

ASSOCIATED FACTORS INVESTIGATIONS  
AND MANAGEMENT OF UNDESCENDED  
TESTIS (UDT) AT KNH, A PROSPECTIVE  
STUDY.

A DISSERTATION SUBMITTED IN PART FULFILMENT FOR THE  
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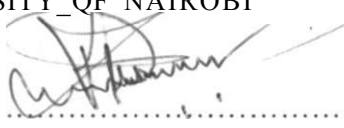
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## **DEDICATION**

I dedicate this thesis:

To my Lord Jesus Christ. In Him I trust

To My lovely wife Leah, my daughters Faith and Rexella for their understanding support,  
patience and prayers.

## **rTST OF CONTENTS**

	<b>PAGE</b>
1. Title.....	i
2. Declaration.....	ii
3. Acknowledgements .....	iii
4. Dedication.....	iv
5. List of contents.....	v
6. List of figures.....	vi
7. List of tables.....	vii
8. List of abbreviations.....	viii
9. Summary.....	1
10. Introduction.....	3
11. Literature review.....	5
12. Rationale of the study.....	16
13. Objectives.....	17
14. Materials and methods.....	18
15. Results.....	22
16. Discussion.....	46
17. Conclusions.....	53
18. Recommendations.....	55
19. Appendix I.....	56
20. Appendix II.....	61
21. References.....	62

**pcT OF FIGURES**

	<b>PAGE</b>
Figure I	.23
Figure 2.....	24
Figure 3.....	25
Figure 4.....	27
Figure.....	31
Figure 6.....	32
Figure 7.....	34
Figure 8a.....	39
Figure 8b. . . . .	39
Figure.	
Figure.	
Figure 10.....	43

## **list of tables**

Table 1.....	22
Table 2.....	25
Table 3.....	26
Table 4.....	28
Table 5.....	29
Table 6.....	30
Table 7.....	31
Table 8.....	33
Table 9.....	35
Table 10.....	36
Table 11a.....	37
Table 11 b.....	38
Table 12.....	42
Table 13.....	44
Table 14.....	44
Table 15.....	45

## I 1ST OF ABBREVIATIONS

ABP	Androgen binding protein
CS	Caesarean Section
CTEV	Congenital talipes equinovarus
CT Scan	Computerized tomography scan
FSH	Follicular - stimulating hormone
GFN	Genito-femoral nerve
GnRH	Gonadotropin releasing hormone
hCGH	Human chorionic ganadotropin hormone
IP.No.	In-patient number
LH	Luteinizing hormone
LHRH	Luteinizing hormone-releasing hormone
KEPI	Kenya Expanded Programme of Immunization
KIU	* • Kilo international units
KNH	Kenyatta National Hospital
MRI	Magnetic Resonance Imaging
OBGyn	Obstetric and Gynaecology
RVF	Recto-vesical fistula
SVD	Spontaneous vertex delivery
UDT	Undescended testes
US	Ultrasonography
VSD	Ventriculo-septal -defect
WHO	World Health Organisation



## SUMMARY

A descriptive prospective study was done to review the associated factors, investigations and management of undescended testes at the Paediatric Surgical Unit of Kenyatta National Hospital from June 2003 to November 2003.

There were a total of 57 patients with undescended testes seen within the period of study and out of these 40 patients had had orchidopexy and the other 17 were still pending.

Twenty patients had their serum gonadotropins evaluated and was compared to 20 other children of similar age with normal (descended testes) who had been admitted to paediatric surgical ward for other reasons e.g. incision and drainage of an abscess.

The ages ranged from 1 month to 13 years with mean age of 5 years.

Most of the children with undescended testes were delivered during the month of September 18%, June 12% and October 10%. None was delivered during the month of January and 2% were delivered in the month of February.

First born males were 62%, second born 36%, none was sixth born or above.

The median mothers' age at delivery of children with undescended testes was between 21-25 years (54%). None was 41 years of age and above who delivered a child with undescended testes. None of the mothers gave a history of smoking at any one time before or during pregnancy.

The abnormality detected at a health institution were 58%. The family members detected 42%. Among the family members, the mothers detected 54%, the fathers 21%, the grandmother 21 % and the aunties 4%.

The patients who had other associated anomalies were 28.1% of which hypospadias accounted for 8.7%, umbilical hernia 5.2% and prune belly syndrome 5.2%.

Ultrasound was performed on 42.1% of the patients to locate the testis. None had Computerized Tomography Scan or Magnetic Resonance Imaging.

There was no significant statistical difference in the gonadotropin levels of children with undescended testes and those with normal (descended) testes at all age groups. P values of LH and FSH were 0.790 and 0.070 respectively.

Among the 40 patients who had orchidopexy performed, 33 were first operations and 7 were redo operations. Among the 7 redo orchidopexy, 5 (71.4%) had bilateral undescended testes and 2 (28.6%) had unilateral undescended testes.

Two patients had complication at surgery; one had avulsed vas deferens and the other had testicular artery injured.

## INTRODUCTION

Undescended testes is one of the common childhood disorders, independent of race and geographical region. It affects 3 - 4% of full term newborn boys and by 1 year of age, the incidence of cryptorchidism decreases to slightly less than 1%.

The incidence of undescended testes appears to be increasing <sup>(1)</sup>. There are several theories of the aetiology of undescended testes; however, majority can be summarised into genetic, endocrine imbalance and mechanical factors.

By definition undescended testes is failure of the testis to descend completely into the scrotum; therefore the diagnosis of undescended testes is by physical examination and may be confirmed by ultrasound, Computerized tomography scan, Magnetic Resonance Imaging or Laparoscopy which are also used to locate the position of the testis.

Ideally, undescended testes should be noted during delivery, then subsequently followed up since most of them i.e. 75% of them will descend spontaneously by 1 year.

For the testis to produce viable mature spermatozoa; the temperature of the local environment must be 3-4° C below the body temperature. Testicular maldescent therefore is believed to be responsible for infertility in patients with cryptorchidism.

Recent studies with electron microscope have found out that pathological changes do occur at an early age of life.

Hadziselomovic and Sequchi found ultrastructural changes in the seminiferous tubules after one year and in the Leydig cells within the first year <sup>(2)</sup>. These changes include the degeneration of mitochondria, loss of cytoplasmic ribosomes and smooth endoplasmic reticulum, and an increase in the collagen fibres in the spermatogonia and Sertoli cells.

At present there are two types of hormonal therapy i.e. Human chorionic gonadotrophin and gonadotrophin releasing hormone. Gonadotrophin releasing hormone is used under the premise that children with undescended testes have abnormality in the secretion of gonadotrophin releasing hormone from the hypothalamus as shown by the decrease in basal luteinizing hormone levels while human chorionic gonadotrophin is given under the premise that stimulating Leydig cells will result in an increase in plasma testosterone, which will promote testicular descent.

The mainstay of treatment of undescended testes is surgery. The goal is to achieve placement of the testis in the scrotum at optimum time to allow normal maturation of the germinal epithelium. Surgery at the age of two years is ideal because there is evidence of microscopic deterioration from the age of one year. At one year, the vas deferens and vessels are still very delicate and routine orchidopexy at the age of eighteen months is recommended<sup>(3)</sup>Orchidopexy at ages 6 months - 1 year however is acceptable.

## LITERATURE REVIEW

Historical background

- o John Hunter<sup>14\*</sup> in 1786 described the earliest account of undescended testis. He found the testis in the abdomen in the 7<sup>th</sup> month of fetal life and in the scrotum in the 9<sup>th</sup> month.
- o In 1899 Bevan<sup>(5)</sup> described the operation for undescended testicle and congenital hernia on a child.
- o Keetley (1894)<sup>(6)</sup> and Torek (1909)<sup>(7)</sup> described the Keetley-Torek operation, which embedded the testis into the thigh temporarily then brought back into the scrotum after three to six months,
- o Ombredanne (1910)<sup>(4)</sup> described an operation in which the undescended testis was replaced in the opposite compartment of the scrotum,
- o In 1945 Bishop P.M.F.<sup>(8)</sup> described clinical endocrinology versus the management of the undescended testicle,
- o Fowler R and Stephens F.D. (1963)<sup>(9)</sup> described the role of testicular vascular anatomy in the salvage of high testis,
- o Benson and Lofti (1967)<sup>(10)</sup> described the Dartos Pouch technique of orchidopexy.
- o Enrich R.M. et al (1969)<sup>(11)</sup> described the effects of gonadotrophin in cryptorchidism.
  
- o Persky L. (1971)<sup>(12)</sup> and Firor H.V. (1971)<sup>(1?)</sup> described the two stage orchidopexy.

### **Embryology of normal testicular descent**

The human male and female gonads are indistinguishable before seven weeks gestation. The testes determining gene (SRY, sex determining region on the Y chromosome), initiates testicular development and testicular hormone production. Mullerian inhibiting substance causes Mullerian duct regression.

Testosterone secreted by the Leydig cells of the fetal testis stimulates Wolffian duct development into epididymis, vas deferens and seminal vesicles. Testosterone potency is enhanced ten fold by 5 alpha reductase conversions in genital tissues to dehydrotestosterone, which promotes phallus enlargement and genital fold fusion to form the scrotum.

The testis migrates from the urogenital ridge on the posterior abdominal wall to the region of the future inguinal canal by 15 weeks gestation (first stage). No further movement occurs until 28-35 weeks when the gubernaculum protrudes into the developing anterior abdominal wall creating the inguinal canal, swells into a bulb and then migrates towards the scrotum.

The tunica vaginalis develops into the gubernaculum, creating a space for the testis to descend into the scrotum, aided by abdominal pressure, by week 35 (the second stage).

Mullerian inhibiting substance may cause transabdominal migration and gubernacular swelling. It probably also causes testicular gonocyte transformation to type A spermatogonia during infancy, which may be significant for normal testicular maturation and later sperm production.

Testosterone appears to cause inguino-scrotal descent by its action on the genito-femoral nerve (GFN).

Miniberg and Schlossberg <sup>(14)</sup> noting the large number of epididymal abnormalities suggested that the epididymis may have a role in testicular descent. The view is shared by Hadziselimovic <sup>(15)</sup>.

The portion of the vas deferens close to the testicle grow more rapidly, producing development of the epididymis which pushes or carries the testis caudally. As this is also under androgenic stimulation, it is likely that abnormalities of the epididymis and vas deferens are due to local or general areas of androgen insensitivity.

### **Control of testicular function**

Follicular-Stimulating Hormone is tropic to the Sertoli cells, in which the developing spermatozoa are embedded. Follicular-stimulating hormone acts on the Sertoli cells to facilitate the last stages of spermatid maturation. It also stimulates the secretion of Androgen Binding Protein and inhibin. Inhibin feeds back to inhibit follicular-stimulating hormone secretion. Luteinizing hormone is tropic to the Leydig cells and stimulates secretions of testosterone, which in turn feeds back to inhibit luteinizing hormone secretion.

### **Causes of maldescent**

There are several theories on the aetiology of undescended testes. However, majority can be summarized into three main categories, which include:

#### **Endocrine imbalance**

Present evidence indicates that the process of descent is mediated by the foetal hypothalamic-pituitary-testicular axis <sup>(16)</sup>. There is evidence that cryptorchidism frequently is associated with an abnormality of hormonal production <sup>(17)</sup>.

#### **Mechanical factors:**

These factors include:

- Absence of gubernaculum or abnormal gubernaculum attachment.
- Abnormal epididymis
- The inability to generate intra-abdominal pressure, such as with prune-belly syndrome or omphalocele
- Short testicular vessels or short vas deferens, which cannot allow the testicle to descent into the scrotum.

- Mechanical; obstruction e.g. a fascial sheet blocking the entrance to the scrotal canal.

Usually these patients have normal testes<sup>(18,19)</sup>.

### **Hereditary factors**

Children in this group are thought to be genetically predisposed to have undescended testes, and their testes are grossly and histologically abnormal<sup>(18,19)</sup>. Hecker and Hienz found up to 30% to 60% of contralateral descended testes to be abnormal<sup>(20)</sup> majority of this group will probably be infertile.

### **Diagnosis of Undescended Testes**

Physical examination should differentiate between palpable or impalpable, (cryptorchid) or retractile testis. One hand attempts to 'milk' the testis down, while the other hand tries to feel the testis. To differentiate a retractile from a true cryptorchid testis, manual traction is gently placed on the testis to try to bring it down to the bottom of the scrotum; and if it does reach the scrotum this is considered a retractile testis. If it does not a second attempt to bring the testis down (at another visit) is necessary. If it fails a second time, the testis must be considered cryptorchid.

Ultrasonography is helpful in locating the testis if it is within the inguinal canal, if it is underneath the aponeurosis of the external oblique muscle or is intra-abdominal and also measures the size of the testicle. CT and MRI can be used to locate the impalpable testis; however the rate of false negative results from CT and MRI are reported as 38 - 87%<sup>(21)</sup>.

Laparoscopy for diagnosing the intra-abdominal testis has an accuracy of 88-100%<sup>(22)</sup>.

The laparoscopic technique combines the advantage of diagnosing an intra-abdominal or absent testis with the possibility of therapeutic procedure such as the first stage of the Fowler-Stephens technique.



Hormonal tests can be used to distinguish bilateral anorchia from bilateral undescend. After a short course of (3HCG 2KIU (kilo international units) daily for three days in patients with bilateral anorchia there is no increase in testosterone and basal gonadotrophin levels are extremely high.

### **Complications of Undescended Testes**

#### Neoplasia

About 10% of testicular tumors occur in an undescended testis. Statistically, the likelihood of malignant transformation of an undescended testis is reported to be 35 - 48 times higher than that of a normal testis <sup>(23)</sup>.

As to the location, an intra-abdominal testis is more likely to undergo malignant transformation than an inguinal testis. In bilateral cryptorchidism, there is a 15% chance of developing a tumor on the opposite side if one testis develops a tumor <sup>(24)</sup>. The probability increases to 30% if both testes are intra-abdominal.

Orchidopexy does not prevent carcinogenesis but allows the surveillance of the gonad in patients with impalpable testis possible. Therefore all impalpable testes should be either removed or placed in a position to allow palpation. For all the patients with cryptorchidism, the age of treatment of an undescended testis appear to have no effect on the risk of testicular cancer <sup>(25)</sup>.

#### **Torsion**

Many gonadal remnants obtained at exploration for impalpable testes contained haemosiderin and calcium deposits, thus supporting the theory that, testicular absence may have been caused by testicular torsion <sup>(26)</sup>.

## Testicular atrophy

Initially the testicular volume in cryptorchidism is relatively normal. However, long-term follow up show that testicular atrophy eventually results if the condition is untreated (27)

## Tj^uma

There is increased trauma to a testicle located at the pubic tubercle. Orchidopexy offers protection (19).

## Hernia

After testicular descent, the processus vaginalis closes between the eighth month of foetal life and the first month after birth. However, when the testis fail to descend, the processus vaginalis remains patent. Clinically, the patent processus vaginalis appears as a hernia or hydrocele (25).

## Fertility

Increased testicular temperature, may reduce fertility. The higher temperature to which an intraabdominal testis is exposed may severely retard normal germinal maturation. Eighty percent of men with bilaterally descended testis are fertile compared with 75% with unilateral descent and less than 30% with bilateral undescended testes. A unilateral undescended testes may reduce the sperm count from the normally descended testis, so called 'endocrinopathy of undescended testes' (28). Studies have suggested that autoantibodies to the cryptorchid testis may be produced, which can cause degenerative changes in the descended testis (29).

## **gffrrt\* of Temperature**

Spermatogenesis requires a temperature considerably lower than that of the interior of the body. The testes are normally maintained at a temperature of about 32°C. They are kept cool by air circulating around the scrotum and probably by heat exchange in a counter current fashion between the spermatogenic arteries and veins. When the testes are retained in the abdomen, or when, in experimental animals they are held close to the body by tight cloth binders, degeneration of the tubular walls and sterility result.

## **Histological aspects.**

In the classic paper by Cooper <sup>(30)</sup>, smaller seminiferous tubules, fewer spermatogonia and more tubular tissue were found in cryptorchid testes appearing 1.5 years of age. The histological alterations were more pronounced, the higher the testis was from the bottom of the scrotum and the longer the testis remained cryptorchid. More recently, electron microscopy has shown ultrastructural changes of the seminiferous tubules at the beginning of the second year of life. These changes include the degeneration of mitochondria, loss of cytoplasmic ribosomes and smooth endoplasmic reticulum, and an increase in the collagen fibres in the spermatogonia and Sertoli cells <sup>(31)</sup>. On light microscopy, the basement membrane of the seminiferous tubules is thickened, the tubules are narrow and the interstitium shows severe fibrosis. Some of these histological studies of cryptorchid gonads reveal that there is a normal number of germ cells during the first six months of life but progressive deterioration thereafter. By puberty, progressive histological damage has reduced the size of undescended testis and spermatogenesis is severely impaired. The authors analysed whole testicular specimens removed in post-pubertal men for cryptorchidism, more than two thirds of cryptorchid testes (36/52) had no germ cells <sup>(32)</sup>. This rate increased significantly with age. Even if germ cells were present, as in the minority of specimens, they would not contribute to fertility.

## **Hormonal aspects**

Cryptorchid infants and children have been shown to have lower levels of basal luteinizing hormone and testosterone, as well as blunted hormonal response after stimulation with gonadotrophin releasing hormone. Histomorphological analyses of biopsies from undescended testes taken before the third month of life showed a hypoplasia of the Leydig cells, normal total germ cell counts and fewer adult dark spermatogonia, which have a distinct appearance and may serve as the most easily recognized marker of the successful transformation of gonocytes into spermatogonia.

These morphometric support the hypothesis that the primary injury to the undescended testes is a decreased gonadotrophin level at age 60 - 90 days. Consequently, the interruption of the hypothalamic- pituitary-gonadal circuit produces the primary histological lesion in the testes i.e. hypoplasia and regression of foetal Leydig cells. The depletion of Leydig cells persists through to the age of 12 years. These abnormalities are present in the cryptorchid and contralateral descended testis but they are more severe of earlier onset and more progressive in the cryptorchid testis <sup>(33)</sup>.

## **Laboratory Evaluation**

The collected results of various endocrined studies in infants with unilateral or bilateral cryptorchism show plasma LH and the postnatal rise in testosterone levels to be significantly lower than of normal males. A plasma FSH hyper-response to LH RH (i.e. GnRH) has been found in pre pubertal boys with both unilateral and bilateral cryptorchidism. There is no difference between the cryptorchid groups <sup>(34)</sup>.

## Treatment

### Reasons for correcting undescended testes

- 1) Fertility may be improved <sup>(35)</sup>.
- 2) The undescended testes has an increased susceptibility to malignant transformation and consequently should be placed in a position where it can be palpated or monitored easily.
- 3) To allay the patients or/and parents psychological feeling
- 4) Prevent torsion.
- 5) Prevent diminished testicular hormone production.
- 6) Correct associated hernia.
- 7) Correct associated hydrocele.
- 8) Achieve cosmetic result.
- 9) Minimize potential trauma to the testis.

Two avenues of therapy are available for placing the undescended testis into the scrotum: hormonal and surgical. However the optimal time for attempting to place the retained testis into the scrotum and the treatment policy for post pubertal patients is debatable. Sperm analysis represents the functional status of the testis but to the patient, paternity is a true gauge of fertility although it is a less specific measure of testicular function. Both variables have been used to evaluate the treatment of cryptorchidism and controversial results obtained with both variables <sup>(19)</sup>.

### Hormonal treatment.

At present there are two types of hormonal therapy, i.e. Human chorionic gonadotrophin and gonadotrophin releasing hormone. Gonadotrophin releasing hormone is used under the premise that children with cryptorchidism have an abnormality in the secretion of gonadotrophin releasing hormone from the hypothalamus, as shown by a decrease in basal luteinizing hormone levels, while human chorionic gonadotrophin is given under

the premise that stimulating the Leydig cells will result in an increase in plasma testosterone, which will promote testicular descent<sup>(1)</sup>.

The success rate for human chorionic gonadotrophin therapy is 14-50% and for GnRH is 6- 70% <sup>(35)</sup>. There appears to be no difference in success rate between unilateral and bilateral cryptorchidism. Hormonal therapy is contraindicated in boys with associated inguinal hemias, those with scar fixation following previous surgery, or those with ectopic testes, because these gonads are mechanically prevented from entering the scrotum.

If the testis has not descended after completion of the recommended course, then surgery is indicated, as excessive doses of Human Chorionic Gonadotrophin may be associated with precocity and accelerated epiphyseal maturation.

### **Surgery**

Surgery before the age of two years is ideal because there is evidence of microscopic deterioration and failure of gonocyte transformation from the age of one year<sup>(1)</sup>.

### **Palpable testis**

Provided the testicular vessels are adequately mobilized retroperitoneally, the testicle should come into the scrotum using a standard orchidopexy technique. There is nearly always a complete hernia sac requiring herniotomy.

### **Impalpable testis**

Laparoscopy is recommended. In most patients an intra-abdominal testis is seen and the testicular vessels can be clipped and divided well above the testis as the first stage of a Fowler- Stephens, two stage orchidopexy. This allows testicular blood supply to develop from the artery of the vas, and the testis can be brought down on the vas six months later. Avoiding groin exploration at the first stage prevents damage to the delicate connection

between vasal and testicular vessels in peri-testicular tissues. The success rate is 70-85%. Parents should be warned of potential testicular loss.

Allotransplantation involves high testicular vessel ligation, the testis is brought down to the scrotum and microvascular anastomosis, to the reflected inferior epigastric vessels is performed.

### **Prognosis**

Whether early orchidopexy will reduce the risk of malignancy or improve fertility is unknown. With adverse histological change being found in the second year of life, orchidopexy before the age of two years seems attractive. This may also allow gonocyte transformation to type A spermatogonia to proceed. These potential benefits have to be weighed against the risk of performing clinically difficult orchidopexy especially in patients under one year of age, which may result in vessel or vasal damage <sup>(1)</sup>.

## RATIONALE OF THE STUDY

Undescended testis affects 3-4% of full term boys. It is common in boys born prematurely and has a decreased incidence as the infant matures; until 6-12 months of age after which incidence stays relatively stable at slightly less than 1%.

present evidence indicates that the process of descent is mediated by the foetal hypothalamic pituitary testicle axis. There is evidence that undescended testes frequently is associated with abnormality of hormonal production <sup>(16)</sup>.

This study wants to determine the gonadotrophin (follicular-stimulating hormone, luteinizing hormone) levels of children with undescended testes and compare with the levels of children of the same age group with normal (descended) testicles.

If luteinizing hormone, follicular-stimulating hormone levels are low in children with undescended testes, then the abnormality would be hypothalamic-pituitary-testicular axis and these children may benefit from exogenous Gonadotrophin releasing hormone treatment. If luteinizing hormone, follicular-stimulating hormone levels are high in children with undescended testes, then most probably testosterone levels are low due to the morphological changes of the undescended testes affecting the Leydig cells.

If follicular stimulating hormone, luteinizing hormone- levels are within the levels of those with normal (descended) testicles at all age groups then the proportion of those with abnormality with hypothalamic-pituitary-testicle axis are low, also the morphological changes of the undescended testes does not affect the hormonal (testosterone) production by the Leydig cells.

This study will also look into the associated factors of undescended testes e.g., smoking by the mother during pregnancy, age of the mother, birth order, seasonal variations, associated anomalies. The person who noticed the abnormality (or place) where the abnormality (undescended testes) was noticed first would be noted; i.e. by the medical personnel or the family members.



# OBJECTIVES

## **Broad objective**

To determine the associated factors of UDT and investigations specifically hormonal evaluation in children with UDT and compare to those of normal children (with descended testes)

## **Specific objectives**

1. To relate birth order, season of delivery, age of mother and smoking by the mother during pregnancy to the occurrence of UDT.
2. To find out the person who commonly first notices the condition of UDT i.e. whether they are parents, grand parents, caretaker or medical personnel.
3. To show commonly associated anomalies of UDT.
4. To determine the serum gonadotrophin (LH, FSH ) levels in children with UDT and compare to the levels in children without cryptorchidism within the same age group.
5. To show the common immediate surgical complications encountered during orchidopexy at KNH

## METHODOLOGY

### **Study Design**

A descriptive study was undertaken at the Paediatric Surgical Unit of the Kenyatta National Hospital covering a period of 6 months.

### **Study population**

All children with undescended testes seen at the Paediatric Surgical Unit of the Kenyatta National Hospital within the study period were included in the study. Twenty patients with normal (descended) testicles who had been admitted to the Paediatric Ward for other reasons were randomly selected and had their gonadotrophin levels tested for comparison with those of children with undescended testes of same age groups.

### **Inclusion criteria**

All children who presented to the Paediatric Surgical Unit with undescended testes were included in this study and the randomly selected children with normal (descended) testes.

### **Exclusion criteria**

Patients with retractile testes, ectopic tests or anorchia and ambiguous genitalia were not included in the study.

## SAMPLE SIZE

Sample size estimation was derived from the formula:

$$N = \frac{Z_a \times Z_b [P(P-1)]}{D \times D}$$

Where:

Z<sub>a</sub> = Standard deviation of the 95<sup>th</sup> percentile (1.96)

D = Width of the confidence interval (0.05)

P= Expected proportion of patients with undescended testes as calculated from the number of total operations per year done at the Paediatric Surgical Unit at Kenyatta National Hospital=3.5% (ranges from 0.9-6.1%)

N = Sample size

$$N = \frac{1.96 \times 1.96 [0.035(0.035-1)]}{(0.05)^2}$$

52 patients

## DATA COLLECTION

Information was obtained from the parents/guardians regarding the patient/s birth history; family, social history including age of mother during delivery of the patient and whether she smoked during the pregnancy.

The person who first noticed the condition (of undescended testes) was noted i.e whether it was by the parents, grandparents, and caretakers or was noticed at a health facility.

Physical examination was conducted to confirm the diagnosis of undescended testes and to check for any other associated anomalies e.g. hypospadias. This was done by the main researcher who again referred to the patients' file to certify the findings.

Twenty patients of those with undescended testes were selected randomly and 20 patients with normal (descended) testicles of the same age group as those with undescended testes were selected randomly out of the patients admitted to paediatric surgical ward for other reasons e.g. incision and drainage of an abscess, paraphimosis etc and had their gonadotrophins evaluated together.

Informed consent was obtained from the parents/guardians for blood to be withdrawn from their children and gonadotrophin levels evaluated. The blood was taken to the laboratory where analysis was to be done and the serum stored at  $-21^{\circ}\text{C}$  until the time of analysis.

Serum follicular-stimulating hormone, luteinizing hormone were analysed by enzyme immuno assay as per World Health Organisation standards and the controls was by internal quality control method.

These tests were done at the Department of Obs Gyn University of Nairobi laboratory. Laboratory, which deals mostly with hormonal evaluation of patients with infertility.

## **PERSONNEL**

All information was collected by the main researcher.

## **DATA PROCESSING AND ANALYSIS**

Data obtained was carefully studied and entered into an IBM computer. Analysis was carried out using SAS computer statistical software to derive descriptive statistics .

## **ETHICAL CONSIDERATIONS**

1. The proposal was submitted to the Ethical and Research Committee of Kenyatta National Hospital and clearance was given after the standards required were met before data collection.
2. The parents/guardians gave informed consent for the evaluation of gonadotrophin levels of their children.
3. All the information obtained was treated with confidentiality

### **Study limitation**

1. The sample size was limited by time factor.
2. Some surgeons did not mention or document the size of the testicle during orchidopexy and whether there was any intraoperative complication that occurred or not.
3. The assay of serum gonadotrophin was very expensive and its was the main factor that limited the number of patients who had their gonadotrophin levels checked.

## RESULTS

During the period of this study, a total of 57 patients with undescended testes were seen and out of these, 40 patients had orchidopexy performed on them ( 7 redo orchidopexy and 33 first orchidopexy).

Gonadotrophin levels (luteinizing hormone, follicular-stimulating hormone) were tested on 20 patients and were compared with the levels of 20 children of similar age group with normal (descended) testes.

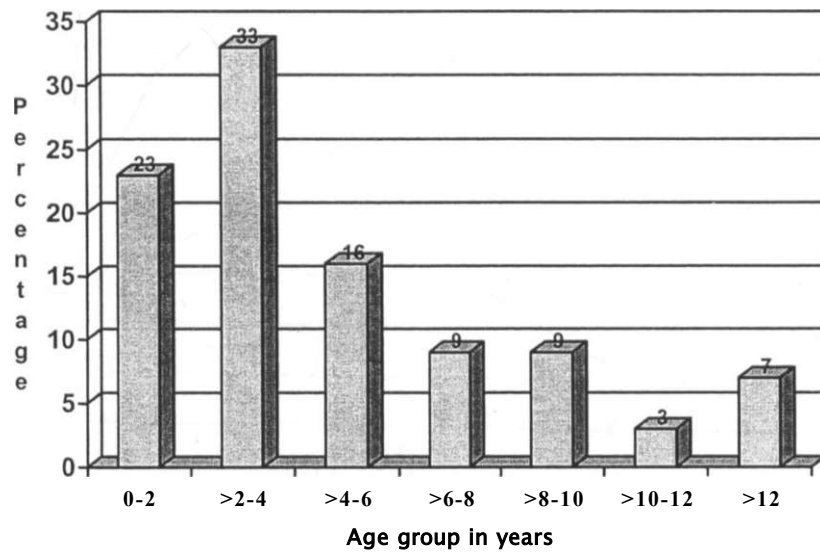
### 1. Age

The ages of the patients seen ranged from 1 month to 13 years with a mean age of 5 years (60 months).

**Table 1: Age distribution.**

Ages of the patients(Yrs)	Frequency	Percentage
0 - 2	13	23
2-4	19	33
4-6	9	16
6-8	5	9
8-10	5	9
10-12	2	3
12	4	7
Total	57	100

Figure 1: Age distribution



## Month of delivery

The month when the patient was delivered was noted. Most of the deliveries 10 (18%) occurred in the month of September, and non occurred in the month of January.

**Figure 2: Distribution of month of delivery of the patients with undescended testes**



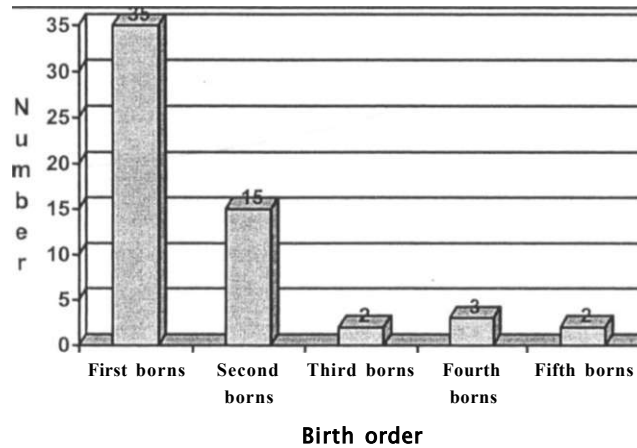


**Table 2: Birth order:**

The table below shows that birth order of the patients in the family. First born males accounted for 62% and second born males accounted for 36% of the patients with UDT. Both first and second born males accounted for 98%.

Birth order	Number of patients	Percentage
First borns	35	62
Second borns	15	36
Third borns	2	3.5
Fourth borns	3	5
Fifth borns	2	3.5
Total	57	100

**Figure 3: Birth order**



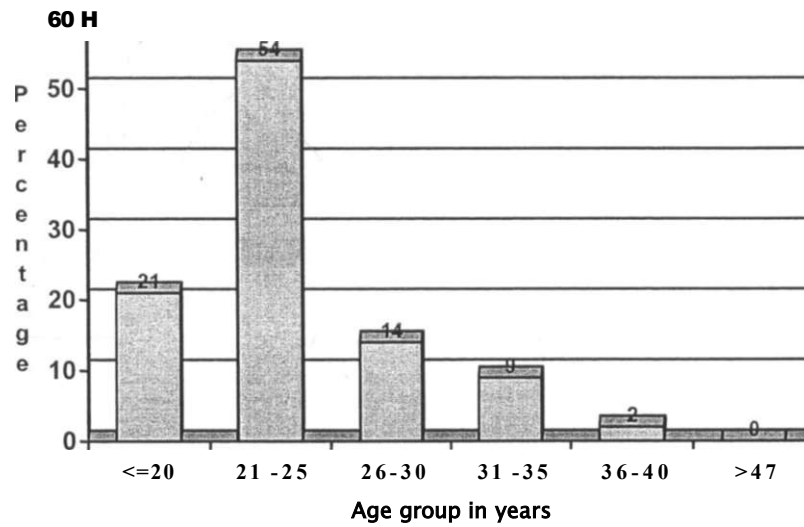
#### 4. Mothers' age at delivery of the patient

The table below shows the ages of the patients' mothers at delivery. The youngest mother at delivery was 17 years and the eldest was 39 years. Average age of the mothers at delivery of a patient with undescended testes was 24 years.

**Table 3: Age of the mother**

Age of mother at deliver (years)	Number of patient mothers	Percentage
<20	12	21
21 -25	31	54
26 -30	8	14
31 -35	5	9
36 -40	1	2
>41	0	0
Total	57	100

Figure 4: Distribution age of mother



**Mothers who smoked during pregnancy**

None of the mothers are smokers or admitted ever smoking cigarettes in their lifetime.

### **Patients with positive family history of undescended testes**

Patients who had one or more relatives with or who had undescended testes were 10 (7.5%) .Four (7%) of the patients each had a brother with or had undescended testes which has been corrected surgically.

Three (5.25%) of the patients each had a paternal grandfather with undescended testes. We could not establish whether the abnormality (undescended testes) was corrected or not and also whether they had unilateral or bilateral undescended testes, but what we know is that they reared children of their own i.e. they were fertile.

One patient had both paternal and maternal cousins with undescended testes. One patient had a paternal cousin and one patient had a paternal uncle with undescended testes.

**Table 4: Relationship to the patient of those relatives with or had undescended testes.**

Relative with undescended testes	Number of patients with the relationship	Percentage
Brother	4	7.0
Paternal grandfather	3	5.25
Paternal uncle	1	1.75
Maternal cousin	1	1.75
Both paternal and maternal cousin	1	1.75
Total	10	17.5

**The place where the abnormality was first noted.**

The abnormality was first noted in a health institution in 58% of the patients and 42% was noted by the family members

**Table 5: Where the abnormality was first noted.**

Place where the abnormality was first noted	Number of patients	Percentage
Health institution	33	58
Home by family members	24	42
Total	57	100

## 8. The person who first noted the abnormality at home

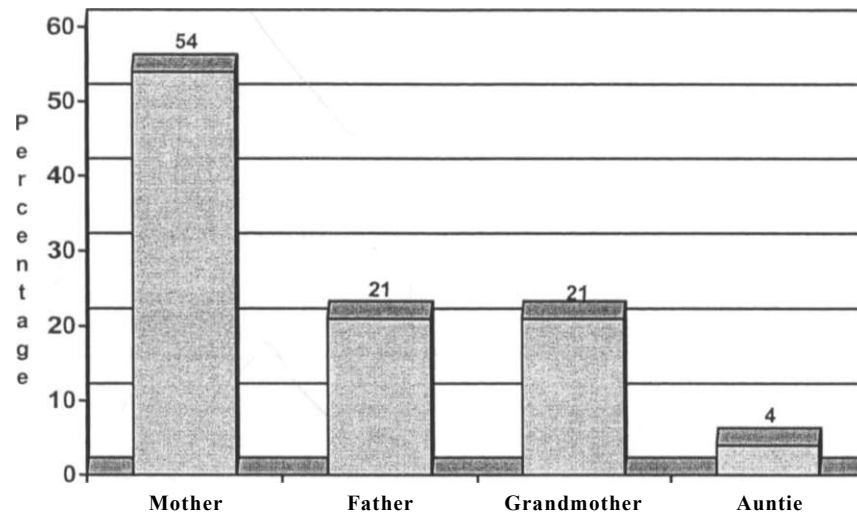
In the study the abnormality of UDT was noted by the family members in 24(42%) of the patients. Of the 24 patients, the mothers noted 54%, the fathers noted 21%, the grandmothers noted 21%, and the aunties noted 4%.

**Table 6: Person who noted the abnormality first at home**

The table below shows the family members who first noted the abnormality

Person who noted the abnormality first at home	Number of patients	Percentage
Mothers	13	54
Fathers	5	21
Grand mothers	5	21
Aunties	1	4
Total	24	100

**Figure 5: Person who noted the abnormality first at home**



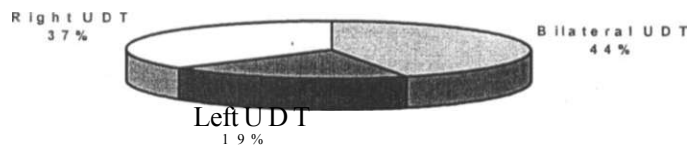
**Side of the undescended testes**

Out of the 57 patients, 21 patients (36.8%) had right undescended testes and 25 patients (43.9%) had bilateral undescended testes.

**Table 7: Side involved**

Side involved	Number of patients	Percent
Right undescended testes	21	36.8
Left undescended testes	11	19.3
Bilateral undescended testes	25	43.9
Total	57	100

Figure 6: Side involved





### Associated anomalies

The table below shows the associated anomalies of undescended testes, out of the 57 patients; 16 (28.1%) had other associated anomalies

**Table 8: Associated anomalies**

Associated anomalies	Number of patients	Percent
Hypospadias	5	8.7
Umbilical hernia	3	5.2
Prune belly syndrome	3	5.2
Congenital Talipes Equinovarus	1	1.8
Anorectal Malformation with recto-vesical fistula	1	1.8
Ventriculo-Septal-Defect	1	1.8
Pyloric stenosis	1	1.8
Achalasia cardia	1	1.8
Total	16	28.1%

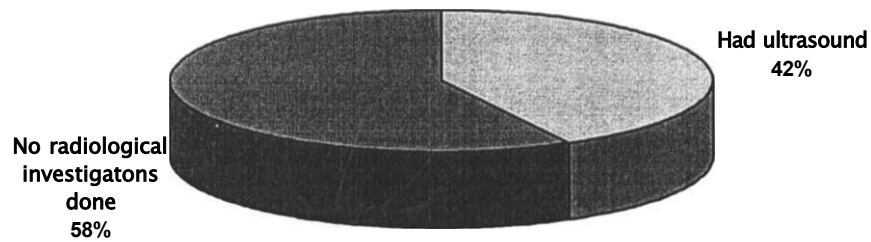
## INVESTIGATIONS

### 11. Radiological investigations done

The radiological investigation that was done to locate the position of the testicle was ultrasound, which was performed on 24 (42.1%) of the patients, whose testis/testes could not be palpated

None had Computerized tomography scan or Magnetic Resonance Imaging done to locate the position of the testicle.

**Figure 7: Proportion of patients who had ultra sound done on them.**



**Blood Investigation**

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**12. Gonadotrophin levels**

The table below shows the gonadotrophin levels of 20 children with undescended testes and other 20 children with normal (descended) testes.

**Table 9: Gonadotrophins of children with undescended testes (n = 20)**

Age	Luteinizing Hormone Levels mmol/l	Follicular-Stimulating Hormone levels mmol/l
1 month	1.56	1.11
1 (Yrs)	0.16	1.86
1 "	0.05	0.40
1.5 "	0.08	0.16
1.5"	0.11	0.95
2 "	0.01	1.50
2 "	0.34	0.05
2.5 "	0.12	0.05
3 "	0.14	0.40
3 "	0.14	0.09
3 "	0.35	0.91
4 "	0.24	0.60
4 "	0.05	0.01
5 "	0.09	0.45
5 "	0.09	0.77
5.5 "	0.80	3.01
6 "	0.02	0.01
7 "	0.07	0.09
8	0.01	1.25
13	1.08	2.12

**Table 10: Gonadotropin levels of children with normal (descended) tested**

**(n=20).**

Age	Luteinizing Hormone-Levels mmol/l	Follicular-Stimulating Hormone Levels mmol/l
9 months	0.36	0.21
1.5 (Yrs)	0.25	1.37
1.5 "	0.61	0.39
1.5 "	0.03	0.15
1 8/12	0.51	0.44
2 "	0.44	0.19
2 "	0.31	0.48
2.5 "	0.22	0.40
2.5 "	0.04	0.35
3 "	0.28	0.44
3 "	0.36	0.54
3.5 "	0.26	0.43
4 "	0.28	0.15
5 "	0.11	0.27
5.5 "	0.01	0.43
6 "	0.02	0.06
7 "	0.10	0.14
8	0.47	0.22
8	0.17	1.22
10	0.15	0.53

### Average gonadotropin levels at different age groups

Average gonadotropin levels of the children with undescended testes and the children with normal (descended) testes at different age groups of <2 years; 2 - < 5 years and > 5 years of age.

**Table 11 a: Average gonadotropin level at different age groups**

Age group	Patient character	Luteinizing Hormone Levels mmol/1	Follicular-Stimulating Hormone Levels mmol/1
< 2 years	A n = 7	0.33	0.86
	B n = 7	0.36	0.46
> 2 - < 5 years	A n = 6	0.17	0.34
	B n = 6	0.24	0.39
> 5 years	A n = 7	0.31	1.11
	B n = 7	0.15	0.41

Patient character

A = with undescended testes (uni/bilateral)

B = with normal (descended) testes

**Table lib****P Values**

Age group	Gonadotrophin	P Values
< 2 years	Luteinizing Hormone	0.90
	Follicular-Stimulating Hormone	0.21
> 2 - < 5 years	Luteinizing Hormone	0.22
	Follicular-Stimulating Hormone	0.77
> 5 years	Luteinizing Hormone	1.30
	Follicular-Stimulating Hormone	0.16

There is no Significant statistical difference of luteinizing hormone and follicular-stimulating hormone levels at different age groups of children with undescended testes and those children with normal (descended) testes.

Figure 8 (a): Luteinizing Hormone levels

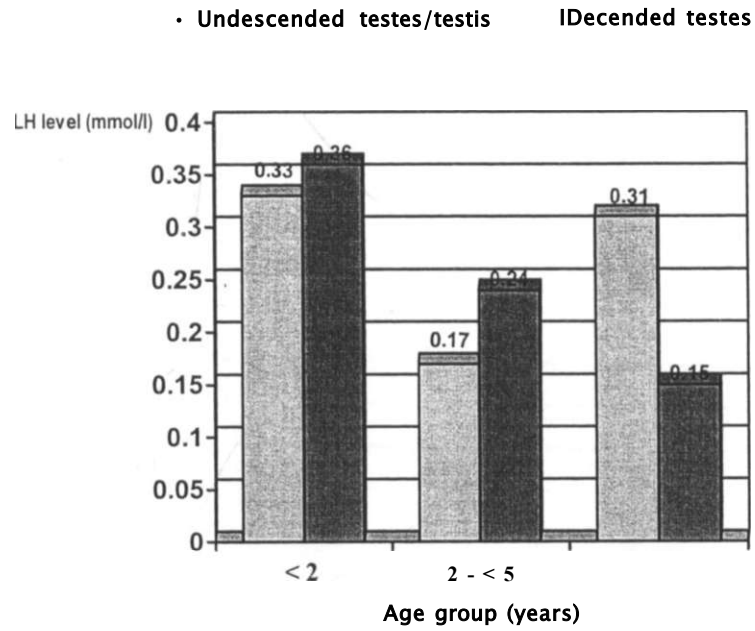
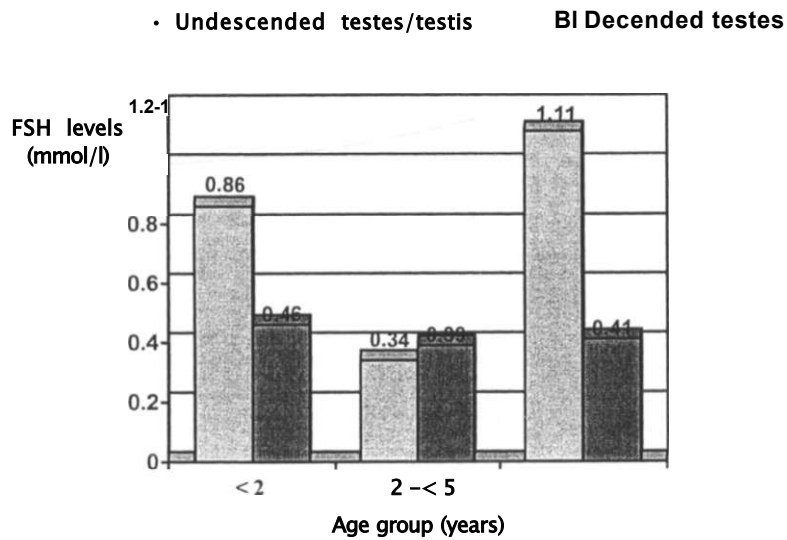
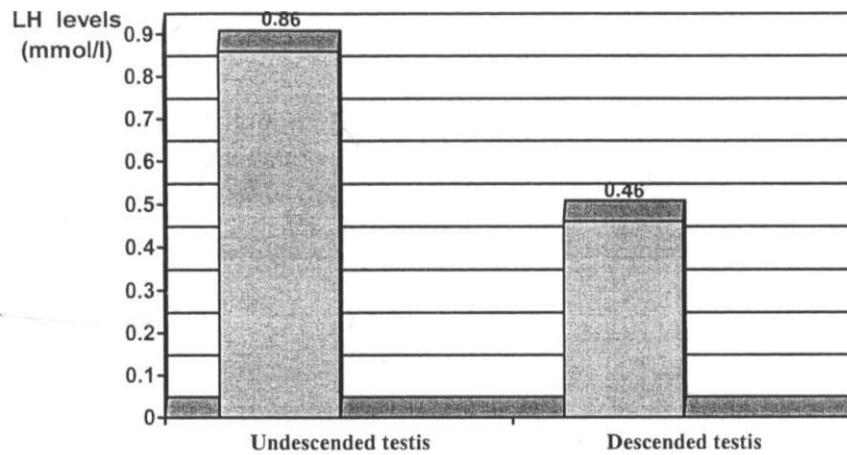


Figure 8 (b) Follicular-Stimulating Hormone levels



Average gonadotropin levels of these children with undescended testis and the ones with descended testes.

Fig. 9a: Luteinizing Hormone Levels

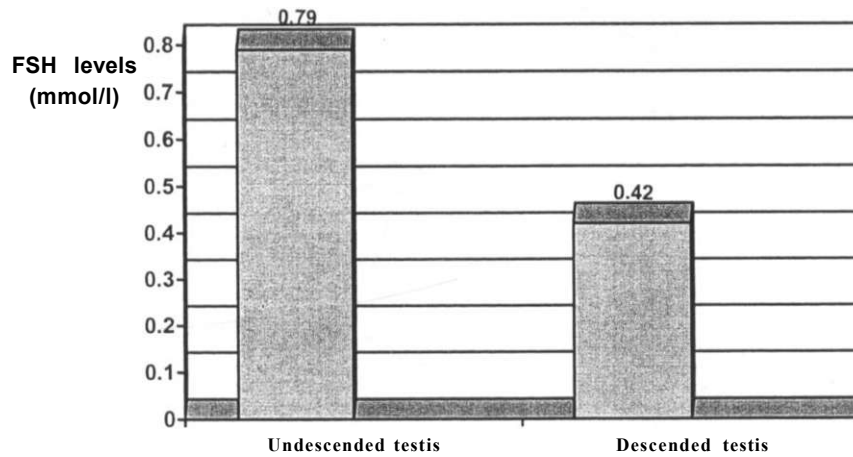


There is no significant statistical difference in the levels of follicular-stimulating hormone in children with undescended testes and those children normal = descended testes. P value = 0.79



Fig. 9b: Follicular-Stimulating Hormone Levels

There is no significant statistical difference in the levels of follicular-stimulating hormone in children with undescended testes and those children with normal descended testes. P value = 0.70



**13. Number of redo orchidopexies performed.**

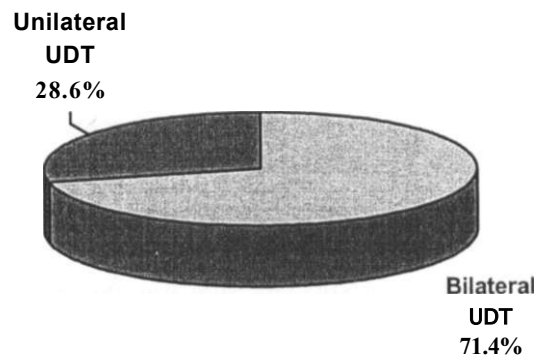
Within the period of this study, 40 patients had orchidopexy performed on them and out of these 33 (82.5%) were first operations and 7 (17.5%) were redo operations.

Of the 7 patients who had redo orchidopexy, 5 (71.5%) had bilateral undescended testes and 2(28.6%) had unilateral undescended testes.

**Table 12: Patients who had redo orchidopexy**

side of undescended testes	Number of patients	Percentage
Bilateral undescended testes	5	71.4
Unilateral undescended testes	2	28.6
Total	7	100

**Fig. 10. Side involved in patients who had redo-orchidopexy**



#### **Complications of undescended testes noted before or at surgery**

Of the 57 patients 7(12.3%) had inguinal hernia and 3 of these had presented initially with irreducible inguinal hernia and undescended testes. Herniotomy was performed as an emergency and schedule for elective orchidopexy. Of the 40 patients who had orchidopexy 8 (20%) were noted to have atrophic testes.

**Table 13: Complications of undescended testes**

Complication	Number of patients	Percentage
Inguinal hernia	7(n=57)	12.3%
Atrophic testis/testes	8 (n=40)	20%

**Table 14: Patients with atrophic testes/testis categorised into ages < 2 years, 2-5 years and > 5 years.**

None of the patients less than 2 years of age was found to have atrophic testes at operation. Most of the patients with atrophic testes were ages 5years and above.

Age group	Number of patients (n=40)	Percent
< 2 years	0	0
2 - < 5year	2	5
> 5 years	6	15
Total	8	20

#### **15. Complications that occurred during orchidopexy**

In this study out of the 40 patients who had orchidopexy only 2 (5%) of the patients had a complication. One had avulsed vas deferens and the other had testicular artery injured. The one whose testicular artery was injured, the gubernaculum was left intact so as to provide blood supply to the testis.

**Table 1 5: Immediate surgical complications of orchidopexy (n = 40)**

Two patients had intraoperative surgical complications

Complication	Number of patients	Percentage
Avulsed vas deferens	1	2.5
Injured testicular artery	1	2.5
Total	2	5

## DISCUSSION

The main concern of the parents to their children with undescended testes is infertility. The parents do want to know the chances of their children becoming infertile; whether the testicle/testicles will descent on its/their own without operation; the right age of orchidopexy; what are the causes of testicular maldescent and will the other child to be born have undescended testicle? These are among the common questions parents do ask concerning their children with UDT.

## **Age**

In this study the ages at presentation of the patients ranged from 1 month to 13 years. The mean age was 5 years and median of ages 2-4 years (33%). Those less than 2 years of age were 23%.

In his study of 1985 at Kenyatta National Hospital Nyambuyi <sup>(36)</sup> found a mean age of 7 years and a median age of 4-6 years. Paul A. Kisanga <sup>(37)</sup> in his study of 1996 at Kenyatta National Hospital found a mean age of 6.1 years and median ages of 4-5 years. From this study; the mean age at presentation has gone down from 6.1 years in 1996 to 5 years; also the peak age group at presentation has gone down from 4-5 years to 2-4 years.

The general population has been enlightened to the importance of seeking early medical consultation of their children with Undescended Testes and also the medical facilities are easily available to the people than 10 years ago.

## **Associated factors**

### **Month of delivery**

In this study, most patients with undescended testes were delivered in the month of September, 10 (18%) followed by the month of June 7 (12%). Though there was no patient whose delivery was in the month of January, the other months had more less the same number of patients. There is no study which has been done before to correlate the month of delivery to the occurrence of undescended testes.

### **Birth order**

First-born males were 62% and second borns were 36% and both (1<sup>st</sup> and 2<sup>nd</sup> borns) account for 98% of the patients with undescended testes.

Kisanga <sup>(37)</sup> in his study found that both first borns and second borns accounted for 53% of the patients with undescended testes. From this study we can say that being a first or second born is a high risk of developing undescended testes.

This is reflected in the ages of the mothers at delivery of children with undescended testes. Fifty four percent of the mothers at delivery were between the ages of 21-25 years i.e. most women deliver their first borns at these age group (21-25 years). Those who were less than 21 years of age at delivery were 21% and those less than 25 years of age at delivery were 75%.

### **Smoking by the mother**

None of the mothers of the patients with undescended testes gave any history of smoking in their lifetime. There is no study, which has been done to show the effects of smoking during pregnancy to the occurrence of undescended testes.

From this study we cannot conclude that smoking during pregnancy predisposes the unborn child to the development of undescended testes.

### **Familial aspects of cryptorchidism**

This study showed that 17.5% of the patients had one or more family members with or who had undescended testes. Brothers were affected in 7% of the patients and 3% of the patients had paternal grandfathers affected. One patient had both maternal and paternal cousins affected, one had paternal cousin affected. None of the patients had a father with a history of undescended testes.

The findings of this study are in line with studies done earlier. Perret and O'Rourke <sup>(28)</sup> presented three brothers with an undescended testicle who had a history of the same disorder going back three generations. Rezvani et al <sup>(39)</sup> presented two half brothers with an undescended testicle, the common parent being the mother. A maternal cousin also had cryptorchidism. Waaler <sup>(40)</sup> examined 168 patients with cryptorchidism, including six pairs of brothers. Jones and Young <sup>(41)</sup>, evaluated the family history of 51 boys with cryptorchidism and found that the fathers were likely to be affected in 4% of the boys and male siblings in 10%. This study supports multifactorial aetiological based on a higher incidence in relatives than would be expected from chance alone.

### **The persons/place where the abnormality was noted**

Of the 57 patients, 33 (58%) of the patients had the abnormality (of undescended testes) noted at a health facility and 24 (42%) of the patients had the abnormality noted by a family member.

Of those noted by the family member; 54% of the patients had the abnormality first noticed by the mothers, 21% noted by the fathers and 21% noticed by the grandmothers and 4% were noted by the aunts.

Most of the children more than 95% were delivered at a health institution and all attended child health clinic for immunization as per Kenya Expanded Programme



Of Immunization schedule. One would expect then that more than 95% if not 100% of undescended testes would be detected at a health facility, either during delivery or at the child health clinic and be followed up since most of the undescended testes will descend by 1 year of age, those that would not have descended by 2 years of age to have orchidopexy performed on them.

### **Associated anomalies of undescended testes**

In this study twenty eight percent of the patients had at least one or more associated anomalies of undescended testes. Hypospadias was seen in 8.7% of the patients. This figure reflects the figure of 9% in a paper written by J. Leissner et al <sup>(42)</sup> which they concluded that the association of hypospadias with cryptorchidism supports the hypothesis that a form of endocrinopathy is involved in the aetiology of cryptorchidism.

Umbilical hernia was seen in 3% of the patients and Prunebelly syndrome was also seen in 3% of the patients with undescended testes. This findings supports the theories that inability to generate intraabdominal pressure contributes to the development of undescended testes.

The presence of cryptorchidism in association with abdominal wall defects has been well known. Kaplan et al <sup>(43)</sup> noted the greatly increased incidence of cryptorchidism with gastroschisis; omphalocele or umbilical hernia. The presence of bilateral undescended testis in the prunebelly or triad syndrome has been well known. Other associated anomalies found in this study were Congenital Talipes Equinovarus, Anorectal Malformation, Ventriculo-Septal defect; infantile hypertrophic pyloric stenosis and achalasia cardia, each accounted for 1.8%. One child had prunebelly syndrome, hydroureters; and Congenital Talipes Equinovarus on top of the undescended testes. These findings support the fact that many patients with cryptorchidism have associated chromosomal abnormalities, which account for 1% of all congenital anomalies<sup>(44)</sup>.

## **Gonadotropin levels**

Although hormonal involvement in testicular descent appears clear cut there are discrepancies in the literature as to whether patients with cryptorchidism have discernible abnormalities in their hypothalamic - pituitary - testicular axis.

In this study the gonadotrophin (luteinizing hormone, follicular-stimulating hormone) levels in children with undescended testes were not statistically different with those of normal children. P value of LH and FSH were 0.790 and 0.070 respectively).

Walsh et al <sup>(45)</sup> found no difference in basal plasma testosterone levels between cryptorchid and normal children: Cacciari et al <sup>(46)</sup> used Gonadotrophin releasing hormone to stimulate the hypothalamic - pituitary axis and they found no difference in pituitary luteinizing hormone and follicular-stimulating hormone secretion between the cryptorchid and normal children. This was confirmed by Sizonenko et al <sup>(47)</sup> and Van Vliet et al<sup>(34)</sup>.

The results of this study do not support the theory that there is a deficit in the hypothalmo pituitary axis in patients with undescended testes, though a higher sample size is needed for definitive conclusion.

We can conclude from this study that in the treatment of undescended testes, hormonal therapy (use of GnRH, HCG) may not play a role. This is supported by a more recent controlled study comparing human chorionic gonatrophin and gonodotrophic releasing hormone (GnRH) in the treatment of cryptorchidism, which was found that hormone therapy had no significant effect on true undescended testes, but 100 percent effect on retractile testes <sup>(48)</sup>.

## **Complications of undescended testes**

The complications of undescended testes are infertility, testicular atrophy, trauma, torsion, hernia and malignancy.

In this study 8(20%) patients were found to have atrophic testicles during orchidopexy; 6(15%) patients were ages 5 years and above and 2 (5%) patients were ages between 2 - 5 years, none was less than 2 years of age.

Nyambuyi <sup>(36)</sup> in his study found the incidence of atrophic testis to be 12.16% and Kisanga <sup>(37)</sup> found it to be 5.5%.

The clinically demonstrated hernia was found in 12.3% of the patients, 3 of the children had presented earlier with irreducible inguinal hernia and had herniotomy performed at the time, and booked for orchidopexy as an elective operation. This stresses the importance of early orchidopexy in children with hernia to avoid strangulation which can occur (Gonzales)<sup>(49)</sup>.

Although the true incidence of hernia is unclear, hernia sac can be found in > 90% of the patients with cryptorchidism <sup>(50)</sup>.

Kisanga <sup>(?)</sup> in his study found out that hernia was the presenting complaints in 10.3% of the patients. He found that 11.6% of the patients had patent processus vaginalis and 61.2% had hernia sac.

Cendron et al <sup>1511</sup> suggested that fertility is gauged by paternity rather than sperm count. Then one would have to follow these patients till when they marry hopefully, and find out their fertility, assuming no extramarital affair would occur on the side of the wife.

### **Intra-operative complications**

Of the 40 patients who had orchidopexy 1 (2.5%) had avulsed vas deference and 1 (2.5%) had injured testicular artery.

In Kisanga's <sup>(42)</sup> study, 1 (0.3%) had vascular injury and 1 (0.3%) had injury to the vas deference, 1 (0.3%) had urinary bladder injury and 1 (0.3%) had one ureter injured.

### **Redo operations**

All the operations performed in this study were the subdartors pouch technique. Of the 40 orchidopexies performed; (7) 17.5% were reoperations, five with bilateral undescended testes and two with unilateral undescended testes. The operations first done to all the seven patients were the subdartos pouch technique of fixation. From this study, it seemed that the chances of failure of orchidopexy was higher in patients with bilateral undescended testes. The reason of failure of orchidopexy could have been the surgical technique of the surgeons in the subdartos pouch fixation; but the main factor is short vascular pedicle and rarely due to short vas deferens. It seems that short vascular pedicle or short vas is common in bilateral undescended testes, hence higher failure rate of orchidopexy in bilateral undescended testes.

In Nyambuyi's <sup>(36)</sup> study he found that 67 (25%) patients had inadequate length of spermatic vessels and Kisanga <sup>(37)</sup> in his study found recurrence rate of 3.8% in the subdartos technique.

## **CONCLUSIONS**

The mean age at presentation of children with undescended testes is still higher than the recommended age of orchidopexy of 2 years.

Though there is slight improvement from a mean age of 6.1 years as per Kisanga's <sup>(43)</sup> study of 1996 to the current one of 5 years a lot of health education to the general public as to the importance of early orchidopexy needs to be done. Our health workers in the primary institutions should refer patients early as soon as the diagnosis of undescended testes is made, to the secondary or tertiary institutions where definitive management can be done.

During delivery, or when children present to the clinic or any health institution few health workers including doctors examine the children's scrotum to document the presence or absence of the testicles. This is shown by the low percentage of 58%, the number of patients whose abnormality of undescended testes were detected by health workers. It is expected that the abnormality of undescended testes should have been detected by health workers in > 95% of the patients, given that > 95% of the children were delivered in a health institution, and 100% attended the child health clinic section for immunizations. Ref. questionnaire (3v,vi).

The family members first noticed the abnormality of undescended testes in 42% of the patients. The mothers noticed most of them. This stresses the importance the mothers play in helping a doctor make a diagnosis in a child and they should be listened to.

First and second born males have a high risk of developing undescended testes. This emphasised the point that the causes of undescended testes are multifactorial. The reasons why first borns and second borns are at a high risk needs further study.

The gonadotrophin levels of children with cryptorchidism and those with normal (descended) testes were the same. More sample size is needed for definitive conclusion to be reached. The study support those who say that hormonal therapy does not play a

role in the management of undescended testes and surgical management is the only way to bring the testicles to the scrotum.

The incidence of testicular atrophy was significant in patients at or above 5 years of age, none had testicular atrophy at ages below 2 years. This finding stresses the importance of early orchidopexy at or below the age of 2 years.

The redo orchidopexy of 17.1% was high, especially to those who had bilateral undescended testes. This shows that patients with bilateral undescended testes have mechanical factors e.g short spermatic vessels or vas deferens as the main factor causing the testes not to descend and 2 stage orchidopexy should be performed on patients with bilateral undescended testes or those with intraoperative finding of short spermatic vessels.

## RECOMMENDATIONS

1. All male children presenting to health institutions for either immunization or for medical check up should have their scrotums checked, and documentation made whether the testicles are present or not. This will enhance early diagnosis of undescended testes to be made and hence early correction
2. Health workers in the peripheral institutional should be encouraged to refer patients as soon as the diagnosis of undescended testes is made to the tertiary institutions, or where orchidopexy or further evaluation can be done.
3. Paediatric surgeons should aim at 2 years or less as the ideal age of orchidopexy and those children who are first or second borns with undescended testes or those with anterior abdominal wall defects e.g. omphalocele, whose spontaneous descent is unlikely should have orchidopexy even earlier.

Those patients with undescended testes and inguinal hernia should have hernia repair and orchidopexy as soon as the diagnosis is made to prevent strangulation of hernia which can occur.

4. A case control study needs to be done on the effects of mothers smoking during pregnancy to the occurrence of undescended testes.
5. Androgen receptors need to be evaluated if they are defective or low in number in patients with undescended testes, so as the whole picture of hormonal influence on testicular descent can be known.
6. Orchidopexy in patients with bilateral undescended testes should be performed by a senior surgeon or by a paediatric surgeon, and not a registrar, given the higher incidence of recurrence and also if a complication was to occur e.g. avulsion of the vas deferens or injury to the testicular vessels even unilaterally, it would greatly compromise the fertility of the patient.

## APPENDIX t

### DATA SHEET

Serial Number

#### A: IDENTIFICATION

1. Name:
2. IP NO:
3. Birth History:

(i) Date of birth: ..... Month ..... Year

	Yes	No
(ii) Full term	•	•
Preterm	•	•
(iii) Delivery		
Caesarian Section	•	•
Spontaneous vaginal delivery	•	•
(iv) Cephalic presentation	•	•
Breech	•	•
(v) Place of delivery		
Home	•	•
Health facility	•	•
(vi) Do/Did the child attend child health clinic for Immunization	•	•



Family social history:

(i) Number of:

Brothers

Sisters

(ii) Which birth number is he?.

(iii) Any relative with undescended testes: (Yes/No)

If Yes name the relationship

(iv) Age of the mother today..... at delivery

**Yes**

**No**

(v) Did the mother smoke during

Pregnancy?

EH

I-I

(Yes/No) If Yes, how many: -

Cigarettes per day

Duration she had smoked by the time of pregnancy

**HISTORY OF PRESENTING COMPLAIN**

(i) Abnormality was noticed by: -

(1) Father

•

•

(2) Mother

•

•

(3) House help

•

•

(4) Others (specify)

(ii) How old was the child when abnormality was noted?.





**Complications noted: -**

- That occurred before surgery;

- |                     |     |   |    |   |
|---------------------|-----|---|----|---|
| a) Torsion          | Yes | • | No | • |
| b) Trauma           | Yes | • | No | • |
| c) Hernia           | Yes | • | No | • |
| d) Others (specify) |     |   |    |   |

- That occurred due to surgery:

- |              |     |   |     |   |
|--------------|-----|---|-----|---|
| a) Immediate | Rt: | • | Lt: | • |
| b) Late      | Rt: | • | Lt: | • |

# CONSENT FORM

I \_\_\_\_\_ the parent/guardian of.....have understood the purpose of the investigation to be carried on my child. I accept blood to be withdrawn from my child and the hormonal evaluation (Follicular-Stimulating Hormone, Luteinizing Hormone) to be carried out.

Signed:

Parent/guardian ..... Date

Doctor ..... Date

## APPENDIX 11

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