (IDD), of endplate origin (Bogduck)
- Considered not clinically relevant, even with indentation of the thecal sac.

- Culprit more likely to be disc that ISN’T bulging.

? Numbers of ‘PLP’ patients treated by ‘conservative’ measures

- Herniations reasoned to be low
- PT clinicians can consistently pick up disc extrusions versus the ‘typical PLP’
- If not a bulging annulus WHAT IS a ‘typical’ P/L disc protrusion?
- How does McKenzie’s passive extension protocol so effectively manage the ‘typical PLP?’

Lumbar discs act as hydrostatic, pressurised cylinders



Loading creates increased NP pressure.

Dispersed equally 3 dimensionally

Restrained by the annulus and endplates

Orientation of annular fibres

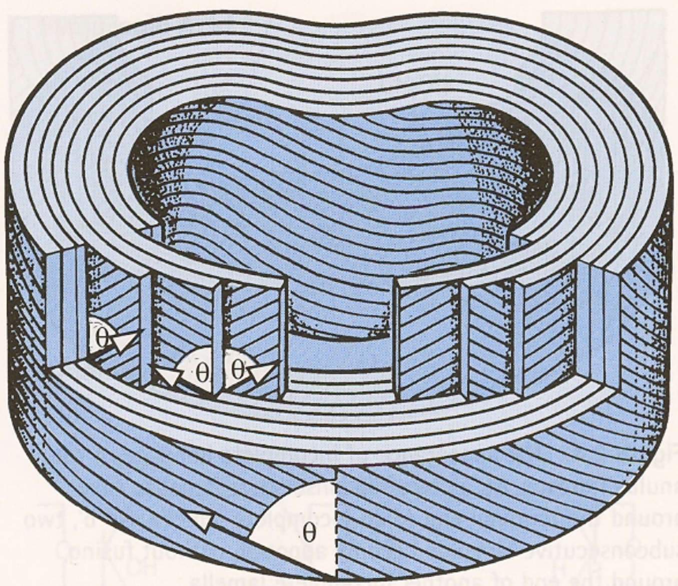
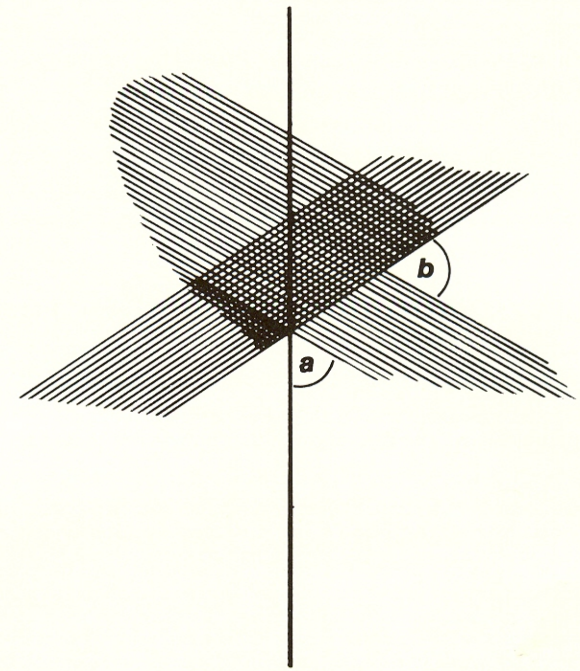


Figure 2.3 The detailed structure of the anulus fibrosus. Collagen fibres are arranged in 10–20 concentric circumferential lamellae. The orientation of fibres alternates in successive lamellae but their orientation with respect to the vertical (θ) is always the same and measures about 65°.



gles "a" and "b" are both about 60° in lumba

When annular fibres under tension, especially during flexion and rotation

WHEN tensed at an optimal angle of 60 degs

Can restrict motion in ALL planes

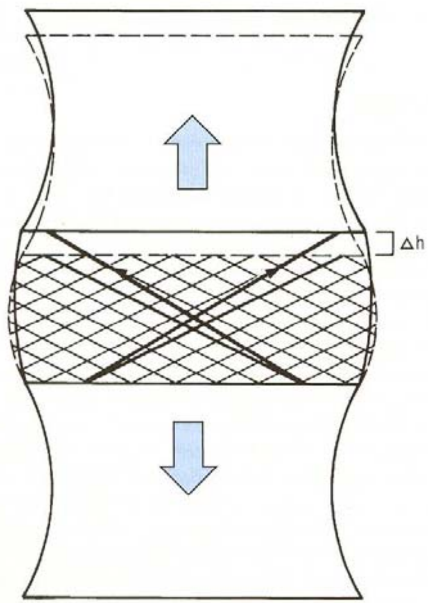


Figure 2.14 Distraction of the interbody joint. Separation of the vertebral bodies increases the height of the intervertebral disc (Δh), and all the collagen fibres in the anulus fibrosus are lengthened and tensed, regardless of their orientation.

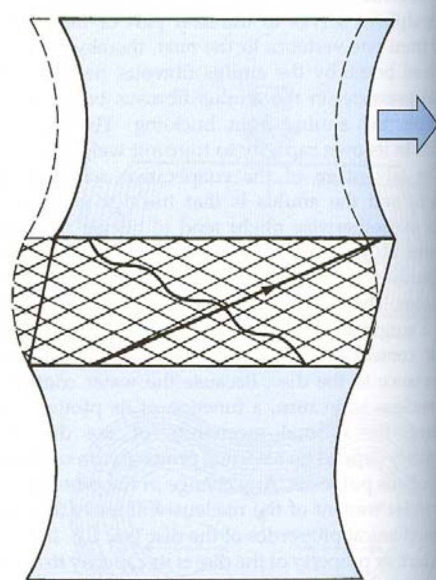


Figure 2.15 Sliding movements of an interbody joint. Those fibres of the anulus that are orientated in the direction of movement have their points of attachment separated, and therefore they are stretched. Fibres in every second lamella of the anulus have their points of attachment approximated, and these fibres are relaxed.

Lumbar FLEXION versus forward bending of the trunk

- Many studies erroneously assume the two are the same
- Forward bending can occur in neutral and even extension (Gracovetsky)
- FLEXION must be defined as a flattening of the lumbar lordosis in <u>any</u> trunk position

Neromuscular and osseoligamentous response to lumbar flexion

- Creates co-contraction of pubococcygeus and transversus abdominus
- Partially to contain abdominal and pelvic viscera during forward bending
- Secondary effect on the thoraco-lumbar fascia

Neuromuscular and osseoligamentous response to lumbar flexion

Co-contraction of pubococcygeus (counter-nutation of the sacrum)
and transversus abdominus tenses the thoraco-lumbar fascia <u>vertically</u>
and <u>transversely</u>

Resultant inhibition of erector spinae and decreased rotational displacement (Gracovetsky, Farfan, Bogduk)

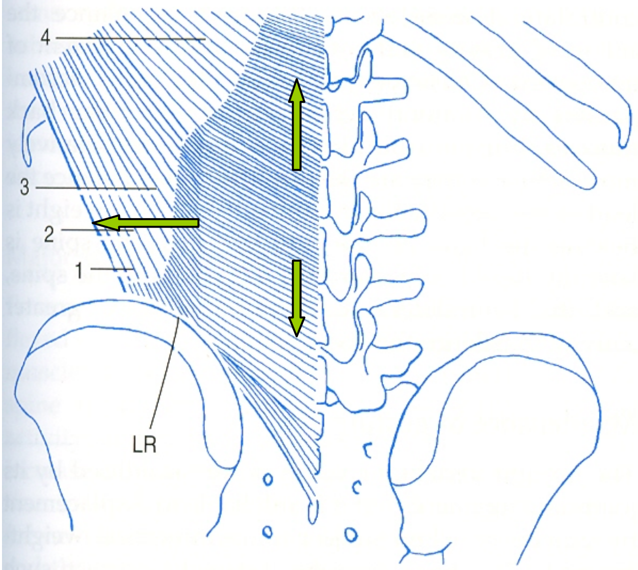


Figure 9.13 The superficial lamina of the posterior layer of thoracolumbar fascia. 1, aponeurotic fibres of the most lateral fascicles of latissimus dorsi insert directly into the iliac crest; 2, aponeurotic fibres of the next most lateral part of the latissimus dorsi glance past the iliac crest and reach the midline at sacral levels; 3, aponeurotic fibres from this portion of the muscle attach to the underlying lateral raphe (LR) and then deflect medially to reach the midline at the L3 to L5 levels; 4, aponeurotic fibres from the upper portions of latissimus dorsi pass directly to the midline at thoracolumbar levels.

Neuromuscuar and osseoligamentous response to lumbar flexion

Increased tension in the 'suprapinous ligament' creates an anti-shearing mechanism, especially at L5 (Bogduk)

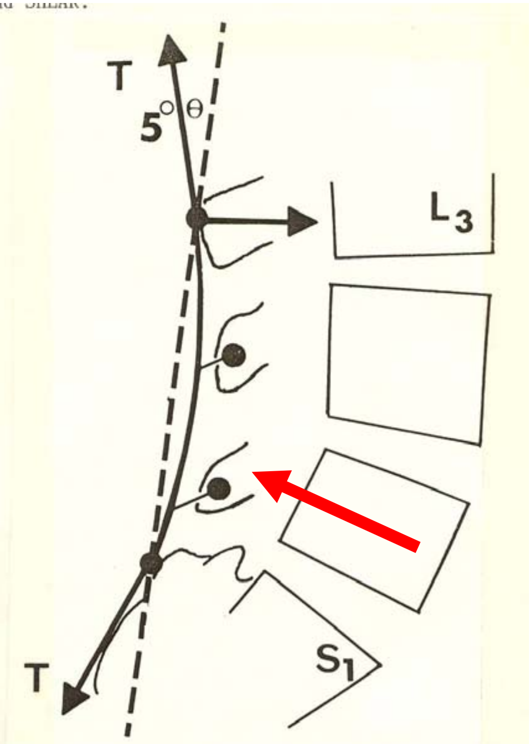


Fig. 8-8

As the lumbar spine is flexed, the lumbodorsal fascia (LDF) is tightened. This exerts a backwards pull on the vertebra. This effect may be estimated:

Total extension force	= 3640 lb
subtract muscle force	= 372
net tension LDF (T)	= 3268
if $\theta = 5°$ backwards pull	= 3268 × 2 cos 85
	588 lb

Neuromuscular and osseoligamentous response to lumbar flexion

If mutifidus contracts during lumbar flexion the increased muscle diameter tenses the thoraco-lumbar fascia further with two responses:

1) 30% increase in muscle strength

2) Increased compression/friction of 'Z' joints – transference of loading to neural arch

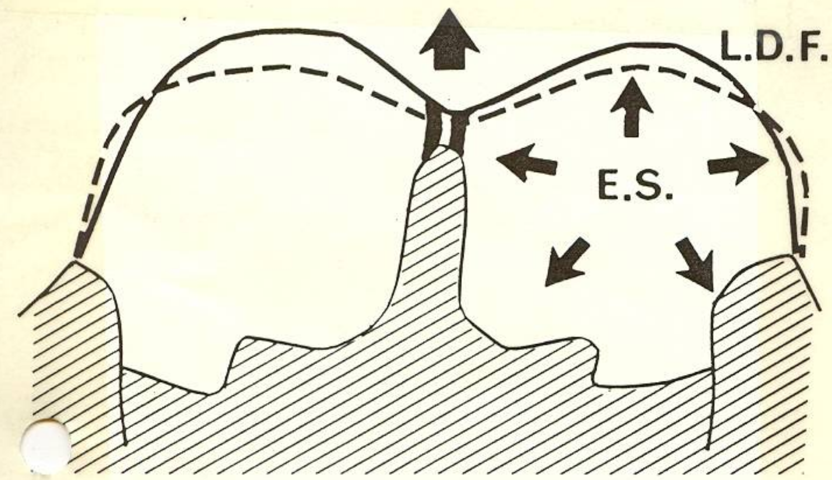


Fig. 8-9
The bone-fascia compartment enclosing the lowest portion of the erector muscles of the spine.

Neuromuscular and osseoligamentous response to lumbar flexion: SUMMARY

- Lumbar flexion increases tension in the thoracolumbar fascia:

  1) Inhibition of erector spinae – decreased trans-articular compression

  2) Decreased shearing and torsion

- During lifting, if erector spinae does contract:

  Tranference of loading away from the discs and intervertebral bodies into the cortical bone of the neural arches

Conclusion:

- True flexion of the lumbar spine IS its ‘position of power’
- Forward bending of the trunk, especially with rotation, in the absence of true lumbar flexion might be hazardous

- However, this still doesn’t explain endplate disruption

The mechanism of endplate disruption

- Vast majority of studies have focused on compression fractures of the endplate ***towards*** the vertebral body
- All have been in vitro

- What if the lesion is an ***avulsion fracture*** of the endplate? How could it occur?

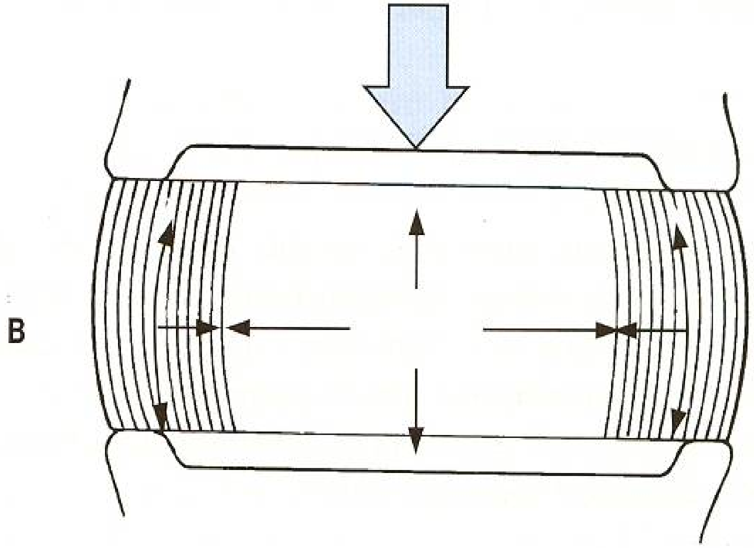
The mechanism of endplate disruption

- Main consideration is the *anchorage* of endplate annular fibres

- To sustain adequate fixation the endplate must be reinforced both internally (through increased intradiscal pressure) and externally through increased intravertebral pressure

The mechanism of endplate disruption

Which intravertebral mechanism reinforces the endplate?



The mechansm of endplate disruption

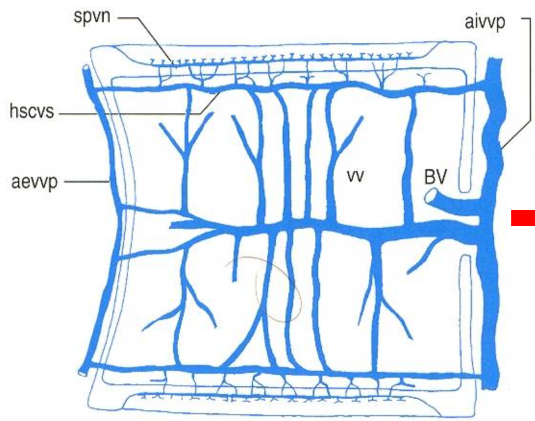


Figure 11.7 The intraosseous veins of the lumbar vertebral bodies. (Based on Crock et al. 1973.[4]) aevvp, anterior external vertebral venous plexus; aivvp, anterior internal vertebral venous plexus; BV, the basivertebral veins; hscvs, horizontal subchondral collecting vein system; spvn, subchondral postcapillary venous network; vv, vertical veins within the vertebral body.

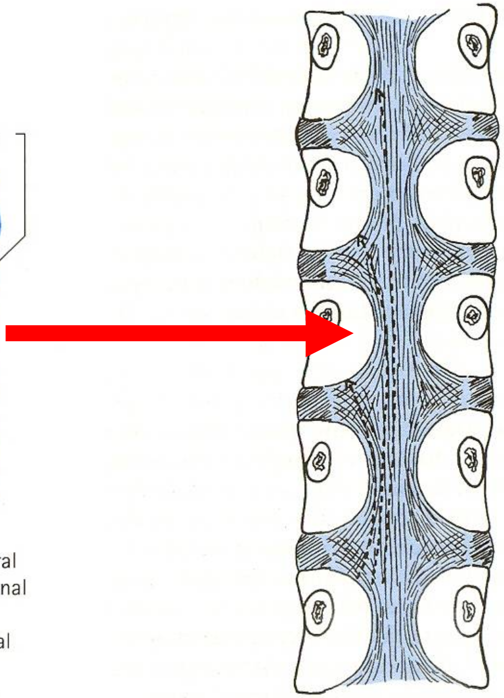


Figure 4.4 The posterior longitudinal ligament. The dotted lines indicate the span of some of the constituent fibres of the ligament arising from the L5 vertebra.

The mechanism of endplate disruption

Relative tensile strengths of the posterior ligaments (Bogduk)

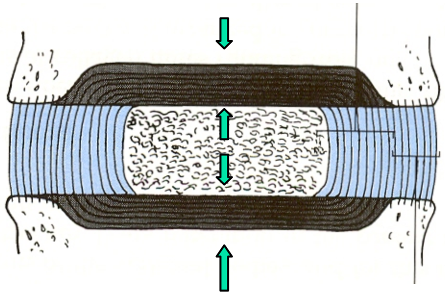
Ligament	Ref.	Average force at failure (N)	Moment arm (m)	Maximum moment (Nm)
Posterior longitudinal	96	90	0.02	1.8
Ligamentum flavum	96	244	0.03	7.3
Zygapophysial joint capsule	96	680	0.04	27.2
	97	672		
Interspinous	96	107	0.05	5.4
Thoracolumbar fascia	96	500	0.06	30.0
Total				71.7

Conclusion:

- True lumbar flexion tightens the posterior longitudinal ligament
- This reinforces the outer layer of the annulus and
- Partially occludes basivertebral venous drainage leading to
- Increased intravertebral pressure

The mechansim of endplate disruption

- True lumbar flexion creates a reciprocating increase in BOTH intradiscal and intravertebal pressure
- This reinforces the endplate annular fibres optimally



The mechanism of endplate disruption: Conclusion

Sustained or repetitive forward bending or rotation of the trunk without true lumbar flexion minimizes annular fixation at the endplate, increasing the risk of an endplate *avulsion fracture*

Avulsion lesions of the endplate

FIGURE 8. Detail of the longitudinal anastamosis between adjacent lumbar arteries shown on coronal section; these vessels pass over the intervertebral disc but do not supply it.

← Direct vascular supply to outer annulus is non-existent (Crock)

Endplate vascular supply is profuse

Avulsion lesions of the endplate: Summary

- Avulsion fracture of the endplate exposes cartilagenous matrix to blood
- Cartilagenous matrix is antigenic
- Resultant inflammatory reaction ensues within the vertebral spongiosa

Symptomatology of a ‘typical’ poterolateral disc protrusion

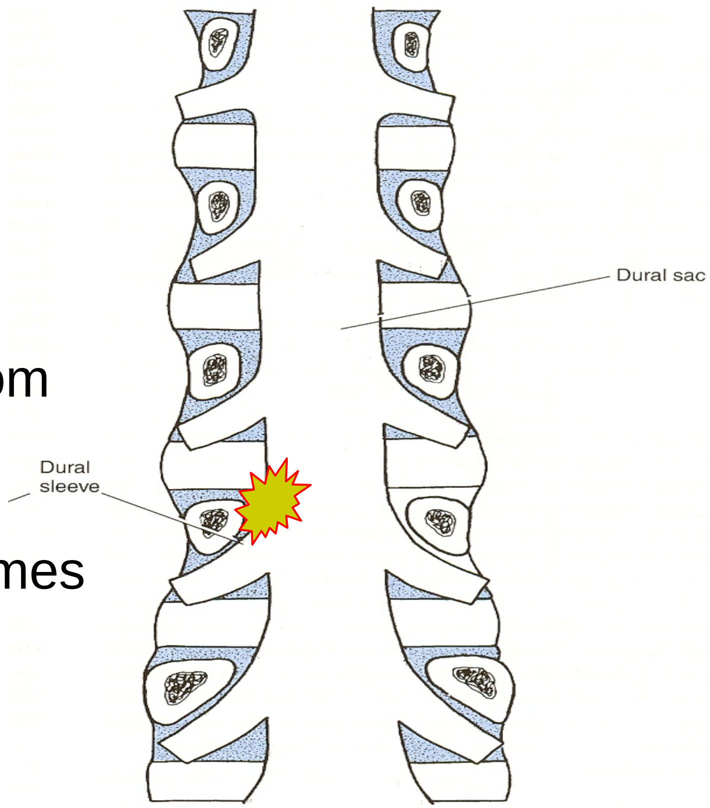
- Sudden onset of acute, localised pain. May be able to ‘work it off’ with continued activity
- Later pain increases in intensity and area
- Often the trunk deviates <u>away</u> from the pain
- Radicular pain ensues with or without neurological signs of nerve compression

Why the radicular symptoms in the absence of nuclear herniation?

Dura NOT sensitive to pressure or traction unless inflamed.

Permeable annulus fibrosus allows diffusion of inflammatory agents from the endplate ‘trauma zone’ to any tissues in close proximity i.e., the exiting nerve roots. The dura becomes inflamed.

Increased intradural pressure may lead to a loss of nerve conduction



Pathomechanical and histological events leading to the 'typical' posterolateral disc protrusion. Summary:

- Repetitive trunk rotation and/or flexion, in the absence of true lumbar flexion leads to an *avulsion* disruption of the vertebral endplate
- Blood within the vertebral spongiosa triggers an *autoimmune inflammatory response*, leading to increasing back pain with or without lateral deviation.
- Migration of *inflammatory agents* across the annulus *sensitize the dura mater* leading to peripheralization of leg pain and/or nerve root signs

So, to the *real* question, how does the passive extension protocol innovated by Robin McKenzie alleviate symptoms and restore function?

- The inflammatory response within the vertebral spongiosa increases the *osmotic* pressure within the region
- To combat this an increase in the *hydrostatic* pressure within the zone of trauma is needed
- This is provided by repeated, passive lumbar extension

- Osmotic pressure draws water IN
- Increased hydrostatic pressure drives water OUT
- But where does the water go?

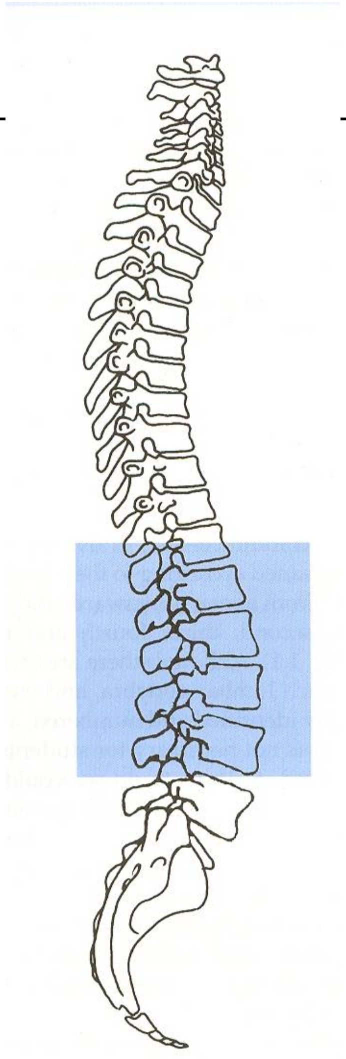
FIGURE 10. Radiograph of a thin coronal section through the center of a lumbar vertebra showing details of the intraosseous distribution of the arteries in the centrum region and, in particular, in the metaphyseal region.

It is clear that excess water from inflammatory exudate is more likely driven cranially or caudally through the collecting vessels adjacent to the mataphyseal region of the vertebral body/endplate junction. NOT across the disc.

Decreased exudate pressure equates to decreased pain and increased motion.

How does *early* use of this protocol decrease the likelihood of post-traumatic lumbar segmental instability?

'New' ideas about collagen synthesis and adaptation (Eyre)



- In scoliotic spines type I collagen density is increased on the side of compressed concavity.
- Supported by the normal development of the lumbar lordosis

‘New’ ideas about collagen synthesis and adaptation (Eyre)

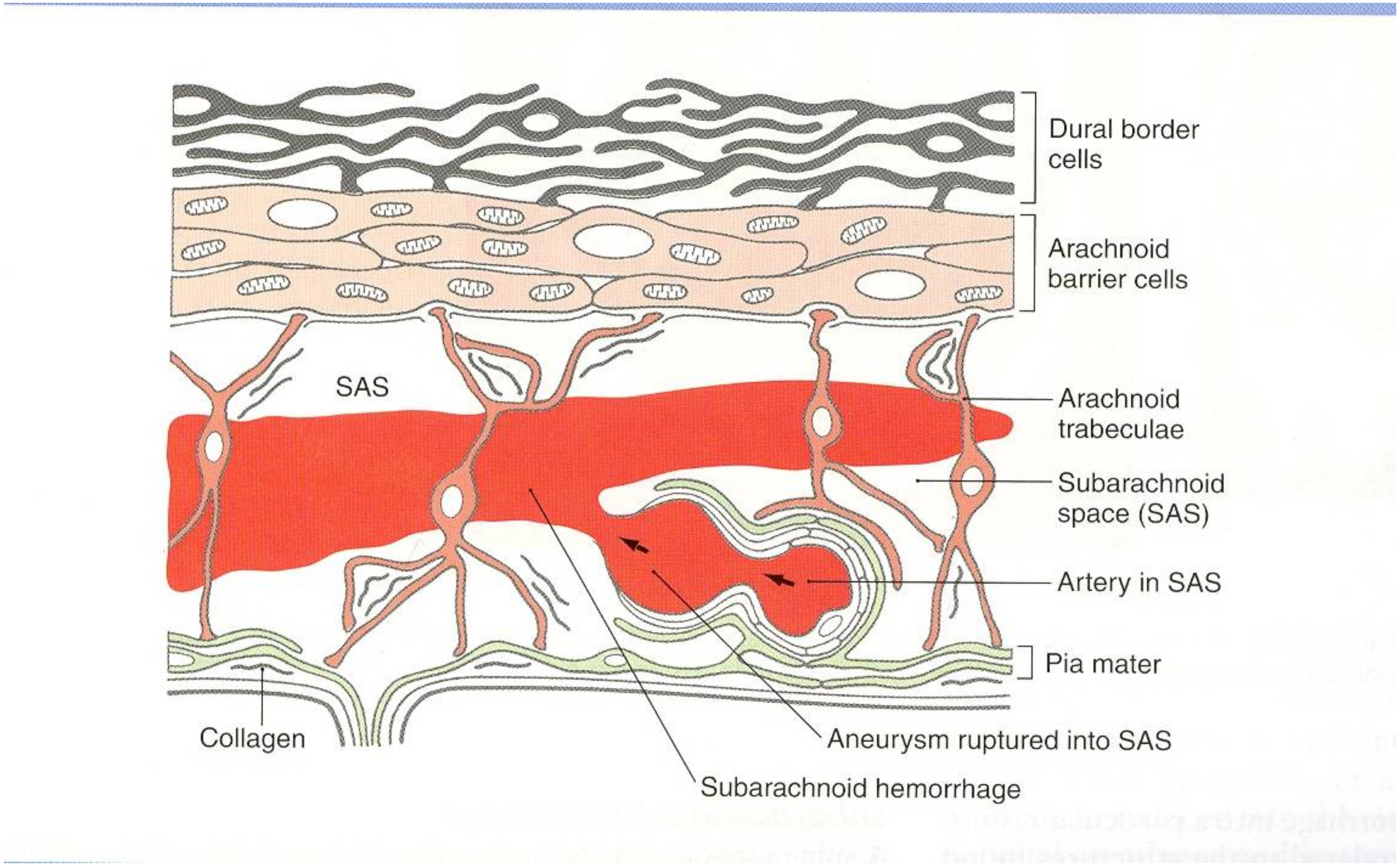
It is proposed that following a traumatic endplate annular disruption early, repetitive, passive lumbar extension and postural maintenance of the lumbar lordosis facilitates ‘1st intention’ healing of the annulus fibrosis.

This would significantly decrease the risk of secondary lumbar segmental instability.

How is the ‘centralization phenomenum’ achieved?

- In a ‘typical’ posterolateral disc protrusion it is accepted that the passive extension protocol can centralize leg pain and improve function.
- Immediately following such treatment most patients will still present with a ‘pre-treatment’ slump test result.
- This suggests alleviation of intradural pressure without decreased inflammatory sensitivity.

How is the 'centralization phenomenum' achieved?



The passive extension protocol works to alleviate pain and improve function:

1) Increasing hydrostatic pressure to exceeds osmotic pressure within the inflamed ‘zone of trauma’ within the vertebral spongiosa.

   This will drive water and inflammatory agents towards the ‘collecting system’ of the vertebral body’s capillary network.

   Decreased pressure within the inflamed vertebral spongiosa will relieve localised back pain.

The passive extension protocol works to alleviate pain and improve function:

2) Restoration and repetitive maintenance of the lumbar lordosis probably enhances type I collagen synthesis resulting in a better prognosis.

The passive extension protocol works to alleviate pain and improve function:

3) Repeated passive lumbar extension creates differential interstitial motion within the meninges, thus reducing intradural pressure and producing the ‘centralization phenomenum’

How do you feel?

- Evidence based studies
- Clinical research
- Scientific dogma

Let clinical reasoning and an open mind be your guide