

# **ADULT NEOPLASTIC SPINAL CORD COMPRESSION**

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**A Dissertation in Fulfilment of Part  
Three of the M.Med (Neurosurgery)**

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## CHAPTER 1

### INTRODUCTION

Spinal cord compression ( SCC ) constitutes a neurological emergency, and if left untreated, can result in permanent irreversible neurological dysfunction. Disabilities can range from mild weakness to complete quadriplegia with the inherent associated mental, physical and emotional suffering .The burden of cost to the individual and community is enormous. ( De Villiers )

SCC can be either Acute or Chronic. In Acute compression, the history of paresis is short ( <72hours). (Johnston; Black 1985; Maurice-Williams). A Chronic history implies a more benign course and type of compression and a thorough investigation can be completed in order to arrive at a diagnosis.

Hughes and Langford initially classified spinal tumours based on their anatomical location within the spinal canal as well as their histological type. (Hughes) They maybe either intra- or extradural and the intradural group either extra- or intramedullary. The ratio of intradural to extradural tumours is approximately 3 to 2. The terms 'extradural' and 'epidural' will be used interchangeably to mean the same thing.

Intradural spinal tumours are uncommon with an incidence of from 3 to 10 per 100 000 population (Kurland; Slooff). Of these intradural spinal tumours, about 70 percent are extramedullary (Okazaki) and the most common of these are the nerve sheath tumours ( 30% ), and meningiomas ( 25% ) (Slooff). The predominant intramedullary tumours are astrocytomas and ependymomas with an approximately equal incidence.

Ninety percent of all intradural spinal tumours are benign and potentially resectable. Thus, with early diagnosis and management, the outlook after surgical removal is excellent ( **Mork; Russell**). A recent review of current literature shows more and more neurosurgeons are changing to a more aggressive treatment of intramedullary tumours (**Cristante**)

Epidural tumours are more often malignant and metastatic lesions of the spine represent the large majority. Nearly 20% of patients with neoplastic involvement of the vertebral column develop spinal compression ( **Schaberg**). The origin of metastases cannot be identified in 9% of patients, and an additional 8% present with spinal cord compression as the initial symptom of cancer (**Black 1998; Gilbert**)

Pain is an early symptom regardless of the location of the tumour (**Gilbert; Levy 1986; McCormick 1990A+1990B**). There may be local pain in the vertebral column or radicular pain if a nerve root is involved especially in the epi - and intradural extramedullary tumours. Patients with intramedullary tumours often have a relative sparing of pain and temperature appreciation because of the topographic arrangement of the fibres, unless the tumour is in close association with the dorsal root ganglion. (**Howe**)

Weakness, usually in the lower limbs is common by the time of diagnosis (**Gilbert**) A clinical grading system which allows for assessment and objective interpretation of the clinical signs and functional ability of the patient was introduced by Frankel (**Frankel**). This allows clinicians to prognosticate more objectively the functional outcome of a patient presenting with limb paralysis as a result of spinal cord dysfunction.

Radiological investigations include plain radiographs, isotope bone scans, myelography, computed tomography (CT) and CT myelography (CTM). At present, magnetic resonance imaging ( MRI ) has become the neuroimaging modality of choice for most disorders of the spine and spinal canal. It offers high lesion detection sensitivity, multiplanar imaging, superior soft tissue contrast and the lack of ionising radiation. Some patients may need sedation or anaesthesia to undergo MRI. There are few

contraindications to MRI but large, obese patients, patients with metal implants and patients who suffer from claustrophobia are unsuitable (Johnston; Hoffmann; Edelman)

CT still remains an important diagnostic tool for patients in whom MRI is contraindicated or unavailable especially when combined with myelography (CTM ). CT is superior to MRI in outlining bony anatomy.

Conventional myelography may still be used in clinical practice when MRI is not available or in patients with metal implants, although it has assumed a lesser role in the diagnosis of spinal tumours especially in intradural types. Radioisotope imaging may also be used in aiding the diagnostic work-up, when MRI is not available as it aids in directing CT to the appropriate segments of the spine when diffuse involvement of the spine is suspected.

Victor Horsely is credited with the removal of the first intraspinal tumour (Gowers), and since then, management of spinal tumours has varied according to location of tumour and tumour histology. Advances in technology and surgical techniques have introduced a wide range of options in the modern day management of spinal cord compression.

The assessment and value of these options in our practice at Groote Schuur Hospital is the object of this investigation.

## CHAPTER 2

### ANATOMY AND PATHOPHYSIOLOGY

#### ANATOMY OF THE SPINAL CORD ( Carpenter; Barr) (Figure 1 – Pg 5)

The spinal cord is an elongated, cylindrical mass of nerve tissue that occupies the upper two-thirds of the adult spinal canal within the vertebral column. The cord is usually 42-45 cm long in adults. It extends from the foramen magnum to the lower border of the first lumbar vertebra.

The **conus medullaris** is the conical distal (caudal) end of the spinal cord; the **filum terminale** extends from the tip of the conus and attaches to the distal dural sac. The filum terminale consists of pia and glial fibres; it often contains a vein.

The **central canal** extends the length of the spinal cord during development. It is lined with ependymal cells and filled with cerebrospinal fluid (CSF). It opens upward into the inferior portion of the fourth ventricle in the lower brain stem. In adults, the canal usually disappears except at the cervical level; central canal remnants of ependymal cells are found elsewhere in the cord.

The spinal cord is shorter than the vertebral canal, which causes a discrepancy between spinal cord segments and their analogous vertebral bodies. This discrepancy increases caudally. For example the C8 spinal cord level lies between vertebral bodies C6 and C7. The fourth thoracic segment of the spinal cord is opposite the third thoracic vertebra. The twelfth thoracic cord segment is opposite the T10-11 interspace, and the L5-S1 spinal cord segment lies opposite the L1 vertebral body.

The spinal cord is anchored to some extent by the dentate ligaments and the dorsal and ventral roots. Masses displacing the spinal cord produces stresses within the interior of the spinal cord that may be altered by these tethering structures.

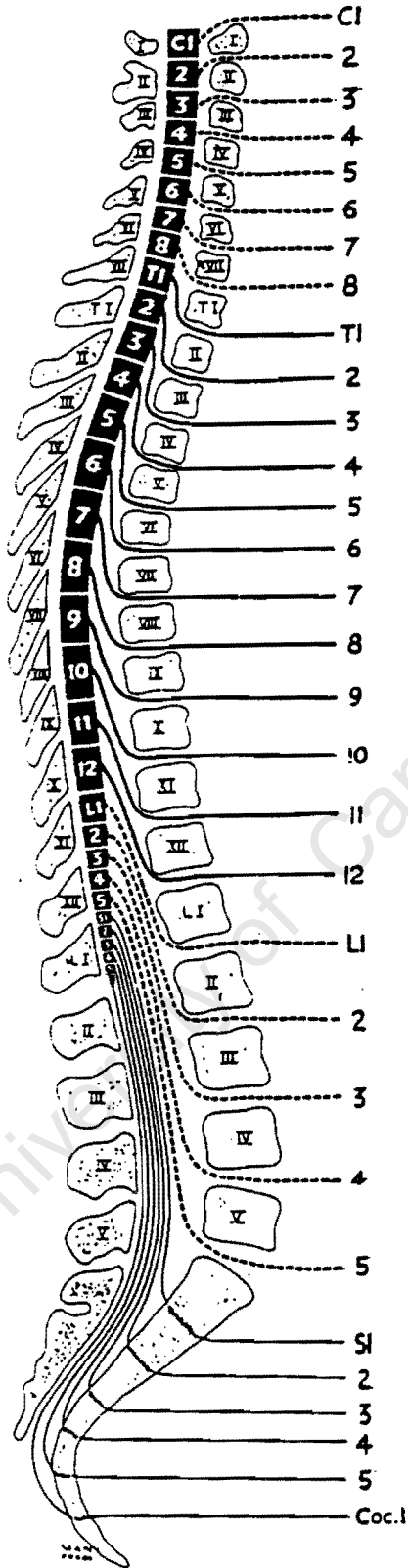


FIGURE 1: SPINAL CORD SEGMENTS

### **Enlargements**

The spinal cord expands in 2 regions: the **cervical** and the **lumbar** enlargement. The latter tapers off to form the conus medullaris. The enlargements of the cord correspond to the origins of the nerves of the upper and lower extremities. The nerves of the brachial plexus originate at the cervical enlargement; the nerves of the lumbosacral plexus arise from the lumbar enlargement.

### **Segments**

While the spinal cord is a continuous unsegmented structure, the 31 pairs of spinal nerves associated with localised regions produce an external segmentation. On the basis of this external segmentation, the spinal cord is considered to consist of 31 segments, each of which receives and furnishes paired dorsal and ventral root filaments. The spinal cord is divided into the following segments: 8 Cervical, 12 Thoracic, 5 Lumbar, 5 Sacral and 1 Coccygeal. At birth the conus medullaris is located near the L3 vertebra; in the adult it is between the L1 and L2 vertebral bodies. The sites of emergence of the spinal nerves do not change, but there is a lengthening of root filaments between the intervertebral foramina and the spinal cord, which is most marked for the lumbar and sacral spinal roots. These roots descend for a considerable distance within the dural sac before reaching their respective intervertebral foramina. Collectively the lumbosacral roots surrounding the filum terminale are known as the **cauda equina**.

### **Longitudinal Divisions**

A cross section of the spinal cord shows a deep anterior **median fissure** and a shallow **posterior median sulcus**, which divide the cord into symmetric right and left halves that are joined in the central midportion. The anterior median fissure contains a fold of pia and blood vessels; its floor is the **anterior white commissure**. The dorsal nerve roots are attached to the spinal cord along a shallow vertical groove, the **posterolateral sulcus**, which lies a short distance anterior to the posterior median sulcus. The ventral nerve roots exit in the **anterolateral sulcus**.

## **SPINAL ROOTS & NERVES**

Each of the 31 pairs of spinal nerves that arise from the spinal cord has a ventral and a dorsal root. In the dorsal root of a typical spinal nerve close to the junction with the ventral root, lies a **dorsal root ( spinal ) ganglion**, a swelling that contains nerve cell bodies. The spinal nerves are divided into groups that correspond to the spinal cord segments.

### **Ventral Root**

The ventral roots carry the large diameter alpha motor neurone axons to the extrafusal striated muscle fibres and the smaller gamma motor neurone axons that supply the intrafusal muscle of the muscle spindles, among others.

### **Dorsal Root**

The dorsal roots contain a variety of sensory fibres which carry pain, temperature, proprioception and touch from cutaneous and deep structures.

## **INTERNAL DIVISIONS OF THE SPINAL CORD**

### **Gray Matter**

**A. Columns:** A cross section of the spinal cord shows an H-shaped internal mass of gray matter surrounded by white matter. The **ventral gray column** lies anterior to the central canal. It contains the cells of origin of the ventral roots. The **intermediolateral gray column (or horn)** is the portion of the gray matter between the dorsal and ventral gray columns, prominent in the thoracic and upper lumbar regions, but not in the midsacral region. It contains preganglionic cells for the autonomic nervous system.

The **dorsal gray column ( posterior )** reaches almost to the posterolateral sulcus. A compact bundle of small fibres, the **dorsolateral fasciculus ( Lissauer's tract )**, part of the pain pathway, lies on the periphery of the spinal cord.

## **B Laminae**

A cross section of the grey matter of the spinal cord shows a number of cellular laminae numbered Lamina I – X This is the most satisfactory way to describe the nuclear groups in the spinal grey matter. The laminae constitute regions with characteristic properties, but their boundaries are zones of transition, where changes may occur either gradually or abruptly.

## **White Matter**

**A. Columns:** Each lateral half of the spinal cord has white columns ( funiculi ) - dorsal, lateral, and ventral - around the spinal grey columns. In the cervical and upper thoracic regions, the dorsal column is divided into a medial portion, the **fasciculus gracilis**, and a lateral portion, the **fasciculus cuneatus**.

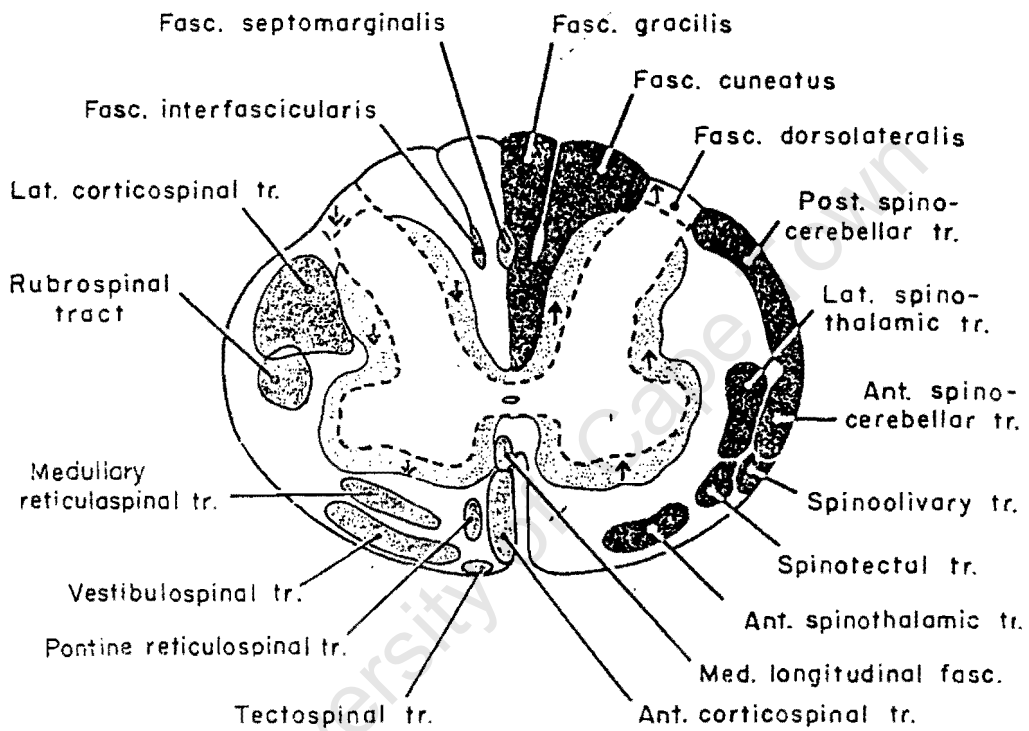
**B. Tracts:** The white matter of the cord is composed of myelinated and unmyelinated nerve fibres. The fast- conducting myelinated fibres form bundles (fasciculi) that ascend or descend for varying distances. Glial cells ( mostly oligodendrocytes ) lie between the fibres. Fibre bundles with a common function are called tracts. The dorsal column tracts are sharply defined by glial septa but the tracts of the lateral and ventral columns are not.

## **PATHWAYS IN WHITE MATTER (Figure 2 – Pg 9)**

### **Descending Fibre Systems**

**A. Corticospinal Tracts:** They originate in the motor cortex of the cerebral hemisphere and terminate on the anterior horn cells of the ventral grey matter and its interneurons. They are located in the lateral and ventral columns of the spinal cord and their function is primarily motor with some modulation of sensation.

**B. Vestibulospinal Tracts:** Their fibres arise from the lateral vestibular nucleus in the brain stem and course downward uncrossed in the ventral column of the white matter of the cord. Fibres of this tract project directly to the motor neurones for extensor muscles. This system is involved in modulating postural reflexes.



**FIGURE 2: SPINAL CORD PATHWAYS**

**C. Rubrospinal Tracts:** Arise in the contralateral red nucleus in the brain stem and course in the lateral white column. They project to interneurons in the spinal grey column and play a role in motor function.

**D. Reticulospinal system:** Arises in the reticular formation of the brain stem and descends in both the ventral and lateral white columns. The fibres of this tract are involved in the modulation of sensory transmissions ( especially pain) and spinal reflexes.

**E. Descending Autonomic System:** Arises from the hypothalamus and brain stem and projects to preganglionic sympathetic neurones in the thoracolumbar spinal cord ( lateral column ) and to preganglionic parasympathetic neurones in sacral segments.

**F. Tectospinal Tracts:** arise from the roof (tectum) of the midbrain, and course in the contralateral ventral white column to end on ventral grey interneurons. They are responsible for head turning in response to sudden visual or auditory stimuli.

**G. Medial Longitudinal Fasciculus:** Arises from vestibular nuclei in the brain stem; some fibres descend into the cervical spinal cord to terminate on ventral grey interneurons to co-ordinate head and eye movements.

#### **Ascending Fibre Systems**

All afferent axons in the dorsal roots have their cell bodies in the dorsal root ganglia.

**A. Dorsal Column Tracts:** These tracts, the **medial lemniscal system**, convey well-localised sensations of fine touch, vibration, 2-point discrimination, and proprioception ( position sense ) from the skin and joints; they ascend, without crossing, in the dorsal white column of the spinal cord to the lower brain stem. The **fasciculus gracilis** and **fasciculus cuneatus** contain fibres arranged in an orderly fashion from medial to lateral; such an arrangement is called **somatotopic organisation**.

**B. Spinothalamic Tracts:** Small-diameter fibres conveying the sensations of sharp (noxious) pain, temperature, and crude touch course upward for one or two segments at the periphery of the dorsal grey horn. These short, ascending stretches of incoming fibres in the dorsolateral fasciculus, or Lissauer's tract, synapse with dorsal column neurones, especially in laminae I, II, and V. After synapsing, the fibres cross to the opposite side of the cord and ascend as the spinothalamic tract or ventrolateral tract to terminate in the contralateral thalamus. These fibres also display a somatotopic arrangement.

**C. Spinoreticular Pathway:** This ill-defined tract which courses within the ventrolateral portion of the cord, arising from cord neurones and ends (uncrossed) in the reticular formation of the brain stem. This tract plays an important role in the sensation of pain, especially deep, chronic pain.

**D. Spinocerebellar Tracts:** Two ascending pathways provide input from the spinal cord to the cerebellum. These dorsal and ventral spinocerebellar tracts convey information about movement and position mechanisms.

### **INVESTING MEMBRANES**

Three membranes surround the spinal cord: The outermost is the dura (dura mater), the next is the arachnoid, and the innermost is the pia (pia mater). The dura is also called the pachymeninx, and the arachnoid and pia are called the leptomeninges.

**Dura**

This is a tough, fibrous, tubular sheath that extends from the foramen magnum to the level of the second sacral vertebra, where it ends as a blind sac. The dura of the spinal cord ( **theca spinalis** ) is continuous with the cranial dura. The epidural, or extradural, space separates the dura from the bony vertebral column; it contains loose areolar tissue and a venous plexus. The subdural space is a narrow space between the dura and the underlying arachnoid.

**Arachnoid**

This is a thin, transparent sheath separated from the underlying pia by the subarachnoid space, which contains CSF.

**Pia**

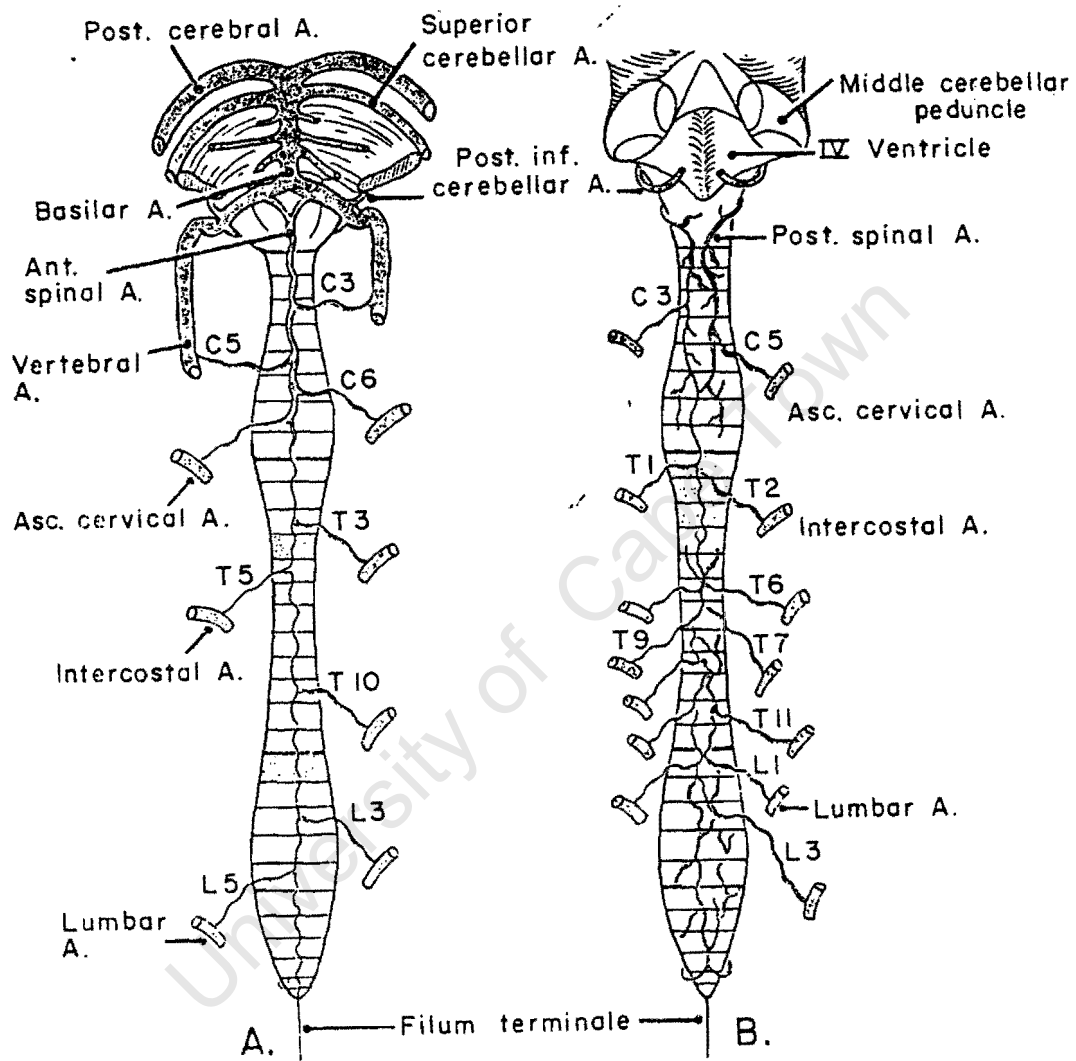
The pia closely surrounds the spinal cord and sends septa into its substance. The pia also contributes to the formation of the **filum terminale internum**, a whitish fibrous filament that extends from the conus medullaris to the tip of the dural sac. The filum is surrounded by the cauda equina, and both are bathed in CSF. Its extradural continuation, the **filum terminale externum**, attaches at the tip of the dural sac and extends to the coccyx. The filum terminale stabilises the cord and dura lengthwise.

**Dentate Ligaments**

The dentate ligaments are a long flange of whitish, mostly pial tissue that run along both lateral margins of the spinal cord, between the dorsal and ventral rootlets. Their medial edge is continuous with the pia at the side of the spinal cord, and its lateral edge pierces the arachnoid at intervals ( 21 on each side ) to attach to the inside of the dura. The dentate ligament helps to stabilise the cord from side to side.

**SPINAL CORD CIRCULATION (Lazorthes) (Figure 3 – Pg 14)**

The arterial blood supply to the spinal cord derives from different sources at different levels. The anterior spinal artery is supplied predominantly by branches of the intracranial portions of the vertebral arteries, and it nourishes the upper cervical spinal cord, assisted by a major radicular artery at C6. In the thoracic region the major arterial supply is at T7, and in the lower spinal cord segments, the predominant supply is via the artery of Adamkiewicz, which may enter the cord anywhere from T8 to L4. Therefore, the intermediate areas of the spinal cord have a watershed blood supply; if the predominant arterial supply related to this is compromised by tumour, the watershed area at some distance from the primary lesion will be the first to suffer. These vascular relationships may explain the necrosis of the central areas of the spinal cord in the lower cervical region at some distance from tumours of the high cervical area.



**FIGURE 3: SPINAL CORD CIRCULATION**

## Arteries

**A. Anterior Spinal Artery:** This artery is formed by the midline union of paired branches of the vertebral arteries. It develops along the ventral surface of the cervical spinal cord, narrowing somewhat near T4.

**B. Anterior Median Spinal Artery:** This is the prolongation of the anterior spinal artery below T4.

**C. Posterolateral Spinal Arteries:** These arise from the vertebral arteries and course downward to the lower cervical and upper thoracic segments.

**D. Radicular Arteries:** Some (but not all) of the intercostal arteries from the aorta supply **segmental (radicular)** branches to the spinal cord, from T1 to L1; the largest of these branches, the **great ventral radicular artery**, also known as the *arteria radicularis magna*, or **artery of Adamkiewicz**, enters the spinal cord between segments T8 and L4. This artery usually arises on the left, and in most individuals, supplies most of the arterial blood supply for the lower half of the spinal cord. Although occlusion in this artery is rare, it might result in major neurologic deficits. Some radicular arteries derived from the lumbar, iliolumbar, and lateral sacral arteries are present in the lumbosacral area. The largest such vessel appears to enter the intervertebral foramen at vertebra L2 to form the lowermost portion of the anterior spinal artery-the terminal artery-which runs along the filum terminale.

**E. Posterior Spinal Arteries:** These paired arteries are much smaller than the single large anterior spinal artery; they branch at various levels to form the posterolateral arterial plexus. The posterior spinal arteries supply the dorsal white columns and the posterior portion of the dorsal grey columns.

**F. Sulcal Arteries:** In each segment, the branches of the radical arteries that enter the intervertebral foramen accompany the dorsal and ventral nerve roots. These branches unite directly with the posterior and anterior spinal arteries to form an irregular ring of arteries with vertical connections. Sulcal arteries branch from the coronal arteries at most levels. Anterior sulcal arteries arise at various levels along the cervical

and thoracic cord within the ventral sulcus; they supply the ventral and lateral columns on either side of the spinal cord.

### **Veins**

An irregular external venous plexus lies in the epidural space; it communicates with segmental veins, basivertebral veins from the vertebral column, the basilar plexus in the cranium, and- by way of the pedicular veins- a smaller internal venous plexus that lies in the subarachnoid space. All venous drainage is ultimately into the venae cavae. Both plexuses extend the length of the cord.

### **PATHOPHYSIOLOGY OF SPINAL CORD COMPRESSION**

A tumour mass located anywhere in the spinal canal, in close proximity to the spinal cord can cause compression of the spinal cord and possible vascular occlusion. Compression can be as a result of the tumour mass itself or as a consequence of the vertebral collapse and subsequent cord compression. The former is more common. Vasogenic oedema is one of the early pathophysiological changes occurring, initially in the white matter, and subsequently involving the grey matter. (Ushio 1977B;McAlhany) The blood supply of the cord gradually, becomes diminished with the onset of venous stasis and subsequent occlusion of the epidural venous plexus. (Ikeda)

Mechanical distortion of the neural tissues is another mechanism resulting in disturbed neurological function. The spinal cord can adjust to slow compression by benign, slow growing tumour or spondylosis; but is unable to tolerate rapid compression by malignant tumour or trauma. The rate of compression by malignant tumour varies widely and it is not uncommon for total paralysis to occur within hours or a few days from the onset of symptoms.

## CHAPTER 3

### NEUROLOGY OF SPINAL CORD COMPRESSION

#### **Patterns of Spinal Compression (Walton)**

The manifestations of SCC at a given level consists of:

#### **Lower motor neurone paralysis**

Atrophic paralysis with diminution or loss of tendon reflexes in the muscles innervated by the affected segments.

#### **Upper Motor Neurone Paralysis**

Spastic paralysis with exaggeration of the tendon reflexes, diminution or loss of the abdominal and cremasteric reflexes and an extensor plantar reflex on one or both sides below the level of the compression.

#### **The Sphincters**

The sphincters are not usually affected in the early stages of SCC, but later precipitancy or hesitancy of micturition develops: later still, urinary retention is common, or the bladder may empty automatically. Constipation is usual, but with severe paraplegia there may be faecal incontinence. Sphincter changes often occur earlier in tumours involving the cauda equina and conus medullaris than when compression occurs at a higher level.

#### **Monoplegia**

Refers to weakness or paralysis of all the muscles of one leg or arm.

#### **Hemiplegia**

Is the commonest form of paralysis involving the arm and leg on one side of the body.

#### **Paraplegia**

Indicates weakness or paralysis of both legs.

**Quadriplegia or Tetraplegia**

Denotes weakness or paralysis of all four extremities.

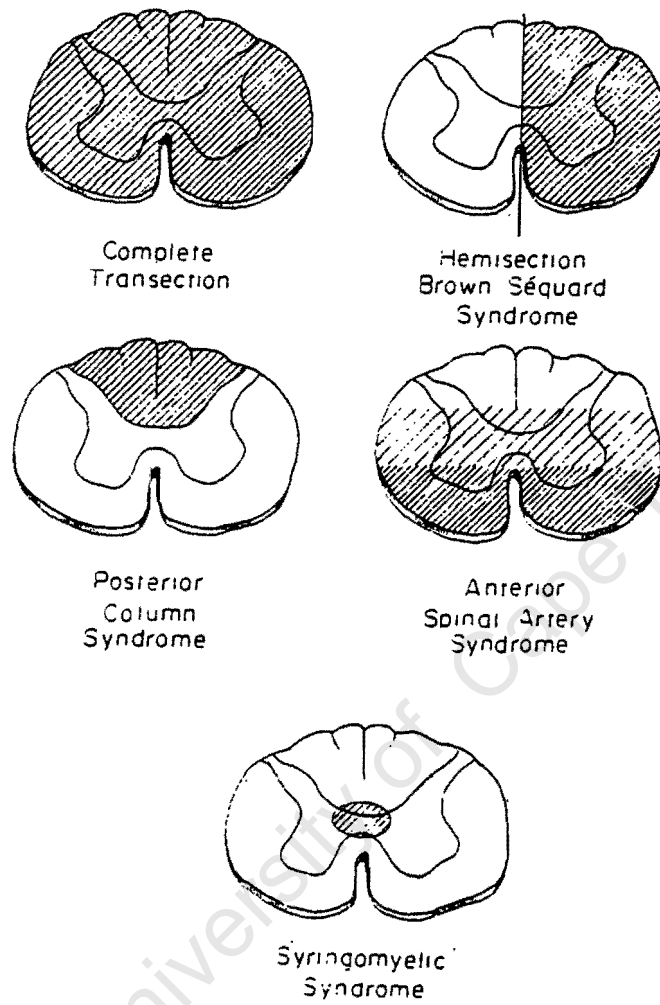
**Spinal Cord Syndromes (Elsberg/Walton)****Fig 4 – Pg 19****Complete Transverse Lesion**

Usually caused by rapid cord compression or cord transection with complete loss of function (sensory, motor and autonomic) below a lesion of the spinal cord.

**Brown - Sequard Syndrome (Hemisection of the spinal cord)**

The essential findings, below the level of the lesion, are as follows :

ipsilateral spastic paresis with increased reflexes and pyramidal tract responses; ipsilateral loss of proprioceptive sensation with sensory ataxia; contralateral loss of pain and temperature sensations extending below the level of the lesion. There may be little or no objective evidence of change in tactile sensation.



**FIGURE 4: SPINAL CORD SYNDROMES**

**Central Cord Syndrome**

Results in a lower motor neurone paralysis at the level of the lesion and upper motor neurone paralysis below the lesion in the cord. There will be loss of pain and temperature appreciation with sparing of tactile sensation because the former fibre tracts cross the cord close to the central canal in the anterior commissure (disassociated sensory loss). In addition, this peculiar sensory loss occurs over only the involved segments (dermatomes) resulting in a suspended sensory loss.

**Anterior Spinal Syndrome**

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

**Posterior Column Syndrome**

Loss of vibratory and position sense occurs below the lesion, but the perception of pain and temperature is affected relatively little or not at all.

**Cauda Equina Syndrome**

Asymmetrical flaccid weakness and wasting of lower limb muscles in the final stages and absent knee, ankle and plantar responses are found on clinical examination. Sensory loss is in the saddle area, involves sacral and lumbar dermatomes so that rectal, vaginal and urethral sensation may be absent. Bladder and rectal sphincter loss may be late and less marked.

**Conus Medullaris Syndrome**

Displays a mixture of upper and lower motor neurone paralysis with absence of tendon reflexes but extensor plantar responses associated with saddle anaesthesia and retention of urine. Bladder and rectal sphincter impairment may be early and marked.

## CHAPTER 4

### PATHOLOGY ( Adams )

#### INTRAMEDULLARY TUMOURS

##### Incidence

Intramedullary tumours of the spinal cord represent about 4% of central nervous neoplasms (Sloof). Astrocytomas and Ependymomas represent the majority of the intramedullary tumours (80-90%), while miscellaneous glial tumours ( oligodendroglioma, ganglioglioma, subependymoma), hemangioblastomas, the rare intramedullary nerve sheath tumour, metastases, inclusion tumours and cysts account for the remainder of intramedullary tumours.

##### Astrocytomas

Spinal cord astrocytomas account for approximately 2-4% of central nervous system astrocytomas. They occur most commonly in the first three decades of life and are four to five times more common than ependymomas in early childhood and adolescence (Epstein 1990). Eighty-eight percent of spinal cord astrocytomas in adults are histologically benign ( Kernohan I-II ). The majority of intramedullary astrocytomas are fibrillar but juvenile pilocytic astrocytomas and gangliogliomas are also frequently found (Epstein 1990; Sandler). These tumours are noted for their indolent behaviour and occur more frequently in children.

Astrocytomas may vary from well circumscribed to diffusely infiltrative without a well defined margin and tumour mass. However, many low-grade fibrillary tumours, especially in children, may be well circumscribed. Intratumoural cysts are encountered more frequently in spinal astrocytomas than in spinal ependymomas. Polar cysts are common and may be lined by non-neoplastic gliotic tissue.

### Ependymomas

Ependymomas are the most commonly occurring intramedullary spinal cord neoplasms in adults over 30 years of age (McCormick 1990C; Stein). These tumours are rare in childhood and adolescence. Approximately half of all CNS ependymomas originate within the spinal canal. Spinal ependymomas occur throughout the spinal cord, although a cervical predominance is reported in most series of intramedullary lesions (McCormick 1990C; Fisher; Hermann). One-third to one-half arise from the filum terminale. The majority are histologically benign (Mork).

Unlike spinal astrocytomas, ependymomas in younger patients tend to be clinically more aggressive. Histological features such as cellularity, vascular proliferation, cellular and nuclear pleomorphism, and necrosis, which may correlate with clinical behaviour in astrocytomas, have not been shown to have prognostic significance for ependymomas (Russell). Even histologically malignant ependymomas may be associated with relatively long survival.

Differentiated features of ependymomas are true rosettes and perivascular pseudorosettes. A variety of histological subtypes may be encountered. The cellular ependymoma is the most common form, but epithelial, tanycytic (fibrillary), subependymoma, myxopapillary, or mixed examples may occur. Almost all are benign. (McCormick 1990C; Mork; Russell) Although unencapsulated, these glial-derived tumours are usually well circumscribed and do not infiltrate adjacent spinal cord tissue.

In the conus region, the tumour may be partially intramedullary and partially exophytic. Those arising in the filum are most common in the third to fifth decades and men are slightly more commonly affected. Myxopapillary ependymoma is the most frequent type encountered in the filum. The distinguishing histological features consist of a papillary arrangement of cuboidal or columnar tumour cells surrounding a vascularised core of hyalinized and scanty cellular connective tissue.

### **Hemangioblastoma**

Haemangioblastomas account for 3-8% of intramedullary tumours. **(Guidetti)** They are most common in the cervical or thoracic region especially when found in association with the von Hippel-Lindau (vHL) syndrome **(Neumann)**. They arise at any age but are rare in early childhood.

Haemangioblastomas are benign tumours of vascular origin that are sharply circumscribed but not encapsulated. Almost all have a pial attachment and are found dorsal or dorsolateral in the cord. They are often associated with cysts.

### **Miscellaneous Pathology**

Uncommon intramedullary tumours include teratomas and dermoid and epidermoid cysts which occur both in an intramedullary location and in the region of the cauda equina. These tumours are slow growing, avascular, encapsulated, and usually resectable. Lipomas account for about 1% of intramedullary tumours. They are not true neoplasms but probably arise from inclusion of mesenchymal tissue and are subpial in location. Metastases account for approximately 2% of intramedullary tumours and lung and breast are the most common primary sources **(Edelson)**

### **EXTRAMEDULLARY TUMOURS**

In adults, about 70% of intradural spinal tumours are extramedullary. **(Okazaki)**. The most common extramedullary tumours are Nerve Sheath tumours and Meningiomas, which occur with approximately, equal frequency **(Slooff; Nittner)**. The next most common tumours are Filum Terminale Ependymomas (15%). With a few exceptions, extramedullary tumours are benign and amenable to complete surgical resection.

### **Nerve Sheath Tumours**

Nerve Sheath Tumours are categorised as either schwannomas or neurofibromas. In patients with Neurofibromatosis ( NF-1 ), the tumours are usually multiple neurofibromas; whereas in the absence of neurofibromatosis, the tumours are almost always schwannomas. (Russell; McCormick 1990A). Neurofibromas and schwannomas have different demographical, histological, and biological characteristics. Schwannomas originate from a Schwann cell precursor cell. It is most likely that neurofibromas originate from cells of mesenchymal ( i.e fibroblast) origin (McCormick 1990A)

They are found more often at thoracic levels. Nerve sheath tumours have an equal incidence in males and females and occur most often between the third and fifth decades.

#### **- Neurofibromas**

Neurofibromas are generally found in the region of the dorsal root ganglia, presenting as a fusiform dilatation of the nerve (Okazaki; Russell)

Microscopically, they have a moderately cellular pattern with a wavy arrangement of nerve fibres, frequently separated by a matrix that is positive for mucin. The nuclei are a dark- staining and elongated. Fibrous strands are positive for both reticulin and collagen. The nerve fibres may be either myelinated or un-myelinated. (Russell)

#### **- Schwannomas**

Schwannomas also usually arise from sensory nerve roots. Grossly, however, they are better delineated from the nerve roots than neurofibromas and are subtly connected to a minority of fascicles within the nerve root without a fusiform enlargement of the nerve itself.

Microscopically they demonstrate two histological patterns; 'Antoni A' and 'Antoni B' types. The 'Antoni A' pattern consists of compact interlocking bundles of elongated bipolar spindle elements. The nuclei stain darkly. The 'Antoni B' pattern consists of a much looser arrangement, without the bundles and palisades seen in the Antoni A type. The cells are often stellate and irregular, with coarsely staining eosinophilic processes. Microcystic degeneration is common. (Russell)

### **Meningiomas**

Seventy-five to 85 percent of meningiomas occur in women, and about 80% are thoracic. (Levy 1982A; McCormick 1990A) Most occur between the 5th and 7th decades of life. They presumably arise from arachnoid cluster cells and therefore are located at the exit zones of nerve roots or the entry zones of the arteries into the spinal canal. They are often lateral or ventrolateral to the spinal cord, especially in the upper cervical spine and foramen magnum where they may be adherent to the vertebral artery near its intradural entry. Ten percent of spinal meningiomas are found in both intradural and extradural components simultaneously or are entirely extradural (Nittner)

The gross characteristics range from smooth and fibrous to the more common variegated, fleshy, and friable appearance. Microscopically, meningiomas are often calcified. Psammomatous meningiomas are the most frequent type. (McCormick 1990A) In younger patients, meningiomas may grow more aggressively and are more often angioblastic (Russell) The growth rate of meningiomas may be affected by female sex hormones (Black 1993)

### **Other Benign Intradural Tumours**

Lipomas are usually found in the thoracic region and are usually intradural and extramedullary and account for about 1% of all spinal tumours. They can be completely intramedullary or partially intramedullary and partially extramedullary. Spinal dermoids and epidermoids can be either extramedullary or intramedullary and are usually located in the lumbosacral region.

### **Malignant Extramedullary Tumours**

Pathological subtypes include malignant schwannomas, neurofibrosarcomas, malignant epithelioid schwannoma, and malignant melanotic schwannomas (**Okazaki**)

Malignant neurofibromas can arise either de novo or as malignant degeneration of pre-existing neurofibromas in NF-1 patients. Malignant degeneration occurs primarily in adults in approximately 2.4 to 4.6 % of patients, (**Sorensen 1986**)

Intradural metastatic lesions are usually associated with the dissemination of tumour via the cerebrospinal fluid pathway. Common tumour types that present in this manner include leptomeningeal carcinomatosis, cerebral ependymomas, medulloblastomas, neuroblastomas, pineoblastomas, lymphoreticular tumours, and leukemia (**Okazaki**)

Other rare types include myosarcomas, chondrosarcomas, and osteogenic sarcomas (**Okazaki**)

### **EPIDURAL TUMOURS**

Metastatic lesions of the spine account for the majority of spinal epidural tumours. . Benign tumours of the vertebral column are very rare and comprise only 1-2% of all primary lesions in the skeleton (**Moran**). Of primary lesions specifically within the vertebrae, benign tumours represent 10-40% (**Malawski; Moran**)

Primary malignant tumours of the spine, excluding lymphoreticular tumours, are extremely rare with an incidence of less than 1 per 100 000 per year (**Dahlin 1978;Huvos; Sundaresan 1990A**)

### **Metastatic Tumours**

In adults, the common primary sources are breast, lung, prostate, kidney and lymphoma. There is a slight male preponderance (60%) (**Gilbert; Törmä**) and the highest incidence occurs in the age range 40-65 years.

## Benign & Malignant Primary Spine Tumours

### - *Malignant Tumours*

Among adults, primary malignant tumours of the spine may be divided into:

- a) Lymphoproliferative conditions
- b) Solid tumours of cartilage, bone, or mesenchymal elements.

- a) Lymphoproliferative conditions

- *Myeloma / plasmacytoma*

Myeloma and plasmacytoma are the most common of all primary malignant spine tumours. The reported incidence is 2-3 per 100 000 in the general population. (**Dahlin**). There is a malignant proliferation of plasma cells in bone marrow, spleen and all lymphoid tissues. About 3% may present as solitary plasmacytomas in either bone marrow or soft tissue and the prognosis is better than for multiple myeloma of the spine. Plasma cell spinal malignancies may have lytic lesions on x-ray, pathological compression fractures or severe osteopaenia. The anterior vertebral elements are usually involved.

- b) Solid Tumours

- *Chordoma*

Chordomas are the commonest solid primary spinal malignancies occurring in less than one-third of patients (**Dahlin**), with approximately 85% occurring in either the sacrum or clivus (**Sundaresan-H**).

They are thought to arise from the notochordal remnants in the vertebral discs and usually have an indolent course.

*-Non-plasma-cell lymphomas ( 10 % )*

They rarely come to the attention of the surgeon. When they do, it is usually because of epidural rather than bony disease.

*-Chondrosarcomas ( 10 % )*

Chondrosarcomas range from almost benign-appearing round-cell tumours to very malignant spindle-cell sarcomas. They usually occur on the lateral side of the vertebral body near the costovertebral junction and are thought to arise from the cartilaginous proximal end of the rib. The majority of cases involve both the anterior and posterior elements by the time of clinical presentation.

*-Osteosarcoma & Ewing's sarcoma ( 10 % )*

These sarcomas are extremely malignant lesions. Ewing's sarcoma usually affects children and primary osteosarcomas occur most frequently in the growing ends of long bones of adolescents. Osteosarcomas arising in pagetoid or irradiated bone usually occur after the age of 60 years.

**Benign Tumours**

*-Osteochondroma ( 3-4 % )*

Osteochondromas are sessile or pedunculated exostosis with a cartilage cap affecting mainly the posterior elements.

*-Haemangioma ( 4-5 % )*

Haemangiomas are vascular tumours consisting of newly formed blood vessels and usually affect the lower thoracic or upper lumbar spine. Approximately two-thirds are solitary.

*-Aneurysmal bone cyst ( 10,5 % )*

Aneurysmal bone cysts are lytic, expansile tumours with a thin cortical shell which affect the posterior elements and are more common in the lumbar spine. They may involve multiple levels.

*-Eosinophilic granuloma ( 3,8 % )*

Eosinophilic granuloma is not a true neoplasm, but rather part of a spectrum of diseases due to a defect in lipid metabolism of the reticulo-endothelial system of bone. Common sites of involvement are the skull and the body of the thoracic or lumbar spine.

*-Giant-cell tumour ( 1-4 % )*

Giant cell tumour is a locally aggressive tumour and histological features of note are multi-nucleated giant cells in a mononuclear stroma. It usually affects the posterior elements and the sacrum is most commonly involved.

*-Osteoid osteoma ( 9 % )*

The distinguishing features of osteoid osteoma consist of a nonprogressive radiolucent nidus with rim of sclerosis. This tumour usually is <2cm in diameter and involves the posterior spinal elements of the lumbar and thoracic spine.

*-Osteblastoma ( 10 % )*

Osteblastomas present as an expansive, radiolucent nidus with or without a sclerotic margin. The nidus is usually >2cm in diameter. They may undergo malignant transformation and the posterior elements at all levels may be affected.

## CHAPTER 5

### **STUDY: A RETROSPECTIVE EVALUATION OF ADULT NEOPLASTIC SPINAL CORD COMPRESSION IN THE DEPARTMENT OF NEUROSURGERY AT GROOTE SCHUUR HOSPITAL FROM 1987 - 1997**

#### **OBJECTIVE**

The objectives of this retrospective study were:

- a. To assess the incidence and therapies used in the management of adult patients with various spinal tumours who were managed in the Neurosurgery Department at Groote Schuur Hospital.
- b. To determine the outcome of adult patients with spinal tumours, causing symptomatic spinal cord compression.
- c. To evaluate specifically the effectiveness of spinal decompressive surgery with respect to the different types of lesions.
- d. To evaluate the usefulness of the Frankel Scale which was originally described for spinal injury, in predicting functional outcome and comparing results in tumoural spinal cord compression.

## Patients and Methods

A 10-year retrospective review was performed of all adult patients with symptomatic neoplastic spinal cord compression admitted to the Groote Schuur Hospital / University of Cape Town Health Complex from 1987-1997. Hospital records from the departments of Neurosurgery and Medical Informatics were reviewed and demographic data regarding level of compression, type of tumour, pre-admission neurological status and post-operative neurological status one year after treatment, type of surgery, type of antitumour therapy and complications of the different modes of therapy, were recorded.

A Frankel score was assigned to each patient retrospectively, based on the initial and follow-up neurological examinations as documented in the hospital records of each patient. (Fig 5)

**Figure 5**

### FRANKEL SCALE

- |           |                      |   |  |
|-----------|----------------------|---|--|
| <b>A.</b> | <b>COMPLETE</b>      | - | motor and sensory loss below the lesion.                                       |
| <b>B.</b> | <b>MOTOR USELESS</b> | - | some sensory preservation below the zone of injury.                            |
| <b>C.</b> | <b>MOTOR USELESS</b> | - | motor and sensory sparing, but the patient is non-functional.                  |
| <b>D.</b> | <b>MOTOR USEFUL</b>  | - | motor and sensory sparing and the patient is functional<br>(stands and walks). |
| <b>E.</b> | <b>RECOVERY</b>      | - | complete functional recovery; reflexes may be abnormal.                        |

We included adult patients older than 15 years of age, but excluded patients without any evidence of neurological abnormalities and non-compressive causes of paraplegia.

The patient data was extracted from records obtained in the following ways:

- a. Records from the Department of Neurosurgery were reviewed from 1987-1997.
- b. A cross check was performed with a computer generated-search from the Department of Medical Informatics from 1987-1997, using the following key words:  
  
spine, compression, spinal tumour, paraplegia, spinal cord tumour, paralysis, metastases, spine surgery.
- c. Reference was also made to previous University of Cape Town publications on spinal compression by Buwembo (1996) and Hoffman et al (1993).

## **RESULTS**

### **Epidemiology**

Between 1987 and 1997, 123 patients affected by neoplastic spinal cord compression underwent treatment in the Department of Neurosurgery at the Groote Schuur Hospital /University of Cape Town complex. Of these patients, 62 were male and 61 female. Their ages ranged from 15-50 years. The age incidence of malignant tumours peaked at 55-59 years (5/123; 4%) and 70-80 years (7/123; 5,7%). Benign tumours peaked at 35-39 years (16/123; 13%).

### Clinical Presentation

The most common complaint was leg weakness (75/123; 61%) followed by pain in either the back, leg or arm (60/123; 49%). Upper limb weakness was present in 9 patients (7,3%) and total paraplegia was present in 4 patients (3,2%).

**Table 1**

#### Symptoms of Spinal Compression ( n = 123 )

Symptoms	No.	%
Leg weakness	75	61
Pain	60	49
Upper Limb Weakness	9	7,3
Paraesthesia/Dysaesthesia	16	13
Sphincter Abnormality	17	13,8
Total Paraplegia	4	3,2
Sensory Loss	14	11,4
Limb Swelling	3	2,4

Pre-operative Frankel Scores were: (Table 6)

**A: 20 (16,3%)    B: 6 (4,9%)    C: 19 (15,5%)    D: 57 (46,3%)    E: 21 (17,0%)**

From the above it can be seen that although only 4 patients (3,2%) complained of total leg weakness, on examination we found that 20 (16,3%) patients had complete motor and sensory loss (Frankel A).

### Diagnostic Imaging

Up to 1980, only myelogram and CT/myelogram was used at Groote Schuur Hospital. MRI was introduced in the early 80's and in our series, MRI was the most common mode of diagnostic imaging (60/123); 10 patients had a MRI and a myelogram combination and 39 patients had a myelogram only.

**Table 2**

### Radiological Investigations

Mode	No.
MRI	60
CT/Myelogram	14
Myelogram	49
Isotope Bone Scan	3
Angiogram	1

There was only 1 patient who deteriorated post-myelogram and had to undergo surgery within 24 hours.

### Level of Compression (Table 3) (Graph 1)

Forty-one patients (33,3%) presented with cervical lesions with the larger proportion occurring in the lower cervical spine (26/123; 21,1%). Thoracic lesions were the most common (55/123; 44,7%) with the majority in the lower thoracic region (34/123; 27,6%). There were 23 Lumbar (18,7%) and 4 sacral tumours.

### Site of Compression

There were 40 patients with extradural tumours (32,5%). Seventy-two with intradural extramedullary tumours and 11 with intramedullary tumours .

**Table 3**

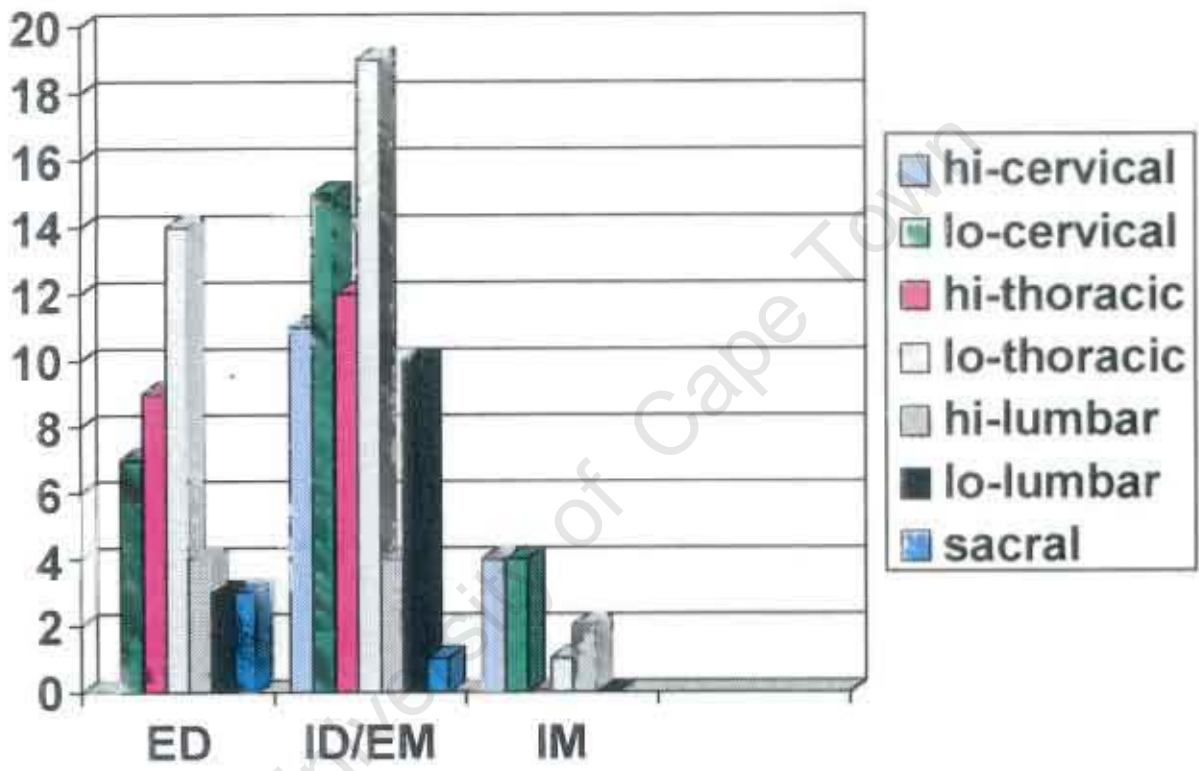
**Level and Site of Compression (Graph 1 – Pg 37)**

Level	Site			Total	%
	ED	ID/EM	IM		
Upper cervical		11	4	15	12,2
Lower cervical	7	15	4	26	21,1
Upper thoracic	9	12		21	17,1
Lower thoracic	14	19	1	34	27,6
Upper lumbar	4	4	2	10	8,1
Lower lumbar	3	10		13	10,6
Sacral	3	1		4	3,3

ED = extradural

ID/EM = intradural extramedullary

IM = intramedullary



GRAPH 1: LEVEL & SITE OF COMPRESSION

## HISTOLOGY/TUMOUR TYPES

Histological diagnosis of the neoplastic processes is summarised in **Table 4**.

**Table 4**

### Histology of Tumour Types

Histology	No	%
Neurofibroma	39	32
Metastatic tumour	27	22
Schwannoma	13	10,5
Meningioma	12	9,7
Lipoma	7	5,7
Myeloma	6	4,9
Astrocytoma	5	4,0
Ependymoma	5	4,0
Non Hodgkins Lymphoma	2	1,6
Chondrosarcoma	2	1,6
Acute Lymphocytic Leukaemia	1	0,8
Ganglioglioma	1	0,8
Neurilemmoma	1	0,8
Hemangioma/Blastoma	1	0,8
Chordoma	1	0,8
	123	100

The largest majority of tumours in our series were benign (78/123; 63,4%). The most common tumour types identified were neurofibroma (39/123; 32%); metastases-spinal (27/123; 22%); meningioma (12/123; 9,7%); and Schwannoma (13/123; 10,5%). Some patients with neurofibroma and Schwannoma had Neurofibromatosis Type-1 and underwent several procedures during their lifetime (15/123; 12,2%). There was an equal incidence of spinal cord astrocytoma and ependymoma (5/123; 4%). Two-fifths of the astrocytomas were high grade.

The most common primary source for metastasis was the lung and bronchus (9/27; 33,3%). There was an equal incidence from the kidney, colon and prostate (3/27; 11,1%). Breast primary accounted for 2/27 (3,6%). In 6 patients (6/27; 22,2%), there was no identifiable primary cause.

#### **Surgical Treatment (Table 5 – Pg 40)**

The majority of surgical procedures consisted of laminectomy and complete excision of tumour in 50 patients (50/123; 40,6%). Laminectomy and debulking of tumour accounted for the second most common operative procedure (24/123; 19,5%). Laminectomy only was done in 18 patients (18/123; 14,6%).

All 3 patients who had a vertebrectomy, underwent a transthoracic approach for metastatic spinal disease. Fifteen patients underwent repeated surgical procedures for tumour recurrence (15/106; 14,1%).

#### **Adjuvant Therapy**

Steroid therapy was commenced preoperatively / intraoperatively in 85 patients and continued in the post-operative phase. It is not clear from the patient notes what dose, duration and type of steroid was used, but it is most likely that dexamethasone was used in the majority of the cases. Seven patients (5,7%) received radiation therapy only to the spine and 41 (33,3%) patients received radiotherapy after a surgical procedure. Eight patients (6,5%) received chemotherapy and radiotherapy.

### Morbidity and Mortality

The most common complications encountered were wound sepsis (3); meningocoele - post-laminectomy (3); post-operative pneumonia (3); unstable spine (2); and meningitis (2).

There were 17 deaths over 2 years and no intraoperative death, but 1 patient died within one month of surgery; thus our operative mortality was 1/106 ( 0.9% ). Of the 16 deaths in the first year, 8 were attributed to metastatic spinal tumour.

**Table 5**

Operative Procedures	No.	%
Laminectomy/Complete Excision	50	40,6
Laminectomy/Debulking	24	19,5
Laminectomy only	18	14,6
Biopsy only	11	8,9
Vertebrectomy/Corpectomy	3	2,4

In those patients with a pre-operative Frankel Score - B - (6), 4 patients underwent a laminectomy and complete excision of the tumour and 2 underwent laminectomy and debulking of tumour.

In those patients with a pre-operative Frankel Score - A - (20), 4 patients underwent a laminectomy and complete tumour excision and 8 patients underwent laminectomy and debulking only.

In those patients with metastatic spinal tumour who died within a year of diagnosis (8); (8/27; 29,6%); only 2 patients underwent a laminectomy and debulking, whilst 3 patients underwent biopsy only and 3 had radiotherapy only.

All 16 deaths occurred in patients with malignant disease with the exception of 2 patients who died from recurrent neurofibromas and schwannomas and had recurrent surgical procedures over a period of 5 years. Of these deaths, overall, 3 patients had a Frankel Score - A - and 1 had a Frankel B.

### **Functional Outcome**

At the end of 1 year, follow-up notes were available for 103 patients (103/123%; 83,7%). Sixteen patients had died and 4 patients were lost to follow-up.

At the end of 5 years, follow-up notes were available for approximately 40 patients only and it was decided not to include a 5-year follow-up Frankel table in this series.

**Table 6 – Pg 42** summarises the 1-year follow-up data available on 103 patients.

**Table 6****Frankel Scores pre-operative and 1-year post-operative****Table 6A****Pre-Operative Frankel Scores**

<b>LEVEL</b>	<b>A</b>	<b>B</b>	<b>C</b>	<b>D</b>	<b>E</b>	<b>TOTAL</b>
Upper cervical	1	1	4	7	2	15
Lower cervical	4		2	18	2	26
Upper thoracic	5	3	2	8	3	21
Lower thoracic	8	1	9	12	4	34
Upper lumbar	1	1	1	4	3	10
Lower lumbar	1		1	7	4	13
Sacral				1	3	4
<b>TOTAL</b>	<b>20</b>	<b>6</b>	<b>19</b>	<b>57</b>	<b>21</b>	<b>123</b>

**Table 6B****1 Year Post-Operative Frankel Scores**

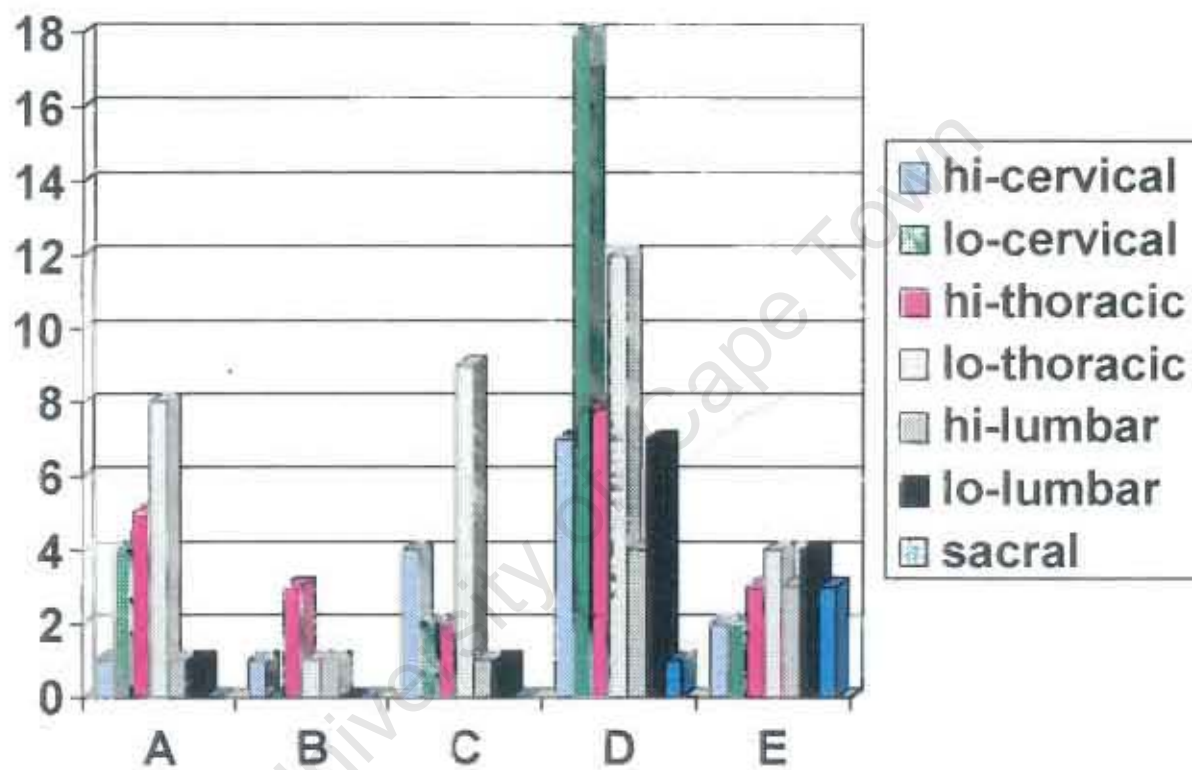
<b>LEVEL</b>	<b>A</b>	<b>B</b>	<b>C</b>	<b>D</b>	<b>E</b>	<b>TOTAL</b>
Upper cervical	1	1	5	4	4	15
Lower cervical	4			8	8	20
Upper thoracic	5	1		4	8	18
Lower thoracic	3	1	4	9	10	27
Upper lumbar	1	1	1	4	3	10
Lower lumbar	2	3		4	4	13
Sacral						
<b>TOTAL</b>	<b>16</b>	<b>7</b>	<b>10</b>	<b>33</b>	<b>37</b>	<b>103</b>

In the category of patients with Frankel A at pre-operative evaluation (20), only 1 patient (out of 4 who had undergone complete excision of tumour), recovered to Frankel D at 1 year post-operatively (meningioma = symptoms - 2 months). In the same category, 1 patient (out of 8 who underwent debulking of tumour) recovered to Frankel D (myeloma = symptoms - 2 months).

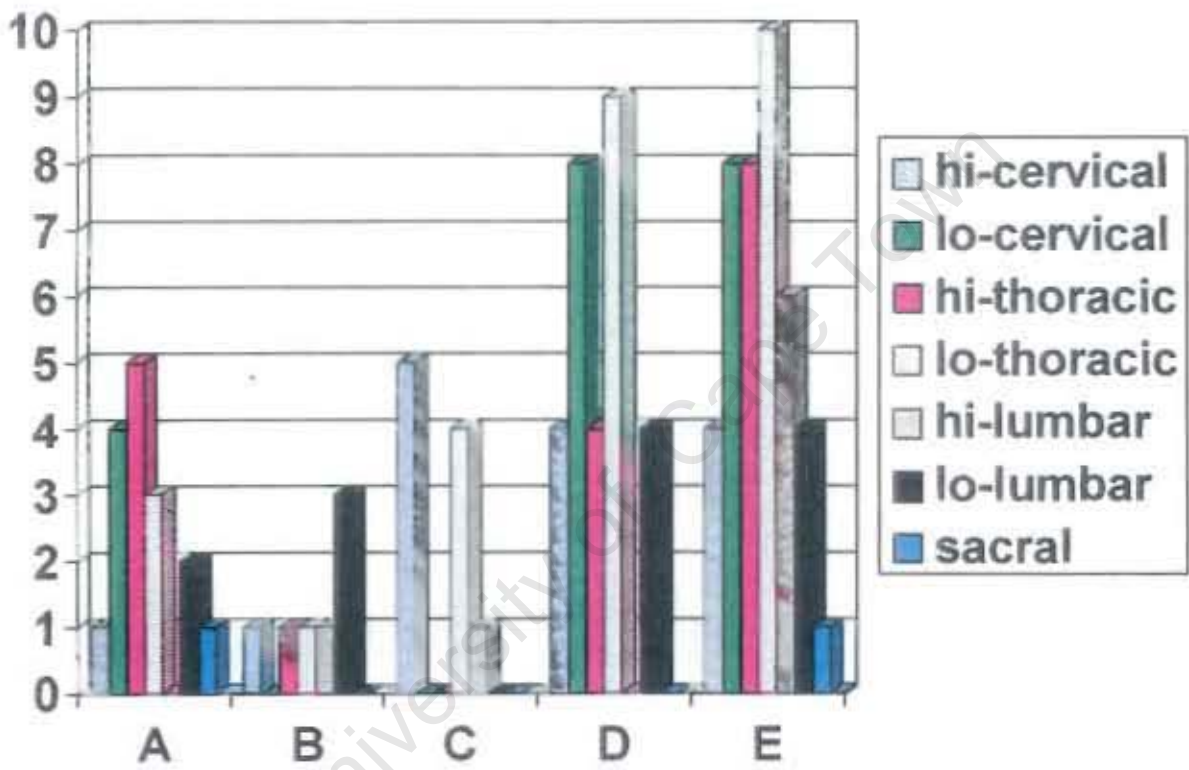
In the category of patients with a Frankel B at pre-operative assessment (6), only 2 patients improved to Frankel E at 1 year follow-up. Histological tumour types in the latter cases were meningioma (duration of symptoms = 9 months) and neurofibroma (symptoms = 3 months). However, 2 other patients with similar benign tumours and symptom duration failed to recover (Schwannoma = 3 months; neurilemmoma = 2 months). Both groups had undergone complete excision of tumour.

In the category Frankel C (19), there was only 1 death due to non-Hodgkins lymphoma at 1 year follow-up. Four of these patients had deteriorated to Frankel A at 1-year follow-up, 3 patients had neurofibromatosis and repeated surgical procedures and 1 patient had a high grade glioma of the cord. The remaining patients (14) had improved to Frankel D or E. **(Graphs 2 & 3 – Pgs 44 & 45)**

University of Cape Town



GRAPH 2: PREOPERATIVE FRANKEL SCORES



GRAPH 3: 1 YR POSTOPERATIVE FRANKEL SCORES

## CHAPTER 6

### DISCUSSION:

#### GENERAL

The quoted ratio of intradural to extradural tumours is approximately 3:2 (**Kurland; Slooff**). Most studies separate the incidence of epidural tumours and intradural tumours. In our series, the ratio of intradural:extradural tumours is 83:40 (2.07:1). This slightly different result may reflect our selection bias in excluding cases without neurological abnormalities.

#### Metastatic Spinal Tumours

Metastatic lesions of the spine represent the large majority of spinal epidural tumours. Some reports have suggested that approximately 5% of patients with a primary malignancy - both adults and children - develop spinal secondaries (**Barron; Sundaresan (A); Galasko; Sundaresan (G)**). Others have said that approximately 20-100% of all patients with malignancies develop skeletal metastases during their lifetimes and the majority of these patients have spinal lesions (**Jaffe, Schaberg; Wong**). One in 10 patients with symptomatic spinal metastases presents without a known primary tumour (**Botterell; Livingston; Gilbert**). In 6 (6/27; 22.2%) of our patients, we were unable to diagnose the primary source of the spinal metastases.

Not all patients with spinal secondaries develop neurological deficits (**Cobb**). Older studies have suggested that as many as 70% of patients with spinal tumours had objective weakness by the time of definitive diagnosis (**Weinstein**). However, with the introduction of MRI, it is now possible to detect spinal lesions before neurological deficits develop and the incidence of neurological deficits at the time of diagnosis is now no more than 10%. (**Sarpel**). According to Schaberg, 20% of the patients with neoplastic involvement of the vertebral column develop spinal cord compression (**Schaberg**).

There is a slight preponderance of metastatic tumours in males (60%) (Gilbert; Törmä) and the peak incidence at 40-65 years of age. We found that our incidence of symptomatic spinal metastatic disease was 27/123 (22%) and the ratio of males to females was 62:61 with age peaks at 55-59 years and 70-80 years.

We also found that the primary source of the metastatic tumour was most commonly from the lung and bronchus (9/27; 33.3%). The kidney, colon and prostate were the next most common (3/27; 11.1%). This is comparable with most adult series where primary sources in the breast, lung, prostate, kidney and lymphoma were most common (Constans; Sunderesan (F)), although in our series, breast primary accounted for only 3.6% of cases (2/27).

Our most common level of metastatic spinal involvement was at the thoracic level and the least common site was the cervical area and this reflects the findings of other studies. (Livingston)

### **Intradural Spinal Tumours**

Spinal cord tumours account for approximately 15% of central nervous system neoplasms (Slooff). Some series have quoted the incidence of approximately 3 to 10 per 100 000 population (Kurland).

Helseth & Mork reported a large population based survey of primary intraspinal neoplasms from the Norwegian Cancer Registry and quoted an annual incidence of five per million for females and three per million for males. Of these intradural spinal tumours, about 70% are extramedullary (Okazaki; Slooff; Russell) and the most common of these, are the nerve sheath tumours and meningiomas which occur with approximate equal incidence. Nittner et al reviewed 4885 cases of spinal cord tumours and other space occupying lesions in their series. Meningiomas accounted for 22.4% of the cases, Schwannomas for 23.1% and ependymomas for only 2.5%. Slooff et al in 1964 reported 1322 tumours and the incidence of meningiomas was 25.5% and of Schwannoma 29%.

In our series, the most common tumour type found was neurofibroma (39/123; 32%) followed by Schwannoma (13/123; 10,5%) and meningioma (12/123; 9,7%). As a group, nerve sheath tumours accounted for (52/123; 42,5%) and meningioma just under 10%.

Astrocytomas and ependymomas represent the majority of the intramedullary tumours (80-90%) and occur with approximately equal incidence (Slooff). Astrocytomas occur with equal frequency in men and women in the third and fourth decades. Ependymomas occur between the second and sixth decades with a peak incidence in the fourth decade and they are slightly more common in males than females. The results of our series show that the incidence of astrocytomas peak at 30-39 years and ependymomas peak at 40-49 years.

#### **Distribution of Spinal Tumours**

We found that the ratio of intradural to extradural tumours was 83:40, metastatic tumours were most common in the thoracic region (n=17), intradural extramedullary tumours were most common in the thoracic region too (n=31) and intramedullary tumours most common in the cervical region (n=8).

From the literature, metastatic tumours involve the thoracic spine in about 60% of cases. Symptomatic spinal metastases most commonly localise in the thoracic spine (Livingston). Nerve sheath tumours are found more often at thoracic levels and they have an equal incidence in males and females and occur most often between the third and fifth decades. Seventy-five to 85% of meningiomas occur in women and about 80% are thoracic (Levy 1982A; McCormick 1990A). Most occur between the fifth and seventh decades of life. Astrocytic intramedullary tumours occur with equal frequency in men and women in the third and fourth decades and they are found most commonly in the cervical segments (Webb).

Ependymomas occur throughout the spinal cord, although a cervical predominance is reported in some series of intramedullary lesions (Greenwood). About 40% of spinal canal ependymomas arise within the filum terminale (Sloof; McCormick 1990C). They are the most common intramedullary spinal cord neoplasms in adults over 30 years of age. (McCormick 1990C).

In our series, 11 of the 12 meningiomas were found in the thoracic region and 10 of them occurred in females.

## **PRIMARY SPINAL TUMOURS**

### **Malignant Spinal Tumours**

These include lymphoproliferative conditions (myeloma, plasmacytoma) and solid tumours (chordoma, chondrosarcoma). In our series we had 1 sacral chordoma, 2 lumbosacral chondrosarcomas, 6 myelomas with 4 found in the thoracic region, 1 non-Hodgkins lymphoma and 1 acute lymphocytic leukaemia - both found in the thoracic area.

Primary malignant tumours of the spine are rare. Excluding lymphoreticular malignancies, the incidence of other primary spinal tumours is less than 1 per 100 000 per year (Dahlin 1978A); Huvos; Sundaresan 1990A).

Our series reflects the results of other large series where malignant primary bone tumours of the spine are uncommon, accounting for a small percentage of all primary bone tumours (Dahlin 1986; Huvos).

### **Benign Spinal Tumours**

These are exceedingly rare. It has been estimated that they account for about 1-2% of primary skeletal tumours (Dahlin 1986; Huvos). We did not record any case of benign primary spinal tumour in our series.

## CLINICAL SIGNS AND SYMPTOMS

The most common complaints of patients, in our series, were leg weakness (61%) and pain (49%). Paraesthesias and dysaesthesias (13%) and sphincter abnormalities (13,8%) were the next most common presenting symptoms. In 14 patients (11,4%), there was evidence of sensory loss. Most series report that pain is usually the initial symptom (96% of cases) preceding other symptoms by approximately 5 days-2 years (median 7 weeks). (Gilbert; Levy 1986; Shapiro).

Pain, however, may be absent but usually with epidural and intradural, extramedullary tumour, pain is felt in a segmental distribution. often before neurological deficits occur (Gilbert; Livingston) Stretching or compression of the pain sensitive structures viz, longitudinal ligaments, annulus fibrosus, joint capsules and dura may result in pain in a localised area in the back. Compression of the spinal cord causes very little or no pain, but compression or tumour infiltration of the nerve roots may result in severe radicular pain in a dermatomal distribution.

The majority of patients with metastatic spinal disease experience midline pain localised to the level of spinal involvement. Pain due to spinal compression may be dull, aching and continuous with a wide distribution. Pain of spinal root origin is described as shooting, stabbing or burning.

Motor disturbances are often the second clinical feature to be noticed with the legs affected first and the patient complaining of stiffness of muscles, gradually followed by weakness.

The term 'paraplegia' causes great confusion when assessing reported results (Findlay 1984). In some papers, the term 'paraplegia' is used to include those with sensory preservation but no movement (Shaw 1980) and other papers include people with retained movement but no antigravity function (Siegal 1985A). Charting of individual muscle strengths does not give a good functional picture, and it is best to assess a patient's disability in terms of his or her ability to walk. (Menezes). Thus the usage of some universal

classification system would help toward resolving the present confusion and ensure uniformity. The Frankel Scale was developed to assess and classify post-traumatic paraplegic patients and its usage has now been extended to assess paraplegic patients with spinal tumours and this modified scale is the ASIA / IMSOP scale (Frankel; Menezes; Tokuhashi) (Fig 6). Other classification systems have been used in spinal tumours but have not received universal acceptance (Cooper 1985).

In our series, complete paraplegia was recorded in 4 patients, leg weakness in 75 patients and hand weakness in 9. In order to ensure uniformity, a Frankel Scale grading was assigned to all patients retrospectively after analysing their reported clinical notes.

The pre-operative Frankel Scores in our patients were: (See Table 6 – Pg 42).

The Frankel Scale, also, allows better assessment of the patient's disability and functional status. This facilitates comparison of the preoperative functional ability to the postoperative functional outcome to be made. Different therapies such as surgery, or surgery and radiotherapy, or radiotherapy alone can then be assessed.

**ASIA/IMSOP Impairment Scale\***

<b>Grade A—complete</b>	<b>No motor or sensory function is preserved in the sacral segments S4 and S5.</b>
<b>Grade B—incomplete</b>	<b>Sensory but not motor function is preserved below the neurological level and extends through the sacral segments S4-S5.</b>
<b>Grade C—incomplete</b>	<b>Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade less than 3.</b>
<b>Grade D—incomplete</b>	<b>Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade greater than or equal to 3.</b>
<b>Grade E—normal</b>	<b>Motor and sensory function are normal.</b>

\* Classification of spinal cord injury based on the International Standards for Neurological and Functional Classification of Spinal Cord Injury by the American Spinal Injury Association (ASIA) and the International Medical Society of Paraplegia (IMSOP).

**FIGURE 6: ASIA / IMSOP SCALE**

### **LABORATORY INVESTIGATIONS**

These should begin with a complete blood count, erythrocyte sedimentation rate and urinalysis. Serum and urine protein electrophoresis may confirm or rule out multiple myeloma. Measurement of levels of prostate-specific antigen (PSA) may help in the diagnosis of metastatic prostate carcinoma.

## **DIAGNOSTIC IMAGING**

### **X-rays**

Although Magnetic Resonance Imaging (MRI) is the radiological procedure of choice, diagnostic imaging should still include plain films. A minimum of approximately 50% of local bone destruction must occur in order for a lesion to be detected on plain x-ray films. (Charkes). Plain radiology of the spine shows evidence of metastatic disease in approximately 60% of patients with cord or root compression (Black 1985) and helps establish the exact level of a lesion. Plain films complement MRI in the visualisation of spinal bony structure and should include the entire spine to detect skip lesions at different levels. Plain x-ray changes are usually visible in either the pedicle or the vertebral body, showing either pedicular destruction or loss or vertebral collapse. Multiple spinal deposits can occur in up to 17% of cases (Gilbert).

### **Computed Tomography Scans ( CT )**

CT Scans are far more sensitive than plain x-ray films, but they are relatively non-specific and impractical to use as screening tests because of sequential axial-only imaging. The two most common uses of CT lies in the guidance of needle biopsy in cases where pathological diagnosis is desirable before a therapeutic programme is embarked upon and for surgical planning. CT myelography (CT myelogram) is particularly useful in delineating the plane between the tumour and the spinal cord. CT gives clarity regarding the status of the pedicles, facets and laminae. (Hoffman).

### **Myelography**

Myelography, using water-soluble contrast medium, is readily available in most of our hospital but is invasive and has complications. At times, it is not clear from the myelogram if the lesion is anterior or posterior to the cord, especially when a complete myelographic block is encountered. An acute increase in neurological deficit may occur after myelography (14-20%) (Hollis). We included 1 patient with increased neurological deficit after myelography.

In our series, myelogram only was performed in 39 patients and combined MRI/myelogram was done in a further 10 patients to provide clarity on the site and type of lesion and to show up multiple sites of involvement. CT myelogram was performed in 14 patients on an emergency basis because of the unavailability of on-site MRI and logistic difficulty of arranging MRI Scanning in private hospitals on an emergency basis.

### **Isotope Bone Scans**

Isotope bone scanning can be useful in assessment. Bone scans can detect most vertebral tumours including skip lesions and can detect metastases in other parts of the skeleton. Therefore, they are used for the early detection of vertebral lesions, planning of surgery, long-term follow up of patients and to detect potential pathological fractures elsewhere in the body. However, it is a non-specific screening test and both false positive and false negative results are not uncommon. In our series, we utilised isotope bone scans in only 3 patients.

**MRI (PLATES 1, 2, 3 – Pgs 56, 57, 58)**

Magnetic Resonance Imaging (MRI) is established as the modality of choice for evaluating patients with suspected spinal and intradural pathology. It provides excellent anatomical definition in multi-planar views and is useful for planning surgery (Hoffman). It is non-invasive and uses radiofrequency energy, which is non-ionizing and the entire spine can be imaged, although this can be quite expensive. Multiple separate metastases are relatively common (17-33%) (Gilbert; Ruff; van der Sande), so it is desirable to image the entire spine. Gadolinium enhancement has further increased the sensitivity of MRI.

Most intradural tumours are iso-intense or slightly hyperintense with respect to the spinal cord and cauda equina on T1-weighted images. Nerve sheath tumours are more likely to be hyperintense to the spinal cord than meningiomas on T2-weighted images. Cauda equina tumours usually demonstrate increased signal intensity with respect to CSF on both T1 and T2-weighted images. Almost all spinal cord tumours demonstrate some degree of contrast enhancement on MRI.

In our series, a total of 60 patients underwent MRI. These included 10 patients who had an MRI post myelogram, 2 patients with a prior CT Scan and 1 patient with a prior bone scan. This demonstrates the increasing usage of MRI at Groote Schuur Hospital despite the fact that there is no MRI facility on site. One of the major drawbacks in our regional state hospitals is the lack of availability of MRI and the very high cost of obtaining images from MRI Scanners situated in private hospitals in our area. Currently, we have been allocated only 2 paid sessions at private hospitals in the region on 2 separate days, morning sessions only and emergency MRI requests which are fraught with management and transport problems.

**Angiography**

Angiography is still used in evaluation of cases where the vascular architecture is important for planning of the surgery and in our series 1 patient underwent an angiogram. It will outline the level of the artery of Adamciewicz. Selective arterial embolization, preoperatively, can decrease intra-operative bleeding and can be used in the treatment of some spinal tumours.



PLATE 1: MRI- T1WI - EXTRADURAL TUMOUR ( 2 METASTATIC DEPOSITS )

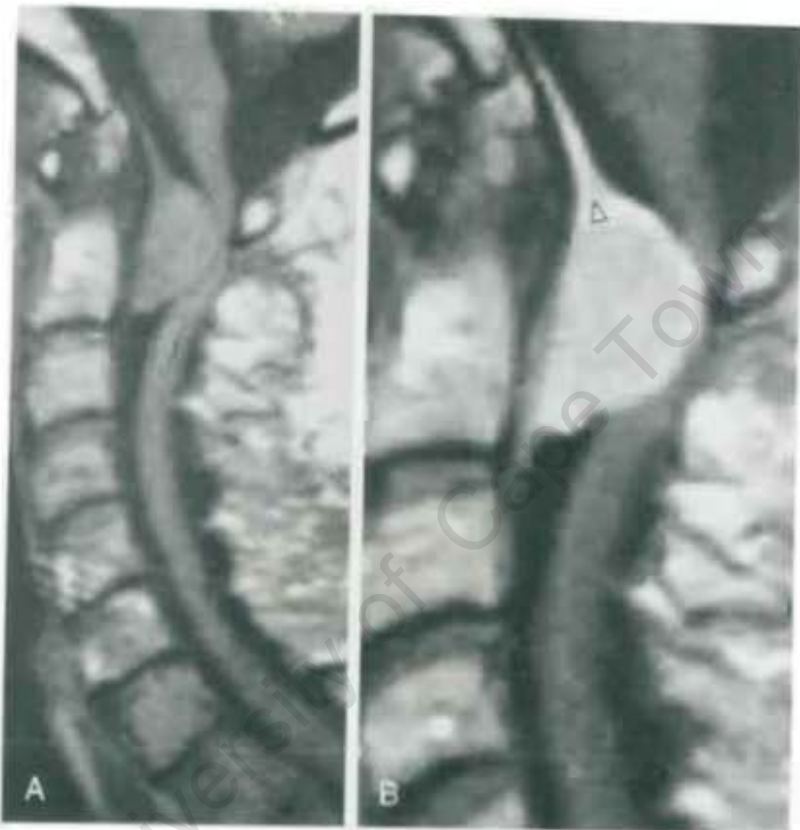


PLATE 2 : MRI- T1WI ( PRE & POST CONTRAST ) - INTRADURAL / EXTRAMEDULLARY  
TUMOUR ( MENINGIOMA )



PLATE 3 : MRI- T1W1 - INTRAMEDULLARY TUMOUR ( ASTROCYTOMA )

## CHAPTER 7

### MANAGEMENT

A multi-disciplinary approach should be adopted in management of adults with spinal tumours and decisions on management should be made by a combined group including neurosurgeons, orthopaedic surgeons, histopathologists and oncologists.

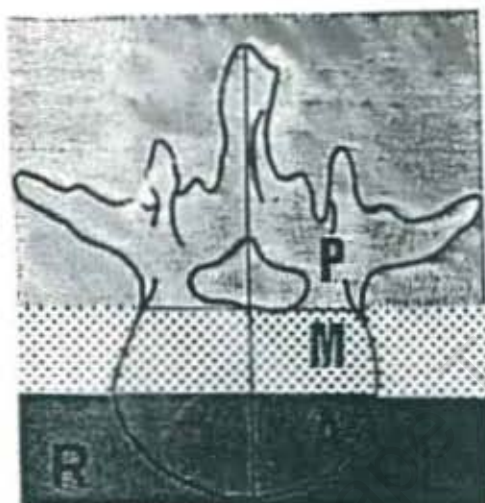
### EPIDURAL TUMOURS

#### Spinal Metastases

In patients with spinal metastases, cure is not possible and palliation is a reasonable goal. Siegel & Siegal (Siegal 1985A) defined the criteria of successful therapy in metastatic spinal cord compression as: "Preservation or restoration of neurological function - ambulation and control of bowel and bladder." Pain relief is also an important but secondary goal that may often be achieved by treatment of the metastatic tumour.

Radiotherapy and surgery are the keystones of management. The relative merits of each have not yet been clarified. To date, there has not been any large, prospective, controlled series comparing radiotherapy and surgery; a group of prospective, clinical trials were inconclusive (Young; Sundaresen 1991, Siegal 1985A).

Most physicians treating this disorder assume that surgical decompression is the only logical choice. Thus, most large series evaluating treatment of malignant extradural tumours have been surgical with patients subjected to laminectomy with or without post-operative radiotherapy (Gilbert).



Classification systems for the evaluation of spinal stability. The three-column system of Denis has been devised for assessment of spinal cord stability in trauma and divides the spine into the anterior (A), middle (M), and posterior (P) columns. The spine is considered unstable if two of the three columns are disrupted. The six-column system of Kostuik and Errico is devised for the evaluation of stability in spine tumors. The three columns, as defined by Denis, are subdivided into left (L) and right (R) halves. The spine is unstable if three to four of the columns are destroyed.

**FIGURE 7: CLASSIFICATION SYSTEMS FOR SPINAL STABILITY**

**- Surgery Only**

For years the only therapy used was urgent laminectomy followed, if appropriate, by radiotherapy. The proportion of patients who significantly benefitted from traditional decompressive laminectomy alone was 17% in one large series (Constans) and 20% in another study (Sorenson 1990A). The operative mortality has ranged from 3-14% in various series with an average mortality of 9% (Black 1985A). Worsening of neurological status was reported in various studies, with an overall mean of 12% (Black 1985A). Surgical morbidity following laminectomy included wound infection, CSF leak and instability with subluxation of the spine in approximately 11% (Black 1985A). In summary, the hazards of laminectomy alone consist of an estimated 9% risk of death, 12% risk of neurological worsening and 11% risk of other complications (Denis; White 1978 ; Kostuik ) (Fig 7 – Pg 60).

In our series, the primary mode of therapy was laminectomy, either with complete excision of tumour (40,6%) or debulking of tumour mass (19,5%), or laminectomy alone (14,6%). Our operative mortality was  $(1/106 = 0,9\%)$  which is favourable when compared to others . Our surgical morbidity ( 16 % ) was slightly higher , but, mirrored the results of other series . Wound infection, CSF leak or meningocele and instability of the spine resulted in a total complication rate of (16%).

At one time, the prognosis after decompressive laminectomy for epidural metastases was so poor that Elsberg and Shenkin considered laminectomy contra-indicated in patients with metastatic epidural tumours. (Elsberg; Shenkin) Torma in the largest series to date, reported 250 cases in 1957 and concluded that if the patient was known to have extradural metastases from systemic cancer, decompressive laminectomy was not warranted. (Törmä) However, advances in surgical technique and improved post-operative radiotherapy, have changed this picture (Smith; White 1971).

Hall and McKay (Hall) in a series of 118 patients with spinal metastases, reported that decompressive laminectomy improved 39% of the patients where the tumour was posterior; 35% where the lesion was lateral, 25% where the tumour was circumferential and only 9% when the lesion was in the anterior epidural space.

Why are the results of surgical decompression so poor? The reason lies in the anatomy of the metastatic spinal cord compression. The majority of epidural tumours arise in the vertebral body (Arseni; Barron; Törmä), invade the epidural space anteriorly, and remain largely anterior to the spinal cord. The vertebral body at the level of the cord compression is often destroyed. The surgeon approaches the tumour posteriorly and is usually unable to remove substantial parts of the mass without damaging the spinal cord. If the cord is not adequately decompressed, worsening compression eventually causes spinal cord infarction, usually within the white matter (McAlhany). This suggests that because laminectomy is effective primarily for posteriorly and laterally placed tumours, an anterior or antero-lateral surgical approach to the spine is needed when the tumour or bone mass is located ventrally.

In our series of 27 cases of metastatic spinal disease, 8 patients (29,6%) died within 1 year of diagnosis. Two of these patients had undergone laminectomy only and the remaining 6 underwent biopsy and radiotherapy (3), or radiotherapy only (3). Of those patients with malignant epidural spinal disease who survived beyond 1 year (14,8%), 3 of these survivors with spinal metastases had undergone an anterior approach with a vertebrectomy and stabilization and postoperative radiotherapy. The remaining survivor presented with multiple myeloma and underwent laminectomy combined with postoperative radiotherapy and survived beyond 5 years.

**- Radiotherapy Only**

Gilbert et al (Gilbert) based on a study of 235 patients suggested that primary radiotherapy might be as effective as laminectomy. They compared their radiotherapeutic results against a surgical group who were pre-selected, in that they had either had previous radiotherapy, a more rapid onset or did not have a proven primary. Gilbert et al concluded that the primary radiotherapy was as effective as laminectomy, but the scientific foundation of that conclusion was suspect because it was not a double - blind randomized controlled prospective study. Other studies have also shown that radiotherapy to the area of the spine involved with metastatic tumour produced significant neurological improvement (over pre-treatment status) in 30-45% of patients . (Black 1985; Constans; Gilbert; Young). Findlay in 1984 approached the question by reviewing all the publications on the topic over the previous 25 years (Findlay 1984). In summary, there was accurate data concerning the over 1800 cases available. No significant difference in outcome between patients treated by either primary radiotherapy or by laminectomy and subsequent radiotherapy, was found. Overall, 71% of those who were still ambulant at the time of therapy, retained their ability to walk. Of those who were paraparetic and non-ambulant, only 37% regained the ability to walk. Overall, 20% of the patients in that review actually deteriorated, either from ambulant to paraparetic but non-ambulant, or to become paraplegic despite apparently optimum therapy.

Radiosensitive tumours such as Hodgkins and non-Hodgkins lymphoma, multiple myeloma, seminoma, and neuroblastoma, have slightly better results with an improvement rate of about 50% (Black 1985). Tumours that are less sensitive to radiotherapy, including the carcinomas, melanomas, and soft tissue sarcomas, have a less favourable outlook (Gilbert).

There was some concern that radiation therapy may have resulted in cord swelling/oedema and caused neurological deterioration. Experimental studies, however, do not support the concept of radiation oedema. It has been found that more modern, high-dose radiotherapy regimes actually did cause acute shrinkage of the tumour without significant cord oedema. (Rubin; Ushio 1997A; Ushio 1997B).

Further, Gilbert et al (Gilbert) actually looked at their cases who presented with rapid deterioration and found that radiotherapy was markedly superior to laminectomy in outcome. To minimize the risk of radiation myelopathy, fractionation schedules of 2000 cGy in 5 fractions or 3000 cGy in 10 fractions have been recommended as safe regimes for the thoracic spinal cord (WARA).

Little information exists in the radiotherapy literature about the effect of vertebral collapse on outcome, but, Tomita et al (Tomita) certainly showed a much poorer outcome from radiotherapy in this situation, even in the presence of apparently radiosensitive lesions.

In our series, 7 patients received radiotherapy only and the majority were patients with advanced malignant spinal disease and poor neurological status preoperatively. Forty-one of our patients received radiotherapy after a surgical procedure for malignant spinal disease. We did not compare the results of surgery vs radiotherapy or combined therapy in our series as it was standard that all patients with malignant spinal disease and a relatively good prognosis for survival would get radiotherapy post-surgery. All of the 7 patients who received radiotherapy only, died within one year of treatment.

**- Surgery and Radiotherapy**

Brady et al (**Brady**), compared surgery with radiotherapy from the same institution. They noted an improvement of 61% in 90 patients treated with surgical decompression followed by radiotherapy and compared that with the response rate of 29% in 24 patients receiving decompressive laminectomy alone and 47% in 19 patients receiving radiotherapy alone. They concluded that the combination of surgery and radiotherapy was likely to yield the best response.

In various reported series of combined therapy in which laminectomy was the surgical technique employed, pooled data revealed a mean improvement rate of 44% (**Black 1985**); **Constans; Sorensen 1990**). In 2 of the large retrospective series, the combination of laminectomy plus radiotherapy resulted in improved neurological status in a mean of 36% of patients, compared with 26% of those treated with radiotherapy alone and 18% with surgery alone (**Constans; Sorensen 1990**). A prospective study of 29 patients comparing the combination of laminectomy plus radiotherapy vs radiotherapy alone, revealed improvement in 16% of patients in both groups (**Young**).

**Sundaresan et al (Sundaresan 1991)**, in a prospective trial involving 54 patients undergoing surgery for spinal compression, showed that all 54 patients improved after surgery with 37 of those patients undergoing external radiation therapy after surgery. Twenty-three of 25 patients surviving at 2 years continued to be ambulatory (**Sundaresan 1991**).

For tumours of the lymphoma type, the results after combined therapy were considerably better, with a pooled improvement rate of 68%. (**Black 1985**).

Wright (**Wright**) achieved a 50% ambulatory rate among 17 patients treated with post-operative radiotherapy, but only 40% in patients treated by surgery alone. Wild and Porter (**Wild**) reported that 44% of their patients irradiated after surgery became ambulatory, whereas only 26% of those not irradiated did so.

#### *- Anterior Surgical Approaches*

Little attention has been paid to the significance of the presence of vertebral body collapse on the outcome with either therapies. Brice & McKissock in 1965 (**Brice**) did observe that no patients with severe neurological deficit with collapse improved following laminectomy. Hall and McKay in 1973 (**Hall**) found that only 9% of their patients with collapse improved after laminectomy. Findlay in 1987 (**Findlay 1987A**) examined the role of vertebral collapse in a series of patients undergoing laminectomy. He found that those with collapse had a much worse outcome, with only 15% remaining ambulant and over 50% becoming paraplegic. In addition, 22% of those undergoing laminectomy in the presence of vertebral body collapse, develop spinal instability (**Denis; White 1978**).

The reason for the apparent failure of posterior techniques to improve outcome, lies in the fact that the majority of patients have an anterior extradural mass as either the sole cause or part of the spinal cord compression. (**Siegal 1985A**). In early 1980's, reports began to appear concerning an anterior approach to the spine for metastatic epidural cord or cauda equina compression. The technique consists of vertebral body resection and stabilisation. (**Cooper 1993; Harrington 1984; Siegal 1982; Sundaresan 1985; Siegal 1985B; Siegal 1985A; Sundaresan 1986A; Perrin; Turner**).

These studies show remarkably consistent results with between 76% and 83% of the patients being able to walk following anterior operation. A high percentage of patients also experienced pain relief (Harrington 1984; Conley). Mortality ranged from 0-7%, neurological worsening 0-6%, and other morbidity 10-15%. (Cooper 1993; Harrington 1984; Siegal 1982; Sundaresan 1985).

Although the average mortality and operative morbidity are roughly comparable for both the anterior and posterior (laminectomy) approaches, the anterior approach seems to be superior with respect to the prospect of neurological improvement and the lower chance of neurological worsening.

In our series, 3 cases of metastatic spinal involvement underwent an anterior approach with vertebrectomy and fusion with good results. All survived to 1 year follow-up with Frankel Scores of D and E. The majority of our patients with metastatic spinal lesions underwent laminectomy and post-operative radiotherapy. One patient improved from Frankel B to Frankel C and another improved from Frankel C to Frankel D. Our 1-year follow-up revealed 16 deaths and of these, 8 were due to metastatic spinal disease. Our results could have been better if the anterior approach had been an option in patients with a relatively good prognosis and pre-operative neurological function.

*- Chemotherapy*

Chemotherapy has not been shown to be of benefit for spinal metastatic carcinoma, but may be useful when combined with radiotherapy in cord compression secondary to lymphoma Hodgkin's disease, seminoma, Ewing's sarcoma (Posner).

**- Conclusion**

A wide range of therapies are available for the modern management of metastatic spinal disease, ranging from symptomatic treatment to aggressive surgery. No single approach will accommodate all patients and the therapeutic choice must be tailored to the individual situation. Surgical decompression - by laminectomy or by an anterior approach - is carried out initially to effect prompt relief of cord compression. An internal stabilisation procedure (with methyl methacrylate and/or bone graft, metal rods, wire or plates) is added when spinal stability needs to be restored (Conley; Harrington 1984; Siegal 1982; Sundaresan 1985).

**- Indications for surgical intervention are:**

- (i) Spinal instability or compression by bone.
- (ii) Failure to respond to radiotherapy.
- (iii) Known radioresistance of the tumour.
- (iv) Previous radiation exposure of the spinal cord.
- (v) Diagnosis in doubt/tissue biopsy.
- (vi) Repeat surgical decompression for recurrent tumour.
- (vii) Intractable pain which does not respond to radiotherapy.

These indications are relative rather than absolute because other considerations must be taken into account including general medical condition, histology of the primary tumour, extent of metastatic disease elsewhere and the patient's attitude concerning aggressive treatment. Minimum life expectancy of 3-4 months appears reasonable in an ideal circumstance (Cooper 1993). However, in our environment, this may not be totally applicable.

Post-operative radiotherapy is intended to eradicate or suppress the growth of residual tumour tissue. Reduction of the tumour burden (by surgical resection) is also thought to enhance the effectiveness of radiotherapy (**Brice; Harrington 1984**). Irradiation, however, may compromise fusion with bone so that, in some cases, the total dose of radiation may need to be reduced (**Harrington 1984**). Some authors, however, report the use of bone graft without the mention of their being affected by subsequent radiotherapy (**Conley; Siegal 1982; Sundaresan 1985**).

Radiotherapy may be the primary mode of therapy for the management of metastatic disease of the spine in the absence of neurological deficit, or where pain is the main symptom. It is most clearly indicated for radiosensitive tumours such as lymphomas and myelomas. In our series, where we had 6 patients with myeloma and 1 with lymphoma, only 1 patient with myeloma had survived up to 5 years after surgery and radiotherapy.

*- Adjuvant Therapy*

*- Corticosteroids*

Corticosteroids are a vital adjunct in the treatment of metastases causing spinal cord compression (**Cantu; Delattre; Posner**). The improvement in neurological function after steroid administration, documented in both animal investigations and human studies, is attributed to a reduction of spinal cord oedema near the compression site (**Ushio 1977B; Ushio 1977A; Delattre; Greenberg**).

Serious complications, thought to be secondary to high dose steroids, occurred in 40% of patients (**Heimdal**). It has been reported that steroids (dexamethosone) may have an oncolytic effect on spinal epidural metastases (**Posner**). In common practice, corticosteroids in standard dosage are recommended for their protective effect on the spinal cord and nerve roots, and serve as a supplement to radiotherapy or surgical decompression.

### **Primary Malignant Tumours Of The Spine**

As with most lymphoproliferative diseases, plasma cell neoplasms are usually quite radiosensitive and tend to melt away with even low doses of conventional radiotherapy (Weinstein 1992). It is uncommon for these lesions to come to surgery. Surgical indications would be in those new patients who present with progressive neurological deficit secondary to canal encroachment by vertebral compression fracture and associated soft tissue mass or in those with known myeloma and recurrent lesions in areas already maximally radiated.

Surgical removal is the standard treatment for chordoma. Aggressive surgical resection offers the best chance for a prolonged survival. These tumours are highly resistant to standard radiotherapy and chemotherapy. Complete resection of sacral chordomas can be achieved by a combined anterior and posterior surgical approach (Stener; Sundaresan 1990B).

A sacrectomy should be performed above the highest level of involvement in the non-sacral spine, resection of the involved vertebrae and all gross tumours where possible (Sundaresan 1990B). Proton and Photon irradiation therapy has been reported to achieve a significant success rate in local disease control.

In our series, there was one case of chordoma of the sacrum that required several operations to debulk tumour (anterior and posterior) and was resistant to radiotherapy. The tumour displayed aggressive growth patterns over more than a 10-year period.

The majority of chondrosarcomas involve both anterior and posterior elements of the vertebrae by the time of clinical presentation. Most are quite resistant to conventional radiotherapy and chemotherapy. This combination of resistance to adjunctive agents and the tendency to recur locally for prolonged periods before any evidence of metastases appear, makes a radical surgical approach the most appropriate for this lesion (Sundaresan 1990A).

We had 2 patients with chondrosarcomas and both underwent debulking surgery and both died within a year of treatment.

In view of the dismal prognosis using traditional techniques of debulking or decompression followed by radiation therapy for Ewing's Sarcoma and Osteosarcoma. Sundaresan, Weinstein (Sundaresan 1988; Weinstein) and others have recently advocated radical spondylectomy followed by radiation therapy and aggressive polychemotherapy, using agents that have proved successful in appendicular lesions. A few long-term survivors have been reported using these techniques.

### **Benign Epidural Tumours**

The treatment of neurilemmomas, neurofibromas and meningiomas is total surgical excision.

### ***- Benign Tumours of the Bony Spine***

The mainstay of treatment is complete surgical excision when possible, although in select instances intralesional curettage may be curative. Complete excision may not be possible, depending on the location, size and aggressive nature of the tumour. An anterior, posterior or combined approach may be indicated and, depending on the amount of resection and decompression obtained, stabilisation with fusion and/or instrumentation may be necessary. Adjuvant radiation therapy, chemotherapy, pre-operative embolisation and the use of other treatments such as cryosurgery, are decided upon on an individual basis. We did not have any patient with a benign bony tumour in our series, and this was probably due to our selection bias.

## CHAPTER 8

### INTRADURAL SPINAL TUMOURS

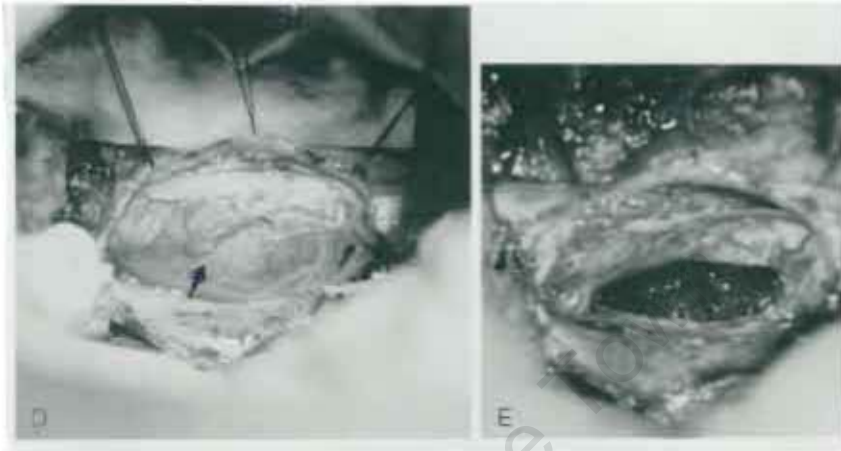
#### **Intradural Extramedullary (Plate 4 – Pg 74)**

Common extramedullary-intradural tumours which include meningiomas and nerve sheath tumours, are benign and, if they are carefully and thoroughly removed, leads to an excellent prognosis. The immediate results and prognosis of these tumours have been well established (Mork; Russell; Sloof; McCormick 1990A). The first tumour removed was probably a Schwannoma (Gowers). The intraoperative photograph clearly shows the extent to which the cord can be compressed by a benign intradural tumour.

Spinal intradural extramedullary meningiomas and schwannomas have similar surgical results although the meningiomas have a higher incidence of recurrence (Solero). Levy et al (Levy 1982A) reported a 4% incidence of symptomatic recurrence. Philippon in 1986, had a similar experience with recurrences occurring from 5-16 years after complete tumour removal. (Philippon) Mirimanoff et al in 1985 computed a risk of recurrence after total tumour removal of nil within the first 5 years and 13% at 10 years. (Mirimanoff) We had 1 recurrent meningioma within 5 years after total excision. This patient had multiple meningiomas which showed particularly aggressive behaviour (1/12=8,3%).

The prospects of post-operative neurological recovery in patients with Schwannomas are good even in those who had severe pre-operative neurological deficits. Levy et al (Levy 1982A), reported that 85% of their patients had a good neurological recovery 6 months after surgery, and on long-term follow-up, 91% had a good functional result. This is similar to the series reported by Solero et al where 92% of their patients made a good functional recovery on prolonged follow-up

**(Solero).** Solero et al had a series of 174 spinal meningiomas. They observed that 31 of their 37 patients with severe pre-operative neurological disability showed significant improvement on long-term follow-up, and only 6 patients were left generally disabled. Eighty-nine of their cases had mild pre-operative neurological dysfunction and 4 (4.5%) deteriorated after surgery.



Tumour pre-incision

Post excision

PLATE 4 INTRAOPERATIVE PHOTOGRAPH - INTRADURAL / EXTRAMEDULLARY  
TUMOUR (SCHWANNOMA)

In our series, of 12 spinal meningiomas, there were 2 patients who presented with a poor neurological grade prior to surgery (1 x Frankel A and 1 x Frankel B). Both underwent laminectomy and complete excision. The Frankel A patient (symptoms 2/12) improved to Frankel D and the Frankel B patient (symptoms 9/12) improved to Frankel E at 1-year follow-up. The remaining 10 patients presented with good neurological grades (D & E) and remained at good neurological grades after surgery.

In our 53 patients with nerve sheath tumours, 6 patients presented with poor neurological grades pre-operatively (3 "B" and 3 "A"). However, only 1 of these 6 (16,7%) (Frankel B - neurofibroma - symptoms 3/12) improved post-operatively to Frankel E. The others failed to recover even with similar symptom duration. Three of these patients had Type I neurofibromatosis.

From the above, it can be seen that meningiomas may have a slightly better prospect of recovery after total excision. The reoperation rates are higher in this series for nerve sheath tumours because of the relatively high incidence of patients with familial neurofibromatosis (15).

Giuffre, in 1976, reviewed the surgical results of 132 intradural spinal lipomas - 27% of the patients improved after decompression and biopsy, whereas 58% improved when the tumour was debulked (Giuffre). It is claimed that early prophylactic resection of the lipoma and untethering of the spinal cord is sufficient to allow long-lasting neurological stability or even improvement in many cases (McClone). Of our 7 cases of lipoma, all cases survived to 1 year follow-up with debulking surgery of the lipoma and untethering of the spinal cord in 5 patients with lumbosacral lipoma, and 2 patients had debulking surgery of the lipoma for thoracic cord compression.

## **Intramedullary Tumours (Plate 5 – Pg 78 )**

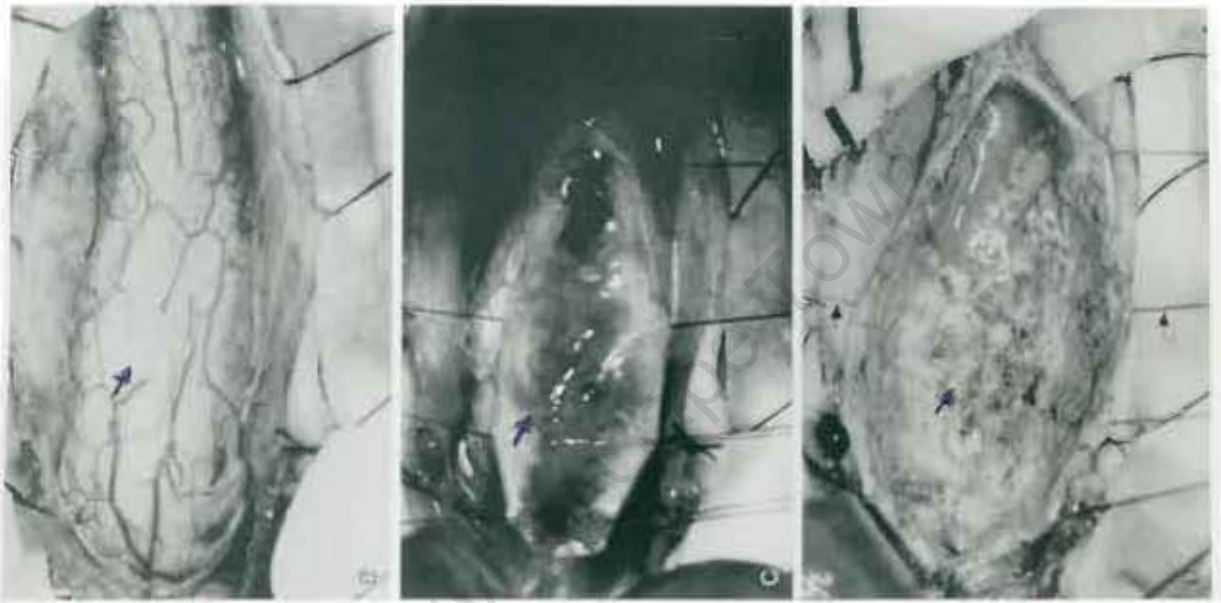
### **- Surgery**

The role of surgery in the management of intramedullary tumours of the spinal cord has been expanded in recent years. Previously, surgical management of an intramedullary mass was limited to biopsy, cyst aspiration or visual inspection alone, usually followed by radiation therapy (Slooff; Wood). Pioneering neurosurgeons, such as Elsberg and Frazier (Elsberg; Frazier), recognised the resectability of certain intramedullary tumours several decades ago. Recent advances in imaging and microsurgical technique as well as accumulating experience with the benign nature of most intramedullary growths, have established microsurgical removal as the single most effective treatment for most intramedullary tumours. (McCormick 1992C; McCormick 1990C; McCormick 1990B; Rawlings).

Recovery from a significant, long-standing defect rarely occurs. Neurological improvement from a pre-operative deficit is usually modest. Unfortunately, it is the minor deficits which are most likely to improve. Thus, the major benefit of intramedullary tumour removal is prophylactic. Worsening of an existing motor deficit is also common, but usually transitory. Recovery to pre-operative level or better usually occurs 3-6 months after surgery. (McCormick 1990B, 1992; 1990C; Stein).

In our series of 10 intramedullary tumours (5 astrocytoma; 5 ependymoma), 2 patients with high-grade glioma died within 2 years of treatment. Four patients presented with severe neurological deficits (Frankel A). Two of these patients had ependymomas - 1 died after surgery, and on follow-up the other remained at Frankel B. Both cases of glioma remained at Frankel A on follow-up at 1 year. In the single patient with spinal cord glioma presenting as Frankel C, there

was post-surgical neurological deterioration to Frankel A. Three patients with low grade ependymomas that were resected, survived beyond 1 year.



Cord-pre-incision

myelotomy- tumour visible

post excision-tumour bed

**PLATE 5 : INTRAOPERATIVE PHOTOGRAPH – INTRAMEDULLARY TUMOUR  
(EPENDYMOMA)**

This underscores the importance of early diagnosis and aggressive initial surgical treatment before a significant neurological deficit develops. Ependymomas that are totally removed rarely recur whereas subtotally resected ependymomas, whether irradiated or not, slowly progress (Schweitzer; McCormick 1990C). In treating adult benign spinal cord astrocytomas, approximately 40% are resectable (McCormick 1990B; Stein).

Long-term follow-up of Stein & McCormick's published series of 23 patients who underwent gross total removal of benign intramedullary ependymomas, reveals only 1 radiographic tumour recurrence, 6 years after total removal and was completely removed at the second operation (McCormick 1990C; Stein).

The recurrence rates following various degrees of removal of benign intramedullary astrocytomas is about 25-30% in 5 years. Age, rather than the degree of resection, seems to be the most important prognostic factor.

Cristante (Cristante) reported that tumour location seems to have had a significant influence on final outcome in their series of patients. These results showed that patients affected by tumour located in the cervicothoracic and upper thoracic region, developed more pronounced post-operative deficits and their recovery was less satisfactory. Other authors have found that cervical tumours are associated with the least post-operative morbidity but surgical morbidity appears to be greatest for tumours located in the lumbar enlargement and conus medullaris cord segments (McCormick 1992 & 1990C).

When only a portion of the tumour has been removed, the subsequent course depends upon the growth pattern of the tumour. Most patients affected by tumours with a large solid component, have a tendency for more pronounced post-operative sensory deficits (Cristante). Their recovery, although satisfactory, is delayed.

In our series, we found that tumours in the lower thoracic segments had slightly better outcomes when compared to the other segments.

(Pre-operative Frankel D + E = 16)

(Postoperative Frankel D + E = 19)

Malignant tumours are associated with significant operative morbidity and carry a poor prognosis. Surgical manipulation may result in tumour seeding via the CSF. (McCormick 1992 & 1990C; Cristante).

#### ***- Radiotherapy***

The results of radiotherapy for a primary malignancy of the spinal cord have been disappointing. The role of radiotherapy in the management of benign intramedullary spinal cord tumours, is uncertain (Epstein 1992; Cristante). Nevertheless, radiation therapy may provide a degree of tumour control in some patients with low-grade ependymomas or astrocytomas (Whitaker; Garcia; Schwade; Shaw 1986; McCormick 1990B). Routine radiation therapy is no longer accepted by some (Cristante). Patients with low-grade tumours are best treated with aggressive surgery and watchful waiting than adjunctive therapy (Epstein 1992; Wood; Stein; Garrett).

Clearly the use of radiotherapy to treat intramedullary tumours is controversial (**McCormick 1990B & 1990D; Whitaker**). Some reports reveal that the follow-up is purely clinical and too short relative to the normal evolution of these tumours. (**Schwade; Wood**). Lacking definite evidence of a beneficial effect of radiation upon benign intramedullary tumours, most authors do not recommend such treatment for benign pathology. Rather consider a second operation, should the clinical and radiological situation so dictate.

In the presence of tumour recurrence, a second surgical procedure may be carried out without any greater morbidity than the primary one and, following this, radiation might be considered or again deferred (**Epstein 1992; Cristante**).

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## CHAPTER 9

### OUTCOME

(Table 6b – Pg 42)

#### **Metastatic Epidural Tumours**

The factor of prime significance in determining outcome is the histology of the primary neoplasm which has an important bearing on both survival and the potential for recovery of neurological function (**Barcena; Brice**). Approximately 30% of patients with malignant spinal cord compression may survive beyond a year, and, rarely some may survive up to 4 - 9 years (**Gilbert; Hall**). Those with widespread malignant disease usually survive for only a few months.

Patients with myeloma, lymphoma, Ewing's sarcoma, neuroblastoma or carcinoma of the breast, have a favourable prognosis for recovery of function. Secondaries from bronchogenic lesions have a poor prognosis. (**Black 1985; Gilbert; Greenberg**). After radiotherapy, the median survival of patients with a lung primary is 1,5 months and for patient with breast primary the median survival is 9,2 months. (**Sorenson 1990**).

Perhaps the most important prognostic indicator is the clinical condition of the patient at the time of the surgery (**Karnofsky**).

There is general agreement that the more severe the pre-treatment neurological deficit is, the worse is the outcome. (**Barcena ; Black 1985; Findlay 1984; Gilbert; Levy 1982B**). Seventy percent of the patients who can walk at the time of diagnosis retain that ability after treatment; 35% of those who are initially paraparetic become ambulatory, and only 5-7% (range 0-25%) of paraplegic patients regain the ability to walk. These success rates show no significant difference

between the two modalities of management when the proportion of tumours with favourable histology is comparable. (Barcena; Findlay 1984).

The success rates achieved by all therapies in paraplegic patients is different, depending on whether the patient has complete functional cord transection or whether there is preservation of some neurological function. (Barcena). The fact that only 2% of the former patients recover the ability to walk after treatment, as compared with 20% of the latter, indicates a strong prognostic significance of residual neurological function. Complete paraplegia carries a bad functional prognosis regardless of the mode of therapy employed.

Camins, Oppenheim & Perrin report the results of their series of 353 consecutive cases and state that patients who are walking at the time of surgery do best and have the highest likelihood of maintaining ambulation. Patients who are paralysed at the time of surgery do worse and have less than a 10% chance of recovering ambulation. (Camins 1996)

Findlay in 1984 (Findlay 1984), reported that of those patients who were still able to walk even with aids at the onset of treatment, 71% were still able to walk after the completion of therapy. The success in regaining ambulation in paraplegic patients fell to 30%. Patients who were truly paraplegic only rarely regained the ability to walk after therapy.

Unfortunately, the terms us to describe paralysis vary, which makes comparison of the different series difficult with regards to patients with mild weakness to complete paralysis. Hence our use of the Frankel Scale to try and standardise results. Our overall rate of ambulation at the 1-year follow-up mark was 70/103 (68%) patients compared to 98/123 (79%); patients pre-operatively when comparing all tumour types.

Four patients 4/26 (15,4%) with Frankel A & B grades pre-operatively regained ambulation in our series.

For the individual categories of tumours in patients with malignant tumours presenting with Frankel A & B, the number of patients regaining ambulation were 1/13 (7,7%) whilst in those with benign tumours and presenting with Frankel A & B, 3/13 (23,1%) regained ambulation.

There is general agreement that, in the presence of true paraplegia of greater than 24 hours duration, functional recovery is extremely rare (Findlay 1984; Black 1985). Sixty percent of patients who can walk at the time of diagnosis retain that ability after treatment, whereas only 35% of those who are initially paraparetic (mild to moderate weakness) become ambulatory (Bruckman).

Gilbert and Posner (Gilbert) analysed the results in 22 patients who had weakness that developed over less than 48 hours. Of the 9 patients who underwent surgical decompression (decompressive laminectomy), none improved. Of the 13 patients irradiated without surgery, 7 improved. The difference in response was significant, although the grades of neurological function was not compared pre-therapy.

Brady et al (Brady) reported that 61% of their patients who were ambulatory continued to be ambulatory after treatment, whether the treatment was surgical decompression or radiotherapy. Only 5% of their patients who were paraplegic regained ambulation after therapy, no matter what the therapy. They had a 37% recovery rate from "marked paresis" after surgery and radiotherapy. They also noted that the more severe the degree of involvement, the less good the recovery. Brady's patients who had surgery alone had a 29% response rate as opposed to 61% with post-operative radiotherapy.

Sundaresan et al (Sundaresan 1995), state that patients who have rapidly evolved deficits (less than 24 hours) probably are destined to have a poor outcome regardless of therapy, especially if the deficits progress on high dose steroid therapy. Surgery offers the potential of neurologic palliation, even though it may have little impact on overall survival. They recommend that emergency surgery be considered only in patients with unstable neurologic deficits on steroid therapy. They say that most patients stabilise long enough for more complete radiologic assessment.

### **Malignant Epidural Tumours**

In patients with multiple myeloma, the median survival time after diagnosis is 2 to 5 years although this tumour is sensitive to both chemotherapy and radiotherapy. In our series, 6 patients presented with multiple myeloma and only 1 patient survived 5 years.

For sacral chordomas, the median survival is about 5 years with 20-40% survival at 10 years post-treatment. (Sundaresan 1990B). For Ewing's sarcoma, Bradway & Pritchard (Bradway) in 1990 reviewed 19 patients with spinal Ewing's sarcoma treated by the combination of radiation and chemotherapy. Thirteen patients had died within 33 months and only 1 patient had survived more than 5 years. This indicates the very poor prognosis associated with this tumour, particularly when it presents with spinal involvement.

Radical surgical excision is the principal method of treatment for chondrosarcoma (Camins 1978). The prognosis with this tumour is related to the incidence of local recurrence. Debulking of the tumour, almost invariably results in recurrence. Chondrosarcomas are radioresistant although there may be some benefit in treating patients with radiotherapy for unresectable tumours or following inadequate surgical excision. (Suit).

### **Intradural Extramedullary Tumours**

The intradural extramedullary tumours surgical results for benign intradural tumours of the spine are usually excellent. The outcome is primarily related to the patient's pre-operative neurological condition, age and duration of symptoms. (McCormick 1990A; Solero). In meningioma surgery, following total resection, the reported recurrence rates for meningiomas is 1,3% at 5 years and 6% at 14 years, with recurrence rates of <15%, even with subtotal resection. (Levy 1982A; Solero).

Unresectable (or subtotally resected) spinal meningiomas may be treated using the adjunctive radiation therapy. (Barbaro; Kupersmith; Taylor). However, it remains to be shown that radiation therapy will be helpful in cases of residual or recurrent meningiomas of the spine.

### **Intramedullary Tumours**

The long-term outlook following removal of intramedullary spinal cord astrocytomas is variable. Prognostic factors such as patient age, pilocytic histology, gross circumscription and extent of surgical removal, have been cited by various authors. It is probably that these factors are inter-related and not independent variables. (Stein; McCormick 1992 & 1990C; Sandler).

#### ***- Other factors which may affect outcome are:***

##### ***- Rate of onset and progression***

Rapid onset and progression of neurological symptoms are associated with a worse prognosis than are gradual onset and slow progression. (Brice) Smith et al reported that patients with preoperative symptoms longer than 2 months showed greater reversibility of their deficits after surgical decompression. (Smith)

The rate of onset and progression is a more critical determinant than the duration of symptoms or deficits. Once paraplegia is complete, however, the duration of paralysis has a prognostic significance. Paraplegia lasting longer than 24 hours is often viewed as hopeless, especially if the paralysis has been of rapid onset.

*- Vertebral collapse*

The presence of vertebral collapse and compression of the cord combined with compression by the tumour mass makes the prognosis for recovery, less favourable. (Wright)

*- Segmental level of spinal cord or cauda equina compression*

There are differences in opinion regarding the critical level of the spinal cord where compression results in ischaemia of the cord and paralysis. White et al found that results were more favourable with tumours in the lower 2/3 of the thoracic cord (White 1971) Livingston et al found the reverse, with a more favourable result with tumours in the upper thoracic spine (Livingston) The disparity in results may reflect the variability in the collateral blood supply to the spinal cord. (Hughes)

*- Location of the tumour within the spinal canal.*

In the past, most of the approaches to tumour within the canal was done via a posterior route, and at the stage, produced relatively better results when compared to the anterior approaches (Brice; Hall; Black 1985). Some authors claimed a significantly better prognosis for a posterior, rather than an anterior compression (Barcena; Findlay 1984; Hall).

With the advance in microsurgical techniques and instrumentation, far better results are now being achieved with the anterior approaches (Sundaresan 1991).

- General condition of the patient has an obvious effect on outcome. In an attempt to assess the advisability of surgery in a more rational manner, Tokuhashi et al (1990) devised a scoring system based on 6 variables.( Tokuhashi)

- a. Karnofsky performance status.
- b. Number of extraspinal bony metastases.
- c. Number of metastases in the vertebral body.
- d. Metastases to major organs.
- e. Primary site of the cancer.
- f. The neurological status.

They found that patients scoring 5 or less points did not survive for more than 3 months after surgery, while patients scoring 9 or more had a high chance of surviving for longer than 12 months.

## CONCLUSIONS

The present scientific basis for decision making in the management of Spinal Cord Compression is incomplete, contradictory and, at best, based on class 3 data. Our retrospective study suffers from the same inadequacy, but concurs with the following observations.

1. Patients with meningiomas may have a slightly better prospect of recovery than nerve sheath tumours after excision, if both groups present with marked neurological deficits preoperatively (**FRANKEL A & B**).
2. Lower thoracic segments have a better outcome after treatment of spinal cord compression in those segments.
3. Some factors have emerged as important determinants of functional prognosis, viz:
  - a. The pretreatment neurological status of the patient is important in determining outcome after treatment. Patients with minimal neurological deficits or **FRANKEL D & E** fared better than the rest.
  - b. Tumour type and biology plays an important role, with benign intradural extramedullary tumours displaying the best prognosis for long term survival after complete excision.
  - c. Complete paraplegia of >24 hours and an abrupt onset <48 hours has a poor prognosis, hence the role of emergency surgery may be limited to those with unstable deficits on steroid therapy.
4. Operative treatment for spinal metastases should not be relegated to the realms of "last resort" or "salvage" surgery and the most appropriate operative approach (including anterior surgery) with stabilisation should always be considered.
5. Patients with ependymomas have a better chance of long-term survival than patients with spinal cord astrocytomas after surgical excision.

- In addition, we feel the use of the Frankel Scale offers a convenient and practical way of recording functional performance, both pre- and postoperative and is useful for the standardisation of comparative reporting.
  
- Decision making in the management of spinal tumours should rest on multi-disciplinary teamwork that involves neurosurgeons, orthopaedic surgeons, histopathologists and oncologists.
  
- Our study also emphasises the need for an on-site MRI to adequately evaluate our patients.
  
- Overall, the results of this series confirms that aggressive treatment of patients with poor neurological and clinical condition preoperatively (excluding benign intradural extramedullary tumour types), may not be appropriate and that conservative management may be more economically viable in our circumstances of limited resources.

**BIBLIOGRAPHY**

Adams JH. *Brain Biopsy: The Smear Technique for Neurosurgical Biopsies*. London: Chapman & Hall, 1981: 92-97.

Arguello F, Baggs RB, Duerst RE, Johnstone L, McQueen K, Frantz CN. Pathogenesis of vertebral metastasis and epidural spinal cord compression. *Cancer* 1990; **65**: 98-106.

Arseni CN, Simionescu MD, Horwath L. Tumors of the spine. *Acta Psychiatr Neurol Scand* 1959; **34**: 398-410.

Barbaro NM, Gutin PH, Wilson CB, Sheline GE, Boldrey EB, Wara WM. Radiation therapy in the treatment of partially resected meningiomas. *Neurosurgery* 1987; **20**: 525-528.

Barcena A, Lobato R, Rivas J, *et al.* Spinal metastatic disease: Analysis of factors determining functional prognosis and the choice of treatment. *Neurosurgery* 1984; **15**: 820-827.

Barr ML. *The Human Nervous System. An Anatomical Viewpoint*. 5<sup>th</sup> ed. Maryland: Harper & Row, 1988: 63-83.

Barron KD, Hirano A, Araki S, Terry RD. Experiences with metastatic neoplasms involving the spinal cord. *Neurology (Minneap)* 1959; **9**: 91-106.

Batson OV. The function of the vertebral veins and their role in the spread of metastases. *Ann Surg* 1940; **112**: 138-149.

Black P. Spinal metastases: Current status and recommended guidelines for management. *Neurosurgery* 1985; **5**: 726-746.

Black PM. Meningiomas. *Neurosurgery* 1993; **32**: 643-657.

Botterell EH, Fitzgerald GW. Spinal cord compression produced by extradural malignant tumors: Early recognition, treatment and results. *Can Med Assoc J* 1959; **80**: 791-796.

Bradway J. Ewing's tumor of the spine: diagnosis and clinical management. In: Sundaresan N, Schmidek H, eds. *Tumours of the Spine: Diagnosis and Clinical Management*. Philadelphia: WB Saunders, 1990: 235-239.

Brady LW, Antoniades J, Prasasvinichai S, *et al*. The treatment of metastatic disease of the nervous system by radiation therapy. In: Seydel ND, ed. *Tumors of the Nervous System*. New York: John Wiley & Sons, 1975: 177-188.

Brice J, McKissock W. Surgical treatment of malignant extradural spinal tumours. *Br Med J* 1965; **1**: 1341-1344.

Bruckman JE, Bloomer WD. Management of spinal cord compression. *Semin Oncol* 1978; **5**: 135-140.

Buwembo JEB. *Spinal compression in childhood*. Cape Town: University of Cape Town Press, 1996.

Camins M. Tumours of the Vertebral Axis: Benign, Primary Malignant, and Metastatic Tumours. In: Youmans JR, ed. *Neurological Surgery*. 4th ed. Philadelphia: WB Saunders Co, 1996: 3134-3162.

Camins MB, Duncan AW, Smith J, Marcove RC. Chondrosarcoma of the spine. *Spine* 1978; **3**: 202-209.

Cantu RC. Corticosteroids for spinal metastases. *Lancet* 1968; **2**: 912.

Carpenter MB. *Core Text of Neuroanatomy*. 3rd ed. Baltimore: Williams & Wilkins, 1985: 52-101.

Charkes ND, Sklaroff DM, Young I. A critical analysis of strontium bone scanning for detection of metastatic cancer. *Am J Roentgenol* 1966; **96**: 647-656.

Cobb C, Leavens M, Eckles N. Indications for non-operative treatment of spinal cord compression due to breast cancer. *J Neurosurg* 1977; **47**: 653-658.

Conley FK, Britt RH, Hanbery JW, Silverberg GD. Anterior fibular strut graft in neoplastic disease of the cervical spine. *J Neurosurg* 1979; **51**: 677-684.

Constans JP, de Divitiis E, Donzelli R, Spazianti R, Meder JF, Haye C. Spinal metastases with neurological manifestations: review of 600 cases. *J Neurosurg* 1983; **59**: 111-118.

Cooper PR, Epstein F. Radical resection of intramedullary spinal cord tumors in adults. Recent experience in 29 patients. *J Neurosurgery* 1985; **63**: 492-499.

Cooper PR, Errico TJ, Martin R, Crawford B, DiBartolo T. A systematic approach to spinal reconstruction after anterior decompression for neoplastic disease of the thoracic and lumbar spine. *Neurosurgery* 1993; **32**: 1-8.

Cristante L, Herrmann HD. Surgical Management of Intramedullary Spinal Cord Tumors: Functional Outcome and sources of Morbidity. *Neurosurgery* 1994; **35**: 69-76.

Dahlin D. *Bone Tumors. General Aspects and Data on 6221 Cases*. 3rd ed. Illinois: Charles C Thomas, 1978.

Dahlin D. *Bone Tumors: General Aspects and Data on 8,542 Cases*. Illinois: Charles C Thomas, 1986.

Delattre JY, Arbit E, Thaler HT, Rosenblum MK, Posner JB. A dose-response study of dexamethasone in a model of spinal cord compression caused by epidural tumor. *J Neurosurg* 1989; 70: 920-925.

Denis F. The three column spine and its significance in the classification of acute thoracolumbar spine injuries. *Spine* 1983; 8: 817-821.

De Villiers JC. Spinal Cord Compression. In: Mieny, Mennen, eds. *Principles of Surgical Patient Care*. Pretoria :Academica, 1990: 544-549.

Edelman RR, Warach S. Magnetic Resonance Imaging. *NEJM* 1993; 328: 707-715.

Edelson RN, Deck MDF, Posner JB. Intramedullary spinal cord metastases: clinical and radiological findings in 9 cases. *Neurology (Minneap)* 1972; 22: 1222-1231.

Elsberg CA. *Surgical Diseases of the Spinal Cord, Membranes, and Nerve Roots*. New York: Hueber, 1941: 499-502.

Epstein FJ, Farmer JP. Pediatric spinal cord tumor surgery. *Neurosurg Clin North Am* 1990; 1: 569-590.

Epstein FJ, Farmer JP, Freed D. Adult intramedullary astrocytomas of the spinal cord. *J Neurosurgery* 1992; 77: 355-359.

Findlay GFG. Adverse effects of the management of malignant spinal cord compression. *J Neurol Neurosurg Psychiatry* 1984; 47: 761-768.

Findlay G. The role of vertebral bodies collapse in the management of malignant spinal cord compression. *J Neurol Neurosurg Psychiatry* 1987A; 50: 151-154.

Findlay GFG. Compressive and Vascular Disorder of the Spinal Cord. In: Miller JD , ed. *Northfields Surgery of the Central Nervous System*. London: Blackwell Scientific Publications, 1987: 707-759.

Fisher G, Mansuy L. Total removal of intramedullary ependymomas: Follow-up study of 16 cases. *Surg Neurol* 1980; 14: 243-249. In: Cristante L, Herrmann HD , eds. *Surgical Management of Intramedullary Spinal Cord Tumors*. *Neurosurgery* 1994; 35: 69-76.

Frankel HL, Hancock D, Hyslop G, *et al*. The value of postural reduction in the initial management of closed injuries to the spine with paraplegia and tetraplegia. *Paraplegia* 1969; 7: 179-192.

Frazier CH. *Surgery of the Spine and Spinal Cord*. New York: Appleton, 1919. In: Menezes A, Sonntag V, eds. *Principles of Spinal Surgery*. New York: McGraw-Hill, 1996: 1369.

Galasko CSB. Skeletal metastases and mammary cancer. *Ann R Coll Surg Engl* 1972; 50: 3-28.

Garcia DM. Primary spinal cord tumors treated with surgery and post-operative irradiation. *Int J Radiat Oncol Biol Phys* 1985; 4: 1933-1939.

Garrett PG, Simpson WJK. Ependymomas: Results of radiation treatment. *Int J Radiat Oncol Biol Phys*, 1983; 9: 1121-1124.

Gilbert RW, Kim JH, Posner JB. Epidural spinal cord compression from metastatic tumour: diagnosis and treatment. *Ann Neurol* 1978; **3**: 40-51.

Giuffre R. Tumours of the Spine and Spinal Cord. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology Vol 20 Part II*. 1<sup>st</sup> ed. Amsterdam: North-Holland, 1976: 389-412.

Gowers WR. A case of tumour of the spinal cord. Removal: recovery. *Med-Chir Trans* 2nd s 1888; **53**: 377-428.

Greenberg HS, Kim JH, Posner JB. Epidural spinal cord compression from metastatic tumor: Results with a new treatment protocol. *Ann Neurol* 1980; **8**: 361-366.

Greenwood J Jr. Surgical removal of intramedullary tumors. *J Neurosurg* 1967; **26**: 276-282.

Guidetti B, Fortuna A. Surgical treatment of intramedullary hemangioblastoma of the spinal cord. Report of 6 cases. *J Neurosurg* 1967; **27**: 530-540.

Hall AJ, Mackay NNS. The results of laminectomy for compression of the cord and cauda equina by extradural malignant tumor. *J Bone Joint Surg [Br.]* 1973;**55**: 497-505.

Harrington KD. Anterior cord decompression and spinal stabilization for patients with metastatic lesions of the spine. *J Neurosurg* 1984; **61**: 107-117.

Harrington KD. Metastatic disease of the spine. *J Bone Joint Surg [Am]* 1986; **68**: 1110-1115.

Heimdal K, Hirschberg H, Slettobo H, *et al.* High incidence of serious side effects of high-dose dexamethasone treatment in patients with epidural spinal cord compression. *J Neuro-oncol* 1992; **12**: 141-144. In: Wilkins RH, Rengachary SS, eds. *Neurosurgery*. 2nd ed. New York: McGraw-Hill, 1996: 1804.

Helseth A, Mork SJ. Primary intraspinal neoplasms in Norway 1955 to 1986. A population-based survey of 467 patients. *Neurosurg* 1989; **71**: 842-845.

Herrmann H-D, Neuss M, Winkler D. Intramedullary spinal cord tumors resected with CO<sub>2</sub>-laser microsurgical technique: Recent experience in 15 patients. *Neurosurgery* 1988; **22**: 518-522.

Hoffman EB, Crosier JH, Cremin BJ. Imaging in children with spinal tuberculosis. *J Bone Joint Surg (Br)* 1993; **75**: 233-239.

Hollis PH, Malis LI, Zappulla RA. Neurological deterioration after lumbar puncture below complete spinal subarachnoid block. *J Neurosurg* 1986; **64**: 253-256.

Howe JF, Loeser JD, Calvin WH. Mechanosensitivity of the Dorsal Root Ganglia and Chronically Injured Axons: A Physiological Basis for the Radicular Pain of Nerve Root Compression. *Pain* 1977; **3**: 25-41. In: Buwembo JEB. ed. *Spinal compression in childhood*. Cape Town: University of Cape Town Press, 1996.

Hughes JT. *Pathology of the Spinal Cord*. London: Lloyd Luke, 1978: 218-241.

Huvos AG. *Bone Tumors, Diagnosis, Treatment and Prognosis*. 2<sup>nd</sup> ed. Philadelphia: WB Saunders, 1979:523-653.

Ikeda H, Ushio Y, Shimizu K, Mogami H, Hayakawa T. Experimental spinal cord compression by epidural neoplasms. *Neurol Surg (Tokyo)* 1978; **6**: 891-898. In: Black P, ed. *Spinal Metastases. Neurosurgery* 1985; **5**: 726-746.

Jaffe HL. Tumors and Tumorous Conditions of the bones and Joints. In: Menezes A, Sonntag V, eds. *Principles of Spinal Surgery*. New York: McGraw-Hill, 1996: 1419.

Johnston RA. The Management of Acute Spinal Cord Compression. *J Neurol, Neurosurg & Psychiat* 1993; **15**: 1046-1054.

Karnofsky DA. Karnofsky Scale. In: Greenberg MS, ed. *Handbook of Neurosurgery*. 3rd ed. Florida: Greenberg Graphics, 1994: 378.

Kostuik JP. Differential Diagnosis and Surgical Treatment of Metastatic Spine Tumours. In: Schmidek HH, Sweet WH, eds. *Operative Neurosurgical Techniques*. 3rd ed. Philadelphia: WB Saunders Co, 1995: 1997-2025.

Kupersmith MJ, Warren FA, Newall J, Ransohoff J. Irradiation of meningiomas of the intracranial anterior visual pathway. *Ann Neurol* 1987; **21**: 131.

Kurland LT. Frequency of intracranial and intraspinal neoplasms in the resident population of Rochester, Minnesota. *J Neurosurg* 1958; **15**: 627-641.

Lazorthes G, Gouaze A, Zadeh JO, Santini JJ, Lazorthes Y. Arterial vascularization of the spinal cord: recent studies of the anastomotic substitution pathways. *J Neurosurg* 1971; **35**: 253-262.

Levy WJ, Bay J, Dohn DF. Spinal cord meningioma. *J Neurosurg* 1982A; **57**: 804-812.

Levy WJ Jr, Latchaw J, Hahn JF, Sawhny B, Bay J, Dohn DF. Spinal neurofibromas: a report of 66 cases and a comparison with meningiomas. *Neurosurgery* 1986; 18: 331-334.

Levy WJ, Latchaw JP Jr, Hardy RW, Hahn JP. Encouraging surgical results in walking patients with epidural metastases. *Neurosurgery* 1982B; 11: 229-233.

Livingston KE, Perrin RG. The neurosurgical management of spinal metastases causing cord and cauda equina compression. *J Neurosurg* 1978; 49: 839-843.

Malawski SK. The results of surgical treatment of primary spinal tumors. *Clin Orthop* 1991; 272, 50.

Maurice-Williams RS, Richardson PL. Spinal cord compression; delay in the diagnosis and referral of a common neurosurgical emergency. *Brit J Neurosurg* 1988; 2: 55-60.

McAlhany HJ, Netsky MG. Compression of the spinal cord by extramedullary neoplasms. *J Neuropathol Exp Neurol* 1955; 14: 276-287.

McClone DG, Naidich TP. Laser resection of fifty spinal lipomas. *Neurosurgery* 1986; 18: 611-615.

McCormick PC, Post KD, Stein BM. Intradural extramedullary tumors in adults. *Neurosurg Clin North Am* 1990A; 1: 591-608.

McCormick PC, Stein BM. Intramedullary tumors in adults. *Neurosurg Clin North Am* 1990B; 1: 609-630.

McCormick PC, Stein BM. Evaluation of recovery following complete removal. In: Holtzman RNN, Stein BM, eds. *Surgery of the Spinal Cord: Potential for Regeneration and Recovery*. New York: Springer-Verlag, 1992: 245-260.

McCormick PC, Torres R, Post KD, Stein BM. Intramedullary ependymoma of the spinal cord. *J Neurosurg* 1990C; 72: 523-532.

Menezes AH, Sonntag VKH. *Principles of Spinal Surgery*. New York: cGraw-Hill, 1996: 785-799.

Mirimanoff RV, Dosretz DE, Lingood RM, Ojemann RG, Martuza RL. Meningiomas: analysis of recurrence and progression following neurosurgical resection. *J Neurosurg* 1985; 18-24.

Moran RM, Webb JK. Primary tumors of the spine. In: Findlay G, Owen R. eds. *Surgery of the spine: A Combined Orthopedic and Neurosurgical Approach*. Oxford : Blackwell Scientific Publications, 1992: 539-556.

Mork SJ, Loken AC. Ependymoma. A follow-up study of 101 cases. *Cancer* 1977; 40: 907-915.

Neumann HPH, Eggert HR, Weigel K, Friedburg H, Wiestler OD, Schollmeyer P. Hemangioblastomas of the central nervous system: A 10-year study with special reference to von Hippel-Lindau syndrome. *J Neurosurg* 1989; 70: 24-30.

Nittner K. Spinal meningiomas, neurinomas and neurofibromas, and hourglass tumours. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Vol 20 Part II. 1<sup>st</sup> ed. Amsterdam: North-Holland, 1976: 177-322.

Okazaki H. *Fundamentals of Neuropathology*. New York: Igaku-Shoin, 1983.

Perrin R, McBroom R. Spinal fixation after anterior decompression for symptomatic spinal metastasis.

*Neurosurgery* 1988; **22**: 334-327.

Philippon J, Cornu PR, Grob R, Rivierez M. Les meningiomes récidivantes. *Neurochirurgie* 1986; **32**: 54-

62. In: Findlay G, Owen R, eds. *Surgery of the spine: A Combined Orthopedic and Neurosurgical*

*Approach*. Oxford :Blackwell Scientific Publications, 1992: 586.

Posner JB, Howieson J, Cvitkovic E. "Disappearing" spinal cord compression: oncolytic effect of

glucocorticoids (and other chemotherapeutic agents) on epidural metastases. *Ann Neurol* 1977; **2**: 409-413.

Rawlings CE, Giangaspero F, Burger PC, Bullard DE. Ependymomas: A clinicopathological study. *Surg*

*Neurol* 1988; **29**: 271-281. In: Wilkins RH, Rengachary SS, eds. *Neurosurgery*. 2nd ed. New York:

McGraw-Hill, 1996: 1781.

Rubin P. Extradural spinal cord compression by tumour: Part I. Experimental production and treatment

trial. *Radiology* 1969; **93**: 1243-1260.

Ruff RL, Lanska DJ. Epidural metastases in prospectively evaluated veterans with cancer and back pain.

*Cancer* 1989; **63**: 2234-2241.

Russel DS, Rubinstein LJ. *Pathology of Tumors of the Nervous System*. 5<sup>th</sup> ed. Baltimore: Williams &

Wilkins, 1989: 192-214.

Sandler HM, Papadopoulos SM, Thornton AF, Ross DA. Spinal cord astrocytoma: Results of therapy.

*Neurosurgery* 1992; **30**: 490-493.

- Sarpel S, Sarpel G, Yu E, *et al*. Early diagnosis of spinal epidural metastasis by magnetic resonance imaging. *Cancer* 1987; **59**: 1112-1116.
- Schaberg J, Gainor BJ. A profile of metastatic carcinoma of the spine. *Spine* 1985; **10**: 19-20.
- Schwade JG, Wara WM, Sheline GE, Sorgen S, Wilson CB. Management of primary spinal cord tumors. *Int J Radiat Oncol Biol Phys* 1978; **4**: 389-393.
- Schweitzer JS, Batzdorf U. Ependymoma of the cauda equina region: Diagnosis, treatment, and outcome in 15 patients. *Neurosurgery* 1992; **30**: 202.
- Shapiro W, Posner J. Medical versus surgical treatment of metastatic spinal cord tumours. In: Thompson R, Green J, eds. *Controversies in Neurology*. New York :Raven Press, 1983: 57-65.
- Shaw EG, Evans RG, Scheithauer BW, Ilstrup DM, Earle JD. Radiotherapeutic management of adult intraspinal ependymomas. *Int J Radiat Oncol Biol Phys* 1986; **12**: 323-327.
- Shaw B, Mansfield FL, Borges L. One-stage posterolateral decompression and stabilization for primary and metastatic vertebral tumors in the thoracic and lumbar spine. *J Neurosurg* 1989; **70**: 405-410.
- Shaw M, Rose J, Paterson A. Metastatic extradural malignancy of the spine. *Acta Neurochir* 1980; **52**: 113-120. In: Youmans JR, ed. *Neurological Surgery*. 4th ed. Philadelphia: WB Saunders Co, 1996: 3122.
- Shenkin HA, Horn RC, Grant FC. Lesions of spinal cord epidural space producing cord compression. *Arch Surg* 1945; **51**: 125-146.

Siegel T, Siegal T. Surgical decompression of anterior and posterior malignant epidural tumours compressing the spinal cord: a prospective study. *Neurosurgery* 1985A; 17: 424-432.

Siegel T, Siegal T. Current considerations in the management of neoplastic spinal cord compression. *Spine* 1989; 14: 223-228.

Siegel T, Siegal T, Robin G, Korn IL, Fuks Z. Anterior decompression of the spine for metastatic epidural cord compression: a promising avenue of therapy? *Ann Neurol* 1982; 11: 28-34.

Siegel T, Tiqva P, Siegal T. Vertebral body resection for epidural compression by malignant tumours. *J Bone Joint Surg* 1985B; 67A: 375-382.

Slooff JL. *Primary Intramedullary Tumors of the Spinal Cord and Filum Terminale*. Philadelphia: Saunders, 1964:29-115.

Smith R. An evaluation of surgical treatment for spinal cord compression due to metastatic carcinoma. *J Neurol Neurosurg Psychiatry* 1965; 28: 152-158.

Solero CL, Fornari M, Giombini S, *et al*. Spinal meningiomas: Review of 174 operated cases. *Neurosurgery* 1989; 25: 153.

Sorenson PS, Borgesen SE, Rohde K, *et al*. Metastatic epidural spinal cord compression. Results of treatment and survival. *Cancer* 1990; 65: 1502-1508.

Sorensen SA, Mulvihill JJ, Nielson A. Long-term follow-up of von Recklinghausen neurofibromatosis: Survival and malignant neoplasms. *N Engl J Med* 1986; 314: 1010.

Stein BM, McCormick PC. Intramedullary neoplasms and vascular malformations. *Clin Neurosurg* 1992; **39**: 361-387.

Stener B, Gunterberg B. High amputation of the sacrum for extirpation of tumors: principles and technique. *Spine* 1978; **3**: 351-366.

Suit H, Goitein M, Munzenrider J, *et al.* Definitive radiation therapy for chordoma and chondrosarcoma of base of skull and cervical spine. *J Neurosurg*, 1982; **56**: 377-385.

Sundaresan N, DiGiacinto G, Hughes J. Surgical treatment of spinal metastases. *Clin Neurosurg* 1986A; **33**: 503-522.

Sundaresan N, DiGiacinto GV, Hughes JE, Cafferty M, Vallejo A. Treatment of neoplastic spinal cord compression: Results of a prospective study. *Neurosurgery* 1991; **29**: 645-650.

Sundaresan N, Galicich J, Lane JM, Bains MS, McCormack P. Treatment of neoplastic epidural cord compression by vertebral body resection and stabilisation. *J Neurosurg* 1985; **63**: 676-684.

Sundaresan N, Galicich J, Lane J, Scher H. Stabilisation of the spine involved by cancer. *The Unstable Spine*. New York: Grune & Stratton, 1986B. In: Findlay G, Owen R. eds. *Surgery of the spine: A Combined Orthopedic and Neurosurgical Approach*. Oxford : Blackwell Scientific Publications, 1992: 572.

Sundaresan N, Krol G, Hughes JEO. Primary malignant tumors of the spine. In: Youmans JR, ed. *Neurological Surgery*. 3d ed. Philadelphia : Saunders, 1990A: 3548-3573.

Sundaresan N, Rosen G, Huvos AG, Krol G. Combined treatment of osteosarcomas of the spine.

*Neurosurgery* 1988; **23**: 714-719.

Sundaresan N, Rosenthal D, Schiller A, *et al.* Chordomas. In: Sundaresan N, Schmidek H, eds. *Tumors of the Spine: Diagnosis and Clinical Management*. Philadelphia: WB Saunders, 1990B: 192-213.

Sundaresan N, DiGiacinto G, Hughes J. Surgical Management of Primary and Metastatic Tumours of the Spine. In: Schmidek HH, Sweet WH, eds. *Operative Neurosurgical Techniques*. 3rd ed. Philadelphia: WB Saunders Co, 1995: 1981-1984.

Taylor BW Jr, Marcus RB Jr, Friedman WA, Ballinger WE Jr, Million RR. The meningioma controversy: Postoperative radiation therapy. *Int J Radiat Oncol Biol Phys* 1988; **15**: 299.

Tokuhashi Y, Matsuzaki H, Toriyama S, Kawano H, Ohsaka S. Scoring system for the preoperative evaluation of metastatic spine tumour prognosis. *Spine* 1990; **15**: 1110-1113.

Tomita T, Galicich J, Sundaresan N. Radiation therapy for spinal epidural metastases with complete block. *Acta Radiol Oncol* 1983; **22**: 135-143.

Törmä T. Malignant tumors of the spine and the spinal epidural space: a study based on 250 histologically verified cases. *Acta Chir Scand* 1957; **225**: 1-138.

Turner P, Prince H, Webb J, Sokal M. Surgery for malignant extradural tumours of the spine. *J Bone Joint Surg* 1988; **70B**: 451-456.

Ushio Y, Posner R, Kim JH, Shapiro WR, Posner JB. Treatment of experimental spinal cord compression caused by epidural neoplasms. *J Neurosurg* 1977A; **47**: 380-390.

Ushio Y, Posner R, Posner J, Shapiro W. Experimental spinal cord compression by epidural neoplasm.

*Neurology (Minneap.)* 1977B; 27: 422-429.

Van der Sande JJ, Kroger R, Boogerd W. Multiple spinal epidural metastases: an unexpectedly frequent finding. *J Neurol Neurosurg Psychiatry* 1990; 53: 1001-1003.

Walton J. *Brain's Diseases of the Nervous System*. 10<sup>th</sup> ed. Oxford: Oxford University Press, 1993: 478-512.

Wara WM, Phillips TL, Sheline GE, Schwade JC. Radiation tolerance of the spinal cord. *Cancer* 1975; 35: 1558-1562.

Webb JH, Craig WM, Kernohan JW. Intraspinial neoplasms in the cervical region. *J Neurosurg* 1953; 10: 360-366.

Weinstein JN, McLain RF. Tumors of the spine. In: Rothman RH, Simeone FA, eds. *The Spine*. 3<sup>rd</sup> ed. Philadelphia : Saunders, 1992.

Whitaker SJ, Bessell EM, Ashley SE, Bloom HJG, Bell BA, Brada M. Post-operative radiotherapy in the management of spinal cord ependymoma. *J Neurosurg* 1991; 74: 720-728.

White AA III, Panjabi MM. *Clinical Biomechanics of the Spine*. Philadelphia: Lippincott, 1978: 423-431.

Wild WO, Porter RW. Metastatic epidural tumor of the spine. *Arch Surg* 1963; 87: 137-142.

Wong D, Fornasier V, MacNab I. Spinal metastases: the obvious, the occult, and the imposters. *Spine* 1990; **15**: 1-4.

Wood EH, Berne AS, Taveras JM. The value of irradiation therapy in the management of intrinsic tumors of the spinal cord. *Radiology* 1954; **63**: 11-24.

Wright RL. Malignant tumors in the spinal extradural space: results of surgical treatment. *Ann Surg* 1963; **157**: 227-231.

Young RF, Post EM, King GA. Treatment of spinal epidural metastases. Randomised prospective comparison of laminectomy and radiotherapy. *J Neurosurg* 1980; **53**: 741-748.