LUMBAR ROOT ENTRAPMENT SYNDROMES

****

a correlative study of

CLINICAL, EPIDUROGRAPHIC & OPERATIVE FINDINGS .

by

M. F. GUIRGIS

M.B., B.CH., D.S., D.U., D.Orth., (Cairo),
F.R.C.S. (Eng.), F.R.C.S. (Ed.).

Submitted for the degree of

M.CH. ORTH.

LIVERPOOL

1976

PROTOCOL

1. Acknowledgements.
2. Introduction.
3. Historical Review of Literature.
4. Anatomy of Lumbar Nerve Root Tunnels and pathology of Lumbar Nerve Root Entrapment Syndrome.
5. Patients and Methods.
6. Findings:- Clinical
   Epidurographic
   Operative
7. Analysis of cases and discussion.
8. Summary and conclusions.
9. References.

THIS WORK IS DEDICATED

TO

EL -ZATOUN ORTHOPAEDIC HOSPITAL

CAIRO, EGYPT

ACKNOWLEDGEMENTS

I would like to take this opportunity to express my deepest gratitude to my friend , Dr. Gamal Soliman, M.CH.Orth., Cairo, Senior Consultant Orthopaedic Surgeon whose const--ructive advice and help - depending on the range of his wide experience - were essent-ial to accomplish this work.

My thanks are also to Dr. Nabil Fawzy , Senir Consultant Radiologist for all the , facilities and help he kindly offered in , doing the epidurograms.

In this context also I would like to , thank my wife for reading the manuscript , and checking the references and in general, being tirelessly helpful and supportive.

At last I wish to thank my secretary , Mrs. Christine Robinson who typed the manu-script and who proved to be very patient , co-operative and skilful.

INTRODUCTION

The term "Entrapment" was first used by Russel Brain in 1947 to describe compression of a nerve in a closed compartment created in a fibrous or fibro-osseous tunnel through which the nerve should pass in its normal anatomical course.

The nerve roots gain their exit to the exterior through conduits or tunnels and not through simple intervertebral foramina as was previously conceived.

The attitude in tackling the lumbo-sciatic problem should be changed according to this concept and we should now manage this problem in terms of decompression of the affected nerve roots rather than in terms of simple disc surgery. This attitude has been made more feasible with the use of contrast examination of the epidural canal - epidurography - in investigating these cases.

The aim of this thesis is to discuss the anatomy, pathogenesis and pathology of lumbar nerve root entrapments and to present and discuss the clinical, epidurographic and operative findings in thirty patients treated at El-Zatoun Orthopaedic Hospital in Cairo for lumbo-sciatic problems.

HISTORICAL REVIEW

Sciatica or radiated pains in the lower limbs with or without lower back pain have been known to trouble the human race probably since man adopted his erect posture, as it seems that the spinal column was not designed initially to withstand the compression and flexion forces of the upright attitude and natural evolution did not help the spine as much as it did with other systems in the body.

It was as far back as Imhotep - "He who comes in peace" - the ancient Egyptian god of medicine when this sort of trouble appeared first in the medical literature, in Ebers Papers which showed that he manipulated his patients, and this also appeared on one of the walls of a tomb in Sakarah (a small town near the city of Giza in Egypt). Fig. 1

Other earliest appearances in medical literature were in Hippocrates Papers 460-377 B.C. and in Galenus Papers 132-200 A.D. In the year 1000 Avisenna, the famous Arab physician and scientist manipulated his patients suffering from sciatica in Bagdad using the Hippocratic methods.

It was Vesalius in 1555 who first described the intervertebral disc in his document "De humani corporis fabrica libri septem."

Cotunius in 1764 wrote the first book on sciatica titled "De Ischide Nervosa commentarius.", and he did not mention the intervertebral discs as an underlying cause of sciatica, but he attributed it to neuritis with oedema of the sciatic nerve sheath and subsequent compression of the nerve producing the pain.

In 1838 Key of Guy's Hospital gave the first impression to correlate between disc pathology, lumbar nerve roots and sciatica in his article "On paraplegia depending on the ligaments of the spine", reporting a case of intraspinal disc herniation from the 11th thoracic interspace causing paraplegia and death.

Virchow in 1857 reported a post mortem finding of a herniated intervertebral disc and he attributed it to traumatic causes but his observations passed unnoticed. In the following year Luschka reported another two cases and he thought that if the disc herniations were large enough they might have caused compression of the cord. These observations passed unnoticed as well.

The second book on sciatica was published by Lasègue in 1864, 100 years after Cotunius' book - and was titled "Contribution a l'etude clinique de la sciatique."

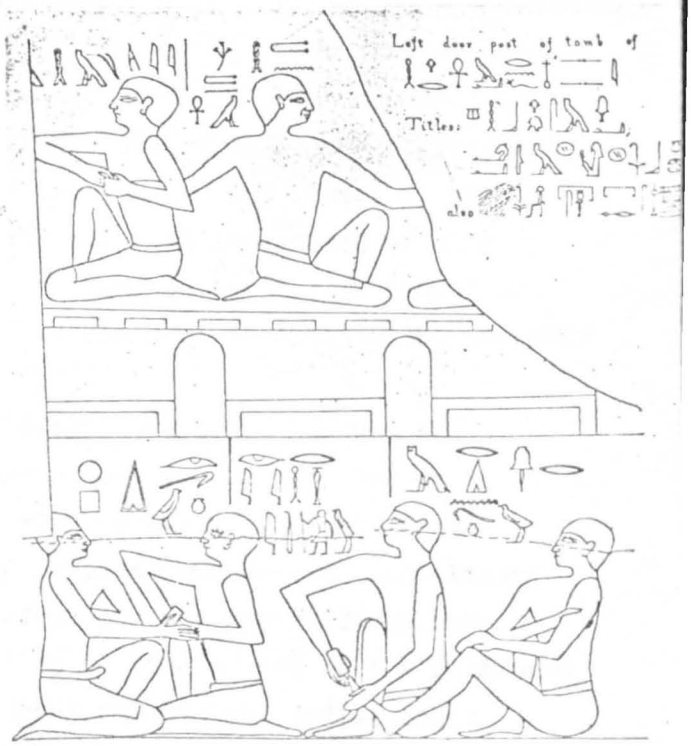
The first description of the characteristic spinal deformity in sciatica patients was given by Charcot in 1888.

In 1896 Kocker reported a case of traumatic rapture of the second lumbar intervertebral disc after a fall from a hieght of 100'. Since then the aetiology of lumbo-sciatic pain ha been the subject of much speculation and with the advances made in the spinal-surgical field the condition of many of these patients operated upon was thought to be due to extra-dural chondromata or fibrochondromata in the spinal cord (Openheim and Krause 1909, Adson and Ott 1922 and Elsberg 1925).

Imhotep *'Ij-m-ḥtp*

" HE WHO COMES IN PEACE "

He lived in the reign of, King Zoser of the IIIrd , dynasty (2800 B.C.). He, was the visier of that , king and the originator , of the magnificent step , pyramid at Saqqara. He was the oldest medical , figure in history writing many books which were con -sidered to be the origin of Ebers, Ed, Smith and Berlin Medical Papyri. He was worshipped later as , the God of Healing.



A drawing of one of the pictures on a wall , of a tomb in Saqqara near the step pyramid , showing manipulation of one of the patients , in progression according to Imhotep's scribes Later scribes used to pour few drops of water before starting to write as an act of respect to his soul.

In 1911 Middleton and Teacher described the case of a patient who felt a sharp snapping pain in his back while lifting a heavy object from the ground and developed paralysis of both lower limbs during the following night. This patient died later from urinary tract infection and the post mortem examination revealed retropulsion of the disc between the 12th thoracic and the 1st lumbar vertebrae and they described the bulging mass as "white and firm and particularly resembled the central part of the intervertebral disc".

In the same year Goldthwait described the relations of lumbo-sacral anomalies to the development of sciatica syndrome.

In 1918 Sicard felt that sciatica was due to radicular affection and he assumed that the site of affection was situated peripheral to the spinal ganglion and was due to changes in the intervertebral foramen which by pressure against the spinal nerve gave rise to sciatica, and he called it "Sciatique Funiculaire". His views were shared by Putti in 1927 who attributed sciatica to funiculitis. Two years earlier in 1925 Danforth and Wilson claimed in an article that sciatica was a symptom of injury to the lower lumbar region, commonly localised to the junction between the 5th lumbar and the 1st sacral vertebrae and that the 5th lumbar root was the one particularly involved.

Then came the famous Mixter and Barr's article in 1934 showing that root compression due to disc herniation in the lumbar region was the commonest cause of sciatica.

Since then a large number of articles appeared in the English, American and European literature, providing a wealth of knowledge about the anatomy, physiology and pathology of disc prolapse and its relations to the nerve roots. (Burns 1937, O'Connel 1943, Inman and Saunders 1947, Charnley 1951 and Armstrong 1952)

The concept that nerve roots gain their exit to the exterior through tunnels rather than simple intervertebral foramina was not stressed until recently in 1975 when Dommisse described the morphological aspects of these tunnels, though earlier observations by Larmon in 1944 gave dimensions to the foramina, yet he did not think in them in terms of tunnels. The clinical importance of this tunnel concept was stressed by Pheasant 1975 in those patients with failed or negative disc surgery, when the cause of the trouble was not dealt with, probably due to failure to appreciate this tunnel concept.

Since the relationship between disc pathology, radiculopathy and the lumbar sciatic syndrome was established, every effort was made to try to establish a system of clinical investigation which accurately localised the site and side of the lesion to facilitate the surgical management. Anciliary methods of investigation, including plain radiography and contrast myelography were used but the degree of accuracy was only up to 85% in most of the published series. The concept of use of the epidural canal in contrast radiographic examination, the so called epidurography, was introduced by Knutsson in 1941 and then Yates in 1965 and Luyendijk and Voorthuisen 1966 published two series using the same technique.

ANATOMY OF LUMBAR NERVE ROOT TUNNELS

Each lumbar nerve root conduit or tunnel is a capacious structure with an inlet, a canal and an outlet. The inlet is large and obliquely placed leading from and forming an integral part of the spinal canal. The length of the tunnels increases proximo - distally and at the level of L5 - S1 the average length in an adult is estimated to be about 2½c.m. The direction of the tunnels is almost vertical at the level of L1 and L2 and then becomes more and more oblique towards the lumbo-sacral level and as a result, exposure to the intervertebral disc is over a greater surface area here.

The boundaries are as follows:- Fig. 2

Superiorly: The under surface of the pedicle of the vertebra above.

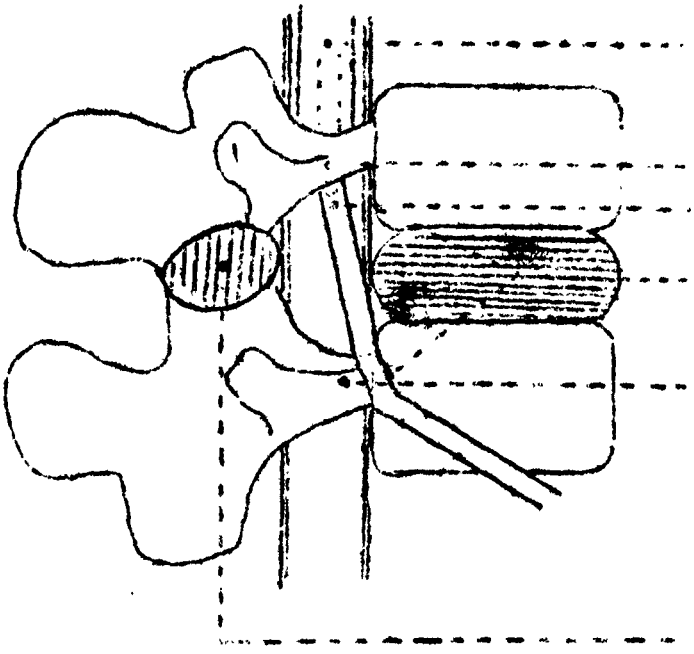
Inferiorly: The superior surface of the pedicle of the vertebra below.

Anteriorly: The upper half is related to the posterior surface of the vertebral body and the lower half to the intervertebral disc. The intervertebral discs are almost rounded at L1 and L2, oval at L3 and kidney shaped at L4 and L5.

Posteriorly: The paravertebral joint (and in particular the anterior surface of the superior articular facet), and the ligamentum flavum.

The S1 nerve root differs in having its walls entirely bony within the body of the first sacral vertebra. Actually it is inaccessible within its bony tunnel except at the inlet of the tunnel where it has a medial relationship to the 5th lumbar nerve root.

The contents of the tunnel are the lumbar nerve roots ensheathed in dural sleeves, radicular vessels, intraspinal branches of the segmental arteries and Batson's plexus of veins. The nerve root occupies only the upper fourth of the tunnel, the rest of the space being occupied by fatty areolar tissue penetrated by the vessels. The outlet of the tunnel is called the "Distribution Point", of the segmental vertebral arteries, a site which requires due attention during surgical procedures. Frequently the transforaminal ligaments (unyielding cords of collagenous tissue that pass anteriorly from various parts of the neural arch to the body of the same or the adjacent vertebra and may be as much as 5mm wide (Larmon 1944)) encroach on the medial parts of the conduits.



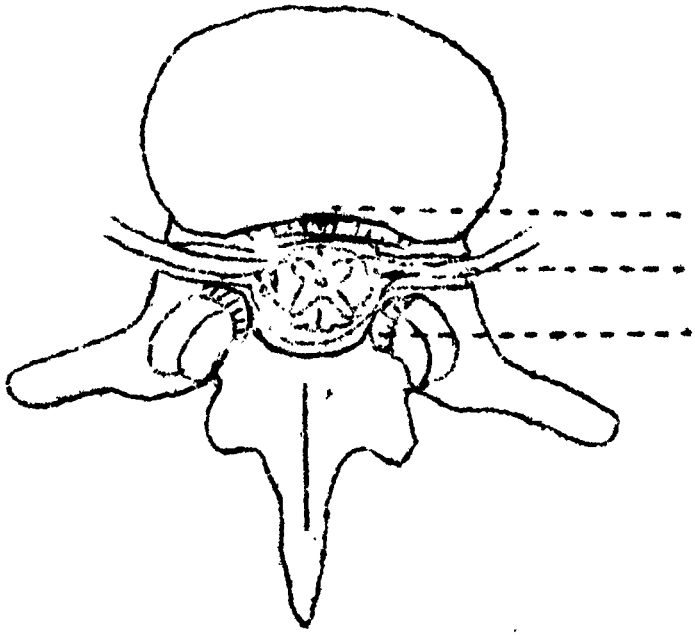


Fig. 2 ANATOMY OF LUMBAR NERVE ROOT TUNNELS

PATHOGENESIS OF ENTRAPMENT RADICULOPATHY

The ensheathed nerve root occupies only the upper quarter of the space in the tunnel. At the point where the nerve penetrates the dura to form the sleeve, the sleeve is firmly attached and immovable. The dural sleeve is also firmly attached where it ends at the outlet of the tunnel. Between those two fixed points very little movement is allowed for the nerve root away from pressure by a prolapsed I.V.D. Commonly this happens in a postero-lateral direction where there is greater exposure of the tunnels to the discs due to their increasing obliquities. Other factors include the progressive narrowing of the posterior longitudinal ligament as it approaches the lumbar region so that at the level of L4-5 &L5-S1 it has half of its original width. At those levels the discs are kidney shaped, therfore the poles of the kidney will protrude lateral to the posterior longitudinal ligament. All these factors plus the fact that the facet planes of the zygo-apophyseal joints in the lumbar region lie in the vertical sagital plane, thus permitting flexion-extension movements and to a much lesser extent lateral flexion and rotation; particularly on foreward flexion - form the basis of the biomechanical theory of Farfan 1970. He beleives that flexion and rotation of the spine causes shearing at the discs especially at the 2 lowest levels.

Those cases without a positive history of trauma come to lie under the second theory of disc pathology namely the biochemical theory of Naylor 197 . It was discovered that the interstitial fluid of the disc matrix contains at least 2 independent glycoproteins. After embryological development the nucleus pulposus becomes sequestrated from contact with the blood stream and hence the immunological tolerance to its proteins is lost. The theory postulates that interference with mucoplysacchsride synthesis/depolymerisation in the disc leads to collagen fibrillation and degeneration. Annular tears thus result with subsequent nuclear prolapse. These annular tears are repaired by ingrowth of granulation tissue and the contact of this with its blood vessels with the nuclear glycoproteins initiates an antigen-antibody reaction of the cell mediated type leading to the development of a hypersesitivity state. The latter will account for the initiation of symptoms in those patients without a positive history of trauma and the chronicity of inflamation in all patients. (Fig. 3)

In any case compression of a nerve root which is inflamed, hypersensitive and irritable from contact with the herniated disc material will result within the closed compartment created in the tunnels.

The fourth lumbar nerve root emerges at the interspace between the fourth

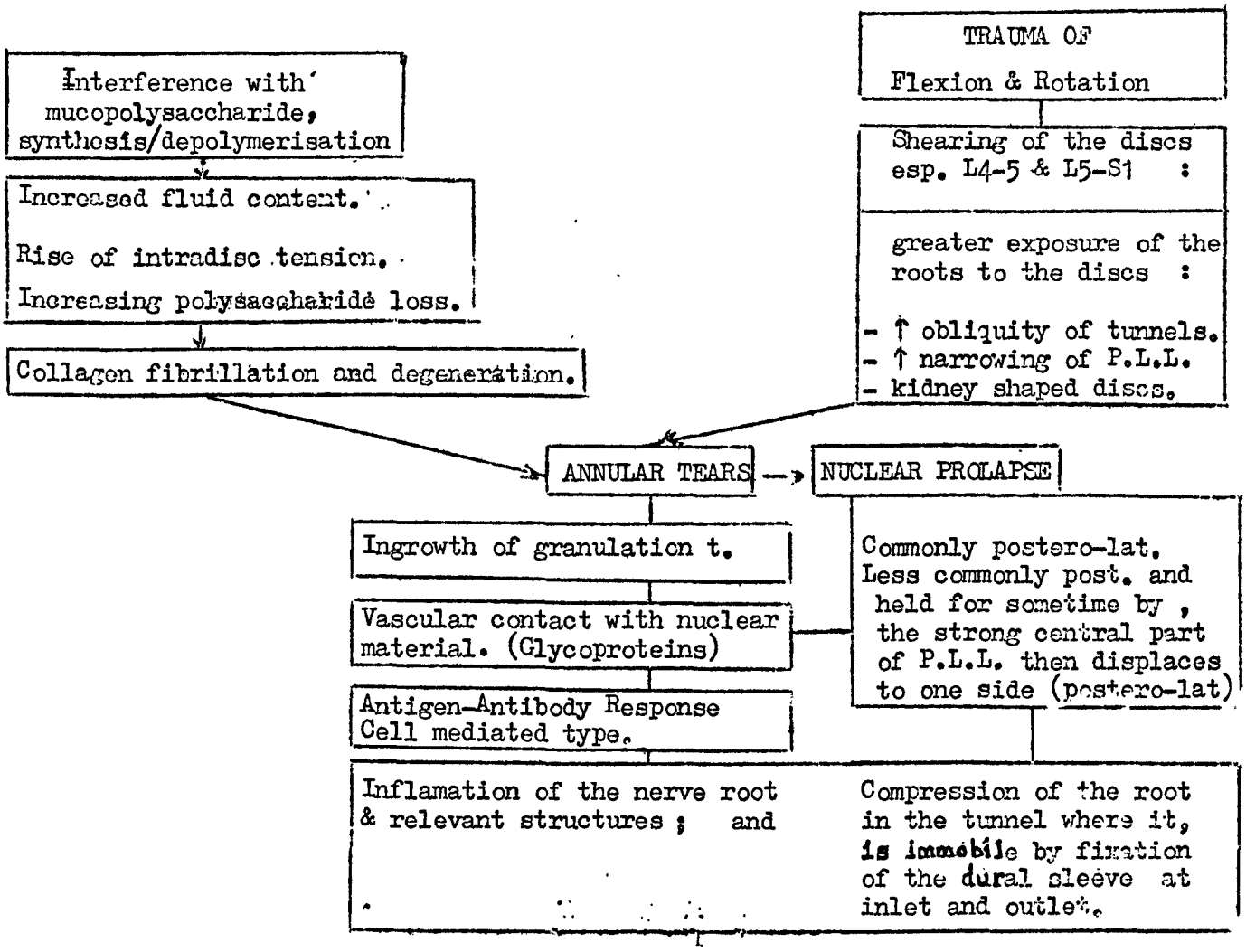


Fig. 3 Pathogenesis of disc pathology.

the fourth and fifth lumbar vertebrae, but the inferior vertevral notch is so deep that the nerve leaves the vertebral canal posterior to the lower end of the fourth lumbar vertebra and thus passes above rather than across the disc at this interspace. In consequence unless the disc protrusion is so massive that it pushes the fourth lumbar nerve against the pedicle or the ligamentum flavum, this root escapes and it is the fifth root - the most lateral nerve to cross the disc that is commonly involved. Since all the nerves below the fifth lumbar nerve root also cross the disc between the fourth and fifth lumbar vertebrae, the first sacral and sometimes even the other sacrals may be involved at this level (Hollinshead 1965).

The root protrusion relationship varies, but as the root crosses the affected disc obliquely from above downwards, it may either be stretched over the prolapse or actually compressed from the inner side (parasaudal) or from the outer side (pararadicular) Harvey Jackson 1947. Fig. 4.

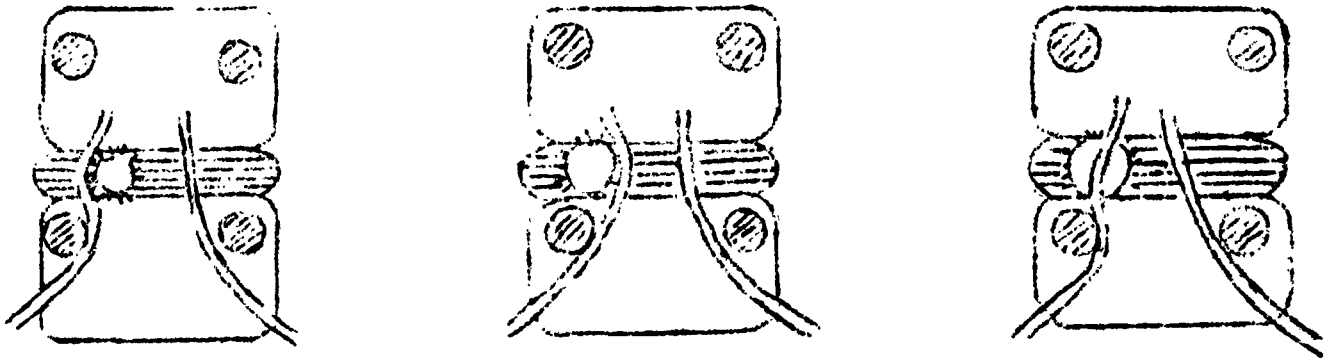
Sometimes though the protrusion is unilateral, yet it might affect two roots if it projects directly lateral into the tunnel at the same level, compressing the roots above and below, or it may be a large dissecting one extending up or down, compressing more than one root. Sometimes even what is called foraminal migration happens when the disc lesion becomes sequestrated and displaced migrating down into the tunnel causing the compression radiculopathy. In these cases at surgical exploration the relevant disc will be found soft and appearing normal, while the affected nerve root will show marked signs of tension, and decompression will not be effected unless this migrating sequestrated part is found and removed, (Macnab 1971).

In some patients a far laterally bulging prolapsed intervertebral disc might compress the nerve root at or immediately after leaving the outlet.

Even in the earliest stages adhesions form binding the root down quite firmly to the protrusion. Histological examination of these early adhesions showed infiltration with lymphocytes and plasma cells conforming with the autoimmune theory for the disc pathology. Later these adhesions become massive and fibrous, and when the protrusion reaches the calcified stage the root may be so adherent that it appears to be partly burried in the bony nodule.

It might not be the primary disc protrusion which causes the entrapment radiculopathy but the secondary consequential effects on all surrounding structures sharing in the formation of the nerve tunnels might provide the basic factors in producing the closed compartment syndrome.

Thus, as a result of disc degeneration and thinning, the pedicles are brought more closely together, decreasing the cephalocaudad diameter of the tunnel. If asymmetrical collapse of the disc results, the pedicle of the upper vertebra as it descends, might kink the emerging nerve root - pedicular kinking. Fig. 5



Paracaudal Pararadicular Subrhizal

Fig. 4 ROOT PROTRUSION RELATIONSHIP

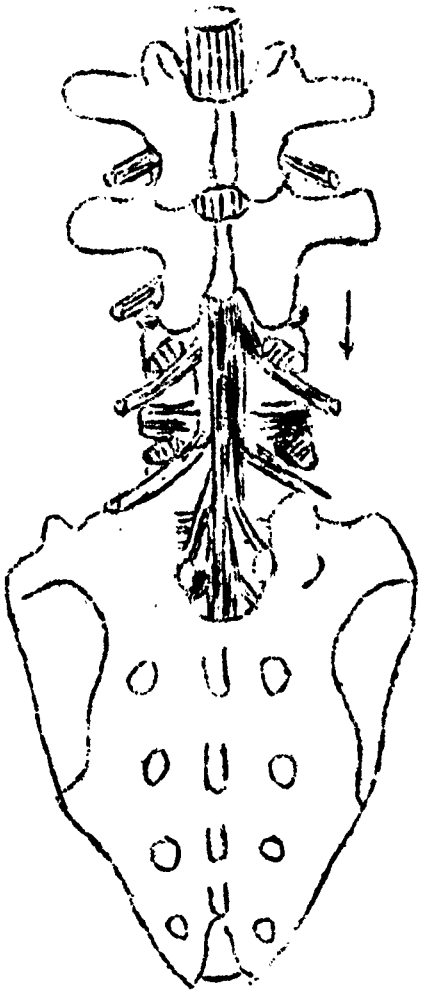


Fig. 5 Pedicular kinking of the root caused by asymmetrical narrowing of the disc space allowing 2 vertebrae to, approach each other more on one side.

As the nerve root begins to move out laterally, still closely applied to the pedicle but now under the pars it might be entangled in the fibro-cartilagenous or fibro-osseous reaction forming at the site of a spondylolisthetic defect. (Gill 1955)

Another secondary effect of the disturbed biomechanics of the region is the resultant thickening and buckling and in late cases, ossification of the ligamentum flavum and its intrusion into the tunnel, causing the entrapment syndrome. Fig. 6

Due to the disturbed biomechanics the zygoapophyseal joints react and undergo osteoarthritic changes. The capsule and synovial membrane of these joints will become distended and allow for the over-riding of the articulations. As a result bony impingement may occur between the tip of the superior articular process and the pedicle or transverse process of the vertebra above. There may be also bony impingement between the tip of the inferior articular process and the lamina of the arch above. These bony impingements may result in the development of new articulation with fibrocartilage bumper formation. Erosion and sclerosis of underlying bone occurs and spur formation results, encroaching on the tunnels. Fig. 7 In these cases the posterior articulation affection might lead also to anterior subluxation of the lower vertebra, causing what is called reverse spondylolisthesis and this may allow the superior articular process from below to subluxate forwards and upwards, into the tunnel, causing a decrease in its anterior posterior diameter. Bony spurs from the backs of the vertebral bodies may be an aggravating factor in these cases. Fig. 8

Hypertrophy of the superior articular facet as a secondary change to the spondylotic process may compress the nerve as it passes underneath its medial border before swinging around the pedicle between the superior articular facet and the dorsal aspect of the vertebral body. This is particularly the case with advanced degenerative disc disease, degenerative spondylolisthesis and degenerative spinal stenosis. Fig. 9

Congenital narrowing of the spinal canal and tunnels produced by thickening of the neural arches in association with interpedicular narrowing and a trifoil configuration of the laminae might produce bilateral radiculopathy but usually they do not present any evidence of impairement of root conduction on recumbance. (Verbiest 1955)

In the case of the 5th lumbar nerve root, it might be trapped at the outlet between the corporo-transverse ligament and the ala of the sacrum.

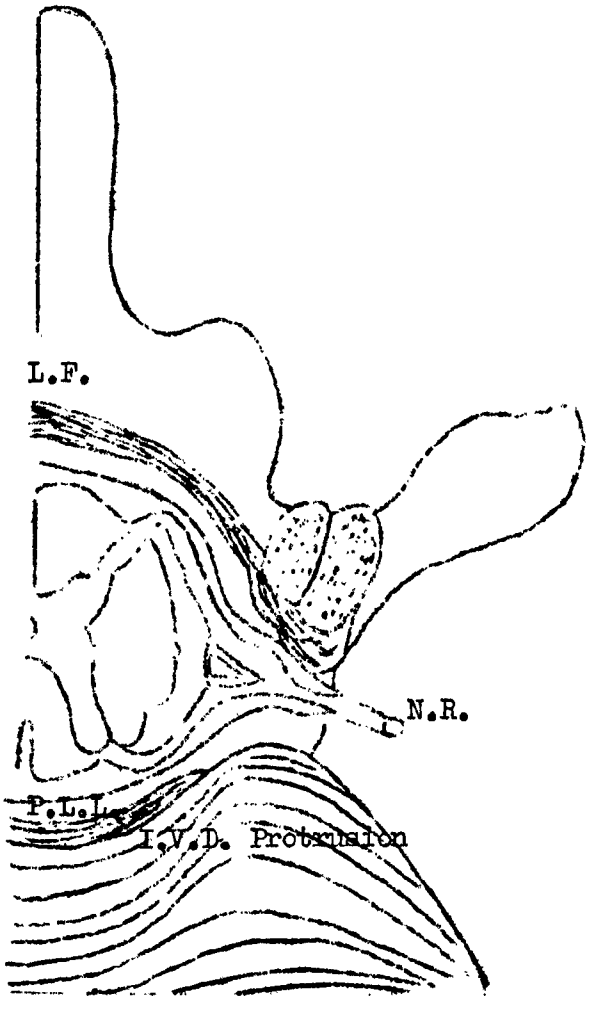


Fig. 6 Lig. Flav. thickening & entrusion into the, tunnel causing entrap.

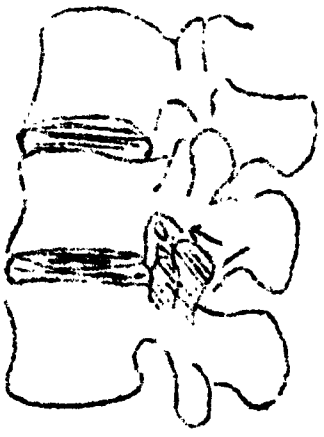


Fig. 7 Post. subluxation of the zygo--apophyseal joint with ostephyte formation encroaching on the , tunnels.

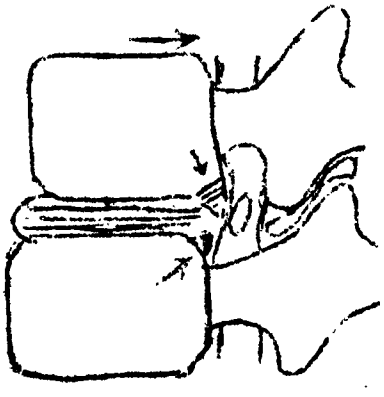


Fig. 8 Retro-displacement of the vertebra above with spur, formation from the back , of the vertebral bodies , encroaching on the tunnels.

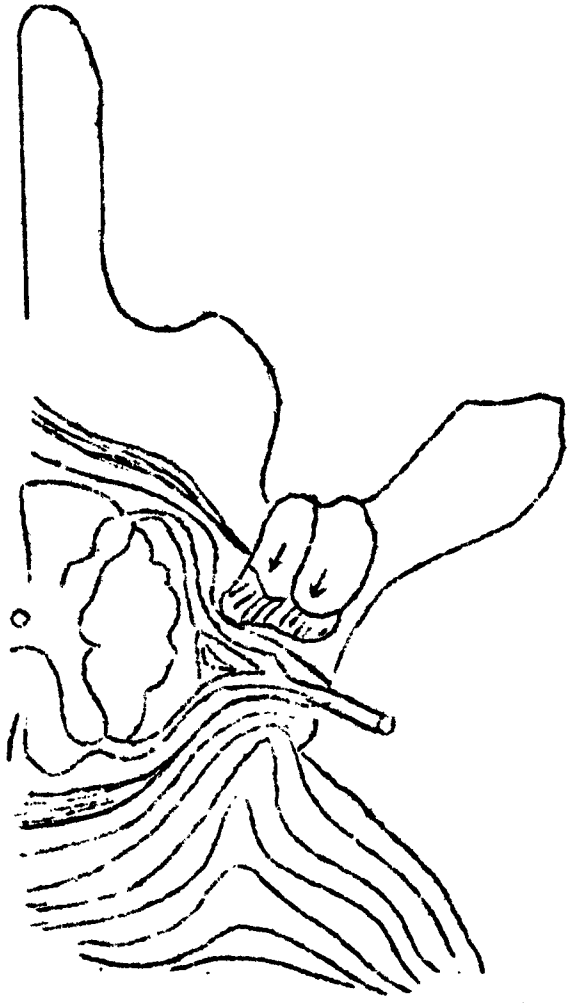


Fig. 9 Entrapment of the nerve root in the subarticular gutter , created by hypertrophy of the superior articular process .

PATHOLOGY OF NERVE CHANGES IN ENTRAPMENT RADICULOPATHY

Macroscopically the compressed nerve may show some alteration in its shape such as flattening, concavity or triangulation incidental to the external pressure. The nerve disc relationship has been discussed before. The root appears congested, oedematous and petechial haemorrhages may appear in its sheath. The epineurium which surrounds the entire nerve is thickened and adhesions firmly attach this to the tunnel walls on the outside. Fibrosis extends inwards between the nerve bundles, leading to thickening of the periureum as well. Microscopically various morphological changes with various stages of nerve degeneration and regeneration were noted. Certain nerve bundles and ganglion cells were found flattened. Many Schwann tubules outlined by the neurilemma were empty of myelin and axons where the macrophages had removed these degenerated elements. In other sections of the same nerve bundle these tubules were seen already filled with multiple rods of Schwann protoplasm and neuclei. This probably represents a later stage in the process of nerve regeneration. Some evidence of oedema of the endoneurium was observed with haemorrhages beneath the perineurium. Sclerosis of arterioles within the nerve bundle was also observed. (Hadley 1964)

HISTOCHEMICAL CHANGES IN THE MULTIFIDOUS IN PATIENTS WITH LUMBAR ROOT ENTRAPMENT

Normally the multifidous muscle contains two main types of muscle fibres, slow twitch ones (with little adinosine triphosphotase, aerobic metabolism and serving the function of sustained tonic activity), and fast twitch ones (rich in adinosine triphosphotase with anaerobic metabolism and serving the function of powerful phasic activities of short duration).

With root involvement, the fast fibres become angular, then atrophied with subsequent reinnervation by collaterals from the slow type motoneurons which are limited from spreading beyond their fascicle of origin by the boundary of the perimysium. These findings suggest that the multifidous adopts an increasing postural role in lumbar root entrapment syndrome. (Jowett et al 1975).

PATHOGENESIS OF PAIN IN ENTRAPMENT RADICULOPATHY

The intervertebral disc itself is a none-pain- sensitive tissue. The total disc is an inert tissue and the nucleus has been found completely free of any sensory type of nerve endings. Nerve endings have been found in the outer-most layer of the annulus fibrosis but neurophysiologic studies have failed to discover pain sensory transmission from these nerves. The discs, therefore, both annulus and nucleus must be considered insensitive to pain sensations.

The sinuvertebral nerve, a recurrent branch of each spinal nerve, is reflected back through the intervertebral tunnel to supply fibres to the articular connective tissue, periosteum, meninges and vascular structures associated with the vertebral cannal. The nerve originates just distal to the dorsal root ganglion where it is frequently united with a branch from the ramus communicans, revealing its dual spinal and autonomic composition. The sinuvertebral nerve passes through the superior part of the intervertebral tunnel, usually between the dorso-lateral surface of the vertebral body and its expected spinal nerve root. Curving upward around the base of the pedicle it divides into a superior and an inferior branch approaching the posterior longitudinal ligaments. There is marked overlap in the levels of the sinuvertebral nerve ramifications so that branches from each level anastamose with those of the adjacent segments; thus it is quite probable that discogenic pain from a single level may involve more than one recurrent branch of the spinal nerves.

The vertebral epidural venous sinuses receive numerous nerve branches from the sinuvertebral nerves (Hence the name given by Luschka). Since these thin-walled venous elements show little or no smooth muscle, the numerous fine, free nerve endings can reasonably be regarded as sensory terminations. When these venous sinuses are compressed or inflammed they might provide an additional source of pain attending disc disorders.

Sensory innervation of the zygoapophyseal articulation is derived from the posterior ramus of the spinal nerves. This branch supplies filaments to the articular capsules of the facets and to the ligamentum flavum and interspinous ligaments. Thus irritation of these synovial joints can produce dull to severe pain, depending on the severity and extent of inflammation.

The ligamentum flavum and interspinous ligaments, although showing nerve endings on their surfaces, yet no such endings can be found within their layers and they are considered as insensitive to pain. On the contrary the posterior longitudinal ligament is richly supplied by nerve endings from the sinuvertebral nerve and it is considered that irritation of that ligament can happen from an increased intradiscal pressure within an affected disc producing pain.

The concomitant muscle spasm that accompanies the spinal disability is in itself capable of eliciting pain.

Mere contact with a normal nerve root is not enough to cause radiation of pain. Pressure on a normal nerve usually does not cause pain. Pressure must be upon a nerve already inflammed and thus hypersensitive. With a nerve already hypersensitive any mechanical irritation such as movement, pressure or traction upon the nerve can initiate a response which can be radiated to the specific nerve dermatome involved.

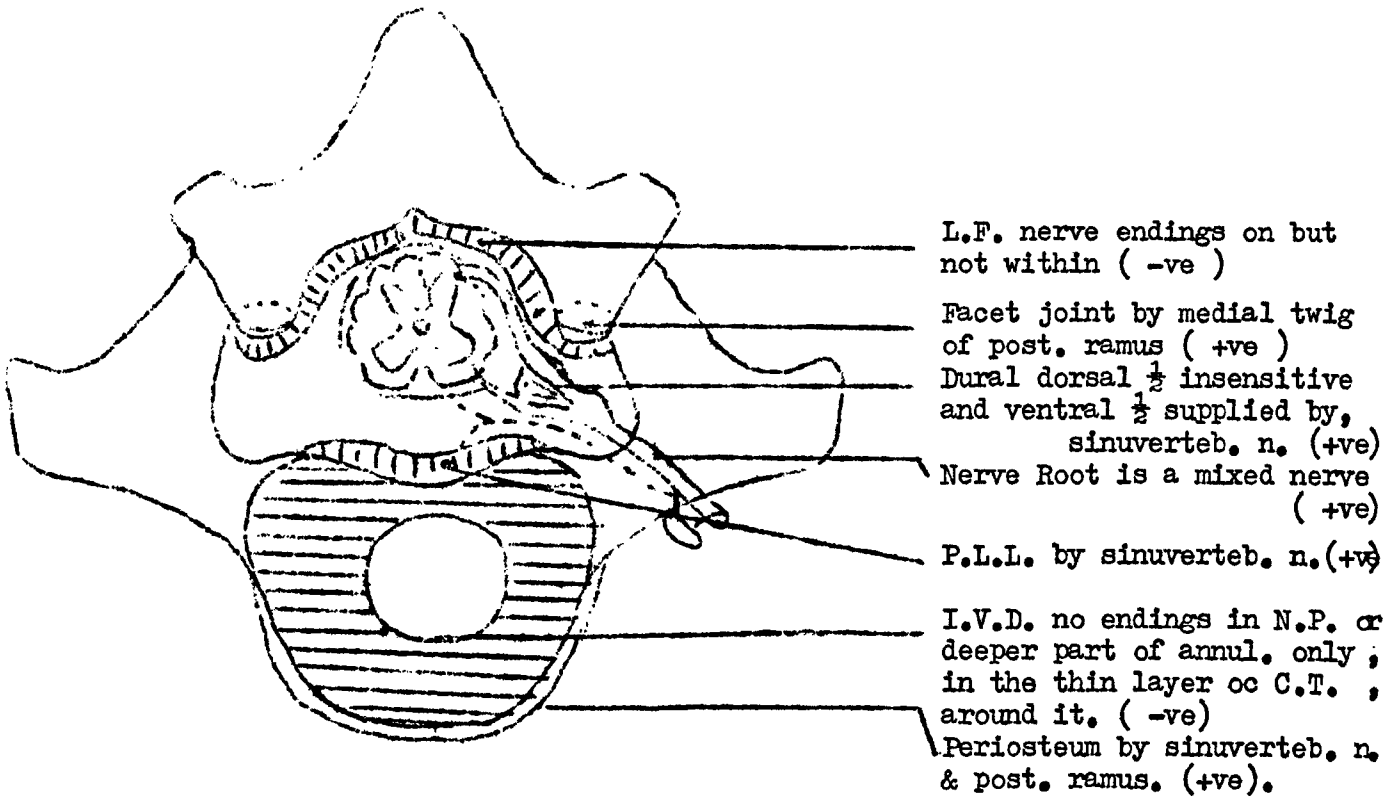


Fig. 10 Anatomy of pain sensitive structures related in the production of lumbo-sciatic pain.

PATIENTS AND METHODS

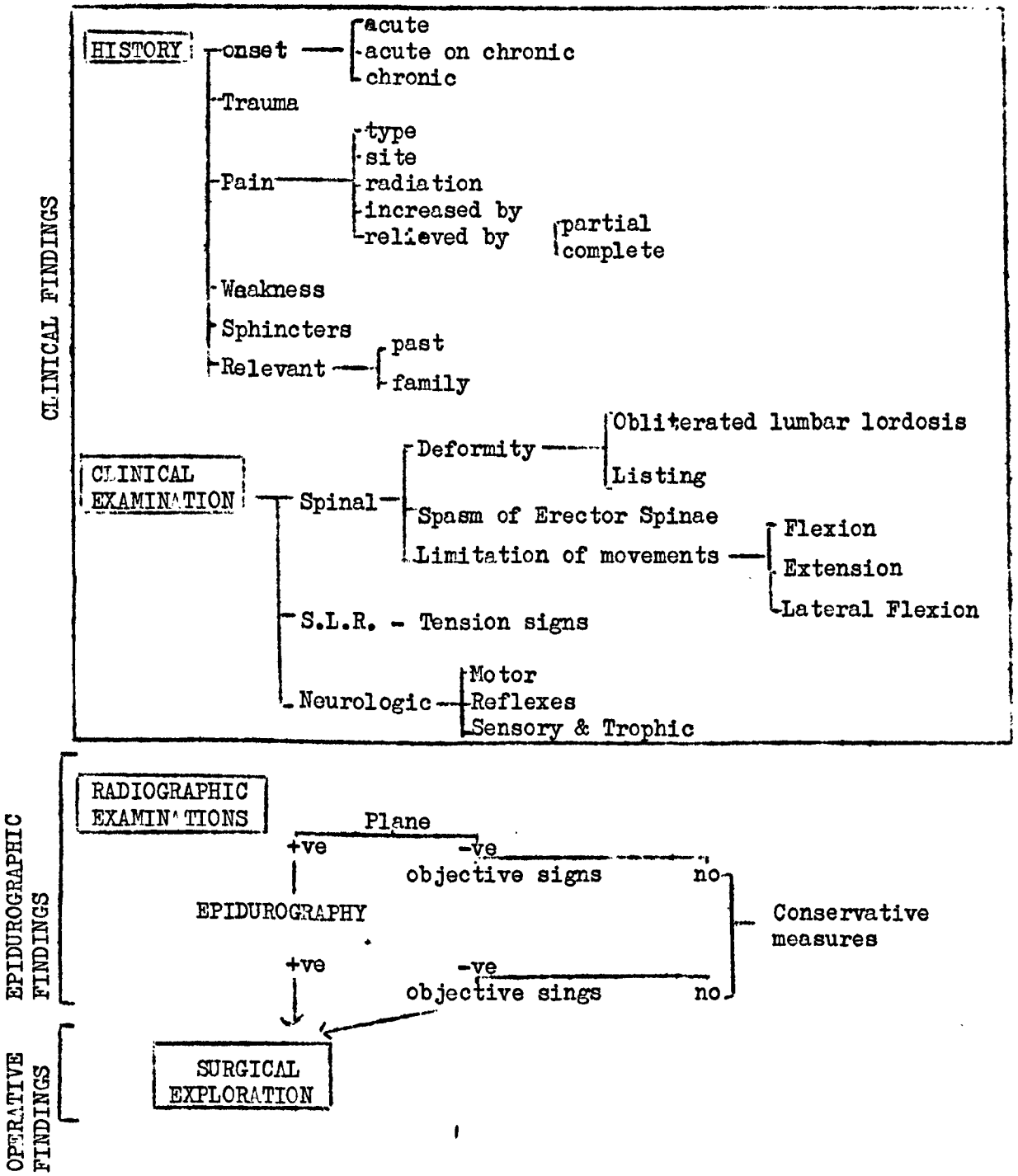
Patients:

This series includes thirty patients who have been chosen according to the following criteria:-

1) They are all male patients whose ages range between 20 and 40 years.
2) Their lumbo-sacral spine was supposedly normal before the onset of symptoms.
3) Their conditions were diagnosed as sciatica with or without low back pain.
4) They were investigated by standardised scheme which includes epidurography.
5) They were operated upon for their condition.

Methods

I Scheme of clinical management



II Epidurography

This means contrast examination of the lumbo-sacral epidural space i.e. the space that lies between the dura matter and the walls of the spinal conal. Before discussing the technique I think it is worthwhile describing the anatomy of the lumbar spinal canal:-

a) Membranous lining of the canal:-

i) posterior longitudinal ligament:-

a number of connective tissue layers arranged both longitudinally and circumferentially and containing within its layers some of the venous channels of extradural plexus of Batson. As mentioned before, it becomes more and more narrower as it approaches the lumbo-sacral region.

ii) periosteal membrane of the neural arches:-

this completes with the above the membranous lining of the canal.

b) Dural membrane and sac:-

The dural sac reaches its terminal extremity at the second sacral segment. The conus medullaris occupies the dural sac from the level of L1 - L2 disc to the level of L2 - L3 disc and ends in the filum terminale. The roots of the cauda equina embrace the conus and proceed to their respective nerve root tunnels and they carry with them the radicular arteries and veins. The roots, as mentioned before, are ensheathed with dural outpouches or sleeves in their tunnels and the junction between the outpouch and the dural sac is usually proximal to the level of the root tunnel.

c) Extradural portion of Batson's plexus of veins:-

This is in the form of two major longitudinal plexiform venous chanels that occupy the space between the membranous lining of the canal and the dural membrane, and embracing the ensheathed nerve roots in their tunnels. The channels communicate with each other across the mid-line, both ventral and dorsal to the dural sac. The ventral ones lye at the mid-level of the vertebral bodies and into them drain the basivertebral veins. The dorsal ones are vulnerable to injury during laminectomy.

As mentioned before, the peridural space thus contains spinal nerves and blood vessels embedded in areolar connective tissue and fat and is connected with the paravertebral and presacral connective tissue via the intervertebral tunnels. In the tunnel connective tissue attaches the spinal nerves and blood vessels to the tunnel walls, the so called Charpy's ligaments. Furthermore the epidural space is connected with the subcutaneous tissue via the sacral hiatus.

Technique:

1. A sedative (Pethidine 50 mg), half an hour before examination.
2. Patient prone with a pillow under his lower abdomen.

3. Prepare the skin:
   - Shave any hairs present.
   - Cleanse with cetavlon, then paint with Tr. Iodine and remove the excess using alcohol. (Sensitivity to Iodine should be excluded)
   - Mark the site of entry by feeling the two sacral cornua and between them lies the dip of the sacral hiatus.
4. Local anaesthetic infiltration with 1% zylocaine.
5. A spinal needle is introduced into the spinal canal through the sacral hiatus in a cranio-anterior direction so that the tip of the needle should not be higher than S2 to avoid dural sac penetration. The patient is asked to cough and gentle suction with a syringe applied to be sure that the needle does not puncture one of the external veins (blood will come in the syringe), or more important, the dural sac (cerebrospinal fluid will come in the syringe) A lateral radiogram is then taken to show the position of the needle exactly. It should be noted that the dye is not injected in the subarachnoid space as this will result in painful persistant cramps from nerve root irritation. It has been shown that injections of Urographin in the subarachnoid space of cats produced fatal paroxysms depending on the dose, and this can be completely prevented by intraperitoneal injection of Nembutal after a lag period of 3 to 5 minutes, depending on the dose (Luyendijk et al 1966)

Fig. 11 & 12

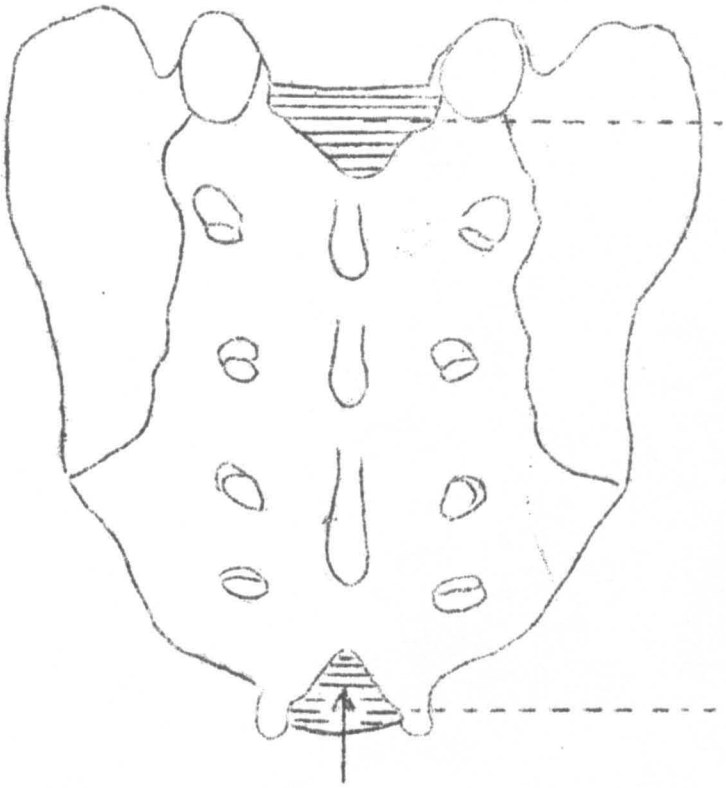
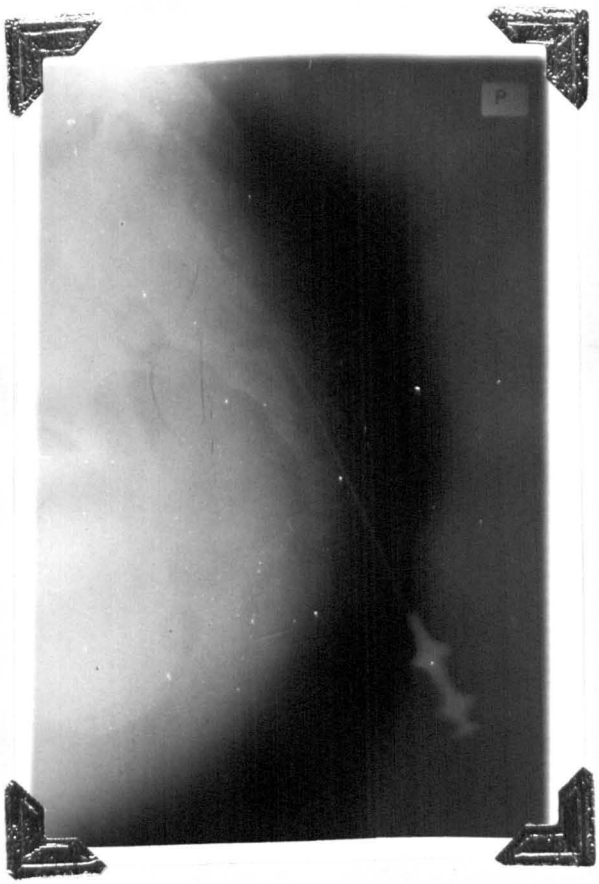


Fig. 11 Site of entery of the needle.



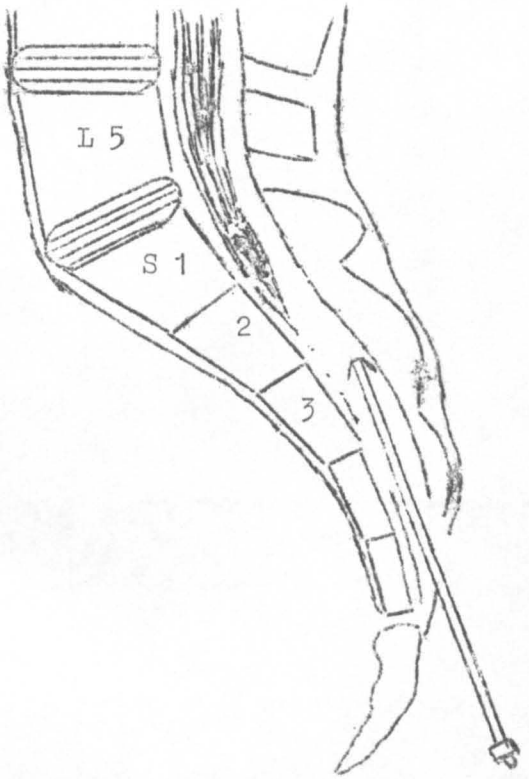


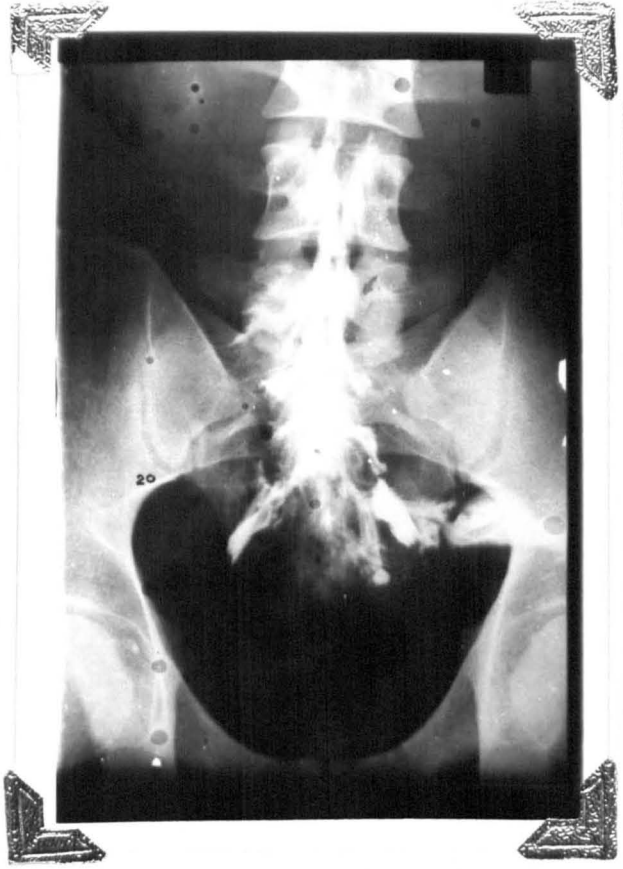
Fig. 12 Needle in situ ready for injecting the contrast medium.

6. Inject 20 c.c.'s of 76% Aqueous solution of sodium Diatrizoate (Urographin) diluted with 5 c.c.'s of 1% novocane, slowly over 5 minutes, and then take the radiographic exposures within the next 15 minutes as the dye is rapidly absorbed and excreted by the kidneys (Pyelogram). Intravenous hydrocortisone (to deal with any allergic manifestations to the dye) and intravenous pentothal (to combat nerve root irritation if the dye is accidentally injected into the subarachnoid space) should be available. Luckily enough no single case of 110 patients who have been exposed to this investigation suffered any of these untoward effects and no other complications or side effects were encountered in them or reported in literature.

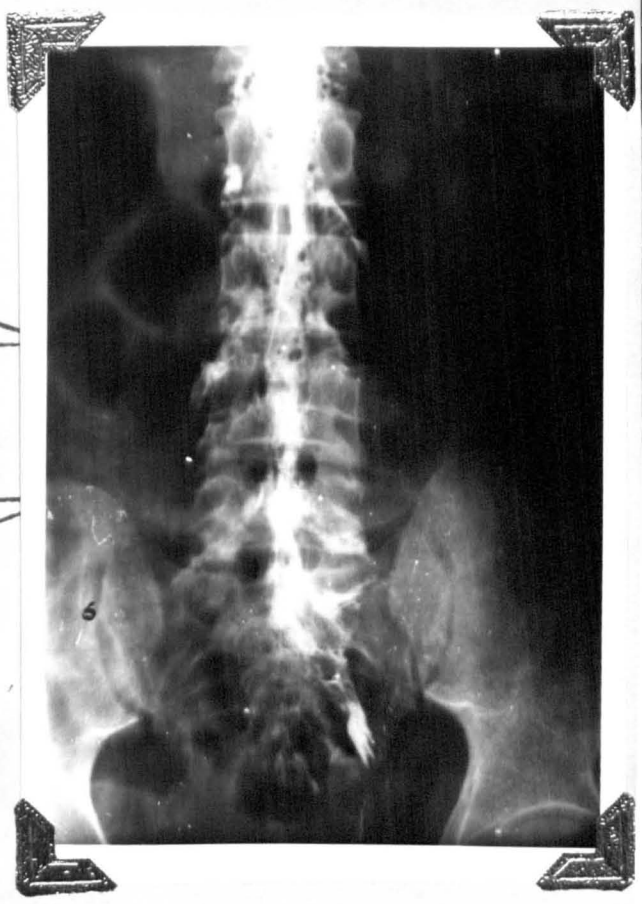
Interpretation of epidurographic findings

The shape of the peridural space is determined by the manner in which the dural sac is attached to the walls of the spinal canal. The attachment is effected on the ventro-lateral aspect by the spinal nerves and in the median plane dorsally, where it is attached to the vertebral arches. In the lateral peridurogram the dye is spread in a thin layer between the dural sac and the walls of the spinal canal. Anteriorly this distribution is not uniform:- dorsal to an intervertebral dis, it is small, but behind a vertebral body it is usually larger, increasing at every segment toward the centre of the latter. The width and intensity of the dorsal contrast band indicates that the peridural space is larger in size in the dorsal than in the ventral region. The abnormalities which can be seen in the lateral peridurogram may be either an anterior filling defect or a posterior vertebral canal narrowing.

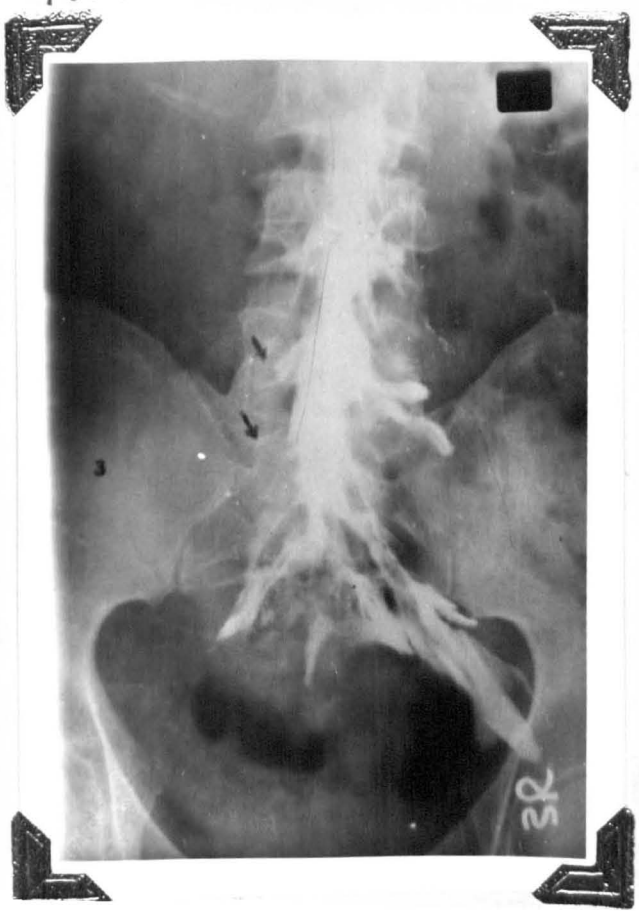
In the antero-posterior peridurogram there is a central contrast column denser in the lateral than in the central zone, and the dye can be seen trickling along the spinal nerves through the tunnels. The abnormalities which can be seen may be a centrally placed filling defect, a peripheral filling defect or an interruption or even amputation of the nerve root outline. (Fig13)



A-P : Amputation of Rt L5 root.

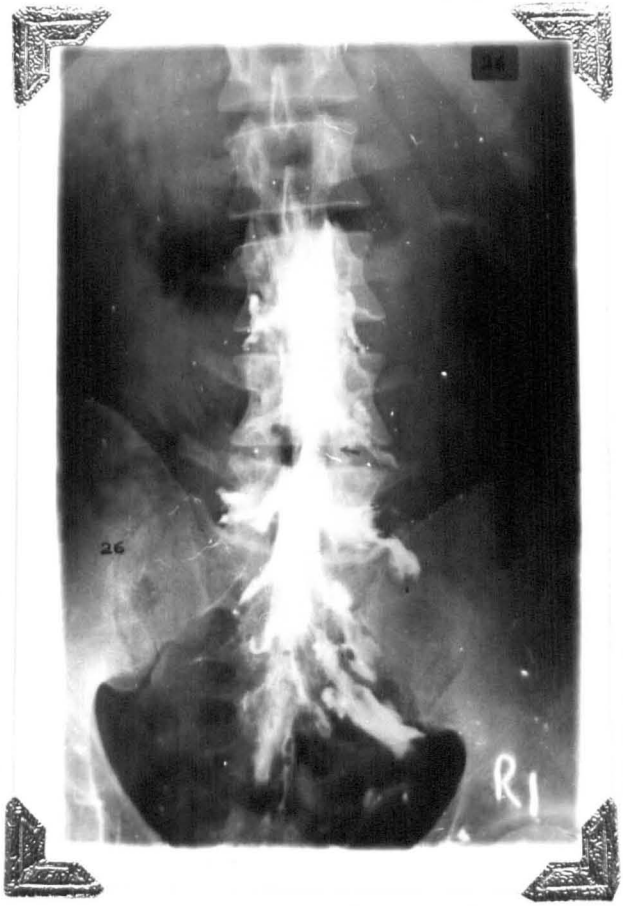


A-P : Amputation of Lt S1 root.

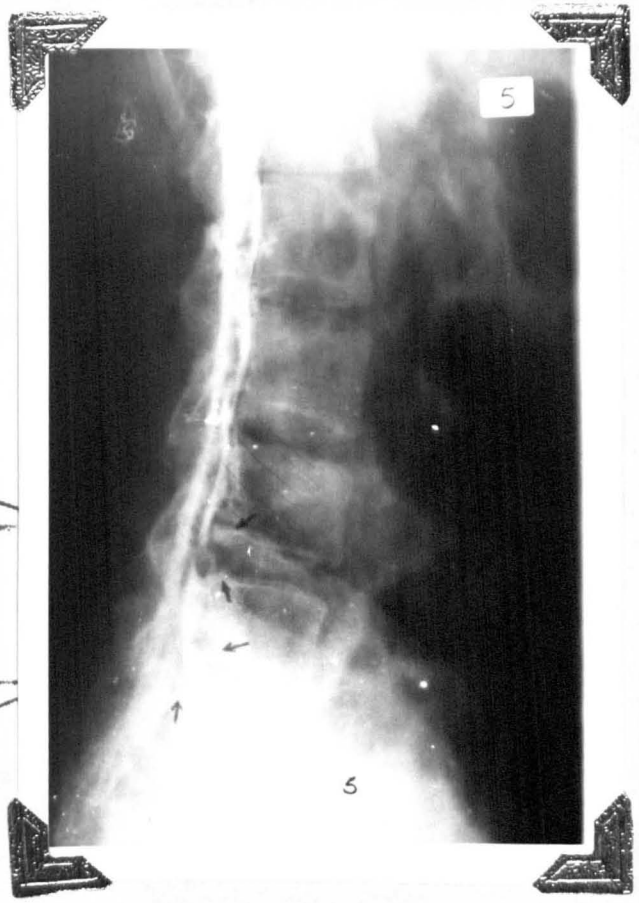


A-P : Amputation of Lt L5 & S1 roots
+ a peripheral filling defect

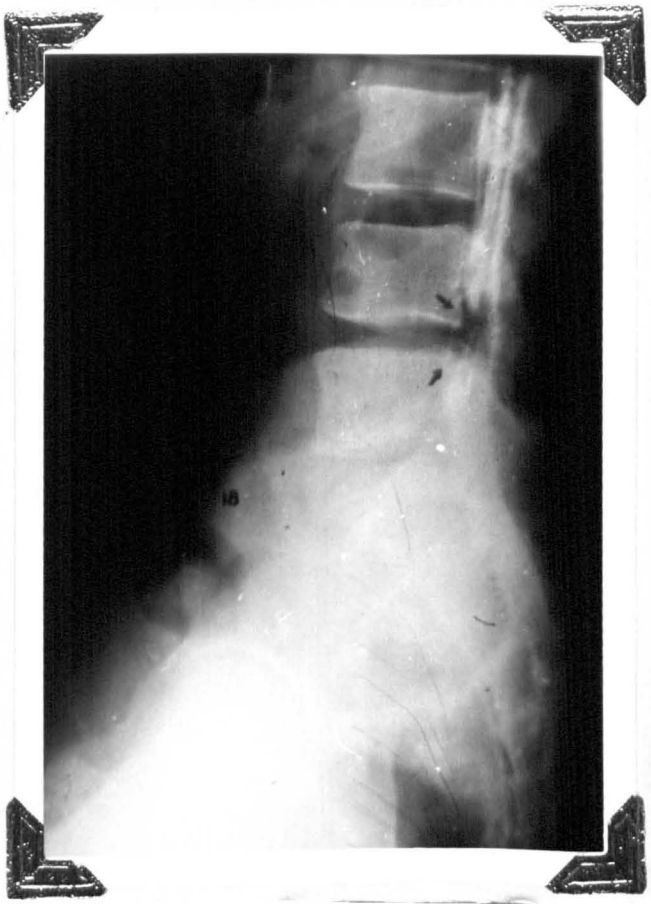
Fig. 13 Examples of Epidurographic Findings.



A-P : Peripheral filling defect



Lat. : Anterior invagination.



Lat. : Anterior filling defect +
Post. invagination.

III Surgical Technique

- General anaesthesia (Hypotensive technique)
- Lateral position (The lumbar spine is kept flexed by putting the patient in the knee - chest position).
- Curvy-linear exposure slightly convex towards the affected side.
- Deep dissection to expose the spinus process, laminae and ligamentum flavum.
- Hemilaminectomy (The interspinus ligament is divided, the spinous process of L5 removed, the uppermost half of L5 lamina excised and the ligamentum flavum is removed).
- Dealing with the pathology and the most important step is to be sure that decompression of the affected nerve root is complete, so after removal of the disc prolapse, if found, the suspected nerve root is displaced medially. Normally it should be freely mobile. If it is found to be taught and cannot be displaced medially then it is teathered somewhere between the point of exposure and its emergence from the spinal canal, in the hidden zone, and it should be freed (Macnab 1971) Fig.14.
- Haemostasis and closure in layers with drainage.
- Post operatively the patient lies on his back, flat, for the first few days until the acute pain of the operation settles down and then he is allowed out of bed and early physiotherapy is started.

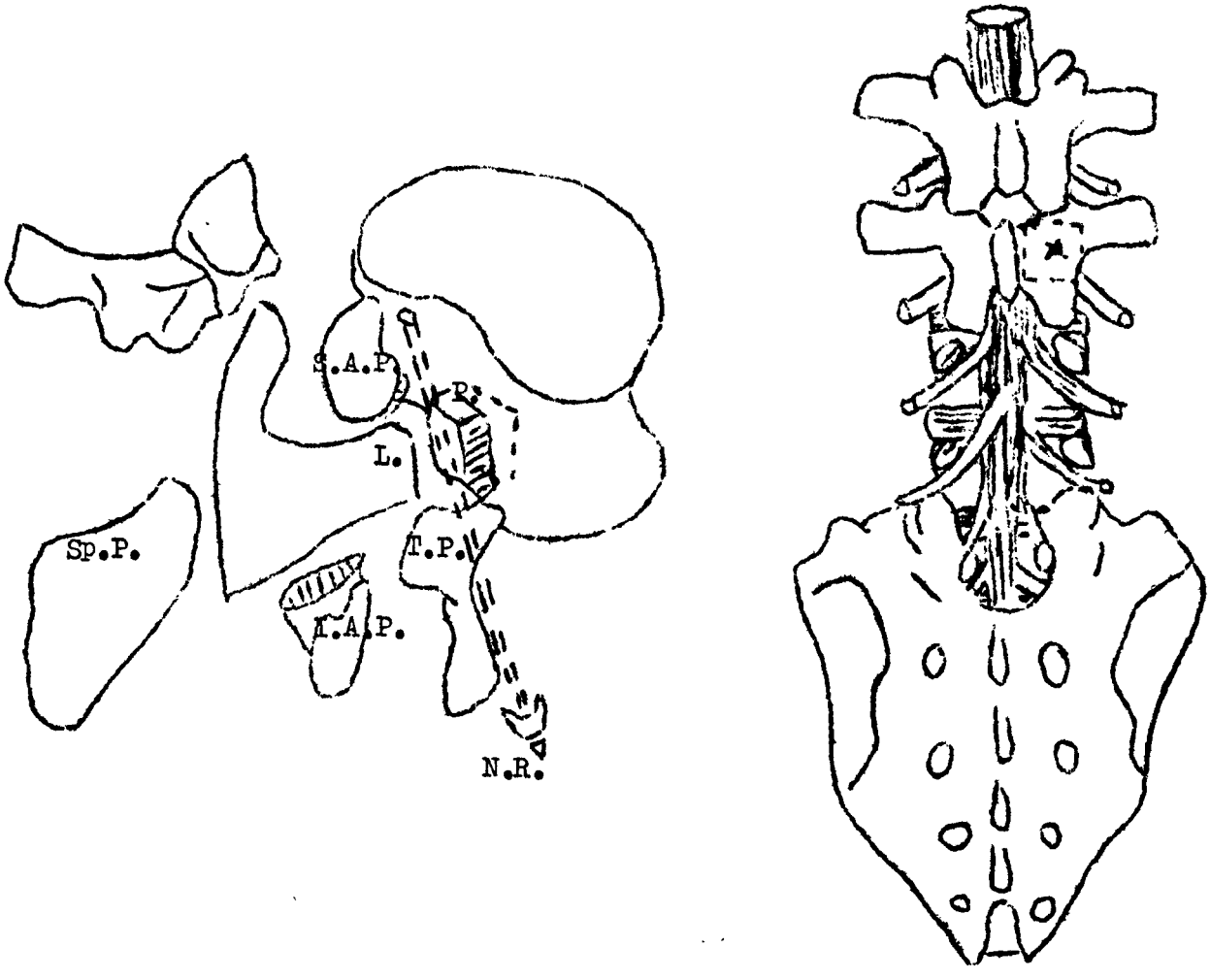


Fig. 14 The hidden zone of Macnab.

ONSET : A Acute of sciatica.
B Acute of L.B.P. & sciatica.
C Gradual of sciatica.
D Gradual of L.B.P. & sciatica.
E Gradual of L.B.P. followed in weeks or months by sciatica.
F Gradual of reversed sciatica.

Neurological Signs :

E.H.L. Extensor Hallucis Longus.
E.D.L. Extensor Digitorum Longus.
T.A. Tibialis Anterior.
A.J. Ankle Jerk.
Kn. J. Knee Jerk.
Post. Tib. Ref. Posterior Tibial Reflex.
? Doubtful.

Radiation of Pain :

U uncertain

Epidurographic Findings :

C.F.D. Central Filling Defect.
P.F.D. Peripheral Filling Defect.
Amput.N.R. Amputation of nerve roots.
Ant.F.D. Anterior filling defect.
Post. F.D. Posterior vertebral narrowing.

Operative Findings :

P.I.V.D. Prolapsed intervertebral disc.
Lig. Flav. Ligamentum flavum thickening.
P.L.L. Posterior longetudinal ligament thickening.
Others : * Bands.
@ Varices.

No.	Age (Y)	Onset	Trauma	Duration (months)	Rad. of pain Hallux	Heel	Sp. Deformity Lord	List	S.L.R. R	L	Neurological Signs Motor Weakness of	Reflexes ↓ or absent	Sensory Signs	A-P C.F.D.	EPIDUROGRAPHIC FINDINGS P.F.D.	Amput.NR.	Ant.F.D.	LAT Post.F.D.	OPERATIVE FINDINGS Root	P.I.V.D.	Adhesions	Lig.Flav	P.L.L.	Others
1	27	C	+	7	+	-	+	L	70	30	E.H.L.	-	L 5	-	-	-	+	-	LL 5	L 4-5	+	+	-	-
2	22	D	+	9	-	-	+	L	80	40	E.H.L., E.D.L., T.A.	Post. Tib.	L 5	-	-,	+	+	-	LL 5	L 4-5	+	+	+	-
3	34	C	+	12	+	-	+	-	90	60	-	-	? L 5	-	+	+	-	+	LL 5	L 4-5	+	+	-	-
4	20	C	-	11	-	+	+	L	50	70	E.H.L.	A.J.	S 1	-	-	+	+	-	R L5&S1	L 5- S1	+	-	-	+ @
5	23	F	+	8	-	U	+	-	80	80	Wasted calf	A.J.	? S 1	-	-	+	+	-	RS 1	L5 -S1	+	+	-	-
6	25	E	-	15	U	-	+	R	50	60	E.H.L.,	-	L 5	-	-	+	+	-	RL 5	L5 -S1	-	+	-	-
7	32	C	+	20	+	-	-	R	90	30	-	-	? L 5	-	-	-	+	+	LL 5	L 4-5	+	+	-	-
8	30	B	+	17	-	+	+	-	80	40	Wasted calf	A.J.	S 1	-	-	+	+	+	LS 1	L5-S1	+	-	-	+ *
9	25	F	-	16	U	-	+	L	90	60	E.H.L., T.A.	-	L 5	-	-	-	-	-	LL 5	L 4-5	-	-	-	-
10	38	B	-	7	-	U	+	L	90	70	Triceps Suris.	A.J.	? S 1	-	-	-	+	-	LS 1	L5-S1	-	-	-	-
11	25	D	+	18	-	-	+	R	60	90	Quadriceps	Knee J.	L 4	-	+	+	+	-	RL 4	L3-4	-	+	-	-
12	23	B	+	12	+	-	+	R	50	80	E.H.L., E.D.L.	-	L 5	-	-	+	+	-	RL 5	L4-5	+	+	-	-
13	29	E	+	20	-	U	-	R	30	80	Wasted calf	A.J.	S 1	+	-	+	+	-	RS 1	L5-S1	-	+	-	-
14	24	B	+	10	-	-	+	L	60	60	E.H.L., T.A.	Post. tib.	? L 5	-	+	+	+	-	LL 5	L 4-5	+	-	-	-
15	35	C	+	23	+	-	+	-	30	90	-	A.J.	L 5	-	-	+	+	+	RL 5	L 4-5	-	+	+	-
16	29	A	+	14	-	+	+	L	70	30	Wasted calf	A.J.	S 1	-	-	+	+	-	LS 1	L5-S1	+	+	-	+ *
17	27	E	+	18	+	+	+	R	40	20	E.H.L.	A.J.	? S 1	-	-	+	+	+	RL5&S	L5-S1	+	+	-	-
18	21	A	+	16	U	-	+	L	90	80	E.H.L.	-	? L 5	-	-	+	-	+	LL 5	L 4-5	+	+	-	-
19	32	B	+	4	+	-	+	L	70	30	E.H.L., E.D.L.	-	L 5	-	-	+	+	-	LL 5	L 5-S1	+	+	-	-
20	30	E	+	22	U	-	+	L	85	80	-	-	L 5	-	-	+	+	-	RL 5	L 4-5	-	+	-	-
21	25	C	+	10	-	+	-	L	60	40	Triceps Suris.	A.J.	S 1	-	+	+	+	-	LS 1	L5-S1	+	+	-	-
22	28	C	-	23	U	-	+	R	0	60	E.H.L.	-	? L 5	-	-	+	-	+	RL 5	L 4-5	+	+	-	-
23	32	A	+	12	-	U	+	R	20	20	Wasted calf	A.J.	S 1	-	-	-	+	-	RS 1	L5-S1	-	+	-	-
24	21	E	+	14	-	-	+	-	60	80	-	-	L 5	-	-	+	+	-	RL 5	L5- S1	+	+	-	+ @
25	24	A	+	8	+	-	+	R	80	30	-	-	L 5	-	-	+	+	+	LL 5	L4-5	+	+	+	+ *
26	31	A	+	7	-	-	+	R	40	80	E.H.L., E.D.L.	-	L 5	-	+	+	+	-	RL 5	L4-5	+	+	+	+ *
27	29	D	-	11	-	-	+	L	20	80	E.H.L.	A.J.	? L5	-	-	+	+	+	R L5	L5-S1	+	+	-	-
28	22	C	+	9	-	+	+	-	20	50	Wasted calf.	A.J.	? S 1	-	+	+	+	+	RS 1	L5-S1	+	+	-	+ *
29	27	C	-	12	-	-	+	L	70	80	-	A.J.	? L 5	-	-	+	+	-	-	-	-	-	-	-
30	32	E	+	11	-	-	+	R	60	90	E.H.L.	-	L 5	-	-	+	+	+	RL 5	L4-5	+	+	-	-

ANALYSIS OF CASES AND DISCUSSION.

The material consisted of the findings in 30 male patients within the age group 20 - 40 years old, operated upon for sciatica during the years 1971-72. This particular age group was chosen to avoid the superadded effects of the ageing process and onset of spondylotic changes on the primary and secondary disc pathology causing radiculopathy. The age-level relationship is shown in Fig.15. There appears to be no significant relationship between the age of the patient and the level or nature of the lesion in this particular age group when correlated with the operative findings.

It appears that trauma is an important underlying factor in the production of symptoms in this age group as evidence of a positive relevant history of trauma wasfound in 23 patients i.e. 76.6%. The type of trauma varied from lifting heavy objects, to falling awkwardly, to direct injury and whatever type the effects were the same.

The patterns of onset and the nature of the initial attack of sciatic pain varied. In nine patients i.e. 30% it was an insidious onset with gradually increasing discomfort, starting as a dull, unpleasant ache in the buttock and posterior aspect of thigh, later increasing in intensity and spreading below the knee to the calf and foot. In six patients out of these nine there was a positive history of trauma.

In another two patients i.e. 6.6%, this picture was reversed with the same mode of onset but starting in the heel or ankle and spreading up the leg and the positive history of trauma was found in one patient.

In a further six patients i.e. 20%, the pain was very severe from the start, occurring suddenly and was constant all the time and could not be relieved by rest or the assumption of any particular posture. The positive history of trauma was found in five patients of this group.

In three patients, i.e. 10%, the onset was insidious with low back pain and sciatica occurring at the same time, and in two out of these three patients it started after trauma.

In a further five patients, i.e. 16.6%, low back pain and sciatica started simultaneously acutely and in all the five patients it was definitely related to trauma.

In the last five patients, i.e. 16.6% the condition started as low back pain which lasted fof weeks or months and then the sciatica began to make its appearance insidiously and in four patients the syndrome was attributed to trauma.

The pattern of onset - level relationship is shown in Fig. 16.

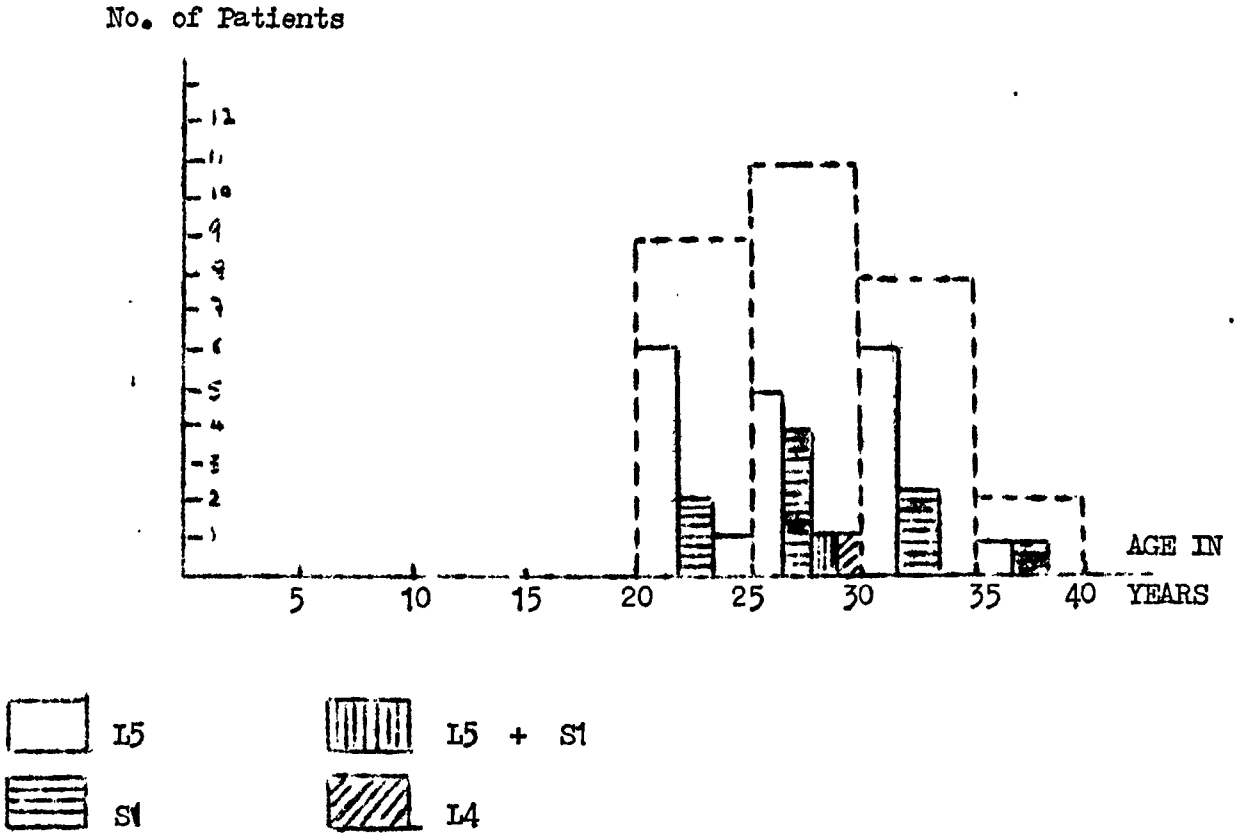


Fig. 15 AGE - LEVEL Relationship.

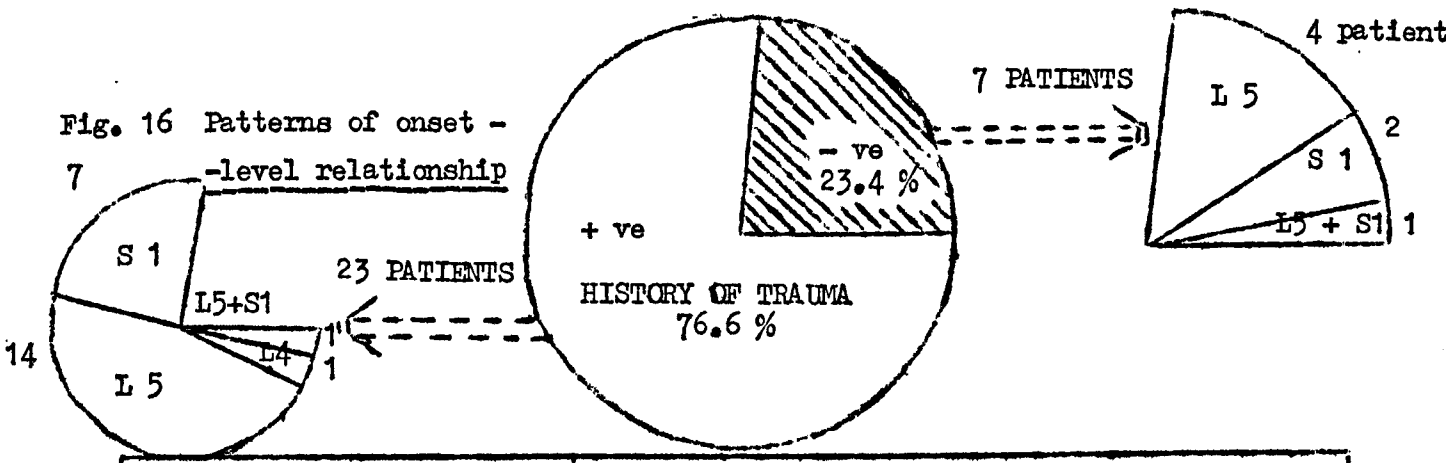


Fig. 16 Patterns of onset - -level relationship

A ACUTE				GRADUAL							
Sciatica		Simult. of B.P. & Sciat.		Sciatica Up - down		Sciatica Reversed		Simult. of B.P. & Sciat.		B.P. followed by sciatica	
6		5		9		2		3		5	
+	-	+	-	+	-	+	-	+	-	+	-
5	1	5	0	6	3	1	1	2	1	4	1
+ ve History of TRAUMA											
LEVEL											
L5	S1	L5	S1	L5	S1	L4	S1	L5	S1	L5	S1
4	2	3	2	6	2	1	1	2	1	4	1

The duration of symptoms before operative intervention ranged between six months and two years and as appears from Fig.17, the relation between the level of the lesion and the duration is insignificant. The picture, as a whole, appeared to be rather hot and acute in this age group with considerable disability almost all the time and even if there was some improvement at some stage, acute exacerbations happened, the frequency, duration and intensity of which appeared to have no relation to the level or age but was found to be definitely related to the local tissue reaction in response to the disc pathology. (Case Nos. 6,8,10,12,13,14,17,19,20,24 and 30).

The extent to which pain radiated along the lower limb was found to be of little significance in localising the level unless it radiated either in the heel (S1 affection) or in the big toe (L5 affection), as it seemed that most of these patients were either uncertain or unaware of the extent to which pain had radiated along the leg. This appears in Fig. 18.

Sometimes (in cases 4 and 17) an L5/S1 disc lesion caused entrapment of L5 and S1 roots with motor paralysis of L5 and sensory affection (objective and subjective) of S1 distribution, adding to the perplexity of the clinical picture.

When acute sciatica is present the patient usually lists away from the side of his sciatica, producing a "sciatic scoliosis". Fig. 19. When the disc herniation is lateral to the nerve root the patient will incline away from the side of the irritated nerve root in an attempt to draw the nerve root away from the disc fragment. When the herniation is in an axillary position (medial ot the nerve root), the patient will list toward the side of the lesion in an effort to decompress the nerve root. However, this segmental spasm of the errector spinae muscle, which is supposed to be due to nerve root recurrent nerve irritation, is not always consistent with the above description, as appears in Fig. 20. which correlates between the sciatic list and operative findgins.

The gait and stance of patients with an acute entrapment was also characteristic. The patients usually held the painful leg in a flexed position and were reluctant to place their feet completely flat on the floor. Presumably flexion of the leg relaxed the nerve roots in an effort to decompress them. When they walked they had an antalgic gait, putting as little weight on the extremity as possible and quickly transferring their weight to the unaffected side.

Loss of lumbar lordosis and paravertevral muscle spasm were also seen during the acute phase of the disease. Limitation of motion was noted during the symptomatic phase of the disease. Movements in all directions (namely flexion, extension and lateral flexion) were found to be limited to a varying extent. I found that forward flexion was of

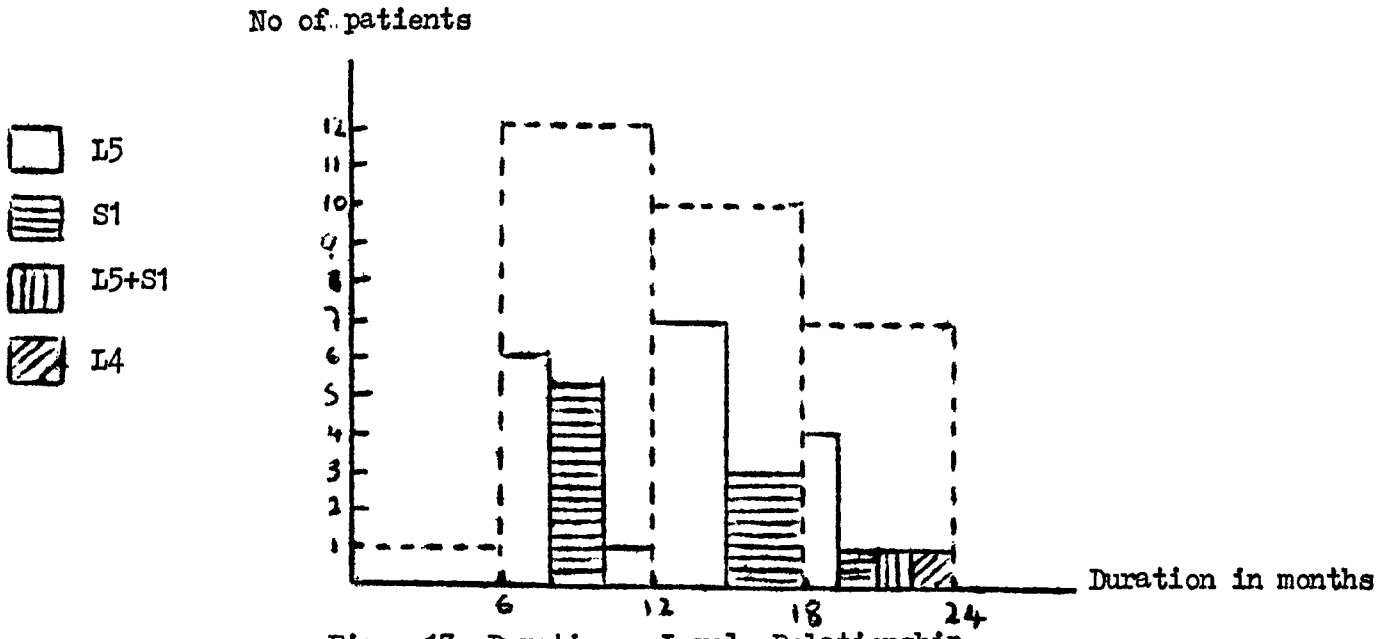


Fig. 17 Duration - Level Relationship.

Root Affected	Total No.	RADIATION OF PAIN TO			
		Leg	Hallux	Heel	Uncertain
L 5	18	13	7	-	5
S 1	9	7	-	4	4
L5 + S1	2	2	1	2	-

Fig. 18 Radiation of PAIN

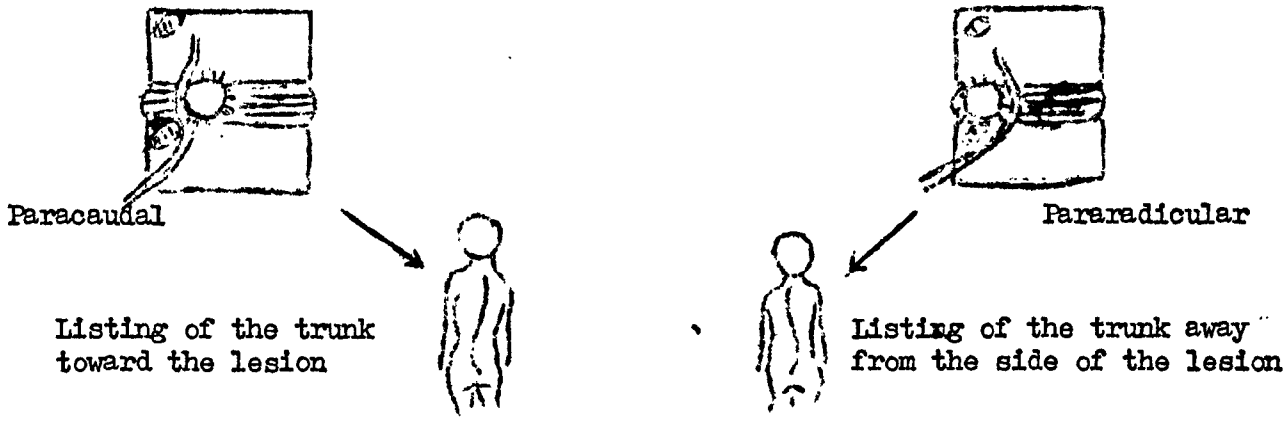


Fig. 19 Sciatic List

Consistent with the above		Not consisting with the above	Other operative findings
Pararadicular	Paracaudal		
6	9	6	9

Fig. 20 Relation between sciatic listing and operative findings.

particular diagnostic importance. Flexion is initiated by contraction of the psoas and abdominal muscles; then gravity takes over, aided by gradual relaxation of the sacrospinalis muscles. This was confirmed by Flint (1965) using electromyographic techniques.

Limited trunk flexion because of sacrospinalis muscle spasm acting as a protective mechanism can be understood when it is realised that trunk flexion normally causes traction on the dural sac. Flexion of the trunk causes an upward movement of the spinal nerves. This movement is most marked at the L1/L2 level and this movement of the nerves decreases caudally, becoming insignificant at the L5 level. Fig. 21.

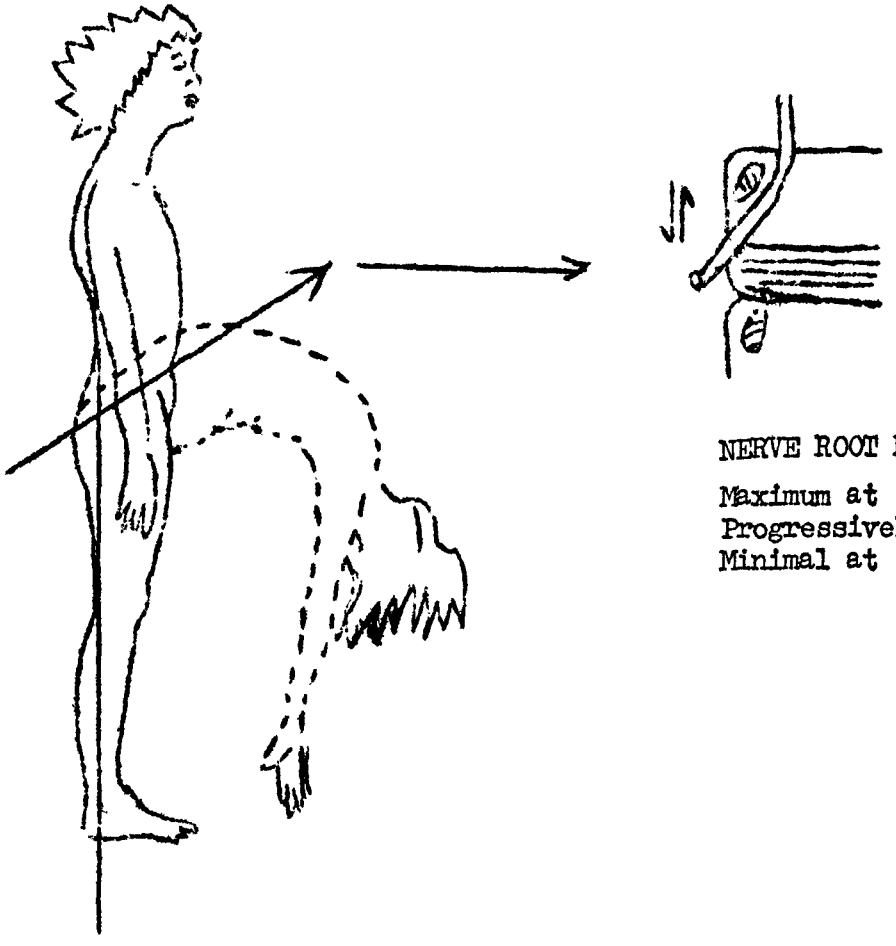
There are several methods which tighten the sciatic nerve and in doing so, further compress an inflammed nerve root against a herniated lumbar disc or any other space occupying lesion in its closed compartment. The most significant tension sign was found to be the simple straight leg raising test. The degree of nerve root movement that occurs at the intervertebral tunnel in the straight leg raising test is exactly the reverse to what happens in trunk flexion, thus maximum movement occurs at the level of L5/S1 and no movement at the level of L2/L3. This was why it appeared that the test was strongly positive in cases of S1 affection and less positive in cases of L5 affection. This is especially the case when the contralateral straight leg raising is positive.

The neurological examination had shown the correct level of the lesion in 22 cases out of 30.(73.3%)

Compression of the motor fibres of the nerve root may result in weakness or paralysis of the affected muscle groups associated with loss of tone and bulk of the muscle belly. In the case of the first sacral nerve nerve the triceps suris muscle is the one affected mostly but because it is a very strong muscle this could not be detected clinically in terms of weakness of plantar flexion of the foot, which was present only in 2 cases out of 8. Wasting of the calf was more easily detected in the remaining six cases by measuring the bulk of the muscle and comparing it with the opposite side. The hamstring group of muscles was also affected in 2 patients out of 8 with S1 affection, and this was easily detected clinically.

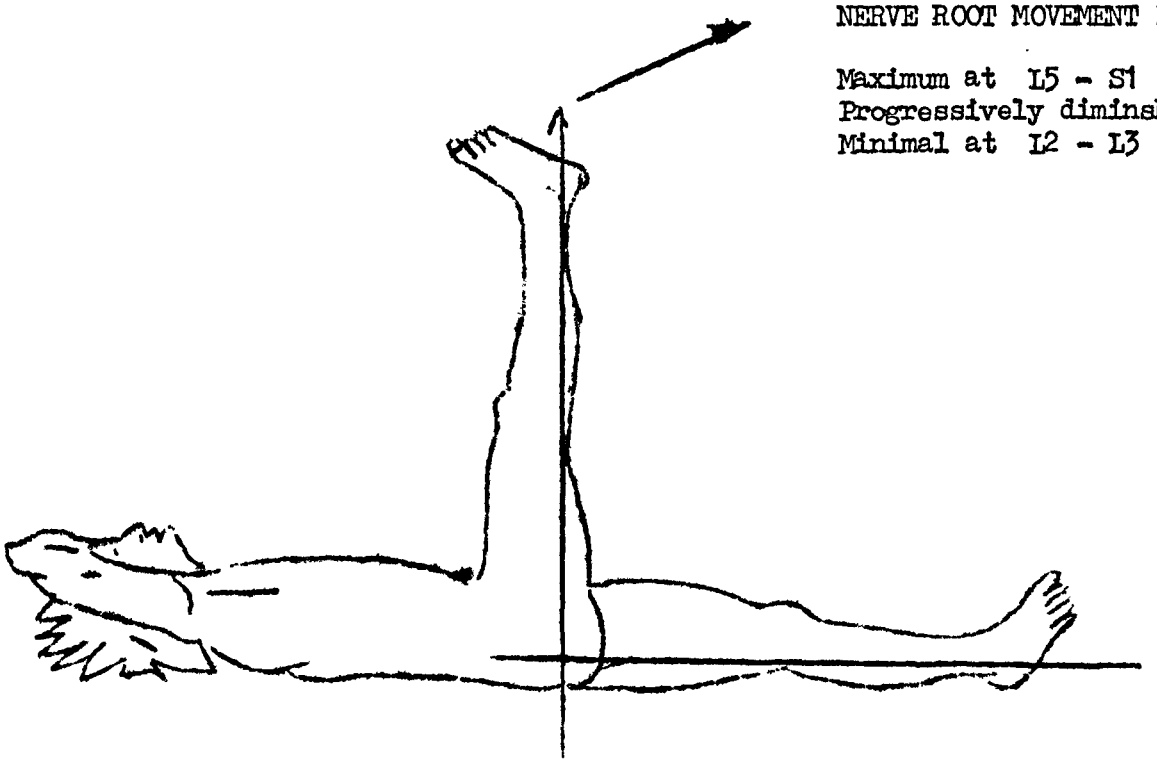
In cases of L5 affection, motor changes included weakness of dorsiflexion of the hallux in 13 cases out of 20 and weakness of dorsiflexion of all the toes and the foot in 4 cases. The tibialis anterior was clinically affected in 3 cases.

Compression of L4 resulted in wasting and weakness of the quadriceps muscle.



NERVE ROOT MOVEMENT IN FOREWARD FLEX.

Maximum at L2 - L3
Progressively diminishing to become
Minimal at L5 - S1



NERVE ROOT MOVEMENT IN S. L. R.

Maximum at L5 - S1
Progressively diminishing to become
Minimal at L2 - L3

Fig. 21 Nerve root movement with trunk flexion and S.L.R.

The pattern of sensory involvement when nerve root compression was present, usually followed the dermatome of the affected nerve root. In general there is considerable overlap between adjacent dermatomes, except when these are separated by the axial lines, and this is particularly the case with L4,L5, and S1. Fig. 22
In cases of L5 affection this was along the antero-medial aspect of the leg and foot including the medial four toes. In cases of S1 affection this was along the postero-lateral aspect of the leg, heel and sole of the foot. In cases of L4 affection this was along the postero-lateral aspect of thigh, across the patella and along the upper half of the anteromedial aspect of the leg.

The deep tendon relfexes were altered in nerve root compression. The Achilles reflex was diminished or absent in 7 cases out of 8 with compression of the S1 nerve root.

Compression of the fifth lumbar nerve root most commonly caused no reflex change (18 out of 22). Occasionally an absent posterior tibial reflex was detected but this should be asymmetrical to be of any clinical importance. (2 cases out of 22). An absent Tento-Achilles reflex was discovered in 2 cases proved at operation to be due to affection of the 5th lumbar root solely.

Compression of L4 (1 case) resulted in diminished patellar reflex.

The corresponding figure for epidurographic examination was 28 cases out of 30. (93.3%) One case (Case No.9) showed negative epidurographic findings and on exploration the lesion was discovered to be adhesive radiculitis, and in addition a band was found stretching across the dural sac and obliterating the inlet of the tunnel on the left side. Excision of that band and removal of adhesions relieved the pressure effects on the nerve. The second case showed positive epidurographic findings localising the lesion to be at the level of L5 but at operation no abnormality could be detected, neither at that level or above or below. The epidurographic findings are collectively shown in Fig. 22.

The collective operative data is tabulated in Fig.23. It appears that in this younger age group the actual pathology of lumbar nerve root entrapment is mainly due to soft tissue pathology, either in the form of disc lesions, ligamentus thickening, zygoapophyseal capsular and synovial thickening, or adhesions, which proved to form quite early in the course of the disease. The disc lesions were in the form of either herniation (6 cases) or prolapse of nuclear material, either wholly (20 cases) or in fragments (3 cases) into the spinal canal. Whatever the nature of the disc lesion discovered at operation, the outcome was the same. i.e. Evoking the secondary inflammatory reaction in the relevant structures with the ultimate outcome of compression of the affected roots in their closed compartments. None of the 30 patients showed an intact degenerated disc.

A-P	Central filling defect	Peripheral filling defect	Amputation of N.R.
	1	6	25

LAT	Anterior invagination	Posterior invagination
	26	11

Total No. of Patients 30	+ve 28	-ve 2

Fig. 22 Collective epidurographic findings

	P.I.V.D.				Adhesions	Lig.Flav.Thick.	P.L.L.Thick.	Bands	Varices
	L3-4	L4-5	L5-S1	-ve					
N.R.affected	1	14	14	1	21	24	4	5	2
	L4	L5	L5 4 S1 8 L5&S1 2						

Fig. 23 Collective operative data.

From the above tables it appears that the commonest epidurographic sign is an anterior filling defect on the lateral epidurogram which is present in 26 cases out of 29 proved at operation to have a disc prolapse at the site shown on the film.

The second important epidurographic sign is interruption of nerve root outlines which is present in 25 cases out of 29 proved at operation to have entraped nerve roots consistent with the findings on the film.

Posterior filling defects are present in 11 cases out of 24 proved at operation to have a thickened ligamentum flavum.

Adhesions, bands and varices seem to present no spicific epidurographic findings apart from the above mentioned signs.

The findings in the 2 -ve cases are discussed before.

Despite the many factors that may play a role in the ultimate outcome of an operation for a lumbo-sciatic syndrome - such as the patients' psychological status and his motivation to return back to his work - the importance of achieving proper effective nerve root decompression should be overemphasized. This cannot be done through a simple fenestrectomy operation but it needs proper exposure through a standard laminectomy operation as previously described in the surgical techniques and the extent of the exposure should depend on the local findings, and so, after accomplishing excision of what appears to be the offending disc lesion, careful inspection of the epidural space around the nerve root is made. The nerve root should be now moveable with a minimum of force. If there is resistance to movement, the aim of the operation is not achieved: a search must be made for the extruded disc fragments, perhaps remote in the tunnel. If the nerve root continues to be tense a foramenotomy should be performed. This may ultimately require removal of the medial portion of the interarticular facet joint. It should be noted that significant portions of this joint can be excised at one level unilaterally without producing subsequent instability. If the persistent tension on the root is due to an underlying spondylotic spur this foramenotomy may be the only solution to afford decompression. Ultimately by extending the laminectomy and foramenotomy it is always possible to relax the nerve root in its tunnel behind the hidden zone, as mentioned before.

SUMMARY AND CONCLUSIONS

1. A historical review of literature on the lumbo-sciatic syndrome was presented.
2. The concept of Lumbar Nerve Root Entrapment was introduced and discussed giving an account on the anatomy of lumbar nerve root tunnels. The pathology of nerve root entrapment was discussed and this came under two big headings, namely soft tissue lesions and bony lesions. The soft tissue lesions included primarily disc pathology and in addition it included adhesions thickened ligamentum flavum, thickened posterior longetudinal ligament, zygoapophyseal capsular and synovial thickenings. The bony lesions included subarticular entrapment, pedicular kinking, entrapment in the chondro-osseous mass at the site of the breach in the pars in cases of spondylolisthesis, entrapment by osteophytic formation and entrapment due to spinal stenosis.
3. A scheme of clinical management was introduced including Epidurography as an ancillary method of investigation. The technique of epidurography was discussed and its ease, safety and high diagnostic value was shown.
4. The clinico-neurologic, epidurographic and operative findings in 30 male patients treated at El-Zatoun Orthopaedic Hospital in Cairo during the years 1971-1972, were presented, analysed and discussed. The age of these patients ranged between 20 and 40 years.
5. This age group was chosen to avoid the perplexity of the superadded effects of the aging process which usually become more manifest over 40. In this age group, trauma appeared to play an important role in inducing and starting the pathological process. The radiculopathy in these patients is not only caused by disc lesions but the secondary effects in the form of inflammation of the nerve roots, adhesion formation and thickening of ligamentum flavum and of posterior longitudinal ligament - (all of which probably represent a fixed cell type hypersensitivty reaction consisting with the autoimmune theory of pathology of disc lesions)- play an important role in the production of symptoms. The clinical picture in these patients was rather acute and the disability was considerable almost all the time. Consequently, the duration of symptoms before the surgical treatment was relatively short ranging between 6 months and 2 years; during this period all sorts of conservative treatment failed to control the disease. The concept of tunnel entrapment also altered our approach to the surgical procedure and the primary aim became to effect and achieve proper decompression of the nerve root rather than dealing with disc lesions only. This could not be done except through a wide exposure provided by a laninectomy.

REFERENCES

ADSON, W.F. and OTT, W.O. (1922) Arch. Neurol. & Psychiat. 8 530-538:
Results of the removal of tumours of the spinal cord.

ARMSTRONG, J.R. (1965) Edinburgh: Livingstone::
Lumbar Disc Lesions. 3rd ed.

BABECHKO, W.P. and HIRSCH, C. (1965) J.B.J.S. 47B 574:
Autoimmune response to nucleus pulposus in the rabbit.

BARR, J.S. (1947) J.B.J.S. 29 429:
Ruptured intervertebral disc and sciatic pain.

BATSON, O.E.(1940) Annals of Surgery. 112 138:
The function of the vertebral veins and their role in the spread of metastases.

BERRY, R.J. (1961) J.B.J.S. 43B 387
Genetically controlled degeneration of the nucleus pulposus in mice.

BRADY, L.P., PARKER, L.B. and VAUGHEN, J. (1969) J.B.J.S. 51A 539:
An evaluation of the electromyogram in the diagnosis of the lumbar disc lesion.

BRAIN, W.R., WRIGHT, A.D. and WILKINSON, M. Lancet. i 227 (1947):
Spontaneous compression of both median nerves in carpal tunnels:
6 cases treated surgically.

BURN, J.M.B. and LAGDON, L. (1970) Rheum. Phys. Med. 10 368:
Lumbar epidural injection for the treatment of chronic sciatica.

BURNS, B.H. and YOUNG, R.H. (1945) Lancet. 6 424:
Protrusions of intervertebral discs.

(1947) Lancet. 623 6454:
Backache

(1951) Lancet. 1 245:
Results of surgery in sciatica and low back pain.

CAILLIET, R. (1975) Philadelphia: Davis. 2nd ed.
Low Back Pain Syndromes.

CATHELIN, M.F. (1901) C.R. soc. biol. Paris 53 450:
Une nouvelle voie d'injection rachidienne. Methode des injections épidurales par le procede du canal sacre. Applications a l'homme.

CHARCOT J.M. (1888) quoted by Miller, H. (1967) Proc. R. Soc. Med. 60 399:
Three great neurologists.

CHARNLEY, J. (1951) Lancet. 1 186:
Orthopaedic signs in the diagnosis of disc protrusions.

(1958) Lancet. 1 1277:
Physical changes in the prolapsed disc.

COTUNIUS, D. (1764) Vienna: Graffer:
De ischiade nervosa commentarius.

CROCK, H.V., YOSSKIZAWA, H. and KAME, S.K. (1973) J.B.J.S. 55B 528:
Observations on the venous drainage of the human vertebral body.

CYRIAX, J. (1942) B.M.J. i 578:
Perineuritis.
(1949) St. Thomas Hosp. Rep. 5 68:
Pressure on the nerves of the neck and upper limbs.
(1971) London: Gower press: 2nd ed.:
The slipped disc.
(1975) London: Baillier Tindall: 6th ed:
Textbook of Orthopaedic Medicine.
DANFORTH, M.S. and WILSON, P.D. (1925) J.B.J.S. 7 109 - 160:
The anatomy of the lumbosacral region in relation to sciatic pain.
DAVIDSON, J.J. and ROBIN, G.C. (1961) B.J. Anaesth. 33 595:
Epidural injections in the lumbosciatic syndrome.
DAWKINS, C.J.M. (1969) Anaesthesia. 24 554:
Complications of extradural and caudal block.
DE PALMA, A.F. and ROTHMAN, R.H. (1970) Philadelphia: Saunders:
The intervertebral disc.
DOMMISSE, G.F. (1974) J.B.J.S. 56B 225:
A critical vascular zone in spinal surgery.
(1975) Orthop. Clin. N. America. Vol. 6 No 1 163 - 173:
Morphological aspects of lumbar spine and lumbosacral region.
EDGAR, M. (1966) J. neurol., neurosurg. & psychiat. 29 530:
The innervation of the spinal dura.
ELLIOT, F.A. (1944) Lancet. 1 47:
Tender muscles in sciatica: electromyographic studies.
ELSBERG, G.A. (1913) Surg. Gyn. Obst. 16 117:
Observations upon 60 laminectomies for spinal disease.
(1916) Philadelphia & London: Saunders:
Diagnosis and treatment of surgical disease of the spinal cord and its membranes.
EVANS, W. (1930) Lancet. 2 1225:
Intrasacral epidural injection in the treatment of sciatica.
EYRING, E.J. (1969) Clinical Orthopaedics. 67 16:
The biochemistry and physiology of the intervertebral disc.
FARFAN, H.F., COSETTE, J.W., ROBERTSON, G.H., WELLS, R.V. AND KRAUS, H (1970) J.B.J.S. 52A 468:
FLINT, M.M. (1965) Phys. Ther. Rev. 45 248:
Electromyographic comparison of iliacus and rectus abdominis muscles.
FORD, L.T. (1968) J.B.J.S. 50A 418:
Local complications of disc surgery.
FORST, J. (1881) Thes de Paris. 33:
Confribution a l'etude clinique de la sciatique.
GALENUS (130 - 200 A.D.) Venetiis, 1525: Aldus et Andreas:
Opera Omnia 1 - 5.

GILL, G.G., MANNING, J.G. and WHITE, H.L. (1955) J.B.J.S. 37A 496:
Surgical treatment of spondylolisthesis without spinal fusion.
Excision of the loose lamina with decompression of the nerve roots.

GOLDTHWAITE, J.E. (1911) Boston med. surg. J. 164 365:
The lumbosacral articulation: an explanation of many cases of lumbago, sciatica and paraplegia.

GRAINGER, R.G. (1975) Recent advances in Radiology. Vol.5 37:
Water soluble lumbosacral radiculography.

HADLEY, L.A. (1955) J.B.J.S. 37A 787:
Bony masses projecting into the spinal canal opposite a break in the neural arch of the fifth lumbar vertebra.

HARRIS, R.I. (1954) J.B.J.S. 36B 304:
Structural changes in the lumbar intervertebral disc.

HIRSCH, C.H. (1965) J.B.J.S. 47A 991:
The efficiency of surgery in low back pain.

HIPPOCRATES. af M.K., Lowergren 1- 2 Gleerup Lund (1910)
De hippokratiska. 460 - 377 B.C.

HODGSON, A.R. and YAN, A. (1969) Recent Advances in Orthopaedics. London: Churchill
Surgical approaches to the spinal column.

HOLLINSHEAD, W.H. (1965) J.B.J.S. 47A 214:
Anatomy of the spine: Points of interest to Orthopaedic Surgeons.

HOWORTH, M.B. (1964) J.B.J.S. 46 A 1845:
Low backache and sciatica: Results of surgical treatment.

IMHOTEP, "God of Healing", "HE WHO COMES IN PEACE" 2800 B.C.
Ebers Pap. quoted by Dr. HASSAN KAMAL in his book "Dictionary of Pharaonic Medicine".
One of the walls of a tomb in Sakarah, Giza, Egypt.(Vis. picture on page 1)

INSTRUCTIONAL COURSE LECTURES. Various Authors. (1968) J.B.J.S. 50A
- 167 - 210: Low back and sciatic pain.
- 382 - 428: Complications of lumbar disc surgery.

JACKSON, H.C., WINKELMAN, R.K. and BICKEL, W.H. (1966) J.B.J.S. 48A 1272:
Nerve endings in the human lumbar spinal column and related structures.

JACKSON, R.K. (1971) J.B.J.S. 53B 609:
The long term effects of wide laminectomy for lumbar disc excision.

JOWETT, R.L., MALCOLM, W.F., AND TROUP, J.D.G. (1975) Orthop. Clin. N.Am. Vol. 6 No.1 145:
Histochemical changes in Multifidus in mechanical derangements of the spine.

KELMAN, H. (1944) Am. J.S. 64 183:
Epidural injection therapy forsciatic pain.

KEY, C.A. (1938) Guy's Hosp. Rep. 111 17:
On paraplegia depending on disease of the ligaments of the spine.
KEY, J.A., (1945) Ann. Surg. 121 534:
Intervertebral disc lesions are the commenest cause of low back pain with or without sciatica.
KEYES, D.C. and COMPERE, E.L. (1932) J.B.J.S. 14 897:
The normal and pathological physiology of the nucleus pulposus of the intervertebral disc.
KNUTSEN, D. and YGGE, H. (1971) Acta Orthop. scand. 42 338:
Prolonged extradural anaesthesia with bupivacaine for lumbago and sciatica.
KNUTSSON, B. (1959) Acta orthop. scand. 28 290:
Electromyographic studies in the diagnosis of lumbar disc herniations.
KNUTSSON, F. (1941) Acta. rad. 22 694:
Experiences with epidural contrast examination of the lumbosacral canal in disc prolapse.
KREMPTON, J.F. and SMITH, B.S. (1974) J.B.J.S. 56A 1435:
Nerve root injection.
LARMON, W.A. (1944) Ann. Surg. 119 892:
An anatomic study of the lumbosacral region in relation to low back pain and sciatica.
LASEGUE, C. (1864) Archs. gen. med. 2 558:
Consideration sur la sciaticque.
LUYENDIJK, W. and VAN VOORTHUISEN, A.E. (1966) Acta Rad. Scand. 5 1051 - 1056
Contrast examination of the spinal epidural space.
MACNAB, I. (1971) J.B.J.S. 53A 891 - 903:
Negative disc exploration.
MATHEWS, J.A. (1972) Modern Trends in Orthopaedics. Vol. 6 148-153:
Epidurography.
MIDDLETON, G.S. and TEACHER, J.H. (1911) Glasg. J. Med. 76 1:
Injury of the spinal cord due to rupture of an intervertebral disc during muscular effort.
MIXTER, W.J. and BARR, J.S. (1934) New Eng. J. Med. 211 210:
Rupture of the intervertebral disc with involvement of the spinal canal.
MORRIS, J.M. LUCAS, D.B. and BRESTER, B. (1961) J.B.J.S. 43A 327:
The role of the trunk in maintaining stability of the spine.
NAYLOR, A. (1962) Ann. R.C.S. Eng. 31 91:
The biophysical and biomechanical aspects of intervertebral disc herniation and degeneration.
(1974) J.B.J.S. 56B 17:
The late results of laminectomy for lumbar disc pathology.

- and, HAPPEY, F., TURNER, T.L., SHENTAL, R.D., WEST, D.C. and RICHARDSON, C. (1975) Orthop. Clin. N. Am. Vol. 6 51:
Enzymic and immunological activity in the intervertebral disc.

O'CONNELL, J.E.A. (1951) J.B.J.S. 33B 8:
Protrusions of the lumbar intervertebral discs.

OPENHEIM, H. and KRAUSE, F. (1909) Deutche med. wschr. 35 562:
Uber Einklemmung bzw. Strangulation der Cauda Equina.

PEDERSEN, H.E., BLUNK, C.F.J. and GARDNER, E. (1956) J.B.J.S. 38A 377:
The anatomy of the lumbosacral posterior rami and meningeal branches of spinal nerves. (sinu vertebral nerves)

PHEASANT, H.C. (1975) Orthop. Clin. N. Am. Vol. 6 No.1 322 - 327:
Sources of failure in laminectomies.

PUTTI, V. (1927) Lancet. 2 53 - 60:
New concepts in pathogenesis of sciatic pain.

ROGERS, L. (1955) Ann. R.C.S. Eng. 16 1:
Tumours involving the spinal cord and its nerve roots.

ROTHMAN, R.H. (1971) Orthop. Clin. N. Am. Vol. 2 No. 2 463 - 475:
The clinical syndrome of lumbar disc lesions.

SICARD, J.A. (1901) C.R. soc. biol. Paris 53 369:
Les injections medicamenteuses extra-durales par voie sacro-coccygienne.

(1918) Presse Med. 26 9:
Nevrodociles et funiculites vertevrales.

SMYTH, M.J. (1958) J.B.J.S. 40A 1401 - 1418:
Sciatica and the intervertebral disc: an experimental study.

STOKES, J.M. (1968) J.B.U.S. 50A 394:
Vascular complications of disc surgery.

TENG, P. (1960) J.B.J.S. 42A 392 - 407:
Root compression.

TROTTER, M. (1947) Anaesth. and Analg. 26 192:
The anatomy of the sacrum.

VERBIEST, H. (1955) J.B.J.S. 37B 576:
Further experiences on the pathological influence on the developmental narrowness of the bony lumbar vertebral canal.

VESALIUS, A. (1555) Basilea per J. Oporinus. 71:
De Humani corporis fabrica libri septem.

WARR, A.C., WILKINSON, J.A., BURN, J.M.B., and LANGDON, L. (1972) Practitioner. 209:
Chronic lumbosacral syndrome treated by epidural injection and manipulation.

WILLIS, T.A. (1959) J.B.J.S. 41A 935:
Lumbosacral anomalies.

YATES, D.A.H. (1965) Ann. phys. med. 8 81:
Epidural myelography with water soluble contrast material (Urographin)