"LUMBAR VERSUS CAUDAL EPIDURAL INJECTION OF STEROID IN CHRONIC LOW BACK ACHE & SCIATICA A RANDOMIZED CONTROLLED TRIAL"

By

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MAY - 2012

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I hereby declare that this dissertation entitled **"LUMBAR VERSUS CAUDAL EPIDURAL INJECTION OF STEROID IN CHRONIC LOW BACK ACHE & SCIATICA, A RANDOMIZED CONTROLLED TRIAL"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. V. G. MURAKIBHAVI**MS (Ortho), D (Ortho), FAOI Professor & Head, Department of Orthopaedics, J. N. Medical College, Nehru Nagar, Belgaum-590010.

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LIST OF ABBREVIATIONS

1. Beck's Depression Inventory Scoring – BDIS
2. Oswestry Disability Index Score – ODIS
3. Numerical Pain Intensity Score - NPIS
4. Straight Leg Raising Test - SLRT
5. Visual Analogue Scale - VAS

ABSTRACT

Purpose: To compare the efficacy of epidural steroid injection, using either the lumbar or the caudal route in patients suffering from low back pain & sciatica.

Methods: 133 cases of severe low back pain with sciatica > 3 months attending the orthopaedic outpatient clinic at our tertiary care hospital were selected after screening for inclusion criteria. All cases answered Oswestry Disability Index questionnaire, Becks Depression Inventory Questionnaire, Numerical Pain Intensity Score, Visual Analogue Score & underwent physical examination, before & immediately after intervention & then at 3 weeks, 3 months & 6 months. Pain relief, disability, & activity levels were assessed & statistically evaluated. The patients of Group A underwent lumbar epidural injection while Group B underwent caudal epidural injection both Xylocaine (2 ml) & Steroid (2 mL).

Results: Symptoms improved in both the groups, with group A having a statistical significance. The mean ODIS, the mean BDIS, the mean NPIS, as well as the VAS score were statistically significant lower than at prior to time of enrolment. Seven patients underwent a second caudal injection with six of them reporting improvement while one did not respond well & underwent decompression (n=1). Twelve patients underwent a second lumbar injection with eight of them reporting improvement while four did not respond well & underwent decompression (n=4).

Conclusion: Caudal epidural steroid injections seem to be effective when treating patients with low back pain & sciatica. They are easy to perform, less technically demanding, with lower complications compared to other routes of epidural injection placement & less expensive way of treating chronic low back pain.

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INTRODUCTION

"For all happiness mankind can give, is not in pleasure but in rest from pain"

- John Dryden

Pain may serve a number of functions including protection (the burnt fingers in a patient of Hansen), it could be defensive (immobilization of an inflamed joint), or diagnostic (acute abdomen). Backache has haunted mankind since time immemorial. Its history can be traced back to the evolution of bipedal gait.

With or without sciatica, backache is a symptom of some underlying pathology, be it merely a soft tissue strain or disc protrusion or more sinister conditions such as neoplasm or ankylosing spondylitis.

It is of such a common occurrence that at some time or other, at any age, most of us have experienced it [1]. Apart from suffering a heavy drain on manpower, it wrecks the economy of the industry. Not only that, it is costly to the National Health Service & the tax payers, to keep such a patient in a hospital [2].

The variable character of low back pain, its multiplicity of causes & difficulties in its treatment render this affection one of the most perplexing & frequent problems that confront the orthopaedic surgeon. The forms of treatment used are numerous. At one end of the spectrum is the regime of bed rest on hard boards & analgesics, forming the main pillar of the treatment while at the other, there is the operative removal of the prolapsed disc. Even in the conservative line of treatment, there is no universally accepted policy & the best form of treatment. This wide & rather haphazard spectrum of treatment suggests that there is no single satisfactory method of treatment that ensures permanent & long lasting cure.

The above facts are all the more pertinent in a country like ours, for three main reasons:

1. Non-availability of specified medical aid in places remote from cities.
2. Shortage of beds in Hospitals & consequent heavy strain on the existing ones, necessitating a fast turn over.
3. The inability of an average patient to stay for long in the hospital even if it were possible, as he may be the only bread earner in the family.

AIMS & OBJECTIVES

1. To compare the efficacy of epidural steroid injection using either the lumbar or the caudal route in low backache & sciatica.
2. To assess the outcome in terms of pain relief, neurological status, activity levels, disability using Oswestry Disability Index, Visual Analogue Scale, Numerical Pain Intensity Scores.
3. To assess the depression associated with low back & sciatica using the Beck's Depression Inventory Scores.
4. To find out any predictors of good outcome

REVIEW OF LITERATURE

Historical Review

Backache & sciatica are symptoms that have been recorded for centuries, but their common pathology & relationship has been recognized only recently. Various related points were known in isolation, but it was left to Mixter & Barr in 1934, to correlate them & put them in a comprehensive form [3].

In 1555, Andreas Vesalius, described the intervertebral disc in his treatise, "Dehumani Corporis Fabrica".

Cotugnio described "sciatica" as a clinical entity in 1764, in his book "De ischiade nervosa commentarios", & also differentiated it from the pain generated from hip disease.

In 1854, Virchow described traumatic herniation of the fibrocartilage of the disc during an autopsy, which later came to be known after him as a tumor. The intervertebral disc was described in detail by Von Luschka in 1858. Kocher also described traumatic rupture of the intervertebral disc reported from an autopsy in 1898.

The first attempt to correlate sciatica with backache was in 1867, by Laseque who described the posture & gait in sciatica, & also devised the "Sciatic nerve stretch test". In 1888, Charcot described the spinal deformity associated with sciatica, & Brissot in 1890 called it "Sciatica Scoliosia".

Early in the twentieth century, Goldthwait noticed a sudden development of cauda equina lesion, in a patient under treatment for lumbosacral strain. He attributed the symptoms to subluxation at the lumbosacral joint, & suggested that it was

posterior displacement of the discs. The same year, Middleton & Teacher described sudden paraplegia in a man due to disc retropulsion while lifting heavy weight.

In 1916, Sicard put forth his theory which stated that sciatica was due to irritation of the nerve roots, which he termed as "neurochorditis". His theory of root irritation was supported by Putti (1927), who felt that the lesion commonly was at the intervertebral foramen & due to arthritis of the facet joints following variations in their planes. In the late 1920's, Schmorl with Junghans, described the pathological anatomy of the spinal column & intervertebral discs, & that the herniation of the disc can occur in any direction, including posteriorly into the spinal canal, & anteriorly into the prevertebral region. During the same period Dandy confirmed by his studies that the tumors earlier described by Elsberg were in fact cartilaginous loose fragments extruded from the disc. In 1934, came the first published report by Mixter & Barr summarizing the previous knowledge regarding disc lesions & they suggested surgical removal for relief of the symptoms. They documented their findings in 30 clinical cases with unilateral sciatica.

In 1941, Dandy described the "Concealed Disc" or intermittent herniation of the nucleus pulposus. After the 2nd world war Key, Burns & Young pointed out that, disc herniations could produce low back pain without sciatica. In the next few years, scientists from all over the world evaluated causes for these herniations. Charnley suggested fluid imbibition; Scott stated the factor of mental stress, & Lindblom suggested compression as a causative factor. With these postulations, factors other than mere trauma came to receive increasing attention, & aided in establishing the present view of multifactorial origin of low back pain & sciatica.

History & Evolution of Epidural Injections

In 1855, Wood of Edinburgh popularized the use of a hollow needle, which has been described 10 years earlier by Rynd of Dublin, & the use of the glass syringe, which has been devised by Paraviz of France. Introduction of the syringe & needle into clinical practice proved a big milestone in the management of many ailments & disease disorders, which were treated by injection of opiates, chloroform, bromides, etc., near the nerve trunks.

Leonard Corning (New York) used the epidural space in 1885 to produce spinal analgesia with injections of cocaine in the dog & subsequently used the same in man for seminal incontinence & spinal weakness [4].

In 1901, Sicard employed extradural cocaine by the caudal route to treat cases of lumbago & sciatica. He subsequently described the interspinous approach to the epidural space [5].

During this period, there were various attempts to establish epidural injection as modality of treatment for sciatica One such attempt was made by Caussade & Queste in 1909, reviewed several cases of sciatica relieved by spinal injection of stovocaine, but their selection criteria & technique cases & description of technique were vague [6]. Page described the technique of Lumbar epidural analgesia [7].

Almost a quarter century after Sicard, Viner published a small series of cases of sciatica which was treated by caudal extradural injection of procaine & 50-100 ml normal saline or liquid petroleum [8].

Dogliotti (1933), an Italian described at greater length the technique of lumbar epidural injections, & described the loss of resistance sign [9].

Further research came from Karl Koller in 1929, who described the clinical use of cocaine for anesthesia [10]. Evans (1930) reported a case series where he successfully used varying amounts of fluid (normal saline, 1% procaine, 2% procaine, & 1% procaine followed by normal saline) [11]. Around 1950, Cyriax described the use of epidural injections with procaine as a diagnostic test of differentiate between lesions outside the canal & those inside it, & at the same time noted its therapeutic value. He later laid down compressive indications for selection of patients & advocated epidural anesthesia as the conservative treatment of choice in patients of low lumbar disc lesions with nerve root pressure & neurological signs, the one contra indication being the presence of bladder symptoms [12]. Liever et al, in 1953 were the first to explore the space via the sacral route [13]. Goebert et al. in 1961 were the first to report use of hydrocortisone acetate in 113 patients with painful radiculopathy [14]. In 1961, Coomes compared the results of bed rest & epidural injections only to conclude the superiority of the latter. Barry & Kendal in 1962 got similar results as Goebert with the use of cortisone [15].

The largest study of the 20th century came from Swerdlow & Sayle-Creer in 1970, who described a series of 5000 cases of backache. They were treated with saline & lignocaine epidural injections, saline injections only, & injections containing lignocaine with methyl prednisolone, through the lumbar & caudal routes & were followed up for at least 12 months. They conclude from their studies that in time of recovery from severe pain, that hospitalization or long periods of rest should be avoided & physiotherapy should be started early. They stated that the epidural injection may avoid the need for surgery [16].

There was more research with different combinations by Pamela Daly, Beliveau (1971) & Warr et al (1972). The latter opined though the epidural injection is "not the cure-all of any back pain", it is the "best method currently available, short of laminectomy, for all cases of syndrome, young or old, mild or severe, acute or chronic, but for the contraindications". They concluded by saying that "the ability to achieve in hours what may other-wise take weeks or months, commends this form of treatment" [17,18].

Dilke, Burry & Grahame in 1973, published the results of a study of a double-blind controlled trail on 100 cases given epidural corticosteroid injections by the lumbar route & 100 control cases. They reported statistically highly significant differences with respect to relief of pain & resumption of normal duties in favor of the group treated by extradural injections & feel that it "seems to be a valuable adjunct to the management of lumbar nerve root compression syndromes, associated with degenerative disc disease" [19].

In 1977, Dr. R. K. Sharma has reported on a series of 201 cases of low back pain with sciatica, given 40 ml of saline, 0.5% lignocaine hydrochloride with 80 mg of methylprednisolone via the caudal route he obtained good to very good results in 56.2%, fair in 23.9% & no improvement in 19.9% of cases. In the same year Brevick et al discussed a series of 53 low backaches with sciatica cases which they treated with Caudal Epidural injections using Depomethylprednisolone & Bupivocaine with excellent long & short term results [20].

Kelman in 1994 reported on 486 injections & remarked that the relief of pain lasted the known effect of anesthesia [21]. In 1997 Carette et al reported their results in the New England Journal of Medicine on a 158 cases with disc prolapse & radicular

pain, given methyl prednisolone (80 mg) & saline (8 ml). They remarked significant improvement following these lumbar injections & reported a good short term follow up [22].

In the beginning of the twenty first century, Manchikanti et al reported a case series of 62 patients with discogenic pain, who were given caudal epidural injections & remarked a positive short as well as long term follow up [23]. In 2005, Arden et al reported [24] on a series of 228 cases of low back ache with unilateral sciatica, given 80 mg of Triamcinolone Acetate & 10 ml of 0.25 % Bupivocaine via the lumbar route they obtained excellent results in the short term. At the same time, Wilson-Mcdonald et al [25] reported on a series of 93 patients with MRI evidence of a disc prolapse, spinal stenosis, with lumbosacral nerve root pain which had not resolved within a minimum of 6 weeks.

Ackerman et al published their findings in discogenic pain using different routes of epidural injections in 2007 [26]. Salahadin Abdi et al in a study conducted in Miami stated that there is limited evidence in the lumbar spine for long-term relief by interlaminar lumbar injections while both lumbar transforaminal & caudal had moderate long-term relief in managing nerve root pain & chronic low back pain [27]. Abdi et al once again evaluated the effect of lumbar interlaminar epidural injections with or without steroids in managing various types of chronic low back & sciatica [28].

In 2009, Sayegh et al reported a case series of 193 cases with low back pain & sciatica. They evaluated these cases after using Caudal Epidural injections with or without steroid & concluded that steroid containing preparations demonstrated better & faster efficacy [29].

Anatomy [30-33]

The central axis of the human skeleton is formed by the vertebral column. At its upper & through two modified vertebrae – the Atlas & the Axis it supports the skull. In the thoracic region it articulates with the rib cage which, in turn, articulates with the pectoral girdle & upper limbs. Finally through sacral vertebrae, it articulates with the pelvic girdle to which the lower limbs are attached. The lumbar vertebrae do not have any articulations. The variety in its size & architecture lends special advantages to each region, viz. , mobility & range of movements in the cervical & lumbar regions, & the comparative rigidity in the thoracic an sacral regions, required for their articulations which the upper & lower limbs respectively. The various ligaments & muscles attached to the column lend to its great strength & ruggedness & also great flexibility.

"**The backbone is flexible because it has so many joints so close together**"

- Last

The familiarity with anatomical relationship & the lesion in the region of the spinal column is essential to an understanding of prolapse of an intervertebral disc. Kester has introduced the concept of the functional spinal unit, which consists of:

(a) Two adjacent vertebrae with their articulations

(b) An intervening Disc

(c) Ligaments (Figure 1)

- Anterior Longitudinal Ligaments
- Posterior Longitudinal Ligaments
- Ligamentum Flavum
- Interspinous Ligaments

In the position of normal stance, balance is maintained by intrinsic structural stability & supported by the tone of the various postural muscles, which are controlled by various receptors, the semi-circular canals, the neck, the skin, muscles & joints, & the sole of the foot.

The weight of the trunk is centered over its base by three curves (Figure 2):

(i) Cervical Lordosis

(ii) Thoracic Kyphosis

(iii) Lumbar Lordosis

Lumbar Lordosis is functional & can be reduced, reversed or increased by muscular actions of gravity. Normally lumbar lordosis is reduced on forward flexion & accentuated on extension. These various curves are produced due to variations in the size & width of the vertebral bodies. Kyphosis starts at L3 due to reversal of wedge form. The vertebral ridges & articular processes thereby lie closer together & the shape & the size of the foramen are also affected.

Spinal Canal (Figure 3, 4)

Spinal canal is formed by superimposition of vertebrae, bounded ventrally by the dorsal surfaces of the bodies of the vertebrae, intervertebral discs, & the posterior longitudinal ligament. Dorsally & laterally it is bounded by pedicles, laminae, transverse processes & spinous processes, & the ligamentum flavum, which can be quite thick at times. The posterior longitudinal ligament is less well developed over the lower two vertebrae, which is loosely attached to the bodies but firmly attached to the inter-vertebral disc (Malmros).

Figure 1 – A Functional Spinal Unit

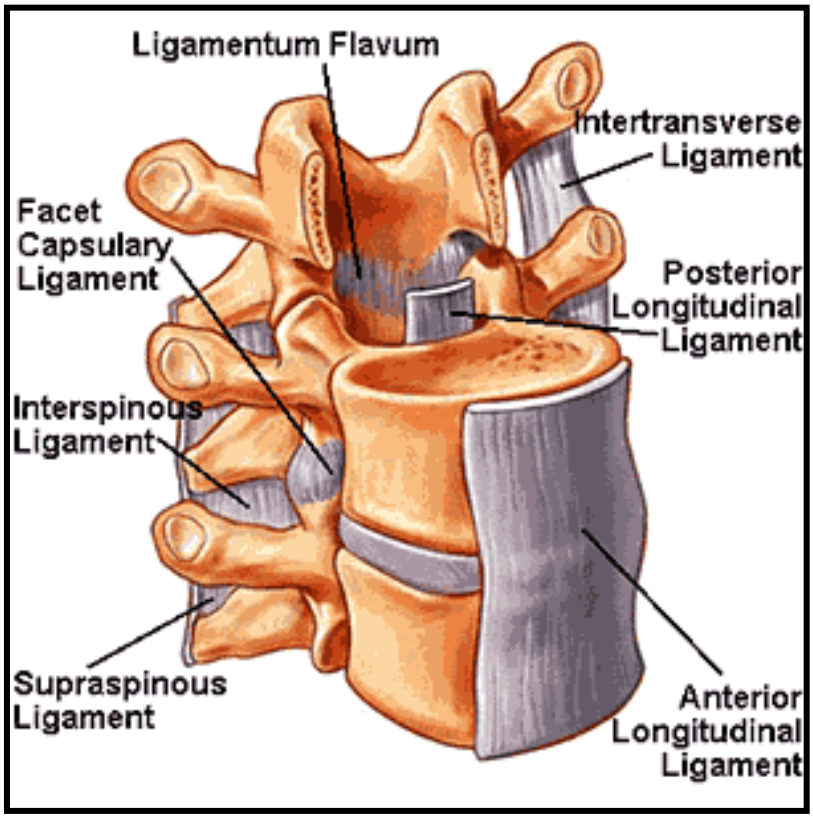
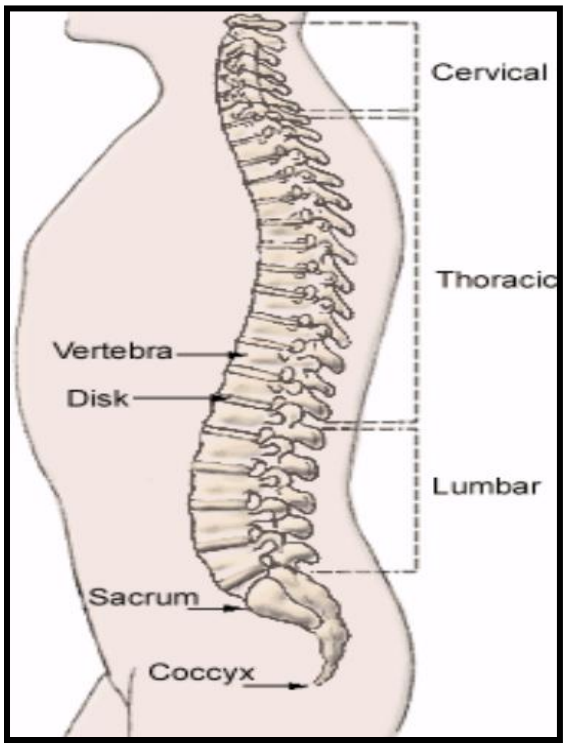


Figure 2 – The Spinal Column



Contents – Lumbo - Sacral Spinal Canal:

(a) The Dural Sac containing the spinal cord & the nerve roots, which ends at S2 – S3, & becomes filum terminale

(b) Cauda Equina.

(c) Cerebro-spinal fluid.

(d) The Epidural Space. This is wider at the dorsal side as a result of the dural sac lying more closely against the cerebral bodies. It contains fat, connective tissue, venous plexuses, & the emerging spinal roots.

The spinal cord ends between L1 & L2. From here, the dural sac contains only the lower nerve roots & the conus medularis, which together form the cauda equina. Protection of the delicate nervous system from shocks is afforded by:

1. The Bony Spinal Canal
2. Cerebrospinal fluid
3. Epidural cavity

Spinal Nerve Roots (Figure 5):

The lumbar nerve roots emerging from the dural sac reach the intervertebral foramina after descending obliquely in the sulci lateralis & along the pedicles. From the point of exit from the dural sac they are surrounded by the dural sheath. They are susceptible to be compressed in the sulci lateralis as there is little room for displacement.

Figure 3 - Contents of the Spinal Canal

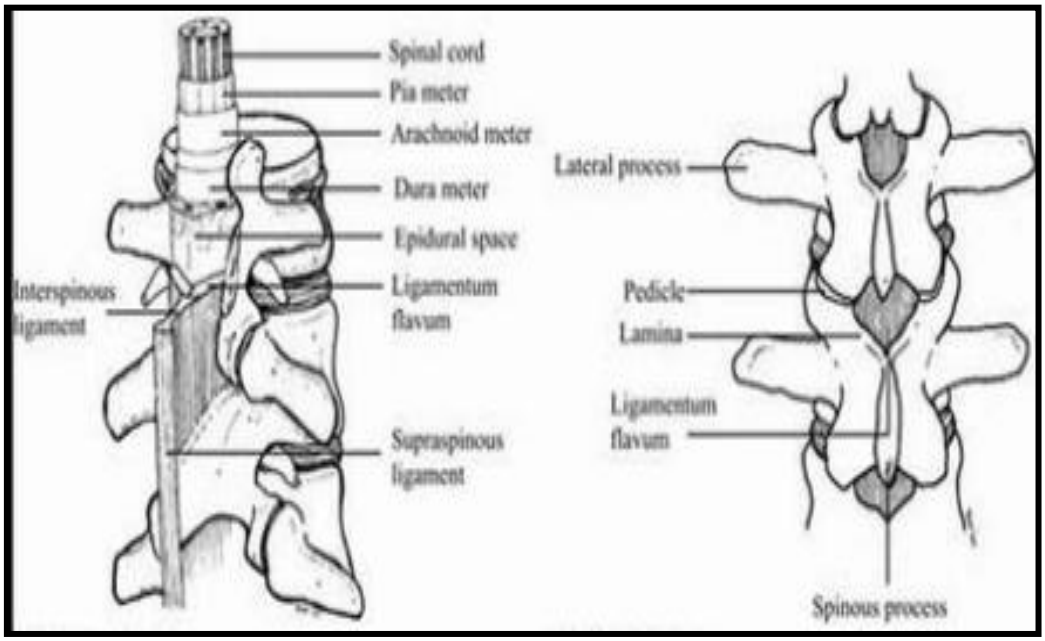


Figure 4 – Sacral Canal

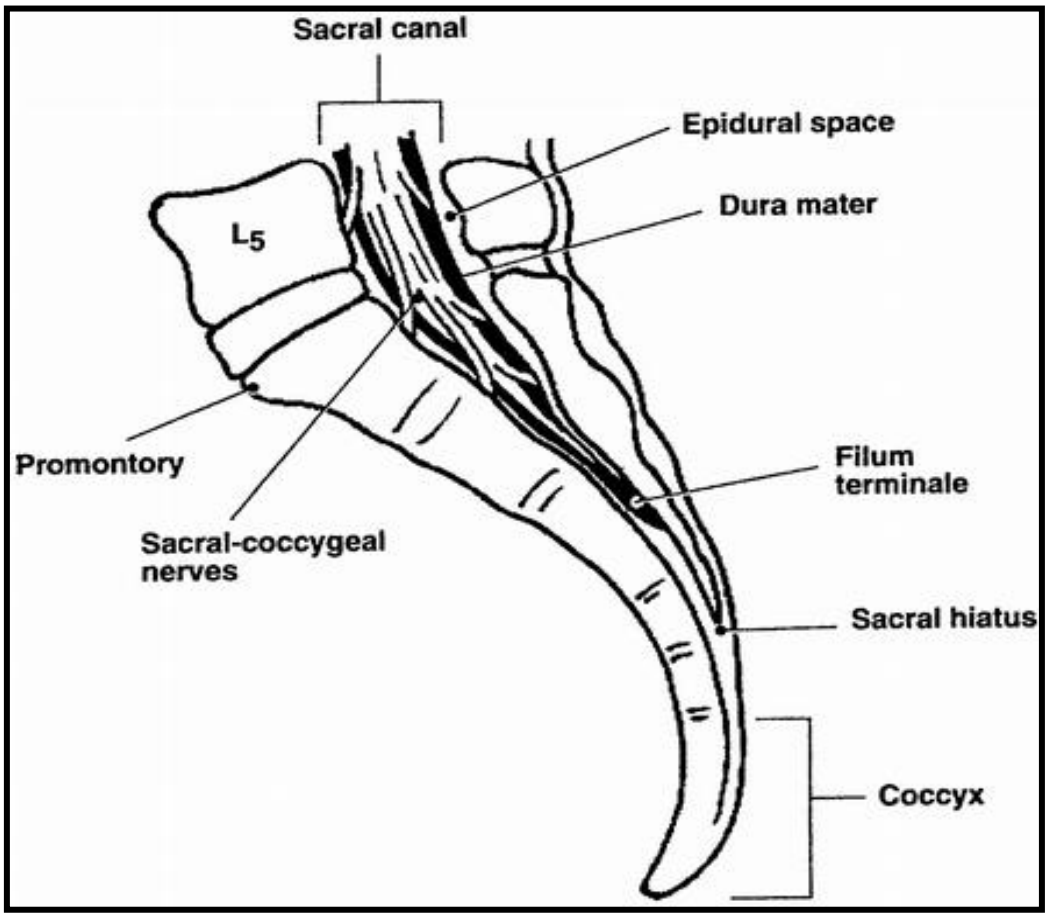
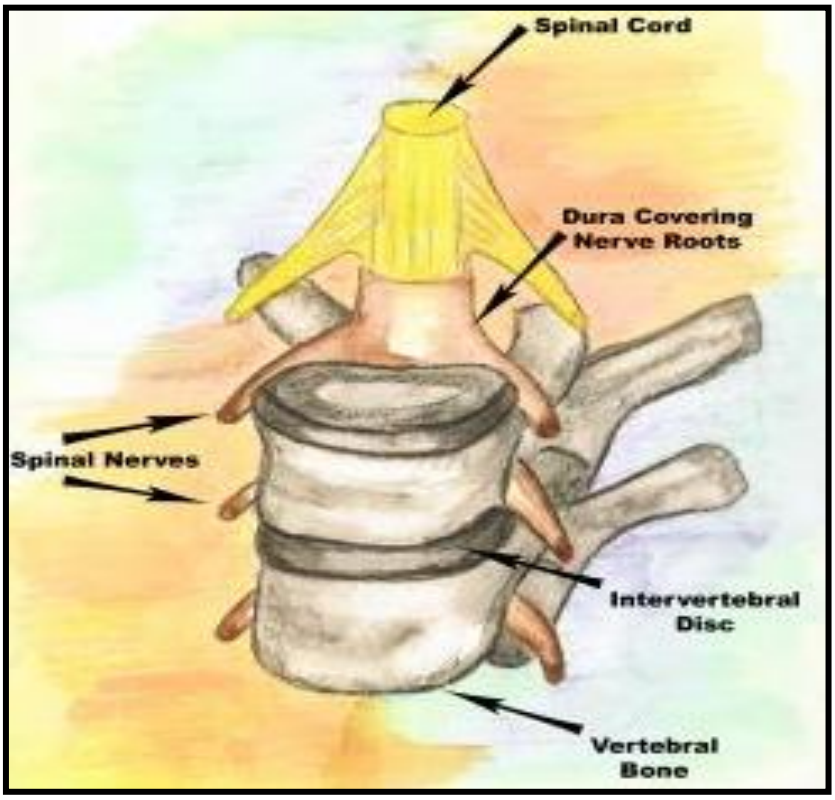


Figure 5 – Spinal Nerve Roots



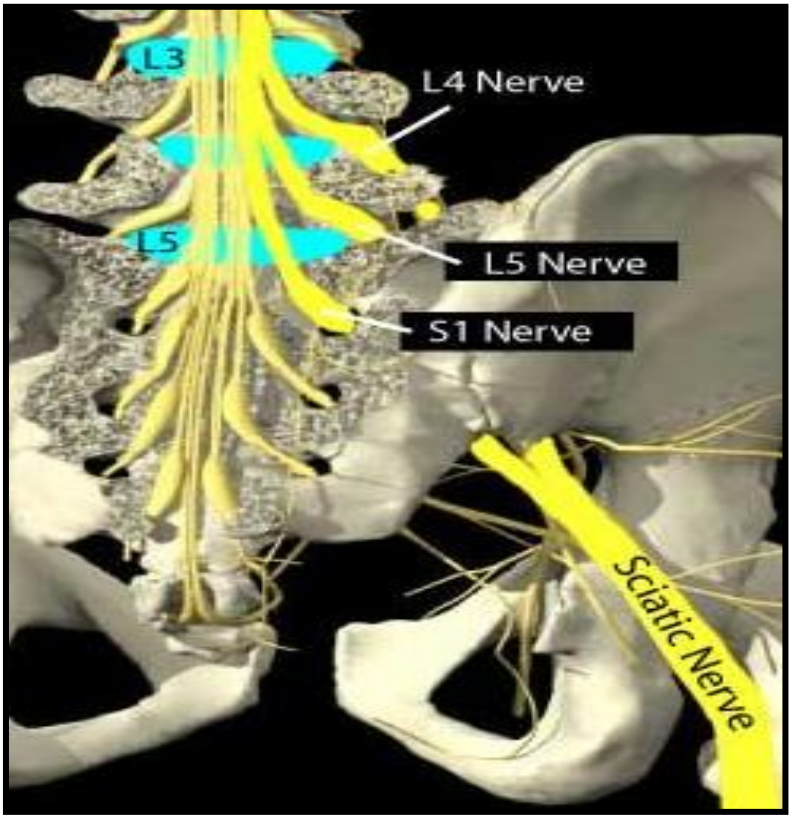
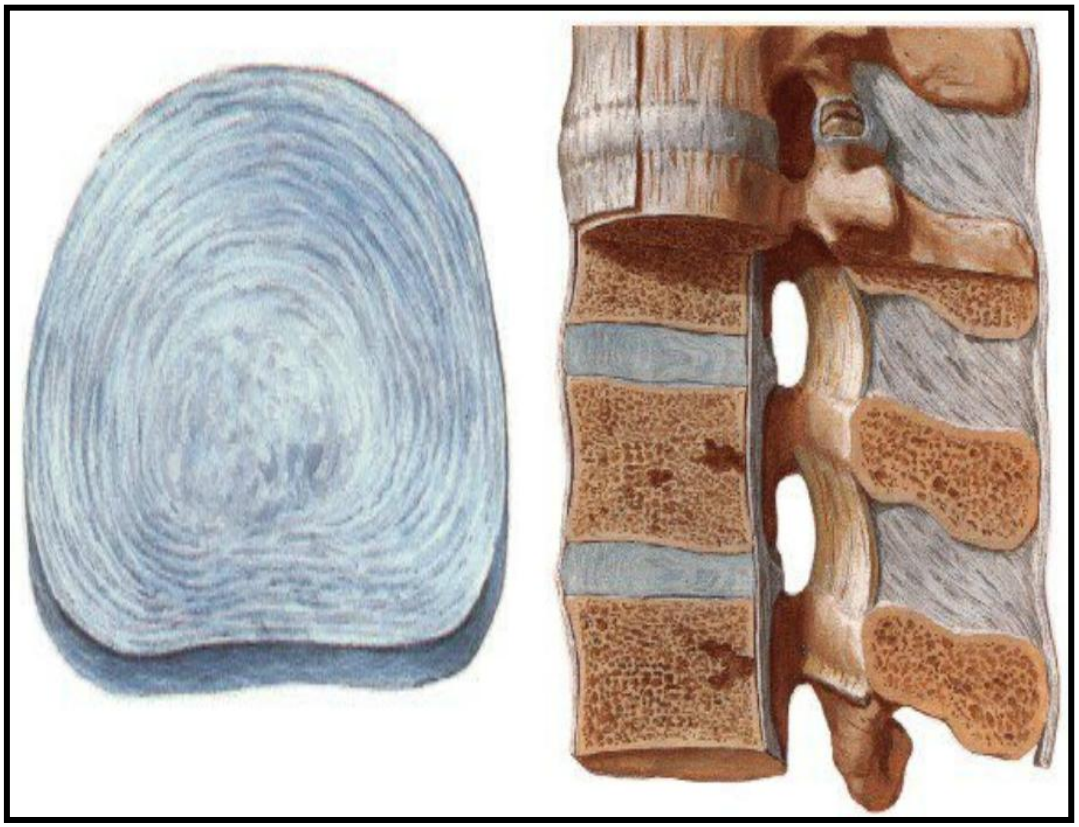


Figure 6 – The Intervertebral Disc



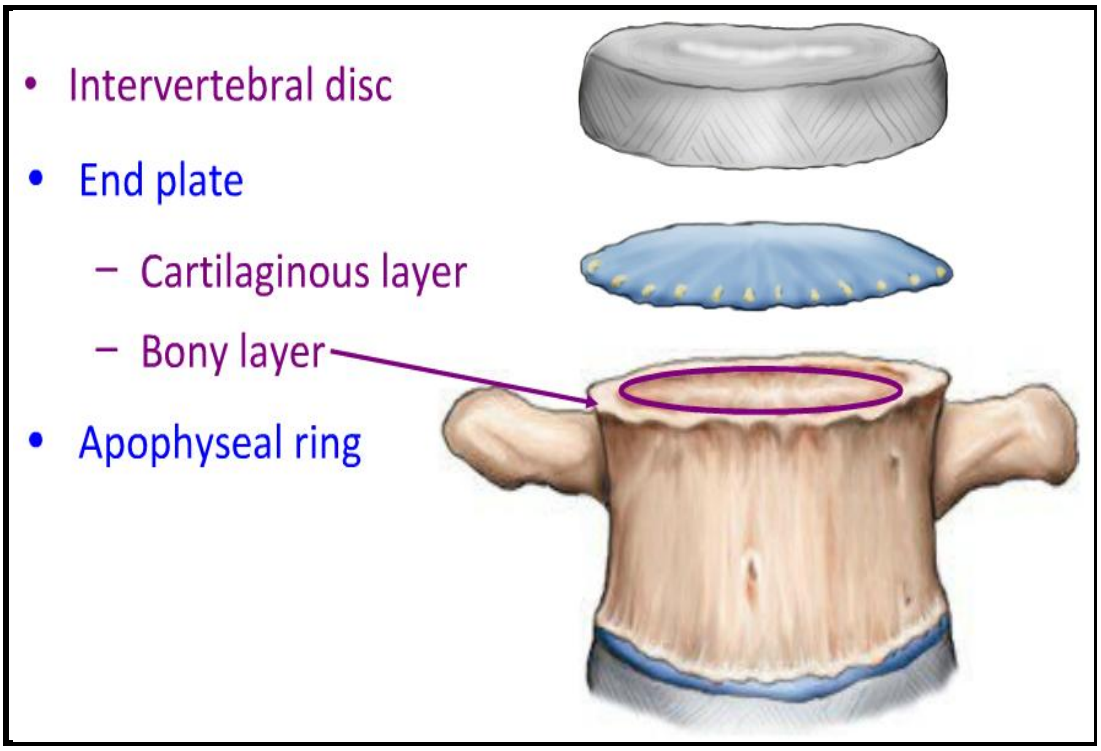


Figure 7 – Annulus Fibrosus

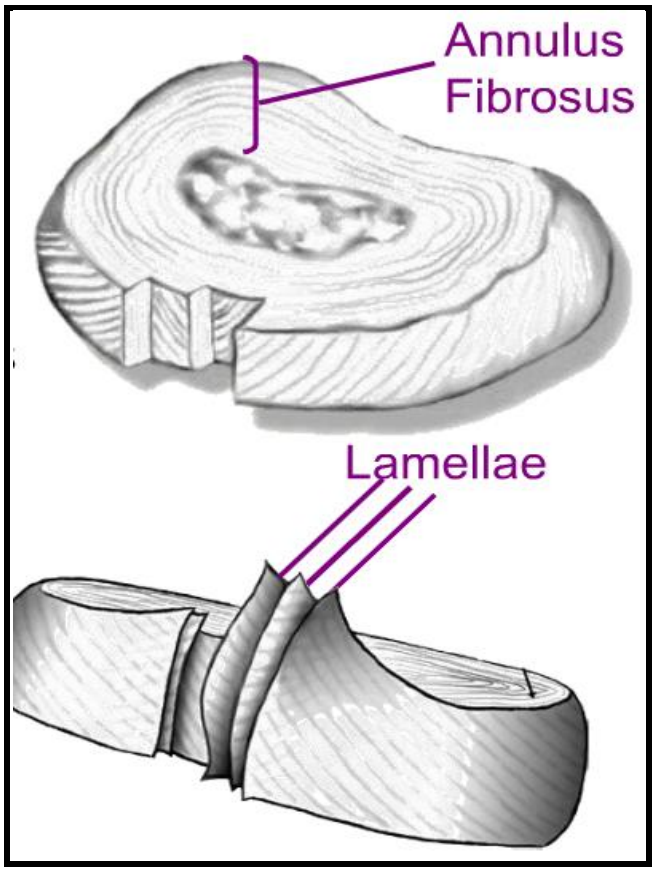
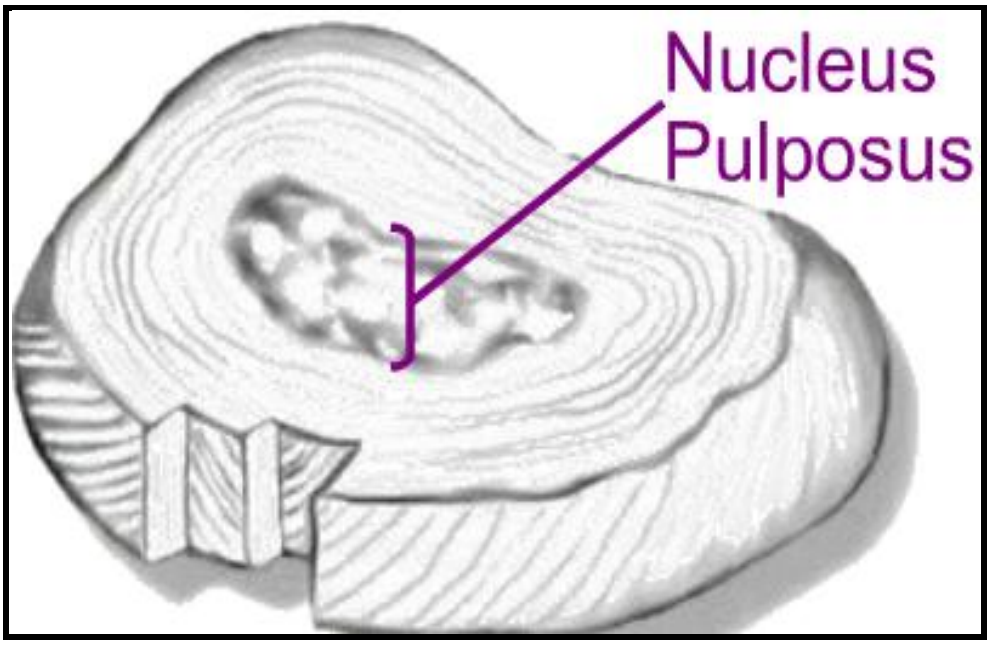


Figure 8 – Nucleus Pulposus



Intervertebral Foramina:

These are formed by two adjacent vertebrae, the dorsal boundary of which is formed by articular processes both superior & inferior, their joint capsules, & ligaments. The ventro–medial boundary of the foramen is bounded by the bodies of adjacent vertebrae & the intervertebral disc.

L5 root is more likely to be compressed because of the anatomical peculiarities of the intervertebral canal, through which the root passes (Dubs). For example:

(a) Lowest intervertebral foramen is much narrowed due to obliquity of the promontory & shallow inferior notch.

(b) The disc lies nearer the inferior notch of L5.

(c) Broad roof formed by pedicles

Due to longer, narrow & oblique canal thus formed, the nerve root remains in close contact with the disc over a greater distance. In lateral prolapse of the disc, there will be little chance for the nerve to escape from pressure. This is in contrast with the other intervertebral canals, which are roomy & less oblique.

The Intervertebral Discs (Figure 6)

The discs contribute to a third or fourth of the length of the articulated vertebral column, & variations in their thickness anteriorly & posteriorly play a great role in maintaining the primary & secondary curves of the column.

Each intervertebral disc consists of **two plates of hyaline cartilage**, united by a ring of fibrous tissue, the **annulus fibrous**, in the middle of which lies the **nucleus pulposus**.

The plates of **hyaline cartilage** are 1 mm thick & are placed horizontally. They are attached to the bodies of adjacent vertebrae. On completion of skeletal growth, they fuse with the surroundings epiphyseal ring. Until the age of about 30 years they contain vascular channels which later disappear & are replaced by scar tissue.

The **annulus fibrosis** (Figure 7) forms a rigid firm bond between the vertebral bodies by being attached to the vertebral margins & to the anterior margins of the cartilage plates. It consists of fibrous tissue arranged in 10-12 concentric laminae. The fibers of the laminae are all placed obliquely at an angle of about 45 degrees to the adjacent vertebral bodies; but the layers contain fibers lying alternately in the 45 degrees slope, at right angles to each other, thus offering it enough strength to withstand strain in any direction (Inman & Saunders). Some fibers are attached to the anterior & posterior longitudinal ligaments & are reinforced by them. Puschel (1930) stated that its water content is 78% at birth, reduces to 70% in the third decade, & remains fairly constant thereafter.

The **nucleus pulposus** (Figure 8) is a greyish white, translucent, & semi-gelatinous. It consists of a polysaccharide/protein gel (which gives it the high capacity for imbibition of water) in which are interspersed collagen fibrils, & cartilage cells. The notochordal cells present at birth disappear by about the 10^{th} year, following which fibrous tissue gradually increases. Its water content is normally about 80% of its total weight, varying from 88% in the newborn to 67% at 77 years. In other words with time & abuse, it tends to lose its fluid consistency & become more fibrous. Due to water imbibition during the night, man is 1-2 cms taller in the morning than at night. The turgescence of the disc being responsible for its intradiscal pressure varies

with posture, time of day & age. As the nucleus pulposus is situated not centrally but a little posteriorly the annulus fibrosis is thinner posteriorly.

The **nourishment** of the disc (Figure 9) is through the lymph which diffuses from the marrow cavity of adjacent vertebrae, through perforations in the cartilage plates. Some investigators period suggests their existence through adult life. The pattern of the vascular supply of the annulus fibrosis has interested some workers, such as Larcher, Prader & Tondury, 1947, who described that layers of the annulus fibrosis are permeated during fetal life by numerous vessels of different sizes. This occurs almost exclusively in the postero-lateral part of the annulus & they branch out into capillary system without penetrating further into the disc. With the growth of the child, these vessels gradually disappear, their place being taken by scar tissue. It may be that the site of these vessels leaves a point of lessened resistance for eventual prolapse of the disc. According to Thurel, fibers in the posterior part of the annulus fibrosis are less strongly developed than in ventro-lateral part, another factor also tending to weaken the annulus in this region.

The **nerve supply** (Figure 10) of the disc is from it posterior aspect, a branch of the nerve sinu vertebralis of von Luschka which arising from the posterior primary division, re-enters the spinal canal & supplies the posterior longitudinal ligaments two segments below its origin.

The intervertebral disc, as a whole acts as an excellent shock absorber, by virtue of its different components, for example, a vertical compression force (as experienced when each foot is put forwarded on one after in walking or running), is transmitted by the fluid nucleus in a centrifugal direction, due to its incompressibility. This stretches the fibers of the annulus fibrosis, only to regain its original position

Figure 9 – Nutrition of the Disc

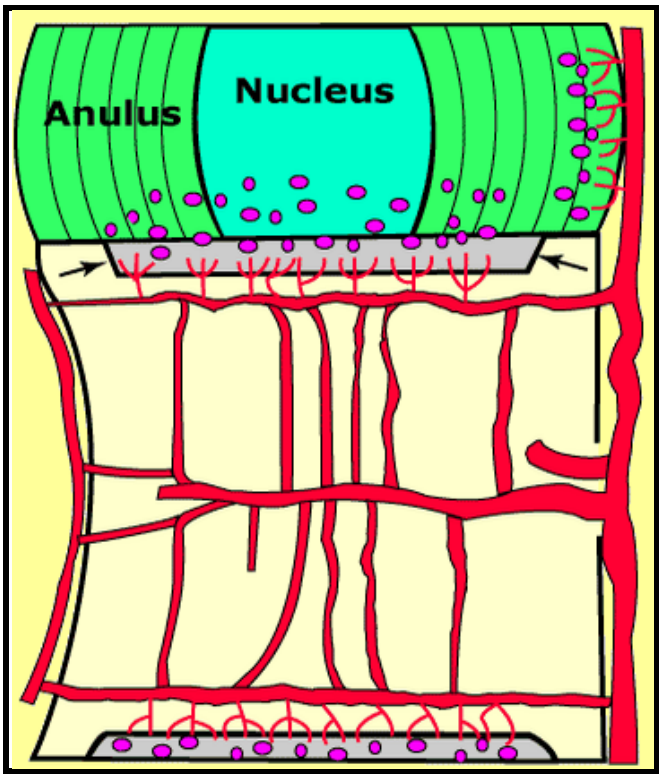
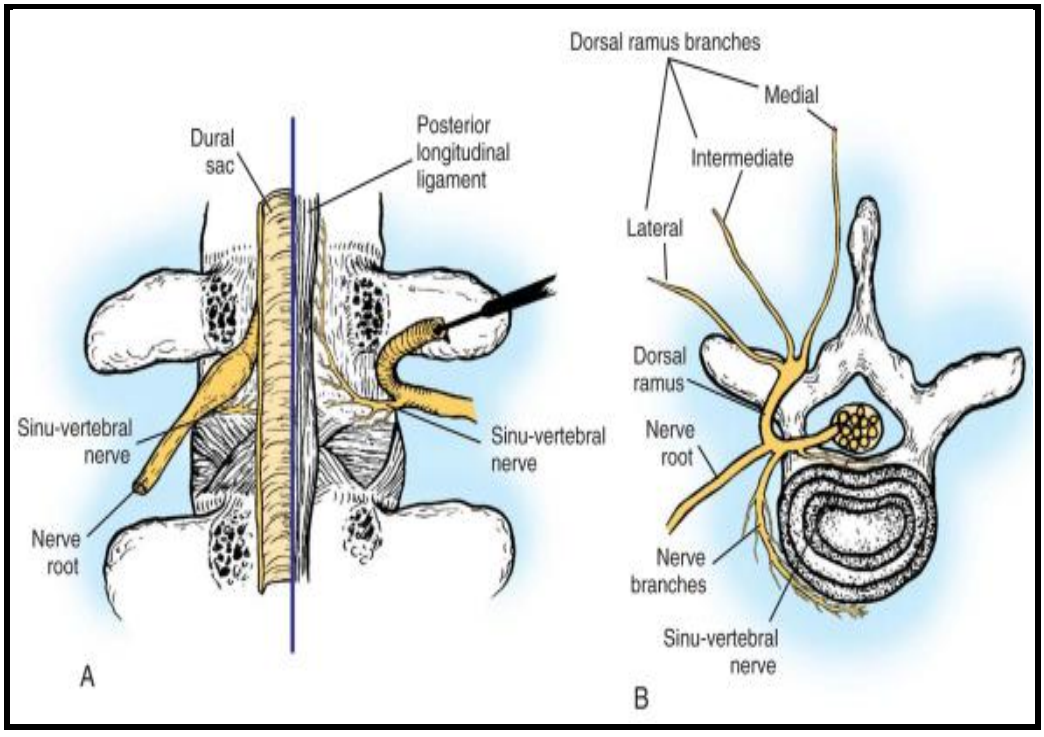


Figure 10 – Nerve Supply of the Disc



when the force is removed. Similarly in flexion or extension, the annulus fibrosis on the concave side is not compressed as it is pushed away by the nucleus pulposus.

Types of Disc Lesions (Figure 11)

There is no clear-cut distinction between degenerative changes due to age & pathological conditions of the intervertebral disc. The various disc lesions can be classified as follows:

1. **Atrophia Simplex of Bartschi**, which is the simplest form of primary degeneration of the disc, which may or may not be characterized by persistent low back pain.
2. **Protrusion Annuli Lesi of Bartschi**, which is characterized by protrusion of the disc through the weakness of the annulus fibrosis & consequently resulting in stretching of the sensitive posterior longitudinal ligament. It is accompanied by severe low back pain & sciatica & other radicular compression symptoms, depending on whether or not the nerve roots are pressed. In this stage, there is also new cartilaginous tissue formation of the margin of the vertebral body, probably as a result of irritation, when it ossifies & forms osteophytes
3. **Prolapse of intervertebral disc**, in this stage, there is complete eruption of the nucleus pulposus through the annulus fibrosis, whereby it comes to lies on the posterior longitudinal ligament. Two types are recognized.

   (a) The mobile type (concealed ruptured disc or intermediate prolapse). In this type, the prolapsed portion returns to the same tear intermittently

   (b) The fixed prolapse, when the prolapsed nucleus can no longer be reduced.

   In long-standing disc protrusion, the following changes can occur:

   (a) A mobile protrusion may become fixed.

   (b) Calcification of the herniated disc.

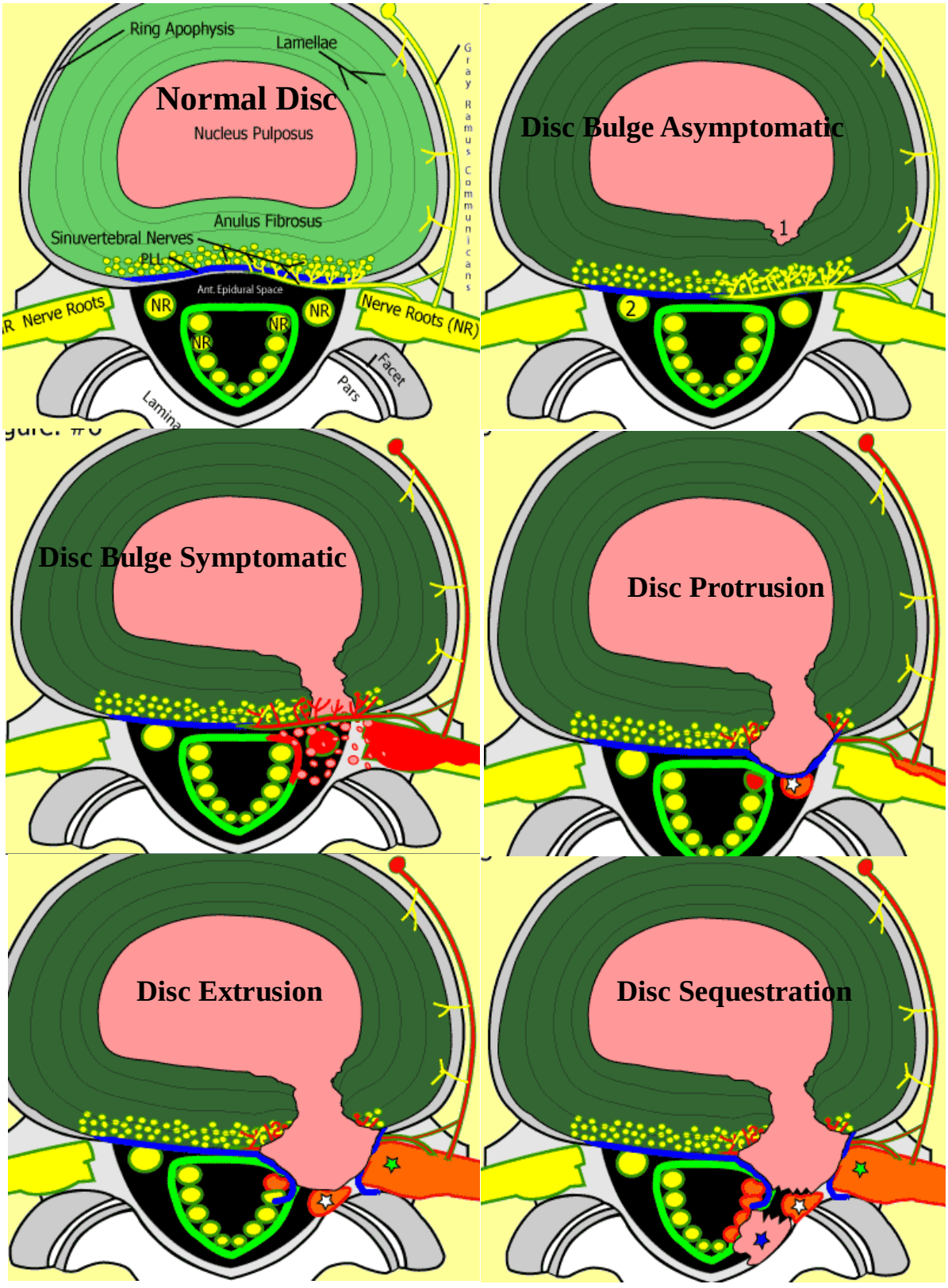
(c) Adherence of the disc to the dura mater & extra-dural roots, & finally adhesions & fibrosis secondary to inflammatory reactions

Disc Protrusion

Cyriax, in an attempt to find a suitable classification that would help in dictating decisively the appropriate treatment in the management of prolapsed disc to be instituted, classified them mainly into two groups:

1. Prolapse of the fibro-cartilage by the classical history of the patient being sized with sudden severe & acute back pain whilst trying to lift a heavy load, which may or may not be associated with the production of an audible "click" & sciatica. The patient is usually unable to straighten himself up & manifests the list. He considers it to be amenable to manipulative reduction.
2. Prolapse of the nucleus pulposus. In this group the onset of symptoms is very insidious. The patient may give a history of minor injury to his back or being involved is some unaccustomed work. The patient may feel a little backache at the time of injury, but may carry on working, the pain gradually getting worse by the end of the day & reaching the peak of severity the next morning, so that the patient is even unable to get up.

Figure 11 – Types of Disc Lesions



Aremstron (1963) classifies disc protrusion into three stages. This is more or less pathological classification from one similar to that of Bartschi.

- **First Stage**
    - In the first stage there is progressive degeneration of the disc, accompanied by disturbances of the water content. The actual sequestration starts in the nucleus pulposus & spreads to the posterior annulus & ends with annulus rupture.
- **Second Stage**
    - In this stage, the nucleus escape is characterized by episodic extrusions of sequestrated nuclear fragments through the annular defect.
- **Final Stage**
    - This is the stage of fibrosis & repair & it may overlap with the second stage & progress for several years, until the abnormal disc is replaced by fibrous tissue.

Presentation of Disc Protrusion

(a) The commonest protrusion is fairly well circumscribed bulging of the disc, yellowish or white glistening covered with attenuated annulus fibrosus or with soft elastic summit.

(b) Less commonly free lying completely extruded disc material in the epidural space, which may or may not be embedded in dense fibrosus tissue.

(c) Intermittent herniation of Falconer, or concealed disc of Dandy.

Nerve Sensitivity of the Structures of the Intervertebral Canal

The pain secondary to disc protrusion may be either due to irritation, be it chemical, mechanical or auto-immune, of the adjacent direct irritation of the inflamed nerve or even a combination of all the factors, or permutation of several. The **pain sensitive tissues** in this area are:

(a) Anterior longitudinal ligaments

(b) Vertebral bodies

(c) Synovium

(d) Capsule of the intervertebral joint

(e) Nerve roots & the muscles

(f) Inter-spinous ligament – this may or may not contain pain fibers.

The **non-sensitive tissues** are:

(a) Ligamentum flavum

(b) Annulus fibrosis & nucleus pulposus of the disc

(c) Inter-spinous ligament – this may or may not contain pain fibers

The Nucleus of the Back

Intimately related to the functional unit of the spine are the spinal muscles, which for the sake of simplicity can be divided into two groups.

Short Stabilizers

The short stabilizers bind together the adjoining & thus enable the long muscles to move a number of segments as one group.

The muscles concerned are:

(a) The spinalis connecting two adjoining spinous processes

(b) Transversalis, joining two adjacent transverse processes

(c) Multifidus, arising from the spinous process & inserting into the transverse process of the adjacent vertebra below.

Long Stabilizers

Sacrospinalis is the primary muscle in this group

The Epidural Space & Sacral Canal (Figure 12)

The epidural space is a potential, elliptical, or annular space between the spinal dura & the bony vertebral canal.

Formation:

In the cranial cavity, the dura is arranged in two layers, the periosteal & investing layers, which are finally adherent to each other expect where they split to enclose the venous sinuses. The outer periosteal layer is the periosteum of the inner surface of the skull which in spine, acts as the periosteum of the spinal canal. The inner investing layer is continued on form the brain in the cranium to the spinal dura blends with the periosteal layer. The space between the two is therefore present layer. The space is therefore present only in the spinal canal.

Boundaries & Extent:

Its upper boundary is the foramen magnum, inferiorly at the end of the sacral canal. It ends where the hiatus is closed by the sacro-coccygeal membrane. Anteriorly, the space is bounded by the posterior longitudinal ligament, laterally by

the pedicles of the vertebral laminae. It communicates laterally with the paravertebral space through the intervertebral foramina. Since a few fibrous bands hold the dura against the posterior longitudinal ligament, there is very little of epidural space there & a tenth of the epidural space is on the lateral & posterior aspects of the dura. The actual dimensions of the space vary at different levels being inversely proportionate to the dilatations of the cord. The cervical portion of the space is widest at C 3; varying in width from 1.0 to 1.5 mm the thoracic portion is widest at T-6 (4 to 5 mm). The lumbar portion is widest at L-2 varying in width from 5.0 to 6.0 mm sometimes even 8.0 mm due to the triangular shape of the body vertebral canal.

Contents:

Contents of the epidural space include the dural sac & spinal nerve roots, & the extradural plexus of veins, spinal arteries, lymphatic's & fatty tissue. The extradural plexus of veins, also called the venous plexus of Batson, are places of the space. They are relatively mainly in the anterior & lateral parts of the space. They are relatively large, thin walled, & contain no valves. They are arranged in 4 vertical channels, two on either side of the posterior longitudinal ligament & two in front of the vertebral arches, & are interconnected by venous rings at each segment level. At each segment, they receive the basivertebral veins from the spinal cord. Serially they also receive communicating branches from the vertebral, cervical, deep cervical, intercostals, lumbar iliolumbar & lateral sacral veins through the intervertebral & sacral foramina. They form connecting limbs with the cerebral veins above & the pelvic below.

Importance:

Due to the large number of communications that this valve-less venous plexus receives any factor which raises the intra-abdominal or intra-thoracic pressure will cause a shunt of blood from the thoracic & abdominal veins into this plexus, thus decreasing the effective volume of the extradural space & also causing an increase in the C.S.F pressure. The vast communication network also favors spread of any infection of malignant cells from one portion to another.

The spinal arteries supply the various structures which form the vertebral canal as well as its contents. These arteries correspond to the veins enumerated above; enter at each intervertebral foramina, & lie chiefly along the lateral part of the epidural space, where they supply the adjacent vertebrae, ligaments, & spinal cord.

Theories of Negative Pressure in the Epidural Space:

Though the epidural space was first used in 1885 by Corning to inject cocaine & produce analgesia in dogs, the sacral approach used in 1901 by Sicard & by Cathelin, & the interspinous approach by Sicard & Forrester in 1906, it was Janzen who first described the negative pressure in the epidural space in 1926. Since then, various theories have been advanced to explain this negative pressure.

In conclusion of various theories put forward it can be said that the size of the size of the space varies with:

a) The height of the individual & therefore the length of the spinal canal

b) The amount of loose areolar tissue & fat in it

c) The degree of flexion of the spine

d) The position of the patient, being more in the lumbar than in the thoracic region in the vertical position

e) The length of time that the person is maintained in the lying down position, as the influx of venous blood reduces the space if there is undue delay in entering

Sacral Canal

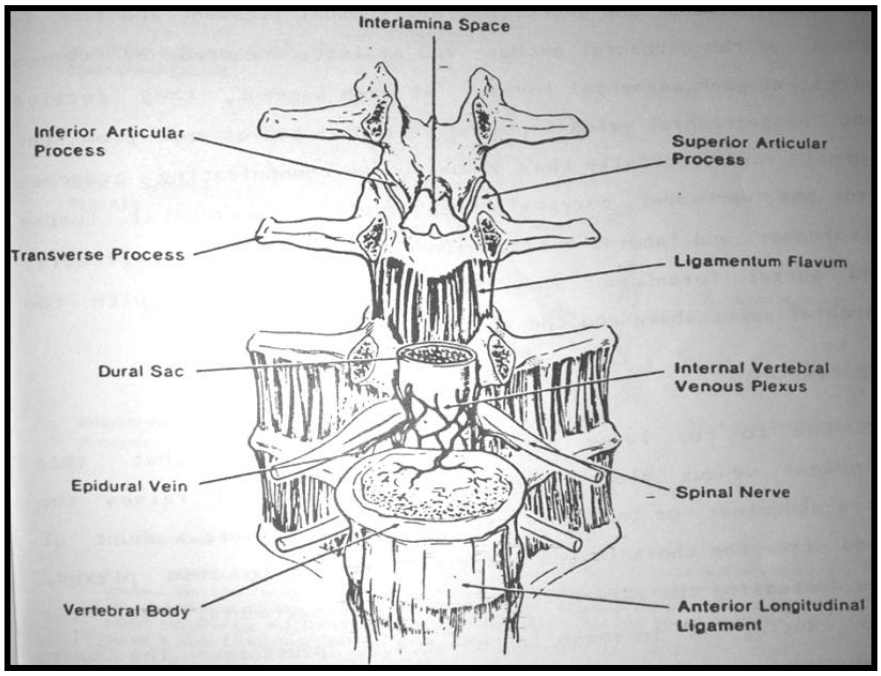
The sacral canal (mainly based on a description by Trotter) is the continuation of the vertebral canal through the sacrum & being in the sacrum it is quite naturally curved, like the bone. It is triangular in cross section at the base, & flattened towards the apex. The anterior wall is formed by the posterior surface of bodies of the sacral vertebrae & posterior longitudinal ligaments. The lateral & posterior wall is formed by pedicles & laminae of sacral vertebrae, which are fused at the base where the elastic ligamentum flavum extends between the 5^{th} lumbar & the 1^{st} sacral vertebra. At the caudal extremity, the lamina of the 5^{th}, & often of the 4^{th} sacral vertebra, fail to meet in the midline, leaving the sacral hiatus at the termination. The hiatus is covered by:

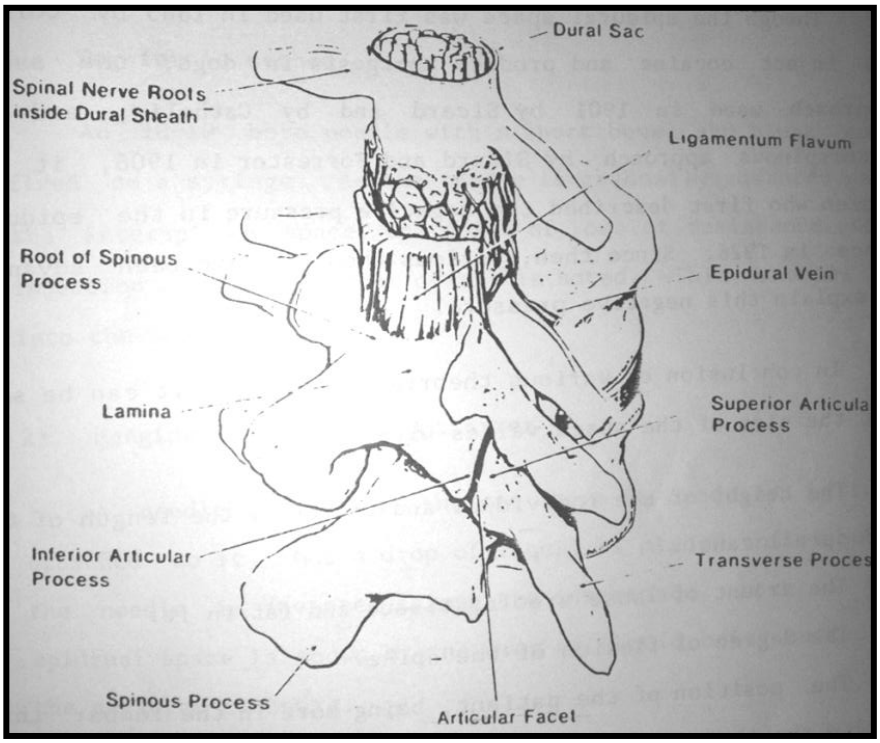
(a) Prolongations of the supra spinous ligament

(b) Superficial posterior coccygeal ligament, which serves as a roof

(c) Over the end of the sacral canal.

Figure 12 – Epidural Space





The content of the canal are:

(a) Dural sac, which terminates at S2 or S3 & continues as filum terminale, to be attached to the coccyx.

(b) The spinal nerve & dural sheets.

(c) The epidural space with its contents.

The level of the termination of the dural sac is an important factor which may have some bearing in the accidental puncture of the dura mater in the caudal approach of epidural injection. According to Trotter, the level of termination, of the distal end of the sac to the middle third of S2 was 46 percent.

The other factor which may determine the accidental puncture of the dura mater is the distance from the apex of the hiatus to the dural sac, which of course depends on the type of the dorsal wall. In the study of Trotter, this distance was about 47 mm (42 %).

Abnormalities of the Sacral Hiatus [34] (Figure 13)

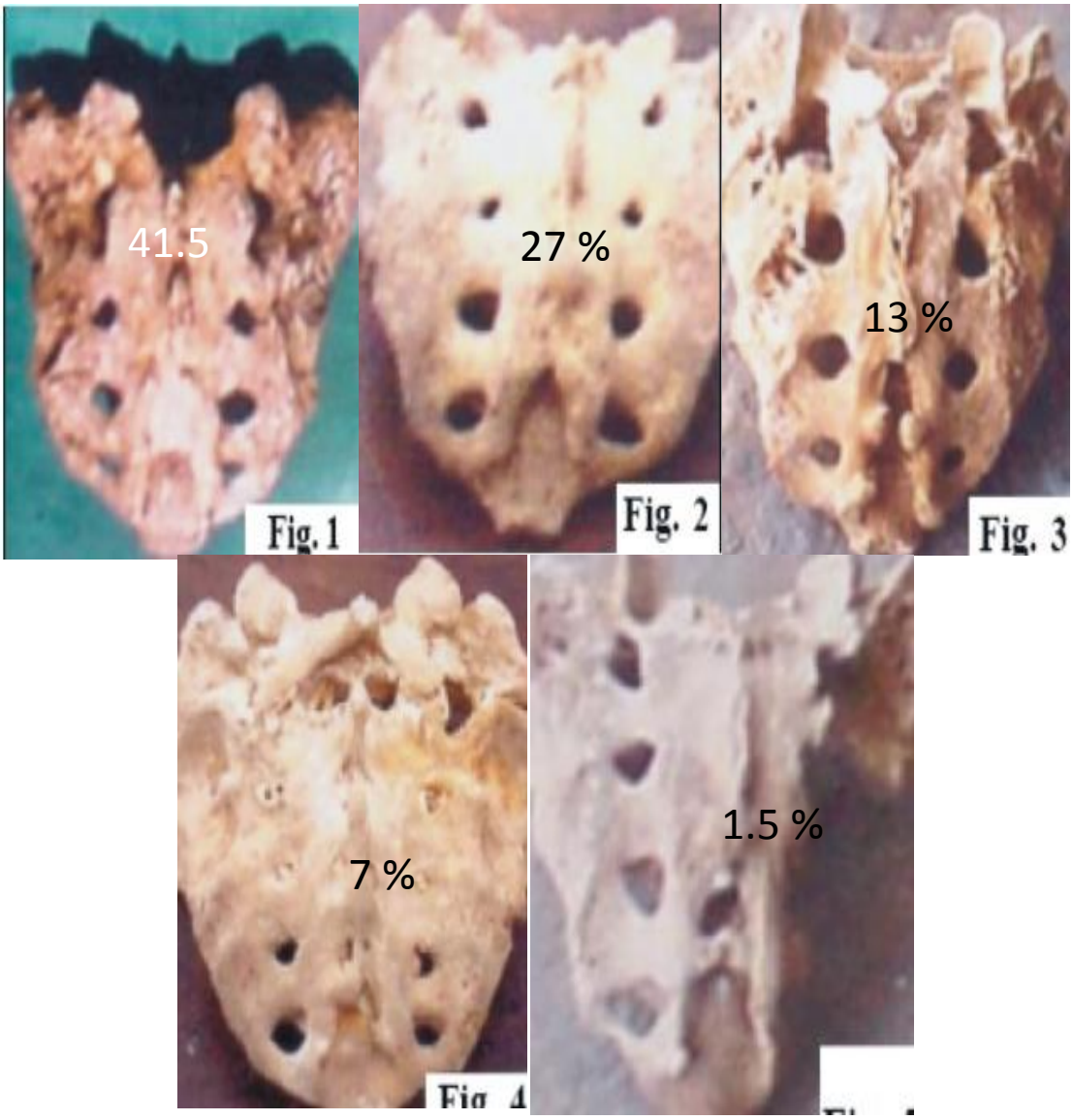
Abnormalities of the sacral hiatus can cause difficulty in the insertion of the needle into the epidural space. Various abnormalities may be:

(a) Obliteration of the hiatus, in which there are two lateral approaches instead of a single central opening.

(b) Unilateral aperture.

(c) Overgrowth of the bony landmarks.

Figure 13 – Abnormalities of Sacral Hiatus



Methods of Identifying the Epidural Space

Though there are a large number of methods, they are but variations in the methods enumerated below.

1. Dogliott's Method:

An 18 - 19 bore needle with a short bevel & blunt end is fixed on a syringe. As the needle is gradually advanced across the interspinous space, the point of **loss of resistance** to the injection of a liquid or of air is noted. This denotes entry into the space.

2. Hanging drop sign of Gutierrez

A needle similar to the one above is used, no syringe is attached to it, but a drop of liquid is placed in its hub. As the needle is advanced across the interspace, entry in to the epidural space is shown by the **drop of liquid being sucked in by the negative pressure**.

3. Odom's method

To the hub of the needle is attached a three way cannula. The second limb of the cannula is attached to the pre-flattened rubber balloon. Initially, the knob is kept in such a way that the two do not communicate with each other. As the tip of the needle traverses the interspace the knob is turned so that the balloon communicates with the needle. When the epidural space is encountered the **balloon collapses due to the negative pressure**.

4. Spring Loaded Syringe

This special syringe makes use of a spring on its position I such a way that it contracts as soon as the negative pressure of the epidural space is reached by the needle tip.

5. Whoosh Test

Once the needle has been placed in the epidural space, air is injected into the space & a **sound (gush of air)** can be palpated with a stethoscope just above point of insertion of the needle. A 1-year prospective study, using fluoroscopic imaging to identify needle position, was conducted to compare the sensitivity & specificity of the whoosh test with that of clinical impression alone in assessing correct needle placement in the caudal space. Of 131 patients studied, correct needle placement was achieved in 121 on the first attempt (92%). Clinical impression alone had a sensitivity of 94% & a specificity of 20%. The whoosh test had a sensitivity of 80% & a specificity of 60%. The whoosh test is superior to clinical judgment in detecting incorrect caudal needle placement [35].

Fluoroscopic Contrast Dispersion Method

Isohexol is used as a die to document placement of the needle in the epidural space & also to study the dispersion of the steroid & the extent of its reach. The advantage is highly accurate needle placement & the study of dispersion pattern also helps to decide the extent. Disadvantages include the need of a fluoroscopic table & die related complications [36, 37].

Considering the various factors which contribute towards the negative pressure of the epidural space, its absence of in a small percentage of persons, its

variations from person to person & also from region to region in the same person, Hewer & Lee (1958) conclude that "Techniques which rely upon negative pressure is identifying space tend to be unreliable, whereas those which depend upon positive pressure from without or loss of resistance are not only preferable but also applicable at any level of the vertebral canal". When the space is identified without fluoroscopic guidance, which is the gold standard for accurate needle placement other methods as well palpating the right landmarks (For Caudal Injections – Sacral Landmarks) can be used for accurate needle placement in 92% of cases [30]. Stitz & Sommer support a high rate of success, as long as readily palpable anatomic landmarks are properly recognized [30].

Applied Anatomy

It is pertinent to consider here a few of the important facts which influence the high incidence of disc lesions as well as the involvement of nerve roots in the lumbosacral region.

1) The intervertebral disc is smallest in the cervical region, bigger in the thoracic, & biggest in the lumbar, the cervical being half of the thoracic & ¼ of the lumbar approximately. Hence, their protrusions are commonest in the lumbar, & least in the level.

2) The lumbar & the sacral regions are in a straight line in the early fetus. The lumbosacral angle begins to form in the mid-fetal life & continues growing rapidly increases when the child begins to sit up, & reaches about 120 degrees in the average adult, with wide variations from the average. The L5 – S1 disc is consequently wedge shaped, the base of the wedge being about 2 ½ times the posterior.

3) The lumbosacral articulation is at the junction of a mobile segment (Lumbar) with a fixed one (the sacrum). In conjunction with the angle at this site present is responsible for the heavy shearing forces present here normally.

4) The intervertebral foramina decreases in size from above downwards, L1-L2 being the biggest & L5-S1 the smallest.

5) The Lumbar nerves increase in size from above downwards from L1 to L5.

6) The sciatic nerve roots are L4, S1, S2 & S3 of which the major contributions are from L4 & L5, since the major portion of the lower limb bud develops from these somatic segments.

7) The nerve roots of the lumbar & sacral regions have a comparatively longer intra-spinal course.

8) Those of L1 & L2 arise at their respective vertebrate bodies, but those from L3 onwards at a higher level viz., at the L3 disc level, the corresponding root exist but L4 root has to cross this disc to reach its foramen, similarly at subsequent segmental levels. Hence the neurological disturbances may occur not only over the root distribution at the level of disc protrusion, but also over the next lower one, from irritation of deeper structure e.g., L5 in L4 – 5 disc lesions.

9) The fourth lumbar nerve passes in front of the 5^{th} lumbar transverse process to join the 5^{th} lumbar nerve & form the lumbosacral trunk. It can therefore be irritated by a large L5 transverse process.

10) The lumbosacral trunk lies in front of the sacroiliac joint & hence its disease may cause sciatic symptoms (though this theory is disputed).

11) As the L4 & L5 nerves exit through their respective foramina, they are surrounded by a venous plexus which may irritate if they are engorged or over developed.

12) Again according to Dubs, the L5 root likely to be compresses due to the following anatomical peculiarities of its intervertebral foramina.

    a) The disc is nearer the inferior of L5.

    b) The thick pedicles form a broad roof.

    c) The obliquity of the sacrum & shallow inferior notch of L5.

    d) The root remains in close contact with the disc over a greater distance as the foramen is rendered narrower, longer & more oblique.

13) Fibrous bands hold the dura against the posterior longitudinal ligament so that the nerve roots are comparatively tethered.

14) Congenital anomalies are commonest in the lumbar region, of all the regions of the, viz., sacralization of lumbar vertebra, Pre post fixation of the lumbosacral plexus, spina bifida, spondylolysthesis, & so on.

15) The pain-sensitive tissues in this region are: anterior longitudinal ligament, vertebral bodies, synovium & joint capsule of the apophyseal joints, nerve, & muscles. The interspinous ligament may or may not contain nerve fibres. The non-sensitive tissues are: The ligamentum flavum, annulus fibrosus & nucleus pulposus.

Etiopathogenesis

Sciatica is a symptom. It is defined as radicular pain along the sciatic nerve. According to the causative factor it is classified as:

1) Reflex Sciatica:

This type of neuralgia is due to visceral causes, e.g. diseases of the bladder, prostrate, ovaries, or of structures like joints, ligaments & muscles. The main features of this type of neuralgia are that neurological disturbances do not correspond to known patterns of nerve distribution & there are no objective neurological signs.

Primary Sciatic Neuritis:

In this form, it is a manifestation of either a general systemic disease like diabetes or syphilis, or of a generalized toxemia like alcoholism, lead & arsenic poisoning etc. It can be diagnosed by other signs & symptoms of the underlying disease or toxemia.

2) Secondary Sciatica:

This is due to peripheral neuritis & is due to pressure on the nerve, which may be.

a) Outside the spinal canal such as by pelvic tumors

b) Non-disc lesions inside the spinal canal such as extra/intra-dural masses

c) Intervertebral disc lesions

Depending upon the extent of nerve involvement there may be parasthesia / anesthesia, muscle weakness, atrophy, & depressed or diminished reflexes. Their diagnostic feature is the segmental distribution of neurological signs.

In the words of Duplessis, "It is becoming increasingly evident that nuclear retropulsion is the villain of the pace of sciatica & it is with this form called the "Lumbo - Sciatic syndrome" that we are concerned here [40].

The etiological factors as well as pathophysiology can be conveniently considered under the following headings though the division is but arbitrary, & each cannot be separated from the other:

a) Mechanical causes
b) Changes in the annulus fibrosis
c) Changes in the nucleus pulposus
d) Changes in the adjacent structure

Mechanical Causes:

Cyriax blames nature for failure to redesign the spinal column to suit the erect posture [12]. This view is also held by Williams, who tracing the evolution of a biped man from his quadruped ancestors, compares anatomical details like changes in the spinal column, lumbosacral angle, shift of muscle attachments & consequently a change in their function, & so on. According to him, lumbar disc prolapse is another penalty that man pays, like hemorrhoids & varicose veins, for his erect posture. These views again are disputed by Finneson et al [41], who also feel that unaccustomed exercise is the culprit, as when a man of sedentary habits & a pampered spine, indulges in a vigorous round of sports during the weekend & insults his spine. This theory, however, seems to be inapplicable in so far as a large number of patients who fall prey to the lumbosciatic syndromes are those with well-developed musculature e.g.: Farmers, Laborers. It would be more appropriate to blame "A life time of constant abuse, stress & strain" which gradually & progressively lay, the foundation for the production of the syndrome.

During 1969, Kester [42] has broadly divided the mechanical causes into "Static" & "Kinetic"

a) Static:

In the majority of cases, there is an increased lordosis, either postural or secondary leading to:

- An increase in the lumbosacral angle with an increase accordingly in the shearing forces & an overstretching of the anterior longitudinal ligament.
- An increase in the weight borne by the posterior apophyseal joints, much more than they are normally meant for, with consequent changes.
- Hyperextension with compression of the joint facets, narrowing of intervertebral foramen, & therefore increased changes of root pressure by the protruding disc, & with "Kissing spines".

b) Kinetic:

Due to initiation of pain-sensitive tissues during movement, Callietes (1957) further differentiates between these causes:

- Abnormal stress on a normal back, e.g.: Lifting of a heavy weight to which the person is not accustomed
- Normal stress on an abnormal back e.g.: a defective vertebral column, weak muscles due to paralysis etc.
- Normal stress on a normal unprepared back, e.g.: lifting a full suitcase expecting it to be empty.

Changes of degeneration are more common in the lower lumbar discs & at a much earlier date compared to the higher ones. Love & Walsh noted in a series of 500 cases that the proportion of disc prolapse at L 1 - L 2 - L 3 - L4 - L5 is 1:1.5:

13:87: 79 [41]. Observations of O'Connell (1951 - 500 Cases) [44] & Armstrong (1958 - 10,000 Cases) also bear out a higher incidence in the lower discs with the L 5 – S 1 accounting for almost 50 % of the cases [45].

According to Roaf (1960), disc degeneration is more common in the lumbosacral region due to the facts enumerated in the earlier section, due to greater range of mobility & a higher frequency of congenital anomalies [46].

Changes in the Annulus Fibrosus:

It has been stated earlier that the annulus is narrower posteriorly. Malmos points out that the posterior longitudinal ligament also is not as well developed in the lower lumbar region as higher up, at the same time being narrower. In 1951, Thurel stated that the fibers of the annulus are well developed anteriorly & laterally.

Larcher, Prader & Thubury (1947) described vessels permeating the posterolateral aspects of the annulus, & then branching out without penetrating the disc. According to them, these vessels disappear with growth, usually in the second decade, & are replaced by scar tissue. Weak points are thus formed, facilitating of the nucleus at a later date.

When, due to bad posture, abnormal stresses or other mechanical causes, the line of weight bearing lies in the posterior part of the disc & hence over the annulus instead of over the nucleus as in the normal, the annulus is weakened further. The nucleus being one-fourth of the total size of the disc in the lower lumbar region makes matters worse. It may be repeated minor stresses result in small tears which form weak spots (Lindblom, 1952, 1957) through which herniation of the disc is later facilitated. A single trauma, however violent, is unlikely to cause annular rupture, as evidence by the fact that in trauma sufficient to produce compression fractures &

fracture dislocation of the vertebrae, the discs are very often intact. Rather, such trauma may be the precipitating factor for protrusion of the nucleus through an annulus weekend as described above [47].

Cause in the Nucleus Pulposus:

Paulson, Sylven, Snellman & Hirsch et al (1952) from their clinical, anatomical, microscopic studies stated that more than mere mechanical stress is responsible for the nuclear degeneration [48].

The reduction in water content has already been noted. There is a decrease in the mucopolysacchrides & an increase in the proteins, which leads to a decrease in the water-binding capacity of the disc. As the protein content increases, there is a loss of delineation between the annulus & the nucleus. In such degenerated discs, pain perception increases [47].

Hirsch demonstrated that it is difficult to inject even 0.5 ml of normal saline into a normal disc & that the process is painless in a normal disc. Where degeneration commenced, more than 1.0 ml can be injected, but the process is painful, presumably due to inflammation of the disc & to stretching of pain-sensitive tissues. He along with Nachemson (1979) in experimental studies using pressure gauges, displacement pickups, high speed cameras, etc., have shown that very little bulging occurs in normal discs, whereas bulging is much more in the degenerated discs, when pressure is exerted [49].

Disc degeneration starts from the center of the nucleus & proceeds posteriorly or postero-laterally, undermining its strain-absorbing capacity. Part of the disc thus becomes necrotic. The entire nucleus does not totally change, even in advanced cases; certain areas are still recognizable as having normal structure.

However, whether such changes in the nucleus are physiological or pathological or at what age they can be called physiological & at what age pathological, is still debated. The earliest recorded changes have been noted at 8-10 years, & definitely at 20 years. Such changes are present in almost all individuals, particularly in the L5-S1 disc. Hirsch therefore suggests that the term "Nuclear degeneration" should be applied only when there is evidence of rupture of the annulus fibrosis.

Now, considering the changes in the annulus & in the nucleus together, the nucleus gradually escapes, from the confines of the annulus & herniation occurs.

- Anteriorly, No structures near enough to be affected hence no symptoms
- Posteriorly, which is the most important is considered in detail
- A third type of herniation is vertically into the adjacent vertebral bodies, which again produces no symptoms. It is commonly seen in adolescent Kyphosis & the radiological appearance is known as "Schmorl's Nodes"

Further, the posterior herniation can occur in the midline (which is least common due to the presence of the posterior longitudinal ligament), paramedially or laterally. The more lateral the protrusion, the greater the occurrence of pain radiating down the lower limb. In a series of cases where clinical & radiological findings were correlated with the operative findings, it has shown that:

- Central protrusions mostly produce back ache & rarely radiating pain
- Lateral protrusions produce radiating pain more frequently than back ache
- The intermediate protrusions produce back ache & radiating pain

Changes in the annulus fibrosis & the nucleus pulposus do not always go hand in hand. Annular rupture can occur when clear changes are minimal; in such cases, under favorable circumstances, the still semi-fluid nucleus which has protruded can partly return to the confines of the annulus; this is called a "Mobile" or "Intermittent" protrusion. After repeated protrusions, the nucleus slowly becomes more & more sequestrated, until it returns no more; it is then called a "Fixed protrusion". Such fixity naturally occurs earlier when the nucleus is already sequestrated at its first herniation.

Changes in the Adjacent Structures:

As the line of weight bearing shifts posteriorly, there is more stress placed on the apophyseal joints, leading to an incongruity of their articular surfaces & early onset of degenerative changes. This produces pain, both, in the low-back & of the referred type. Osteophytes at the joint may reduce the size of the intervertebral foramen.

The intervertebral foramen may also be reduced in size by a laterally placed nuclear protrusion as well as osteophytosis at the vertebral body margins. In either case, there is obstruction to the flow of venous blood & pressure on the nerve roots, with edema &/or hemorrhage. As the exudates get organized there is fibrosis around the nerve root & they gradually get fixed.

Diminution in the interbody space results in stretching of the posterior (inter & supra spinous) ligaments which is another source of pain. In course of time, they may undergo hypertrophy.

All of these changes, either singly or in combination, can cause irritation of the pain sensitive structures, the stimuli operating through chemical, mechanical or

autoimmune mechanisms, producing the characteristic clinical signs & symptoms of the lumbosciatic syndrome.

Progression to Multilevel Pathology

Generalized Spondylosis may arise in two ways: Simultaneous pathological changes at several levels, commonly considered to be the end-result of aging, & progression of changes from one level to those immediately above & below. Altered mechanism at one level due to instability or decreased mobility may throw abnormal forces on adjacent joints.

Degenerative spinal stenosis occurs in some cases of spondylosis, with the difference that those parts of the stenotic spine are contiguous to cauda equina due to osteophytic new bone formation.

Degenerative Spondylosis:

As in other degenerative joint lesions, the main changes are degeneration & dis-integration of the articular cartilage of the posterior joints & of the constituents of the discs, along with osteophyte formation.

The Posterior Joints:

The articular cartilage undergoes progressive degeneration. Small fragments of separated cartilage may form loose bodies in the joints. Osteophytes & new bone on the surface of the joints result in large irregular processes bulge posteriorly into the spinal canal.

Intervertebral Discs:

Undergo degeneration with reduction in height & loss of disc space, sometimes there is fragmentation of the cartilage plates.

Scoliosis:

Degeneration of the discs & deformity of the vertebral bodies not infrequently results in scoliosis with rotational deformity of the spine.

Subperiosteal new bone formation:

Results in the formation of osteophytes at the edges of the bodies which may in the end, produce fusion of one or more vertebrae by bone.

Severe low back pain may occur in several ways. The posterior primary rami may be compressed, irritated or stretched by irregularities of the transverse processes & posterior articular processes. There may be compression or irritation of the branches of the sino vertebral nerves in the foramina or lateral recess; they may be affected by osteophytes at the posterior margins of the vertebral bodies adjacent to the discs.

Leg pain may be referred from the posterior primary ramus or sino vertebral nerve, or due at any site in the nerve canal.

The Effect of Degenerative Changes in Soft Tissues & Bone on the Vascular Supply of the Lumbar Spine:

In degenerative spondylosis & particularly in spinal stenosis several factors cause occlusion or partial occlusion by pressure on the intermediate spinal arteries, which supply the contents of the spinal canal, on the radicular arteries, supplying the nerves in the canal & the internal venous plexus of Batson. These are osteophyte formation, narrowing of the discs, thickening of the laminae, internal protrusion of enlarged articular processes & narrowing of the lateral recesses & foramina. Radicular veins have valves but those of Batson's plexus are without valves. The normal

pressure in the arterioles supplying the cauda equina & spinal nerves is only slightly higher than the colloid osmotic pressure. The funiculi of the spinal nerves carry an abundant blood supply from radicular arterioles & capillaries. The blood vessels supplying the nerves are extremely sensitive to compressive forces. The ischemic changes in nerves in spinal stenosis may be such that no recovery of neural function is possible even after removal of the cause of compression.

Innervation of the Lumbar Spine:

The innervation is by the sino vertebral nerve & the posterior primary ramus. The sino vertebral nerve was first described by Von Luschka. It arises from the anterior aspect of the spinal nerve a few mm distal to the dorsal root ganglion. It is joined almost immediately by a branch from the sympathetic ramus communicans. The composite nerve about 1 mm thick passes back through the intervertebral foramen into spinal canal to divide into ascending, descending & transverse branches which anastomose with the nerves of the contralateral side & with those from adjacent levels.

The post-primary ramus arises from each spinal nerve outside the foramen & divides into medial & lateral branches. Medial branch gives a twig to the interior capsule of the posterior joint, & to the dorsal spinal muscles & anastomoses with the nerves from adjacent levels.

The lateral branches of the posterior primary rami of the upper three lumbar levels supply the skin as far distally as the greater trochanter, but there is no cutaneous supply from the lower two posterior rami.

The spinous processes & laminae are supplied by branches from the posterior primary rami. The posterior joints are supplied by the medial branches of the posterior

primary rami. There is an overlap of innervation. At each level the ramus supplies branches to that level & also the joint at the level below.

Clinical Application:

The poor localization & radiation of low back pain may well be related to the fact that each sino vertebral nerve & each posterior primary ramus supplies at least two levels.

Entrapment of spinal nerves is an obvious cause of pain, sensory disturbances & muscle weakness, but the distribution of the branches of the sino vertebral nerve & posterior primary ramus gives rise to a more complex problem. Irritation of these branches may be relevant in the pain of the disc degeneration & herniation especially in spinal stenosis. On the other hand irritation of the posterior primary rami is a feature of segmental instability.

In performing an inter-transverse spinal fusion, the posterior primary rami are nearly always sectioned. This may be one of the beneficial effects of this operation.

Irritation of sino vertebral nerve may cause back pain & sciatica. This has special relevance to the variety of conditions that produce lateral recess narrowing at L 4-5 & L 5 -S l levels.

Production of Pain

There are various theories as to how pain is produced by the prolapsed intervertebral disc. These fall into the following groups:

Derangement of Mechanics of the Spine:

The pain may be secondary to loss of integrity of the disc. This is substantiated by experiments where pain has been produced in normal, healthy volunteers by stimulating these structures chemically & also mechanically.

- Apophyseal joints via the recurrent sino vertebral nerve of Von Luschka
- The stretched (or compressed) ligaments mainly the supra & inter spinous

Inflammatory Changes around Nerve Roots:

As observed during the operation & by histological studies, where operation was done in patients with recent symptoms, acute inflammatory reactions were noted. In chronic cases, evidence of subsided inflammatory reactions like adhesions & scar-tissue formation was observed.

Microscopically, the features noted were perivascular infiltration of lymphocytes & fatty degeneration of myelin sheath & Schwann cells. Clinically, this theory is substantiated by the intermittent nature of symptoms & also the favorable response to local hydrocortisone infiltration, which probably acts by suppressing the local inflammatory process.

Dural Origin:

Edgar & Nundy (1966) showed fine nerve trunks on the ventral surface of the dura, derived from the ramus meninges of the gray ramus communicans & anterior primary rami, which are perivascular in distribution [50]. They are in longitudinal anastomoses with branches from one segment running to adjacent ones, so that pain may be felt not only in the affected segment, but also in adjacent ones.

Pressure on Nerve Roots:

Pressure produces irritation, stretching, compression, occlusion of vasa nervosum, &/or degeneration of nerve fibers. Murray & Falconer dispute this theory on the ground that relief of symptoms often occurs even when the protrusion persists, as shown by radiography. Another strong opponent is Kelley (1956), who points out that axon cylinders cannot be receptors of pain, as in the following conditions where

weakness & not pain is the presenting symptom though sensory deficit & altered tendon reflexes are common to all conditions in the more advances stages:

- Radial nerve compression in Humeral fractures.
- Tuberculous spondylosis with neurological involvement.
- Tardy ulnar palsy, etc.

Hence, the exact mechanism by which pain is produced is still a matter conjecture. Similarly, the intermittent nature of the symptoms is explained as due to one or more of the following factors:

- Rest offered by forced inactivity
- Partial or complete reduction of the disc (mobile)
- Healing of tears in the annulus fibrosis
- Desiccation & hence reduction in the size of extruded disc material
- Relief of compression of the nerve roots due to suppression of inflammatory reaction & venous stasis
- Adaptation of nerve roots to pressure & tension with lengthening.
- Variation of root – protrusion relationship as when postural variation
- Causes the root to slip off the summit of the protrusion & subsequently either the protrusion shrinks or patient continues to maintain the pain free posture (list).

Clinical Symptoms & Signs

It is only possible to arrive at an accurate diagnosis in 80 % of cases of backache. The actual lesion often remains obscure in spine of accurate clinical & radiological investigations. Accurate diagnosis demands careful, thorough & detailed elicitation of history, meticulous physical examination, including neurological assessment as well as interpretation of x-rays & other laboratory investigations.

The clinical diagnosis of low back pain & sciatica (Stevens J, 1968) covers a large number of different disorders, the commonest being prolapsed intervertebral disc (90 %), then soft tissue disorders, such as lumbo-sacral strain, fibrositis, myosititis, or Newman's spring back, osteoarthritis, & miscellaneous, in that order of frequency [51]. Other, although rare, conditions are equally important, e.g. specific infections such as pyogenic osteomyelitis, tuberculous spondylitis & brucellosis, & non-specific conditions, such as ankylosing & brucellosis, & non-specific conditions, such as rheumatoid arthritis, osteomalacia, paget's disease etc., neoplastic such as myelomatosis, neurofibromatosis, hemangiomas, lipomas, & primary or secondary malignant tumors of the spine, & metabolic disorders such as severe osteoporosis.

Lumbar disc disease may manifest itself with predominantly mechanical or 'orthopedic' signs, or neurological signs or both. The onset of backache or lumbago may be sudden, after lifting a weight or after bending forwards to pick up an object, or it may have an insidious onset & progress slowly. Often the backache has an insidious onset & progress slowly. Often the backache is mild & intermittent, brought on by exertion & relieved by rest. This persists for a few days or weeks till a sudden strain aggravates the trouble. This pain may be felt over the spine, the sacro-iliac joints or the iliac crest or occasionally even the groin, & is due to the stretching of the annulus & the posterior longitudinal ligament. Tender areas may be felt over the sacral or the gluteal region, but tenderness of the sciatic nerve is uncommon in disc lesions. The backache is accompanied by stiffness of the spine due to reflex-muscle spasm. Bilateral spasm leads to scoliosis, which may be either towards or away from the side of the disc. In some cases alternating scoliosis is observed. The patent assumes the posture, which gives him the least pain. Because of muscle spasm, all movements of the spine are limited especially forward bending. In case of a disc

prolapse, generally only one movement is restricted. A generalized restriction should make us investigate for alternative causes of pain. All these symptoms get relieved by bed rest. However, while resting in bed, the patient may find it difficult to turn over unless he lifts his whole pelvis & turn it as one unit. Gardner's test for malingerers states that if the patient is able to sit with his legs & trunk at right angles, it is probable that he has a straight leg raising test (SLRT) to 60-70 degree.

The progress of symptoms may stop at this stage with no neurological sins or may progress further neural involvement.

Neurological Symptoms & Signs:

Neurological symptoms may appear simultaneously with pain & stiffness of the spine or often much later. The earliest evidence of root involvement in a lumbar disc prolapse is radiating pain along the course of the sciatic nerve, commonly called sciatica. The exact distribution of the pain depends upon the particular root involved. Usually it is a shooting, episodic pain, related to movement, & going down the back of the thigh a varying distance either along the posterior or postero-lateral aspect of the calf. The distribution of pain will naturally be different if the disc prolapse is at a higher level. The radiating pain is usually often aggravated by coughing, sneezing, or straining.

Besides pain, compression of the root may produce parasthesia, tingling or numbness in the area of the distribution of the particular root. As the compression progresses objective neurological deficits i.e. sensory, motor or reflex appear. Usually the symptoms & signs are restricted to one root, but occasionally other adjacent roots or the whole of the cauda equina may be compressed (Tandon &

Sankaran, 1967) [52]. The signs & symptoms of specific root involvement are summarized below in Table 1.

An important feature of the disease is the intermittent exacerbation & remission of varying duration & severity. The sciatic pain in some rare instances may get suddenly relieved leaving behind motor weakness & numbness, if the nerve root loses its conductivity due to extreme compression or ischemia. Occasionally the backache may disappear with the appearance of the sciatic (root) pain & is due to the fact that with the extrusion of the disc, the stretch on the annulus has eased.

Table 1 – Common Root Involvement

Root	Cause	Sensory Deficit / Parasthesia	Motor Deficits	Reflexes
L 4	L 3 – L 4 PIVD, L 4 Foraminal Stenosis	Postero-lateral Thigh, Anterior Knee, Medial Leg	Quadriceps Hip Adductors	Patellar tendon
L 5	L 4 – L 5 PIVD, L 5 Foraminal Stenosis	Anterolateral Leg, Dorsum of the foot, Great Toe	Extensor Hallucis, EDL, EDB, Gluteus Medius	Usually None
S 1	L 5 – S 1 PIVD, S 1 Foraminal Stenosis	Lateral Malleolus, Lateral foot, Heel, Web of 4^{th} & 5^{th} Toes	Peroneus Longus & Brevis, Gastro - Soleus, Gluteus Maximus	Achilles Tendon

Straight-Leg Raising Test (SLRT - Lasegue's Test):

Compression or stretching of any one of the nerve root constituting the sciatic nerve, results in limitation in straight leg raising due to the extra stretch that this maneuver causes. This sign is present in the vast majority of cases of root compression due to protruded discs. Usually, the pain begins when the leg is raised to about 40 degrees. Extension of the contralateral leg may also be limited by the onset

of pain on the affected side. This happens in 43% of cases in unilateral disc lesions & 88.3% of cases where the disc has protruded en mass [53]. The raising of the contralateral leg tugs at the sensitive nerve root causing pain. In our country, limitation of straight leg raising may not be seen in some patients thought a root compression by a prolapsed disc is proved later at surgery. This is explained by the fact that in India most people, especially women, adopt the stooping posture to perform many of their daily chores. Thus the nerve is elongated & has a certain amount of elastic flexibility which permits full straight leg raising despite the compression by the disc protrusion [39].

To differentiate the pain arising from stretching of the sciatic nerve roots from that of lumbosacral or sacro-iliac disease, the straight leg raising test can be modified. The patient is made to do the straight leg raising test, till the pain just appears, the leg is now lowered a little to make the pain disappear. At this angle, a dorsiflexion of the ankle will reproduce the pain, if it is due to nerve root stretching & not from any other causes. The Kernig's test also is useful in such cases to confirm root irritation. With the patient lying supine the hip & knee are flexed; when the hip flexion is about 90 degrees, the knee is slowly extended. This causes the sciatic pain to appear in cases of ruptured discs.

The commonest roots to be involved are the L5 & S1 nerve roots. Sarin & Chandy (1959) found L5 root involvement in 58% of their cases whereas the commonly reported figure is about 40%. Balaparameswar Rao & Dinakar (1970) also reported involvement of L5 in 72% of cases [53].

Occasionally bladder symptoms may appear without the other characteristic features of a disc syndrome (Love & Emmett, 1957). This syndrome has been

reported only in women. Urinary retention is usually uncommon in women except when due to cystitis or gynecological causes. This report assumes significance in stressing the importance of proper investigation of cauda equina function in cases of urinary retention in women (BMJ, Edit, 1967). Removal of protruded lumbar disc relieves the symptoms.

Investigations

Plain X - Rays

Straightening of the normal lordosis, scoliosis, narrowing of the affected disc space & osteophyte formation are the usual features. Oblique x-ray films may reveal associated spondylolysis in the lumbar region but are more useful in cervical disc lesions to show osteophytes & foraminal narrowing. Well marked calcification of the affected discs may indicate alkaptonuria; rarely the ruptured & extruded discs may calcify & thus show up on plain films. These changes in the plain x - rays localize the actual prolapsed disc responsible for the patient's symptoms. Neurological signs aided when necessary by MRI / Myelography gives more accurate information. Plain x-rays may often be normal in a case of disc prolapse.

Plain x-rays are particularly useful in excluding other lesions causing backache & sciatica, like spondylolisthesis, arthritis, tuberculosis & secondary tumors.

In India, one must always keep in mind the possibility of tubercular lesion of the spine mimicking the picture of a disc prolapse. In reviewing x-rays of the spine in a case of sciatica, very often the surgeon is so occupied with the height of the intervertebral disc & the pedicle or transverse processes, which may be the seat of a tuberculous focus [52].

A preliminary estimation of the size of the canal can be made by observing the ratio of the size of the canal to the size of the adjacent vertebral body at each segment. In cases presenting with symptoms of intermittent claudication, such a study helps to determine the cause of the claudication as neural rather than vascular (Jones & Thompson, 1968). It may, however, be mentioned that routine plain x-rays may not reveal this lesion. Axial tomography is required to demonstrate the shape & size of the spinal canal.

Myelography:

This is performed only in doubtful cases, after careful thought. Any study that introduces a foreign material, however, innocuous, into the subarachnoid space should be advised only when absolutely necessary. In case neurological localization of the affected root is definite there is no need for myelography.

The indications in a suspected case of disc prolapse are as follows:

- When neurological signs are not definite;
- When there are bilateral signs;
- When there are signs of multiple root involvement;
- When there is a bladder involvement;

Computed Tomography (CT):

This is an excellent non-invasive method of investigating the spine. This can be either Plain or Contrast enhanced. The invention of CT has helped to accurately diagnose any abnormality in the vertebral body & its appendages. Thinner sections or cuts can be taken in the later generation of CT scanners which help to delineate the pathology better. The main disadvantage being the expense & availability but with due course of time this is increasing exponentially.

Magnetic Resonance Imaging (MRI): (Figure 14, 15, & 16)

MRI is currently the standard for advanced imaging of the spine. MRI is superior to CT in most circumstances, in particular, identification of infections, tumors, & degenerative changes within the discs. More importantly, MRI is superior for imaging the disc & directly images neural structures. Also, MRI typically shows the entire region of study (i.e., cervical, thoracic, or lumbar). Of particular value is the ability to image the nerve root in the foramen, which is difficult even with post - myelography CT because the subarachnoid space & the contrast agent do not extend fully through the foramen. Despite this superiority, there are circumstances in which MRI & CT, with or without myelography, can be used in a complementary fashion.

One of the difficulties with MRI is showing anatomy that is abnormal, but may be asymptomatic. Gibson et al. found disc degeneration in all symptomatic adolescent patients & in four of 20 asymptomatic adolescents. Boden et al. found cervical spinal abnormalities in 14% of asymptomatic patients younger than 40 years old & in 28% of asymptomatic patients older than 40. Cervical disc degeneration was found in 25% of patients younger than 40 years & in 60% of patients 60 years & older. They studied lumbar MRI of 67 asymptomatic patients & found that 20% of patients younger than 60 years had a herniated nucleus pulposus, which also was present in 36% of patients older than 60. Asymptomatic abnormalities were found in 57% of patients 60 years old or older. Lumbar disc degeneration was found in 35% of patients 20 to 39 years old & in 100% of patients older than 50. The demonstrated findings must be carefully correlated with the clinical impression. The importance of this concept cannot be overstated. The best way to obtain meaningful clinical information from MRI of the spine is to have a specific question before the study. This question is

derived from a patient's history & a careful physical examination & is posed using the parameters of (1) neural compression, (2) instability, & (3) deformity. In each case, the specific location of the abnormality should be suspected before MRI & confirmed with the study. Only abnormalities in one or a combination of these categories are important because surgical techniques can treat only these problems. Failure to interpret an imaging study in this way, especially MRI, which is sensitive to anatomical abnormalities, would inevitably lead to poor clinical choices & outcomes.

Research using MRI after the administration of a contrast agent has shown differences in the diffusion of the contrast material into the discs. Rajasekaran et al. found consistent differences dependent on the end plate in the pattern of gadodiamide diffusion into the nucleus pulposus. These pattern differences correlated more with degenerative changes & not with age. This pattern may allow for development of criteria to distinguish normal aging of the disc from pathological disc degeneration.

T1 is used for vertebral body structure, while T2 is used to enhance the spinal cord. It is much more expensive & availability is restricted to a few centers in the country.

Discography:

Myelograms do no locate disc prolapse less than 8x8x8 mm especially when such prolapse is sited far laterally. In such cases discography has been advocated. Contrast medium may be injected directly into the suspected intervertebral discs under nuclear clefts (a sign of disc degeneration) & demonstrated the affected disc by the leakage of contrast medium along the tear. Discography is a painful procedure liable to complications, like iatrogenic disc prolapse along the needle track, aggravation of neural damage by extruding additional disc tissue through the annular

& infection in the disc space. False positive & negative results also occur. Hence, discography has not attained the popularity of myelography, MRI & has no adherents in this country. Intraosseous vertebral venography is useful when myelography fails to confirm a lateral disc prolapse despite clinical signs. Absence of filling of the intervertebral, being at the concerned level confirms the presence of a compressing lesion (Schobinger, et al. 1961).

Electromyography:

Examination of paravertebral muscles in the appropriate zone may help to localize the exact level nerve root compression. Segmental denervation is easy to detect with this technique & a few surgeons have been confident enough to operate on these findings.

While a diagnosis based on clinical signs & myelography offers no problems in the majority, the reminder may tax the ingenuity & wits of the most experienced clinician. A large disc protrusion with marked nerve root compression may produce in a patient with a wide spinal canal, only scanty neurological signs while 'orthopedic' signs or backache is prominent. Most clinician are unwilling to embark upon myelography in such cases. In some of these cases, electromyography may help. In others, unless the symptoms are severe enough, the patient might be put through a fairly long & painful period of conservative treatment before myelography is undertaken. With the advent of MRI, all of this has changed & it has become the Gold Standard for Disc lesions.

Figure 14 – MRI Image of a Protruded Disc (Transverse Section)

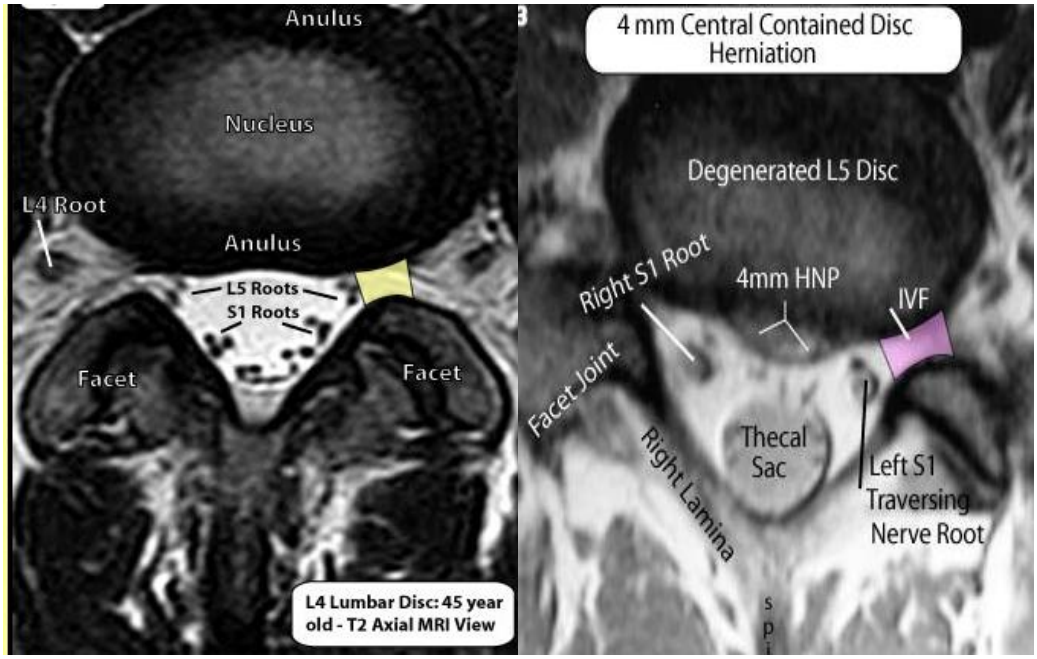


Figure 15 - MRI (Saggital Section)

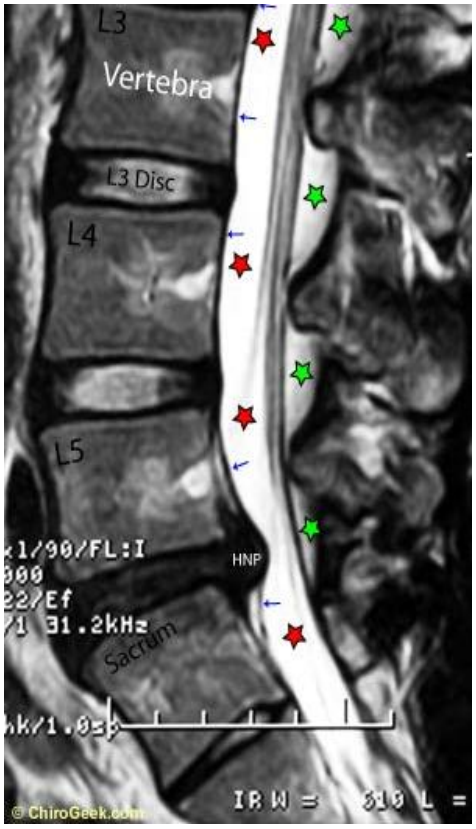
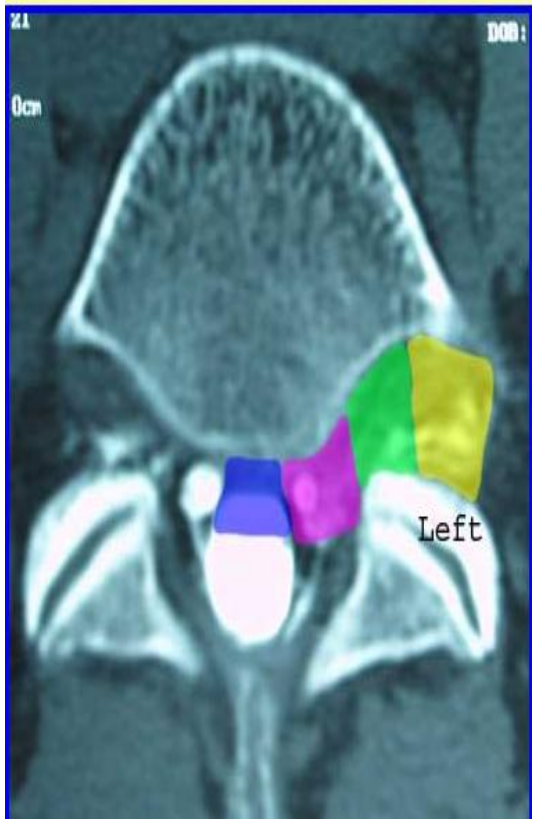


Figure 16 – Zones of Disc Protrusion



Treatment

From the onset it must be re-iterated that there is no a single treatment for backache & sciatica offering a complete & permanent cure. The three most effective components are (Cyriax)

(a) Exercise & heat to lumbar spine

(b) Manipulation & traction

(c) Epidural injection – which is unfortunately not very widely practiced

Broadly speaking total management of backache falls into three groups:

1. Prophylactic

2. Operative

3. Conservative.

Prophylactic Treatment

There is no prophylaxis in so far as there are no means available to prevent backache from occurring. The main role of prophylaxis comes after the completion of conservative & operative treatment & is aimed at prevention of recurrence of symptoms. Prophylaxis consists of the correction of both the dynamic & static posture & to avoid undue & unnecessary stresses & strains which predispose the herniation of the disc.

Surgical Treatment

This involves removal of the extruded disc either through fenestration surgery or wide laminectomy, which may or may not be followed by arthrodesis of the spine.

The indication for surgical intervention, the technique & management, are controversial & much has yet to be found out before the fundamental principles become universally accepted.

Operative removal of the prolapsed disc tissue for the relief of persistent sciatica is now accepted as a valuable & safe procedure, attended with very good prospect of cure & minimal morbidity. Even with surgical removal the cure obtained is only about 40-50 percent, Shinners & Hamby (1949) [54, 55], Dunkerley (1971), Love (1949), Raaf & Berglund (1949) [56]. On the other hand the relief from sciatica is in the region of about 80 percent of cases. Better results of surgery are obtained in cases who displayed objective neurological evidence than in those without neurological signs (Shinner & Hamby). In my opinion there is no logic in the wait & watch policy, a more realistic & aggressive attitude is more desirable & beneficial for the patient once conservative treatment has failed to relieve the symptoms.

MRI can form a useful pre-operative investigation in locating & defining lesions. A cauda equina lesion is an indication per excellence for urgent exploration.

Conservative Treatment

This, in almost all the cases suffering from disc syndrome, forms the first line of treatment. Traditionally it consists of bed rest, analgesics & muscle relaxant drugs, traction, physiotherapy consisting of short wave diathermy, ultrasonic heat, massage, intermittent traction, & exercises, & epidural injections. There appears to be general agreement in that all forms of conservative treatment are effective & beneficial to the patient in some form or other. The choice & plan of conservative treatment is very much a better of the clinician's own likings & dislikings, & is indeed his ideas, interests & beliefs.

Bed rest on Hardboard:

It is by far the most widely form of conservative treatment aimed at both physical & mental rest. Putting the patient in bed in itself reduces the intradiscal pressure considerably & also, by minimizing the movement, reduces the chances of nerve irritation & thereby affords relief of displaced cartilage (Cyriax), eliminates the work & the gravity stresses, & diminishes the muscle spasm. For the treatment to be effective the patient must be at complete rest, not only physically but also mentally. The patient cannot rest if agitated or worried (Raaf) [56]. The obvious disadvantages if prolonged rest in bed is that the patient should ideally be hospitalized which entails the patient moving out of his home surroundings with its accompanied disadvantages.

Other disadvantages are:

(i) slowness in affording relief,

(ii) economic factors both involving the hospital & the patient,

(iii) extra workload for nursing staff

To this are added further risks, particularly to the patient who is on traction, such as psychological upsets, nausea, vomiting, tension secondary to the patient's orientation to horizontal plane, difficulty of micturition & defecation, feeding, dressing & boredom, etc. In short it takes a minimum of two or three days for the patient to settle down & occasionally these symptoms may be severe enough to warrant the stopping or taking off the traction. To this may be added other risks of prolonged decubitus such as deep venous thrombosis with all its attendant complications & hypostatic pneumonia, etc.

Medication

Various medicaments that have been used fall mainly into 3 groups:

1. Simple analgesics,
2. Muscle relaxants – these were used in the hope to combat painful spasm & also to produce sedation & tranquillization (Krayenbuld & Zender). Good results have been claimed by some.
3. Anti - inflammatory drugs – use of these drugs is based on the fact that no respective inflammatory processes be that due to mechanical, chemical or of auto-immune nature has been confirmed by histological means (Rexed & Lindahl). The obvious logical step was to use anti-inflammatory drugs to combat this inflammatory process.

Other drugs that have been tried are:

1. Cortisone – systemic use is hazardous & dangerous for obvious reasons. Locally its use it effective & useful, both in reducing inflammatory process & reducing the subsequent fibrosis & adhesion formation,
2. Non-steroid drugs – such as:

    Phenylbutazolodin

    Tanderil

    Indomethacin

Physiotherapy

Heat, & massage according to many is soothing & may help to reduce the spasms, but Cyriax believes that in disc lesions heat & massage, though futile are quite harmless. Hydrocollator packs-moist heat-is more comfortable than dry heat.

Exercises in the acute stage are not only undesirable & illogical, but are positive harmful. In the acute stage, the patient needs rest rather than mobility but in the convalescent period exercises should from the integral part of the prophylactic managements by correcting the posture, by improving the tone & muscle balance, undue strain of movements & gravity are minimized.

Supports:

Cyriax considers it a logical treatment to be advocated after reduction of the disc & this he believes is to maintain the reduction. Supports can be given either as:

(a) Surgical appliances such as corsets or lumbo-sacral supports.

(b) Plaster of Paris jacket.

Advantages of Supports

(a) They permit earlier mobilization, ambulation & resumption of activities.

(b) They restrict movements of the deranged segment of the spine - a permanent factor in the production of pain, i.e. it immobilizes spine.

Disadvantages & Limitations of the Supports

(a) If continued without exercise, the muscles waste in a very short time.

(b) They do not eliminate the stresses of gravity

(c) Proper molding & fit of the corset is of the utmost importance to that they can position & maintain the spine in the proper physiological curve. Short corsets are no good. They in fact increase lordosis.

Back Braces

Back braces often offer no advantage over corset. On the other hand, they are less comfortable.

Manipulation

They are a great deal of controversy & debate as to the indications, value & mechanism by which manipulation affords relief. Some people believe that by manipulation of the spine, one is breaking the peri articular adhesions. Cyriax believe that manipulation reduces the displaced fiber cartilage part of the disc & therefore is only successful in those cases, & not when there is protrusion of the nucleus pulposus can be carried out with or without anesthetic.

Manipulation is contraindicated in conditions such as.

(a) Signs of involvement of both sacral roots.

(b) Pregnancy.

(c) Spinal claudication.

(d) Neurosis.

It is likely to be a useless procedure if the disc protrusion is large & soft. The value of manipulation in post laminectomy cases is doubtful.

Epidural Injections

The effectiveness & utility of nerve blocks as diagnostic & therapeutic measures in the management of disc sciatica syndrome is depended upon inherent properties of local anesthetic to interrupt specific sensory & somatic, motor & anatomic pathways & also on the anti-inflammatory & anti-proliferative properties of hydrocortisone.

In the recent years, epidural injections are gradually finding their well-deserved place in the management of backache & sciatica. The results of relief from the treatment so far published are quite encouraging.

Mechanism of Action of Epidural Injections

Epidural injections have been used successfully for decades in treatment of low back pain & sciatica. The mechanism of action of the drugs is uncertain. Hence, there have been many theories as to what should be injected & what is being accomplished by the injection as well as the best way to approach the space, these include the following.

Mechanism of Action:

Anti-Inflammatory Effect:

For more than a decade, orthopedic surgeons have considered the likelihood of chemical irritation of the nerve root in association with disc prolapse as the cause of very acute pain following injury. This view has arisen from the frequent finding at operation of a swollen, inflamed, nerve root without bone pressure. The chemical content of the nerve root lists glycoprotein as a constituent. Direct tests of nucleus pulposus pharmacologically show the presence of 1-4 mg Histamine per gram, but no

tryptamine & no slow reacting substances or kinins. This cannot account for acute pain. However extract of the Glycoprotein of Histamine, edema fluid, protein, & another amine with four times the mobility of Histamine from the isolated perfused lung of guinea pig. This spot on the chromatogram corresponds to the amine spot found in red wine, which accounts for severe pain in migrant type of neuralgia. This substance has also been shown to be the cause of acute pain in pancreatitis. The acute pain in a disc lesion occurs due to local irritation of the nerve root producing edema & release of protein & 'H' substance at the site of disc injury. The corticosteroids act by inhibiting the peripheral response to 'H' substances (Marshal, L.L. & Taylor, A.)

Inhibition of the Autoimmune Response to Nucleus Pulposus:

The nucleus pulposus is normally contained tightly within the annulus & after its embryological formation it no longer normally takes any vascular contact with the systemic circulation. This system is analogous to other human situation in which derangement is known to produce an autoimmune response. The thyroglobulin in Hashimotos disease of the thyroid & the similarly enclosed Vitreous humor of the eye in the production of sympathetic opthalmitis are to such autoimmune diseases.

At present, emphasis has been focused on regional lymph nodes as the site of antibody production in many immunological responses (Scothorne & McGregor, 1955). This antibody does not enter the systemic circulation to give humoral antibody titres but remains fixed within the cytoplasm of certain lymphocytes in the cell bound antibody state. In this cell bound state the antibody can be carried from the site of production within the antibody can be carried from the site of production within the primary regional lymph nodes by efferent lymphatics to the site of the antigen (or auto antigen) & here produce a chronic inflammatory reaction of antigen-antibody. A new

hypothesis based on this type of autoimmune response has been formulated as a possible explanation for the perplexing cases of chronic inflammatory reaction so often encountered as nerve adhesions during a "Negative disc exploration" in patients with chronic low back pain. The concept depends upon whether autoantibodies can in fact develop to ones own nucleus pulposus autoantibodies can in fact develop to one's own nucleus pulposus if this is exposed to a blood supply. It has been shown in experimental findings in the rabbit that autogenous nucleus pulposus material can excite an autoantibody response in regional lymph nodes & if this is true, corticosteroids can suppress this antigen antibody reaction & also chronic inflammation proved by this reaction.

The Mechanical Effect:

Evans in 1930 published intra sacral epidural injection in the treatment of sciatic. By injecting high volumes either of an local anesthetic or normal saline (up to 100 ml), he observed displacement of the posterior & lateral aspects of the dural sac & reasoned that the physical displacement of the neural elements caused by the injected fluid may load to stretching & lysis of the neural adhesions & even to anaesthesia from compressive effects. This breaking down of scar tissue in the epidural space by causing neurolysis can cause relief of radiating pain, but this has never been proved by any study. Also it was found by the injection contrast media into the epidural space that high volumes simply pass out of intervertebral foramen. When there has been previous surgery the contrast is guided away from the scar tissue site & takes the path of least resistance.

Indications/Contraindications & Routes of Epidural Steroid Injection

Indications can be conveniently divided into two main groups:

1. Diagnostic

Epidural injection is useful in determining the cause of:

(a) Uncharacteristic Backache

(b) Referred pain, which will not be affected or altered

(c) Contradictory opinion & medico legal cases

(d) Psychoneurosis

2. Therapeutic indications

a) Hyper Acute Lumbago - in this condition, the real value lies in providing the patient quick & prompt relief & thereby avoiding unnecessary heavy sedation & drugging of the patient.

b) Intractable backache

c) Chronic backache

d) Pregnancy with disc symptoms where other conservative treatment may not be advisable for obvious reasons

e) Root pain with or without neurological signs

f) Nocturnal cramp & coccydinia

g) A patient with discogenic pain with or without sciatica, not relieved by adequate conservative methods (Acute / Chronic)

h) As a non-operative treatment in patients with lumbar canal stenosis.

Routes

Though the epidural space can be theoretically approached at any segmental level, the lumbar (interspinous) & the caudal routes are the commonly preferred to the others, in the lumbosciatic syndrome.

In the caudal route, first used by Sicard & by Cithelin in 1901, the epidural injection is made through the sacral hiatus. It has the important advantages of a comparatively lesser risk of accidental dural puncture, but has certain disadvantages. Higher rate of congenital abnormalities of the sacrum, variations in the sacral curvature, difficulty in identifying the hiatus, particularly in an obese individual, & larger volume of fluid required. Forty ml of fluid spreads from the caudal region to the thoracic segments, as confirmed by using a radio-opaque dye.

The lumbar route has a slightly greater risk of dural puncture which can however be overcome by a patient & careful technique, but the advantages are more ease of identification, a lesser degree of variation in the anatomy of the lumbar region, nearer to the probable site of lesion, thus enhancing its therapeutic value.

Hence, the controversy of which route is superior continues to exist & requires further studies to arrive at a conclusion.

Contraindications:

a) Inflection at the site of needle.

b) Septicemia.

c) Progressive neurological deficit.

d) Hemorrhagic diasthesis.

e) Cauda Equina syndrome, which is a surgical emergency.

Complications & Its Management

1) Due to technical difficulties -

a) Most common complication is that the injection becomes an intrathecal, with recovery of spinal fluid. In these situations the needle can be withdrawn & the next level above or below selected. An occasional intrathecal injection is accompanied by transitory "Spinal" headache which usually is relieved within 24 hours by the use of oral analgesics.

b) Epidural needle may break & should be removed surgically as soon as possible

2) Commonly transitory mild weakness in one or both lower extremities is noted secondary to the addition of local anesthetic in the epidural space. This usually subsides after 15-20 minutes after which the patient is able to resume normal ambulatory activities

3) Complications after intra-spinal use of long acting steroid preparation have included tuberculous meningitis, adhesive arachnoiditis, aseptic meningitis, sclerosing spinal patchy meningitis & hyper cortisolism

4) Lignocaine sensitivity

5) Transient Neurogenic Bladder

6) Paraparesis

METHODOLOGY

The role of epidural injection in low back ach e & sciatica was taken up in this study. The study was conducted for a period of one & half year, from January, 2010 to June, 2011, with the last six months being allocated to follow up under the department of Orthopaedics at a tertiary care hospital. The total period of follow up was ranging from six months to one & a half year up to the time of analysis.

Study design: A Randomised Controlled Trial

Study period: The study is being conducted from January, 2010 to June, 2011.

Sample size: The total number of cases studied was 133. 68 cases are belonging to the caudal group & 65 cases belonged to the lumbar group.

Randomisation is done by using Computer assisted Random Allocation Software.

During this period, patients were selected on the basis of criteria laid down by McCulloch in 1977 (3 or more),

1. Unilateral Leg Pain in a typical sciatic root distribution including discomfort below the knee. The leg pain has to be more severe than or at least equal to the pain in the lower back. If the roots of the femoral nerve were involved, pain would be produced in front of the thigh.
2. Specific neurological symptoms incriminating a single nerve for example, numbness over the dorsum of the foot into the great toe region or flapping of the foot on walking signifying the involvement of the 5^{th} Lumbar root.
3. Limitation of straight leg raising test (SLRT) owing to pain in the leg, by at least 50 % of the normal or cross leg pain from the unaffected leg to the

symptomatic leg or radiating to the thigh or back discomfort or calf & foot numbness on bowstring pressure on the medial or lateral popliteal nerve.

4. At least two of four possible neurological changes; muscle wasting, weakness, sensory alteration & reflex changes.
5. A positive MRI shows a disc lesion at the level suspected clinically.

Patients were selected as per the selection criteria listed below in Table 2.

Table 2 – Inclusion / Exclusion Criteria

Inclusion Criteria	Exclusion Criteria
Chronic Low Backache with Unilateral or Bilateral Sciatica > 3 months.	Cases with history of previous surgery
Refractory to Analgesics	Cases with severe motor weakness, rapidly progressing neurological deficits, cauda equina syndrome, neurogenic claudication.
Cases of age 18 yrs & above	Local infection at the site of injection
	Use of steroids 3wks or less before the study
	Allergy to Steroids, Bleeding diatheses, Pregnancy
	Uncontrolled hypertension, uncontrolled diabetes mellitus, & or were not included in the study.

After selecting the cases on the basis of the above mentioned criteria, they were counselled about the study & were included after a written informed consent to participate in the study.

All the patients underwent a thorough clinical evaluation in way of a history of the illness, including the details of pain, as well as the nature of the conservative treatment they have received in the past. This was followed by a complete physical

examination including neurological assessment of the lower limb as per a proforma prepared for the study (attached later). They were subjected to following questionnaires including the Visual Analogue Scale, Oswestry Disability Index Score (ODIS), Numerical Pain Intensity Score (NPIS), Beck's Depression Inventory Scoring (BDIS) & the scores were evaluated before & after the intervention, & at every follow up [57, 58].

The investigations done formed a part of the routine assessment protocols at the tertiary care center. All the cases were not made to undergo any investigation or procedure apart from the routine protocol followed at this center. The investigations that were done for every patient included an X-ray Lumbo-Sacral Spine – AP / LAT, MRI Lumbosacral Spine, Routine Hemogram, & a Urine Examination. An X-ray Pelvis with both hips was performed only in the cases in which there was a difficulty in approaching the sacral hiatus as a part of the caudal epidural injection to study the abnormalities of the hiatus & their significance.

The selection of the cases & assessment, clinically, radiological & laboratory investigations was done by a doctor at the department of orthopedics in the tertiary care center & were tabulated by the principal author at a later date. Following which all the cases were randomized into the two intervention groups (namely 'Group A' i.e. Caudal Epidural Injection & 'Group B' i.e. Lumbar Epidural Injection). This was performed based on a list generated by a computer assisted programme (Random Allocation Software). The randomization was unbiased & double blinded (i.e. to the patient as well as to the principal author/researcher). For every subsequent patient entering the study, the group was assigned as per this list. Once the patient agreed to

participate in the study, he was included in the group assigned to him by the number on the list & then was treated by the allocated protocol.

Materials Required (Figure 17, 18)

A pre-set Tray with cover, containing the following was kept ready for use.

a. Hole Drape (50 *50 cms size with a 10 cm hole)
b. Sponge Holding Forceps
c. Four Syringes
    I. 2 ml – For Local Infiltration with preservative free Xylocaine 2 %
    II. 10 ml – For Detection, Loss of Resistance
    III. 2 ml – For Steroid
    IV. 2ml – For Preservative Free Xylocaine 2 %
    V. 10 ml – For Saline
d. Needles
    I. 18 gauge Tuohy spinal needle (7.6 cms in length)
    II. 22 gauge Hypodermic needle (Interspinous infiltration)
    III. Other Hypodermic needles (Local infiltration)
e. Swabs
f. Bowl

Other Materials kept ready were:-

g. Steroid Triamcinolone Acetate 2 ml (40 mg / ml)
h. Normal Saline
i. 2 % Xylocaine without adrenaline & methyl parabane (Xylocard)
j. Rectified Spirit
k. Povidine Iodine

Figure 17 – Epidural Tray Set

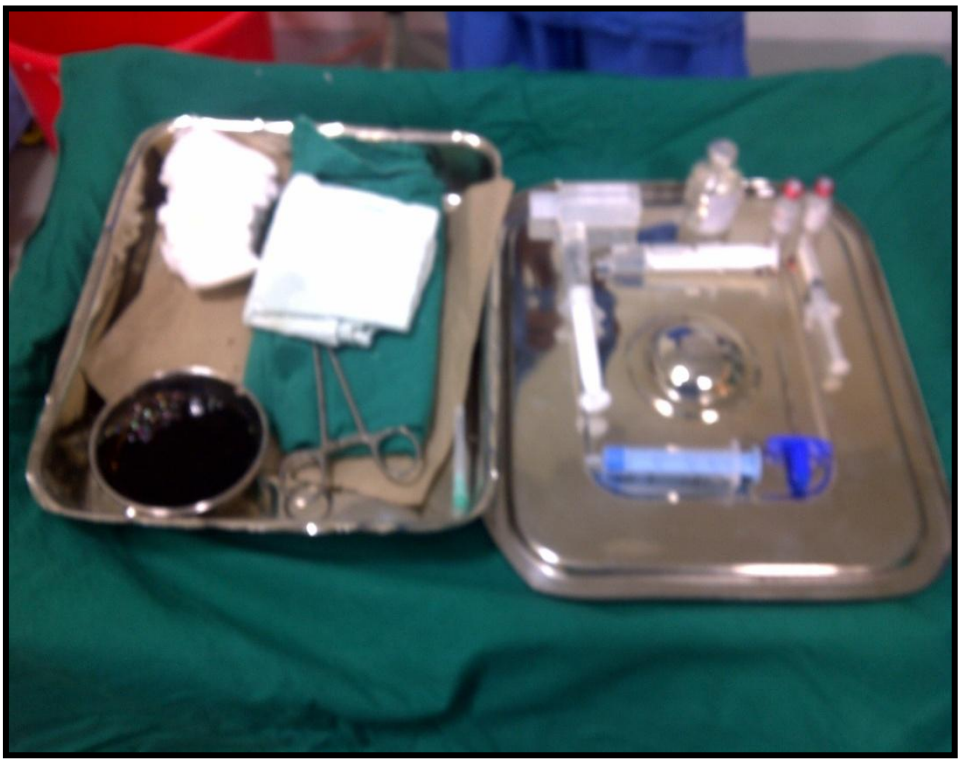
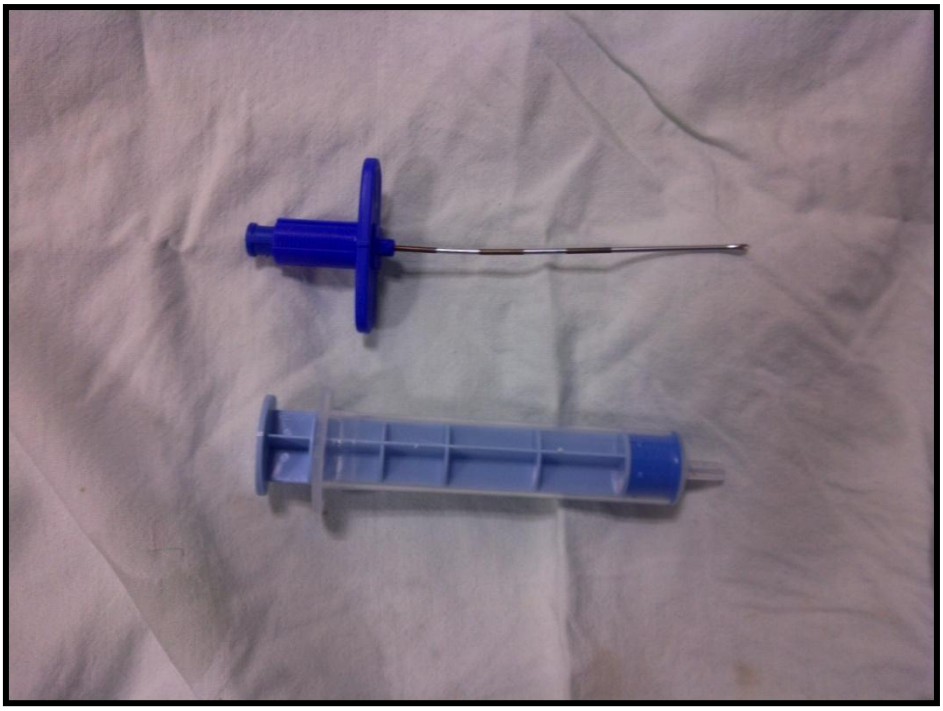


Figure 18 – Epidural Needle



The steroid used was Triamcinolone Acetate because of its marked anti-inflammatory action & relatively low incidence of side effects. Normal Saline was used to obtain wider dispersion of the steroid. Xylocaine was used for local as well as epidural infiltration. Adrenaline was avoided owing to presence of side effects & preservative free avoided certain complications attributed to the methyl parabane.

Preparation & Position of the Patient

The procedure was explained to the patient in order to obtain in order to obtain his/her full cooperation.

Before the injection is given the procedure is carefully explained to the patient, who is told to expect increase in intensity of his symptoms during the injection. It is stressed that sudden movements are likely to cause complications & that these movements must be avoided whilst the injection is in progress. The patient is also assured that intensification of his symptoms is to be regarded as a welcome sign. The principal aim in this exercise is to obtain the patient's confidence, & to sustain this confidence whilst the injection is being given by a quietly continued conversation.

All the injections were carried out in the operation theatre with dry, sterile materials. Neurological Status & SLRT are re-assessed at this stage. Aseptically the skin area was be prepared with spirit & povidine iodine several segments above & below the laminar interspace to be injected. The patient is draped in a sterile fashion.

The position used for the caudal epidural injection was the prone position most commonly or the left lateral position. In addition to these positions, sitting position was also used while giving the lumbar epidural injection. The patient lies routinely in the prone position with a pillow under the chest & one placed under the ankles to

release tension on the sciatic nerve. If the patient is pregnant, a lateral position is adopted with the affected side nearest to the table. Prone was preferred for the caudal epidural steroid injection as the give way of the resistance could be easily appreciated & the anatomical landmarks could be well palpated. Sitting position was preferred for the lumbar epidural steroid injection, as the fully flexed spine lead to opening of the interspinous spaces, especially L 3 – L 4 & below which were being used for the injection.

Procedure

Caudal Route (Figure 19 – 22)

After positioning the patient & preparation of the skin, the sacral hiatus is identified by palpation, which is located between the two horns of the sacral cornu. The hiatus also forms an inverted orthopaedic triangle with the two posterior superior iliac spines which help in identifying the location. The injection is made through the sacral hiatus which is located by palpation using the index finger or thumb. The finger or thumb is placed over the suspected area, firmly pressed down & rolled from side to side. In this way the cornu are usually felt & the hiatal space can then be determined with certainty. Difficulty may sometimes arise when there are congenital sacral abnormalities, or an unusual amount of overlying fat. In these circumstances an approximate location of the sacral hiatus can be made by pressing the buttocks together. This narrows & elongates the natal cleft, & the sacral hiatus usually lies beneath the upper end of the narrowed cleft. Alternatively the hiatal membrane can be taken to lie at a rough guess about 1-1 5 in (4 cm) from the upper end of the natal cleft. Further difficulty may arise when the sacral hiatus membrane is replaced by a bony mass. In this event the sacral approach is not possible &should be abandoned.

Using a needle, the skin over the target interspace was injected with 1 to 2 ml of 2% preservative-free Xylocaine without epinephrine & was anaesthetized. Keep the C-arm positioned so that the fluoroscopic beam remains lateral. Insert a 3½-inch, 22-gauge spinal needle between the sacral cornu at about 45 degrees, with the bevel of the spinal needle facing ventrally until contact with the sacrum is made in the "Orthopaedic Triangle". Anesthetize the area with 2 ml of 2% preservative free Xylocaine without epinephrine. This prepares the tract for a larger bore Tuohy needle. Then anesthetize the soft tissue with 2 ml of 2% Xylocaine as the spinal needle is withdrawn. Then insert a 3½-inch, 18-gauge Tuohy epidural needle & advance it an angle of 60 to 80 degrees within the anesthetized soft tissue track until contact with the sacrum has been made. At this point the needle is withdrawn 1 mm & its direction changed to about 30 to 40 degrees & gently pushed forward more cephalad, advancing it into the sacral canal through the sacrococcygeal ligament & into the epidural space using the loss of resistance technique. A spot x-ray under C arm was taken to document placement. The other methods used to document the needle placement included the Whoosh test as well as the Hanging drop test. Remove the stylet. Aspirate to check for blood or spinal fluid. Inject slowly a 10-ml volume of Normal Saline followed 2 ml of 40 mg/ml of Triamcinolone Acetate & 2 ml of Preservative Free Lignocaine, & 10 ml of sterile normal saline again, to get a total of 24 ml. Finally the tuohy needle is withdrawn & the spot was sealed with tincture benzoin.

Difficulty in Caudal Injection

Where the sacral curve is acute, advancement of the needle may be impeded. This can sometimes be overcome by bending the needle about 2-5 cm from its tip by a

few degrees or by simple manoeuvre shown in the figure. Sub-periosteal insertion is suspected if a sensation of scratching or grating is felt when the needle is advanced in a cephalad direction, & fluids cannot be injected. In this event the needle should be withdrawn & reinserted. Occasionally the needle may be located completely outside the sacrum & lie in the nearby muscles. This mistake can be suspected during the injection when there is little or no resistance to the injection & it can be confirmed by putting the 'watching' hand over the sacrum whilst the injection is made. If the injection is made sub muscularly the hand will be lifted & it will receive a vibrating sensation as the injection is made. In this situation the needle should be withdrawn completely & a further attempt should be made to insert it into the epidural space. Before the injection is given the patient is again warned against any sudden movement which could cause the needle to puncture the dura. Having estimated that the point of the needle is approximately at the S2 level & that no blood vessels or the dura mater have been punctured, i.e. by the coughing test & by withdrawing the piston of the syringe to ensure that neither C.S.F. nor blood is withdrawn, the solutions are injected. The rate of injection should be a slow, stop-&-go procedure. Too rapid injection may produce syncopal attack. Aggravation of the patient's symptoms during the injection, apart from confirming that the injection is being made at the desired level, is usually associated with a better response than when no aggravation occurs.

Figure 19 – Palpation of Anatomical Landmarks

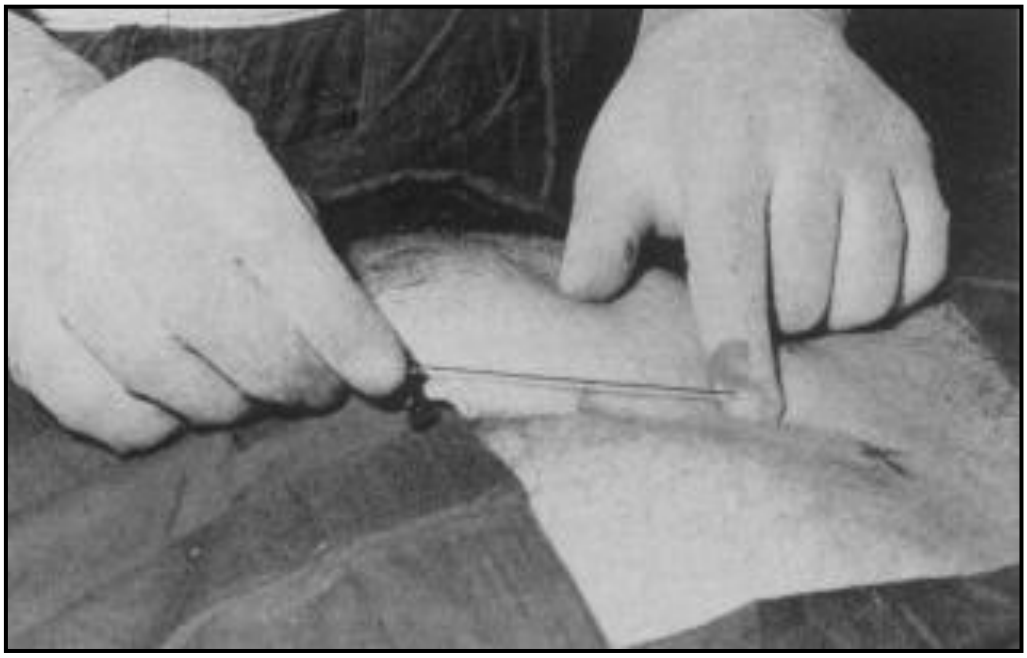


Figure 20 – Method of Insertion of Needle

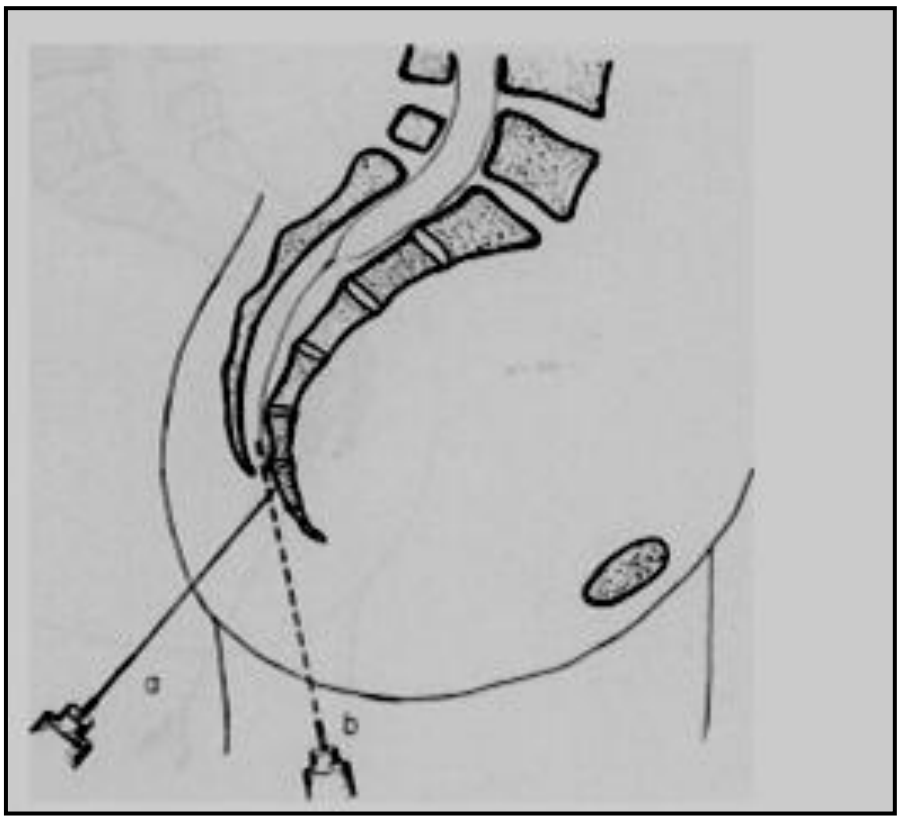


Figure 21 – Difficulties During Caudal Epidural

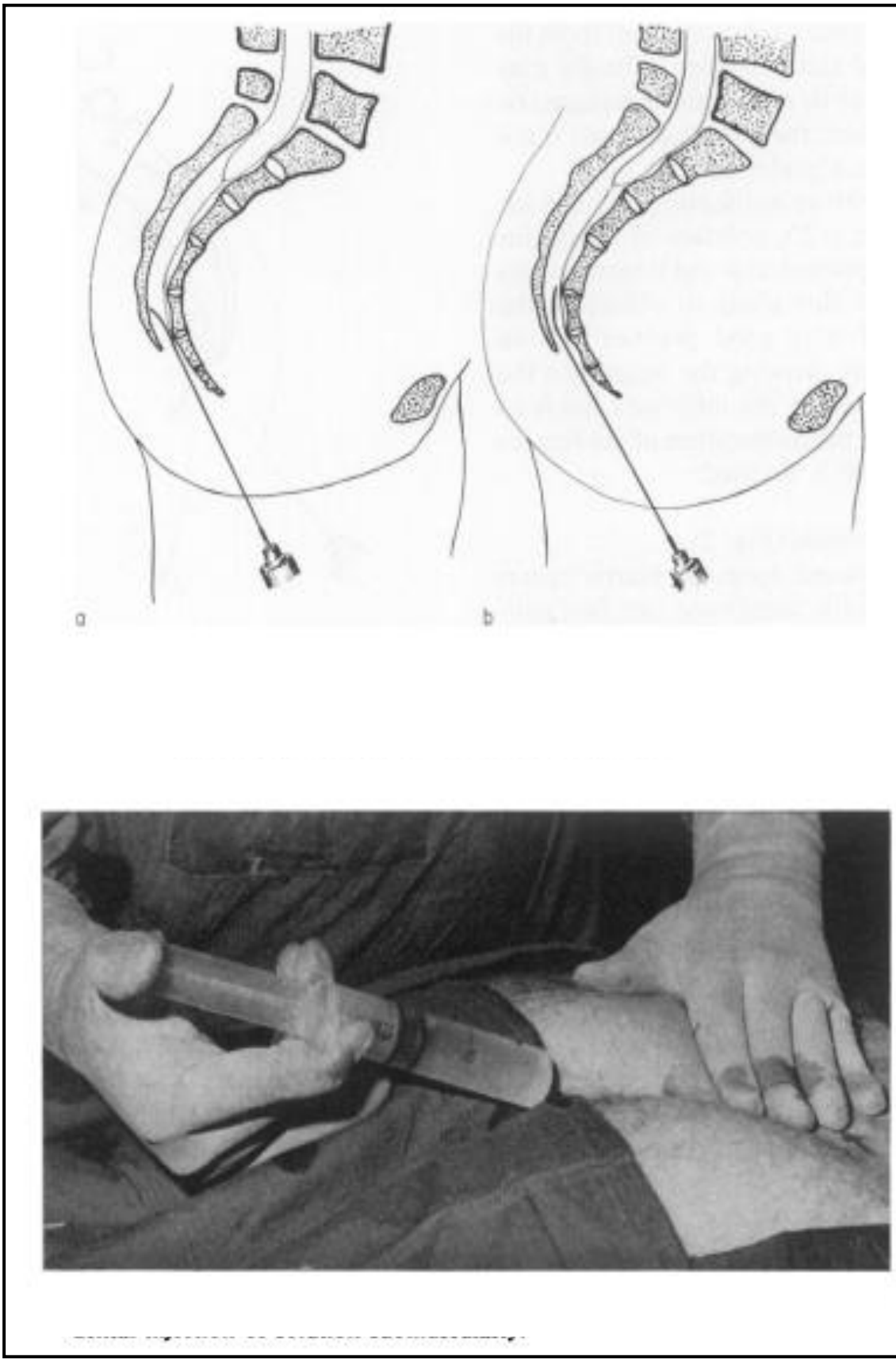


Figure 22 – Loss of Resistance / Whoosh Test

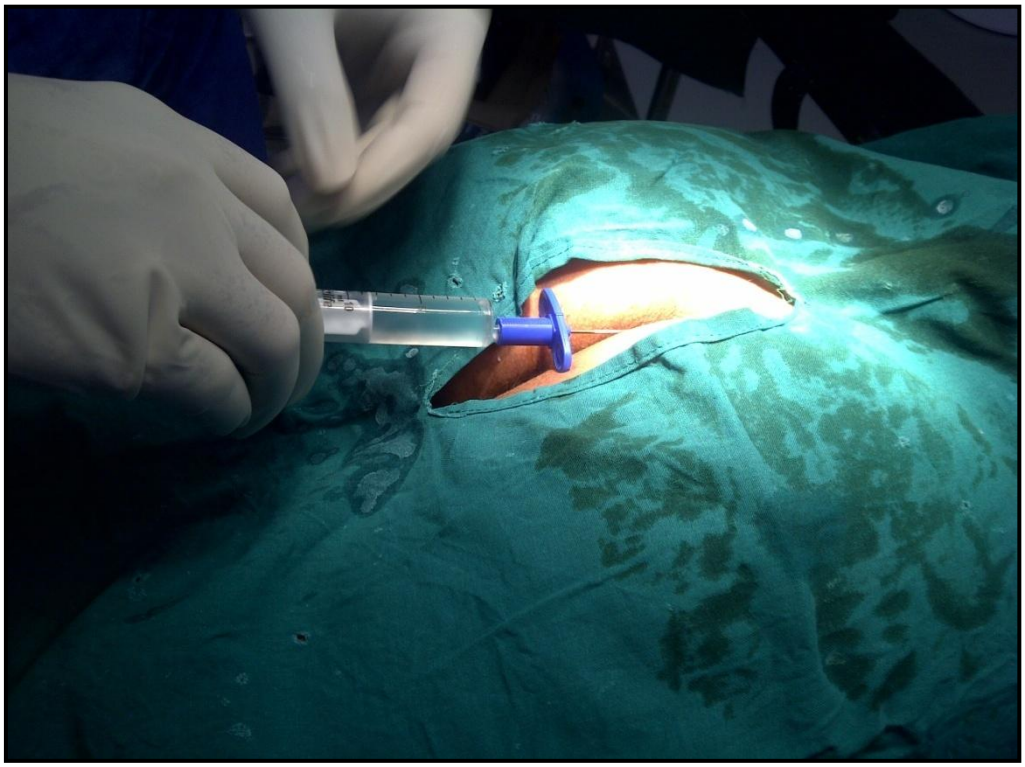
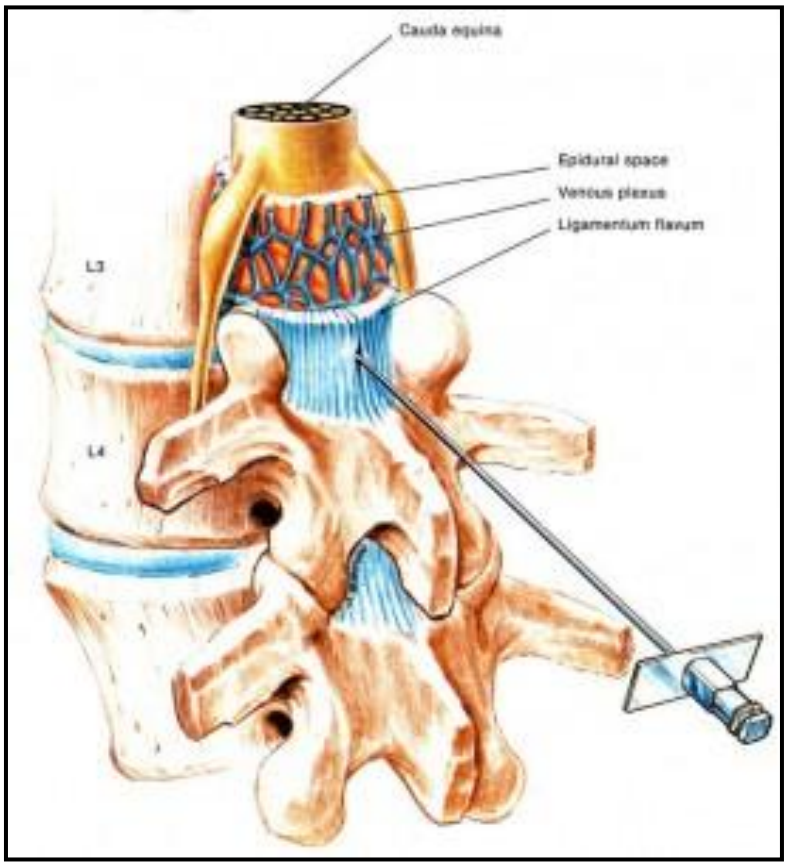


Figure 23 – Lumbar Route



Lumbar Route (Figure 23)

After positioning the patient & preparation of the skin, the target laminar interspace was identified. Commonly used anatomical landmarks were the highest point of the iliac crest, with L 4 spinous process. Using a needle, the skin over the target interspace was injected with 1 to 2 ml of 2% preservative-free Xylocaine without epinephrine & was anaesthetized. This was followed by inserting a 3½-inch, 22-gauge spinal needle vertically until contact is made with the upper edge of the inferior lamina at the target interspace. Anesthetize the lamina with 2 ml of 2% preservative-free Xylocaine without epinephrine. This prepares the tract for a larger bore Tuohy needle. Then anesthetize the soft tissue with 2 ml of 2% Xylocaine as the spinal needle is withdrawn. Then insert a 3½-inch, 18-gauge Tuohy epidural needle & advance it vertically within the anesthetized soft tissue track until contact with the lamina has been made. "Walk off" the lamina with the Tuohy needle onto the ligamentum flavum. Remove the stylet from the Tuohy needle & attach a 10-ml syringe filled halfway with air & sterile saline to the Tuohy needle. Advance the Tuohy needle into the epidural space using the loss of resistance technique. A spot x-ray under C arm was taken to document placement. The other methods used to document the needle placement included the Whoosh test as well as the Hanging drop test. This was followed by securing the needle & then injecting using a 10-ml syringe containing 4 ml Normal Saline, 2 ml of 40 mg/ml Triamcinolone Acetate & 2 ml of preservative free Xylocaine, a total of 8 ml. Finally the tuohy needle is withdrawn & the spot was sealed with tincture benzoin.

Post Injection Protocol

At the conclusion of the injection a note is made of the following: relief of pain & its extent measured subjectively as well as by straight leg raising test, & motor & sensory examination. The VAS Scale, Oswestry Index are administered & the scores recorded. The patient is advised that apart from a feeling of warmth in the legs & perhaps a sensation of walking on cotton wool, there should be no other neurological signs or untoward effect. The patient is further warned that as after any hydrocortisone injection the pain may be worsened for a few days before it begins to settle. The patient is advised to lie flat for at least 45 min after the injection which helps to avoid headache developing on sitting up. The patient should be advised to pass urine before leaving the hospital as urinary retention. Physiotherapy & Back extension exercises were continued after the injection as a routine protocol.

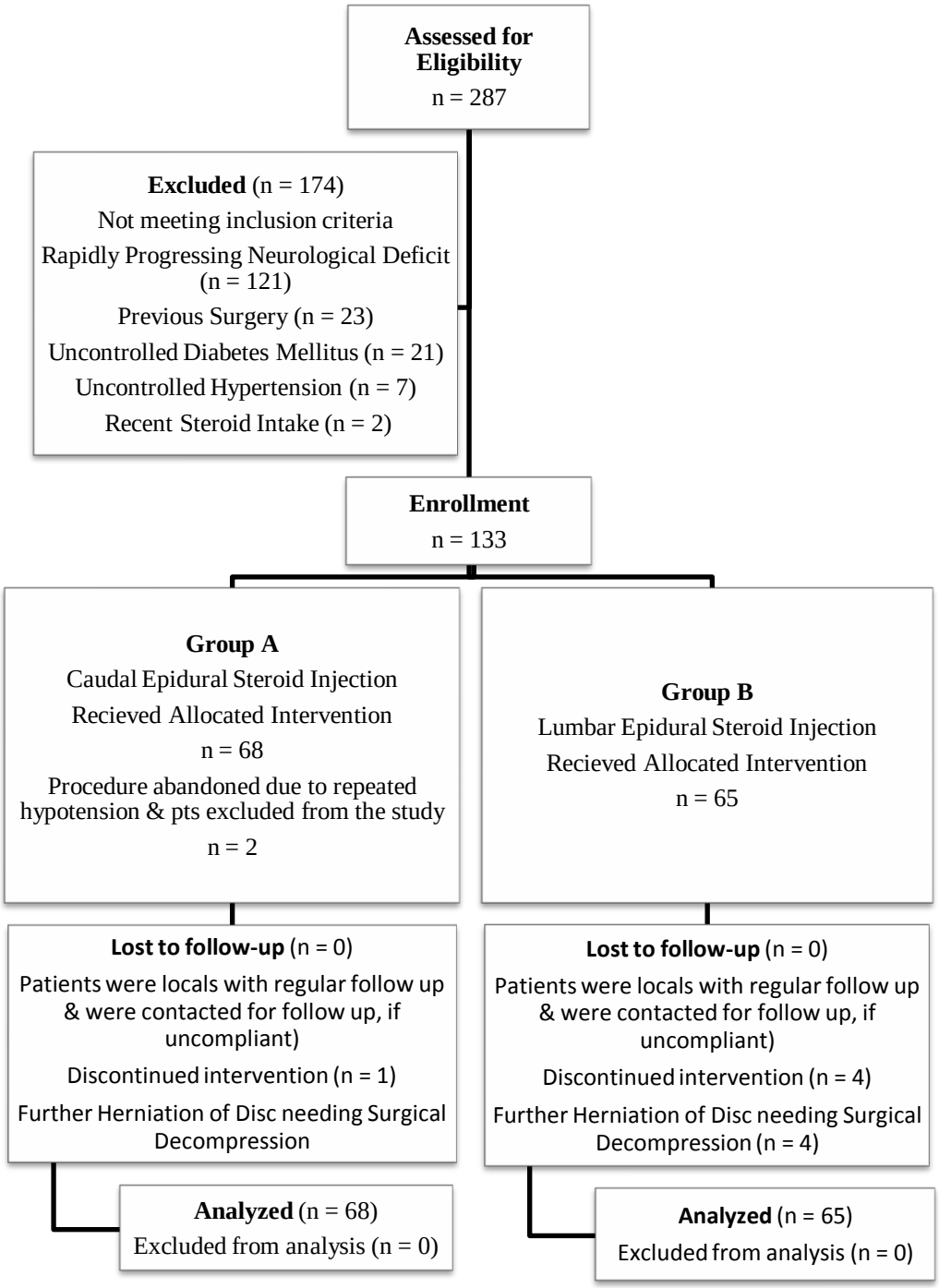
Follow Up & Repeat Injections

Clinical evaluations were performed immediately after the injection for patients, & at 3 weeks (visit 2), 3 months (visit 3), & 6 months (visit 4) for both the groups. The Visual Analogue Score, Oswestry Disability Index score & the Straight Leg Raising Test (SLRT) (positive < 60°) were used to differentiate patients whose symptoms improved from those who remained symptomatic. At re-evaluation if a patient had a complete or no pain, then no further injection therapy was done. If a patient had partial pain relief in a week from the time of the injection with a visual analogue scale score reduction not more than 20% a repeat injection was done on an average 2-3 weeks after the first Injection. If the first injection fails to relieve symptoms, further injections can be given at 2-week intervals. The number of injections is a matter of personal choice, but a total of three injections would appear to be a reasonable limit.

RESULTS

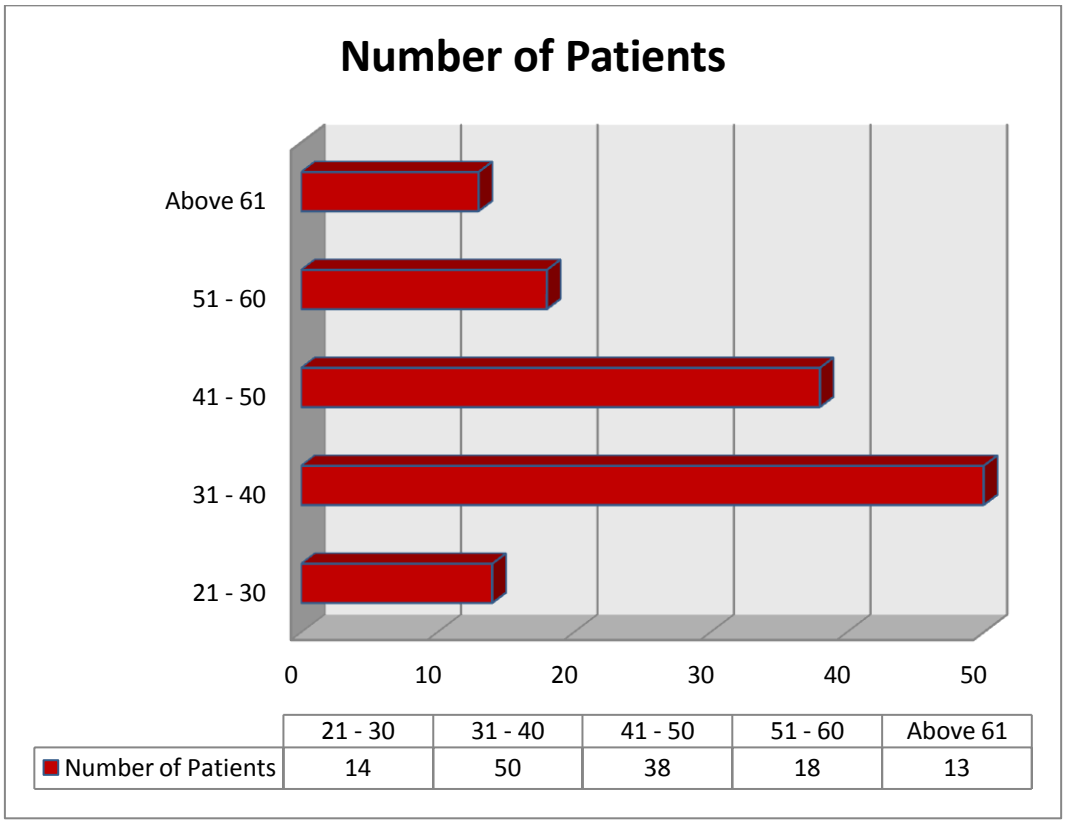
Two hundred & eighy-seven patients were screened for study inclusion (Graph 1). A total of 133 patients were enrolled & completed the study.

Graph 1 – Flow Chart for Study Sampling & Selection



In this study of 133 cases the maximum incidence of low back ache & sciatica was found in the 4th decade of life. The youngest patient in the study was 22 years where as the oldest was 84 years. The distribution was as tabulated below (Graph 2).

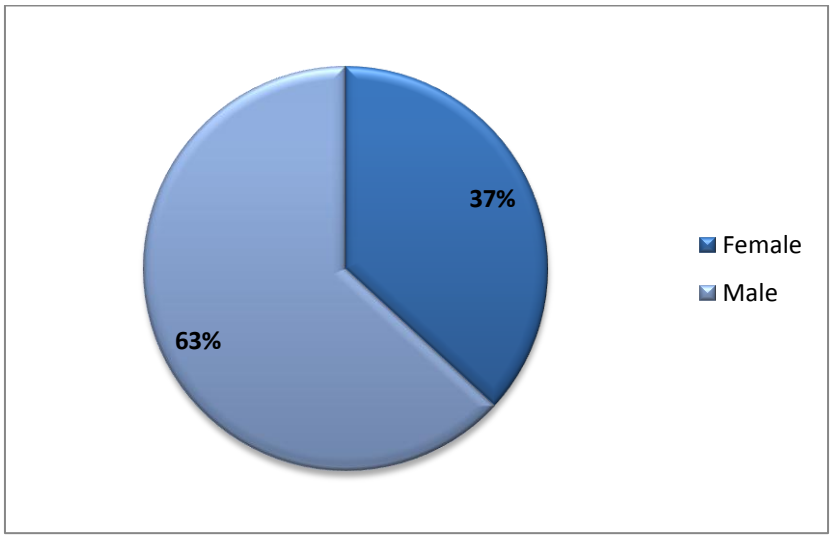
Graph 2 – Age Distribution



	21 - 30	31 - 40	41 - 50	51 - 60	Above 61
Number of Patients	14	50	38	18	13

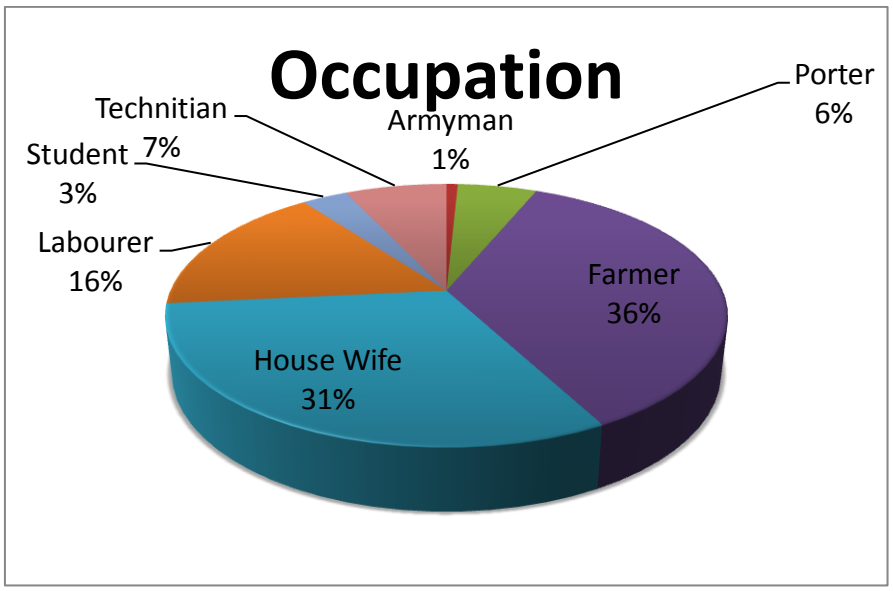
Overall the maximum incidence of back ache was seen in the 3rd & 4th decade. These results coincide with O Connel's results in a series of 500 cases as well as Jackson's series [59, 60].

Graph 3 – Sex Distribution



In this series of 133 cases, males accounted for 63 % of the cases while females formed a substantially lower 33 %. The distribution ratio is represented above (Graph 3). These results were in accordance with Robinson's series [61] where the number of males was substantially higher as well as O'Çonnels series [59]. The reason for the higher incidence in males in our study was linked to the nature of their occupation.

Graph 4 – Occupation Distribution



Occupation was a major contributory factor to the chronicity of low back pain with sciatica. Occupations like of farming & heavy weight lifting by labourers was deemed as the major cause for disc prolapse (Graph 4). Majority of the women suffering from disc disease were housewifes with a sedentary lifestyle. These results matched those of O'Çonells with large proportion of patients giving history of occupational strain [59].

Pain was the most common presenting symptom in the series with majority of the patients presenting with low back ache & unilateral or bilateral sciatica, while only about 20 % of the patients presented with isolated sciatica. This reproduced O'Connell's results (Table 3).

The Duration of symptoms was varied. The number of cases with a chronic history of > 2 years was seen in most of the cases. The average duration of the symptoms was around 31 months (Table 3).

A theory states that sciatica is much more common in the left lower limb than on the right owing to the incompleteness of protective mechanisms on this side but our results showed a more even distribution with nearly 30 % cases presenting with left or right sciatica respectively (Table 3).

A large number of patients (60 %) presented with stiffness of the back. This was not typical but was vague complaint, present more or less throughout the day only to reduce after lying down. This was probably attributed to a protective paraspinal muscle spasm (80 %), preventing jerky movements at the lumbar spine & was related to the pathology (Table 3).

Table 3 – Demographic & Clinical Data of Patients

Age (yrs)*	44.10 (11.96)
Sex #	
Men	84
Women	49
Duration of Symptoms before injection (mo)*	31.04 (21.82)
Signs & Symptoms (Visit 1) ^^	
Sciatica without low back ache	26 (19.55 %)
Left Sciatica with low backache	38 (28.57 %)
Right Sciatica with low back ache	40 (30.08 %)
Bilateral with low back ache	29 (21.81 %)
Stiffness	76 (57.14 %)
Sensory Parasthesia	105 (78.95 %)
Spinal Tenderness	104 (78.20 %)
Paraspinal Muscle Spasm	109 (81.95 %)
Previous Treatment ^^	
Rest/Analgesics	122 (91.73 %)
Traction	105 (78.95 %)
Orthosis (Lumbo-Sacral Belt)	106 (79.70 %)
Physiotherapy	106 (79.70 %)
Epidural Injections	17 (12.78%)
Number (Mean)# - 01	
Interval (Mean)# - 01 year	
Plane X - Ray Findings #	
Muscle Spasm	70 (52.63 %)
Reduced Disc Space	33 (24.81 %)
No Abnormality	30 (22.56 %)
Magnetic Resonance Imaging Findings #	
Disc Degeneration (IDD)	80 (60.15 %)
Disc Bulge	35 (26.31 %)
Disc Herniation (Protrusion)	18 (13.53 %)

*The values are given as the mean with the standard deviation in parentheses

The values are given as raw numbers

^^ The values are given as raw numbers with the percentages in parentheses

Other significant findings on physical examination included sensory parasthesia as well as a spinal tenderness (Seen in > 80 % cases). These findings were similar to those found by O'Connell in his series [59] (Table 3).

When history of previous treatment for the low backache & sciatica was elicited from the patient, majority (> 80 %) revealed that they had tried Analgesics, Rest, Traction, & Physiotherapy without significant relief. This was one of our inclusion criteria as well. As a result chronicity & unrelenting nature of problem was well established. With only 12 % of the patients giving a history of epidural injection earlier it was clear that the literature was very sparse on epidural injections (Table 3).

Plain X-rays were done in all of our patients. It was seen that only about 25 % of the patients showed reduced disc space. While majority of the findings including loss of lordosis, spondylotic changes did not guide us to the actual diagnosis although a paraspinal muscle spasm was suggestive of a lumbar strain commonly seen in disc prolapse (Table 3). Lumbosacral X-rays as a diagnostic tool have been used by Andre Hakelin & Friberg in their studies & have got slightly better results than our study [62].

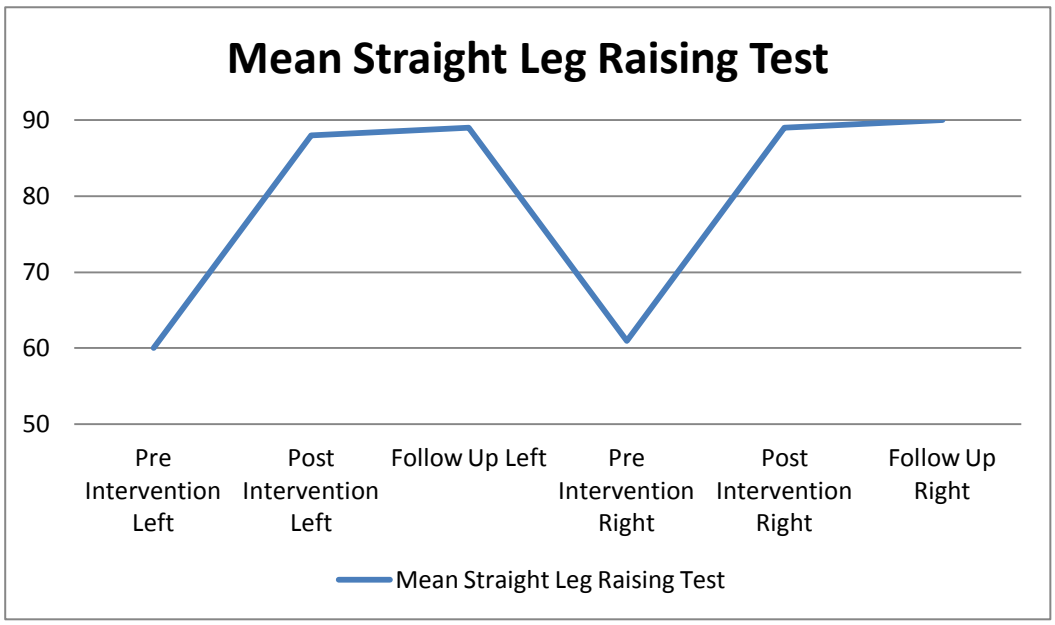
MRI being the gold standard for diagnosis of prolapsed intervertebral disc was done for all our cases. It showed that a large number of our patients (60 %) presented with Internal Disc Derangement. This ranged from Grade 3 to Grade 5. The rest of the cases were evenly distributed for Disc Bulge or Protrusion. We considered Disc Sequestration or Extrusion absolute indications for surgery & did not include any of these cases in our study. These results are supported by Abdi et al in their large scale epidural steroid injection study in 2009 [27, 28] (Table 3).

On the basis our literature search, we determined that our sample size was sufficient for this study using a desired power of 0.8 & error of 0.05 [28]. The primary

analysis of power was the pain score. Statistical analysis was done using the Student's paired *t*-test & unpaired t-test where appropriate with $P < 0.05$ required to reject the null hypothesis. The SPSS statistical software (Version 17) was used. Also the total amount incurred on the treatment of both groups was calculated & analysed.

Straight Leg Raising Test (SLRT) was measured in both the groups for all cases prior to the intervention, immediately following it as well as at every subsequent visit. It was not used to include the patient in the study as we felt that it was not diagnostic. A positive straight leg raising test was defined as < 60 Degrees. All cases had positive tests prior to the intervention. Post intervention & follow up results held us to determine the efficacy of the treatment. But it was an added outcome measure. The main outcomes were measured using the structured scores. Starting at visit 2 & continuing until visit 4, the straight leg raising test kept improving in both the groups. There was statistically significant improvement noted in the straight leg raising test. The Graphs below show the change in SLRT for each leg in the Groups (Graph 5).

Graph 5 – Straight Leg Raising Test (SLRT)



Pain Relief was the primary index for evaluating the outcome of the study. 3 weeks was deemed as Short term & 24 week deemed as Long term for the purpose of evaluation. It was observed that cases in both the groups had a large number of patients reporting complete pain relief even at the end of the deemed period of 6 months (Table 4). Complete relief was defined as VAS < 3 while Partial relief was defined as VAS < 5 for the purpose of the study. The results were also classified as per the patient's opinion as follows:

1. Good: The cases who obtained complete relief (residual symptoms being occasional twinges in the back), improvement being maintained over a year, & which in the opinion of the patient was marvelous, excellent or very good.
2. Fair: Cases with complete relief or partial relief, improvement being maintained up to 4 – 5 months (< 6 months) & opinion being expressed as fair.
3. None: No better or worse

It was seen that the percentage of cases with complete relief was fairly large in the both the groups but was higher in the group with caudal epidural steroid injection (90 % compared to 80 %). The same was seen for partial relief which was higher in Group A i.e. the caudal epidural steroid injection group (10 % vs. 16 %) at the end of 3 weeks (short term) compared to the lumbar epidural steroid injection group. Also the number of cases where VAS was zero was much higher in the caudal epidural steroid group compared to the lumbar epidural steroid group. At the end of 6 months, it was seen that 70 % of the patients in group A had persistence of complete relief compared to 50 % in group B which was significantly lower (long term).

Oswestry disability index scores (ODIS) were significantly improved within both the groups. The patients' mean scores kept decreasing (representing

improvement of their symptoms) at all follow- up re-evaluations. The mean Disability score was statistically significantly lower, when compared with the score prior to the injections in both the groups. The observed decrease in the mean ODI scores (a) between visit 1 & 2, & (b) between visit 1 & 4, were statistically significant with a p value < 0.01 for the 99 % confidence interval (Table 5).

Table 4 – Pain Relief Evaluation #

	Short Term		**Long Term**	
	Group A	**Group B**	**Group A**	**Group B**
Complete Relief Good	60 (88.24 %)	51 (78.46 %)	48 (70.59 %)	32 (49.24 %)
Partial Relief Fair	07 (10.29 %)	10 (15.39 %)	19 (27.94 %)	29 (44.62 %)
No Relief None	01 (01.47 %)	04 (06.15 %)	01 (01.47 %)	04 (06.15 %)

The values are given as raw numbers

Becks Depression Inventory Scores & Function evaluated by Visual Analogue scale (VAS) & Numerical pain intensity score improved within both the group (Table 5). This change was also statistically significant with p < 0.01, for the 99 % confidence interval.

A statistical analysis by the Independent samples T test or the Unpaired T test was done for the groups. The Oswestry disability index scores (ODIS) taken in both the groups were statistically comparable prior to the intervention. The results of the analysis for the short & long term follow up scores showed that there was a statistically significant change (p < 0.05 for the 95 % CI) in group A (Caudal Epidural Steroid Injection) compared to group B (Lumbar Epidural Steroid Injection). The

Beck's Depression Scores (BDIS) also showed a statistical significance for the results in group A (Caudal Epidural Steroid Injection) compared to group B (Lumbar Epidural Steroid Injection). Scores of the Visual Analogue Scale (VAS) & the Numerical Pain Intensity Score (NPIS) were not statistically significant.

Table 5 - Visual Analogue Pain Score, Disability, Depression & Numerical Pain Intensity Scores *

	Mean Score		**Standard Deviation**		**Significance within group 95 % CI P Value**	
	Group A	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
Visual Analogue Score Pre Injection	8.29	8.4	+/- 0.87	+/- 0.68	------	------
Visual Analogue Score Post Intervention	2.84	2.96	+/- 1.42	+/- 1.82	P < 0.01	P < 0.01
Visual Analogue Score Follow Up	3.46	3.62	+/- 0.91	+/- 1.74	P < 0.01	P < 0.01
ODIS Pre Intervention	36.34	36.68	+/- 1.93	+/- 2.12	------	------
ODIS Post Intervention	12.21	14.92	+/- 4.96	+/- 9.86	P < 0.01	P < 0.01
ODIS Follow Up	12.43	13.93	+/- 2.4	+/- 2.79	P < 0.01	P < 0.01
BDIS Pre Intervention	18.55	19.39	+/- 2.73	+/- 2.41	------	------
BDIS Follow Up	8.66	9.48	+/- 2.14	+/- 2.08	P < 0.01	P < 0.01
NPIS Pre Intervention	8.36	8.61	+/- 0.71	+/- 0.49	------	------
NPIS Follow Up	3.21	4.80	+/- 0.99	+/- 1.1	P < 0.01	P < 0.01

*Group A – Caudal Epidural Injection, Group B – Lumbar Epidural Injection

There were a few complications documented after the intervention in both the groups. They are listed in Table 6 below.

No patient reported any late complication(s) following the lumbar or caudal epidural steroid injection which have been documented in literature.

The difficulty asscociated with both the interventions was not a technical error because the surgeon performing the procedure was skilled in the same. The 23 cases in which there was a documented difficulty in Group A (Caudal epidural Steroid Injection) was attributed to negotiating the sacrococcygeal ligament due to its calcification & the abnormalities of the sacral hiatus. 5 of these patients were subjected to Pelvis X-rays after the procedure & it was seen that the Sacral hiatus was of the closed variety & the place was very narrow, hence the difficulty. But the difficulty experienced in the Lumbar epidural steroid group (B) was higher nearing almost 50 %. This was attributed to calcified & tight ligamentum flavum & the narrowed disc space as well as the arthritis of the posterior vertebral elements (Table 6).

Dural Puncture is a known complication of the Lumbar Epidural Injection & was seen in 25 % of the patients (Table 6). This also read to higher chances of infection & meningitis thus leading to a higher cost incurred & side effects on the antibiotics post operatively which were used in all these patients. This was completely avoided in cases with the Caudal route cause dural sac ends at the S 2 level much above the point of entry of the needle (Table 6).

Table 6 – Complications

Complication^^	Group A - Cases	Group B – Cases
Attempts required for steroid placement		
One	45 (66.18 %)	25 (38.46 %)
Two	15 (22.06 %)	21 (32.31 %)
Three	08 (11.77 %)	19 (23.23 %)
Difficulty in approach	23 (33.28 %)	40 (61.54 %)
Dural Puncture (CSF tap)	00 (00.00 %)	16 (24.62 %)
Headache	09 (13.24 %)	26 (40.00 %)
Hypotension (during the procedure)	19 (27.94 %)	06 (09.23 %)
Bleeding (at the time of injection)	02 (02.94 %)	02 (03.08 %)
Repeat Injections	07 (10.29 %)	12 (18.46 %)
Number Required (Mean)	01	01
Surgery Required	01	04

^^ The values are given as raw numbers with the percentages in parentheses

Headache was seen in 9 & 26 cases in Group A & B respectively. The complication in group B was accounted for by the dural puncture but in the caudal group it was attributed to the effect of xylocaine & transient increase in pain following the injection. The headache was managed with increase intake of fluids & analgesics only.

Hypotension was encountered during the procedure, was seen in nearly 30 % of the patients & was deemed as a complication of the needle placement in the caudal region leading to a vaso-vagal response. It was managed promptly by stopping the procedure & monitoring the patients vitals, following which a second attempt was made. If the hypotension repeated then the procedure was abandoned. In literarure

hypotension has been attributed to Xylocaine but we believed it was not so in our cases as the volume of the injectate was really small & sensitivity was done earlier.

The number of patients requiring repeat injections totalled up to seven in group A (Caudal epidural steroid injection) & six of them recovered completely following a second injection three weeks later, while in one patient there was no pain relief & the repeat MRI showed deterioration of the herniation following which surgical decompression was done (Table 6). The results of group B (Lumbar epidural steroid injection) were statistically worse compared to the former group as almost 12 patients recieved a second injection & 4 deteriorated & ultimately underwent an operative decompression (Table 6).

Twenty patients reported experiencing transient bilateral lower extremity numbness immediately after the injection. None reported any lower limb dysfunction in terms of loss of sensation & / or reduced motor power, or bladder & bowel dysfunction(s).

DISCUSSION

There has been a high morbidity asscociated with Chronic Low back ache & sciatica [63]. Its aetiology remains controversial [23, 64]. Degeneration, herniation, or by an inflammatory reaction could be responsible for lower backache & sciatica [65]. In 1901, Sicard introduced the injection of cocaine through the caudal route into the epidural space & ever since caudal epidural steroid injections are commonly used when dealing with chronic low back &/or radicular pain [66]. This approach to the epidural space is the earliest known technique for epidural steroid injection or blocks [67]. It however, did not gain universal recognition until 1925, when Viner popularized its use [67]. The first published report came from Evans who reported good results of Caudal epidural injections containing saline in patients of low back pain [66, 67]. The results were attributed to the physical displacement of the nerves & to the lysis of the neuronal adhesions caused by the saline injected [63]. In some patients with lumbar disk herniations, conservative pharmacologic &/or physical therapy may not provide adequate pain relief & alternative therapies such as lumbar epidural steroid injection may be helpful. An epidural injection can decrease inflammation in the epidural space as well as the pain in the affected nerve root [68, 69].

Since then numerous studies tried to evaluate the efficacy of epidural steroid injections in patients of chronic low back pain & sciatica. Extensive literature research revealed only a few randomized, double-blind prospective studies assessing the efficacy of the injection [69]. Dashfield *et al* [70], evaluated epidural injection & root blocks, but concluded that both treatments were effective & & were no significant differences. Breivik *et al* [71] evaluated caudal epidural injections with limited success. Bush & Hillier [71] evaluated the injections containing steroid & saline & concluded

that short term they very effective but long term potency was variable. Matthews *et al* [71] did a similar study with variable results but favoured steroid placement. Epidural Injection Therapy should form the next line of treatment before any surgery is contemplated.

We assessed the efficacy of Epidural steroid injections containing a preparation of local anaesthetic & steroid via the lumbar & the caudal route in a group of patients with chronic low back pain & sciatica.

Our results showed that 60 patients from group A (Caudal epidural steroid injection) responded well to the first injection itself while there was a large number seen in the group B (Lumbar epidural steroid injection) as well which totalled upto 51 cases. They had Complete / Good Results as laid down by our criteria. These reults were similar to the results obtained by Abdi et al in 2007 [27].

Recovery from symptoms was evaluated by the Oswestry disability index score primarily & was steadily observed from the first week following the injection. The main therapeutic result of the injection in both the groups appeared during the first week itself, when an immediate decrease in the mean Oswestry disability index score of the patients was noticed (Table 6). Our results support the existence of both short & long-term (up to 6 months) relief from symptoms for both the groups which is statistically significant. The results we obtained for the scores in group A were statistically more significant compared to group B. Similar statistically significant findings were also seen in Beck's Depression Scores. These results were significant & were similar to the efficacy of the injection demonstrated by Ackerman in 2007 [26].

Complications were seen in both the groups. The major compication of the Caudal epidural steroid injection we encountered was Hypotension was was attributed to a vasovagal effect rather than the solution used. It was promptly managed with fluids & did not lead to any further complication. Whereas the number of complications in Group B (Lumbar epidural steroid injection) were higher & more statistically significant. They included dural punture & headache which accounted for most of the complications. They were mangaed with antibiotics & fluids as well as head low position.

Steroid & xylocaine containing preparations seem to act faster & more efficiently, & improvement should probably be attributed to the preparation used in both the groups.

All our patients had Magnetic Resonance Imaging confirmation for the pathology. Although the efficacy of epidural steroid injections in the treatment of low back pain & sciatica has been clearly demonstrated, mechanisms of such benefits continue to lack scientific validity. It is hypothesized that corticosteroids exert their anti-inflammatory actions either by inhibiting the synthesis or release of inflammatory substances. Membrane stabilization, inhibition of neural peptide synthesis or action, of phospholipase A2 activity, & prolonged suppression of ongoing neuronal discharge are also possible actions exerted by corticosteroids. The administration of any solutions clears the locally concentrated chemical irritants [68, 70 & 71]. The weak solution of xylocaine effectively blocks finer sensory & autonomic fibres without affecting the motor fibres thereby aboloishing the effect of a noxious stimulus i.e. protruding disc, at all four possible levels;

a. Locally, by blocking the pain it should reduce the local inflammation & edema of the affected nerve root & the dura sheath. With the reduction of local inflammation & edema one would assume that incidence of a subsequent fibrosis should be correspondingly diminished.

b. By blocking the response at a segemental level it abolishes the reflex spasms.

c. By removing pain it also removes the psycosomatic sensation.

The indivisual response to the injection is variable both in distribution & duration & has been attributed to the following factors as given by Bromage in 1954.

a. Volume of the extra dural space

b. Size of the escape routes

c. Level of the injection

d. Volume of the injectate which in turn is determined by the route

e. Speed of the injection

f. Gravity

The advantages of our study should be considered the large number of patients enrolled, the use of validated questionnaires, as outcome measures instead of subjective criteria as well as the detailed statistical analysis.

The chance of puncturing the dura is much less with the caudal method. The lumbar method carries a risk of trauma to the nerve root during needle placement & also includes the risk of paraplegia if steroid is injected into a radicular artery that reinforces the anterior spinal artery & other complications. Furthermore, disk entry can be a complication of the lumbar route as well. Based on our results & higher

number of complications in the lumbar epidural steroid injection group we preffered to use the caudal epidural steroid injection group.

This study however, has also certain limitations. Epidural injections were performed without fluoroscopic guidance, which is the gold standard for accurate needle placement. Nonetheless, there are several methods that can be used when trying to achieve the proper placement & confirmation of the right position of the needle in the sacral hiatus without fluoroscopic guidance such as the whoosh test & aspiration as well palpating the right landmarks. Successful injections without fluoroscopic guidance may be achieved by these methods in 92% of cases. Stitz & Sommer support a high rate of success, as long as readily palpable anatomic landmarks are properly recognized.

Our article would have probably been better if we had used fluroscopic guidance but we believed that the other methods are adequete for accurate needle placement. We did not use a placebo - controlled group because the patients complained of severe pain & we did not feel that a placebo injection would be ethical in these circumstances.

The sucess of epidural injection therapy depends on careful seletion of patients & understanding that it is not a cure all simple remedy. Post injection Back extenstion excercises, with special caution to patients engaged in heavy manual activity will go a long way in determining the sucess of the injection.

Caudal epidural steroid injection is a simple, rapid, & easily performed day care procedure that can offer significant pain relief. It may even be considered as an alternative to operative procedures in patients not responding well to conservative treatment, of high operative risk, or when they refuse to be operated on. Following

injection, patients are discharged, thus avoiding long periods of hospitalization & bed rest. The combination of local anaesthetics, steroids & saline could be of an additional benefit leading to greater & faster relief during the first week & this improvement is maintained even 6 months later. We preffered using this route owing to the ease of the procedure, lesser number of complications, compared to Lumbar epidural steroid injection & our results also showed statistically significant results. More studies are certainly needed in order to better understand the potential action of steroids when treating patients with low back pain & sciatica with epidural steroid injections.

SUMMARY & CONCLUSION

Back ache & sciatica, together constitute a major problem to our society. A majority of the cases are due to an intervertebral disc prolapse.

Diagnosis is made mainly by clinical examination & by excluding other conditions through laboratory & other investigations. MRI is diagnostic.

A brief account of the relevant anatomy, physiology, pathology & neuropathology is given in the hope that it will make the understanding of the disc problems more comprehensible.

It is appreciated that all forms of conservative treatment of this problem are effective in some way or the other. Hence, attempt has been made to under or over value any of the treatment.

I have analysed 133 cases of chronic low back ache & sciatica due to disc protrusion which failed to respond to other forms of treatment & thus were subsequently treated with epidural injections.

The caudal route is preferred because of its relative freedom from complications, simplicity, ease with which it can be perfected by an average man & the treatment can be practised on an out-patient basis.

In this unselected series the follow up period varied from 6 months to a year & the results were classified as Good, Fair & None. They were as follows:

I. In this study of 133 cases the maximum incidence of low back ache & sciatica was found in the 4^{th} decade of life.

II. Males accounted for 63 % of the cases, the reason was linked to the nature of their occupation like of farming & heavy weight lifting (Table 6).

III. Straight Leg Raising Test (SLRT) showed a statistically significant improvement before & after the intervention & on follow up.

IV. It was seen that the percentage of cases with complete relief was fairly large in the both the groups but was higher in group A (90 % compared to 80 %).

V. The same was seen for partial relief which was higher in Group A (10 % vs. 16 %) at the end of 3 weeks (short term) compared to Group B.

VI. Also the number of cases where VAS was zero was much higher in the caudal epidural steroid group compared to the lumbar epidural steroid group. At the end of 6 months, it was seen that 70 % of the patients in group A had persistence of complete relief compared to 50 % in group B which was significantly lower (long term).

VII. Oswestry disability index scores were significantly improved in both groups.

VIII. Becks Depression Inventory Scores & Function evaluated by Visual Analogue scale & Numerical pain intensity score improved within both the group (Table 6). This change was also statistically significant with $p < 0.01$, for the 99 % confidence interval.

IX. A statistical analysis showed that the Oswestry disability index scores taken in both the groups were statistically comparable prior to the intervention. The results of the analysis for the short & long term follow up scores showed that there was a statistically significant change ($p < 0.05$ for the 95 % CI) in group A (Caudal Epidural Steroid Injection) compared to group B (Lumbar Epidural Steroid Injection).

X. The number of patients requiring repeat injections totalled up to seven in group A & six of them recovered completely following a second injection three weeks later, while in one patient required surgery. The results of group B were statistically worse compared to the former group as almost 12 patients recieved a second injection & 4 deteriorated & ultimately underwent an operative decompression.

XI. The difficulty asscociated with both the interventions, in Group A was attributed to negotiating the sacrococcygeal ligament due to its calcification & the abnormalities of the sacral hiatus or a lipoma. 5 of these patients were subjected to Pelvis X-rays after the procedure & it was seen that the Sacral hiatus was of the closed variety & the place was very narrow, hence the difficulty. But the difficulty experienced in the Group B was higher nearing almost 50 %. This was attributed to calcified & tight ligamentum flavum & the narrowed disc space as well as the arthritis of the posterior vertebral elements.

XII. Hypotension was encountered during the procedure, was seen in nearly 30 % of the patients & was deemed as a complication of the needle placement in the caudal region leading to a vaso-vagal response.

Thus Concluding,

1. On the basis of our results obtained plea is made to use epidural injections much more often than hitherto for a therapuetic purpose.
2. The route preffered for the epidural steroid injection is the Caudal route when compared to the lumbar route.

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ANNEXURE – I: INFORMED CONSENT

Mr./Mrs./Ms._____

You are invited to participate in this study

Title of the Study: "LUMBAR VERSUS CAUDAL EPIDURAL INJECTION OF STEROID IN CHRONIC LOW BACK ACHE & SCIATICA – A RANDOMIZED CONTROLLED TRIAL"

Principal Investigator: Dr. Aditya Khemka

Assessment & Investigations

They include a detailed history, physical examination (as per the tailored proforma, attached separately), scoring by the Oswestry disability index, Visual analogue scale, Numerical pain intensity score, Beck's depression inventory, radiography including lumbosacral spine x-rays, lumbosacral spine MRI's, pelvis x-rays (if difficulty during procedure), routine hemogram, urine examination.

Procedure of Epidural Steroid Injection:

1. **LUMBAR ROUTE:** The patient will be placed in the prone / lateral / sitting position on an operating table. Neurological Status & SLRT are assessed. Aseptically the skin area will be prepared with Spirit & Povidine-iodine several segments above & below the laminar interspace to be injected. The patient is draped in a sterile fashion. The target laminar interspace is identified. Using a needle, the skin over the target interspace on the side of the patient's pain with 1 to 2 ml of 1% preservative-free Xylocaine (Xylocard) without epinephrine is anaesthetized. Insert a 3½-inch, 22-gauge spinal needle vertically until contact is made with the upper edge of the inferior lamina at the target interspace. Anesthetize the lamina with 2 ml of 1% preservative-free Xylocaine (Xylocard) without epinephrine. Then anesthetize the soft tissue with 2 ml of 1% Xylocaine (Xylocard) as the spinal needle is withdrawn. Then insert a 3½-inch, 18-gauge Tuohy epidural needle & advance it vertically within the anesthetized soft tissue track until contact with the lamina has been made. "Walk off" the lamina with the Tuohy needle onto the ligamentum flavum. Remove the stylet from the Tuohy needle & attach a 10-ml syringe filled halfway with air & sterile saline to the Tuohy needle. Advance the Tuohy needle into the epidural space using the loss of resistance technique. A spot X-ray under C arm is taken, Whoosh Test, & Hanging Drop test are used to document accurate placement. Take a 10-ml syringe containing 2 ml Normal Saline, 2 ml of 40 mg/ml Triamcinolone Acetate & 2 ml of Preservative Free Lignocaine, a total of 6 ml. Neurological Status is reassessed along with SLRT.

2. **CAUDAL ROUTE:** The patient is placed in a prone / lateral position on the operating table. Neurological Status & SLRT are assessed. Aseptically, the skin area is prepared from the lumbosacral junction to the coccyx with Spirit & Povidine-iodine. The patient is draped in a sterile fashion. Try to identify by palpation the sacral hiatus, which is located between the two horns of the sacral cornu. The soft tissues & the dorsal aspect of the sacrum with 2 to 3 ml of 1% preservative-free Xylocaine (Xylocard) without epinephrine. Keep the C-arm positioned so that the fluoroscopic beam remains lateral. Insert a 3½-inch, 18-gauge Tuohy needle between the sacral cornu at about 45 degrees, with the bevel of the Tuohy needle facing ventrally until contact with the sacrum is made in the "Orthopaedic Triangle". The needle is then redirected more cephalad, horizontal & parallel to the table, advancing it into the sacral canal through the sacrococcygeal ligament & into the epidural space with a loss of resistance technique. A spot X-ray under C arm is taken, Whoosh Test, & Hanging Drop test are used to document accurate placement. Remove the stylet. Aspirate to check for blood. Inject slowly a 10-ml volume of Normal Saline followed 2 ml of 40 mg/ml of Triamcinolone Acetate & 2 ml of Preservative Free Lignocaine, & 10 ml of sterile normal saline. Pain is elicited during the injection. Neurological Status is reassessed along with SLRT.

ALTERNATIVES:

1. **VOLUNTRY PARTICIPATION/WITHDRAWAL:**

   Taking part in the study is voluntary. I may choose not to take part on this study. Or if I decide to take part I can later change my mind & withdraw from the study. My decision will not change the present or future healthcare or other services that I can receive. The study doctor or the sponsor may stop my participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. I f choose not to take part in the study I will receive the standard treatment for the participants with my condition.

2. **COSTS**

   There will be no extra cost incurred by the participant. The participant will have to pay for the investigations which are part of the existing treatment protocol for this ailment. Each procedure will cost around Rs 1,000/-

3. **COMPENSATION**

   As the subject voluntarily consents to be a part of the study, no compensation will be given. The patient can withdraw from the study anytime or can be removed from this study.

4. CONFIDENTIALITY

All information collected about the subject during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify the subject in this research record. Information from this study may be published but the subject's identity will be confidential in any publication.

QUESTION:

If any enquiries in the future or in case of research related injury illness, you may contact following person.

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CONSENT FORM

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, & had all my questions answered. I will be given a copy of this consent form.

Signature of the Participant or legally authorized representative.

Participant's Name : …….

Signature : …….

Name of the legally authorized representative : …….

Signature : …….

Witness's Name : …….

Signature : …….

Investigators name & Signature : …….

Date & Place : …….

ANNEXURE – II: PROFORMA

Case No:

NAME: **AGE/SEX:**

IP No: **OCCUPATION:**

ADDRESS:

HISTORY OF PRESENT ILLNESS:

BACK PAIN:

Character:

Location: Dorso-Lumbar ☐ **Mid-Lumbar** ☐ **Low Back** ☐

Duration: Days ☐ **Weeks** ☐ **Months** ☐ **Years** ☐

No of Attacks ☐ **Last Attack** ☐

Radiation: RIGHT\LEFT\BILATERAL

Aggravated By:

1 – Yes.

2 - No.

Cough, Sneezing & Straining: ☐

Stooping & Bending ☐

Lifting ☐

Recombining ☐

Night Pain ☐

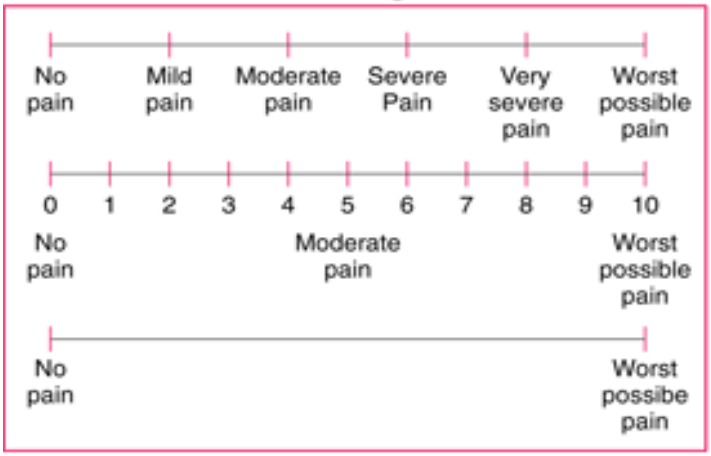
Stiffness ☐

Cause of Pain: Injury **1. Yes** ☐

2. No

If Yes Details:

Visual Analog Scale



Word Descriptor Scale

0 = No pain
1 = Mild pain
2 = Distressing pain
3 = Severe pain
4 = Horrible pain
5 = Excruciating pain

Graphic Scale

Verbal Scale

"On a scale of 0 to 10, with 0 meaning no pain and 10 meaning the worst pain you can imagine, how much pain are you having now?"

Functional Pain Scale

0 = No pain
1 = Tolerable and pain does not prevent any activities
2 = Tolerable and pain prevents some activities
3 = Intolerable and pain does not prevent use of telephone, TV viewing, or reading.
4 = Intolerable and pain prevents use of telephone, TV viewing, or reading.
5 = Intolerable and pain prevents verbal communication

SENSORY SYMPTOMS: **1. Yes** ☐

2. No

If YES Details:

MOTOR SYMPTOMS: **1. Yes** ☐

2. No

If YES Details:

PAST HISTORY: History of Previous Surgery: 1. Yes ☐

2. No **If YES Details:**

Diabetes, Hypertension, Asthma, Epilepsy: **1. Yes** ☐

2. No

If YES Details:

FAMILY HISTORY:

PREVIOUS TREATMENT:

1 – Yes. ☐

2 - No.

Bed Rest ☐

Analgesics ☐

Traction ☐

Lumbosacral Belt ☐

Physiotherapy (Exercises / Heat / Shortwave) ☐

Epidural Injections ☐

Number: ☐ **Interval b/w Injections:** ☐

Result: **Cured** ☐ **Improved** ☐ **Not Better** ☐

Period of Remission: ☐

GENERAL EXAMINATION: **a) Pulse Rate:** ☐

b) Blood Pressure:

c) Respiratory Rate:

1 – Yes. ☐

2 - No.

Pallor: ☐

Cyanosis ☐

Icterus ☐

Clubbing ☐

Pedal edema ☐

Lymphadenopathy ☐

LOCAL EXAMINATION:

SCOLIOSIS: **1. Yes** ☐

2. No

If Yes details:

ANY OTHER SWELLING/DEFORMITY: **1. Yes** ☐

2. No

If Yes details:

SPINAL TENDERNESS: **1. Yes** ☐

2. No

If Yes details:

PARASPINAL TENDERNESS: **1. Yes** ☐

2. No

If Yes details:

PARASPINAL MUSCLE SPASM: **1. Yes** ☐
2. No
If Yes details:

SPINAL RANGE OF MOTION: **Flexion:** ☐ **Extension:** ☐
Lateral Bending: ☐
STRAIGHT LEG RAISING ACTIVE: Right: ☐
Left: ☐
PASSIVE: Right: ☐
Left: ☐
SACROILIAC JOINT TESTS: FABER:
PUMP HANDLE:
GANSLEYS:
NEUROLOGICAL EXAMINATION: MOTOR: Left: ☐ **Right:** ☐
SENSORY: Left: ☐ **Right:** ☐
REFLEXES: Left: ☐ **Right:** ☐
MODIFIED OSWESTRY SCORE: ☐
BECK'S DEPRESSION INVENTORY SCORE: ☐
NUMERIC PAIN INTENSITY SCORE: ☐
SYSTEMIC EXAMINATION:

X-RAY Findings:
MRI Findings:
ROUTE OF EPIDURAL STEROID INJECTION:
DATE OF EPIDURAL STEROID INJECTION:
COMPLICATIONS: 1.Yes 2. No ☐
Difficulty of reaching Epidural Space ☐
Number of Attempts to reach Space ☐
Puncture of Dura Mater ☐
Bleeding ☐
Headache ☐
Convulsions ☐
Neurological ☐
Infection ☐

Results Immediately after the Intervention:

1. **Score:** **VAS** ☐ **ODI** ☐
2. **SLRT:** **Left** ☐ **Right** ☐

Signature of Examiner **Signature of Guide**

- **Repeat Injection: Due to lack of relief**
  1. **YES** ☐
  2. **NO**

  If Yes details:

  Number (Max 2 Then Surgery)
- **AT FOLLOW UP 1st (3 weeks / 3 months / Final 6 monthly Follow up):**

  Grade Of Subjective Relief Of Pain: Visual Analog Score

  SLR: Left: ☐ **Right:** ☐

  Neurological Status Deterioration: 1. Yes ☐

  2. No (Unchanged)

  If Yes Details:

  Modified Oswestry Score: ☐

  Beck's Depression Inventory Score: ☐

  Numerical Pain Intensity Score: ☐

  Improvement in Spinal Motion: 1. Yes ☐

  2. No

  If Yes Details:
- **AT FOLLOW UP 2nd (3 weeks / 3 months / Final 6 monthly Follow up):**

  Grade Of Subjective Relief Of Pain: Visual Analog Score ☐

  SLR: Left: ☐ **Right:** ☐

  Neurological Status Deterioration: 1. Yes ☐

  2. No (Unchanged)

  If Yes Details:

  Modified Oswestry Score: ☐

  Beck's Depression Inventory Score: ☐

  Numerical Pain Intensity Score: ☐

  Improvement in Spinal Motion: 1. Yes ☐

  2. No

  If Yes Details:
- **AT FOLLOW UP Final (3 weeks / 3 months / Final 6 monthly Follow up):**

  Grade Of Subjective Relief Of Pain: Visual Analog Score ☐

  SLR: Left: ☐ **Right:** ☐

  Neurological Status Deterioration: 1. Yes ☐

  2. No (Unchanged)

  If Yes Details:

Modified Oswestry Score:

Beck's Depression Inventory Score: ☐

Numerical Pain Intensity Score: ☐

Improvement in Spinal Motion: **1. Yes** ☐

2. No

If Yes Details:

Signature of Examiner **Signature of Guide**

Oswestry Low Back Pain Disability Index

Fairbank (1980) & his colleagues originated the Oswestry index. A more recent version of the index by Fritz & Irrgang makes certain items easier for patients to answer & for clinicians to score.

The questionnaire contains six statements (denoted by the letters A through F) in each of ten sections. The sections concern impairments like pain, & abilities like personal care, lifting, reading, driving, & recreation. For each section, subjects choose the statement that best describes their status.

The chosen statements receive scores: statement A=0; statement B=1; C=2; D=3; E=4; F=5. Total scores can range from 0 (highest level of function) to 50 (lowest level of function). To accommodate patients who do not respond to every section, clinicians can calculate a "percentage of disability" on the basis of the total possible points. Fairbank & his colleagues (1980) interpret "percentage of disability" scores in this manner:

- 0% to 20% Minimal Disability
- 20% to 40% Moderate Disability
- 40% to 60% Severe Disability
- 60% to 80% Crippled
- 80% to 100% Bed bound (or exaggerating symptoms)

QUESTIONNAIRE

SECTION 1-- Pain Intensity

A. The pain comes & goes & is very mild.
B. The pain is mild & does not vary much.
C. The pain comes & goes & is moderate.
D. The pain is moderate & does not vary much.
E. The pain comes & goes & is severe.
F. The pain is severe & does not vary much.

SECTION 2-- Personal Care

A. I would not have to change my way of washing or dressing in order to avoid pain.
B. I do not normally change my way of dressing even though it causes some pain.
C. Washing & dressing increases the pain, but I manage not to change my way.
D. Washing & dressing increases the pain & I find it necessary to change my way.
E. Because of the pain, I am unable to do some washing & dressing without help.
F. Because of the pain, I am unable to do any washing or dressing without help.

SECTION 3-- Lifting

A. I can lift heavy weights without extra pain.
B. I can lift heavy weights, but it causes extra pain.
C. Pain prevents me from lifting heavy weights off the floor.
D. Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, e.g., on a table.
E. Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.
F. I can only lift very light weights, at the most.

SECTION 4-- Walking

A. Pain does not prevent me from walking any distance.
B. Pain prevents me from walking more than one mile.
C. Pain prevents me from walking more than 1/2 mile.
D. Pain prevents me from walking more than 1/4 mile.
E. I can only walk while using a cane or on crutches.
F. I am in bed most of the time & have to crawl to the toilet.

SECTION 5—Sifting

A.I can sit in any chair as long as I like without pain.
B. I can only sit in my favorite chair as long as I like.
C. Pain prevents me from sitting more than one hour.
D. Pain prevents me from sitting more than 1/2 hour.
E. Pain prevents me from sitting more than ten minutes.
F. Pain prevents me from sitting at all.

SECTION 6 -- Standing

A. I can stand as long as I want without pain.
B. I have some pain while standing, but it does not increase with time.
C. I cannot stand for longer than one hour without increasing pain.
D. I cannot stand for longer than 1/2 hour without increasing pain.
E. I cannot stand for longer than ten minutes without increasing pain.
F. I avoid standing, because it increases the pain straight away.

SECTION 7 -- Sleeping

A. I get no pain in bed.
B. I get pain in bed, but it does not prevent me from sleeping well.
C. Because of pain, my normal night's sleep is reduced by less than one-quarter
D. Because of pain, my normal night's sleep is reduced by less than one-half.
E. Because of pain, my normal night's sleep is reduced by less than three-quarters.
F. Pain prevents me from sleeping at all.

SECTION 8--Social Life

A. My social life is normal & gives me no pain.
B. My social life is normal, but increases the degree of my pain.
C. Pain has no significant effect on my social life apart from limiting my more energetic interests, e.g., dancing, etc.
D. Pain has restricted my social life & I do not go out very often.
E. Pain has restricted my social life to my home.
F. I have hardly any social life because of the pain.

SECTION 9-- Traveling

A. I get no pain while traveling.
B. I get some pain while traveling, but none of my usual forms of travel make it any worse.
C. I get extra pain while traveling, but it does not compel me to seek alternative forms of travel.
D. I get extra pain while traveling which compels me to seek alternative forms of travel.
E. Pain restricts all forms of travel.
F. Pain prevents all forms of travel except that done lying down.

SECTION 10-- Changing Degree of Pain

A. My pain is rapidly getting better.
B. My pain fluctuates, but overall is definitely getting better.
C. My pain seems to be getting better, but improvement is slow at present.
D. My pain is neither getting better nor worse.
E. My pain is gradually worsening.
F. My pain is rapidly worsening.

Beck Depression Inventory

Twenty-one question survey

Answers: Scored 0 to 3 Scale

Minimal: 0

Severe: 3

Questions

1. Sadness
2. Hopelessness
3. Past failure
4. Anhedonia
5. Guilt
6. Punishment
7. Self-dislike
8. Self-blame
9. Suicidal thoughts
10. Crying
11. Agitation
12. Loss of interest in activities
13. Indecisiveness
14. Worthlessness
15. Loss of energy
16. Insomnia
17. Irritability
18. Decreased appetite
19. Diminished concentration
20. Fatigue
21. Lack of interest in sex

Interpretation

Score <15: Mild Depression

Score 15-30: Moderate Depression

Score >30: Severe Depression

ANNEXURE – III: RANDOMIZATION LIST

0001: Lumbar Route 0026: Lumbar Route 0051: Lumbar Route 0076: Caudal Route 0101: Caudal Route 0126: Lumbar Route

0002: Lumbar Route 0027: Caudal Route 0052: Caudal Route 0077: Caudal Route 0102: Lumbar Route 0127: Lumbar Route

0003: Caudal Route 0028: Caudal Route 0053: Caudal Route 0078: Caudal Route 0103: Caudal Route 0128: Lumbar Route

0004: Lumbar Route 0029: Lumbar Route 0054: Lumbar Route 0079: Caudal Route 0104: Lumbar Route 0129: Caudal Route

0005: Lumbar Route 0030: Lumbar Route 0055: Lumbar Route 0080: Lumbar Route 0105: Caudal Route 0130: Caudal Route

0006: Lumbar Route 0031: Lumbar Route 0056: Lumbar Route 0081: Lumbar Route 0106: Caudal Route 0131: Caudal Route

0007: Lumbar Route 0032: Lumbar Route 0057: Caudal Route 0082: Caudal Route 0107: Caudal Route 0132: Caudal Route

0008: Lumbar Route 0033: Lumbar Route 0058: Caudal Route 0083: Lumbar Route 0108: Caudal Route 0133: Caudal Route

0009: Lumbar Route 0034: Caudal Route 0059: Caudal Route 0084: Lumbar Route 0109: Caudal Route 0134: Caudal Route

0010: Lumbar Route 0035: Caudal Route 0060: Caudal Route 0085: Lumbar Route 0110: Lumbar Route 0135: Lumbar Route

0011: Lumbar Route 0036: Lumbar Route 0061: Caudal Route 0086: Caudal Route 0111: Lumbar Route 0136: Lumbar Route

0012: Lumbar Route 0037: Caudal Route 0062: Lumbar Route 0087: Lumbar Route 0112: Caudal Route 0137: Lumbar Route

0013: Caudal Route 0038: Lumbar Route 0063: Lumbar Route 0088: Caudal Route 0113: Caudal Route 0138: Lumbar Route

0014: Caudal Route 0039: Caudal Route 0064: Lumbar Route 0089: Caudal Route 0114: Lumbar Route 0139: Lumbar Route

0015: Caudal Route 0040: Lumbar Route 0065: Lumbar Route 0090: Lumbar Route 0115: Caudal Route 0140: Caudal Route

0016: Caudal Route 0041: Lumbar Route 0066: Caudal Route 0091: Caudal Route 0116: Caudal Route 0141: Lumbar Route

0017: Caudal Route 0042: Caudal Route 0067: Caudal Route 0092: Caudal Route 0117: Caudal Route 0142: Lumbar Route

0018: Caudal Route 0043: Lumbar route 0068: Lumbar route 0093: Lumbar Route 0118: Lumbar Route 0143: Lumbar Route

0019: Lumbar Route 0044: Caudal Route 0069: Caudal Route 0094: Caudal Route 0119: Lumbar Route 0144: Caudal Route

0020: Caudal Route 0045: Lumbar Route 0070: Caudal Route 0095: Lumbar Route 0120: Lumbar Route 0145: Caudal Route

0021: Caudal Route 0046: Lumbar Route 0071: Lumbar Route 0096: Caudal Route 0121: Lumbar Route 0146: Caudal Route

0022: Caudal Route 0047: Caudal Route 0072: Caudal Route 0097: Lumbar Route 0122: Caudal Route 0147: Caudal Route

0023: Lumbar Route 0048: Caudal Route 0073: Caudal Route 0098: Caudal Route 0123: Lumbar Route 0148: Caudal Route

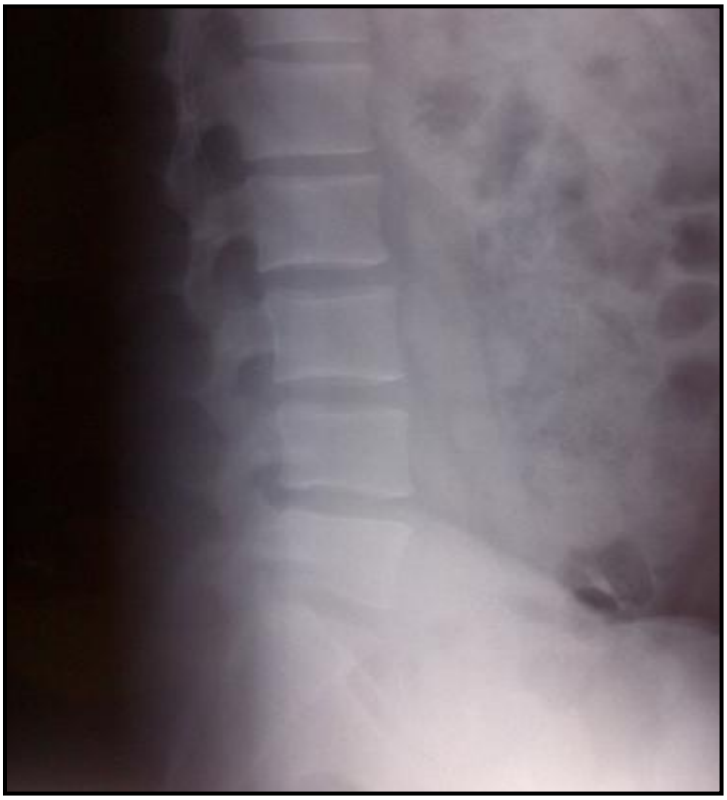
0024: Lumbar Route 0049: Lumbar Route 0074: Lumbar route 0099: Lumbar route 0124: Caudal Route 0149: Lumbar Route

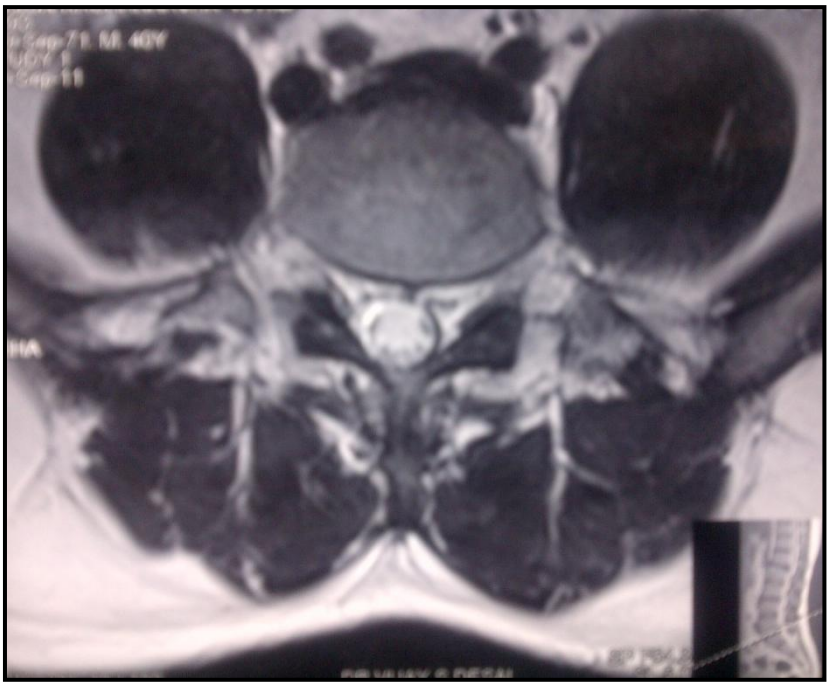
0025: Caudal Route 0050: Lumbar Route 0075: Caudal Route 0100: Lumbar Route 0125: Caudal Route 0150: Lumbar Route

ANNEXURE – IV: PHOTOGRAPHS

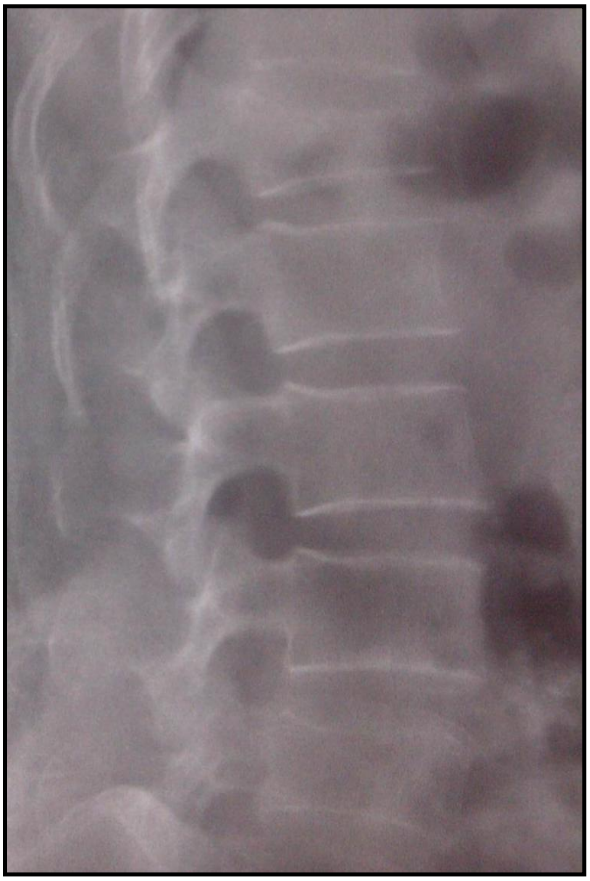
FEW TYPICAL CASES

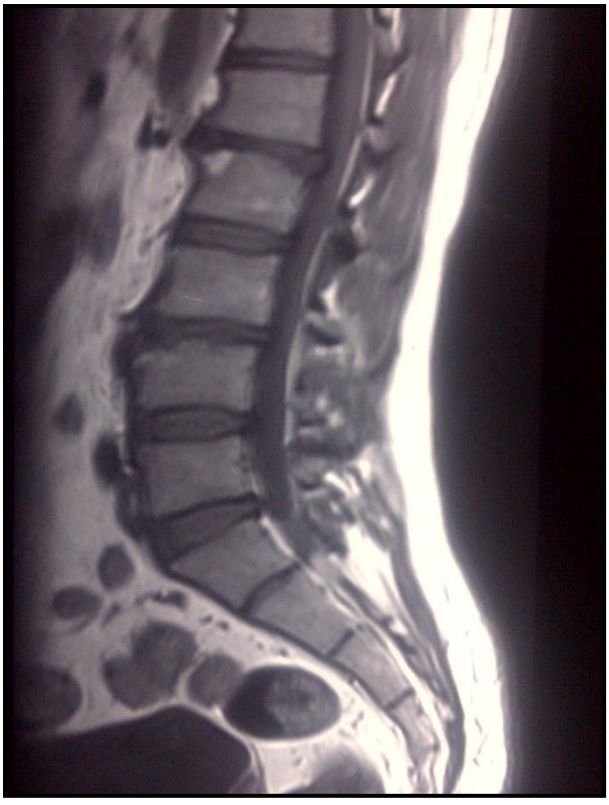
Case 1 – Normal Radiographs, with IDD Type 2 on MRI, with Severe L 5 Radiculopathy



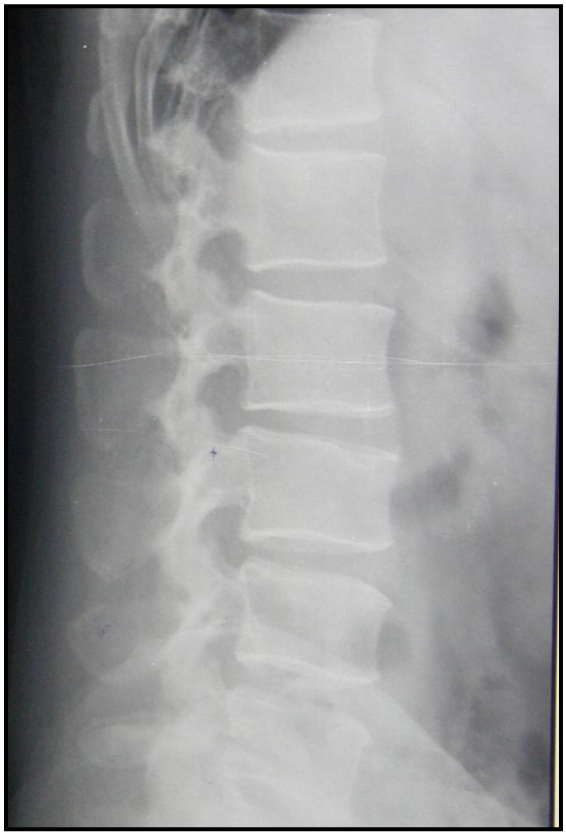


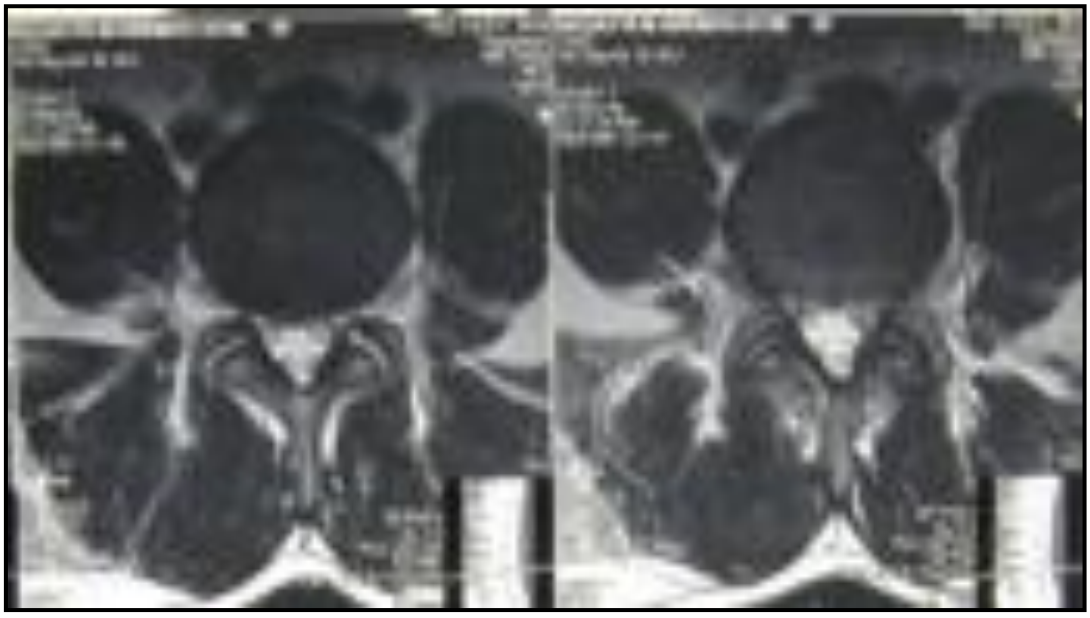
Case 2 – Reduced Disc Space L 4 - L 5, with Multiple Disc Bulges on MRI, with Bilateral Radiculopathy along the S 1 Root



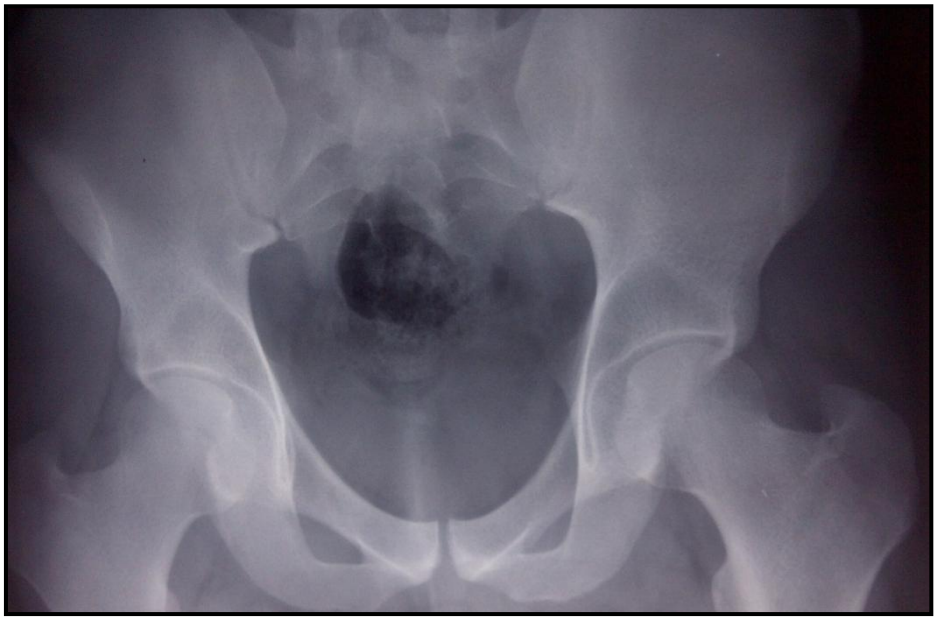


Case 3 – Reduced Disc Space L 3 - L 4, with Central + Bilateral Para-central Disc Bulge on MRI, with only Left sided sciatica

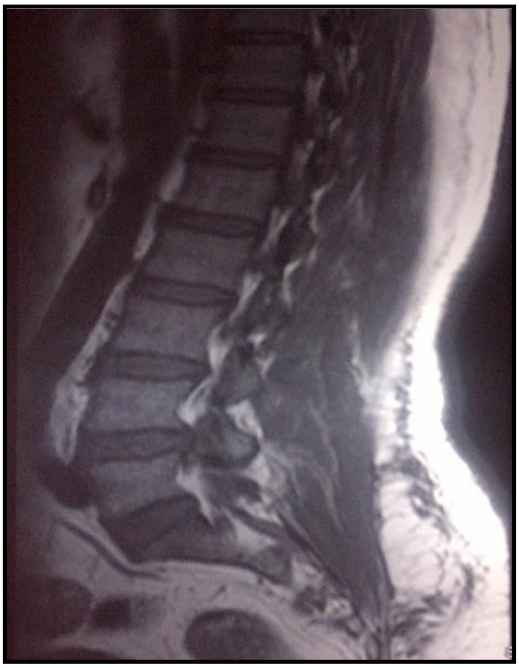


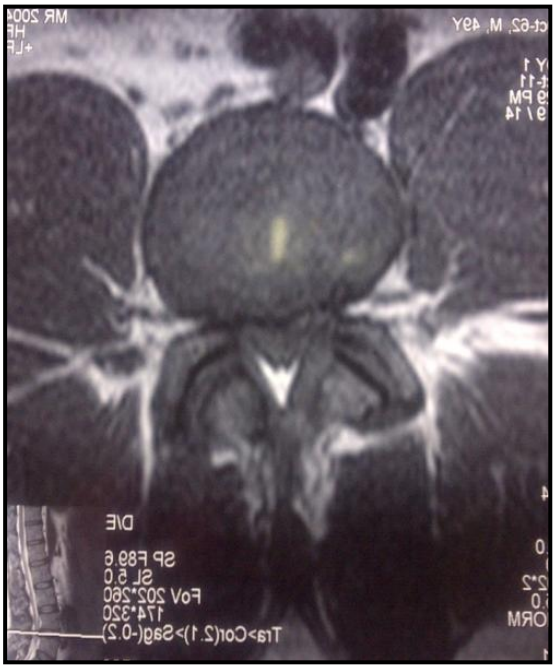


Case 4 – Normal Pelvis X-Ray - V shaped Sacral Hiatus, Difficulty in the Procedure



Case 5 – Typical L 4 - L 5 Para-central Disc Bulge on MRI, with Low Back Ache





ANNEXURE-V: MASTER CHART

Lumbar Epidural Steroid Patient List

Name	A	S	Occupation	I.P. No.	Primary Symptoms						Previous Treatment					Spinal Signs												MOS	BDIS	NPIS	MRI Findings	Lumbar Route						Short Term Results				Rep Inj		Surg	Follow Up						
					Sciatica	Dur	VAS	SS/s	MS/s	R/A	Tr	Ph	Epidural Injections			Tenderness		Spasm	Spinal ROM			SLRT		CNS		O/e						Complications						VAS	ODIS	SLRT		Y/N	No		Dur	VAS	ODIS	BDIS	NPIS	SLRT	
													P/NP	No	Interval	P/A	Location		F	E	Lat F	Lt	Rt	M	S	R					D	At	DP	H	Hy			L	R										L	R
Vimal Katawale	40	M	Farmer	340900	None	24mo	8	2	2	1	1	1	1	3	1yr	1	L 4,5	1	105	30	30	70	70	N	N	N	40	18	9	L 5 - S 1 Bulge	1	2	2	2	2	3	10	90	90	1	1	None	6mths	4	16	8	4	90	90
Patrevva Hosali	40	F	Housewife	345678	Right	48mo	8	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	90	40	N	N	N	39	20	9	L 4,5 Bulge	1	4	2	2	2	4	16	90	90	2	None	1	None	Sx	Sx	Sx	Sx	Sx	Sx
Nirmala Cloudapannavar	46	F	Housewife	346678	Right	6mo	9	1	2	1	2	1	2	None	None	1	L 4,5 - S 1	1	100	25	30	80	60	N	N	N	38	22	9	L 5 - S 1 Bulge	1	2	2	1	2	2	8	90	90	2	None	None	6mths	3	10	6	2	90	90
Parshuram	40	M	Labourer	342046	Left	48mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	20	30	60	90	N	N	N	40	24	8	L 4,5 Bulge	1	2	2	2	2	1	17	90	90	2	None	None	6mths	4	21	10	4	90	90
Ranaki F Bhimappa	60	M	Cooli	342514	None	3mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	20	20	N	N	N	39	18	9	L 4,5 Bulge	2	2	2	2	2	16	90	90	2	None	None	6mths	4	16	10	4	90	90	2
Ahilya B Kesnukar	57	F	Farmer	349154	None	48mo	8	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	30	20	60	60	N	N	N	34	14	8	L 4,5 Bulge	1	3	2	1	2	2	10	90	90	2	None	None	6mths	3	10	8	3	90	90
Virupakshi Raje	50	M	Farmer	337448	B/l	6mo	8	2	2	1	1	1	2	None	None	2	None	1	90	30	30	60	50	N	N	N	37	20	9	L 4,5 Bulge	2	3	1	1	2	3	13	90	90	2	None	None	3mths	3	14	10	3	90	90
Magdum Shankar	45	M	Farmer	388814	Right	6mo	10	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	40	N	N	N	38	21	9	L 5 - S 1 Bulge	2	1	2	2	2	2	12	90	90	2	None	None	6mths	3	12	10	3	90	90
Solomon Kodamala	40	M	Labourer	345679	B/l	84mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	60	60	N	N	N	38	18	9	L 5 - S 1 Bulge	2	1	2	2	2	3	16	90	90	2	None	None	6mths	3	14	10	3	90	90
Adiveppa Manikeri	46	M	Farmer	353326	None	8mo	8	1	2	2	2	2	2	None	None	2	None	2	105	30	30	90	50	N	N	N	38	18	9	L 4,5 Bulge	1	3	2	1	2	3	14	90	90	2	None	None	6mths	4	14	12	4	90	90
Anirudh Patil	23	M	Clerk	343245	Right	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	2	100	30	30	90	40	N	N	N	36	18	8	L 5 - S 1 Bulge	1	2	2	1	2	3	14	90	90	2	None	None	6mths	4	16	10	4	90	80
Parvati Kalganavar	48	F	Housewife	342345	None	24mo	7	1	2	1	2	2	2	None	None	1	L 4,5	1	105	30	30	70	70	N	N	N	34	16	8	L 4,5 Bulge	2	1	2	2	1	2	10	90	90	2	None	None	9mths	4	12	10	4	90	90
Ratnawwa Patil	40	F	Housewife	333456	B/l	12mo	9	1	2	1	1	1	2	None	None	1	L 3,4	2	100	30	30	40	40	N	N	N	34	18	8	L 3,4,5 Bulge	2	3	2	2	2	3	12	90	90	2	None	None	12mths	4	14	10	4	90	90
Mahadev Kohli	35	M	Labourer	354237	Left	3mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	90	N	N	N	38	18	9	L 3,4,5 Bulge	1	3	2	1	2	3	20	90	90	1	1	None	12mths	4	16	8	4	90	90
Revappa Chandti	45	M	Labourer	356206	Left	6mo	8	1	2	1	1	2	2	None	None	1	L 4,5	1	100	30	30	90	90	N	N	N	36	20	9	L 4,5 Bulge	1	3	1	1	2	3	14	90	90	1	1	None	6mths	3	16	12	3	90	90
Surekha Patil	38	F	Housewife	336589	Right	24mo	7	1	2	1	2	1	2	None	None	1	L 4,5 - S 1	1	70	10	10	90	60	N	N	N	38	20	9	L 5 - S 1 Bulge	1	2	1	1	2	3	16	90	90	1	1	None	6mths	4	14	11	4	90	90
Manjula Sunkod	36	F	Housewife	356138	Left	6mo	9	1	2	1	2	2	2	None	None	1	L 4,5	1	100	30	30	40	90	N	N	N	36	20	8	L 4,5 Bulge	2	1	2	2	2	4	18	90	90	2	None	1	None	Sx	Sx	Sx	Sx	S	Sx
Fakirappa Hoovanavar	73	M	Clerk	334567	Left	12mo	9	1	2	1	1	1	1	1	6mths	1	L 5 - S 1	1	105	30	30	40	70	N	N	N	38	18	9	L 3,4,5 Bulge	2	1	1	1	2	5	14	90	90	2	None	None	6mths	4	14	12	4	90	90
Jose Dias	45	M	Labourer	340244	Left	36mo	8	1	2	1	1	1	1	1	2yr	1	L 4,5	1	100	30	30	60	90	N	N	N	38	20	9	L 4,5 Bulge	1	1	2	1	2	3	14	90	90	1	1	None	6mths	4	16	10	4	80	80
Shivagouda Patil	40	M	Farmer	334521	B/l	24mo	8	1	2	1	1	1	2	None	None	2	None	1	105	30	30	40	40	N	N	N	33	22	8	L 3,4,5 Bulge	1	2	2	2	1	3	16	90	90	2	None	None	6mths	4	16	12	4	90	90
Kasturi Mallapa	30	F	Housewife	363676	None	24mo	8	1	2	1	1	1	1	1	2yr	1	L 4,5	1	105	30	30	30	90	N	N	N	38	20	9	L 4,5 Bulge	2	2	2	1	2	3	16	90	90	1	1	None	6mths	4	16	12	4	90	90
Annapa Mallanavar	38	M	Labourer	358080	Right	6mo	9	1	2	1	2	1	2	None	None	1	L 4,5 - S 1	1	100	25	30	80	60	N	N	N	38	22	9	L 5 - S 1 Bulge	1	2	2	1	2	2	8	90	90	2	None	None	6mths	3	10	6	2	90	90
Savitri Yellangoudar	40	F	Housewife	358184	Left	48mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	20	30	60	90	N	N	N	40	24	8	L 4,5 Bulge	1	2	2	2	2	1	17	90	90	2	None	None	6mths	4	21	10	4	90	90
Roopa Fadte	50	F	Labourer	362341	None	3mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	20	20	N	N	N	39	18	9	L 4,5 Bulge	2	2	2	2	2	3	16	90	90	2	None	None	6mths	4	16	10	4	90	90
Gangawwa Hugli	57	F	Housewife	358639	None	48mo	8	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	30	20	60	60	N	N	N	34	14	8	L 5 - S 1 Bulge	1	3	2	1	2	2	10	90	90	2	None	None	6mths	3	10	8	3	90	90
Pandurang Pawar	69	M	Farmer	371626	B/l	6mo	8	2	2	1	1	1	2	None	None	2	None	1	90	30	30	60	50	N	N	N	37	20	9	L 4,5 Bulge	2	3	1	1	2	3	13	90	90	2	None	None	3mths	3	14	10	3	90	90
Parvetteva Angadi	55	F	Housewife	368471	Right	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	2	100	30	30	90	40	N	N	N	36	18	8	L 5 - S 1 Bulge	1	2	2	1	2	3	14	90	90	2	None	None	6mths	4	16	10	4	90	80
Revappa Hoskatti	45	M	Farmer	372649	None	24mo	7	1	2	1	2	2	2	None	None	1	L 4,5	1	105	30	30	70	70	N	N	N	34	16	8	L 4,5 Bulge	2	1	2	2	1	2	10	90	90	2	None	None	9mths	4	12	10	4	90	90
Sardar Sanadi Madar	38	M	Police	374412	B/l	12mo	9	1	2	1	1	1	2	None	None	1	L 3,4	2	100	30	30	40	40	N	N	N	34	18	8	L 3,4,5 Bulge	2	3	2	2	2	3	12	90	90	2	None	None	12mths	4	14	10	4	90	90
Mallapa Naik	70	M	Farmer	377953	Left	3mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	90	N	N	N	38	18	9	L 3,4,5 Bulge	1	3	2	1	2	3	20	90	90	1	1	None	12mths	4	16	8	4	90	90
Neelawwa Somappa	35	F	Housewife	378563	Left	6mo	8	1	2	1	1	2	2	None	None	1	L 4,5	1	100	30	30	90	90	N	N	N	36	20	9	L 4,5 Bulge	1	3	1	1	2	3	14	90	90	1	1	None	6mths	3	16	12	3	90	90
Shakuntala Topalkatti	35	F	Housewife	334790	B/l	6mo	8	2	2	1	1	1	2	None	None	2	None	1	90	30	30	60	50	N	N	N	37	20	9	L 4,5 Bulge	2	3	1	1	2	3	13	90	90	2	None	None	3mths	3	14	10	3	90	90
Mayappa Pandrali	55	M	Farmer	394100	Right	6mo	10	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	40	N	N	N	38	21	9	L 5 - S 1 Bulge	2	1	2	2	2	2	12	90	90	2	None	None	6mths	3	12	10	3	90	90
Mahesh Kudali	25	M	Labourer	392270	B/l	84mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	60	60	N	N	N	38	18	9	L 5 - S 1 Bulge	2	1	2	2	2	3	16	90	90	2	None	None	6mths	3	14	10	3	90	90
Kasturi Patil	49	F	Housewife	391422	None	8mo	8	1	2	2	2	2	2	None	None	2	None	2	105	30	30	90	50	N	N	N	38	18	9	L 4,5 Bulge	1	3	2	1	2	3	14	90	90	2	None	None	6mths	4	14	12	4	90	90
Sajadbi Nadaf	42	F	Farmer	390106	Right	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	2	100	30	30	90	40	N	N	N	36	18	8	L 5 - S 1 Bulge	1	2	2	1	2	3	14	90	90	2	None	None	6mths	4	16	10	4	90	80
Nagappa Bassapa	48	M	Farmer	300997	Left	6mo	9	1	2	1	2	2	2	None	None	1	L 4,5	1	100	30	30	40	90	N	N	N	36	20	8	L 4,5 Bulge	2	1	2	2	2	4	18	90	90	2	None	1	None	Sx	Sx	Sx	Sx	S	Sx
Laxmi More	52	F	Clerk	388901	Left	12mo	9	1	2	1	1	1	1	1	6mths	1	L 5 - S 1	1	105	30	30	40	70	N	N	N	38	18	9	L 3,4,5 Bulge	2	1	1	1	2	5	14	90	90	2	None	None	6mths	4	14	12	4	90	90
Kamallava Harale	40	F	Housewife	344567	Left	36mo	8	1	2	1	1	1	1	1	2yr	1	L 4,5	1	100	30	30	60	90	N	N	N	38	20	9	L 4,5 Bulge	1	1	1	1	2	3	14	90	90	1	1	None	6mths	4	16	10	4	80	80
Mahadevi Sabasuddi	40	F	Housewife	380286	B/l	24mo	8	1	2	1	1	1	2	None	None	2	None	1	105	30	30	40	40	N	N	N	33	22	8	L 3,4,5 Bulge	1	2	2	2	1	3	16	90	90	2	None	None	6mths	4	16	12	4	90	90
Seetaram Dixit	74	M	Farmer	281660	None	24mo	8	1	2	1	1	1	1	1	2yr	1	L 4,5	1	105	30	30	30	90	N	N	N	38	20	9	L 4,5 Bulge	2	2	2	1	2	3	16	90	90	1	1	None	6mths	4	16	12	4	90	90
Channabasappa V Kamat	40	M	Farmer	396758	Right	12mo	8	2	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	70	70	N	N	N	40	18	9	None	1	2	2	2	1	3	10	90	90	2	None	None	6mths	4	16	8	4	80	80
Rajaappa M Dabadi	61	M	Farmer	397859	Right	48mo	8	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	90	40	N	N	N	39	20	9	L 4,5 Bulge	1	3	2	2	2	4	16	90	90	2	None	None	6mths	3	10	6	3	80	80
Shekappa S Bandrolli	60	M	Farmer	402494	Right	6mo	9	1	2	1	2	1	2	None	None	1	L 4,5 - S 1	1	100	25	30	80	60	N	N	N	38	22	9	L 5 - S 1 Bulge	2	1	2	2	2	2	8	90	80	2	None	None	6mths	3	21	10	3	90	90
Shobha P Magasuli	42	F	Housewife	399153	Left	12mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	20	30	60	90	N	N	N	40	24	8	L 4,5 Bulge	1	2	2	2	2	1	17	90	90	2	None	None	6mths	3	16	10	3	90	80
Pundalik B Holi	26	M	Farmer	402456	None	3mo	8	2	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	20	20	N	N	N	39	18	9	L 4,5 Bulge	2	2	2	2	2	3	16	90	90	2	None	None	6mths	2	10	8	2	90	90
Ishwar K Minache	50	M	Coolie	407354	None	48mo	8	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	30	20	60	60	N	N	N	34	14	8	None	1	3	2	2	1	2	10	90	90	2	None	None	6mths	2	14	10	2	90	90
Sominga B Pattanshetty	50	M	Clerk	407337	B/l	6mo	8	2	2	1	1	1	2	None	None	2	None	1	90	30	30	60	50	N	N	N	37	20	9	None	2	1	2	2	2	3	13	80	80	2	None	None	6mths	3	12	10	3	90	90
Yallawwa Siddappa	45	M	Farmer	388814	Right	6mo	10	2	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	40	N	N	N	38	21	9	None	2	1	1	2	2	2	12	90	70	2	None	None	6mths	3	14	10	3	90	80
Sangamath G Rameshswa	42	M	Coolie	411108	B/l	48mo	8	2	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	60	60	N	N	N	38	18	9	None	2	1	2	1	2	3	16	80	70	2	None	None	6mths	5	14	12	5	90	90
Ramu K G Govindappa	41	M	Farmer	399643	None	8mo	8	2	2	2	2	2	2	None	None	2	None	2	105	30	30	90	50	N	N	N	38	18	9	L 4,5 Bulge	1	3	2	1	2	3	14	90	90	2	None	None	6mths	3	16	10	3	90	90
Maruti Patil	23	M	Student	396021	Right	6mo	9	2	2	1	1	1	2	None	None	1	L 4,5	2	100	30	30	90	40	N	N	N	36	18	8	None	1	2	1	1	2	3	14	90	90	2	None	None	6mths	3	12	10	3	90	90
Ashok Hukkeri	45	M	Shopkeeper	397351	Right	8mo	8	2	2	1	1	1	2	None	None	1	L 4,5	2	105	30	30	90	60	N	N	N	34	20	8	None	2	1	2	2	2	3	16	90	80	2	None	None	6mths	3	14	10	3	90	90
Ahmed Khan Pathan	35	M	Shopkeeper	396447	Left	24mo	9	1	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	30	20	60	60	N	N	N	37	24	8	None	1	2	2	2	2	3	17	80	70	2	None	None	6mths	3	16	8	3	90	90
Khairunnissa K Badeghar	47	F	Housewife	410453	Right	6mo	8	1	2	2	1	1	2	None	None	2	None	1	90	20	20	60	50	N	N	N	34	24	8	None	2	3	2	2	2	2	8	90	90	2	None	None	6mths	2	16	12	2	90	90
Indra Singh Bhayal	42	M	Businessma	410454	Right	8mo	9	1	2	2	2	2	2	None	None	1	L 4,5	1	90	20	20	60	50	N	N	N	36	22	9	None	2	1	2	2	2	2	10	90	90	2	None	None	6mths	4	14	11	4	90	80
Shantawwa	45	F	Housewife	410413	Right	6mo	8	1	2	1	1	1	2	None	None	2	None	1	100	25	30	50	50	N	N	N	32	18	8	None	2	1	2	2	2	3	14	90	90	1	1	None	6mths	4	13	11	4	70	70
Ujjawala Patil	34	F	Housewife	404015	Right	8mo	8	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	90	30	20	60	50	N	N	N	34	18	8	L 5 - S 1 Bulge	2	1	2	2	2	3	14	90	90	2	None	None	6mths	2	8	4	2	90	90
Sushila Mallyannavar	36	F	Housewife	402897	Left	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	25	30	60	60	N	N	N	36	20	8	None	2	1	2	2	2	3	12	90	90	2	None	None	6mths	2	12	8	2	90	90
Siddawwa Somkanavar	54	F	Housewife	399987	Right	8mo	9	1	2	1	1	1	2	None	None	1	L 4,5 - S 1	2	105	20	20	80	40	N	N	N	37	16	8	None	2	1	2	2	2	2	16	90	90	2	None	None	9mths	3	14	7	3	90	90
Siddawwa H Hukkeri	60	F	Housewife	399957	Right	12mo	8	1	2	1	1	1	2	None	None	1	L 4,5 - S 1	2	100	20	20	50	50	N	N	N	37	18	9	L 5 - S 1 Bulge	2	1	2	2	2	2	14	90	90	1	2	Sx	Sx	Sx	Sx	Sx	Sx	Sx	Sx
Ramesh H Mudalgi	42	M	Clerk	397854	B/L	24mo	9	2	2	1	1	1	2	None	None	1	L 4,5	1	90	30	20	60	70	N	N	N	34	22	8	L 4,5 Bulge	2	1	2	2	2	3	12	90	90	2	None	None	6mths	4	10	8	4	90	90
Subhash M Patil	24	M	Shopkeeper	407398	Right	2mo	10	1	2	2	2	2	2	None	None	2	None	1	90	20	20	40	30	N	N	N	32	18	9	None	2	1	2	2	2	1	12	90	90	2	None	None	6mths	3	10	4	3	90	90
Nirmala S Gavade	42	F	Housewife	405515	Left	6mo	8	2	2	2	2	2	2	None	None	1	L 4,5	2	100	30	30	50	50	N	N	N	34	22	9	None	1	2	2	2	2	2	14	90	90	2	None	None	6mths	3	11	8	3	90	90
Shivappa B Holemani	60	M	Clerk	408534	Right	24mo	9	1	2	2	2	2	2	None	None	1	L 4,5	2	105	20	20	60	60	N	N	N	34	22	9	L 4,5 Bulge	1	3	2	2	2	3	14	90	90	2	None	None	6mths	3	9	6	3	90	90

Caudal Route Epidural Steroid Patient List

Name	A	S	I.P. No.	Occupation	Primary Symptoms					Previous Treatment					Spinal Signs												MOS	BDIS	NPIS	MRI Findings	Caudal Route						Short Term Results					Rep Inj		Surg	Follow Up						
					Sciatica	Dur	VAS	SS/s	MS/s	R/A	Tr	Ph	Epidural Injections			Tenderness		Spasm	Spinal ROM			SLRT		CNS			O/e					Complications					VAS	ODIS	SLRT		Y/N	No		Dur	VAS	ODIS	BDIS	NPIS	SLRT	
													P/NP	No	Interval	P/A	Location		F	E	Lat F	Lt	Rt	M	S	R					D	At	DP	H	Hy			L	R										L	R
Santosh Nikam B	63	M	303626	Farmer	B/l	24mo	7	1	2	1	1	1	2	None	None	2	None	1	100	30	30	40	40	N	N	N	38	12	8	L 4,5 Bulge	2	1	2	2	2	3	12	90	90	2	None	None	6mths	5	12	10	5	90	90
Lata Gurav	40	F	302413	House Wife	B/l	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	20	80	70	N	N	N	36	18	8	L 5 - S 1 Bulge	2	1	2	2	2	4	16	90	90	2	None	None	6mths	5	16	10	4	90	90
Darnendra Saptasagar	37	M	301818	Farmer	Left	24mo	9	1	2	1	2	1	2	None	None	2	None	1	105	30	10	30	50	N	N	N	40	24	9	L 4,5 Bulge	2	2	2	2	1	4	12	90	90	2	None	None	6mths	3	14	12	5	90	90
Bhadruddin	30	M	306398	Farmer	Right	24mo	9	1	2	1	1	1	1	1	1yr	2	None	1	105	30	30	90	20	N	N	N	38	19	8	L 3,4,5 Bulge	2	2	2	2	2	3	9	90	90	2	None	None	4mths	5	13	11	5	90	90
Shiraya Bhosagi	35	M	306018	Farmer	Left	24mo	9	1	2	1	1	1	2	None	None	2	None	1	100	30	30	20	90	N	N	N	34	21	9	L 3,4,5 Bulge	2	1	2	2	1	4	12	90	90	2	None	None	6mths	3	14	12	3	90	90
Vasant Lad	38	M	305939	Labourer	B/l	30mo	8	1	2	1	1	2	2	None	None	1	L 4,5	1	105	30	30	40	30	N	N	N	36	20	8	L 5 - S 1 Bulge	2	1	2	2	2	8	22	40	40	1	1	None	6mths	3	14	9	3	90	90
Vasant Patil	76	M	311641	Farmer	Left	36mo	8	1	2	1	1	1	2	None	None	1	L 3,4,5	1	100	30	30	70	90	N	N	N	36	21	8	L 3,4,5 Bulge	1	2	2	2	2	3	13	90	90	2	None	None	6mths	3	14	11	3	90	90
Kamalkant Sawant	44	M	311549	Armyman	Left	24mo	8	1	2	1	1	1	1	1	1yr	1	L 4,5	1	100	20	20	20	90	N	N	N	34	21	8	L 3,4,5 Bulge	2	1	2	1	2	3	10	90	90	2	None	None	6mths	3	13	10	4	90	90
Kasshava Patil	48	F		Labourer	B/l	24mo	7	1	2	1	2	2	2	None	None	2	None	1	105	30	30	30	30	N	N	N	36	20	7	L 3,4,5 Bulge	2	1	2	2	1	3	14	90	90	2	None	None	6mths	3	16	10	4	90	90
Shobjan Torasgar	55	F		House Wife	Right	24mo	7	1	2	1	2	2	2	None	None	1	L 3,4,5	1	105	30	30	90	20	N	N	N	36	14	7	L 4,5 Bulge	2	1	2	2	1	3	14	90	90	2	None	None	6mths	3	14	6	3	90	90
Hanumant Singh	34	M		Labourer	Left	24mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	30	90	N	N	N	36	18	9	L 3,4 Bulge	2	1	2	2	2	3	12	90	90	2	None	None	4mths	3	13	10	4	80	90
Racchaya M Hiremath	45	M	333417	Labourer	B/l	3mo	7	1	2	1	1	1	2	None	None	1	L 3,4,5	1	100	30	30	60	45	N	N	N	39	24	7	L 3,4,5 Bulge	2	1	2	2	2	2	14	90	90	2	None	None	6mths	3	14	4	2	90	90
Yamnurappa Iligeri	22	M	336545	Student	Right	8mo	7	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	80	40	N	N	N	34	18	7	L 5 - S 1 Bulge	2	1	2	2	2	2	8	90	90	2	None	None	6mths	3	12	8	2	90	90
Devappa Hardalekar	46	M		Farmer	None	18mo	6	2	2	1	1	2	2	None	None	2	None	2	100	30	30	70	90	N	N	N	34	16	7	L 3,4,5 Bulge	2	1	2	2	2	2	8	90	90	2	None	None	6mths	2	9	5	3	90	90
Sunanda Hukkeri	38	F		House Wife	Left	6 mo	8	2	2	1	2	2	2	None	None	1	L 4,5	1	110	30	30	60	80	N	N	N	36	19	9	L 4,5 Bulge	2	1	2	2	2	2	7	90	90	2	None	None	6mths	2	10	8	2	90	90
Alisaab Appasaab Yarkhund	62	M		Farmer	Left	3 mo	9	1	2	1	1	1	2	None	None	1	L 5 - S 1	1	100	30	30	60	90	N	N	N	34	18	8	L 5 - S 1 Bulge	2	1	2	1	2	2	9	90	90	2	None	None	6mths	3	11	9	2	80	90
Ajjappa Basavantappa	62	M		Farmer	Left	36 mo	8	1	2	1	1	1	2	None	None	1	L 5 - S 1	1	100	30	30	60	90	N	N	N	37	18	8	L 5 - S 1 Bulge	2	1	2	2	2	3	10	90	90	2	None	None	6mths	3	12	8	3	90	90
Laxmikant Badiger	46	M	354238	Farmer	Left	12mo	7	2	2	1	2	2	2	None	None	2	None	2	90	30	30	70	90	N	N	N	32	17	7	L 5 - S 1 Bulge	1	3	2	2	2	2	7	90	90	2	None	None	6mths	3	10	6	2	90	90
Suhas Kamdar	74	M	353033	Farmer	Right	6mo	10	1	2	1	1	1	1	2	3yrs	1	L 4,5	1	110	30	30	90	30	N	N	N	38	20	9	L 4,5 Bulge	2	1	2	1	2	1	6	90	90	2	None	None	6mths	2	8	4	2	90	90
Renuka Yallappa Patil	37	F	352729	House Wife	B/l	18mo	9	1	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	80	20	20	70	70	N	N	N	37	18	9	L 4,5 - S 1 Bulge	2	2	2	1	2	2	11	90	90	2	None	None	6mths	3	12	8	3	90	90
Shabbir Deshnur	40	M	352567	Labourer	B/l	24mo	10	1	2	1	1	1	2	None	None	1	L 5 - S 1	1	90	30	30	60	80	N	N	N	36	17	8	L 5 - S 1 Bulge	1	2	2	2	2	2	8	90	90	2	None	None	6mths	3	14	7	3	90	90
Prakash Asif	38	M	352097	Farmer	None	24mo	9	1	2	1	1	1	1	1	2yrs	1	L 4,5	1	100	30	30	30	90	N	N	N	36	12	9	L 4,5 Bulge	2	1	2	2	1	2	13	90	90	1	1	Yes	None	Sx	Sx	Sx	Sx	Sx	Sx
Shankar Hanchimal	55	M		Technitian	Right	12mo	8	1	2	1	2	2	2	None	None	2	None	1	110	30	30	60	60	N	N	N	38	18	8	L 5 - S 1 Bulge	2	1	2	2	1	2	10	90	90	2	None	None	6mths	3	10	8	2	90	90
Ashok Shinde	50	M		Farmer	Left	8mo	7	2	2	1	1	1	2	None	None	2	None	2	105	30	30	80	40	N	N	N	35	16	8	L 4,5 Bulge	2	1	2	2	2	1	6	90	90	2	None	None	6mths	2	10	4	1	90	90
Gurunath Basavantappa	38	M		Labourer	B/l	24mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	60	60	N	N	N	34	18	9	L 4,5 Bulge	2	2	2	2	2	2	10	90	90	2	None	None	6mths	3	11	8	4	90	90
Nazeer Appalal Gotyal	44	M		Labourer	None	3mo	9	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	60	60	N	N	N	34	16	9	L 4,5 Bulge	2	1	2	2	2	2	8	90	90	2	None	None	6mths	3	9	6	3	90	90
Kenchappa Shivappa Khot	62	M		Farmer	None	24mo	8	1	2	1	2	2	2	None	None	1	L 4,5	1	105	30	30	60	50	N	N	N	36	14	8	L 5 - S 1 Bulge	2	1	2	2	2	6	24	80	80	1	2	None	6mths	3	14	10	4	90	90
Shakeela Bashirahmed	45	F	359467	House Wife	B/l	24mo	9	1	2	1	2	2	2	None	None	1	L 5 - S 1	1	100	30	30	50	50	N	N	N	38	16	9	L 5 - S 1 Bulge	1	3	2	1	1	8	30	60	60	1	2	None	6mths	3	16	10	4	90	90
Madhavi Shubam	40	F	358591	House Wife	B/l	60mo	9	1	2	1	2	2	2	None	None	1	L 3,4	2	100	30	30	40	40	N	N	N	34	16	9	L 3,4,5 - S 1 Bulge	2	1	2	2	2	3	8	90	90	2	None	None	6mths	3	10	8	4	90	90
Dundappa Badakar	57	M		Farmer	Right	3mo	8	1	2	1	2	2	2	None	None	1	L 3,4,5	1	105	30	30	90	50	N	N	N	39	20	8	L 4,5 - S 1 Bulge	2	1	2	2	2	2	8	90	90	2	None	None	6mths	3	10	8	4	90	90
Kammalawa Harde	40	F	361197	House Wife	B/l	24mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	60	N	N	N	36	18	8	L 4,5 Bulge	2	2	2	2	2	2	9	90	90	2	None	None	6mths	3	12	12	4	90	90
Hari Tukaram Desai	35	M		Farmer	Right	36mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	40	N	N	N	36	16	9	L 3,4,5 - S 1 Bulge	2	1	2	2	2	3	8	90	90	2	None	None	6mths	3	8	7	3	90	90
Tangewwa Bhajarthi	25	F	361157	Student	Left	3mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	60	90	N	N	N	37	18	9	L 5 - S 1 Bulge	2	1	2	2	1	2	10	90	90	2	None	None	6mths	2	12	6	2	90	90
Balu Bachate Rajare	50	M	360421	Labourer	Right	3mo	8	1	2	1	1	1	2	None	None	1	L 3,4	1	90	10	10	90	60	N	N	N	38	18	8	L 5 - S 1 Bulge	2	1	2	2	2	2	7	90	90	2	None	None	6mths	3	8	8	3	90	90
Mahshyana Raut	35	F	361264	Farmer	Left	36mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	20	20	N	N	N	38	18	9	L 3,4,5 Bulge	1	3	2	2	2	3	10	70	90	2	None	None	6mths	3	12	8	4	90	90
Mahadevi G	40	F	362363	House Wife	Right	36mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	90	30	30	90	60	N	N	N	36	18	7	L 4,5 Bulge	1	1	2	1	1	3	13	90	90	2	None	None	6mths	4	12	8	4	90	90
Ganesh M	30	M		Labourer	Left	4mo	7	1	2	1	1	1	2	None	None	1	L 5 - S 1	1	60	10	10	40	90	N	N	N	38	15	7	L 5 - S 1 Bulge	1	1	2	2	2	2	12	90	90	2	None	None	6mths	4	12	8	4	90	90
Kamal Behale	34	F	363891	Farmer	Left	48mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	60	70	N	N	N	36	18	8	L 4,5 Bulge	2	1	2	2	2	4	9	90	90	2	None	None	6mths	5	13	9	5	90	90
Malu Chougale	40	F	363383	House Wife	Left	12mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	60	90	N	N	N	36	16	9	L 4,5 Bulge	2	1	2	1	2	2	11	90	90	2	None	None	6mths	3	12	8	4	90	90
Kallapa Kanthi	84	M	368470	Farmer	Right	3mo	9	1	2	1	1	1	1	1	2yrs	1	L 4,5	1	105	30	30	70	70	N	N	N	34	18	9	L 4,5 Bulge	2	2	2	2	2	3	10	90	90	2	None	None	6mths	3	10	8	3	90	90
Shivram Barappa Ghasti	55	F	363936	Farmer	Left	8mo	6	1	2	1	1	1	2	None	None	1	L 3,4	1	105	30	30	40	90	N	N	N	38	18	9	L 5 - S 1 Bulge	1	2	2	2	2	8	32	60	90	1	1	None	6mths	3	11	7	3	90	90
Kasturi Sharangouda Patil	40	F	372280	House Wife	Left	36mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	40	90	N	N	N	36	18	9	L 4,5 Bulge	2	1	2	2	2	2	9	90	90	2	None	None	6mths	4	10	7	4	90	90
Sharanappa Shivashimpi	44	M	375257	Cooli	Left	24mo	9	1	2	2	2	2	2	None	None	1	L 4,5	1	90	20	20	50	90	N	N	N	34	14	7	L 4,5 Bulge	2	1	2	2	1	3	12	90	90	2	None	None	6mths	3	14	10	3	90	90
Raju Patil	32	M	352295	Farmer	B/l	60mo	8	1	2	1	1	1	1	1	2yrs	1	L 5 - S 1	1	100	20	20	60	60	N	N	N	38	18	8	L 5 - S 1 Bulge	1	1	2	1	2	3	8	90	90	2	None	None	6mths	4	9	12	2	90	90
Parvati Hannavar	50	F	353035	House Wife	None	36mo	9	2	2	1	1	1	1	1	6mo	1	L 4,5	1	105	30	30	50	50	N	N	N	34	16	9	L 4,5 Bulge	2	1	2	1	2	2	10	90	90	1	1	None	6mths	3	16	10	3	90	90
Suresh Halkatti	40	M	377705	Cooli	Left	24mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	20	90	N	N	N	36	14	8	L 4,5 Bulge	1	2	2	2	1	4	12	90	90	2	None	None	6mths	4	12	10	4	90	90
Babasaheb Iragouda	45	M		Technitian	None	24mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	70	10	10	60	60	N	N	N	34	16	9	L 5 - S 1 Bulge	2	1	2	2	2	4	24	90	90	2	None	None	6mths	5	20	12	5	90	90
Sharawwa Biradi Basappa	40	F		House Wife	B/l	36mo	7	1	2	1	1	1	1	3	1yr	1	L 3,4,5	2	100	30	30	90	40	N	N	N	32	22	9	L 4,5 Bulge	2	2	2	2	2	4	16	90	90	2	None	None	6mths	5	18	12	5	90	90
Prakash Yehechawandi	38	M		Farmer	None	24mo	8	1	2	1	1	1	1	2	6mo	2	None	2	105	30	30	40	60	N	N	N	34	24	9	L 3,4,5 - S 1 Bulge	1	3	2	2	1	4	12	90	90	2	None	None	6mths	4	12	10	4	90	90
Ganesh Gawde	34	M		Labourer	B/l	11mo	9	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	40	40	N	N	N	40	24	9	L 5 - S 1 Bulge	2	1	2	2	2	2	12	90	90	2	None	None	4mths	4	14	9	2	90	90
Megha S Heddurshetti	36	F	397994	Housewife	None	12mo	8	2	2	1	1	1	2	None	None	1	L 4,5	1	105	30	30	70	60	N	N	N	39	18	9	L 5 - S 1 Bulge	2	2	2	2	1	3	10	90	90	2	None	None	6mths	3	12	10	3	90	90
Laxman K Hulimani	35	M	396573	Farmer	Right	6mo	8	1	2	1	1	1	2	None	None	1	L 3,4,5	1	105	30	30	90	40	N	N	N	39	20	9	None	1	3	2	2	2	3	16	90	90	2	None	None	9mths	3	16	10	3	90	90
Gajanan S Malik	51	M	346678	Mechanic	Right	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	25	30	80	60	N	N	N	38	22	9	L 5 - S 1 Bulge	2	1	2	2	2	2	8	90	90	2	None	None	6mths	2	14	12	2	90	90
Vitthal T Jadhav	26	M	342046	Labourer	Left	4mo	8	1	2	1	1	1	2	None	None	1	L 4,5	1	100	20	30	60	90	N	N	N	40	24	8	L 4,5 Bulge	2	1	2	2	2	1	17	90	90	1	1	None	6mths	4	13	11	4	90	90
Mallappa B Bedasur	32	M	400204	Labourer	None	3mo	8	1	2	1	2	1	2	None	None	1	L 4,5	1	105	30	30	50	40	N	N	N	39	18	9	None	2	1	2	2	2	3	16	90	90	2	None	None	6mths	3	14	12	3	90	90
Shankar C Navalgi	37	M	404073	Farmer	None	48mo	8	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	1	100	30	20	60	60	N	N	N	34	14	8	L 5 - S 1 Bulge	2	1	2	2	2	2	10	90	90	2	None	None	6mths	2	14	9	2	90	90
Mallappa B Kajagar	43	M	405456	Farmer	B/L	6mo	8	2	2	1	1	1	2	None	None	2	None	1	90	30	30	60	50	N	N	N	37	20	9	None	2	2	2	2	2	3	13	90	90	2	None	None	6mths	3	14	11	3	90	90
Shetteppa S Dhali	65	M	404825	Farmer	Right	6mo	10	1	2	1	1	1	1	1	1yr	1	L 4,5	2	100	30	30	90	40	N	N	N	38	21	9	L 5 - S 1 Bulge	2	1	2	2	2	2	12	90	90	2	None	None	6mths	2	13	10	2	90	90
Raju M Savatgi	37	M	407354	Liftman	B/L	48mo	8	1	2	1	1	2	2	None	None	1	L 4,5	1	105	30	30	60	60	N	N	N	38	18	9	L 5 - S 1 Bulge	2	1	2	2	2	3	16	90	90	2	None	None	9mths	3	16	10	3	90	90
Prakash A Virupaxappa	43	M	406977	Farmer	None	8mo	8	1	2	1	1	1	2	None	None	2	None	2	105	30	30	90	50	N	N	N	38	18	9	L 4,5 Bulge	1	3	2	2	2	3	14	90	90	2	None	None	9mths	4	14	6	4	90	90
Shrinath S Nandikol	23	M	407456	Clerk	Right	6mo	9	1	2	1	1	1	2	None	None	1	L 4,5	1	100	30	30	90	40	N	N	N	36	18	8	L 5 - S 1 Bulge	1	2	2	2	2	3	10	90	90	2	None	None	9mths	2	13	10	2	80	90
Savakka S Pujeri	31	F	411530	Housewife	None	3mo	8	1	2	1	1	1	2	None	None	2	None	2	100	30	30	60	60	N	N	N	38	20	9	None	2	1	2	2	2	2	14	90	90	2	None	None	9mths	2	14	4	2	90	90
Geeta Mohan Patil	40	F	412416	Housewife	Left	6mo	9	1	2	1	1	1	2	None	None	2	None	1	105	25	30	50	60	N	N	N	37	21	8	L 4,5 Bulge	1	2	2	2	2	2	12	90	90	2	None	None	6mths	3	12	8	3	90	90
Saleema Begum Borgi	55	F	411136	Housewife	Left	36mo	9	2	2	1	1	1	2	None	None	1	L 4,5 - S 1	2	90	20	20	40	50	N	N	N	34	22	8	L 5 - S 1 Bulge	1	4	2	2	1	1	10	90	90	2	None	None	6mths	3	9	5	3	90	90
Rakshi R Hallappa	52	M	408063	Farmer	Right	24mo	8	1	2	1	1	1	2	None	None	2	None	1	100	25	30	50	50	N	N	N	37	22	8	None	2	1	2	2	2	2	12	90	80	2	None	None	9mths	3	10	8	3	90	90
Bhirappa B Tadasalur	25	M	405558	Labourer	Right	12mo	9	1	2	2	2	2	2	None	None	1	L 4,5	1	100	30	30	70	60	N	N	N	34	20	9	None	2	1	2	2	2	2	13	80	80	2	None	None	9mths	4	11	9	4	80	90
Mahantesh B Ingale	29	M	404211	Shopkeeper	Right	3mo	8	2	2	2	1	1	2	None	None	2	None	1	100	25	30	50	50	N	N	N	36	21	8	None	2	1	2	2	2	3	14	90	90	2	None	None	9mths	4	12	8	4	90	90
Annapurna B Kaddappanava	53	F	403258	Housewife	B/L	6mo	8	1	2	1	1	1	2	None	None	2	None	1	105	30	25	70	70	N	N	N	37	22	9	None	1	3	2	2	2	2	16	80	80	2	None	None	6mths	1	10	6	1	90	90

KEY TO MASTER CHART

i. 1 : Present

ii. 2 : Absent

iii. A : Age

iv. S : Sex

v. I. P. No : Inpatient Number

vi. Dur : Duration

vii. SS/s : Sensory Signs & Symptoms

viii. MS/s : Motor Signs & Symptoms

ix. R/A : Rest / Analgesics

x. Tr : Traction

xi. Ph : Physiotherapy

xii. P/NP : Present / Not Present

xiii. No : Number

xiv. ROM : Range of Motion

xv. F : Flexion

xvi. E : Extension

xvii. Lat F : Lateral Flexion

xviii. O/e : On / Examination

xix. M : Motor

xx. S : Sensory

xxi. R : Reflexes

xxii. N : Normal

xxiii. D : Difficulty

xxiv. At : Attempts

xxv. DP : Dural Puncture

xxvi. H : Headache

xxvii. Hy : Hypotension

xxviii. L : Left

xxix. R : Right

xxx. Inj : Injection

xxxi. Surg : Surgery Decompression