A STUDY OF THE EFFECTS OF CHIROPRACTIC THERAPY ON THE DIAMETER OF THE SPINAL CANAL IN PATIENTS WITH LOW BACK PAIN AND RADICULOPATHY

A Dissertation submitted in partial compliance with the requirements for the Master's Diploma in Technology in the Department of Chiropractic at the Technikon Natal.

I, Bradley Stuart Beira, do hereby declare that in respect to the following dissertation, titled 'A Study Of The Effects Of Chiropractic Therapy On The Diameter Of The Spinal canal In Patients With Low Back Pain And Radiculopathy'; as far as I know and can ascertain no other dissertation nor thesis exists, and all references detailed in the dissertation are complete in terms of all personal communications engaged in and published works consulted.

The completed paper represents original research compiled by the author.

BY

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Approved for final submission by

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DEDICATION

This work is dedicated to my wife Camilla, whose understanding, patience and encouragement remained unwavering.

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ABSTRACT

Focus during this investigation was directed towards determining alterations in the size of the lumbar intervertebral disc in patients symptomatic with low back pain and sciatic distribution pain, in response to chiropractic management of this condition. The efficacy of chiropractic management for low back pain with associated radiculopathy has been examined. Flexion distraction technique and side posture rotatory adjustment technique of the lumbar spine have been used during this trial.

An unblinded experimental clinical trial was designed, making use of thirty subjects who presented to the Chiropractic clinic at Technikon Natal with low back pain and radicular symptoms and signs. Fifteen patients were included into each group. No controls were used. Patients were assessed using recognised orthopaedic and neurological testing procedures. Numerical rating scale 101 and the Oswestry Back Disability Index were used to obtain subjective patient responses. Computed tomographic examination of the lumbar spine was conducted on an Elscint 2400 scanner by a team of independent radiographers.

Pre and post treatment computed tomographs were conducted on all patients participating in this programme.
Data was statistically evaluated using Parametric paired and unpaired t-tests within the 95 percent confidence interval. Critical values for percentage occupancy of the spinal canal by the intervertebral disc at the fourth intervertebral disc was evaluated as 0.008, and at the fifth intervertebral disc as 0.763. ($t =_{13;0,05}$ 1,771)

No statistically significant changes were noted in the percentage occupancy of the spinal canal by the intervertebral disc at any level. Changes in the percentage occupancy included increased occupancy in ten cases and decreased occupancy in twenty cases.

Intervertebral disc lesions were noted at the third, fourth and fifth lumbar intervertebral discs. Most commonly the lesion was located at the level of the fifth intervertebral disc, followed by the fourth intervertebral disc, followed by the third intervertebral disc.

Thirty eight lumbar intervertebral disc levels were found to be pathological prior to onset of chiropractic treatment.

Both flexion distraction technique and side posture rotatory adjustment of the lumbar spine provided symptomatic relief for the presenting condition. Neither technique proved effective in altering the percentage occupancy of the intervertebral disc within the spinal canal.
Chiropractic care may be viewed as a favourable approach in the management of low back pain with radiculopathy. The compressive role of the intervertebral disc lesion as a protagonist to the signs and symptoms of low back pain with radiculopathy needs be reevaluated.

The role of inflammatory agents and chemical mediators should be more carefully considered as causative agents in neurological fallout, associated with lower limb radiculopathy. Capsulitis of the facetal joints at the level of the disc lesion is most likely to be the source of the low back pain experienced.

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CHAPTER ONE

INTRODUCTION

Chiropractors have reported success in the treatment of intersegmental dysfunction, posterior facet joint problems, lateral recess stenosis, central canal stenosis and other aspects of spinal degenerative disease, which form part of the mosaic of low back and leg pain syndromes. (Cassidy **et al.** (1993), Cox **et al.** (1993)).

Questions exist as to the relationship of pain and discomfort to the anatomical and pathophysiological circumstances which may or may not be found in those suffering from these complaints. (Howe **et al.** (1987)) In an age of rising health care costs and subsequent insurance limitations, it has become increasingly important to determine which diagnostic studies are most sensitive, specific and accurate in diagnosing lower back pathology, because it is not always possible to obtain all the desired diagnostic procedures. (Bischoff **et al.** 1993)

Pain experienced by the patient, both radicular and referred, have been reported to diminish as a direct result of chiropractic intervention, without any apparent change in the extent of the foraminal stenosis. (Cassidy **et al.** 1993). The presence of disc herniations in asymptomatic population of patients has been described by researchers since 1984. (Wiesel **et al.** (1984)). A literature review presented by Chapman-Smith (1993) suggests that up to 40 % of people over the age of forty are asymptomatic, with an abnormal computed tomography showing a disc herniation.

Changes in occupancy of the spinal canal by the intervertebral disc has been the topic of investigation over a number of years. Literature throughout the professions suggests that the intervertebral disc herniation decreases post therapy. (Bush **et al.** 1991, Cassidy **et al.** (1993), Cox & Aspegren (1987), Cox **et al.** (1993), Delauche - Cavallier **et al.** (1993), Maigne **et al.** (1992), Saal **et al.** 1989)).

The purpose of this investigation was to evaluate the effectiveness of chiropractic treatment in the management of low back pain with associated radiculopathy, in terms of objective and subjective clinical findings, as well as any alterations to the spinal canal, as determined by computerised tomographic examination. This was performed in order to arbitrate a more conservative management approach toward the treatment of mechanical low back pain with associated radiculopathy.

Objectives to be achieved included the measurement of alterations to the objective and subjective presentation of the patient, with respect to physical dysfunction. Orthopaedic testing for pathology in the lumbar spine and sacroiliac joints were undertaken. Subjective patient responses were measured using questionnaires and diagrams. Computed tomography was used to measure the size alterations of the spinal canal and intervertebral disc. Measuring procedures, as described by Cox & Aspegren (1987), were used to obtain these results. This technique was selected since it provided what appears to be a more conservative, digitally reproducible measurement procedure as opposed to visual assessment of alterations in intervertebral disc herniation size. It is felt that the mechanism towards the cause of pain should be re-evaluated, with less emphasis on the radiological impression.

This study has served to place emphasis on the zygapophyseal joints, chemical mediators and associated soft tissue structures as precursors for nociceptive stimuli. The incidence of surgical intervention for patients presenting with radicular symptoms may well decrease as a greater number of practitioners place greater emphasis on returning the biomechanics of the spine to within normal limits, rather than surgically removing soft tissue lesions from the lumbar spinal canal. Patient response time is far greater with conservative care than with surgical intervention, with a decrease in expense and time lost from work. These implications bode well in a society with escalating medical expenses and only partial renumeration from medical aid schemes.

There will always be place for surgical intervention in the management of severe non responsive radiculopathy. Greater emphasis should be placed on active conservative intervention for soft tissue spinal lesions in patients with low back pain and lower limb radiculopathy.

CHAPTER TWO

REVIEW OF RELATED LITERATURE

INTRODUCTION

Low back pain is a pandemic complaint, with between 80 % and 90 % of the population suffering from this condition, in one of its forms, at some time in their lives. (Bogduk (1990), Bush **et al.** (1992), Cassidy (1993), Cox (1990), Fischgrund (1993), Hession (1993), Jonsson & Stromqvist (1993), Jull (1990), Kaupilla (1993), Maigne (1992), Neault (1992), Olmarker **et al.** (1993), Simmons Jr. **et al.** (1993), Weber (1993)).

Despite the major socioeconomic and medical significance of low back pain, the knowledge of the basic underlying pathophysiological mechanisms is fairly limited. (Olmarker **et al.** 1993). The common, simultaneous occurrence of back pain and sciatica is not really understood. Compression of nerve roots was recognised as early as 1931 by Towne and Reichert. Simmons Jr. **et al.** (1993) are of the opinion than low back pain is frequently accompanied by radiation and/or dysesthesias down the length of one or both of the lower extremities, in either a dermatomal or somatotopic (myotomal or sclerotomal) sensory pattern.

Weber **et al.** (1993) feel that sciatica with nerve root symptoms and signs is a frequent illness. They feel that the pathophysiology of radicular pain due to nerve root compression is still unclear. Simmons Jr. **et al.** (1993) are of the opinion that emphasis has been misplaced on defining a dermatomal distribution, but should rather be focused towards the existence of myotomal or sclerotomal neurosensory fallout. A combination of a large vertebral canal, small dural sac, no ligamentum flavum, or facet hypertrophy with a small disc protrusion may be asymptomatic with minimal pressure on the nerve root. A similar combination of a small vertebral canal, a large dural sac area, and facet hypertrophic stenosis, with ligamentum flavum thickening, could lead to the presentation of severe pain with the same degree of nucleus pulposus herniation. Cox (1990:258)

The following chapters are designed to present the anatomical structures within the lumbar spine, followed by the pathoanatomy of the discussed structure with its mechanism towards potential nociception. The possibility of the discussed structure contributing to referred or radicular pain in the lumbar spine and lower limb will also be explored. A review of related literature pertaining to the lumbar manipulation and flexion distraction techniques and their efficacy shall follow the anatomical discussion. A brief discussion of the imaging modality used, computed tomography, with plates of the pathologies discussed will complete this section.

2.1 - THE VERTEBRAL CANAL

Early in embryological development, there is an intimate segmental relationship established between each nerve and its correlating dermatomal, myotomal and sclerotomal components. (Simmons Jr. **et al.** 1993)

2.1.1 - The Peridural membrane

Wiltse **et al.** (1993) describe the peridural membrane as a fibrovascular sheath lying external to the dura lining the vertebral canal. The membrane extends up the medial sides of the pedicles and around on the undersurfaces of the laminae and ligamentum flava. No neural structures have been identified within the ligamentum flavum. (Giles 1991). The peridural membrane crosses the disc level laterally at the entrance to the bony lateral canal. (Wiltse **et al.**1993).

This membrane truly surrounds the dura leaving a potential space between itself and the dura. This area may be referred to as the epidural space. Along with the lateral expansions of the posterior longitudinal ligament, the peridural membrane is continuous with the sheath that lines the bony lateral canals, referred to as the circumneural canal. (Kikuchi 1982)
Within the circumneural canal lies the circumneural sheath. This sheath appears to start at the entrance of the neurovascular canal and, in the lumbar spine, continues out eight to ten millimetres beyond the lateral foramen. The circumneural sheath was first described as the "epiradicular sheath" by Kikuchi in 1982.

The peridural membrane surrounds the dura and extends through the lateral aspects of the vertebral canals (lateral recesses) and surrounds the spinal nerves. There is no peridural membrane on the posterior aspect of the annulus fibrosus. The peridural membrane passes posterior to the superficial layer of the posterior longitudinal ligament and is attached to the undersurface of the deep layer of the posterior longitudinal ligament. The dural sleeves surround the dorsal and ventral roots and accompany them to just beyond the sensory ganglion. At this point the dura blend with and becomes continuous with the epineurium of the spinal nerve (Wiltse **et al.** 1993).

Wiltse **et al.** (1993) proposed the name "dural root sleeve" for the tightly bound area immediately preceding the ganglion. This area is tightly bound to the radicles, preventing even water soluble contrast from flowing into this space. This small segment of tissue extends from the lateral border of the main dural sac and ends just beyond the sensory ganglion. Contained within are the two nerve roots inside a dural sheath. (Wiltse **et al.** (1993)) On the medial border and caudal border of the pedicle, the peridural membrane is loosely attached. The lateral side of the pedicle is covered with periosteum which appears to be a homologue of the peridural membrane.(Wiltse **et al.** 1993).

2.1.2 - Lumbar Vascular Supply

Upper and middle parts of the lumbar spine receive their blood supply from the paired lumbar arteries. Branches of the iliolumbar arteries and the middle sacral artery supply the lower part of the lumbar spine. (Kauppila & Tallroth 1993) Blood enters the vertebral bodies through the arterial system, exiting through the venous system, through a large foramina on either side of the bony septum near the central point of the posterior face of the vertebral body. With changes in intraabdominal pressure, the direction of blood flow will reverse.

2.1.2.1 - Vertebral ischaemia

Saal & Saal (1989) postulate that stenosis would cause an interruption of blood flow to the nerve root with resultant venous congestion, ischaemia, axonal damage and eventual intraneural fibrosis. Olmarker **et al.** (1993) noted that central fascicular lesions was seen when the endoneurial circulation is compromised by vasculitis. This may indicate an element of ischaemia. Epineurial and endoneurial bleeding as well as the hyperaemia noted in their subjects suggest that these may influence the function of nerve fibres. Although the cause of the inflammatory reaction is unclear in these studies, an inflammatory reaction in the spinal canal may be of pathological significance, regardless of the cause.

Olmarker **et al.** (1993) concluded that the presence of autologous nucleus pulposus in close proximity to the epidural sheath, without mechanical compression, may induce pronounced changes in the nerve root structure and function. Stenotic conditions may create pressure levels on nerve roots, but these might be insufficient to cause either motor or sensory findings. Functional changes induced by nerve root compression can be caused by mechanical nerve fibre deformation, associated with intervertebral disc herniation and spinal stenosis. These changes may be a consequence of changes in nerve root circulation, leading to ischaemia and formation of intraneural oedema. Kauppila & Tallroth (1993) are in agreement with Olmarker **et al** . (1993) and suggest atherosclerosis of the radicular arteries as a possible cause of the patients' discomfort.

Compression of normal peripheral nerve, or nerve root, may induce numbness but rarely induces pain. This numbness may be as a result of ischaemia, not mechanical nerve fibre compression. (Dorwart **et al.** 1983, Kirkaldy Willis 1984). If a nerve root is the site of chronic irritation, even minor mechanical deformity may induce radiating pain. Compression of peripheral nerve trunks and spinal nerve roots will induce marked changes in the microcirculation and nutrition of the nerve trunk and nerve root. The resulting endoneurial oedema, along with direct mechanical stresses, can contribute to the observed structural changes in the nerve and thus the presentation of clinical signs and symptoms. (Ghormley 1983).

Kauppila & Tallroth (1993) evaluated, by postmortem angiography, the differences in the arteries supplying the lumbar spine between subjects who, during their lifetimes, were with or without low back pain. They concluded that the lumbar and middle sacral arteries are more likely to be narrow or absent in persons with low back pain history than those without.

2.1.2.2 - Internal vertebral venous plexus

The Internal vertebral venous plexus, is a confluence of valveless veins that run longitudinally and lie largely on the dorsal surface of the peridural membrane, but penetrate it at several points to form the basivertebral veins that enter the vertebral bodies through the nutrient foraminae. (Wiltse **et al.** 1993).

Internal vertebral venous plexus, (Batson's venous plexus), is associated mainly with the anterior, and to some extent the anterolateral parts of the epidural space of the fourth and fifth intervertebral disc levels. (Giles 1991). These veins cross the posterior longitudinal ligaments and the annulus fibrosis complex without interruption at the disc level. Yasuma **et al.** (1993) believe that the presence of small blood vessels accompanied by loose fibrous tissue is sometimes noted in the marginal regions of free, extruded pieces of annulus fibrosis. The authors feel that these blood vessels may be causative of extradural haematomas, as also described by Delauche - Cavallier (1992). These sentiments are echoed by Wiltse **et al.** (1993). There is a rich vascular supply to the dorsal root ganglion in the form of microvascular network. Radicular veins connecting the internal vertebral veins with the external vertebral veins, (epivertebral veins), are positioned anteriorly on the dorsal root ganglion. (Hasegawa 1993)

2.1.3 - Posterior Longitudinal Ligament

At the mid vertebral body level, the superficial layer of the posterior longitudinal ligament is wider than its deep layer. The superficial layer becomes wider at its midpoint as it progresses cranially. The long fibres of the central portion of the posterior longitudinal ligament form a band approximately eight to ten millimetres wide, traversing two to three segments. The Posterior Longitudinal Ligament attaches to the annulus at the vertebral margins but is separated from the annulus as it crosses the intervertebral disc space. The remainder of the superficial posterior longitudinal ligament is hour glass shaped and blends with the annulus at the disc level. (Ogata & Whiteside 1981)

The peridural membrane passes underneath the superficial layer of the posterior longitudinal ligament and is attached to the undersurface of the deep layer of the posterior longitudinal ligament. The deep posterior longitudinal ligament membranes are attached loosely to the bony septum so blood or disc fragments usually stay on one side or the other. The bony septum is not complete. At these areas of incongruity, there is no septum and blood or disc fragments may migrate across. (Ogata & Whiteside 1981)

2.1.4 - Hoffman's Ligaments

Hoffman's ligaments stretch between the dura and the superficial layers of the Posterior Longitudinal Ligament. They are very narrow, almost threadlike at the level of the fifth lumbar vertebral, and may be absent at the level of the first sacral segment. There are usually two at each level, a right and a left. There is considerable anatomical variation and multiple fibrous attachments may be present. (Spencer **et al.** (1983), Wiltse **et al.** (1993)).At the level of the upper lumbar spine, the ligaments often meet in the middle to form one wide band. These bands attach to the area of the Posterior Longitudinal Ligament where it blends with the Posterior Longitudinal Ligament annulus complex, and pass posterior and cranially to attach to the dura. (Spencer **et al.** 1983) Spencer **et al.** (1983) describe the function of lateral Hoffman's ligaments, as binding the dural radicular sleeve to the vertebral canal.

These ligaments have the function to prevent posterior movement of the spinal nerve root when a disc bulges against it anteriorly. Thus pain is produced even though there is plenty of room for the nerve in the bony canal posteriorly.

2.1.5 - Lateral Recess

Hasegawa **et al.** (1993) describe the lateral spinal canal by dividing the spinal canal into three regions: the entrance zone, the midzone and the exit zone. The entrance zone, also known as the lateral recess area is the subarticular area medial to the pedicle. The midzone is located under the pars interarticularis and the pedicle, while the exit zone is synonymous with the intervertebral foramen.

2.1.5.1 - Entrance zone

Located ventral to the superior articular process of the facet joint and medial to the pedicle, the entrance zone lies inferior to the facet joint. The lateral recess begins at the lateral aspect of the thecal sac and runs obliquely downwards and laterally to the intervertebral foramen. Nerve root compression in this region is referred to as lateral recess syndrome, superior facet syndrome or nerve root canal stenosis. (Hasegawa **et al.** 1993). The lateral recess is bordered laterally by the pedicle, posteriorly by the superior articular facet, anteriorly by the posterolateral surface of the vertebral body and adjacent intervertebral disc. Medially the lateral recess is bordered by the thecal sac. The narrowest portion of the lateral recess lies between the superior border of the pedicle and the broad portion of the superior articular facet. The length of the lateral recess is longer in the lower lumbar levels. (Hasegawa **et al.** 1993)

In this region the nerve root is covered by cerebrospinal fluid and is encased by the root sleeve. The lateral margin of the nerve root sleeve comes into contact with the medial cortical bone of the pedicle. The medial margin of the nerve root sleeve is surrounded by epidural fat tissue. Hasegawa **et al.** (1993) suggests that the nerve roots exit from the lateral recess at varying inclinations, dependant on the level in the lumbar spine. This inclination will increase in the vertical direction for the more caudal nerve roots. The authors lists the angles of inclination in the lumbar spine as follows: seventy to eighty degrees to the vertical at the first and second lumbar vertebral bodies, sixty degrees for the third and fourth nerve root levels, forty five degrees for the fifth lumbar nerve root and thirty degrees for the first sacral nerve root.

2.1.5.2 - Midzone

The midzone is located under the pars interarticularis and just below the pedicle. Anteriorly it is bounded by the posterior aspect of the vertebral body, posteriorly by the pars interarticularis. Medially the midzone opens into the spinal canal. Spondylolysis and pedicular anomalies may result in nerve irritation or impingement within this zone as the nerve root travels obliquely downwards through the lateral recess into the intervertebral foramen. (Hasegawa **et al.** 1993)

The nerve roots travel under the subpedicular notch and contacts posteriorly with the ventral wall of the pars interarticularis at the attachment of the ligamentum flavum.

2.1.5.2.1 - Spinal nerve roots

The dorsal and ventral nerve roots are covered with the common epineural sheath. Within this sheath, the motor nerve roots lie anterior to the dorsal root ganglion. The cross sectional area of a typical nerve root varies from twenty nine to forty two millimetres squared, and is larger at the more caudal levels of the lumbar spine. At these levels, the nerve roots occupy twenty three to 30 % of the area of the foramen. Within the intervertebral foramena the dorsal root ganglion, ventral root and dural sleeve are covered with adipose tissue. (Hasegawa **et al.** 1993) Each spinal nerve is formed by the union of the anterior and posterior roots which arise from the spinal cord as rootlets. The spinal nerve passes through the intervertebral foramen and divides to form the anterior and posterior primary rami.

The mean length of the spinal nerve roots from the thecal sac to the proximal margin of the dorsal nerve root ganglion varies from six millimetres, at the level of the first lumbar nerve root, to fifteen millimetres at the second sacral level. The average size of the dorsal nerve root ganglion measures thirteen millimetres long and six millimetres wide, with a gradual increase in dimensions from the first lumbar to first sacral levels. (Hasegawa **et al.** 1993)

The anterior rami form the lumbosacral plexus and the posterior rami divides further to form medial and lateral branches. The medial branch passes dorsally and caudally through the intertransverse space toward the superior border of the root of the subjacent transverse process. From here it continues dorsally and caudally, lying against the groove formed by the junction of the root of the transverse process with the root of the superior articular process. This medial branch of the posterior ramus is held in the groove formed by the mamillary process and the accessory process by the mammilloaccessory ligament and has been thought to be causative in some degree to low back pain.

At this point the medial branch of the posterior primary division of the nerve divides into

i) an ascending branch which goes to the adjacent zygapophyseal joint capsule in the region of the inferior recess,

ii) a branch to the Multifidi muscles

iii) a branch to the superior zygapophyseal joint one segment caudal.

Giles (1991) found that the posterior primary ramus only occupies a small percentage of the space enclosed by the mammiloaccessory ligament and concludes that it is unlikely that the medial branch of the posterior primary ramus could be trapped beneath the mammiloaccessory ligament causing pain. Irritation of the nerve root may occur, accounting for nociceptive stimuli, perceived as pain, by the patient. Macnab (1986) and Wilste **et al.** (1993) note entrapment of the L5 nerve with the transverse process of the fifth lumbar vertebra, impinging on the nerve against the sacrum in spondylolisthesis, degenerative scoliosis and asymmetrical disc disease.

Because of the ability of the dorsal root ganglia to synthesise substance P and other neuropeptides, they are considered to be pain sensitive structures and are implicated in the pathogenesis of low back pain. (Hasegawa **et al.** 1993). Immunohistochemistry revealed substance P antibody immunoflourescent nerve fibres within the capsule and the synovial folds.

2.1.5.2.2 - Segmental structures innervated

Nerves emerging from the lumbar spine innervate the following various segmental structures: the annulus fibrosis of the intervertebral disc; spinal ligaments, fascia, tendons of the paraspinous and lower limb muscles; periosteum and synovial membrane within the joints of long bones; the dermis and subcutaneous tissue with dermatomal, myotomal and sclerotomal extensions corresponding to that nerve. (Bogduk (1983), Simmons Jr. **et al.** (1993)) Sinuvertebral nerves are sensory afferent nerves relaying nociceptive stimuli from free nerve endings. These nerves are seen to branch from the postganglionic anterior primary ramus to innervate a portion of the segmental sclerotome, sending fibres to the intervertebral disc, posterior longitudinal ligament, anterior dura and periosteum.

There are afferent autonomic sympathetic branches from the sympathetic ganglion, above the level of the second lumbar vertebra, and from the sympathetic chain, below the level of the second lumbar vertebra, that develop connections to the sinuvertebral nerve. Bogduk (1983) describes three branches ascending, descending and transverse branches. The ascending branch relays information from the posterior longitudinal ligament, intervertebral disc and anterior dura of the segment above. The descending and transverse branches supply the entry level disk, posterior longitudinal ligament and anterior dura. In addition, there may be interaction with as many as two to three segments below. Thus there may be significant overlap between as many as three levels. (Bogduk 1983)

Nerve endings have been identified within the periosteum of the developing embryo as within ligaments and synovial membrane of human joints. (Simmons Jr. **et al.** 1993). These are separate from the cutaneous nerve endings supplying the dermatomes. Sclerotomal symptoms may result from trauma, during which time an object may come into contact with the periosteum. These fibres can be directly stimulated leading to a dull aching sensation post trauma. This sclerotomal information passes into the ventral primary ramus and dorsal root ganglion. Constitutional manifestations such as nausea, vomiting, sweating and vasoconstriction may result concomitant with the sclerotomal involvement. (Simmons Jr. **et al.**1993)

Myotomal pain may be regarded as being similar to sclerotomal pain in that both are a combination of perception and elicitation, being deep, dull and aching in nature. What characterises myotomal pain is that it may be well localised. As in the case of an acutely tender motor point, myotomal pain may be elicited by direct pressure, not uncommonly associated with palpable muscle spasm. (Simmons Jr. **et al.** 1993) Mesoderm overlying the myotomes also receives sensory innervation from various peripheral nerves, giving rise to dermatomes. Dermatomes lie directly beneath the skin and are formed by the sensory afferents located within the subcutaneous tissues and dermis of the skin.

If a nerve root is compressed, the patient experiences numbness. Inflammation of the nerve will result in pain referred to a portion of the limb innervated by that segment. (Simmons Jr. **et al.** 1993) The above mentioned authors propose a mechanism whereby severe leg pain may be present in a "dermatomal" pattern with negative tension signs and little radiographic evidence of nerve compression. Chemical mediators; Angiotensin, Bombesin gastric related peptide, Calcitonin gene related peptide, Cholecystokinin, Enkephalin, Neurotension, Somatostatin, Substance P, Vasoactive intestinal polypeptide, may act as irritants around the dorsal root ganglion. Chemical mediators, as well as free nerve endings within the various structures, when stimulated will result in referred pain (dysasthesias) without tension signs nor neurological deficit. Simmons Jr. **et al.** (1993) labels the phenomena of dysasthesias without neurological findings as convergence. The author further concludes that dermatomal charts should be utilised only as general references, not as a guide to specific diagnosis by their use alone. (Simmons Jr. **et al.** (1993))

Two theories have been forwarded by Simmons Jr. **et al.** (1993), as first described by Ruch and Patton, with regard to referred pain mechanisms; Convergence facilitation, Convergence projection These mechanisms related primarily to visceralsomatic referred pain patterns, but their relationship to radicular pain can be appreciated.

2.1.5.2.3 - Convergence - facilitation

Impulses from exteroceptive nociceptor fibres in the skin, that are given a continuous stimulus from their exposure to environmental stimuli, can be amplified by the addition of a stimulus from the nociceptive fibres within an acutely torn annulus fibrosis.

2.1.5.2.4 - Convergence - projection

Afferent axons from two different regions synapse upon the same spinothalamic tract cells that receive their primary input from other structures. Once stimulated, the spinothalamic tract cell projects the information to the thalamus and sensory cortex, through the anterolateral tract. This pain is perceived as arising from one structure.

For example - a primary spinothalamic tract cell receives dermatomal information from cutaneous receptors, but also receives sensory input from the sclerotomal (annulus fibrosis) and myotomal components. Once stimulated, the pain may be perceived as arising from one single structure. Intersegmental and intrasegmental spinal reflexes, which are primarily related to large diameter fibres, terminate in the motor nuclei. These fibres are important as they mediate both stretch and pain reflexes. Thus these pathways may be important in generating muscle spasm reflexes in response to pain. (Simmons Jr. **et al.** 1993).

2.1.5.3 - Exit zone

The tunnel formed between the lumbar intervertebral canal and the spinal canal, bounded superiorly and inferiorly by the pedicles of the adjacent vertebrae, posteriorly by the pars interarticularis and ligamentum flavum, and anteriorly by the posteroinferior margin of the superior vertebral body, the posterior margin of the intervertebral disc and the posterosuperior margin of the inferior vertebral body, is referred to as the exit zone.

Transforaminal ligaments may be present in the inferior portion of the intervertebral foramen, arising from the superior posterolateral intervertebral disc margin, to attach to the superior articular process. The normal foraminal height varies from twenty to twenty three millimetres. (Hasegawa **et al.** 1993) The width at the upper foraminal area, where the dorsal nerve root ganglion is located, varies from eight to ten millimetres (Hasegawa **et al.** 1993). The costotransverse ligament has been implicated as a potential source of nerve entrapment syndrome and neural irritation when accompanied by other pathological changes, such as spondylolisthesis, L5 disc degeneration or lateral herniation. The ligament which has been noted to be up to five millimetres thick, bisects the lateral aspect of the foraminal canal into two compartments. In his study, Church **et al.** (1991) discusses the relation of the costotransverse ligament to the intervertebral canal and investigates various diagnostic imaging modalities. Anatomical dissection consistently found the gray ramus communicans to enter the anterosuperior compartment, and the ventral ramus of L5 exiting through the posteroinferior compartment. The segmental nerve root occupies 35 % to 50 % of the intervertebral foramen.

Pathology in and about the foramen may reduce its dimensions and compress or irritate the nerve. In situations in which reduction in the height of the foramen may occur, the costotransverse ligament may encroach or even entrap the ventral ramus against the ala of the sacrum. (Church **et al.** 1991)

2.2 - Spinal Stenosis

Spinal stenosis in the lumbar spine is described, by DuPriest (1993), as one of the end processes in the pathogenesis of low back pain. The author feels that lumbar spinal stenosis is the end result of progressive degenerative change. These changes produce symptoms which may be debilitating.

2.2.1 - Classification of Spinal Stenosis

Two points must be considered when defining and classifying spinal stenosis:
(1) narrowing of the osseoligamentous spinal canals which can
(2) cause compression or irritation of neural structures.

The morphological shape or size of the canal is less important than the presence of neurologic compromise. It is only when the narrowing of the canal reaches the point where it encroaches on the nervous system that spinal stenosis can be considered significant. (Yang **et al.** 1993)

2.2.2 - Types of Spinal Stenosis

Cox (1990:272) subdivided degenerative stenosis into:

I) Acquired (degenerative) stenosis

A. thickened irregular laminae,

B. ligamentum flava hypertrophy,

C. soft tissue hypertrophy mechanical instability, degenerative disease,

D. posterior articular joint disease,

E. trefoil configuration of the intervertebral canal,

F. intervertebral disc protrusion,

G. spondylolisthesis with forward L5 displacement on the sacrum,

H. posterior intervertebral body hypertrophic osteophytes into the foramina,

I. narrowing of lateral recess due to hypertrophic articular processes,

II) Stenosis resultant from excessive stress placed on a motion segment above a level of spinal fluctuation causing:

A. The ligamentum flavum and interspinous ligament to become thickened,

B. the spinous process base to project into the canal,

C. the lamina to protrude ventrally.

There may be proliferation of bone under the fused area at the level of spinal fluctuation, with thickening of the lamina and ligamentum flavum associated with bulging of the posterior portion of the affected articular process. Disc herniation is common in both sets of circumstances. Laminectomy and discectomy may also cause progressive deterioration of the intervertebral disc, with consequent migration of the superior articular process and continued degenerative changes. Scar formation at the operative site may contribute to local stenosis.

III) Foraminal (lateral recess) stenosis, trauma or recurrent inflammation

This leads to hypertrophy and intrusion of the superior facet into the lateral recess.

Central canal stenosis is most common at L2/3, L3/4, L4/5 levels and cause radiculopathy, especially myopathy, often with bilateral lower extremity claudication on exertion (Atlas 1991:850).

Foraminal stenosis can occur when there is a bulging of the intervertebral disc, hypertrophic facets, vertebral body osteophyte formation or inflammation of the ligamentous structures causing encroachment on the neural elements within the foramina. Lateral recess stenosis is usually a result of a hypertrophic superior facet encroaching on the lateral recess causing compression on a nerve root before it exits the neural foramen.

Inferior facet hypertrophy tends to narrow the spinal canal posteriorly by reducing the interlaminar angle (Hammerschlag 1976).

Mikheal **et al.** (1981) defined lateral recess stenosis as present when the distance between the superior facet and the posterior aspect of the vertebral margin is less than four millimetres.

2.3 - MYOFASCIAL PAIN AND DYSFUNCTION

Referred pain from trigger points in the Gluteus Maximus musculature projects into the buttock. Pain from trigger point one extends along the gluteal cleft, including the sacroiliac joint. A patch of pain may spill over onto the adjacent lateral thigh. Trigger point two refers pain to the entire buttock but may also refer pain deep into the buttock. Symptoms from active trigger points in the Gluteus Maximus musculature are aggravated by walking uphill, especially in a forward bent position. Discomfort may be experienced when the patient is seated. (Travell & Simons 2:133-137)

Myofascial trigger points in the Gluteus Medius musculature commonly refer pain into the lower lumbar spine and across the iliac crest on the ipsilateral side. Pain may extend into the midgluteal region, upper thigh posteriorly and laterally. discomfort may be experienced during walking and sleeping on the affected side. Lying supine may painfully compress the posterior Gluteus Medius trigger points. (Travell & Simons 2:151) Pain referred from trigger points in the Gluteus Minimus musculature can be intolerable and excruciatingly severe.

Trigger points in the anterior portion of the musculature project both pain and tenderness to the lower lateral part of the buttock, the lateral aspect of the thigh and knee, and to the peroneal region of the leg as far as the ankle. rarely the referred pain zone may include the dorsum of the foot. Myofascial trigger points in the posterior portion of the musculature may refer pain and tenderness in a pattern that includes most of the buttock (concentrating on the lower medial aspect), and that covers the posterior aspect of the thigh and calf. This referred pain pattern sometimes includes the back of the knee. Symptoms experienced resulting from trigger points in the posterior portion of the Gluteus Minimus Muscle are pain in the hip, causing a limp during walking. Lying on the affected side may be painful, with sleep interrupted as the patient changes position. Difficulty rising from a chair after sitting for a period and standing up straight due to pain if experienced with involvement of the anterior portion of this muscle. The pain may be excruciating and constant. "The patient may not be able to find a stretching movement or change of position that relieved the pain and can neither lie down comfortably nor walk normally." (Travell & Simons 1992 2:171)

2.3.1 - Piriformis syndrome

A sequelae of ipsilateral sacroiliac joint fixation, Myofascial trigger point syndrome within the Piriformis musculature and possible neurological irritation of the Sciatic and Pudendal nerves may refer pain deep into the buttock, posterior and posterolateral thigh, popliteal fossa and lateral foot. If the pudendal nerve is affected, the patient may present with groin pain, inability to sit comfortably on the affected side and, at times, pseudo neurogenic claudication. Onset of these symptoms may occur after a lifting injury with increased axial pressure, sometimes with rotation of the trunk.(Travell & Simons 1992 2:201)

Trigger points located in the Tensor Fasca Lata is known to refer pain to the lateral thigh. Active trigger points in the Extensor Hallucis Longus musculature may refer pain onto the dorsal aspect of the foot and to the big toe. Biceps Femoris, Semimembranosus and Semitendonosus trigger points may refer pain into the buttock, over the ischial tuberosity, along the posterior thigh and into the popliteal fossa. (Travell and Simons 1992 2:171)

2.4 - PATHOGENESIS OF BACK PAIN ORIGINATING FROM THE ZYGAPOPHYSEAL JOINTS

The facets may also be responsible for buttock and greater trochanter pain, pain down the back of the thigh and to the knee occasionally down into the posterior and lateral portions of the foot. (Simmons Jr. **et al**. 1993) "The zygapophyseal joints are probably the most carefully studied synovial joints of the body for specific referred pain patterns." (Travell & Simons 1987 2:24)

2.4.1 - Pathomechanics

The disc and facet combination may only be capable of two to four degrees of rotation, but the forces on the disc at the maximum facet rotation compound as shearing forces which may be the cause of the annular tears. (Goel & Weinstein 1990:165).

2.4.2 - Lumbar and lumbosacral zygapophyseal joints

At the upper lumbar spine, the facet joints are more sagitally orientated, but the lower facet joints are more coronally orientated. The mean angle of the lumbar facet joints relative to the coronal plane is about forty degrees at the fourth lumbar segment and thirty five degrees at the lumbosacral junction. The articular surface of the superior articular process is concave and that of the inferior articular process is convex. The hyaline cartilage that covers the articular surface of each facet joint normally varies in thickness between two to four millimetres. An elastic and fibrous tissue capsule covers the facet joint posteriorly, but there is no capsule anteriorly. Here the facet joint is covered by the ligamentum flavum (Hasegawa **et al.** 1993)

The joint space commonly extends away from the joint along the superior or inferior articular process, below the ligamentum flavum, ventral to the lamina, or under the capsule dorsal to the lamina. A meniscus has been described within the joint space but this is substantiated in only 40 % of adult facet joints from the first lumbar to first sacral segments, as described by Hasegawa **et al.** (1993). The anatomical arrangement of the lumbosacral zygapophyseal joint differs from those joints between the fourth and fifth lumbar vertebra. Each lower lumbar zygapophyseal joint is innervated by the medial branches of two adjacent posterior primary rami. Synovial folds within the zygapophyseal joints have been noted to contain nerve fibres or fasciculi weaving between the fat cells of the subsynovial tissue. Occasionally, encapsulated nerve endings were seen in the fibrous capsule closely related to the attachment of synovial folds. (Bogduk 1990).

Giles (1989) proposes two mechanisms whereby pain may be produced in the synovial folds as a probable result of the nociceptive nerves within the synovial folds.

A - Mechanical pinching of synovial fold tissue, which will result in traction of pain sensitive tissues. Tissue damage with cell rupture and the release of pain producing substances, which results in nerve impulses arising from nociceptors.

B - Traumatic synovitis causing ischaemia of the synovial membrane, with the genesis of ischaemic pain.

Kirkaldy Willis (1984) suggests that the majority of patients presenting with low back pain have the origins of this pain as a result of dysfunction. The term dysfunction implies that at one anatomical level, the three components of the joint the intervertebral disc and two posterior facet joints, are not functioning normally. The author divides the presentation of this condition into three types: rotational or compressive strain as a result of major or minor trauma; pain occurring after unusual activity for that patient; and recurrence of pain due to a very minor episode of trauma.

An episode of trauma (major or minor) causes posterior joint sprain, resulting in small capsular tears. A small degree of joint subluxation takes place causing synovitis because of trauma to the posterior joint synovium. Sustained protective hypertonic contraction of the posterior segmental muscles results in muscle ischaemic and increased perceived pain by the patient. (Kirkaldy Willis (1984)) The changes in this dysfunctional stage affect primarily the facet joints, but small annular tears may also occur. The presenting symptoms and signs may originate primarily from the posterior facet joints and the segmental muscles posterior to them. (Bogduk 1990)

The percentage of weight bearing compressive load transmitted through the articular facets, in persons with normal intervertebral discs, showing no evidence of degeneration and a slightly flattened lumbar lordosis, has been measured at 16 % in two studies (Adams & Hutton (1980); Pal & Routal, (1987)) and between 3 % to 25 % in another (Yang & King 1984). In degenerative joint disease the articular weight bearing in as high as 47 % (Yang 1984) or up to 70 %.

The capsule of the articular facets is richly innervated with sensory fibres. (Bogduk 1983). The posterior primary division of the spinal nerve and recurrent nerve of the anterior primary division innervate the capsule. This sensory nerve supply is sufficiently developed to support the hypothesis that irritation of the capsule of the lumbar articular facets could well produce pain stimuli, which could return to the central nervous system through the posterior primary division, and produce referred pain associate with the dermatomes of the involved nerves. This could possibly correspond to the pathway of sciatic radiation (fourth and fifth lumbar nerves), and this could thus serve to mimic radiculopathy caused by disc entrapment of these nerves.

Ghormly (1983) used the term facet syndrome to describe the sudden onset of low back pain brought on by some activity usually, involving a twisting or rotatory strain of the lumbosacral region. Giles (1989) concluded that in the absence of positive neurological findings and radiological evidence of intervertebral disc prolapse, traumatic synovitis of the synovial folds may be a cause of low back pain, with or without leg pain. Localised reflex muscle spasm is a likely consequence.

The pathological involvement, subluxation or fixation, of the facet joints during disc degeneration, might contribute to the low back pain experienced during lateral entrapment syndrome. If the joint complex has been damaged by torsion then it is likely that the disc and posterior elements are injured simultaneously. Damage to the neural arch may be structural, and observable after computed tomography examination. (Gormley 1983) When rotation is forced, the inferior articular process of the rotated vertebra is forced into the superior facet of the vertebra below, bending the inferior articular process backwards and medially. The pedicle on the opposite side is displaced medially, taking the nerve root with it. Rotation of the pedicle nine degrees could stretch the nerve root up to one centimetre, sufficient to result in compromised function leading to a neuropathic syndrome without disc protrusion/extrusion. (Gormley 1983) "While pathology in and about the foramen may reduce its dimensions and lead to nerve involvement, more likely causes of nerve involvement at this site are friction over osseofibrous irregularities or traction of the nerve or nerve roots fixed in the foramen by adhesions." Korr (1978).

2.5 - PATHOGENESIS OF THE INTERVERTEBRAL DISC

Large portions of the population have disc protrusions but are asymptomatic with respect to pain or dysfunction related to the level of herniation. (Buirski & Silberstein (1993), Chapman Smith (1993), Giles (1993), Rothman (1984), Wiesel (1984)). About 40 % of individuals over the age of forty live asymptomatically with some degree of spinal canal stenosis resultant from disc herniation. (Bullough (1992)). Olmarker **et al.** (1993) feel that one specific pathoanatomical condition related to back pain syndromes, especially sciatic pain, is intervertebral disc herniation.

Jönsson & Stromqvist (1992) suggest that central disc herniation and lateral spinal stenosis affect much the same age groups. They noted that the proportion of females is higher in lateral stenosis, 68 %, as opposed to disc herniation, central stenosis, 44 %. The incidence of root tension was equal in both central and lateral stenosis. Bogduk (1990:72) suggests that disc herniation accounts for fewer than 30 % of presentation of lumbar pain problems, perhaps as few as 5 % or as low as 1 %. The author postulates that the source of pain, in the vast majority of patients, either lies outside the lumbar disc or involves hitherto unrecognised processes within the disc itself.

Delauche-Cavallier **et al.** (1992:928) are of the opinion that the presence of a large herniated nucleus pulposus, in patients with radicular compression without severe neurologic impairment, should not be considered as overwhelming evidence for a surgical procedure, since 66 % of the twenty one patients treated under conservative care, who responded favourably, showed some degree of definite decrease in the size of the herniation six months following treatment. In Low Back Pain. Mechanisms, Diagnosis and Treatment, Cox (1990, 5th ed.) quotes cases where repeat computerised tomographic scans, taken with the patient asymptomatic, has shown no reduction in size of the disc protrusion, compared to that seen on the symptomatic computerised tomographic scan. Other cases show varying degrees of reduction of the disc bulge with the patient attaining total relief of low back and leg pain.

A study of twenty one patients, performed by Delauche-Cavallier **et al.** (1992) showed that reduction was noted in the lumbar intervertebral disc protrusion in fourteen patients. Five of these patients showed a significant decrease in the size of the herniation, four patients displayed a moderate reduction in the herniation. Complete reduction of the herniation was noted in five patients. No change to the structure of the intervertebral disc was noted in the remaining seven patients.Saal & Saal (1989) feel that the presence of a disc extrusion is not an indication for surgery. Maigne **et al.** (1992) are of the opinion that the herniated disc material could be penetrated by granulation tissue and infiltrated by capillary vessels from the epidural space, transforming this tissue into scar tissue.

Secondly, the authors postulate that these fragments are no longer part of the intradiscal space, therefore absorbing water rapidly but also losing this water rapidly, thereby dessicating. Nuclear material can break through the annulus and collect beneath the Posterior Longitudinal Ligament fairly readily, (Ogata & Whiteside 1981), preventing possible dessication of the nuclear fragment described by Maigne **et al.** (1992). Since the nucleus pulposus comprises up to 80 % water, the compression created by the herniation may be a reversible phenomenon, based on the changes in hydroscopic content of the disc. Gradual dessication of the extruded nuclear fragment would decrease the extent of encroachment and serve as an explanation to the loss of radicular symptoms (Maigne **et al.** 1992, Delauche - Cavallier **et al.** 1992).

Added to this phenomenon, inflammation caused by the presence of nuclear disc material may cause a autoimmune mediated response, resulting in direct toxic injury to the nerve root by the chemical mediators of inflammation. Secondary amplification of intra and extra neural swelling, with resultant venous congestion and conduction block may result from these chemical inflammatory mediators. (Bogduk 1990). As the inflammatory response is controlled, the degree to which the above mechanisms play a role in the dynamics of pain production is reduced.

Disc herniation may be simulated by the presence of an extradural haematoma. Lumbar spine haematomas may mimic clinical symptoms and Computerised tomograph appearances of disc herniations (Delauche Cavallier **et al.** 1992). Local circulatory changes secondary to inflammation may cause the extruded nuclear fragment to maintain its osmotic pressure, sustaining the stenotic conditions and concomitant clinical pattern.

Bogduk (1990) is of the opinion that a tear in the annulus would evoke an inflammatory repair response. The chemicals involved would constitute the trigger to nociception in the area. Saal & Saal (1989) are of the opinion that inflammation, because of nuclear material in the epidural space, may lead to nerve injury for two reasons. Firstly, direct toxic injury may occur secondary to chemical mediators. Secondly, intra and extraneural swelling may lead to venous congestion and conduction block. Resolution of inflammation would allow for resolution of irritation. The duration of vascular compromise to the nerve root would be less in this inflammatory situation than in stenosis resultant from osseous entrapment. If stenosis is present and results from osseous structures, then dessication of the intervertebral disc would increase the degree of spinal canal stenosis, possibly furthering the symptom pattern. (Cox 1990).

The lumbar disc is endowed with the necessary apparatus for nociception. Provocation discography has demonstrated that the patients symptoms can be reproduced by provoking the disordered disc with injections of contrast medium or normal saline. (Bogduk (1990)) Olmarker **et al.** (1993) suggest that the nucleus pulposus may induce nerve tissue injury by mechanisms other than mechanical compression.

These mechanisms may be based on direct biomechanical effects of nucleus pulposus components on nerve fibre structure and function and microvascular changes including inflammatory reactions in the nerve roots. In their experiment, Olmarker **et al.** (1993) showed that autologous nucleus pulposus, applied epidurally to the sacrococcygeal cauda equina in hogs, induced profound effects on the structure and function of nerve roots. A marked reduction in nerve conduction velocity, as well as nerve fibre degeneration in spinal nerves exposed to the nucleus pulposus was noted.

Besides mechanical deformation of the nerve roots, it has also been suggested that various tissue components of the intervertebral disc itself, namely nucleus pulposus and annulus fibrosis, might biochemically affect the nerve roots resulting in an autogenic inflammatory response. The authors conclude that epidural application of autologous nucleus pulposus without mechanical compression may induce pronounced changes in the nerve root structure and function. The pathophysiology of such effects of the nucleus pulposus on nerve tissue is likely to be complex, comprising of factors such as directly irritating substances, autoimmune reactions, microvascular changes and increased inflammatory phenomena. (Olmarker **et al.** 1993) Howe **et al.** (1987) feel that sequestrated fragments lying adjacent to a nerve root may stimulate a dilated root sheath, producing variable radicular signs and symptoms.

2.6 - RADICULOPATHY IN THE LOWER LIMB

The original description of sciatica, has been ascribed to Cortugno, who differentiated leg pain with associated neurologic symptoms of numbness or weakness from less discreet leg pain without associated neurologic symptoms (Frymore **et al.** 1991). Lumbar pain is most commonly associated with lumbar canal stenosis, especially nucleus pulposus herniation, which involves noxious stimulation of the anterior primary rami. Pain originating in the lumbar spine has also been related to those structures supplied by the posterior primary rami ie. facet joint capsules and ligaments.These are recognised as other less common causes of radicular\referred symptoms, "sciatica". The pain resulting from the structures innervated by the posterior primary rami is referred to as somatic pain.

Studies completed by Frymore **et al.** (1991) state that 40 % of their male subjects reported leg symptoms. If the subjects experienced low back pain of duration greater than two weeks, the lifetime incidence of sciatica was 11 %.(Mayer 1991:129).

2.7 - REFERRED PAIN vs RADICULAR PAIN

Simmons Jr. **et al.** (1993) define referred pain as pain experienced in a region other than its source of origin. The pain originating from a motion segment may be felt at a site removed from its origin. Deep tenderness of the muscles and altered sensation, usually hyperalgesia, occurs in an area of referred pain. Referred pain may not always be resultant from direct nerve root involvement, but may result form other adjacent structures. (Simmons Jr. **et al.** 1993) In the lumbar spine, pain is often referred to the anterior aspects of the groin, lower abdomen and foot as well as to the posterior buttock, skin over the greater trochanter, down the posterior and lateral aspects of the thigh. (Cox 1990: Cox 1992) The difficulty in identifying the pain is made greater because not only is there overlap of innervation at each level, but also an overlap of pain patterns.

Radicular pain due to nerve root irritation is experienced in a dermatome, myotome or sclerotome, as a result of direct involvement of a lumbar spinal nerve. These phenomena are characterised by perceived objective or subjective sensory, motor or reflex change. Irritation of the spinal nerve root may occur as a result of interaction with the intervertebral disc anteriorly or laterally or the posterior facets posteriorly. (Goel & Weinstein (1990:1-65)) Simmons Jr. **et al.** (1993) are of the opinion that referred pain may not always be resultant from direct nerve-root involvement. Structures that lie within close proximity to the neural canal may give rise to these symptoms and signs. The pain described as numbness and aching is not specifically radicular; it may radiate from the low back downwards or there may be retrograde progression. Neurological examination is often normal or may reveal minimal motor, reflex, or sensory changes. (Camis 1987).Pain develops when the patient is in the upright position or is ambulatory. Mayer **et al.** (1991:129) describes the condition as pseudoclaudication characterised by leg pain, numbness and weakness after the patient has walked short distances. The role of nonoperative management of these clinical conditions remains open for debate. (Saal & Saal 1989)

2.8 - SUMMARY

(1) The clinical signs and symptoms attributed to nerve root entrapment due to compression may in fact originate from rotational facets of the zygapophyseal joints. This latter condition may respond strongly to chiropractic care. An alteration of the intensity of the patients symptoms could be attributed to restoration of normal anatomical relationships between adjacent vertebrae and their soft tissue supports.

(2) Symptoms and signs arise from the posterior joints in several ways:

a - the patient may present with signs and symptoms characteristic of the posterior facet syndrome or with the symptoms and signs of that syndrome in combination with those of a disc herniation.

b - the symptoms and signs of a posterior facet syndrome may be complicated by those of a sacroiliac syndrome.

c - the symptoms and signs may result partly from a posterior facet syndrome and partly from a disc herniation.

This coupled with the possibility that the nerve root is affected by:

a) paravertebral muscle spasm causing referred pain patterns into the low back ,buttock , posterior and lateral thigh down to and sometimes including the calf,

b) irritation of the nerve root by the costotransverse or mammiloaccessory ligaments, as the nerve root passes out of the intervertebral foramen, may prove to be the cause of the radicular symptomatology (motor, sensory and reflex) that the patient presents with.

2.9 ROTATORY MANIPULATION AND FLEXION DISTRACTION IN THE LUMBAR SPINE

Moderately acute lower limb radiculopathy or radiculopathy that has been present for a long period of time presents the best indication for manipulation. These manipulations must be carried out in the pain free direction. Useful manipulation techniques for lower limb radiculopathy include rotation techniques, lateral flexion and forward flexion techniques.

Maigne (1992) suggests measuring the patient's response to Lasegue sign (straight leg raising), before and after the manipulation in order to gauge any degree of change in patient response. He states that in some cases patient response is excellent with an increased straight leg raising from thirty degrees to seventy degrees. The first session may be followed by a painful reaction twelve to twenty four hours after the first manipulation in one third of the cases. Treatment usually includes three to five treatments three to seven days apart. Maigne (1992) suggests further that manipulation," even in severe sciatica" can be of great help in addition to other conservative measures such as bed rest and the use of anti-inflammatory medication. Maigne (1992) suggests that low back pain and\or sciatica persisting after surgery may be helped by manipulation of the lumbar spine. There is an ongoing debate among chiropractic physicians as to the superiority of flexion distraction manipulation versus rotatory manipulation in the treatment of disc lesions. (Hession & Donald 1993)

Patients with low back pain often have absence of motion or abnormal coupling patterns of the lower lumbar spine. The etiology of fixation dysfunction is most likely multifactorial. (Plaugher 1993:198) Therapeutic effects of rotatory manipulation may include release of trapped intraarticular synovial folds. This may explain the often dramatic relief from pain and muscle spasm sometimes experienced. Stretching of the joint capsule, with possible regions of fibrotic scarring of the synovium will lead to increased excursion of the joint. (Giles 1991)

2.9.1 - MECHANISMS OF PAIN RELIEF BY MANIPULATION

Haldeman (1992) proposes a mechanism whereby manipulation is thought to relieve pain which includes a change in the pain threshold, release of muscle spasm, and a reduction in disc protrusion as the primary mechanism for the results of manipulation and an increased range of motion. Restoration of normal motion characteristics reduces the likelihood of persistent stenosis. The tendency towards prolonged compression and adverse mechanical tension on nerve and perineural tissue would be diminished if the pliability of movement returns to the involved articulation. (Lopes 1993:67) "The adjustment is designed to reduce the dyskinesiologic position of the segment. This will normalise the axis of motion around which joints move and decrease tension in the stressed soft tissues. Normalisation of position of individual segments may have an effect of the position of the lumbar spine as a whole." (Plaugher 1993:198-199) Kuo **et al.** (1986) feel that manipulation of the spine can be effective treatment for lumbar disc protrusion, stating that 76.8 % of five hundred and seventeen patients had satisfactory results after receiving manipulation in the lumbar spine. Spinal manipulation has been described by Sandoz, cited by Giles (1991) as a passive manual manoeuvre during which the zygapophyseal joint complex is suddenly carried beyond the physiologic range of movement without exceeding the boundaries of anatomical integrity.

Giles (1991) regard specific rotatory manipulation as essential to mobilize the affected joint in order to provide more rapid relief from pain. Regardless of the mechanism by which the joint becomes fixated, a restriction of mobility is pathologic and should be remedied. Plaugher (1993) states that in the acute stages of injury, the facetal joints and the intervertebral disc may restrict motion at the affected joint. If left in an immobilized state, biomechanical changes may occur. There will be a reduction in glycosaminoglycans and water and an increase in intermolecular collagen crosslinkages. This author is of the opinion that the collagen crosslinkages may be broken down with an adjustment, thus restoring mobility. The concomitant reduction in oedema within the disc or apophysial joints would likely increase mobility and decrease pain by decreasing tension on the pain sensitive outer annular fibres and synovial joint capsule. Bogduk (1990) suggests that impaction of facetal joint surfaces limits the movement of joints about the longitudinal axis to approximately three degrees rotation. If sufficient force is further applied, a lateral shear force on the underlying disc may be introduced.

2.9.2 - RESPONSE OF LUMBAR DISC LESIONS TO MANIPULATION

Since most of the current writing groups herniation of the intervertebral disk almost exclusively with spinal canal stenosis (degenerative stenosis excluded), the treatment of herniated nucleus pulposus with concomitant symptoms, with rotatory manipulation and flexion distraction will be dealt with here. An essential component in manual therapy is presynaptic nociceptive inhibition by proprioceptors after mechanical stimulation of group I, II, III proprioceptors, located primarily in the zygapophyseal joints. A spinal manipulation may selectively stimulate the proprioceptors without nociception stimulation, thereby blocking the perception of pain. (Lopes 1993) Reduction in inflammation may also reduce adverse tension or chemical irritation to neural elements. Lopes (1993) postulates that removal of mechanical irritation by correction of abnormal biomechanics likely interrupts the inflammatory cycle. Increasing motion within an area of fixation or dysfunction with oedema may help circulate inflammatory by products, reducing pressure and chemical irritation.

Henderson (1952) felt that the upset caused by the disc protrusion was essentially subjective. Furthermore, the results of conservative treatment are difficult to assess. In his study of five hundred patients with low back pain and sciatica, four hundred and six patients improved, fifty eight patients had surgery and sixteen patients showed no improvement but were not deemed surgical cases. Henderson (1952) found that of patients with signs and symptoms of nerve root tension, three out of those fifteen patients enjoyed relief from their discomfort. Ten patients had surgical intervention. More than half the patients with little nerve root involvement and moderate to severe signs and symptoms showed dramatic improvement with manipulation of the lumbar spine. A further twenty patients who were receiving exercise therapy and physiotherapy only improved after the lumbar manipulation By way of conclusion, Henderson felt that the mechanism of the manipulation was unclear. (Henderson 1952)

In 1955, Mensor conducted a study of two hundred and eighty five patients with symptoms of protruded lumbar spine intervertebral discs (Mensor 1955). Mensor (1955), concluded that a conservative regimen including manipulative therapy of the lower lumbar spine in intervertebral disc syndromes is an essential part of conservative therapy, although a certain percentage of patients will not be amenable to manipulation. Furthermore, treatment by manipulation should take precedence over surgical intervention unless contraindicated. The author feels that a higher percentage of patients enjoy complete relief from symptoms when undergoing manipulative therapy as opposed to spinal surgery. He felt that satisfactory results should be obtained after one to two manipulations of the lumbar spine. (Mensor 1955)

Chrisman **et al.** (1964) confirmed Mensor's results. Twenty of the thirty nine patients with unequivocal clinical picture of ruptured intervertebral disc had good to excellent results after rotatory manipulation of spine, under anaesthetic. In these patients the appearance of the pre and post treatment myelograms remained unchanged. Ten of the twenty seven patients with positive myelograms showed good to excellent results greater than three years post manipulation. Patients with normal myelographs were shown to do better than those patients with defects detected by myelogram. It was concluded by Chrisman **et al.** (1964) that the immediate subjective effects of manipulation were dramatic in greater than half the patients, saying that the sciatic syndrome symptoms were greatly improved within twenty four hours.

Kou and Loh (1986) examined and treated five hundred and seventeen patients with protruded lumbar spine intervertebral discs. 76.8 % of these patients showed satisfactory results. 14.1 % of the patients had recurrences of their symptoms. Forty seven cases showed no response to therapy. Kou & Loh (1986) concluded that manipulation of the spine is effective treatment for lumbar spine intervertebral disc protrusions. It was noted by these researchers, (Kou & Loh 1986), that the intervertebral disc between the fourth and fifth lumbar vertebrae was the most common site for breakdown of the integrity of the intervertebral disc. The fifth lumbar intervertebral disc was the second most common site, followed by the third lumbar intervertebral disc.

Saal & Saal (1989) show results for the total group treated in their study to include 90 % good or excellent outcome with a 92 % return to work rate. For the subgroups with extruded discs and second opinions, 87 % and 83 % good or excellent outcomes respectively, all of whom returned to work.It was concluded that lumbar herniated nucleus pulposus can be treated nonoperatively with a high degree of success. Saal & Saal (1989) showed successful treatment of disc extrusion in thirteen out of fifteen cases. It is felt by these authors that the optimum time for conservative care before surgical intervention is between six to eight weeks. Failure of passive nonoperative treatment is not sufficient for the decision to operate. The authors believe that the presence of disc extrusion does not adversely affect the outcome of nonoperative therapy and should not be used as overwhelming evidence that surgery is necessary. Studies by Kuo & Loh (1986), Merrill **et al.** (1955), Chrisman **et al.** (1964) and Henderson (1952) all show similar responses to manipulation for the treatment of lumbar spinal canal stenosis resultant from intervertebral disc encroachment.

Weber (1983), reaches the following conclusions in his study:

1. Patients who have unequivocal signs and symptoms and clear cut indications for surgery are best treated operatively.

2. Patients who have certain signs of sciatica and confirmatory tests can be treated conservatively but at least 25 % will require surgical intervention within the year.

3. Patients treated nonoperatively who fulfil liberal criteria for operative intervention will not fare as well as their counterparts for a period of at least one year.

A case report completed by Quon **et al.** (1989) show the authors to be of the opinion that manipulation of the lumbar spine may be an effective form of therapy for some patients with lumbar disc herniation. The authors do caution that some complications of this form of therapy exist.

Posterior to anterior forces applied to a vertebra through its centre of mass, when indicated, are utilized to influence position of disc material that has been displaced posteriorly into the area occupied by neural components, without compromising annular integrity. (Lopes 1993:67) The pain experienced during a disc protrusion may be associated with an increase in stress and strain on the posterior facet joints of the functional spinal unit. This strain related pain might be relieved by the lumbar adjustments and flexion-extension distraction techniques. It is unlikely that the pain experienced could be as a direct result of marginal osteophyte formation projecting to a limited degree into the spinal canal.

In a case report and literature review by Hession and Donald (1993), approaches to treatment for lumbar intervertebral disc herniation are investigated. The authors establish from the clinical trials of Nwuga and Quon and the writings of Adams and Hutton (1980) that the rotational manipulation is unlikely to introduce sufficient torsion into the three joint complex to damage an intervertebral disc. (Hession & Donald 1993) Nwuga's success in treating herniated nucleus pulposus presentation with rotatory adjustment has been echoed by the review of the literature published by Cassidy **et al.** (1993) in which they conclude that the treatment of intervertebral disc herniation by side posture is both safe and effective. Hession and Donald present a case to support the idea that the presence of a disc herniation radiographically does not contradict the use of manipulation. By way of conclusion Hession and Donald write, "the presence of a lumbar disc herniation radiographically does not contraindicate the judicious use of manipulation, provided there is no evidence of cauda equina syndrome or progressive neurological deficit." (Hession & Donald 1993)

Cox **et al.** (1993) note that eighty to 90 % of herniated lumbar disc cases are successfully handled by nonsurgical treatment. Cassidy **et al.** (1993) conclude that in order to fully understand the effects of side posture manipulation on the lumbar intervertebral disc, further research is required.

2.9.3 - RESPONSE OF LUMBAR DISC LESIONS TO FLEXION DISTRACTION

Flexion distraction in the lumbar spine has the postulated effects of increasing spinal canal volume, decreasing the neural ischaemia that results in neural dysfunction. (DuPriest 1993). Neural ischaemia, secondary to soft tissue hypertrophy, may result in intermittent neural compression, relieved by flexion distraction techniques. (DuPriest 1993). Cyriax in 1950 popularised traction for lumbar disc lesions. He proposed that by decompressing the joint, compression strain would be reduced and the symptoms diminished.

Studies done by Christie (1955) into the effectiveness of traction in the treatment of acute and chronic lumbar backache with and without root signs, comparing traction in sixty patients to a bland pill. Dormosan, in sixty patients. The study showed that traction when effective was most effective in chronic backache with root signs. Weber (1983) conducted a double blind study with seventy two patients suffering from sciatica due to prolapsed disc. The treatment group received modulated intermittent traction with a traction force corresponding to one third of the body weight for twenty minutes a day for five to seven days. In the control group he applied a force of seven kilograms sufficient only to tighten the harness. Weber (1983) concluded that comparison between the treated group and control group failed to show any significant difference in pain, mobility of the lumbar spine, or the presence of neurological signs such as weakness, reflex changes or sensory deficit.
Case reports undertaken by Cox **et al.** (1993), Hession and Donald (1993) and Neault (1992) into the efficacy of flexion distraction techniques in the treatment of intervertebral disc herniation have yielded favourable results.

Cox **et al.** (1993:342) conclude that "Chiropractic distraction manipulation is an effective treatment of lumbar disc herniation, if the chiropractor is observant during its administration for patient tolerance to manipulation under distraction and any signs of neurological deficit demanding other types of care." In a discussion by Cox and Aspegren (Cox 1990), the authors reported a 14 % reduction in the disc bulge with complete relief of low back pain and sciatica. Cox reports excellent results utilizing flexion distraction manipulation in thirty one of thirty five cases of lateral herniation and twenty two of twenty six cases of medial herniation.

DuPriest (1993) reports successful management of a patient with lumbar spinal stenosis. The patient received flexion distraction at L4/5 and L5/S1 levels. DuPriest reports that the patient was discharged asymptomatic post treatment. In conclusion, the author feels that the management of spinal stenosis appears to be plausible. Furthermore it is felt that the efficacy of flexion distraction in the lumbar spine cannot be accurately determined. (DuPriest 1993). Neault (1992) in a case study on a single patient with an intervertebral disc prolapse including a sequestrated segment, at the fourth lumbar intervertebral disc on the left, indicated favourable response to conservative care over a six week period. Soft tissue therapy and flexion-extension distraction manipulation were used in the treatment of this case. Neault (1992) feels that the size of the spinal canal in relation to the size of the disc prolapse was an important factor in the outcome of the reported case. Hession and Donald (1991) are of the opinion that the effectiveness of flexion distraction manipulation awaits randomised clinical trial. Theories that the distraction of the lumbar spine draws the nucleus to the centre of the joint is supported by Onel (1993). In eleven out of fourteen medial herniations, the herniated nucleus pulposus regressed during traction. In four of the seven cases of far lateral herniation the lesion regressed during traction. Clinical findings of range of motion, straight leg raising, deep tendon reflexes, sensory impairment and lower extremity muscle power improved in twenty eight of the twenty nine cases studied. (Onel (1993))

2.10 - COMPUTED TOMOGRAPHY IDENTIFICATION

Computed tomography is frequently used as the first line of investigation in cases of suspected lumbar prolapsed intervertebral disc disease. (De Vos Meiring **et al.** 1994). One of the first applications of spinal computer tomography was the evaluation of spinal canal stenosis (Dorwart **et al.** (1983). Hasegawa **et al.** (1993) believe that lateral spinal canal stenosis is a common cause of lumbar radicular symptoms and an indication for radiographic evaluation.

Eisenstein (1976) measured the sagittal diameters of two thousand one hundred and sixty six lumbar vertebra in four hundred and thirty three adult negro and white skeletons and found an overall lower limit of normal sagittal diameter to be fifteen millimetres. Of the two thousand one hundred and sixty six vertebrae, 6.3 % of these vertebrae showed midsagittal stenosis with none less than eleven millimetres. Midsagittal stenosis was found to be twice as frequent as other types.

Changing the tube angle twenty degrees in either direction significantly and irregularly, altered both the pattern and values of anteroposterior diameter and cross-sectional area in both spines. (Bayley **et al.** 1991). This poses potential measurement difficulties if the tube angle is not maintained. Differences between pre and post treatment computer tomographs measurements for the same patient, also between patients, may result providing inappropriate results.

Studies conducted by Wiesel **et al.** (1984) found that 35 % of fifty two asymptomatic patients with respect to low back or leg pain had computerised tomographic scans showing abnormalities including herniated nucleus pulposus. 24 % of people with no history of neither back pain nor sciatica had abnormal myelograms as well. Mikhael **et al.** (1981) defined lateral recess stenosis as present when the distance between the superior facet and the posterior aspect of the vertebral margin is less than four millimetres.Wegener **et al.** (1993:529) suggest relative stenosis to exist when the anteroposterior distance equals a diameter of twelve to fifteen millimetres. The authors regard a diameter of ten millimetres or less as absolute stenosis. Techniques of obtaining these measurements had not been described.

Axial visualisation of the spine afforded by computerised tomography has allowed increased ability to appreciate many aspects of spinal anatomy. The spinal canal and intervertebral canals may be viewed in their entirety, along with their contents. Bulging of disc material, or herniation, and thickening of the ligamentum flavum are well demonstrated by computerized tomography. (Howe **et al.** 1987:273) Howe **et al.** (1987:310) conclude that computerized tomography had become the imaging technique of choice in the diagnosis of disc herniation, particularly in patients who have not undergone previous spinal surgery, although Magnetic Resonance Imaging is also favourable. A study by Bischoff **et al.** (1993) concluded that Myelo-C/T scanning was the most accurate and sensitive in the diagnosis of herniated nucleus pulposus. A report by Epstein **et al.** (1990) concluded similar results.

The computerised tomographic scan can help to identify conformational changes contributing to more focal stenotic processes, especially in the lateral recesses and subarticular regions (Delauche-Cavallier **et al.** 1993)

The costotransverse ligament was identified using axial computed tomography, using both soft tissue windows and negative images. Computed tomography blink mode may be of benefit in differentiating the costotransverse ligament from the adjacent soft tissue. (Church **et al.** 1991) Hasagawa **et al.**(1993) feel that transaxial computerised tomographic scans are not effective in demonstrating the lumbar intervertebral foramen.

2.10.1 - BULGING ANNULUS

Usually symmetrical but occasionally asymmetrical. There is a generalised extension of the disc margin beyond that of the adjacent vertebral bodies, usually resulting in convexity of the midportion of the posterior disc margin. This is in contrast to the central concavity that characterises the normal disc margin. When asymmetrical, the disc bulge may simulate a small herniation of the nucleus pulposus. (Howe **et al.** 1987) (Plate 1 - Bulging Annulus)

This phenomenon is felt to be fairly common, especially with increasing age (Howe **et al.** 1987). Since neurological structures are rarely compromised, disc bulges are of questionable clinical significance. (See Plate 1)

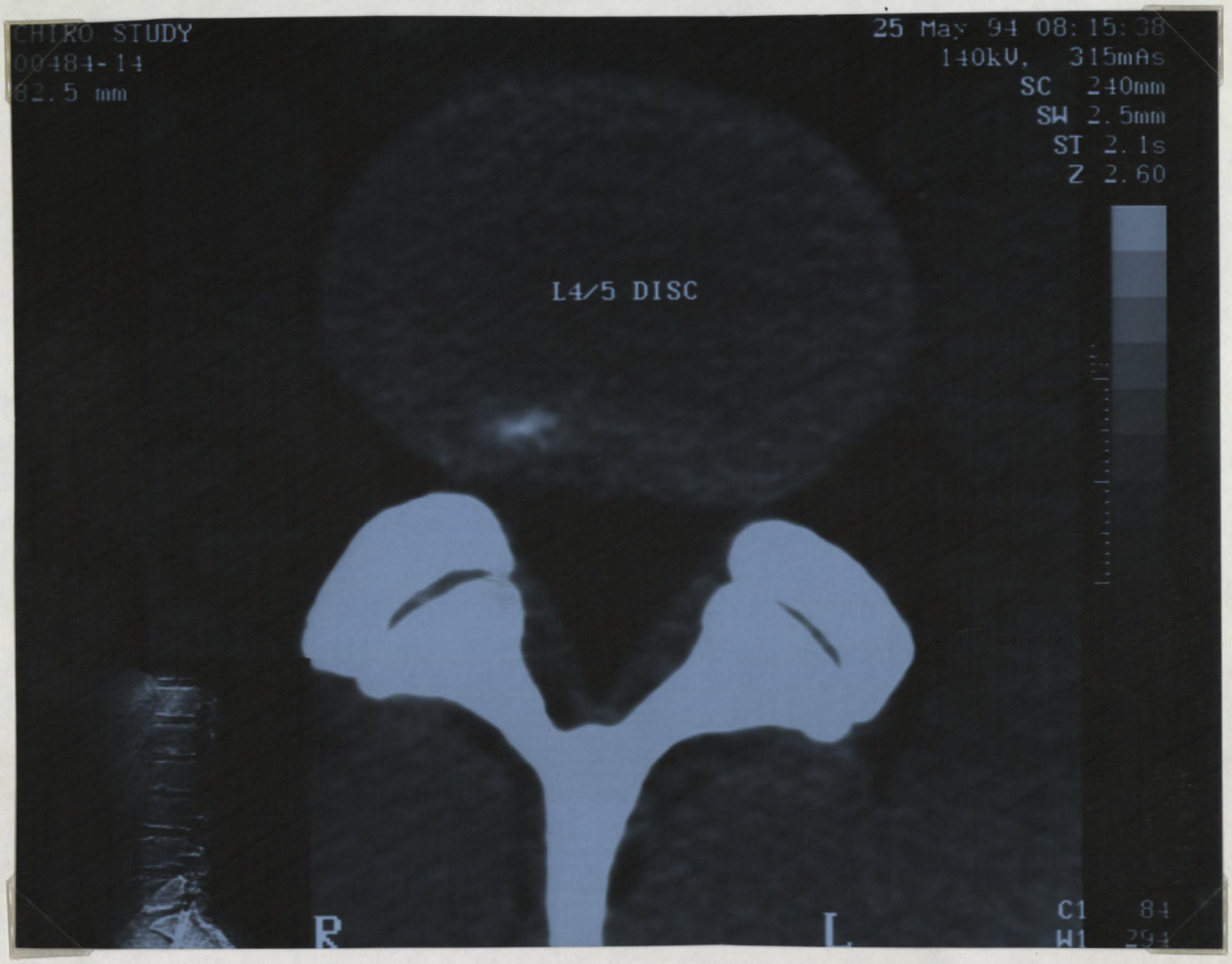


PLATE 1: BULGING ANNULUS

2.10.2 - HERNIATED NUCLEUS PULPOSUS

Actual herniation or prolapse of nuclear material through the annulus is a relatively common cause of low back pain, often associated with sciatica. Howe **et al.** (1987) feel that computerised tomography, myelography and magnetic resonance imaging are definitive for this diagnosis. On computerised tomography the diagnosis of disc herniation is made easier by the abundant epidural fat at the anterior of the dural sac.

The authors are of the opinion that deformity of the dural sac by the herniated nucleus pulposus is fairly common and is afforded clinical significance. It is felt by these authors that since the annulus is thinner posteriorly and the posterior longitudinal ligament is not substantial, protrusion of the nucleus pulposus through the annulus fibrosis most commonly occurs posteriorly or posterolaterally. Central herniations will probably not be accompanied by radiculopathy, although low back pain may be associated. (See Plate 2) This may be a result of the sensory innervation to the meninges, posterior longitudinal ligament and outer two thirds of the annulus fibrosis. (Howe **et al.** 1987, Bogduk 1990).

Herniated nuclear material beneath the posterior longitudinal ligament produces focal protrusion, well demonstrated on computerized tomographic scans. At the level of the fourth intervertebral disc, deformity of the dural sac by the herniated nucleus pulposus is common and is afforded clinical significance. At the level of the fifth intervertebral disc where the epidural space is greater, herniated nucleus pulposus may not cause dural impingement, making its significance less certain. (Howe **et al.** 1987:287315). It is felt by Saal & Saal (1989) that local circulatory changes secondary to inflammation could cause an extruded disc fragment or contained herniation to maintain high water content, maintaining the compressive injury. Reduction of the inflammation would reduce the size of the presenting herniation.

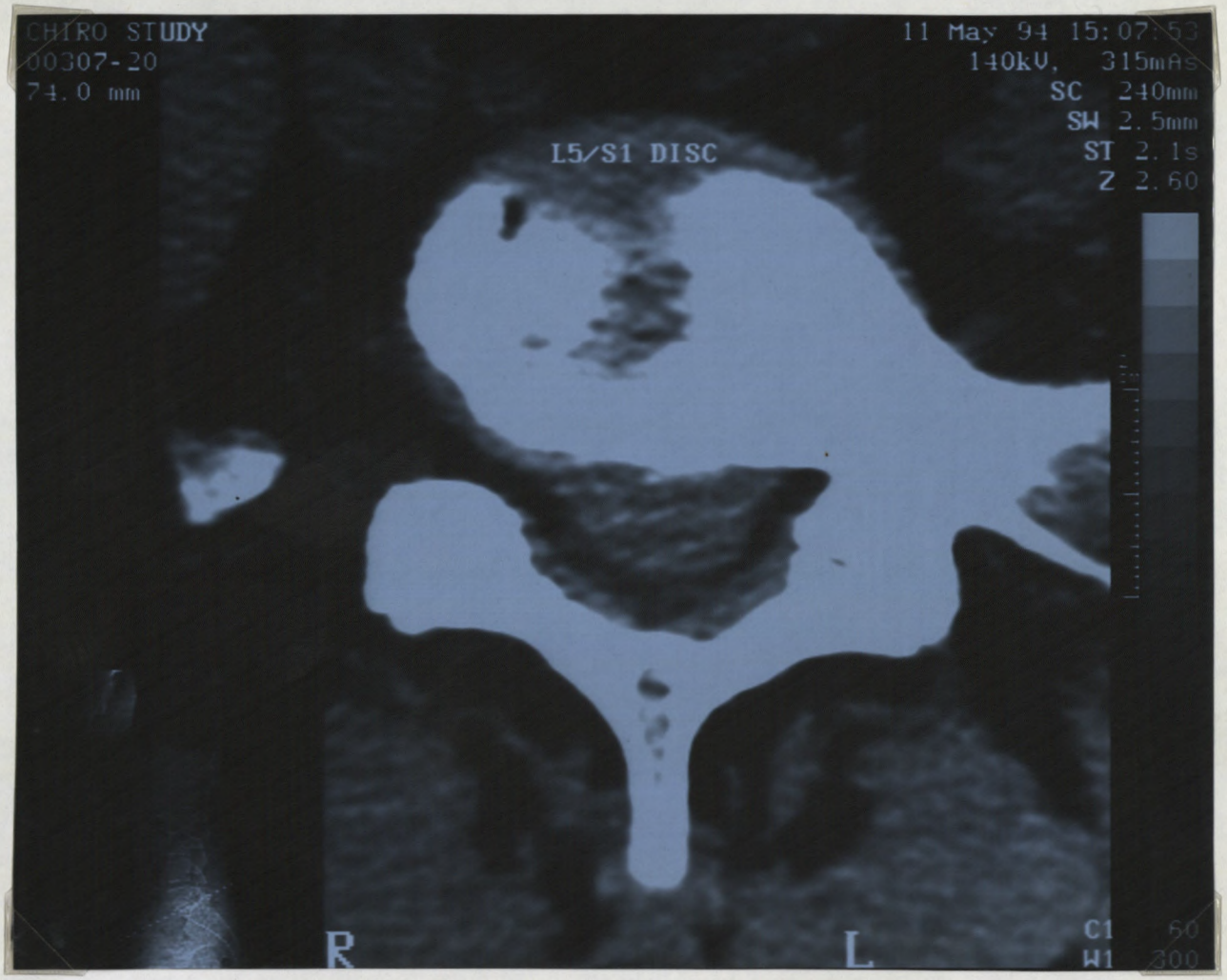
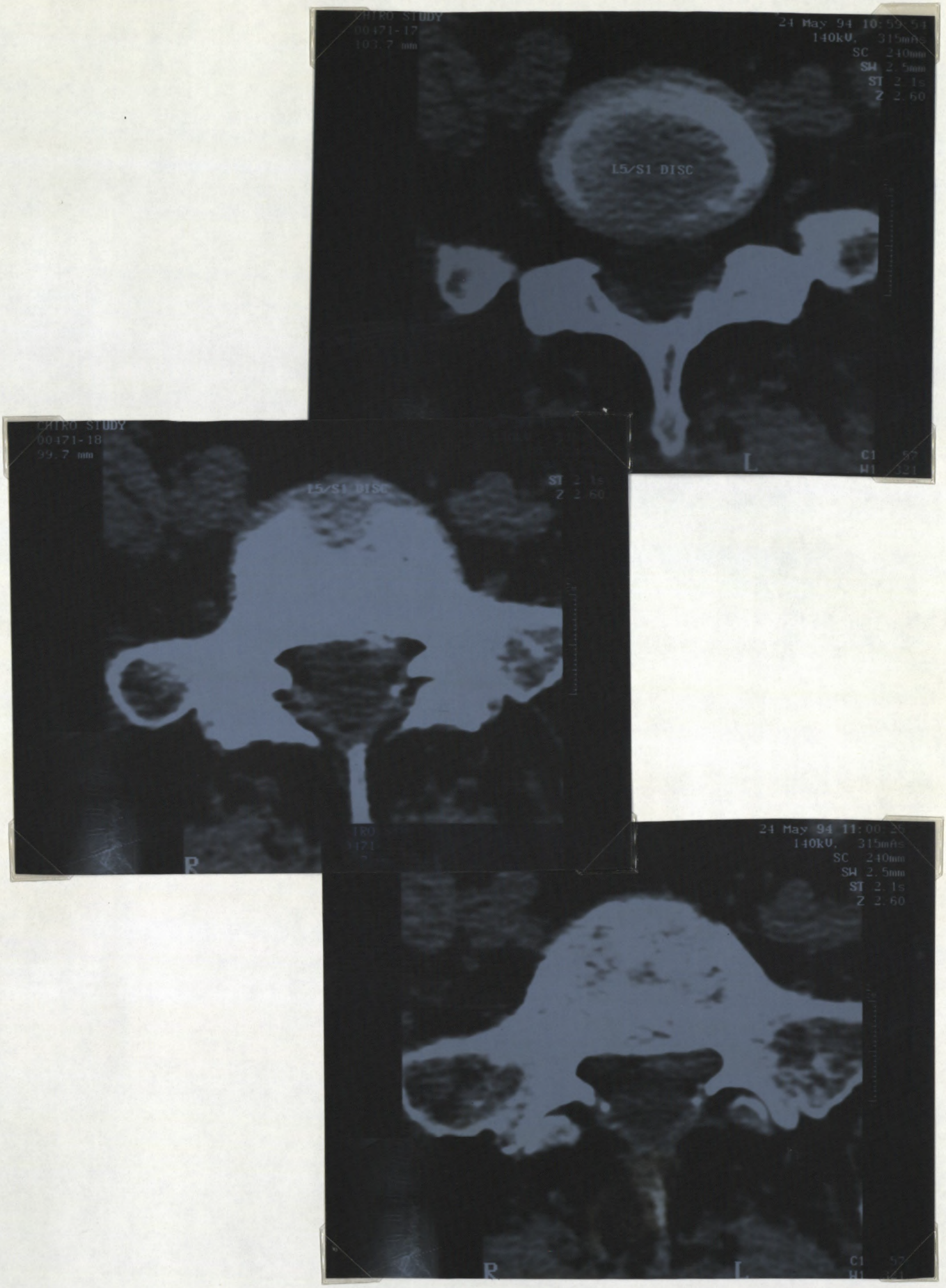


PLATE 2 - HERNIATED NUCLEUS PULPOSIS

2.10.3 - DISC EXTRUSION / SEQUESTRATION

Nuclear material that has passed posteriorly through, or around, the posterior longitudinal ligament and then separated from the bulk of the protrusion is referred to as a sequestrated fragment. These free fragments are thought to be relatively rare and almost exclusively exist in the lumbar spine. Once free in the epidural space, the free fragment may migrate cranially or caudally. (Howe **et al.** 1987) (See Plate sequence 3-5)

The author feels that the computerised tomographic scans should include more than just the sections through the disc interspaces so as to record these sequestrations. The lateral recesses should also be demonstrated for better viewing sequestrated material.



PLATES 3-5 - SEQUESTRATED NUCLEAR FRAGMENT

2.11 MEASUREMENT TECHNIQUE USING COMPUTED TOMOGRAPHY

Lateral recess measurements have been well described on computerised tomography, as described by Hasegawa **et al.** (1993) in their paper on imaging anatomy of the lateral lumbar spinal canal. A height of five millimetres or more in the lateral recess is regarded as falling within normal limits. A height of three to four millimetres suggests lateral recess stenosis. Since the images are derived from digital matrices, accurate measurements can be made using computerised tomography. The significance of these measurements may be questioned in light of clinical findings. (Howe **et al.** 1987:273 315)

Cox (1990:261) proposes a technique to measure the disc protrusion size and to evaluate the size of the bulge. The technique involves obtaining three consecutive parallel two millimetre cuts through the disc with the gantry angulation set to obtain axial scans in the plane of the disc. Perpendicular sections to the rostocaudal axis may also be best. These cuts were repeated at later dates for comparison. The first measurement is from the posterior margin of the vertebral body to the most posterior aspect of the disc bulge (A). The second measurement is from the posterior margin of the vertebral body to the posterior spinal canal where the laminae join with the spinous processes (B). These two measurements are used to form a percentage (A\B). (See Plate 6) The three disc-bulge-to-spinal-canal percentages obtained were then averaged for each date. The sagittal diameter of the spinal canal may be measured by computed tomography, with cursors placed on the posterior surface of the vertebral body and anterior surface of the lamina.This technique was first presented by Cox & Aspegren in 1987.

At the level of the fourth intervertebral disc, deformity of the dural sac by the herniated nucleus pulposus is common and is afforded clinical significance. At the level of the fifth intervertebral disc where the epidural space is greater, herniated nucleus pulposus may not cause dural impingement, making its significance less certain. (Howe **et al.** 1987:273 - 315)

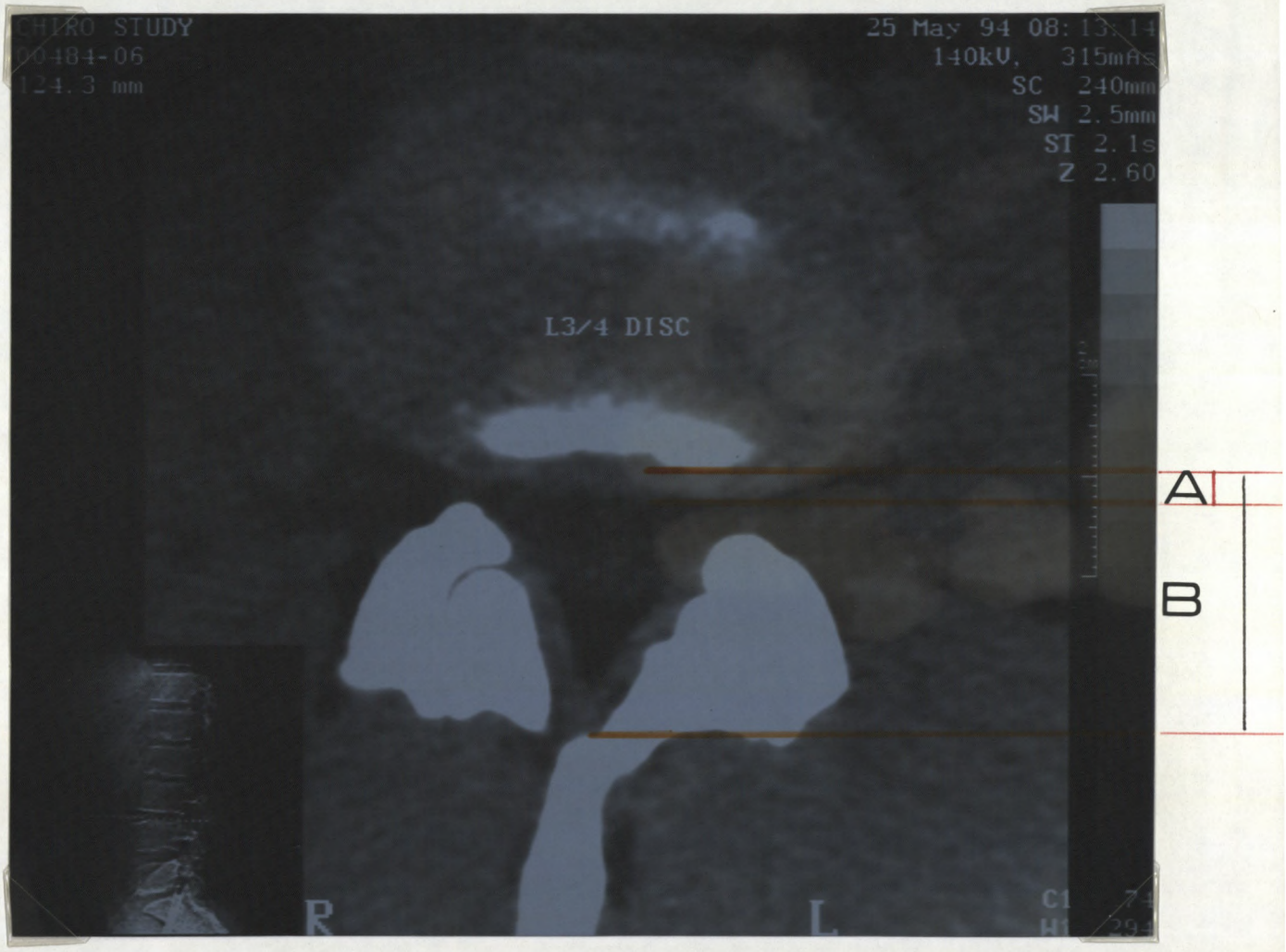


PLATE 6 - MEASUREMENT PROCEDURE FOR OCCUPANCY OF INTEVERTEBRAL DISC IN SPINAL CANAL.

CHAPTER THREE

MATERIALS AND METHODS

INTRODUCTION

The following chapter provides a detailed overview of the nature, location, capture, processing and statistical evaluation of the data that has been used in this dissertation for the year 1994 from the months spanning May to December.

3.1 - METHODS, TECHNIQUES AND MEASUREMENTS

3.1.1 - PROCESS OF RANDOMISATION

3.1.1.1 - SAMPLE SELECTION

1 All patients had to satisfy the delimitations described below. This study did not include patients with clinical presentations indicative of:

a) cauda equina syndrome,

b) conditions constituting a surgical emergency,

c) congenital foraminal stenosis requiring aggressive therapy,

d) infectious diseases of the spine,

e) neoplasms,

f) sequestrated nuclear fragments,

g) stenosis as result of abnormal osteological growth that poses a surgical emergency.

All patient accepted presented to the outpatient clinic at Technikon Natal with low back pain and lower limb pain unilaterally or bilaterally.

2 - Patients were divided into blocks of four. Two patients in every four received rotatory adjustments of the lumbar spine as the focus of the therapy and two patients received McManis traction of the lumbar spine.

3 - It was assumed that the variables under consideration were distributed as normal.

4 - Let the treatment = T

Let the control = C

(i)TTCC	(ii)CCTT	(iii)TCCT
(iv)TCTC	(v)CTCT	(vi)CTTC

A sequence of random numbers was then chosen by tossing a die, which precipitated the allotment of patients into the two sample groups.

3.1.1.2 - PATIENT MANAGEMENT

All patients were screened before the initial consultation, excluding those patients with conditions delimited by the constraints of the research framework. Once accepted into the study group of thirty patients, each patient was then allocated into one of two treatment groups of equal number, using random assortment.

3.1.1.3 - PATIENT PROCEDURE

A letter of introduction and a brief explanation of procedures to be followed during the treatment period were provided prior to the initial examination and inclusion into the study group.

The informed consent form was completed by all participants in the research in both categories, before any treatment was initiated.

Patients completed a Numerical Rating Scale101, (appendix E), an Oswestry Low Back Pain Disability Index, (appendix D), and a Pain diagram, (appendix F), before the initial treatment proceeded.

3.1.1.3.1 - NUMERICAL RATING SCALE 101

Once the treatment period was initiated, the patient completed a Numerical Rating Scale 101 at each consultation, before treatment commenced, in order to assess the progression of the treatment with respect to the patient's perception of pain.

3.1.1.3.2 - OSWESTRY LOW BACK PAIN DISABILITY INDEX

These questionnaires were completed prior to initiation of the treatment period, and again once the treatment period was been completed. Fairbank **et al.** (1980) found the Oswestry Low Back Pain Disability Index to be a valid indicator of disability. The authors concluded that the Oswestry questionnaire avoids observer bias and ensures uniformity of presentation. The Oswestry Low Back Disability Index was reported by Fairbank **et al.** (1989) to be reliable, if its score closely reflects the patientís observed disability and symptoms.

3.1.1.3.3 - COMPUTED TOMOGRAPHY

The duration of the treatment period was 21 days during which time the patient attended a total 9 consultations: the initial consultation and 8 followup consultations. An initial computer tomographic examination of the lumbar spine from the level of the third lumbar intervertebral disc to the second sacral segment was performed prior to initiation of treatment. Follow up computed tomography examinations of affected areas, identified on each patient individually, were performed after the treatment period.

3.2 - CAPTURING AND SECURING THE DATA

3.2.1 - OBJECTIVE DATA CAPTURE

The first objective was to determine the effectiveness of chiropractic care, in the management of low back pain with radiculopathy, in terms of objective clinical findings in order to assess the effects of chiropractic treatment on recognised selected diagnostic criteria. The data needed was obtained using clinically acceptable orthopaedic tests. Patients underwent examination during each consultation at which time all the relevant orthopaedic tests were repeated. Results were recorded by the clinician examining the patient. Examination of the lumbar spine included protocol as laid out in the Chiropractic Clinic, Technikon Natal, Durban. (Appendix B, C)

3.2.1.1 - Orthopaedic tests performed

1)PATRICK FABERE

2)GAENSLEN'S TEST

3)ERICHSEN'S TEST

4)STRAIGHT LEG RAISE

5)THOMAS'S TEST

6)PHEASANT'S TEST

Only the patient responses, as measured by the examiner during the initial and final consultations, were used for statistical evaluation. All the data needed for statistical evaluation was collected as measured responses to clinical orthopaedic and diagnostic tests that the patients performed in the presence of the clinician. No control group was used. Two differing techniques were investigated. The patients in both groups received soft tissue therapy for the management of muscular spasm and pain relief, including myofascial therapy. Those patients divided into group one received rotatory adjustments in the lumbar spine comprising of Diversified Adjustment techniques embracing either a spinous push or spinous pull technique executed in the lateral recumbent position. (Gitelman & Fligg 1992:483-502) Patients assigned to group two received Mcmanis flexion extension distraction techniques in the lumbar spine.

Each orthopaedic test that proved positive, as a result of pain, received a score of five points. Each orthopaedic test that was negative with respect to pain received a score of zero points. A total combined positive pain score of thirty points was the maximum score obtainable, and a total combined score of zero was the minimum score obtainable. Once the scores for the initial and final examinations were received, they were converted to percentages and statistically analysed. Statistical evaluation of each set of patient scores was undertaken using PAIRED-t tests.

3.2.2 - SUBJECTIVE DATA CAPTURE

The second objective was to determine the effectiveness of chiropractic care, in the management of low back pain with radiculopathy, in terms of subjective clinical findings in order to assess the patient's perception of the treatment. The data needed was obtained from subjective patient responses, during a detailed case history, as is in use at the Chiropractic Clinic, Technikon Natal, Durban (Appendix A). Subjective responses were recorded on pain diagrams (Appendix F), Numerical Pain Rating Scale 101 questionnaires (Appendix E) and Oswestry Low Back Pain Disability Index questionnaires (Appendix D).

3.2.2.1 - NUMERICAL RATING SCALE 101

For the Numerical Rating Scale101, patient response was noted at every consultation prior to treatment, from the initial consultation until the final consultation. Screening of all the questionnaires was done to ensure that all questionnaires were filled out correctly. Patients completed one form before every consultation, prior to receiving treatment for their condition. This served to record the patient's perception of pain prior to initiation of that treatment. The mean of each patient was calculated for all nine consultations. The data obtained during the initial and final consultations has been statistically analysed using parametric PAIRED-t tests for each of the patient sample groups. Statistical comparison was made for the treatment period, measuring rate of change, within each sample group, and between the two sample groups using UNPAIRED-t tests.

3.2.2.2 - OSWESTRY LOW BACK PAIN DISABILITY INDEX

For the Oswestry Low Back Pain Disability Index all the patient responses were measured at the initial consultation and at the final consultation. A maximum score of five points could be scored in each of the sections (one through ten) and a minimum score of zero was obtainable for each section. Once all the sections had been completed the total patient score was divided by the maximum score of fifty to obtain a percentage figure. This was done for the initial as well as final consultations and the percentages were correlated and statistically evaluated. Scores were correlated for initial and final consultation questionnaires. The percentages were correlated, statistically evaluated using PAIRED-t tests. PARAMETRIC UNPAIRED-t tests were used to draw statistical comparison between the two treatment groups.

3.2.2.3 - PAIN DIAGRAMS

Pain diagrams were completed by patients, during the initial screening procedure. Pain diagrams are an effective aid to the patient in expressing themselves. The Pain diagram is effective for feedback and patient follow-ups. (Margoles (1980))

3.2.3 - COMPUTERISED TOMOGRAPHIC SCANNING

The third objective was to determine the effectiveness of chiropractic care, in the management of low back pain with radiculopathy, in terms of any alteration in the size of the spinal canal (central canal and\or intervertebral foramina) in order to determine the effects of chiropractic treatment on size alteration of the spinal canal. The data needed was obtained using pre and post treatment computerised tomographic examinations of the affected areas of the lumbar spine. An ELSCINT 2400 elite Computed Tomography scanner was used for all examinations and subsequent measurement procedures. All the spinal canals were measured for their anteroposterior diameters Alteration in size of the disc protrusion were measured preceding and succeeding treatment.

Measurement technique involved obtaining three consecutive parallel four millimetre cuts through the vertebral end plates and the intervertebral disc with the gantry angulation set to obtain axial scans in the plane of the disc. These cuts were repeated at later dates for comparison. The measurements were used to form a percentage. The disc-bulge-to-spinal-canal percentages obtained were then averaged for each date. The first measurement was taken from the posterior margin of the vertebral body to the most posterior aspect of the disc bulge. The second measurement was taken from the posterior edge of the vertebral body to the posterior spinal canal, where the laminae join with the spinous processes. In an attempt to standardise the measurement procedure, magnification of two point six zero was used in all viewing and measuring procedures. Tube angulation was not in excess of ten degrees, between the pre and post treatment examinations of any of the pairs of tomographs examined. Bayley **et al.**(1991) feel that changes to the tube angle of less than twenty degrees caudally or cephalad, is unlikely to significantly alter the pattern or absolute values of the anteroposterior diameter and cross sectional area in the spine. Swivel was reduced to between zero and zero point two degrees.

Data recorded from the pre and post treatment computer tomographic examinations, within each treatment group, was statistically integrated using PAIRED-t tests. Data comparing the two treatment groups both pre and post treatment has been analysed using UNPAIRED-t tests. Measurements of the spinal canal, as well as those denoting intervertebral disc size, have been used for statistical evaluation.

CHAPTER FOUR

RESULTS

This was not a blinded study.

The author performed the clinical examinations and orthopaedic testing. Measurements of the intervertebral discs and spinal canals were performed by same. Personal interpretation in location of exact extent of the herniated discal material is difficult. Attempts were made to err in favour of conservative measurements.

It should be noted that at the time the percentage occupancy measurements were performed, no direct correlation to case presentation was available to the author. Cases were numbered individually by the radiologists. These number sequences were used during measurement procedures.

4.1. DEMOGRAPHIC DATA

TABLE 1: DEMOGRAPHIC DATA

RACIAL GROUP	NUMBER TREATED	%
BLACK	2	6.67%
COLOURED	1	3.33%
INDIAN	12	40.00%
CAUCASIAN	15	50.00%
TOTAL	30	100.00%

The ratio of females to males was twelve males to eighteen females. Mean age of the treatment group was noted at forty four point zero three years. The youngest patient was twenty eight years of age as of January 1994. The oldest patient was aged seventy years as of January 1994.

Age distribution and the mean age noted in this study compare favourably with studies presented by Bush **et al.** (1992), Buirski & Silberstein (1993), Chrisman **et al.** (1964), Jonsson & Stromqvist (1993), Kuo & Loh (1986), Mensor (1955), Saal & Saal (1989), Weber **et al.** (1993).

No studies reviewed have made mention of racial distribution. Gender distribution corresponds loosely to the studies mentioned in the previous paragraph.

TABLE 2:ORTHOPAEDIC TESTS - RESULTS

TEST		Mean. Difference Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ERICHSEN'S TEST	ADJ.	2.67	2.58	15	4.000
	MNS.	3.00	2.54	15	4.583
GAENSLEN'S TEST	ADJ.	2.33	2.58	15	3.500
	MNS.	2.00	2.54	15	3.055
PATRIC FABERE	ADJ.	2.33	2.58	15	3.500
	MNS.	3.00	2.54	15	4.583
PHEASANT'S TEST	ADJ.	1.00	2.07	15	1.871
	MNS.	2.33	2.58	15	3.500
STRAIGHT LEG RAISE	ADJ.	3.33	2.44	15	5.292
	MNS.	2.00	2.54	15	3.055
THOMAS'S TEST	ADJ.	0.33	1.29	15	1.000
	MNS.	2.00	2.54	15	3.055

$t_{14;0,05} = 1,761$

ADJ - ADJUSTMENT GROUP
MNS - MCMANIS GROUP

In only one case is there no statistically significant change at the 5% significance level. This is noted in the Adjustment group for Thomas's Test. This result may be due to the small sample size. All other cases show statistically significant bettership, following treatment, at the 5% level of significance.

4.3. SUBJECTIVE CLINICAL TRIALS

4.3.1 OSWESTRY LOW BACK PAIN DISABILITY INDEX

TABLE 3: OSWESTRY LOW BACK PAIN DISABILITY INDEX - ONE SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	7.93	32.91	15	0.934
McMANIS	17.13	30.22	15	2.196

$t_{14;0,05} = 1,761$

TABLE 4:OSWESTRY LOW BACK PAIN DISABILITY INDEX - TWO SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	48.40	19.02	15	2.106
INITIAL McMANIS	35.33	14.69	15	

$t_{28;0,05} = 1,701$

TABLE 5:OSWESTRY LOW BACK PAIN DISABILITY INDEX - TWO SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	31.27	31.78	15	0.333
FINAL McMANIS	27.40	31.89	15	

$t_{28;0,05} = 1,701$

OSWESTRY PAIN DISABILITY INDEX - STATISTICAL EVALUATION

At 5% level of significance, the McManis group showed bettership that may be regarded as statistically significant. The Adjustment group showed no statistically significant changes. This is thought to result from the large variation in scored percentages obtained. The two sample testing at 5% significance level showed no statistically relevant differences, when comparing the final Oswestry Low Back Pain Disability Index scores between the two treatment groups.

4.3.2 NUMERICAL RATING SCALE 101

TABLE 6:NUMERICAL RATING SCALE 101 - ONE SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	35.10	23.16	15	5.871
McMANIS	32.30	30.68	15	4.077

$t_{14;0,05} = 1,761$

TABLE 7:NUMERICAL RATING SCALE 101 - TWO SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	59.80	19.41	15	0.867
INITIAL McMANIS	54.00	16.74	15	

$t_{28;0,05} = 1,701$

TABLE 8:NUMERICAL RATING SCALE 101 - TWO SAMPLE RESULTS

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	27.50	36.20	15	0.706
FINAL McMANIS	18.90	30.31	15	

$t_{28;0,05} = 1,701$

NUMERICAL RATING SCALE 101 - STATISTICAL EVALUATION

At 5% level of significance, both Adjustment and McManis groups showed bettership that may be regarded as statistically significant. Two sample testing showed no statistical significant differences between the two treatment groups, at the 5% significance level.

4.4. COMPUTED TOMOGRAPHY

4.4.1 FOURTH INTERVERTEBRAL DISC AND SPINAL CANAL

TABLE 9:MEASUREMENTS OF SPINAL CANAL L4/5 - ONE SAMPLE (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	-0.21	0.65	8	-0.869
McMANIS	-0.08	0.49	7	-0.430

$t_{7;0,05}$ = 1.895 ADJUSTMENT GROUP

$t_{6;0,05}$ = 1.943 McMANIS GROUP

No statistical bettership was noted within either treatment group at a 5% level of significance.

TABLE 10: MEASUREMENTS OF INTERVERTEBRAL DISC L4/5 - ONE SAMPLE (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	-1.05	1.94	8	-1.431
McMANIS	1.14	1.18	7	2.751

$t_{7;0,05}$ = 1.895 ADJUSTMENT GROUP

$t_{6;0,05}$ = 1.943 McMANIS GROUP

No statistical bettership was noted within the Adjustment group at a 5% level of significance. Bettership of statistical significance occurred in the McManis treatment group at this intervertebral disc level, at a 5% level of significance, with respect to alteration of intervertebral disc size. No procedural nor anatomical explanation can be given as to this observed phenomenon.

TABLE 11:PERCENTAGE OCCUPANCY OF SPINAL CANAL BY INTERVERTEBRAL DISC (L4/5) - ONE SAMPLE MEASUREMENT

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	5.93	5.76	8	2.908
McMANIS	-4.10	10.43	7	-1.041

No statistically significant bettership, at the 5% significance level, was noted in either treatment group following completion of the prescribed period of conservative care. The statistical test performed was a left tailed test.

TABLE 12:MEASUREMENTS OF SPINAL CANAL L4/5 - PRE TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	18.64	1.93	8	-0.206
INITIAL McMANIS	18.85	1.94	7	

$t_{13;0,05} = 1.771$

TABLE 13:MEASUREMENTS OF SPINAL CANAL L4/5 - POST TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	18.72	2.11	8	-0.327
FINAL McMANIS	19.06	1.96	7	

$t_{13;0,05} = 1.771$

At a 5% level of statistical significance, there is no appreciable difference between the two treatment groups, before or after completion of the treatment period. No bettership was noted between the pre and post treatment canal measurements, at a 5% level of significance.

TABLE 14:MEASUREMENTS OF INTERVERTEBRAL DISC L4/5 -PRE TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	6.36	2.32	8	1.437
INITIAL McMANIS	4.82	1.74	7	

$t_{13;0,05} = 1.771$

TABLE 15:MEASUREMENTS OF INTERVERTEBRAL DISC L4/5 - POST TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	5.23	2.20	8	-0.444
FINAL McMANIS	5.87	3.44	7	

$t_{13;0,05} = 1,771$

At a 5% level of statistical significance, there is no appreciable difference between the two treatment groups before or after completion of the treatment period.No bettership was noted between the pre and post treatment disc measurements, at a 5% level of significance.

TABLE 16:PERCENTAGE OCCUPANCY OF THE SPINAL CANAL BY INTERVERTEBRAL DISC (L4/5) - TWO SAMPLE TESTING

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	28.55	12.91	8	0.008
FINAL McMANIS	28.49	19.78	7	

$t_{13;0,05} = 1,771$

Alteration in the percentage occupancy of the spinal canal by the intervertebral disc at the L4/5 level was found to be not statistically significant at a 5% level of significance.

4.4.2 FIFTH INTERVERTEBRAL DISC AND SPINAL CANAL

TABLE 17:MEASUREMENTS OF SPINAL CANAL L5/S1 - ONE SAMPLE (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	0.43	1.00	6	1.216
McMANIS	-0.41	0.92	8	-1.084

$t_{5;0,05}$ = 2.015 ADJUSTMENT GROUP

$t_{7;0,05}$ = 1.895 McMANIS GROUP

No statistical bettership was noted within either treatment group at a 5% level of significance.

TABLE 18:MEASUREMENTS OF INTERVERTEBRAL DISC L5/S1 - ONE SAMPLE (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	1.32	1.59	6	2.358
McMANIS	0.22	1.06	8	0.499

$t_{5;0,05}$ = 2.015 ADJUSTMENT GROUP

$t_{7;0,05}$ = 1.895 McMANIS GROUP

No statistical bettership was noted within the McManis treatment group at a 5% level of significance. Bettership within the Adjustment group with regard to alteration in the size of the intervertebral disc, is noted to be statistically significant at the 5% significance level.

TABLE 19:PERCENTAGE OCCUPANCY OF THE SPINAL CANAL BY INTERVERTEBRAL DISC (L5/S1) - ONE SAMPLE

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
ADJUSTMENT	2.20	6.00	6	0.899
McMANIS	8.06	9.36	8	2.434

No statistically significant bettership, at the 5% significance level, was noted in either treatment group, following completion of the prescribed period of conservative care. The statistical test performed was a left tailed test.

TABLE 20:MEASUREMENTS OF SPINAL CANAL L5/S1 - PRE TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	15.39	1.42	6	-2.148
INITIAL McMANIS	17.91	2.58	8	

$t_{12;0,05} = 1.782$

TABLE 21:MEASUREMENTS OF SPINAL CANAL L5/S1 - POST TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	15.80	1.71	6	-1.186
FINAL McMANIS	17.48	3.12	8	

$t_{12;0,05} = 1.782$

At the 5% level of statistical significance, there is no appreciable difference between the two treatment groups pre and post treatment. It would appear that there is no statistically significant bettership in the spinal canal size post treatment, at the 5% significance level.

TABLE 22:MEASUREMENTS INTERVERTEBRAL DISC L5/S1 - PRE TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
INITIAL ADJUSTMENT	4.25	1.58	6	-1.501
INITIAL McMANIS	5.86	2.23	8	

$t_{12;0,05} = 1.782$

TABLE 23:MEASUREMENTS INTERVERTEBRAL DISC L5/S1 - POST TREATMENT TWO SAMPLE TESTING (MEASURED IN MILLIMETERS)

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	4.03	1.43	6	-0.654
FINAL McMANIS	4.53	1.39	8	

$t_{12;0,05} = 1,782$

At the 5% level of statistical significance, there is no appreciable difference between the two treatment groups, pre and post treatment.It would appear that there is no statistically significant bettership in the apparent size of the herniated nuclear material post treatment, at the 5% significance level.

TABLE 24:PERCENTAGE OCCUPANCY OF THE SPINAL CANAL BY INTERVERTEBRAL DISC (L5/S1) - TWO SAMPLE TESTING

	Mean. Descriptive Statistics	Standard Deviation	Sample Size	Critical Value(T)
FINAL ADJUSTMENT	25.16	7.66	6	-0.763
FINAL McMANIS	28.21	7.21	8	

$t_{12;0,05} = 1,782$

Alteration in the percentage occupancy of the spinal canal by the intervertebral disc at the L5/S1 level was found to be not statistically significant at a 5% level of significance.

NOTE

Small sample sizes lend themselves towards non parametric testing. The sample sizes in this study do not lend themselves towards parametric testing. It was not anticipated at the inception of this study, that the subgroups of each level would provide such small sample sizes, as a result of the small number of patients included.Since the sample sizes were small, and parametric testing was used, tabulated values, as stipulated by Stoker (1994), were used for comparison. The computed statistical values were compared to these tabulated values when drawing statistical conclusions.

CHAPTER FIVE

DISCUSSION

INTRODUCTION

At completion of the programme, fifty two patients had been interviewed for inclusion into the study. Twenty two patients were excluded for reasons laid out in the delimitations stated in Chapter Three. Common reasons for exclusion included:

- Gynaecological conditions, two patients,
- Osteophytic degeneration and hypertrophy, seventeen patients,
- Rheumatoid arthritis and Osteoarthritis, three patients.

Thirty patients were introduced into the research programme. Five of these patients did not have follow up computed tomographic examinations.

- One patient had a sequestrated nuclear fragment
- One patient had osseous canal stenosis
- Three patients had progressive symptoms that required immediate surgical decompression.

Although these patients did not complete the required number of consultations, the data is of statistical relevance. Twenty five patients remained to complete the duration of the study and undergo both the pre and post treatment computerised tomographic examinations. Nineteen patients reported complete resolution of symptoms following completion of the programme. One patient reported 70 % improvement. Three patients reported up to 80 % improvement, but the pain returned during a period of one month following termination of treatment. Two patients underwent decompressive surgery after completing the treatment period since there was recurrence of symptoms. Subjective changes of those patients who received treatment, were measured using the Numerical Pain Rating Scale 101 (appendix E) and Oswestry Low Back Pain Disability Index (appendix D) questionnaires. Objective clinical changes were measured using orthopaedic testing. The orthopaedic tests performed included Straight leg raise, Patric FABERE test, Gaenslen's test, Thomas's test, Erichsen's test, Pheasant's test. Measurements of the spinal canal and intervertebral disc-to-spinal canal occupancy ratio were taken on the computed tomography examinations performed pre and post treatment. No visible changes were detected on initial examination of both the pre and post treatment computerised tomographic examinations.

5.1. - OBJECTIVE SIGNS

5.1.1 - Dermatomal symptoms

Twenty two patients examined in this study experienced dermatomal symptoms. This is higher than the 26 % described by Mensor in 1955, and the 46 % cited by Onel **et al.** (1991). Dermatomal distribution hypoaesthesia was noted in fourteen cases, 63.64 %. There were two cases of hypoaesthesia following conclusion of the treatment period. Both patients displayed hypoaesthesia along the S1 dermatomal distribution of the lateral foot and distal portion of the fifth toe. This is highly comparable with the statistically significant improvement of 66 % described by Onel **et al.** (1991).

The dermatomal distribution of pain followed the S1 dermatome in twelve patients, six patients in each treatment group, the L5 dermatome in thirteen patients, five patients in the adjustment group and eight in the McManis traction group, the L4 dermatome in five patients, one in the adjustment group and four in the McManis traction group. Multiple levels were noted in seven patients, two in the adjustment group and five in the McManis traction group. Ill-defined dermatomal patterns were noted in two patients. Eight patients experienced neither hypoaesthesia nor anaesthesia in the affected lower limb.

5.1.2. - Myotomal signs

Motor weakness, (+3) to (+4), was noted in seven patients, 23.33 %. Quadriceps musculature was affected in three instances, Extensor Hallucis Longus in three patients and Gastrocnemius muscle in one patient. These findings are closely related to those of Mensor (1955) but are lower than those cited by Saal & Saal (1989), who describe motor weakness in 64 % of the patient population. After treatment there was improvement in the muscle strength of three patients, 10 %. Onel **et al.** (1991) report decreased muscle strength in 29 % of the patients examined. Post treatment findings showed a statistically significant improvement of 53 % in the study by Onel **et al.** (1991). This was not found to be the case in this study.

5.1.3. - Deep tendon reflexes

Ten patients, 33.33 %, exhibited a decreased or absent deep tendon reflex in the affected lower limb. This compares favourably with the 40 % noted by Bush **et al.** (1992) and Onel **et al.** (1991), also 40 %, but is lower then the 53 % cited by Mensor (1955). Decreased patella reflex was noted in two cases, one in each treatment group. Absent patella reflex was noted in two patients, both in the McManis Traction group. Decreased Achilles reflex was noted in two cases, one in each treatment group. Absent Achilles reflex was noted in four patients, two in each treatment group. These findings did not correlate closely with those reported by Jonsson & Stromqvist (1993). This discrepancy is thought to be a result of the vastly differing sample sizes. In addition, Jonsson and Stromqvist divided the patients into those with central spinal stenosis as opposed to disc herniation, or lateral stenosis. This study did not make use of these classifications, possibly accounting for the lack of corroborative results. No change was noted post treatment to those patients with absent deep tendon reflexes. Patients with decreased deep tendon reflexes all regained full strength (+2) reflexes post treatment. This finding may indicate myofascial involvement of the Quadriceps or Gastrocnemius musculature on the affected side, which resolved post treatment.

Neurogenic intermittent claudication has been described by Cox (1990:264) as lower extremity pain in patients with no ischaemia of the leg muscles, but having compression of the cauda equina in the lumbar spine. Mayer **et al.** (1991) describes the condition as pseudoclaudication characterised by leg pain, numbness and weakness after the patient has walked short distances. Neurogenic claudication was noted in all thirty patients prior to onset of treatment. These findings are in keeping with those of Onel **et al.** (1993). With the exception of the three patients referred for immediate decompressive surgery, the claudication resolved following treatment.

5.1.4 - ORTHOPAEDIC TESTING

5.1.4.1. Straight leg raise

Straight leg raising (reproduction of spontaneous pain below sixty degrees of elevation) (Delauche-Cavallier **et al.** 1992) was positive in twenty four patients, 80 %. Nine patients, 30 % had bilateral straight leg raise positive with respect to pain in the lumbar spine with lower limb radiation. Twenty eight patients, 93.33 %, experienced pain as a direct result of straight leg raise testing prior to onset of treatment. Ten patients, 33.33 %, experienced pain post treatment, during the same test. Eleven patients in the Adjustment group experienced pain during this testing procedure at less than sixty degrees. Three patients experienced discomfort with each leg tested, at below sixty degrees.
Thirteen patients in the McManis traction group experienced pain during this testing procedure at less than sixty degrees. Six patients experienced discomfort in both lower extremities, at below sixty degrees.

5.1.4.2. Fabere-Patrick's test

A classically positive test constitutes pain within the hip joint, especially at the hip flexor attachment. Pain located in the region of the sacroiliac joint, provides indication of a sacroiliac lesion. (Gerard & Kleinfield 1993:454) Fabere-Patrick's test was positive in fifteen patients, 83.33 %, prior to the onset of treatment. Following treatment, 23.33 % of the patients, experienced pain during the same testing procedure.

5.1.4.3. Gaenslen's test

'Re-creation of pain in the sacroiliac joint on the side of the extended leg indicates a sacroiliac joint lesion.' (Gerard & Kleinfield 1993:404) Gaenslen's test was positive in nineteen patients, 63.33 % prior to the onset of treatment. Seven patients experienced pain during the same testing procedure following treatment, 13.33 %.

5.1.4.4. Erichsen's test

Erichsen's test is considered positive if the patient indicates the pain to lie over the sacroiliac joint (Gerard & Kleinfield 1993:402). This procedure was positive with respect to pain in twenty seven patients, 90 %, prior to the onset of treatment. Following treatment, 23 % of the patients experienced pain during the same examination procedure.

5.1.4.5. Thomas's test

Elevation of the lower limb or an increase in flexion of the knee on the side not being tested signifies a hip flexor contracture (Gerard & Kleinfield 1993:384). In addition, restriction of the sacroiliac joint of the same side will cause contralateral hip elevation with sacroiliac joint pain. Thomas's test was positive in eleven patients, 36.67 %, prior to the onset of treatment. Following treatment, two patients experienced pain during the same examination procedure, 6.67 %.

5.1.4.6. Pheasant's test

Pheasant's test was positive in fourteen patients, 46.67 % of the pre treatment group. One patient, 3.33 % , displayed a positive finding for this test, during the same testing procedure, following treatment.

5.1.5. - Myofascial trigger points

Myofascial trigger points (as defined in chapter 2) were noted in twenty five of the thirty patients who underwent treatment, 83.33 %. Thirteen patients fell into the McManis traction group. The remaining twelve patients were in the Adjustment group. Sixteen patients, 64 %, had multiple myofascial trigger point involvement. Nine patients in the McManis group and seven patients in the Adjustment group.

Musculature involved included the Biceps Femoris, Semimembranosus, Semitendinosus (Hamstring) - four patients, Lateral Gastrocnemius - two patients, Gluteus Maximus - seven patients. Gluteus Medius - eight patients, Gluteus Minimus - seven patients, Piriformis - four patients. Quadratus Lumborum - nine patients, Tensor Fasca Lata and Iliotibial band - four patients, Tibialis Anterior - one patient, Vastus Lateralis - three patients. Pain referral patterns of these muscles, as laid out by Travel & Simons (1992) have been shown by the authors to result in lower limb pain, mimicking sciatic distribution pain in the buttock, posterior thigh, lateral foot as well as anterior thigh pain and at times Quadriceps weakness possibly resultant from Vastus Medialis trigger points. (Travell & Simons 1992 2:23-350).

Localised pain was noted over the greater trochanter in two patients with L4 radiculopathy. Digital pressure reproduced the radicular symptoms in the posterior thigh, medial calf and lateral calf. Needling of trigger points around the area provided temporary relief. Ambulation reproduced the symptoms. One patient presented with a macular rash over the Quadratus Muscle on the affected side immediately following the computed tomography examination preceding treatment. The rash cleared over a period of three days and did not recur. No satisfactory conclusion was reached in this regard.

5.2. - SUBJECTIVE TESTING

5.2.1. - Numerical Rating Scale 101

Use of the Numerical Pain Rating Scale 101 showed decrease of pain registered by patients in twelve cases in the McManis group, 80 %, and thirteen cases, 86.67 %, in the Adjustment group.

5.2.2. - Oswestry Low Back Pain Disability Index

Use of Oswestry Back Pain Disability Index prior to initiation of treatment at the initial consultation and again immediately following treatment at the final consultation were of varying significance. Due to the large standard deviation noted in the adjustment group, one set of scores was shown to be not statistically relevant. No satisfactory explanation can be provided for this phenomenon.

5.2.3. - PAIN DIAGRAMS

Inconsistency with patient completion of the pain diagrams rendered this method of data capture invalid for conclusive assessment. These diagrams may be of limited use within a clinical environment. Perhaps restructuring of these diagrams to show the lateral thigh and sole of foot may ensure greater success in their usage.

5.3 - COMPUTERISED TOMOGRAPHIC EXAMINATIONS

A minimum of twenty one days elapsed between initial and followup computerised tomographic examinations. The changes in spinal canal diameter were noted visually, by a team of independent Radiographers, from Durban Radiological Services, who were blind to the nature of the study. A number of patients presented with previous radiographic evaluation of the lumbar spine.

- Magnetic Resonance Imaging: five patients
- Computerised tomographic evaluation: nine patients
- Plain film Xrays: twenty three patients
- Radioisotope Bone scans: one patient

Initial scans were viewed for comparison when interpreting the final scans.

TABLE 25: DISC ANALYSIS PRE TREATMENT

LEVEL	NUMBER	%
L3/4	2	5.26
L4/5	16	42.11
L5/S1	20	52.63
TOTAL	38	100.00

Ninety intervertebral disc levels were examined prior to initiation of treatment. Thirty eight levels, 42.22 %, showed some form of pathological change resulting in perceived spinal canal stenosis. These changes were resultant from the intervertebral disc bulging or herniating, disc degeneration with decrease in the intervertebral disc height, facet joint hypertrophy, post surgical scarring and/or soft tissue hypertrophy including Ligamentum Flava hypertrophy. In one instance the intervertebral disc had sequestrated into the spinal canal.

Of the initial thirty eight pathological levels noted, two were noted at the level of the third lumbar intervertebral disc, 5.26 %, sixteen were noted at the level of the fourth lumbar intervertebral disc, 42.11 %, twenty were noted at the level of the fifth lumbar intervertebral disc, 52.63 %. (Refer Table 25) Thirty two intervertebral disc levels were re-evaluated post treatment. The number of intervertebral disc lesions examined post treatment was reduced due to three patients referred for decompressive surgery, one patient with osseous spinal canal stenosis and one patient delimited due to the presence of a sequestrated segment. Four patients, 13.33 %, were referred for decompressive surgery at the affected levels in the lumbar spine. One lesion was noted at the level of the fourth intervertebral disc. Two of these lesions were located at the fifth lumbar intervertebral disc. The fourth patient had osseous spinal canal stenosis. No surgical intervention was felt to be expedient in this case.

In total, two pathologies, 6.25 %, were noted at the level of the third lumbar intervertebral disc. This result is not dissimilar to the results published by De Vos Meiring **et al.** (1994:323). These authors found an incidence of 8 % to 10 % of all disc pathology occurred at the level of the third intervertebral disc. Fifteen pathologies, 46.875 %, were noted at both the levels of the fourth and fifth lumbar intervertebral discs. Ten patients, 26.32 %, were noted to have multiple intervertebral disc lesions. Buirski & Silberstein (1993:1810) quotes 58.26 % of the one hundred and fifteen patients examined to have multiple level lesions. This result is higher than was noted here.No intervertebral disc lesion was noted in three cases, 7.89 %. This compares closely with Bush **et al.** (1992:1209) who noted no pathology in 4 % of the intervertebral discs examined. These cases were included for statistical evaluation due to the adjacent soft tissue structures which could contribute to soft tissue spinal canal stenosis in the lumbar spine. Twenty five lesions, 65.79 %, were noted to be central. Four lesions, 10.53 %, were noted to lie in the lateral recess. Twelve lesions, 31.57 %, were mixed in location. Onel **et al.** (1993) showed 41 % of the lesions to be central, 36 % to be mixed and 23 % to have occurred in the lateral recess.

5.3.1. - Reduction in size of intervertebral disc

Reduction in the size of the disc herniation was noted in twenty cases out of thirty two, 62.5 %. Bush **et al.** (1992) state that 76.19 % of the herniated discs showed some degree of resolution. (Refer Table 26) Reduction was more common at the fifth intervertebral disc level, twelve out of twenty cases, 60 %. Eight lesions, 40 %, at the level of the fourth intervertebral disc were noted to reduce in size. These results correspond fairly closely to the above mentioned study by Bush **et al.** (1992).

A mean change of 1.31 millimetres was noted at the level of the fourth intervertebral disc. Reduction in herniation size varied between 0.3 millimetres and 3.5 millimetres.

Mean change: 1.31 millimetres

$$0.3 < X < 3.5 \text{ millimetres}$$

A mean change of one point one nine millimetres was noted at the level of the fifth intervertebral disc. Reduction in herniation size varied between 0.15 millimetres and 4.0 millimetres.

Mean change: 1.19 millimetres

$$0.15 < X < 4.0 \text{ millimetres}$$

5.3.2. - Increase in size of intervertebral disc

Increase in the size of the herniation was noted in ten cases out of the thirty two cases, 31.25 %. Two herniations, 20 %, at the third intervertebral disc increased in size. (Refer Table 26) A mean change of 0.33 millimetres was noted. The size increase fell between 0.2 to 0.45 millimetres.

Mean change: 0.33 millimetres

$$0.2 < X < 0.45 \text{ millimetres}$$

Six herniations, 60 %, at the fourth intervertebral disc, increased in size. A mean change of 1.44 millimetres was noted. Size increase at this level was noted as between 0.05 millimetres and 4.85 millimetres.

Mean change: 1.44 millimetres

$$0.05 < X < 4.85 \text{ millimetres}$$

Two herniations, 20 %, at the fifth intervertebral disc exhibited size increases. The mean change at this level was measured to be 0.95 millimetres. Size increases ranged between 0.4 millimetres and 1.5 millimetres.

Mean change: 0.95 millimetres

$$0.4 < X < 1.5 \text{ millimetres}$$

TABLE 26:SIZE ALTERATIONS OF INTERVERTEBRAL DISC POST TREATMENT

Type of change	Number of levels	Affected level	Number I.V.D.s	Percentage patients
Decrease in size of nucleus pulposus	20 Levels	L3/4	0	ZERO
		L4/5	8	25.00%
		L5/S1	12	37.50%
Increase in size of nucleus pulposus	10 Levels	L3/4	2	6.25%
		L4/5	6	18.75%
		L5/S1	2	6.25%
No change in size of nucleus pulposus	2 Levels	L3/4	0	ZERO
		L4/5	1	3.12%
		L5/S1	1	3.12%
Total	32		32	100.00%

5.3.3. - Percentage evaluation of spinal canals

The mean percentage for the Adjustment group, pre-treatment, showed the intervertebral disc to occupy 30.98 % of the spinal canal. Followup examination showed the intervertebral disc to occupy a mean of 26.29 % of the spinal canal. This mean is for all the levels combined. Mean percentage occupancy of the intervertebral disc, within the spinal canal, in the McManis group, was noted to be 33.51 % prior to onset of treatment. Following completion of treatment, the percentage occupancy was averaged at 29.28 %.

5.3.3.1. - Third intervertebral disc level

At the third intervertebral disc level, for the patient in the Adjustment group, the pre-treatment percentage was measured as 14.52 %. Followup examination at the same level showed a small increase to 14.90 %. At the third intervertebral disc level, for the patient in the McManis group, the pre-treatment percentage was measured as 40.72 %. Followup examination at the same level showed a small increase to 43.41 %.

5.3.3.2. - Fourth intervertebral disc level

Eight patients in the Adjustment group showed the intervertebral disc to occupy a mean area of the spinal canal of 34.48 %, at the level of the fourth intervertebral disc, prior to the onset of treatment. Following the treatment period, this percentage decreased to 25.31 %. At the same level, the mean intervertebral disc-to-spinal canal percentage of the eight patients in the McManis traction group, prior to onset of treatment, was measured as 27.01 %. Following treatment, the percentage had increased slightly to 28.49 %. One patient from this group did not complete their followup examination. The intervertebral disc-to-spinal canal measurement showed the intervertebral disc to occupy 45.37 % of the spinal canal at this level.

5.3.3.3. - Fifth intervertebral disc level

Nine intervertebral disc-to-spinal canal percentages were calculated at the level of the fifth intervertebral disc, in the Adjustment group. Prior to treatment, the percentage of intervertebral disc-to-spinal canal was recorded as 29.69 %. Following treatment, this percentage dropped to 25.16 %. Three patients did not have followup examination. The percentages of the pre-treatment measurements, of the above patients, was 29.62 %, 26.49 % and 46.95 % occupancy of the intervertebral disc of the spinal canal in each of those patients. Eleven intervertebral disc-to-spinal-canal percentages were calculated at the level of the fifth intervertebral disc, in the McManis group. Prior to treatment, the percentage of intervertebral disc-to-spinal canal was recorded as 35.39 %. Following treatment, this percentage dropped to 26.14 %.

Comparison at this point can be drawn with a case study presented by Cox **et al.** (1993). The spinal canal, at the fifth intervertebral disc, was measured at 40 % occupancy in the sagittal diameter, prior to initiation of treatment. Follow-up examination showed that 33 % of the vertebral canal was occupied by intervertebral disc and the patient was asymptomatic. The 7 % reduction, reported by Cox **et al.** (1993), is slightly lower than the mean decrease in percentage occupancy of 8.99 % show in this study, at the equivalent spinal level.

Two patients in the above group did not have followup examination. The percentages of the pretreatment measurements in these cases were 46.32 % and 43.39 % occupancy of the spinal canal by the intervertebral disc. Although some percentile changes are noted between the two treatment groups throughout the various intervertebral levels, no statistical trend was noted.

5.4. - Vertebral levels adjusted

A total of fourteen patients were adjusted in this group. One patient had been delimited out of the study due to the presence of a sequestrated fragment. Eight patients were adjusted on the right side, four on the left, and one bilaterally. In five cases, the adjustment was performed on the opposite side to the detected intervertebral disc herniation, when correlated with computed tomography. Eleven of the patients had central herniations, as well as left or right sided lesions. Two patients had no identified lesion on computerised tomographic examination.

CHAPTER SIX

CONCLUSIONS

The results of this investigation compare favourably with those obtained by Delauche - Cavallier **et al.** (1992), Bush **et al.** (1992), Buirski & Silberstein **et al.** (1993), Chrisman **et al.** (1964), Mensor (1955), Jonsson & Stromqvist (1993), Saal & Saal (1989). Kuo & Loh (1986) note that the level of the disc lesion was most common at the level of the fourth intervertebral disc. This was not the case in this study. The mean ages, male to female ratio, and results showing improvement post treatment, of 76.8 %, reported by Kuo & Loh (1986), correlated closely with this study. Maigne **et al.** (1992) presented a paper in which the authors relate reduction in size of the herniation of the lumbar intervertebral disc in all forty eight of the patients accepted into the study. The results of this study did not seem to correlate in this regard to those described by Maigne **et al.** (1992).

It was noted, however that the greatest reductions in the lumbar herniations appeared at those levels where the herniations were greatest in size. This is in keeping with the findings by Maigne **et al.** (1992). These findings may indicate that the intervertebral disc lesion does in fact dessicate or that the noted lesion was a haematoma that has subsequently resorbed. (as discussed in chapter 2)

6.1. - ORTHOPAEDIC TESTING

Of all the orthopaedic tests that proved positive, those specific for sacroiliac lesions were most frequently positive, ie. Erichsen's test, Fabere-Patrick's test, Gaenslen's test. Motion palpation revealed fixation over the ipsilateral sacroiliac joint in all patients with low back pain and radicular signs and symptoms. Kemp's test was positive in all thirty patients. Pain on extension of the lumbar spine, at the level of the lesion, was regarded as a positive test. Kemp's test has been used to indicate facet syndrome or differentiate between medial and lateral discal pathology (Gerard & Kleinfield 1993:344). It can be concluded that in addition to the lumbar spine facet syndrome, sacroiliac syndrome is common. It is likely that the pain distribution into the buttock, posterior thigh, even into the lateral calf and foot, may be associated with this syndrome.

If the piriformis muscle is also involved, a Piriformis syndrome may result with the sequelae of sacroiliac fixation, myofascial pain and dysfunction, involving the Piriformis musculature and neurogenic irritation of the nerves exiting through the sciatic notch, viz. sciatic and pudendal nerves (Travell & Simons 1992 2:201). Sacroiliac joint adjustment would greatly relieve the symptoms associated with this syndrome. It is recommended that the sacroiliac joint is adjusted in conjunction to the fixated lumbar segment.

6.2. - SUBJECTIVE TESTING

Results of the Numerical Rating Scale 101 show that both treatment groups responded favourably to the type of conservative treatment received. It is informative to note that the patients within the Adjustment group achieved asymptomatic status more rapidly than those patients receiving McManis flexion distraction. Over the three week period however, no significant differences were noted between the two differing treatment groups. The results noted within this study correspond closely with reports published by Cassidy **et al.** (1993), Cox **et al.** (1993).

Oswestry Low Back Pain Disability Index results demonstrated a degree of variation between the two groups prior to onset of treatment. Although these scores are statistically significant, the scores demonstrating the final results showed no statistical variance.

It is felt that the use of pain diagrams as presented by Margoles (1980) may have had greater accuracy in illustrating the patientís discomfort. This is due to the fact that the pain diagrams presented by Margoles provide facility for the patient to indicate nociceptive sensation on the plantar aspect of the foot and over the lateral aspect of the thigh and leg

6.3. - COMPUTED TOMOGRAPHY

Computed tomography may be effective in the demonstration of soft tissue intrusions into the spinal canal, but there are certain inherent errors in using the technique as a measurement procedure to compare two different scans of the same person.

Firstly, disc material may progress into the lateral recess, in which case it may not be visible, if the scan is only performed between the two vertebral endplates. Scanning should include the inferior aspect of the superior pedicle to the superior aspect of the inferior pedicle.

Secondly, measurements may vary between observers, as it is difficult to accurately identify the boundaries between the annulus fibrosis, Posterior Longitudinal ligament, thecal sac, epidural haematoma (Delauche - Cavallier **et al.** 1992) and post operative fibrous scar tissue. This phenomenon may be a result of the little difference of absorption coefficients that exists amongst these tissues. (Hirofuji & Tanaka 1983) (Refer Plates 7, 8, 9, 10) If repeated, this study would benefit from independant observers performing the measurements, so as to reduce validity. Since the measurements are subjective, reliability would be difficult to sustain.

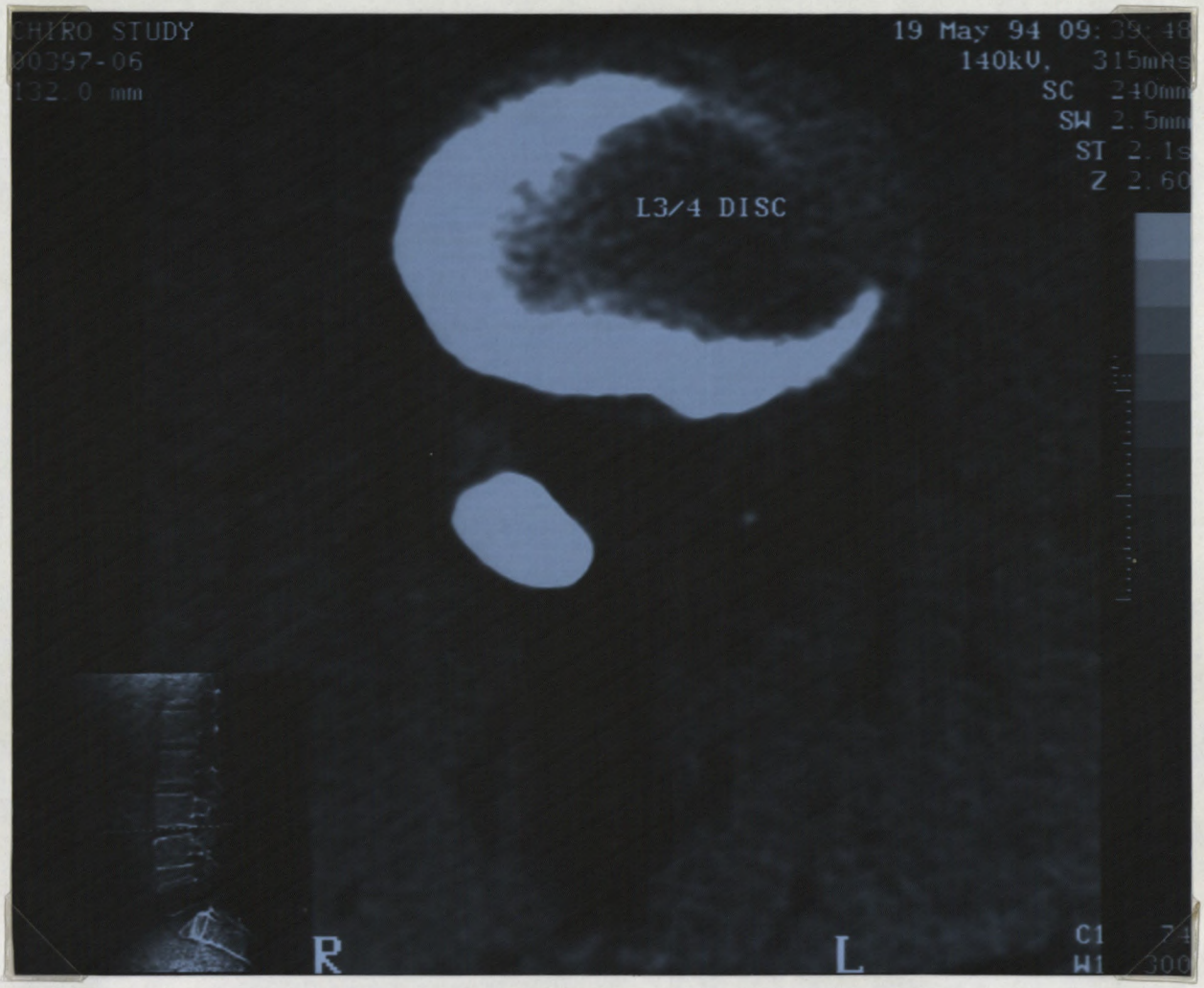


PLATE 7:

PRE TREATMENT COMPUTED TOMOGRAPH 1

SLICE VARIATION

(PATIENT A)

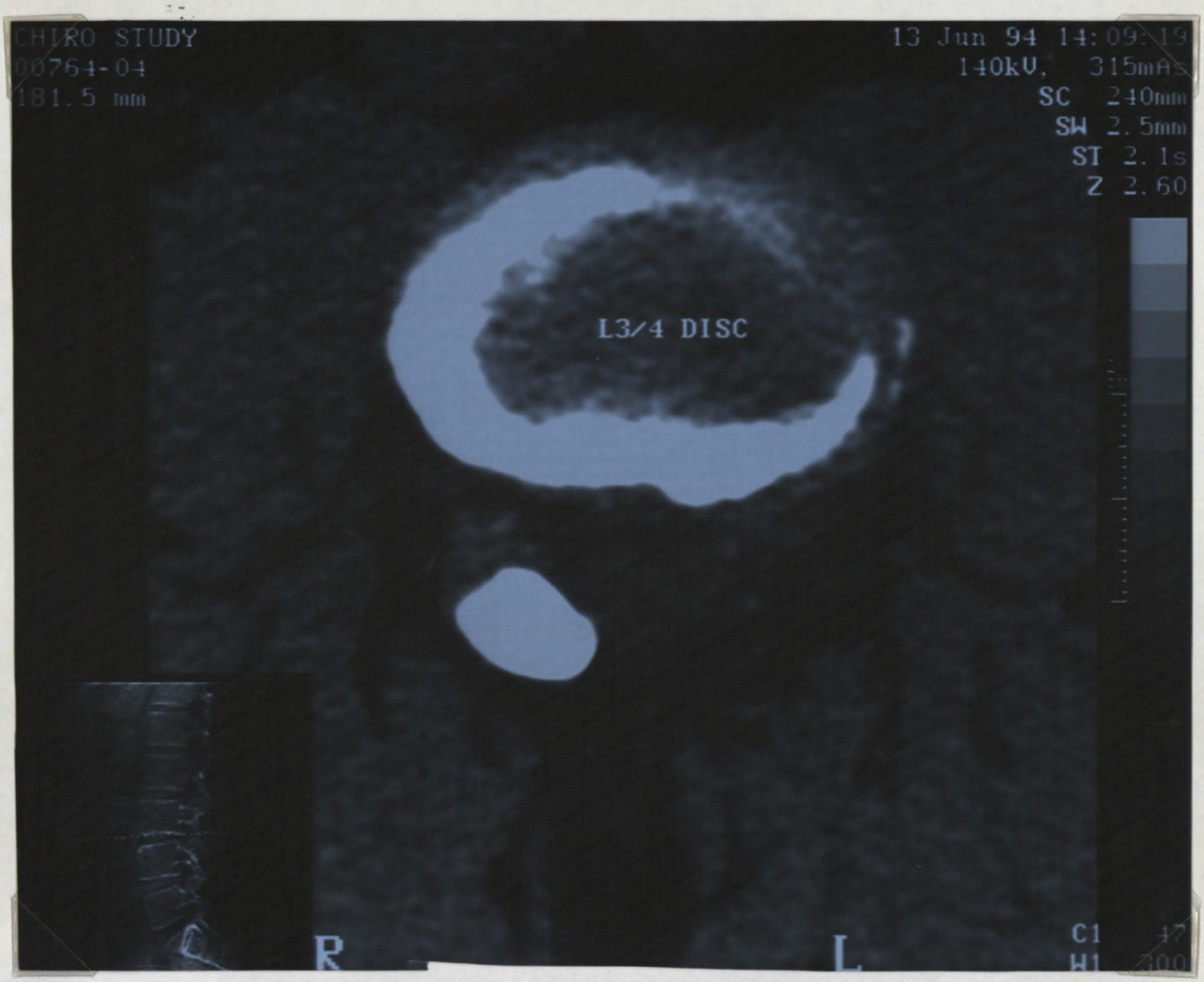


PLATE 8:

POST TREATMENT COMPUTED TOMOGRAPH 1

SLICE VARIATION

(PATIENT A)

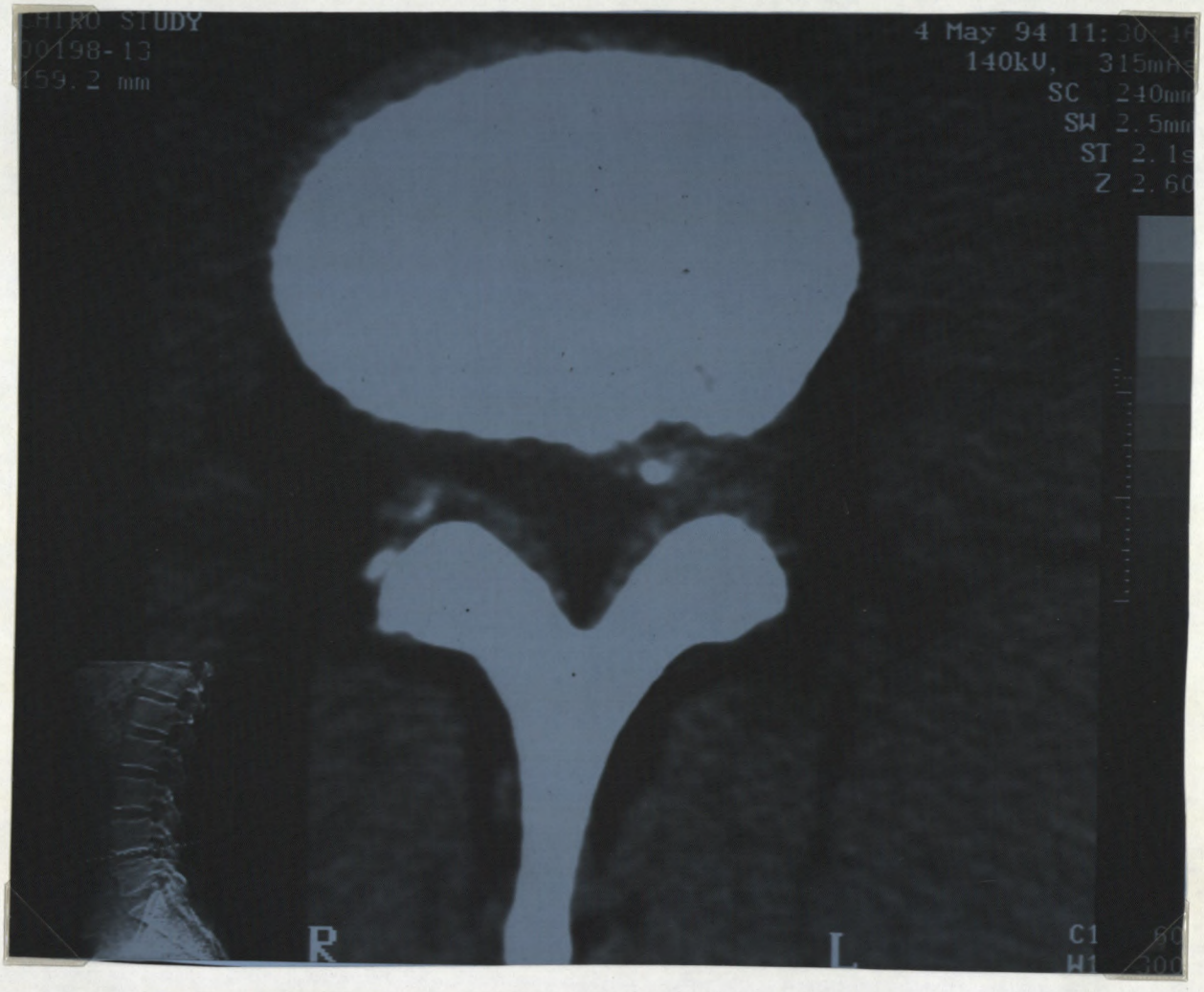


PLATE 9:

PRE TREATMENT COMPUTED TOMOGRAPH 2

SLICE VARIATION

(PATIENT B)

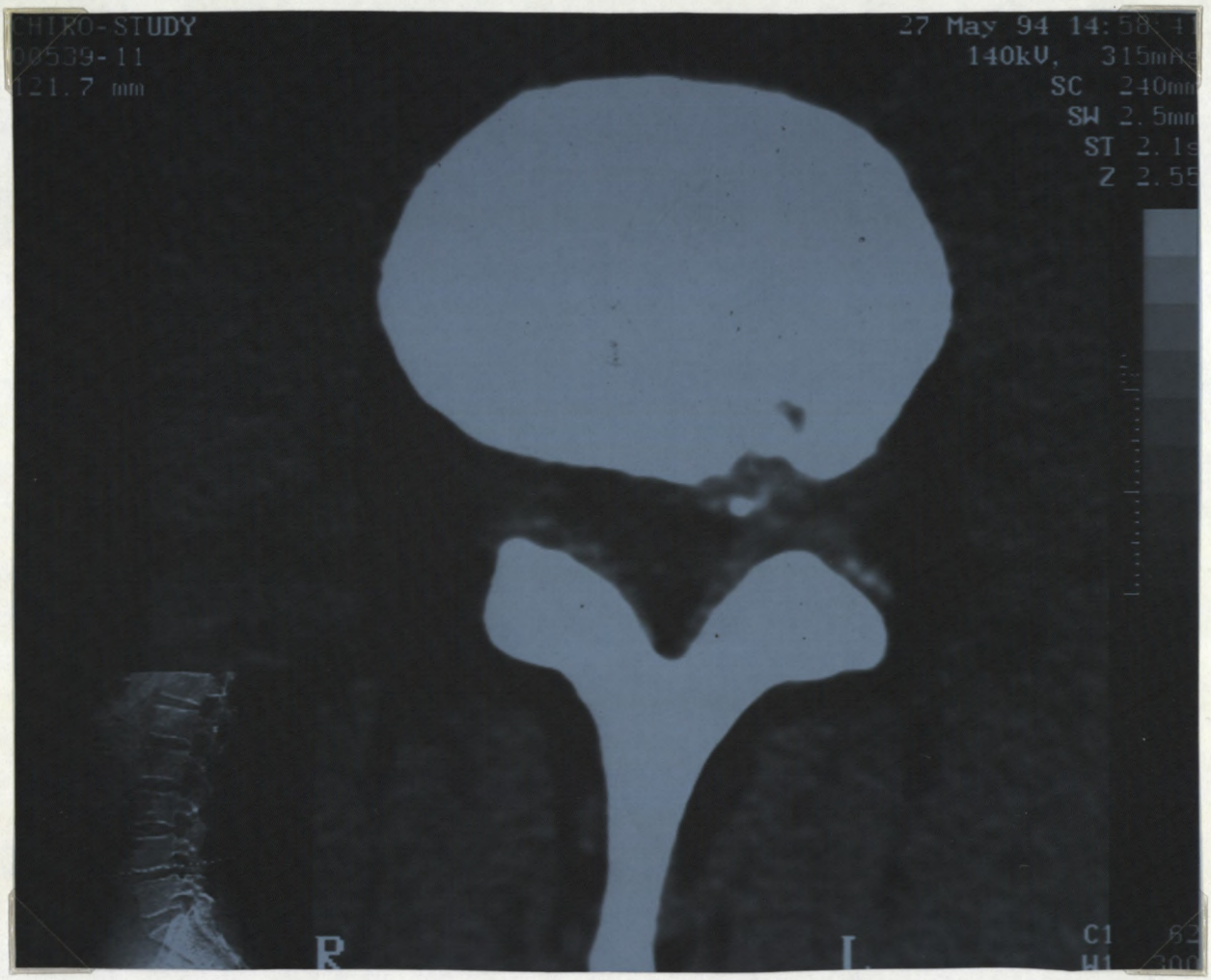


PLATE 10:

POST TREATMENT COMPUTED TOMOGRAPH 2

SLICE VARIATION

(PATIENT B)

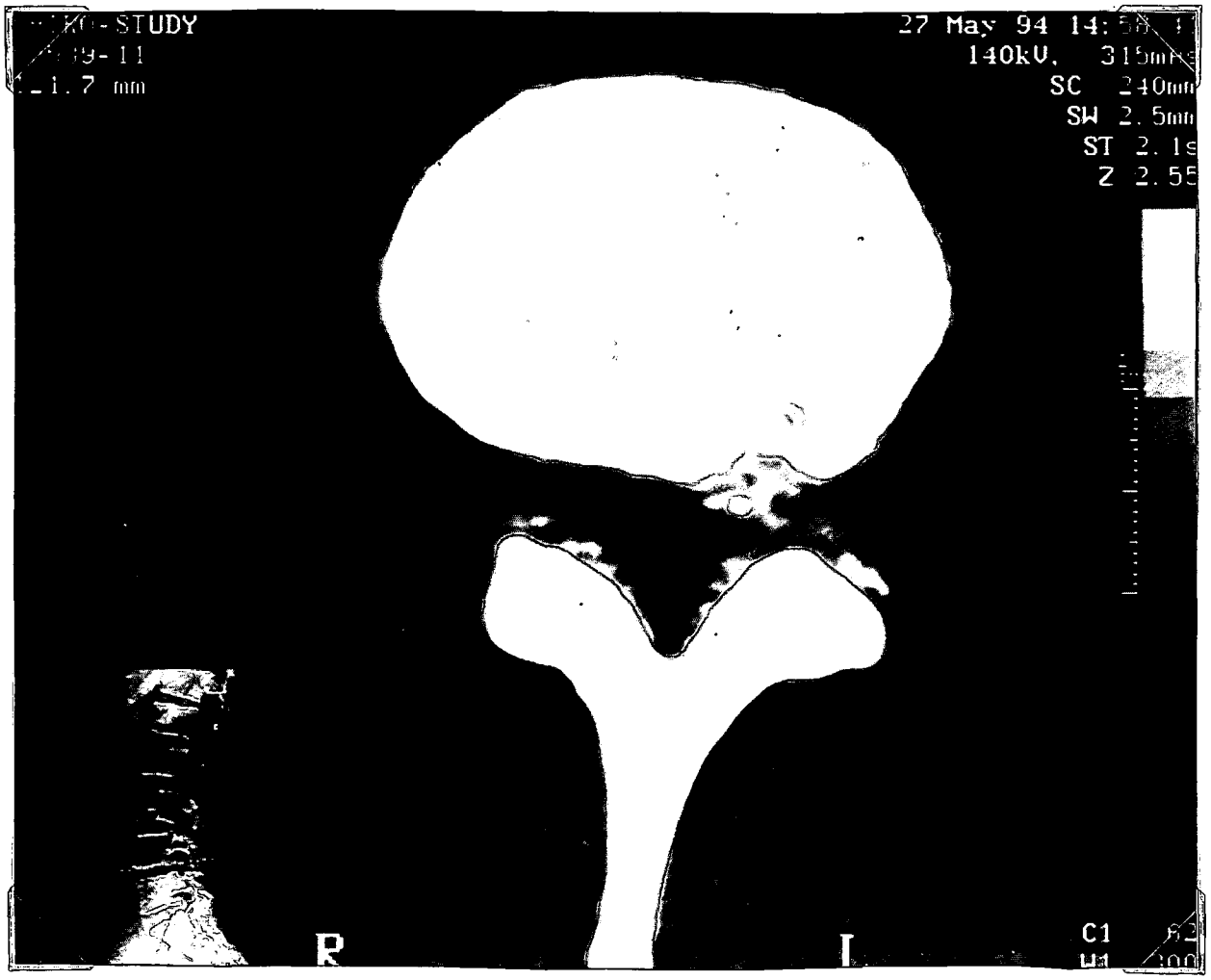


PLATE 10:

POST TREATMENT COMPUTED TOMOGRAPH 2

SLICE VARIATION

(PATIENT B)

Computed tomography does not accurately display inflammation, possibly resultant from introduction of nucleus pulposus into the spinal canal. It is not impossible that this inflammation mimics the appearance of a herniated nucleus pulposus, explaining the apparent resolution of herniation as described in reports by Maigne **et al.** (1992), Delauche - Cavallier **et al.** (1992). Magnetic resonance imaging might be used in future similar studies. Cost constraints prevented the use of M.R.I. in this study.

The pain associated with a disc herniation is felt by Cassidy **et al.** (1993) to be a result of the inflammation. This pain is chemically mediated and is not necessarily dependant on the size of the herniation. Saal, Saal and Hertzog (1990) propose that the pain experienced may be due to the presence of pro-inflammatory enzymes, phospholipase A2, in lumbar disc herniations. These views are supported by Bogduk (1990), Cox (1990:111-112) & Delauche - Cavallier **et al.** (1991). By increasing mobility in the facetal joint, the side posture manipulation may be effective for decreasing the capsulitis associated with zygapophyseal joint fixation (refer to chapter 2)

It may be expedient to measure the thecal sac for deviation, to assess any degree of inflammatory compression acting directly on the sac, rather than measuring the degree of occupancy of the intervertebral disc in the spinal canal. These measurements have been advocated by Cox (1990:261). Sagittal and coronal measurements of the thecal sac may be performed. Extradural fat collected anterior to the apex of the neural arch (anterior to the spinous process) may also affect the perceived diameter of the spinal canal. Generalised fat loss throughout the body may cause alterations in the amount of extradural fat, altering the size of the spinal canal.

Thirdly, when performing measurements on the follow up examination, it is unlikely that exactly the same area was scanned during the follow up examination. This may result in an apparent increase or decrease in the perceived size of the herniated fragment, when in actuality this does not exist. Difficulty is also experienced in obtaining accurate measurements of the herniation if it is not mid-centre in the spinal canal. Observer error may arise in choosing the area of greatest size of the herniation for measurement, as well as clearly defining these soft tissue boundries.(Refer Plates 11, 12, 13, 14).

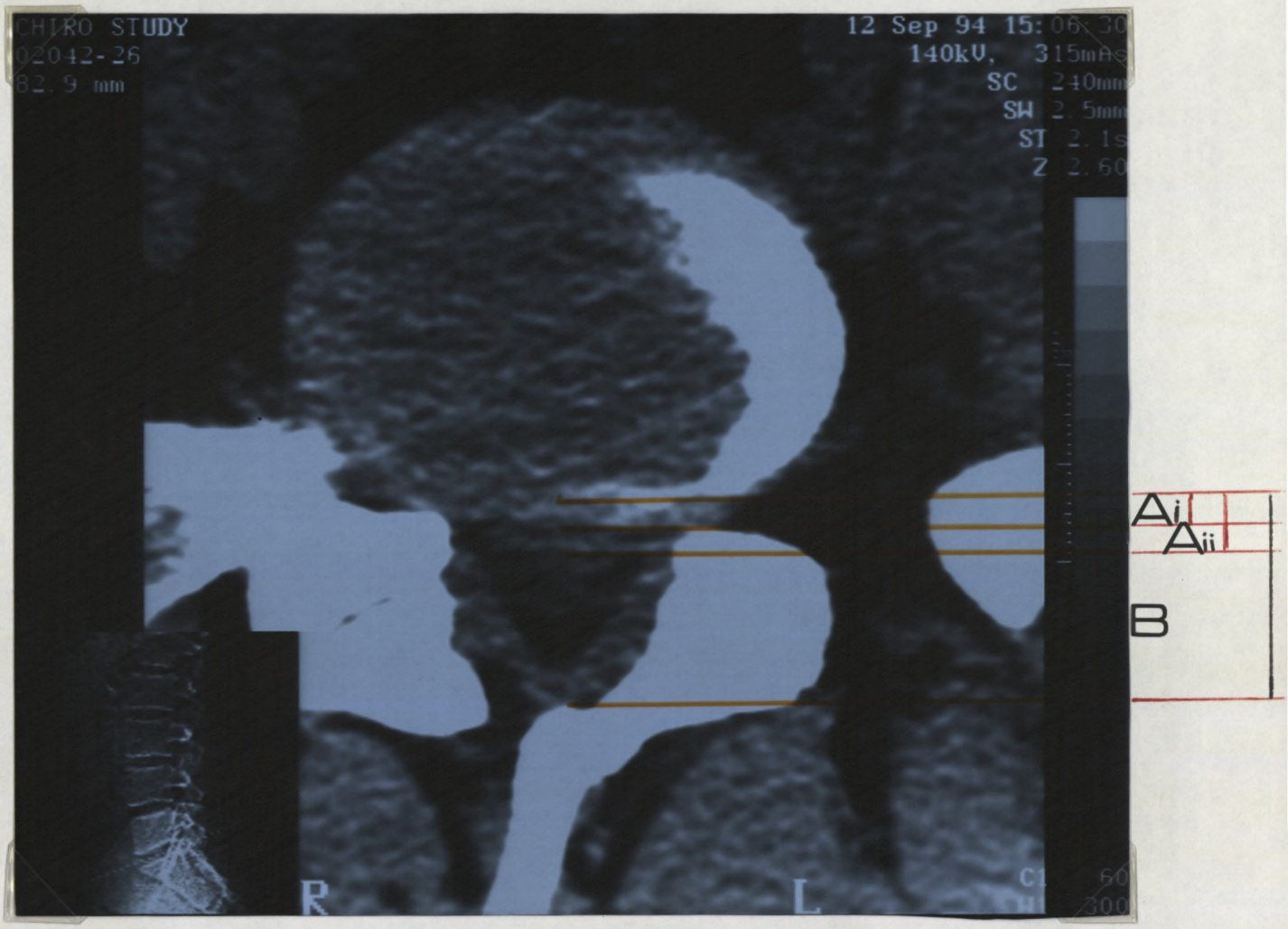


PLATE 11:

PRE TREATMENT COMPUTED TOMOGRAPH 3

MEASUREMENT VARIATION

(PATIENT C)

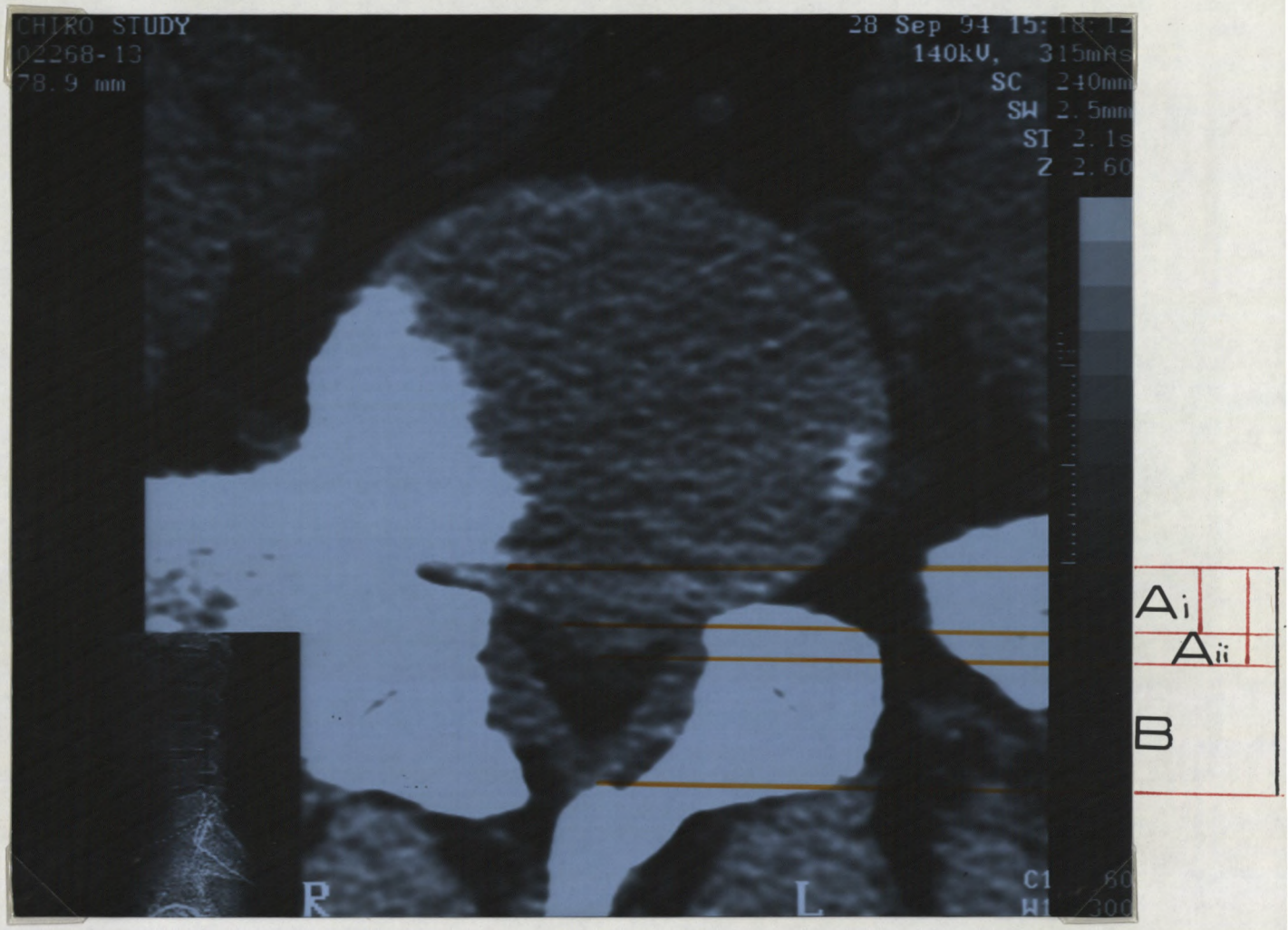


PLATE 12:

POST TREATMENT COMPUTED TOMOGRAPH 3

MEASUREMENT VARIATION

(PATIENT C)

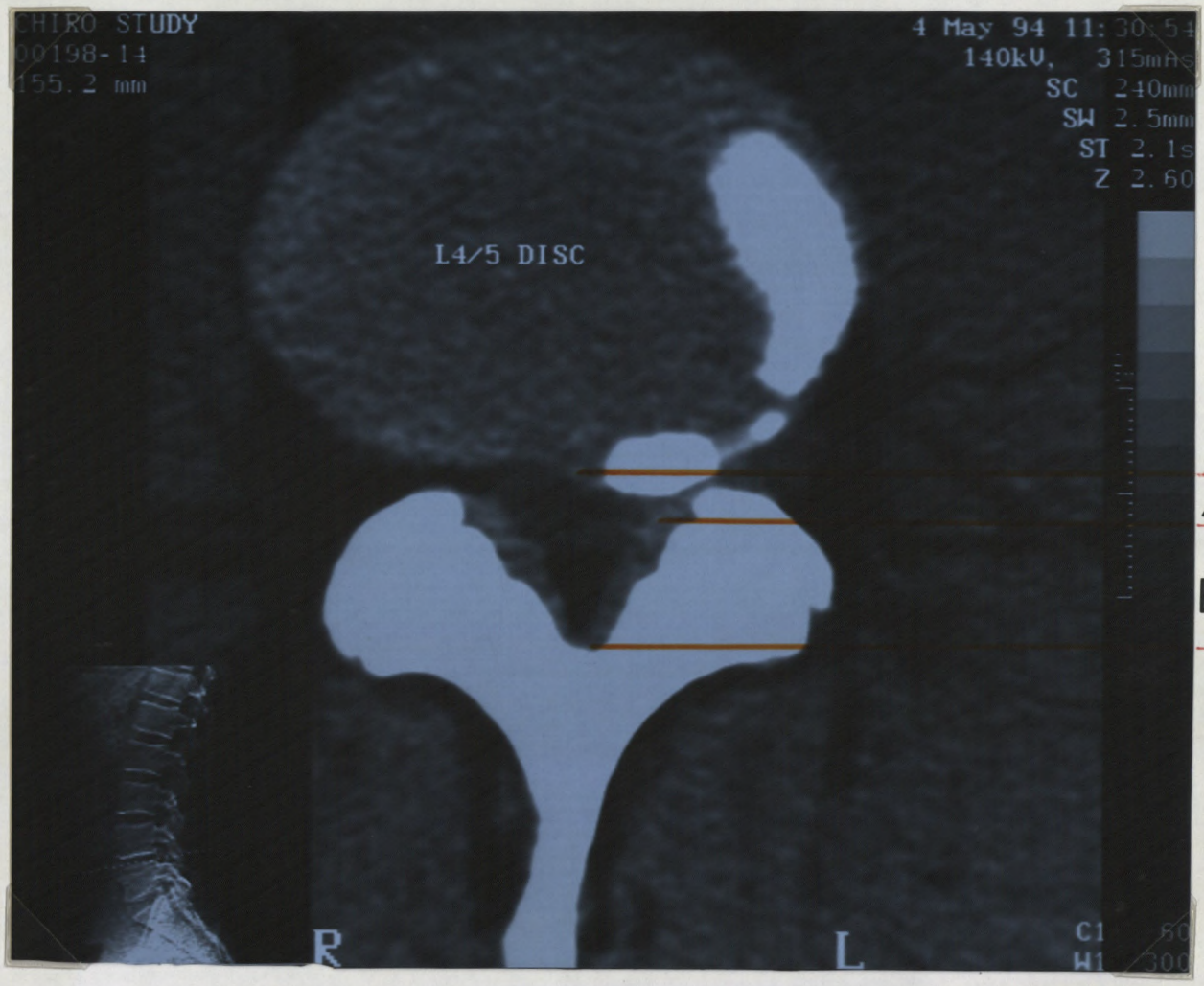


PLATE 13:

PRE TREATMENT COMPUTED TOMOGRAPH 4

MEASUREMENT VARIATION

(PATIENT D)

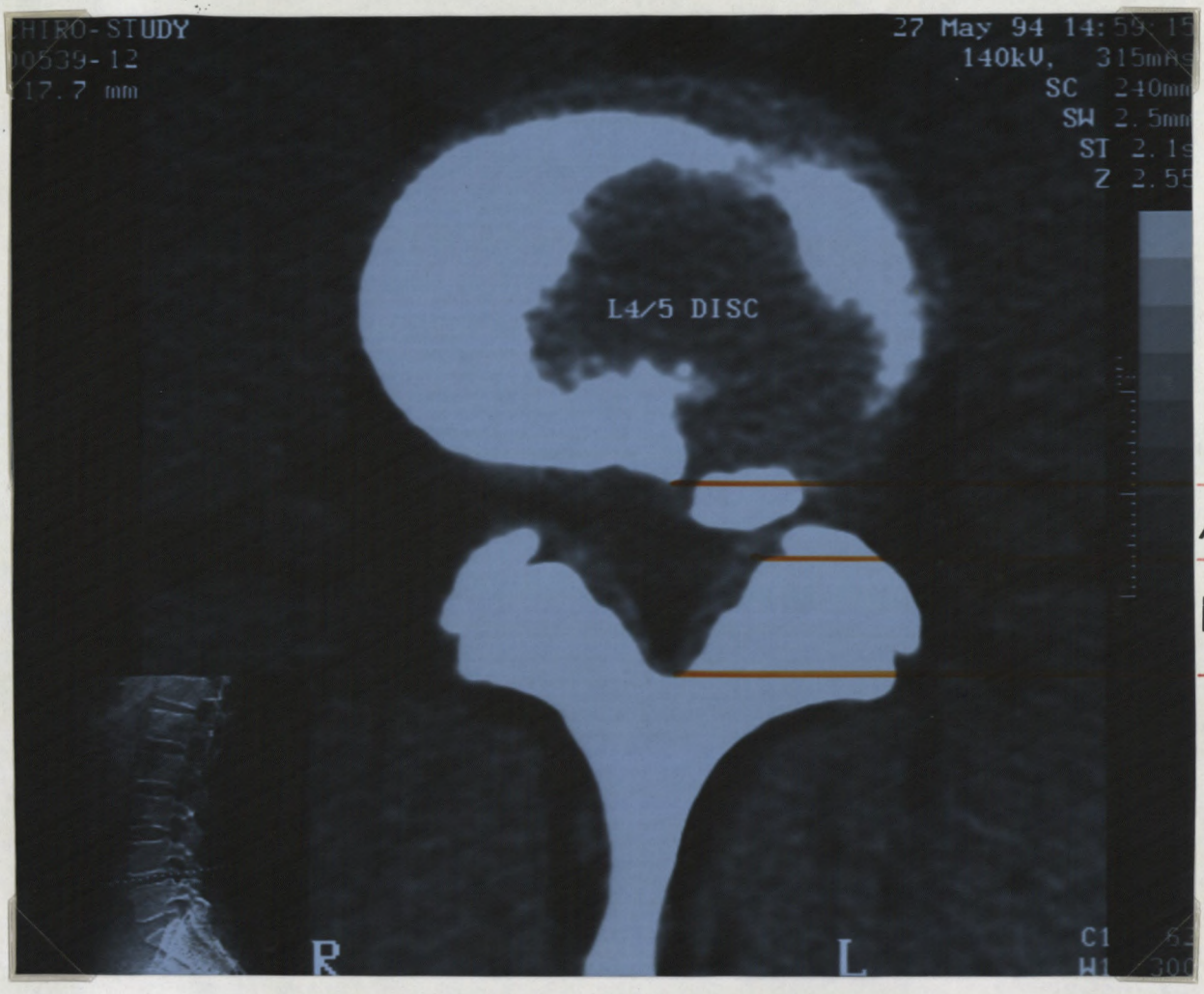


PLATE 14:

POST TREATMENT COMPUTED TOMOGRAPH 4

MEASUREMENT VARIATION

(PATIENT D)

In the two scan sequences presented, the initial slice was taken close to the superior endplate of the inferior vertebral body. The slices are at slightly differing levels and consequently present marginally different intervertebral disc herniation sizes in each case.

Bayley **et al.** (1991) state that the spinal canal is sinusoidal. They suggest that the computed tomographic scans need to be sequential, without gaps, in order to avoid skipping areas of spinal cord compression. This study made use of 3 consecutive parallel 4 millimeter cuts through the plane of the intervertebral disc. The underlying question that remains is still `To what degree does the presence of a herniated nuclear fragment correlate to the objective and subjective clinical presentation of the patient ?' Wiesel **et al.** (1984) concluded that 35 % of asymptomatic patients were found to have abnormal computed tomography examinations. Over 19 % of these patients, under the age of forty years were diagnosed as having herniated nucleus pulposus. Patients over forty years of age showed a 50 % abnormal rate, with the most common diagnosis being canal stenosis and facet degeneration. A literature review published by Chapman-Smith (1993) reinforces these statements.

Of the fourteen patients treated by Cassidy **et al.** (1993), in most cases the computed tomograph post treatment remained unchanged. In five of the cases, a small decrease in the size of the herniation, and in one case a large decrease was observed. Maigne **et al.** (1992), noted that the greatest reductions in the lumbar intervertebral disc herniations were located at those levels where the herniations were greatest in size. The results of this research correspond more closely with those of Cassidy **et al.** (1993) than with those of Delauche - Cavallier **et al.** (1991) and Maigne **et al.** (1992), who reported greater than 80 % of the treated patients exhibiting reduction in the size of the intervertebral disc. These reports, as with that of Saal, Saal & Hertzog (1990) have follow up periods of many months before the second computed tomography examination. Perhaps the three week period observed during this study was too short to observe the resolution of the proposed inflammatory reaction, or dessication due to separation from the discal nutrient supply and blood supply, as proposed by Saal, Saal & Hertzog (1990)

It was noted that there was no statistically significant alterations in the percentage intervertebral disc occupancy of the spinal canals, at the level of the lesion, in any of the levels measured. In addition, no significant variance was noted in the percentage occupancy of the spinal canal by the intervertebral disc, between the two treatment groups, at any of the levels investigated.

The incidence of myofascial trigger points, in patients presenting with lower limb pain, needs further examination. Involvement of the sacroiliac joints also need further study. Since the three joint complex involves two facetal joints and the intervertebral disc, and the centre of rotation of the complex is located in the centre of the vertebral body, focus on the detrimental effects of facet fixation as a precursor to intervertebral disc lesion formation needs to be examined. The facetal joint rotated between 2 degrees and 3 degrees during normal motion.(refer to chapter 2) Reduction in motion of a facetal joint may place a shearing force on the intervertebral disc due to a shift in the centre of rotation towards the fixated joint, This may lead to a greater likelihood of annular tear formation and later internal disc disruption. The pathophysiological role of the facetal joints in low back pain with associated lower limb radiculopathy needs to be further examined.

REFERENCES

REFERENCES

REFERENCES

Adams, M.A. & Hutton, W.C. 1980. The effects of posture on the role of the apophysial joints resisting intervertebral compressive forces. Journal of bone and joint surgery, 62B:358362

Atlas S.W. (Ed).1991, Magnetic Resonance Imaging Of The Brain And Spine, p795864. Raven Press, New York. 1137p. ISBN 0881676942

Basmajian J.V. 1985. Manipulation, Traction and Massage 3rd ed., Baltimore:Williams and Wilkins. 333p. ISBN 068300378

Bayley, Cpt. J.C., Kruger, D.M. & Schlegel, J.M. 1991. Does The Angle Of The Computed Tomographic Scan Change Spinal Canal Measurements? SPINE, 16(10), supplement:S526-S529

Bischoff, R.J., Rodriguez, R.P., Gupta, K., Righi, A., Dalton, J.E. & Whitecloud T.S. 1993. A Comparison Of Computed Tomography-Myelography, Magnetic Resonance Imaging, and Myelography In The Diagnosis Of Herniated Nucleus Pulposus and Spinal Stenosis. Journal of Spinal Disorders, 6(4):289-295.

Bogduk, N. 1983.The Innervation of the Lumbar Spine. Spine, 8(3):286293.

Bogduk, N. 1990. Pathology of lumbar disc pain.

Journal of Manual Medicine, 5:7279.

Bronfort, G.; Chapter 24.

Effectiveness Of Spinal Manipulation

And Adjustments, pp 415-442, in Halderman, S.,1992

Principles And Practice Of Chiropractic. 2nd ed.,

Appleton And Lange, Norwalk, 1992,

ISBN 0-8385-6360-0

Buirski, G. & Silberstein, M. 1993.

The Symptomatic Lumbar Disc In Patients With Low

Back Pain. Magnetic Resonance Imaging

Appearances In Both A Symptomatic And Control

Population. SPINE, 18(13):1808-1811.

Bullough, P.G. & Vitoritas 1992.

Atlas Of Orthopaedic Pathology With Clinical And

Radiologic Correlations. 2nd ed, New York:Gower

Medical Publishing. ISBN 0397446217

Bush, K., Cowan, N., Katz, D.E. & Gishen, P. 1992.

The Natural History Of Sciatica Associated With

Disc Pathology. A Prospective Study With

Clinical And Independent Radiologic Follow-up.

SPINE, 17(10):1205-1212

Cassidy, J.D., Theil, H.W. & Kirkaldy Willis, W.H. 1993. Side posture manipulation for lumbar intervertebral disc herniation. Journal of Manipulative and Physiological Therapeutics, 16(2):96-102, Feb.

Camins, M.B. & O'leary, P.F. 1987: The Lumbar Spine Raven press,New York. p489. ISBN 0881672084

Chapman-Smith, D. (Ed.) 1993. Lumbar Disc Herniation - New Research and New Conclusions. The Chiropractic Report. 7(4):1-6. May

Chrisman, O.D., Mittnacht, A. & Snook, G.A. 1964. A study of the Results Following Rotatory Manipulation In The Lumbar Intervertebral Disc Syndrome. The Journal of Bone and Joint Surgery, 46A(3):517.524, Apr.

Church, C.P. & Buehler, M.T. 1991. Radiographic evaluation of the costotransverse ligament at the L5 intervertebral foramen:A cadaveric study. Journal Of Manipulative And Physiological Therapeutics, 14(4):240-248, May.

Cox, J.M. & Aspegren, D.D. 1987. A Hypothesis Introducing A New Calculation For Discal Reduction: Emphasis on Stenotic Factors And Manipulative Treatment, SPINE, 10(6):287-294 Dec.

Cox, J.M.,1990. Low Back Pain : Mechanism, Diagnosis and Treatment, 4th ed. Baltimore: Williams and Wilkins. p645.ISBN 0-683-02152-4

Cox, J.M.1992. Traction and Distraction Techniques. In Halderman, S. Principles And Practice Of Chiropractic. 2nd ed. pp503-518. Norwalk:Appleton And Lange. 640 p. ISBN 0- 8385-6360-0

Cox, J.M., Hazen, L.J. & Mungovan, M. 1993. Distraction Manipulation Reduction of an L5S1 Disc Herniation. Journal of Manipulative and Physiological Therapeutics, 16(5):342-346, June

Delauche-Cavallier, M., Budet, C., Laredo, J., Debie, B, Wybier, M., Dorfmann, H. & Ballner, I. 1992. Lumbar Disc Herniation, Computed Tomography Scan Changes After Conservative Treatment Of Nerve Root Compression. SPINE, 17(8):927-933.

De Vos Meiring P., Gandhi M., Nakielny R.A. 1994, Computed Tomographic Scanning Of L3/4 In Suspected Disc Prolapse: Is It Necessary?, The British Journal of Radiology, 67(796):323-324, Apr.

Dorwart, R.H., Volger III, J.B. & Helms, C.A. 1983. Spinal Stenosis. Radiologic Clinics Of North America, 21(2):301-325, June

DuPriest, C.M. 1993 Nonoperative Management Of Lumbar Spinal Stenosis. Journal Of Manipulative And Physiological Therapeutics, 16(6):411-414, July/Aug.

Eisenstein, S. 1976. Measurement Of Lumbar Spine Canal In Two Racial Groups. Clinical Orthopaedics, 115:4245, Mar./Apr.

Epstein, N.E., Epstein, J.A., Carras, R. & Hyman, R.A. 1990. Far Lateral Disc Herniations And Associated Structural Abnormalities. An Evaluation In 60 Patients Of The Comparative Value Of The CT, MRI, And Myelo-CT In Diagnosis and Management.SPINE, 15(6):534-539

Fairbank, J.C.T., Davies, J.B., Couper, J., & OíBrien, J.P. 1980. The Oswestry Low Back Pain Disability Questionnaire,Physiotherapy. 66(8):271-273, Aug.

Fischgrund, J.S. & Montgomery, D.M. 1993. Diagnosis and Treatment Of Discogenic Low Back Pain. Orthopaedic review, 311-318, Mar.

Frymore, J.W., Ducker, T.B., Hadler, N.M., Kostuik, J.P., Weinstein, J.N., Whitecloud III, T.S. 1991. The Adult Spine Principles and Practices. vol2, New York:Raven press. ISBN 0881676896

Gerard, J.A., Kleinfield, S.L., 1993. Orthopaedic Testing,A Rational Approach to Diagnosis, New York:Churchill Livingstone. ISBN 0-443-08876-4, p344, 402, 404, 454, 478.

Ghormley, R.K. 1993. Low Back Pain With Special Reference To The Articular Facets With Presentation Of An Operative Procedure, J.A.M.A.,101:17731777

Giles, L.G.F.1991. The Relationship Between The Medial Branch Of The Lumbar Posterior Ramus And The MammiloAccessory Ligament. Journal Of Manipulative And Physiological Therapeutics , 14(3):189-192. Mar.\Apr.

Gitelman, R. & Fligg, B. 1992. Diversified Technique. In Halderman, S.; Principles And Practice Of Chiropractic. 2nd ed, pp 483-502. Norwalk:Appleton And Lange. 640p. ISBN 0-8385-6360-0.

Goel, V.K. Weinstein, J.N. 1990. Biomechanics Of The Spine:Clinical and Surgical Perspective. Boston:CRC Press 787p. ISBN 044303365X

Hammerschlag, S.B., Wolpert, S.M. & Carter, B.C. 1976. Computed tomography of the spinal canal. Radiology, 121:361367

Hasegawa, T., An, H.S. & Haughton, V.M.1993. Imaging Anatomy of the Lateral Lumbar Spinal Canal. Seminars in Ultrasound, CT, and MRI, 14(6):404413. Dec.

Henderson, R.S. 1952. The treatment of lumbar intervertebral disc protrusion. An assessment of conservative measures. British Medical Journal, 13:597598. Sept.

Hession, E.F. & Donald, G. 1993. Treatment Of Multiple Lumbar Disk Herniations In An Adolescent Athlete Utilizing Flexion Distraction And Rotational Manipulation. Journal of Manipulative and Physiological Therapy, 16(3):185-191. Mar.\Apr.

Hirofuji, E. & Tanaka, S. 1983. Computed Tomography Of Lumbar Disc Herniations. Spine, 8(3):300304.

Howe, J.W., Rowe, L.J. & Yochum, T.R.1987. Diagnostic Imaging Of Spinal Stenosis And Intervertebral Disc Disease. In Rowe, L.J., Yochum, T.R. In Essentials Of Skeletal Radiography, 2 vol. pp 273 315. Baltimore:Williams and Wilkins. ISBN 0-683-09329-0.p537

Jonsson, BO. & Stromqvist, B. 1993. Repeat Decompression Of Lumbar Nerve Roots, A Prospective Two-year Evaluation. The Journal Of Bone And Joint Surgery, 75-B(6):894-897. Nov.

Jull G.A. 1990, Aspects Of Conservative Therapy For The Acutely Painful Lumbar Disc, Journal of Manual Medicine, 5:8488.

Kauppila LI, Tallroth K, Postmortem angiographic findings for arteries supplying the lumbar spine: Their relationship to low back symptoms. Journal of spinal disorders, 6(2):124129.

Kikuchi, S. 1982: Anatomical and experimental studies of nerve root infiltration, Journal of Japanese Orthopaedic Association, 56:605-614.

Kirkaldy-Willis W.H.; Relationship Of structural pathology on nerve root. Spine 1984;9(1):49-51

Korr I.M. 1978, The neurobiological mechanisms in Manipulative therapy, Aspects of Manipulative Therapy. p137167

Kuo, P.G.F. & Loh, Z.C. 1987. Treatment of lumbar Intervertebral disc protrusions with manipulation. Clinical Orthopaedics and related research. 215:47:55 Feb.

Lopes, M. 1993, in, Plaugher, (ed.) Textbook of Clinical Chiropractic 527p. ISBN 0683068970

Macnab, I. McCulloch, J. 1986. Backache 2nd ed.,Baltimore: Williams and Wilkins.

Maigne, J., Rime, B. & Delignet, B. 1992. Computed Tomographic Follow-up Study Of Forty Eight Cases Of Nonoperatively Treated Lumbar Intervertebral Disc Herniation, SPINE, 17(9):1071-1074

Margoles, M.S. 1980. Pain Charts, Pain, 8(1):115-117, Feb

Mayer, T.G., Mooney, V. & Gatchel R.J., 1991. Contemporary Conservative Care For Painful Spinal Disorders, Philadelphia:Lea and Febiger:129

Mensor, M.C. 1955. Non-operative treatment including manipulation for lumbar intervertebral disc syndrome. Journal of Bone and Joint Surgery, 37A(5):925:936, Oct.

Mikhael, M.A., Ciric, I., Tarkington, J.A. & Vick, N.A. 1981 Neuroradiological Evaluation of Lateral Recess Syndrome. Radiology 1981; 140 pp97107

Neault, C.C., 1992. Conservative Management Of An L4L5 Left Nuclear Disk Prolapse With A Sequestrated Segment. Journal of Manipulative and Physiological Therapeutics. 15(5):318-321, June

Ogata, K. & Whiteside, L.A. 1981. Nutritional Pathways In The Intervertebral Disc: An Experimental Study Using Hydrogen Washout Technique. Spine 6:211-216

Olmarker, K., Rydevik, B. & Nordborg, C. 1993. Autologous Nucleus Pulposus Induces Neurophysiologic and Histologic Changes In Porcine Cauda Equina Nerve Roots. SPINE, 18(11):1425-1432.

Onel, D., Sari, H. & Donmez, C. 1993. Lumbar Spinal stenosis: Clinical\Radiologic Therapeutic Evaluation in 145 Patients. Conservative Treatment or Surgical Intervention? SPINE, 18(2):291298

Pal, G.P. & Routal R.V. 1987. Transmission Of Weight Through The Lower Thoracic And Lumbar Regions Of The Vertebral Column In Man. Journal of anatomy, 152:93-105

Plaugher, G.(ed.)1993 Textbook of Clinical Chiropractic - A Specific Scientific Approach Williams and Wilkins, pp 198-199 ISBN 0683068970

Rothman, R.H. 1984, A Study Of Computer-Assisted Tomography, Introduction. SPINE, 9(6):548

Saal, J.A. & Saal, J.S. 1989. Nonoperative Treatment Of Herniated Lumbar Intervertebral Disc With Radiculopathy. An Outcome Study. SPINE, 14(4):341437

Saal, J.A., Saal, J.S. & Hertzog, R.J. 1990. The Natural History of Lumbar Intervertebral Disc Extrusions Treated Nonoperatively. SPINE, 15(7):683-686

Simmons, Jr. J.W., Ricketson, R. & McMillin, J.N. 1993. Painful lumbosacral sensory distribution patterns: Embryogenesis to adulthood, Orthopaedic review, pp1110-1118, Oct.

Spencer, D., Irwin, G. & Miller, J., 1983. Anatomy and significance of fixation of the lumbosacral nerve roots in sciatica. Spine, 8(6):672679

Stoker, D.J. Prof. 1977 Statistical tables, 3rd Revised Edition, Academia, Tenth Impression 1994 p.8

Towne, E.B. & Reichert F.L.:Compression of the lumbosacral roots of the spinal cord by thickened ligamentum flava. Annuls of Surgery, 94:327336,1931.

Travell, J.G. & Simons, D.G., Myofascial Pain And Dysfunction, The Trigger Point Manual, vol 2:23-350, Williams and Wilkins, Baltimore, 1992, ISBN 0-683-08367-8, p607

Weber, H. 1983. Lumbar Disc Herniation. A Controlled, Prospective Study Over Ten Years Of Observation. SPINE, 8(2):131-140.

Weber, H., Holme, I. & Amlie, E. 1993. The Natural Course Of Acute Sciatica With Nerve Root Symptoms In A Double-Blind Placebo-Controlled Trial Evaluating The Effect Of Piroxicam. SPINE, 18(11):1433-1438

Wegener, O.H., Fassel, R. & Welger, D. 1993. Whole Body Computed Tomography. Boston:Blackwell Scientific Publications. 710p. ISBN 0865422230

Wiesel, S.W., Tsourmas, N., Feffer, H.L., Citrin, C.M. & Patronas N., 1984. A Study Of Computer-Assisted Tomography, I. The Incidence of Positive CAT Scans In An Asymptomatic Group Of Patients, SPINE, 9(6):549-551.

Wiltse, L.L., Fonseca, A.S., Amster, J., Dimartino, P. & Ravessoud F.A., 1993. Relationships Of The Dura, Hoffman's Ligaments, Batson's Plexus, And A Fibrovascular Membrane Lying On The Posterior Surface Of The Vertebral Bodies And Attaching To The Deep Layer Of The Posterior Longitudinal Ligament. An Anatomical, Radiologic, and Clinical Study. SPINE, 18(8):1030-1043.

Yang, K.H., & King A.I., 1984. Mechanism of facet loaded transmittion as a hypothesis of low back pain. SPINE, 9:557565

Yasuma, T., Arai, K. & Yamauchi, Y., 1993. The Histology Of Lumbar Intervertebral Disc Herniation, The Significance Of Small Blood Vessels In The Extruded Tissue. SPINE, 18(13)1761-1765.

APPENDICES

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

CASE HISTORY

Patient: _____ Date # _____

File #: _____

X-ray #: _____

Age: _____ Sex: _____ Occupation: _____

Intern: _____ Signature: _____

FOR CLINICIAN'S USE ONLY

Initial visit clinician: Signature:

Case History:

Examination:
Previous: TN Current: TN
Other Other

X-ray Studies:
Previous: TN Current: TN
Other Other

Clinical path. lab.:
Previous: TN Current: TN
Other Other

Case status:
PIT: Conditional: Signed off: Final sign out:

Recommendations:

Intern's case history

1. Source of history:

2. Chief complaint: (patient's own words)

3. Present illness:

Location

Onset

Duration

Frequency

Pain (character)

Progression

Aggravating factors

Relieving factors

Associated S & S

Previous occurrences

Past treatment and outcome

APPENDIX A2

4. Other complaints:

5. Past history:

General health status

Childhood illnesses

Adult illnesses

Psychiatric illnesses

Accidents/injuries

Surgery

Hospitalizations

6. Current health status and life-style:
   - Allergies
   - Immunizations
   - Screening tests
   - Environmental hazards
     (home, school, work)
   - Safety measures
     (seat belts, condoms)
   - Exercise and leisure
   - Sleep patterns
   - Diet
   - Current medication
   - Tobacco
   - Alcohol
   - Social drugs

7. Family history:
   - Immediate family:
     - Age
     - Health
     - Cause of death
     - DM
     - Heart disease
     - TB
     - HBP
     - Stroke
     - Kidney disease
     - CA
     - Arthritis
     - Anaemia
     - Headaches
     - Thyroid disease
     - Epilepsy
     - Mental illness
     - Alcoholism
     - Drug addiction
     - Other

8. Psychosocial history:

   Home situation

   Daily life

   Important experiences

   Religious beliefs

9. Review of systems:

   General

   Skin

   Head

   Eyes

   Ears

   Nose/sinuses

   Mouth/throat

   Neck

   Breasts

   Respiratory

   Cardiac

   Gastro-intestinal

   Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematologic

Endocrine

Psychiatric.

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

PHYSICAL EXAMINATION

Underline abnormal findings in RED and elaborate on back of relevant page, if necessary.
Mark "WNL" if normal.

Patient:_____ File #_____

Last name First name

Clinician:_____ Signature:_____

Intern:_____ Signature:_____

Date:_____

Height:_____ Weight:_____ Temp:_____

Rates: Heart:_____ Pulse:_____ Respiration:_____

Blood pressure: Arms: L / R /

Legs: L / R /

General appearance:

STANDING EXAMINATION.

Minor's sign
Skin changes
Posture
  erect
  Adam's
Ranges of motion:

T/L spine: Flexion: 90 Fingers to floor
Extension: 50
R.lat.flex.: 30 Fingers down leg
L.lat.flex.: 30 Fingers down leg
Rot.to R.: 35
Rot.to L.: 35

Flex.

L.Rot. R.Rot.

L.lat flex. R.lat. flex.

Ext.

/ = pain-free limitation; // = painful limitation.

Romberg's sign.
Pronator drift.
Trendelenburg's sign.
Gait.
  rhythm
  balance
  pendulousness
  on toes
  on heels
  tandem
Half squat.
Scapular winging.
Muscle tone.
Spasticity/Rigidity.

APPENDIX B2

Shoulder:
- skin
- symmetry
- ROM - glenohumeral
  - scapulo-thoracic
  - acromioclavicular
  - elbow
  - wrist

Chest measurement
- inspiration
- expiration

Visual acuity

Breast examination:
- Inspection:
  - skin
  - size
  - contour
  - nipples
  - arms overhead
  - hands against hips
  - leaning forward.
- Palpation:
  - axillary lymph nodes.

SEATED EXAMINATION.

Spinal posture

Head
- scalp
- skull
- face
- skin

Eyes
- conjunctiva
- sclera
- eyebrows
- eyelids
- lacrimal gland
- nasolacrimal duct
- alignment
- corneal reflex
- ocular movement

	L			R	
III	IV	VI	III	IV	VI

- visual fields
- accomodation
- iris
- pupils
- red reflex
- optic disc

APPENDIX B3

vessels
general background
macula
vitreous
lens

Ears:
auricle
ear canal
drum
auditory acuity
Weber test
Rinne test

Nose:
external
internal
septum
turbinates
olfaction
Sinuses (frontal & maxillary):
tenderness
transillumination
Mouth and pharynx:
lips
buccal mucosa
gums and teeth
roof
tongue
inspection
movement
taste
palpation
pharynx
inspection
CN X
Neck:
posture
size
swelling
scars
discoloration
hair line

ROM:

Flexion:	45	chin to larynx
		chin to sternum
Extension:	55	forehead parallel to floor
L.lat.flex:	40	
R.lat.flex:	40	
L.rot.:	70	
R.rot.:	70	

Flex.

L.Rot. R.Rot.

L.Lat. flex. R.lat. flex.

Ext.

lymph nodes
trachea
thyroid
carotid arteries (thrills, bruit)
CN V
CN VII
CN VIII (nystagmus)
CN IX
CN XI
TMJ --
Inspection
ROM
deviation
Palpation
crepitus
tenderness

Neurological:
- Dermatomes
  - C5
  - C6
  - C7
  - C8
  - T1
- Tendon reflexes
  - biceps
  - triceps
  - brachioradialis
- Muscle strength
  - C5
  - C6
  - C7
  - C8
  - T1
- Coordination:
  - point-to-point
  - dysdiadochokinesia

Thorax:
- Chest:
  - Inspection:
    - skin
    - shape
    - respiratory distress
    - rhythm (respiratory)
    - depth
    - effort
    - intercostal/supraclavicular retraction
  - Palpation:
    - tenderness
    - masses
    - respiratory expansion
    - tactile fremitus
  - Percussion:
    - lungs (posterior)
    - diaphragmatic excursion
    - kidney punch
  - Auscultation:
    - breath sounds
      - vesicular
      - bronchial
    - adventitious sounds
      - crackles (rales)
      - wheezes (rhonchi)
    - voice sounds
      - broncophony
      - whispered pectoriloquy
      - egophony

APPENDIX B6

Cardiovascular:
auscultation (aortic murmurs)
Allen's test

SUPINE EXAMINATION

JVP
PMI
auscultation heart (L.lat.recumbent)
respiratory excursion
percussion chest (anterior)
breast palpation
The abdomen:
Inspection:
skin
umbilicus
contour
peristalsis
pulsations
hernias (umbilical/incisional)
Auscultation:
bowel sounds
bruit
Percussion:
general
liver
spleen
Palpation:
superficial reflexes
cough
light
rebound tenderness
deep
liver
spleen
kidneys
aorta
intra-/retro-abdominal wall mass
shifting dullness
fluid wave
Acute abdomen:
where pain began and now
cough
tenderness
guarding/rigidity
rebound tenderness
Rovsing's sign
psoas sign
obturator sign
cutaneous hyperaesthaesia
rectal exam
Murphy's sign.

Male genitals and hernias.

Inspection:

skin

prepuce

glans

meatus

nits/lice

scrotum

inguinal/femoral bulges

Palpation:

penis (tenderness/induration)

testes

epididymis

inguinal canal

femoral canal

cremasteric reflex

Auscultation:

scrotal mass.

Peripheral vasculature:

Inspection:

skin

nail beds

pigmentation

hair loss

Palpation:

pulses - radial, brachial, femoral, popliteal, post.tibial, dorsalis pedis

lymph nodes - epitrochlear, femoral (horizontal & vertical)

temperature (feet & legs)

Manual compression test

Retrograde filling (Trendelenburg) test

Arterial insufficiency test

Musculoskeletal:

ROM

hip

flex. 90/120

ext. 15

abd. 45

add. 30

int rot 40

ext rot 45

knee

flex. 130

ext. 0/15

ankle

plantar flex 45

dorsiflex 20

inversion 30

eversion 20

leg length

APPENDIX B8

Neurological:
- dermatomes
  - L1
  - L2
  - L3
  - L4
  - L5
  - S1
- muscle strength
  - hip flexion
  - knee extension
  - ankle dorsiflexion
  - plantar flexion
- tendon reflexes
  - patellar
  - Achilles
- plantar reflex

Rectal examination:
- Inspection
  - sacrococcygeal & perianal areas
- Palpation
  - sphincter tone
  - tenderness
  - indura:
  - nodules
  - prostate
  - seminal vesicles

Mental status
- Appearance and behaviour:
  - level of consciousness
  - posture and motor behaviour
  - dress, grooming, personal hygiene
  - facial expression
  - affect
- Speech and language:
  - quantity
  - rate
  - volume
  - fluency
  - aphasia (pra)
- Mood
- Thought processes (logical, relevant, organised)
- Memory and attention:
  - orientation (time, place, person)
  - remote memory
  - recent memory
  - new learning ability

APPENDIX B9

- Higher cognitive functions:
  - information and vocabulary (general & specialised knowledge)
  - abstract thinking.

TECHNIKON NATAL

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC.

REGIONAL EXAMINATION -- LUMBAR SPINE AND PELVIS.

PATIENT:_____

FILE # :_____ DATE:_____

INTERN/RESIDENT:_____

SUPERVISING CLINICIAN :_____

STANDING :

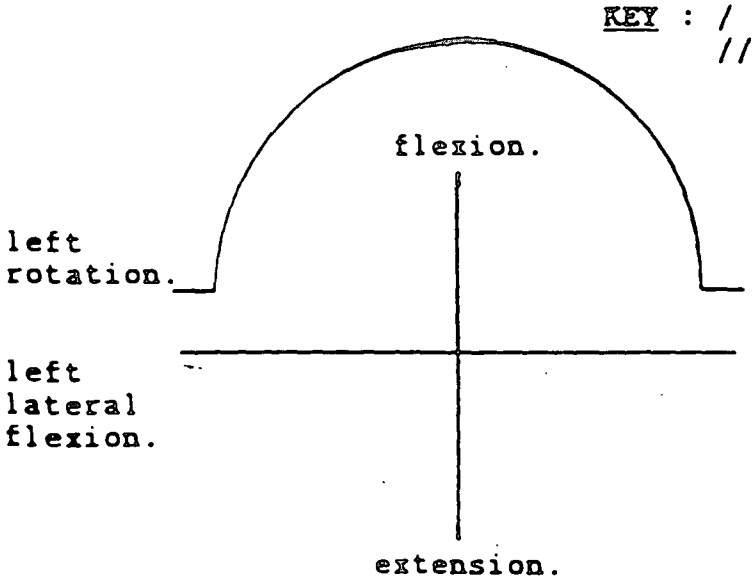
Posture
Minor's Sign
Skin
Scars
Discoloration
Muscle tone
Bony and soft tissue contours

Spinous percussion
Schober's Test (6cm)
Treadmill
Body Type
Attitude

RANGE OF MOTION.

Forward Flexion = 40-60 degrees.(15cm from floor)
Extension = 20-35 degrees.
L/R Rotation = 3-18 degrees.
L/R Lateral flexion = 15-20 degrees.

KEY : / PAINLESS LIMITATION.
// PAINFUL LIMITATION.



SUPINE :

Skin.
Hair.
Nails..

Observe abdomen
Fasciculations
Abdominal reflexes
Auscultate abdomen/groin
Palpate abdomen/groin
Pulses (abdomen)
Pulses (extremities)

SLR
Bowstring
Plantar reflex
Circumference (thigh, calf)
Leg length :
- actual
- apparent

Sciatic notch
Patrick Faber
Gaenslen's Test
Gluteus Maximus Stretch
Hip medial rotation
Psoas Test
Thomas' Test :
- hip joint
- rectus femoris

LATERAL RECUMBENT :

S-I compression
Ober's Test
Femoral nerve stretch
Myotomes :
- QL
- Gluteus Medius

PRONE :

Gluteal skyline
Skin rolling
Iliac crest compression
Facet joint challenge
S-I tenderness
Erichson's Test
Pheasant's Test
Myotomes :
- GluteusMaximus

Active MF Trigger Points:
- QL
- Glut. Med.
- Glut. Max.
- Glut. Min.
- Piriformis
- Hamstrings
- TFL

NON-ORGANIC SIGNS :

Pin Point Pain.
Axial Compression.
Trunk Rotation.
Burn's Bench Test.
Flip Test.
Hoover's Test.
Ankle Dorsiflexion Test.

MOTION PALPATION :

Jt.play		Left						Right					Jt.play	
A	Lat	Fle	Ext	LF	AR	PR		Fle	Ext	LF	AR	PR	P/A	Lat
							T10							
							T11							
							T12							
							L1							
							L2							
							L3							
							L4							
							L5							
					U	L	SI	U	L					

GAIT :

Rhythm
On toes (standing)
On heels (standing)
Half-squat on one leg

Remarks : _____

_____

_____

_____

NEUROLOGICAL EXAMINATION :

DERMATOMES: Left	Right.	MYOTOMES: Left	Right.	REFLEXES: Left	Right
T12		hip flex		C5	
L1		hip int rot		C6	
L2		hip ext rot		C7	
L3		hip abd			
L4		hip add			
L5		knee flex			
S1		knee ext			
S2		dorsiflex			
S3		plantarflex			
		eversion			
		ext.hall.long			

Tripod
Kemp's Test

COMMENTS: _____

_____

_____

_____

OSWESTRY BACK DISABILITY INDEX

PATIENT NAME:_____ FILE #:_____ DATE:_____

This questionnaire has been designed to give the doctor information as to how your back pain has affected your ability to manage in everyday life. Please answer every section and mark in each section only the ONE box which applies to you. We realize you may consider that two of the statements in any one section relate to you, but please just mark the box which most closely describes your problem.

Section 1 - Pain Intensity

- ☐ I have no pain at the moment.
- ☐ The pain is very mild at the moment.
- ☐ The pain is moderate at the moment.
- ☐ The pain is fairly severe at the moment.
- ☐ The pain is very severe at the moment.
- ☐ The pain is the worst imaginable at the moment.

Section 2 - Personal Care (Washing, Dressing etc.)

- ☐ I can look after myself normally without causing extra pain.
- ☐ I can look after myself normally but it causes extra pain.
- ☐ It is painful to look after myself and I am slow and careful.
- ☐ I need some help but manage most of my personal care.
- ☐ I need help every day in most aspects of self care.
- ☐ I do not get dressed, I wash with difficulty and stay in bed.

Section 3 - Lifting

- ☐ I can lift heavy weights without extra pain.
- ☐ I can lift heavy weights but it gives extra pain.
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table.
- ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.
- ☐ I can lift very light weights.
- ☐ I cannot lift or carry anything at all.

Section 4 - Walking

- ☐ Pain does not prevent me walking any distance.
- ☐ Pain prevents me walking more than 1 mile (2.2 km).
- ☐ Pain prevents me walking more than 1/2 mile (1.1 km).
- ☐ Pain prevents me walking more than 1/4 mile (0.5 km).
- ☐ I can only walk using a stick or crutches.
- ☐ I am in bed most of the time and have to crawl to the toilet.

Section 5 - Sitting

- ☐ I can sit in any chair as long as I like.
- ☐ I can only sit in my favorite chair as long as I like.
- ☐ Pain prevents me from sitting more than 1 hour.
- ☐ Pain prevents me from sitting more than 1/2 hour.
- ☐ Pain prevents me from sitting more than 10 minutes.
- ☐ Pain prevents me from sitting at all.

Section 6 - Standing

- ☐ I can stand as long as I want without extra pain.
- ☐ I can stand as long as I want, but it gives me extra pain.
- ☐ Pain prevents me from standing for more than one hour.
- ☐ Pain prevents me from standing for more than 30 minutes.
- ☐ Pain prevents me from standing for more than 10 minutes.
- ☐ Pain prevents me from standing at all.

Section 7 - Sex Life

- ☐ My sex life is normal and causes no extra pain.
- ☐ My sex life is normal but causes some extra pain.
- ☐ My sex life is nearly normal but it is very painful.
- ☐ My sex life is severely restricted by pain.
- ☐ My sex life is nearly absent because of pain.
- ☐ Pain prevents any sex life at all.

Section 8 - Social Life

- ☐ My social life is normal and gives me no extra pain.
- ☐ My social life is normal but increases the degree of pain.
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests, for example, dancing.
- ☐ Pain has restricted my social life and I do not go out as often.
- ☐ Pain has restricted my social life to my home.
- ☐ I have no social life because of pain.

Section 9 - Sleeping

- ☐ I have no trouble sleeping.
- ☐ I can sleep well only by using pills.
- ☐ Even when I take pills I have less than six hours sleep.
- ☐ Even when I take pills I have less than four hours sleep.
- ☐ Even when I take pills I have less than two hours sleep.
- ☐ Pain prevents me from sleeping at all.

Section 10 - Travelling

- ☐ I can travel anywhere without extra pain.
- ☐ I can travel anywhere but it gives me extra pain.
- ☐ Pain is bad but I manage trips over two hours.
- ☐ Pain restricts me to trips of less than one hour.
- ☐ Pain restricts me to trips under 30 minutes.
- ☐ Pain prevents me from travelling, except to the doctor or hospital.

NUMERICAL RATING SCALE

Please indicate on the line below the number between 0 and 10 that best describes the pain of your major problem at this point, when it is at its worst. A zero (0) would mean "no pain at all" and ten (10) would mean "pain as bad as it could be."
Please write only one number.

0 _____ 10

DATE: _____

DIAGNOSIS _____

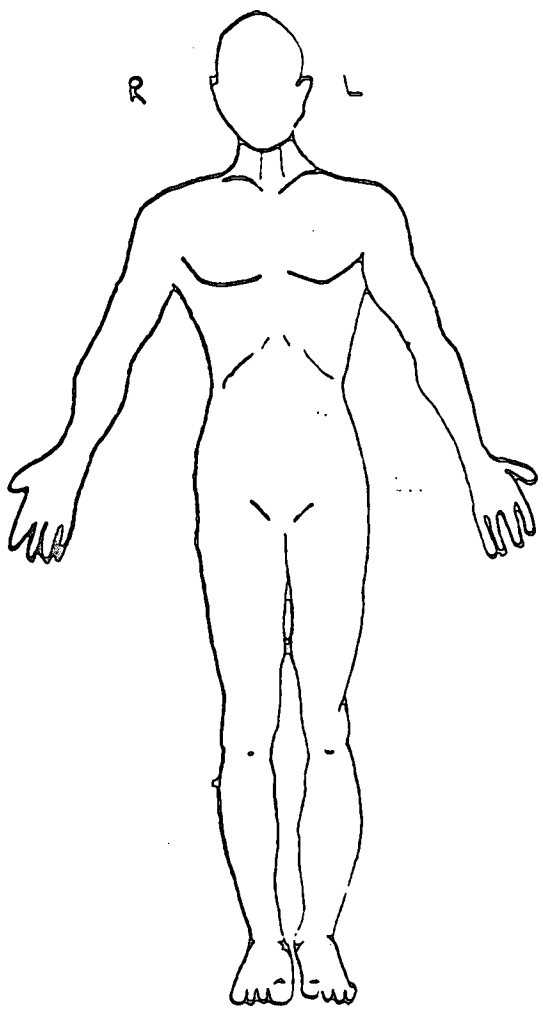
APPENDIX E1

PATIENT NAME: _____ **FILE #:** _____ **DATE:** _____

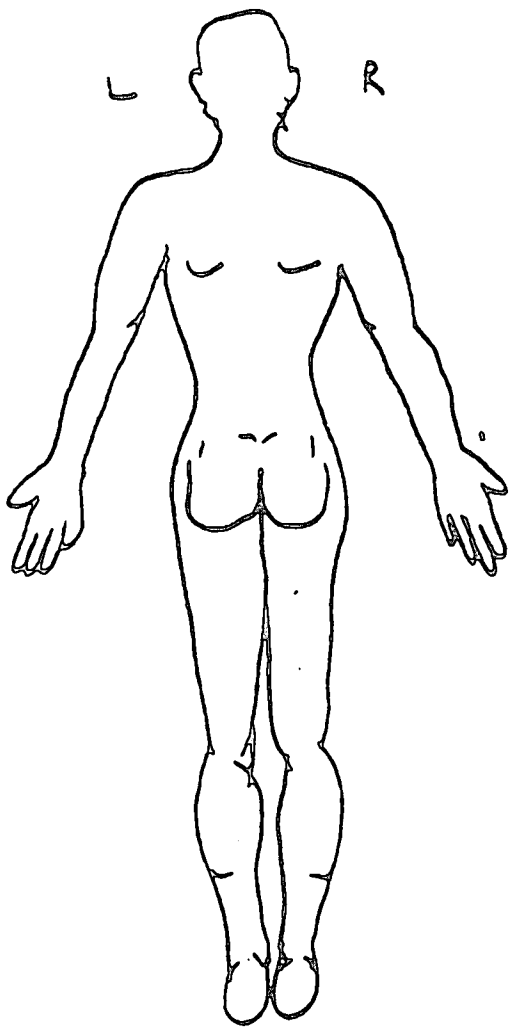
In the diagram provided below, please mark the areas on your body which you feel best represent the pain(s) or sensation(s) you are experiencing. Please include all areas. Use the symbols provided below.

SYMBOLS

numbness	=== ===	pins and needles	....
burning	XXX XXX	stabbing and sharp	//// ////
dull and aching	+++ ++-	stiff and tight	ZZZ ZZZ



FRONT



BACK

APPENDIX F1