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The Intervertebral Disc and Low Back Pain

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THE INTERVERTEBRAL DISC AND LOW BACK PAIN

by

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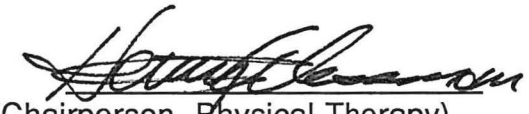

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ABSTRACT

Low back pain has an enormous socioeconomic impact in this country. Even with advancement in diagnostic technology, the incidence and severity of low back pain continues to increase. The intervertebral disc plays an important causative role in the production of low back pain. The intervertebral disc may cause direct discogenic pain by mechanical and/or chemical irritation of the nociceptor receptors found within the outer one-third of the annulus fibrosis, or cause back pain by an indirect method. The incidence of low back pain is first reported around the age of 25 and is most prevalent from ages 35-60. It is during this time that the intervertebral disc is in its semi-fluid state and possesses high intradiskal pressure.

The results of this research of the literature suggest that it is crucial to maintain the integrity of the intervertebral disc to prevent low back pain. This is done by avoiding the positions and activities that increase intradiskal pressure, and by an adequate exercise routine consisting of walking and isometric trunk strengthening.

CHAPTER I

INTRODUCTION

Historical Perspective

Low back pain, with or without leg pain, is not new. Jacob, in Genesis 32, was the first person reported to suffer from sciatica.[1] Since that time the incidence and disabling effect of low back pain has greatly increased, attracting significant investigation as to possible cause.

Vesalius in 1555 and Cotungno in 1765 first explained sciatica as a result of a change in cerebrospinal fluid.[1] Forst[1] related sciatica to a inflammatory reaction following chronic neuralgia. Laseque[1] described the straight leg test, as related to sciatica in 1880. In 1857, Virchow[2] described what is now known as a disc prolapse.

In the early 1900's, Schmorl[3] identified nucleus pulposus herniation through the bony end plate into the cancellous vertebral body. Posterior disc displacement was identified as a cause of low back pain in 1911 by Goldthwait.[3] In 1929, both Alagomaninte and Dandy reported the removal of a "enchondroma" in patients with sciatica.[3] In 1932, Barr[4] subjected the material removed to pathological studies and found that the enchondroma was in reality disc hernia. Since then, major advances in both diagnosis and treatment of discogenic low back pain have occurred.

Epidemiology of Low Back Pain

Despite increased technology, low back pain and disability resulting from low back pain continues to escalate. Between 1971 and 1981 in the United States, the number of persons disabled with low back pain increased to 14 times that of the growth population.[5] Currently in the United States, there are 5.2 million persons disabled by low back pain, one half of whom are temporarily disabled, and one half of whom are chronically disabled.[6] At any given time an additional 9 million[7] are impaired in the U.S.; low back pain is the most common cause of disability in those aged less than 45 years.[8]

Another measure of the magnitude of the problem of low back pain is the annual incidence and point prevalence. At any given time, between 12.2% and 52% of the population indicate they are experiencing back pain.[7] Recurrence of symptoms has been reported in as many as 85% of patients,[9] and in as few as 60%.[10]

Epidemiology of Sciatica

The lifetime prevalence of sciatica is stated by some authors[11,12] to be as high as 40%. Other studies have yielded a smaller prevalence. Hirsch and associates[13] found that 13.8% of women in their study had experienced sciatica. This figure compares favorably with the 11% lifetime prevalence reported by Gyntelberg[14] in Denmark. The majority of these patients had involvement of either the L_{4-5} or L_5-S_1 disc, although a proportionately higher incidence of L_{3-4} disc herniation was identified in the older population.[15] The level of disc

herniation moves cephalad with increasing age, but more than 98% of disc herniations occur in the lowest three disc spaces (15) of L_{3-4}, L_{4-5}, or L_5-S_1.

The natural history of patients with sciatica favors recovery.[12] No more than 5 to 10 percent of patients with unrelenting sciatica eventually require surgery.[16] This corresponds to the natural history of all patients with low back pain. After three months of low back pain, only 5 percent of patients have persisting symptoms, yet it is this population that accounts for 85% of the costs in terms of compensation and loss of work due to low back pain.[11,17,18]

The Socio-Economic Impact

The National Center for Health Statistics reports that 14.3% of all new patient visits to physicians are for low back complaints.[19] Annually, 12.9 million visits are made for chronic low back pain, and 4.114 million are for back symptoms.[19] Orthopedists see 15% of these patients.[19] In addition to physicians, chiropractors report 50 million office visits per year for back complaints,[19] and Physical Therapist's report an additional 5.2 million visits per year.[19] Symptoms severe enough to require hospitalization account for 2.8% of all hospital discharges in the United States.[1] The cost of low back pain has been reported to range from 15 to 50 billion dollars per year.[17] Back pain is the single greatest cause of compensable injury in the working age population, and the second most common cause of work loss time.

Three questions are appropriate here: How can a self limiting disease have such a profound socioeconomic impact? How can a self-limiting disease

disable 5.4 million Americans? How can a self-limiting disease actually reach epidemic proportions despite a prolific increase in knowledge and diagnostic equipment?

Uncertainty with Diagnosis

The answer to these questions may lie in part in the fact that only 10 to 20 percent of patients suffering from low back pain can be given a precise pathoanatomical diagnosis.[3,20] The most common diagnoses given are nonspecific, such as strain or sprains. There has been an increase in diagnoses, such as bulging discs, spondylolythesis, and muscle tear, with the advent of computerized tomography and magnetic resonance imaging. However, the problem with these imaging studies is they do not reveal the source of pain, but rather identify possible structural abnormalities thought to be consistent with the pain complaints. In other words, imaging studies do not distinguish between symptomatic and asymptomatic abnormalities. The other major problem in making a diagnosis is that several quite distinct lesions commonly yield much the same symptom complex. Other lesions that typically produce quite characteristic symptoms and signs may on occasion present in a quite atypical way.

Structures involved in Low Back Pain

Pain is the most common of all clinical symptoms encountered in medical and surgical practice.[21] No matter where it is felt in the body or what the etiology, it is always an expression of a disturbance of neurological function.

Therefore, the pain arising from the low back must originate in the structure that is innervated. The pain producing structures in the low back are: 1) paraspinal musculature; 2) facet joints; 3) spinal ligaments; 4) annulus fibrosis; and 5) neurological structures, such as the dura mater and nerve roots.[21]

To what extent is the intervertebral disc involved in the production of low back pain? According to Nachemson,[22] the intervertebral disc is the central structure in the understanding of low back pain. He offers the following reasons as indirect proof:

1) Disc hernia is usually preceded by one or more attacks of low back pain.
2) Following intradiscal injection of either hypertonic saline or contrast media, it is often possible, in patients with complaints as well as in symptom free subjects, to artificially cause the same type of pain as that which occurs naturally.
3) Investigations have been performed in which thin nylon threads were surgically fastened to various structures and around the nerve root. Three to four weeks after surgery these structures were irritated by pulling on the threads, but pain resembling that which the patient had experienced previously could be registered only from the outer part of the annulus and the nerve root.
4) Pathoanatomically radiating ruptures are known to occur in the posterior part of the annulus, reaching out toward the areas in

which naked nerve endings are located. The presence of such single ruptures in the lumbar disc are first seen around age 25, the same age at which the low back pain syndrome becomes clinically important. Various theories exist as to how these ruptures conceivably elicit pain.

5) Of all the structures that theoretically could be involved in the pain process, only the discs shows any changes that could account for the anatomic changes at such an early age. Such changes in other structures in the region generally show up much later in life and then as a rule only secondary to sever disc degeneration.

6) Although a late sign, disc degeneration as noted on radiograms in patients between 50 and 60 years old is seen significantly more often in those who have had back pain than those who have not.

The purpose of this independent study report is to thoroughly investigate the literature relating to the intervertebral disc in search of clues that may indicate a causal relationship between pathoanatomical changes in the intervertebral disc and low back pain.

CHAPTER II

STRUCTURE, FUNCTION, AND COMPONENT PARTS OF THE INTERVERTEBRAL DISC

Embryology

The developing embryo is composed of three germinal layers. Specific tissue will be derived from these three primary germ layers. The ectoderm develops into the epidermis and its appendages and nervous system. The mesoderm develops into connective tissue, muscle tissue, bone, and tissues of the vascular and lymphatic systems. The endoderm develops into the epithelial lining of the digestive tract.

The longitudinal neural groove forms between two neural crests progressing cephalo-caudally. Fusion of this crest results in neural tube formation by the 29th embryonic day.[3] The neural tube gradually differentiates into nerve tissue becoming the spinal cord and peripheral nerves. The notochordal plate develops from the endoderm ventral to the neural groove. The vertebral column develops in the embryonic mesoderm at four weeks.[23] The individual vertebrae develop under the combined inductive influence of the notochord and neural tube by migration of the sclerotome cells which subsequently undergo differentiation into chondrocytes.[24] Between the vertebrae, the notochord expands as cells within a proteoglycan matrix forming the nucleus pulposus.[23] The nucleus is then surrounded by the annulus

fibrosus which is derived from the perichordal mesenchyme.[23] Together these two structures constitute the embryonic intervertebral disc.

Anatomy

The human spine contains 23 intervertebral discs.[24] The lumber discs are larger than the cervical and thoracic discs, but each have the same general anatomy. The Intervertebral disc consist of three parts: the nucleus pulposus, the annulus fibrosis and the cartilaginous end plates.

The nucleus pulposus is semi-gelatinous containing about 80% water and ground substance consisting of collagen and protein polysaccharide.[25] Because of this high fluid content, the nucleus distributes pressure evenly in all directions to the annulus and end-plates.[24] In the infant or young child, the nucleus appears more rectangular; whereas in the adult, it is variable in shape ranging from oval to biocular.[23] The positioning of the nucleus pulposus varies from cervical to lumbar, being centrally located in cervical discs and posteriorly located for lower lumbar discs.[23] There is a transitional zone between the nucleus pulposus and annulus fibrosis which represents the growth plate of the nucleus pulposus and is similar to epiphysial growth plates.[23]

The annulus fibrosis consists of concentric lamella of highly oriented collagen fibers which encapsulate the nucleus pulposus.[24] The fibers of each layer are parallel and run spirally at an angle of 45° to the bodies of the vertebrae, and the fibers of alternate layers are at right angles to each other.[25] This criss-cross arrangement of fibers resists torsional and flexional deformity

and ensures resistance to rupture of the annulus.[25] The fibrous lamellae are closely packed anteriorly and posteriorly, but much less so laterally.[23] The annular rings are firmly attached superiorly and inferiorly to adjacent vertebral bodies and the vertebral end-plates and serve to maintain the nucleus under constant pressure and in a functional position.

The cartilagineous end plates are found at each end of the vertebral centrum and represent the anatomic limit of the disc.[23] This hyaline cartilage is approximately 1 mm thick at the periphery and decreases centrally. The cartilagineous end plates have three main functions:[23] 1) to protect the vertebral center from pressure atrophy, 2) to confine the annulus fibrosis and nucleus pulposus within their anatomical boundaries, and 3) to act as a semipermeable membrane to facilitate fluid exchange between the annulus fibrosis, nucleus pulposus, and vertebral body via osmotic action.

Biochemistry

The normal human nucleus is nearly inaccessible to valid direct biochemical study; therefore, most information of a chemical nature is collected from study of similar tissues of animals.[26] The water content of the nucleus pulposus is about 90% at birth and decreases to 80% at age 20 and 70% at age 60.[24] The annulus fibrosus contains 60-70% water,[24] and this stays constant throughout life. The cartilaginous end plates contain approximately 72% water.[24]

The nucleus contains 15-20% collagen. The annulus contains 50-60% collagen and the cartilaginous end plates contain between 40 and 65% collagen.[24] The collagen content of the intervertebral disc shows little change with age. Collagen is basically a glycoproteinaic with similar basic structure, but with differences in fine structure required for differing functions. The individual collagen molecules assemble themselves into a quaternary structure which is generally fibrillar. These fibrils vary widely in diameter and arrangement depending on several factors. These include the extent of hydroxylysine glycosylation, the interaction of the collagen molecules with other extracellular matrix macromolecules (proteoglycons), and the presence of procollagen molecules in which the amino propetide has not been cleaved.[24]

There are at least 15 genetically distinct collagens found in connective tissue.[24] The intervertebral disc consists of type I, II, V, IX, and XI collagen. The collagen of the intervertebral disc is similar to the collagen content of cartilage. However, the collagen of the nucleus pulposus has a higher glucosylgalactose to galactose ratio and, therefore, will be more hydrated than that of cartilage.[24]

Proteoglycans make up 65% of the nucleus, 20% of the annulus, and 18% of the cartilagineous end plate.[24] Proteoglycans within the disc enable it to imbibe water and hence have an essential role in regulating the mechanics of the intervertebral disc. In general, disc proteoglycans are of smaller size and different composition than those of normal hyaline cartilage.[23,24,26] Disc

proteoglycans contain keratin sulphate and chondroitin sulphate attached to a protein core.[23] When comparing the proteoglycans of cartilage to disc, those of the disc contain more keratin sulphate and less chondroitin sulphate.[23] With age, the total proteoglycan content decreases. The rest of the biochemical composition of the intervertebral disc consists of non-collagenous protein 5-25% in the annulus and 20-45% in the nucleus, elastin, extracellular enzymes, age pigment, and the cells themselves.[24]

Nerve Supply

All structures capable of producing pain are supplied with nociceptors. Activation of these nerve receptors can be caused by mechanical stress and exposure to chemical substances released from traumatized, inflamed, or metabolically abnormal tissues.[3,21]

The structures in the low back supplied with nociceptors and capable of pain production are as follows: 1) skin, subcutaneous, and adipose tissue, 2) fibrous capsules of facet and sacroiliac joints, 3) longitudinal spinal, interspinous, flava, and sacroiliac ligaments, 4) periosteum covering vertebral bodies and arches, 5) dura mater and epidural fibro-adipose tissue, 6) walls of blood vessels supplying the spinal and sacroiliac joints and in vertebral cancellous bone, 7) walls of epidural and paravertebral veins, and 8) walls of intramuscular arteries within lumbosacral muscles.[3,21,27] Also, more recent studies have identified nerve endings up to as far as a third of the way into the

cadaveric annulus fibrosis.[28] Therefore, nearly all the tissues in the low back may give rise to pain.

To complicate the clinical picture of low back pain even further, consider the following. The sinuvertebral nerve supplies at least two intervertebral discs.[27] The individual dermatome receives nociceptive innervation from a minimum of three and maximum of five dorsal nerve roots.[21] Nowhere in the vertebral column does a single facet joint receive its nociceptive innervation from a single dorsal nerve root.[21] The sinuvertebral branch of the second lumbar nerve gives off a long descending collateral branch that extends caudally as far as the fifth lumbar vertebrae.[21] Due to this complex anastomotic innervation of spinal tissues, the origin of back pain is difficult to isolate.

Vascular Supply

The intervertebral disc is the largest avascular tissue in the body.[24] The disc depends on the dual function of molecular diffusion and volume flow[29] to provide its nutrition supply. There are two nutritional pathways into the disc. One is from the blood vessels at the margins of the discs and one is from the vertebral bodies.[23,24,29,30] The nucleus and inner annulus depend on diffusion from the vertebral body, whereas the outer annulus derives its nutritional supply only from their blood vessels.[30] The arteries that feed the nucleus via the vertebral bodies are subject to degeneration and hence the blood supply to the nucleus decreases with age.[30] One consequence of decreased nutrition to the

disc is the loss of proteoglycans.[30] This affects the disc hydration and may lead to disc degeneration.

Function

The primary function of the intervertebral disc is to maintain the space between the vertebral bodies, thus dissipating compressive forces while at the same time facilitating flexibility.[23] The disc also has a secondary function of protection for the neural structures due to its anatomic location.[31]

The fibrous tissue is able to stretch and accommodate movement. It responds poorly to compressive forces.[23] The hydrostatic properties of the intervertebral disc account for the shock absorption function.[23]

Biomechanics

Mechanical low back pain is a common diagnosis given to patients who have increased pain when increased mechanical demands are placed on the spine. Mechanical structures fail when they are unable to support the stress induced by the load applied. The intervertebral disc is affected by both compressive and shear forces. The disc is able to withstand high compressive forces, but appears much weaker in shear.[32]

In axial compression, the increased intradiscal pressure is counteracted by annular fiber tension, disc space narrowing and disc bulge.[31] Degenerative discs tend to bulge more than healthy discs.[23] Adams and Hutton[33] showed that discs do not rupture under compression loading alone; in fact, it is usually

the vertebral end-plate that is the site of failure if compressive loads become excessive.

There is still controversy as to whether the nucleus actually moves forward or backward with trunk flexion and/or extension. Various authors have reported posterior movement of the nucleus with flexion and anterior movement with extension.[33,34] Others report expansion of the annulus on the concave side with simultaneous retraction on the convex side, but no actual movement of the nucleus, just increased pressure that stretches the annulus.[26,35]

In axial rotation, the annulus fibers of one orientation are stretched while those on the opposite side are shortened or crimped.[33] These shearing or torsional stresses are mainly absorbed by the facet joints, and under normal circumstances it is doubtful that much shear is felt by the disc.[32] However, when torsion is combined with trunk flexion, or in the presence of severe deterioration of the facet joints, there is a significant concentration of stress in the posterolateral disc which is a frequent site of disc failure.[33]

Normal Disc Aging

At birth, the water content is approximately 90% in the nucleus pulposus and 80% in the annulus fibrosis.[3,36] The disc consists almost entirely of nucleus with only a thin rim of surrounding annulus.[37] The nucleus is shiny, translucent gray, and amorphous. The inner annulus is white, and the periphery, where sharpey fibers are evident, is dark gray or brown.[38] Both structures contain fibrocartilage and are sharply demarcated from each other.[38,39] The infantile

nucleus pulposus contains cells originating from the notochord which are surrounded by fine fibrous tissue.[40] These cells are located centrally in the nucleus. The cartilagineous end plate consists of two layers; one a growth layer, analogous to the growth plate of a growing long bone, and an articular cartilage layer facing toward the nucleus.[40] Blood vessels are present in the cartilage end plates.

The discs of children and adolescents are different than those at birth. The nucleus pulposus covers approximately one-half the area of the disc, and is located more posteriorly in the disc of the upper lumbar spine and more anteriorly in those of the lower lumbar spine.[41] The boundary between the nucleus and annulus is less distinct as dense fibrous tissue starts to appear at the periphery of the nucleus.[40]

The cells derived from the notochord are still present in the central region of the nucleus, but their numbers decrease with age and are absent by age 20.[40] The nucleus pulposus is gelatinous and turgid in nature and will bulge spontaneously from the cut surfaces of a disc at autopsy.[37] The cartilagineous end plate still has two layers, but shows a reduced growth layer.[40] There is a reduction in the number of blood vessels with many being closed and replaced by cartilaginous tissue.[40] At this stage, the annulus fibrosus first starts to show concentric tears.[39] These are characterized by a simple separation of annular rings without an interruption in their longitudinal

courses. Other than this, the annulus does not appear to change until much later in life.

The adult disc is more semi-solid, having lost much of its gelatinous texture and turgescence.[37] The nucleus shows a much less homogeneous and translucent appearance. At this stage, there is a slight decrease in the water content of the disc.[39] There is an increase in fibrocartilage and dense fibrous tissue near the periphery of the nucleus resulting in an indistinct separation of nucleus and annulus. There is less fiber and more ground substance near the center of the disk and a proliferation of chondrocytes near the cartilagineous end plate.[41] Vertical fibers, which were not present earlier, appear and extend from the end-plate to the nucleus.[40] The cartilagenous end-plate has lost the growth layer and is composed of only the articular layer.[40] The articular layer begins to show areas of calcification accompanied by blood vessels from the vertebral body.[40] This is the beginning of the bone-forming process that eventually penetrates the entire cartigenous end plate and compromises the nutritional supply to the nucleus. The annulus of the normal adult disc may start to show transverse tears in addition to concentric tears.[39] A transverse tear is oriented perpendicular to the fibers of the annulus fibrosus, extends through its outermost fibers, but does not extend centrally to reach the nucleus.

The transformation of the gelatinous infantile intervertebral disc to the fibrous adult disc is considered normal aging. According to a study by Ho et al,[41] these normal age changes were seen in 100% of the people up to the age

group of 30-39. After that, the incidence of normal age changes dropped to 45% in subjects over the age of 70, while the incidence of degenerative disc disease increased to 38%.

Degenerative Disc Changes

It is sometimes difficult to differentiate between normal disc aging and degenerative changes. Age is not a reliable indicator of degenerative disc change. Many elderly discs prove to be just as strong in torsion or compression as their younger counterparts.[32] However, degenerative disc changes are rarely present in persons under the age of 30. According to a study by Ho et al,[38] degenerative changes first appeared in the age group 40-49.

Degenerative discs are characterized by radial tears, a brownish discoloration, and usually narrower disc spaces.[38,39] Radial tears of the annulus have the same orientation as transverse tears; however, they extend through the innermost fibers of the annulus to reach the nucleus pulposus. These radial tears rarely contain nuclear material and therefore appear unlikely to have been formed as a result of disc herniation. It is not uncommon to find vascular ingrowth around the margins of the tears indicating a repair process.[37] Radial tears are postulated to be a result of trauma rather than of aging process.[25,37,42]

In the degenerated disc, there is a weakening of the anchoring of the annulus to the bony end plates. Rather than being attached deeply to the cartilagineous end plates by horizontally coursing collagen fibers as seen in the

normal disc, they are only superficially embedded in the bony surfaces of the end plate.[43] These annular changes are accompanied by a loss of proteoglycans and water and an increase in the glycoproteins of the nucleus.[43] As the nucleus loses its content and becomes semisolid, it carries less and less load. At the same time, support for the inner annulus layers diminishes with the end result of an inward bulging of the inner layers of the annulus resulting in further reduction of the disc height and increased load on the facet joints.[44] These internal derangements of the degenerative disc are more severe when there is also evidence of true disc prolapse.[25]

The progression of the degenerative disc is also accompanied by changes in the vertebral end plates and alterations in the vertebral bodies. The end-plate shows fissures and a disappearance of cartilage as it begins to ossify.[37] This bone formation inside the end-plate means a reduction in the nutritional supply to the disc and may actually accelerate the degeneration of the nucleus pulposus.[10]

The vertebral bodies tend to become relatively lower and broader with age.[45] Osteophyte formation at the peripheral margins of the vertebral bodies is seen with degenerative disc changes; the more severe the degenerative changes the more marked are the osteophyte formations.[37] These changes in the vertebral body are thought to be a compensatory mechanism broadening the base of support for degenerative discs.[45]

CHAPTER III

DISCUSSION

Direct Disc Pain

The unresolved question is how does the intervertebral disc cause low back pain? The classical signs and symptoms associated with intervertebral disc prolapse are easily recognized. However, this accounts for a very small percentage of cases of low back pain.[36] The less well defined diffuse pain complaints make up a much larger percentage of the population of people suffering from low back pain.

The fact that nerve fibers have been identified in the outer third of the annulus fibrosis paves the way for primary disc pain. There are different types of nerve fibers within the intervertebral discs having different functions. The unmyelinated nerves running with blood vessels have a vasomotor role, the free nerve endings have a nociceptive role, and the complex receptors found on the surface of the annulus may have a proprioceptive role.[28]

It is possible that the nociceptor endings are mechanically irritated during bulging of the disc.[46] However, there is a significant number of individuals with bulging discs that are asymptomatic. It is possible that nociceptor receptors are mechanically irritated by concentric and radial tears of the annulus fibrosis associated with disc aging and disc degeneration. However, individuals with concentric tears are often asymptomatic, and radial tears are generally seen in

the elderly when the incidence of low back pain actually decreases.[46] It is also possible that the nociceptor receptors are irritated chemically as it has been shown that some people with LBP have an altered PH.[22] There is continued controversy about whether this alteration in PH is due to the build up of lactate, or is from the breakdown of the glucosaminoglycans found within the nucleus. If the irritation is from lactate, then the pain should be transient, dissipating as the excess lactate is absorbed. If the irritation is from the glucosaminoglycons seeping through the tears of the annulus, then this could be cause for chronic pain. But again, as with the speculation that tears themselves may be painful, the peak incidence of low back pain and the occurrence of these tears do not coincide. In addition, most individuals with radial tears in the annulus are completely asymptomatic.[39]

Indirect Disc Pain

While it appears possible that true discogenic pain does exist, and in fact can be confirmed by discography, it seems more likely that the discs cause low back pain in a secondary manner.

Low back pain generally begins around the age of 25[47] with the peak incidence of disabling symptoms occurring between the ages of 35 and 55.[48] This corresponds to the timeframe when the nucleus is in its semi-fluid state and possesses high intradiskal pressure. At this stage, the nucleus behaves as a non-compressible fluid, and therefore must follow the mechanical laws of a contained viscous fluid:[49] (a) any force that changes shaped will change the

shape of the container (annulus or cartilage end plate), (b) pressure at any point in the fluid remains equal, and (c) displacement of the fluid and container will take place first in the area of least resistance.

This paves the way for bulging of the annulus. Bulging of the annulus generally takes place in the posterolateral margins of the disc.[50] Speculation for this has been attributed to the thinness of the posterior longitudinal ligament, and also that the disc is weakest at its posterolateral margins due to the bunching up of the annular rings. It is also possible that this is the area of least resistance, given that most back injuries are precipitated by being in the flexed position or flexed and twisted position, which increases intradiskal pressure and causes a change in the shape of the container. When this happens, the annulus impinges on pain sensitive structures--primarily the posterior longitudinal ligament and the dural sheath. This gives rise to somatic pain and somatic referred pain. Somatic pain is perceived deeply and is described as dull, aching, or pressure-like in quality.[50] Somatic referred pain is also felt deeply and is aching in quality, and in the context of lumbar spinal pain may occur in the groin, buttock, or lower limb.[50]

The fact that 30% of bulging discs are asymptomatic[51] may lead some to discount the bulging disc as a source of back pain. However, the size of the spinal canal is crucial when dealing with a symptomatic vs. asymptomatic bulge. Patients who undergo surgery after failure of adequate conservative care seem more likely to have small and/or abnormally shaped spinal canals or other

anatomic variants that do not accommodate the bulging disk.[51] Conversely, those who spontaneously recover from a herniated disk usually seem to have large spinal canals and foramina. In addition, a bulging disc should be more symptomatic when it occurs in a younger individual when intradiskal pressure is high as compared to a bulging disc in the elderly when the nucleus is semi-solid.

As one ages, the disc degenerates and becomes semi-solid. Osteophyte formation is present, and there is fibrosis of the posterior joints and capsules.[48] This is the period of stabilization when movement is reduced and the incidence of back pain actually decreases. For some, this stabilization phase progresses to spinal stenosis. It appears that the integrity of the intervertebral disc plays an important role in the prevention of spinal stenosis as the narrowing process is accelerated and more marked when there is bulging of the annulus or internal derangements, such as radial tears or schmorl's nodes.[26]

The integrity of the intervertebral disc is also important in the prevention of osteoarthritic changes to the posterior facet joints of the lumbar spine, with loss of disc height either from disc prolapse or decreased hydration, the contact forces on the facets, increase leading to degenerative changes.[26]

Prevention

It is obvious that the integrity of the intervertebral disc needs to be maintained in order to prevent low back pain. The fact that the majority of low back pain occurs during the ages of greatest intradiskal pressure suggests that

decreasing intradiskal pressures should help in the prevention of low back pain. Nachemson's[22] work on intradiskal pressures shows that different postures will increase intradiskal pressure. Patients do complain that different positions and movements cause increased pain. It has been shown that when the lumbar spine is moved toward lordosis, there is a decrease in pressure and that movements into flexion increase intradiskal pressure.[52] Therefore, to maintain the integrity of the intervertebral disc during early to mid-adulthood, lumbar flexion needs to be avoided, and the normal lordosis should be maintained as much as possible. In order to decrease Intradiskal pressure while seated, the optimal position would be to recline 20° from vertical, use a 4 cm lumbar support, and use armrests.[31] In addition to this, back pain prevention needs to incorporate isometric strengthening of the trunk musculature, as this may prevent the initial onset of back pain.[53] The individual should be subjected to adequate aerobic exercise to ensure sufficient nutritional supply for the disc.[54] Walking is a good exercise that is considered safe from an intradiskal pressure standpoint. In addition, walking causes 5 to 7 degrees rotation at the lumbosacral joint which puts tension on the annular fibers and leads to enhanced strength of the disc collagen, slowing the normal aging development of degenerative disc disease.[54]

CHAPTER IV

SUMMARY

According to Wyke,[21] pain in not a primary sensation; rather, it is an unpleasant emotional state. There can be a large discrepancy between the degree of tissue disturbance and the subjective intensity of the resulting pain. However, a complaint of pain is always indicative of some variety or degree of tissue dysfunction. Unfortunately, when dealing with low back pain, it is very difficult to ascertain which tissue is at fault. It is because of this that only 10-20% of patients suffering from low back pain are given an actual pathophysiological diagnosis for the cause of pain.

The intervertebral disc can cause low back pain either as primary discogenic pain or as indirect pressure pain. Primary disc pain is caused by mechanical or chemical stimulation of the nociceptors found in the outer third of the annulus fibrosus. Indirect disc pain is caused by direct pressure on the nerve root during intervertebral disc prolapse; by a bulging disc putting pressure on the surrounding structures; decreased disc height causing an increase in contact forces on the facet joints leading to degenerative changes; and by accelerating the rate of spinal stenosis when internal derangements of the intervertebral disc are present.

Maintaining the integrity of the intervertebral disc is absolutely crucial to decreasing the socioeconomic impact that low back pain has on this country. It

seems reasonable that more effort needs to go into the prevention of low back pain as a means to control this problem. Prevention starts with the maintenance of normal lumbar lordosis during activities of daily living and is complemented by an adequate exercise program consisting of isometric trunk strengthening and walking.

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