Involvement of the Lumbo-Sacral Plexus in Mechanically Induced Pelvic Pain and Organic Dysfunction. An Overview of the PPOD Syndrome.

A Literature Review

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Abstract

Objective:

The purpose of this study is to review the literature concerning treatment of lumbo-sacral plexus dysfunction, particularly regarding the involved anatomy, associated conditions, specific therapy protocols, and safety.

Methods:

A qualitative literature review and clinical experiences by licensed physicians are the basis for this overview of the condition known as the PPOD syndrome. Largely based on the work of Dr. James Browning D.C., this review will discuss the symptoms, involved anatomy, neurologic dysfunction, treatment protocols, and patient outcomes associated with the PPOD syndrome.

Results: The literature is rich with research about the etiology of many low back and pelvic pain generators such as the intervertebral disc, spinal stenosis, and various chemical irritations to associated nerve roots. However, when it comes to research correlating back and pelvic pain and organic dysfunction there is significantly less research. There is, however, strong clinical correlation involving pelvic pain and organic dysfunction reported by Dr. Browning and many other physicians.

Conclusion:

By looking at the concepts of abnormal sensory and motor fiber function and applying them to the other structures supplied by the various branches of the pudendal, pelvic splanchnic and unnamed nerves, we can begin to see how mechanical insult to the lower sacral nerve roots caused by a mechanical disorder in the region of the lumbar spine can result in the development of not only wide spread chronic pelvic pain, but also the wide range of disturbances of bladder, bowel, gynecologic, and sexual function that are so commonly found in mechanically induced PPOD syndrome patients. [16]

Introduction:

The mechanically induced Pelvic Pain and Organic Dysfunction (PPOD) syndrome as discussed by Dr. Browning is "a disorder of spinal origin characterized by a wide range of symptoms of pelvic organic dysfunction. Caused by a mechanical disorder of the lumbar spine with secondary impairment of the lower sacral nerve root function, its clinical presentation is highlighted by various combinations of symptoms of pelvic pain and disturbances of bladder, bowel, gynecologic, and sexual function." [15]

PPOD syndrome is described as "a syndrome within a syndrome." Meaning a patient will present to a physician's office with a leg or low back pain complaint, but not necessarily correlate their pelvic pain symptoms to their current back dysfunction. Patients will often see a urologist or gynecologist for their pelvic pain, and a chiropractor for what seems to be a separate low back or leg complaint. Dr. Browning's challenge to physicians is to sift through their back pain patients to identify those who also demonstrate the spectrum of over 30 symptoms that comprise the PPOD syndrome.

Discussion:

Although on the surface these problems seem to be completely unrelated, the following discussion will clearly demonstrate that all of them are likely being caused by a common underlying disorder. These symptoms can be broken down into four categories; Bladder, Bowel, and Gynecologic/ Sexual Dysfunction.

Bladder Dysfunction

Frequency - Increased number of urinations during a given amount of time.

Urgency - Elevated feeling of needing to urinate.

Dribbling - post micturition dribbling.

Urinary Incontinence- Which can be subdivided into several types.

True urinary stress incontinence- occurs during activities that would increase the intraabdominal and intravesical pressures such as coughing, sneezing, laughing, bearing down or stooping down with flexion, and or rotation of the lumbar spine.

Urge Incontinence- Strong, sudden, uncontrolled urge to urinate.

Mixed Incontinence- as the name suggests encompasses those patients that have urine loss from either or both increased intravesical pressure or a sudden urge to void the bladder.

Overflow or Paradoxical incontinence- A sudden urge to void the bladder that only occurs once the bladder has reached an excessively, elevated level of filling.

Difficulty- some patients have issues either initiating urination or they may have difficulty completely emptying the bladder.

Sluggishness- often associated with a reduction in the decreased in force of the urine stream.

Retention- can occur with some patients not being able to completely empty the bladder or in sever cases the patient requires catheterization in order to empty the bladder.

Nocturia- some patients experience problems with getting up frequently during the night to urinate, or they experience bed wetting (enuresis).

Dysuria- Painful urination can occur with or without the presence of an active bacterial infection in the upper or lower urinary tract

Loss of Bladder sensory perception- An inability to sense normal urinary filling or a total loss of bladder filling is possible.

Bowel Dysfunction

Constipation – infrequent bowel movements or difficultly in passage of stools.

Diarrhea – loose, watery stools that occur more frequently than usual.

Alternation constipation and diarrhea- which can mimic the commonly diagnosed irritable bowel syndrome.

Excessive intestinal gas

Anal sphincter spasm- which can prevent normal passing of the stool.

Encopresis- Fecal incontinence

Mucorrhea- mucus discharge from the rectum.

Loss of rectal sensory perception- which can be partial or total loss of the sensation of rectal filling.

Spontaneous bowel discharge- can often be uncontrollable discharge.

Gynecologic and Sexual Dysfunction

Miscarriage- significant if miscarriage is recurrent. However it is considered normal for 1 in 5 females trying to achieve pregnancy for the first time to miscarry.

Vaginal discharge

Vaginal spotting

Painful/ Irregular menstruation

Menstrual migraine

Decreased genital sensitivity- partial or complete loss of genital sensitivity.

Decreased or loss of orgasm

Dyspareunia- painful intercourse

Genital pain/ parethesias- most commonly females will experience tenderness or throbbing of the labia, but sometimes patients will experience pain around the vestibular glands (vestibular vulvodynia).

Pelvic pain on orgasm

Deficient coital lubrication- decreased function of the greater vestibular or Bartholin's glands.

Depressed libido- decreased desire for sex.

Impotence- erectile dysfunction

The Anatomy

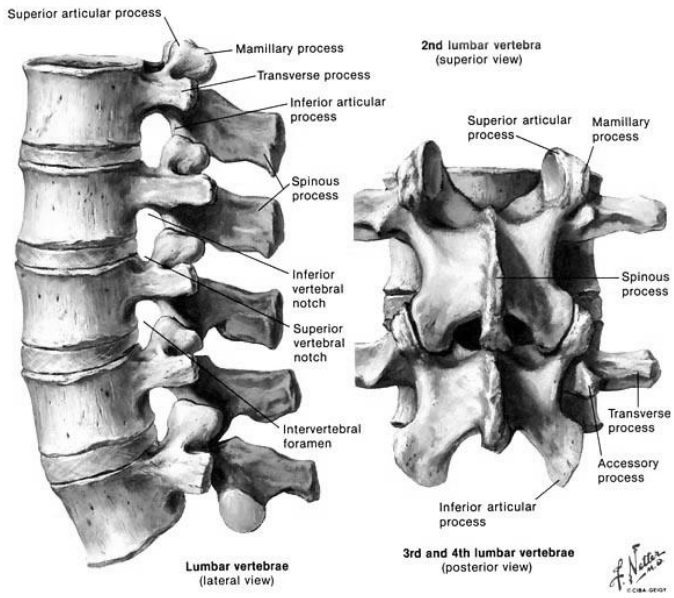
The symptoms of the PPOD patient may seem like isolated complaints, but as we discuss the involved anatomy it will become clear how a single mechanical deficit can be responsible for a multiple symptoms described above.

The basic anatomy of the spinal column, cord, nerves, and associated musculature that is involved in the PPOD patient in the following discussion is by no means an all inclusive look at these structures, but the essential anatomy is necessary to realize the link between a mechanical dysfunction of the spine and the 'numerous accompanying disturbances of bladder, bowel, gynecologic, and sexual function.' [16]

The spine is made up of 24 vertebrae separated by intervertebral discs. When seen from the side the spine forms the 3 natural curves including the cervical lordotic curve, the thoracic kyphotic curve, and lumbar lordotic curve. The sacrum and coccyx are 2 fused bones at the bottom of the spine. The fused vertebrae along the sacrum and coccyx, in conjunction with the cervical, thoracic, and lumbar spine vertebrae, form the

spinal column. The posterior elements of the spinal column form a bony tunnel called the spinal canal, which protects the spinal cord. It's the area of the low back or lumbar spine that, from the standpoint of the mechanically induced PPOD syndrome, the majority of our interest exists.

Adjacent vertebrae along the spinal column form interlocking structures (joints) which vary by region and are separated by intervertebral disc. The anterior portion of the joint consists of the vertebral body and intervertebral disc. Discs are the shock absorbers between the vertebrae, and are the major weight bearing structures. In combination the discs and vertebral bodies carry 70-80% of the body's weight in a neutral position. Each disc is composed of two distinct parts, an outer region called the anulus, which in the human is formed from approximately 10-12 layers of cartilage [11], and an inner portion called the nucleus pulposus. The nucleus is comprised of a gelatinous protein matrix which is approximately 80-90% water in its overall composition.[7,33] Upon removal of the nucleus to observe its physical properties it would be found to be similar in consistency to the white portion of an egg. The disc provide flexibility by changing shape allowing for flexion, extension, rotation, lateral flexion, and circumduction.



Lateral and posterior view of the Lumbar spine. Netter.

The spinal cord tapers to a point called the conus medullaris, and the spinal nerves continue inferiorly through the spinal column as a structure called the cauda equina. Those nerves which compose the cauda equina will exit their appropriate intervertebral foramen and form the lumbar and sacral plexi. It's important to understand the complexity of the lumbo-sacral plexus, especially the lower sacral nerve roots, when discussing the PPOD syndrome. The common somatic pain patterns of the pelvis as described earlier, as well as the 30 plus symptoms associated with the mechanically induced PPOD syndrome are intricately related to the anatomy of the lumbo-sacral plexus.

Lumbo-Sacral Plexus

Leaving the level of S4 there are a number of unnamed nerves, which supply motor and sensory function to the muscles of the pelvic floor. These muscles are the predominate structural support for the pelvic floor, help maintain the proper position of pelvic organs, and allow proper urinary and rectal continence. Also from the lower sacral nerve roots (S2-S4) levels is the origin of the Pudendal and Pelvic Splanchnic nerves.

The Pudendal nerve is derived from the anterior primary division of the S2-S4 sacral spinal nerves, and supplies extensive neurological connections to organs throughout the pelvis Major divisions of the Pudendal nerve include the **Inferior Rectal nerve, Dorsal nerve** of penis/clitoris, and **Perineal nerve** which divides into the **posterior scrotal/labial nerve** and a **Muscular (deep) branch**. The Pudendal nerve leaves the inner pelvis by traveling inferior between the Piriformis muscle and Coxygeus muscle through the lower portion of the greater sciatic foramen. At this point it joins the internal

Pudendal artery and dives into the greater sciatic foramen and into the Pudendal canal. Along its course the Pudendal nerve gives off two branches called the **Inferior Rectal nerve and dorsal nerve**. The **Inferior Rectal nerve** supplies somatic sensory and motor fiber to all layers of the external anal sphincter, sensory to the mucus membrane of the lower portion of the anal canal and sensory to skin of anus and perineum. In the female the inferior rectal nerve supplies the lower third of the vaginal wall. **The Dorsal nerve** in the female supplies somatic sensory fibers to the corpus, crus, glans, prepuce, and frenulum of the clitoris and anterior regions of the labia minora and majora. In the male the dorsal nerve runs along the dorsal lateral aspect of the penal shaft supplying sensory to the skin, fascia, and corpus cavernosum. Upon reaching the distal region of the penis the dorsal nerve supplies the glans and frenulum. The **Perineal nerve** divides into **the posterior scrotal/ labial nerve** and **Muscular (deep) branch**. The **Posterior scrotal nerve/ labial nerve** are sensory to the posterior two-thirds of scrotum or labia majora. The **Muscular (deep) branch** is sensory and motor to anterior half of pelvic floor, and sensory and motor to (bulbospongiosus, ischiocavernosus, transverse perineus profundus, perinea superficialis, urethral sphincter, and anterior portions of the external anal sphincter and levator ani.) Prior to reaching the bulbospongiosus a small division called the **nerve to the urethral bulb**. From the origin of this nerve will pierce Buck's fascia and supply sensory input to the corpus spongiosum. It continues on to supply terminal sensory branches to the mucus layer of the urethra.

Pelvic Splanchnic nerves are derived from the anterior primary division of the S2-S4 sacral spinal nerves, and supplies extensive neurological connections to organs throughout the pelvis. These nerves supply parasympathetic innervations pelvic organs

with **visceromotor, vasomotor, and secretomotor** functions. **Visceromotor** fibers to: the distal half of the large bowel, rectum, muscular wall of the urinary bladder, ureters, renal pelvis, lower portions of the uterus, cervix, upper region of the vagina, and inhibitory fibers to the vesicle and urethral sphincters. **Vasodilator** fibers to: testes, prostate, seminal vesicles, vas deferens, uterus, ovaries, fallopian tubes, greater vestibular glands, and erectile tissues of both the penis and clitoris. **Secretomotor** fibers to: the prostate, seminal vesicles, and ejaculatory ducts. Most of these structures listed also receive a visceral afferent supply though fibers of various branches of Pelvic Splanchnic nerves. These fibers represent the afferent limb of the pelvic parasympathetic reflex arch, thus are important in maintaining and regulating normal pelvic organ function. Some of these fibers however terminate in free nerve endings, which terminate within the muscular wall of the various organs and tissues previously discussed. When activated, are responsible for creating impulses associated with the transition of painful or noxious stimuli

The connections made by the Pudendal, Pelvic Splanchnic, and Unnamed nerves that innervate the pelvic floor supportive musculature makes it clear how a mechanical dysfunction of the lower lumbar and sacral nerve roots could results in pelvic pain and organic dysfunction.

Discerning pain generators of the Low Back

It's important to discern the various tissues of the low back and their pain distributions. "An in vivo study of the pain production in the human lumbar spine," published in *The Origin of Low Back Pain* describes an experiment on 67 patients that studied the pain response of various tissues to mechanical irritation. Patients underwent microsurgical

nerve root decompression for herniated discs, and the experimenters would stimulate various tissues by means of pressure, stretch, or electrocautery and record the responses of the patient to a few simple questions. The study points out a few structures that reproduced significant low back and leg pain in most patients.

Low Back Pain

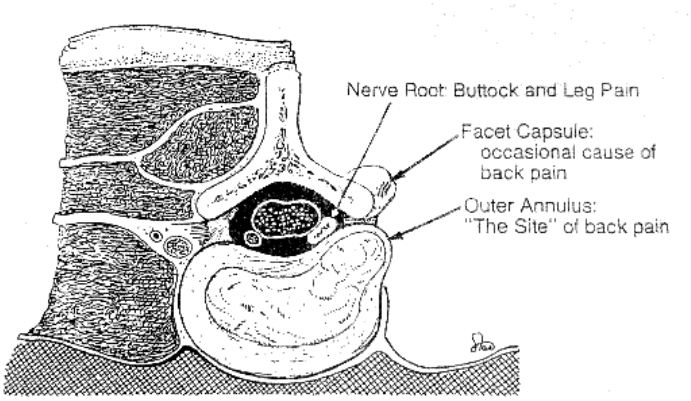
Outer Layers of Anulus Fibrosus (outer layers of the intervertebral disc)
Anterior Epidural tissues when inflamed
Facet capsule (occasionally)

Buttock Pain

Compressed Nerve Root
Nerve Root dura

Sciatica (shooting leg pain)

Compressed stretched, or inflamed nerve root.



Primary Sources of Low Back Pain and Sciatica

According to this study the only true and reproducible site of sciatica is nerve root compression. The pain may be produced by either pressure or stretch at the caudal dura, the nerve root sleeve, at the ganglion, or distal to the ganglion.

The study also suggested that only about one third of the participants experienced pain with stimulation on the outer layers of the anulus fibrosus. However, when it did produce pain it always reproduced the patients preoperative low back pain pattern.

Mechanical Dysfunction of the spine causes pelvic pain and organ dysfunction.

Mechanisms of Nerve Root Compression

Intervertebral Disc Derangement
Chemical Stimuli/Irritation
Spinal Canal Stenosis/ IVF Constriction

As concluded in the Williams study printed in the text, *The Origin of Low Back Pain,* "the only tissue in the low back that is capable of producing leg pain is direct pressure or stretch on the inflamed, stretched or compressed nerve root."[23] Several sources of nerve root compression, stretch, and inflammation exist. The discussion that follows will investigate the mechanisms of intervertebral disc derangement, chemical stimuli/irritation, spinal canal stenosis, and various etiologies of intervertebral foramen constriction.

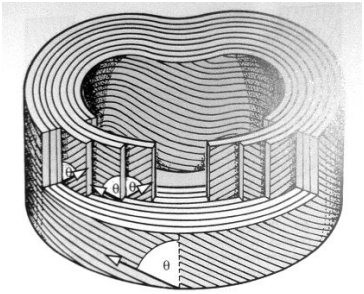
Degeneration of the intervertebral discs, especially in the lumbar spine, which leads to protrusion and eventual nerve root irritation is a well documented. Posterolateral protrusion is the most common type of disc protrusion, and is also most commonly found in the lower lumbar (L4 & L5) and upper sacrum (S1 & S2). These protrusions are responsible for a large number of back and leg complaints seen in clinical practice. However, the lower sacral nerve roots (S2, S3, & S4) will often protrude centrally (medially). These lower sacral nerve roots are the origin for many important pelvic organ functions. It will be important to understand intervertebral disc pathology to make the connection between mechanical dysfunctions the disturbances of bladder, bowel, gynecologic, and sexual function.

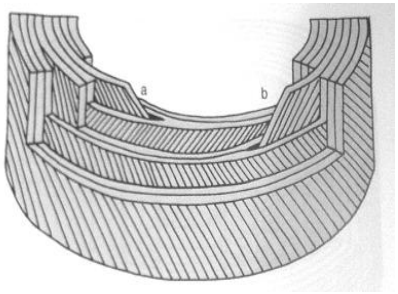
Intervertebral Disc derangement.

> The most exhaustive contribution to the literature on the anatomy and pathology of the intervertebral disc was made by Schmorl, whose incomparable postmortem studies of the spine appeared between 1927 and 1932. In 1929 Dandy reported two cases of nerve root compression caused by extruded cartilaginous fragments of the intervertebral disc. However, it remained for Mixter and Barr in 1934 to report the first removal of herniated disc material, which they initially thought was tumor. They provided, however, the first description of sciatica caused by nerve root compression from disc herniation. Although accurate, that mechanical model was unfortunately accepted for decades as the sole origin for discogenic back pain and nerve root irritation. Studies by Franson et al., as well as other investigators, have shown that other important mechanisms also play a role in the production of radicular and disco-vertebral pain. [79]

During developmental and early adult years, in the absence of any significant trauma or injury to damage the spine, the disc remains anatomically and functionally intact. Over time however, usually beginning about the mid to late teenage years, the anulus begins to sustain internal damage. Lumbar Disc disease begins in adolescence. A study of 12,000 people before the age of 15 demonstrated degenerative disc disease.[81] Another study looking at 20,888 individuals aged 12-41 found that 50% of men and women experienced at least one episode of low back pain by 20. (18 in females and 20 in males.)[40] Low Back pain is the most expensive work injury[13] accounts for 149 million work days lost per year in the United States,[34], and the second most common cause to

industrial absenteeism.[34] One of the first signs of degenerative disc disease is loss of hydration capacity, which decreases IVD pressures of the nucleus and in disc height.[22] Compromise to the anular fibers often occurs. Anular fibers are damaged by tensile stress, compressive, bending, and twisting loads. The fibers of the anulus fibrosus are arranged in layers which are oriented in alternating 45 degree angles to each other.

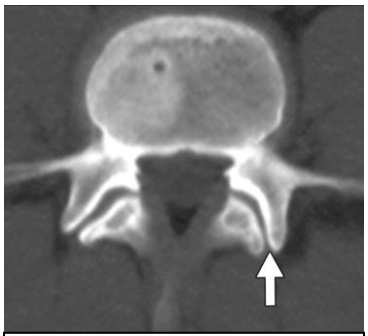




Images from: Clinical Anatomy of the Lumbar Spine and Sacrum. Nikolai Bogduk 4th edition.

Due to this crisscrossing pattern of annular fibers the disc is predisposed to tensile stress, compressive loads, bending, and twisting loads.

The anular fibers are considered to have the same mechanical properties as tendon; thus the stresses required to produce a given deformation and which irreversibly damage the fibers can be predicted. Hickey reported in his Volvo Award winning paper that the maximal amount of rotation that would not damage the anulus was 3.6 degrees.[35] The facet joints of the lumbar spine are designed to withstand such rotational forces, but variations in normal anatomy may predispose the disc to unusual stresses.



Unilateral facet tropism of the lumbar spine.

Facet tropism often occurs unilaterally, which would allow for abnormal wearing of the disc. Depending on the angulations of the affected facet the disc may be allowed to move a few degrees more on one side versus the other. This would create a local area of weakness in the annulus and allow for destruction of

anular fibers.

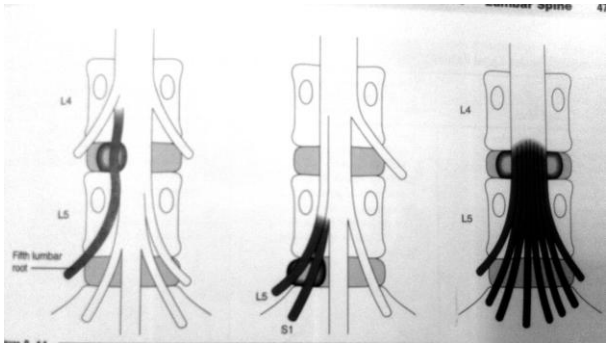
As the spine is exposed to mechanical strain, small cracks and tears (micro-trauma) can begin to develop throughout the weaker inner layers on the anulus. Since the inner layers of the disc don't contain pain fibers the early destruction doesn't become apparent to the patient until the degeneration progresses into the outer layers of the anulus, which are innervated by pain fibers. Once this damage begins to affect the outer pain sensitive layers of the disc the nerve endings can become irritated and cause pain to develop at the region of the spine and back.

As this process of developing micro-trauma progresses to the outer layers of the disc, causing pain in the process, the region of anulus affected gradually become weakened and less capable of restraining and containing the nucleus in the center region of the disc. As a result, under the pressure associated with weight bearing strain (vertically erect spine), which causes the nucleus to push outward against the inner layers of the anulus, the nucleus begins to slowly push into the weakened portion of the anulus and cause a bulge to gradually form. As this occurs, the intensity of the back pain experienced by the individual may become more sever due to the effects of the nucleus pushing back and straining against the outer layers of the anulus and irritating the nerve endings further. [2,16] As reported by Tampier and Moore RJ in their issues of Spine, 'damage to the outer anulus fibrosus results in degenerative disc disease. In 4 months the outer 1/3 of the anulus healed, but the inner 2/3 did not heal. Progressive damage develops with the small cleft inside the 1st layer of anulus. Nuclear material pressed through this cleft will create a fluid filled, delaminated pocket between collagen fibers

with the lamellar bundle. This is the1st stage of damage with observed herniated nucleus pulposus at a microscopic level.[69,47]

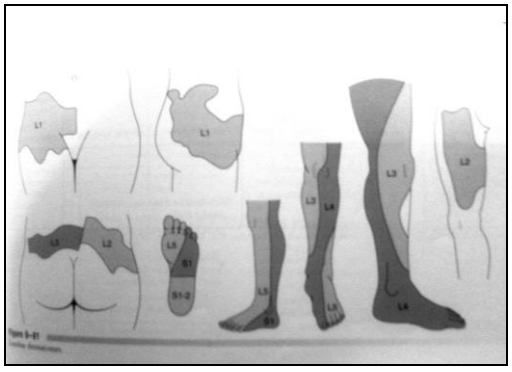
The anulus fibrosus of the intervertebral disc is considered the most pain sensitive tissue in the low back.[11,39,63] Mechanical stimulation of the lumbar discs may not always produce pain, where as inflammatory changes may cause the disc to become sensitive to mechanical stimuli, resulting in nociceptive information being transmitted as discogenic low back pain to the spinal cord through the lumbar sympathetic trunk with a variety of symptoms. The same study also demonstrated that nociception is increased when fissures in the outer anular fibers begin to heal after an initial injury. New nerve growth has been discovered at such anular fissures, which magnifies the pain response should this tissue be injured again.[68] Growth of new nerve fibers provides a morphologic basis for true discogenic pain. However, the responsible mechanism for the penetration of neural structures deeper into the discs is still poorly understood. This is a problem considering after the initial acute episode of low back pain, 25% of patients have a recurrence within 12 months.[2] Moore states in a September 1996 issue of Spine that revascularization leads to resorption of fragments by macrophage, vascular, and fibroblast converting nucleus to scar tissue.[47]

Over time, accumulating micro-trauma weakens the annulus further and allows the bulge to progressively enlarge. At some point the enlarging bulge may begin to come in contact with the nerve roots that pass behind the disc en route to leaving the spine.



(From left to right) Posterolateral disc bulge affecting a single nerve root, multiple nerve roots, and a central disc bulge affecting multiple roots.

As this occurs the nerve roots fibers can easily become irritated from the effects of the enlarging bulge. Once the nerve roots become affected, the symptoms felt by the individual can change from that of primary back pain and stiffness, caused by the damage and bulging occurring in the disc itself, to back pain, as well as leg pain, tingling and numbness, that can develop anywhere along the course of the nerve root being irritated. [16]



Dermatomes associated with lumbar spine nerve root irritation. i.e. disc herniation. [44]

In the area of the low back, the nerve roots that are susceptible to the effects of teh bulge or protrusion that develops in the posterolateral region of the discs are the L4, L5, and S1 nerve roots. These nerves, which contribute to the formation of the sciatic nerve, distribute their fibers to the buttocks, thighs, legs, feet, and toes. As a result, symptoms that develop from irritation or pressure of one or more of these roots can be felt in any or all of these regions. [44]

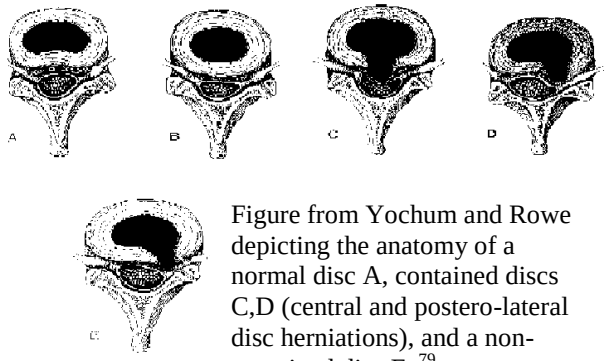
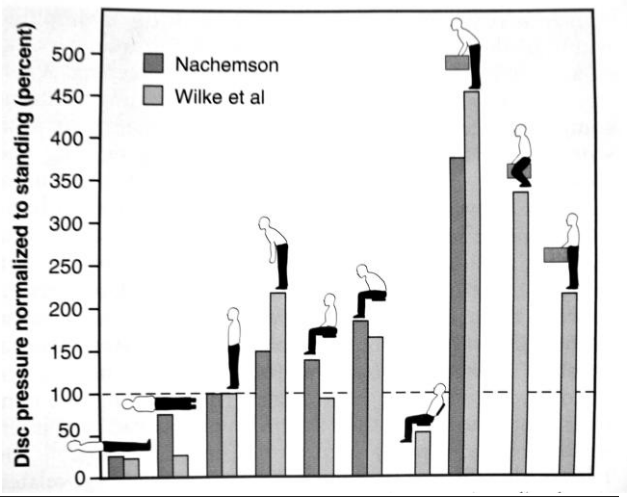


Figure from Yochum and Rowe depicting the anatomy of a normal disc A, contained discs C,D (central and postero-lateral disc herniations), and a non-contained disc E. [79.]

Over the past several decades there have been many interchangeable and often confusing terms to describe disc derangements. Post [57] suggested a clear and succinct classification of three basic types of disc lesions. Patients can suffer from a **bulging disc** (bulging anulus, disc bulge, balloning disc), **Contained disc** anulus, disc bulge, ballooning disc), **contained disc** (protrusion, herniated disc, herniated nucleus pulposus, slipped disc, subligamentous disc herniation) , and **non-contained disc** (prolapse, extruded disc, sequestered disc, fragmented disc, floating disc, free fragment, ruptured disc, amputated disc, wrapped disc, migrated disc).



Mean change in load on L3. Nachemson, Wilke et al. [49]

Independent studies by Nachemson and Wilke et al demonstrate very similar findings looking at mean change in load on L3 disc with various activities, compared with upright posture. As depicted on the chart the highest loads occur in a flexed and loaded position, which puts up to 4.5 times greater stress on the disc compared to an upright standing posture. The second position with the highest pressure on the disc, as depicted in the chart, is when carrying a unilateral load (i.e. carrying a box with both hands and the torso rotated to the right or left and using the

hip to support the load.) The chart depicts the flexion and rotation positions in isolation, which result in the highest strain on the intervertebral disc. The combination of these motions with a load would likely cause significantly higher loads approaching 7-8 times the load of a standing posture. (700-800 percent, which would off the chart in the Nachemson, Wilke el al studies.)

Chemical Stimuli/Irritation

It seems unlikely that discogenic pain is merely generated by mechanical irritation of sensory nociceptive terminals. Chemical stimuli in a degenerated disc have been reported to play a substantial role as well. In this context, the observations of extremely high phospholipase A2 enzyme activity in herniated disc tissue are very interesting.[61] In addition, a wide variety of substances, with the ability to excite or increase the excitability of primary sensory neurons have been reported in the interstitial fluid of the disc. These include prostaglandin E, histamine-like substances, potassium ions, lactic acid, and several polypeptide amines.[12,45,48,71] In this respect, Weinstein et al.[71,72,74,73] emphasized the important role of the dorsal root ganglion, which is located in the intervertebral foramen and serves as warehouse for all kinds of peptides. It is very likely that the dorsal root ganglion has a pain-modulating function around each motion segment.

Normally, the adult human disc is avascular. However, angiogenesis associated with disc disorders is not an uncommon finding. Experimental studies have shown that anular lesions heal by the formation of granulation tissue containing blood vessels.[55] In experimentally injured porcine intervertebral discs, Kääpä et al.[37] even found a dense network of capillaries in the healed anular area 2 weeks to 2 months after operation.

Vascularization of the inner parts of the disc has also been reported in degenerated discs.[76] In the current study, blood vessels were found only in the anterior longitudinal ligaments. They possessed a perivascular nerve network with probably a vasomotor or vasosensory function.

SP-immunoreactive nerves were always found running freely in the supportive connective but not in the vicinity of blood vessels, although SP is known to act as a vasodilator in other tissues.[4,10,41] Conclusive evidence that SP is present in the human intervertebral disc fibers hasn't been well established. More recently, SP activity was demonstrated in the outer 3 mm of the anulus fibrosus of human intervertebral discs.[5] These studies have postulated that it's reasonable to see limited amounts of SP around a relatively healthy disc, and a "painful" disc contains a larger amount of SP given the neovascularity in the inflamed tissue around the injured disc. The relation between painful discs, neurotransmitter distribution, and SP staining needs further elaboration.

In the event of a transligamentous herniated nucleus pulposus (HNP) the patient will usually not have any low back pain anymore since the pressure is off the outer, pain sensitive anular fibers. As the low back pain resolves in this patient they will likely have thigh, leg, and foot pain as the HNP comes into contact with lumbar nerve roots. Some patients experience motor weakness, but this is not an indication for surgery. Only in the case of cauda equina syndrome when the a large HPN, usually a medial lower lumbo-sacral disc, has caused a loss in bladder or bowel function is surgery warranted. 87% of extrusions are treatable without surgery.[60] Even with foot drop the patient doesn't require surgery.[6] A non-contained HNP can be treated without surgery if patient can tolerate symptoms for 2 months.[36] Within that time frame, however, a patient may experience

spontaneous regression by retraction of the HNP back within the boarders of the anular fibers, dehydration of the HNP, or by resorption due to inflammatory reaction.[65] Although the inflammatory process can bring some discomfort to the patient it will help resolve disc material the is extruded into the spinal canal. The patient's choice to undergo steroid injections to relieve the symptoms will cause a suppression in resorption of HNP material.[46] The composition of the nucleus pulposus (NP) is mainly water, but it also contains proteoglycans with will swell and potentially adhere to nerve roots and cause chemical and mechanical stress to that neural tissue. Additionally the proterglycans may cause autoimmune reactions from the increased lactic acid and local pH changes from the HNP. [38]

Spinal Canal Stenosis/ IVF Constriction

Spinal stenosis is slowly progressing condition which is responsible for the most low back surgeries in older adults, and is a major cause of pain and disability. There is no apparent gender bias, and is characterized by a spinal canal circumference of 10mm or less. Lumbar spinal stenosis affects three main areas including the IVF, lateral recess, and spinal canal. It's possible to be born with narrowing at any of these locations, but most cases include acquired spinal stenosis. The most common symptoms associated with lumbar spinal stenosis are neurologic and vascular claudication.

It's possible to differentiate these two symptoms by a series of orthopedic and functional tests. Vascular claudication is usually described as aching, cramping, or tired feeling. It is most evident when activity occurs that increased vascular demand of the tissues, and rest often relieves symptoms quickly. There is generally no night pain or sensory loss, but the hallmark sign is decreased or absent pedal pulses and calf pain.

There are strong correlations between vascular claudication and systemic diseases like diabetes mellitus, hypertension, and ischemic heart disease.

Neurogenic claudication causes compression of the DRG which will diminish reflexes, the patient experiences low back pain which is relieved with flexion, sitting, and squatting. Patients often experience weakness, but pulses are typically normal. The stoop test is used to assess if the patient suffers from neurogenic claudication by requiring the patient to briskly walk for 50 yards and ask about pain in the buttock, and lower limb. To relieve the pain the patient flexes forward. These symptoms may also be relieved when the patient is sitting and forward flexing. A study showed that 42 out of 47 patients with neurogenic claudication experienced pain relief within 2 minutes by sitting, and leaning forward.[80] If flexion does not relieve the symptoms, the test is negative and it's likely the patient has vascular claudication.

Lumbar spinal stenosis associated with bladder dysfunction, interstitial cystitis, and chronic cystitis is well documented in the literature.[26,32,67,19,18,17,27] Details about how stenosis affects patients will be discussed in the next section. Patients are often treated with conservative care at first to include: modalities, meds (NSAIDS/corticosteroids) chiropractic (flexion-distraction, cautions against extension manipulation), epidural injections, active physical therapy/ flexion based exercises. The only indications for surgery are if the patient can not tolerate the pain, progressive neurological symptoms, or if conservative treatment fails.[3]

The PPOD Syndrome

After investigating common somatic pain patterns, the symptoms associated with the PPOD syndrome, the basic involved anatomy, and mechanisms of nerve root irritation, we're finally able to discuss the specifics about the PPOD syndrome from a clinical perspective. The discussion that follows will focus specifically on the development of PPOD syndrome in patients, some basic neurology, and clinical significance of nerve irritation to the previously mentioned lumbo-sacral plexus.

While the most common, posterolateral, disc bulge can be extremely painful for a patient to endure, a medial disc bulge affecting the lower sacral nerve roots (S2,S3, & S4) has the capability of affecting the origin of several extremely important nerves with respect to pelvic organ function.

The lower sacral nerve roots distribute fibers to structures located throughout the pelvis. As mentioned in detail previously, the nerves in this area give rise to the pudendal, pelvic splanchnic and unnamed nerves that supply virtually all motor and sensory input to the muscles of the pelvic floor, integument, and organ functions within the pelvis.[16] It's because of the mechanisms previously discussed (disc herniations, stenosis, etc) that cause abnormal neurologic functions and the production of individual PPOD symptoms. Some of these fibers mediate impulses that function on a conscious level to allow us to be aware of certain sensations or initiate voluntary movement, while others mediate impulses that function completely on a subconscious level to control and regulate the function of the internal organs.[21]

Basic Neurologic Control Systems

Sensory nerve fibers mediate impulses toward the spinal cord and to the brain about the current status of touch, pressure, tension, temperature, and pain of the involved structure. Most of these fibers remain at a subconscious level, which provide feedback to control centers about the functioning of body parts or organs.

Motor nerve fibers mediate impulses that travel away from the brain and spinal cord, which mainly serve to initiate voluntary or involuntary muscular contraction in one form or another.

Both sensory and motor functions must be working in sync in order to maintain proper organ and tissue function. [16] Disruption in either sensory or motor pathways can cause improper information to be transmitted to the spinal cord of brain and prevent the brain and cord from returning appropriate information to the acting structure. The reverse scenario is also possible. A proper signal can be sent to the cord and brain, but the message can be garbled while returning to the target tissue.

These explanations are basic, but essential in understanding the mechanically induced PPOD patient. The following descriptions are common clinical presentations of somatic pain patterns of the pelvis. These presentations are the result of neurologic abnormalities in the sensory-motor arc responsible for keeping organs and tissues working properly.

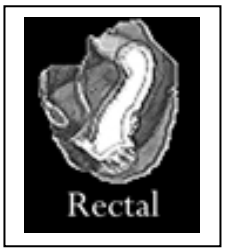
Common Somatic pain patterns of the pelvis.

Inguinal pain- Can be found unilaterally or bilaterally. When found bilaterally the pain tends to be more intense on one side. Most patient will experience some sort of lower extremity pain on the same side of

greatest inguinal involvement. Female patients often describe the pain as coming from the area of the ovary. Male patients are often misdiagnosed with a developing or existing inguinal hernia, which shares a similar pain distribution.

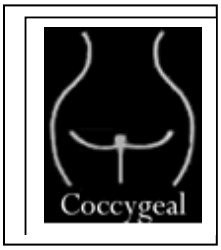
Suprapubic pain- Often found directly immediately above the pubic bone, tends to be superficial abdominal wall pain, and is usually reproduced with light palpation. However, some patients may have an additional complaint of deep suprapubic pain that is aggravated by emptying the bladder and can mimic interstitial cystitis. In some cases the patient will experience burning during urination along the urethra while voiding. This can be caused by a lower bacterial urinary infection, but in the absence of bacteria is often the result of hypersensitivity of the nerve fibers terminating at the bladder neck and urethra. Acidic urine can irritate this mucosa causing sharp or burning pain.

Para-anal- Can be found unilaterally or bilaterally and is usually described as a deep muscular ache. Palpation of the medial gluteal or levator ani muscles often reproduces the pain.



Rectal pain- Can have some variation in its presentation. Some patients have isolated pain near the opening of the anal canal, but others have referred pain higher up into the pelvis. Both pain

distributions tend to be a deep, burning pain, but some patients also experience sharp, abrupt pain that radiates into the rectum. Least likely is the patient that describes a cramping, muscular pain in the rectum, which is then usually associated with other enterologic conditions.



Coccygeal- Is often confined to the area directly over the coccyx, but can also be found in conjunction with Para-anal or rectal pain.

The extent to which a patient will experience these particular referral patterns depends not only on the affected anatomy, but the magnitude to which it is affected. Meaning the symptoms will vary depending on the amount of time and the amount of pressure, stretch, or irritation on the involved nerve roots. An experiment by Triano looked at the magnitude and duration of nerve irritation in mice, and observed that under a consistent amount of pressure for one day the mice experienced a certain level of hyperirritability. Keeping that level of nerve pressure constant the mice demonstrated increased irritation the second day, and even higher irritation by the sixth day. When the magnitude of the nerve irritation was increased the nerve fiber became mechanically deformed, and the mice were observed demonstrating depressed or inhibited function in the affected nerves.[70]

Several experiments on pigs [51,52,53,81] demonstrate that increasing pressure on nerve roots results in variations in symptoms. Over a two hour period 5-10 mmHg of pressure was applied to target nerve roots and resulted in microscopic reductions in blood flow to the arties supplying the corresponding nerve root, but this time period failed to demonstrate any detectable level of decreased nerve function. As the pressure was

increased to 50-75 mmHg for a two hour period a significant reduction in both sensory and motor conduction began to occur. The symptoms remained constant throughout the entire two hour period, but after the pressure was released the symptoms quickly dissipated and nerve function returned to normal. When pressure was increased to 100mmHg of pressure the affected nerves' ability to conduct impulses were significantly reduced. Motor function suffered a 50% reduction in function, while sensory function decreased by 75%. Upon releasing the pressure from the nerve roots motor function returned to normal, but sensory function only rebounded to 57% of it's baseline functional value. At 200mmHg pressure there was an instant reduction in motor and sensory function to 3.9% and 0%. Upon releasing the pressure there was only a 13.8% and 10.2% recovery of nerve root function.

These studies demonstrate a mechanism very similar to that of a lumbar disc herniation with a single insult affecting a single or multiple nerve roots. However, there is another mechanism of injury which has potential to impair the function of an affected nerve root to a much greater degree. An experiment by Olmarker [53] suggests that two points on insult along the course of a single nerve has significantly greater affects than a single insult along the same path. It was reported that with only 10 mmHg pressure at two points along the same nerve caused a 60% reduction in nerve function. Remember that the studies using only single insults to a nerve required nearly 50 mm Hg pressure to reduce nerve function to that level. It's this "double crush" mechanism, such as mild bulging of multiple lumbar discs, that is believed to be the mechanism behind production of the various symptoms found in many PPOD syndrome patients and likely accounts for the "atypical" or "occult" nature of the underlying disorder. [16]

The 'take home' from these experiments is that with a common patient presentation such as multiple mild disc bulges into the spinal canal will affect spinal nerve roots. Macroscopically only a single nerve root may be affected, but within that single nerve root, on a microscopic scale, there is a mix of thousands of motor and sensory nerves. It varies by patient which fibers will be affected, thus a variety of reported symptoms depending on the person. Some patients may have mild sensory fiber involvement and severe motor fiber involvement, while another patient may present in the opposite manner. Either presentation will vary depending on the type of fiber involved, the magnitude of pressure involved, and the length of time the insult has occurred.

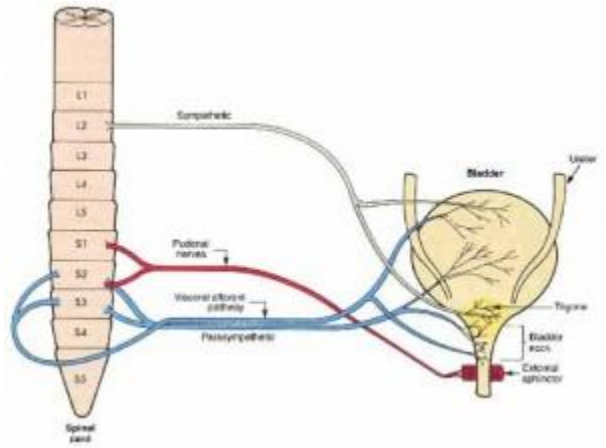
Up until now the discussion has focused on how pain can be generated throughout the pelvic and low back. Although patients will often present with pain symptoms, it is the imbalance in facilitation and inhibition to target tissues which is the root of their bladder, bowel, gynecologic, and sexual dysfunction.[16] A full explanation of the 30 plus symptoms involved in the spectrum of PPOD syndrome patients would be quite lengthy and well beyond the scope of this discussion. A brief look into the mechanism of urologic dysfunction should serve as a foundation for understanding the events behind the production of other pelvic pain and organic dysfunctions.

Normal bladder function involves a gradual filling, which will cause the internal pressure in increase as the walls of the bladder expand. At about 300-400 ml of urine there will sufficient internal pressure to stimulate bladder receptors, which are sensitive to increasing pressure. As the bladder continues to fill the number of receptors being stimulated increases also. At some point there is sufficient stimulation that a signal

reaches the cord and later the brain, giving us the ability to consciously perceive the normal urge to void. As discussed earlier, organ and tissue functions are dictated by both motor and sensory inputs working in sync. The sensory impulse of the bladder filling triggers a companion motor reflex from the brain to the muscular lining of the bladder, and causes it to begin contractions. [16]

This illustration demonstrates the relationship between specific spinal levels and how they correlate to a specific organ.

Insult to these spinal levels may cause a decreased function of the associated organ and prompt the onset of symptoms. This sensory-motor arc continues to sustain bladder contraction and successful bladder voiding. The ability to consciously sense the bladder filling allows people to override the voiding reflex and prevent urine flow until desired voiding. However, at some point the stimulation will be so great that bladder contraction will exceed the urethral sphincter's ability to block urination and spontaneous urination will occur. Overall this normal process occurs automatically without our conscious effort, and only the ability to inhibit this process in conscious. [16]



This illustration demonstrates the relationship between specific spinal levels and how they correlate to a specific organ. Insult to these spinal levels may cause a decreased function of the associated organ and prompt the onset of symptoms.

With this understanding and our knowledge of abnormal neurologic function caused by mechanically induced facilitation and inhibition, let's look at how some of the

symptoms of urologic dysfunction can develop in the mechanically induced PPOD syndrome patient.

Urologic Dysfunction: frequency, urgency, difficulty, & retention.

Paradoxical Urologic Dysfunction
Stress Incontinence in PPOS Patients
Overflow or Paradoxical Incontinence in PPOS Patients
Enuresis
Urinary Retention

Paradoxical Urologic Dysfunction

In normal urologic function the bladder has sensory receptors that are sensitive to increasing bladder filling. If these receptors have hyperactive sensory nerves then it would take a significantly less amount of urine to reach a stimulus level great enough to trigger an impulse to the brain which initiates a muscular contraction in the bladder wall. This would result in an issue with urologic frequency and urgency.

Conversely an inhibition of motor fibers would result in weakness of the bladders contractile muscles despite adequate sensory input. resulting in symptoms of difficulty and retention.

A second mechanism of urinary frequency and urgency exists also. Inhibition of the urethral sphincter would result in an inability to resist the contractile forces of the bladder wall and result in the urge to void more frequently.

Conversely a hyper excitation of the urethral sphincter would result in an inability to of the normal bladder contractions to overcome the urethral sphincter. Some patients require urethral dilation in order to void the bladder. [16]

Stress Incontinence in PPOS Patients

Stress incontinence is often the result of inhibited motor fibers to the urethral sphincter. In this scenario anything that might increase intraabdominal pressure would overpower the weakened urethral sphincter and cause urinary loss. [16]

Overflow or Paradoxical Incontinence in PPOD Patients

In this scenario the patient has an inhibition of the sensory fibers of the bladder filling receptors. The bladder will fill to the point that the urethral sphincter can not withstand the excessive pressure and spontaneous voiding will occur. Since there is such an inhibition of sensory input to the brain the voiding reflex never occurs, and patients often have no sensation of bladder fullness. Some patients can sense suprapubic fullness, which will prompt them to self catheterize several times per day, which also leads to increased risk of urinary tract infections. [16]

Enuresis

Inhibition of both sensory and motor fibers allows a patient to be asleep, which is also a state of maximum muscle relaxation, and their bladder fills to the point it overpowers the urethra sphincter and voiding occurs. Filling is allowed to occur due to inhibited sensory input to the brain, thus a breakdown in voiding reflex, and no conscious perception of bladder filling will wake the patient. Again, inhibited motor fiber allows the urethral sphincter to be overpowered and allow voiding. [16]

Urinary Retention

Sensory inhibition and motor facilitation prevents the voiding reflex from occurring, and prevents the urethral sphincter from relaxing to allow urine to flow freely.

The result is a bladder that has no means to stimulate it's muscular wall to contract and no means of relax the urethral sphincter. These patients often must be catheterized. [16]

By looking at the concepts of abnormal sensory and motor fiber function and applying them to the other structures supplied by the various branches of the pudendal, pelvic splanchnic and unnamed nerves, we can begin to see how mechanical insult to the lower sacral nerve roots caused by a mechanical disorder in the region of the lumbar spine can result in the development of not only wide spread chronic pelvic pain, but also the wide range of disturbances of bladder, bowel, gynecologic, and sexual function that are so commonly found in mechanically induced PPOD syndrome patients. [16]

TREATMENT

Before getting to the specifics of treatment protocols it's important to understand some of the research presently available about Cox flexion distraction. Afterwards a brief overview of Dr. Browning's protocols in treating PPOD patients will be discussed.

Chiropractic is one of the fastest segments of professional health care today. Several adjusting styles exist that deal with the use of mechanical forces on short levers, or combination of short and long levers. Of these methods, Cox flexion distraction has the most peer-reviewed literature to support its claims to provide relief to low back pain patients. The Agency for Health Care Policy and Research practice guideline on acute low back pain in adults indicates that manipulation of the spine suing short or long leverage methods is safe and effective for patients in the first month of acute low back pain symptoms without radiculopathy. The policy says treatment after 1 month is probably safe, but was inconclusive. Overall the policy suggests that a 4 week trial of

care is appropriate for low back pain patients, but should be stopped and a reevaluation performed if no significant improvement is demonstrated. [9,24]

Weber concludes that conservative care for patients with disc herniations for 2-3 months before surgical consideration is reasonable. Patients with signs and symptoms of herniated lumbar discs should be observed before the final decision to go the surgical route is taken. 90% of sciatica patients will get well with months of energetic, nonoperative, conservative care. Surgical indications include cauda equina syndrome, intolerable pain, and/or progressive muscle weakness. Patients presenting with there indications should be recommended for surgical intervention. [78,77]

Although there are a few indications for surgery, Schvartzman and associates state that surgical interventions have the same outcomes as continued conservative care when it comes to herniated lumbar disc patients. Several other sources share this view point, and Dr. Cox states that "the decision to continue with a conservative regimen or go to surgery should ultimately be the choice of the well-informed patient." [9]

Cox distraction adjusting is a form of chiropractic adjustment directed at the intervertebral disc, posterior facet elements, and osseoligamentous canal allowing the following potential benefits:

- Increase the intervertebral disc height so as to remove anular distortion within the pain-sensitive peripheral portion. The anulus fibrosus bulges into the concave side,[14,42,29] or the posterior lordotic curve of the lumbar spine, and distraction under slight traction reduces this protrusion.[20,54]

- Decrease intradiscal pressure with creation of a centripetal force on the protruding nucleus pulposus to allow it to assume its more central position within the anulus fibrosus.[20,58]
- Remove subluxation of the facet articulations and restore physiologic motion to the posterior elements of the vertebral segment.
- Improve posture and locomotion while relieving pain, improving body function, and restoring a state of well-being.

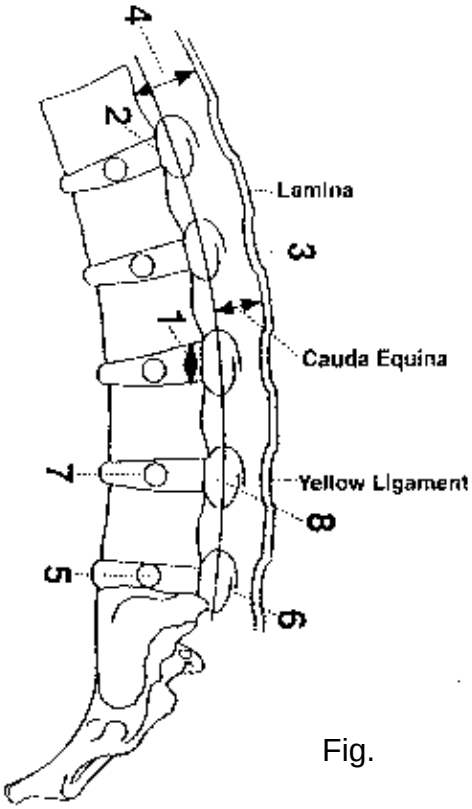


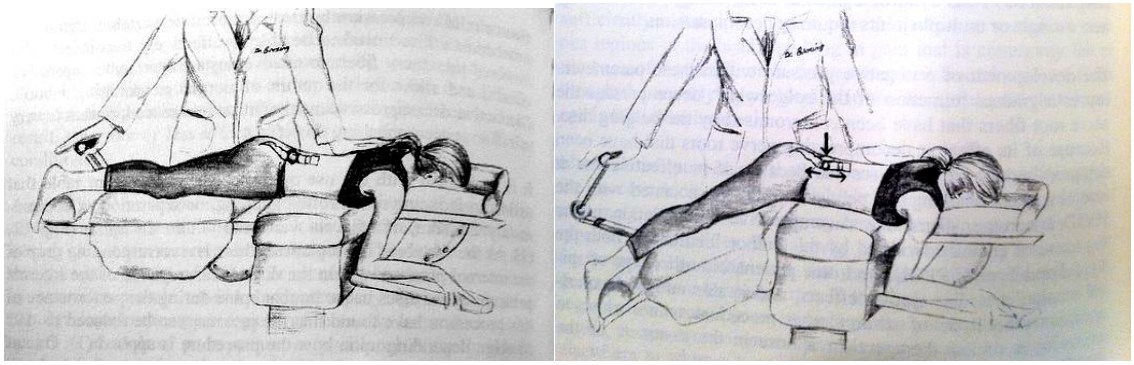
Fig.

1. The posterior disc space increases in height.[29,20,54,25]
2. Flexion decreases disc protrusion and reduces stenosis.[29,20,54,28] (Discs protrude and degenerate into the concavity of a curve, into the side of extension.[42] Thus, reduction of lumbar lordosis reduces disc protrusion and spinal stenosis.
3. Flexion stretches the ligamentum flavum (yellow ligament) to reduce stenosis.[29,54]
4. Flexion opens the vertebral canal by 2 mm (16%) or 3.5 -6 mm more than extension.[62,43]
5. Flexion increases metabolite transport into the disc.[1]
6. Flexion opens the apophysial joints and reduces posterior disc stress. [1,56]
7. The nucleus pulposus doesn't move on flexion.[75] Intradiscal pressure drops under distraction[20] to below 100mm Hg.[58] On extension the nucleus or anulus is seen to protrude posterior into the vertebral canal.[20,28,31,64]
8. Intervertebral foraminal openings enlarge giving patency to the nerve.

Computed tomography (CT) scans during a 45 kg distractive experiment performed in a 1989 issue of Spine by Onel et al demonstrates the regression of herniated nuclear material back into the discal space and withdrawn from the neural foramina. The osseoligamentous canals show increased sagittal diameter during distraction, further reducing canal stenosis.

How to perform flexion distraction.

1. Adjust the table height to allow the patient to easily get on it.
2. Have the patient tighten their abdominals and gluteal muscles and lay down in the prone position.
3. The headpiece should be slightly lowered to patient comfort.
4. The ASIS should be place 2 inches above the split of the table.
5. Perform Tolerance Test to see if the nerve can handle stress – traction the good leg down and hold for 4 seconds, then traction the bad leg down for 4 seconds.
6. If Tolerance Test is negative, cuff ankles to increase the traction.
7. Perform soft tissue tension test to find the new neutral – this is the point at which the interspinous ligament becomes taut.
8. Contact the spinous process of the affected vertebrae, with a thenar contact.
9. Perform 3 sets of 5 distractions in flexion – if the patient's leg pain does not go below the knee, lateral flexion and circumduction may be performed.



Proper hand placement and operation of the Cox flexion distraction table.

Distributions of injuries associated with adjusting techniques and specifically training-related activities at Parker College of Chiropractic, 2006 (student perception of injury sources)

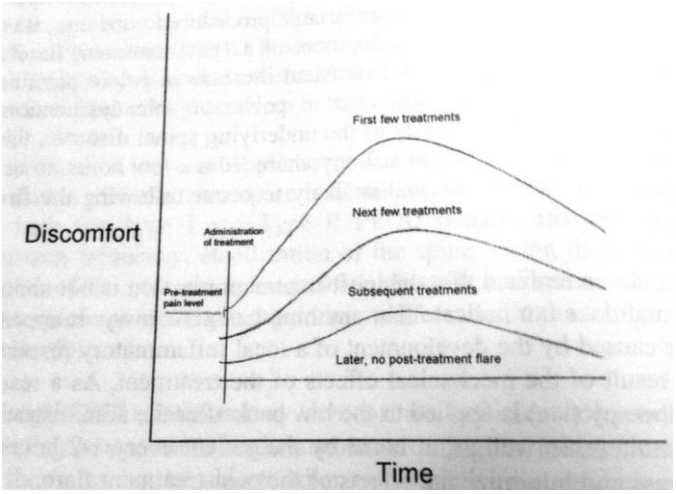
Adjusting Technique	*# of cases*	*# of injuries from adjusting to doctor*	*# of injuries to patient from being adjusted*
Diversified	280	61	74
Thompson	142	3	25
Gonstead	135	31	39
Cox F/D	54	0	1

30

Tailoring Cox to meet the needs of the PPOD patients and protocols.

In the treatment of patients with the PPOD syndrome the type of manipulation must focus on reducing the mechanical irritation or mild compression by one or more intervertebral discs, the procedures utilized need to be able to effectively decompress the involved nerve root fibers in resolve any irritive or compressive effects, and allow for the return of normal neurologic function. Distractive decompressive manipulation is a procedure that is very effective at accomplishing this effect.

A common finding among PPOD patients is the post treatment flare of symptoms that may last one or two days after the initial treatment. Depending on the severity of the underlying spinal disorder, the patient may experience pain for a couple hours or a couple days after treatment. Ice is usually used to help relieve symptoms.



Depicted are response curves observed by Dr. Browning during the treatment of PPOD patients. Initially the discomfort can be quite high after treatment, but over the course of the treatment plan the post treatment flares will often subside.

A key component of the initial treatment of the PPOD patient is the strict recumbency policy. Because of the deleterious effects associated with weight -bearing strain a patient with a mild case (Type1) will remain recumbent for a usually only a couple days. More severe cases may have to remain recumbent for several days.

Another important feature of Dr. Browning's treatment of PPOD patient's is the myofascial component of the treatment plan. Trigger points and fascial torque can complicate the patients presentation, so extensive work on reliving these insults is vital to effective treatment.

The final factor in treating PPOD patient's is the frequency of care. If treatment is terminated too early there is a high incidence of recurrence and can be deleterious to the

patient's condition. In mild cases (Type 1) patients are seen every other day for a couple weeks until post treatment flares subside. These flares are monitored by the patient's feed back on symptom questionnaires. (see Appendix). Once the post treatment flares subside then treatment decreases until the patient is on a stable one every six weeks treatment plan. PPOD patients generally will require some regular life long care at about once every six to eight weeks.

More severe cases (type 2), whose condition is dominated by their organic involvement will be seen daily until the post treatment flares subside. And then a similar protocol will be used as with the type 1 PPOD patient.

Conclusion:

By looking at the concepts of abnormal sensory and motor fiber function and applying them to the other structures supplied by the various branches of the pudendal, pelvic splanchnic and unnamed nerves, we can begin to see how mechanical insult to the lower sacral nerve roots caused by a mechanical disorder in the region of the lumbar spine can result in the development of not only wide spread chronic pelvic pain, but also the wide range of disturbances of bladder, bowel, gynecologic, and sexual function that are so commonly found in mechanically induced PPOD syndrome patients. [16]

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Appendix 1

PPOD SCREENING QUESTIONNAIRE

PELVIC PAIN

- ☐ Pain on one side of the front of the pelvis
- ☐ Pain on both sides of the front of the pelvis
- ☐ Pelvic pain immediately above the pubic bone
- ☐ Pelvic floor pain on one side of the anus
- ☐ Pelvic floor pain on both sides of the anus
- ☐ Pain internally within the rectum
- ☐ Genital pain, numbness or tingling
- ☐ Pelvic, rectal, vaginal or genital pain, numbness or tingling that is aggravated by sitting
- ☐ Laparoscopy without identification of specific cause for pelvic pain
- ☐ Partial or complete hysterectomy without significant relief of pain

Appendectomy, hernia repair, or removal of bladder, testicle or labia without significant relief of pain (click one box for each individual operation that did not provide significant pain relief).

- ☐ Individual Operation 1
- ☐ Individual Operation 2
- ☐ Individual Operation 3

BLADDER DYSFUNCTION

- [ ] Urinary frequency
- [ ] Urinary urgency
- [ ] Urinary dribbling
- [ ] Urinary incontinence
- [ ] Urinary sluggishness or difficulty emptying bladder
- [ ] Urinary retention
- [ ] Recurring urinary tract infections
- [ ] Waking more than twice at night to empty the bladder
- [ ] Wetting the bed
- [ ] Normal urge to void (empty bladder) replaced by a sense of bladder pressure or pelvic distention
- [ ] One bladder surgery for urinary incontinence
- [ ] Two or more bladder surgeries for recurring incontinence

GYNECOLOGIC AND SEXUAL DYSFUNCTION

- [ ] Pelvic pain with intercourse
- [ ] Pelvic pain with intercourse that is felt in the same location as pelvic pain experienced apart from intercourse
- [ ] Impaired, altered or complete inability to achieve normal orgasm
- [ ] Deficient sexual lubrication
- [ ] Loss of libido or sex drive

- [ ] Persistent vaginal discharge

- [ ] Difficulty or inability attaining or maintaining erection

BOWEL DYSFUNCTION

- [ ] Chronic or recurrent constipation

- [ ] Chronic or recurrent diarrhea

- [ ] Alternating constipation and diarrhea

- [ ] Rectal incontinence

- [ ] Painful spasm of the anal sphincter

BACK AND LEG PAIN

- [ ] History of chronic or recurrent low back pain

- [ ] Buttock or leg pain, numbness or tingling that is dominant (most intense) on the same side as dominant accompanying pelvic pain

- [ ] Pain in both buttocks or both legs
