Osteopathic Considerations of Discogenic Problems

Christian Fossum, D.O.

Lecture Content

- Anatomy, physiology and biomechanics of the intervertebral disc
- Categories of low back and referred pain
- Pathogenesis of disc pain
- Epidemiology and prevalence
- Natural course and prognosis of intervertebral disc diseases
- Manipulative care and the intervertebral disc
  - Spinal manipulative therapy
  - Traction for disc herniations (vertebral axial decompressive therapy)
- Safety and complications
- Osteopathic considerations in management of intervertebral disc conditions
  - Biomechanical considerations
  - Neuroreflexive considerations
  - Circulatory considerations
- Conclusion
Introduction

Classification of Spinal Disorders

Classification of Spinal Disorders (after Boos, 2008)

<table>
<thead>
<tr>
<th>Specific Spinal Disorders</th>
<th>Non-specific Spinal Disorders</th>
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<tbody>
<tr>
<td>With clearly identifiable pathomorphological correlate such as (10 – 15 %)</td>
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<tr>
<td>• Congenital</td>
<td></td>
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<tr>
<td>• Developmental</td>
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<tr>
<td>• Traumatic</td>
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<td>• Infectious</td>
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<tr>
<td>• Tumorous</td>
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<tr>
<td>• Metabolic</td>
<td></td>
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<tr>
<td>• Degenerative (depending on the disorder)</td>
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<tr>
<td>Without clearly identifiable pathomorphological correlate (85 – 90 %)</td>
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<tr>
<td>• Non-specific axial neck pain</td>
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<tr>
<td>• Non-specific axial dorsal pain</td>
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<tr>
<td>• Non-specific axial back pain</td>
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There are many potential causative and aggravating factors associated with this category, but no structural pathology can with certainty be held responsible

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<tr>
<th>Structure / Pathology</th>
<th>Prevalence</th>
<th>Source</th>
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<tbody>
<tr>
<td>Discogenic pain (IDD)</td>
<td>39%</td>
<td>Schwarzer et al 1995</td>
</tr>
<tr>
<td>Sacroliliac Joints</td>
<td>6 – 13% (16 – 21%)</td>
<td>Schwarzer et al 1995, Bogduk 1995</td>
</tr>
<tr>
<td>Nerve root (radicular pain)</td>
<td>2 – 10%</td>
<td>Deyo et al 1994, Govind 2004</td>
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</table>
Patients with low back pain can present with a variety of signs, symptoms and clinical findings of variable significance relative to degree of severity of the problem, injury, pathoanatomical chances and frank pathology. Low back pain where sinister pathology (spinal infections, fracture, metastases and tumors) as the cause is relatively rare in primary care with an estimated prevalence of less than 1%.

Since patients with low back pain often presents with a clinical picture that may be multifactorial, it is important for the clinician to be able to recognize and identify conditions and possible comorbidities that may have serious consequences for the patient, or may hinder full or optimal recovery from the presenting problem. Numerous Red Flags indicating varying degrees of possible pathology or factors influencing the treatment of the patient is well described in the medical literature.


1. **Nociceptive back pain**
   - Ligaments in the lumbosacral spine
   - Lumbar zygapophyseal joints
   - Sacroiliac joints
   - Posterior surface of the lumbar intervertebral disc

2. **Somatic referred pain**
   - Lumbar zygapophyseal joints
   - Lumbar intervertebral disc
   - Myofascial triggerpoints (MTrP)

3. **Radicular pain**
   - Pain evoked by ectopic discharges emanating from a dorsal root or its ganglion → disc herniations and inflammation of affected nerve seems to be the critical pathophysiological process

4. **Radiculopathy**
   - Neurological state in which conduction is blocked along a spinal nerve or its roots. It is not defined by pain but by its objective neurological signs
A symptomatic lumbar disc herniation occurs during the lifetime of 2% of the general population.

90 – 95% of all lumbar discogenic problems are confined to L4 – L5 or L5 – S1.

Risk factors for development of discogenic problems are:
- Male gender aged 30 – 50 years
- Physical labour with heavy lifting and torsional movement
- Low job satisfaction and low socioeconomical status / income
- Smoking
- Long-term exposure to vibration

Not all disc pathology has clinical importance.

Abnormal MRI findings in lumbar IVD in asymptomatic subjects (Vacarro 2005)

<table>
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<tr>
<th>Age</th>
<th>Prolaps</th>
<th>Protrusion</th>
<th>Disc degeneration</th>
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<tr>
<td>20 – 39</td>
<td>21 %</td>
<td>56 %</td>
<td>34 %</td>
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<tr>
<td>40 – 59</td>
<td>22 %</td>
<td>50 %</td>
<td>59 %</td>
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<tr>
<td>60 - 80</td>
<td>36 %</td>
<td>79 %</td>
<td>53 %</td>
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Anatomy of the Lumbar IVD

- Each IVD consists of three components
  - I. Central gelatinous nucleus pulposus
  - II. Surrounding anulus fibrosus
  - III. Pair of vertebral endplates that sandwich the NP
- Nucleus Pulposus
  - Consists of a matrix of proteoglycans that bind a considerable amount of water
- Anulus Fibrosus
  - Concentric laminae of Collagen fibers
  - Inner fibers of the AF envelop the NP and are attached to the vertebral endplate
  - Outer fibers of the AF are attached to the margins of the vertebral bodies and constitute the ligamentous portion of the AF
- Vertebral endplates
  - Cartilaginous structures that are circled by the ring apophysis. Covers the NP and attaches to AF
  - Only weakly attached to the vertebral bodies

Disc Degeneration Lumbar Spine

- Disc degeneration may be a sequel to injuries and/or incompetence of the disc tissue to bear normal load
  - Pathological disc degeneration or deterioration of the disc is characterized by dysfunctional cells and a decrease in extracellular components, leading to a gradual loss of intradiscal fluid
  - The initiating events are not fully understood, but it may involve poor nutrition as well as mechanical injuries to the disc. In addition, biochemical, immunologic, genetic and nociceptive factors that predispose the IVD to dysfunction has been proposed
- Depending on grade of degeneration, several changes can be seen with use of imaging modalities
  - Decrease of disc height, irregular disc contour with bulging due to deterioration of the anulus fibrosus, centrally abundant presence of gas, osteophyte formation and endplate erosion
**General Classification of Disc Lesions**

- Normal
- Congenital / developmental variant
- Degenerative / traumatic lesion
  - Anular tear
  - Herniation
    - Protrusion / Extrusion
    - Intravertebral
  - Degeneration
    - Spondylosis deformans
    - Intervertebral osteochondrosis
- Inflammation / Infection
- Neoplasia
- Morphologic variant of unknown significance

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Innervation: normal and degenerated IVD

- In human and animal models of degenerating IVDs, especially painful IVDs, it has been observed that innervation is increased and that the nociceptive nerve fibers grow into what are usually aneural inner parts of the AF and even into the NP, sometimes together with blood vessels.

- It has also been observed in degenerated IVDs an increase in the number of Golgi-tendon organ-like structures such as Ruffini’s and Pacinian corpsules.

- In addition to sensory nerve fibers, there is growing evidence that sympathetic afferents are also increased in degenerated IVDs and that they play a significant role in LBP.

Garcia-Cosamalon et al (2010)
Innervation of a normal IVD


Innervation of the disc

- Each lumbar disc is innervated by multiple sources
- Anterior and lateral the anulus receives nerves derived from branches of the sympathetic trunk and its grey rami communicantes
- Posteriorly the annulus receives branches derived from the sinuvertebral nerves, which are the recurrent meningeal branches of the lumbar ventral rami

Innervation of the disc

Sympathetic trunk and its grey rami communicantes (gr)

Anterior

Posterior

Sinusvertebral nerve

Innervation Lumbar ZAJs: Dorsal Rami

Giles (1989, 2009)
IVD Innervated by Multiple Segments

- It has been widely believed that the lumbar IVD is segmentally innervated by the dorsal root ganglion (DRG) neurons through the sinuvertebral nerve
- However, data from animal studies in the 1990s demonstrated that the L5 – L6 disc in rats was innervated by upper (L2) DRG neurons and other studies confirmed the L1 and L2 innervation (Takahashi et al, 1993, Moringa et al, 1996)
- It was also shown that the posterior potion of the lumbar disc was innervated by sympathetic nerves multisegmentally and bilaterally (Nakamura et al, 1996)
- Discogenic groin pain (L2 segment) has been confirmed in human subjects (Oikawa et al, 2012) adding credibility to data from animal studies

Core Message:

- The Lumbar IVD receives multisegmental innervation through the sinuvertebral nerve and sympathetic trunks via the dorsal root ganglion (DRG)
- Treating discogenic pain from the osteopathic perspective should include evaluation and treatment of the thoracolumbar region

The Role of Neurotrophins in Discogenic Pain

- The increased innervation of the injured lumbar IVD is not fully understood
- Neurotrophins (NTs), known to have both neurotrophic and neurotropic properties and regulate the density and distribution of nerve fibers in peripheral tissue, seems to play an important role
- Indeed, biological evidence suggests that NTs play a role in the genesis and maintenance of painful stimuli from degenerating IVDs
- NTs, together with extracellular matrix modifications and some cytokines, regulate the nerve ingrowth into the IVD, the synthesis of pain-related peptides in the IVD itself but especially in the DRGs

The Degenerative Cascade (after Kirkaldy-Willis 1983)

The progress of degeneration can be divided into three phases:

- **Stage I (Dysfunction):** changes in biochemical composition, physiology and biomechanics of the motion segment may result in the clinical symptoms
- **Stage II (Instability):** increased mobility at the affected level
- **Stage III (Stabilization):** spinal osteophyte formation (symptoms due to facet joint hypertrophy and spinal stenosis)

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Cartilage Degeneration

Facet hypertrophy and narrowing of spinal canal

Asymmetrical Disc Injury at One Motion Segment

Disturbed Kinematics of the Motion Segment

Unequal sharing of facet loads

Before changes in the physiology of the disc: physiological movements of the spine as described by Fryette (1918, 1954) may apply

Coupled motion pattern of the motion segment influenced by changes in weight distribution

Lumbar facet joints Load Transfer:
Normal: ca. 18% Now: ca. 70%
Manipulative Care and the Intervertebral Disc


James Cyriax, M.D.:
Major advocate of manipulative therapy for treatment of discs

- Used Mixter and Barr’s seminal 1934 NEJM on intervertebral disc problems to discredit the theories and methods of osteopathic physicians, osteopaths and chiropractors in treating back pain
- The advocacy of his approach was not based so much on existing pathology but on the clinical model he proposed
- His manipulative techniques in the spine can best be described as long-lever, low specificity, high-velocity thrusts with excessive traction and multiple assistants
- Based on the premise that tightening up the anterior and posterior longitudinal ligaments would “reposition” the disc pathology (protrusion, prolaps, extrusion, or sequestration)
- Little or no experimental or clinical data to support this claim and practice

- “Since manipulation, as practiced by orthopedic surgeons, has proved unsuccessful in most cases, the general attitude to disc lesions now is to prescribe rest and, if this fails, to operate and remove part of the disc”
- “This is unfortunate, because manipulation has a most important place in the treatment of intervertebral disc conditions”
- “There is a great deal of difference between accurate skilled manipulation and the haphazard snapping of spines under anesthesia”
- “By correct and appropriate manipulation it is often possible to reduce prolapsed discs, to re-align joints and to remove mechanical stresses and strains”
- “The essential point about the manipulation is that it should never aggravate the condition”

**Risk Management: Considerations**

- The prevalence of symptomatic disc herniation has been estimated at 1 and 3 %, with 95 % occurring in the lower lumbar spine (Andersson, 1997)
- Sciatica accompanies about 10 % of low back pain episodes, and nerve root compression by disc herniation is regarded as the most frequent cause of sciatica (Vroomen et al, 2000)
- Symptomatic disc herniation is therefore probably a common presentation in osteopathic clinics (Snelling, 2006)
- With regard to manipulation, a great deal of controversy exists
  - Some authors state that it is absolutely contraindicated in cases of disc herniations (Corrigan and Maitland, 1983)
  - Others advocate its use and clinical efficacy (Cyriax, 1985, Cox, 1993)
  - Attitudes of UK osteopaths in a survey indicated that 54 % of respondents would sometimes employ manipulation in the presence of disc herniation, and most others described this practice as “dangerous” (Rebain et al, 2003)
  - In the United States it is reported that disc herniation is the leading cause of claims against chiropractors (Jagbandhansingh et al, 1997)

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Patients</th>
<th>Intervention</th>
<th>Follow-up</th>
<th>Outcome measures</th>
<th>Main result</th>
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<tbody>
<tr>
<td>Colthead, 1993</td>
<td>RCT,</td>
<td>Pain in sciatic distribution, but including just buttock pain. Disc herniation n = 322 hospital outpatients</td>
<td>(a) Manipulation (Maitland), (b) exercise, (c) traction, (d) corset</td>
<td>4 weeks, 3-4 months</td>
<td>Self perceived improvement, pain scale, return to work</td>
<td>No significant difference in outcomes</td>
</tr>
<tr>
<td>Matthews, 1997</td>
<td>RCT,</td>
<td>LBP and reduced SLR. Pain and /or nerve root pain. NOT image confirmed</td>
<td>(a) Manipulation (n = 132) up to 2 weeks, daily if necessary, (b) heat (n = 101) 3 times a week, for 2 weeks</td>
<td>2 weeks, 1, 3, 6 and 12 months</td>
<td>Pain scores</td>
<td>(a) &gt; (b) at 2 weeks, other results not presented</td>
</tr>
<tr>
<td>Liu and Zhang,</td>
<td>RCT</td>
<td>112 in-patients with CT confirmed disc herniation</td>
<td>5 weeks of (a) manipulation (n = 62), (b) traction (n = 50)</td>
<td>3, 4 and 5 weeks</td>
<td>Unclear</td>
<td>(a) &gt; (b)</td>
</tr>
<tr>
<td>Burton, 2006</td>
<td>RCT</td>
<td>40 sciatica with MRI confirmed disc herniation</td>
<td>(a) Osteopathic care (n = 20), over 12 weeks, or (b) chemonucleolytic (n = 29)</td>
<td>2 and 6 weeks, 12 months</td>
<td>Roland–Morris + pain scores</td>
<td>(a) &gt; (b) at 2-6 weeks, no differences at 12 months</td>
</tr>
</tbody>
</table>

RCT, randomised controlled trial; LBP, low back pain; SLR, straight leg raising.

Osteopathic Manipulative Considerations of Discogenic Problems
Specific Spinal Pathology:
- Biomechanical Theory
- Imaging Findings

Patient Response Characteristics:
- Subgroup Identification
- Clinical Outcome

Exclusion of red flags (<1%)
Diagnostic work-up
Classification of LBP
- Nociceptive back pain
- Somatic referred pain
- Radicular pain
- Radiculopathy
Management plan specific to patho-anatomical and functional changes

DIRECT METHODS
INDIRECT METHODS
COMBINED METHODS
HOMEOSTATIC METHODS

Biomechanical – Postural Model
Neurological Model
Respiratory – Circulatory Model
Biopsychosocial Model
Metabolic Model

DIRECT METHODS
INDIRECT METHODS
COMBINED METHODS
HOMEOSTATIC METHODS

Postural Control and Movement
Integrated Autonomic Function
Circulation
Behavior and Adaptation
Metabolic, Immune and Endocrine

The Total Musculoskeletal System:
"Primary Machinery of Life"

"Motion is Lotion"

Medical Dimensions of Manual Medicine and its role in the management of LBP

Pain perception level:
Modification or assistance through various other treatment interventions

Functional level:
Manual medicine intervention, exercise program, reconditioning therapy, physical therapy

Structural level:
Structural diagnosis, Risk-benefit ratio, potential contraindications to intended treatment and the patient's candidacy for other medical interventions

Perspectives

Prevalence
- LBP: one of the leading causes for why patients seek medical care
- 97% of all LBP are of a mechanical nature (Deyo et al, 2001)
- “Tissue causing symptoms” prevalence lists three major progenitors of LBP:
  - Lumbar IVD 39%
  - Lumbar ZAJ 15 – 40%
  - Sacroiliac Joints 16 – 21%
- Patients with CLBP have greater severity of diagnostic findings characterizing the somatic dysfunction (TART)

Effectiveness of Care
- Studies and Guidelines support that Osteopathic Manipulative Treatment (OMT) is effective in management of LBP
  - NICE Guidelines. Low back pain: the acute management of patients with chronic (longer than 6 weeks) non-specific low back pain. Issued May 2009, (The National Collaborating Center for Primary Care and the Royal College of General Practitioners, United Kingdom)

OMM for Pain Reduction
- Pharmacologic studies in humans and animals have begun to decipher potential mechanisms in the central nervous system underlying the analgesia produced by joint manipulation
- The non-opioidergic descending inhibitory pathways (serotonergic and noradrenergic) seems to be the major players
- Clinical context
  - Treatment of non-injured adjacent areas may result in pain reduction through these mechanisms
  - This has been confirmed in experimental studies (Skyba et al, 2003, Hoeger-Bement and Sluka, 2006, Sluka, 2006)
- Effect is most likely non-segmental but more depending on CNS response
Snider KT, Johnson JC, Snider EJ, Degenhardt BF
Increased Incidence and Severity of Somatic Dysfunction in Subjects With Chronic Low Back Pain

Table 1
Incidence and Severity of Somatic Dysfunction for Chronic Low Back Pain and Non-Low Back Pain Groups (N=60)

| Palpation Examination | Chronic LBP (n=50) | Non-LBP (n=10) | P Value
|-----------------------|-------------------|----------------|--------
| Tissue texture changes | 61 (95.3)         | 174 (92.6)     | .54    |
| Static rotational asymmetry | 61 (95.3) | 166 (88.3)     | .21    |
| Resistance to anterior springing | 59 (92.2) | 133 (70.7)     | <.001  |
| Tenderness | 33 (51.5)         | 29 (15.4)      | .002   |

Severity Rating of Somatic Dysfunction/ Movement
50%: Normal; >50%: Favorable; Absence of Mechanics

Results: Resistance to anterior springing (P<.001) and tenderness (P=.002) were found at significantly greater incidence in the chronic LBP group than in the non-LBP group, but there were no significant differences between groups for incidence of tissue texture changes or static rotational asymmetry. Significantly greater severity of tissue texture changes (P=.006), static rotational asymmetry (P=.008), resistance to anterior springing (P<.001), and tenderness (P=.001) were observed in the chronic LBP group than in the non-LBP group.

Conclusion: When compared with non-LBP subjects, chronic LBP subjects had overall greater severity for each of the four elements of somatic dysfunction evaluated, as well as greater incidence of resistance to anterior springing and tenderness. Somatic dysfunction is more severe in individuals with chronic LBP than in individuals without chronic LBP.

Natural history

Favorable indications for non-operative treatment*
- sequestered disc herniation
- young age
- minor neural compromise
- small herniation
- mild disc degeneration
- mild to moderate sciatica

*Although based more on anecdotal experience than scientific evidence, several factors have been associated with a favorable outcome of non-operative treatment


- The natural history of discogenic problems and radiculopathy is usually benign
- Acute episodes of sciatica takes a brief course: this phase is normally followed by a subacute or chronic period of residual symptoms but most patients recover within 1 month, but the recurrence rate is approximately 10 – 15 %

Prognostic factors of positive outcome with conservative intervention for lumbar disc herniation*

<table>
<thead>
<tr>
<th>Favorable</th>
<th>Unfavorable</th>
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<tbody>
<tr>
<td>Absence of crossed SLR; spinal motion in extension does not reproduce leg pain; relief or &gt; 50% reduction in leg pain within 6 first weeks after onset; limited psychosocial issues; self-employed; educational level &gt; 12 y; absence of spinal stenosis; progressive return of neurological deficit within first 12 weeks</td>
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<tr>
<td>Positive crossed SLR; leg pain produced with extension; lack of &gt;50% pain reduction in first 6 w; overbearing psychosocial issues, Worker’s compensation; Educational level &gt;12 y; concomitant spinal stenosis; cauda equina syndrome</td>
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</tr>
</tbody>
</table>

*Saal JA. Natural history and nonoperative treatment of lumbar disc herniation. Spine 21: 25 – 95, 1996
**Effective office management of LBP**

**Discogenic Pain:**
- Internal disc disruption
- HNP
  - Protrusion, Extrusion or Sequestration

**Lumbar Zygaphophyseal or Facet Joint Pain (FJP)**

**Sacroiliac Joint Pain (SIJP)**

- Pain intensity and pain distribution
- Degree of neurological involvement
- Contributing factors: what is amenable to osteopathic manipulative treatment?

**Predicted probabilities and 95% CI for internal disc disruptions (IDD), facet joint pain (FJP), sacroiliac joint pain (SIJP) and other sources of LBP**

Diagnostic Considerations: Radicular Pain and Radiculopathy

- The affected nerve root and its degree of involvement can usually be determined through sensory, motor and reflex testing.
- Sensitivity for sciatic distribution of pain in the diagnosis of lumbar disc herniation of 90% and calculated likelihood ratio of disc herniation being present in the absence of sciatic pain as 0.1%
- Radiculopathy is such a sensitive finding (95%) that its absence almost rules out a clinically important disc herniation.
- The SLR and its variations are good markers for radicular pain / radiculopathy involving the 4th and 5th lumbar as well as 1st to 3rd sacral nerve roots.
- SLR may be positive with 3rd lumbar nerve root involvement, but the likelihood is greater that the femoral nerve stretch test will be positive.


Radicular Pain and Radiculopathy

- **L3 – L4 Disc → L4 Nerve Root**
  - Pain in L4 dermatome (radicular pain)
  - Sensory loss in L4 dermatome (radiculopathy)
  - Loss of muscle force: inversion of foot (radiculopathy)
  - ↓ DTR: Patellareflex

- **L4 – L5 Disc → L5 Nerve Root**
  - Pain in L5 dermatome (radicular pain)
  - Sensory loss in L5 dermatome (radiculopathy)
  - Loss of muscle force: Extensor Hallucis Longus Muscle
  - ↓ DTR: None

- **L5 – S1 Disk → S1 Nerve Root**
  - Pain in S1 dermatome (radicular pain)
  - Sensory loss in S1 dermatome (radiculopathy)
  - Loss of muscle force: eversion of foot (radiculopathy)
  - ↓ DTR: Achillesreflex

- **Central Prolaps → Symptoms may be as in CES**
Pain Patterns: IDD, ZAJ and SIJ

- This study looked at the predictive value of pain localization relative to structures causing it
- $N = 170$, Average age 54.4 yr, LBP average 12 months
- Provocative discography, ZA- and SI joint blocks
- Discogic (IDD), Lumbar ZAJ og SI joint pain
- Calculated sensitivity, spesificity, positive and negative predictive value, diagnostic accuracy and LR +/-
- Pain median (over the SPs) $\rightarrow$ IDD and reduces the likelihood for ZAJ and SIJ as pain generators
- Isolated paramedian pain $\rightarrow$ increased likelihood for ZAJ and SIJ as pain generators

Distribution of pain in internal disc disruption (IDD):
Axial midline pain (over SPs) with possibility for pain in buttock and groin region (Skogsbergh et al, 2001)

Use of Translatory Motion for Pain Provocation with IDD

If tolerated by the patient, translatory motion in seated position may be used to induce mechanical stress on the IVD with IDD, and may be used for pain provocation. In addition, resistance to motion at segment with pain is noted.

Traditional motion testing using flexion, extension, SB and rotation, may, despite potential displacement of the nucleus, be insufficient to provoke pain from a lumbar IVD with IDD. With a HNP these tests, especially flexion and extension may produce pain.

Summary: Findings with IDD

A provocative discogram is considered the “gold standard” for determining pain of discogenic origin (IASP). However, there are numerous clues from the physical examination that may help indicate the condition:

- Age: <40 years
- Translatory motion testing to stress the lumbar IVD
  - Motion stress to the IVD to provoke pain with IDD
- Compression and traction test
  - Positional loading testing to reproduce the pain: midline pain, well-localized and well-defined
- Provocative AP pressure in prone position
  - Overpressure at vertebra with well-localized, well-defined midline pain corresponds well with IDD, especially in the lower lumbar spine and lumbosacral junction
- Vibratory pain provocation on spinous processes
  - In lateral recumbent position: symptomatic side down→ vibratory stimulation applied to SP
  - Can elicit pain associated with internal disc disruption: agrees with provocative discography in 70.9% to 85.9% of discs. Comparatively, MRI specificity was 55.7


Yima M et al. Bony vibration stimulation test combined with magnetic resonance imaging. Can discography be replaced? Spine 1997; 22(7): 808 – 803
Somatic Dysfunction

- From a biomechanical perspective the spinal somatic dysfunction is to be considered a tripod system where dysfunction involves all active and passive structures of the functional spinal unit (FSU).
- The sensorimotor control of the spine depends on normal interaction between the active and passive components of the FSU.
- Somatic dysfunction alters the sensorimotor control of the spine through the FSU.
- The three legs of support in the FSU is the intervertebral disc and the two zygapophyseal joint.
- They are all three involved in the mechanics of somatic dysfunction.
- Although the osteopathic literature describes Type I and Type II mechanics in the lumbar spine, natural aging and degeneration of the structures of the FSU will alter the coupling behavior of the motion segment making it less predictable.

Instantaneous Axes of Rotation: Lumbar Spine

- Flexion and extension
  - Caudal portion of intervertebral disc close to the intervertebral endplate
- Sidebending
  - In the central portion of the intervertebral disc slight displacement to the left or the right during sidebending
- Rotation
  - Central (Panjabi, 1978) or posterior (Farfan, 1986) portion of the intervertebral disc.

Key Message: The lumbar IAR all seem to be located in the IVD

- The IVD is weight bearing element and controls movement of the Functional Spinal Unit
- The facet joints are only in spatial apposition whose function is to guide movement
- Coupled motions are less influenced by contractile elements
- Type I (NS, R₁) and Type II (E or F, R₂, S₂) mechanics would apply here

- With disc degradation or degenerative changes, the weight bearing of the facet joints can be up to 70% of imposed weight (Adams et al 2001)
- The facet joints are in apposition through the arc of movement
- Type I (NS, R₁) and Type II (E or F, R₂, S₂) mechanics would not necessarily apply here
Osteopathic Considerations: Biomechanical

- Muscle Energy Techniques (MET)
  - If tolerated by the patient → seated position
  - Use translatory motions
    - A→P translation for flexion and extension: may prevent unwanted movement of the nucleus pulposus as with pure F or E
    - Lateral translation: with the sidebending component the coupled rotation will follow automatically. Less concerns about altered coupling behavior at FSU
- PIR variation: restore motion at FSU
- Ruddy variation: reduce venolymphatic congestion
- MET can also effectively be used to improve sensorimotor control of the spinal motion segment
  - Ensure to keep to contraction effort from the patient light in order to prevent recruitment from the polyarticular muscles

Combined Leverage and Thrust in the Lumbar Spine in Patients with Discogenic Pain

- The spine is kept in a neutral position and the technique utilizes physiological locking of the spine from above and below (e.g. myofascial locking as opposed to approximation of joint surfaces)
- Why neutral position?
  - Mathematical analyses of the lumbar IVD shows that with bending moments (flexion) the stress on the AF is 450 times greater than with spinal rotation and they conclude that there is no radial expansion or extrusion (bulging) during twisting
  - Extension may aggravate the patients symptoms
  - In neutral there is less stress or strain on inflamed or injured tissue (pain ↓)

Thoracolumbar Junction: A major player in LBP and Discogenic Pain

LBP of Thoracolumbar Origin

- LBP of thoracolumbar origin is common in clinical practice, it may be acute or chronic, and its character is similar to that of pain of lumbosacral origin
- This may also include pseudovisceral abdominal pain, pubic and trochanteric tenderness associated with this
- Note the anterior ramus (1): groin pain of discogenic origin has recently been demonstrated in L2 distribution

Distribution of spinal nerves T12 and L1:
1. Anterior ramus
2. Posterior ramus
3. Perforating lateral cutaneous branch

**TL Junction: Biomechanical Stress Inducer**

- The lumbar zygapophyseal joints (ZAJ) are designed to limit or block axial rotation.
- The protects the intervertebral disc from excessive torsion.
- Although approximating the sagittal plane, the surface of the lumbar ZAJ are either flat, C-shaped or J-shaped.
- On average there is 2–3° of axial rotation possible per segment.
- There is a sudden change of joint structure occurring in the region of a single vertebra T12: orientation of ZAJ changes abruptly.
- Lack of axial rotation in the lumbar spine is compensated at this junction.
- Motion restriction at T12 may increase the biomechanical stress in the lumbar spine.


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**Stabilizing Role of the TL Junction**

- An early signal to the stabilizing role of the human thoracolumbar junction (TLJ) appears in the sequence of ossification of the vertebral centra.
- A number of reports have indicated that the first sites of ossification are consistently located in the lower two thoracic and first lumbar vertebral bodies before a progressive cranial and caudal pattern of ossification commences in the adjacent vertebrae.
- The reciprocal change in curvature of the thoracic kyphosis and lumbar lordosis produces an inflexion point commonly located between T11 and L1.
- The morphology of the TLJ is highly variable, but the morphology may aptly be described as an “anti-torsion” device limiting torsional stress in the region.
- Data from Donish and Basmajian (1972) indicates that the multifidi muscles in the TLJ are antagonistic to axial rotation and thereby prevents torsion.
- In addition to resisting axial rotation, in the TLJ extension is “closed-packed position” → mechanics of spinal HVLA in the region may needs revisiting.


HVLA in the Thoracolumbar Region

- Arguably, HVLA techniques using extension and rotational stress may be difficult to apply with a high degree of specificity in the TLJ
- A modification of the Kirksville Crunch (aka Dog Technique) to involve the sagittal plane only can successfully applied to separate or cavitate the ZAJ of the TLJ
- This technique is also very useful for treatment of the TLJ in patients with pain of discogenic origin as it places minimal stress on the lumbar spine

So what then is the function of the psoas muscle?

- One of the primary functions is stability of the lumbar spine (Bogduk et al, 2002, Gibbons, 2007)
- This is because the axial compression is bigger than the shear forces acting on it (McGill, 2002)
- Because the muscle crosses the pelvis and the SIJ it may contribute to stability of the SIJ
- Because of its pivot point on iliopectineal eminence and the resulting leverage it will cause a posterior rotation of the innominate (Gibbons et al, 2001, Gibbons, 2007)
- The iliac muscle will rotate the innominate anterior in an anterior direction (Neumann, 2010)
- The antagonistic functions of these two muscles on the pelvis contributes to the axial compression of the lumbar spine and stability of the lumbosacral junction (Neumann, 2010)
Psoas Muscle and Discogenic Pain

- The axial compression on the lumbar IVD from the psoas muscle may negatively influence both the cell mechanics and the extracellular matrix mechanics in the annulus fibrosus of the IVD.
- Unilateral axial compression from psoas muscle on one side only may shift the nucleus pulposus (bulging or extrusion) and also increase the torsional stress on the annulus fibrosus (→ pain from radial fissures).
- The axial compression on the lumbar spine from the psoas muscle may also restrict motion segmentally in the lumbar ZAJ with exacerbates the condition at the involved FSU.

TLJ, Lumbar IVD and Lumbosacral Mechanics

- Typical Pattern:
  - The Progenitor: Type II Somatic Dysfunction TL Junction Region → Psoas Tension +++
  - Axial Compression or Rotational Stress Lumbar IVD
  - Sacral Rotation on Oblique Axis / Sacral Tension or Unilateral "Shear" in the form of a Unilateral Sacral Flexion
  - Piriformis Tension +++
  - SD and Stress L5 – S1
Suggestions on treating the Psoas Muscle

- Treatment of the thoracolumbar junction and upper lumbar spine prior to treating the psoas muscle in patients with discogenic pain
- Counterstrain: this technique is helpful in addressing the psoas muscle specifically. Although the position requires a great deal of flexion with SB and rot, patients with discogenic pain usually tolerates it well
- If condition is very acute, consider holding the position of comfort for longer than 90 seconds
- James Cyriax, M.D. used the “Dallison Technique” (see picture) on patients with acute discs
- This technique bears resemblance to how osteopathic physicians treat the psoas muscle using counterstrain

Thoraco-Abdomino-Pelvic Pump

- Respiration is an activity where numerous systems and all body tissues are involved (Cathie, 1965, 1974)
- Thoracoabdominal junction: vertical and transverse plane where we have integration of somatic, respiratory, vascular, neural and visceral functions (Cathie, 1974)
- < 23,000 respiratory cycles per day
- The diaphragm is the extrinsic pump of the venous and lymphatic system: asymmetries with reduce the respiratory efficacy through distortion of the cylinder (Zink, 1970, 1973, 1977, Mitchell, 1984)
- Important in LBP and discogenic conditions
Circulatory Considerations

- The vessels of the venous system can also play a role in the generation or worsening of disk-related symptoms

- Relevant anatomy
  - The valveless veins of the spinal canal form an uninterrupted anastomotic chain running from the skull base to the sacrum
  - The degree of filling of the lumbar epidural veins depends on the central venous pressure
  - The venous plexus of the spine are also a venous pathway connecting the superior and inferior vena cava
  - Together with the azygos system, they form a collateral venous circulation that operates beyond the local level, coming into play physiologically whenever the venous pressure is elevated in the thoracic, abdominal, or intracranial cavities
  - Elevations of pressure in the chest and abdomen worsen disk-related pain because they increase the degree of filling of the epidural veins

- Clinical considerations
  - Reduce congestion
  - Influence absorption of material from extrusion & sequestration

Osteopathic Considerations: Circulatory

- Opening up the Thoracic Inlet Region
  - Anecdotal information that this may be beneficial in patients with LBP: Drs. Alexander McWilliams, D.O. and J. Gordon Zink, D.O., FAAO

- Thoracic Lymphatic Pump
  - Influences respiration and thereby the central venous pressure

- Treatment of the Respiratory Diaphragm

- Treatment of the Lesser Omentum
  - To influence the portal venous system

- Liver Pump

- Releasing the Mesenteries

- Abdominal Pump with Respiration
  - Potential stronger effect on intracavital pressures

- Supine rhythmic traction Lumbar Spine
  - Move fluid at a segmental level (FSU)

Example: Treatment Protocol Lumbar IVD

Respiratory – Circulatory Model:
- Venolymphatic drainage ↑
- Arterial supply ↑
- Pressure nerve root ↑
- Absorption disc material ↑
- Improved biochemical environment around nociceptors

1. Treat the thoracic inlet
2. Treat the respiratory diaphragm
3. Release the lesser omentum
4. Perform the liver pump
5. Release the mesentery
6. Move fluids: lymph pumps

Biomechanical – postural model and neurological model:
- Nociception and pain ↓
- Function and motion ↑
- Biomechanical stress ↑
- Optimizing load transfer
- Sensomotor control ↑

1. Treat the thoracolumbar junction
2. Treat the psoas muscle
3. Treat motion segment restrictions in the lumbar spine
4. Treat the pelvic girdle
5. Rhythmic traction lumbar spine
6. Treat the neural tissues

LBP: Why not try a little Osteopathy?

Thoracolumbar Junction:
- LBP → Sup. cluneal nerve (T12 – L2)
- Biomechanical stress TLZ A-joints
- SD → Tension psoas muscle +++
- Somatosympathetic reflexes IVD

Lumbar IVD
- OMM for centrally mediated pain ↓
- Decrease mechanical stress on FSU
- Restore function to FSU
- Treat tissue causing symptoms: IVD and neural tissues

Greenman's Dirty-Half Dozen:
- Leg-length discrepancy / sacrum tilt
- Type II SD in the lumbar spine
- SD Pubic symphysis
- SD Innominate shear
- Sacral dysfunctions
- Muscle imbalance LPH - region

Respiratory - Circulatory:
- Impaired central venous pressure
- Epidural veins → discogenic pain
- Decreased tissue drainage: pain ↑
- Sensitization in area of inflammation
- Cranial, thoracic and lumbar cavities

SIJ and Radicular Pain
- Substance P and CGRP identified SIJ
- Extravasation pattern from SIJ
- Inflammation → L5 root / sheath
- No neurological findings
- Untypical SIJ pain pattern