TOPIC:

COMPRESSIVE LESSIONS OF THE SPINAL CORD IN KENYA, AS SEEN AND TREATED IN THE NEUROSURGICAL DEPARTMENT IN KENYATTA NATIONAL HOSPITAL, JANUARY 1981 TO DECEMBER 1984.

A THESIS SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE (SURGERY) IN THE UNIVERSITY OF NAIROBI.(KENYA)

BY

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DECLARATION:

THIS THESIS IS MY ORIGINAL WORK AND HAS NOT BEEN PRESENTED FOR A DEGREE IN ANY OTHER UNIVERSITY COLLEGE.

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THIS THESIS IS SUBMITTED FOR EXAMINATION WITH MY APPROVAL AS A UNIVERSITY SUPERVISOR.

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CONTENTS:

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My special thanks go to Susan Thuo for secretarial help in the preparation of this thesis.

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22. Comparison of causes of cord compression.

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COMPRESSIVE LESIONS OF THE SPINAL CORD AS SEEN AND TREATED IN THE SECTION OF NEUROSURGERY, KENYATTA NATIONAL HOSPITAL.

OBJECTIVES:

I. TO DOCUMENT:

1. The symptomatology and clinical presentation.
2. The results of various clinical, laboratory and radiological procedures.
3. The diagnostic yield after clinical, laboratory, radiology, surgery and histology.
4. The immediate post operative outcome.
5. The various data for example the ages of patients, durations of illness, sex ratios, spinal levels, and causes of spinal compression as a base line for further studies on paraplegia.

II. To Compare and contrast some aspects of compressive spinal cord lesions as seen in other tropical and non tropical countries.

INTRODUCTION:

Compressive spinal cord lesions present by producing progressive paralysis with or without muscle wasting, sphincter disturbances, sensory loss, local, girdle, or radiating pain. One would expect such symptoms to be taken seriously by the patient and hence report early to hospital but this is not always so. In Kenya, as in many other developing countries, diagnosis is made late. Part of this delay may be due to great distances one has to travel to reach a specialist centre, or referral to the Neurosurgical unit in Kenyatta National Hospital. Part of this delay may be due to delay in referral from peripheral hospitals due to various problems, for example lack of suitable transportation.

The Neurosurgical unit at Kenyatta National Hospital is the only one in Kenya. Ideally patients from peripheral hospitals should be referred to the Medical Department, especially to the Neurological Clinic or wards for investigations before being referred to the Neurosurgeon. Fully investigated patients from Provincial Hospitals may however be referred to the Neurosurgical clinics directly. In practice this works well but occasionally univestigated patients reach the Neurosurgical clinic and have to be worked up from there. Worked up patients in the medical wards may be transferred to the Neurosurgical ward prior to surgery, after surgery or may be operated on and subsequently managed from the Medical wards.

This applies especially to those who have, in addition, a significant medical problem, for example diabetes, or those likely require medical treatment after surgery.

The patients reviewed here have been examined, investigated, and managed by several people. It is, therefore, not suprising that there is no uniformity, as the standard plan of management of such patients is often not adhered to at Kenyatta National Hospital. This being a retrospective study, and due to the various difficulties alluded to above, there was some difficulty in interpreting various records. Some records were incomplete or missing. Adequate records were however available for the patients studied.

Due to the small number at beds available to the Neurosurgical Section in Kenyatta National Hospital patients are discharged early either to the referring hospital or to the hospital nearest their rural homes for convalescence. The early operative outcome may therefore change with time. An incompletely removed tumour may recur, or paralysed sphincters may regain function. This information was not available for this study.

Review of the literature on non traumatic paraplegia from various developing countries is also presented.

SUMMARY:

The results of a retrospective study of 100 patients with proven compressive spinal cord lesions, operated on in the Neurosurgical Unit at Kenyatta National Hospital, Nairobi, Kenya, between January 1981 to December 1984 are presented.

The mode of presentation, the results of various investigations, the findings at surgery, and the results of surgery together with the histological diagnosis are presented.

The diagnostic yield of clinical, radiological, surgical methods and histology are evaluated. A detailed list of compressive cord lesions found in this study is presented and compared and contrasted with similar studies from other tropical and non tropical countries.

MATERIALS AND METHODS.

All the patients operated on between January 1981 to December 1984 with compressive spinal cord lesions whose complete records were available were included in this study. 100 such files were available. 20 files were either missing or were incomplete.

When the records were doubtful or ambiguous, confirmation was sought from the various departments. The radiology, pathology, operation theatre, and ward registers were checked in such cases.

The National Research Council muscle power gradation charts are used by most doctors, and physiotherapists in this country (Table 1). The lowest figure for the paralysed limb (s) was used. This figure was obtained from the referral letter, or notes of the admitting doctor, and confirmed from the physiotherapy chart.

Most laboratory investigations were available at the Kenyatta National Hospital during the period covered by the study.

Plain x-rays and tomograms were available during the period under study. Myelography was occasionally not available during the period under review due to lack of dye, or a non functioning machine. Histology services were available through out the period under review.

TABLE I

THE NATIONAL RESEARCH COUNCIL MUSCLE POWER GRADATION CHART.

GRADE 5	Contraction against powerful resistance	=	100%
GRADE 4	Contraction against gravity and some resistance	=	75%
GRADE 3	Contraction against gravity only	=	50%
GRADE 2	Movement only possible with gravity eliminated	=	25%
GRADE 1	Flicker of contraction but no movement	=	10%
GRADE 0	Complete paralysis	=	0%

TABLE II. THE AETIOLOGY OF SPINAL CORD COMPRESSION.

I.	CONGENITAL.....		16
	Craniovertebral anomaly	14	
	Narrow cervical spinal canal	1	
	Haemangioma	1	
II.	AQUIRED.....		79
	- Degenerative		27
	Cervical spondylosis	12	
	Prolapsed intervertebral disc	15	
	- Primary cord tumours		23
	- Intramedullary		2
	Cystic glioma	1	
	Astrocytoma	1	
	- Extramedullary		21
	Meningioma	10	
	Neurofibroma	6	
	Neurofibrosarcoma	1	
	Ganglioneuroma	4	
	- Secondary tumours -(Table IIa)		9
	Inflammatory		13
	Non specific inflammation	8	
	Tuberculosis	5	

- Trauma		3
	Old compression fractures	2
	Haemorrhage	1

III. MISCELLANEOUS 5

Bone spur	1
Acellular intramedullary tumours	3
Anular constriction of dura	1

TABLE II B. LIST OF SECONDARY TUMOURS

Anaplastic carcinoma	2
Lymphocytic lymphoma	2
Multiple myeloma	2
Unspecified metastatic tumour	1
Epithelial tumour(? hepatoma)	1
Embryonal tumour (? wilms)	1
	9

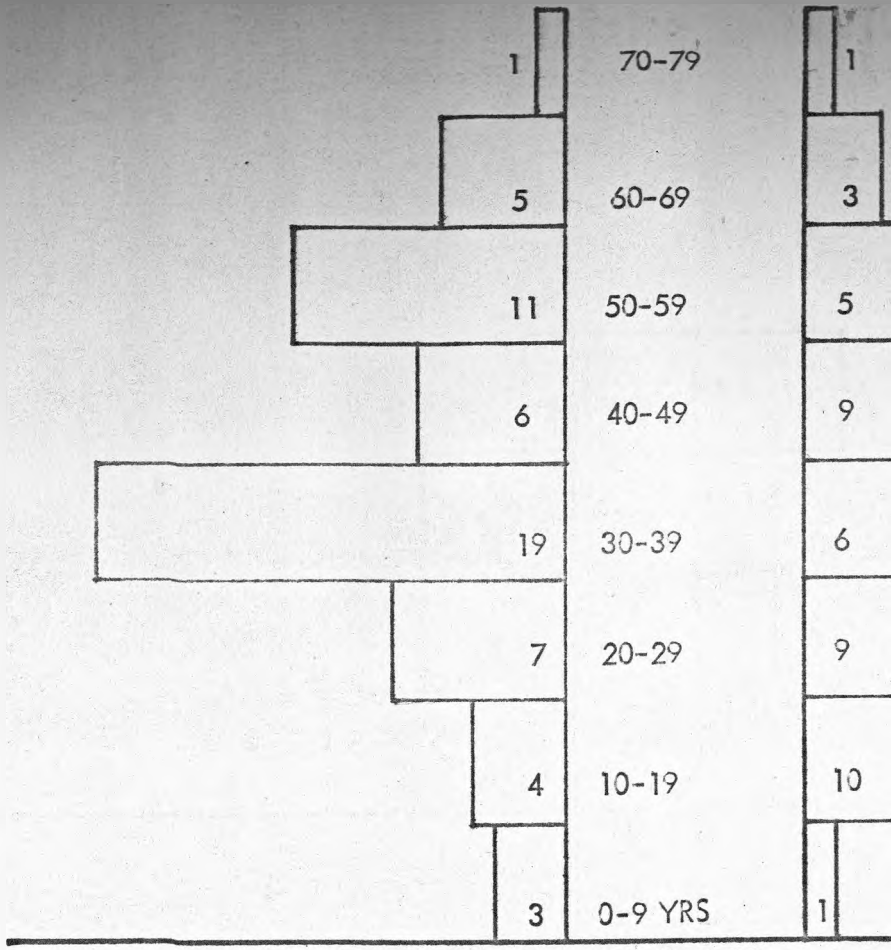
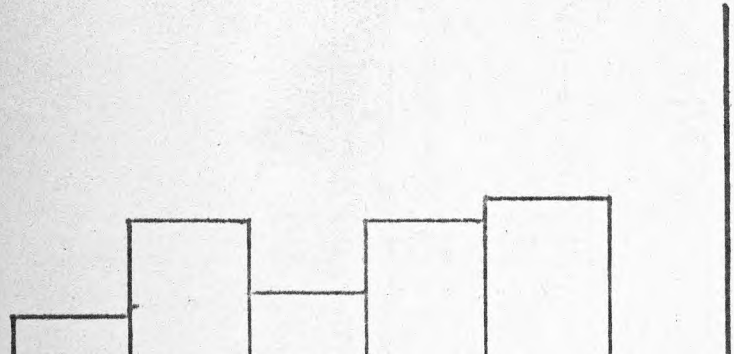


TABLE III. AGE DISTRIBUTION.


FEMALE

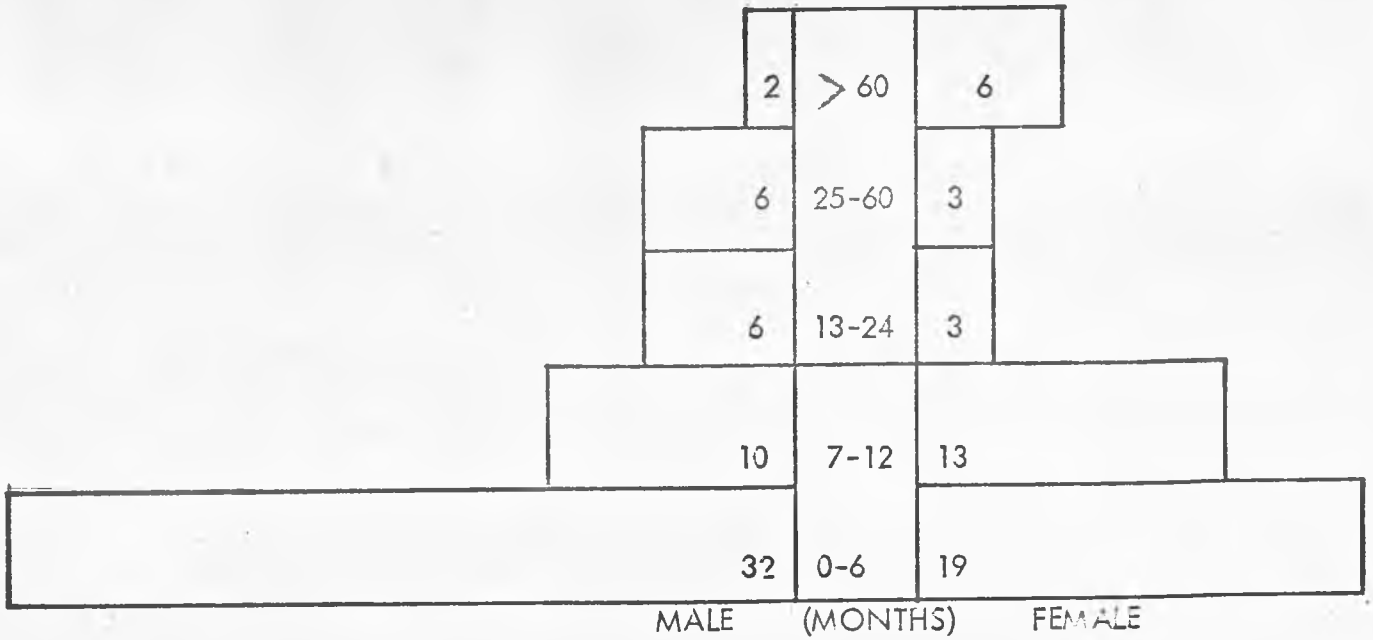


TABLE IV. DURATION OF ILLNESS (MONTHS)

TABLE V. FINDINGS ON ADMISSION

PARESIS	93%
SENSORY CHANGES	57%
BACK OR NECK PAIN	35%
INCONTINENCE OF URINE	33%
INCONTINENCE OR URINE + STOOL	14%
BACK OR NECK STIFFNESS	13%
DEFORMITY OF SPINE	5%

TABLE VI. TYPES OF PARESIS OR PARALYSIS

		FEMALE	MALE	TOTAL
PARAPLEGIA OR PARAPARESIS				
	SPASTIC	14	15	29
	FLACCID	11	17	28
QUADRAPLEGIA OR QUADRAPARESIS				
	SPASTIC	8	6	14
	MIXED	7	13	20
HEMIPLEGIA	SPASTIC		1	1
	MIXED		1	1
				93

TABLE VII. CAUSES OF MIXED QUADRAPLEGIA OR QUADRAPARESIS.

		FEMALE	MALE	TOTAL
1.	CERVICAL SPONDYLOSIS	4	7	11
2.	SCHWANNOMA	-	2	2
3.	NEUROFIBROSARCOMA	1	-	1
4.	MENINGIOMA	1	-	1
5.	PROLAPSED DISC C_6 C_7	-	1	1
6.	SECONDARY EPITHELIAL TUMOUR	-	1	1
7.	*INTRAMEDULLARY TUMOUR	-	1	1
8.	TUBERCULOSIS	-	1	1
				20

* HISTOLOGY: NECROTIC PARTLY HAEMORRHAGIC ACELLULAR TUMOUR.

TABLE VIII.

NRC POWER GRADE	F	MIXED M
5	-	-
4	2	3
3	1	2
2	2	4
1	2	3
0	-	2

A COMPARISON OF POWER AND MUSCLE TONE.

TYPE OF PARALYSIS.

FLACCID		SPASTIC		TOTAL
F	M	F	M	
-	-	-	-	4 FEMALE 3 MALE
4	1	4	3	17
2	6	3	5	19
1	7	5	7	26
4	1	6	5	21
1	1	4	2	10
				100

TABLE IX COMPARISON OF MUSCLE POWER IN VARIOUS AGE GROUPS.

MALE								FEMALE						
NRC			POWER		GRADE		AGE	NRC		POWER			GRADE	
5	4	3	2	1	0		(YRS)	0	1	2	3	4	5	
1	-	-	1	-	-		0-9	-	-	-	-	-	1	
-	-	2	-	1	1		10-19	1	4	2	1	2	-	
1	-	2	3	-	1		20-29	2	3	3	-	1	-	
1	3	4	3	5	3		30-39	-	2	-	1	3	-	
-	2	-	2	2	-		40-49	1	1	2	3	1	1	
-	1	4	6	-	1		50-59	-	-	-	1	2	2	
-	1	1	3	-	-		60-69	-	1	1	-	1	-	
-	1	1	1	1	-		70-79	-	1	-	-	-	-	
56	3	7	13	18	9	5		4	12	8	6	10	4	44

TABLE X MUSCLE POWER COMPARED TO DURATION OF ILLNESS

MALE					NRC MUSCLE POWER	FEMALE				
DURATION OF ILLNESS MONTHS						DURATION OF ILLNESS MONTHS				
60	25-60	13-24	7-12	0-6		0-6	7-12	13-24	25-60	60
-	-	-	3	3	0	1	2	1	-	-
-	1	2	-	6	1	5	5	-	1	1
-	3	2	1	13	2	5	1	-	2	-
1	1	1	4	6	3	2	1	1	-	2
1	1	1	2	3	4	6	1	1	1	1
1	-	-	1	-	5	-	2	-	-	1

TABLE XI. LESIONS COMPRESSING THE CORD WITHOUT CAUSING PARESIS

		MALE	FEMALE
1.	PROLAPSED INTERVERTEBRAL DISC	2	2
2.	CRANIOVERTEBRAL ANOMALY	-	1
3.	TETHERED CORD	1	-
4.	COMPRESSION FRACTURE LUMBAR VERT.	1	-
	TOTAL	7 PATIENTS.	

TABLE XII.

CERVICAL

THORACIC

LUMBAR

SACRAL

SENSORY LEVELS
6
37
10
4

TABLE XIII PRESENCE OF BACKACHE COMPARED TO DURATION OF ILLNESS

BACKACHE		DURATION OF ILLNESS (MONTHS)	NO. BACKACHE	
MALE	FEMALE		MALE	FEMALE
12	5	0-6	20	14
3	5	7-12	8	8
2	-	13-24	4	3
3	1	25-60	3	3
1	1	60	1	4

TABLE XIV PRESENCE OF BACKACHE IN VARIOUS AGE GROUPS.

BACKACHE		AGE OF PATIENTS (YRS)	NO.BACKACHE	
MALE	FEMALE		MALE	FEMALE
-	-	0-9	3	1
1	1	10-19	3	9
2	3	20-29	5	6
10	1	30-39	9	5
3	3	40-49	3	6
3	2	50-55	9	3
2	1	60-69	3	2
1	1	70-79	-	-

TABLE XV. DURATION OF ILLNESS COMPARED TO URINARY SPHINCTER CONTROL.

CONTINENT		DURATION OF ILLNESS (MONTHS)	INCONTINENT	
MALE	FEMALE		MALE	FEMALE
20	11	0-6	14	18
7	9	7-12	2	3
4	2	13-24	2	1
3	3	25-60	2	1
3	5	60		

TABLE XVI.

DIAGNOSTIC FINDINGS

SUGGESTIVE FINDINGS

NON SPECIFIC CHANGES

NORMAL

RESULTS OF PLAIN X-RAYS	
	34%
	11%
	9%
	46%

TABLE XVII - RESULT OF MYELOGRAM

TOTAL BLOCK	38
PARTIAL BLOCK	19
NO. BLOCK	3
EXTRADURAL DYE	3
	65

TABLE XVIII DIAGNOSTIC YIELD

		CLINICAL IMPRESSION	PRE-OP DIAGNOSIS	POST-OP DIAGNOSIS	FINAL DIAGNOSIS
1.	CORRECT SPECIFIC	45%	54%	76%	84%
2.	CORRECT NON SPECIFIC DIAGNOSIS	50%	42%	15%	16%
3.	INCORRECT SPECIFIC DIAGNOSIS	5%	4%	9%	-

TABLE XIX NON SPECIFIC DIAGNOSES

1. Intramedullary tumour
2. Necrotic partly haemorrhagic acellular tumour - intramedullary.
3. Arachnoiditis 4 cases.
4. Anular constriction of dura.
5. Non specific inflammatory extradural compression.
6. Necrotic inflammatory extradural tissue.
7. Haemorrhagic inflammatory extradural tissue.
8. Hyalinised fibrovascular fatty tissue.
9. Anaplastic Ca. 2 cases.
10. Metastatic tumour (unspecified).
11. Epithelial malignant tumour (? hepatoma).
12. Embryonal tumour (? Wilms).

TABLE XX - OUTCOME OF SURGERY

PROCEDURE	IMPROVED	NO. CHANGE	WORSE	DEAD	LOST TO FOLLOW UP
BIOPSY ONLY	-	1	-	4	-
EXPLORATION	-	4	-	1	-
DECOMPRESSION	25	9	4	3	1
PARTIAL REMOVAL	2	7	1	2	2
TOTAL REMOVAL	25	4	-	2	2
	52	25	5	13	5

TOTAL 100 patients

TABLE XX1 PRESENTING FINDINGS COMPARED

	KNH SERIES	KAMPALA* SERIES (4)
NUMBER OF PATIENTS	100	23
WEAKNESS	93	6
INCONTINENCE (URINARY)	33	7
BACKACHE	35	5
SENSORY LEVEL	57	4

*p 53

TABLE XXIII PRIMARY TUMOURS

	KNH SERIES	KAMPALA* SERIES (4)	MALAWI* SERIES (1)
NO. OF PATIENTS	100	23	102
MENGIOMA	10	5	4
NEUROFIBROMA	6	1	4
GLIOMA	2	-	-

*p 53

RESULTS:

100 patients with compressive cord lesions were studied. There were 56 males and 44 females. Their age distribution is shown in Table III. The range was from 8-70 years, 8-70 years for males, and 9-70 years for females. The average ages were 38.5 years for the male patients, and 34.8 years for the female patients. The duration of illness ranged from 3 days to 10 years.

The average duration of illness was 1.6 years for male patients, and 1.3 years for female patients. There was no correlation between the ages of patients and the duration of illness as shown in Table IV. The aetiologies of the spinal cord compression have been listed in Table IIA and IIB. The findings on admission are listed on Table V.

(I) WEAKNESS (PARESIS)

93 patients had a N.R.C. power gradation of 4 or less, consisting of 53 male patients and 40 female patients. There were 57 patients with paraplegia, or paraparesis, 29 of them having a spastic paraplegia or paraparesis, and 28 having a flaccid paraplegia or paraparesis. There were 34 patients with quadraplegia

or quadraparesis, 14 of them having spastic upper and lower limbs, and so patients having a "mixed" quadraplegia or quadraparesis with floccid upper limbs and spastic lower limbs. 13 of the 14 patients having spastic upper and lower limbs had a craniovertebral anomaly. The 14th patient had a cervical neurofibroma. 11 of the 20 patients who had quadraplegia, or quadraparesis with flaccid upper limbs and spastic lower limbs had cervical spondylosis. There were two hemiplegic patients. One a 34 years old male patient with spastic left upper and lower limbs had syringomyelia presenting as a Brown-Sequard syndrome.

The other was a 30 years old patient with a flaccid left upper limb, and a spastic left lower limb due to cervical spinal cord compression at CV4 caused by a bone spur. The types of paralysis or paresis is shown in Table VI. The lesions resulting in mixed quadraplegia or quadraparesis with flaccid upper limbs and spastic lower limbs are shown in Table VII. The type paresis or paraplegia classified according to the N.R.C. muscle power gradation is shown on Table VIII. There was no correlation between the ages of the patients and the N.R.C. muscle power gradation (Table IX), or the

the duration of illness and the N.R.C. muscle power gradation (Table X). The compressive spinal cord lesions that did not result in paralysis are listed in Table XI.

(II) SENSORY SYMPTOMS:

On admission, 57 patients had loss of sensation. Only the sensation to touch and pin prick were tested in all the files reviewed. The sensory levels are listed in Table XII. As expected the sensory levels located in the thoracic dermatomes predominated.

(III) BACK OR NECK PAIN:

35 of the 100 patients with compressive cord lesions had back or neck pain. There were 22 male patients, and 13 female patients. 17 patients had neck pain and 18 patients had back pain. There was no correlation between presence or abscence of backache or neck pain and the duration of illness (Table XIII) or the age of the patient (TABLE XIV). Back or neck pain was present in presence of degenerative structural or traumatic lesions rather than primary spinal cord tumours.

(IV) INCONTINENCE:

33 of the 100 patients were incontinent of urine. There were 20 male and 13 female patients. 14 of the patients with urinary incontinence had fecal incontinence as well, 11 male, and 4 female patients. There was no patient who had fecal incontinence only.

(V) DEFORMITY OF THE SPINE:

5 patients with compression spinal cord lesions presented with deformity of the spine. Three patients had kyphoscoliosis. One of the three patients with kyphoscoliosis had a craniovertebral anomaly which was the cause of the spinal cord compression. The other two were congenital and were the cause of spinal cord compression and traction. The two other patients who did not have kyphoscoliosis had tuberculous destruction of the vertabra leading to a gibbus.

(VI) INVESTIGATIONS:

All the patients reviewed had haemoglobin level, a total white blood cell count, urea and electrolytes, and a routine urine analysis for protein and sugar.

47 patients had a serological test for syphilis done, (mainly Kahn test) which were all negative. No test for syphilis was carried out on cerebraspinal fluid.

3 Mantoux tests are reckorded as having been done. All were positive, and were done on patients who turned out to be having tuberculous spinal cord compression. A fourth patient had a very positive mantoux test, done at a peripheral hospital, leading to a presumed diagnosis of tuberculous paraplegia, for which he got antituberculous treatment, at the peripheral hospital resulting in a delay in the definitive treatment of his spinal compression, which was actually caused by a meningioma.

49 patients had records of lumbar punctures being done on them and all were normal except one case of tuberculous compression of the spine with 50 grams of protein per litre of cerebrospinal fluid. Cerebrospinal fluid pressures were estimated from the rate of flow of cerebrospinal fluid from the lumbar puncture needle a highly inacurate method. No manometric measurements were made. Quicknestedts' test was not done on any of the patients and is rarely done in Kenyatta National Hospital. There were no records of typical Froin's syndrome.

Urine was tested for Bence Jones' protein on three patients, and was negative in all of them. Two of the patients were later

proven to be having multiple myeloma.

Brucella agglutinin test was done only once, and was negative. There were no records of a casoni skin test being done on any patient.

(VII) RADIOLOGICAL EXAMINATION:

All the patients had plain x-ray examination of the vartabra, at the relevant level or levels. 34% of the plain x-rays had a diagnostic finding. 11% of the plain x-rays had non specific changes unrelated to the spinal cord compression. 46% of the x-rays were normal (Table XV).

65 myelograms were done (Table XVII). There were three extradural injections of dye. 6 ml of myodil was used for adults and 3 ml for children. No attempt was made to recover the dye at the end of the procedure. In four patients the myelogram provided the specific diagnosis. These were syringomyelia, neurofinroma, ganglioneuroma, and a prolapsed intervertebral disc.

THE DIAGNOSIS:

To assess in what percentage of the patients, a diagnosis was made, three categories have been empirically adopted.

1. Correct specific diagnosis: The cause of the spinal cord compression and the cord level were correctly identified.
2. Correct non specific diagnosis: The cause of the spinal cord compression was not identified but may have been suggested. The spinal cord level was correctly identified.
3. Incorrect specific diagnosis: The specific cause of the spinal cord compression was misdiagnosed even though the correct spinal level may have been identified.

The diagnostic yields are shown at various stages of treatment (Table XVIII). The final diagnosis for the 16 patients for who a specific diagnosis was not arrived at are listed in Table XIX.

THE OUTCOME OF SURGERY:

The outcome of surgery according to the procedure performed is shown in Table XX. 52% of the patients improved. In 25% of the patients there was no change at the time of discharge. 5% of the patients deteriorated, 13% of the patients died, and 5% of the patients were discharged early and lost to follow up.

DISCUSSION.

In Kenyatta National Hospital patients paralysed from trauma are admitted, managed and rehabilitated at a nearby special spinal injury unit. Perhaps their occurence in a dramatic manner has helped to convince the planners, and the administrators of the need for a special unit for such patients. This is a worldwide practice.

However this does not apply to non traumatic paraplegics. Most are admitted to theneurological ward, but quite a significant number end up in general medical wards. When nursed in a general medical ward paralysed patients take a disproportionate time of the nursing staff, and the resources of the institution. The insidious nature of most non traumatic paraplegias does not help much either as it leads late presentation, and hence a delayed diagnosis.

Neurological assessment of a patient with paraplegia requires considerable expertise, and a sound knowledge of the anatomy of the spinal canal and its contents. The data yielded by a detailed chronological history, a careful general examination,

and a methodical neurological assessment should serve to identify the structure and level affected. A plan of investigations should be drawn up on admission. Ralatively few tests are required by identify the main possibilities. When spinal radiography and myelography are available a rational working diagnosis should soon be established.

This further emphasises the need to manage paralysed patients at one point from where they can be attended to by all who are concerned with their management. Where this is not possible then a standardised approach to examination and management would be helpful. Perhaps if the above methods were practiced in Kenyatta National Hospital, probably the investigations in this series would have been more systematic.

Compressive cord lesions form 47% (23 of 49 patients (4) to 75% (75 patients out of 100 patients (3) of patients with non traumatic paraplegia. In the present series, 20 files were either missing entirely or in relevant information. Therefore 120 patients were operated on in 4 years, an annual average of about 30 patients. This means that an estimated 40-60 patients with non traumatic paraplegia

are admitted annually. For a 1200 bed hospital with the only neurosurgical ward in Kenya, this number are small.

The estimated incidence of non traumatic paraplegia in Europe is 10 per 1,000,000 (9). Using the above figures, our annual admission represent 4-6 million of the population in Europe Non traumatic paraplegias in developing countries are significantly contributed to by poliomyelitis, tropical spastic paraplegias, and nutritional myelopathies which are reported to be rare in East Africa (10), (11). This surely means that many of our patients with non traumatic paraplegias are not reaching us.

Our investigation compare well with those of other series from East, and Central Africa (1-4). However the comparison can not be too close, due to the bias toward aetiology necessarily introduced from the source of the patients in a neurosurgical unit.

In Kenyatta National Hospital cerebrospinal fluid manometry is rarely done, and Quickenstedts' test is almost never done. Murphy and Hardikar (4) had 11 positive Quickenstedts' test in their series with 12 patients with complete obstruction of the spinal canal on myelography. In a North American series of 262 patients with

spinal cord tumours, Quickenstedts' test was positive in 83.6% of the patients (6). In a related series, an elevated cerebrospinal fluid protein level of 0.6g/l or above was present in 82.4% of the patients.

In this series 45% of the plain x-rays showed a relevant finding, 34% being diagnostic. Brown (1) had 44/102, and Murphy and Hardikar (4) had 4/23 relevant finding on plain x-rays.

65 myelograms were done, of which 38 had a total block, 19 a partial block, and three myelograms were normal. One of the normal myelograms belonged to a patient with craniovertebral anomaly. The other two belonged to patients with prolapsed intervertebral discs. Out of 45 myelograms Brown (1) reported 18 with a total block, 19 with a partial block, and 8 were normal.

The presentations as found by Murphy and Hardikar (4) are similar to those in series (TABLE XXII).

Tuberculosis is the commonest cause of paraplegia in developing countries (1), (2), (3), (7), (12) accounting for 32.5% (1), 45% (2), 54% (3) of the paraplegia. In this series it was responsible for only 5% of the paraplegia. Tuberculous paraplegia is usually treated by Orthopaedic Surgeons and hence its infrequent occurence in this series, from a neurosurgical unit. The pathogenesis of tuberculous paraplegia has been discussed in detail by Butler (17) and Roaf et al (12). Compression of the cord is by granulation tissue, abscess formation, or displaced vertebral or disc tissue, or by occlusion of radicular arteries.

The present trend of treatment of non complicated tuberculosis of the spine has moved from the prolonged immobilization in plaster shells with or without surgery as used years ago to ambulant treatment (8) (14-17). However there is divergence of opinion as to the best form of treatment once complications have set in.

In presence of paraplegia, decompression is obligatory. Anterior approaches have been claimed to result in less instability with the advantage that in presence of instability an anterior fusion can be carried out at the same time. Earlier healing, better stability,

and less final deformity are claimed to result (13) (18), (19). This is the route used on patients with tuberculosis in the series from Kilimanjaro Christian Medical Centre (2),(3). However the Malawi experience is different where laminectomy (posterior approach) has been used for many years with few problems (1). The patients in this series were all operated from the posterior approach, and instability was not a problem.

The clinical features of patients with spinal tuberculosis noted in this series were similar to those of the other series. They tended to be young though the average age was 36.4 years due to the inclusion of a 58 years old man, and an average E.S.R. of 55 mm/hr as compared to an average age of 28 years and an E.S.R. of 51 mm/hr (2), (3). The result of treatment was gratifying as has been found in almost all other series. Neoplasms were the largest cause of paraplegia, and the second commonest cause of paraplegia in the series from Tanzania, and Malawi. They ranged from 13 and 15% (3), (2) to 32% in this series and 33% is the small series from Kenyatta National Hospital by Ojiambo (5).

The various series have different findings as to which is the

commonest primary cord tumour. This to some extent depends on the age distribution within each series. Paediatric series are dominated by embryonic neural tumours especially medulloblastoma and neuroblastoma. In the adult series, there is some controversy as to which is more common, neurofibroma or meningioma. In this series meningioma almost twice as common as neurofibroma, 10 and 6 respectively. Austin (6) Murphy et al (4) and Bull (20) had similar findings.

In contrast, Harison et al (1949) Kernohan and Syre (23), Tucker (1956) (24) Ojiambo (1966) (5) found more neurofibromas than meningiomas. Murphy et al (1967) (4) in their series had all the meningiomas in their series occuring in females, in the thoracic region as did Bull (1953) (10). This is in variance with findings of this study where there were 6 females (60%) and 4 males (40%). One meningioma each were found in the cervical, lumbar, and cauda equina regions, with 7 meningiomas occuring in the thoracic region. 75% of meningiomas are reported to occur in the posterolateral region of the cord, and present early with sensory symptoms. This was not found in this series. 2 patients with meningiomas presented

late in pregnency, and one early after delivery. In two patients, a 19 years old male patient, and a 21 years old female patient, the tumour had increased mitotic figures which indicated aggressive behaviour. However in both the followup was short and recurence was therefore not found.

In this series one patient with a neurofibroma presented as a Brown-Sequard syndrome. Several patients in the series by Austin (6) presented in a similar manner.

Schistosomiasis is an important cause of paraplegia in nothern Tanzania resulting in 6-8% of the paraplegia (3) (2). Schistosomasis was not found as a cause of paraplegia in this series or in the series from Malawi (1).

The possibility of schistosomiasis causing obscure paraplegia was raised by Ojiambo (5) in 1966, and Haddock (11) citing Bird, even suggested a trial of anti-schistosomal treatment is obscure cases of paraplegia, with negative stool, sigmoidoscopy, and rectal snip. Schistosomiasis causes paraplegia as a result of the formation of granulomatous massess around the ova in or around the cord, or secondary to acute hypersensitivity to worms or ova resulting in

oedema interfering with cord perfussion and viability. When intra-abdominal pressure rises, venous blood flows from the common iliac veins to the vertebral venous plexus (23).

Brucellosis is a known cause of paraplegia due to formation of granulomatous masses around the cord. A case treated in Kenyatta National Hospital is reported by Ojiambo (5). Only one serological test for Brucellosis was done in this series which was negative. There was no report of typical radiologic findings on the vertebral x-rays in this series (26). However there were three cases with obscure non-specific inflammatory masses and any of these could have been due to Brucellosis.

It is generally accepted that metastatic tumours of the cord are commoner than primary cord tumours, as a cause of paraplegia (27-28). This was the finding by Scrimgeour (3), Brown (1), Hulme-Moir (2) Austin (6), and many other investigators (27-28) (30). However in this series there were 9 secondary tumours (Table IIB) as compared to 14 primary cord tumours. The suprising finding is the rarity of secondaries from the prostate in contrast to the findings of Scrimgeour (3), Brown (1), and Murphy (4). There was an incidental

carcinoma of the prostate in a 60 year old patient with meningioma.

Diffuse adhessive arachnoiditis may follow acute menengitis, chronic tuberculous, syphilitic or cryptocorcal menengitis or as a sequel to myelography. Most of them however are idiopathic. In the four cases encountered in this series no primary cause was found.

No patient presented with an anterior spinal syndrome in this series. This syndrome may be a manifestation of syphillis or a presentation of trauma, or tuberculosis. All the serological tests for syphilis were negative. Anterior spinal artery thrombosis in syphilis results in a sudden onset of an inicially areflexic flaccid paraplegia, with diffuse meningomyelitis which produces a slowly progressive spastic paraplegia. In Jamaica, a type of paraplegia with an association of nutritional deficiency and positive serological test for syphilis is common, Montgomery (25).

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