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Low Back Pain

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University of North Dakota

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LOW BACK PAIN

by

Jeff Large
Bachelor of Science in Physical Therapy
University of North Dakota, 1993

An Independent Study
Submitted to the Graduate Faculty of the
Department of Physical Therapy
School of Medicine
University of North Dakota
in partial fulfillment of the requirements
for the degree of
Master of Physical Therapy

Grand Forks, North Dakota
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1994
This Independent Study, submitted by Jeff Large in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

(Faculty Preceptor)

(Graduate School Advisor)

(Chairperson, Physical Therapy)
PERMISSION

Title Low Back Pain

Department Physical Therapy

Degree Masters of Physical Therapy

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Date April 21, 1994
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ABSTRACT

Low back pain (LBP) is one of the most common disorders seen in the health care setting today. The cost of rehabilitation of LBP makes up a significant proportion of today's health care economy. Both physical and social factors have been associated with the etiology of LBP. Today's advanced diagnostic equipment has allowed the underlying pathologies of LBP to be better understood. In the past, different theories and techniques for rehabilitation have been utilized. Current rehabilitation of LBP has emphasized effective, cost-efficient prevention and education.

The purpose of this independent study is to research current theories of risk factors, pathologies and rehabilitation programs associated with LBP. The procedure being used to perform this independent study will be a literature review. Results of this independent study will provide health care workers with information to assist them with effective management of LBP patients.
Chapter 1
Introduction

Low back pain (LBP) is one of the most common disorders in developed countries. Studies have found 60 to 90% of the population are affected by LBP at some time during their lives.\textsuperscript{1-5} Hult\textsuperscript{1} found 60% of males from the general population in the neighborhood of Munkfors, Sweden had at some time suffered from symptoms related to the low back. de Girolamo's study\textsuperscript{2} showed up to 75% of the general population to experience LBP at some point in their lives. Frymoyer and coworkers\textsuperscript{3} found the prevalence of LBP to range up to 90%.

Most episodes of LBP do not seriously incapacitate people. Dixon\textsuperscript{6} reported 44% of people seeking help from family physicians improve within one week, 86% improve within one month, and 92% improve with in two months regardless of treatment. Only 5-10% of persons having LBP develop chronic illness.\textsuperscript{2,7} However, these persons are primarily responsible for the high costs associated with low back injuries.
Spengle and coworkers performed a study on 31,200 employees at Boeing Company in Western Washington between 1979 and 1980. Claims related to back injuries were responsible for 41% of the total injury costs. Ten percent of all back injury claims were responsible for 79% of the total back injury costs. Snook found similar results among industrial workers. He found 25% of LBP cases to account for 90% of the costs. One of the goals of the health care community today is to control these outlying costs.

It is difficult to quantify the total cost of LBP in the United States today. It has been estimated to be from 11.1 billion dollars per year to over 100 billion dollars per year. The total cost includes direct medical expenses and indirect expenses. Indirect expenses include; lost earnings for the employee, personal suffering, and production losses for the employer. In workers' compensation cases indirect expenses have been estimated to be 3 to 4 times greater than the direct medical expenses. In 1984, Glazier and coworkers performed a comprehensive analysis. They came up with the estimated total cost for low back disorders to be about 16 billion dollars per year in the U.S. Webster and Snook reviewed 98,999 claims of LBP reported to Liberty Mutual Insurance Company in 1986 and found the average cost per claim to be $6,807. Direct
medical costs represented 31.5% of this average cost. Frymoyer and Cats-Baril\textsuperscript{12} have estimated the direct and indirect expenses of LBP to be in the range of more than 50 billion dollars per year, and could be as high as 100 billion dollars per year in 1990.

The economic impact on the health care community of LBP is enormous. LBP is the main reason for job-related absenteeism.\textsuperscript{10} Successful prevention and treatment is the goal to controlling the prevalence and expense of LBP. Today’s physical therapist plays a major role in both the prevention and treatment of LBP. I believe a physical therapist should have a well informed understanding of the risk factors, pathology, and treatment of LBP.

There are many risk factors associated with LBP including occupational risk factors. Some of the prevalent risk factors include lifting and twisting, heavy physical activity, smoking, age, alcohol use, vibration associated with driving machinery and trucks, poor posture, prolonged sitting, obesity, inactivity, anxiety and depression.\textsuperscript{14-17} The pathology, applied anatomy and associated risk factors and rehabilitation of LBP will be discussed in the following chapters.

This independent study will research current literature pertaining to risk factors, pathologies and rehabilitation programs
associated with LBP. The procedure being used to perform this independent study will be a literature review. The purpose of this independent study is to compile information to assist health care workers with effective management of LBP patients.
Chapter II
Origin of Pain

To understand entities causing LBP, it is essential to review basic features of pain and the innervation of tissues in the low back. Pain is defined as, "the sensory and emotional experience associated with actual or potential tissue damage." Pain, being subjective, is hard to research. How the body interprets pain is not clearly known.

Pain transition occurs via two basic types of sensory nerve fibers, type A delta and C fibers. Type A delta fibers are large and myelinated fibers which transmit touch and pressures. They transmit signals more rapidly than type C fibers. Type C fibers are fine and unmyelinated which transmit pain and temperature. Both fibers run cephalad to the cortex. Coding of the fibers is explained by the specificity theory. The specificity theory suggests that sensory receptors have specific terminals which are activated by certain types of stimulus. There are two types of receptors of pain stimulus in the low back. One type of receptor is located in the ligaments, peripheral annulus fibrosis, facet capsules, and
periosteum of the bone. Another type of receptors are the nerve roots, sensory ganglia, and spinal nerves that course through the spinal canals. These are not normally nociceptors, but become the source of ectopic pain stimulus under pathological conditions. Once a terminal is activated the stimulus travels on "labeled" fibers to the cortex. The cortex interprets the intensity by the frequency of the impulses received.$^{14,19,20}$

The innervation of the lumbar spine has been studied by Paris$^{21}$ and Bogduk$^{3,22}$. In the lumbar spine mixed spinal nerves off the spinal cord form within the intervertebral foramen beyond the dorsal root ganglion. The first branch off of the mixed spinal nerve is called the sinuvertebral nerve. This nerve branches off the spinal nerve within the intervertebral foramen and re-enters the spinal canal sending branches to the posterior longitudinal ligament, posterior and posterior lateral disc, ligamentum flavum, anterior facet joint, anterior meninges, and the nerve root sleeves as far out as the intervertebral foramen.

Further distally, a branch from the mixed spinal nerve innervates the superior facet joint. Then the mixed nerve continues and divides into an anterior and posterior primary ramus. It also sends one or two ascending branches to the posterior lateral and lateral aspects
of the intervertebral disc. The anterior ramus is larger containing more fibers. It goes on to form the lumbar and sacral plexuses. The posterior ramus travels laterally to the base of the transverse process. It sends an ascending branch intramuscularly to the posterior aspect of the facet joint and lateral and intermediate branches off to the multifidus, iliocostalis, and longissimus muscles and interspinous ligament. The psoas, quadratus and intertransversarri are innervated by the anterior ramus.22,23

All of the structures of the lumbar spine, with the possible exception of the posterior dura, nucleus pulposus, and central annulus fibrosis, are innervated by at least two, and usually three segmental nerves. As a result, pain perceived from the lumbar spine may have a variable distribution of perceived pain.

Acute Conditions

Acute injury to the soft tissue surrounding the spine may result from a traumatic event or mechanical overload. The body's basic response to the initial primary injury includes a secondary response and repair phases. The primary injury is caused by some type of trauma, such as a direct blow, rotational stress, forced abnormal motion and overstretching or tearing forces. The trauma inflicts damage to muscles, tendons, ligaments, bones, nerves or the skin when the forces are greater than these tissues can withstand. This
damage may stimulate pain receptors and result in perceived pain. A certain amount of hemorrhaging will be present if blood vessels are damaged.\textsuperscript{24,25}

The body's secondary responses include bleeding, hypoxic damage to tissues, and inflammation. The clotting mechanism is activated to control the bleeding. Cells are damaged or torn lose from their nutrition supply, and as a result, die from the inability to maintain necessary cellular activities required for normal function. Cells may also be injured by hypoxia from a decreased flow of blood secondary to initial vasoconstriction responses of the body. These necrotic cells and the extravasated blood develops into a hematoma. Increased vascular permeability and structural damage allows plasma and plasma proteins to leak out into the extracellular area. This leakage of blood fluids accounts for much of the edema and may continue for 24-48 hours after the initial trauma has occurred. Chemical contents of this edema may stimulate nerve endings resulting in pain. This inflammatory process is an important defense mechanism of the body.\textsuperscript{24,25}

Muscle spasms may also contribute to LBP. Muscles contract in an attempt to splint the area surrounding the injured area. The resulting contraction may increase pressure on nerve endings, or
create hypotonic conditions resulting in more LBP. The body may respond to the increased pain by increasing muscle spasms, resulting in a viscous pain-spasm cycle.\textsuperscript{24,25}

The lumbar erector spinae and paravertebral muscle complexes are ensheathed in layers of fascia. These fascia layers define compartments in the lumbar spine. Pressure can be raised in these compartments secondary to micro or macrotraumatic injury to the muscles. The inflammatory process and exercising these muscles may increase compartmental pressures.\textsuperscript{26} Pressures as high as 175 mmHg have been measured in paraspinal muscles.\textsuperscript{27} Styf\textsuperscript{28} looked at 12 patients with characteristics of possible paraspinal compartment syndromes. Only one of these 12 patients had an elevation of compartment pressure associated with pain. Paraspinal compartment syndromes can occur, although these conditions are rare.

Spondylolysis results from an overload of forces to the pars interarticularis which causes a stress fracture. This condition typically occurs at the L\textsubscript{4} on L\textsubscript{5} or L\textsubscript{5} on S\textsubscript{1} vertebral levels. Spondylolysis can progress to involve both pars interarticularis regions and include anterior translation of the vertebral body on the lower vertebral segment. This occurrence is named
spondylolisthesis.29

The mechanism of injury involves hyperextension of the normal lumbar lordosis and increased stresses on the spine. If the normal sacral inclination of 30° is increased to 40°, the shear forces increase from 50% to 65% of the upper body weight. Gymnastics and football blocking activities can duplicate this mechanism of injury.29

Risk factors such as lifting and twisting, heavy physical activity30, alcohol use, obesity, and inactivity increase stress on lumbar structures and tissues and promote deconditioning. The presence of these risk factors can significantly increase the occurrence of LBP in an individual.

Chronic Conditions

The intervertebral disc has been associated with most LBP. The disc, along with the two adjacent vertebrae, make up the functional unit of the spine. This unit functions to transmit load, allow motion, and protect the spinal cord. The intervertebral disc's role in this unit is to distribute forces evenly between the adjacent vertebral bodies.31

The intervertebral disc consists of an outer annulus fibrosis surrounding a nuclear pulposus and cartilage endplates. Anteriorly,
the annulus fibrosis is thicker than posteriorly. The annulus fibrosis consists of lamellar layers. Each layer contains collagen and elastin fibers aligned at approximately 65° to the vertebral body endplate. The lamellar layers alternate with adjacent layers, rotated approximately 180°. The lamella of the outer portion of the annulus inserts into the vertebra as Sharpey's fibers. The lamella become tense with compression, bending and twisting of the spine.

Cartilage endplates and nucleus pulposus are inside the annulus fibrosis. The cartilage endplates of the vertebral bodies also are made up from collagen fibers. These fibers do not connect directly to the vertebral bodies though. This makes the cartilage bone interface a potential site for damage from shear forces. The nucleus pulposus is completely surrounded by the collagen network of annulus fibrosis and cartilage endplates. It consists mainly of water and proteoglycans. Water content of the nucleus pulposus makes up 88-90% at birth and diminishes to approximately 70% in old age, whereas water content for the annulus fibrosis remains constant at 60-70% throughout life. The nucleus pulposus is avascular. It relies on diffusion of nutrients from the cartilage endplates and surrounding annulus periphery. Compression and distraction help dispel and absorb water, metabolites and
nutritional supplies respectively. Up to 5% fluid loss has been reported during certain compressive loads. Although Urban and coworkers found that, "fluid ‘pumping’ during movement has an insignificant effect on transport of nutrients into the disc. Small solutes (eg. oxygen, glucose, and sulphate) are transported into the disc chiefly by diffusion." They did find “pumping” to increase the rate of transport of larger solutes into the disc because of their low diffusivities.

Proteoglycans help inflate the network with water through their hydrophilic properties. This helps distribute loads evenly between vertebra and maintain disc height. The proteoglycan matrix prevents serum proteins such as immunoglobulins and other large molecules from entering the disc matrix. It also prevents the ingrowth of nerves and blood vessels into normal discs. In degenerated discs, nerves have been seen to proliferate in the proteoglycan matrix. Irritation of these ingrown nerves may explain LBP associated with some degenerated discs.

The decrease in water content of the nucleus pulposus with age has been associated with a natural physiological degenerative cascade. Most LBP episodes occur between the ages of 25 and 55 years. Kirkaldy-Willis and coworkers have described aging
changes of the disc. They described the nucleus pulposus becoming increasingly fibrous in nature with increasing age. The circumferential collagen fibers of the annulus fibrosis begin to extend toward the center of the disc into the nucleus pulposus. Eventually, the whole disc shows marked fibrosis. This degeneration is contributed to both increasing age and repetitive trauma.

Disc degeneration is profuse in Western industrialized societies; however rare in non-industrialized cultures.\textsuperscript{5,14,40,41} This can be associated with western society’s frequency of sitting and standing posture. The distribution of forces on the intervertebral segment is different when sitting in chairs than when squatting and sitting on the ground. Fahri and Trueman\textsuperscript{41} concluded the incidence of degenerative changes in the intervertebral disc in primitive squatting populations is considerably less than found in civilized peoples. They compared American and Swedish radiographic studies to studies of a tribe of jungle dwellers, a squatting tribe in West Central India. White\textsuperscript{40} stated, “in more primitive cultures, where people use their backs differently, the degenerative segment does not become a painful clinical entity until much later in life.”

McKenzie\textsuperscript{5} suggests LBP can be associated with sitting posture, loss of extension and frequency of flexion. During sitting for
prolonged periods the lumbar spine becomes fully flexed, which stresses the posterior ligamentous structures and eventually becomes painful. The stress on the intervertebral disc increases with loss of extension and frequent flexion. Dolan and coworkers stated, "many commonly adopted postures reduce the lumbar lordosis when compared with erect standing or sitting." Nachemson measured the intradiscal pressure in vivo and found forward leaning, and weight lifting increased the pressure within the disc by more than 100%. When he compared sitting and standing postures he found sitting postures resulted in 40% more intradiscal pressure. He also found normal discs subjected to vertical loads demonstrated higher strains in the posterior part of the annulus fibrous. The additional fact, that the annulus fibrous is thinnest posteriorly, further explains why lesions occur predominantly in the posterior and posterior lateral annulus fibrous. Various studies have tried to prove that mechanical compression played a significant role in the etiology of disc degeneration, but were not able to prove this theory. Farfan and coworkers reported, "it is postulated that in vivo disc degeneration is due to imposed torsional strains rather than to compressive loads."

The degeneration of the intervertebral disc predisposes it to
injury. Rotational injuries in the form of circumferential tears of the annulus fibrosis are common. The outer annulus is associated with most causes of low back pain. Kuslich and coworkers reproduced back pain by stimulating various tissues in the lumbar spine while performing operations with local anesthesia. They found the outer layer of the annulus fibrous and posterior longitudinal ligament to be the most common tissue to produce LBP. The outer annulus is innervated by the sinuvertebral nerves from multi-levels. Nuclear pulp can bulge or break through the distorted lamella and cause the outer most lamella and the adhering posterior longitudinal ligament to protrude into the spinal canal or intervertebral foramen. Repeated minor trauma produce circumferential tears which enlarge to form one or more radial tears. Further trauma leads to internal disruption and/or disc herniation. Once complete internal disruption of the disc occurs the incidence of herniation is low. With the internal disruption of the disc the intradiscal pressure is reduced secondary to loss of disc height. If the disc herniates, it is likely to happen before complete internal disruption of the disc occurs.

With the compromised annulus fibrosis the nucleus pulposus may leak out. McCarron and coworkers studied the effect of the
nucleus pulposus in the dural sac, spinal canal, and its roots. They found evidence of an inflammatory response in dogs. Compression of the nerve roots can bring about clinical conditions through biomechanical and microvascular mechanisms during the inflammatory response.

Compression of spinal nerve roots may induce painless neurologic deficit in terms of sensory changes and motor weakness. Observations in vitro have been made by placing balloons close to nerve roots in the lumbar spine at the time of a laminectomy and then inflating these balloons after surgery. Inflation elicited neurological deficits, but the patients did not complain of pain.\textsuperscript{48} Pedowitz and coworkers\textsuperscript{49} found both the magnitude and duration of compression to affect the degree of efferent and afferent conduction deficit during acute spinal nerve root compression. They suggest pressure-time thresholds may be related to various biomechanical and microvascular abnormalities in the nerve tissue. Chronic compression may induce structural damage to the nerve fibers, impairment of intraneural blood flow, and formation of intraneural edema as well as axonal transport block. Olmarker and coworkers\textsuperscript{50} showed the minimum pressure required to stop flow in the capillaries was 40 mmHg and 30 mmHg in the venules. Venous
congestion can induce an intraneural edema. Pedowitz and coworkers\textsuperscript{49} found significant nerve conduction deficits 1.5 hours after 2 or 4 hours of 100 and 200 mmHg compression on nerve roots. No significant deficits in verve conduction were observed with 0 and 50 mmHg compression. Mechanical deformation may then result in radiating nerve root pain.\textsuperscript{21,48}

Herniation of the nucleus pulposus may be another cause of edema in the epidural space. Homogenized autogenous nucleus pulposus injected into the epidural space caused a chemical or immunologic inflammation of the neural sac and decreased nerve conduction velocity.\textsuperscript{47,51} Theories for this inflammatory response include an autoimmune mechanism against the herniated disc.\textsuperscript{52} Specific substances in the nucleus pulposus such as glycoproteins\textsuperscript{53}, immunoglobulin G molecules\textsuperscript{54}, human phospholipase A\textsubscript{2}\textsuperscript{55} and stromelysin\textsuperscript{56} have been associated with the inflammatory response. Because the nucleus is not normally in contact with the systemic circulation after development of the spine in the fetus, it may indeed elicit an autoimmune response like any other foreign substance would.

The nerve root proximal to the dorsal root ganglia (DRG) differs from peripheral nerves, it generally lacks a perineurium and only has
a very sparse epineurium.\textsuperscript{38,57} This allows for a greater permeability of plasma proteins and other macromolecules. With the inflammation, caused by the nucleus pulposus, and the increased permeability of the nerve roots and DRG, chronic edema can occur. Chronic edema can lead to venous congestion and ischemia which can cause chemical irritation of the nerve root from the presence of waste materials such as lactic acid and proteins associated with inflammation, (i.e. bradykinins, serotonin, histamine, ect.). The chemical irritation may be perceived as LBP with or without radicular pain.\textsuperscript{58}

Nerve roots are not static structures but move with functional activities. The L5, S1, and S2 nerve roots have an excursion of approximately 2-6 mm with a 70° unilateral straight leg raise.\textsuperscript{34} This micromotion helps the nerve root receive nutrition from the CSF. Chronic edema and subsequent fibrosis around and in nerve roots secondary to a herniated disc and/or stenosis can impair this movement and result in tissue irritation.\textsuperscript{59} This pain can be perceived in the low back and/or the lower extremities.

The degenerated discs' most marked change is the loss of proteoglycans. Ng and coworkers\textsuperscript{60} found prolapsed discs to have a change in the normal pattern of collagenolytic enzymes. It may be
possible that this is a consequence of disc prolapse, but it may be
the precipitating factor in prolapsed discs. Lipson and Muir's
study\textsuperscript{61} suggested that loss of confined fluid of the disc signals an
absorptive repair attempt leading to disc degeneration rather than
biomechanical changes in proteoglycans. Ohshima and Urban\textsuperscript{36} linked
the decrease of proteoglycans to the decrease of pH in the
intervertebral disc. The disc, the largest avascular structure in the
body, relies on diffusion from adjacent vertebral bodies for it's
nutritional needs. Ohshima and Urban\textsuperscript{36} stated, decreased pH levels
were associated with increased lactic acid levels from anaerobic
metabolism in the disc. The decrease of proteoglycans in the
intervertebral disc, leads to decreased water holding capability, and
breakdown of the proteoglycan matrix in the disc. A decreased
water content, and compromised disc matrix results in less
efficient transfer of loads between vertebra, and decreased disc
height, stressing other structures within the spine.

Increased pressure across facet joints or impingement may be a
source of pain in patients with reduced disc spaces. The results of
Dunlop, Adams, and Hutton's study\textsuperscript{62} showed pressure between the
facets increased significantly with narrowing of the disc space, and
with increasing angles of extension. Yang and King\textsuperscript{63} calculated
normal facet joints to carry 3-25% and arthritic facet joints to carry as much as 47% of a compressive load. Mooney and Robertson\textsuperscript{64} found structures related to the facet joint (eg. joint capsule and meniscus) can be a persistent contributor to the chronic pain complaints of individuals with LBP and radicular leg pain. Extrarticul ar impingement was found to be caused, or worsened, by disc space narrowing.

Hedtmann and coworkers\textsuperscript{65} studied length changes in lumbar spine ligaments. They found the center of rotation to move to the concave side of motion after destruction of the disc. As a result, extension in the lumbar spine lead to stretching of the posterior longitudinal ligament instead of the expected shortening.

Smoking has been shown to promote disc degeneration by increasing lactic acid levels in the disc. Holm and Nachemson\textsuperscript{66} found that 20-30 minutes of cigarette smoking resulted in constriction of the capillaries underneath the hyline cartilage of the vertebral body and also a reduction of transport of sulfate, methyl glucose and oxygen. The recovery time required for oxygen levels to reach 95% of previous levels was 37 minutes. This oxygen depleted environment further lowers the pH level promoting the degenerative process.
Vibration, such as from driving a truck or bus, is also linked with accelerated disc degeneration. Ishihara and coworkers\textsuperscript{67} subjected fresh disc-cartilaginous end plate complexes of porcine coccygeal vertebrae to vibration at loads at 5, 10, and 35 Hz in vitro. Common construction vehicles generate vibrations between 3.5-9 Hz. They found proteoglycan synthesis in the nucleus pulposus was depressed when exposed to vibration for a long period of time. Vibration has also caused fatigue of the dorsal muscles when subjected to seated vibration. This eventually contributes to degenerative changes within the spine.\textsuperscript{68}

Degenerative spinal stenosis of the lumbar spine is another cause of LBP. Degenerative changes in the facet joints, ligamentum flavum, intervertebral discs, epidural venous structures, laminae, and pedicles are commonly seen in the lumbar spine. Symptoms usually do not develop until the seventh decade of life. The decrease in the volume of the spinal canal occurs slowly, allowing time for accommodation of the neurological structures. Most patients who have advanced degenerative stenosis of the lumbar spine may have few neurological manifestations. Narrowing of the spinal canal, increased pressure within the narrowed canal, inflamed tissues within the canal, ischemia of the cauda equina, and exiting nerve
roots and redundant nerve roots result in a friction neuritis.

Another risk factor for LBP is individual psychosocial factors. Individual psychosocial factors are commonly found in patients with LBP. A history of depression, alcoholism, divorce, low educational level, disabled relatives, extreme religious attitude, job dissatisfaction, family problems, low level of recreational activity, early work history, previous back surgery, abnormal Minnesota Multiphasic Personality Inventory scores, and other psychosocial entities are frequently associated with individuals suffering from chronic LBP. Although psychosocial factors are unproven as predictors of industrial injuries, there are reports that psychosocial factors can predict an individual's response to treatment. Further research needs to look at whether these psychosocial factors can provide a predictive key to industrial injury, or whether they are only the result of the injury.
Chapter III

Rehabilitation

The rehabilitation of LBP has varied throughout history. Hot poultices, massage, various liniments, and some herbal sedative medications have been some of the few treatments used in the past. Today's rehabilitation of LBP has emphasized education and prevention. It can be divided into acute and recovery phases. Acute care consists mainly of pain reduction. Pain control methods are based on the patients ability to function and comply with a prescribed exercise program. The recovery phase utilizes back schools and exercise programs to rehabilitate and educate patients and to prevent reoccurrences of LBP.

Acute Phase

The initial stage of treatment involves a pain control program. This includes ice application, rest, and instruction of basic body mechanics to facilitate pain free movements for ADLs and rest. Trials of traction, extension exercises and TENS may be effective in reducing pain.

Medications can be an effective component in reducing pain.
Saal\textsuperscript{71} frequently used non-narcotic analgesics and NSAIDS. Occasionally he used a class III narcotic analgesic like Tylenol with codeine. He did not use sedative hypnotics or muscle relaxant medications for pain control. Injection of corticosteroide may be utilized for treatment of persistent radicular pain. Decisions to inject or reinject are based on the patient's progress with an exercise program. Injection therapy is used to facilitate functional progress.

Bed rest is not recommended. A level of activity that does not exacerbate radicular pain or worsen a neurologic deficit is desired. Deyo and coworker's study\textsuperscript{72} suggests early mobilization of selected patients with acute LBP is beneficial. Potential adverse effects of bed rest including physical deconditioning and decreased ROM are reduced with early activity.\textsuperscript{72,73}

Gentle stretches help maintain lumbar mobility. William's and McKenzie's exercises can help decrease pain. Both William and McKenzie believe improper posture is the main risk factor for LBP.\textsuperscript{5,74} William's flexion exercises were popular in the 1970's for treatment of LBP.\textsuperscript{74} William\textsuperscript{74,75} believes erect standing redistributes body weight to the posterior aspect of the intervertebral disc in the lumbar spine. This causes the posterior
disc to rupture and put pressure on the spinal nerves in the spinal canal and intervertebral canal. Extension of the spine increases the likelihood of the nerves being impinged.

Sinaki and coworkers\textsuperscript{76} retrospectively compared the efficacy of flexion exercises and extension exercises in the conservative treatment of spondylolisthesis. They found flexion exercises to be more beneficial than extension exercises for symptomatic relief. At three years the recovery rates, measured in terms of having only rare episodes of mild pain, being able to perform job and leisure activities without restriction and no longer using back support, were 0\% for the extension group and 62\% for the flexion group.

Later McKenzie's extension exercises replaced William's flexion exercises in popularity. McKenzie\textsuperscript{5} believes all spinal pain can be attributed to alternation in the position of the disc's nucleus pulposus, in relationship to the surrounding annulus, mechanical deformation of the soft tissue about the spine, which has undergone adaptive shortening, or mechanical deformation of soft tissue caused by postural stress. He contributes these abnormalities to our lifestyle and an almost universal loss of extension in the low back.\textsuperscript{5}

Studies\textsuperscript{77,78} have compared the effectiveness of William and McKenzie's protocols in treatment of LBP. The results indicated that
patients receiving the McKenzie protocol improved significantly faster and to a greater extent than the patients receiving the William’s protocol. DiManagio and coworkers\textsuperscript{79} found the McKenzie program to be twice as effective as traction and back schools in alleviating LBP in a prospective comparison trial. Stankovic and Johnell\textsuperscript{80} concluded the McKenzie method of treatment for acute LBP to be superior to the mini-back school. McKenzie’s protocol has been deemed successful for decreasing LBP in 83\%\textsuperscript{79} and 97\%\textsuperscript{81} of the patients. Donelson and coworkers\textsuperscript{81} reported centralization of radicular pain to be a very accurate predictor of successful treatment outcomes with McKenzie’s program.

Controversy exists regarding indications for surgical intervention and the surgical procedure.\textsuperscript{71} Factors should be taken into consideration when surgical intervention is proposed. Factors including the functional status of the patient, the outcome of a conservative program, the accuracy of the diagnoses, and the neurological signs and symptoms influence the outcome of surgery.\textsuperscript{82} A compliant patient whose neurological signs and symptoms have become worse and who has not responded to a conservative program should consider surgery. Neurological loss, such as sensory, motor and reflex changes, in the lower extremities and disruption of
sphincter control, may indicate surgery. Pain alone is not an indication for surgery. A patient with severe pain, without a clear clinical picture, should complete a psychological test. Any pending litigation should also be taken into consideration. An accurate diagnosis should be made prior to surgical intervention.\textsuperscript{71} A complete history, physical exam, and tests such as MRI, CT scans, myelograms, EMGs and x-rays may help identify pathological structures. Some of the more common surgical procedures performed are laminectomy, chemonucleolysis, discectomy and fusions. Basically, these techniques attempt to relieve pressure off the nerve roots and stabilize the spine. All forms of lumbar spinal surgery increased in frequency from 1979 through 1987. Spinal fusion rates grew the most, 200%; although laminectomy and discectomy surgeries were still performed more frequently.\textsuperscript{83}

Weber\textsuperscript{84} compared a group of conservatively treated sciatic patients with patients who were exposed to surgery. The two groups were followed retrospectively for ten years. His results indicated surgery was more efficient than conservative management in the first year. Ten years into the study there was no significant difference between the two groups.

Shvartzman and coworkers\textsuperscript{85} looked at 55 white, male truck
drivers with a herniated lumbar intervertebral disc. The medical costs and compensational payments for lost working time were analyzed over five years. They found no significant difference in cost-effectiveness between surgically and conservatively treated groups. Their results showed an 80% "good" or "fair" success rate for both groups with average costs estimated at $55,000 +/- $1,000 per case.

Social and psychological factors have been found to influence the outcome of surgery for lumbar intervertebral disc herniations more than physical factors.76

Recovery Stage

The recovery stage utilizes patient education and exercise programs. The goal of the recovery stage is to educate the patient and to attain adequate, dynamic control of the lumbar spine to prevent repetitive injuries.

Back schools play a big part in the recovery stage. They help to increase a patient's self care and functional capacity through increased knowledge of anatomy, improved posture and body mechanics, and improved strength and flexibility. Pain reduction is not a goal, but can be considered a long term result.70

Various clinicians have contributed to the evolution of back
schools. Kraus began formalized exercise programs for LBP sufferers in the 1950's. These programs stressed relaxation and stretching of tight muscles and strengthening of weak muscles to improve physical fitness and relieve symptoms. In the 1960's, Mooney focused his treatment of LBP on reinforcement of healthy behaviors at Rancho Los Amigos Hospital in California. At this same time Fahrni was organizing his concepts in the book, Backache Relived. This book attributed poor posture to LBP. The term “back school” was first used by Marianie Zachrisson Forssel in 1969 at Danderyd Hospital in Sweden. A. White, Mattmiller, and L. White started the California Back School in San Francisco in 1976. In 1977, Berquist-Ullmann and Larson published a study from work done at the Volvo factory in Sweden that showed education could reduce the incidence of LBP. In the 1980's, the back school concept began to spread across North America.

The typical back school consists of three or more one-hour classes in a two-three week time period. The classes proved information on anatomy and biomechanics of the discs, facet joints, ligaments, muscles, bones and nerves of the spine and lower extremities. Correct posture is discussed in both dynamic and static position. The concept of neutral spine, the position when the
spine is most pain free, is defined. Abnormal posture or movements outside physiologic curves and how they will increase strain on spinal segments are discussed.

Stabilization training has become the current trend to help strengthen the trunk muscles. Stabilization training was conceptualized by Ellen Vollowitz, a physical therapist, in Norway. She brought the concept back to the San Francisco area. There Michael Moore, a physical therapist, and she further developed better techniques. A definition of stabilization training is: “to train an individual to move his spine in such a way as to find the least painful position. The spine is held in that “neutral position” while performing ever-increasing tasks.”89 Neutral spine and stabilization training begins in a supine position and progresses to prone, kneeling, standing and transition movements as the patient advances. Emphasis is placed on techniques and the ability of the patient to hold ‘neutral position’ while performing activities. The activities are designed to help strengthen the trunk and lower extremity muscles. They are also based on functional movements or positions of which the patient is involved. Engram motor planning is developed through the precise repetition of activities.

The activities work on strengthening muscles through isometric and dynamic exercises. The trunk extensors and abdominals play an
important part in maintaining a neutral spine during challenging activities. Studies of the thoracolumbar fascia and its biomechanical functions have found the thoracolumbar fascia to be capable of resisting flexion and lateral flexion of the lumbar spine through abdominal and latissimus dorsi muscles.\textsuperscript{90-93} The internal oblique and transverse abdominal are attached to the thoracolumbar fascia through tendonous attachments.\textsuperscript{90,91,93} Muscle and tendonous attachments are the main stabilizers of the spine. Lucus and Bresler\textsuperscript{94} studied intact cadaver spines devoid of their muscles. They found the specimens to buckle at compressive loads of less than four kg.

Soft tissue flexibility and joint mobility are essential components of the stability of the spine. Muscles attaching to the spine and pelvis can influence the spine's symmetry. Muscles act as guy wires on a telephone post. Tight muscles can change postural alignments, increasing stress on certain spinal segments. Stretching of the trunk extensors, hamstrings, quadriceps, iliopsoas, rectus femoris, gastroc-soleus complex, external and internal hip rotators is essential. Stretching should be performed in a relaxed, sustained stretch with attention focused on the muscles being stretched. At first, the stretch may be performed passively by the
therapist. Active stretching, by the patient, is performed as the patient progresses.

Back school education and stabilization exercises have been proven to be effective methods of treatment for patients with LBP.\textsuperscript{71,96-100} The cost-effectiveness of back school education in industry was studied by Versloot and coworkers\textsuperscript{96} in a Dutch bus company over a six year period. Results showed a back school program reduced absenteeism by at least five days per year per employee. The decrease in absenteeism wasn't due to decreased incidence of LBP, but rather to mean length of absenteeism. Rud\textsuperscript{101} eluded that 80\% of funds related to work injuries is due to loss of work days. Klaber Moffett and coworkers\textsuperscript{100} further found that back school was a more effective method of managing chronic LBP than just exercise alone. Brown and coworkers\textsuperscript{99} found the back school to be a cost-effective method of reducing injuries in municipal workers. In fact, their findings showed back school participants to have half as many reinjures as nonparticipants. Sirles and coworkers\textsuperscript{96} found significant improvements in back strength, back flexibility, and psychological well being after participation in back school by municipal workers. Harris' study\textsuperscript{102} analyzed the factors predicting the outcome of the Swedish back school and spontaneous
recovery in chronic LBP. She found the best predictor for the outcome of the treatment and for spontaneous recovery to be work satisfaction.

Prevention of low back injuries has been shown to be an effective, cost-saving entity in industry. The incidence of LBP can be decreased significantly by matching a worker to his/her job. An accurately tested and properly matched worker is 14 times less likely to be injured than an untested or unmatched worker. Ninety-five percent of the tested work force, identified as physically capable of performing physically demanding jobs, worked injury free.¹⁰³
Chapter IV

Conclusion

Results of this independent study shows LBP to be common in Western countries and to have an enormous economical impact on industry and the health care community. Many studies have been performed to study the epidemiology, incidence and costs of LBP. Through these studies many risk factors for developing LBP have been identified.

The pathology of LBP can vary from person to person. The lumbar spine has many structures that have the potential to produce LBP. The most common source of LBP involves the intervertebral disc. The intervertebral disc undergoes an accelerated degenerative process due to increased stress placed on it. This stress occurs secondary to many factors including flexed postures associated with ADLs in most Western societies. Other factors include smoking, vibration, deconditioning, depression, and increased loads associated with heavy physical activity and obesity.

Rehabilitation of LBP is divided into an acute and recovery phase. The acute phase utilizes modalities, exercises and in extreme cases
surgery to provide back first aid and relieve symptoms. The main
goal of this phase is pain reduction and prevention of secondary
complications like deconditioning. The use of back first-aid
techniques including modalities help decrease LBP, and speed up the
healing phase while minimizing complications. The recovery phase
utilizes education and exercises to prevent the recurrence of LBP.
Education on risk factors such as correct posture and lifting
techniques along with strengthening and flexibility exercises help
decrease the reoccurrence of LBP. Strengthening exercises focus on
strengthening the abdominal and paraspinal muscles which help
support the spine. Flexibility exercises work on eliminating tight
musculature that changes postural alignments in static and dynamic
activities.

The cost-effectiveness of back schools and exercise programs
has been proven. Back school and exercise programs have decreased
episodes of LBP and demonstrated a faster return to work. A
significant amount of medical and industrial costs can be saved by
the use of the back school and exercise programs for LBP sufferers.
A back school and exercise program can also be used as a means of
prevention. Matching an employer to his/her job has also been
proven as another cost-effective tool to decrease the incidence of
LBP.
REFERENCES


