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Examination and Treatment of Low Back Pain with Intervertebral Disc Pathology

Introduction

Low back pain is a worldwide public health issue due to its widespread prevalence and economic impact. It has been estimated that each year, 10-20 percent of Americans experience acute back pain, 2-8 percent experience chronic back pain, 3-4 percent are temporarily disabled, and 1 percent of working-age Americans are permanently disabled from back pain.[1] Low back pain affects almost all Americans during the lifespan (80%), and the recurrence of low back pain is common.[1,2] Of all disabling conditions, low back pain contributes most to disability in working-age populations worldwide.[3] In the United States, it has resulted in a staggering estimated $635 billion spent yearly in cost of health care and lost work productivity.[4] Addressing low back pain has been identified as a national public health priority through Healthy People 2020, and physical therapists can play an important role in its management.[1] This paper will address the physical therapy examination and treatment of low back pain with an emphasis on intervertebral disc pathology.

Intervertebral Disc

The intervertebral disc has three distinct structural regions: the annulus fibrosus, nucleus pulposus, and cartilage end-plates.[5] The annulus fibrosus is composed of fibrous cartilage and 15-25 ring-like lamellae composed of collagen fibers.[5] The collagen fibers are oriented in parallel at a 60 degree angle, and alternate direction in adjacent lamellae.[5] Elastin fibers positioned between lamellae bind adjacent layers, and may help the disc retain its shape after movement.[5] The nucleus pulposus is the gelatinous core of the intervertebral disc. It is composed of randomly oriented collagen fibers, and radially oriented elastin fibers which are contained in an aggrecan

gel.[5] Finally, the cartilage end-plates are composed of hyaline cartilage, are <1 mm thick, and contain and protect the nucleus pulposus. Their collagen fibers run parallel and horizontally and continue into the intervertebral disc. The end-plates are composed of hyaline cartilage, and thus lack vascularity or innervation.[5] The discs have little blood supply, and have some nerves that start in the outer region of the annulus fibrosis and have proprioceptor endings.[5]

The composition of the intervertebral disc includes collagen, the proteoglycan aggrecan, and water. Collagen provides tensile strength, is mainly type I and II collagen, and makes up 70% of the annulus dry weight and 20% of the nucleus dry weight. Aggrecan and water provide hydration to the disc, and are present mostly in the nucleus pulposus. There is a constant breakdown and remodeling of matrix in the intervertebral disc that allows the disc to maintain its mechanical properties.

The intervertebral disc functions to absorb shock and protect adjacent vertebrae. It transmits body weight and muscular forces through the spine.[5] Postures that increase intervertebral disc compression include postures like standing in flexion, carrying a load, or sitting in flexion (see appendix figure 2).[6] Biomechanically, with lumbar flexion, the annulus moves posteriorly, causing disc compression anteriorly and tension posteriorly.[7] The opposite occurs with lumbar extension, in which the nucleus moves anteriorly, causing posterior compression and anterior tension (see appendix figure 3).[7] Due to tapering in the posterior longitudinal ligament, posterolateral disc herniation can occur with lumbar flexion, potentially causing nerve root impingement and pain.[8]

With aging, lamellae fibers become less organized, there is cross-linkage of proteoglycans, and the nucleus loses its gel-like properties and begins to resemble the fibrotic annulus.[5] One cadaveric study suggested that blood supply to the end-plates begins to diminish

early in life during the second decade, initiating degeneration.[9] In typical adult intervertebral discs, more than half of cells are necrotic.[5] Due to degeneration including cell death and tearing that occurs with typical aging, it is challenging to distinguish between a pathologic versus typically aging disc.[5] Typical age-related degeneration in intervertebral disc structure can be visualized in appendix figure 1.[9]

Much is still unknown about the etiology of intervertebral disc degeneration, but a prevalent hypothesis is that limited blood supply results in lack of nutrients and subsequent cell death.[5] Further decreases in blood supply may be caused by atherosclerosis, too little or too much physical activity, or calcification of cartilaginous end plates.[5] These characteristics may be related to why patients with hypertension, smoking, or excess body weight are at higher risk for experiencing lumbar radiculopathy.[2] The mechanisms that occur with disc degeneration are complex. Enzymes break down matrix molecules, causing tearing throughout the disc, especially in the nucleus.[5] There is often a presence of increased innervation and vascularity with disc degeneration, which is associated with chronic back pain.[5] Proteoglycan content decreases, causing decreased osmotic pressure and decreased hydration.[5] Due to decreased hydration, the discs lose height more quickly with loading and do not hydrostatically disperse pressure, resulting in excess focal stress concentration in the end-plate/annulus and pain.[5] These changes influence surrounding structures, which can lead to subsequent pathology. Changes in height cause abnormal loading of facet joints, predisposing individuals with disc degeneration to osteoarthritic changes.[5] Reduced height of the entire spinal column with degeneration, and therefore less tension on the ligamentum flavum, causes thickening and bulging of this ligament into the spinal canal, leading to stenosis.[5]

Degenerated discs are often associated with intervertebral disc herniation. Because only very large forces greater than those imposed with physiologic loading cause rupture in healthy intervertebral discs, it has been inferred that disc degeneration is a precursor to herniation.[5] One likely mechanism of herniation is that portions of the nucleus pulposus move through fissures in degenerated annulus fibrosus with loading.[5] It is important to note that many people with disc bulging on nerve roots do not experience pain, so there must be other mechanisms that contribute to pain.[5]

Clinical Examination

Although few patients who present to physical therapy experience low back pain due to serious medical pathology, it is important to screen for serious conditions that could present as low back pain. Potential conditions requiring referral could include but are not limited to tumor, cauda equina syndrome, infection, compression fracture, and abdominal aneurysm.[2] Key indicators of back-related tumor upon clinical examination are reports of constant pain that is worse at night and unaffected with positional change, age >50, history of cancer, failure to improve with conservative treatment, unexplained weight loss, and no pain relief with bed rest.[2] Signs of cauda equina syndrome include bowel and bladder changes like urine retention and fecal incontinence, saddle anesthesia, and sensory/motor deficits in the L4-S1 distribution.[2] A back-related infection could be indicated by reports of recent infection, IV drug use, immunosuppressive disorder, constant deep pain, spinal rigidity, and fever.[2] Spinal compression fracture should be suspected with history of major trauma, age >50, prolonged corticosteroid use, point tenderness, and increased pain with weight bearing. Finally, serious abdominal aortic aneurysm (≥4 cm) could be indicated by risk factors such as smoking, family history, peripheral vascular disease, diabetes, coronary artery disease, age >70, minority race, female; and objective

signs like abdominal girth <100 cm or palpation of abdominal aortic pulse that is 4-5 cm or greater.[2] A single red flag for these conditions does not warrant referral, but in the presence of multiple red flags and suspicion of a more serious condition, the patient should be referred to an appropriate medical provider.[2] Additionally, if a patient presents with paresthesia, weakness, and signs of CNS disorder, a thorough neurological exam is warranted.[2]

Clinicians should also be aware of potential yellow flags that are associated with poor prognosis and disability from low back pain. Symptom-related yellow flags include previous episodes of pain, pain of high intensity, and presence of leg pain.[2,10] Lifestyle factors include high body mass, smoking, and little physical activity.[10] Psychological indicators include depression, catastrophizing, and fear avoidance beliefs.[2,10] Finally, social factors include higher physical workloads, lower educational attainment, compensation for work injury, and poor work satisfaction.[10] Although not all of these yellow flags are modifiable, physical therapists should screen for and address modifiable yellow flags through direct intervention or referral.

When interpreting imaging findings, clinicians should be aware that pathological changes appearing on imaging are often not correlated with pain. There is a high likelihood of false positives for low back pain on CT, MRI, and myelography.[2] Savage et al found that only 47% of subjects with low back pain had abnormal imaging findings, whereas 32% of subjects without low back pain had abnormal imaging findings; therefore imaging should be interpreted with caution.[11] Many patients without lumbar radiculopathy (between 20 to 76%) show signs of herniated disc on imaging.[2] Lumbar MRI is most helpful to detect suspected pathology such as a fracture or tumor, or when patients are planning for surgery.[2] Regular clinical imaging without sufficient need can cause more harm than good, and is advised against by the American College of Physicians.[12]

Clinical presentation of lumbar disc degeneration or herniation with low back pain is heterogeneous. Common signs and symptoms include radiculopathy, weakness, and pain produced with movement.[13] Due to biomechanical behavior of intervertebral discs with sagittal plane motions as described in the section above, flexion often exacerbates symptoms.[13] Clinical examination should serve to determine specific functional deficits for each individual patient.

Many self-report outcome measures are validated clinical tools that can be used to assess key domains of pain, function, work disability, health status, and patient satisfaction.[2] The Medical Outcomes Survey Short-Form-36 (SF-36) can be used to evaluate health and patient response to treatment in a general, comprehensive manner.[14] However, it may be less specific to certain patient populations and does not measure change specifically in the low back.[2] The Oswestry Disability Index includes 10 items that describe pain intensity, personal care, lifting, walking, sitting, standing, sleeping, sex, social, and travel.[15] Its psychometric properties have been extensively studied, showing an MCID of 12.8 points (of total 100), adequate to excellent test-retest reliability, and variable construct validity but excellent correlation with the visual analog scale.[15] The Roland-Morris Disability questionnaire has 24 items that assess the impact of pain on social activity, functional movement, activities of daily living, and wellness such as mood, appetite, and sleep.[16] Varying MCIDs have been established, including 5-7 points (of total 24) or 30% change in score from baseline.[16] It has strong construct and criterion validity, and has strong inter-rater and intra-rater reliability of $r = 0.83$.[16] The patient-reported Visual Analog Scale (VAS) or the Numeric Pain Rating Scale (NPRS) may be used in conjunction with these self-report measures to specifically assess pain.[2] The VAS MCID is 15 of 100 mm, and the NPRS MCID is 2 of 10 points.[17,18]

In addition to administering outcome measures related to back pain and function, psychosocial health should be considered. Using a holistic approach to care, every patient with low back pain should be screened for depression and referred to a mental health provider as necessary.[2] Although clinicians may assume that they can detect patient depression based on judgment, a formal screen should be administered because clinical impression is not sufficient to detect depression.[19] Depression is especially prevalent in people with chronic back pain, and is associated with poorer clinical outcomes.[2,10] The Patient Health Questionnaire-2 is a very brief measure that asks the patient, "over the past two weeks, how often have you been bothered by any of the following problems?"[20] The patient then responds to two items, "little interest or pleasure in doing things," and "feeling down, depressed, or hopeless."[20] The scores range from 0 to 6, with a cut point of 3 indicating depression is likely. Two other measures to assess factors associated with poor prognosis[10] are the Fear-Avoidance Beliefs Questionnaire and Pain Catastrophizing Scale.[2]

Objective examination techniques may include a variety of tests and measures corresponding to subjective history. Some recommended measures in the APTA clinical practice guideline on low back pain include lumbar active range of motion, segmental mobility testing, centralization testing, the prone instability test, straight leg raise, slump test, trunk muscle strength, and passive hip motions.[2] This is not an all-inclusive list of measures, but can be used as a clinical tool box of some available examination methods.

Lumbar active flexion, extension, and side bending range of motion as measured with an inclinometer can be measured, which correlated with radiographic measurements and had high interrater reliability.[2]

Segmental spinal mobility can be assessed by performing a posterior-anterior mobilization on each spinous process of the lower thoracic and lumbar spine with the patient in prone (appendix figure 4).[2,21] This tool can be used to assess for pain provocation and amount of movement (normal, hypermobile, or hypomobile) based on clinician judgement. Clinicians should be aware that categorization of spinal motion (k = 0.38-0.48) and pain provocation (k = 0.25-0.55) have weak to moderate reliability.[22,23,24]

When assessing presence of pain centralization/peripheralization, therapists should determine a baseline location of symptoms.[2] Centralization is defined as symptoms that become more proximally located after movement, whereas peripheralization refers to symptoms that become more distally located.[2] Repeated active movements of flexion, extension, and lateral trunk shifts should be performed to determine presence of directional preference, with the clinician applying overpressure if centralization is present.[2] Interrater reliability for detecting centralization is k = 0.70 for a new therapist and k = 0.90 for an experienced therapist.[25,26]

The prone instability test involves the patient lying prone on the table, but with "legs over the edge and feet resting on the floor."[2] The therapist first performs a posterior-anterior mobilization to the lower lumbar spinous processes in this position, then repeats the mobilizations with the patient lifting both legs off the floor (appendix figure 5).[2,27]A positive test is indicated by pain reproduced in the first position but reduced in the second; whereas a negative test is indicated by no pain provocation or no change in pain.[2] This test has good interrater reliability (k = 0.87) but is better used as part of a comprehensive examination, not independently.[2.28]

The straight leg raise test is performed with the patient supine, and the therapist passively flexes the patient's hip with an extended knee (appendix figure 6).[2,] A positive sign is indicated

by concordant radiating or radicular lower extremity pain.[2] This test can identify patients with radiating pain in a dermatomal distribution and assess peripheral nerve mobility.[2] The test has fair reliability (k = 0.68) for patients with radiating leg symptoms.[29]

Trunk muscle performance, including of the trunk flexors, trunk extensors, hip abductors, and hip extensors should be assessed. Trunk flexor musculature can be examined with the patient supine and the therapist lifting the patient's straight legs until the sacrum elevates.[2] The patient then actively and slowly lowers the legs to the table while maintaining low back contact with the table.[2] The test is terminated when the patient can no longer maintain low back contact.[2] This test could be used to measure progress, since cut points of >50 degrees hip flexion for males and >60 degrees for females at test termination were able to distinguish patients with chronic low back pain.[30] Trunk extensor endurance should be measured, with the patient in prone and actively raising chest up to 30 degrees for as long as possible. Males who hold the position for less than 31 seconds and females who hold the position for less than 33 seconds are at risk for back pain. The extension measure has good reliability (ICC = .89-.90).[31] Hip abductor muscle testing, and glute bridge or hip extensor muscle testing should also be performed to assess muscular performance about the hip.[2]

Passive hip motions should be assessed. Hip internal and external rotation can be measured in prone or short sitting, ensuring stabilization of the pelvis.[2] Hip flexion should be measured with the patient supine.[2] Hip extension can be measured passively through the Thomas test position by measuring the angle between the femur and trunk, or in prone.[2] Other objective examination measures should be performed based on individual patient presentation. The therapist may wish to observe functional movements that provide information beyond body structure and function levels, such as gait or lifting mechanics.

Treatment

In patients with low back pain resulting from intervertebral disc pathology, further injury should be prevented. Overall health should be addressed with attention to factors that can modify therapy prognosis.[10] Such factors may be modified by screening for and referring for psychological contributors to pain, encouraging active lifestyle habits to address lack of physical activity and excess body weight, and smoking cessation counseling. High-intensity aerobic exercise should be performed by patients with chronic low back pain.[2] For patients with generalized pain due to central sensitization, or pain resulting from increased neural sensitivity with a lack of tissue damage, lower intensity, progressive exercise should be incorporated.[2] Undesirable postures that increase disc compression should be minimized, such as carrying loads with spine flexed, or sitting in flexed and slumped positions.[6]

A variety of conservative physical therapy treatment options exist for patients with low back pain. Evidence-based interventions may include manual therapy, strengthening, directional preference exercises, nerve mobilization, education and counseling, and general endurance exercise.[2] Treatment should be selected based on low back pain classification[2] instead of anatomic characteristics due to heterogeneity in clinical presentation of patients with intervertebral disc pathology.[11] A variety of classification systems for low back pain have been proposed, but the one proposed by the APTA categorizes types of pain with qualifying characteristics like mobility deficits, movement coordination, lower extremity pain, radiating pain, generalized pain, cognitive or affective tendencies, and acuity level.[2] Patient progress should be continuously evaluated throughout treatment, with intervention strategies modified as needed.[2]

Manual therapy does not yield significant treatment effects in all patients, but is best for certain subgroups and when used in combination with other treatments.[2] Spinal thrust manipulation, such as general lumbopelvic manipulation or side-lying rotational manipulation, is beneficial for patients with "duration of symptoms less than 16 days and no symptoms distal to the knee."[32,33,34] Patients who meet this criteria may benefit from manipulation combined with exercise to a greater extent than patients who receive exercise treatment alone.[32]

Motor control, stabilizing, and strengthening exercises for the trunk muscles are also supported by evidence in randomized controlled trials.[35,36,37,38] Patients with at least 3 of the following: "age less than 40 years" with "positive prone instability test, presence of aberrant movements with motion testing, and straight leg raise greater than 91 degrees" are likely to benefit from stability exercises based on a clinical prediction rule.[36] Multifidus and transversus abdominis motor control exercises appear to be modestly more beneficial than modalities in improving activity and recovery for patients with chronic low back pain.[37] There is significantly less recurrence of first-episode low back pain for patients who perform multifidus and transversus abdominis exercises for 4 weeks in comparison to solely receiving advice and medication (30% vs. 84% at 1 year; 35% vs. 75% at 3 years).[38]

McKenzie therapy exercises for patients with pain centralization and directional preference can also be an effective therapeutic intervention.[2] These exercises involve repeated movements in a specific direction that is determined by response to treatment or examination (appendix figure 7).[39] It is theorized that centralization responses result from relocation of herniated intervertebral disc.[39] Patients with centralization of symptoms tend to have good prognosis, and it was determined in a meta-analysis that many patients fall into this category (70% of patients with subacute pain, 52% of patients with chronic pain).[40] Patients should be

treated with an approach that matches their directional preference; for example, patients with an extension directional preference should be treated with extension exercises.[41] These interventions may be limited to short-term benefits, as a systematic review with meta-analysis showed that McKenzie therapy showed superior short-term outcomes, but that simple advice to continue physical activity resulted in better outcomes than McKenzie treatment at 12 week follow-up.[42]

Evidence for lower-quarter nerve mobilization is generally of lesser quality than for the above interventions, but still has potentially beneficial treatment effects. Patients who do not exhibit centralization but do have low back pain with a positive slump test and symptoms distal to the gluteus fold benefit from exercise and stretching in the slump position.[43]

Patient education and counseling with the goal of reducing fear of movement and maintaining physical activity levels should be implemented.[2] Patient education should include advice to remain active and should frame recovery in a positive approach rather than describing specific anatomic pathology.[44,45] Patients should be encouraged about the strength of their spine, educated on pain neuroscience principles, good prognosis for low back pain, active coping strategies, early return to functional activity, and the significance of bodily function over pain relief.[2]

Patients with low back pain may also consider more invasive approaches to pain management. Transforaminal epidural steroid injections combined with McKenzie therapy may be sufficient to prevent need for surgery for herniated lumbar discs, but patients should be discouraged from receiving repeated steroid injections without active therapy.[46] Spinal fusion surgery is not recommended because although it limits unstable motion, it shifts forces to adjacent levels and can lead to future need for further surgery.[47] Microdiscectomy with or without radicular decompression is effective in reducing pain, but may not be necessary for

many patients who can successfully manage pain with advice and other conservative approaches.[47]

In conclusion, low back pain caused by intervertebral disc pathology is a prevalent condition that results in significant economic impact. Physical therapists can play a crucial role in management of low back pain through understanding of intervertebral disc anatomy and evidence-based examination and treatment principles.

Appendix

Figure 1: Age-related changes in intervertebral disc structure[9]

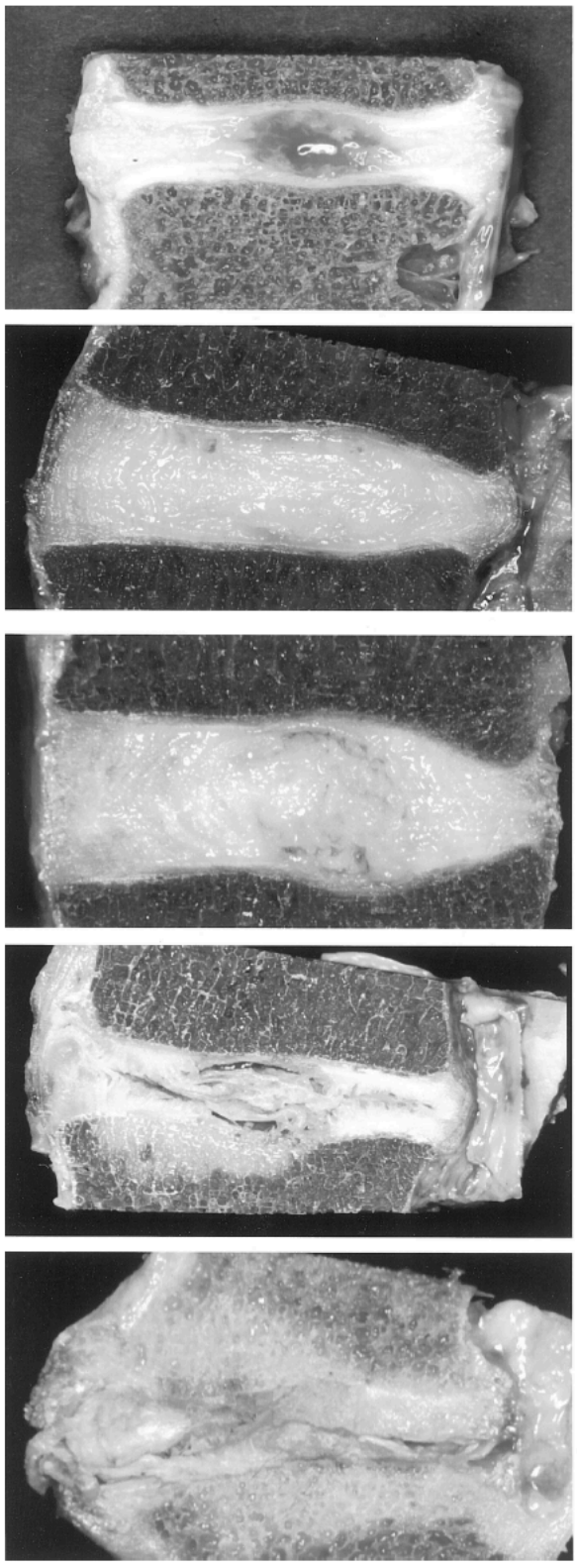

Figure 2: Disc Pressure with Postural Changes[6]

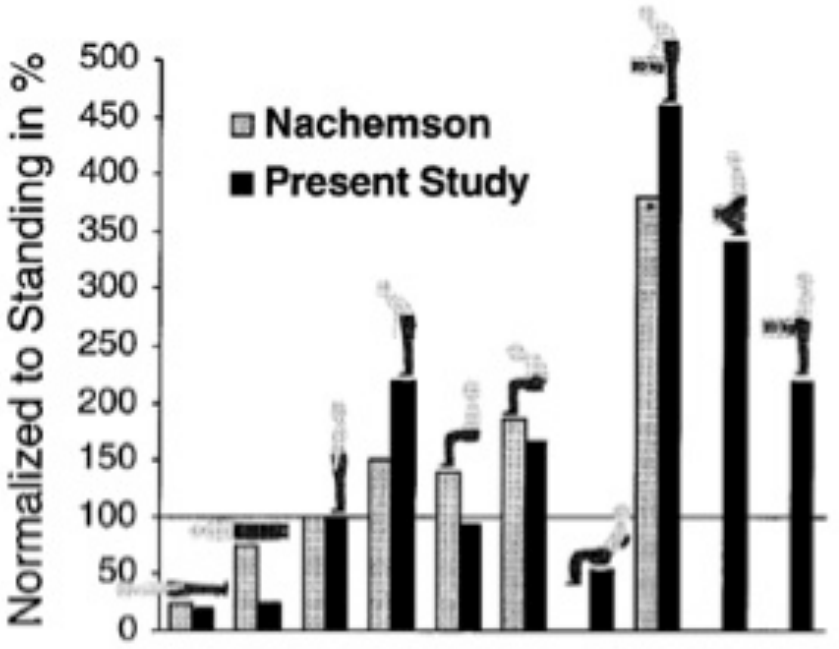

Figure 11. A comparison between data of Nachemson[17,19] and those of the current study (both for 70-kg individuals) regarding intradiscal pressure in common postures and activities, normalized to standing. Lifting weight = 20 kg in the current study; *lifting weight = 10 kg in Nachemson study.

Figure 3: Disc Movement with Lumbar Extension and Flexion[7]

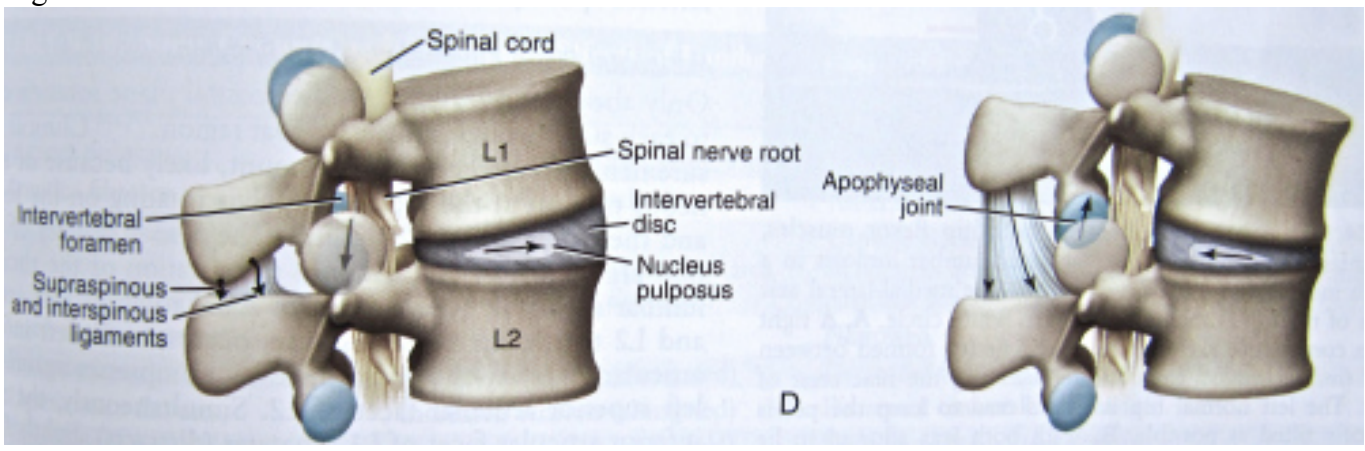

Figure 4: Segmental Spinal Mobility Examination[21]

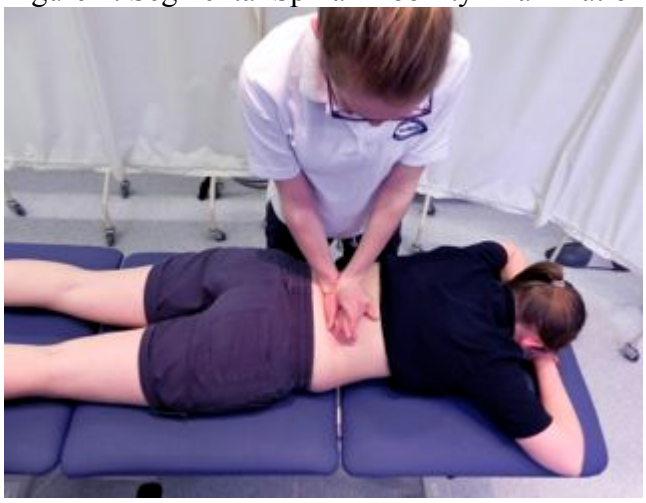

Figure 5: Prone Instability Test[27]

Figure 6: Slump Test[29]

Figure 7: Examples of Directional Preference/McKenzie Exercises[39]

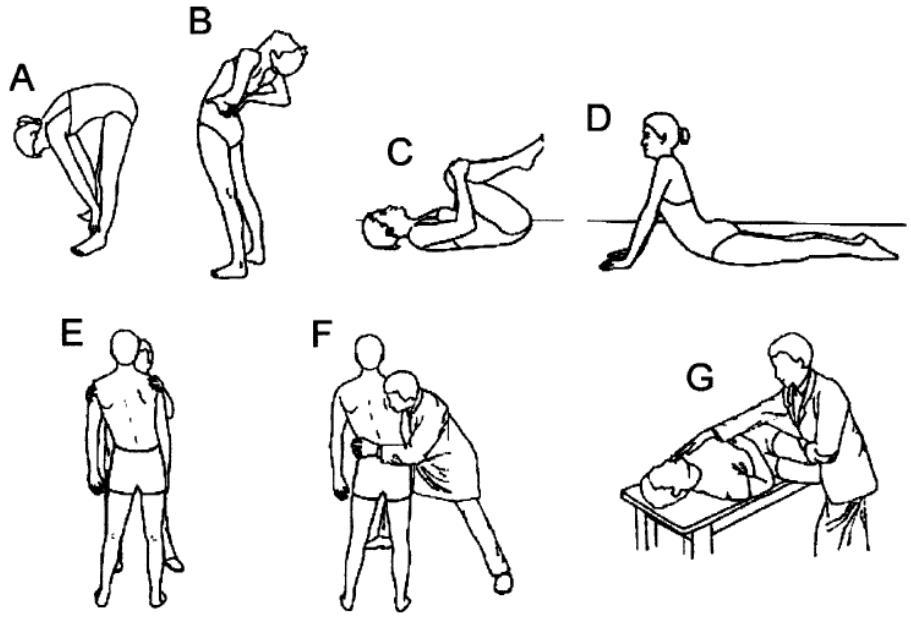

Fig. 1. Standing and recumbent directional lumbar test movements performed repeatedly to the patient's available end-range: (A) Flexion while standing. (B) Extension while standing. (C) Flexion while lying. (D) Extension while lying. (E) Side-gliding while standing. (F) Side-gliding with over-pressure. (G) Flexion-rotation with overpressure. During testing, changes in patient's pain location are continuously monitored in order to identify a directional "preference" (pain centralizes or is abolished) and directional "vulnerabilities" (pain is produced or peripheralizes). A direction of testing that centralizes or abolishes pain is continued and defines a patient's direction of treatment, and those directions that produce or peripheralize pain are temporarily avoided.

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