An Osteopathic Approach to Patients with Degenerative and Herniated Discs

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Abstract

This paper reviews the basic science of disc disease (DD), which suggests; osteopathic manipulative medicine addresses the causes and symptoms of DD, can reverse its pathologic cascade, prevent its side effects and future episodes. The paper will use the modern scientific understanding of disc disease to show the body is a self-regulating, self-healing unified whole, structure and function are interrelated at all levels, and intervertebral disc cells contain the biochemical and physiologic capacity for self-healing. This evidence suggests osteopathic intervention initiates and magnifies this healing process.

The paper will also discuss the authors' experience with a treatment protocol for DD, based on osteopathic principles.

Introduction

Disc degeneration and herniation are common causes of back pain.[1-4] Most current treatment for DD addresses its symptoms rather than causes.[2,4] Osteopathic physicians focus not only on disease, but also, its causation. Comprehensive treatment for patients with DD should decrease the pain and reverse the causative pathological cascade.[1,2] It should put the patient in a position to heal, normalize function, and prevent recurrences. We will show an osteopathic approach is well suited to treat patients with degenerative and/or herniated discs.

Summary of Evidence

Although surgery has been successful for the treatment of disc herniation, 15% of patients have recurrences.[5] A limitation of surgery is that it doesn't address the abnormal biomechanics contributing to or resulting from the herniated disc.[5] Following surgery, there is often ongoing pain, interfering with the quality of life.[5,6] Regenerative therapies have been studied but even if a herniated nucleus pulposus (NP) were regenerated, it would still be surrounded by an incompetent annulus fibrosis (AF).[2,4] AF repair has not yet been proven successful,[5-7] but a small recent ovine, in-vitro study shows some promise in AF repair.[8] The authors' OMM protocol decreases intradiscal pressure (IDP), corrects abnormal biomechanics, and may improve cellular function. It treats the somatic dysfunction (SD) to increase range of motion (ROM), circulation, and disc nutrition.[9] It addresses the lifestyle issues, which increase the IDP, neovascularization and symptomatology. Our treatment protocol uses modalities to decrease edema and local inflammatory mediators, and change disc diseases' postural and habitual biomechanical causes.

Motion is vital for intervertebral disc (IVD) nutrition.[10] However, too much motion, especially with cyclic compression[11] and/or flexion, prolonged static posture with too much or too little loading.[12,13]. Worst yet is hyper-flexion with rotation.[14,15] These motion patterns cause tissue injury (Figure1).[14] Isolated rotation within normal ROM doesn't seem to cause significant IVD damage.[11,14] Asymmetric loading, even with small loads, is more injurious than balanced loading with greater magnitude.[14,16]

There is a "sweet spot" where the vector and nature of disc loading are beneficial.[12,17] Varying from this causes damage to IVDs[12] due to mechanical stress, inflammation, cellular apoptosis,[4,11,13,14,17] and changes in genetic expression and fluid dynamics (nutrition).[2,12,18]

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Discs at some segments more frequently undergo degeneration than at other levels,[19] implying mechanical stress plays a role in (DD).[1,3,4,12] This is supported by the increase of (DD) adjacent to surgically fused segments[2] and to fused sacra.[3]

And so, SD may be part of the cause of the degenerative disc disease (DDD) and annular tear.[1,11,15,20] OMT addresses abnormal biomechanics (SD), which limits motion and causes asymmetric loading.

The fascia forms continuous chains from head to toe and from the mitochondria to the superficial fascia[21-23] (Figure 2). Intracellular elements of this fascial system are deformed[21-23] and cellular function is changed when external forces are applied to the body.

The body's trillions of cells communicate biomechanically, chemically, neurologically, and genetically to coordinate and unify the body. At the cellular level, these interactions cause changes in mechanotransduction and affect gene expression, ion channels, and the secondary messenger system (cAMP).[21,22,24] These changes in mechanotransduction affect the natural history of the cell,[21,22,24] determining its shape and function.[24,25]

As SD and abnormal posture change fascial tension, it likely affects all of these modes of cellular communication, worsening nutrition, cellular communication, and abnormal biomechanics, and beginning disc degeneration by profoundly changing cellular function.

It is difficult to predict the cellular effects a particular structural stress or its treatment would have. Nevertheless, it is reasonable to assume more normal vectors of fascial stress would result in more normal cellular function.

Normalizing posture is therefore important in the treatment of DD[12] and prevention of radiculopathy.[26] Normalizing posture improves disc nutrition and decreases AF strain. Slight decreases in lordosis decreases IDP,[27] improves postural and fascial stresses, and presumably normalizes mechanotransduction, cyclic AMP, and ion channel function. There is also a biomechanical effect on both ligamentum flavum (LF) and nucleus pulposus cytokine expression,[28] which should be improved by normalizing biomechanics. By normalizing posture, we decrease angiogenesis[29] and neurogenesis, major causes of disc pain. Decreasing lordosis is thus beneficial.[11,29-33]

Postural stress affects spinal mechanics segmentally and in full body patterns. A postural retraining exercise program can decrease this effect and help determine IVD health at the cellular level.[12] In the primary author's experience, the "sweet spot" of lumbar posture is that in which the spine functions symmetrically and in neutral mechanics.

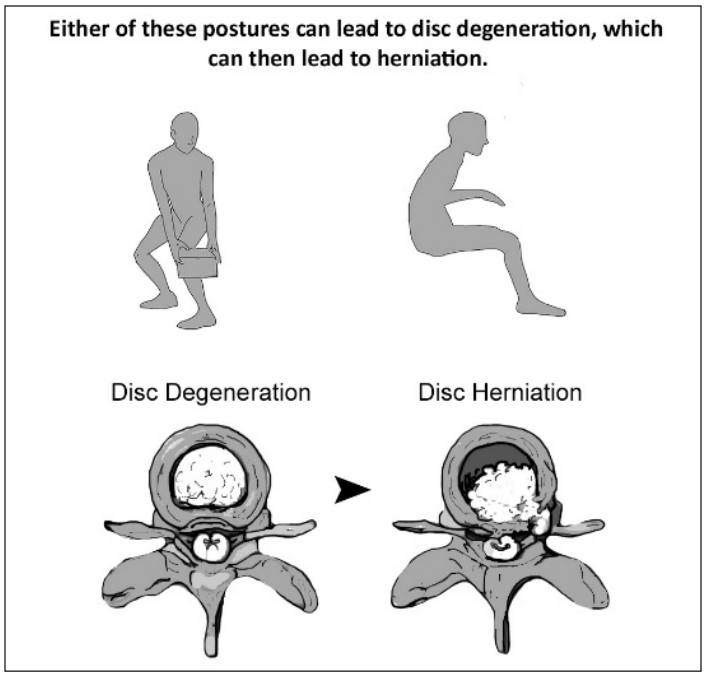

Figure 1. *Non optimum motion and postural patterns lead to disc disease. With permission from Rachel Kessler*

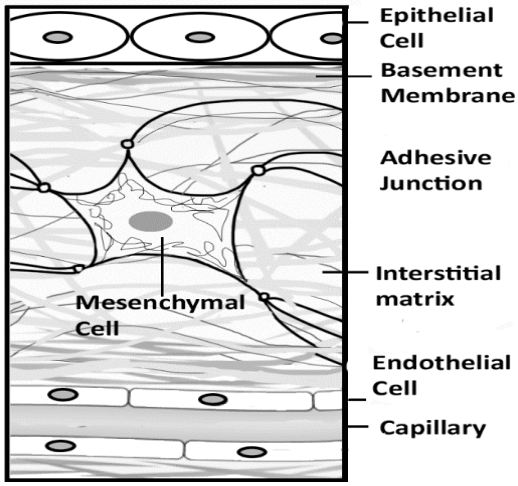

Figure 2. *Fascia continuity extends from the superficial fascia to the cell nucleus. With permission from Rachel Kessler*

Maintenance of neutral spinal posture lessens facet articular pressure,[27,30] improving facet syndrome's symptoms and lessening facet damage.[27,34,35] This decreases inflammatory compounds and growth factors, which are causes of facet and LF hypertrophy. Decreasing lordosis directly increases LF size; together, these symptomatically improve foraminal stenosis.[30,35,36]

Lumbar lordosis causes LF shortening and folding.[37] These further decrease the normal space in the spinal canal.[9,35,38] Decreasing lordosis straightens the LF, which reduces the mechanical impact on the

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spinal canal.[37] These factors decrease spinal stenosis symptomatology.[37]

The authors' clinical experience confirms many patients with spinal stenosis improve with the Dynamic Lumbar Stabilization Program (DLSP), which decreases lumbar lordosis. This improvement is more predictable if their symptoms improve when they change body mechanics (neutralize posture) during activities of daily living (ADL), i.e., bending over a shopping cart while walking. The combination of postural correction and application of OMT may address several of the underlying causes of disc herniation, stop or slow its progression,[3,39] and lessen several sequelae of the degenerative process.

Postural stress affects full body patterns of spinal mechanics. Within these patterns, segmental mechanics are changed. Improving posture decreases the negative effects of this stress, improving cellular level IVD health.[12] Chronic postural adaptations make the process of change challenging. Postural compression causes osteoblastic over osteoclastic predominance and instigates bony remodeling.[40] This makes correcting postural imbalance difficult, necessitating a process of re-education over time.[40]

The deep multifidus may be retrained with selective exercise, such as abdominal hollowing[41]-and deep breathing.[42] A retrained deep multifidus may allow spinal stabilization without limiting movement.[41]

The DLSP re-educates patients and helps them maintain neutral spinal mechanics during ADLs in patients with herniated lumbar.[31-33,43] Patients are taught to maintain a slightly decreased lumbar lordosis (neutral posture) by using abdominal hollowing and bracing, the same muscular efforts which retrain the deep multifidus. This yields excellent clinical results in patients with herniated discs.[31-33,43]

The (DLSP) trains patients to maintain neutral spinal mechanics. It starts with a pain relief phase using extension exercises.[31] Besides pain relief,[44] it helps the ultimate goal of maintaining a neutral lumbar sagittal curve during ADLs.

Neither flexion nor extension strength alone, but a balance between them determines the lumbar sagittal curve.[26] Extension increases pressure at the posterior aspect of the disc, which causes disc migration back within the AF.[45,46] After the initial phase, the DLSP focuses on movement in neutral posture. It returns 92% to 96% of patients with herniated discs, and 87% of patients with extruded discs (80% of those previously scheduled for surgery), to work without pain.[31-33] More recent studies show other types of stabilization exercises also have excellent results.[47]

Movement in neutral posture requires using muscles in proper sequence.[39] SD, besides limiting ROM, causes patterns of muscle imbalance, inhibiting normal movement,[15,48] as can degenerated and herniated discs.[48] For example, sacroiliac SD inhibits the gluteal muscles, causing both delayed gluteus maximus firing during leg extension, and gluteus medius and minimus during hip abduction, and abnormal firing patterns of trunk muscles.[48] These muscle imbalances lead to wider neuromusculoskeletal compensations and further injuries.[26] Layering on injury and adaptation in an ever expanding cycle ultimately causes whole body patterns of dysfunctional imbalances and posture.[39] This centralized sensory motor change can outlive the initial injury's healing.[48]

Abnormal muscular firing patterns cause chronic lumbar pain.[49] Treatment of these imbalances includes OMT for SD, a sensory motor retraining program, stretching hypertonic muscles, and retraining inhibited muscles.[39,50] This approach treats the dysfunctional pattern, pain caused by the SD and postural stress, and in the authors' experience, decreases pain associated with DD and its sequela. The author uses this approach prior to the DLSP discussed above. But, there are still other causes of pain in these cases.

Studies have shown that 70% of cases of herniated discs do not cause pain, despite applying pressure to nerve roots.[51,52] Clearly, pressure is not the major cause of pain. Inflammatory compounds released by the herniated disc[4,7,53-56] act synergistically with nerve compression[19] to cause pain. Epidural steroids for the pain of herniated discs with radiculopathy is a common treatment. Systemic reviews show these help in the short term (less than 2 weeks), but the difference between steroid and placebo is small[57,58] and do not change the rate of surgical intervention.[57] Any long-term changes may be due to DD's natural history.[57] The authors only refer patients for epidural steroids if short-term pain relief is needed to allow patients to start a rehabilitative exercise program.

Degenerated discs release inflammatory compounds, causing migration of new blood vessels and nociceptive nerve endings into the disc, increasing sensitivity to painful stimulation.[4,24] Neovascularization occurs at symptomatic degenerated discs and not surrounding segments.[4] Neovascularization also occurs in herniated discs. Neovascularization predicts post-operative pain.[24] In one study, 100% of those patients without angiogenesis at the extruded disc improved function after surgery, while 16% of those with angiogenesis did not.[24]

Proteins and inflammatory mediators in the extravascular tissues must be returned to the central circulatory system via the lymphatic circulation.[57,59] In order to form and move lymph through the lym-

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phatic vessels, a gravitational pressure gradient must be overcome. Inherent tissue motion and the fluctuation of extracellular fluid begin this process.[59] The lymphatic pumps include inherent peristalsis of the lymph vessels (regulated by sympathetic nervous input), and respiratory movements, among others.[59] A lymphatics approach utilizes many manipulative techniques, which improve lymphatic flow.[59] This increases the return of lymphatic fluid to the venous system three fold.[59] A lymphatics OMM approach to decrease disc edema, perineural inflammatory mediators, and angiogenesis has not been studied. However, the known physiology in other areas suggests it would be effective.[59] It should decrease the pain of a herniated disc and hasten healing. Reabsorption of herniated NP material is caused by an immune response,[53,55,60] which is increased when the NP material is exposed to the epidural space.[24,55] Because a lymphatic approach increases immune responses[59] it should speed disc reabsorption. This is consistent with the authors' clinical experience.

Lifestyle changes are important in a holistic approach to DD. Smoking decreases oxygenation,[61] reduces nutrition and disc healing.[61,62] It disrupts cellular metabolism[24,62] and thus, decreases the rate of proteoglycan production.[4,15,62,63]. Smoking increases angiogenesis and migration of nociceptive fibers,[4,24] worsening disc pain and disability.[24] Smoking is a risk factor for non-healing after disk surgery and predicts postoperative pain.[24] Smokers with DD should be helped with cessation programs. Smoking cessation reduces angiogenesis by 32%, decreasing disc pain and improving surgical results.[24] Several other modifiable lifestyle issues are additional risk factors for angiogenesis, including obesity, heavy lifting, and sedentary lifestyle. A holistic approach should help patients change these lifestyle factors.

SD limits motion and limiting motion decreases disc nutrition. SD causes muscle imbalance and postural changes. It increases fascial stress and so may cause changes in genetic expression and cellular biochemical activity. Together these effects may both begin and magnify the processes of DD and herniation. It seems likely that OMT with muscle balance and postural training exercise, lessens future problems to the disc in question and surrounding discs. In the authors' experience, it decreases pain from the degenerative and/or herniated disc, the facet, and the SD, and improves pain from foraminal and spinal stenosis in many patients. Our current understanding of the changes in disc physiology with treatment confirms the body is self-regulating and self-healing.

Motion increases disc diffusion and mobility,[64] which are factors that are beneficial to patients with DD. Despite its benefits, disc herniation has been called a relative contraindication to manipulation.[65-67] Osteopathic manipulation has few side effects in the general population[56] or in patients with herniated discs.[68-70] Current guidelines suggest manipulation is only contraindicated if there are progressive neurologic signs.[70]

Manipulation at the segment of the herniation can be performed with greater safety using techniques that decrease IDP during the procedure. The Cox Technique uses manual traction with articulatory low velocity mobilization.[71] It lowers IDP while being performed and improves symptoms.[71]

A modification of the classic facilitated positional release technique for discogenic pain has been used successfully in patients with herniated lumbar discs. It uses manual traction with both longitudinal and lateral traction vectors localized to the herniated disc. It induces these forces with caudal traction of the sacrum and by using the patient's legs as levers.[72]

Traction has long been used for herniated discs.[73-75] It lowers IDP, with the greatest decline when applied with flexion.[76] Traction decreases lumbar disc herniation size.[74] While we can't claim that other OMT techniques would induce the same changes in IDP, manipulation should be safer and have increased efficacy when performed with traction across the herniated segment. By using traction, these measures should be able to lower IDP while performing OMT.

It is possible to use many osteopathic techniques with a traction vector across the dysfunctional segment. In the author's experience, the Still technique is effective and can use a traction vector as an activating force. In patients with herniated discs, the authors perform these techniques with a pillow under the prone patient's abdomen to neutralize lumbar position.

In most patients with DDD, OMT with a muscle balance exercise program, followed by a DLSP takes 4-8 weeks. It is noninvasive, inexpensive, and generally without side effects. The indication for surgery for a herniated disc is a progressive neurologic deficit, cauda equina syndrome or intractable pain unresponsive to conservative care.[77] While surgical approaches give excellent results, at moderate and long-term follow-up, conservative care is equally good.[36,43,78] Surgical results are improved with better patient selection. Magnetic resonance spectroscopy has improved surgical outcomes when used to select patients and segments for surgery.[79]

Even patients with very large disc herniation do well with conservative care.[60] In fact, disc herniation protruding past the posterior longitudinal ligament shows more reabsorption than bulging discs.[53,60] This reabsorption is due to an immune response secondary to the "liberated" IVD nuclear material and neovascularization.[53,55,60] The AF and posterior longitudinal ligament seem to protect the nuclear

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material from this immune response until it has herniated past these,[55,60] exposing it to the epidural space.[29] In one seven-year follow-up study, there was a long-lasting regression rate averaging over 70%,[53] occurring largely in the first six weeks.[53] And yet, lumbar disc surgery is the second most common surgical procedure.[80]

Conclusion

SD may start a pathologic cascade of DDD, annular tear, disc herniation, spinal and foraminal stenosis. When one considers the basic science of somatic dysfunction and DD it seems likely that early intervention in this process should be able to alter its progression.

Absent an indication for immediate surgery, the treatment approach should be to address the pathology's symptoms and root causes with conservative, holistic care. OMT, decreases rotational stress on the disc and increases movement across it. As OMT improves ROM it improves IVD nutrition.[9] In the authors' experience, it lessens the pain of a herniated disc and its causative factors. Retraining muscle imbalance normalizes biomechanics and makes postural re-education possible. Movement in neutral posture likely provides the balanced duration, direction and magnitude of compression needed to maintain disc health, improves disc degeneration and herniation's fellow sequela of SD; spinal stenosis, foraminal stenosis and facet syndrome. Smoking cessation and dietary improvement increase disc nutrition and oxygenation and prevents neovascularization. Decreasing obesity, controlling activity, and other lifestyle modifications prevent disc damage and should be part of the treatment of these patients.

A herniated IVP may be reabsorbed without treatment, or the patient may ultimately require surgery. But, even if these things had occurred, with the treatment program outlined above there should be improved surgical outcomes. The causative abnormal biomechanics of the original problem will have been normalized and there should be less risk for future DD.

The approach discussed in this paper has been successful in the primary authors' clinical practice. While this is only testimonial evidence, as a demonstration of this I would tell a story. In 2004, a 66-year-old man presented to the primary author's office with lumbar spinal stenosis for a second opinion prior to a scheduled surgery. After receiving treatment with the approach outlined in this paper he was pain free and able to play golf nearly daily since. From that time, he has referred 2-3 patients a year from his golf club, with differing combinations of DDs and is sequela, all of whom had been scheduled for back surgery. Two have required surgery after this type of treatment.

Even though surgeries have been increasingly successful, and there have been advances in NP regenerative cellular interventions,[2,4,52,81] the authors are reminded of the wisdom of A.T. Still when he said "God had certainly placed the remedy within the material house in which the spirit of life dwells,"[82] and "we can access these remedies by adjusting the body in such a manner that the remedies may naturally associate themselves together, hear the cries, and relieve the afflicted."[82]

This paper is based on the physiology of the IVD and pathophysiology of DD as well as treatment physiology. We make assumptions from these to predict what outcomes should be. Anecdotal evidence from our experience treating these patients is also cited. There is, however a lack of objective outcomes data available, especially for the cellular level effects and clinical outcomes of the treatment protocol. Magnetic resonance spectroscopy (MRS) has been used to examine intracellular biochemistry.[79] Intracellular lactate and proteoglycan can be measured and used as biomarkers for anaerobic metabolism and discogenic pain.[79] Further studies should be performed using MRS pre-and post OMT. These would provide objective evidence of the effects of OMT and OMM.

References

1. Choi Y-S. Pathophysiology of Degenerative Disc Disease. *Asian Spine J*. 2009;3(1):39. doi:10.4184/asj.2009.3.1.39
2. Smith LJ, Nerurkar NL, Choi KS, Harfe BD, Elliott DM. Degeneration and regeneration of the intervertebral disc: lessons from development. *Dis Model Mech*. Jan 2011;4(1):31-41. doi:10.1242/dmm.006403
3. Saleem S, Aslam HM, Rehmani MA, Raees A, Alvi AA, Ashraf J. Lumbar disc degenerative disease: disc degeneration symptoms and magnetic resonance image findings. *Asian Spine J*. 2013;7(4):322-334. doi:10.4184/asj.2013.7.4.322
4. Freemont AJ. The cellular pathobiology of the degenerate intervertebral disc and discogenic back pain. *Rheumatology (Oxford)*. 2009;48(1):5-10. doi:10.1093/rheumatology/ken396
5. Guterl CC, See EY, Blanquer SB, et al. Challenges and strategies in the repair of ruptured annulus fibrosus. *Eur Cell Mater*. 2013;25:1-21. doi:10.22203/ecm.v025a01
6. Bron JL, Helder MN, Meisel HJ, Van Royen BJ, Smit TH. Repair, regenerative and supportive therapies of the annulus fibrosus: achievements and challenges. *Eur Spine J*. 2009;18(3):301-313. doi:10.1007/s00586-008-0856-x
7. Adams MA, Stefanakis M, Dolan P. Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: implications for physical therapies for discogenic back pain. *Clin Biomech (Bristol, Avon)*. 2010;25(10):961-971. doi:10.1016/j.clinbiomech.2010.07.016
8. Pennicooke B, Hussain I, Berlin C, et al. Annulus Fibrosus Repair Using High-Density Collagen Gel: An In Vivo Ovine Model. *Spine*. 2018;43(4):E208-E215. doi:10.1097/BRS.0000000000002334
9. Lueck M. Lumbar spine manipulation and water diffusion in the intervertebral disk (pt. 2). *Dynamic Chiropractic*. 2016;34(1)

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(continued from page 39)

10. Gautam D. Herniated Nucleus Pulposus. Background, Anatomy, Pathophysiology. Medscape. Updated May 20, 2019. https://emedicine.medscape.com/article/1263961-overview
11. Barbir A, Godburn KE, Michalek AJ, Lai A, Monsey RD, Iatridis JC. Effects of torsion on intervertebral disc gene expression and biomechanics, using a rat tail model. *Spine*. 2011;36(8):607-614. doi:10.1097/BRS.0b013e3181d9b58b
12. Chan SC, Ferguson SJ, Gantenbein-Ritter B. The effects of dynamic loading on the intervertebral disc. *Eur Spine J*. 2011;20(11):1796-1812. doi:10.1007/s00586-011-1827-1
13. Maclean JJ, Lee CR, Alini M, Iatridis JC. Anabolic and catabolic mRNA levels of the intervertebral disc vary with the magnitude and frequency of in vivo dynamic compression. *J Orthop Res.* 2004;22(6):1193-1200. doi:10.1016/j.orthres.2004.04.004
14. Walter BA, Korecki CL, Purmessur D, Roughley PJ, Michalek AJ, Iatridis JC. Complex loading affects intervertebral disc mechanics and biology. *Osteoarthritis Cartilage*. 2011;19(8):1011-1018. doi:10.1016/j.joca.2011.04.005
15. Snider KT, Johnson JC, Snider EJ, Degenhardt BF. Increased incidence and severity of somatic dysfunction in subjects with chronic low back pain. *J Am Osteopath Assoc*. 2008;108(8):372-378.
16. McGill SM, Marshall L, Andersen J. Low back loads while walking and carrying: comparing the load carried in one hand or in both hands. *Ergonomics*. 2013;56(2):293-302. doi:10.1080/00140139.2012.752528
17. Stokes IA, Iatridis JC. Mechanical conditions that accelerate intervertebral disc degeneration: overload versus immobilization. *Spine*. 2004;29(23):2724-2732. doi:10.1097/01.brs.0000146049.52152.da
18. Urban JP, Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther*. 2003;5(3):120-130. doi:10.1186/ar629
19. Blankenbaker DG, Haughton VM, Rogers BP, Meyerand ME, Fine JP. Axial rotation of the lumbar spinal motion segments correlated with concordant pain on discography: a preliminary study. *AJR Am J Roentgenol.* 2006;186(3):795-799. doi:10.2214/AJR.04.1629
20. DiGiovanna EL, Schiowitz S, Dowling DJ, eds. *An Osteopathic Approach to Diagnosis and Treatment.* 3rd ed. Lippincott Williams & Wilkins; 2004.
21. Swanson RL, 2nd. Biotensegrity: a unifying theory of biological architecture with applications to osteopathic practice, education, and research--a review and analysis. *J Am Osteopath Assoc.* 2013;113(1):34-52. doi:10.7556/jaoa.2013.113.1.34
22. Ingber DE. Tensegrity-based mechanosensing from macro to micro. *Prog Biophys Mol Biol*. 2008;97(2-3):163-179. doi:10.1016/j.pbiomolbio.2008.02.005
23. Stiles E. Science and Osteopathic Research. Presented at: American Academy of Osteopathy Convocation; 2005; Louisville KY.
24. Huang S, Ingber DE. A non-genetic basis for cancer progression and metastasis: self-organizing attractors in cell regulatory networks. *Breast Dis*. 2006;26:27-54. doi:10.3233/bd-2007-26104
25. McBeath R, Pirone DM, Nelson CM, Bhadriraju K, Chen CS. Cell shape, cytoskeletal tension, and RhoA regulate stem cell lineage commitment. *Dev Cell*. 2004;6(4):483-495. doi:10.1016/s1534-5807(04)00075-9
26. Kim HJ, Chung S, Kim S, et al. Influences of trunk muscles on lumbar lordosis and sacral angle. *Eur Spine J*. 2006;15(4):409-414. doi:10.1007/s00586-005-0976-5
27. Shirazi-Adl A, Parnianpour M. Effect of changes in lordosis on mechanics of the lumbar spine-lumbar curvature in lifting. *J Spinal Disord*. 1999;12(5):436-447.
28. Indahl A, Kaigle AM, Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine*. 1997;22(24):2834-2840. doi:10.1097/00007632-199712150-00006
29. David G, Ciurea AV, Iencean SM, Mohan A. Angiogenesis in the degeneration of the lumbar intervertebral disc. *J Med Life*. 2010;3(2):154-161.
30. Wu L, Cruz R. Lumbar spinal stenosis. StatPearls. Updated December 20, 2020. https://www.ncbi.nlm.nih.gov/books/NBK531493
31. Saal JA, Saal JS. Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy. An outcome study. *Spine*. 1989;14(4):431-437. doi:10.1097/00007632-198904000-00018
32. Saal JA. Dynamic muscular stabilization in the nonoperative treatment of lumbar pain syndromes. *Orthop Rev*. 1990;19(8):691-700.
33. Saal JS, Saal JA, Yurth EF. Nonoperative management of herniated cervical intervertebral disc with radiculopathy. *Spine*. 1996;21(16):1877-1883. doi:10.1097/00007632-199608150-00008
34. Malanga G. Lumbosacral facet syndrome treatment & management. Medscape. Updated November 19, 2018. https://emedicine.medscape.com/article/94871-treatment
35. Kuchera ML. Postural considerations in osteopathic diagnosis and treatment. In: Seffinger MA, eds. *Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications, and Research*. 4th ed. Wolters Kluwer; 2018:706.
36. Vleeming A, Mooney V, Stoeckart R, eds. *Movement, Stability & Lumbopelvic Pain: Integration of Research and Therapy*. 2nd ed. Churchill Livingstone Elsevier; 2007.
37. Chung SS, Lee CS, Kim SH, Chung MW, Ahn JM. Effect of low back posture on the morphology of the spinal canal. *Skeletal Radiol*. 2000;29(4):217-223. doi:10.1007/s002560050596
38. Chokshi FH, Quencer RM, Smoker WR. The "thickened" ligamentum flavum: is it buckling or enlargement? *AJNR Am J Neuroradiol.* 2010;31(10):1813-1816. doi:10.3174/ajnr.A2241
39. Page P, Frank C, Lardner R. *Assessment and Treatment of Muscle Imbalance: The Janda Approach.* Human Kinetics; 2010.
40. O'Connell J. *Bioelectric Fascial Activation and Release: The Physician's Guide to Hunting with Dr. Still*. American Academy of Osteopathy; 2000.
41. MacDonald DA, Moseley GL, Hodges PW. The lumbar multifidus: does the evidence support clinical beliefs? *Man Ther*. 2006;11(4):254-263. doi:10.1016/j.math.2006.02.004
42. Ko MJ, Jung EJ, Kim MH, Oh JS. Effects of deep breathing on internal oblique and multifidus muscle activity in three sitting postures. *J Phys Ther Sci*. 2018;30(4):504-506. doi:10.1589/jpts.30.504
43. Saal JA, Saal JS, Herzog RJ. The natural history of lumbar intervertebral disc extrusions treated nonoperatively. *Spine*. 1990;15(7):683-686. doi:10.1097/00007632-199007000-00013
44. Broetz D, Hahn U, Maschke E, Wick W, Kueker W, Weller M. Lumbar disk prolapse: response to mechanical physiotherapy in the absence of changes in magnetic resonance imaging. Report of 11 cases. *NeuroRehabilitation*. 2008;23(3):289-294.
45. Takasaki H, May S, Fazey PJ, Hall T. Nucleus pulposus deformation following application of mechanical diagnosis and therapy: a single case report with magnetic resonance imaging. *J Man Manip Ther.* 2010;18(3):153-158. doi:10.1179/106698110X12640740712455
46. Balkovec C, McGill S. Extent of nucleus pulposus migration in the annulus of porcine intervertebral discs exposed to cyclic flexion only versus cyclic flexion and extension. *Clin Biomech (Bristol, Avon)*. 2012;27(8):766-770. doi:10.1016/j.clinbiomech.2012.05.006

(continued on page 41)

(continued from page 40)

47. Jeong DK, Choi HH, Kang JI, Choi H. Effect of lumbar stabilization exercise on disc herniation index, sacral angle, and functional improvement in patients with lumbar disc herniation. *J Phys Ther Sci*. 2017;29(12):2121-2125. doi:10.1589/jpts.29.2121
48. DeStefano LA. *Greenman's Principles of Manual Medicine*. 5th ed. Wolters Kluwer; 2017.
49. Suehiro T, Mizutani M, Ishida H, Kobara K, Osaka H, Watanabe S. Individuals with chronic low back pain demonstrate delayed onset of the back muscle activity during prone hip extension. *J Electromyogr Kinesiol*. 2015;25(4):675-680. doi:10.1016/j.jelekin.2015.04.013
50. Janda V. Treatment of chronic back pain. *J Man Med*. 1992;6(5):166-168.
51. Ozer AF, Oktenoglu T, Sasani M, Kaner T, Ercelen O, Canbulat N. Unusual cause of acute low-back pain: sudden annulus fibrosus rupture. *Orthop Rev*. 2012;4(2):e22. doi:10.4081/or.2012.e22
52. Tavakoli J, Diwan AD, Tipper JL. Advanced strategies for the regeneration of lumbar disc annulus fibrosus. *Int J Mol Sci*. 2020;21(14):4889. doi:10.3390/ijms21144889
53. Benson RT, Tavares SP, Robertson SC, Sharp R, Marshall RW. Conservatively treated massive prolapsed discs: a 7-year follow-up. *Ann R Coll Surg Eng*. 2010;92(2):147-53. doi:10.1308/003588410X12518836438840
54. Saal JS. The role of inflammation in lumbar pain. *Spine*. 1995;20(16):1821-1827. doi:10.1097/00007632-199508150-00013
55. Shamji MF, Setton LA, Jarvis W, et al. Proinflammatory cytokine expression profile in degenerated and herniated human intervertebral disc tissues. *Arthritis Rheumatol*. 2010;62(7):1974-82. doi:10.1002/art.27444
56. Seffinger MA. The safety of osteopathic manipulative treatment (OMT). *J Am Osteopath Assoc*. 2018;118(3):137-138. doi:10.7556/jaoa.2018.031
57. Chou R, Hashimoto R, Friedly J, et al. Epidural corticosteroid injections for radiculopathy and spinal stenosis: a systematic review and meta-analysis. *Ann Intern Med*. 2015;163(5):373-381. doi:10.7326/M15-0934
58. Chou R, Hashimoto R, Friedly J, et al. AHRQ Technology Assessments. *Pain Management Injection Therapies for Low Back Pain*. Agency for Healthcare Research and Quality (US); 2015.
59. Seffinger MA. *Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications, and Research*. 4th ed. Wolters Kluwer; 2018:175-178,906-910.
60. Cribb GL, Jaffray DC, Cassar-Pullicino VN. Observations on the natural history of massive lumbar disc herniation. *Journal Bone Joint Surg Br*. 2007;89(6):782-784. doi:10.1302/0301-620X.89B6.18712
61. Huang W, Qian Y, Zheng K, Yu L, Yu X. Is smoking a risk factor for lumbar disc herniation? *Eur Spine J*. 2016;25(1):168-176. doi:10.1007/s00586-015-4103-y
62. Harrer SW, Carlson WO. Spinal health and smoking. *S D Med*. 2009;62(8):309,311-313.
63. Gore DR, Carrera GF, Glaeser ST. Smoking and degenerative changes of the cervical spine: a roentgenographic study. *Spine J*. 2006;6(5):557-560. doi:10.1016/j.spinee.2005.12.003
64. Thiry P, Reumont F, Brismee JM, Dierick F. Short-term increase in discs' apparent diffusion is associated with pain and mobility improvements after spinal mobilization for low back pain. *Sci Rep*. 2018;8(1):8281. doi:10.1038/s41598-018-26697-7
65. M. WJ. Massage, traction, and manipulation. Medscape. Updated April 1, 2020. https://emedicine.medscape.com/article/324694-overview
66. Malone DG, Baldwin NG, Tomecek FJ, et al. Complications of cervical spine manipulation therapy: 5-year retrospective study in a single-group practice. *Neurosurg Focus*. 2002;13(6):ecp1. doi:10.3171/foc.2002.13.6.8
67. Wainapel SF, Fast A. *Alternative Medicine and Rehabilitation: A Guide for Practitioners*. Demos Medical Publishing; 2003.
68. Peterson CK, Schmid C, Leemann S, Anklin B, Humphreys BK. Outcomes from magnetic resonance imaging-confirmed symptomatic cervical disk herniation patients treated with high-velocity, low-amplitude spinal manipulative therapy: a prospective cohort study with 3-month follow-up. *J Manip Physiol Ther*. 2013;36(8):461-467. doi:10.1016/j.jmpt.2013.07.002
69. Shokri E, Kamali F, Sinaei E, Ghafarinejad F. Spinal manipulation in the treatment of patients with MRI-confirmed lumbar disc herniation and sacroiliac joint hypomobility: a quasi-experimental study. *Chiropr Man Therap*. 2018;26:16. doi:10.1186/s12998-018-0185-z
70. Canadian Chiropractic Association (CCA); Canadian Federation of Chiropractic Regulatory and Educational Accrediting Boards (CFCREAB); Clinical Practice Guidelines Development Initiative (CCA. CFCREAB-CPG); A clinical practice guideline update from the CCA. CFCREAB-CPG. *J Can Chiropr Assoc*. 2008;52(1):7-8.
71. Gudavalli MR, Cambron JA, McGregor M, et al. A randomized clinical trial and subgroup analysis to compare flexion-distraction with active exercise for chronic low back pain. *Eur Spine J*. 2006;15(7):1070-1082. doi:10.1007/s00586-005-0021-8
72. Dowling D. A modification of the classic facilitated positional release technique for discogenic pain. Presented at: American Academy of Osteopathy Convocation; March 11-15, 2020; Colorado Springs, CO.
73. Furman M. Cervical Disc Disease Treatment & Management. Medscape. Updated April 16, 2020. https://emedicine.medscape.com/article/305720-treatment
74. Ozturk B, Gunduz OH, Ozoran K, Bostanoglu S. Effect of continuous lumbar traction on the size of herniated disc material in lumbar disc herniation. *Rheumatol Int*. 2006;26(7):622-626. doi:10.1007/s00296-005-0035-x
75. Yang HS, Yoo WG. The effects of stretching with lumbar traction on vas and oswestry scales of patients with lumbar 4-5 herniated intervertebral disc. *J Phys Ther Sci*. 2014;26(7):1049-1050. doi:10.1589/jpts.26.1049
76. Gudavalli MR, Potluri T, Carandang G, et al. Intradiscal pressure changes during manual cervical distraction: a cadaveric study. *Evid Based Complement Alternat Med*. 2013. doi:10.1155/2013/954134
77. Sahrakar K. Lumbar disc disease. Medscape. Updated September 25, 2018. https://emedicine.medscape.com/article/249113-overview
78. Gugliotta M, da Costa BR, Dabis E, et al. Surgical versus conservative treatment for lumbar disc herniation: a prospective cohort study. *BMJ Open*. 2016;6(12):e012938. doi:10.1136/bmjopen-2016-012938
79. Gornet MG, Peacock J, Claude J, et al. Magnetic resonance spectroscopy (MRS) can identify painful lumbar discs and may facilitate improved clinical outcomes of lumbar surgeries for discogenic pain. *Eur Spine J*. 2019;28(4):674-687. doi:10.1007/s00586-018-05873-3
80. Sipko T, Chantsoulis M, Kuczynski M. Postural control in patients with lumbar disc herniation in the early postoperative period. *Eur Spine J*. 2010;19(3):409-414. doi:10.1007/s00586-009-1082-x
81. Sloan SR, Jr., Wipplinger C, Kirnaz S, et al. Combined nucleus pulposus augmentation and annulus fibrosus repair prevents acute intervertebral disc degeneration after discectomy. *Sci Transl Med*. 2020;12(534) doi:10.1126/scitranslmed.aay2380
82. Still AT. *Autobiography of Andrew Taylor Still*. Jolandos EK. Paehl; 2006 reprint from 1908 ed.■