"EFFECT OF BUPIVACAINE VERSUS NORMAL SALINE IN CAUDAL EPIDURAL STEROID INJECTION IN CHRONIC LOW BACK ACHE: A ONE YEAR RANDOMIZED CONTROLLED TRIAL"

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ABBREVIATIONS

1. Straight Leg Raising Test - SLRT
2. Oswestry Disability Index Score – ODIS
3. Beck's Depression Inventory Scoring – BDIS
4. Visual Analogue Scale – VAS
5. Low Back Pain – LBP
6. Chronic Low Back Pain – cLBP
7. Computed Tomography - CT
8. Magnetic Resonance Imaging – MRI
9. Epidural steroid injections – ESIs
10. Cerebrospinal Fluid – CSF
11. Herniated Nucleus Pulposus HNP

ABSTRACT

Background and objectives

Epidural steroid injections have been performed for decades and are one of the most common spinal injection procedures performed. This palliative procedure is a safe and effective means for treating low back pain. This study was aimed to compare the effect of Bupivacaine versus Normal Saline in Caudal Epidural Steroid injection in the treatment of chronic low backache.

Methodology

The present one year randomized controlled trial was conducted at Department of Orthopedics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on a total of 60 patients with chronic low backache. Based on the computer generated randomization the selected patients were randomized into two groups namely Group B (Bupivacaine group) and Group S (Normal saline group). The assessment of pain was done based on VAS, disability on ODIS and depression on BDIS.

Results

In the present study male to female ratio in group B was 2:1 and in group S it was 1.5:1. 50% of patients in group B had right sided radiculopathy and in group S the same was present in 43.33% of patients. The mean duration in group B was 12.63 ± 12.54 months and in group S it was 13.57 ± 12.72 months. The mean pain scores at post injection six months were less in group B (2.60 ± 1.28) compared to group S (3.00 ± 1.31). The mean ODIS score at post injection three weeks, three months and six months were significantly less in group B compared to group S ($p<0.001$). The mean BDIS score were comparable in both the groups at post injection three weeks. But at post injection three months and six months mean BDIS

scores were significantly less in group B compared to group S. Requirement of repeat injection was less in group B (13.33%) compared to group S (36.67%). Also less number of patients in group B (6.67%) underwent surgery compared to group S (13.33%).

Conclusion and interpretation

Overall the present study showed caudal steroid injection with bupivacaine provided better outcome with regards to pain, disability and depression. Also caudal steroid injection with bupivacaine resulted in fewer repeat injections and avoided surgery.

Key words

Bupivacaine; Epidural steroid injections; Low back pain; sciatica

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INTRODUCTION

Like a modern skyscraper, the human spine defies gravity, and defines us as vertical bipeds. It forms the infrastructure of a biological machine that anchors the kinetic chain and transfers biomechanical forces into coordinated functional activities. The spine acts as a conduit for precious neural structures and possesses the physiological capacity to act as a crane for lifting and a crankshaft for walking.

Subjected to aging, the spine adjusts to the wear and tear of gravity and biomechanical loading through compensatory structural and neurochemical changes, some of which can be maladaptive and cause pain, functional disability, and altered neurophysiologic circuitry. Some compensatory reactions are benign; however, some are destructive and interfere with the organism's capacity to function and cope. Spinal pain is multifaceted, involving structural, biomechanical, biochemical, medical, and psychosocial influences that result in dilemmas of such complexity that treatment is often difficult or ineffective.[1]

Low back pain (LBP) is defined as chronic after 3 months because most normal connective tissues heal within 6-12 weeks, unless pathoanatomic instability persists. A slower rate of tissue repair in the relatively avascular intervertebral disk may impair the resolution of some persistent painful cases of chronic LBP (cLBP). An estimated 15-20% develop protracted pain, and approximately 2-8% have chronic pain. Of those individuals who remain disabled for more than 6 months, fewer than half return to work, and after 2 years of LBP disability, a return to work is even more unlikely.[2]

Recent studies suggest that one third to one fourth of patients in a primary care setting may still have problems after 1 year.[3,4] cLBP is the most common cause of disability in Americans younger than 45 years.[5,6] Each year, 3-4% of the US population is temporarily disabled, and 1% of the working-age population is totally and permanently disabled.[7,3,8] LBP has been cited as the second most frequent reason to visit a physician for a chronic condition[3,8,9,10], the fifth most common cause for hospitalization[3,11,12,13] and the third most frequent reason for a surgical procedure.[3,11,12,13] The socioeconomic impact of cLBP is massive. Ironically, a minority of patients with cLBP and disability due to cLBP account for the majority of the economic burden.[14,15,16]

Most commonly, diagnoses of acute painful spinal conditions are nonspecific, such as neck or back strain, although injuries may affect any of several pain-sensitive structures, which include the disk, facet joints, spinal musculature, and ligamentous support[17, 18]. The origin of chronic back pain is often assumed to be degenerative conditions of the spine;however, controlled studies have indicated that any correlation between clinical symptoms and radiological signs of degeneration is minimal or nonexistent.[6, 17, 18, 19, 20, 21] Inflammatory arthropathy, metabolic bone conditions, and fibromyalgia are cited in others as the cause of chronic spine-related pain conditions.[17, 18]

Although disc herniation has been popularized as a cause of spinal and radicular pain, asymptomatic disc herniations on computed tomography (CT) and magnetic resonance imaging (MRI) scans are common.[21,22,23,24] Furthermore, there is no clear relationship between the extent of disk protrusion and the degree of clinical

symptoms.[25] Degenerative change and injury to spinal structures produce lower back and leg pain that vary proportionally.

There are no interventions which provide definite and long-term improvement in chronic low back pain. In most of the cases the expectations of the patient and the spinal surgeon are not met following lumbar spine surgery.

Most people with low back pain do not seek medical care. Many self treat with over the counter medications and lifestyle changes.[10] Several systematic reviews have concluded that strong evidence supports the use of non-steroidal anti-inflammatory drugs for non-neuropathic low back pain, though the treatment effect is small and the evidence is greater for acute than chronic pain. Paracetamol (acetaminophen) is slightly less effective than non-steroidal anti-inflammatory drugs but has fewer or less severe side effects. Minimal evidence exists that non-steroidal anti-inflammatory drugs are effective for radiculopathy, or that one drug is better than others.[11] Spinal injection procedures serve as an important adjunct to the diagnostic process by providing functional and physiologic information that is not obtained from physical examination and imaging studies.

Epidural steroid injections (ESIs) have been performed for decades and are one of the most common spinal injection procedures performed. This palliative procedure is a safe and effective means for treating low back pain due to spinal stenosis, degenerative disk disease and herniated nucleus pulposus, nerve root compression, or nerve root inflammation due to trauma or infection such as herpes zoster.

OBJECTIVES

1. To compare the efficacy of Bupivacaine or Normal Saline in caudal epidural steroid injection in low backache and sciatica.
2. To assess the outcome in terms of pain relief and disability Visual Analogue Scale using and Oswestry Disability Index.
3. To assess the depression associated with low backache & sciatica using the Beck's Depression Inventory Scores.
4. To find out any predictors of good outcome.

REVIEW OF LITERATURE

The term low back pain (LBP) is very broad and covers a large heterogeneous group of disorders. These cannot only be characterized by pain, but also by discomfort and/or stiffness. LBP probably covers several sub-groups with differing etiologies and prognoses, but since current knowledge does not allow us to determine the exact medical cause of LBP in most patients, ´nonspecific´ LBP is determined by exclusion. In fact, it has been estimated by some that a somatic cause is found in 10-20% of cases with LBP,[26] whereas others find that as much as 97% of LBP is "non-specific" or "sprain/strain".[27] Thus, LBP refers to a set of symptoms or a syndrome rather than a diagnosis.[28]

Low back pain is any back pain between the ribs and the top of the leg, from any cause.[29] Low back pain is an important public health problem in all industrialized nations. Although most people appear to recover quickly from an episode of LBP, disability resulting from back pain is more common than any other cause of activity limitation in adults aged less than 45 years and second only to arthritis in people aged 45 to 65 years.[29]

Historical Review[30]

Discomfort and pain in the low-back was first described on paper in 1500 BC by Edwin Smith's papyrus writings. Prior to the 19th century the possible relationship between the facet joints, the discs, and nerve irritation and low-back pain was unknown.

The first successful laminectomy was probably performed by Paul of Aegina in the 7th century A.D., but this feat was not repeated until 1829 by Alban Smith of Danville, Kentucky.

In conjunction with the development of the British railway system (1800-1850) a relationship between heavy work and damage to the back was acknowledged. Prior to this time low-back pain was never seen in association to an injured spine. The term "wear and tear of the back" became accepted and individuals were entitled to compensation in some instances. Research activity in this field increased markedly but it was still not possible to establish a direct cause and effect relationship.

In 1929, Walter Dandy published two cases of sciatic pain associated with herniated disc fragments which responded to surgical discectomy.

In 1934, Mixter and Barr published a paper describing their observations regarding the role of lumbar disc herniation as a common etiology of sciatic pain. It became clear that the bulging of discal material could result in pressure on the spinal nerves which could in turn result in loss of muscular function and sensory disturbances. This groundbreaking new knowledge regarding the pathoanatomical relationships of spinal structures unfortunately led many physicians to believe that all spinal problems were discal in origin. Many were therefore of the opinion that surgery would be the answer for most back ailments. As great advances were being made in anaesthetics and surgical specialties during this period many low-back pain patients underwent surgery; many of them up to several times. The tendency to overutilise a

newly developed treatment modality for a period of time has also been seen in other areas of medical science.

In the 20th century it was quickly established that the nervous system could be involved in the development of low-back pain and later on it was widely accepted that low-back pain was possibly caused by an "irritation" of the nervous system. Due to the difficulty in establishing a physical cause many of the symptoms were considered to be of hysterical (psychological) nature. The most commonly held belief was that symptoms were a result of an irritated nervous system and research focused on this area.

During this time, the medical specialty orthopaedic surgery was developed. As regards low-back pain, bed rest was the most commonly prescribed treatment. Low-back pain was not treated with bed rest in earlier times, but was considered to be a valid treatment in this period as symptoms appeared to improve in many patients. The use of bed rest was not based upon scientific documentation but rather on empirical evidence (experience). Current knowledge dictates that it is both wrong and clinically ineffective to treat almost all low-back ailments with bed rest of up to several weeks' duration.

During the past 30 years much energy has been focused upon reducing workloads as a result of the increased number of low-back pain episodes occurring at the work place.

In the US population, the third National Health and Nutrition Examination Survey (NHANES III) (1988–1994) estimated that the 12-month period prevalence of back pain episodes lasting for at least 1 month was 17.8%.[31]

An estimated 4.1 million Americans had symptoms of an intervertebral disc disorder between 1985 and 1988, with an annual prevalence of about 2% in men and 1.5% in women. A study of 295 Finnish concrete workers aged 15-64 years revealed that 42% of men, and as many as 60% of the men aged 45 years or older, reported having sciatica. When interviewed approximately 5 years later, the lifetime prevalence had increased from 42% to 59%.[32]

In 1988 a study conducted at Philadelphia stated that LBP is the most expensive, benign condition in industrialized countries.[7] Experts have estimated that approximately 80% of Americans will experience LBP during their lifetimes.[7,33]

In 1999, a study conducted in USA stated that the annual prevalence of LBP is 15-45% with a point prevalence of approximately 30%.[2] Sixty percent of those who suffer from acute LBP recover in 6 weeks and up to 80-90% recover within 12 weeks; however, the recovery of the remaining patients with LBP is less certain.[2]

In 2000, a study done at Athens, Greece stated that in the adult Greek population, the 1-month prevalence of back pain has been estimated as 32%. This figure is somewhat higher than that reported of other population surveys and may reflect the relatively high proportion of the Greek population engaged in manual work such as agriculture.[9]

In 2001, a study conducted at Rohtak, North India reported that back pain is the second most common reason for visits to office based physicians. Out of 11234 patients, there were 2594 (23.09%) patients of low back pain. A total of 4348 roentgenograms were done in these patients, a mean of 1.68 per patient. Six hundred and eighty four patients (26.36%) had to change/leave the profession. In 1738 patients (67%) nonorganic or psychosocial issues were present. One thousand four hundred and sixty five (57%) were heavy manual workers. Nine hundred and seventy three patients were compared with 1000 young adults attending orthopedics outdoor for reasons other than back pain.[34]

In 2004 a study conducted at Lubeck, Germany, recorded a direct comparison of back pain between the United Kingdom and Germany not only showed differences in prevalence between the two countries (22% compared with 44.9% in women) but also demonstrated marked differences in the prevalence of current back pain within each country or region.[35] West Germans carry a risk of back pain 2.5 to 3.5 times higher than the British, even after adjusting for potential confounders. The authors hypothesize that intercultural differences between the British and Germans in pain perception or pain reporting may be a plausible explanation for the variation where none was expected to occur.[35]

In a 2004, nationwide study of 1-year prevalence of musculoskeletal disorders among workers in Taiwan, pain in the lower back and waist was among the most frequently cited symptom, occurring in 18% male workers and 20% of female workers. Workers between the ages of 45 and 64 years had the highest prevalence of back pain in both sexes.[36]

In 2004, a study conducted in Shanghai, China published that overall prevalence of low back pain lasting more than 1 day among Chinese workers was relatively high:50%.[37] The prevalence declined considerably as the period of recall shortened, however, from 61% (lifetime) to 20% in the past week. The most frequent occurrence of low back was among garment workers, who showed a four-fold increase in comparison with teachers.

According to Datamonitor estimates, in 2010 there were about 55.7 million total prevalent cases of chronic low back pain in people aged 18 and over in the seven major countries (the US, Japan, France, Germany, Italy, Spain, and the UK). The majority of cases were in those aged between 40 and 59. Between 2010 and 2020, Datamonitor expects to see an increase in the number of total prevalent cases of chronic low back pain in those aged 18 and over in the seven major countries (the US, Japan, France, Germany, Italy, Spain, and the UK), from 55.7 million cases to 59.1 million cases. Japan will be the only country to witness a decrease in cases. Datamonitor estimates that the largest number of cases of chronic low back pain will be in those aged between 40 and 59, the age group in which low back pain is most prevalent. Of the prevalent cases in men, 45% will be in this age group; in women, 43% of cases will be in this age group.[38]

In 2010, a cross-sectional study was conducted at Sibu, Malaysia among 931 health care providers to investigate the prevalence, the consequences and the risk factors associated with LBP among hospital staff. It was reported that, the cumulative life-prevalence of LBP was 72.5% and the yearly prevalence was 56.9%. Chronic LBP prevalence was noted 5.1% of the cases. Treatment was sought in 34.1% of LBP

sufferers and 7.3% required sick leave or absence from work due to LBP. Risk factors associated with LBP were professional categories, bad body posture, lifting objects or patients and the increased levels of lifting, levels of job satisfaction and stressful job demands.[39]

Studies suggest that low back pain may arise from any one of a number of anatomical structures, including bones, intervertebral discs, joints, ligaments, muscles, neural structures and blood vessels. In a minority of instances, approximately 5–15%, low back pain can be attributed to a specific cause such as an osteoporotic fracture, neoplasm or infection. For the remaining 85–95% of cases, the specific cause of low back pain is unclear. The search for causes of low back pain continues through causal inference, which is a process whereby several criteria are examined to assess causation. These include assessment of bias and confounding; demonstrating a temporal relationship (the cause must precede the effect); plausibility; consistency with other studies; the strength of the association (the relative risk); the dose response relationship (i.e., does increased exposure lead to increased effect) and reversibility (does removal of the exposure reduce the disease risk). Due to methodological heterogeneity in studies that have investigated causation of low back pain, it is difficult to draw any conclusions about causality. Many studies have significant bias and confounding and relatively few cohort studies have been conducted. It has also been difficult to determine a temporal relationship between risk factors and low back pain (depression and low back pain), and it has been difficult to quantify exposure variables (in the occupational setting: the frequency/amount of lifting).[40]

Risk Factors

Risk factors are variables associated with an increased risk of disease. Examining risk factors for a particular disease may involve measuring the occurrence of disease in two or more groups of people who have experienced different levels of exposure. While research into risk factors for low back pain is often challenging due to heterogeneity across research methods, case definitions and study populations, it is clear there are a number of environmental and personal factors that influence the onset and course of low back pain. Some of these are modifiable and some are not.[40]

Age is one of the more common risk factors for low back pain. Some studies have found incidence is highest in the third decade and overall prevalence increases with age until ages 60 or 65 years, and then gradually declines. As noted above, Dionne et al. found prevalence continues to increase with age for more severe forms of low back pain, and an increasing number of studies are showing low back pain to be a very common problem among adolescents.[40]

While most studies have found no significant gender differences in the prevalence of low back pain our systematic review found that both the mean and median prevalence of low back pain was higher in women. Some studies have found a higher prevalence amongst older women compared with older men and several studies have shown that women are more likely to take time off work and use health-care because of their low back pain, and more likely to develop chronic low back pain. Less is known about age patterns in low- and middle-income countries, where introduction to the workforce takes place at earlier ages and life expectancies are

significantly lower. Of the studies that have taken place in these settings, few have found any significant gender differences in the prevalence of low back pain.[40]

Manahan et al. found in their study of 1685 villagers in the Philippines that low back pain was not related to gender, [41] while Anderson found no significant differences in the prevalence of back pain between men and women in Nepal.[42]

Low educational status has been shown to be associated with an increased prevalence of low back pain. This association is an even stronger predictor of episode duration and poor outcomes from low back pain. In one study in Russia, those with a low educational level were shown to have significantly more low back pain complaints ($p<0.05$). Other studies have found an inverse relationship between social status and the occurrence of low back pain.[40]

In a systematic review[43] of risk factors for low back pain, Leboeuf-Yde found that body weight was a weak risk factor. Two studies found obesity or high body mass index (BMI) (>30 BMI) to be associated with an increased occurrence of low back pain. This association may be stronger in women than in men. Two further studies concluded that heredity plays a major role in lumbar disc degeneration and that heavy physical loading, through occupation or sport, is no longer considered as influential as previously thought.

There are a number of psychosocial factors associated with low back pain, including stress, anxiety, depression and certain types of pain behavior; however, the direction for these relationships is often unclear. Evidence shows that psychosocial factors are also significantly associated with the transition from acute to chronic low

back pain. Psychosocial workplace factors have also been shown to be important risk factors for low back pain. In two systematic reviews, it was found that job dissatisfaction, monotonous tasks, poor work relations, lack of social support in the workplace, demands, stress and perceived ability were associated with an increased occurrence of low back pain. Job dissatisfaction has also been shown to be associated with transition from acute to chronic low back pain.[40]

Other occupational factors have been shown to be associated with low back pain. Matsui et al. demonstrated a clear correlation between physical demands of work and the prevalence of low back pain (with the exception of lifetime prevalence in female workers).[44] They found the point prevalence of low back pain was 39% in manual workers, but only 18.3% in male sedentary workers. More recently, a systematic review found that manual handling, bending, twisting and whole-body vibrations are risk factors for low back pain.[45] Although the data on occupational risk in low-income countries are relatively limited, it has been estimated that 80–90% of the population in these areas are involved in 'heavy work', which suggests this may have a significant impact on the occurrence of low back pain.[40]

LBP is most prevalent in industrialized societies. Genetic factors that predispose persons of specific ethnicity or race to this disorder have not been clearly identified with respect to mechanical, discogenic, or degenerative causes. Men and women are affected equally, but in those older than 60 years, women report LBP symptoms more often than men. The incidence of LBP peaks in middle age and declines in old age, when degenerative changes of the spine are universal. Sciatica

usually occurs in patients during the fourth and fifth decades of life; the average age of patients who undergo lumbar discectomy is 42 years.

Epidemiological data suggests that risk factors, including extreme height, cigarette smoking, and morbid obesity, may predispose an individual to back pain. However, research studies have not clearly demonstrated that height, weight, or body build are directly related to the risk of back injury. Weakness of the trunk extensor muscles, compared with flexor strength, may be a risk factor for sciatica. Fitness may be correlated with the time to recovery and return to work after LBP; however, in prospective studies controlled for age, isometric lifting strength and the degree of cardiovascular fitness were not predictive of back injury.

Inconsistencies remain in the literature over the relative contributions of physical and psychological risk factors to the occurrence of back disorders and back pain. Relatively little is known about risk factors for the transition from acute to chronic low back pain. Broadly, the variables associated with nonspecific low back pain can be classified as individual, psychosocial, or occupational factors. More recently, genetic and bio-mechanical models have contributed to the understanding of the development of back disorders that present as back pain.

History and Evolution of Epidural steroid injections

In 1855, Wood of Edinburgh popularized the use of a hollow needle, which was described 10 years earlier by Rynd of Dublin, and the use of the glass syringe, which was 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 and subsequently used the same in man for seminal incontinence & spinal weakness.[46]

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.[47]

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.[48] Page described the technique of Lumbar epidural analgesia.[49]

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.[50]

Dogliotti (1933), an Italian described at greater length the technique of lumbar epidural injections, & described the loss of resistance sign.[51]

Further research came from Karl Koller in 1929, who described the clinical use of cocaine for anesthesia.[52] 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).[53] 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.[54] Liever et al, in 1953 were the first to explore the space via the sacral route.[55] Goebert et al. in 1961 were the first to report use of hydrocortisone acetate in 113 patients with painful radiculopathy.[56] 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.[57]

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.[58]

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."[59,60]

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"[61]

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 & Bupivacaine with excellent long & short term results.[62]

Now, Epidural Steroid Injections (ESIs) have been endorsed by the North American Spine Society and the Agency for Healthcare Research and Quality (formerly, the Agency for Health Care Policy and Research) of the Department of Health and Human Services as an integral part of nonsurgical management of radicular pain from lumbar spine disorders.

ESI's have been used for decades to treat both LBP and sciatica. Unfortunately, they have been, and likely will continue to be, over utilized. Because they have a very safe track record and are viewed as an alternative to surgery, some practitioners have been willing to perform them without careful clinical deliberation. Since all anesthesiologists are trained to enter the epidural space without fluoroscopic guidance, such "blind" ESI is readily available. But although there are indeed many clinical reports of excellent outcomes and low risk, controlled studies of outcomes have been – at best – limited and the best designed studies have utilized the outdated and unreliable blind interlaminar approach to the epidural space. Indeed, several of these studies have refuted the benefits of ESIs in this patient population.[63,64]

A study done to compare the effectiveness of caudal vs. transforaminal epidural steroid injections in patients with primary lumbar radiculopathy concluded that the effectiveness of transforaminal epidural steroid injection is comparable to that of caudal epidural steroid injection (approximately 60%). However the increased

complexity of transforaminal epidural steroid injection is not justified for primary cases and may have a more specific role in recurrent disease.[65]

In 2008 a study done at Surrey to evaluate the effect of caudal epidural steroid injections with/without bupivacaine in patients with lumbosacral radiculopathic pain gave evidence that epidural bupivacaine injection appears to enhance the effect of epidural steroid injection.[5]

ANATOMY

Vertebral column

The central axis of the human skeleton is formed by the vertebral column. Main function of the vertebral column is to protect the spinal cord. The vertebral column comprises of 33 vertebrae.[66]

- Cervical - 7
- Thoracic - 12
- Lumbar - 5
- Sacrum - 5 (fused) to 1
- Coccyx - 4 (fused) to 1

Curves of spine

In adult, the vertebral column has four curves which have significant effect on spread of drugs in sub arachnoid space namely.[66]

- Cervical curve - Convexity anterior
- Thoracic curve - Concave anterior
- Lumbar curve - Convexity anteriorly

In adults the curves of the spine are important when patient is supine or horizontal. The highest point of cervical and lumbar curves in supine position are at cervical (C) five and lumbar (L) five; lowest points of thoracic and sacral are at thoracic (T) five and sacral (S) two respectively.[66]

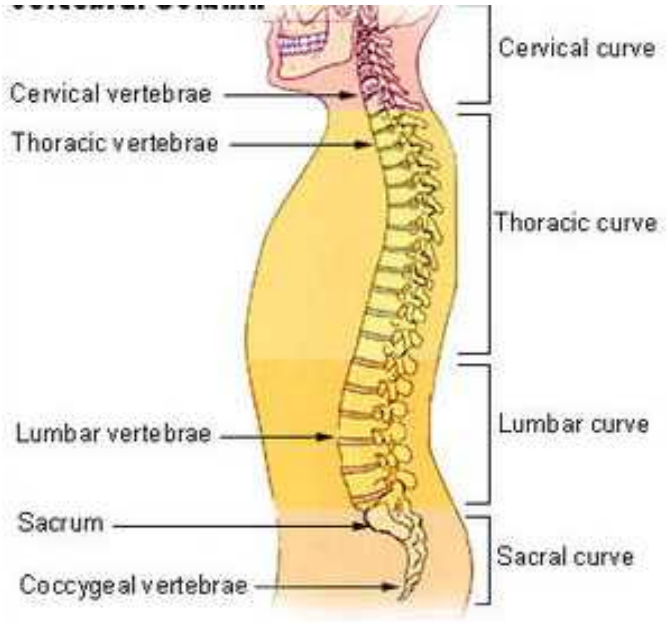


Figure 1 The Spinal Column

The various ligaments & muscles attached to the column lend to its great strength & ruggedness & also great flexibility.

The familiarly 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 2)

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

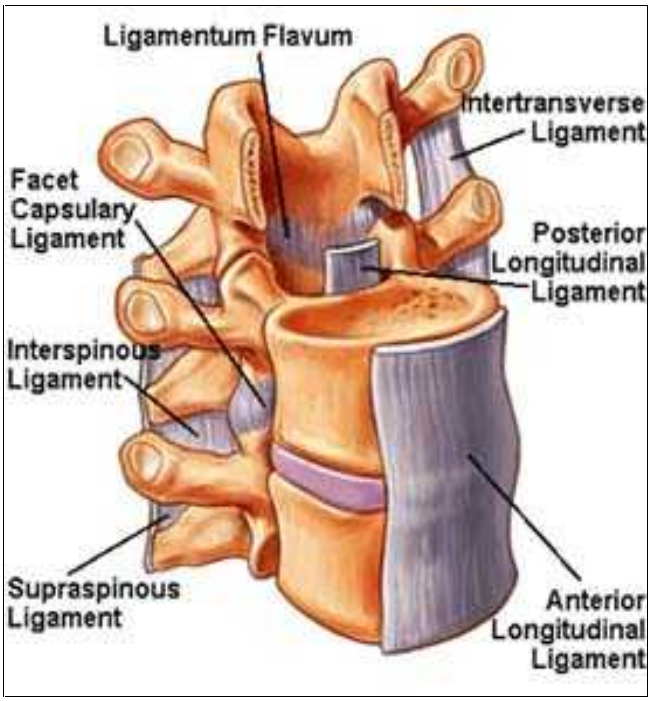


Figure 2. Functional Spinal Unit

Vertebral ligaments[67]

Vertebral column is bound together by following ligaments which give stability and elasticity.

Supraspinous ligament: This is a strong fibrous cord which connects apices of spinous processes from where it continues as the ligamentum nuchae (Figure 2).

Interspinous ligament: This is a thin membranous ligament which connects spinous processes blending anteriorly with ligamentum flavum and posteriorly with supraspinous ligament (Figure 2).

Ligamentum flavum: This ligament comprises yellow elastic fibers and connects adjacent lamina. Laterally this ligament begins at the root of articular processes and extends posteriorly and medially to the point where laminae join to form spinous process (Figure 2).

Longitudinal ligaments: There are two longitudinal ligaments (anterior and posterior) that bind vertebral bodies together (Figure 2).

Lumbar vertebrae[68]

A typical lumbar vertebra consists of (Figure 3);

- A kidney shaped body.
- Two pedicles directed backwards from the upper part of the body.
- Two transverse processes which are slender

- Two laminae meeting posteriorly and enclosing the triangular vertebral foramen.
- Spinous processes which are thick, broad and quadrilateral in shape.
- Two upper and lower articular processes which prevent rotation but allow limited flexion and extension between contiguous vertebrae.

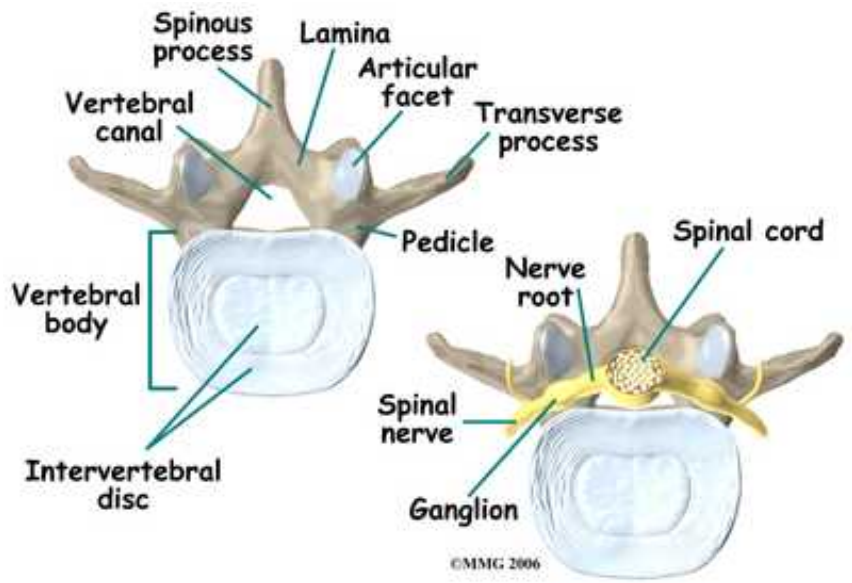


Figure 3. Lumbar Vertebra

Topographical Line of Tauffier[69]

This is a horizontal line across the back between the crests of the ilia passing over the spine of the 4th lumbar vertebra in the upright position. In a patient lying in the lateral position it may also pass through L4 and L5 interspaces. The highest point of iliac crest is used to identify the L4 and L5 interspace during spinal anesthesia.

Intervertebral Discs [68]

These are principle connecting link between vertebral bodies. The intervertebral discs account for about 25% of the length of the spine. They have two parts. The outer fibrous part called the annulus fibrosus is made mostly of fibrous tissue, while the softer core of the disc is the nucleus pulposus. Atrophy of the discs along with osteoporosis of the vertebra leads to decreased height and kyphotic deformity of old age.

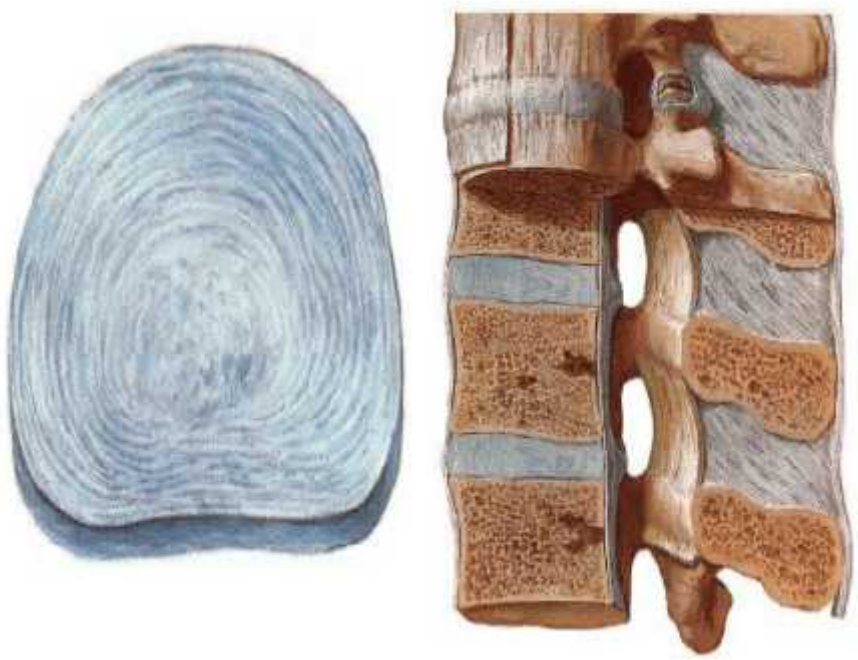


Figure 4. The Intervertebral Disc

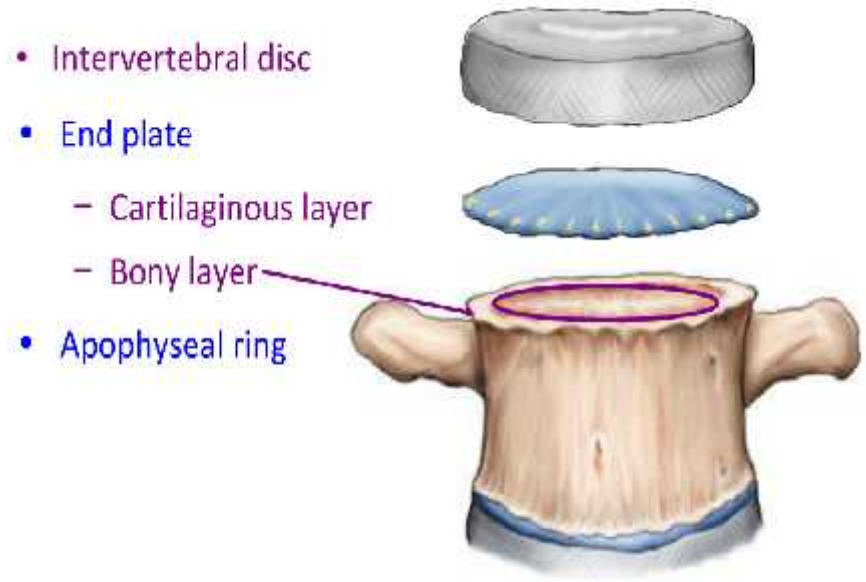


Figure 5. Intervertebral disc

The discs contribute to a third or fourth of the length of the articulated vertebral column, and variations in their thickness anteriorly and 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 fibrosus** (Figure 6) 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 and 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.

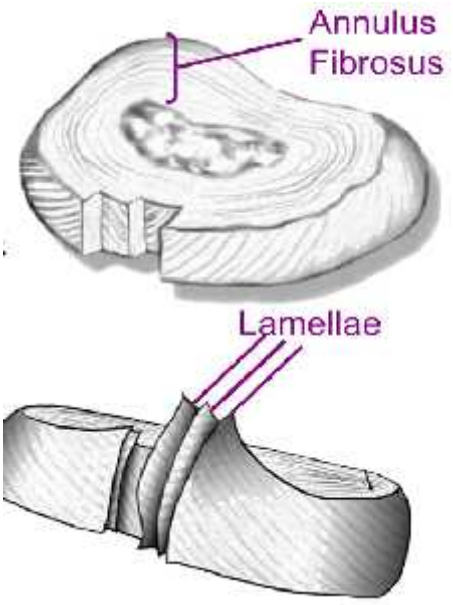


Figure 6. Annulus fibrosus

The **nucleus pulposus** (Figure 7) 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.

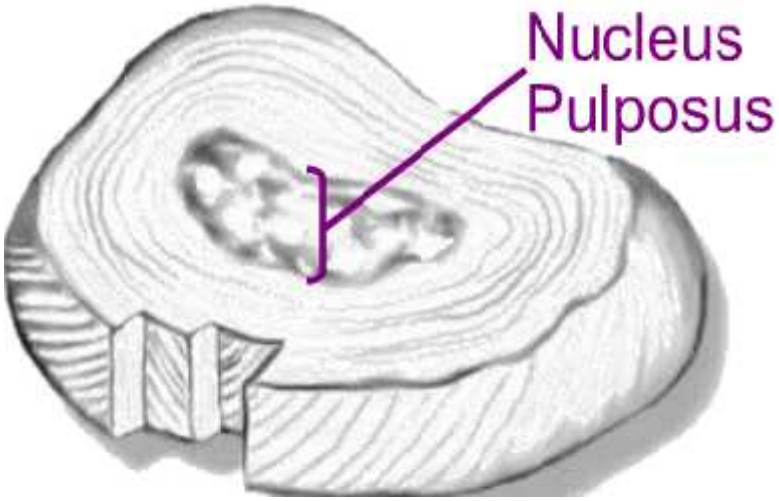


Figure 7. Nucleus pulposus

The **nourishment** of the disc (Figure 8 a) is through the lymph which diffuses from the marrow cavity of adjacent vertebrae, through perforations in the cartilage plates. Some investigators suggest 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 fibrosus 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.

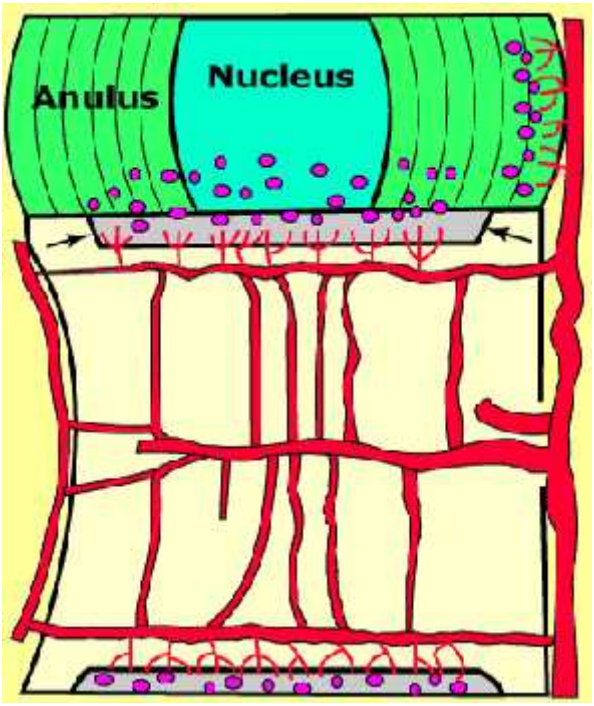


Figure 8 a. Nutrition of the disc

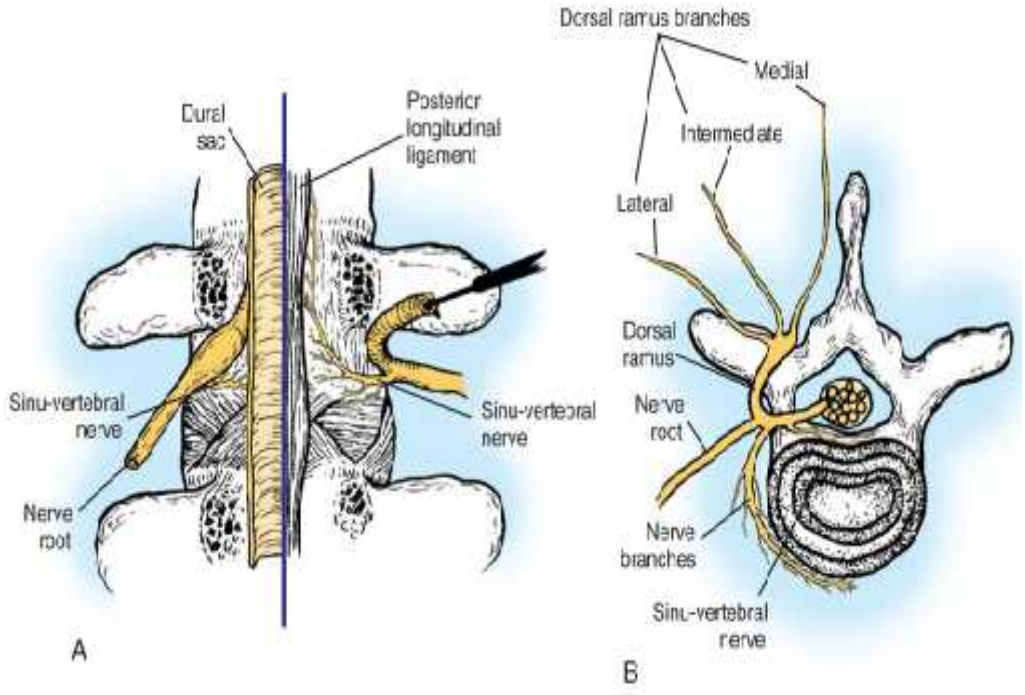


Figure 8 b Nerve Supply of the Disc

The **nerve supply** (Figure 8 b) 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 when the force is removed. Similarly in flexion or extension, the annulus fibrosis on the concave side is not compresses as it is pushed away by the nucleus pulposus.

Types of Disc Lesions (Figure 9)

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:

Atrophia Simplex of Bartschi, which is the simplest form of primary degeneration of disc, which may or may not be characterized by persistent low back pain.

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 and sciatica and 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.

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.

- The mobile type (concealed ruptured disc or intermediate prolapse). In this type, the prolapsed portion returns to the same tear intermittently.
- The fixed prolapse, when the prolapsed nucleus can no longer be reduced.

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

- A mobile protrusion may become fixed.

- Calcification of the herniated disc.
- Adherence of the disc to the dura mater and extra-dural roots, and 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:

- 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.

- 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.

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

- 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.
- Less commonly free lying completely extruded disc material in the epidural space, which may or may not be embedded in dense fibrosus tissue.
- Intermittent herniation of Falconer, or concealed disc of Dandy.

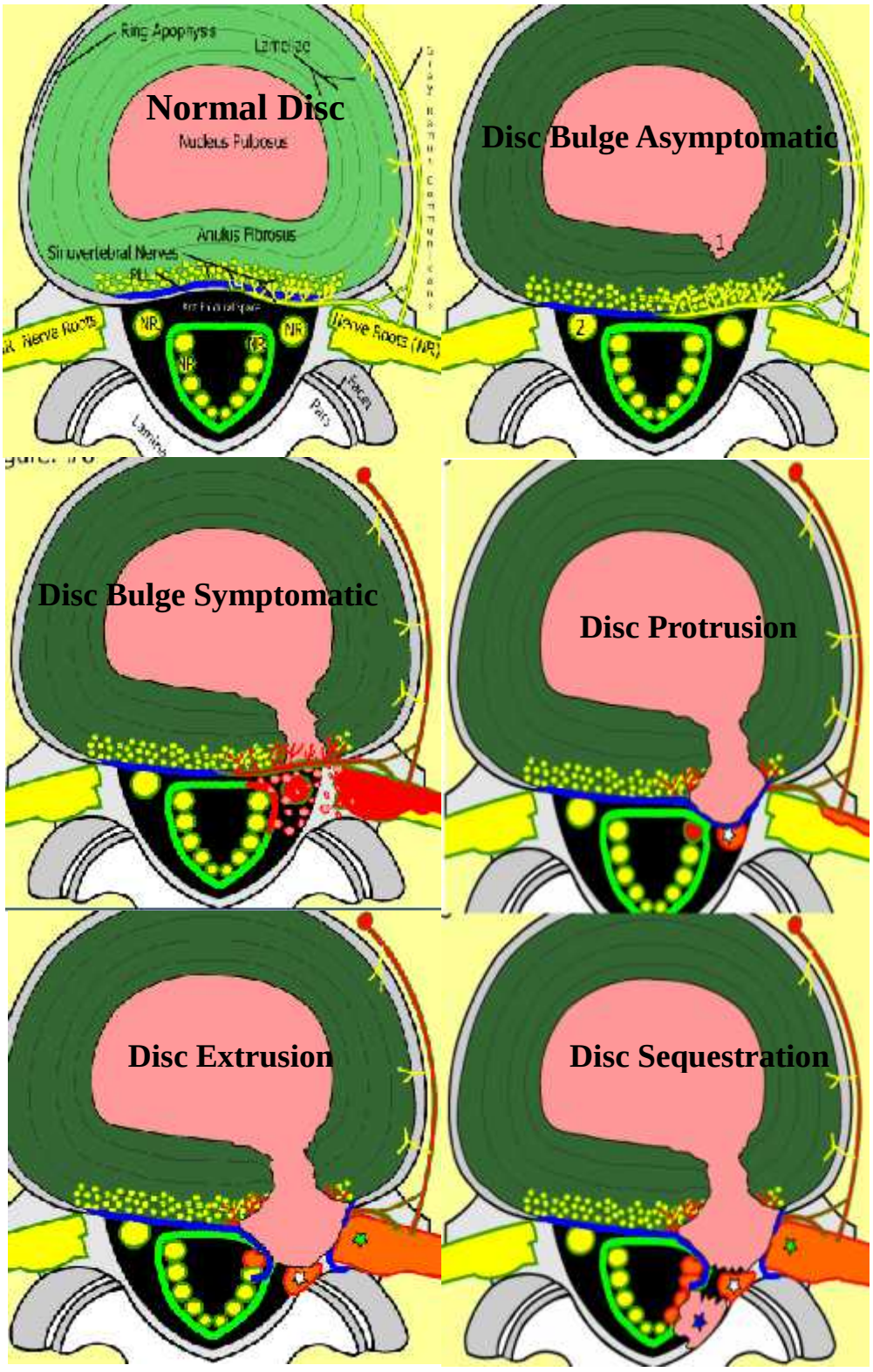


Figure 9 Types of Disc Lesions

Spinal Canal

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).

Contents – Lumbosacral Spinal Canal (Figure 10)

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

(b) Cauda Equina.

(c) Cerebrospinal 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 medullaris, 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

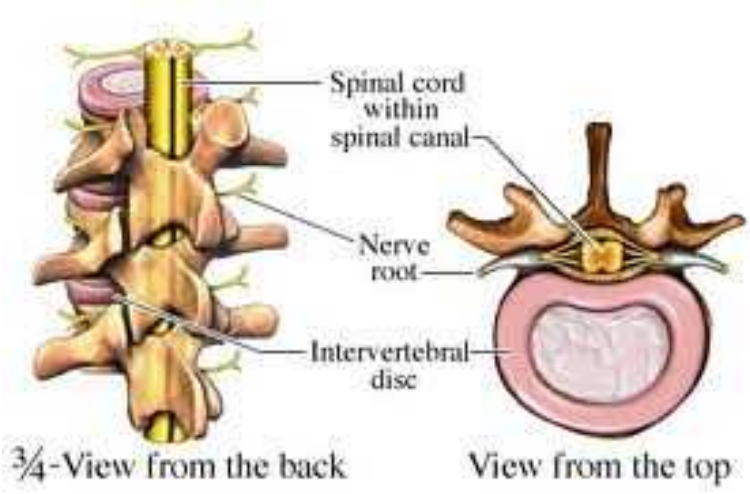


Figure 10 - Contents of the Spinal Canal

The Epidural Space & Sacral Canal (Figure 11,12)

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 favours 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; they enter at each intervertebral foramina, & lie chiefly along the lateral part of the epidural space, where they supply the adjacent vertebrae, ligaments, & spinal cord.

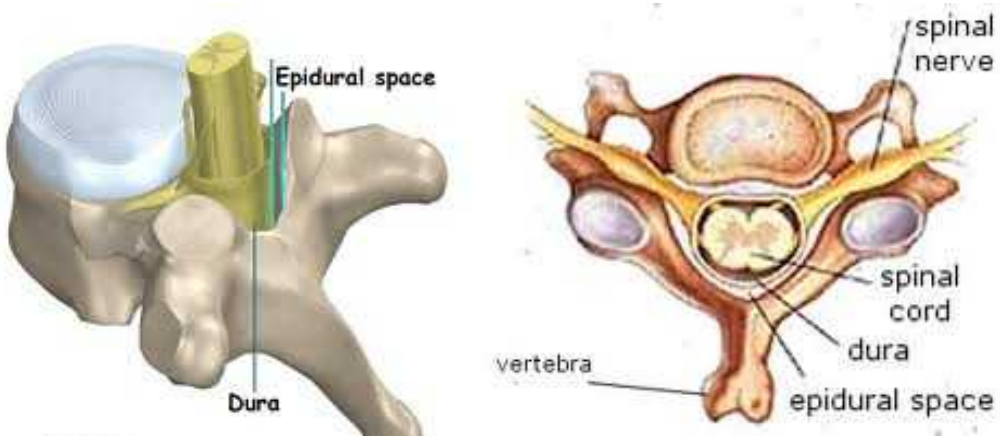


Figure 11 – Epidural Space

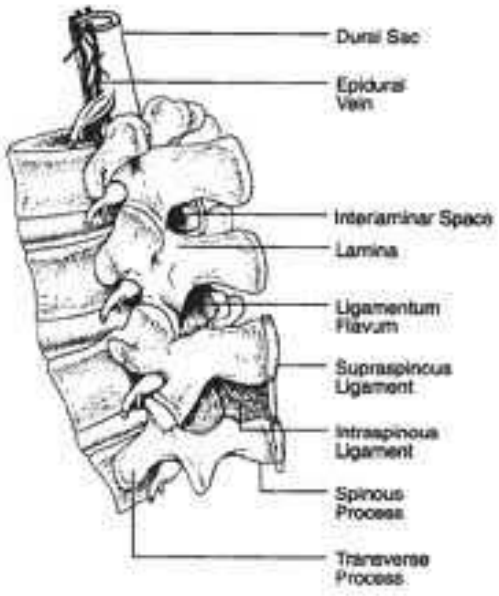


Figure 12 Epidural Space

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 to various theories put forward it can be said that 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.

The contents 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 %.

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 %).

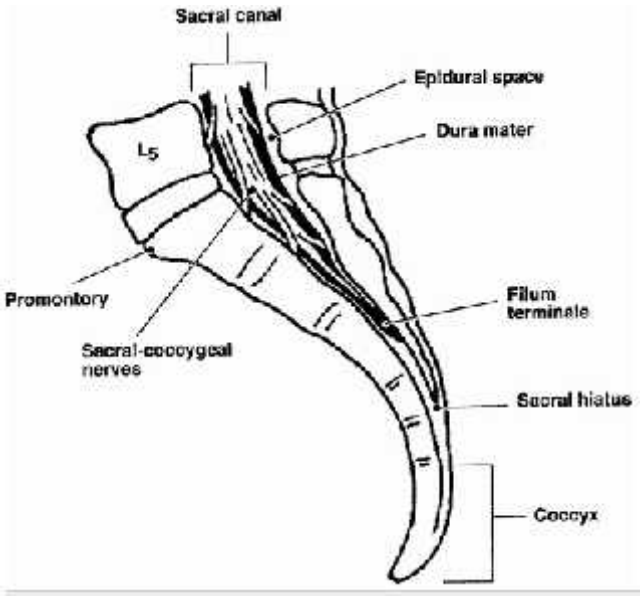


Figure 13 – Sacral Canal

Abnormalities of the Sacral Hiatus [34]

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.

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 along the sacral hiatus, the point of **loss of resistance** to the injection of a liquid or of air is noted. This denotes entry into the space.

2. Spring Loaded Syringe

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

3. 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% and a specificity of 20%. The whoosh test had a sensitivity of 80% and a specificity of 60%. The whoosh test is superior to clinical judgment in detecting incorrect caudal needle placement [35].

4. Fluoroscopic Contrast Dispersion Method

Isohexol is used as a dye 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 & dye related complications.[70,71]

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.[73] Stitz & Sommer support a high rate of success, as long as readily palpable anatomic landmarks are properly recognized.

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 and 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.

Functions

The spine as a whole has static & dynamic functions. It carries the head & shoulder girdles together with the upper limbs & transmits their weight along with the weight of the thorax to the pelvis. The assumption of the erect posture has resulted in the lower lumbar discs being submitted to more shock & strain than the others. In addition, the amount of movement that takes place between the thoracic segment & the sacrum is the most at the lumbosacral junction. The above facts explain the maximum incidence of disc changes at these levels. Similarly, the twisting & torsion strains along with the burden of carrying the weight of the head result in frequent changes in the cervical disc.

The mobility & curvature of the lumbar spine varies with race. The African spine is more mobile than the European.[74] It is equally true of the spine in India & the Orient,

where most people squat on the floor, & where the majority of housework is done by stooping forwards.[75]

The segmental arrangement of the mobile, elastic disc tissue serves as an excellent shock absorber both against individual blows & the continuous stress exerted cephalo-caudally. At the same time this arrangement permits some torsion of the spinal column. All the shearing forces are countered by the construction of the fibrous rings & the strong interlocking between the disc & osseous vertebrae. The S-like curvature of the spine absorbs the forces of blow & shocks. The largest mobile segment of the spine is the disc. Luschka called it an "amphiarthrosis", suggesting that the nucleus pulposus be considered the articular cavity, the annulus fibrosis, the ligamentous apparatus & the cartilaginous plate covering the superior & inferior surface of the vertebrae the articular cartilages. The cartilaginous plates along the edges of the fibers are attached to the vertebral body only by a layer of calcium. The intervertebral disc & the cartilaginous plate therefore, form a physiologic unit & are together subject to chronic diseases & degeneration.

Spinal cord[68]

The average length of the spinal cord in males is 45 centimeter (cm) and females it is 42 cm. The spinal cord is a continuation of the medulla oblongata below the level of foramen magnum and it tapers off into conus medullaris. A delicate fibrous filament descends to the back of first segment of coccyx from apex of conus medullaris. This is known as the filum terminale. At birth, spinal cord ends at the level of lower border of lumbar (L) three vertebrae. In the adult, the vertebral level of termination of spinal cord may be as follows;

- Lower border of L1 - 50%
- Upper border of L2 - 40%
- Upper border of L3 - 3%

From the spinal cord 31 pairs of spinal nerves arises made of a ventral and a dorsal root. These anterior and posterior roots cross the subarachnoid space, pass through the duramater and extradural space independently and unite at the level of intervertebral foramen to form spinal nerve trunks, which soon divide into anterior and posterior primary divisions.

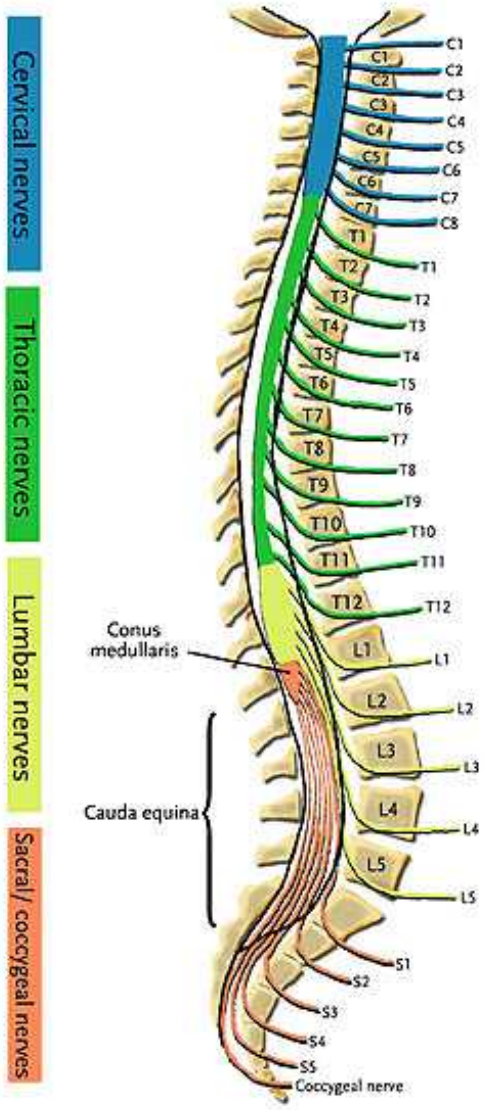


Figure 14. Spinal nerve roots

The amount of white matter declines progressively from the cervical to the lumbar region. The gray matter is greatly increased in the both the lumbar and cervical enlargement

Blood Supply of Spinal Cord[68]

The arterial supply is from the anterior and posterior spinal arteries. The anterior spinal artery is a single vessel lying in front of the anterior median fissure. It arises from the meeting of two small arteries, one given off from each vertebral artery at the level of the foramen magnum. It descends along the whole length of the cord receiving small communications from the intercostal and lumbar arteries; to provide the extra blood supply needed in the cervical, thoracic and lumbar enlargements. There are two posterior spinal arteries-one on each side. They are derived at the base of the brain directly from the vertebral artery or more often from a primary branch of each vertebral artery. They supply the posterior one-third of the spinal cord. This supply is augmented by spinal branches of vertebral, ascending cervical, posterior intercostals, lumbar and lateral sacral arteries, which pass through the intervertebral foramina.

Venous drainage is through a plexus of anterior and posterior veins in the neck, azygous veins in the thorax, lumbar veins in the abdomen, and lateral sacral veins in the pelvis. There is no anastomosis between the anterior and posterior spinal arteries. The longest of the feeder arteries is the radicularis magna (artery of Adamkiewicz), which supplies the anterior spinal artery in the area of the lumbar enlargement of the cord. It enters by way of a single intervertebral foramen (78% of the time on the left) between the T8 and L3 foramina.

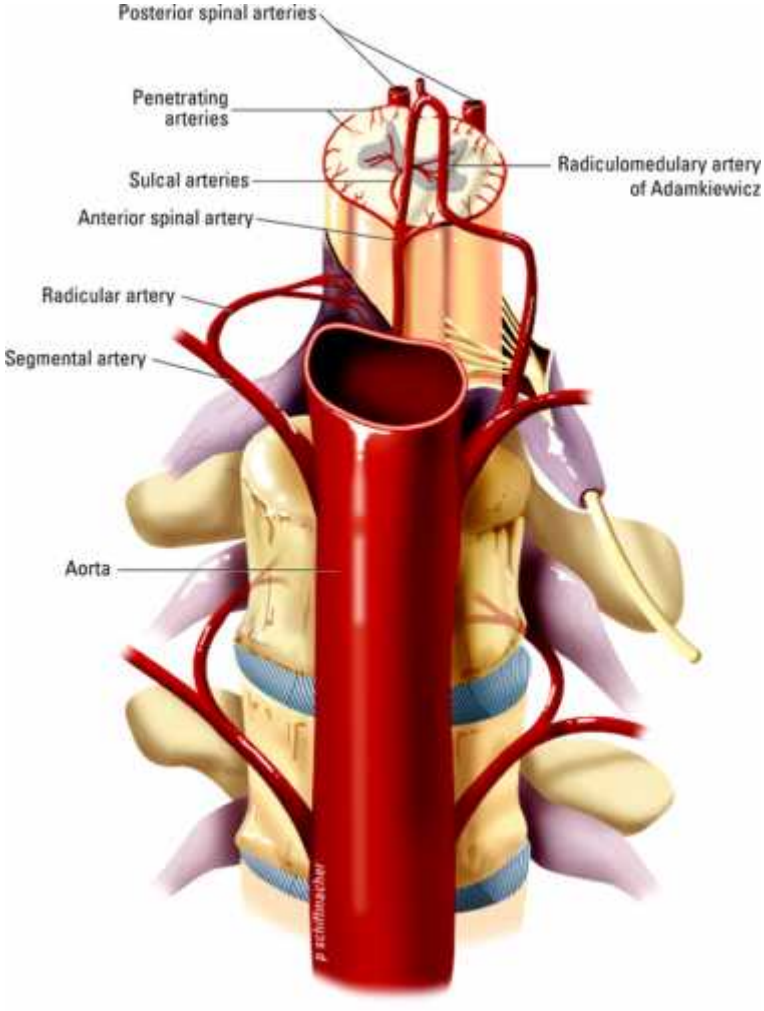


Figure 15. Blood Supply of Spinal Cord

Meninges[76]

The spinal cord has three covering membranes from inward to outward. They are the piamater, the arachnoid mater and the dura mater. The dural sac of spinal duramater is the continuation of meningeal layer of the cranial duramater. It is a circular sac or sleeve surrounding the spinal cord. Above, it is firmly attached to the circumference of the foramen magnum.

Duramater[76]

It is the outermost membrane, the fibers of which run longitudinally. Although continuous, it can be described in two parts: the cranial and the spinal. The cranial dura consists of an outer layer (endosteal), which lines the skull, and an inner layer (meningeal), which invests the brain and folds inward to form the falx cerebri and tentorium cerebelli.

Arachnoid Mater[76]

The arachnoid mater is a delicate non-vascular membrane applied closely to the dura mater. The lower extent of dural sac is as follows;

- S2 vertebra 35%
- Below S2 40%
- Above S2 25%

Below this the dura continues as the filum terminale. The subarachnoid space is the space between the arachnoid and piamater. This space is traversed by cobweb trabeculae and by the cranial and spinal nerves; it is bathed in spinal fluid. The space is annular in the cranial and thoracic vertebrae and is about three mm deep. Below the first lumbar vertebrae it is circular.

Subarachnoid Space[76]

The space between the arachnoid and piamater is called the subarachnoid space and is filled with cerebrospinal fluid and contains numerous arachnoid

trabeculae which form delicate sponge like mass. This space has three divisions which are free communication to each other: cranial (surrounding the brain), spinal (surrounding the spinal cord) and root (surrounding the dorsal and ventral nerve roots). In the spinal cord these nerve roots are covered only by piamater and bathed in CSF. As these spinal nerve roots pass beyond the spinal dura and traverse the epidural space, they carry with them all the three meningeal layers and have a distinct epidural, subdural, subarachnoid and subpial spaces. The subarachnoid space extends separately along both the dorsal and ventral roots to the level of dorsal root ganglion, where arachnoid and piamater continue as perineural epithelium of peripheral nerve.

Piamater [76]

The piamater, the innermost membrane is a vascular sheath which closely invests the brain and spinal cord.

Cerebrospinal Fluid[76]

It is a clear colourless fluid found in the cranial and spinal subarachnoid spaces and in the ventricles. CSF is mainly formed by either secretion or ultra filtration from the choroidal plexus of lateral ventricles. CSF flows from the lateral ventricles into the third ventricle through the foramina of Monro into the fourth ventricle through the Aqueduct of Sylvius into the cerebromedullary cisterna (cisterna magna) through foramen of Magendie and foramina of Luschka. From the cisterna magna, CSF enters subarachnoid space circulating around brain and spinal cord before being absorbed into the arachnoid granulations over the cerebral hemispheres.

Composition of cerebrospinal fluid

- Specific gravity : 1.003 to 1.009 at 37^0C.
- Volume : 120 ml to 150 ml (25 ml to 35ml in spinal space).
- CSF pressure : 60 to 80 mm Hg in lumbar space.
- pH : 7.27 to 7.37
- PCO_2 : 48 mm Hg
- HCO_3 : 23 mEq/L
- Sodium : 135 to 145 mEq/L
- Calcium : 2 to 3 mEq/L
- Phosphorous : 1.6 mg/dl
- Magnesium : 2 to 2.5 mEq/L
- Chloride : 15 to 20 mEq/L
- Proteins : 23 to 38 mg/dl

Evolutionary Mechanisms in Chronic LBP

Spinal pain usually arises from damage to or degenerative changes in the spinal nerves, intervertebral discs, facet joints, muscle/fascia, and dural tissue surrounding the spinal nerve roots.[77]

Facet joints may be responsible for 14–45% of cases of LBP,[77] most often as a result of degenerative changes or trauma that causes inflammation of the joint capsule from overloading. Degenerated and herniated discs are other common causes of LBP and sciatica. Though the mechanism is still not certain, animal studies indicate that

when there is nucleus pulposus tissue in the epidural space, it induces an inflammatory response, neurotoxicity, and thrombosis, all of which can lead to nerve root ischemia and irritation.[78] Fissured, degenerative discs are thought to cause pain by allowing growth of sensory fibers from the sinuvertebral nerve into the inner layer of the annulus fibrosis and nucleus pulposus which are normally not innervated.[79]

It remains unclear to what degree nerve root compression or irritation is responsible for radicular pain and LBP. In general, sciatica type pain is most-likely due to nerve root compromise (radiculopathy), while axial back pain is more indicative of a "mechanical origin" such as facet syndrome, discogenic pain, or muscular pain. Radicular dominant pain is more likely to respond to epidural steroid injections than back-dominant pain.

Chronic LBP (cLBP) is not the same as acute LBP that persists for a greater duration. Usually 6-7 weeks is sufficient for healing to occur in most soft-tissue or joint injuries; however, 10% of LBP injuries do not resolve in this period. The evolution of cLBP is complex, with physiological, psychological, and psychosocial influences. These influences can be divided into 3 major categories, with subcategories, as follows:

- Neurophysiological mechanisms
    - Peripheral
    - Peripheral to central
- Psychological mechanisms
    - Behavioral

  - Cognitive-affective
  - Psychophysiological
- Barriers to recovery
  - Medical and surgical
  - Physical
  - Psychological
  - Neuropsychological
  - Social

Neurophysiological mechanisms

Peripheral mechanisms may reinforce nociception when the source of pain persists. If an ongoing pathological condition causes the peripheral pain stimulus, continuous nociception may induce repetitive stimulation and sensitization of pain receptors and nerve fibers so that they adversely respond to even mild or normal sensory stimuli (i.e., allodynia). Furthermore, the liberation of algogenic and other substances from damaged tissues may induce changes in the microenvironment by means of neuroactive, biochemical, inflammatory, or vasoactive effects that activate or increase the sensitivity of nociceptors.

Peripheral-to-central processing may also modify nociception. Persistent tissue damage may stimulate afferent nerve fibers that project to internuncial neurons in the spinal cord and thereby set up neuronal loops of continuous, self-sustaining abnormal reverberating nociceptive activity. Peripheral inhibition, a mechanism for reducing the intensity of an afferent pain signal, may be impaired owing to persistently

malfunctioning or diseased large peripheral myelinated fibers, which normally dampen nociception (e.g., peripheral neuropathy, epidural scarring, and chronic herniated disk material).

Ectopic impulse generation is a theoretical mechanism Wall and Gutnick proposed.[80] Damaged sensory nerves, affected by conditions such as neuromata or demyelinating lesions in peripheral nerves, produce aberrant signals.

CNS bias of the signal may occur in the spinal cord, brainstem reticular formation, or cortex. The brainstem reticular formation acts to direct the attention of the CNS toward or away from central and peripheral stimuli. Depending on the degree of focus, or the lack thereof, the transmission of pain signals may be either enhanced or inhibited. Furthermore, cortical influences, such as cognitive and affective disorders, may affect the intensity of the processed pain signal.[32]

Psychological mechanisms[32]

Psychological manifestations are 3-fold; they include behavioral, cognitive-affective, and psycho physiological mechanisms. Guarded movements, nonverbal and verbal expressions of pain, and inactivity are called pain behaviors. Normal healthy behavior patterns may become extinguished when these verbal and nonverbal pain behaviors are reinforced by environmental factors.

Cognitive-affective mechanisms often contribute to the perception of chronic pain. Pain complaints are common in depressed individuals, and patients with chronic pain frequently become depressed. Depression acts though biochemical processes

similar to those that operate in chronic pain; this may enhance symptoms through a synergistic relationship. Patients with pain who are depressed may illogically interpret and distort life experiences, further complicating the feasibility of treatment or employment.

Psycho physiological mechanisms naturally triggered by pain and injury can lead to generalized muscle over activity, increased fatigue, and other pain problems (tension myalgia, headache). The emotional stress that pain induces tends to heighten norepinephrine activity and the general activity of the sympathetic nervous system, which may further amplify nociception by means of peripheral or central mechanisms.

Barriers to recovery[32]

Barriers to recovery may be premorbid, result from traumatic injury, or develop over time as a result of psychological and environmental influences. These barriers strongly influence chronicity and the patient's prognosis. For example, medical problems, such as diabetes or heart disease, may make the patient a poor candidate for rehabilitation or surgery. Failed back surgery may create permanent physical and psychological obstacles.

Patients differ in their inherent capacity to exercise. Deconditioning syndrome, a term Mayer coined, is caused by prolonged reduction of physical activity due to chronic low back pain (cLBP). This syndrome is associated with a gradual reduction in muscle strength, joint mobility, and cardiovascular fitness, which over time may become a self-sustaining and independent component of the individual's musculoskeletal illness.

Preexisting psychological factors may combine with lower back injuries to create a pain syndrome with predominantly psychiatric features. Psychiatric interviews of 200 patients with cLBP entering a functional restoration (FR) program revealed that 77% met lifetime diagnostic criteria for psychiatric syndromes, even when the category of somatoform pain disorder was excluded. In addition, 51% met the criteria for at least 1 personality disorder. Psychological barriers to recovery include those listed below.

- Premorbid factors
    - Depression
    - Predisposition toward somatoform pain disorder
    - Psychoactive substance-abuse disorder
    - Personality disorder or traits thereof
    - Anxiety disorders including panic disorder
    - Childhood sexual abuse
    - Cognitive process
    - Psychosis, delusional pain
- Traumatic factors
    - Anxiety/panic
    - Fear
    - Psychophysiological response
    - Loss of control
    - Abnormal dependence

- Posttraumatic factors
    - Anxiety, panic
    - Depression
    - Posttraumatic stress disorder
    - Anger/hostility
    - Iatrogenic substance abuse
    - Somatoform pain disorder
    - Symptom magnification
    - Increasing time since injury
    - Disability mindset

Personality disorders or related traits often affect the prognosis. People with borderline personalities may acquire pain as a method for structuring an otherwise empty existence, whereas patients who are narcissistic may acquire pain and seek medical attention as a way of preventing more serious illness. Those with an antisocial personality are often exploitative and prone to complications. Individuals with depression are prone to chronic pain or to have pain as a symptom. Other personality disorders or disorders that may influence chronic pain include the paranoid, passive-aggressive, and avoidant conditions.

Previous learning and role models also affect the patient's prognosis and treatment outcome. An individual's cognitive or attribution style (the patient's tendency to catastrophize, over generalize, personalize, or selectively attend to negative aspects of the pain experience) heavily influence prognosis and treatment outcomes. The physical and emotional trauma that occurred during the injury or that

was encountered during the ordeal of convalescence may contribute to the psychosocial milieu and create a host of emotional responses, including anxiety and fear.

Psycho physiological responses may be reinforced and include nightmares, palpitations, diaphoresis, headaches, dizziness, irritability, and fatigue. Patients are often overwhelmed and have feelings of abnormal dependence. They perceive a loss of control and look to their physician, attorney, or family for guidance. Some advisors may be over solicitous or encourage compensation-seeking or litigation, creating further barriers to recovery.

Enduring prolonged pain also may cause emotional disturbances. Depression has already been mentioned as a common partner to chronic pain and is enhanced by the loss of physical function, low self-esteem, loss of employment, and financial insecurity. Heightened anxiety may be secondary to continued pain and cause associated life disruption. Fear of injury and panic symptoms may also enhance anxiety and complicate the person's recovery. Anger or hostility directed at the workplace or perceived ineffective medical care may hinder communication with physicians, employers, family, and friends. As the length since the injury increases, the aggregation of posttraumatic emotions becomes increasingly complex; avoidance learning and deactivation further complicate the situation.

As these barriers accumulate, the probability of a poor prognosis rises. Neuropsychological factors may preexist or come into effect due to the injury. Limited cognitive function, either premorbid or from brain injury, may limit the

patient's capacity to make decisions or succeed in a rehabilitation program. Neuropsychological barriers to recovery include the following:

- Intelligence
- Brain injury
- Dementia or other organic mental syndromes

Environmental and social influences may play the strongest role in determining the patient's prognosis for chances of recovery. Job dissatisfaction or conflict is a key predictor of chronic LBP with disability. Compensated unemployment may reinforce chronicity in these cases. Family, financial, and legal issues also affect chronicity. A patient with chronic LBP may be unable to return to a previous job that was strenuous or involved heavy lifting and may be poorly equipped to pursue alternative vocational options because of a lack of education. Older individuals may have reduced capacity for work and less vocational potential; therefore, loss of compensation becomes an overriding issue. Social barriers to recovery include the following:

- Job dissatisfaction or conflict
- Compensated unemployment as a disincentive
- Family or spousal dynamics
- Perception of the norm, ie, family history
- Legal influences
- Financial security
- Limited education or vocational potential

- Age-related factors

Clinical evaluation[32]

In most cases, chronic LBP has been investigated with the appropriate physician evaluation and perhaps imaging studies. Characterization of the pain as mechanical is a primary goal when a history is obtained from a patient with cLBP and sciatica. Mechanical or activity-related spinal pain is most often aggravated by static loading of the spine (prolonged sitting or standing), long-lever activities (vacuuming or working with the arms elevated and away from the body), and levered postures (forward bending of the lumbar spine). Pain is reduced when multidirectional forces balance the spine like, walking or constantly changing positions) and when the spine is unloaded (reclining). Patients with mechanical LBP often prefer to lie still in bed, whereas those with a vascular or visceral cause are often found writhing in pain, unable to find a comfortable position.

Unrelenting pain at rest should suggest a serious cause, such as cancer or infection. Imaging studies and a blood workup are usually mandatory in these cases and in cases with progressive neurological deficits. Nonphysiological or implausible descriptions of pain may provide clues that operant or other psychosocial influences coexist.

Physical Examination[32]

Physical examination is important to confirm a mechanical or benign cause for the patient's LBP. Observations of verbal and nonverbal behaviors suggesting

symptom magnification should be noted. Inspection of the spine requires the patient to disrobe. Open-back gowns give the physician only 1 view of the spine; therefore, swimming attire is often appropriate for complete, 360° inspection. Leg-length discrepancy and pelvic obliquity, scoliosis, postural dysfunction with forward-leaning head and shoulders, or accentuated kyphosis should be noted. Physicians' preferences vary with regard to the importance of testing range of motion; however, just asking the patient to bend forward often enables the most worthwhile observations.

The patient is asked to drop his or her head and shoulders forward and then move slowly into forward bending. Normal forward bending is revealed when the patient recruits from each cephalic segment to the level below, and so on, progressing from the cervical spine through the thoracic and lumbar region, where flexion of the hips completes the excursion into full flexion. Patients with clinically significant mechanical back pain or lumbar segmental instability usually stop cephalic-to-caudal segmental recruitment on reaching the thoracolumbar junction, or sometimes the involved lumbar level. To continue forward bending, they then contract their lumbar muscles to brace the mechanically compromised segment and then continue recruitment in a reverse direction, beginning with motion through the hips, then proceeding cephalad, level to level, completing the excursion of the spine to the erect posture.

In cases of severe mechanical back pain and segmental instability with regional muscular spasm, the patient often reports an inability to perform any flexion below a thoracic spinal level. Any soft-tissue abnormalities and tenderness to palpation should be recorded. Palpation of lumbar paraspinal, buttock, and other

regional muscles should be performed early in the examination. The examiner should palpate and note areas with superficial and deep-muscle spasm, and he or she should identify trigger points and small, tender nodules in a muscle that elicit characteristic regional referred pain.

Dissociation of physical findings from physiological or anatomical principles is the key with patients in whom psychological factors are suspected to be influential. Examples of this phenomenon include non dermatomal patterns of sensory loss, nonphysiological demonstrations of weakness (give-way weakness when not caused by pain, or ratchety weakness related to simultaneous agonist and antagonist muscular contraction), and dissociation between the lumbar spinal movements found during history-taking or counseling sessions from movements observed during examination.

The assessment of Waddell signs has been popularized as a physical examination technique to identify patients who have nonorganic or psychogenic embellishment of their pain syndrome. One of the examination techniques that Waddell proposed is simulated rotation of the hips en masse with the lumbar spine without allowing for spinal rotation;this maneuver normally does not cause pain. Another is the application of light pressure on the head, which should also be painless. Likewise, gentle effleurage of superficial tissues is unlikely to cause pain. Other techniques include a striking dissociation between testing straight leg raising with the patient sitting versus supine and the examiner's discovery of nonphysiological weakness and/or sensory deficits by the patient.

Besides pain, compression of the root may produce paraesthesia, 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).[81] The signs & symptoms of specific root involvement are summarized below in Table 1.

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 5 PIVD, L 5 Foraminal Stenosis	Anterolateral Leg, Dorsum of the foot, Great Toe	Extensor Hallucis, EDL, EDB, Gluteal Medius	Usually None
S 1	L5 – 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):

Straight leg raising with the patient supine should produce ipsilateral leg pain between 10° and 60° to be declared positive. Straight leg raising that produces pain in

the opposite leg carries a high probability of disk herniation, and an investigation should be considered, especially if neurological evidence for radiculopathy is present. Nonspecific complaints, overtly excessive pain behavior, patient contraction of antagonist muscles that limit the examiner's testing, or tightness of buttock and hamstring muscles are commonly mistaken for positive results on straight leg raising.

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 spondylolysthesis, 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.[81]

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 16, 17, 18)

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.

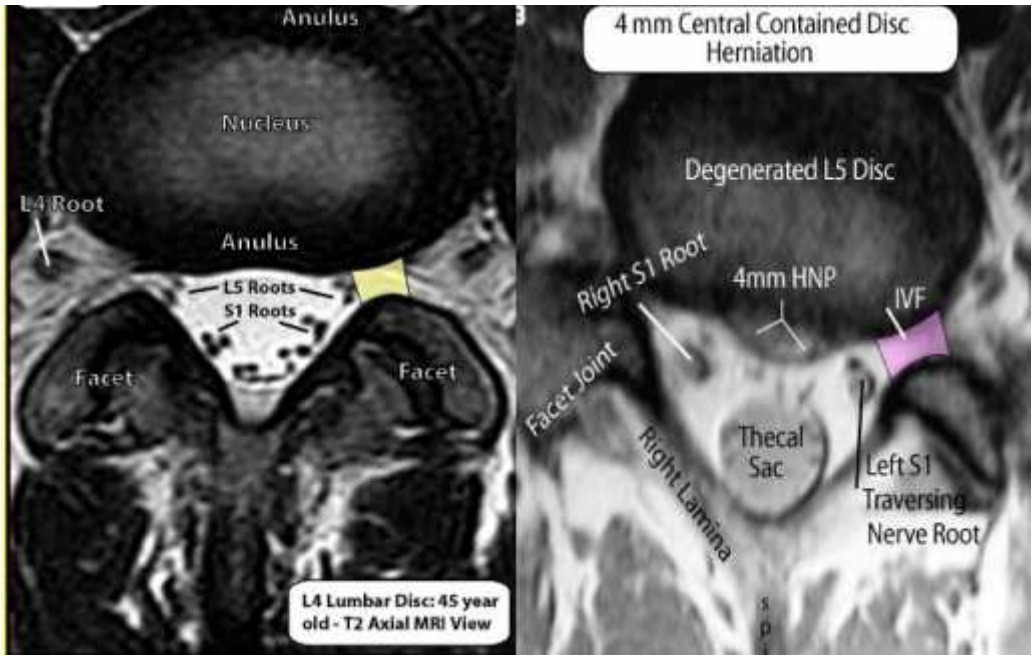


Figure 16 MRI Image of a Protruded Disc (Transverse Section)

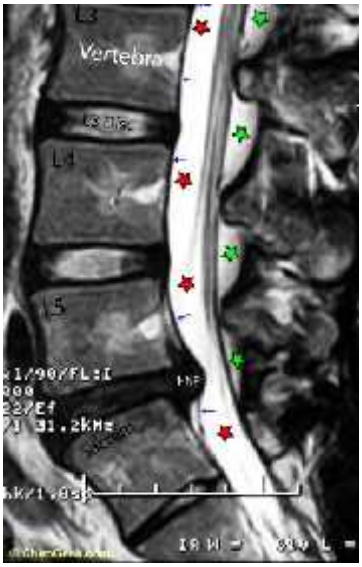


Figure 17 - MRI Image of a Protruded Disc (Saggital Section)

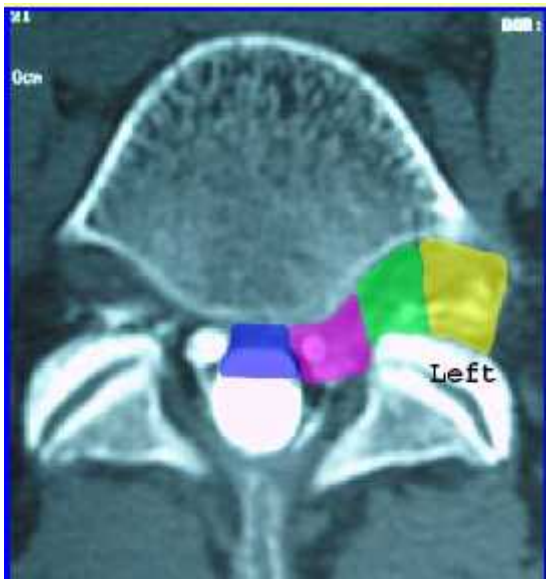


Figure 18 – Zones of Disc Protrusion

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 clinicians 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.

Treatment

General principles[82]

The goals of treating chronic low back pain often change over time, shifting from the initial intent to cure to improving pain and function. Patients often have unrealistic expectations of complete pain relief and full return to their previous level of activity. There is often a large gap between a patient's desired amount of pain

reduction and the minimum percentage of improvement that would make a treatment worthwhile.[83] Documenting goals and expectations and revisiting them on follow-up visits may be helpful.

Patients should receive information about effective self-care options and should be advised to remain active (because muscles that do not move can eventually become hypersensitive to pain).[84] Assessing the response to therapy should focus on improvements in pain, mood, and function.

Treatment should begin with maximal recommended doses of nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen, followed by adjunctive medications. Nonpharmacologic therapies are effective in certain clinical situations and can be added to the treatment program at any time. For those with severe functional disabilities, radicular symptoms, or refractory pain, referral for epidural steroid injection or surgical evaluation may be reasonable

Pharmacologic treatment options

Acetaminophen is first-line therapy because of its high safety profile. NSAIDs provide similar analgesia, but have significant gastrointestinal and renovascular adverse effects.[85] There are several classes of NSAIDs, and if one class fails, medications from other classes can be tried before abandoning them altogether. Tramadol, opioids, and other adjunctive medications may benefit some patients who do not respond to NSAIDs.

Classes of NSAIDS for Chronic Low Back Pain[82]

Class	Generic (brand)	Standard dosage	Maximal dosage (mg per day)
Salicylic acids	Aspirin	325 to 650 mg every four hours	4,000
	Diflunisal (Dolobid)	500 mg two times daily	1,500
	Salsalate	1,500 mg two times daily	3,000
Acetic acids	Choline magnesium trisalicylate	1,500 mg two times daily	3,000
	Diclofenac potassium (Cataflam)	50 mg three times daily	200
	Diclofenac sodium, delayed release (Voltaren)	50 mg two or three times daily	200
	Etodolac	200 to 400 mg two or three times daily	1,200
	Indomethacin (Indocin)	25 to 50 mg three times daily	200
	Indomethacin, extended release (Indocin SR)	25 to 50 mg one or two times daily	150
	Sulindac (Clinoril)	200 mg two times daily	400
	Tolmetin	200 to 600 mg three times daily	1,800
Oxicam	Meloxicam (Mobic)	7.5 to 15 mg once daily	15
	Piroxicam (Feldene)	20 mg once daily	20
Propionic acids	Ibuprofen	600 mg four times daily or 800 mg three times daily	2,400
	Ketoprofen	50 to 100 mg three times daily	300
	Naproxen (Naprosyn)	250 to 500 mg two times daily	1,500
	Naproxen sodium (Anaprox)	275 to 550 mg two times daily	1,650
	Oxaprozin (Daypro)	1,200 mg once daily	1,800
Anthranilic acid	Meclofenamate	50 to 100 mg four times daily	400
Cyclooxygenase-2 inhibitor	Celecoxib (Celebrex)	200 mg two times daily	400
Nonacidic agent	Nabumetone	1,000 to 2,000 mg one or two times daily	2,000

Tramadol is an analgesic that has weak opioid and serotonin-norepinephrine reuptake inhibitor (SNRI) activity. Studies demonstrate short-term improvements in pain and function, but long-term data are lacking.[86,87] Because of its opioid activity, tramadol generally should not be used in patients recovering from narcotic addiction. Adverse effects include drowsiness, constipation, and nausea.

All muscle relaxants provide similar short-term improvements in pain and function, but there is no evidence to support their long-term use for chronic low back pain. Sedation is a common adverse effect, and chronic use of benzodiazepines and carisoprodol (Soma) carries the risk of dependency [17].

A 2006 Cochrane review[88] found that some herbal medications appear effective in short-term randomized trials, but lack long-term safety data. Harpagophytum procumbens (devil's claw) in a dosage of 50 mg daily, Salix alba (white willow bark, a source of salicylic acid) in a dosage of 240 mg daily, and Capsicum frutescens (cayenne) plaster applied topically every day appear to be better than placebo at reducing chronic low back pain. Limited studies have shown that devil's claw and white willow bark appear to be as effective as NSAIDs.

Short-acting (immediate-release) and long-acting (sustained-release) opioid analgesics are sometimes used for chronic low back pain. There have been few high-quality trials to assess the effectiveness and potential risks of these medications in chronic low back pain.[89]

Taking opioids can lead to the development of tolerance, hyperalgesia (enhanced pain response to noxious stimuli), and allodynia (enhanced pain response to

innocuous stimuli).[90] The combination of tolerance and hyperalgesia can decrease opioid effectiveness over time. One of the challenges of treating chronic low back pain is differentiating among tolerance, opioid-induced hyperalgesia, and disease progression. Hyperalgesia involves increasing pain despite increasing opioid treatment, pain that becomes more diffuse and beyond the distribution of the preexisting pain, and an apparent change in pain threshold or tolerability.[90] In this situation, the dosage of opioids should be decreased, or patients should be weaned off the medication altogether.

Selective serotonin reuptake inhibitors and antiepileptic medications have not been shown to help patients with chronic low back pain. Tricyclic antidepressants, however, provide some benefit and can be a useful addition to analgesic therapy.[91] Gabapentin (Neurontin) may provide short-term relief in patients with radiculopathy.

Nonpharmacologic treatment options

Patients commonly use nonpharmacologic treatment options, with or without consulting a physician. Forty-five percent of patients with low back pain see a chiropractor, 24 percent use massage, 11 percent get acupuncture, and 7 percent try meditation.[92]

Acupuncture provides short-term relief of chronic low back pain, improves functioning, and works as an adjunct to other therapeutic options. It has not been shown to be more effective than other treatments.[93,94] Fifty-one to 64 percent of patients are willing to try acupuncture if recommended by their physician.[92]

Exercise therapy, focusing on strengthening and stabilizing the core muscle groups of the abdomen and back, appears to produce small improvements in pain and functioning in patients with chronic low back pain. However, few studies (i.e., six of the 43 studies included in a Cochrane review) have been able to demonstrate clinically important and statistically significant differences between intervention and control groups.[95,96]

Behavior therapy is as effective as exercise therapy for short-term relief of chronic low back pain. Consistent evidence supports cognitive behavior therapy and progressive relaxation for short-term improvement, whereas biofeedback techniques have produced mixed results. Combining behavior therapy with other modalities does not have an additive effect.[97]

Multidisciplinary rehabilitation programs that include a physician and at least one additional intervention (psychological, social, or vocational) alleviate subjective disability, reduce pain, return persons to work five weeks earlier, and after returning to work, reduce the amount of sick time in the first year by seven days. Benefits persist for up to five years.[98,99]

Acupuncture massage and pressure point massage are mildly helpful for reducing chronic low back pain, and the benefits last for up to one year. Massage appears to be most effective when combined with exercise, stretching, and education.[100]

Spinal manipulation provides modest short- and long-term relief of back pain, improves psychological well-being, and increases functioning.[101] The benefits derived

are not dependent on the type of training of the manipulator because osteopathic and chiropractic outcomes appear to be similar.[102]

One therapeutically directed style of yoga (Viniyoga) may provide some relief of chronic back pain. Six weeks of yoga decreased medication use and provided more pain relief than exercise and self-care. Other forms of yoga have mixed results in small studies, and at this time there is not enough evidence to recommend them.[103]

Back schools, low-level laser therapy, lumbar supports, short wave diathermy, traction, transcutaneous electrical nerve stimulation, and ultrasound have negative or conflicting evidence of effectiveness.[103-108]

Surgery

Most patients with back pain will not benefit from surgery. However, if anatomic abnormalities consistent with the distribution of pain are identified, surgery can be considered in persons who have experienced significant functional disabilities and in those with unremitting pain, especially pain lasting longer than 12 months despite multiple nonsurgical treatments. Good evidence supports the use of spinal fusion for treating back pain caused by fractures, infections, progressive deformity, or instability with spondylolysthesis.[107]

Spinal decompression, nerve root decompression, and spinal fusion have been extensively evaluated for the treatment of degenerative disorders of the spine, mostly with short-term outcomes, yielding conflicting results and questionable patient benefit. Disc arthroplasty (replacing the original intervertebral disc with an artificial

one) appears to be as effective as lumbar fusion for short-term relief of chronic low back pain, but there is no evidence of long-term relief, and concerns exist regarding the durability of the artificial disks. Intradiscal electrothermal therapy is a technique that applies heat to a damaged disk through a catheter, causing collagen contraction for structural support and ablating nearby pain-sensing nerves for pain reduction. It has been shown to provide modest pain relief, but little functional improvement.[109]

Epidural Steroid 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 of Epidural Injections

Anti-Inflammatory Effect:

For more than a decade, orthopaedic 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. 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

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.

At present, emphasis has been focused on regional lymph nodes as the site of antibody production in many immunological responses. 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 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 auto antibodies 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 of 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 for ESI

ESIs are indicated for treating radicular and LBP caused by annular tears, herniated nucleus pulposus (HNPs) (with or without image-confirmed nerve root compression), chemical neuritis, spondylosis, and spinal stenosis. ESIs are most effective during the acute phase of pain and inflammation.[110] Response rates that range up to 90% in patients with symptoms for less than 3 months fall under 50% in patients with symptoms for more than a year. As discussed earlier, ESI is best used in conjunction with other conservative measures to relieve painful symptoms with the understanding that most of these conditions will self–heal with time. The longer symptoms persist without responding to conservative measures, the less likely they are to improve, and the more likely they will require surgical treatment. Other than

duration of symptoms, a poorer response rate to ESI is predicted by prior lumbar surgery, and severe compression by spinal stenosis or a large HNP.

ESIs have been repetitively shown to be unreliable and ineffective in treating pure LBP, for which they have little or no indication. Musculoskeletal pain (muscle, joint, disc) can mimic true neurogenic radicular pain, and there is no reliable diagnostic test other than provocative neuro-blockade to identify pain mediators.

Methods and routes of administration

There is little agreement regarding the type, dosage, frequency, or total number of ESIs that will yield the greatest efficacy and safety. The recommendations often quoted are for a series of 3 injections spaced two weeks apart:3 mg/kg body weight;210 mg annually, or 240 mg in a lifetime. These figures are based on old studies done with poorly supported rationale.[111] Considering the plethora of agents available, the three routes of entry, and the countless personal preferences of providers, it is not surprising that analysis of outcome studies often leads to confusion and uncertainty. The three routes of entry to the epidural space are:

1. *Caudal*
2. *Interlaminar*
3. *Transforaminal*

All are actively practiced today and have their own unique risks and benefits. The superiority of one route over the others is controversial because of the documented as well as perceived differences in efficacy and safety.[112] Evidence-based

guidelines have been drafted by the American Society of Interventional Pain Physicians in an ongoing project with continuous updates. For the current management of chronic low back or radicular pain, interlaminar ESIs offer strong short-term relief and limited long-term relief; *Caudal* and *Transforaminal* ESIs provide strong short-term relief and moderate long-term relief. All techniques have limited benefit in managing post laminectomy syndrome and spinal stenosis.[77]

Caudal ESI was the first to be used for sciatica symptoms.[111] It is the easiest route into the epidural space with the lowest risk of inadvertent puncture of the dura, so it was popular among those who had limited training. It is the least target-specific approach; it requires the largest volume of injectate (which dilutes the steroid that reaches the site of pathology);it reaches only the lower lumbar nerve roots; and it is not useful to treat problems above the lower lumbar region. More recent catheter-assisted techniques have made it more useful in patients with prior lumbar surgery.

Interlaminar ESI gained popularity because it permits placement of steroid from the low lumbar region to the cervical spine at the level and site of pathology.[113] Classically, this technique was performed blindly, but those experienced in interventional pain management now performed it with fluoroscopic guidance, which greatly increases the accuracy of drug placement. Still, the drug is mostly confined to the posterior epidural space, with anterior spread in less than 40% of injections.[114] Though there is an increased risk of dural puncture with this technique, the risk is still quite low (approximately 1%).

Transforaminal ESI has been developed only in the past 10-15 years, and is designed to administer the most target-specific agent to the affected nerve root. Injections are made directly into the neural foramen, which allows spread of steroid to the anterior epidural space, which is believed to be the site of disc-nerve interface. Recent outcome studies reveal greater duration of pain relief and avoidance of surgery compared with interlaminar injections.[115]

Complications

The list of most common complications of ESIs can be divided into those caused by needle placement and those caused by the administered drug. Complications from needle placement are fortunately rare, and include infection, dural puncture, spinal cord trauma, epidural hematoma, nerve damage, headache, vascular injury, and death.[112] Relatively, few complications have been reported due to the pharmacology of the injected steroid. Review of the literature on intrathecal steroid injections reveals no proof of clinically significant arachnoiditis.[116] In addition; no direct evidence of neurotoxicity of steroids has been confirmed in the clinical setting.

Transient suppression of the pituitary-adrenal axis has been universally observed with ESIs. Other potential side effects include weight gain, osteoporosis, and avascular necrosis of bone.[79]

The most worrisome complications of ESIs are permanent neurological injury and death. Though rare, there are several case reports of nerve root and spinal cord injury that led to paralysis and death. In particular, several case reports of *transforaminal* injections have described catastrophic complications such as spinal

cord infarction, massive cerebral edema, anterior spinal artery syndrome, and intra-cord injections. This experience has tamed the enthusiasm to embrace transforaminal-ESIs and abandon other approaches, though with proper precautions and standardized techniques, major complications should be rare.[118]

Epidural hematoma is the most frequent concern, since more and more patients are receiving anti-platelet and anticoagulant therapy. Guidelines for spinal injections in patients taking anticoagulants have been drafted by the American Society of Regional Anesthesia.[119] Warfarin should be discontinued 4-5 days in advance, to yield an INR <1.5. Epidural hematomas, though rare, can lead to rapid compression of the spinal cord with paralysis if not recognized promptly and treated aggressively.

In Caudal Epidural injections post injection pain at the sacral hiatus site of entry may be prolonged but usually does not exceed 2 to 6 months when present. There may be associated injection-site ecchymosis. Intrathecal injection can occur despite a reduced risk of same and is associated with prolonged and/or high subarachnoid block, respiratory distress or arrest, and total spinal anesthesia with risk of death; adhesive arachnoiditis may result from solvent of depo-steroid polyethylene glycol, and post dural puncture headache may also occur. An early sign of intrathecal injection is the patient's inability to evaluate the lower extremity soon after the injection has been completed or the inability to push up the knees in a supine position.

Overall, epidural steroid injections are effective and safe in providing short and long term pain relief for sciatica and LBP when proper patient selection and contemporary techniques are used. They are not intended to replace surgical treatment

for spinal disorders, but to aid in avoiding surgery if possible. Surgery should be reserved for those who do not respond to non-surgical treatment or have serious neurological compromise. Unfortunately, patients are usually not given the option of ESI until their condition has become chronic, and after multiple and often more costly conservative therapies have been exhausted. ESIs are most effective during the acute phase of pain and inflammation, and response rates fall as the duration of symptoms increases. For the patient with LBP with a radicular component, they should be utilized early, in combination with other non-surgical treatments, to provide relief of pain and suffering during the period of self-healing.

Epidural steroid injections may help patients with radicular symptoms. Studies have found conflicting results, but the trend is toward a small improvement for up to three months after injection.[82] There is no evidence to support the use of epidural steroid injections in patients without radicular symptoms, and injections are less effective in patients with severe spinal stenosis and those with stenotic lesions encompassing more than three lumbar levels.[82]

Pharmacology of bupivacaine[120]

Local anaesthetics are drugs that produce reversible blockade of conduction of nerve impulses. The primary desirable properties of an ideal local anaesthetic agent are:

1. Short latency
2. High potency or anaesthetic activity
3. Superior penetration or diffusion
4. Low toxicity
5. Complete reversibility of action
6. Prolonged duration of action
7. Stability and ability to withstand heat sterilization.

Bupivacaine (Marcain, Marcaine and Sensorcaine)

Bupivacaine, an amino amide local anesthetic was first synthesized in Sweden by A.F Ekenstam and his colleagues in 1957. First report of its use was in 1963 by L.J Teluvio. It is one of the long acting local anesthetic agents available, which is extensively used for intrathecal, extradural and peripheral nerve blocks.

Chemistry

The molecular weight of chloride salt is 325 and that of base form is 288. pH of plain solutions varied between 4.5 to 6 and pKa 8.16. Bupivacaine Hydrochloride is chemically designated as 2-piperidinecarboxamide, 1-butyl-N-(2,6-dimethylphenyl)-, monohydrochloride, monohydrate and has the following structure:

Presentation

Bupivacaine is presented as 2.5 mg/ml, 5 mg/ml and 7.5 mg/ml solutions. Bupivacaine is also present as 5 mg/ml with 80 mg/ml dextrose in a 4 ml clear ampoule.

Chemical Structure

Bupivacaine has an IUPAC nomenclature of 1-butyl-n-(2, 6-dimethylphenyl) piperidine-2-carboxamide.

Dosage

Bupivacaine can be used in a dose for block upto a maximum of 2 mg/kg depending on the type and duration of surgery.

Properties

The base is sparingly soluble, but the hydrochloride salt is readily soluble in water. In spinal anesthesia, the onset of action is about three to four minutes and complete anesthesia occurs in five minutes and lasts for 3.5 to 4 hours. Because bupivacaine is an amide, the liver is the primary site of metabolism. The most of the drug is metabolized by N-dealkylation.

Physiochemical Properties

- Molecular formula C_{18} H_{28} N_{2O}
- Molecular weight 288.43 g/mol

- Solubility in water 25 mg/ml
- pH of saturated solution 5.2
- pKa 8.1
- Specific gravity 1.021 at 37°c

Mechanism of Action

Mechanism of action is similar to that of any other local anesthetic. The primary action is on the cell membrane axon, on which it produces electrical stabilization. Bupivacaine prevents the generation and the conduction of the nerve impulse. Bupivacaine blocks conduction by decreasing or preventing the large transient increase in permeability of excitable membranes to sodium that normally is produced by a slight depolarization of the membrane. This action of Bupivacaine is due to its direct interaction with the voltage gated sodium channels. As the anesthetic action progressively develops in a nerve, the threshold for electrical excitability gradually decreases, the rate of rise of the action potential declines, impulse conduction slows, and the safety factor for conduction decreases, these factors decrease the probability of propagation of the action potential and nerve conduction fails. The mechanism by which local anaesthetics block sodium conductance is as follows:

Local anesthetics in the cationic form act on the receptors within the sodium channels on cell membrane and block it. The local anesthetics can reach the sodium channel either via the lipophilic pathway directly across the lipid membrane, or via the

axoplasmic opening. This mechanism accounts for 90% of the nerve blocking effects of amide local anesthetics.

The second mechanism of action is by membrane expansion. This is a non-specific drug receptor interaction. Bupivacaine is available in the following concentrations:

- 0.25%. 0.5% and 1%
- 0.25% and 0.5% solution in isotonic saline
- 0.5% solution in 8% dextrose
- Dosage is 2mg/kg limited to 150 mg in four hours. The intrathecal minimum local analgesic dose of Bupivacaine is 2.37 mg.

Basic Pharmacology

Bupivacaine hydrochloride is 2-piperidine carboxamide, 1 butyl N-2, 6 dimethyl phenyl, monohydrochloride, monohydrate. Bupivacaine molecule is a tertiary amine separated from an aromatic ring system that is a benzene ring by an intermediate chain. The tertiary amine is a base that is a proton acceptor. The chain contains an amide linkage (-NHCO-) therefore, it is classified as an aminoamide compound. This amide linkage contributes to the anaesthetic potency.

The aromatic ring system gives a lipophilic character to its portion of molecule whereas, the tertiary amine end is relatively hydrophilic.

Structure – Activity relationship

Bupivacaine being more lipophilic (because of butyl group) it is very potent and produces longer lasting blocks.

pKa of any drug is defined as the hydrogen ion concentration specific for each drug at which the concentration of local anaesthetic base is equal to the concentration of charged cation. pKa of bupivacaine hydrochloride is 8.1 at 36ºC.

Anesthetic Potency

Hydrophobicity appears to be a primary determinant of intrinsic anesthetic potency and Bupivacaine is highly hydrophobic, hence is very potent.

Onset of Action

The onset of conduction blockade is dependent on the dose or concentration of the local anesthetic

Differential Sensory Motor Blockade

Bupivacaine in low concentration (0.125%) produces acceptable analgesia with only mild muscular weakness.

Pharmacokinetics

The concentration of Bupivacaine in blood is determined by the amount injected, the rate of absorption from the site of injection, the rate of absorption from

the site of injection, the rate of tissue distribution and the rate of biotransformation and excretion of Bupivacaine.

Absorption

The site of injection, dose and addition of a vasoconstrictor determine the systemic absorption of Bupivacaine .The maximum blood level of Bupivacaine is related to the total dose of drug administered from any particular site. Absorption is faster in areas of high vascularity.

Distribution

The two-compartment model can describe this. The rapid distribution phase α is believed to be related to uptake by rapid equilibrating tissue i.e., tissues that have high vascular perfusion. The slow distribution phase β is mainly a function of distribution to slowly equilibrating tissue, biotransformation and excretion of the compound.

More highly perfused organs show higher concentrations of the drug. Bupivacaine is rapidly excreted by lung tissue. Though skeletal muscle does not show any particular affinity for bupivacaine it is the largest reservoir of the drug.

Clinical Pharmacology

1. Anaesthetic potency: Hydrophobicity is a major determinant of intrinsic anaesthetic potency and bupivacaine being highly hydrophobic, is very potent.

2. Onset of action: It depends on the pH of the drug and its concentration.
3. Differential sensory/motor blockade:

Bupivacaine 0.25 to 0.75% produces adequate analgesia with less of motor blockade.

Actions

Central Nervous System

Bupivacaine readily crosses the blood brain barrier causing CNS depression following higher doses. The initial symptoms involve feeling of light-headedness and dizziness followed by visual and auditory disturbances. Disorientation and occasional feeling of lightheadedness may occur. Objective signs are usually excitatory in nature, which include shivering, muscular twitches and tremors, initially involving muscles of the face (perioral numbness) and part of extremities. At still higher doses cardiovascular or respiratory arrest may occur. Acidosis increases the risk of CNS toxicity from Bupivacaine, since an elevation of $PaCO_2$ enhances cerebral blood flow, so that more anesthetic is delivered rapidly to the brain.

Autonomic nervous system

Bupivacaine does not inhibit the Nor Adrenaline uptake and hence has no sympathetic potentiating effect. Myelinated preganglionic B fibers have a faster

conduction time and are more sensitive to action of Bupivacaine. When used for conduction blockade, all local anesthetics, particularly Bupivacaine produces higher incidence of sensory than motor fibers.

Cardiovascular System

The primary cardiac electrophysiological effect of a local anesthetic is a decrease in the maximum rate of depolarization in Purkinje fibers and ventricular muscle. This action by Bupivacaine is far greater compared to Lignocaine. Also, the rate of recovery of block is slower with Bupivacaine. Therefore there is complete restoration of Vmax between action potential particularly at higher rates. Therefore Bupivacaine is highly arrythmogenic. Bupivacaine reduces the cardiac contractility. This is by blocking the calcium transport. Low concentration of Bupivacaine produces vasoconstriction whereas high doses cause vasodilatation.

Respiratory System

Respiratory depression may be caused if excessive plasma level is reached which in turn results in depression of medullary receptor center. Respiratory depression may be also caused by paralysis of respiratory muscles of diaphragm as may occur in high spinal or total spinal anesthesia.

Biotransformation and Excretion

Bupivacaine undergoes enzymatic degradation primarily in the liver. The excretion occurs primarily via the kidney. Renal perfusion and factors affecting urinary pH affect urinary excretion. Less than 5% of Bupivacaine is excreted via the kidney unchanged through urine. The major portion of injected agent appears in urine in the form of 2, 6 pipecolyoxylidine which is a n-dealkylated metabolite of bupivacaine. Renal clearance of the drug is related inversely to its protein binding capacity and pH of urine.

Adverse effects are encountered in clinical practice mostly due to overdose, inadvertent intravascular injection or slow metabolic degradation.

Adverse Effects

CNS:Nervousness, dizziness, blurring of vision or tremors, drowsiness, convulsions and respiratory arrest.

CVS:Myocardial depression, hypotension, arrhythmia, ventricular type conduction defect, SA node depression and cardiac arrest

Allergic reactions:Urticaria, bronchospasm, hypotension

Other:Constriction of pupil and tinnitus

METHODOLOGY

The present study was conducted at KLES Dr.PrabhakarKoreHospital and Medical Research Centre, Belgaum during the period of January 2011 to December 2011.

Study design

One year randomized controlled trial.

Study period

Present study was conducted from January 2011 to December 2011.

Place

This study was carried out at Department of Orthopedics, KLES Dr.PrabhakarKoreHospital and Medical Research Centre, Belgaum attached to JawaharlalNehruMedicalCollege, KLEUniversity, Belgaum

Source of Data

The patients with chronic low backache with sciatica attending KLES Dr.PrabhakarKoreHospital and Medical Research Centre, Belgaum were included in the study.

Sample Size

A total sample size of 60 cases divided into two groups.

Sample size calculation

On the basis our literature search, we determinedthat a sample size of 30 participants per group was sufficient for this study using a desired power of 0.8 and an error of 0.05. The sample size was derived from the formula as mentioned below.

$$n = \frac{2(Z_\alpha + Z_\beta)^2 \, x(S.D_1^2 + S.D_2^2)}{(X_1 - X_2)^2}$$

Where,

Z_α = 1.965 for α=0.05

Z_β = 0.84 for β=0.3

S.D. = 6

X_1 = 30

X_2 = 24

Using this formula the sample size was determined as 30 in each group.

Selection Criteria

Inclusion criteria

1. Patients with chronic low backache for more than 3 months and refractory to conservative treatment with analgesics, rest &physiotherapy for at least 2 weeks.

2. Patients aged 18yrs and above.

Exclusion criteria

1. Cases with history of previous lumbar spine surgery.

2. Cases with severe motor weakness, rapidly progressing neurological deficits, caudaequina syndrome, neurogenic claudication.

3. Local infection at the site of injection

4. Use of steroids 3wks or less before the study

5. Allergy to Steroids

6. Bleeding diatheses

7. Pregnancy

8. Patients with uncontrolled hypertension, uncontrolled diabetes mellitus.

9. Not willing to consent

Randomization

Randomisation was done by using Computer assisted Random Allocation Software.

- Group B (n=30) – Bupivacaine group.
- Group S (n=30) – Normal saline group.

Ethical clearance

The study was approved by the Ethical and Research Committee, JawaharlalNehruMedicalCollege, Belgaum.

Informed Consent

Patients fulfilling the selection criteria were briefed about the nature of the study and a written informed consent was obtained from the selected patients (Annexure I).

Data collection

After obtainingwritten informed consent from the selected patients, demographic data, chief complaints at presentation and history was taken. General physical examination, local examination for spine and neurological assessment was done. Patients were subjected to questionnaires including the visual analogue scale(VAS), Oswestry Disability Index Score (ODIS), Beck's Depression Inventory Scoring (BDIS)[57,58]and the scores were evaluated before the interventionand findings were recorded on predesigned and pretested proforma (Annexure II).

Investigations

Routine investigations such as X-ray Lumbo-Sacral Spine (AP and lateral view), blood investigations (Haemoglobin, total count, direct count, erythrocyte sedimentation rate), blood sugar levels, urine analysis (urine albumin, sugar, microscopy) were done. Special investigation such as magnetic resonance imaging (MRI)of lumbar spine was also done in all the cases.

Procedure

Interventions

Group B

Under aseptic precautions patients were infiltrated with an injection of 2 ml triamcinolone acetate and 18 mL 0.25% bupivacaine.

Group S

Patients were infiltrated with 2 ml triamcinolone acetate and 18 mL normal saline.

Technique

Materials Required

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. 20 ml – For 0.25% Bupivacaine/ Normal Saline
d. Needles

I. 18 gauge Tuohy needle (7.6 cms in length)

II. Other Hypodermic needles (Local infiltration)

e. Swabs

f. Bowl

Other Materials kept ready were:-

g. Steroid- Triamcinolone Acetate 2 ml (40 mg / ml)

h. 0.25% Bupivacaine

i. Normal Saline

j. 2 % Xylocaine without adrenaline & methyl parabane (Xylocard)

k. Rectified Spirit

l. Povidine Iodine

The steroid used was Triamcinolone Acetate because of its marked anti-inflammatory action and relatively low incidence of side effects. Xylocaine was used for local infiltration. Bupivacaine and Normal Saline were used in Group B and Group S respectively.Preservative free preparations avoided certain complications attributed to the methyl parabane.

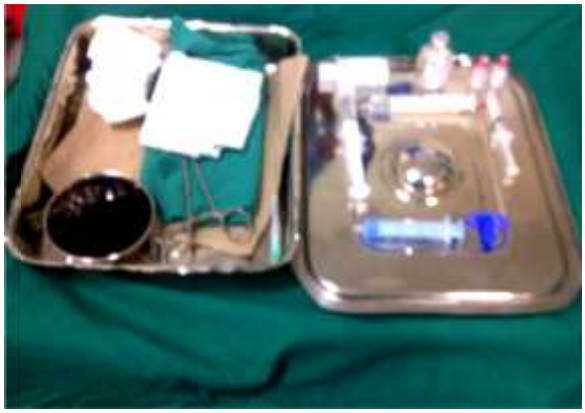


Figure 19 Epidural Tray Set

Figure 20 Epidural Needle & Loss of Resistance Syringe

Figure 21 0.25% Bupivacaine

Figure 22 Normal Saline

Preparation andposition 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 and 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, in presence of an anaesthetist. Neurological Status and SLRT are re-assessed at this stage. Aseptically the skin area was prepared with spirit andpovidine iodine. The patient was draped in a sterile fashion.

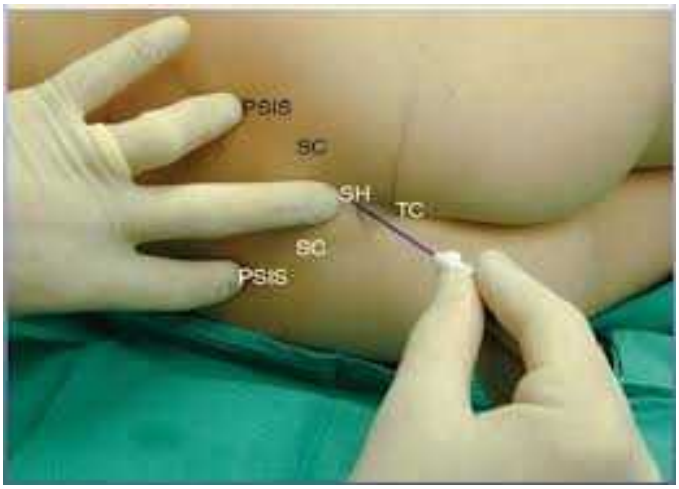


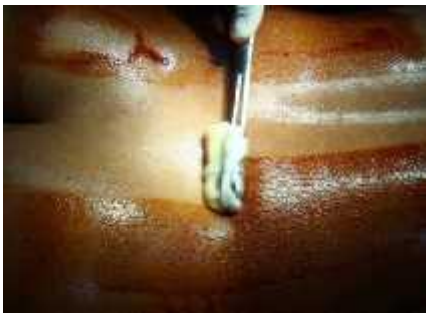
Figure 23 Orthopaedic Triangle

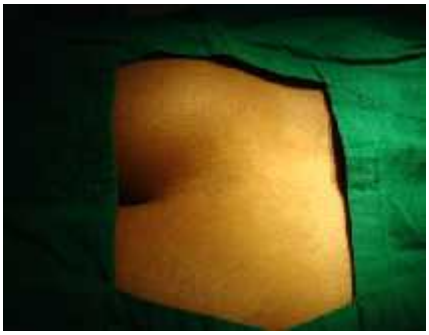
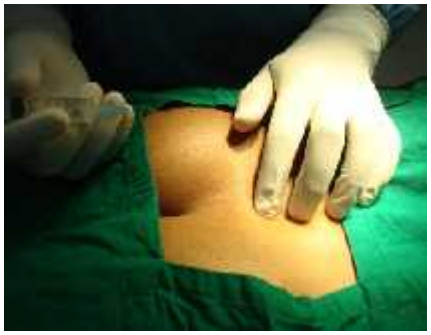
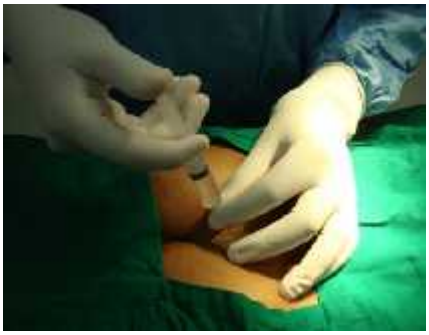
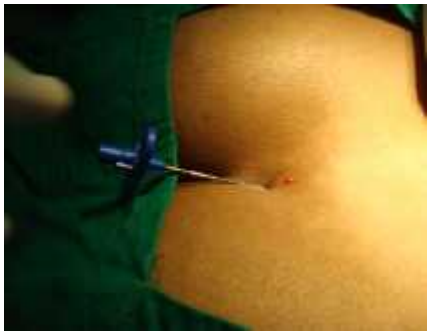
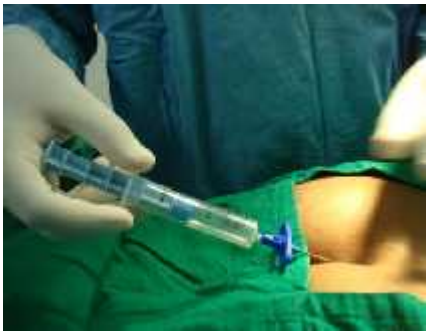
The position used for the caudal epidural injection was the prone position. The patient lies routinely in the prone position with a pillow under the chest and one placed under the ankles to release tension on the sciatic nerve. Prone position was preferred for the caudal epidural steroid injection as the give way of the resistance could be easily appreciated and the anatomical landmarks could be well palpated.

Method

After positioning the patient and preparation of the skin, the sacral hiatus is identified by palpation, which is located between the two horns of the sacral cornu. An inverted orthopaedic triangle is formed by the two posterior superior iliac spines and sacral hiatus which help in identifying the location. The injection is given 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 and rolled from side to side. In this way the cornu are usually felt and 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, and 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 inches (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. 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 and gently pushed forward more cephalad, advancing it into the sacral canal through the sacrococcygeal ligament and into the epidural space. The methods used to document the needle placement included the Whoosh test as well as the Loss of resistance test. Remove the stylet. Aspirate to check for blood or spinal fluid. Inject slowly 2 ml of 40 mg/ml of Triamcinolone Acetate followed by 18 ml of 0.25% Bupivacaine / Normal Saline, to get a total of 20 ml. Finally the tuohy needle is withdrawn and the spot was sealed with tincture benzoin.



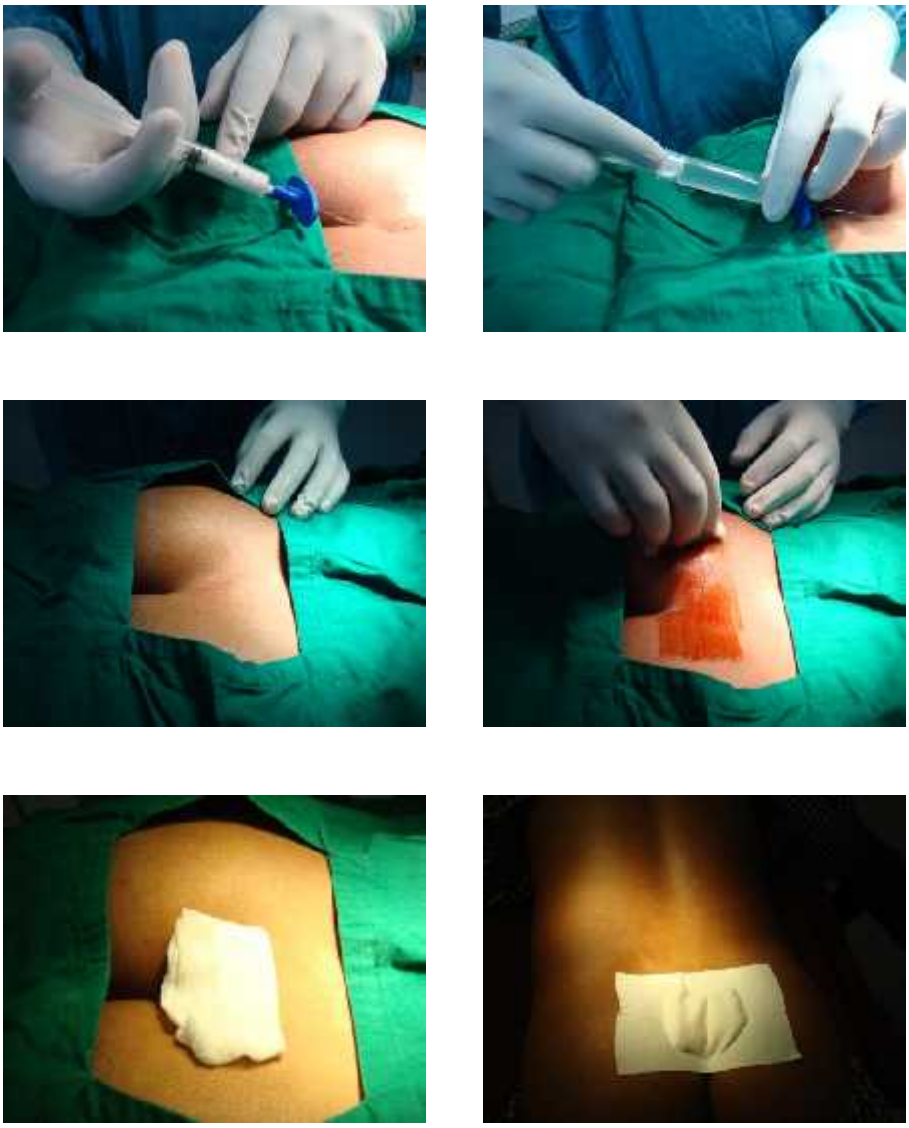



Figure 24 Caudal Epidural Steroid Injection Procedure

Post Injection Protocol

At the conclusion of the injection a note was made of the following:relief of pain and its extent measured subjectively as well as by straight leg raising test, and motor and sensory examination. The patient was advised that apart from a feeling of warmth in the legs and perhaps a sensation of walking on cotton wool, there should be no other neurological signs or untoward effect. The patient was advised to lie flat for at least 45 min after the injection which helps to avoid headache developing on sitting

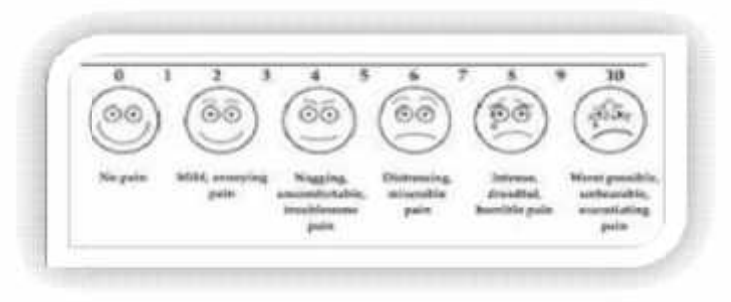
up. The patient was advised to pass urine before leaving the hospital as urinary retention is known to occur after epidural injection. Physiotherapy and Back extension exercises were continued after the injection as a routine protocol.

Outcome variables

Visual analog scale

Outcome evaluation was done by assessment of pain score. Visual analog score (VAS) was used to evaluate pain level (0=no pain to 10=worst pain). Assessment of VAS scores was done as below

- 0 – No pain
- 1 to 3 – Mild pain
- 4 to 6 – Moderate pain
- > 6 – Severe pain



Visual analog scale

Oswestry Disability Index Score

The questionnaire contains six statements (denoted by the letters A through F) in each of ten sections. The sections concern impairments like pain, and 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)

Beck's depression inventory score

Beck's depression inventory score consists of twenty-one question survey. The scores are based on 0 to 3 scale and interpreted '0' as minimal and '3' as severe. The interpretation of BDIS scores was done as below;

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

Follow-up

Clinical evaluations were performed 48 hours after the injection for patients, at 3 weeks (visit 2), 3 months (visit 3), and 6 months (visit 4) for both the groups. Evaluation at 48 hours included VAS and SLRT only.

The Visual Analogue Score, Oswestry Disability Index score, BDIS and 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 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 two to three 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.

Statistical analysis

Data obtained was coded and entered into Microsoft Excel spread sheet. The categorical data was expressed as rate, ratio and percentage. The comparison was done using chi-square test and man Whitney 'U' test. The continuous data was expressed as mean ± S.D. A 'p' value of less than or equal to 0.05 was considered as statistically

RESULTS

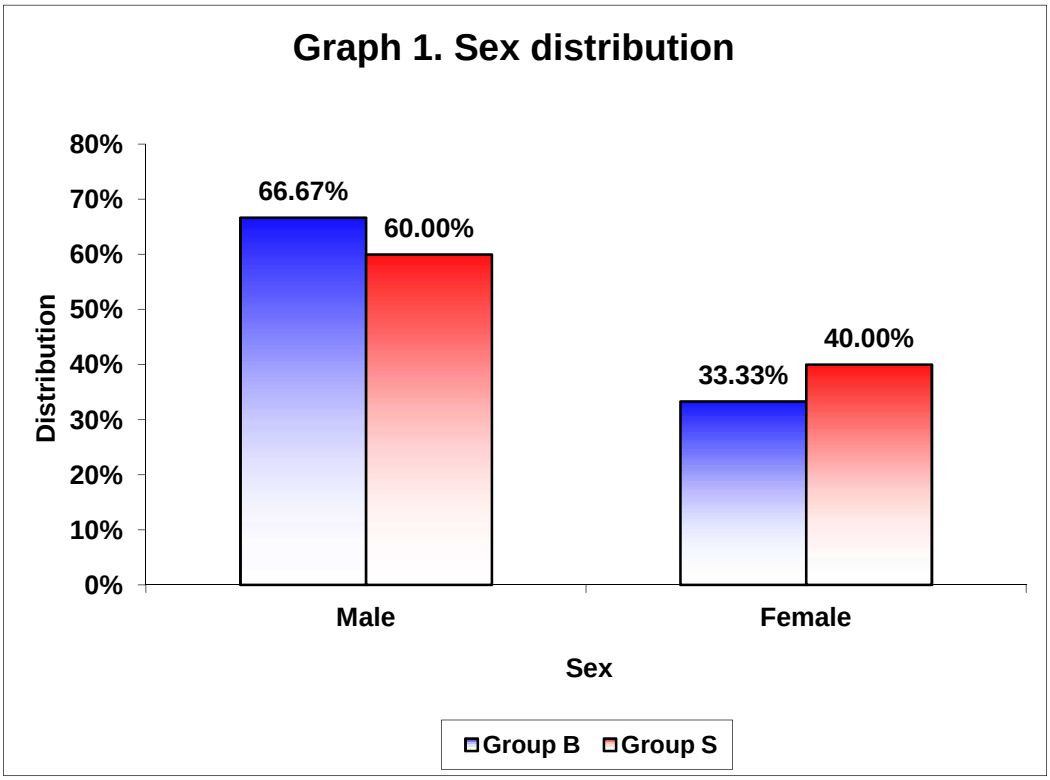
The present one year randomized controlled trial was conducted at at Department of Orthopedics, KLES Dr.PrabhakarKoreHospital and Medical Research Centre, Belgaum attached to JawaharlalNehruMedicalCollege, KLEUniversity, Belgaum.

A total of 60 patients with chronic low backache with sciatica were included in the study. Based on the computer generated randomization the selected patients were randomized into two groups namely Group B (Bupivacaine group) and Group S (Normal saline group).

Data obtained was coded and entered into Microsoft Excel spreadsheet. The data was analysed and the results were tabulated as below.

Table 1. Sex distribution

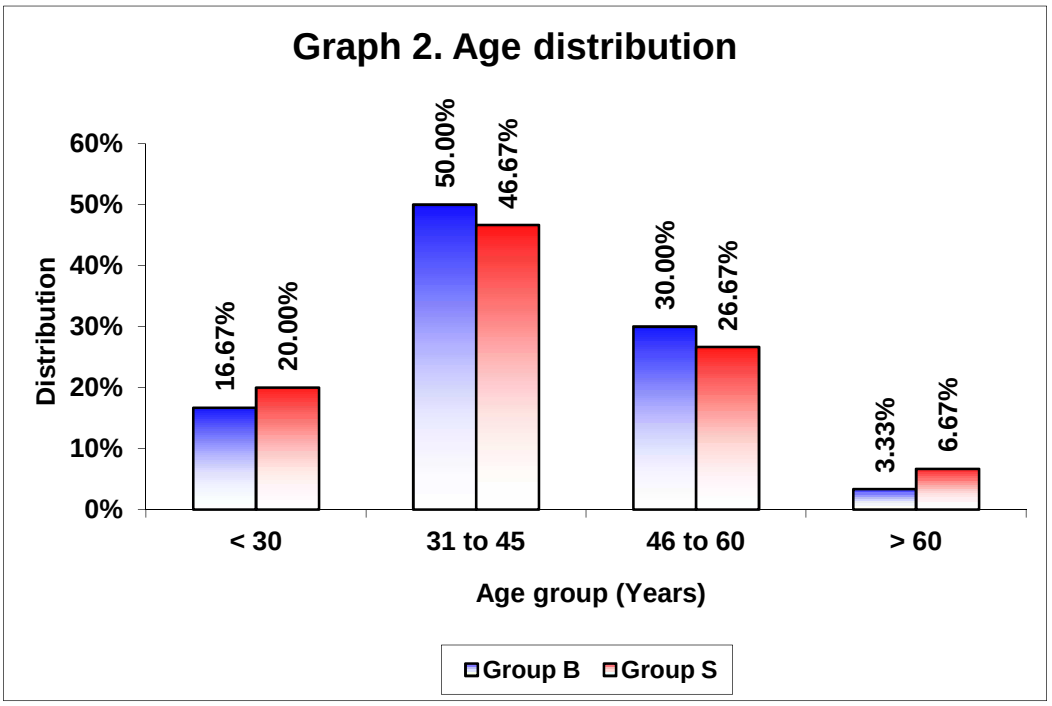
Sex	**Group B (n=30)**		**Group S (n=30)**	
	Number	**Percent**	**Number**	**Percent**
Male	20	66.67	18	60.00
Female	10	33.33	12	40.00
Total	**30**	**100.00**	**30**	**100.00**



In the present study 66.67% in group B and 60% in group S were males. And females constituted 33% and 40% in group B and group S. However both the groups were comparable (p=0.479)

Table 2. Age distribution

Age group (Years)	Group B (n=30)		Group S (n=30)	
	Number	Percent	Number	Percent
< 30	5	16.67	6	20.00
30 to 45	15	50.00	14	46.67
46 to 60	9	30.00	8	26.67
> 60	1	3.33	2	6.67
Total	**30**	**100.00**	**30**	**100.00**

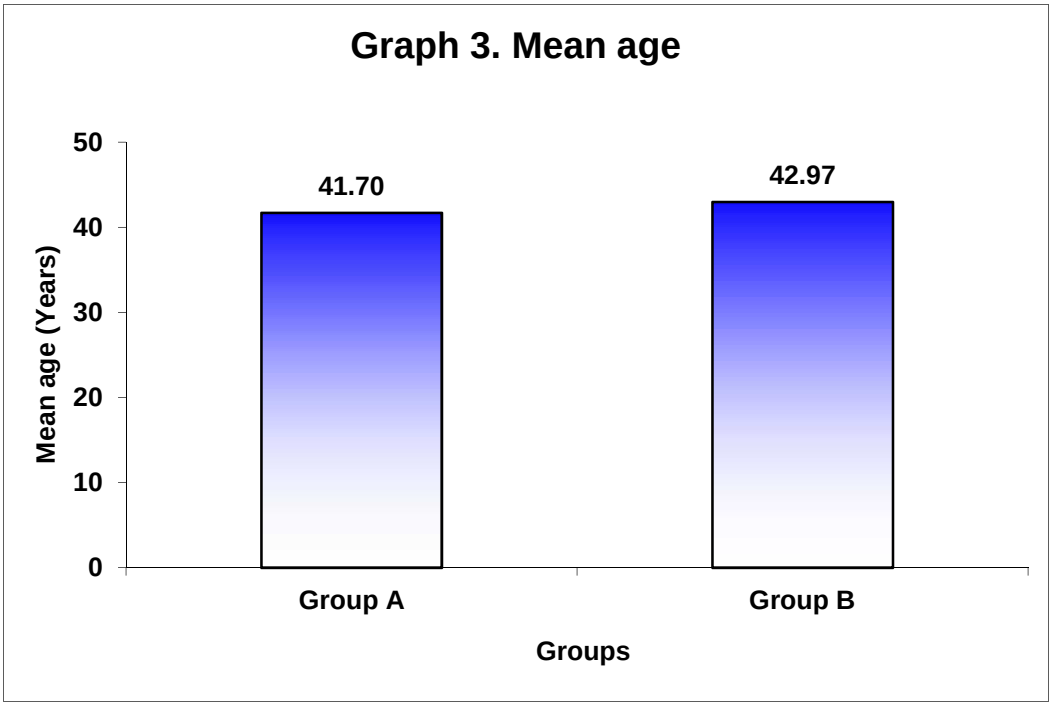


In the present study most of the patients in both the groups (group B-50% and group S- 46.67%) were aged between 31 to 45 years.

Table 3. Mean age

Groups	Mean	SD
Group A	41.70	11.55
Group B	42.97	10.93

p=0.664

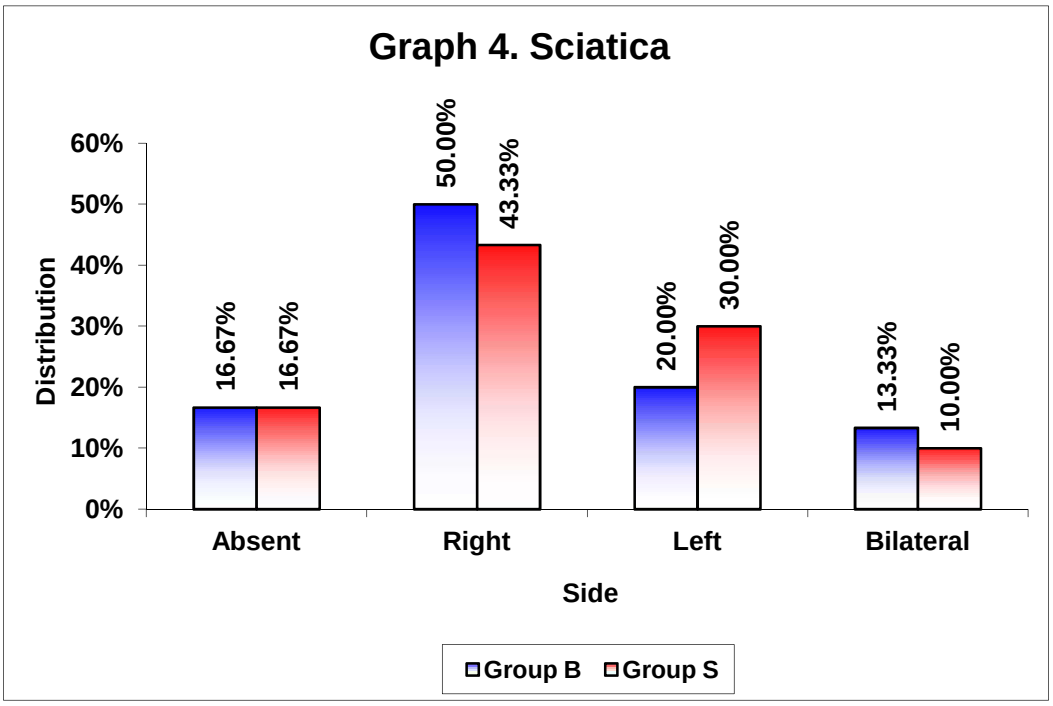


In the present study the mean age in group B was 41.70 ± 11.55 years and in group S it was 42.97 ± 10.93 years.

Table 4. Sciatica

Side	Group B (n=30)		Group S (n=30)	
	Number	**Percent**	**Number**	**Percent**
Absent	5	16.67	5	16.67
Right	15	50.00	13	43.33
Left	6	20.00	9	30.00
Bilateral	4	13.33	3	10.00
Total	**30**	**100.00**	**30**	**100.00**

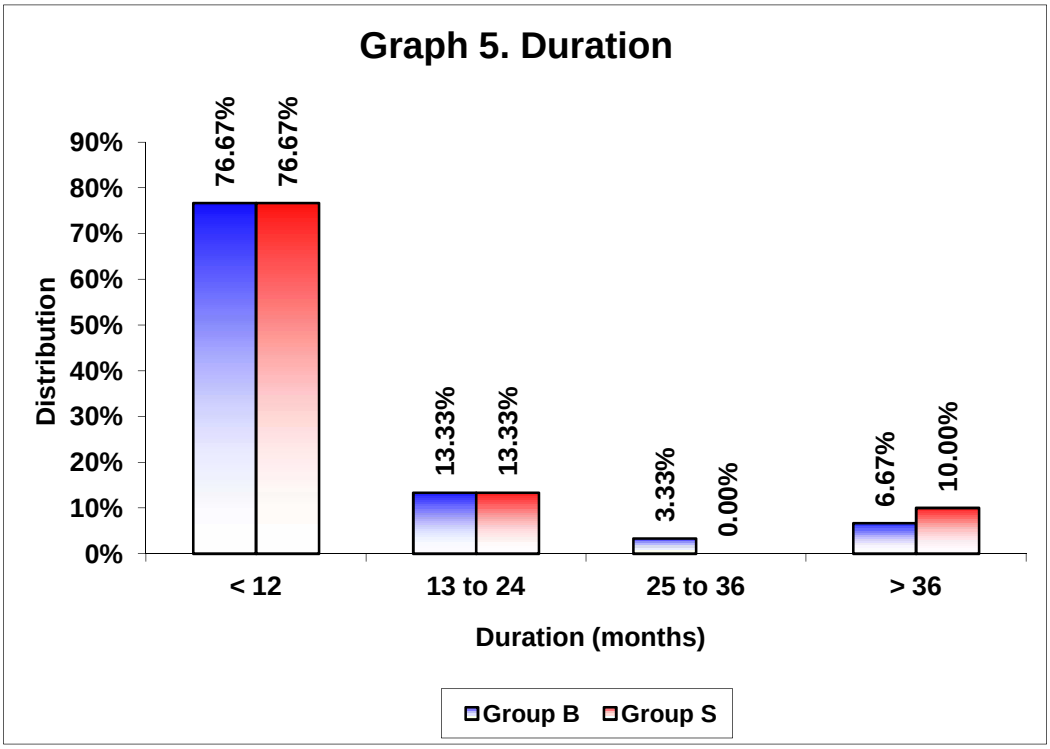
x^2=0.8857 **DF=3** **p=0.8289**



In the present study 50% of patients in group B had right sided sciatica and in group S the same was present in 43.33% of patients. However, 16.67% patients each in both the groups sciatica was absent.

Table 5. Duration

Duration (months)	Group B (n=30)		Group S (n=30)	
	Number	Percent	Number	Percent
< 12	23	76.67	23	76.67
13 - 24	4	13.33	4	13.33
25 - 36	1	3.33	0	0.00
> 36	2	6.67	3	10.00
Total	**30**	**100.00**	**30**	**100.00**

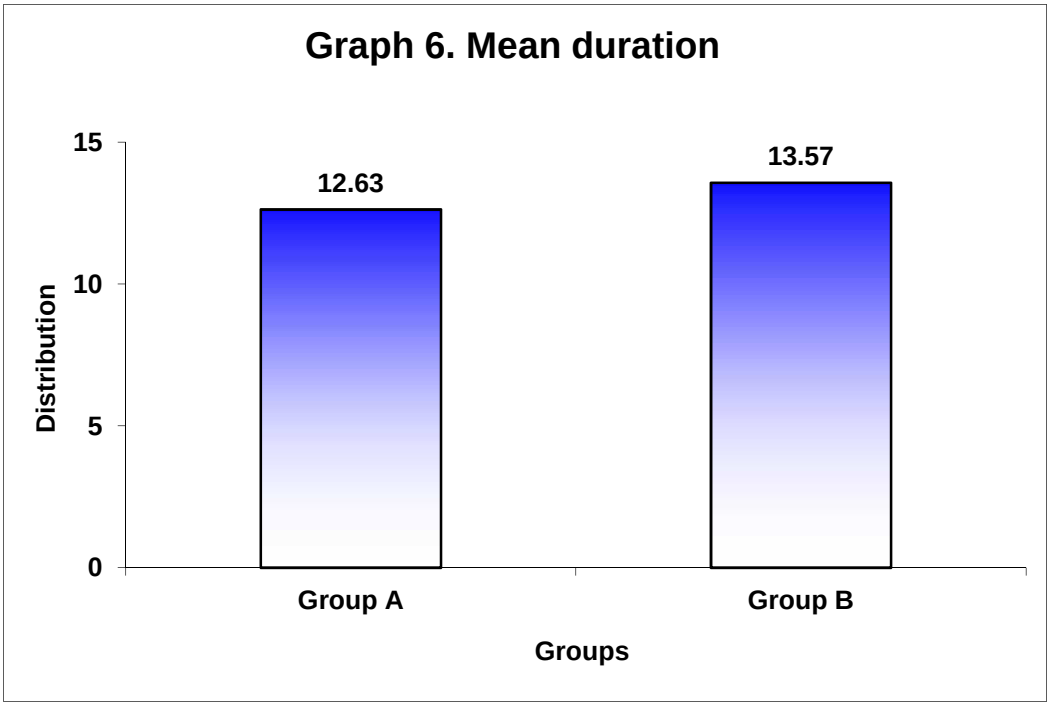


In the present study majority (76.67%) of the patients in both the groups had duration less than 12 months.

Table 6. Mean duration

Groups	Mean	SD
Group A	12.63	12.54
Group B	13.57	12.75

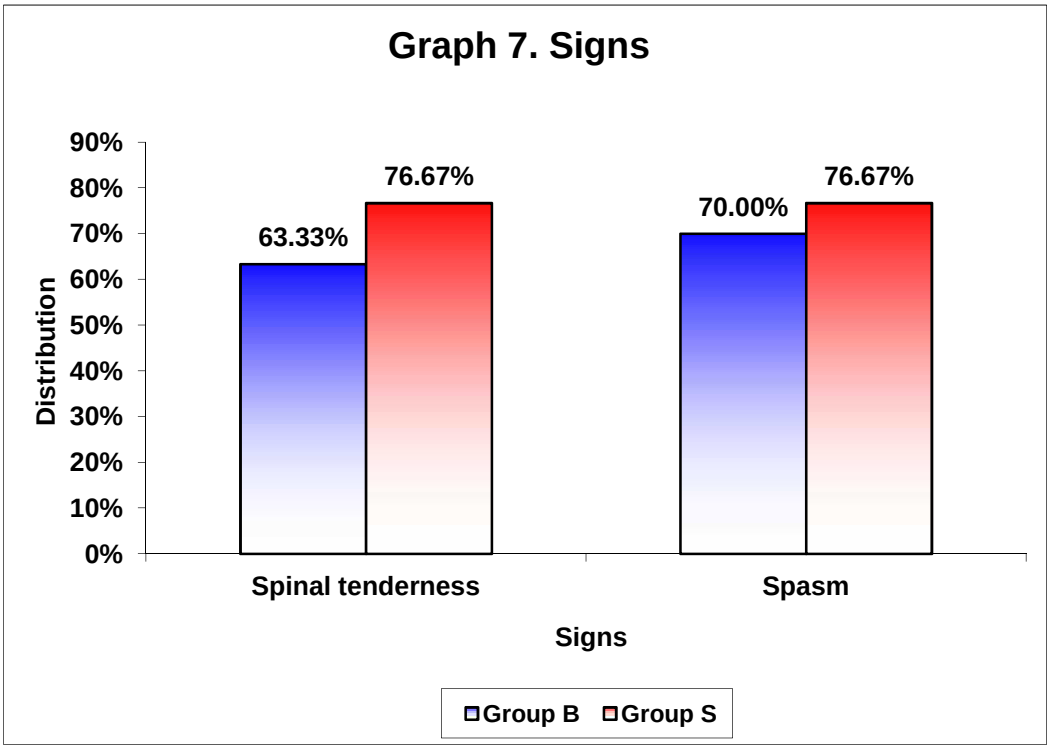
p=0.776



In the present study the mean duration in group B was 12.63 ± 12.54 months and in group S it was 13.57 ± 12.72 months. Both the groups were comparable in terms of duration.

Table 7. Signs

Signs	Group B (n=30)		Group S (n=30)	
	Number	Percent	Number	Percent
Spinal tenderness	19	63.33	23	76.67
Spasm	21	70.00	23	76.67



In the present study 63.33% and 76.67% of patients each in both groups presented with spinal tenderness and spasm.

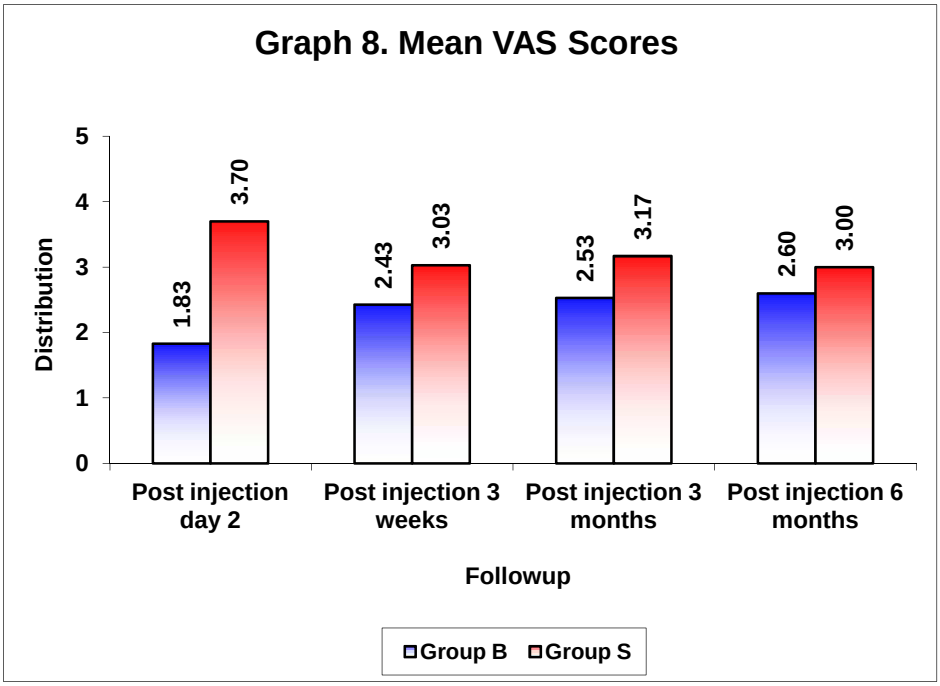
Table 8. Outcome – VAS Scores

Duration	Pain	Group B (n=30)		Group S (n=30)	
		Number	**Percent**	**Number**	**Percent**
Post injection day 2	Mild	30	100.00	11	36.67
	Moderate	0	0.00	19	63.33
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
					p < 0.001
Post injection 3 week	Mild	27	90.00	22	73.33
	Moderate	3	10.00	8	26.67
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
					p=0.0292
Post injection 3 months	Mild	25	83.33	21	70.00
	Moderate	5	16.67	9	30.00
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
					p=0.03511
Post injection 6 months	Mild	25	83.33	25	83.33
	Moderate	5	16.67	5	16.67
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
					p=0.1691

In this study, mild pain was noted in all patients (100%) at post injection day 2 in group B. At third week follow up 90.00% patients reported mild and 10.00% reported moderate pain and at third month follow up 83.33% reported mild and 16.67% reported moderate pain suggesting less pain in group B and the difference was statistically significant. However, pain scores at six months in both the groups were comparable.

Table 9. Mean VAS Scores

Followup	Group B (n=30)		Group S (n=30)	
	Mean	SD	Mean	SD
Post injection Day 2	1.83	0.99	3.7	1.47
Post injection 3 weeks	2.43	1.25	3.03	1.13
Post injection 3 months	2.53	1.04	3.17	1.23
Post injection 6 months	2.6	1.28	3	1.31



In this study the mean pain scores at post injection day two were 1.83 ± 0.99 in group B whereas in group S it was 3.7 ± 1.47, at 3 weeks follow up mean pain scores were 2.43 ± 1.25 in group B and 3.03 ± 1.13 in group S. At 3 months follow up mean pain scores were 2.53 ± 1.04 in group B and 3.17 ± 1.23 in group S whereas the mean pain scores at six months follow up were less in group B 2.60 ± 1.28 compared to group S 3.00 ± 1.31.

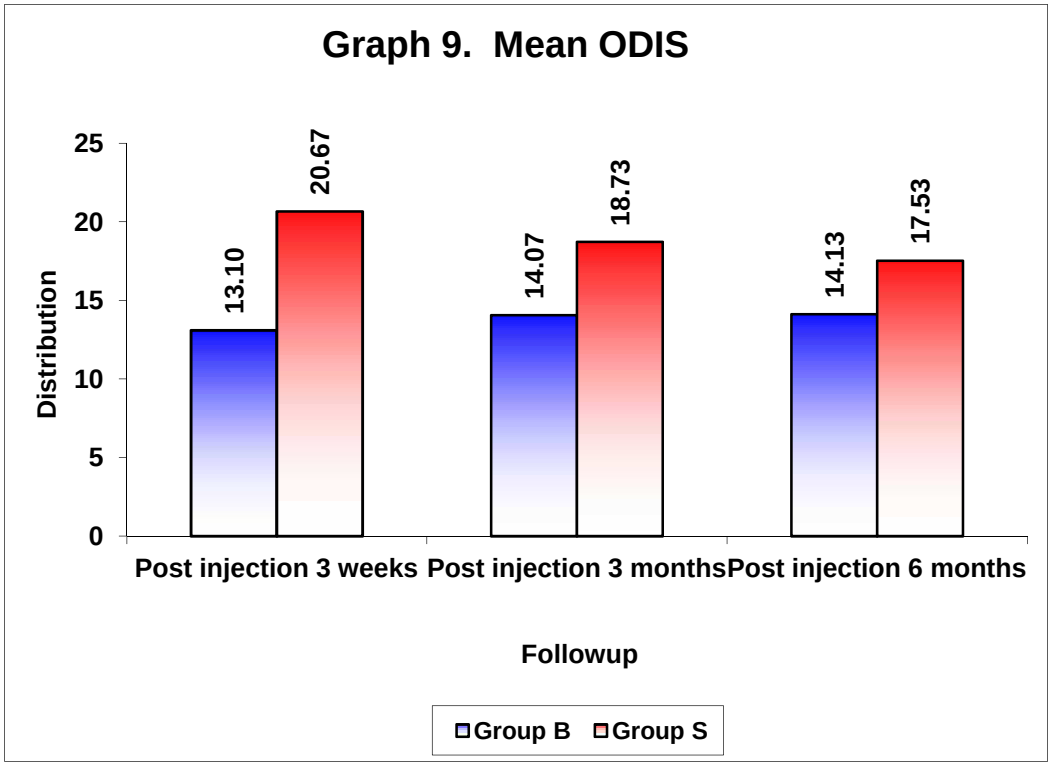
Table 10. Outcome – ODIS

Duration	Pain	Group B (n=30)		Group S (n=30)	
		Number	**Percent**	**Number**	**Percent**
Post injection 3 weeks	Minimal	6	20.00	14	46.67
	Moderate	24	80.00	14	46.67
	Severe	0	0.00	2	6.67
	Crippled	0	0.00	0	0.00
	Bed bound	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=7.831		**DF=2**		**p=0.019**
Post injection 3 months	Minimal	3	10.00	2	6.67
	Moderate	27	90.00	19	63.33
	Severe	0	0.00	9	30.00
	Crippled	0	0.00	0	0.00
	Bed bound	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=10.590		**DF=2**		**p=0.005**
Post injection 6 months	Minimal	3	10.00	5	16.67
	Moderate	27	90.00	22	73.33
	Severe	0	0.00	3	10.00
	Crippled	0	0.00	0	0.00
	Bed bound	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=4.010		**DF=2**		**p=0.134**

In the present study based on ODIS minimal and moderate disability was recorded in 20% and 80% of patients in group B compared 46.67% each with minimal and moderate and 6.67% with severe disability in group S. This difference was statistically significant (p=0.019). At post injection three months minimal disability was present in 10% patients and 90% patients reported moderate disability in group B whereas 6.67%, 63.33% and 30% of patient in group S reported minimal, moderate and severe disability respectively. This difference was statistically significant (p=0.005). At post injection six months minimal disability was present in 10% patients and 90% patients reported moderate disability in group B whereas 16.67%, 73.33% and 10% of patients in group S reported minimal, moderate and severe disability respectively. However this difference was statistically not significant.

Table 11. Mean ODIS

Followup	Group B (n=30)		Group S (n=30)	
	Mean	SD	Mean	SD
Post injection 3 weeks	13.1	2.25	20.67	3.08
Post injection 3 months	14.07	1.86	18.73	3.08
Post injection 6 months	14.13	1.89	17.53	3.39



The mean ODIS score at post injection three weeks, three months and six months were significantly less in group B compared to group S ($p<0.001$).

Table 12. Outcome – BDIS

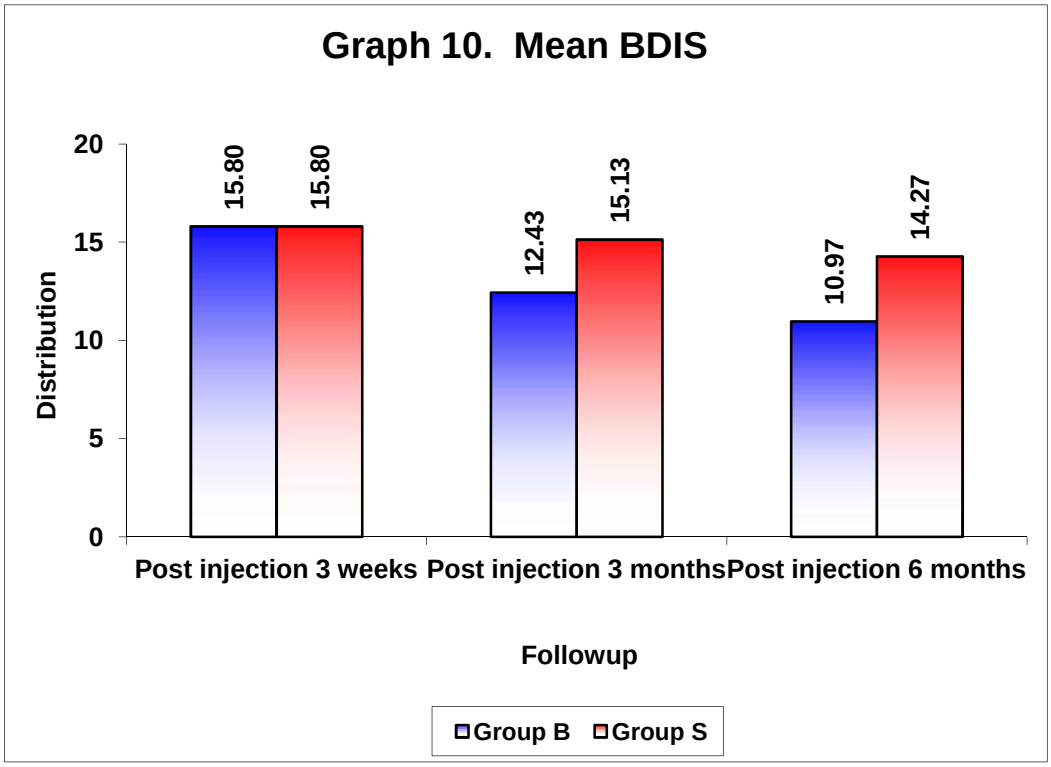
Duration	Pain	Group B (n=30)		Group S (n=30)	
		Number	**Percent**	**Number**	**Percent**
Post injection 3 week	Mild	13	43.33	12	40.00
	Moderate	17	56.67	16	53.33
	Severe	0	0.00	2	6.67
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=2.070		**DF=2**		**p=0.355**
Post injection 3 months	Mild	21	70.00	13	43.33
	Moderate	9	30.00	17	56.67
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=3.325		**DF=2**		**p=0.068**
Post injection 6 months	Mild	25	83.33	18	60.00
	Moderate	5	16.67	12	40.00
	Severe	0	0.00	0	0.00
	Total	**30**	**100.00**	**30**	**100.00**
	x^2=0.085		**DF=2**		**p=0.085**

In the present study the assessment of depression based on BDIS showed 83.33% patients with mild and 16.67% with moderate depression at six months follow up. However, no statistically significant difference was observed between BDIS in group B and S at all the follow ups.

Table 13. Mean BDIS

Followup	Group B (n=30)		Group S (n=30)	
	Mean	SD	Mean	SD
Post injection 3 weeks	15.8	2.95	15.8	3.12
Post injection 3 months*	12.43	3.98	15.13	2.96
Post injection 6 months*	10.97	3.86	14.27	3.18

***p<0.05**



The mean BDIS score were comparable in both the groups at post injection three weeks. But at post injection three months and six months mean BDIS scores were significantly less in group B compared to group S.

Table 14. Mean SLRT

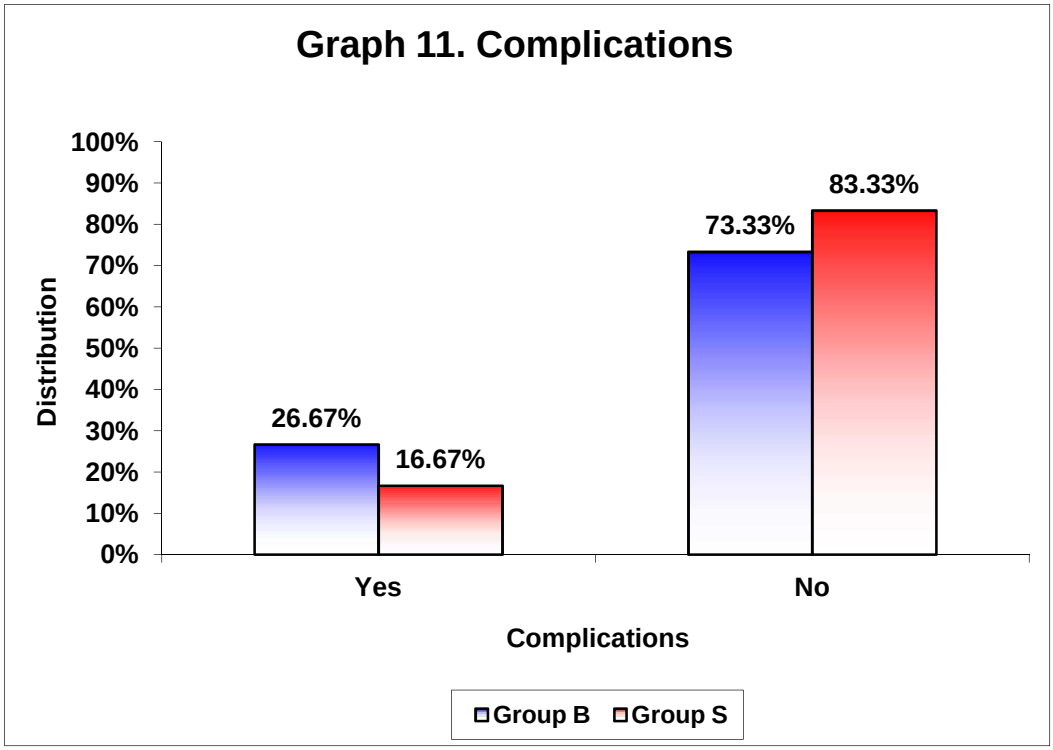
	Right leg				Left leg			
Followup	**Group B (n=30)**		**Group S (n=30)**		**Group B (n=30)**		**Group S (n=30)**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
3 weeks	84	4.98	79	9.6	79.93	14.95	78.67	10.08
3 months	82.67	5.83	79	9.6	81	6.62	78.67	10.08
6 months	80.33	9.28	78	8.87	76.27	16.57	76.67	8.02

The mean SLRT for right leg and left leg are as shown in Table 14. There was no statistically significant difference between the two groups at all the follow ups.

Table 15. Complications

Complications	Group B (n=30)		Group S (n=30)	
	Number	**Percent**	**Number**	**Percent**
Yes	8	26.67	5	16.67
No	22	73.33	25	83.33
Total	**30**	**100.00**	**30**	**100.00**

x^2=0.3928 **DF=1** **p=0.530**

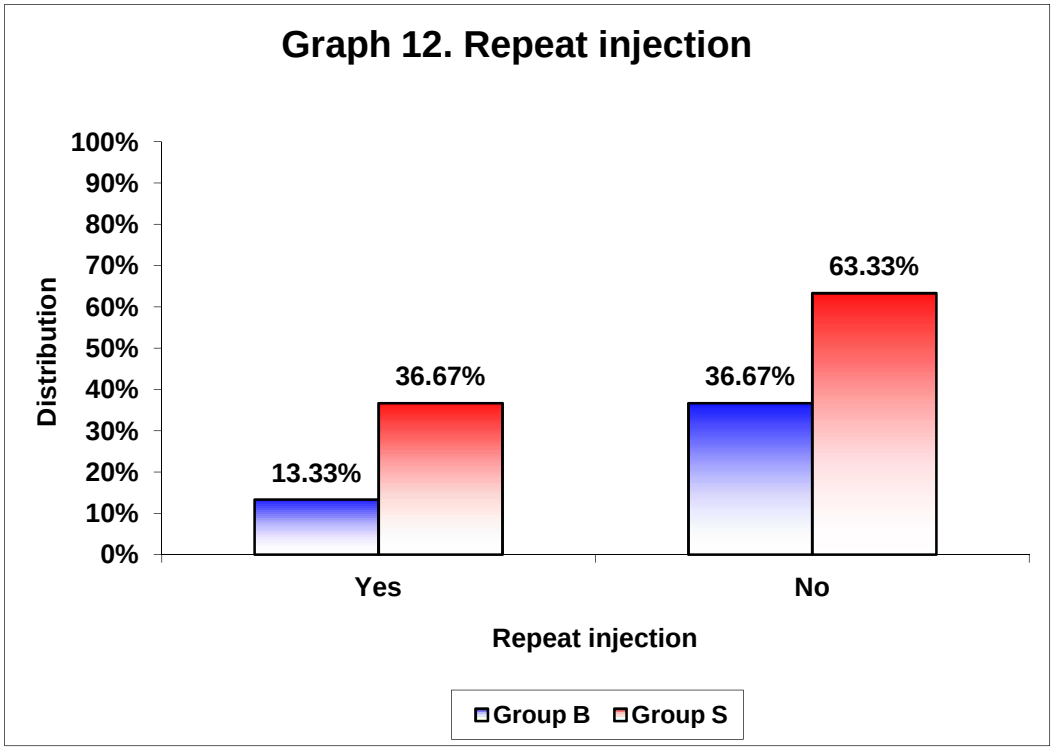


In the present study among the patients with group B 26.67% had complications compared to 16.67% in group S and this difference was statistically not significant.

Table 16. Repeat injection

Repeat Injection	Group B (n=30)		Group S (n=30)	
	Number	Percent	Number	Percent
Yes	4	13.33	11	36.67
No	26	86.67	19	63.33
Total	**30**	**100.00**	**30**	**100.00**

x^2=3.200 DF=1 p=0.073

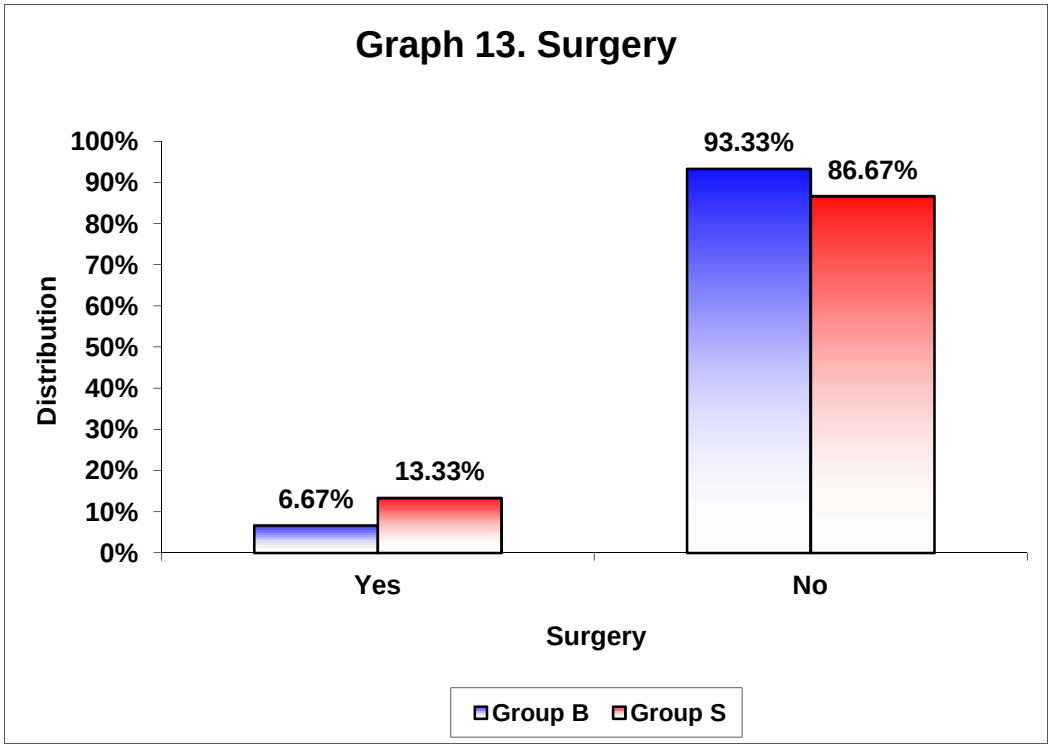


In this study among the patients with group B 13.33% required repeat injections whereas in group S 36.67% required repeat injection. However, this difference was statistically not significant (p=0.073).

Table 17. Surgery

Surgery	Group B (n=30)		Group S (n=30)	
	Number	Percent	Number	Percent
Yes	2	6.67	4	13.33
No	28	93.33	26	86.67
Total	**30**	**100.00**	**30**	**100.00**

x^2=0.1852 **DF=1** **p=0.667**



In this study among the patients with group B 6.67% underwent surgery compared to 13.33% in group S. However, this difference was statistically not significant (p=0.667).

DISCUSSION

Even though the prevalence of persistent Low Back Pain (with or without radicular pain) is high and the morbidity accompanying it is substantial,[121] its etiology remains controversial.[122] Intervertebral disc can contribute to LBP through various processes i.e. degeneration, herniation, inflammatory reactions.

In 1901, Sicard introduced the injection of cocaine through the sacral hiatus into the epidural space.[122,123] Ever since, CEIs are commonly used when dealing with chronic low back and/or radicular pain[124] in order to address potential pathology adjacent to the epidural space.

The caudal approach to the epidural space is the earliest known technique for epidural steroid injection.[125] This approach however, did not gain universal recognition until 1925, when Viner popularized its use for the treatment of sciatica.[125] Evans was the first to report good results in patients undergoing CEI containing only saline,[122] he attributed them to the physical displacement of the neuronal elements and to the stretching and lysis of the neuronal adhesions causedby the injectate.[121]

Extensive literature research revealed only 4 randomized, double blind prospective studies assessing the efficacy of CEI.[126] Dansfield *et al*[126] compared caudal epidural injection with nerve root block and found both treatments to be effective with no significant differences. Breivik *et al*[126] evaluated role of steroid and saline in caudal epidural injection and concluded that during the initial post-CEI period, there was a significant difference between the 2 groups, favoring the steroid group. Bush and Hillier[127] evaluated the injections containing steroid & saline & concluded that short term they very effective but long term potency was variable.

Matthews *et al*[122] compared bupivacaine and methylprednisolone acetate or lignocaine and reported negative short-term and positive long-term relief in favor of the steroid group. Epidural Injection Therapy should form the next line of treatment before any surgery is contemplated.

We assessed and compared the efficacy of CEIs containing a preparation of steroid and local anaesthetic or normal saline in 2 (statistically comparable) groups of patients with chronic LBP and radiculopathy.

The present one year randomized controlled trial was conducted at at Department of Orthopedics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum attached to Jawaharlal Nehru Medical College, KLE University, Belgaum. A total of 60 patients with chronic low backache with radiculopathy were included in the study. Based on the computer generated randomization the selected patients were randomized into two groups namely Group B (Bupivacaine group) and Group S (Normal saline group).

In the present study slight male preponderance was seen with male to female ratio in group B was 2:1 and in group S it was 1.5:1. The reason was linked to the nature of their occupation like farming & heavy labour. 30% of the patients in study were farmers. However both the groups were comparable (p=0.479). Most of the patients in both the groups (50% and 46.67%) were aged between 31 to 45 years. The mean age in group B was 41.70 ± 11.55 years and in group S it was 42.97 ± 10.93 years (p=0.664).

In the present study 50% of patients in group B had right sided radiculopathy and in group S the same was present in 43.33% of patients. However, 16.67% patients each in both the groups radiculopathy was absent (p=0.8289).

In the present study, majority (76.67%) of the patients in both the groups had pain for duration less than 12 months. The mean duration in group B was 12.63 ± 12.54 months and in group S it was 13.57 ± 12.72 months. Both the groups were comparable in terms of duration (p=0.776). 63.33% and 76.67% of patients in both groups presented with spinal tenderness and spasm. Hence in the present study both the groups were comparable.

In this study, at post injection day 2 pain was mild in group B among all the patients (100%) whereas in group S 63.33% patients had moderate pain. This difference was statistically significant suggesting better pain relief in group B at post injection day two follow-up (p=0.001). Post injection third week 73.33% patients reported mild and 26.67% reported moderate pain and post injection three months 70% reported mild and 30% reported moderate pain suggesting less pain in group B. In this study, at post injection day 2, 3 weeks follow up and 3months follow up pain relief on VAS scale was more and statistically significant in group B. However, pain scores at six months in both the groups were comparable.

Also the number of cases where VAS was zero was much higher in the bupivacaine group compared to the normal saline group.

In the present study the Oswestry disability index scores taken in both the groups were statistically comparable prior to the intervention. The mean ODIS score at post injection three weeks, three months and six months were significantly less in group B compared to group S. Minimal and moderate disability was recorded in 20% and 80% of patients in group B compared to 46.67% each with minimal and moderate disability in group S. This difference was statistically significant (p=0.019). At post injection three minimal and moderate disability was present in 10% and 90% of the

patients, respectively in group B as compared to 6.67% and 63% months in group S. At post injection six months minimal disability was present in 10% patients and 90% patients reported moderate disability in group B whereas 16.67% and 73.33% of patient in group S reported minimal and moderate disability respectively. However this difference was statistically not significant. Severe disability was seen only in patients in group S. Oswestry disability index scores improved in both the groups but the improvement was statistically significant in the bupivacaine group.

The mean BDIS score were comparable in both the groups at post injection three weeks. But at post injection three months and six months mean BDIS scores were significantly less in group B compared to group S.

Straight Leg Raising Test (SLRT) was measured in both the groups for all cases prior to the intervention as well as at regular follow ups. 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. It was an added outcome measure. The main outcomes were measured using the structured scores. An improvement in SLRT was seen in both the groups following intervention but there was statistically significant improvement only at post injection day 2 follow up. At subsequent follow ups, in terms of SLRT both groups were statistically comparable.

In this study among the patients with group B 13.33% required repeat injections whereas in group S 36.67% required repeat injection. However, this difference was statistically not significant (p=0.073).

In this study among the patients with group B 6.67% underwent surgery compared to 13.33% in group S. However, this difference was statistically not significant (p=0.667).

Hypotension was encountered during the procedure and was seen in nearly 21 % of the patients. The difference was not statistically significant in both the groups. It was deemed as a complication of the needle placement in the caudal region leading to a vaso-vagal response.

A study[128] enrolled 100 consecutive patients listed for caudal epidural by two spinal orthopedics consultants with lumbosacral radiculopathic pain. Patients were randomized to either steroid injection (80 mg triamcinolone) with 18 ml 0.25% bupivacaine or steroid injection with 18mls Normal saline. Pre-injection maximal (Pmax) and average (Pav) pain scores and Oswestry Disability Index (ODI) scores were obtained for each patient. Pain scores were repeated at 48 hours, 2 weeks and 8 weeks with a second ODI. Results showed a significant improvement in both Pav and Pmax over the study period for both groups. There was no significant difference in either group for Pav or Pmax at any stage of the study. Those patients who received a mixture containing bupivacaine underwent a significant improvement in their ODI in contrast to those who were injected with the saline mixture. There was also a significant difference in the ODI between groups. The study concluded that, epidural bupivacaine injection appears to enhance the effect of epidural steroid injection with adequate levels of monitoring and access to specialist support.

To date, very few studies have reported efficacy of caudal epidural injections of bupivacaine and the findings of the present study could be compared to these studies as the methodology of assessment and outcome variable are different.

CONCLUSION

On the basis of our results obtained, plea is made to use caudal epidural injections much more often than hitherto for a therapuetic purpose

Based on the results of this study it may be concluded that, caudal steroid injection with bupivacaine provided better outcome with regard to pain, disability and depression. Also caudal steroid injection with bupivacaine resulted in fewer repeat injections and avoided surgery.

Plea is also made to use anaesthetic agent Bupivacaine in concentration of 0.25%. There may be a beneficial effect on the local pain pathways by the bupivacaine that outlasts the direct pharmacological effect.

SUMMARY

Low back ache & sciatica together constitute a major problem to our society. The causative factor in majority of the cases is an intervertebral disc prolapse.

Diagnosis is established by clinical examination & by excluding other conditions through laboratory & other investigations. MRI is the gold standard diagnostic tool.

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.

Keeping in view that all forms of conservative treatment of this problem are effective in some way or the other, they have been enumerated.

Epidural steroid injections (ESIs) have been performed for decades and are one of the most common spinal injection procedures performed. This palliative procedure is a safe and effective means for treating low back pain.

The present study was undertaken to compare the effect of Bupivacaine versus Normal Saline in Caudal Epidural Steroid injection in the treatment of chronic low backache.

The caudal route was preferred because of its relative freedom from complications, simplicity and ease with which it can be perfected by an average man.

We have analysed 60 cases of chronic low back ache due to disc protrusion which failed to respond to other forms of treatment & thus were subsequently treated with epidural injections.

. Based on the computer generated randomization the selected patients were randomized into two groups namely Group B (Bupivacaine group) and Group S (Normal saline group).

In the present study slight male preponderance was seen with male to female ratio in group B was 2:1 and in group S it was 1.5:1. 50% of patients in group B had right sided radiculopathy and in group S the same was present in 43.33% of patients. The mean duration in group B was 12.63 ± 12.54 months and in group S it was 13.57 ± 12.72 months.

The mean pain scores at post injection six months were less in group B (2.60 ± 1.28) compared to group S (3.00 ± 1.31). The mean ODIS score at post injection three weeks, three months and six months were significantly less in group B compared to group S ($p<0.001$). The mean BDIS score were comparable in both the groups at post injection three weeks. But at post injection three months and six months mean BDIS scores were significantly less in group B compared to group S. Among the patients with group B 13.33% required repeat injections whereas in group S 36.67% required repeat injection and 6.67% patients in group B underwent surgery compared to 13.33% in group S.

Overall the present study showed caudal steroid injection with bupivacaine provided better outcome with regard to pain, disability and depression. Also caudal steroid injection with bupivacaine resulted in fewer repeat injections and avoided surgery.

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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

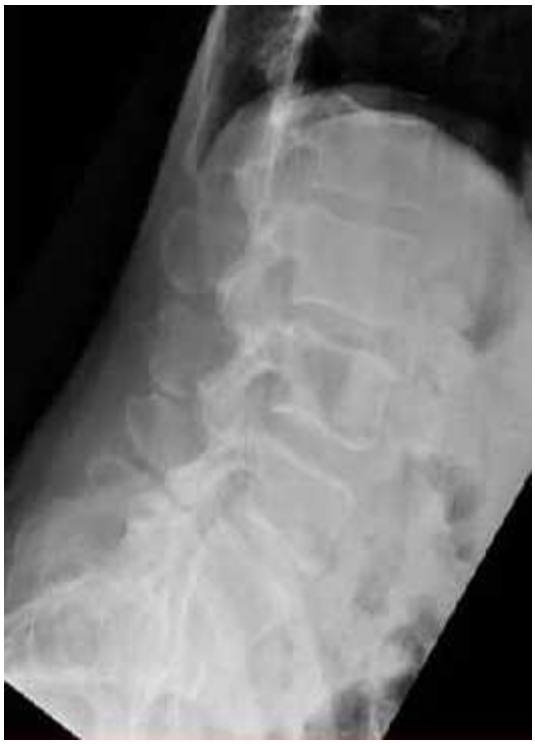
Score <15: Mild Depression

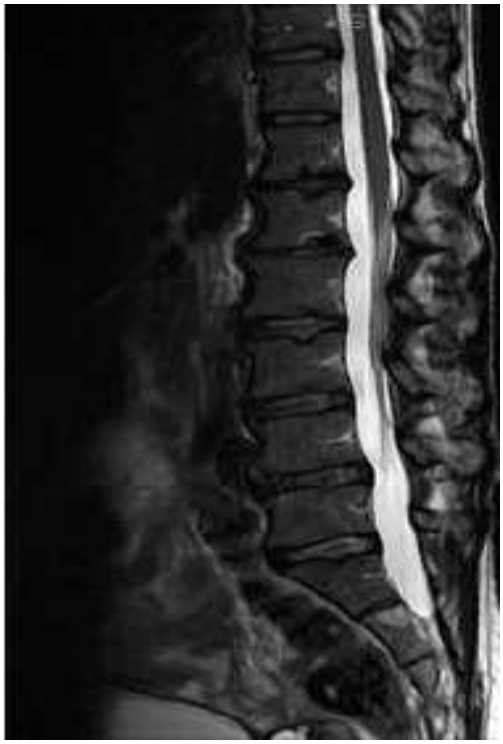
Score 15-30: Moderate Depression

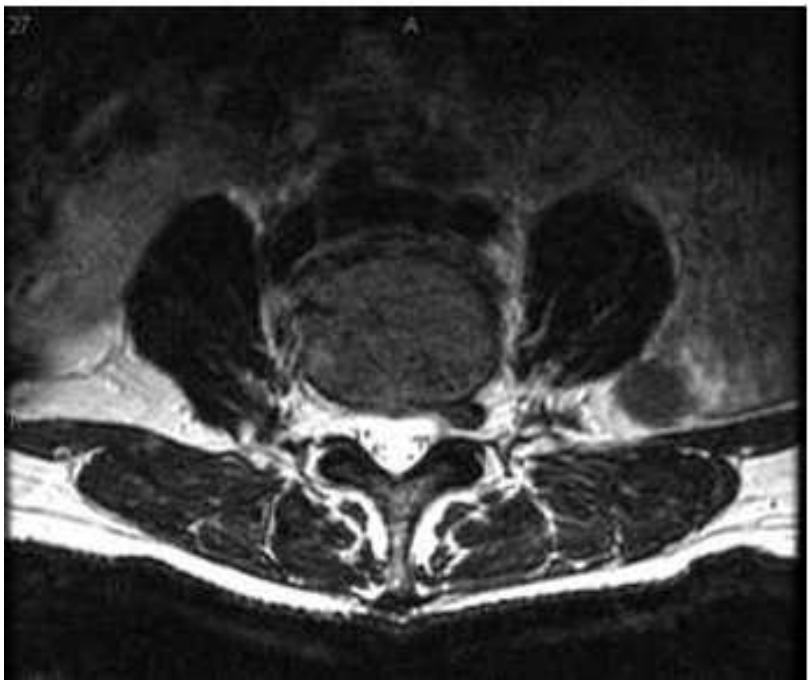
Score >30: Severe Depression

Few Typical Cases

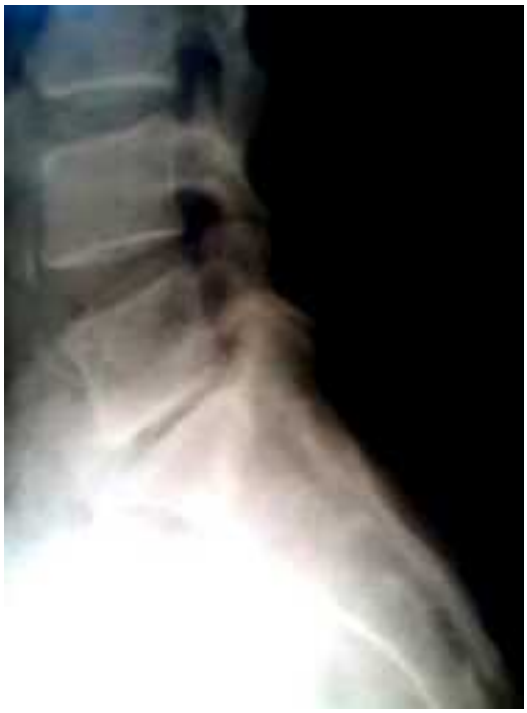
Case 1 – Left sided sciatica, reduced disc space L 4 - L 5, MRI of the lumbar spine shows a far lateral focal disc protrusion at the level of the L4-5 disc.

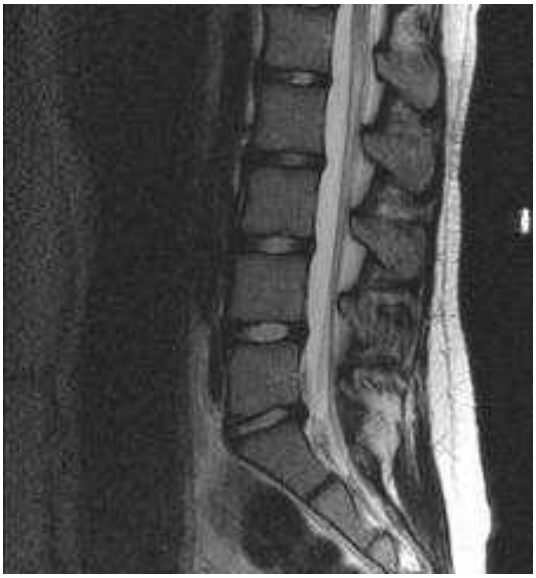


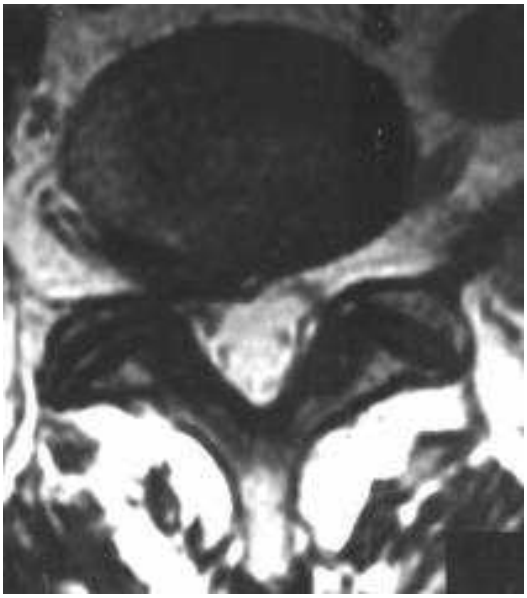




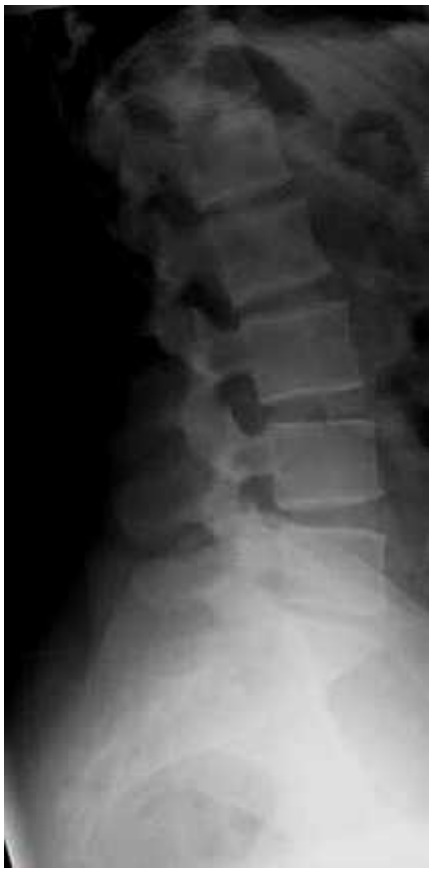
Case 2 – Left sided sciatica, MRI of the lumbar spine shows disc protrusion at the level of the L4-5 and L5, S1

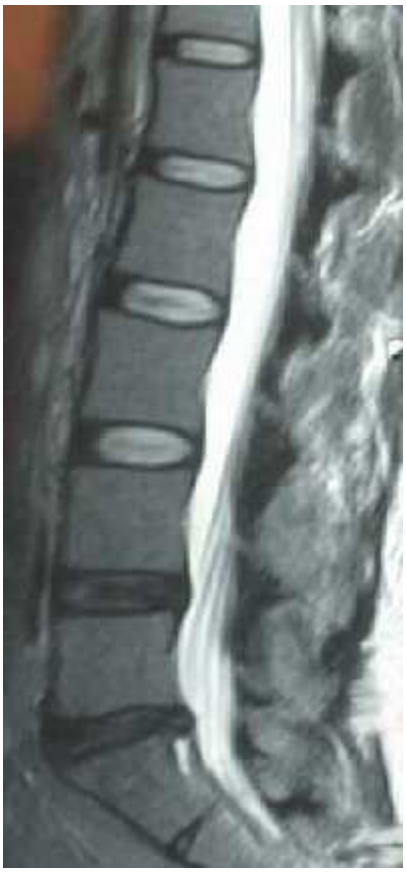


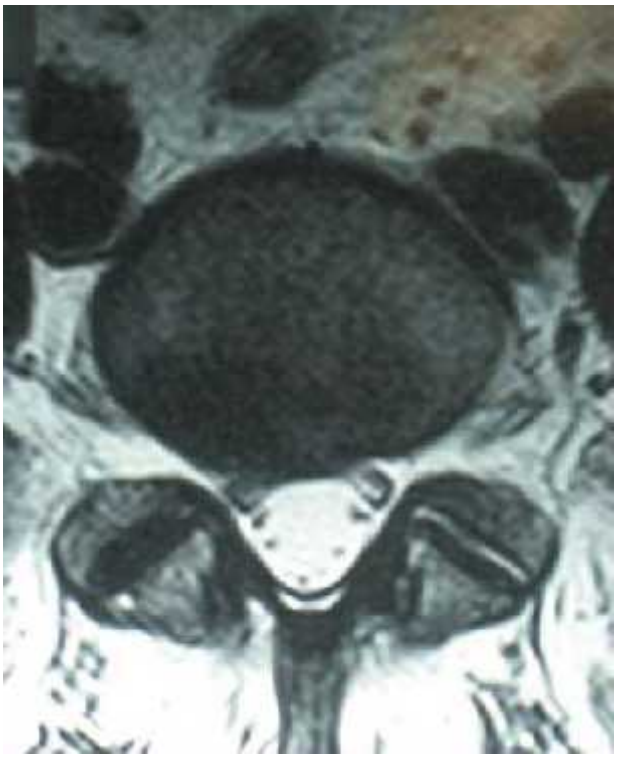




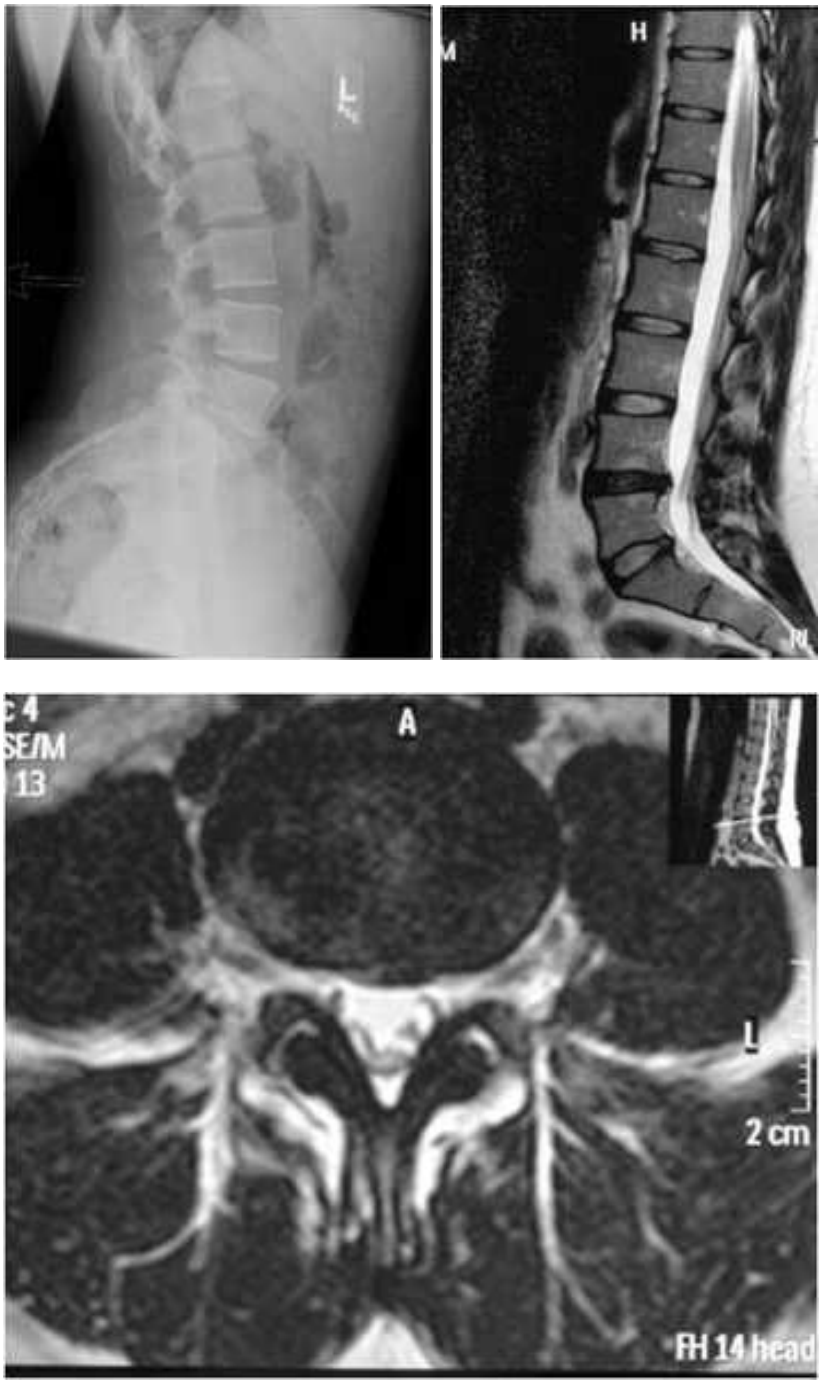
Case 3 – No radiculopathy. Reduced disc space at L5 S1. MRI of the lumbar spine shows Right sided disc protrusion at the level of the L5 S1 disc



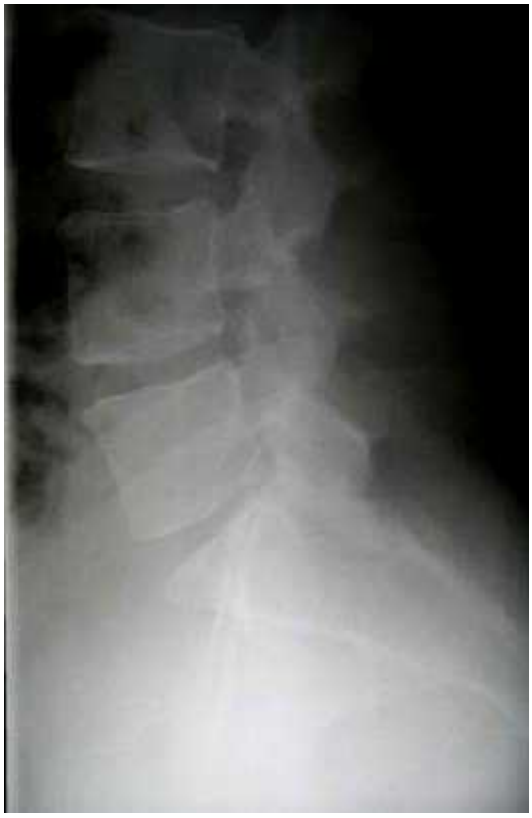


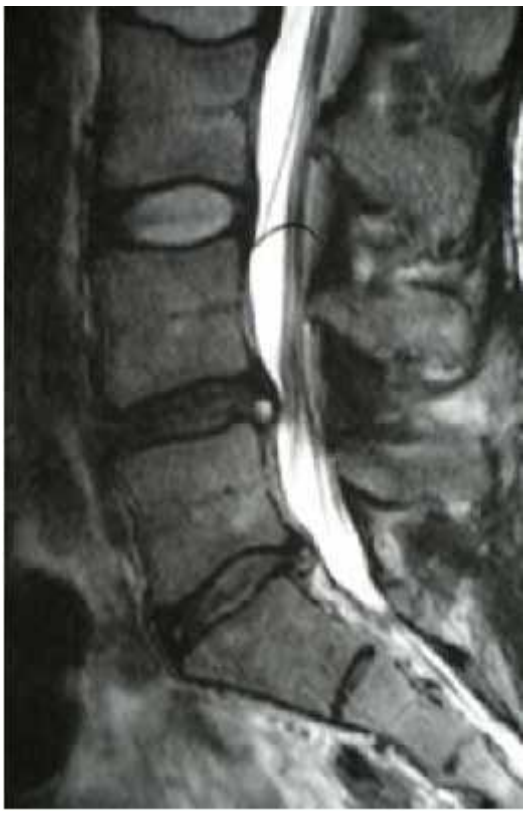


Case 4 – Bilateral radiculopathy. Retrolistheis at L 4, 5. MRI of the lumbar spine shows Central disc protrusion at the level of the L4,5 disc

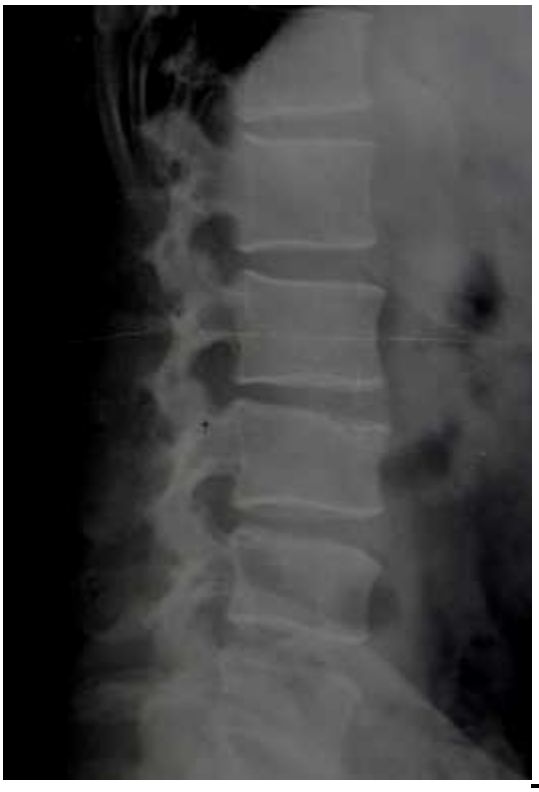


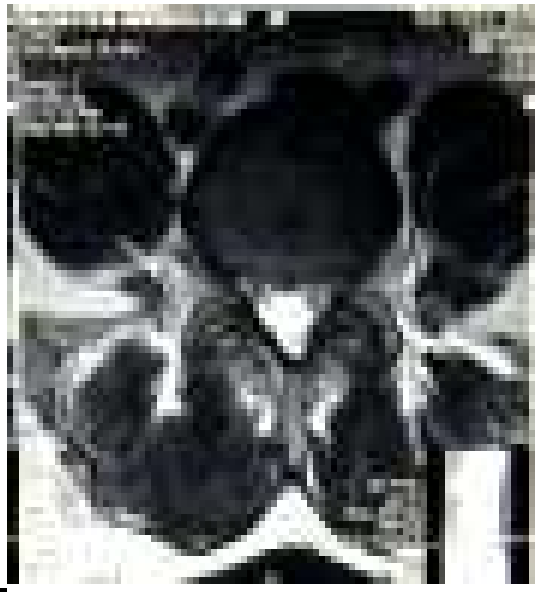
Case 5 – No radiculopathy. Reduced disc space at L4,5 and L5 S1. MRI of the lumbar spine shows Disc bulge at the level of the L4,5 and L5 S1.



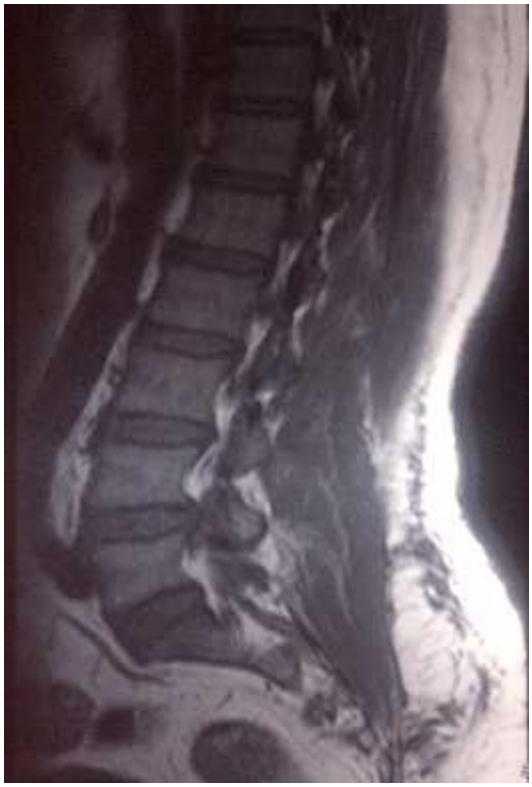


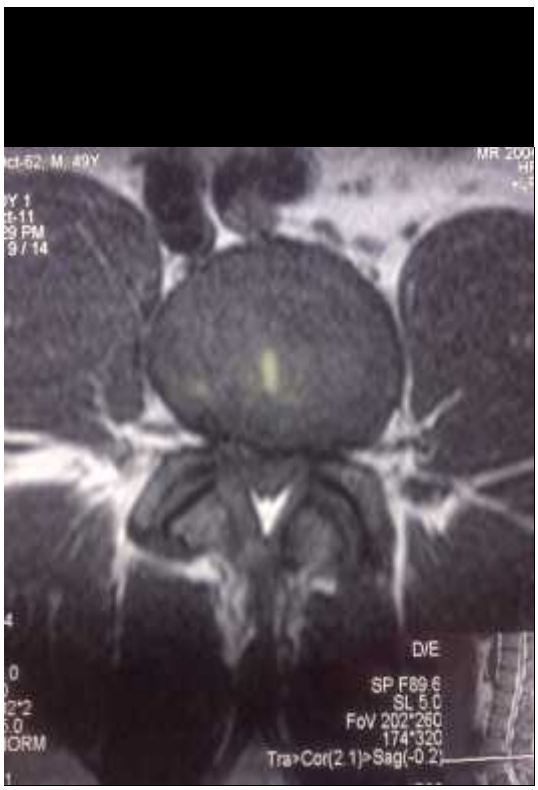
Case 6 – Left sided sciatica, reduced disc space at L 3 - L 4, with para-central disc bulge on MRI.



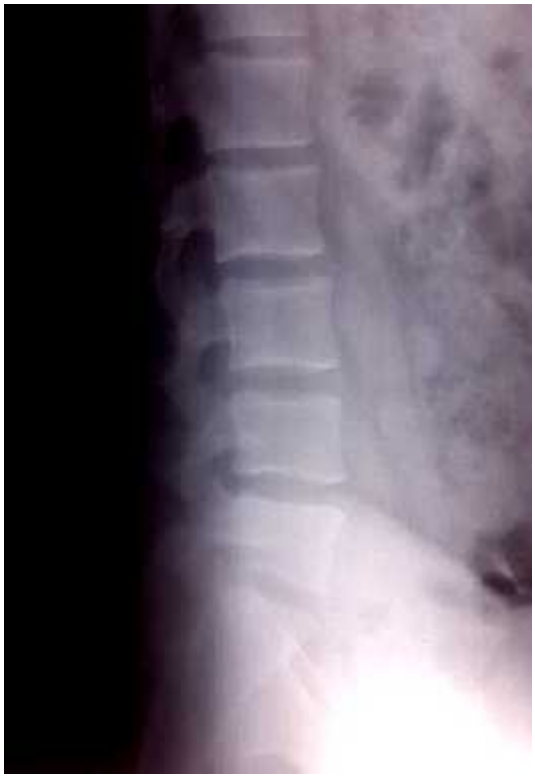


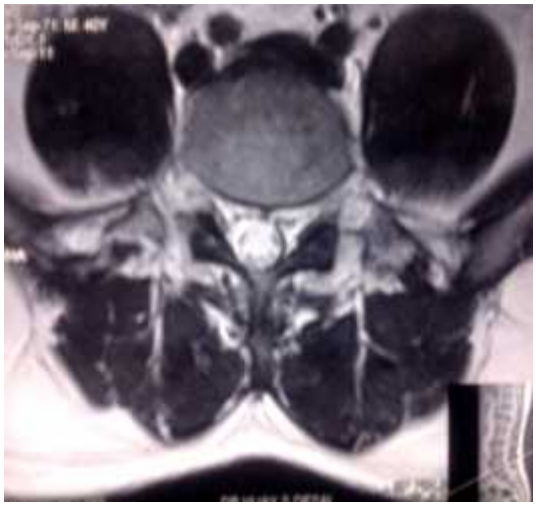
Case 7 – Bilateral radiculopathy. L 4 - L 5 central and para-central disc bulge on MRI



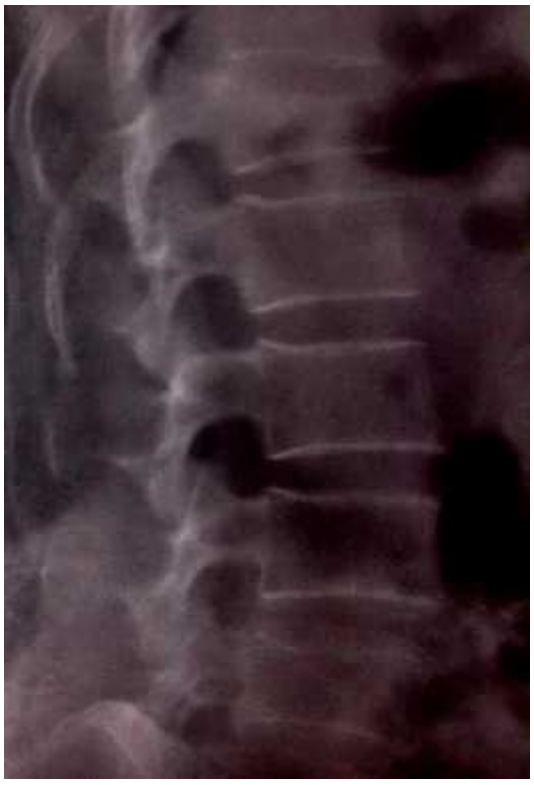


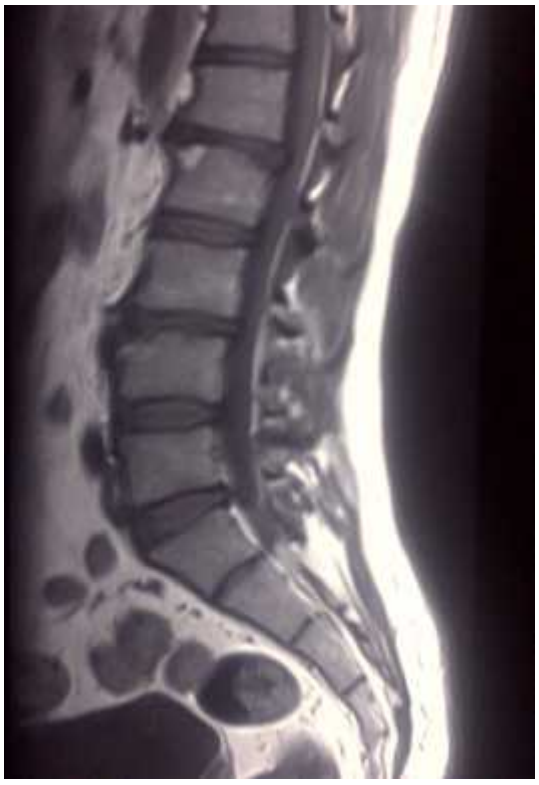
Case 8 – Severe L 5 radiculopathy, normal radiographs. On MRI- paracentral disc protrusion



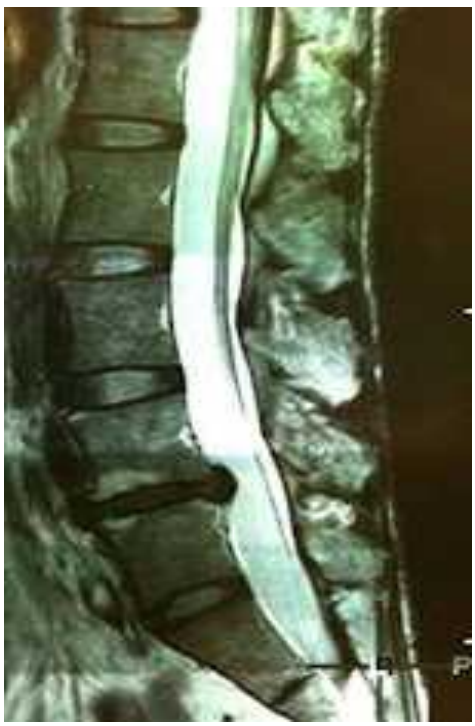


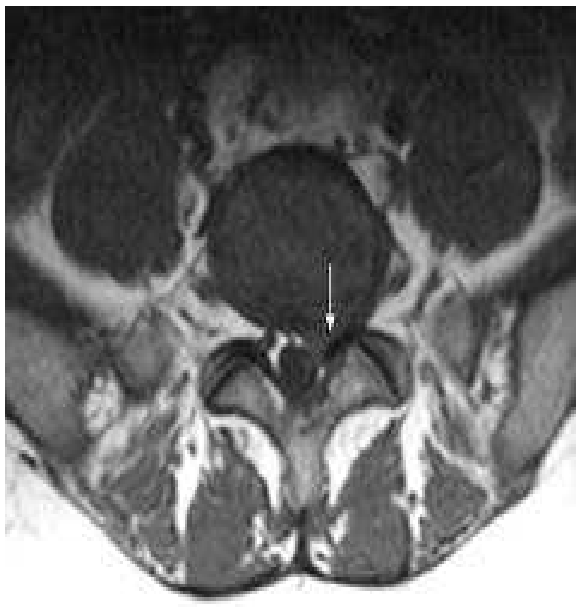
Case 9 – Bilateral radiculopathy, X ray picture shows reduced disc space at L 4 - L 5, with multiple disc bulges on MRI.



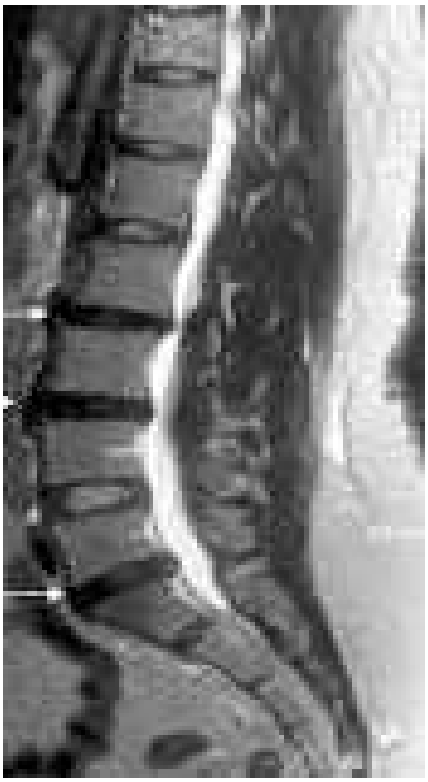


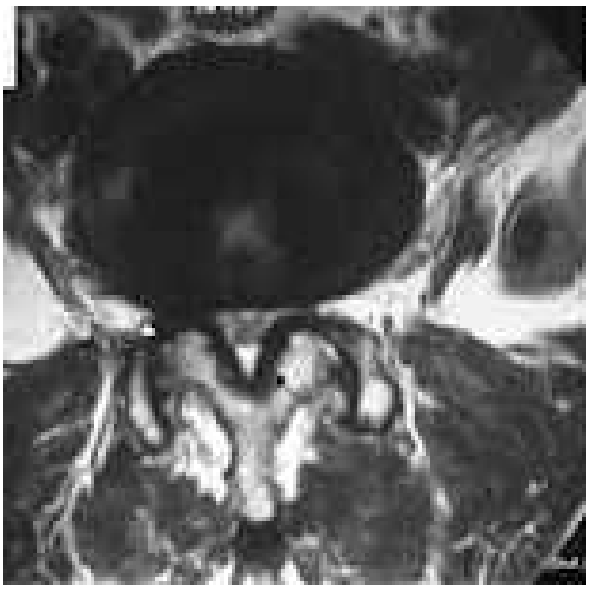
Case 10 – Left sided sciatica, X ray picture shows reduced disc space at L 4 - L 5, with left sided disc prolapse on MRI.





Case 11 Bilateral radiculopathy, multiple level disc bulges on MRI and with paracentral protrusion at L 3,4 level





INFORMED CONSENT

Mr/Mrs/Ms_____

You are invited to participate in this study

TITLE OF THE STUDY:"EFFECT OF BUPIVACAINE VERSUS NORMAL SALINE IN CAUDAL EPIDURAL INJECTION OF STEROID IN CHRONIC LOW BACK ACHE":A HOSPITAL BASED ONE YEAR RANDOMIZED CONTROLLED TRIAL.

Principal investigator: **Dr. Vishal Kohli**

PROCEDURE OF CAUDAL EPIDURAL STEROID INJECTION:

Procedure is to be carried out in OT in presence of Anaesthetist under proper monitoring

1. **Without Bupivacaine:**

The patient will be placed in the prone position on an operating table.

Neurological Status and straight leg raising test (SLRT) are assessed.

The skin area will be prepared aseptically, from the thoracolumbar junction to the coccyx and parts will be draped.

Try to identify by palpation the sacral hiatus, which is located between the two horns of the sacral cornu.

The soft tissues and the dorsal aspect of the sacrum are infiltrated with 2 to 3 ml of 1% preservative-free Xylocaine without epinephrine.

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 "Orthopedic Triangle".

The needle is then redirected more cephalic, horizontal and parallel to the table, advancing it into the sacral canal through the sacrococcygeal ligament and into the epidural space. Placement is checked using the loss of resistance technique.

Remove the stylet. Aspirate to check for blood or spinal fluid. Inject slowly a 18-ml volume of Normal Saline followed by 2 ml of 40 mg/ml of Triamcinelone Acetate.

Pain is elicited during the injection. Neurological Status is reassessed along with SLRT.

2. With Bupivacaine:

The patient will be placed in the prone position on an operating table.

Neurological status and straight leg raising test are assessed.

The skin area will be prepared aseptically, from the lumbar junction to the coccyx and parts will be draped.

Try to identify by palpation the sacral hiatus, which is located between the two horns of the sacral cornu.

The soft tissues and the dorsal aspect of the sacrum are anaesthetized locally with 2 to 3 ml of 1% preservative-free Xylocaine without epinephrine.

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 "Orthopedic Triangle".

The needle is then redirected more cephalic, horizontal and parallel to the table, advancing it into the sacral canal through the sacrococcygeal ligament and into the epidural space. Placement is checked using the loss of resistance technique.

Remove the stylet. Aspirate to check for blood or spinal fluid. Inject slowly an 18-ml volume of 0.25% Bupivacaine followed by 2 ml of 40 mg/ml of Triamcinolone Acetate.

Pain is elicited during the injection.

Neurological Status is reassessed along with SLRT.

COMPLICATIONS:

1. Infection
2. Post injection pain at the sacral hiatus site of entry.
3. Injection-site ecchymosis.
4. Urinary retention and incontinence
5. Adhesive arachnoiditis
6. Post epidural injection headache.
7. Nerve injury may occur.
8. Intravascular or intraosseous injection may lead to toxicity of local anesthetic e.g., metallic taste in the mouth, oral numbness, dizziness, drowsiness and sedation. Also nausea, seizure and respiratory arrest (apnea) may occur.
9. Bupivacaine tends to produce cardiac toxicity.

10. Intrathecal injection can occur despite a reduced risk of same and is associated with prolonged and/or high subarachnoid block, respiratory distress or arrest, and total spinal anesthesia with risk of death.

ALTERNATIVES:

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 and 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.

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/-

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.

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.

Dr. Vishal Kohli

PG in MS Orthopaedics

Dept. of Orthopaedics,

K.L.E. University,

J. N. Medical College, Belgaum 590010.

Telephone :0831-2473777 Ext:1779

Dr. V.G.MURAKIBHAVI **M.S. (Ortho), D (Ortho), FAOI**

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Principal,

K.L.E. University,

J. N. Medical College, Belgaum 590010.

Telephone :0831-2473777 Ext:1779

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. I have also been explained regarding the benefits and the complications which may arise as consequences of the drugs and the procedure used. My signature below indicates that I have read this entire consent form or it has been read to me, and 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 and Signature :…….

Date and Place :…….

KEY TO MASTER CHART

Dur	Duration
VAS	Visual Analogue Scale
SS/s	Sensory symptoms
MS/s	Motor symptoms
N	No
Y	Yes
Tract	Traction
Physio	Physiotherapy
R/A	Rest and Analgesics
Spinal ROM	Spinal Range of Motion
F	Flexion
E	Extension
Lat F	Lateral Flexion
SLRT S	traight Leg Raising Test
Lt	Left
Rt	Right
M	Motor
S	Sensory
K	Knee Jerk
A	Ankle Jerk
P	Plantar response
ODIS	Oswestry Disability Index Score
BDIS	Beck Depression Inventory Score

PROFORMA

Case No:

NAME: **AGE/SEX:**

IP No: **OCCUPATION:**

ADDRESS:

HISTORY OF PRESENT ILLNESS:

BACK PAIN:

Character:

Location:Dorso-Lumbar **Mid-Lumb** **Low**

Duration:Days **Weeks** **Mont** **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

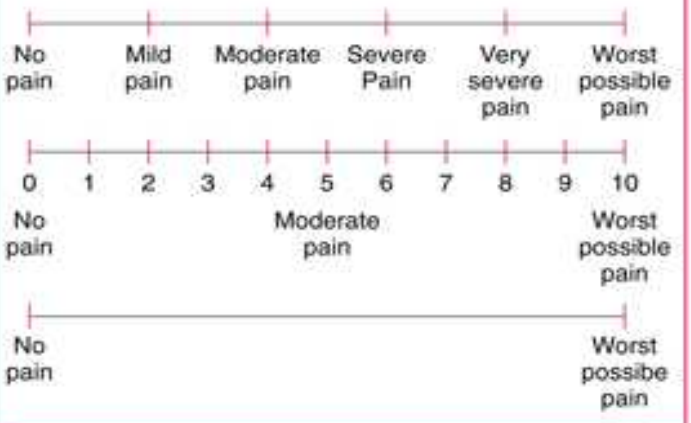
Stiffness

Cause of Pain: Injury **1. Yes**

2. No

If Yes Details:

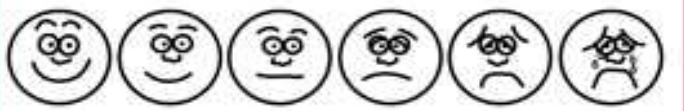
Visual Analog Scale



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: ☐

SYSTEMIC EXAMINATION:

X-RAY Findings:

MRI Findings:

ROUTE OF EPIDURAL STEROID INJECTION:

DATE OF EPIDURAL STEROID INJECTION:

COMPLICATIONS:1.Yes 2. No

Puncture of Duramater ☐

Bleeding ☐

Headache ☐

Convulsions ☐

Neurological ☐

Infection ☐

Results Immediately after the Intervention: ☐

Score: **VAS** ☐ **ODI**

SLRT: **Left** ☐ **Right** ☐

Signature of Examiner **Signature of Guide**

- **Repeat Injection:Due to lack of relief**
  YES ☐

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: ☐

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:

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:

Improvement in Spinal Motion:1. Yes

2. No

If Yes Details:

Signature of Examiner **Signature of Guide**

RANDOMIZATION LIST

001: Bupivacaine Group	011: Normal Saline Group	021: Normal Saline Group	031: Bupivacaine Group	041: Bupivacaine Group	051: Bupivacaine Group
002: Normal Saline Group	012: Bupivacaine Group	022: Normal Saline Group	032: Bupivacaine Group	042: Normal Saline Group	052: Normal Saline Group
003: Bupivacaine Group	013: Bupivacaine Group	023: Bupivacaine Group	033: Normal Saline Group	043: Normal Saline Group	053: Normal Saline Group
004: Normal Saline Group	014: Normal Saline Group	024: Bupivacaine Group	034: Bupivacaine Group	044: Bupivacaine Group	054: Bupivacaine Group
005: Bupivacaine Group	015: Normal Saline Group	025: Bupivacaine Group	035: Normal Saline Group	045: Normal Saline Group	055: Normal Saline Group
006: Normal Saline Group	016: Bupivacaine Group	026: Normal Saline Group	036: Normal Saline Group	046: Normal Saline Group	056: Bupivacaine Group
007: Normal Saline Group	017: Bupivacaine Group	027: Normal Saline Group	037: Normal Saline Group	047: Normal Saline Group	057: Normal Saline Group
008: Bupivacaine Group	018: Bupivacaine Group	028: Normal Saline Group	038: Normal Saline Group	048: Bupivacaine Group	058: Normal Saline Group
009: Bupivacaine Group	019: Bupivacaine Group	029: Bupivacaine Group	039: Bupivacaine Group	049: Bupivacaine Group	059: Bupivacaine Group
010: Bupivacaine Group	020: Normal Saline Group	030: Normal Saline Group	040: Bupivacaine Group	050: Bupivacaine Group	060: Normal Saline Group