SPINA BIFIDA

AS SEEN IN KENYATTA NATIONAL HOSPITAL.
TOPIC

PATTERN OF PRESENTATION OF SPINA BIFIDA AS SEEN AND MANAGED IN KENYATTA NATIONAL HOSPITAL.

BY

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A DISSERTATION SUBMITTED AS PART OF FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE IN SURGERY IN THE UNIVERSITY OF NAIROBI.

2004
DECLARATION

This dissertation is my original work and has not been presented for a degree in any other university

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Date……………………..

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This dissertation has been submitted for examination with my approval as a university supervisor.

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2004
DEDICATION

To both my late grand parents, mother Josephine who encouraged me in times of hardship and instilled the principles of hard work in me as I grew up.

To my wife Millicent and son Ian for their patience, hope, support, love and encouragement.
ACKNOWLEDGEMENT

I wish to express my profuse thanks to my supervisor DR. C.K. MUSAU for his assistance, advice, critique and guidance in the preparation of the protocol and this dissertation.

My thanks also go to the Kenyatta National Hospital Ethical and Research committee and the staff of ward 4C and Neuro surgical out patient clinic for facilitating this research.

I am grateful to all the patients and their mothers who participated and without whose co-operation the study would not have succeeded.
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<th>Abbreviation</th>
<th>Description</th>
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<td>A.F.P</td>
<td>Alpha Fetoprotein</td>
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<td>2</td>
<td>A.N.C</td>
<td>Ante natal clinic</td>
</tr>
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<td>3</td>
<td>C.N.S</td>
<td>Central nervous system</td>
</tr>
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<td>F.G.F</td>
<td>Fibroblast growth factors</td>
</tr>
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<td>5</td>
<td>K.N.H</td>
<td>Kenyatta National Hospital</td>
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<td>6</td>
<td>N.T.D</td>
<td>Neural tube defects</td>
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<td>7</td>
<td>T.G.F</td>
<td>Transforming growth factors</td>
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<td>8</td>
<td>U./ S.</td>
<td>Ultra Sonography.</td>
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<td>C.T. SCAN</td>
<td>Computer Tomographic scan</td>
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<td>10</td>
<td>M.R.I.</td>
<td>Magnetic Resonance Imaging</td>
</tr>
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<td>11</td>
<td>V.P.S.</td>
<td>Ventriculo peritoneal Shunt</td>
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SUMMARY

This was a hospital based descriptive prospective study of patients born in KNH with spina bifida and those referred with similar conditions for further evaluation and possible operation. A total of 75 patients aged 5 days to 15 years with various forms of spina bifida were recruited at Kenyatta National Hospital's Ward 4c and Neuro–surgical outpatient clinic. Assessment of clinical presentations, demographic data timing of surgery and common complications was carried out.

Methodology.

Patients were recruited over a five month period between March 2004 and July 2004, according to the set criteria. Each patient was followed till either discharge or a minimum of 4 weeks in the clinic. Data was entered in a questionnaire and analyzed.

Results

From the data, 65.4% of all patients were aged below two years with a median age of 17 months. Females accounted for 54.7% with a male to female ratio 1:1.2. Many of the patients came from Central province 56% and were first born 41.3%.

Most the mothers were housewives 66.8% with poor social economics status, 52% earned less than two thousands shillings a month. Forty eight percent of the mothers had education up to primary school level and their mean age was 25.8 years.
Majority of the mothers attended ANC during the third trimester 62.7%, and only 1.3% had used folic acid antenatally. Twenty percent had a history of exposure to the known teratogens. Seventy eight point seven percent of the patient had spina bifida cystica with spina bifida occulta accounting for 21.3%. Majority of the lesions were spina bifida myelomeningocele 48.7% . Of all patients, 33.3% of them were paralyzed and of these 76% had spina bifida cystica. Patients with lesion at the lumbar spine were 76%.

Ultrasonography of the skull was the commonest imaging study which was performed 66.7%, followed by ultrasound of the lesion 13.3%. The commonest associated congenital malformation was hydrocephalus accounting for 54.7% . The median age at operation was 20 months . Post operatively, 16% had complications. Wound sepsis accounting for 8% . Ventriculo peritoneal shunt placement was performed in 54.7% of the patients and of these 80.5% had them before the main operation for the spina bifida.

**Conclusion**

The commonest form of presentation of spina bifida is spina bifida myelomeningocele. Spina bifida is common in Central Kenya from mothers of poor social economic status. Use of folic acid in the periconception period and early antenatal attendance can reduce the incidence significantly.

Early operation for the lesion and V.P. shunting for those with hydrocephalus will enable the patient reach adulthood and lead a normal life unaware of their deficiencies.
INTRODUCTION

Spina bifida is congenital abnormality of the spine that occurs due to failure of the neural folds to fuse in the midline and form the neural tube. The subsequent defect is the maldevelopment of the mesoderm which in turn forms the skeletal and muscular structure that cover the underlying neural structures. (1)

Neural tube defects (NTD) can be open (neural structure that communicate with the atmosphere) or closed (skin covered). They can be ventral or dorsal midline defects (2).

This condition presents a tremendous initial shock for the parent on looking at the deformity. They develop a sustained fear of the future of the child.

The condition is often a challenge to the medical personnel particularly when the child has paralysis, stool and urine incontinence. The future quality of life of the child even after surgery is also a challenge for the majority have stool and urinary incontinence. (3).

The condition has been noted to be significantly reduced by the use of periconceptuals folate. In our country there are no records for this observation. But in other countries like China, this have been reported that periconceptual intake of 0.4 gms of folic acid daily reduced the risk of NTD from 41% to 3% of the expectant mothers. (4)

The use of folic acid antagonist such as trimethoprim, carbamazepine during pregnancy has been shown to increase the incidence of cardiovascular defects, oral
defects, neural tube defects and urinary defects. (5)

This shows the condition is a public health concern which can be significantly reduced if the mothers at risk are given periconceptual folate.

In early 1970s and 1980s many doctors were not in favour of doing anything active on the spina bifida lesion S(6). Currently, following the early closure of the defect and efficient treatment of hydrocephalus, these children reach adulthood unaware of their deficiencies. (8)

Currently early antenatal attendance and ultrasonography screening can assist in detection of this condition. The use of in utero repair of the spina bifida and therapeutic abortion for those with severe or multiple anomalies has helped to reduce the incidence and the morbidity associated with this condition.(9)
LITERATURE REVIEW

DEFINITION.

Spinal bifida (dysraphism), is a clinical manifestation of incomplete closure of the neural tube. There is absence of the vertebral arches with or without closure of the overlying skin. (1, 10)

Spina bifida has been shown to exist for more than 1200 years. (11)

Tulpins the teacher in Rembradts’ Anatomy lessons’ described and illustrated it in 1652 and called it spina bifida. (14)

Morgagni in 1761 first associated spina bifida with hydrocephalus.

Almost a century later John Cleland published contributions to the study of the spina bifida, encephalocele and anencephalus. (7)

Eight years latter Hans Chiari, Professor of Morbid Anatomy at Charles University in Prague published similar observations on congenital anomalies in the cerebellum and brain stem.

Anorld in 1894 brought minor contributions to Chiari’s work and brought the name Anorld Chiari’s malformations.

In 1950s and 1960s Laurence in Wales described a cohort of 290 children with spinal bifida left untreated, only 11% of them lived past the 1st decade of life.

In 1960s in the United States most children with myelomeningocele were treated and this resulted in a higher survival rate with more than 80% surviving past the 1st decade. (12)
In 1970s Smithhells advanced a concept that Nutrition may be related to the development of neural tube defects.(24)

Von Ruysch in 1974 distinguished between paralytic and nonparalytic forms.

In 1991 the medical research council of Britain published a research on women taking Folic acid pre conception. It was found that folic acid offered protection against NTD.(4)

In 1999 Vanderbilt University researchers carried out a cohort study of 29 patients with isolated myelomeningocele. The patients underwent intrauterine repair of NTD. This was performed at between 24 to 30 weeks gestation. The results were compared to 23 lesions matched controls who underwent postnatal surgery. The main outcome measure was requirement for a ventriculo peritoneal shunt for the treatment of hydrocephalus. Their results showed that those NTD patients who underwent inutero surgery had lower incidence of hydrocephalus than the control group (59 % versus 91%)(14)
PATHOLOGY

SPINA BIFIDA CYSTICA

The two major types of defects seen in spina bifida cystica are meningocele and myelomeningocele. Cervical and thoracic region are the least common sites and lumbar and lumbar sacral regions are the most common sites for these lesion. (15, 16)

Myelomeningocele is a condition in which the spinal cord and nerve roots herniate into a sac comprising the meninges. The sac protrudes through the bone and musculocutaneous defect. The spinal cord often ends in this sac. It is splayed open exposing the central cord. The splayed open neural structure is called the neural placode (16)

Certain neurologic anomalies such as hydrocephalus and Chiari. II malformation accompany myelomeningocele. In addition myelomeningocele have higher incidence of associated intestinal, cardiac,and oesophageal malformation as well as renal and anorectal anomalies. Most neonates with myelomeningocele have orthopedic anomalies of their lower extremities and urogenital anomalies due to involvement of the sacral nerve roots. (17)

A meningocele is simply herniation of the meninges through the bony defect (spinal bifida). The spinal cord and nerve roots do not herniate into this dorsal dural sac. Neonates with meningocele do not have associated neurologic malformations such as hydrocephalus or Chiari II malformations.

A subtype of spinal bifida is called lipomeningocele or lipomyelomeningocele
which is a common form of NTD. The lesions have a lipomatous mass that herniates through the bony defect. It attaches to the spinal cord, tethering the cord and often the associated nerve roots. Lipomyelomeningocele can envelop both the dorsal and ventral nerve roots. Sometimes only the dorsal nerve roots or simply the filum terminalis and conus medullaris are involved. These lesions do not have associated hydrocephalus but have a more guarded prognosis compared to simple meningocele. (13, 18)

The other type of spina bifida cystica is the rare myelocystocele. The spinal cord has a large terminal cystic dilatation resulting from hydromyelia. The posterior wall of the spinal cord often is attached to the skin and is undifferentiated thus giving rise to a large terminal skin covered sac. (19)

The vast majority of the lesions are dorsal although minorities are ventral. The most common ventral variant is an anterior sacral meningocele which most often is discovered in females as a pelvic mass. (20)
SPINAL BIFIDA OCCULTA

In this group of NTDs the meninges do not herniate through the bony defect. The lesion is covered by skin. These patients do not have associated hydrocephalus or Chiari II malformations. Often a skin lesion such as hairy patch, dermal sinus tract, skin dimple, hemangioma or lipoma may be present. These point towards the underlying spina bifida. Presence of these cutaneous stigmata above the gluteal fold signifies the presence of an occult spinal lesion. Dimples below the gluteal fold signify a benign non neurologic finding such as a pilonidal sinus.(11,21)

Signs and symptoms of occult spinal disorders in children include the following.

(a) Radiologic, hemi vertebrae scoliosis; widening of interpedicular distance; butterfly vertebrae, lamina defect

(b) Cutaneous stigmata

Capillary hemangioma Caudal appendage, Dermal Sinus Hypertrichosis

(C) Orthopedic findings

extremity asymmetry
foot deformities
Neurological problems. (12, 20)

- weakness of leg or legs
- leg atrophy or asymmetry
- loss of sensation with painless sores
- hyporeflexia
- unusual back pain
- abnormal gait
- radiculopathy
- urologic abnormality.
- neurogenic bladder

**EPIDEMIOLOGY**

It is more common in people of Celtic origin and female are more affected

approximately 60 – 70% of the children. (13)

Countries of the British Isles have higher rate than Asian countries. (13)

There has been a significant decline worldwide in NTD births. For example in the United States, New England has seen the incidence of spinal bifida drop from 2.3 per 1000 births during the 1930s to 0.77 per 1000 births during the 1960s.
Reasons for the dramatic drop are not completely clear however certain factors probably play a role. These include prenatal screening tests such as alpha fetoprotein (AFP) and ultrasonography. (U/S) (22)

Termination of pregnancy increased 50 fold in British Isles after the introduction of prenatal screening. The use of periconceptual folate will most likely reduce the incidence of NTD latter in the 20th century. (13.)

The risk of giving birth to second born child with a NTD is significantly high. (3)

**EMBRYOLOGY**

The nervous system develops from the neural plate. (17)

DG.1
The neural plate is a thickened slipper shaped area of embryonic ectoderm. It’s the notochord and paraxial mesoderm that induce the overlying ectoderm to differentiate into the neural plate.

Signaling molecules appear to involve members of the transforming growth factor B(TGF-B) family. Others are activin and fibroblasts growth factors (FGFs) this leads to the formation of the neural folds neural tube and neural crest from the neural plate.

DG.2

The neural tube differentiates into the CNS consisting of the brain and spinal cord. The neural crest gives rise to the cells that form most of the peripheral nervous system and autonomic nervous system consisting of cranial, spinal and autonomic ganglia.

Formation of the neural tube neurulation begins during the early part of the fourth week in the region of the fourth to sixth pairs of somites. At this stage the cranial two thirds of the neural plate and tube as far caudal as the fourth pair of somites
represent the future brain and the caudal one third of the neural plate and the tube represent the future spinal cord. Fusion of the neural folds proceeds in a cranial and a caudal direction until only small area remains open at both ends. Here the lumen of the neural tube – neural canal communicate freely with the amniotic cavity

Diagram 3

The cranial opening the rostral (anterior) neuropore closes as about the twenty fifth day and the caudal (posterior) neuropore 2 days latter. Closure of the neuropores coincides with the establishment of blood vascular circulation for the neural tube. The walls of the neural tube thicken to form the brain and the spiral cord The neural canal of the neural tube is converted into the ventricular system of the brain and the central canal of the spinal cord.
## CENTRAL NERVOUS SYSTEM MALFORMATION

<table>
<thead>
<tr>
<th>DAYS OF GESTATION</th>
<th>EVENT</th>
<th>RESULTANT MALFORMATION</th>
</tr>
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<tbody>
<tr>
<td>18 – 19</td>
<td>Formation of neural plate and groove form</td>
<td>Anterior midline defect</td>
</tr>
<tr>
<td>20 – 23</td>
<td>Appearance of optic vessels</td>
<td>Hydrocephalus</td>
</tr>
<tr>
<td>24 - 26</td>
<td>Closure anterior neuropore</td>
<td>Anencephally</td>
</tr>
<tr>
<td>26 – 28</td>
<td>Closure posterior neuropore</td>
<td>Cranium bifidum</td>
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<tr>
<td></td>
<td></td>
<td>Spina bifida cystica</td>
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<td></td>
<td></td>
<td>Spina bifida occulta</td>
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## ETIOLOGY

Over the last century, teratogens have been implicated in the etiology of NTD in humans and experimental animals. These includes potato blight: hyperthermia, antihistamine and sulfonamide use, nutritional deficiencies, vitamin deficiencies and anticonvulsant use (17, 23). Of all the suspected teratogens, carbamazepine, valproic acid and absence of folate have been
most strongly tied to the development of NTD. Smithells first advanced the concept that nutrition may be related to the development of NTD in 1970. He noted that a low erythrocyte folate and leukocyte ascorbic acid during the first trimester resulted in more pregnancies affected by NTDs than in controls. (24)

A double blind study was carried out by medical research council of Britain to see if women who previously gave birth to children with NTD could lower the recurrence rate with multivitamins or folic at 4mg per day. One thousand eight hundred and seven women who had a previous child with NTD were selected. One thousand one hundred and ninety five women who had not had a child with NTD were also selected. All the women were randomized into four groups. One group received multivitamins, the other group received folic acid, the third group received both and the fourth received neither. The study was terminated early when significant protective effect was observed in the groups that received folic acid but not in the group that did not. Multivitamins alone had no significant protective effect. Folic acid ingestion in the pre conception period prevented an estimated 72% of recurrent NTDs (13)

Hungarian investigators performed a randomized double blind multicenter trial of folic acid to see if it had a protective effect for a first occurrence of NTD. One group of 2104 women received 0.8mg of folic acid with their multivitamins while the second group of 2052 women received no folic acid with their multivitamins.
The folic groups had no cases of NTD while the non folic groups had 6 cases. (13)

**DIAGNOSTIC DETECTION OF NTDs**

Presence of open NTDs can be detected by the measurement of AFP (Alpha Feotal Protein) in the amniotic fluid or maternal blood stream. Alpha feotal protein is the major serum protein in early embryonic life and is 90% of the total serum globulin in a fetus. It is believed to be involved in preventing fetal immune rejection and is first made in the yolk sac and then latter in the gastrointestinal system and liver of the fetus. It goes from the fetal blood stream to the fetal urinary tract where it is excreted into the maternal amniotic fluid. The AFP also can leak into the amniotic fluid from open NTDs such as anencephaly and myelomeningocele in which the fetal blood stream is in contact with the amniotic fluid.(12,25)

In prenatal screening maternal sample for AFP is drawn between 15 and 20 week of gestation. A patient specific risk is then calculated based on gestational age and AFP level. A normal AFP concentration in the maternal serum is usually lower than 500ng. Determining precise gestational age is essential because fetal AFP levels are age specific and can peak in a normal fetus at 12-15 weeks of gestation. For example at 20 weeks gestation a maternal serum AFP concentration higher than 1000ng/ml would be indicative of an open NTD. The measurement of maternal serum AFP levels is more than 75% accurate in detecting an open NTD at more than 15 weeks gestation. Amniotic AFP can also be obtained especially at 15-20 weeks, of gestation.
and detects approximately 98% of all open NTD although this method is not preferred for screening. Amniotic acetyl cholinesterase levels can be used. This adds an increased degree of resolution.

Detection of NTD with fetal ultrasound in the hands of a skilled ultrasonographer usually is 98% specific. False positive result from multiple pregnancies or inaccurate fetal dating. However closed NTD also can sometimes remain undetected especially in cases of skin covered lipomyelomeningocele and meningoceles in which the AFP also may be normal. These closed NTDs comprises about 10% or more of total NTDS discovered.(13,25)

The following is a list of other fetal abnormalities associated with elevated AFP

Anencephaly
Encephalocele (leaking)
Conjoined twins
Omphalocele
Turner syndrome
Gastroschisis
Extrophy of the cloaca
Oligohydramnios
Sacrococcygeal teratoma
Polycystic kidney
Feotal death
Urinary tract obstruction
EVALUATION AND TREATMENT.

Initial examination

Evaluation of

1 Anatomical level of the lesion
2 Motor and sensory level
3 Presence of associated hydrocephalus
4 Presence of associated symptomatic hindbrain herniation
5 Presence of associated orthopedic deformity

The lesion is first examined after the birth of the neonate. Open neural tube defects should be immediately covered with saline moistened sponge to avoid rupture of the sac or drying of the exposed neural placode. The neonate is maintained and examined in the prone or lateral position and treated with antibiotics. Other abnormalities like hydrocephalus and cardiac abnormalities are assessed by use of ultrasonography. Also urological examination is performed by ultrasound. Orthopedic evaluation for hip dislocation, varus or valgus deformities of the extremities are carried out. Attention to the anus helps to assess sacral nerves root function. Flaccid musculature in the second and fourth sacral regions often present with flat buttocks, absence of a well developed gluteal cleft and a patulous anus with no anal wink. The thoracic or lumbar region may have a large lump due to kyphosis or scolosis of the spine. An MRI may reveal defects in cellular migration in the cerebral cortices for example thinning of the corpus collosum or an abnormal white matter finding.
TIMING OF MYELOMENINGOCELE REPAIR.

In 1960s repair of myelomeningocele used to be a neurosurgical emergency. Studies have subsequently shown that closure within 48 hrs was both safe and effective. (26) 

Operative approach

Any major procedure on a neonate with myelomeningocele must be performed in such a condition as to avoid hypothermia hypovolemia and airway compromise. The goals of the operation is to circumnavigate the neural placode without injuring any of the neural elements. Then the neural placode is placed into the spinal canal. Then the dura is identified, dissected and is used to cover the neural placode by a water tight closure.

When dura is absent, a muscle fascial flap is reflected off the para spinal muscles and is used to create a watertight tube. The skin is then closed in layers after careful and adequate undermining of the skin so as to avoid necrosis or ischemia.
**SHUNT PLACEMENT**

Approximately 20% of all patients with myelomeningocele have significant hydrocephalus at birth. Sixty to seventy percent of patients with spina bifida develop hydrocephalus after the closure of spina bifida lesion. Shunting is performed during the same operation for closure of myelomeningocele while others are done after they manifest with the hydrocephalus. (13, 26)

**OUTCOME AND PROGNOSIS**

The natural history of neonates with NTD left untreated leads to a high mortality and morbidity. Most die of meningitis, hydrocephalus and sepsis.

In evaluating the outcome there are various issues that one looks for. These are hydrocephalus, intellect, ambulation, continence, orthopedic problems, employment and independent living status. In intellect, 60-70% of the children with myelomeningocele had intelligence quotient (IQ) greater than 80 (27)

**CONTINENCE**

Only 10-15% of all children with myelomeningocele are continent of urine. This issue often cause children to be separated from their peers which leads to neuropsychological deficit. (14, 29)

Clean intermittent catheterization results in these children being socially continent and significantly decreases the rate of urosepsis. Urinary diversion can also be performed. Use of ant cholinergic drug combined with clean intermittent catheterization results in better self image and greater educational and vocational
opportunities for children with NTDs

Bowel continence is achieved with a combination of medication, diet control, and manual disimpaction and enemas.
RATIONALE OF THE STUDY:

Spina bifida is a common congenital anomaly of the spine seen in our country. It presents a tremendous initial shock to the parents on looking at the deformity when they have been expecting a normal healthy baby.

It is associated with a high morbidity and mortality. Therefore, it has a significant economic impact on this country. Majority of Kenyans have a poor social economic background. Mothers do not attend antenatal clinic on time. This has led to an increase in the incidence and prevalence of such children being born. (6)

Maingi A.W in his study in KNH in 1980 showed that these children were not operated on time. This was due to lack of facilities and personnel. The operation could only be carried out in KNH and children who had paralysis with spina bifida were not normally operated on. (6)

This study intends to evaluate the geographical distribution of these patients, the time mothers attend ANC, the use of periconception folic as a prophylaxis for those at risk and whether the mothers had exposure to the known teratogens.

The study also aim to look at the commonest form of presentation of spina bifida and the timing of surgery for these condition.
OBJECTIVES OF THE STUDY.

MAJOR OBJECTIVE.

To determine the clinical presentation of spina bifida in Kenyatta National Hospital.

MINOR OBJECTIVES.

To determine,

1. The age of the mother and her occupation.
2. Her parity and positive history of similar siblings.
3. Geographical distributions of these patients.
4. Associated congenital anomalies.
5. Diagnostic investigations carried out before surgery.

HYPOTHESIS

The majority of children born with NTD have spina bifida with myelomeningocele. These are born of young mothers less than 25 years age and of poor social economic status.
MATERIALS AND METHODS

Study design.

This was a descriptive hospital based prospective study of patients seen with spina bifida in K.N.H. ward 4C and neurosurgical clinic between March 2004 and July 2004.

During the first visit, the child with spina bifida lesion was examined thoroughly from the neurological point of view and thorough general examination done. The initial examination was usually carried out by one of the neurosurgeons.

History and physical examination was carried out. In the history, mothers geographical location, history of siblings with similar ailment and exposure to any known teratogens was noted.

A general examination of the chest and the abdomen was carried out and any disease present identified and other congenital malformations excluded.

The tone power, reflexes, sensation, bladder or bowel incontinence presence or absence of foot deformity and head circumference noted.

Children who were born in Kenyatta National Hospital maternity wing were examined by a pediatrician or registrar attached to that unit. They were then referred to neurosurgical unit where a further reevaluation was carried out.

This included radiological investigation like ultra sonography to evaluate for associated abnormalities such cardiac defects and urological defects.

A spine X ray may or may not have been done by a referring centre though it was done in cases of suspected spina bifida occulta.

Surgery was booked as early as the children were seen. The operation was
performed by a senior registrar in the unit or the consultant neurosurgeon.

Post operative patients were followed up in the neurosurgical clinic.

Any complications after surgery was noted and these patients were followed up for two weeks postoperatively

**PRESENTATION OF RESULTS**

This was in form of tables, bar charts / graphs and pie charts.

**DATA ANALYSIS**

Data for the age of these patients at time of presentation, the age of the mothers at first delivery, the time of A.N.C. attendance of the mother and the age of the patients was analyzed. This was by frequency calculations, the median age of the patients and the mean age of the mothers.

These data was analyzed using SPSS (Statistical Package for Social Sciences) version 11.0 computer software,

**SAMPLE SIZE**

This was based on the patients presenting in KNH with spina bifida. The patient should fit the eligibility criteria below.

The sample (n) was derived by $n > z^2 \times \frac{pq}{d^2}$

Where $p$ is the incidence, $d$ the confidence limit, $q = (1-p)$ and $z$ is the standard deviation of the 95th percentile (1.96).

Based on Maingi five year retrospective study between 1975 to 1979 with a total incidence of 132 babies and children with mid line congenital abnormality of the spine, the average annual incidence $p$ was 26.

A confidence limit of 0.1. was used. The sample should not have been less than $(1.96)^2 \times \frac{(0.26 \times 0.74)}{(0.1)^2} = 74$ patients.
**Study Limitations**

1 Lack of standardization in clinical assessment as this was subjective and prone to individual bias.

2 Operative outcome as this was still dependent on the surgeon’s competence.

**Inclusion Criteria.**

All babies born with spina bifida in labour ward of K.N.H, those who have been referred with a similar problem to neurosurgical unit for operation or have been operated on during the study period after giving consent constituted the study population.

**Exclusion Criteria**

Patients who fell out of the study period or those who withheld consent for the study.
**Ethical aspect**

Application was made to the Ethical and Research Committee of the Kenyatta National Hospital to permit the study at the institution.

Patients were recruited after signing an informed consent form (appendix iii) This was signed after the parent/guardian read and understood the consent explanation form.

All patients’ records were handled confidentially and used for the intended purpose.
RESULTS

A total of 75 patients were seen during the study period of March 2004 and July 2004.

Table 1: Age distribution (n=75)

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 12 months</td>
<td>27</td>
<td>36.0</td>
</tr>
<tr>
<td>13-24 months</td>
<td>22</td>
<td>29.3</td>
</tr>
<tr>
<td>25-36 months</td>
<td>14</td>
<td>18.7</td>
</tr>
<tr>
<td>&gt; 36 months</td>
<td>12</td>
<td>16.0</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100</td>
</tr>
</tbody>
</table>

The age range was 5 days to 15 years and the median age was 17 months

Figure 1: Gender distribution
Table 2: Distribution of area of residence (province)

<table>
<thead>
<tr>
<th>Province</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
<td>42</td>
<td>56</td>
</tr>
<tr>
<td>Western</td>
<td>11</td>
<td>14.8</td>
</tr>
<tr>
<td>Eastern</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Rift Valley</td>
<td>7</td>
<td>9.3</td>
</tr>
<tr>
<td>Nyanza</td>
<td>5</td>
<td>6.6</td>
</tr>
<tr>
<td>Nairobi</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Table 3: Distribution of age of the mother

<table>
<thead>
<tr>
<th>Age group</th>
<th>Number</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-20</td>
<td>6</td>
<td>8.0</td>
</tr>
<tr>
<td>21-25</td>
<td>37</td>
<td>49.3</td>
</tr>
<tr>
<td>26-20</td>
<td>24</td>
<td>32.0</td>
</tr>
<tr>
<td>&gt;30</td>
<td>8</td>
<td>10.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

The mean age of the mother was 25.8 years at birth of child.

Table 4: Distribution by birth order

<table>
<thead>
<tr>
<th>Birth order</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>31</td>
<td>41.3</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt;</td>
<td>21</td>
<td>28.0</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>15</td>
<td>20.0</td>
</tr>
<tr>
<td>4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td>&gt; 4&lt;sup&gt;th&lt;/sup&gt;</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Table 6: Distribution as per the occupation of the mother

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Number</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Housewife</td>
<td>50</td>
<td>66.8</td>
</tr>
<tr>
<td>Farmer</td>
<td>18</td>
<td>24</td>
</tr>
<tr>
<td>Teacher</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Business</td>
<td>2</td>
<td>2.6</td>
</tr>
<tr>
<td>Clerk</td>
<td>2</td>
<td>2.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
Figure 2: Distribution by level of education of the mother

![Bar Chart]

- None: 5
- Primary: 48
- Secondary: 16
- Above secondary: 6

Education level

- None
- Primary
- Secondary
- Above secondary
### Table 7: Distribution of monthly income of the mother.

<table>
<thead>
<tr>
<th>Income (Kshs)</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 2000</td>
<td>39</td>
<td>52</td>
</tr>
<tr>
<td>2001-4000</td>
<td>10</td>
<td>13.3</td>
</tr>
<tr>
<td>4001-6000</td>
<td>10</td>
<td>13.3</td>
</tr>
<tr>
<td>6001-8000</td>
<td>2</td>
<td>2.6</td>
</tr>
<tr>
<td>8001-10000</td>
<td>4</td>
<td>5.3</td>
</tr>
<tr>
<td>Undisclosed</td>
<td>10</td>
<td>13.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Note: Exchange rate at the time of the study was Kshs 79 per one dollar.

### Table 8: Distribution as per time of first antenatal attendance

<table>
<thead>
<tr>
<th>Time of attendance</th>
<th>Number</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>3rd trimester</td>
<td>47</td>
<td>62.7</td>
</tr>
<tr>
<td>2nd trimester</td>
<td>25</td>
<td>33.3</td>
</tr>
<tr>
<td>1st trimester</td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
Figure 3: Use of folic acid prenatal and antenatal
Figure 4: Distribution of exposure to teratogens during pregnancy

- Not exposed: 80%
- Exposed: 20%
Figure 5: Distribution of type of lesion

- Spina bifida occult: 21.3%
- Spina bifida cystica: 78.7%
Figure 6: Distribution as per the specific type of lesion
Table 9: Distribution as per the lesion by paralysis

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Paralytic</th>
<th>Non paralytic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spina bifida occult</td>
<td>6</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>Spina bifida cystica</td>
<td>19</td>
<td>40</td>
<td>59</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25</strong></td>
<td><strong>50</strong></td>
<td><strong>75</strong></td>
</tr>
</tbody>
</table>

For all patients 33.3% had paralysis while 66.7% had no paralysis

Table 10: Distribution as per the level of lesion

<table>
<thead>
<tr>
<th>Level of lesion</th>
<th>Number</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td>Thoracolumbar</td>
<td>4</td>
<td>5.3</td>
</tr>
<tr>
<td>Lumbar</td>
<td>57</td>
<td>76.0</td>
</tr>
<tr>
<td>Lumbosacral</td>
<td>9</td>
<td>12.0</td>
</tr>
<tr>
<td>Sacral</td>
<td>4</td>
<td>5.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Table 11: Distribution as per preoperative diagnostic investigations

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultrasound of the skull</td>
<td>50</td>
<td>66.7</td>
</tr>
<tr>
<td>Ultrasound of the lesion</td>
<td>10</td>
<td>13.3</td>
</tr>
<tr>
<td>X-ray of the spine</td>
<td>7</td>
<td>9.3</td>
</tr>
<tr>
<td>Ultrasound of the abdomen</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>None</td>
<td>5</td>
<td>6.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
Table 12: Distribution of associated congenital anomalies with type of spina bifida lesion

<table>
<thead>
<tr>
<th>Malformation</th>
<th>Type of spina bifida</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Meningocele</td>
<td>Myelomeningocele</td>
<td>Lipomyelomeningocele</td>
<td>Occulta</td>
<td></td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>10</td>
<td>24</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Congenital talipes</td>
<td>1</td>
<td>5</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>equinovarus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urological abnormalities</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Arnold malformation</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>12</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>35</td>
<td>9</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

Table 13: Distribution as per age of the patient at operation

<table>
<thead>
<tr>
<th>Age in months</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>7-12</td>
<td>12</td>
<td>16</td>
</tr>
<tr>
<td>13-18</td>
<td>14</td>
<td>18.7</td>
</tr>
<tr>
<td>19-24</td>
<td>14</td>
<td>18.7</td>
</tr>
<tr>
<td>Above 24</td>
<td>26</td>
<td>34.6</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100</td>
</tr>
</tbody>
</table>

The median age at operation was 20 months.
Figure 7: Distribution of outcome of surgery

<table>
<thead>
<tr>
<th>Complication</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wound sepsis</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>CSF leak</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Meningitis</td>
<td>2</td>
<td>2.7</td>
</tr>
<tr>
<td>Paralysis</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td>Death</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td>None</td>
<td>62</td>
<td>82.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>75</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

NB. One patient died in theatre after the operation due to anaesthetic complications.
Table 15:  Condition of skin cover of spina bifida lesion versus surgical complication.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Skin cover of spina bifida lesion</th>
<th>Normal skin</th>
<th>Thin skin</th>
<th>Ulcerated skin</th>
<th>CSF leak</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td></td>
<td>16</td>
<td>44</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Wound sepsis</td>
<td></td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Meningitis</td>
<td></td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td></td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td><strong>20</strong></td>
<td><strong>46</strong></td>
<td><strong>6</strong></td>
<td><strong>3</strong></td>
</tr>
</tbody>
</table>

Wound complications were the most common following surgery 8% of the patients (table 14.)

**Figure 8: Shunt placement**

![Shunt placement chart]

- No shunt placement: 45.3%
- Shunt placement: 54.7%
Table 16: Timing of shunt placement and correlation of surgical complication at the Spina bifida lesion

<table>
<thead>
<tr>
<th>Timing of shunt placement</th>
<th>Number of patients</th>
<th>Number of surgical complications</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre operative.</td>
<td>33</td>
<td>9</td>
<td>80.5</td>
</tr>
<tr>
<td>Intra operative.</td>
<td>2</td>
<td>0</td>
<td>4.9</td>
</tr>
<tr>
<td>Post operative.</td>
<td>6</td>
<td>2</td>
<td>14.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>41</strong></td>
<td><strong>11</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
DISCUSSION

There were a total of 75 patients aged between 5 days to 15 years with a median age of 17 months seen during the study period (table 1). 65.4% were aged below 2 years, children aged 2-3 years accounted for 18.6% and above 3 years accounted for 16.0%. This is because most of the children were not referred in the neurosurgical immediately they were born (table 1). This lead to the delay for the operation in these children at median age of 20 months.(table 13).

For those who presented late above 3 years, majority had spina bifida occult which did not manifest at birth but presented later in life with kyphoscoliosis or back pain.

In the study, there were more females than males with a ratio of 1.20:1 (figure 1). This pattern resembles that seen in people of Celtic origin in whom females are more affected approximately 60-70% of the children (13)

Fifty six percent of the patients came from Central Province (table 2) followed by Western Province 14.8%, Eastern Province accounted for 12% with the other provinces contributing 17.2%.

Whether the high contribution by the Central Kenya is due to it’s proximity of the geographical location to K.N.H. or there’s an associated teratogen is still unknown.

Most of the patients were 1st born 41.3% (table 4) with the parents being young mothers of mean age 25.8 years (table 3). The occupation of the mothers were house wife 66.8% (table 6) with primary school level of education 48% (figure 2)

The total gross monthly income for most (52%) of the patients mothers fell below Ksh 2000 with 26.6% of the parents being in the Ksh2001-6000 wage bracket. 13.3% refused to disclose the total monthly gross income while the rest earned between Ksh 6001-10000 (table 7).
This shows that many of the patient’s parents are low income earners, as has been shown in the British isles and many other parts of the world that low social economic status has been implicated as an etiology of Neural tube defects (28).

Majority of the mothers first attended A.N.C. in their third trimester 62.7% (table8), 33.3% in the second trimester and 2.7% in the first trimester. This is shows that majority attended for purposes of delivery when the defects had already occurred.

In this study, only 1.3% of the patients (figure3) had used folic acid antenatally. This was quite low knowing the beneficial effects of folic acid as has been shown by American and Chinese health workers (4,13,22).

Twenty percent of the mothers had exposure to the known teratogens during pregnancy (figure4), but this cannot be directly incriminated as the causes is usually multifactorial including both genetic and environmental factors (28,29).

Spina bifida cystica accounted for 78.7% of the lesion (figure5) with myelomeningocele the commonest lesion 46.7%. Mark R Foster (31) et al in his study found myelomeningocele accounted for over 80% of the cases.

Maingi in his study in K.N.H. 1975-1979 (6) 108 patient had spina bifida cystica and 71% of these had myelomeningocele.

Thirty three point three percent of the patients had paralysis (table 9), of this 78% had myelomeningocele. This is comparable to studies done in U.S.A. in which it showed 80% of the patients with myelomeningocele have paralysis and only 20% of the children were incontinent of urine (13). Of all the patients seen in the study, 76% total had the lesion in the lumbar spine (table 10).

These were followed by patients with lesion in the lumbar sacral region 12.0%. Maingi in
his dissertation found that 82% of the patients had lumbar & lumbar sacral
myelomeningocele (6).

Among the imaging studies performed, ultrasound of the skull was the most common
66.7% of the patients. Radiology despite being the cheapest and the most readily
available was among the least performed in only 9.3% of the patients (table 11). Most of
the spine X-rays were done in cases of suspected spina bifida occult. They are important
as they can show whether there is hemi vertebrae or lamina defects. The vertebrae are still
cartilaginous and bony defects are not well demonstrated in the neonate hence the low
incidence of X ray spine.

Transcranial ultrasound is performed during the neonatal period to evaluate the extent of
ventricular enlargement. Initially the ventricles may be normal or only slightly enlarged.
However, after the N.T.D. is closed surgically, the ventricles enlarge (20, 31).

In this study 72% of the patients had associated congenital anomalies (table 12).

Hydrocephalus was the commonest 54.7%.

Richard G. Ellenb (13) in U.S.A. in 2002 reported that 20% of the patients with
myelomeningocele had significant hydrocephalus at birth, another 60-70% develop it
after myelomeningocele repair. In our study 58% of the patient with hydrocephalus
had associated myelomeningocele (table 12).

Mark R Foster (31) in Turkey in 1999 found hydrocephalus was the commonest
associated malformation 35%. Numerous other malformations also found in this study
but in low frequency were cranio-vertebral junction anomaly, Dandy Walker cyst,
congenital talipes equinoverus, syndactily of all toes, Arnold Chiari malformations and
bilateral inguinal hernias.
In this study, the median age at operation was 20 months (table 13). Majority of the patients were operated on few days after they were seen in the clinic (table 1), and (table 13). In 1960, the birth of a patient with myelomeningocele was neural surgical emergency and immediate closure of the defect was required (2,9, ).

A study by Tryfonas.N.P (32) comparing delayed closure 3-7 days to immediate closure (< 48hr) showed little difference in survival. Currently in utero repair is being carried out with good results as assessed by incidence of developing hydrocephalus post natally (33, 34). In this study, delay was probably due to factors such as late referrals, lack of readily available imaging services, complications at presentation and limitation in available theatre space (6).

Wound sepsis and other related surgical complications accounted for 16% of the patients (figure7).

Of this wound sepsis and C.S.F. leak were the commonest complications occurring in 8% and 4% respectively (table 14). There was no association between the timing of surgery and wound complications (table 15). In studies done by Omulo (35) and Nooran (36) in K.N.H. no association was found between age and development of VP shunt complications.

The rate of wound complication and infection may be due to a number of factors related to the general hospital and theatre environment rather than specific operation at K.N.H. (37). Among the theatre related factors are overcrowding, theatre overuse, and poor aseptic technique and long operation times.

A long waiting period in the wards has a high association with nosocomical infections
(37), patient related factors might also contribute.

Mortality rate was 1.3% (table14) as this study did not include those patients who had spina bifida cystica and died before operation.

In addition to the repair of the spina bifida, 54.7% (figure 8) of the patients had surgery for insertion of V.P. shunt. Patients with myelomeningocele had a higher association with hydrocephalus than meningocele and occulta (table12).

Sutton L.N. (34) found in university of Washington school of medicine that patients who manifest ventriculomegaly after birth undergo shunt placement after myelomeningocele closure but while under the same anaesthetic.

In this study, out of the 41 patients who had shunt surgery, preoperative insertion of V.P. shunt was performed in 80.5% of the patients (table 16) followed by post operative V.P. shunt in 14.6%. In 4.9% V.P. shunt were inserted at the same time as repair of the spina bifida lesion.

Shunt placement not only decreased future anaesthetic risk but also the chances of C.S.F. leaking through the Myelomeningocele closure. Maria Matero (39) found shunt placement to improve neuropsychological functioning of young adult patients with spina bifida and apparent clinically assessed hydrocephalus showing abnormal intracranial pressure.
CONCLUSION

Spina bifida is a common congenital anomaly in this country. Currently Kenyatta National Hospital is the only public hospital in this country that has the capability to treat patients with this condition. Most of the children with this condition are likely to have an associated congenital malformation.

Females have a high preponderance than males in the ratio of 1:20:1.0. Majority of their mothers are housewife with low social economic status. Most of them had primary school level of education and they attended A.N.C. in their third trimester.

There was no awareness of the use of folic acid as a preventive measure for most of the mothers.

Spina bifida myelomeningocele was the commonest type of lesion found in our country and also accounts for the majority of children with paralysis

The predominant lesions were in the lumbar vertebrae. Imaging investigations were under utilized despite being very useful in planning of surgery and preventing intra operative difficulties.

Ultra sound of the skull to assess for hydrocephalus was performed in most of the patients.

Spina bifida cystica is commonly associated with hydrocephalus which may be present before or after repair of the repair spina bifida cystica lesion.

Shunt placement is normally carried out before the operation of the lesion but can be done at the same time or after the repair of spina bifida lesion.

The median age at operation was 20 months. This was due to time given for cystica
lesions to granulate and form healthy skin and time of presentation in the clinic.

Also spina bifida occult were operated on latter if the patient develops neurological deficit.

Wound sepsis, C.S.F. leak and meningitis were the commonest complication encountered in this study.
RECOMMENDATIONS

Preventive health care programmes targeting the region which have the highest incidence should be carried out.

This entails addition of folic acid in ‘enriched’ labeled breads and cereal grains. As most pregnancies are unplanned, making this vitamin available in readily available food sources will increase the likelihood of effective prevention greater than relying on prescribed vitamin use.

Mothers should be encouraged to attend clinic prenatally, in first trimester for ultrasonography screening for those at risk and measurement of amniotic or maternal serum A.F.P..

Doctors, nurses and other staff should be trained on how to manage this condition and be dispatched to peripheral hospitals at least at the level of provincial hospital.

Currently management of in utero repair should be introduced in our country by providing the necessary equipment and personnel in selected institutions.

Imaging investigations like transcranial ultrasound, abdomen should be performed in all patients as a routine to rule out other associated anomalies as it is readily available.

Management of this condition should be multidisciplinary especially when the child has paralysis and large lesion i.e. Neurosurgeon, plastic surgeon, orthopedic surgeon, pediatrician occupational therapist physiotherapist nurses parents and teachers should be involved.
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APPENDIX I

QUESTIONNAIRE

Name of sibling: .................................................................

.................................................................

IP NO: .................................................................

Age: .................................................................

Sex: .................................................................

District: .................................................................

Date of birth: .................................................................

Date of admission: .................................................................

Name of mother: .................................................................

Age: .................................................................

Parity: .................................................................

Age at 1st delivery .................................................................

Position of child in the family .................................................................

Occupation of the mother .................................................................

Attendance of ANC - 1st trimester .................................................................

- 2nd trimester .................................................................

- 3rd trimester .................................................................

...
Diagnostic investigations

Ultra sounds for

<table>
<thead>
<tr>
<th>Spina bifida</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdomen</td>
</tr>
<tr>
<td>Skull</td>
</tr>
</tbody>
</table>

X-Ray of the Spine

If Yes…RESULTS

Any exposure to teratogens during pregnancy. Yes No

- Antihistamine.
- Anticonvulsants.
- Sulphonamides.
- Potato blight.
- Others.

**level of the lesion.**

- Cervical.
- Thoracic.
- Thoracolumbar.
- Lumbar.
- Lumbosacral.
- Sacral.

Mother's level of education.

- primary.
- secondary.
- college.
| USE OF FOLIC ACID  
PRENATAL AND ANTENATAL | Yes | No |
<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paralytic</td>
<td></td>
<td></td>
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<tr>
<td>Non paralytic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinal bifida occult</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spina bifida cystica</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spina bifida myelomeningecele</td>
<td></td>
<td></td>
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<tr>
<td>Spin bifida lipomyelomeningocele</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spina bifida meningocoele</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Conditions associated</strong></td>
<td>yes</td>
<td>No</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arnold Chairi malformations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital talipes equino varus</td>
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<td></td>
</tr>
<tr>
<td>Urological abnormality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine /stool incontinence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other congenital abnormalities</td>
<td></td>
<td></td>
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<tr>
<td>If yes specify...</td>
<td></td>
<td></td>
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<tr>
<td>Age at operation...</td>
<td></td>
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<td>.</td>
<td></td>
<td></td>
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<tr>
<td>Outcome of surgery</td>
<td></td>
<td></td>
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<td>.</td>
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</tr>
</tbody>
</table>
were there any complications  

Yes  No.

wound sepsis.

csf leak.

meningitis.

paralysis.

others.

V P SHUNT inserted  

yes  no.

if yes Intra-op  

pre-op  post-op.
APPENDIX II

CONSENT EXPLANATION

My name is Dr Ndegwa. I am carrying out a research on pattern of presentation of spina bifida at Kenyatta National Hospital. The study will commence on March 2004 till I get the required sample size. Authority to carry out the research will be from Ethical and Research Committee of K.N.H.

This study will enable us to know, the commonest form of presentation of spina bifida. The geographical distribution pattern, the age and the parity of the mother, the time of attendance of ANC, any exposure to teratogens and use of folate periconception and the time of operation.

Information gathered shall be solely for the purpose of medical research. You are under no obligation to either accept or refuse to be enrolled in this study. Your decision shall in no way affect any treatment you may receive in the hospital. You will be enrolled by giving consent and study number. History and physical exam and other investigations will be carried out as per your condition. You will then undergo surgery and any complications arising will be noted.

Apart from the normal risk any other patient undergoing surgery for this condition faces there are no extra risks you are exposed to in the study. As a parent you should feel free to ask any question that may not be clear. If you wish your child to be part of this study, then sign the consent form below.

I can be contacted via the following telephone number 0722-477213.
Consent Form

I _______________ OF ______________

Hereby agree to my child to be enrolled in the study of Spina Bifida. I understand and agree with all the above all the information gathered shall be confidential and used for medical research. I’m under no obligation to accept or refuse to be part of this study and that my decision shall in no way affect any treatment I may receive in this institution.

Signature/Thumb print _______________  Investigator
Dr.Ndegwa
Signature _______________

Date _______________
CHETI CHA KUKUBALI

MIMI____________________________ WA___________________

Nakubali mtoto wangu kuhusishwa kwa utafiti kuhusu ugonjwa wa uti wa mgongo.
imekubali na yote niliyoelezwa. Matokeo yatatumwa tu kwa minajili ya utafiti
wa sayansi ya matibabu na yatakuwa ya kibinafsi

Naweza kukataa au kukubali na uamuzi wangu hautadhuru au kunizuia kupata
matibabu ninayopasa kupata katika hospitali hii.

Sahihi________________________

Tarehe_______________________