What is a ‘typical’ posterolateral disc protrusion and how is it so successfully managed by the passive extension protocol innovated by Robin McKenzie?

An evidence based review

Erl Pettman MCPA. FCAMT. OMT.

Chairman and Chief Examiner of the North American Institute of Orthopaedic Manual Therapy (NAIOMT)
Adjunct professor of the Doctor of Science (DScPT) program, Andrews University, Mich.

The following lecture is an excerpt from the Level I NAIOMT course taught at Andrews University.

With consideration to lumbar discogenic pain, where does pain come from?
The concept that discs could be pain generators goes back as far as 1947 when it was recognised that the outer annulus was indeed innervated (1). This fact has since been substantiated by many other investigators (2-6). Although coming from the grey rami communicantes these nerves cannot be considered sympathetic since no blood vessels penetrate the annulus (7). In spite of this, the exact neurological mechanism underlying discogenic pain remains debated. Even though discography clearly inculpates the disc as the source of pain it cannot be assumed that the outer annulus is the pain generator since the endplate is also highly innervated (8). In fact, in-growing nerve endings, from the endplate have been found in disc material operatively excised following discography that showed the disc to be painful (9-11).
An alternative proprioceptive function was ascribed to the annular innervation by Malinsky (12), a belief shared by Farfan in his book ‘Biomechanics of the Lumbar Spine’ (13). Farfan described how distortion of the disc gives rise to a measurable electrical discharge. He also describes how, during surgery, electrical stimulation of the outer annulus led to discreet contraction of the multifidus. Unfortunately, no backup studies appear to have been done on this clinical observation. However, if these hypotheses are true they would seem to strengthen the argument for the annular innervation being proprioceptive not nocciceptive. Further, it may help to explain why deteriorated discs that have led to segmental instability, also lead to a loss of motor control and even wasting of the multifidus (14-16).

If, in fact, the endplate *is* the main source of pain receptors then a mechanism of injury needs to be proposed.
In a brief review of the embryological development of the disc (17) it is seen that at around 3 weeks (7mm) the mesenchyme surrounding the notochord undergoes segmentation. A banded pattern becomes apparent as the central part of each segment becomes denser (the ‘dark band’) while the adjacent ends of each segment (the ‘light band’) merge with each other. The ‘dark bands’ will ultimately form the discs and endplates, the ‘light bands’ the vertebral bodies.

At around six weeks (12mm) the developing spine undergoes chondrification. And by the 40mm stage of development the 'light bands' begin to ossify. At around the same time the hyaline cartilage of the developing disc becomes invaded by fibroblasts. The collagen laid down by these cells will become the annulus fibrosis.
At this stage the nucleus pulposis consists of notochordal cells surrounded by embryonic cartilage. Notochordal cells will persist in the nucleus until 3-5 years of infancy by which time they are necrosed and replaced by fibrocartilage.
At this point it is worth emphasising that the adult nucleus pulposus is not analogous to the 'jam in a doughnut' as we have all probably explained to our patients. Its matrix is similar to the matrix of the annulus i.e., a gel state not liquified. In vitro, if a portion of the annulus (of an otherwise healthy disc) is removed and the disc subjected to pressure, the nucleus bulges but does not collapse or herniate (18). Later we shall see how nuclear herniations actually are proposed to occur.
The layers of the developing annulus completely surround the nucleus like concentric spheres. As development continues the outer one third of the annular fibres are amalgamated into the ossifying ring epiphysis but the majority of annular fibres remain as concentric spheres around the nucleus, their caudal and cranial portions being firmly embedded into the end plate. This will be a crucial point when reviewing how the lumbar spine resists the forces applied to it during trunk motion.

It is time to review proposals as to how a lumbar disc may become damaged. The traditional view proposed in Farfan's book (13) describes how a deteriorating series of events begins with either 'circumferential' tearing of the annulus or a vertical extrusion through the endplate (a Schmorl's node). With time the 'circumferential' tears amalgamate to form 'radial' tears allowing nuclear material to migrate to the periphery of the disc where a disc protrusion may occur, or even extrusion of nuclear material (disc herniation).
That nuclear extrusions occur is a matter of record. What is interesting is that when the surgically removed material is analysed endplate material is frequently found. So too are blood vessels and nerves, which tends to infer extreme endplate disruption (19-21). What is perhaps more interesting is that between 40% (22) and 90% (23) of surgically removed specimens contained significant amounts of a substance called amyloid. While amyloidosis can be a lethal condition if systemic it is proposed that, in a more mild form, excessive amounts of this substance may be responsible for early deterioration of all cartilaginous structures including intervertebral discs (24). This certainly underscores the concept that many nuclear extrusions may be the result of *biochemical* disruption of a disc, not biomechanical.

I am unaware of any studies that compare the numbers of back pain patient's who are treated conservatively (by manual or mechanical means) versus those who require surgical discectomy but clinical observation and reasoning dictates that the number of biochemically deranged discs must be extremely small.
At least one study shows that tests performed by Physical Therapists are extremely valid and consistent in identifying disc protrusions versus herniations (25). So the question remains how does the more typical 'postero-lateral disc protrusion' occur and why is it so effectively treated by the passive extension protocol as innovated and described by Robin McKenzie?

The lumbar intervertebral disc acts like a hydrostatic cylinder (primarily the nucleus pulposus) supported by the annulus and the endplates (26,27).
The annular fibres are obliquely oriented, each successive lamina's fibres running in opposite directions. When tensed the fibres of the annulus can restrict excessive anterior, posterior, lateral and rotary displacement (27). The key words here are 'when tensed'. The optimal angle of annular fibres appears to 60 degs to the vertical. Obviously, a deteriorated or dehydrated disc could not maintain this angle and explains the progressive degradation of the disc once full vertical height is lost.
In a normal disc the annular fibres will be tensed by increased intra-discal pressure from trunk displacement, especially forward bending. But forward bending is not, necessarily, lumbar flexion. Lumbar flexion must be defined as a flattening of the lumbar lordosis in any position.
It seems well documented now that lumbar flexion, with forward bending, initiates a co-contraction of the pubo-coccygeus and transversus abdominus. A part of the function of these muscles would logically be a muscular 'containment effort' to retain the abdominal and pelvic viscera. However, these muscular groups also have a simultaneous biomechanical effect, predominantly on the thoraco-lumbar (T/L) fascia.
Sacral counter-nutation, in conjunction with lumbar flexion would tighten the T/L fascia longitudinally. Contraction of transversus abdominus would tighten the T/L fascia laterally. This combined action on the posterior ligamentous system acts as an anti-shearing mechanism at the lower lumbar segments (28) as well as minimising torsional stress. Also, in a lumbar flexed position, if the erector spinae is activated in flexion, being contained in an inextensible osseo-ligamentous 'tube' the result would be a 30% increase in erector spinae muscle strength (29) and a corresponding increased compression, and therefore friction, of the zygapophysial joints. This would facilitate transference of load through the cortical bone of the neural arches, decreasing compression within the lumbar vertebrae and discs (30-33).
In summary, flexion would appear to be the 'position of power' of the lumbar spine and, therefore, forward bending of the trunk as well as rotation of the lumbar spine, in the absence of lumbar flexion might be considered hazardous.

It is interesting that the vast majority of investigative literature has focused on discal damage from compression but what if the major cause of endplate damage isn't compression fractures from excessive loading but *avulsion fractures* from inadequate ***anchorage*** of the fibres of the annulus fibrosis.
It is reasonable to assume that to sustain adequate fixation of the annular fibres the cartilaginous endplate (into which most of the annular fibres are embedded) must be reinforced both caudally and cranially.
It is proposed that this occurs when lumbar flexion, while increasing intradiscal pressure, also increases *intra-vertebral* pressure. The paired basivertebral veins (the main venous drainage of the vertebral body) are vulnerable to compression, and therefore partial occlusion, during actual lumbar flexion through tightening of the posterior longitudinal ligament. Analysis of the tensile strength of the posterior ligamentous system suggests that the function of the posterior longitudinal ligament is not primarily restrictive (34). Rather, it reinforces the outer postero-lateral annulus *and* acts as the only valve mechanism within the lumbar venous plexus.
It is feasible that, during lumbar flexion the increased intra-vertebral pressure would match the increased intra-discal pressure, thus securing the endplate attachments of the annular fibres and help to prevent avulsion of these fibres from the endplate.

Thus it is proposed that the primary precipitative mechanism of injury to a lumbar disc is sustained or repetitive lifting (or simply forward bending of the trunk), especially during rotation, with a neutral, non-flexed lumbar spine.
Deficient tautness in the posterior ligamentous system (including the thoraco-lumbar fascia) would lead to excessive segmental rotation. This, in turn would lead to an increased stress placed upon the annular fibres. If intra-vertebral compression has not been increased by lumbar flexion, endplate fixation of the annular fibres will be compromised and an avulsion type injury of the annular/endplate region is envisioned. This may certainly explain internal disc damage but how does it explain clinical signs and symptoms?

Blood supply to the disc itself is non-existent. Nutrition of the disc relies totally on diffusion of water and nutrients from two vessel systems, those surrounding the outer annulus and, more importantly, the complex of capillaries just beneath the vertebral endplates (7).
If an avulsion type lesion of the endplate occurs cartilaginous matrix will be exposed to blood from the vertebral spongiosa. Cartilagenous disc material is antigenic (35-40) and an autoimmune inflammatory reaction will occur.
This helps to explain why a worker may suffer a lifting injury and get an initial sharp, localised pain at the site of the lesion. He may even choose to 'work it off' and finish his shift. As the inflammatory process builds and spreads to the vertebral spongiosa (41), however, the pain intensifies and is almost certainly responsible for the characteristic lateral deviation away from the painful side.
One intriguing question at this point is, in the absence of actual extruded annular material why do we so commonly see increasing peripheralisation of leg pain?
The annulus fibrosis is extremely permeable to most substances (42-44) and it is likely therefore, that the constituents of inflammatory exudate may also diffuse through the annulus towards the periphery. Any exiting nerve root in close proximity to this inflammatory migration would be subsequently affected.
The dura mater is not pain sensitive to pressure or traction (45,46). It requires an inflammatory reaction to sensitise it (47). It has also been demonstrated that as the inflammatory pressure builds with the nerve root it could, in itself, provide the basis for a loss of neurological conduction (48-52).

So a predictable series of pathomechanical and histological events seem to logically explain what we, as Physical Therapists, identify as a 'typical posterolateral protrusion':

1) Repetitive rotation and flexion, with or without loading, with a lumbar spine NOT IN FLEXION ultimately leads to an *avulsion* disruption of the vertebral endplate.

2) Blood within the vertebral spongiosa triggers an autoimmune inflammatory response, leading to increasing back pain with or without lateral deviation.

3) Migration of inflammatory agents across the annulus sensitise the dura mater of an exiting nerve root leading to peripheralization of pain (radicular pain) and/or loss of nerve root conduction.

And now to the point of the paper. How does Robin McKenzie's passive extension protocol alleviate symptoms and regain function?

From the onset of the endplate avulsion lesion the resulting inflammatory exudate raises the osmotic pressure within the zone of tissue damage. The osmotic attraction of water increases the intrinsic pressure, and thereby pain, within the lesion. Interestingly, once outside of the acute phase most patients with such a lesion may complain more of 'stiffness' in the morning because of this phenomenon.

To remove the affects of increased osmotic pressure the intrinsic hydrostatic pressure within the lesion must be increased. This is achieved by passive lumbar extension. This temporary increase in pressure ensures that the patient will experience greater localised pain but this improves as the increased hydrostatic pressure drives water out of the inflamed region. But where does the water go?

Crock (7) has shown the marked density of arteriole, capillary and venule structures interfacing with the endplate. He also indicates that fluid transport across this interface is rapid enough to change the fluid content of a normal disc on an hourly basis. In fact, radio-tracers injected during discography can be seen within the pulmonary circulation within seconds!
It is clear from these observations that water, and presumably inflammatory agents, are more likely to be dispersed by this mechanism than to be displaced *across* the disc.

As a clinician who has worked in the same community for 28 years it has been my observation that patients suffering from a typical posterolateral disc protrusion (now, more correctly called an intrinsic disc disruption, or IDD) treated early with a passive extension protocol do not later tend to deteriorate towards lumbar segmental instability. This suggests that the passive, repetitive extension protocol somehow offers a better healing potential.
One explanation for this may be found in studies on type I collagen synthesis in scoliotic patients. It is has been found that type I collagen synthesis is stimulated on the concave, compressed side of a spinal curve (53). This is consistent within discs of a normal lumbar lordotic curve. If collagen synthesis can be stimulated in this way it strongly reinforces the concept that repetitive extension to regain, and maintain the lumbar lordosis, may promote a better healing response within the damaged endplate annular fibres.

Finally, what mechanisms are in play to explain the 'centralization phenomenon'?
It is clear from immediate follow up examination of those patients who exhibit this change that centralization of radicular pain does not necessarily equate with an improvement in the slump test results. This tends to suggest that repeated passive extension may relieve the *pressure* within the dural sleeve but not affect dural inflammation or sensitivity to stretch.
One explanation for this may be the relative extensibility or elasticity of the various meningeal tissues. The pia mater is the thinnest and therefore, the most elastic, the dura mater the least elastic (54). Furthermore, the dural sac and nerve roots are tethered to the vertebral column by meningovertebral ligaments (55). This allows for a differential rate of interstitial motion between the layers of the meninges during passive extension of the lumbar spine. This would provide a mechanism to explain the dispersal of the intrinsic *pressure* of inflammatory exudate, thus

decreasing intra-dural pressure, while having little or no immediate effect on dural sensitivity to stretch.

In summary, the passive extension protocol works by:

1) Increasing hydrostatic pressure to exceed the inflammatory osmotic pressure thus driving water towards the 'collecting system' of the vertebral capillary network. A decreased pressure within the inflamed vertebral spongiosa will relieve localised back pain.

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

3) Repeated lumbar extension creates differential interstitial motion within the meninges, reducing intra-dural pressure and producing the 'centralization phenomenon'.

I hope what this paper demonstrates is that evidence based information, scientific research, outcome measures and even scientific dogma can be used to propose any number of different theories regarding clinical problems. Only time will tell which ones are valid or invalid. Keep an open and inquisitive mind.

References:

1) Inman VT, Saunders JB. Anatomicophysiological aspects of injuries to the intervertebral disc. J Bone Joint Surg 1947; 29:461-475,534.
2) Groen G, Baljet B, Drukker J. The nerves and nerve plexuses of the human vertebral column. Am J Anat 1990; 188:282-296.
3) Hirsch C, Ingelmark BE, Miller M. The anatomical basis for low back pain. Acta Ortop Scand 1963; 33:1-17.
4) Jackson HC, Winkelmann RK, Bickel WH. Nerve endings in the human spinal column and related structures. J Bone Joint Surg 1966; 48A:1272-81.
5) Bogduk N, Tynan W, Wilson AS. The nerve supply to the human lumbar intervertebral discs. J Anat 1981; 132:39-56.
6) Bogduk N. The innervation of the intervertebral discs. In: Ghosh P, ed. The Biology of the Intervertebral disc, Vol 1. Boca Raton: CRC Press; 1988: 135-49.
7) Crock HV, Goldwaser M, Yoshizawa H. Vascular anatomy related to the intervertebral disc. In: Ghosh P, ed. The Biology of the Intervertebral Disc, Vol 1. Boca Raton: CRC Press; 1988, Ch. 4: 109-33.
8) Brown MF, Hukkanen MVJ, McArthy ID et al. Sensory and sympatheic innervation of the vertebral endplate in patients with degenerative disc disease. J Bone Joint Surg 1997; 79B:147-53.
9) Coppes MH, Marani E, Thomeer RTWM et al. innervation of the 'painful' lumbar discs. Spine 1997; 22:2342-2350.
10) Freemont AJ, Peacock TE, Goupille P et al. Nerve ingrowth into diseased intervertebral discsi in chronic back pain. Lancet 1997; 350:178-81.

11) Johnson WEB, Evans H, Menage J et al. Immunohistochemical detection of Schwann cells in innervated and vascularised human intervertebral discs. Spine 2001; 26:2550-57.
12) Malinsky J. The otogenetic development of nerve terminations in the intervertebral discs of man. Acta Anat 1959; 38:96-113.
13) Farfan H. Biomechanics of the Lumbar Spine.
14) Hides JA, Stokes MJ, Saide M et al. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 1994 Jan 15; 19(2):165-72.
15) Hides JA, Richardson C, Jull GA. Multifidus muscle recovery is not automatic after pain resolution of acute first episode of low back pain. Spine, 1996 Dec1; 21(23): 2763-9.
16) Yoshihara K, Nakayama Y, Fujii N et al. Atrophy of the multifidus muscle in patients with lumbar disc herniation: histological and electromyographic study. Orthopaedics. 2003 May; 26(5):493-5.
17) Taylor JR, Twomey LT. The development of the human intervertebral disc. In: Gosh P, ed. The Biology of the Intervertebral Disc, Vol 1. Boca Raton: CRC Press; 1988, Ch 2: 39-82.
18) Nachemson A, Elfstrom G. Intravital dynamic measurements in lumbar discs. Scand J Rehabil Med. (Suppl 1), 1, 1960.
19) Brock M, Patt S, Mayer HM. The form and structure of the extruded disc. Spine, 1992 Dec; 17(12): 1457-61.
20) Yasuma T, Arai K, Yamauchi Y. The histology of lumbar intervertebral disc herniation. The significance of small blood vessels in the extruded marterial. Spine, 1993 Oct 1; 18(13): 1761-5.
21) Schmid G, Witteler A, Willburger R, et al. Lumbar disc herniation: correlation of histologic findings with Mrrow Signal intensity changes in vertebral endplates at MR imaging. Radiology, 2004 May; 231(2):352-8.
22) Ladefged C, Fedders O, Peterson O. Amyloid in interverebral discs: a histopathological investigation of surgical material from 100 consecutive operations on herniated discs. Ann. Rheum. Dis. 1986. 45, 239.
23) Ladefoged C. Amyloid in intervertebral discs (a histopathological investigation of intervertebral discs from 30 randomly selected autopsies). Appl. Pathol. 1985. 3, 96.
24) Melrose J, Ghosh P. The noncollagenous proteins of the intervertebral disc. In: Ghosh P, ed. The Biology of the Intervertebral Disc, Vol 1. Boca Raton: CRC Press; 1988, Ch 8:189-208.
25) Stankovic R, Johnell O, Maly P et al. Use of lumbar extension, slump test, physical and neurological examination in the evaluation of patients with suspected herniated nucleus pulposus. A prospective clinical study. Man Ther. 199 Feb; 4(1):25-32.
26) Hukins DW. Disc structure and function. In: Ghosh P, ed. The Biology of the Intervertebral Disc, Vol 1. Boca Raton: CRC Press; 1988, Ch 1:1-38.
27) Bogduk N. Clinical Anatomy of the Lumbar Spine and Sacrum, 4th ed. Philadelphia: Elsevier Churchill Livingstone; 2005, Ch 2:11-28.
28) IBID. Ch 4:44-49.
29) Hukins DWL, Aspden RM, Hickey DS. Thoracolumbar fascia can increase the efficiency of the erector spinae muscles. Clin Biomech. 1987; 5:30-34.
30) Gracovetsky S, Farfan HF, Lamy C. The mechanism of the lumbar spine. Spine 1981; 6:249-62.
31) Farfan HF. Muscular mechanism of the lumbar spine and the position of power and efficiency. Orthop Clin North Am 1975; 6:135-44.
32) Gracovetsky S, Farfan HF, Helleur C. The abdominal mechanism 1985; 10:317-324.
33) Gracovetsky S, Farfan HF, Lamy C. A mathematical model of the lumbar spine using an optimal system to control muscles and ligaments. Orthop Clin North Am 1977; 8:135-53.
34) Bogduk N. Clinical Anatomy of the Lumbar Spine and Sacrum, 4th ed. Philadelphia: Elsevier Churchill Livingstone; 2005, Ch 9:118.
35) McCarron RF, Wimpee MW, Hudkins PG et al. The inflammatory effect of the nucleus pulposus: a possible element in the pathogenesis of low back pain. Spine; 12:760-64.

36) Bobechko WT, Hirsch C. Autoimmune response to nucleus pulposus in the rabbit. J Bone Joint Surg 1965; 47B:574-80.
37) Gertzbein SD. Degenerative disk disease of the lumbar spine: immunological implications. Clin Orthop 1977; 129:68-71.
38) Gertzbein SD, Tait JH, Devlin SR. The stimulation of lymphocytes by nucleus pulposus in patients with degenerative disk disease of the lumbar spine.Clin Orthop 1977; 123:149-54.
39) Gertzbein SD, Tile M, Gross A. Autoimmunity in degenerative disk disease of the lumbar spine. Orthop Clin North Am 1975; 6:67-73.
40) Elves MW, Bucknill T, Sullivan MF. In vitro inhibition of leukocyte migration in patients with intervertebral disk lesions. Orthop Clin North Am 1975; 6:59-65.
41) McCall IW, Park WM, O'Brien JP et al. Acute traumatic intraosseous disc herniation. Spine 1985; 10:134-37.
42) Urban J, Maroudas A. The chemistry of the intervertebral disc in relation to its physiological function. Clin Rheum Dis 1980; 6:51-76.
43) Urban J, Holm S, Maroudas A. Diffusion of small solutes into the intervertebral disc. Biorheology 1978; 15:203-23.
44) Maroudas A. Nutrition and metabolism of the intervertebral disc. In: Gosh P, ed. The Biology of the Intervertebral disc, Vol. II. Boca Raton:CRC Press; 1988: Ch 9:1-37.
45) McNab I. The mechanism of spondylogenic pain. In: Hirsch C, ZottermanY, eds. Cervical Pain. Oxford: Pergamon; 1972; 89-95.
46) Smyth MJ, Wright V. Sciatica and the intervertebral disc. An experimental study. J Bone Joint Surg 1959; 40A:1401-18.
47) Bogduk N. Clinical Anatomy of the Lumbar Spine and Sacrum, 4th ed. Philadelphia: Elsevier Churchill Livingstone; 2005, Ch 15:185-86.
48) Olmarker K, Rydevik B, Nordborg C. Autologous nucleus pulposus induces neurophysiologic and histological changes in porcine cauda equine nerve roots. Spine 1993; 18:1425-32.
49) Olmarker K, Byrod G, Cornefjord M et al. Effects of methylprednisolone on nucleus pulposus-induced nerve root injury. Spine 1994; 19:1803-8.
50) Olmarker K, Nordborg C, Larson K et al. Ultrastructural changes in spinal nerve roots induced by autologous nucleus pulposus. Spine 1996; 21:411-14.
51) Olmarker K, Myers RR. Pathogenesis of sciatic pain: role of herniated nucleus pulposus and deformation of nerve root and dorsal root ganglion. Pain 1998; 78:99-105.
52) Kayama S, Olmarker K, Larsson K et al. Cultured, autologous nucleus pulposus induce functional changes in spinal nerve roots. Spine 1998; 23:2155-58.
53) Eyre D R. Collagens of the disc. In: Ghosh P, ed. Bogduk N. The Biology of the Intervertebral disc, Vol 1. Boca Raton: CRC Press; 1988: 171-188.
54) Haines DE. The Meninges. In:Haines D, 3rd edn. Fundamental Neuroscience for Basic Applications. Philadelphia:Churchill Livingstone Elsevier; 2006: 106-22.
55) Bogduk N. Clinical Anatomy of the Lumbar Spine and Sacrum, 4th ed. Philadelphia: Elsevier Churchill Livingstone; 2005, Ch 10:127.