Pain Generators of the Lumbar Spine

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Abstract Different anatomical structures and pathophysiological functions can be responsible for lumbar pain, each producing a distinctive clinical profile. Pain can arise from the intervertebral disc, either acutely as a primary disc related disorder, or as result of the degradation associated with chronic internal disc disruption. In either case, greatest pain provocation will be associated with movements and functions in the sagittal plane. Lumbar pain can also arise from afflictions within the zygapophyseal joint mechanism, as result of synovitis or chondropathy. Either of these conditions will produce the greatest pain provocation during three-dimensional movements, due to maximal stress to either the synovium or joint cartilage. Finally, patients can experience different symptoms associated with irritation to the dural sleeve, dorsal root ganglion, or chemically irritated lumbar nerve root. Differential diagnosis of these conditions requires a thorough examination and provides information that can assist the clinician in selecting appropriate management strategies.

Key Words: Lumbar, Disc, Zygapophyseal, Root

INTRODUCTION

The majority of all adults will complain of low back pain (LBP) at a given time in life with little or misleading evidence of pathology from imaging studies. Holm suggested that mechanical, genetic, chemical, social, nutritional, and psychological factors can influence the onset of LBP. Investigators have deliberated over which structure or process is responsible for the low back symptoms and one can conclude that LBP can arise from a variety of different anatomical sites in concert with complex mechanical and neurophysiological processes that arise in response to trauma and disease. Consequently, a patient’s clinical presentation may initially be misleading to the diagnostician due to apparently similar pain patterns that are produced by different afflictions. The observed similarities in different patients who present with complaints of LBP may be related to the intricacy of the sensory system that innervates the structures in the lumbar region. To insure effective diagnosis and focused management, the practitioner can implement a clinical examination that attempts to reveal the structure or mechanism that is responsible for the patient’s complaints.

Lumbar symptoms can be produced by pathological alterations in the intervertebral disc and zygapophyseal joints. Additionally, afflictions at each of these sites appear to be inter-related. Younger individuals with LBP are commonly afflicted with a primary disc-related disorder where the “soul” of their symptoms resides in acute mechanical and chemical changes in the disc. However, the intervertebral disc demonstrates a symbiotic relationship with the zygapophyseal joints (ZAJ) as these joints form a closed kinematic chain with the disc. This relationship between disc and joints promotes segmental stability, and time-related disc pathology can produce degeneration and subsequent pain within the ZAJ. This chain reaction suggests that sec-
Secondary disc pathology, including ZAJ synovitis and chondropathy, arises from previous episodes of primary disc-related disorders.

Age appears to have a direct influence on the source of a patient’s LBP. The intervertebral disc begins to change at approximately 20 years of age. Thus, it is more common for primary disc-related disorders to afflict younger individuals. For example, lower lumbar primary posterior lateral protrusions are most commonly seen between the ages of 18 and 35 years, where other lower lumbar primary disc disorders, such as protrusions, prolapses, and extrusions, are most frequently seen in patients between 35 and 55 years of age. Additionally, secondary disc-related disorders more frequently emerge clinically in patients over 45-years-old, after a history of primary disc-related disorders. For example, patients who present with lumbar zygapophyseal chondropathy frequently report a recurrent history of symptoms consistent with disc protrusion. In order to treat the progressive pathology of LBP, one must consider specific segmental changes from a physiological and mechanical perspective.

The Lumbar Intervertebral Disc

As previously mentioned, individuals can develop LBP as a result of a compromise to different structures and functions within the lumbar region. A clinician’s clear understanding of the pathoanatomy, physiology, and mechanics of the lumbar spine can serve as foundation for clarity in differential diagnosis. The intervertebral disc appears to be the source of many patients’ symptoms, because individuals can suffer from afflictions of the cartilaginous endplate (CEP), anulus fibrosis, and nucleus pulposis. These afflictions can contribute to the onset and persistence of LBP through pathophysiological and pathomechanical processes within the disc environment.

The CEP is comprised of thin layers of hyaline cartilage that demonstrate parallel, horizontal collagen fibers (see Figure 1a). This thin structure (approximately 1 mm thick) can fracture during acute traumatic loading or demonstrate gradual flattening in response to disc degeneration and accompanying intradiscal water loss. In either case, a patient under 50 years of age may eventually develop radiologically appreciable Schmorl’s nodes due to herniation of nuclear material into the vertebral body (see Figure 1b). This change is predictive of LBP and represents a breech in the integrity of the CEP. In addition to the escape of nuclear material into the vertebral body, the node indicates communication of blood into the nucleus and a subsequent initiation of a clinically-relevant chemical cascade. Whereas the disc’s normal nutritive response is dependent on diffusion of nutrients across the CEP, nuclear envelope disruption and eventual calcification can lead to compromised diffusion and exchange. This deprivation surrenders the disc to
further breakdown and an altered healing potential. Additionally, smoking, vibration, or aphysiological motion in the disc (resulting in a compromised circulatory response) can alter pH and propagate accelerated degeneration. Goldberg et al reviewed 38 epidemiological studies evaluating the relationship between smoking and prevalence of nonspecific low back pain (NSLBP). These investigators found a positive association between current smoking and NSLBP for men in 18 out of 26 studies and for women in 18 out of 20 studies. Additionally, 5 studies illustrated a relationship between past smoking and NSLBP.

The symptoms associated with injury to the CEP are commonly witnessed in a younger population and are propagated through a macrotraumatic event. The CEP can be a source of LBP, but the pain associated with CEP injury is short in duration and limited to the central low back region. Although symptoms resolve spontaneously in 4 to 6 days, the more significant consequence of this breach emerges several years later. The proteoglycan and collagen matrix of the CEP serves as a barrier to all but small, globular molecules, such as glucose. When the endplate is disrupted the disc is invaded by chemical factors, permanently inhibiting matrix synthesis within its inner sanctum (see Figure 1b). This exchange leads to a production of metallo-matrix protease inhibitors (MMPI’s) that are responsible for gradual radial anular fissuring and cell-mediated disc breakdown. Other autoimmune activities accompany this process including the release of bradykinin, serotonin, and collagenase; the aggregation of T-lymphocytes; and the production of phospholipase A2 (PLA2). Not only do these agents heighten the activity of silent nociceptors found within the walls of the anulus fibrosis, but they can also serve as a trigger for increased MMPI concentrations, which can escalate further collagen degradation within the disc. These events can activate a peripheral “sensitization,” where even normal mechanical activity produces a painful response.

This chemical cascade produces clinical manifestations when the nuclear fluid matrix, along with inflammatory agents, reach the outer sanctum of the anulus. These chemical agents aggressively irritate nerve roots and sensitize previously silent nociceptors. This chemical cascade lowers the threshold on nociceptor populations and gradually results in a band-form distribution of LBP as well as nonradicular lower extremity pain. This gradual onset of lower extremity pain is more commonly observed with individuals over the age of 45 as compared to the immediate lower extremity pain observed in younger individuals who are suffering from the mechanical irritation of a dorsal root ganglion associated with an acute primary disc disorder.

One can observe 3 layers within the disc anulus (see Figure 1). The outer third of the anulus attaches to the outer aspect of the V-body approximately 3 mm in distance from the contiguous level of the CEP, while the middle third attaches to the vertebral cortex at the disc space. The connection between the outer ligamentous anular fibers and bony cortex creates a strong, continuous structure that possesses all features of a ligament. Consequently, this structural arrangement provides support for the motion segment with movements in the sagittal and transverse planes.

The steep fibers of the inner anulus are connected to the vertebral CEP without any bony connections at an angle of 37° from the endplate (versus 24° inclination observed in the outer anulus). These inner fibers are comprised primarily of type II collagen and are confluent with the endplate, creating a continuous envelope around the nucleus (see Figure 1). This envelop, or “zone of lability,” is vulnerable to separation after tension loading and horizontal shear due to a weak connection to the vertebral body (see Figure 1). Additionally, as much as 50% of a herniated nucleus is comprised of endplate material, which can be related to the progression of primary disc-related disorders such as disc protrusion, prolapse, and extrusion. This propensity for disruption is escalated by the laminar configuration of the anulus. The lamellar layers of the anulus are discontinuous with one another as they surround the nucleus. These discontinuities are most frequently seen in the posterior lateral corners of the disc, creating a pathway for the anular fissuring and nuclear migration associated with protrusions, prolapses, and extrusions.

Once again, this separation phenomenon is typically observed in younger subjects after macrotrauma. However, this phenomenon can also be seen in older individuals, where the nutritional state in the nucleus is compromised and disc degeneration has accelerated. The required separation forces are much lower with older patients who are suffering from the clinical consequences of disc degeneration, as accelerated discosis is accompanied by greater CEP separation, especially in the posterior disc.

The greatest compression load that is distributed through the spinal segments is imposed on the posterior inner and middle 1/3 of the anulus rather than the nucleus pulposus. Damage to the CEP and subsequent disc alterations produce increased anular stress peaks
and decreased internuclear pressure during weightbearing, which in turn may activate further structural disruption. Clinically, these behaviors are witnessed with posterior radial anular fissures, posterior disc prolapse or any structural change that renders increased space available for the nucleus to migrate. These stress peaks may be further altered during trunk movements, as intradiscal pressure is elevated during both flexion and extension of the trunk. Considering that many patients with these changes experience greatest symptom provocation during trunk movement in the sagittal plane (flexion or extension), changes in stress peaks and intradiscal pressure may be linked to provocation during those movements.

Because a healthy disc is a poro-elastic element, no permanent deformation is ensued when a prolonged load is subsequently released. The healthy nucleus serves as a means of energy dissipation and load redistribution. This behavior is related to its viscoelastic properties, where it behaves both like a solid and a fluid, depending on the stress profile to which it is exposed. This instantaneous behavioral transition becomes more permanent with aging and degeneration, changing the nucleus pulposus from a fluid-like to solid-like material that is more capable of permanent deformation and failure with chronic loading.

The water content within the nucleus is a function of its composition. The nucleus is heavily populated with proteoglycans, which are nonaggregate chains of glycosaminoglycans (GAGs) that do not efficiently bind with water. Within the disc, the inner anulus demonstrates the greatest hydrophilia due to increased density of GAGs and aggregates. Consequently, while the nucleus is contained by the anulus, the anulus functions to stabilize the motion segment in context with balanced nuclear content. Functionally, internuclear pressure activates anular tension. A nucleus that is full of water promotes anular tension and results in segmental motion control. Changes in nuclear water content, alterations in the disc’s mechanical properties, and imposed microtraumatic or macrotraumatic events all contribute to the onset of primary disc-related disorders. In addition to CEP disruptions and external disc disruptions, primary disc-related disorders include acute internal disc disruption, protrusion, prolapse, extrusion, and sequestration. Although these different afflictions are related, they each represent varying degrees of compromise to disc tissue (see Clinical Presentation of 1° Disc-Related Afflictions).

Persistent mechanical stress imposes different time-based changes in structure and composition on each segmental level of the lumbar spine. The upper lumbar disc segments (L3-L4 and above) demonstrate a natural aging process that produces increased disc height due to increased nuclear hydrostatic pressure and subsequent elevated centrifugal force dissipation throughout the disc. This elevated disc height manifests itself as a codfish-shaped appearance in the intervertebral space found with imaging (see Figure 2). Conversely, the L4-L5 and L5-S1 segments frequently demonstrate accelerated degeneration, which is accompanied by decreased hydrostatic pressure within the nucleus, decreased stress dissipation, and altered mechanics. As result, the disc height diminishes and the disc space appears flattened on imaging. This process predisposes the L4-L5 segment to greater shear forces, lending this pathway of least resistance to instability.

Whereas the healthy disc begins with a rich vascular supply to the anular regions, the adult disc is characterized by a relative avascular state. However, when the

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**Figure 2.** Time-based changes in structure and composition on each segmental level of the lumbar spine. (1) Increased disc height and codfish-shaped appearance of the intervertebral space associated with aging; (2) Centrifugal force distribution associated with increased nuclear hydrostatic pressure; (3) Decreased stress dissipation and altered mechanics associated with accelerated degeneration; (4) Decreased disc height associated with degeneration.
disc is injured and the outer anulus is breached, the deeper levels of the anulus become revascularized. Any neurovascular perforation into the inner sanctum of the anulus may be associated with increased paraspinalse muscle activity and the localized LBP associated with internal disc disruption.\textsuperscript{9,31,32} While this change increases a disc’s healing potential, it also increases the potential for pain originating from structures associated with the disc. Additionally, anular closure is rapidly restored with weaker collagen after the outer anulus is disrupted. Conversely, the deeper layers remain compromised as result of poor vascularity. The disc becomes increasingly fibrotic as it attempts to heal itself in the proximity of deep, persistent radial fissures within the anulus.\textsuperscript{33} This fibrosis forms a loose body, which can instantaneously migrate out through the fissure towards the intact anular wall. This migration, or protrusion, can be activated when the patient attempts to lift a load with the spine in a flexed, compressed state. This protrusive event can repeat itself numerous times through the patient’s adult life due to insufficient healing and progressive fibrosis.

### The Zygapophyseal Joint

Lumbar ZAJ are vertically oriented in the parasagittal plane at the L1-L2 and L2-L3 levels.\textsuperscript{34} Orientation at L4-L5 begins to rotate laterally from the sagittal plane towards the frontal plane and the orientation witnessed at L5-S1 is more obliquely oriented at approximately 15° to 60° from the frontal plane.\textsuperscript{34–39} This configuration, in concert with the outer anulus of the disc, constrains axial rotation in the upper lumbar levels.\textsuperscript{34,40}

Approximately 80% of the normal compressive load is born on the IV disc in the adolescent, while the ZAJ only carry approximately 20% of that same load.\textsuperscript{9} However, as disc height decreases with degeneration, there is a reduction of load on the disc, accompanied by a complementary load increase imposed on the ZAJ. The articular facets of the ZAJ can take 3 different tropic forms as seen from a cranial view: Flat, C-form, or J-form.\textsuperscript{5} Flat surfaces are more frequently observed at L5-S1 versus the curved surfaces witnessed in the upper lumbar segments.\textsuperscript{34} The curved portion of the J-form or C-form process contributes to constraint of transatory movement. However, this anatomical configuration can render these cartilage areas to increased aphysiological loading as the articular processes “bottom out” at the extremes of movement in the sagittal plane.\textsuperscript{12}

Asymmetries can be observed between the left versus right ZAJ in both the transverse and frontal planes. Asymmetry produces a torque preference; the greatest variance is at L1-L2 and L5-S1 and the least variance is at L3-L4.\textsuperscript{34} Thus, clinicians cannot compare left-directed rotatory motions with right rotatory motions due to the potential for anatomically based asymmetry. While several investigators attempted to correlate these asymmetries to clinical afflictions, no conclusive relationships have been postulated.\textsuperscript{41–44} However, increased sagittal orientation of the articular processes appears to be related to the development of both herniated nucleus pulposus and degenerative spondylolisthesis; however, controversy continues with regard to the clinical relevance of that relationship.\textsuperscript{1,45,46}

Eisenstein and Parry observed relatively thick cartilage on the surfaces of the zygapophyseal articular processes.\textsuperscript{47} Likened to the patellofemoral joint, these surfaces have the potential to develop pain-producing chondropathic changes. Lumbar segmental rotation is anatomically constrained by the close proximity of this cartilage in concert with the para-sagittal orientation of the ZAJ. Whereas 50% of the 1° to 2° of segmental axial rotation observed at each lumbar level is afforded by compression of the articular cartilage. Sizeable rotation in the transverse plane is only witnessed when a segment is unstable. Radiologically appreciable gapping between articular facet surfaces will clinically manifest this instability when the patient is prepositioned in rotation while recumbent.\textsuperscript{48}

The fibrous capsule of the ZAJ is complex in structure and demonstrates 2 different classes of fibers. Coursing from medial to lateral, the strong outer collagenous fibers serve as a ligament, stabilizing the joint in the transverse plane. The inner fibers share histological features with the Ligamentum Flavum and serve as the elastic component of the capsule due to their extensive elastin content. These fibers are oriented from inferior to superior and are stressed during three-dimensional movements that involved flexion and or extension. Additionally, the superior and inferior recesses of the ZAJ capsule are filled with fat, which can migrate through holes in the capsular wall with changes in intracapsular pressure.\textsuperscript{5} This may lead to infiltrate leakage false positive diagnostic ZAJ injections,\textsuperscript{49} thus explaining the poor correlation between ZAJ injection and resolution of symptoms.\textsuperscript{50} Finally, the medial aspect of the capsule is reinforced by tendinous fibers of the multifidus, presenting the ZAJ capsule as a dynamic structure versus a static restraint.\textsuperscript{51}

According to Bogduk,\textsuperscript{5} various structures project into and around the ZAJ. Among these is the synovial fatty-fibrous meniscoid structure that can be found cra-
nial and caudal at the joint margin appears to be the most clinically relevant. These menisci are well vascularized, are covered with synovium, and serve as sites for rich nocioceptor and mechanoreceptor populations. The meniscal projections may grow in size with degeneration and, therefore, extend further into the articular space. Due to this projection, patients can experience a “catching” central back pain upon returning to an upright position from sustained flexion as the meniscus is caught in-between the articular processes (see Figure 3).  

**Ligaments of the Lumbar Spine**

Ligamentum Flavum is continuous with the anterior capsule of the ZAJ and closes the posterior spinal canal between the lamina. As it is continuous with the anterior capsule, the ligament demonstrates an increased concentration of elastin fibers and is sufficiently flexible to allow for full flexion. Pretensioning can be observed with the spine in all positions, preventing any anterior buckling of soft tissue into the spinal canal. The ligament hypertrophies with chronic inflammation and/or instability and serves as a key factor in nerve root compression syndrome.

The posterior longitudinal ligament (PLL) is clinically significant, although it does not provide an appreciable biomechanical constraint to movement (see Figure 4). The PLL demonstrates 2 groups of fibers. The superficial fibers, also known as the “Kleenex Ligament,” are very narrow and thin. Conversely, the deeper fibers fan out and become confluent with the outer anulus. This ligament can produce a nonradicular pain reference when irritated with a disc protrusion or prolapse, due to the multisegmental innervation by the sinuvertebral nerve. If the disc affliction is a primary postero-lateral protrusion, irritation of the PLL will be avoided and the consequential lower extremity pain will not be initially accompanied by back pain. Additionally, the PLL is consistently connected to dura mater at the lower lumbar disc levels through the Hoffmann ligaments and dense fibrous adhesions. The pain produced by disc protrusion is related in part to disruption of these adhesions due to intermingled branches from the sinuvertebral nerve. So, a positive straight leg raise (SLR) without any radiologically appreciable bulge represents an external disc disruption (especially in young patients with extension trauma).

The anterior longitudinal ligament (ALL) is more extensively developed than the posterior longitudinal ligament, serving as a mechanical restraint to extension and shearing in the transverse plane. The ligament is barely

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**Figure 3.** Right L4-L5 zygapophyseal joint, posterior view. (1) Right inferior articular process, L4; (2) Right superior articular process, L5; (3) Intra-articular meniscal projection; (4) Inferior translation of L4 inferior process upon returning to an upright position after trunk flexion.

**Figure 4.** Posterior Longitudinal Ligament (PLL). (1) PLL, superficial fibers; (2) Right pedicle, L3; (3) L2-L4 intervertebral disc; (4) PLL, deep fibers attached to the posterior annulus of the disc; (5) L3 vertebral body; (6) L4 vertebral body; (7) L5 vertebral body.
connected to the intervertebral disc and closely con-
nected to vertebral body. The gray communicating rami
innervates this ligament via the sinuvertebral nerve,
bringing a rich sympathetic nerve supply to the liga-
ment. The ALL comes under increased stress as result of
disc degeneration, as evidenced by the anterior lipping
demonstrated at degenerated segments. Degeneration
allows the cranial vertebral segment to translate poste-
rior during extension (degenerative retrolisthesis). This
slippage decreases the load imposed upon the ZAJ and
increases the load on the ALL, which results in deep
groin pain associated with activation of sympathetic af-
ferent endings.

The intertransverse ligament can be found between
transverse processes in the lumbar spine. Additionally, it
extends to the lateral aspect of the facet and pars inter-
articularis. However, the term “ligament” is a misno-
er, as this structure is more likened to a “membrane”
consisting of sheets of connective tissue with no distinct
medial or lateral border. It is likely associated with the
complex fascial compartmental system, as the collagen
is more loosely packed and more irregularly organized
than in most true ligaments. The dorsal root ganglion
(DRG) and spinal nerve are embedded in extraforaminal
fat and connective tissue beneath the intertransverse
membrane. Additionally, the posterior primary ramus
of the spinal nerve passes through the medial aspect of
the membrane before distributing its branches to the
dorsal musculature. Any stress to the intertransverse
membrane may irritate the DRG, due to the intercon-
nections through connective tissue and fat. This irrita-
ion may be facilitated through instability and the
aphysiological loading associated with this sequel of a
disc affliction. This configuration merits flossing after
prolonged inflammation, in that it reduces the conse-
quence of potential nerve root entrapment.

Neurological Contributions
The sinuvertebral nerve provides a nerve supply to the
posterior anulus, PLL, and ventral dura mater.5 The
nerve supply to the ventral dura, PLL, and outer anulus
is multisegmental in nature. Irritation to the outer anu-
lus and PLL results in diffuse, nonradicular pain pat-
terns with irritation to these structures.8 Conversely, the
sinuvertebral nerve supplies the inner anulus with a
mono-segmental nerve supply, resulting in local, unilat-
eral, paravertebral pain when the inner anulus is irri-
tated.31 While demonstrating a scarcity of somatic affer-
ent endings, the anterior disc is heavily innervated by
visceral afferent fibers that converge with somatic pri-
mary afferents in the dorsal horn of the upper lumbar
and lower thoracic spinal cord. Chemical irritation in
lower lumbar segments leads to degenerative fissuring in
the anterior anulus, which can activate the visceral af-
ferent endings in the region. This activation commonly
produces referred anterior medial thigh and groin pain,
due to the convergence of those visceral primary af-
ferents with somatic afferents from the thigh and groin.

As previously mentioned, the outer anulus, PLL, and
ventral dura mater are richly innervated with “Silent” noc-
icceptors that possess high activation thresholds.18,19,22
Although normal mechanical stimuli do not activate
these receptors, they can become mechanosensitive after
being “sensitized” by chemical irritants released from the
disc, such as serotonin, histamine, bradykinin, prostag-
landin, and phospholipase A2 (PLA2).18 See Figure 5.
Serotonin, originating from activated platelets, will re-
duce the activation threshold of silent nociceptors
through direct Na+ activation, K+ channel blockade, or
through a cAMP messenger system. Histamine, a bio-
genic amine originating from mast cells, sensitizes the
nociceptor membrane by increasing membrane perme-
ability to Ca++. 

Bradykinin, the most profound of all pain producing
allogens, will influence silent nociceptors through inter-
action with 2 different Types of Receptors: (1) BK1 re-
ceptors, which modulate hyperalgesia, and (2) BK2 re-
ceptors, which mediate inflammatory pain.56 Activation
of the BK1 receptor triggers phospholipase C (PLC) acti-
vation, which in turn activates protein kinase C (PKC)
through diacylglycerol production. This messenger sys-

tem opens membrane cation channels, resulting in mem-
brane depolarization.57 Activation of the BK2 receptor
is responsible for initiating the actions of phospholipase
A2 (PLA2) through increased intracellular Ca++56,57
This activity leads to the release of arachadonic acid
from membrane phospholipids, which ultimately trig-
gers the synthesis of prostaglandins and leukotrienes via
the cyclooxygenase and lipoxygenase pathways, respec-
tively. Finally, prostaglandins and leukotrienes decrease
K+ membrane conductance through cAMP-mediated
inhibition, thus sensitizing silent nociceptors (see Figure
5).56,58

Nociceptive endings can be observed in the capsule of
the ZAJ.59,60 These high-threshold, slowly-adapting
group-III and group-IV capsular afferent endings are not
activated until a threshold of 3 to 5 kg of stress is ex-
ceeded. Some endings contain Substance P, which is an
11-amino acid neuropeptide that triggers afferent sensi-
tization and delayed pain of chemical origin.17,61 Acti-
viation of these endings produces a nonradicular referred pain pattern that is similar to that produced by the posterior anulus and anterior dura mater, making differential diagnosis from the pain patterns extremely problematic. Additionally, the patterns from different ZAJ segmental levels overlap as a function of each joint receiving nerve supply from multiple root segments. Furthermore, these overlapping distributions are compounded by the convergence of terminating afferents in the dorsal horns of various levels in the lumbar, sacral, and thoracic spinal cord. Consequently, symptoms arising from any single ZAJ will be unilateral, paraspinally, relatively vague, and not easily pinpointed by the patient or clinician.

Afflictions of the lumbar nerve roots can compound clinical low back pain, based on 3 factors: (1) compromised protection; (2) nutritional status; and (3) varying responses to mechanical and chemical stimuli. The lumbar nerve roots are internally covered by endoneurium and pia mater. Surrounding these layers is the dura mater, which is less developed in comparison to the epineurium found more distally on the peripheral nerve. Thus, the root is less protected from mechanical deformation and chemical irritation than peripheral nerves.

The root receives nutrition from 2 sources: vascular branching and cerebrospinal fluid, with the primary source arising from the arterial tree surrounding the root. However, this vascular supply demonstrates fewer lateral vascular branches at the level of the root versus the same supply to the dural sac and the peripheral nerve. A secondary nutritional source is provided to the root through the cerebrospinal fluid (CSF) that is found under the dural sleeve. A scarcity of CSF can be observed at the distal insertion of the sleeve, in the region of the root that demonstrates the poorest capillary perfusion (a “zone of lability”). Any mechanism that
compromises both vascular supply and CSF will further reduce the nutritional support and metabolite diffusion, especially at that zone.66 This regional compromise can lend the nerve root to accelerated irritation and resultant mechanosensitivity.

Mechanical root deformation can arise from either the tension loading that is witnessed in a primary disc-related disorder or compression loading that results from secondary disc-related changes.67 The root is repeatedly loaded under tension, as evidenced by the longitudinal orientation of the majority of collagen fibers found in the dura mater.68 Because the root is straight and does not possess the tension-accommodating fountain spirals found in a peripheral nerve, tension loading could have a greater consequence on the root versus the peripheral nerve. However, a root becomes mechano-sensitive only after it has been chemically irritated, as tension loading to a normal root does not produce pain.18 Investigators have suggested that a nerve root can become chemically irritated from exposure to nuclear disc material.22 Additionally, this exposure produced accelerated degenerative responses, accompanied by axonal swelling, increased axoplasmic density, and myelin sheath splitting.69 Thus, any pain provocation from tension loading to the root is delayed after the onset of a primary disc-related disorder due to the time required for developing chemical root irritation.

Although root mechanosensitivity is delayed and gradual in onset, the DRG demonstrates an immediate mechano-sensitivity when it is mechanically deformed. Even slight mechanical pressure on the DRG can induce discharges for up to 25 minutes after the stimulus is removed (known as after-discharges). This propensity is related to the relatively high concentration of Na⁺ channels that can be found in the region of the DRG.70,71 Thus, the sharp, mechanically induced lower extremity pain observed in the early stages of a primary disc-related disorder is typically related to mechanical deformation of the DRG, while the aching lower extremity pain of gradual onset is related to chemically mediated mechanosensitivity of the root itself. This information can be useful in planning management strategies, as a clinician could expect mechanical interventions to alter the DRG-related pain, while the chemical pain associated with the root may better respond to pharmacological management.22

Lumbar Mechanics

The intervertebral disc segment and corresponding 2 ZAJ form a 3-linked closed kinematic chain, demonstrating a functional motion symbiosis.11,12 An affliction in any one of the components can produce compensatory effects and responses in the other two. However, the disc appears to exert a disproportionate influence on the mechanics of the lumbar spine. As previously mentioned, along with the sagittal orientation of the ZAJ articular surfaces, the outer anulus of the disc constrains axial rotation at the upper lumbar segments. Conversely, the oblique orientation of the surfaces at L5-S1 allows more segmental rotation. Additionally, this articular orientation has less influence on controlling flexion or extension, which appear to be primarily constrained by tension loading of anulus fibrosus.72–74

Thus, a functional model can be proposed from these ideas. During lumbar segmental flexion, the disc controls angular motion while the ZAJ limit ventral translation. Extension creates an opposite effect, where the ZAJ control angulation and the disc limits dorsal translation. As previously reported, very little segmental axial rotation is available in the lumbar spine. However, while the disc controls the lateral translation associated with the couple sidebending, the angular motion is limited by the vertical orientation of the ZAJ in the parasagittal plane. Finally, the angular and translational behaviors of sidebending are both controlled by the disc.75 Thus, a clinician could expect a patient with a disc affliction to report the greatest provocation during sagittal or frontal plane motions with no increase in those symptoms when three-dimensional rotation is added to the test movements.

McFadden, in studying the degree of pure axial rotation achieved by the lumbar vertebral column in the sagittal plane, concluded that appreciable pure axial rotation is not available in the lumbar spine and that all rotation in the transverse plane is accompanied by lateral flexion (or sidebending). Additionally, he suggested that no gaping should be observed in the ZAJ during pure axial rotation and if there any gaping was demonstrated, then segmental instability is suspected.76 McFadden’s observations reinforced the concept of coupling. This concept proposes that a kinetic movement of the vertebral body along 1 axis induces a simultaneous synkinetic movement along another axis.77

The strongest lumbar coupling pattern is a synkinetic contralateral axial rotation that accompanies kinetic sidebending, while the lumbar spine is prepositioned in extension. Conversely, as the extended lumbar spine is kinetically rotated it will synkinetically sidebend in a contralateral direction at L1-L2 through L4-L5, whereas L5-S1 will sidebend ipsilaterally. Additionally, the lum-
bar spine will synkinetically rotate in the same direction of sidebending at all segmental levels, when the spine is prepositioned in flexion. Furthermore, the lumbar spine locks when placed in a “combined” position. The combined positions are three-dimensional and opposite any coupled movement. For example, the combined position with the spine in extension is first sidebending, followed by ipsilateral rotation. In flexion, the lumbar spine is locked in a combined position of sidebending and contralateral rotation. These three-dimensional behaviors can be used for differentially diagnosing afflictions of the articular cartilage or synovium of the ZAJ, as those structures are maximally stressed during three-dimensional motion (see Clinical Pathology of 2° Disc-Related Disorders).10 Finally, abnormal or exaggerated coupling patterns have been described as a possible sign of instability or pathology.78–79

CLINICAL PATHOLOGY OF 1° DISC-RELATED DISORDERS

As previously reported, changes in nuclear water content, alterations in the disc’s mechanical properties, and imposed micro or macrotraumatic events all contribute to the onset of primary disc related disorders. Primary disc-related disorders include acute external disc disruptions, acute internal disc disruption, protrusion, prolapse, extrusion, and sequestration. Although these different afflictions are related, they each represent different clinical pictures associated with varying degrees of compromise to disc tissue.

In spite of the relative strength of the outer anulus, this area is vulnerable to injury, especially as the consequence of macrotrauma. Younger individuals are at risk for acute external disc disruptions sustained during high velocity or large impact trauma while participating in sporting activities. These individuals present with variable movement limitations in the sagittal plane (flexion and or extension), due to either tension or compression forces that are imposed on the outer anulus.80 Frequently, these patients will present with low back pain provoked by either a slump or straight leg raise when distally initiated (ie, through the ankle and leg first), due to tension loading on the outer anulus imposed through the root and ligaments of Hoffman. This condition can last for several weeks; however, symptoms are self-limiting and can be reduced through general cardiovascular conditioning (activation), only cautioning the patient to reduce positions and movements that are pain-producing.

Acute internal disc disruption more commonly affects younger individuals as result of compression-load trauma.50,80 This traumatic event can be sustained in the direction of either flexion or extension, which will also be most provocative and limited during the clinical examination in response to tension or compression loading.81 These patients will present with negative du-

ral tests, will typically self-limit and therefore are best treated with trunk stabilization and generalized conditioning (activation), only cautioning the patient to reduce painful positions and movements.

A disc protrusion is produced when an annular fissure forms from within the disc, which allows the nuclear material to migrate either in a posterior-median or posterolateral direction. Because the outer anulus and posterior longitudinal ligament remain intact, the compromised area within the disc remains relatively avascular and forms a dry fibrotic loose body, which is subsequently catapulted out against the inner wall of the outer anulus. Because of the rich population of free nerve endings in the outer anulus, this protrusion produces LBP in a nonradicular distribution, consistent with the polysegmental innervation associated with the sinuvertebral nerve that innervates the area.5,31 Because the fibrotic loose body has a poor healing potential, the patient experiences frequent reoccurrence of symptoms and variable limitations of motion especially in the sagittal plane.

In the event that the protrusion is large enough to tension load an adjacent nerve root, the patient may experience lower extremity pain. Additionally, lower extremity pain will be provoked during the slump and straight leg raise tests especially when the root tension is greatest (ankle/foot dorsiflexed, knee extended, and chin tucked). The patient will experience more provocation with slump versus straight leg raise, due to increased root tension and increased posterior nuclear migration with the slump position. Due to the lack of axonal involvement with the root irritation, these patients will not present with any neurological deficits, such as weakness or true numbness.10

A protrusion can be classified as either a “shoulder” or “axillary” lesion, depending on the location of the protrusion with respect to the corresponding nerve root. A “shoulder” lesion presents with the protrusion lateral to the root (in the “shoulder region” of the root). See Figure 6. These patients will typically demonstrate a postural lateral shift away from the side of pain due to the trunk’s attempt to reduce tension loading in the root. Additionally, the patient will demonstrate increased leg and or low back symptoms with sidebending towards the involved side, due to increased tension in
Conversely, an “axillary” lesion presents with the protrusion located medial to root (in the “axilla region” of the root). See Figure 6. These patients will present with increased symptoms during sidebending away from the painful side, which is again due to increased root tension. Lateral shift is not a reliable means of differentiating these patients from other disorders, because an appreciable number of patients with an axillary lesion may present with a lateral shift away from the side of pain.

A posterior lateral prolapse is similar to the protrusion in that the nuclear material migrates through an anular fissure. However, with a prolapse the outer anulus is breached, leaving only the posterior longitudinal ligament intact (which can serve as a source of LBP with this condition). The migrating nuclear material contains additional anular and endplate material, suggesting a substantial traumatic precursing event and a resulting “sick” condition in the disc. The prolapse is commonly associated with a macrotraumatic event that results in a rapid onset of pain in the low back, thigh, lower leg, and foot, which may be associated with a brisk inflammatory response in and around the posterior disc region and nerve root. These patients will once again present with a positive tension sign during the slump and the straight leg raise that is similar to that observed with a protrusion, but only more severe and provocative. These patients will commonly present with a lateral shift and provocative movements (previously described), as well as positive neurological deficits (weakness and or numbness), which provide the clinician with a distinctive diagnostic indicator for differentiating between protrusion and prolapse. Although this condition could be viewed as more severe than the protrusion, it is potentially eradicated more rapidly and completely versus the protrusion. This may be related to in-growth of a vascular supply into the fissure, promoting subsequent healing.

Management of LBP “... arises from the balance of wisdom obtained through scientific laboratory experiment, and the ‘art’ “developed from clinical experience.” Protrusion and prolapse can be managed with activity counseling, local thermal agents, axial separation or traction, extension exercises (in some cases), stabilization exercises, and generalized cardiovascular conditioning (activation). Activity counseling is essential to effective management of a lumbar condition. Indahl et al reported that “sub-chronic LBP can be managed successfully ... and long-term disability may be reduced with information for patients about the nature of the problem, provided in a manner designed to reduce fear and give them reason to resume light activity.”

Figure 6. Locations of posteriolateral protrusions and prolapses in the lumbar spine. (1) Nerve root; (2) Intervertebral disc, L3L4; (3) Intervertebral disc, L4L5; (4) Left pedicle, L4; (5) Shoulder “lesion of the intervertebral disc, lateral to nerve root; (6) Axillary” lesion of the intervertebral disc, medial to the nerve root.
tients are counseled to avoid sitting and bending for the first few hours of the day, as investigators have demonstrated that the disc is under greatest stress at that time due to imbibition and diurnal stress fluctuations. Thermal agents can be used as a precursor to other interventions resulting in patient relaxation through convergence in the dorsal horn of the spinal cord.

In the event that the patient presents with a shoulder lesion, the patient can be treated with static lumbar traction, if implemented within the first 6 weeks post onset. Although the clinical outcomes associated with the use of traction have been controversial, investigators have demonstrated several positive effects associated with the application of traction. Studies have demonstrated that traction produces (1) increased trunk stature in normal individuals, (2) separated lumbar vertebral segments, (3) reduced disc protrusion, (4) reduced motor function impairment associated with radiculopathy, and (5) reduced symptoms in LBP patients. Winkel et al recommends at least 30 minutes of static traction adjusted to 25% to 50% of the patient’s body weight, along with a thoracic stabilization belt.

 Conversely, the clinician would be wise to use three-dimensional axial separation when a patient presents with an axillary lesion, as traction can increase the tension loading of the root around the protrusion or prolapse and subsequently escalate the patient’s symptoms.

The posterior central prolapse is considerably more serious than the posterior lateral counterpart, in that the posterior longitudinal ligament is disrupted and the spinal canal is occupied by nuclear material. This condition is most frequent at the L3-L4 and L4-L5 disc levels, as reliably witnessed on MRI or CT. The patient describes exquisite LBP and bilateral lower extremity pain. The patient presents with remarkable lumbar kyphosis in an attempt to unload the disc. Dural tests (slump and straight leg raise) will typically be positive for a tension sign (previously described). Additionally, the patient may present with cauda equina symptoms, which include saddle anesthesia, bowel and bladder disturbances, impotence in males, and numbness in the plantar aspects of both feet. These patients may be good surgical candidates, if they present with neurological signs.

The extrusion is a primary disc-related disorder that demonstrates a disruption of both the outer anulus and the posterior longitudinal ligaments, producing a complete disturbance in the hydrodynamic qualities of the disc and subsequent disc migration outside of the outer sanctum of the anulus. The patient presents with inevitable positive tension signs during the dural testing, reliable findings on the MRI, positive neurological deficits and resistance to nonsurgical treatment. A sequestration is similar to the extrusion, except that the protruding material has sheared off and is free floating in the extradural space. This condition has a similar clinical presentation to the extrusion, and can also be reliably diagnosed from MRI.

**Clinical Pathology of 2° Disc-Related Disorders**

**Introduction**

Secondary disc-related pathology can emerge from long-standing primary disc-related disorders. Disc degeneration results in a reduced disc height and subsequent anular buckling. Consequently, the segmental axes for motion in the sagittal plane migrates away from the disc. This shift promotes a physiological loading, a decreased mechanical neutral zone to control, and increased shearing stress on the disc and ZAJ. This stress produces chronic disc irritation, zygapophyseal subchondral overload and chondropathy.

Pain Generators Associated with NSLBP

In the younger patient, a pain-producing motion segment commonly presents with hypermobility, with hypermobility observed at adjacent segmental levels. However, for older patients the painful level is typically hypomobile, resulting in either synovitis or degenerative arthritis. Nonobjectifiable hypermobility produces an increased neutral zone of segmental motion while the total motion range remains unchanged, reflecting a decrease in the segment’s ability to resist movement in the sagittal and frontal planes. This results in a “wobble” during movement and a subsequent painful “catch” during flexion and extension. Shearing load may increase as result of this wobble, facilitating ZAJ chondropathy. Persistent chondropathy can digress into chondromalacia especially in the context of “Bottoming Out” (previously described).

Hypermobile segments are attempting to stabilize in context with progressive disc fibrosis and reduced disc height. The collagen within the disc transitions from type I into type III, resulting in reduced tensile strength. Conversely, the type II collagen in the nucleus transforms into type I, resulting in a dry, hardened mass and a “Vacuum Phenomenon” on CT. These changes produce ventral, dorsal, and lateral liping on radiographs, which represents a segment’s attempt to stabilize a hypermobile segment. Although these bony changes do not
serve as pain generators, they represent changes in the disc and joints that can produce symptoms.

In spite of these changes, many patients lack objectifiable evidence of their chronic LBP. This condition has been called NSLBP by numerous investigators and can originate from the disc, the ZAJ, or an unknown location. Patients with NSLBP present with a long history of intermittent back problems, which may have been repeatedly treated with multiple manipulations. Minor activities provoke major complaints, accompanied by catching sensations, twinges, and sharp shooting pain with movement, including rotational activities.72 This sharp, shooting pain can be related to mechanical deformation of the dorsal root ganglion, which can be compressed and angulated around the pedicle or through the transverse ligament during movement.55,61 Patients also report transient neurological symptoms that suddenly emerge and quickly resolve. Finally, the patients must push themselves back to a standing position using their hands on their thighs or a nearby table.

NSLBP courses through stages, including pseudospondylolisthesis, segmental hypermobility, and remodeling. Segmental hypermobility advances through 2 phases including anterior-posterior hypermobility and three-dimensional sidebending hypermobility.101 Pseudospondylolisthesis produces increased nonobjectifiable segmental axial rotation, with increased segmental axial hypermobility and excessive axial torque along the Y-axis. These movements produce increased rotation, excessive vertical translation, and resultant strain to the anulus and ligaments.11 The pain produced by these changes can originate from chondropathic changes, synovitis, or a chronic disc irritation. Irritation to the ZAJ is vague and diffused, presenting unilaterally in the paraspinal, buttock, gluteal, and posterior lower extremity region. Additionally, groin and anterior thigh pain (as far as the knee) can accompany chronic back pain, due to information from the visceral afferents that innervate the anterior disc.

Patients with chondropathy experience less pain with flexion movements and positions. Pain increases with the course of the day and the greatest pain is produced during three-dimensional movements that lock the facets (eg, extension, sidebending, and ipsilateral rotation). See Figure 7. Conversely, patients with synovial irritation experiences more pain in the evening and will have greatest pain during coupled movements, due to maximized stress of the capsule and its synovial lining (eg, extension, sidebending, and contralateral rotation.) See coupled movements on Figures 8 and 9. Patients suffering from zygapophyseal synovitis or chondropathy may benefit from electrical stimulation to the upper lumbar spine, so to reduce autonomic involvement in persistent symptoms. In addition, oscillatory mobilization to the painful segment may reduce symptoms when accompanied by mobilization to the adjacent hypomobile segment to improve motion. Furthermore, general stabilization training can be implemented to improve segmental stability. Finally, persistent symptoms may be reduced with a medial branch nerve block, cryoneurolysis, chemical neurolysis, or RFTC lesion under fluoroscopic guidance.

![Figure 7. Interpretation of 2° Disc Related Disorders of the Lumbar Spine: Greatest Pain Provocation in Extension, Sidebending and Combined Rotation Movements.](image-url)
Nociceptive information can originate at any location on the chronically irritated disc, producing a band-form pain across the back. Additionally, chronic, nondisrupted disc irritation can produce nonradicular, referred pain in the buttock, posterior thigh, and posterior lower leg. Negative dural tests will differentiate this lower extremity pain from pain associated with root irritation. The pain produced by a chronic disc irritation is most easily provoked during trunk flexion (and possible extension) without significant increased symptoms when coupled or combined movements are added to a flexed or extended position. See Figure 10. Patients suffering from chronic internal disc disruption may benefit from electrical stimulation to the upper lumbar spine, as previously reviewed; mobilization to a painful segment for pain control; mobilization to an adjacent hypomobile segment to improve motion; and general stabilization training to improve segmental stability. Persistent symptoms may be reduced with an IDET procedure.

**Figure 8. Interpretation of 2° Disc Related Disorders of the Lumbar Spine: Greatest Pain Provocation in Extension, Sidebending and Coupled Rotation Movements.**

**Figure 9. Interpretation of 2° Disc Related Disorders of the Lumbar Spine: Greatest Pain Provocation in Flexion, Sidebending and Coupled Rotation Movements.**

**Pain Generators Associated with Specific LBP**

Segmental hypermobility and the progressive degeneration associated with NSLBP can advance into an affliction category known as “Specific LBP.” These conditions include degenerative spondylolisthesis, dynamic
nerve root compression syndrome, dynamic neurogenic claudications, static nerve root compression syndrome, and static neurogenic claudications.

Degenerative spondylolisthesis is an objectified manifestation of sagittal plane instability. This condition includes retrolisthesis, or posterior translatory instability, and anterolisthesis, representing anterior instability and accompanying ZAJ pathology. Any back pain associated with degenerative spondylolisthesis arises from intervertebral disc stress and zygapophyseal chondropathic changes. Degenerative spondylolisthesis is multifactorial, and can be predisposed by genetics, hormonal imbalances in females, general laxity states (such as Marfan’s syndrome), further disc degeneration, and previous surgical fusion at an adjacent level.

Stenosis, either congenital or acquired can predispose a patient for developing nerve root compression or neurogenic claudications. The majority of stenotic patients demonstrate degenerative disc and ZAJ changes, including intervertebral foraminal narrowing linked to an altered association between two lumbar segments. Additionally, stenosis can be central or lateral with central stenosis involving the spinal canal and lateral stenosis involving the lateral recess and or intervertebral foramen. Furthermore, stenosis can be either static or dynamic. Clinical signs and symptoms do not change with altered body position during static stenosis, but can change with different positions and motions of the trunk during dynamic stenosis. This is related to a change in canal area during flexion and or extension. With degenerative changes the flaval ligament demonstrates decreased elastin and increased collagenation, accompanied by increased fat in posterior canal. During extension, a posterior disc bulge is accompanied by an anterior bulge of the flaval ligament and fat pad. These two changes compromise the area of the spinal canal, lateral recess, and intervertebral foramen, promoting the compression of the neural tissue lying within.

Spinal stenosis does not necessarily have to be pathological. Single-level stenosis may not present clinical problems, while symptomology arises when 2 levels are stenotic. Two-level central stenosis produces intermittent neurogenic claudications (INC) as a result of venous plexus pooling in the ventral spinal canal at L3-L4 and L4-L5. This condition is the result of cauda equina compression within the spinal canal as result of increased epidural pressure. Intermittent neurogenic claudications are a function of this pressure and can increase with standing, standing with extension, and walking, while it will typically decrease when sitting or walking with the lumbar spine flexed (a “simeon” posture).

Conversely, nerve root compression syndrome (NRCS) arises out of a combination of a single-level central stenosis at L4-L5 and a one-level lateral recess or foraminal stenosis compromising the L5 or S1 root. Dynamic NRCS is the consequence of nerve root compression resulting in vascular compromise and inflammation. This inflammatory event “sensitizes” the nerve root, producing a source of nonradicular and radicular pain. The L5 nerve root is most commonly involved at the L5-S1 intervertebral level, as a consequence of the L5-S1 root canal being the longest and the narrowest of all canals, along with the L5 root being the thickest of all lumbar roots.

Dynamic INC and NRCS will produce peripheralizing symptoms. Posterior root irritation can result in sensory changes and parasthesias may develop as result of lateral recess compression of the root by venous plexus engorgement. This compression can lead to chemical irritation of the root, thus making the root mechano-sensitive. The patient complains of increased symptoms with walking (especially downhill) or lying in a supine position. Conversely, uphill walking, cycling, and sitting in a recliner will decrease symptoms. Additionally, extensive or long-term root compression can produce motor, sensory, and nociceptive disturbances. The clinician will observe positive dural tests, with those symptoms increasing during distal tension loading and symptom reduction during cranial tension loading, due to a change in compressive irritation of the previously mentioned “zone of lability” as the root is repositioned by different test positions.
SUMMARY

Different structures within the lumbar spine can serve as pain generators. Several different categories of afflictions can produce a similar clinical presentation at face value. A careful clinical history and examination can assist the clinician in differentially diagnosing these different afflictions. Each affliction merits a different management strategy that is target-tissue specific. This tissuespecific approach to diagnosis and treatment of lumbar pain can expedite patient recovery as needless tests and management approaches can be avoided.

REFERENCES


63. Gillette RG, Kramis RC, Roberts WJ. Spinal projec-


95. Guechev G, Guechev A. Fast dynamics of voluntary


