DISOGENIC LOW BACK PAIN

When pain persists

Can we intervene?
The nature of low back pain

• Elusive diagnosis for specific causative lesion
• Lends itself to speculation, theory, patho-anatomical models.
• Discogenic low back pain is a serious medical and social problem, and accounts for 26%-42% of the patients with chronic low back pain.¹,²,³.
• The prevalence of zygapophysial joints, sacroiliac joints, and lumbar discs was 31%, 18%, and 42%, respectively.¹
• The disc as the most common etiology of chronic low back pain in adults.⁴
References


I’m confused... then why...?

• Then why does prolotherapy & our regen med techniques work on so many...?
  – Its about stiffness
  – Stabilization of subtle instabilities
  – A lot of what you are calling SI pain is NOT SI!
  – Multiple pain generators... “the breakdown”
    • 50/50  70/30  60/40  80/20
  – The category of discogenic back pain
  – The mechanical behavior and internal disc dynamics.
    • Some discogenic syndromes are not amenable to prolotherapy of the posterior elements.
Care depends upon the model

- **Manual therapist:**
  - Manipulation, traction, massage
  - FOCUS = facet joint, sacroiliac joint, trigger points, etc.
- **Physical therapy:**
  - Exercise, conditioning
  - Physical modalities
  - “CORE stab”
- **Physician:**
  - Muscle relaxants – muscle spasm
  - NSAIDs – inflammation
- **Pain specialist:**
  - Narcotics, epidural steroids, facet joint steroids, etc.
  - Intra-discal interventions – primarily ablative technologies.
- **Regen Med:**
  - Focus on ligaments and connective tissues.
  - Slow to recognize the disc as a source of pain.
Finding the nociceptive pain foci: can the average clinician accomplish this?

- How often is an MRI diagnostic?
- In the absence of disc herniation and neurologic deficit comprehensive physical assessment, diagnostic technology (MRI, EMG, etc.) identifies cause of low back pain ______% of the time?

How can we use this out?

- X-rays
- EMG/NCV
- CT scans
- MRI
- SNRB, ESI
- Facet & SI blocks
- Pain mapping with local anesthetics
- Ultrasound
- Exam
- Loading
2 types of discogenic back pain etiologies...

Discogenic back pain classified in two types: In our a previous study, according to discography, we classified discogenic low back pain into two types:¹

– Internal annular disruption IAD.

– Internal endplate disruption (IED)

End-plate is innervated by divisions of the gray rami of the sympathetics and sinuvertebral nerve.²

The endplate

- Endplate – cartilaginous and bony transition.
- Insertion of annular fibers into the end plate cartilage at the inner annulus junction.
- Vascular sinusoids in the marrow space adjacent to the end plate.
The endplate defects

A. End plate cartilage avulsion resulting from bending motion that causes traction at the interface between the end plate and inner annulus.

B. Traumatic node with end plate fragment resulting from excessive compression with a healthy, gel-like nucleus pulposus.

C. Central end plate fracture with exposed trabeculae resulting from excessive compression with a degenerate, fibrous nucleus pulposus.
Endplate innervation

- End plate subchondral bone is innervated by basivertebral nerve, the fibers of which reach the bone marrow along with nutrient arteries that enter the vertebra through the posterior basivertebral foramen.
- End plate innervation is comparable to that of the peripheral annulus.
- Nerve density increases at locations of endplate damage.
- Orange dots demo protein gene product 9.5 (PGP9.5) positive nerve fibers across endplate.
Disc Innervation

- DDD → increased nerve fibers in the disc.
- Nociceptive nerve fibers grow into what are usually aneural inner parts of the annulus and even into the nucleus. ⁴⁻⁵
- In addition to the sensory nerve fibers, there is growing evidence that sympathetic afferents are also increased in degenerated disc and that they play a significant role in low back pain. ⁴⁻⁵
- In human normal disc, protein gene product 9.5-positive nerve fibers, innervate the outer layers of the annulus.⁶
- These nerve fibers are also positive for acetylcholinesterase NFP, SP, CGRP, VIP, neuropeptide Y, C-flanking peptide and synaptophysin.
Disc herniation

• Mechanical stimuli which are normally innocuous to disc nociceptors can, in certain circumstances, generate an amplified response which has been termed ‘peripheral sensitization’.

• This may explain why some degenerative discs are painful and others not.

• There is growing evidence that these pain receptors in painful disc are peripherally sensitized by the activity of sympathetic efferents which may initiate a pain impulse in response to ischaemia, pressure changes or inflammatory irritation.\(^7\)
References disc innervation


J. C. Lotz  A. J. Fields  E. C. Liebenberg
Global Spine J

6. The role of the vertebral end plate in low back pain. J. C. Lotz, A. J. Fields, E. C. Liebenberg

Global Spine J

Innervation

• Disc innervated segmentally.
• Ventral portions of lower lumbar discs are innervated by upper (L1-L2) dorsal root ganglion.
• Posterior lateral portion from L3-L6 DRG. ¹⁻²
• Nerve fibers reach the lumbar disc through the sinuvertebral nerves or from branches of the paravertebral sympathetic trunks. ³
• Clinical studies have indicated those local anaesthetic blocks of L2 nerve root can relief discogenic low back pain.⁴
  – Pulsed radiofrequency to L2 DRG...
references


Disc Innervation

- In the early 1980s, Bogduk\(^1\) clarified the innervation of the outer layers of the annulus.
  - The posterior part of the human disc was supplied not only from the sinuvertebral nerve but also received direct branches in its posterolateral aspect from the ramus communicans or the ventral ramus.
  - Branches from the grey ramus communicans also supplied the lateral aspect of the disc.
  - Anterior discal nerves were observed to arise solely from the sympathetic plexus surrounding the anterior longitudinal ligament.

Neural relationships about the disc
The disc and endplate
Michael Modic M.D.
Predicting discogenic pain on MRI using Modic criteria

- Type I:
  - low signal intensity on T1-weighted images and high signal intensity on T2-weighted images when compared with fatty bone marrow
- Type II:
  - high signal intensity with both images
- Type III, low signal intensity with both images. When two types were present on both sides of the intervertebral space, only one diagnosis was applied: first priority, type I; second priority, type II; last priority, type III.
Type I changes:
- Decreased signal intensity on T1 weighted spin echo images and increased signal intensity on T2 weighted images.
- Disruption and fissuring of the endplates and visualized fibrous tissue.
- Endplate disruption.
- Type 1 can convert to type 2.
Categorizing degenerative disc disease: assessment of changes in the cable bone marrow with MRI
Radiology, 1999 January; 166 (1 Pt 1): 193-199.

• Type 1 changes involve replacement of normal cellular vertebral body marrow by fibrovascular marrow.
• Type 1 endplate enhance after injection of Gd-DTPA, reflecting the vascularity of fibrous marrow.
• Type 1: associated with low signal intensity on T2 (desiccation)
• Type II changes:
  – Increased signal intensity on T1 weighted images and isointense or slightly increased signal intensity on T2 weighted images.
  – In golf endplate disruption and yellow marrow replacement in the vertebral body.
Summary of Modic changes

- MRI: endplate changes (Modic & deRoos)
  - Type I (4%): decreased signal on T1; increased signal on T2; vascularized fibrous tissue
  - Type II (16%): increased T1; isointense T2; local fatty replacement of marrow
  - Type III: decreased T1; decreased T2; advanced sclerosis
What causes these endplate changes?

- 3 prevailing theories:
  - Mechanical
  - Auto-immune
  - Bacterial
- MECHANICAL:
  - In case of a herniation and severe degeneration the loss of nuclear material may increase the shear forces on the endplates and micro fractures may occur.
  - Hence, the Modic type 1 changes might initially reflect bleeding, oedema and vascularisation following trauma or the oedema associated with the repair process after micro fractures within the endplate and the vertebral bone.
- AUTOIMMUNE: **THEORY – O - BROWN**
  - Toxic nucleus tissue invades the endplate and vertebral bone through fractures in the endplates and causes an inflammatory response. It may not only be nucleus. \(^1\)
  - Brown: Break in continuity of endplate leads to sequestered antigen presented to immune cells leading to autoimmune inflammation.
    - Release of metalloproteinases into the intervertebral disc digest nuclear proteins causing rapid IDD.

What causes these endplate changes?

• **BACTERIAL / INFECTION:**
  - Are bacteria present in or around the disc?
  - Stirling et al. who removed nuclear tissue under sterile conditions during surgery for lumbar herniated discs found that 53% of the patients were found to be infected with low virulent anaerobic organisms (Propionibacterium acnes and Corynebacterium propinquum) as opposed to none of the patients who were operated for other spinal disorders [1].
  - They therefore, hypothesised that patients with sciatica have a breach in the structural integrity of the spinal disc, possibly from minor trauma, which allows access by low virulent micro organisms [1].

• **Concept:** Reduce transient Bacteremia. VIBRATION TOOTHBRUSH ???

Schmorls nodes

• One type exist prior to skeletal maturity 2\textsuperscript{nd} to cartilage defects that remain after notocold regression and growth plate closure.

• They can occur after skeletal maturity with herniation of NP through the end plate.
  – This causes fibrovascular marrow changes.
  – Traumatic etiology.
  – IDD with Schmorl’s nodes demonstrate dose response. (Amount of nuclear proteins exposure)

The beginning

- Break in continuity of the end plate.
- Introduction of blood born factors, and autoimmune response in the annulus.
- Degradation of internal disc leading to early fissuring of disc.
Endplate references

IDD: Historical perspectives

• IDD first described by Crock in 1970 and again in 1986.
• Described disruption of internal disc architecture without signs of dural tension or nerve root compression.
• 1995 Schwarzer, April, Derby, Bogduk calculated the prevalence of IDD to be between 30% to 50% within 95% confidence limit.
• They concluded that neither traditional examination findings nor patient’s symptoms could predict whether or not the patient had IDD.
• Validated the importance of discography.

Crock, HV. Internal disc disruption. A challenge to disc prolapse 50 years on. Spine 1986; 11:650-3
Provocation discography

• Subjective component:
  – Is critical to evaluate this step carefully.
  – Non-concordant pain
  – Concordant pain
  – And at what pressure!

• Derby & pressure manometry

• Classification of disc lesion and disease

• The annulogram

• Holt & the discogram dark ages
A case of discogenic back pain

- JL is a 33-year-old Caucasian male reached for a roll of paper towels and felt pop in back. Had LBP with radicular left leg pain. L5 disc was noted.
- At the time of the initial MRI and L5-S1 annular fissure was noted but for the most part ignored.
- Transient improvement and then pain returned. Pain then worsened.
- Pain switched to right leg. Shooting leg pains into both legs lasting 1-2 hours. RTO to surgeon who referred him to Pain Med. Specialist. Started on opioids.
- Had several years of chronic low back and leg pain and unable to work.
- C/O: constant pain across the L4-S1 worsens with activity.
- Sense of “crushing sensation” from muscle tension. Pain extends to buttock with L5 distribution.
- Updated MRI shown to the right.
A case of discogenic back pain.

- What is the first question that I want ???????
- He marked relief on REIL !!!
- What does that mean?

A LOOK INTO INTRADISCAL HYDRODYNAMICS
Internal Disc Dynamics
A Study of 100 Specimens

Jay Shepperd
C. Rand
Hastings England
A case of discogenic back pain

Disco demonstrated the primary painful disc was L4-L5.

Tx:

OUT:
It is NOT what you do at the NP but the AF that counts.

• What are you going to do at the AF tissue to resist intra-disc pressure.

• Derangement...

• **Many strategies** based on classification system and intradiscal dynamics of a given patient !!!

• The wrong strategy ... you can make a patient **WORSE!**
What did we learn?

• We can use **symptomatic response to endrange spinal loading** to determine the presence of discogenic back pain.

• We can **utilize endrange spinal loading as a therapeutic movement** in some patients. Those that do not respond may require intervention.

• We learned that **MRI was nondiagnostic** of the symptomatic disc despite demonstrating HIZ and L4-L5 and L5-S1.

• **Discography or analgesic discography was invaluable** in making the diagnosis and planning therapeutic intervention.

• What we did not discuss his we utilize a Stewart Magill, PhD based spine stabilization program for postprocedural rehabilitation strategies.
Pain worsens with REIL + dural tension?

- Uncontained HNP
  - No centralization
  - REIL worsens leg pain
  - You can predict the findings on MRI with a 1 min exam!
Strategies must vary based on presenting pathology...

- How are you going to deal with the nucleus?
- Shrink back nucleus?
- Remove a portion of the nucleus?
- Are you going to fill void?
- How are you going to change the mechanical behavior of the disc in a given presentation?
- Put a bolus of cells in the disc and you can increase IVD pressure and worsen HNP.
NOTE: NO UNIVERSAL PROTOCOL WILL EVER EXIST !!!!

You must tailor the treatment to
The specific pathological state &
Clinical presentation.

No one tool or protocol fits all problems
Modified McKenzie Examination

Sorting out complex discogenic back pain cases & combined syndromes.
Modified McKenzie:

- The full McKenzie examination involves the exploration of various movement patterns and the symptomatic response to endrange loading to classify and characterize patient’s to develop a plan for prescription of movement strategy and exercise.

- Modified McKenzie:
  - Modified for the interventional spine practitioner.
  - Utilized in conjunction with history, MRI, discography, and other advanced diagnostic interventions to categorize complex cases typically unresponsive to movement, exercise, including McKenzie.
  - Provides a means of assisting in the development of categorization system. typically unrecognized by most interventional physicians.
  - DEMO
Let’s practice

- 42-year-old presents with acute low back pain.
- Back pain predominantly in lumbosacral region.
- Patient in the lateral shift antalgic posture. Rises out of chair in slow and guarded manner.
- Severe pain with spinal extension and standing.
- Patient tolerates lumbar extension in prone position with REIL x 10 reps relieving lower back pain.

Diagnosis?
Let’s practice

• 50-year-old female presents with chronic low back pain of seven years duration.
• Back pain occurs over most of lower lumbar spine extends over the posterior buttock and proximal thigh.
• Neurological examination normal.
• Back pain does not improve with REIL, RFIL, Side glides nor manipulation.
• Marked desiccation and disc space narrowing at L4 L5, and L5-S1.
Let’s practice

• 47-year-old male with history of episodes of back pain generally improved with chiropractic care in the past.

• Patient presents with low back and peripheral leg pain with positive girl tension signs.

• REIL release late pain but does not relieve low back pain.

Diagnosis ?
Let’s practice

• 47-year-old with previous history of low back pain generally relieved with chiropractic manipulation in the past.

• Presents with low back and peripheral leg pain with positive dural tension signs.

• Low back pain and leg pain unrelieved by REIL.

• Peripheral leg pain worsens with REIL.

Diagnosis?
Let’s practice

• 43-year-old female with history of low back pain in the past generally relieved with chiropractic manipulation.
• Presents with low back pain with some peripheral buttock and thigh pain bilaterally.
• Negative dural tension signs
• Back pain relieved with REIL but when she stands pain returns within two minutes.

Diagnosis?
McKenzie or not to McKenzie?

- McKenzie method is a popular treatment for low back pain among physical therapists.
- Clinical studies have indicated that the McKenzie method is slightly more effective than manipulation or is equal to strengthening training for patients with chronic low back pain.¹
- You can’t use anything universally!

McKenzie or not to McKenzie?

- You **can and should** use McKenzie to help triage, classify and categorize your discogenic back pain patient !!!
- You can determine if there is a **directional preference**...
- You can determine if it MIGHT be **appropriate to apply** McKenzie exercises.
- If you do not know the symptomatic response to loading... then you only know 2 things about your patient...


Modified McKenzie - categorization

• Examples described here describe simplistic use of this exam technique.
• Time does not permit description of systematically categorizing of multiple pain generators and complex patients.
• Additional techniques described by McGill.
Rehab or Warehab ???

Movement strategies and core stabilization techniques critical to clinical outcomes.
Real core stab...

• Many progressions of exercises to stiffen and balance the anterior chain and posterior chain in a spine sparing manner.

• Concept:
  – Stiffen, brace and protect a balanced neutral position.
  – Exploration of postures and movements that are triggers for pain and eliminate them.
Rehab vs Warehab

• Robin Robinson, PT
• Stuart McGill, PhD
  – Early am.... No stretching, mob, repeated flexion
  – Side plank not cables.
  – Stir the pot... not crunches!
Provocation Discography & Analgesic Discography

Understanding the controversy
Overcoming the barriers
Modifying methodology
Provocation diskography

• Precision injection of contrast into disk nucleus stimulate nerve endings via 2 mechanisms:
  – Chemical stimulus from contact between contrast in sensitized tissues.
  – Mechanical stimulus resulting from fluid distending stress.
• False positive rate increased with patient’s to demonstrate somatizations, psychometric testing should be considered and included.

Provocation using pressure manometry.

- Total volume injected and should not be >3-3.5 cc.
  - Otherwise his increases rate of false positive findings.
- Contract should be injected slowly.
  - Only in certain instances should contrast injected quickly. (Advanced degenerative disk, with leak through endplate or annulus to get to 50 PSI)
- Higher injection speeds may cause rapid pressure elevations leading to increased pressure differences between nucleus and manometer and between the dynamic and static pressures.
- Typical opening pressures 5-25 PSI
  - Opening pressure >30 PSI = needle is within annulus.
- Disk slowly pressurized injecting 0.5 cc increments with syringe attached to pressure manometer.
- Slow injection speed more closely reflects real intradiscal pressures.
- **Endpoint**
  - Subjective pain = 6 last 10,
  - Intradiscal pressure >50 PSI above opening pressure with a grade 3 annular tear
  - 80-100 PSI a normal-appearing disk
  - Total of 3.5 cc contrast
Discography

- Is an extension of the clinical examination.
- Requires a careful clinical analysis and clinical correlation.
- The palpation finger...
Analgesic discography

- More attractive than provocation of discography.
- Personal experience...
- Derby et al compared 4 different techniques:
  - Local alone
  - Local with contrast
  - Local after provocation disco
  - Etc.
- Analgesic discography demonstrated high false-negative rate.
- Fears of provocation discography is high. Positive.

Intradiscal methylene blue


• Study showed a clinically meaningful pain reduction in 89% of IMBI-treated patients.

• The rationale for this treatment is that methylene blue (MB) is neurolytic.
  – Destroys terminal nerve endings.

• 2011 repeated in the Netherlands.
  – 40% responded to the treatment) in a well-selected group of patients suffering discogenic pain.

• 2015 repeated with RTC underway also in the Netherlands.
A case of discogenic pain

- 52 yo female Attorney at law with many years of chronic low back pain.
- Athletic, many sports and recreational activities.
- Extreme skiing
- Partial relief with facet blocks, - SI
- Significant temp relief with REIL
- DISCO...
  - Which disc...?

L5S1 functional autofusion
Positive L4-L5 disc
Case: “My sacroiliac will not hold”

- 49-year-old Caucasian male with history of pain over the right sacroiliac joint.
- Pain provocation maneuvers such as:
  - ASLR-SASLR
  - Ganslens
  - Yoemans
  - Lasslet
  - Shear
  - Etc.

Brown, Derby, Weins 1992
Pseudosacroiliac syndrome

- Mrs K. 40 yo with chronic SI pain:
  - OMT, DC, Rehab
  - Prolo
  - MMB
  - Facet injections
  - ESI
  - Acupuncture
  - ...

**DISC?**
- Which one?
Disk histology & biochemistry

- **Chondrocytes:**
  - Predominate
  - Tolerate an avascular environment
  - Synthesize matrix in which they are suspended then maintained and repair it.

- **Fibrocytes:**
  - Primarily in outer annulus

- **Collagen** confers tensile strength to disk.

- **Proteoglycan:**
  - Stabilized by Link Proteins (LP 1, LP 2, LP 3)
  - Versican, decorin, biglycan, fibromodulin, lumican

Degenerative Disc Disease

• Repeated biomechanical forces may lead to the loss of cohesion between bundles of the annulus, leading to fissuring.
• These fissures are repaired by ingrowth of granulation tissue which leads to vascularization, and that's initiating autoimmune phenomena.
• Fractures of the endplate leading to exposure of sequestered antigen and autoimmune phenomena.

Disc degeneration

- First matrix changes occur in the center nucleus:
  - fragmentation of proteoglycans
  - decreasing proteoglycan and water concentration
- Proteoglycan ends in the in plain regulate movement of solutes into and out of the disc. Roberts et al. 1996
- Removal of proteoglycan from the endplate accelerates the loss of proteoglycan from the nucleus.
- Atherosclerosis with the lumbar artery = decreased blood flow to endplate also contributes. Kauppila et al. 1997
Disc degeneration

- Type I collagen = annulus fibrosis
  - Small amount of II, III, V, VI, IX
- Type II collagen = inner annulus and nucleus pulposus.
  - Small amount of I, II, VI, XI
- Vertebral endplate:
  - Considered part of the disc
  - Composed of thin layer of cortical bone covered by hyaline cartilage
  - Produced by typical chondrocytes.
Increase in intradiscal pressure in IDD

• Pressure build up in the IDD: Onik explains that the intervertebral disc may be considered an osmotic system and because of the breakdown in macro molecules during the fourth and fifth decades of life, the number of particles in the intervertebral disc increases, which in turn causes a concomitant rise in the osmotic pressure secondary to an influx of fluid, which in turn increases the intradiscal pressure.

• In order to relieve the pressure, annular fissures developed that resultant osmotic annular tears and disc protrusions and pain.

Genetics and back pain

• Genetic variations in the structural components of the disc:
  – Collagen IX
  – Aggrecan


• Collagen IX
  – Gln326Trp Alpha 2 chain
  – Arg103Trp Alpha 3


• IL-1 polymorphisms, IL-6, TNF
Disc regenerative injection procedure...?

Pipedreams or reality in our Future?
Foundation of Regen Med

Prolotherapy

- Stem Cells
- Scaffolding
- Growth Factors
- Peptides
Stem cell vs engineering

• STEM CELLS
  – ADSC
  – Autologous BMAC MSC
  – Induced pluripotent SC
  – Endogenous progenitor cells
    • Notochord
  – NP cells
  – AF cells
  – NCP
  – Umbilical cord stem cells
  – Cultured and expanded
Autologous BMC IVD injection

- This study provides evidence of safety and feasibility in the non-surgical treatment of discogenic pain with autologous BMC, with durable pain relief (71% VAS reduction) and ODI Oswestry Disability Index improvements (>64%) through two years.

Hydrogels in IVD DDD

- Certain hydrogels have shown promising results, promoting both the viability of injected cells as well as their differentiation into NP-like cells.\(^1\)
- Transplantation of MSCs in a hydrogel carrier induced regenerative effects in degenerated IVDs.
- SDF-1 incorporation in proper delivery systems is able to promote cell recruitment to an injury site and increase the potential of tissue regeneration.\(^2\)
- Combined SDF-1 thermoreversible hyaluronan-poly(N-isopropylacrylamide) (HAP) hydrogel promote MSC migration to disc.

In search of scaffolding...

• A variety of biomaterials have been used for fabricating scaffolds in NP tissue engineering: 1-7
  – chitosan/hydroxybutyl
  – chitosan, alginate
  – collagen/atelocollagen
  – gelatin
  – Hyaluronic acid
  – calcium polyphosphate
  – poly-D-L-lactide (PDLA)
  – demineralized bone matrix (DBM)
  – small intestine submucosa (SIS)
  – carboxymethylcellulose
  – PGA–hyaluronan
References

Intradiscal PRP?

- 12 good articles 2004-2015 concerning the use of PRP in IDD
- 6 were “in vitro” studies
- 6 were “in vivo” studies
- INVITRO:
  - Studied effect on NP and AF cells and proliferation
  - The most frequently analyzed variables were: cell proliferation and extracellular matrix regeneration in terms of different proteoglycan (PG) levels/PG-mRNA expression.
- Only one paper analyzed PL regenerative potential.
- All the included studies underlined the positive histological results, demonstrating that PRP induces ECM regeneration and cell proliferation.
- Early IDD not late IDD with a dead disc and few cells.

How, what, when

• How does that alter the mechanical behavior of the disc?
• Which category and type of disc lesion is it best suited for?
• Our results have been inconsistent. We are not using intradiscal PRP at this time.
Stem cell & tissue engineering for DDD & discogenic pain

What is the evidence...
Our experience...
What we are doing...
Research activities...
THANK YOU

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