SPINAL COMPRESSION IN CHILDHOOD:

THE UNIVERSITY OF CAPE TOWN EXPERIENCE

A dissertation in fulfilment of Part Three

of the MMed, Neurosurgery

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DECLARATION

I, Joseph E Buwembo, hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other University.

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SIGNATURE

1st March 1996

DATE

DEDICATION

This work is dedicated to my parents, *Alice Manjeri* and the late *Erieza Kabega*, for having provided the basic foundation onto which others have contributed, to make me what I am.

ACKNOWLEDGEMENTS

I sincerely thank the following people whose help was instrumental in accomplishing this work.

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* Secondly, Mrs Celeste Herbert, for wholeheartedly taking on the difficult task of typing the manuscript, arranging the figures and tables and the bibliography.

* Last, but not least, to Professor Jonathan Peter, for supervising this work.

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CHAPTER 1

INTRODUCTION

1.1 Overview of Spinal Compression

Spinal compression is pressure on the intraspinal neural structures by a lesion that either compromises the spinal canal or expands within the cord substance, which at a critical level causes disturbance of function. This will manifest clinically as limb weakness, sphincter impairment, sensory deficits and pain syndromes.

The phenomenon is relatively uncommon in childhood when the frequency is compared to that of adults (Coxe, 1961; de Villiers & Cluver, 1978; Peacock & Lazareff, 1986) and most of the neurosurgical literature on the subject reports on intramedullary spinal cord tumours as the major cause of compression (Craig & Mitchel, 1931; Coxe, 1961; De Sousa et al, 1979). However, there are a multitude of causes that should be known by doctors involved in the treatment of children. Tuberculosis (TB), pyogenic and parasitic infections are common in Africa and other underdeveloped regions (Bailey et al, 1972; Carrea et al, 1975; Johnston, 1992).

In the developing world where tuberculosis is endemic, the spinal type affects mainly children (Kemp, 1976). In the 1973 Medical Research Council (MRC) series in Korea, 78% of the patients were aged under 10 years (MRC, Korea, 1973a). This is unlike the developed world where tuberculosis is rare and the spinal type affects mainly adults. Kemp (1976), found that less than 10% of the patients treated for spinal TB at the Royal National Orthopaedic Hospital, London, were under 20 years.

Hoffman, Crosier and Cremin (1993), reported on 64 consecutive children with spinal tuberculosis that were investigated radiologically at the Red Cross War Memorial Children's Hospital (RXH) over the three-year period under review, giving an annual incidence of about 21 children. In a separate report by Cremin, Jamieson and Hoffman (1993), neurological involvement was estimated to be 30% of all patients with spinal TB seen annually at the RXH.

De Villiers and Cluver (1978), reported on four cases of paediatric spinal epidural abscess over a 10-year period, during which time nine adults were treated for the same condition.

Carrea et al (1975), found one spinal hydatid to 29 cerebral hydatids over a 42-year period at the Children's Hospital for neurosurgery, Buenos Aires.

Whereas brain tumours are only second to leukaemia in frequency as far as childhood neoplasia is concerned, the ratio of spinal to brain tumours is given as 1:5-1:20 (Ingraham & Matson, 1969; Harwood-Nash & Fitz, 1976; Hendrick, 1982).

Svien et al (1954) reported 41 cases of intraspinal tumours in children less than 15 years, to have been treated at the Mayo Clinic over a 20-year period, whereas Peacock and Lazareff (1986) found that over a seven-year period, 20 children under 14 years were treated for spinal tumours in the Neurosurgical Department of the University of Cape Town.

The clinical picture of spinal compression will depend on the anatomical region of the spinal cord or nerve root(s) involved by the compressing process.

The speed of evolution of the compressive lesion determines the rapidity of onset of neurological dysfunction, but the intramedullary tumours initially grow slowly and present in a subtle way, commonly with musculo-skeletal abnormalities e.g. scoliosis, talipes equinus and pes cavus, which are sometimes treated for long periods before the diagnosis of cord compression is suspected (Haft

et al, 1959; Tachdjian & Matson, 1965; Maurice-Williams & Richardson, 1988).

Although successful surgical treatment of a spinal tumour was accomplished by Horsley in 1887 (Gowers & Horsley, 1888) several recent publications have indicated that the problem of spinal cord compression is often misdiagnosed and that many patients are referred to the neurosurgical service after a long avoidable delay, many a time with irreversible neurological impairment (Melvill, 1976; Maurice-Williams & Richardson, 1988).

Neuroradiological investigations such as plain x-rays, soluble contrast myelography, computerised tomography (CT), myelography and magnetic resonance imaging (MRI), aid in the diagnosis of spinal cord pathology and in planning surgery (Bloom, Ellis & Jennett, 1955; Allen, Cosgrove & Millard, 1978; Cremin, Jamieson & Hoffman, 1993), but such investigations can only be done after clinical suspicion of a spinal lesion.

Surgical decompression of the spinal cord can be achieved via a posterior, anterior or lateral approach for respectively placed lesions (Greenwood, 1967; Kemp et al, 1973; Johnston, 1993). This not only relieves cord compression but also allows one to obtain specimens for histological diagnosis and/or microbial culture and antibiotic sensitivity. This information is essential for deciding on adjuvant therapy, which would be radiotherapy or chemotherapy for malignant lesions and antibiotics in case of infections.

Successful treatment should result in the return of normal neurological function and a stable, painless spine. Many times, however, patient, family and surgeon may have to accept lesser degrees of functional recovery.

1.2 Anatomy of the Spinal Cord

1.2.1 Gross Anatomy

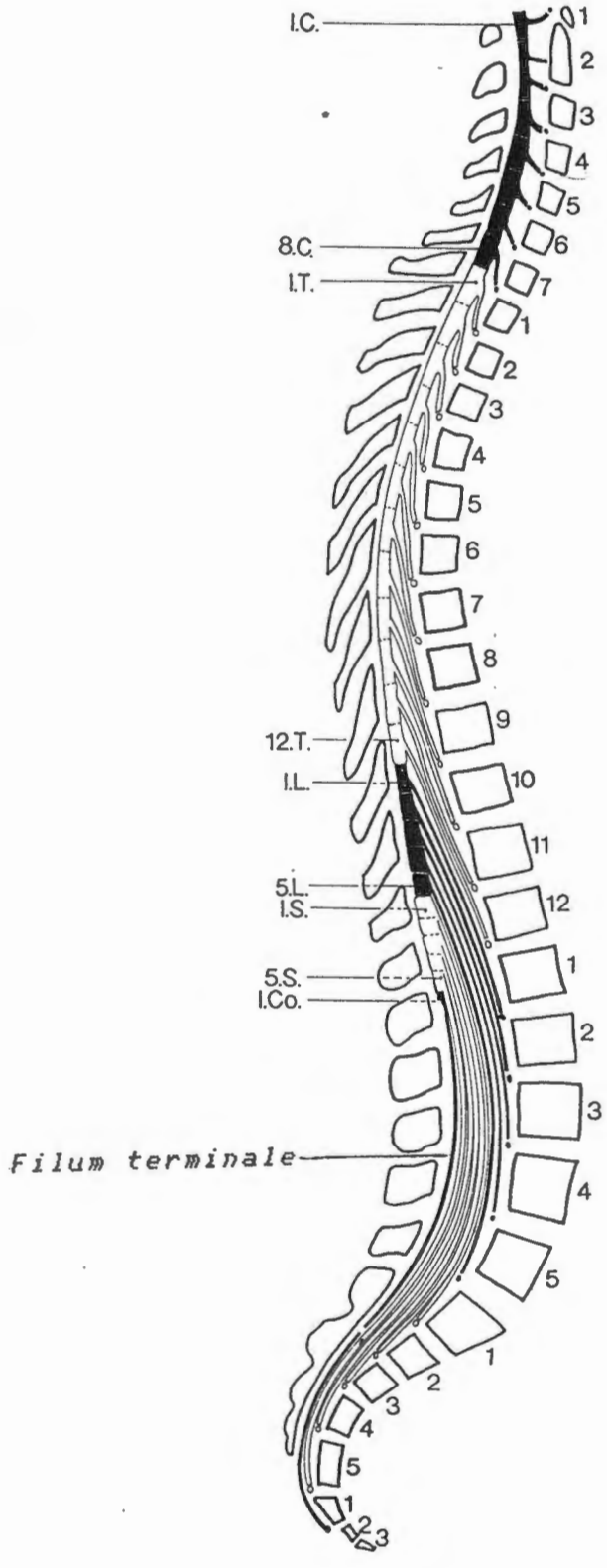


Fig 1: *Diagram of spinal cord and nerve roots, showing the anatomical relationship to the vertebral column. Note the cervical and lumbar enlargements.*

KEY: C = CERVICAL
T = THORACIC
L = LUMBAR
S = SACRAL
Co = COCCYGEAL

The spinal cord is situated within the spinal canal, and commences at the craniocervical junction as the caudal continuation of the medulla oblongata. It normally ends at the lower border of the first lumbar vertebra. Caudally it tapers into the conus medullaris from which a pia mater extension, the filum terminale, descends through the lumbar and sacral subarachnoid space, pierces the dura and is attached at the back of the coccyx. The cord is covered by meninges and bathed in cerebrospinal fluid (CSF) in a manner similar to the brain. The spinal subarachnoid space normally communicates freely with the cisterna magna. Another extension of pia mater, the ligamentum denticulatum, anchors the cord laterally to the dura at regular intervals between the nerve roots (Figure 2). The cord has a deep anterior medium sulcus in which the anterior spinal artery runs, and it has a less prominent posterior median fissure from which arachnoid septi anchor it posteriorly. The postero-lateral sulcus marks the entry of the dorsal roots. A less prominent antero-lateral sulcus marks the exit of the ventral roots. The cord has cervical and lumbar enlargements corresponding to the segments that innervate the upper and lower limbs respectively. These enlargements are due to the greatly increased mass of motor cells in the anterior grey horns in these locations (Barr, 1979). Thirty-one pairs of spinal nerves convey afferent and efferent impulses from and to the body respectively, in a segmental manner i.e. cervical, thoracic, lumbar, sacral and

coccygeal (Figures 1 and 2). Below L1, the anterior and posterior nerve roots pass almost vertically downwards through the subarachnoid space, and together with the filum terminale, constitute the cauda equina (Last, 1978).

Internal Structure

The cord has a central canal which is very narrow, is ependymal lined and contains a very small amount of CSF. Although a direct continuation of the fourth ventricle, in 85% of people the central canal is not patent in the upper three cervical segments (Netsky, 1953). On cross-section, a roughly 'H-shaped' grey matter comprising cell bodies, appears around the central canal. It has larger anterior horns containing motor cells that give rise to the ventral roots or lower motor neurones. The smaller dorsal horns receive most sensory fibres entering via the dorsal root. The Dorsal Root Entry Zone is posterolateral to the posterior greyhorn (Figure 4). Cell bodies in the dorsal horns give rise to second order neurones which ascend in various tracts, and to internuncial neurones for spinal reflexes (Carpenter, 1985). The grey matter is surrounded by the white matter which contains long ascending and descending tracts (Figure 3).

Tracts of Clinical Importance

Ascending Tracts

The lateral spinothalamic tract conveys pain and temperature sensation from the opposite side of the body to the thalamus, from where the information is relayed to the sensory cortex for interpretation. Fibres to this tract enter the Dorsal Root Entry Zone, ascend a few segments, relay in the substantia gelatinosa and the second order neurones cross anterior to the central canal to ascend in these tracts (Barr, 1979).

The anterior spinothalamic tract (crossed) conveys light touch and pressure sensations to the thalamus, from where fibres relay to the sensory cortex.

The dorsal columns comprise the medial fasciculus gracilis and lateral fasciculus cuneatus which convey uncrossed conscious proprioceptive impulses (position sense, vibration) and light touch to the gracile and cuneate nuclei in the medulla from where fibres cross to ascend to the thalamus before being relayed to the sensory cortex.

Descending Tracts

The lateral corticospinal tract conveys motor impulses for voluntary action, from the pyramidal cells of the motor cortex of the opposite side to the anterior horn cells. Fibres cross in the pyramidal decussation in the medulla, descend in this tract and synapse with the anterior horn cells. Thus the tract gets smaller as it descends through the cord. The anterior corticospinal tract is a smaller tract conveying uncrossed motor fibres from the pyramidal cells of the motor cortex but crosses at each segment to synapse with the anterior horn cells of the opposite side. This tract is dissipated in the mid-thoracic segments (Elsberg, 1941). The anterior horn cells are influenced by nearly all descending tracts, but the overwhelming clinical importance of the lateral corticospinal tract has caused it to be equated with the upper motor neurone (Carpenter, 1985). The intermedial lateral column projects as the lateral horn of grey matter between T1 and L2 and contains connector cells for the outflow of the sympathetic nervous system. Fibres reaching this column, project from autonomic centres in the hypothalamus, midbrain, and pons, and descend in the lateral white column. The parasympathetic fibres for bladder and rectal sphincter reflexes, descend adjacent to sympathetic fibres in the lateral white column and relay in the sacral parasympathetic nucleus in S2, S3 and S4 segments (Barr,

1979). The rubrospinal tract originates from the red nucleus in the midbrain, and, having crossed in the ventral tegmental decussation, the spinal fibres descend just anterior to the lateral cortical spinal tract. It is part of the extrapyramidal motor system whose role is to control muscle tone in the flexor muscle groups.

Somatotopic Localisation

The fibres in the white columns are somatotopically arranged (Elsberg, 1941). In the posterior white column (gracile and cuneate fasciculi) the sacral fibres are most medial, followed by lumbar and thoracic, and the cervical are most lateral. In the corticospinal tracts the arrangement is cervical, thoracic, lumbar and sacral from medial to lateral, respectively. In the spinothalamic tract the sacral fibres are most lateral, followed by lumbar and thoracic, and the cervical are most medial. Thus an extramedullary mass compressing the spinothalamic tract is likely to cause sacral sensory impairment initially.

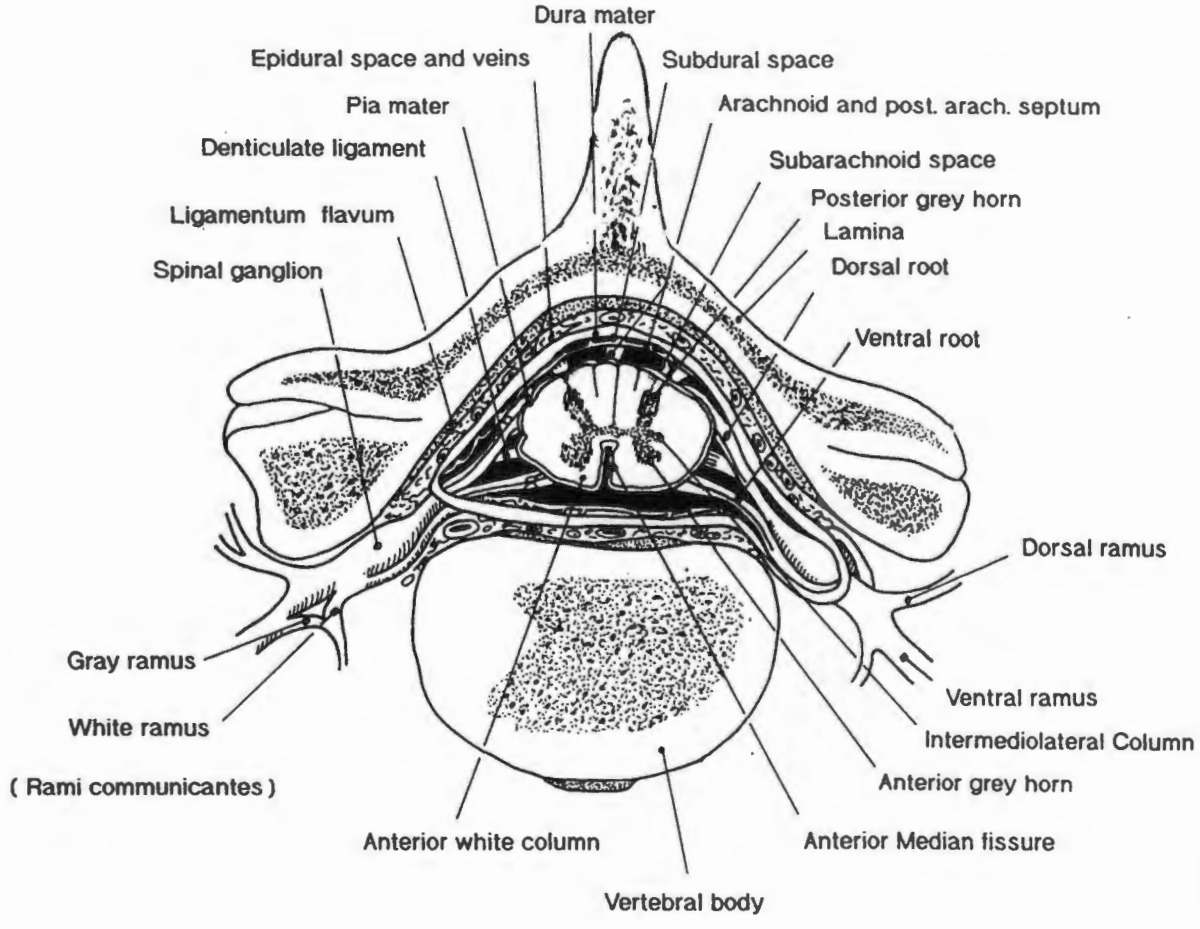


Fig 2: *Diagram of cross-section of spinal cord, showing the relationship to nerve roots and meninges. Modified from Carpenter MB, Core Text of Neuroanatomy, 1985).*

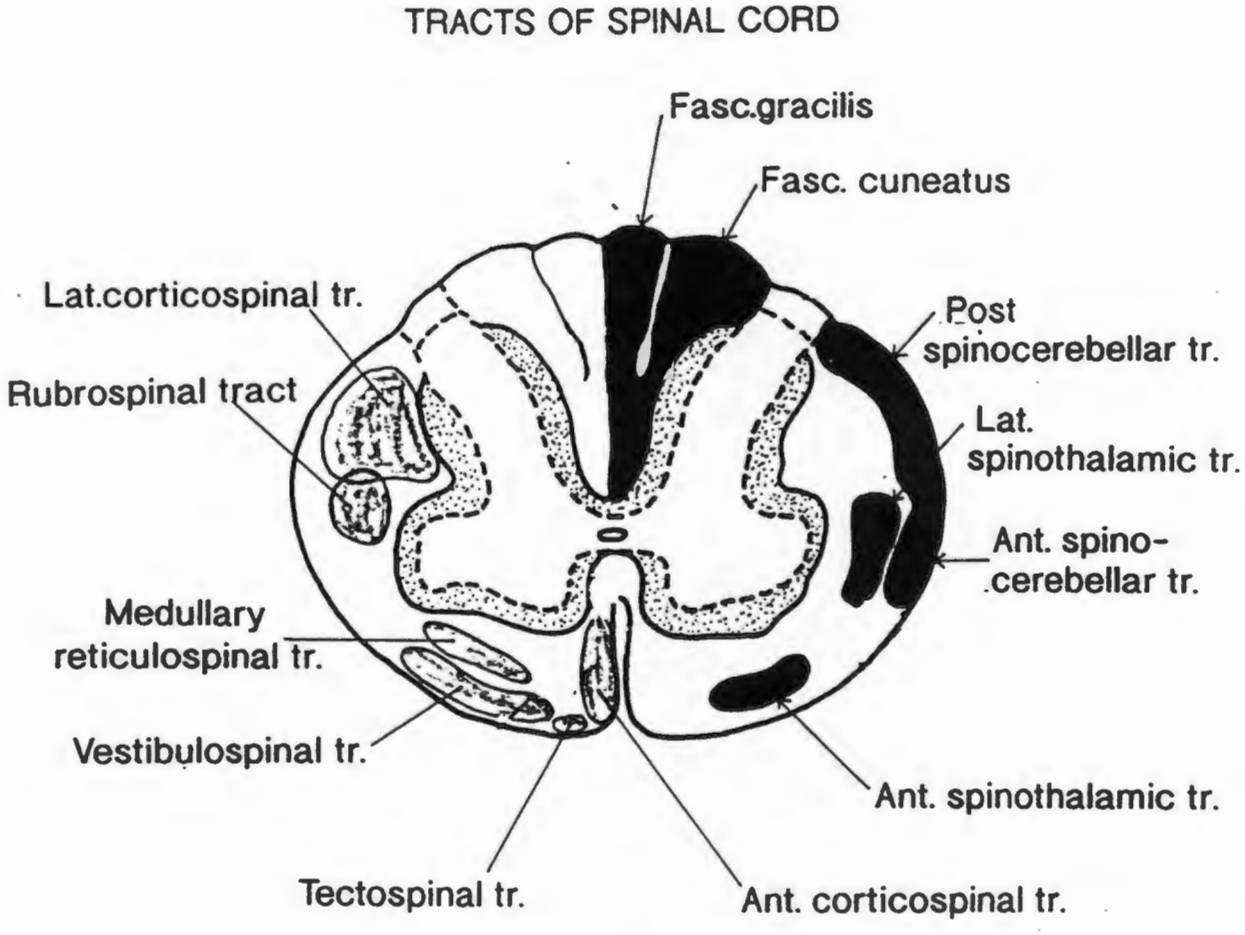


Fig 3: *Ascending and descending tracts.*

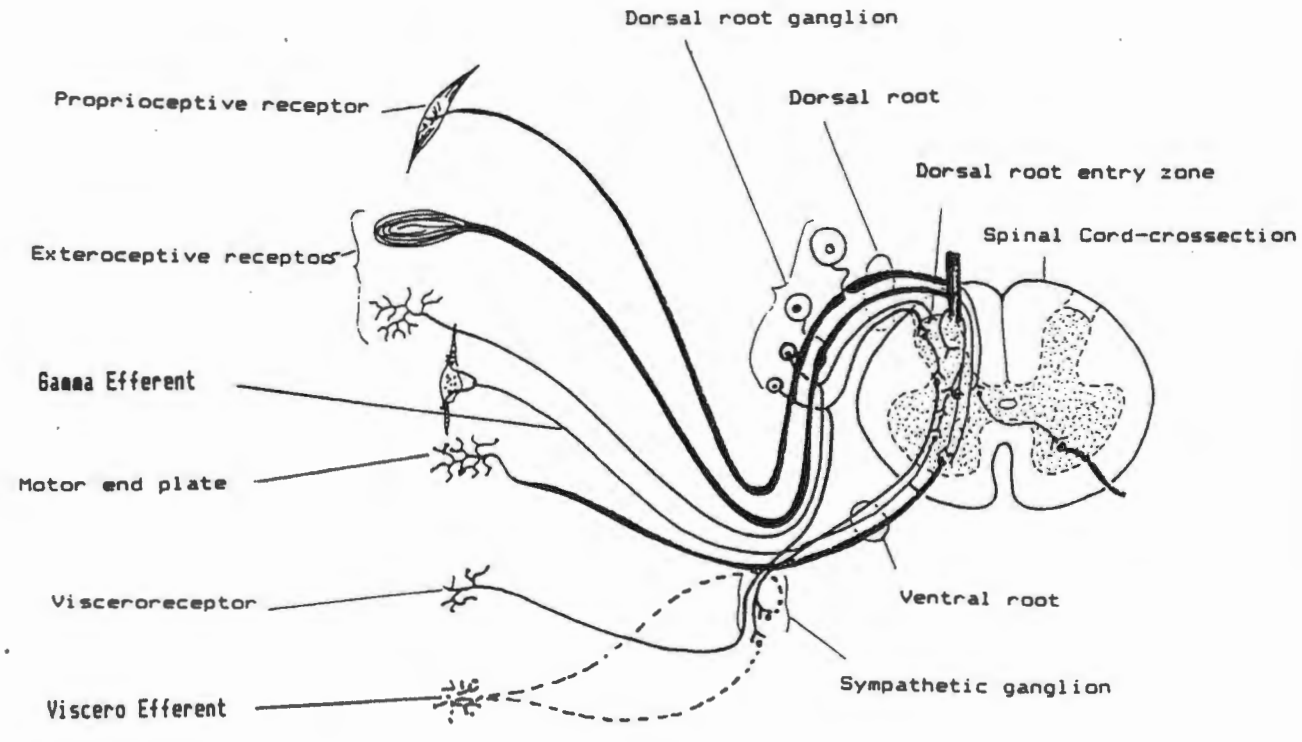


Fig 4: *Dorsal Root Entry Zone.*

1.2.2 Blood Supply

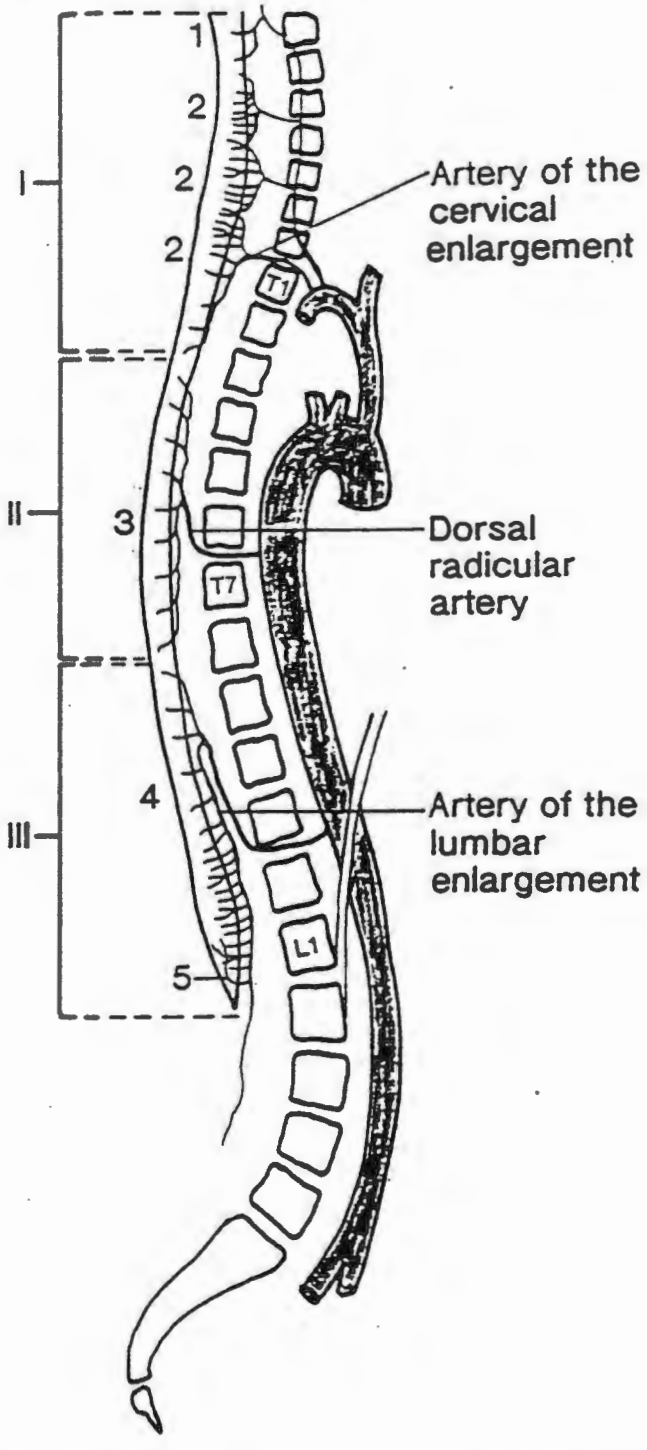


Fig 5: *The three vascular territories of the spinal cord (Modified from Lazorthes G, 1971).*

The cord is supplied by two arterial systems:

a. the anterior spinal artery which supplies the anterior two-thirds of the cord; and
b. the two posterior spinal arteries which supply the posterior one-third.

The anterior spinal artery is augmented by segmental radicular arteries. In the lumbar region one of them is very large, and usually on the left (the artery of Adamkiewicz) and augments arterial supply to the lumbar enlargement. Lazorthes et al (1971) described the three unequal vascular territories of the spinal cord. The mid-thoracic segments are in a watershed zone and are vulnerable to ischaemia in the event of hypotension or occlusion of the radicular arteries.

1.3 Anatomical Pathology of Spinal Compression

The relationship of the spinal lesion to the dural membrane and the spinal cord, has practical surgical implications. Thus lesions are classified as extradural or intradural and the latter can be extramedullary or intramedullary (Elsberg, 1941). (See Figures 8a-c).

1.3.1 **Extradural**

Primary vertebral tumour (e.g. osteo-chondroma), metastases (e.g. neuroblastoma), TB spine, pyogenic abscess, parasitic cyst (e.g. hydatid), fracture dislocation.

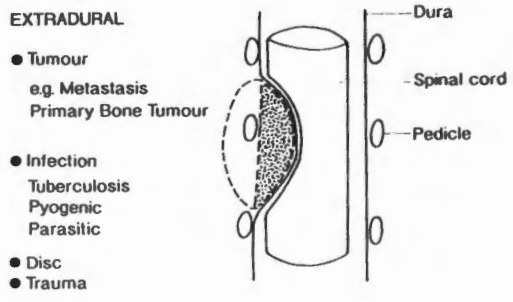


Fig 6a: *Extradural.*

1.3.2 **Intradural**

1.3.2.1 **Extramedullary**

Neurofibroma, meningioma, cysticercosis, arachnoid cyst.

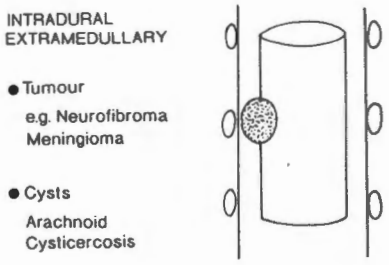


Fig 6b: *Intradural/Extramedullary.*

1.3.2.2 **Intramedullary**

Astrocytoma, ependymoma, haemangioblastoma, juvenile arteriovenous fistula.

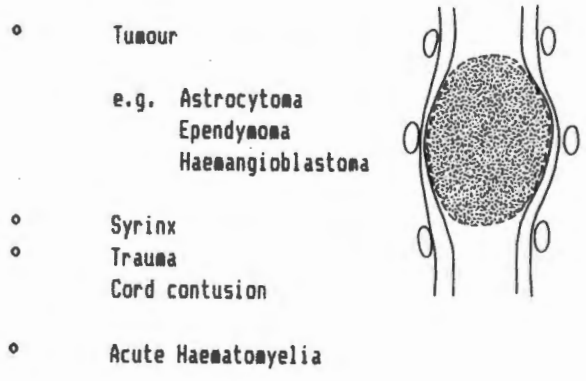


Fig 6c: *Intradural/Intramedullary.*

1.3.3 **Clinical Spinal Cord Syndromes**

These were clearly described by Elsberg (1941).

1.3.3.1 **Transverse**

Usually caused by rapid cord compression or by transsection.

Early: spinal shock. Results in loss of all sensory modalities and motor activity below the lesion.

Late: reflex activity. Lower motor neurone lesion at the level and upper motor neurone signs below.

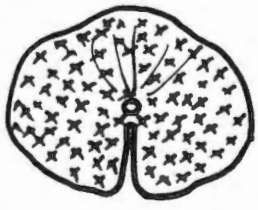


Fig 7a: *Transverse.*

1.3.3.2 **Brown-Séquard (Cord hemisection)**

Below the lesion, there is ipsilateral loss of power and proprioception and contralateral loss of pain and temperature sensation. (Not usually pure in the clinical setting). Usually traumatic, but may be caused by tumours e.g. meningioma.

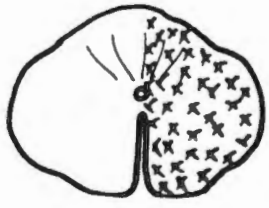


Fig 7b: *Brown-Séquard (Cord hemisection).*

1.3.3.3 **Anterior Spinal Syndrome**

Loss of power, pain and temperature sensation below the lesion while proprioception is preserved. May be caused by anteriorly situated tumours or by anterior spinal artery thrombosis.

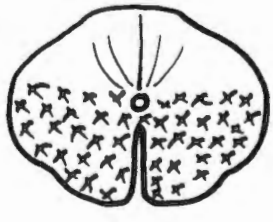


Fig 7c: *Anterior.*

1.3.3.4 **Posterior Cord Syndrome**

Loss of proprioception below the lesion. May be caused by posteriorly situated tumours.

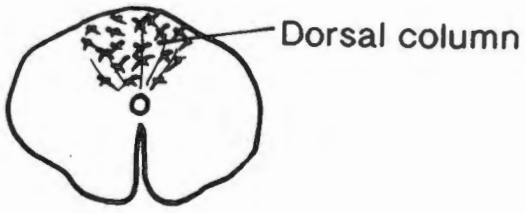


Fig 7d: *Posterior.*

1.3.3.5 **Central Cord Syndrome**

At the level of the lesion:

1. Lower motor neurone signs.
2. Loss of pain and temperature (suspended, dissociated sensory loss).

Below the lesion:

Upper motor neurone signs. Preservation of sensation in sacral dermatomas (sacral sparing) is an important finding.

May be caused by intramedullary tumours, syrinxes.

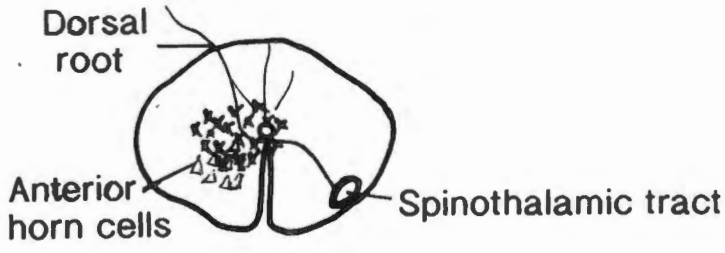


Fig 7e: *Central*.

1.3.3.6 **Conus Medullaris Syndrome**

The conus medullaris is that portion of the cord caudal to, and including the fifth lumbar cord segment.

Lesions may show mixed upper and lower motor neurone signs in the lower limbs, plus a dissociated saddle sensory loss. Deficits are usually symmetrical. Sphincter involvement is early.

Cauda Equina Syndrome

Deficits are usually asymmetrical and include sacral anaesthesia and mono-radicular or poly-radicular signs in the lower extremities. Sphincter impairment is usually late.

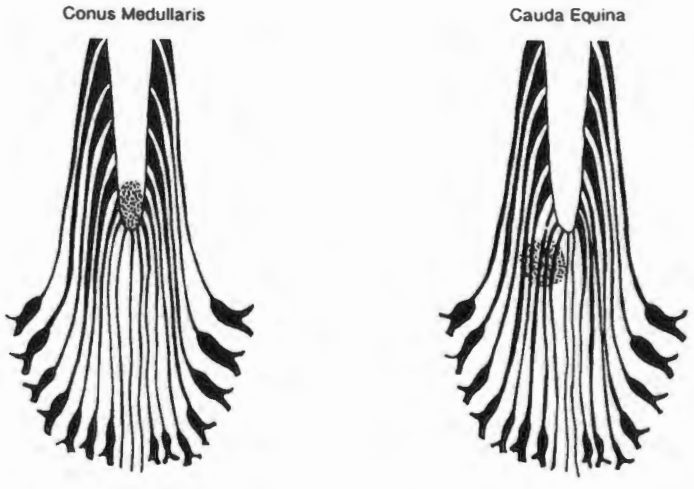


Fig 7f: *Conus Medullaris/Cauda Equina.*

1.3.3.7 **Dorsal Root Entry Zone**

Lesions result in loss of all sensory modalities and a flaccid weakness of a radicular distribution. No atrophy.

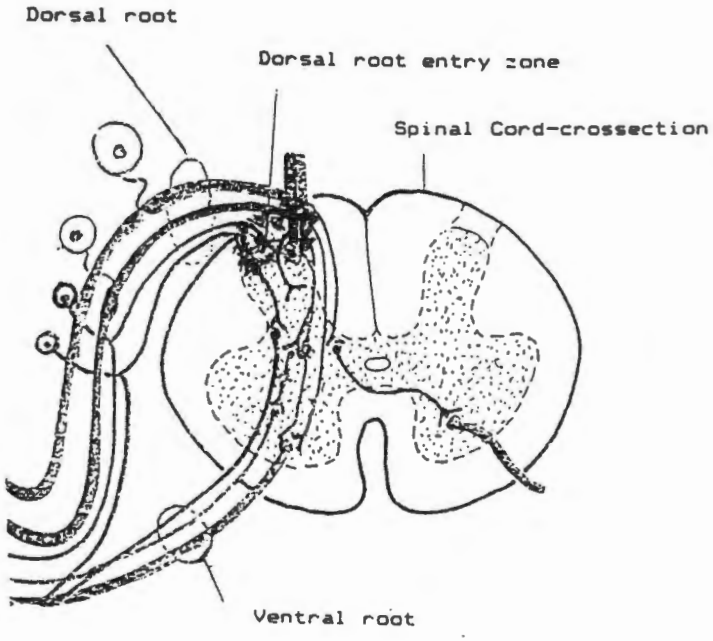


Fig 7g: *Dorsal Root Entry Zone lesion.*

1.4 Pathophysiology of Spinal Compression

Compression, Ischaemia, Hypoxia

There is selective vulnerability of white and grey matter to pressure. Large diameter fibres (15-20 μm) are more susceptible (Gasser & Erlanger, 1929; Tarlov, 1957), whereas grey matter is less affected because it has a richer blood supply; it has less myelin and more glial cells and in cases of extramedullary compression, it is protected by the white matter around it (McAlhany & Netsky, 1955).

Interruption in nerve conduction, resulting in a neurological deficit, can result from pressure per se, and this can be reversed by timely decompression (Hodgson, Skinsnes & Leong, 1967). Compression due to a slowly expanding lesion can result in marked displacement and flattening of the cord while still retaining normal function (Elsberg, 1941). The blood vessels in this case accommodate to the abnormal anatomy (Findlay, 1987).

Long-standing compression impairs neuronal nutrition by compromising the arterial supply and venous drainage, which can result in demyelination (may be reversible following decompression) or infarction (which is irreversible).

Vascular occlusion and cord infarction can also result from a rapidly expanding lesion such as an epidural abscess. This may be on the basis of extramural compression of the vessel (Purves-Stewart & Riddoch, 1923; McAlhany & Netsky, 1955). These authors found from postmortem studies of 106 (1923) and 19 (1955) cases of spinal compression, that the main radicular and pial arteries were collapsed but never thrombosed. The small, scattered cord infarcts followed occlusion of terminal medullary vessels. Feldenzer et al (1988) showed similar findings in an experimental model of spinal epidural abscess developed in rabits.

In the event of traumatic paraplegia due to fracture dislocation of the spine, the cord compression has occurred within a fraction of a second, resulting in disruption of cord tissue, an irreversible lesion (Findlay, 1987).

Grey matter is more susceptible to hypoxia (ischaemia) than white matter (Krogh, 1950) and the small diameter fibres are more susceptible than the larger.

Consistency of Mass

Lesions of hard consistency, such as a hard meningioma or a hard disc, are likely to cause a more pronounced neurological deficit than a soft lesion of similar size, such as a cyst (Findlay, 1987). This may be due to the fact that the hard lesion compromises the cord more during flexion/extension movements of the spine and also during coughing and straining when the engorgement of the epidural veins causes squeezing of the dura and generates a powerful cerebrospinal fluid pressure wave, which is transmitted to the cord.

Late Onset Pott's Paraplegia

The early paraplegia that occurs in the active phase of Pott's disease can be caused by pressure from pus, granulation tissue, sequestra (bony or disc), subluxation and/or dislocation or by vascular compromise (Butler, 1935; Seddon, 1935; Hodgson & Yau, 1967).

The late onset paraplegia on the other hand, occurs after a long interval of quiescence, which may range from four years to as many as 40 years. The cause is most commonly an increasing kyphosis due to a persistent low-grade infection; but a bony ridge at the internal kyphus, epidural fibrosis and at times, granulation tissue and

sequestra, can be the cause of cord compression (Seddon, 1935; Hodgson & Yau, 1967; Hsu, Cheng & Leong, 1988).

Pain

There are three types of pain. Local back pain, radicular pain and central spinal cord pain.

Local pain is due to local dural distension or to local involvement of bony or ligamentous structures (Loeser, 1980).

Radicular pain is of dermatomal distribution and is due to irritation of a nerve root or occasionally the dorsal root ganglion as it lies in the nerve root foramen (Howe, Loeser & Calvin, 1977; Loeser, 1980). It may sometimes be referred to a viscus sharing the same spinal cord segmental supply and may manifest as visceral disease in the abdomen or chest (Bloom, Ellis & Jennett, 1955; Tyson, Grant & Strachan, 1979).

Central pain is due to involvement of central pain pathways by lesions, and is usually of a burning quality and a wide, non-dermatomal distribution (Druckman & Lende, 1965; Guidetti, Mercuri & Vagnozzi, 1981).

CHAPTER 2

STUDY

2.1 Aim

A retrospective study of cases of childhood spinal compression over a 30-year period (1963-1992) was undertaken in order to determine the trend in incidence, aetiology, diagnosis, treatment and prognosis.

2.2 Patients and Methods

The study includes children less than 15 years of age who were treated for spinal compression at the Red Cross War Memorial Children's Hospital, Maitland Cottage Hospital and Groote Schuur Hospital. Children who had tuberculosis of the spine without a neurological deficit, were excluded. The study also excludes dysraphism and non-compressive causes of paraplegia, such as Guillain-Barré Syndrome and poliomyelitis.

The patient data pertaining to demographic characteristics, presenting symptoms, clinical signs, diagnostic work-up, treatment and outcome, was extracted from records which were obtained in the following ways:

* A computer printout for the period January 1986 to December 1992 was obtained from the Medical Informatics Department at the Red Cross War Memorial Children's Hospital, using the following key words: *spinal cord compression, paraplegia, TB spine, epidural abscess, epidural haematoma, spinal cord tumour.*

* The computer data provided the folder number and patient's name, so folders were retrieved from the Records Department and studied together with the relevant radiological investigations.

* The other records were obtained directly from the Department of Neurosurgery and Oncology Unit at Red Cross Hospital, the Department of Neurosurgery at Groote Schuur Hospital and the Maitland Cottage Orthopaedic Hospital.

* Reference was also made to the publications by Melvill (1976), Peacock & Lazareff (1986) and Peter, Kieck & de Villiers (1992).

2.3 Results

2.3.1 Demographic Characteristics

Ninety-seven children fulfilled the study criteria. The male:female ratio was 3:2. The youngest patient was five months.

Of the patients with TB spine, 77.3% were under five years, and all were under 10 years.

2.3.2 **Patient Origin** (see Figure 8)

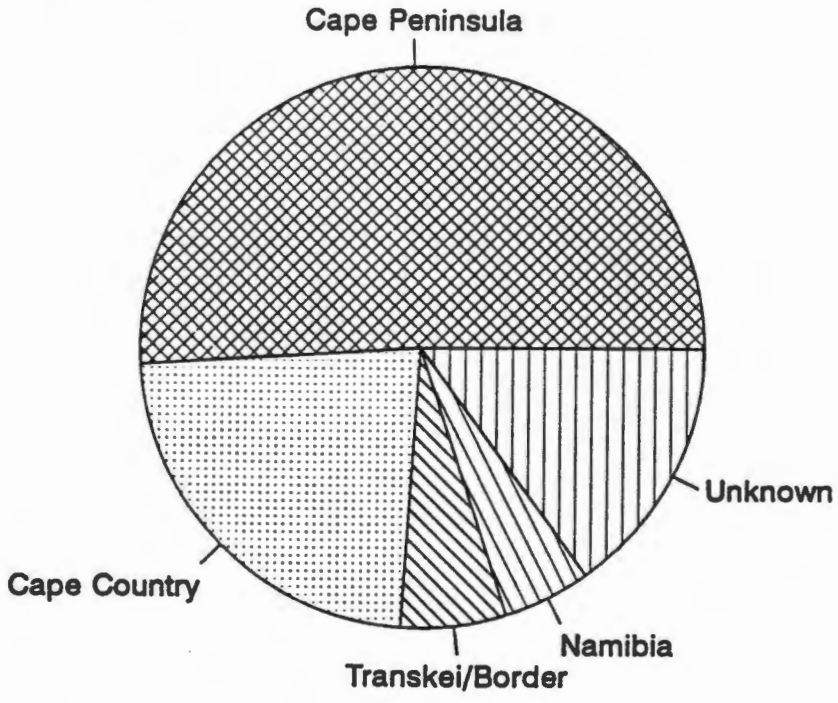


Fig 8: *Patient origin.*
Referral pattern to the Department of Neurosurgery:
10% Private Practitioner
24% Primary Health Care Institution
26% Secondary Health Care Institution
28% Other departments within a tertiary institution
23% Could not be verified

2.3.3 **Causes of Spinal Cord Compression**
(See Table 1 and Figures 11a & b)

Overall, neoplastic lesions were the commonest causes (33 primary and 22 secondary lesions) followed by infective causes (Tuberculosis 22, Epidural abscess 12, Parasitic 4). There were four miscellaneous causes.

Table 1 **Causes of Spinal Compression According to Anatomical Site**

	Extradural	Intradural Extramedullary	Intradural Intramedullary	Total
Cervical	-	6	8	14
Thoracic	52	1	8	61
Lumbar	13	4	2	19
Sacral	3	-	-	3
	68	11	18	97

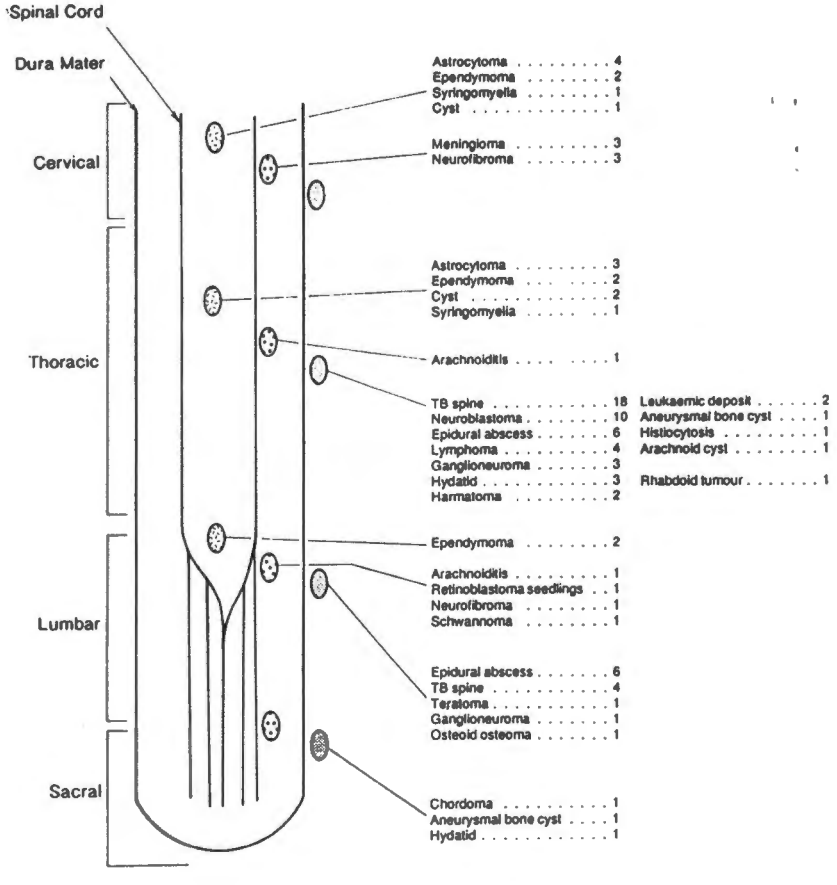
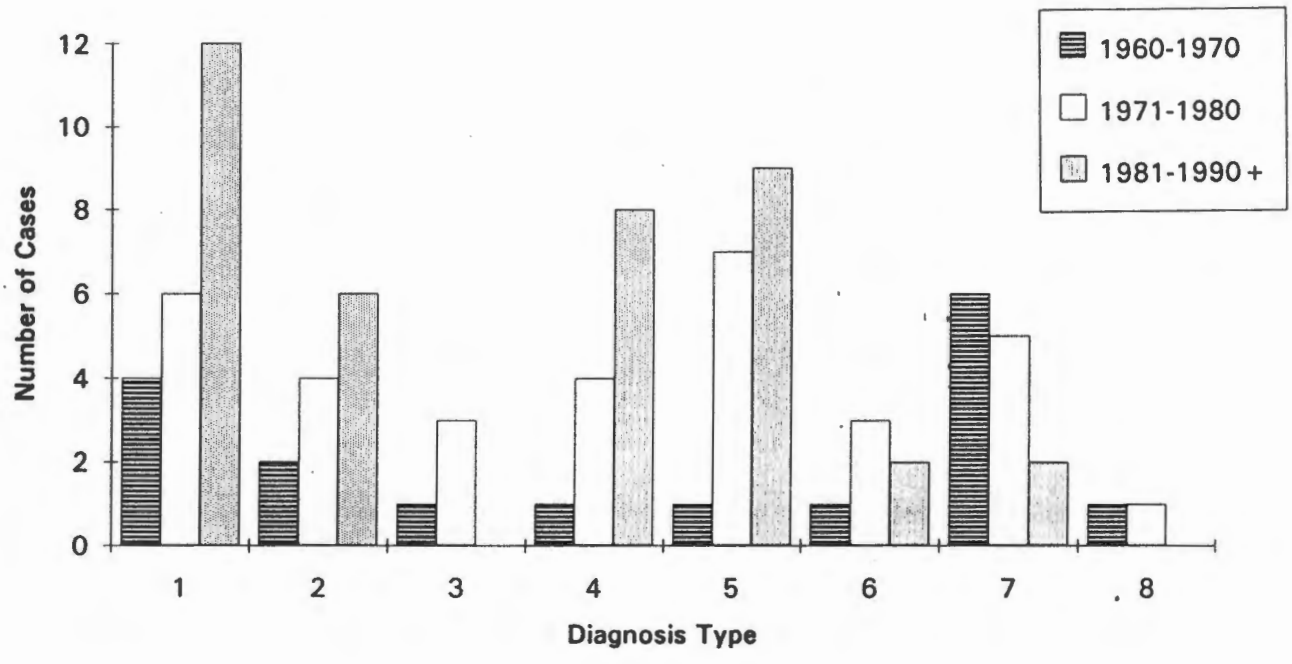


Fig 9a: *Causes of spinal compression.*



Key - Diagnosis type

1 - *TB spine*
2 - *Spinal epidural abscess*
3 - *Parasitic*
4 - *Benign extradural neoplasm*
5 - *Malignant extradural neoplasm*
6 - *Intradural extramedullary neoplasm*
7 - *Intramedullary neoplasm*
8 - *Focal arachnoiditis*

Fig 9b: *Trend in incidence and aetiology of spinal cord compression.*

Extradural (68 cases)

The commonest cause of extradural spinal compression was tuberculosis of the spine with 22 cases, 18 of which were in the thoracic region and four were lumbar. Malignant extradural neoplasms ranked second with 17 cases - all in the thoracic region - and spinal epidural abscess third with 12 cases - six thoracic and six lumbar. The commonest malignant lesion was neuroblastoma (10 cases) followed by lymphoma (four cases) and leukaemia (two cases).

Intradural Extramedullary (11 cases)

These comprised three meningiomas and four neurofibromas. One case of retinoblastoma seedlings, two cases of focal arachnoiditis and one malignant Schwannoma in the lumbar region.

Intradural Intramedullary (18 cases)

The seven astrocytomas all occurred in the cervical and upper thoracic regions.

The six ependymomas had an even distribution in the cervical, thoracic and lumbar regions.

2.3.4 Clinical Features

These are summarised in Tables 2 and 3.

Table 2 **Symptoms**

Age in Years	**Under 5**	**5-10**	**11-15**	**Total**
Symptoms				
Lower limb weakness	8	14	17	39
Backache	5	6	8	19
Sphincter disturbance	2	4	4	10
Radicular pain	1	1	8	10
Upper limb weakness	2	3	4	9
Sensory loss		1	2	3
Central pain		-	1	1
Other	-	4	5	9

Table 3 **Signs**

Age in Years	**Under 5**	**5-10**	**11-15**	**Total**
Signs				
Paraplegia	4	8	6	18
Paraparesis	2	6	8	16
Quadriparesis	1	3	3	7
Lower limb monoparesis	2	4	6	12
Hand muscle atrophy	-	-	2	2
Lower motor neurone	6	7	14	27
Upper motor neurone	3	14	11	28
Sensory	3	4	8	15
Scoliosis	2	1	5	8
Gibbus	17	5	-	22
Foot abnormality	-	2	-	2
Neurocutaneous	3	-	-	3
Other	1	2	-	3

Sudden paraplegia was the way of presentation in eight patients with malignant epidural neoplasms and four patients with spinal epidural abscesses. Neurocutaneous lesions comprised cafe au lait spots in three patients with Type I neurofibromatosis.

A gibbus was noted in all the children with TB, due to various degrees of vertebral body destruction accompanied by anterior wedge collapse.

2.3.5 Radiological Investigations

These are presented in Figures 10-18.

Plain x-rays have been available in all three decades.

Myelography was performed using the oil contrast medium, Myodil, during the period 1960-70, then was discontinued when the water soluble Metrizamide was introduced in the early 70's. The main complication of Myodil myelography was spinal arachnoiditis (2-3% severe, 5-10% minor), and meningeal reaction (? meningism, ? meningitis).

The water soluble contrast media of the 80's (Jopamiron and Omnipaque) which have replaced Metrizamide, have minimal side effects.

Computerised Tomography (CT) myelography was introduced in 1978 and was popularised in the 80's.

Magnetic Resonance Imaging was available since 1986.

Isotope bone scanning, using ^{99}Technetium was used in the last decade under review as part of the work-up for suspected spinal infection, where plain x-rays had not been helpful. It had an accuracy rate of 81%.

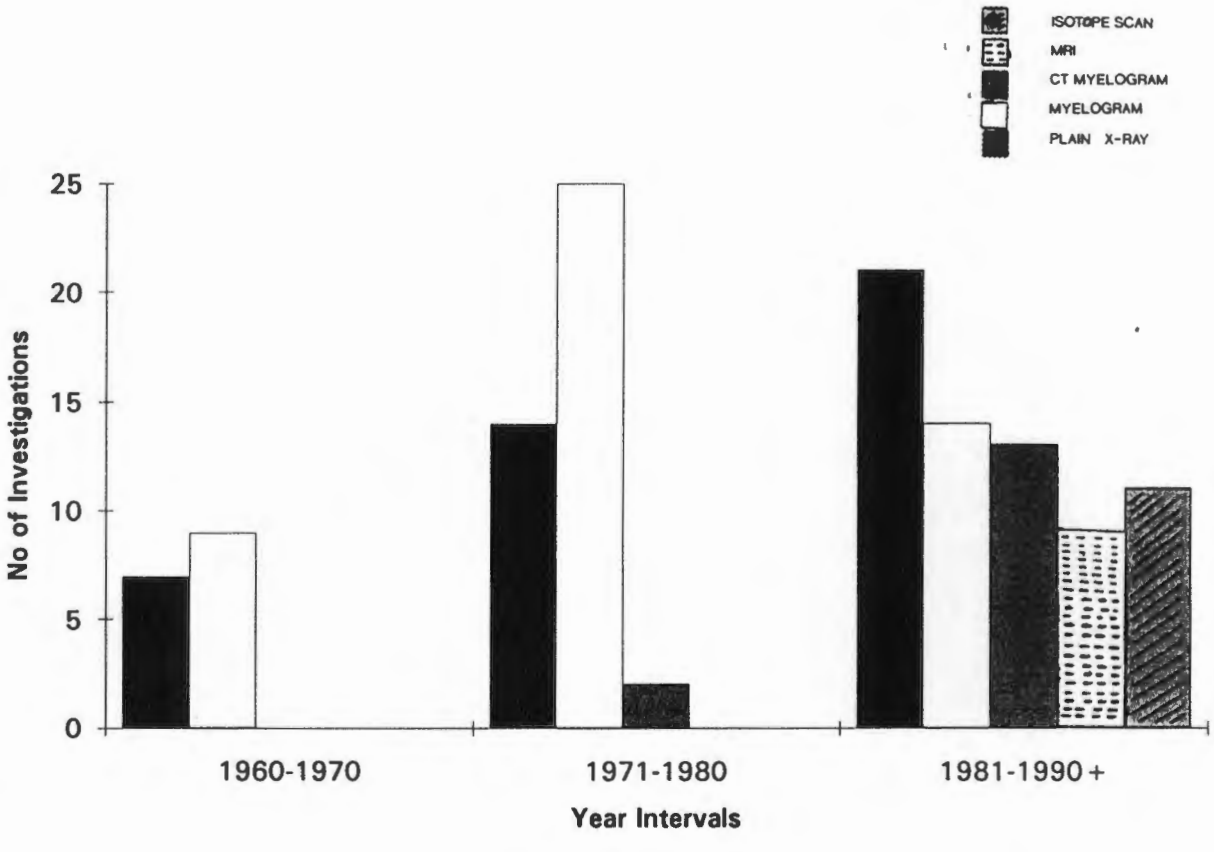


Fig 10: *Radiological investigations.*

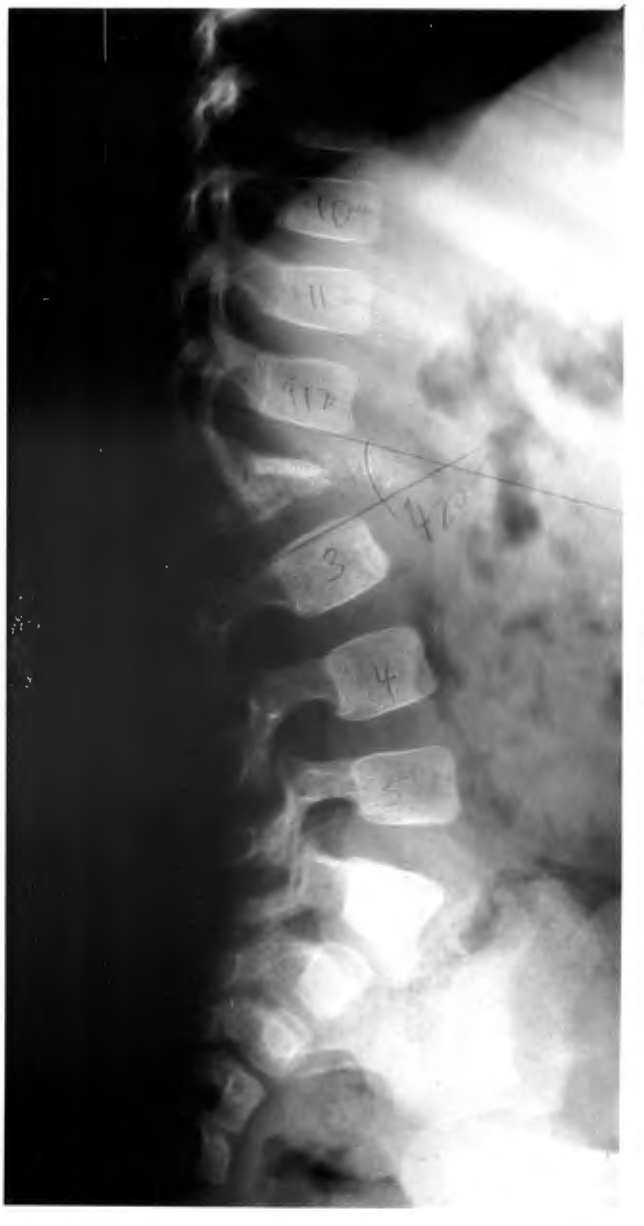


Fig 11: *Plain x-rays of the thoracolumbosacral spine in an eight-year-old child, showing destruction of L1 and L2 bodies with an acute kyphus. The lesion was due to tuberculosis.*

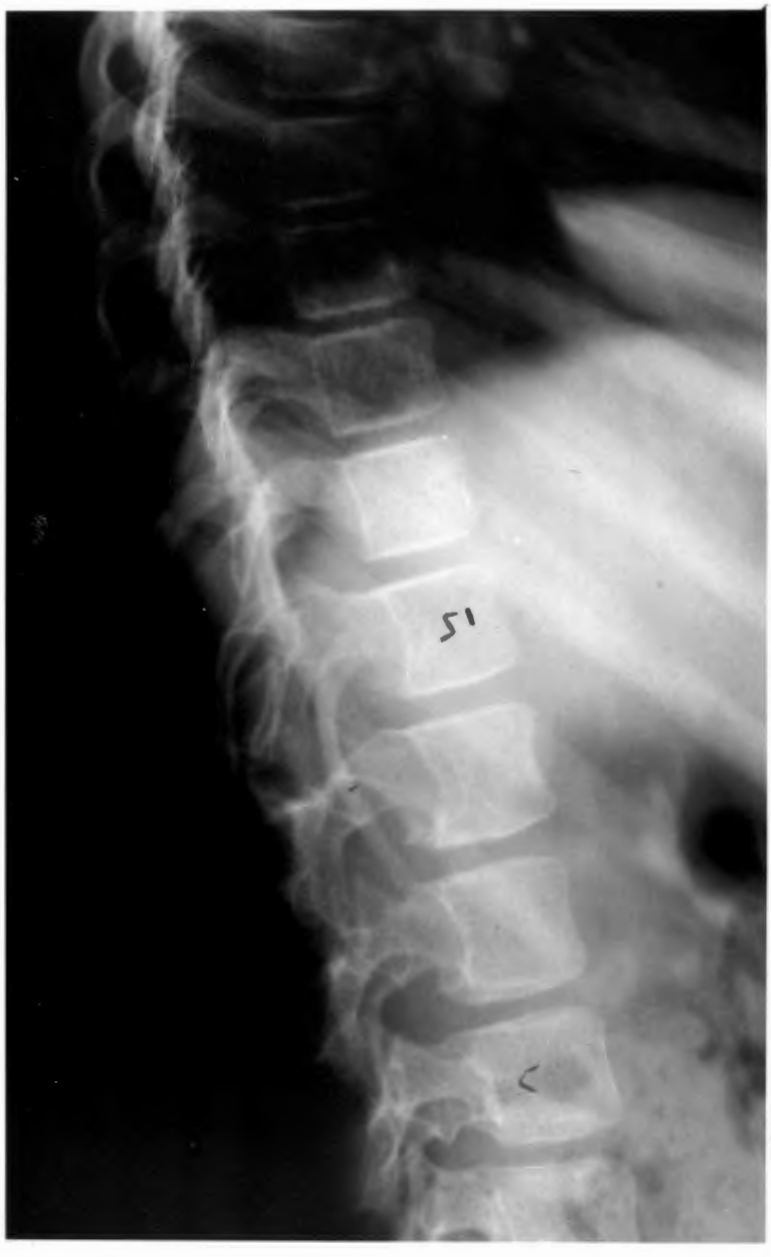


Fig 12: *Plain x-rays show scalloping of the posterior aspect of the bodies of T12-L2 by an intraspinal ganglioneuroma.*

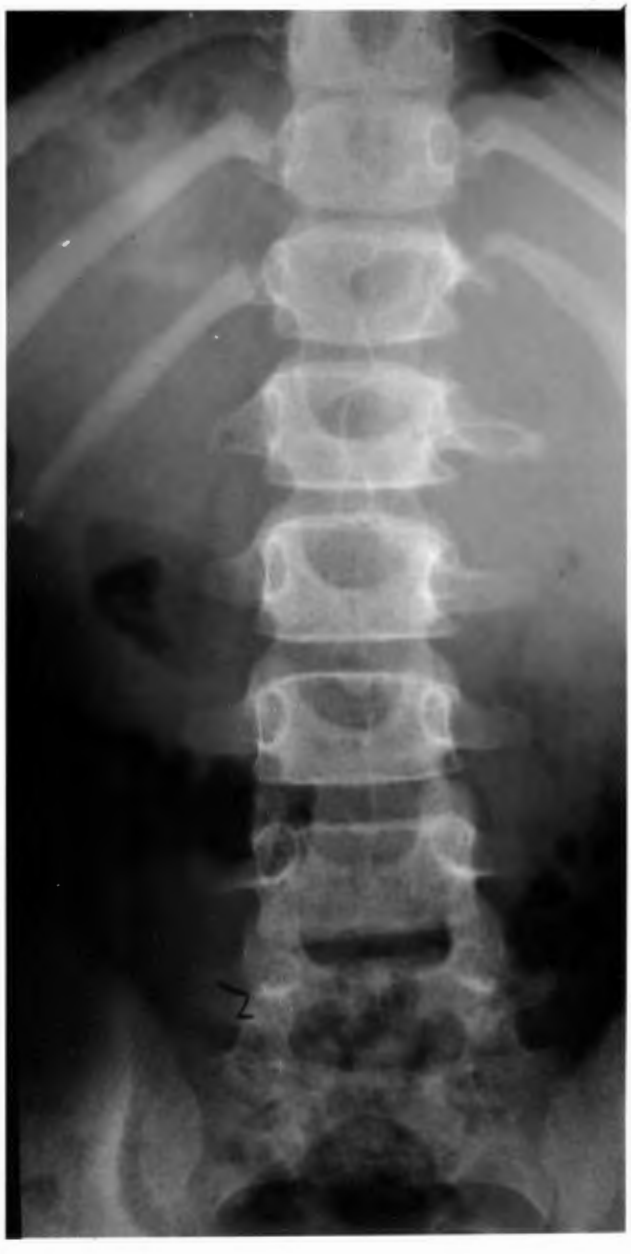


Fig 13: *Plain x-ray of the spine showing erosion of pedicles at L1 and L2 due to a ganglioneuroma. (Same patient as in Fig 12).*

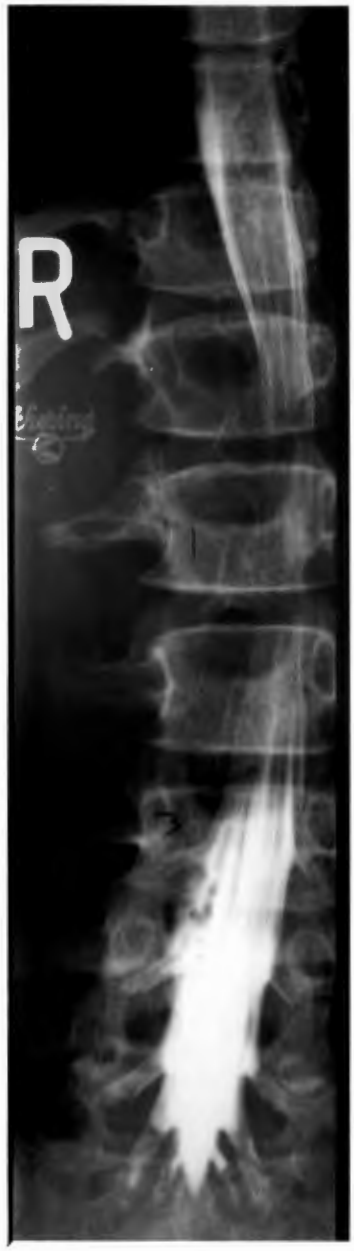


Fig 14: *A soluble contrast lumbar myelogram shows a partial extradural block on the right, due to neuroblastoma direct extension.*

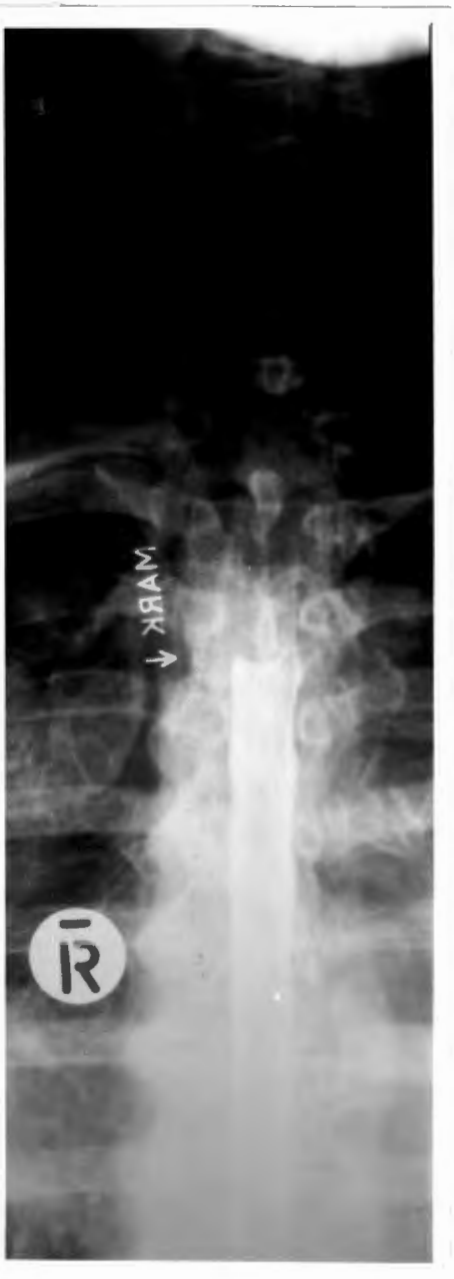


Fig 15: *A water soluble contrast myelogram showing an intradural extramedullary block due to a meningioma in a 14-year-old child.*

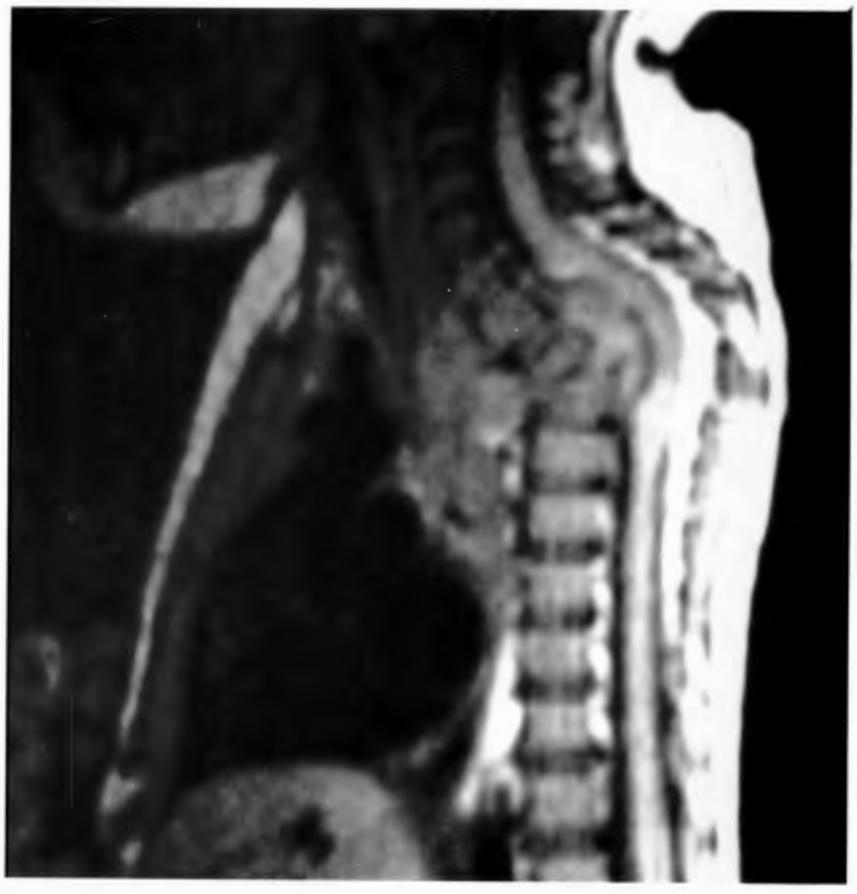


Fig 16: *Sagittal Magnetic Resonance Imaging showing multiple vertebral body destruction in the upper thoracic region due to tuberculosis, with an extradural soft tissue collection pressing on the cord anteriorly. Note the kyphus and curvature in the cord.*

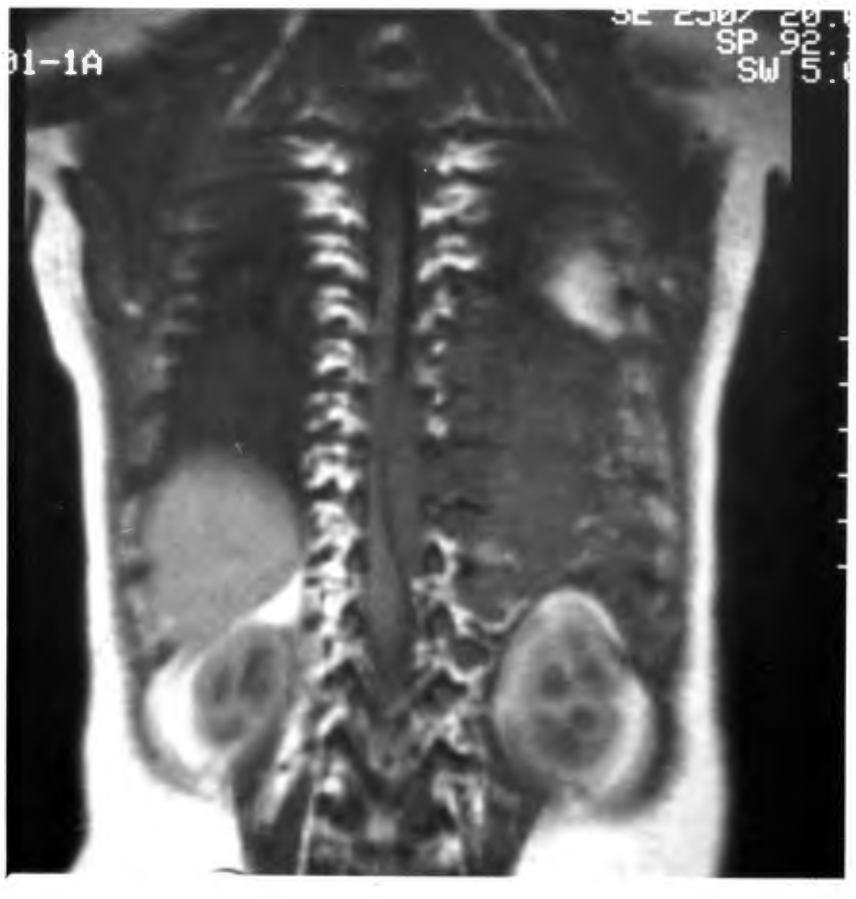


Fig 17: *Coronal Magnetic Resonance Imaging of the thoracolumbar region, showing a left ganglio-neuroma compressing the cord.*

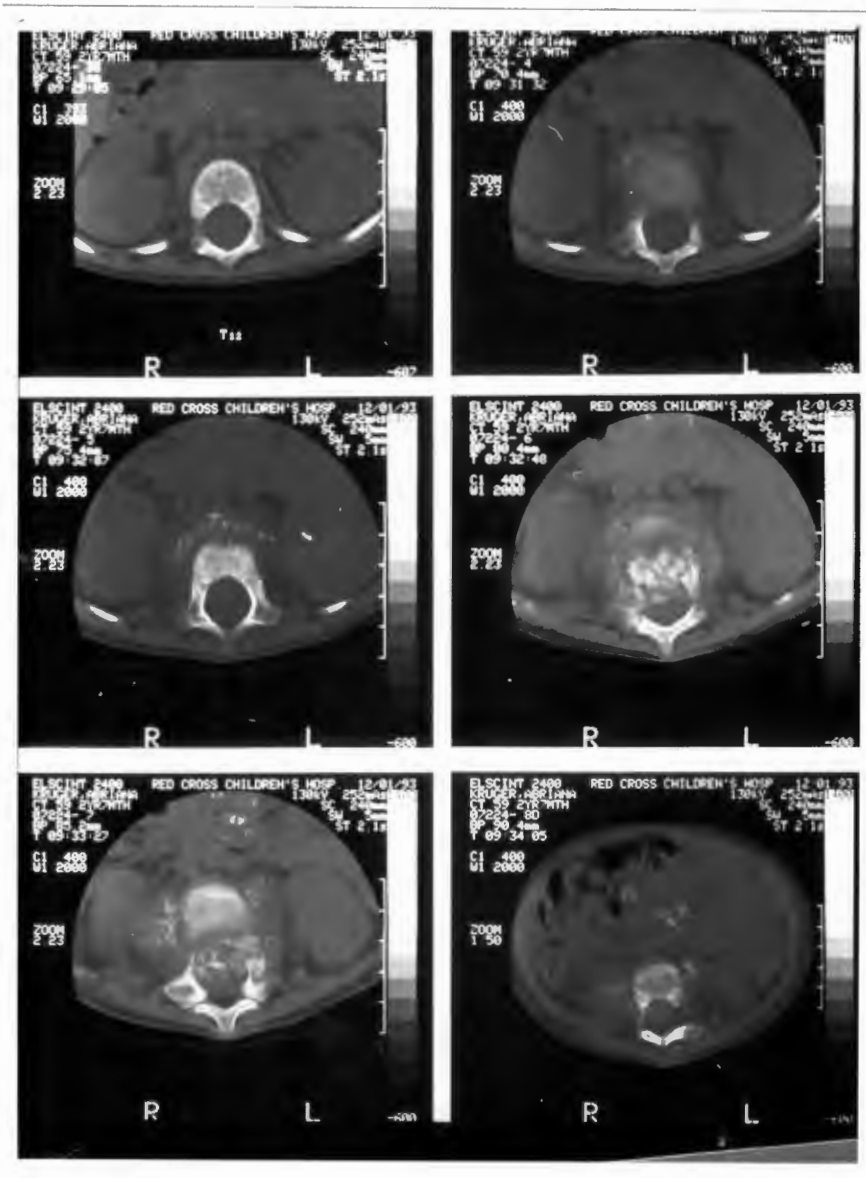


Fig 18: *Plain axial Computerised Tomography of the thoracolumbar region, showing destruction of the bodies of T12 and L1 by tuberculosis.*

2.3.6 Treatment

The surgical procedures are summarised in Table 4.

Table 4 **Surgical Procedures**

Laminectomy + Extradural tumour excision/Decompression	29
Laminectomy + Intradural extramedullary tumour excision	11
Laminectomy + Myelotomy and tumour excision	2
Laminectomy + Myelotomy and tumour biopsy/aspiration	11
Laminectomy for epidural abscess	12
Corpectomy and anterior fusion	7
Costotransversectomy	10
Posterior spinal fusion	2
Microsurgery	4

Infections

Tuberculosis: Initially conservative even when a neurological deficit was present.

Patients received a nine-month course of Rifampicin 10 mg/kg bodyweight/day, Isoniazid 15 mg/kg bodyweight/day and Pyrazinamide 30 mg/kg bodyweight/day. Because of a neurological deficit, patients were kept on bed rest until they recovered or were operated on.

Of the 22 children with tuberculosis, 11 were operated on at intervals of two to eight months after initiating anti-tuberculosis therapy. The indications were either failure to improve or neurological deterioration.

Six children with destroyed vertebral bodies underwent anterior fusion, using autologous fibula strut grafts. Two of these underwent posterior spinal fusion as well.

Epidural Abscess

All 12 children underwent emergency laminectomy as soon as the diagnosis was made, usually within three to seven days of admission. Soft rubber catheters were passed in the epidural space to retrieve loculated collections of pus. All patients received antibiotics.

Parasitic

All four patients who had hydatid cysts underwent laminectomy and excision of the extradural cysts. These children were seen before 1980 and no chemotherapeutic agent was available for treating hydatid.

Tumours

Intramedullary

All were approached via a laminectomy.

Two patients with ependymoma had complete removal of their tumours following a dorsal midline myelotomy and were not subjected to radiotherapy. Three had partial resection and one was only biopsied. The astrocytomas were partially resected and were all subjected to post-op radiotherapy, except one patient who died in the immediate post-operative period.

Intradural Extramedullary

All were approached via laminectomy and were completely excised.

Benign Extradural Masses

The 11 patients had the tumours completely excised via laminectomies.

Malignant Extradural Neoplasms

Thirteen patients underwent laminectomy and decompression of the spinal cord. One patient with undifferentiated non-lymphoblastic lymphoma, underwent an anterior decompression at T9. One patient underwent a needle biopsy at L2 to confirm a diagnosis of histiocytosis X. He was then treated with Prednisone and Vinblastine. The 10 patients with neuroblastoma received one of the three regimens of chemotherapy: Vincristine + cyclophosphamide + Adriamycin; Endoxan + Adriamycin; Vincristine + Adriamycin.

2.3.7 Adjuvant Therapy

Table 5 **Adjuvant Therapy**

Type	No of cases
Chemotherapy	15
Radiotherapy	11
Chemo- and Radiotherapy	5
Anti TB	22
Antibiotic	14

2.3.8 Complications of Treatment (See Table 6)

Table 6: **Treatment Complications**

Treatment Modality	Complication and No. of Cases	
Chemo	Pharyngitis, Pyrexia	(3)
Chemo	Hypokalaemia	(1)
Chemo- and Radiotherapy	Immunosuppression with recurrent Salmonella, Giardia, resistant staph infections	(1)
Surgery	Post-op wound sepsis	(1)
	Wound dehiscence	(1)
	Temporary foot drop	(1)
	Temporary sphincter impairment	(1)
	Temporary neurogenic respiratory depression	(1)
	Meningo ventriculitis (fatal)	(1)

2.3.9 Outcome

Table 7: **Outcome at 18 Months According to Diagnostic Type**

Diagnostic Type	TB Spine	Acute Spinal Epi. Abscess	Hydatid	Intramedul. Tumour	Intradural Extramedul. Tumour	Benign Extradural Tumour	Malignant Extradural Neoplasm	Other	Total
Outcome									
Full recovery	11	7	3	2	4	4	2	-	33
Improved	7	2	-	4	2	4	4	2	25
No change	3	3	1	-	2	2	3	2	16
Deteriorated	1	-	-	2	-	-	2	-	5
Died	-	-	-	4	1	-	6	-	11
Lost to follow-up	-	-	-	-	-	1	4	2	7
TOTAL	22	12	4	12	9	11	21	6	97

TB: = *Tuberculosis*
Epi: = *Epidural*
Intramedul = *Intramedullary*
Extramedul = *Extramedullary*

CHAPTER 3

DISCUSSION

3.1 Demography

There was no significant difference in the Male:Female ratio, either for infective or neoplastic causes. Tachdjian and Matson (1965) found a Male:Female ratio of 2:1 for intraspinal tumours. They also noted that over 50% in their series occurred in the one to four-year age group, with the largest proportion of tumours being of developmental origin. This was also the finding by Hendrick (1984). In this series, the mean age for neoplastic lesions was eight years (range six months to 14 years). The reasons for tumours to present mainly in the older child in this environment are not clear, but it may be that the symptoms and signs in the younger child were attributed to other neurological disorders. (See 3.4).

The findings of this study concur with the observations of Hoffman, Crosier and Cremin (1993) that in this environment, spinal tuberculosis mainly afflicts children under five years.

3.2 Aetiology

a. Neoplasms

In this series, neoplasia was the commonest cause of spinal cord compression in childhood. This has also been the finding by other workers (Craig & Mitchel, 1931; Coxe, 1961; de Sousa et al, 1979; Hoffman, 1980). The significance of neoplasms as a cause of spinal compression will become even greater in Africa as the conditions of living (socioeconomic, cultural, political) improve, resulting in a larger childhood population with a declining incidence of preventable diseases. The cause of neoplasia is believed to be an interaction between genetic and environmental factors such as oncogenic viruses, diet, irradiation and carcinogenic chemicals (Schmidek, 1987).

b. Infections

Tuberculosis still poses a major problem in communities with poor socioeconomic standards. Spinal tuberculosis is rare as compared to the more common pulmonary type (Küstner, 1991). Observations in the developed regions of the world have shown a direct relationship between improved living conditions and a decline in the incidence of tuberculosis (Stead & Bates, 1980). This is also true in the Western Cape, a region

which has the highest incidence of tuberculosis in South Africa (Küstner, 1991). None of the TB sufferers came from an affluent home. The increased incidence in the number of spinal tuberculosis cases over the decades, may reflect the increase in the impoverished population in the Western Cape region, partly due to an influx of job-seekers and partly due to an increased birthrate. It may also reflect improved diagnostic methods and increased physician awareness.

c. Hydatid Disease

Spinal ecchynococcosis (four cases in all) is also preventable by improving personal hygiene and slaughtering methods.

3.3 Clinical Features

Symptoms

Weakness

The majority of children presented with lower extremity weakness, usually of a significant degree. It is important for parents and health workers to recognise that children can adapt to minor degrees of weakness of a limb and walk with a limp, or perform acts with a weak upper limb

in a clumsy fashion. These features should cause one to consider spinal compression until disproved.

Pain

Backache must be regarded as a serious signal in all children (Hoffman, 1980). It might be related to extremes of spinal movement, especially when the child is playing, and may disappear completely when the child is at rest, as was the case in the child with a neurofibroma.

Continuous backache was a presenting feature in most children with spinal epidural abscess, but this was in association with a pyrexia. Backache following a trivial injury should be viewed with suspicion. In such a case, the injury simply draws attention to an underlying pathological process.

It is interesting to note that only two out of 21 children with malignant extradural neoplasms presented with pain. This is unlike in the adult patient, where pain is usually the first symptom of spinal metastasis. This may be due to the fact that all neuroblastomas were direct extension into the vertebral canal without bony deposits and bony destruction.

Sphincter Dysfunction

As a symptom, sphincter dysfunction is difficult to evaluate in the under five-year age group as this is a period of toilet training. However, a child that regresses without a precipitating illness, should be carefully evaluated for the possibility of spinal compression.

Signs

Intrinsic Hand-muscle Atrophy

This is a well recognised phenomena in patients with foramen magnum and upper cervical cord lesions, including cervical syringomyelia, conditions which do not directly involve the T1 segment. It was noted in two of our patients with cervical intramedullary astrocytomas.

It is thought that these pathological conditions result in compromise of the anterior spinal artery leading to cord ischaemia (Symonds & Meadows, 1937).

Similarly, lesions in the thoracolumbar region may compress the artery of Adamkiewicz, giving rise to cord signs above or below the lesion (Findlay, 1987).

Babinski's Sign (Upgoing plantar response)

This is a normal phenomena in children under two years of age, otherwise it is a reliable indicator of an upper motor neurone lesion.

Flaccidity/Spasticity

When symmetrical and involving the lower extremities, flaccidity might be a feature of spinal shock and will give way to spasticity after three weeks or so, but in four of our patients this led to an erroneous diagnosis of the Guillain-Barré Syndrome or Poliomyelitis.

Spasticity implies an upper motor neurone lesion.

Musculoskeletal Features

a. **Scoliosis**

The differential diagnosis of scoliosis may include neuropathic, myopathic, dysembryoplastic, traumatic, degenerative and idiopathic disorders.

Neuropathic scoliosis results either from paraspinal muscle spasm or atrophy, the spinal concavity being ipsilateral to the former and contralateral to the latter, and the spinal lesion being ipsilateral to the muscular abnormality.

In this series there were eight patients who had neuropathic scoliosis as one of the main presenting features. One child presented with painful torticollis. Apart from one case with neuroblastoma where, at the site of the scoliosis, the T1 transverse process was eroded and there was local tenderness, the rest had primary intramedullary neoplasms. One of them was referred from a secondary level institution with flaccid paraparesis and a provisional diagnosis of TB spine. Following a lumbar puncture, the patient developed a flaccid quadriparesis and the diagnosis of ascending polyneuritis was made. This patient required ventilation in the Intensive Care Unit. The diagnostic work-up was only accomplished four months later. This child had a holocord astrocytoma. The child with a painful torticollis had a negative myelogram and normal CSF and was then treated with a cervical brace, followed by traction in extension and improved. A year later, this child presented with a spastic quadriparesis and a C2-T1 astrocytoma was diagnosed.

b. **Pes Cavus**

This is due to imbalance in the power of the intrinsic foot muscles, as a result of denervation of muscles of a functional group. This denervation is due to selective destruction of anterior horn cells of the L4 to S2 spinal cord segments by an intramedullary lesion such as a glioma, or by pressure on the cauda equina by an intradural mass.

In this series, two patients presented with foot abnormalities. One patient with a cauda equina ependymoma had slight atrophy of the left leg and a left pes cavus. This patient was initially diagnosed as a case of muscular dystrophy, but a muscle biopsy was normal and polio antigens were negative. While in a rehabilitation centre, he deteriorated and was brought back to hospital for further investigation. There was a six-month's delay in arriving at the diagnosis. Another child was readmitted at the age of eight years with a T11-L3 ganglioneuroma. At an earlier age, a diagnosis of poliomyelitis had been made and he had had corrective surgery for the equinovarus deformity. The delay in this case was over three years.

These signs can sometimes lead to misdiagnosis and cause patients to be treated for long periods for idiopathic orthopaedic abnormalities. It is clear in this neurosurgical series that in the majority of cases, scoliosis pointed to an underlying primary intramedullary cord tumour, and this must be excluded.

Sensory examination requires maximum patient cooperation and this is very difficult to obtain in children, especially when assessing dorsal column function. Gross spinothalamic abnormalities with a sensory level are, however, readily ascertainable.

Neurocutaneous abnormalities may be found in phakomatoses. Neonates with lumbosacral skin conditions such as naevi, hypertrichosis, skin appendages, dimples or clefts, need to be scanned with Magnetic Resonance Imaging at three months for early diagnosis of occult dysraphic conditions. These skin abnormalities must not simply be dismissed as birthmarks.

3.4 Diagnosis

The differential diagnosis of spinal compression is protean. Several erroneous initial diagnoses were entertained before the final diagnosis of cord compression was made. This obviously led to delay in performing the relevant investigations and in initiating treatment. Poliomyelitis, Guillain-Barré's Syndrome, idiopathic scoliosis, back injury, acute abdomen and meningitis were the major pitfalls, in descending order of frequency.

A high index of suspicion following a careful history and a thorough neurological examination, should lead to timely neuroradiological investigation and neurosurgical treatment, before irreversible compression has occurred.

Plain X-rays should include the entire spine in order not to miss skip lesions and the chest to assess lung lesions such as tuberculosis. Individual vertebrae should be carefully examined for complete destruction, scalloping by adjacent tumours, for widening of intervertebral foramina by a neurofibroma. The pedicles should be examined for medial scalloping implying an intraspinal mass or for widening of the interpedicular distance, implying an intramedullary lesion of long standing. (See Figures 13-15). Loss of pedicles, a feature common in adult spinal metastases, was observed in only one of these children.

Loss of the intervertebral disc height is an early feature in spinal tuberculosis and may be associated with paravertebral soft tissue mass in 30-60% of cases (Kemp, 1976; Allen, Cosgrove & Millard, 1978). It is due to herniation of the disc via the damaged end plate into the affected body. Because of the relative lack of bone repair in tuberculosis, sclerosis of the vertebral margins is more suggestive of pyogenic infection (Kemp, 1976; Allen, Cosgrove & Millard, 1978).

Plain x-rays were adequate for making a diagnosis of TB spine. In the pre-CT and MRI era, surgery for TB spine was carried out on the basis of the plain x-ray features. However, in this era, those patients needing surgery are assessed with CT and MRI. CT gives clarity regarding the status of the pedicles, facets and laminae (Hoffman, Crosier & Cremin, 1993). MRI helps to indicate whether the compressing mass is pus or fibrous tissue and may show a high cord signal which may mean oedema, demyelination or infarction (Corr, Handler & Davey, 1991; Cremin, Jamieson & Hoffman, 1993).

Magnetic Resonance Imaging (MRI) has emerged as the best modality for imaging spinal lesions so far. It gives excellent anatomical definition in multiplanar views and is useful for planning surgery (Hoffman, Crosier & Cremin, 1993). It is non-invasive and uses radio energy which is nonionising and the entire spine can be imaged.

Its major drawbacks at the moment in the Western Cape are the very high cost, lack of availability in academic and major provincial hospitals and inability to image ventilated patients. At Groote Schuur and the Red Cross War Memorial Children's Hospitals, for instance, imaging can only be done on a quota basis at the Medical Research Council unit once a week, and only during working hours. Thus only 10 non-emergency patients can slot into this arrangement.

Myelography using water soluble contrast medium is readily available in most hospitals in the Cape Province, even at night. The procedure is invasive because of the need for lumbar and/or cisternal puncturing and may require some uncooperative children to be heavily sedated or anaesthetised. The myelogram can clearly indicate whether the lesion is extra- or intradural. However, it is at times not clear from the myelogram if the lesion is anterior or posterior to the cord, especially when a complete myelographic block is encountered.

In this series, the main diagnostic drawbacks using myelography, were during the investigation of intramedullary tumours. In two cases it failed to detect possibly early intramedullary neoplasms, causing a diagnostic delay of one year and six months respectively, and in four children there was difficulty in determining the extent of the tumour. One child had a complete myelographic block at T10. Because a cisternal puncture could not yield any fluid, a ventriculogram had to be performed which demonstrated a block at the fourth ventricle with gross dilatation of the rostral part of the ventricle. This led to craniocervical decompression as the initial procedure and a month later to a thoraco-lumbar laminectomy and decompression of the tumour. This child died four months later and a postmortem revealed an astrocytoma extending from the fourth ventricle to the conus medullaris (Figures 19 and 20). This is a case where Magnetic Resonance Imaging would have indicated the extent of the cord involvement and have avoided such invasive diagnostic procedures in this already unfortunate patient.

Fig 19: *A specimen showing the hindbrain and the spinal cord of an eight-year-old child who had a holocordal astrocytoma which also involved the medulla and obstructed the caudal part of the fourth ventricle. Note the diffuse enlargement of the cord. The terminal portion of the cord shows a dorsal mass which was at the site of surgical exploration via a thoracolumbar laminectomy done four months prior to the patient's death. The rostral cervical cord shows a dorsal cystic enlargement, which was at the site of the craniocervical decompression five months before death occurred.*

Fig 20: *Cross-section of the specimen shown in Fig 19 at the cervical level showing diffuse infiltration of the cord by tumour and necrosis in the centre of the cord.*

Another child who had had a tumour removed from the upper thoracic spine five years before, presented with a triparesis with wasting of the intrinsic muscles of the affected hand. The myelogram showed an intramedullary lesion extending from T9-T11 and he had a laminectomy at these levels and decompression of the intramedullary mass. It is quite likely that this tumour extended into the cervical cord to cause left upper limb weakness, but the myelogram did not show this.

Fourteen percent of patients with a complete block who undergo myelography can develop a rapid deterioration in the neurological deficit (Hollis et al, 1986). This necessitates immediate decompression, thus the procedure of myelography in patients with deficits should be undertaken after discussion with a neurosurgeon.

This deterioration may be related to cerebral spinal pressure shifts following lumbar puncture below the mass lesion (Hollis et al, 1986). Three of our children deteriorated within a few hours following lumbar puncture, although one was not related to myelography. All of them had intramedullary masses involving the cervical cord. All needed ventilation and one had to have a tracheostomy. This observation differs from the report by Hollis et al (1986), where the deterioration occurred 24 hours after myelography in six adults with epidural masses and one with an intradural extramedullary mass.

Computerised Tomography (CT) following myelography (CT Myelography) is useful in defining the anatomical relationship of the lesion in the antero-posterior and transverse directions, thus aiding in surgical planning. It also defines better the extent of bone destruction. The usefulness of plain CT scanning in children for assessing intraspinal lesions, is questionable.

3.5 Treatment

Infections

Tuberculosis: The presence of a neurological deficit is not an indication for immediate decompression in patients with spinal tuberculosis. (Kemp, 1976; Fowles, 1979; Pattison, 1986; Hoffman, Crosier & Cremin, 1993). Patients who are on anti-tuberculosis therapy can be monitored for long periods and in the majority of cases, improvement will take place. The indication for surgery is failure to improve or neurological deterioration while on therapy and may be due to a retropulsed sequestrum compressing the cord, a severe kyphus or sometimes vascular compromise. Pattison (1986) reported on 89 consecutive cases of Pott's paraplegia treated in Korea. Only four of these required costotransversectomy in the latter stages of treatment, and of these, three did not benefit. In our series, of the 11 operated cases, four had a full recovery, five improved and two were unchanged.

Acute spinal epidural abscess is a neurosurgical emergency and requires immediate decompression. (Johnston, 1992). A high index of suspicion is essential to make a diagnosis of this relatively rare condition. The key factors are backache, neck stiffness, local spinal tenderness and pyrexia. A child with this tetrad, should be

subjected to MRI of the spine; if not available, to myelography plus CT. The critical period seems to be the interval between the onset of neurological signs and surgical decompression. Children that were operated on within hours of onset of a neurological deficit, had a better outcome than those operated on several days later (Peter, Kieck, de Villiers, 1992).

Neoplasms

Intramedullary Tumours

Epstein and Epstein (1981) have highlighted the fact that intramedullary spinal cord gliomas are invariably multisegmental and occasionally they may be holocordal. They estimated that in children, 60% of the lesions are astrocytomas and 20-30% ependymomas. This ratio is reversed in adults. Ependymomas and some low-grade astrocytomas, lend themselves well to radical excision because a plane of cleavage can be identified after performing a dorsal midline myelotomy (Greenwood 1967; Epstein & Epstein, 1981; Stein, 1983). This should be done with the aid of a microscope, as it not only improves vision through magnification and good lighting, but it also minimises injury to tissue and hence reduces operative morbidity (Greenwood, 1967; Wilson & Harbaugh, 1981; Epstein & Epstein, 1981; Rhoton Jr, 1990; Cristante & Herrmann, 1994). A frozen section diagnosis

helps the surgeon to know the biology of the lesion before attempting gross total removal.

Epstein and Epstein (1981) also concluded that gross total resection was possible in some cases of low-grade astrocytomas and that it was compatible with neurological recovery. This followed treatment of two patients with holocord astrocytomas. In this series, complete removal was achieved in two patients with ependymomas. These patients showed marked improvement in the neurological function post-operatively.

One child had partial resection of a cervical ependymoma, followed by radiotherapy in 1964, but presented in 1971 with a thoracolumbar ependymoma which was excised. He presented again in 1972 with a midthoracic lesion and this time he was moribund. This case shows the usefulness of MRI in evaluating intramedullary lesions, both for surgical planning and follow-up. One patient with ependymoma of the cervical region had biopsy only, followed by radiotherapy and had marked improvement in the right hemiparesis. Of the seven patients with astrocytomas, three had cyst drainage only and four had biopsy only. All had radiotherapy. Four of these died within six months. Obviously the approach to these tumours at that time was very conservative. This was based on the old general view that complete resection of an intramedullary lesion would

inevitably result in damage to the spinal cord and worsening of the neurological deficit. But in view of the report by Epstein and Epstein (1981), one would recommend an attempt at radical excision of these tumours in patients that can tolerate the procedure and have low-grade lesions, with a clear plane of cleavage.

Cywes (1992) has underscored the need for CT or MRI of the spine in children with thoracic neuroblastoma to look for intraspinal extension. He recommends that even if there is no neurological deficit, laminectomy and removal of the intraspinal component be done prior to removal of the mediastinal component.

Radiotherapy has a potential complication of radionecrosis of the spinal cord and should be reserved for malignant neoplasms (Kramer, 1968; Burns et al, 1972). Ependymomas and benign astrocytomas, if only partially resected, should rather be re-operated than irradiated (Stein, 1983).

General

Extensive laminectomy (≥ 3 levels) in a growing child, can lead to several long-term complications. In the cervical region, a swan-neck deformity can result and in all regions a kypho-scoliosis can occur (Lonstein et al, 1976). Instability can develop where bilateral facetectomy has been done (Yasuoka et al, 1981). This needs to be avoided whenever possible and if facet damage is noticed, the child must be fitted with a brace in the immediate post-operative period and referred to the orthopaedic surgeon for follow-up (Tachdjian & Matson, 1965). If an increasing deformity is noted, spinal fusion is indicated. Many workers have recommended laminoplasty as a way of avoiding these complications (Raimondi, 1976; Yasuoka et al, 1981; Cristante & Herrmann, 1994).

3.6 Outcome

Infections

It is evident from this series that infections had a good prognosis. As indicated in Table 7 (p.57), there was no mortality and permanent disability seems to be related to the interval between the onset of neurological deficit and initiation of treatment. In cases of spinal epidural

abscess, decompression should be done within 24 hours, but the earlier the better.

Neoplasms

Benign extradural and extramedullary neoplasms had a good prognosis. There was one death due to meningoventriculitis as a complication of laminectomy and excision of a neurofibroma from the cervical region.

The patients with extradural ganglioneuromas did extremely well, whereas one patient with an extradural sacral chordoma and one with a midthoracic harmatoma did not improve following tumour excision.

Malignant extradural neoplasms evidently had a poor prognosis in spite of a combination of surgery, chemo- and radiotherapy. Patients with neuroblastoma who were less than three years of age, faired better than older patients. Those that recovered fully, were both 10 months of age and the two that died were seven and nine years. One patient with a neuroblastoma diagnosed and treated with surgery and chemotherapy at the age of two years, had regression of the lesion into a ganglioneuroma one year later.

Intramedullary tumours had a poor prognosis. The four deaths were all due to astrocytoma. Patients with ependymoma faired better, with two fully recovering and two improving. Radiotherapy caused improvement in the hemiparesis of one patient with a cervical ependymoma who had only had a biopsy procedure.

CHAPTER 4

CONCLUSIONS

a. Neoplasms are the commonest cause of spinal compression (40%) followed by infections (30%).

b. Of the extradural group, tuberculosis of the spine is the commonest cause (29%) followed by malignant extradural neoplasms (23%).

c. Although neuro-imaging studies are essential in arriving at a diagnosis and planning the surgical approach, clinical suspicion by the primary doctor is the most important factor in preventing delay in appropriate management.

d. An attempt should be made at radical excision of certain intramedullary tumours such as ependymomas and some astrocytomas with a clear plane of cleavage visualised under the surgical microscope.

e. Chemo- and radiotherapy, in addition to surgery, improves meaningful survival in children with neuroblastoma and some lymphomas.

f. The final outcome depends on:

1. the biology of the lesion; and
2. the interval from onset of neurological signs to adequate spinal decompression.

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