RETAINED PLACENTA - MANUAL REMOVAL:

Name:	J.N.	LMP:	20.9.1987.
Age:	30 years	EDD:	27.6.1988.
Unit No:	713441	Admission:	2.6.1988.
Parity:	3+0	Discharge:	4.6.1988.

PRESENTING HISTORY:

Mrs. J.N. was admitted to labour ward on 2.6.1988 at about
5.40 p.m. through casualty with a history of labour pains and
draining of clear liquor since the previous day. There was
no history of vaginal bleeding. She had not attended any
antenatal clinic during the index pregnancy.

PAST MEDICAL HISTORY:

Her past medical and surgical history was not remarkable.

OBSTETRIC AND GYNAECOLOGICAL HISTORY:

Her menarche was at 14 years and subsequently bled for 4 days
every month. She did not use contraception. She was para 3+0. All
were spontaneous vertex deliveries. The last delivery was in
1986. She had two girls and one boy. All were alive and well.
She had post-partum haemorrhage after manual removal of the
placenta during the last delivery, but later recovered. Her
last menstrual period was on 20.9.87, giving her expected
date of delivery to be 27.6.88. Gestation on admission was
therefore 36 weeks and 4 days.

FAMILY AND SOCIAL HISTORY:

She was married and lived in Nairobi with her family. Her
husband was a casual labourer in one of the city's factories.

PHYSICAL EXAMINATION :

Her general conditon was fair. There was no pallor, jaundice,
oedema, dehydration or palpable peripheral lymph nodes. Clinically
she was afebrile. Her blood pressure was 120/80 mmHg; pulse rate
was 20 per minute, and the body temperature 36.7°c.

The cardio-pulmonary and the central nervous systems were essentially normal.

ABDOMINAL EXAMINATION:

The uterine size was 36 weeks. Foetal lie was longitudinal and presentation was cephalic. The foetal head was 5/5 above the pelvic brim (Notelwicz's method), and the foetal heart beat was regular at 138 per minute. She was getting two uterine contractions every 10 minutes lasting 20 - 40 seconds.

Both the liver and the spleen were not enlarged.

PELVIC EXAMINATION:

The external genitalia was normal. On digital exam, the cervix was soft, central and 80% effaced. The os was open about 6 cm. Clear liquor was draining. No cord was felt. The presentation was vertex, and the position was left occipito-anterior. No caput succedaneum or moulding were noted. The pelvis was roomy.

DIAGNOSIS:

A diagnosis of active phase of labour in a multigravida was made.

PROGRESS IN LABOUR:

Partogram was started and the progress of labour was monitored. At 6.50 p.m. she progressed well in labour and had a spontaneous vertex delivery to a male infant weighing 2350 gram. Apgar score was 8 and 10 at 1 and 5 minutes respectively. Intramuscular ergometrine 0.5 mg was given after delivery of the baby. This was repeated after 20 minutes. By 7.30 p.m. the placenta had not yet separated and a diagnosis of retained placenta was made. The vital observations of the patient were, however, stable and there was no active vaginal bleeding. Delivery by cord traction was attempted but was unsuccessful. Blood for grouping and cross-match was taken, and two units of blood were asked for.

Syntocinon 10 units in 500 ml of 5% dextrose infusion was set up,

and the patient prepared for manual removal of the placenta in theatre after obtaining the necessary informed consent.

MANUAL REMOVAL:

The patient was placed in lithotomy position on the operating table, under general anaesthesia. Vulvo-vaginal toilet was done with cetavlon solution, and draped with sterile surgical towels. About 200 ml of clear urine was obtained on catheterisation. Controlled cord traction was again attempted but failed. The left hand then held the umbilical cord taut while the right hand was inserted into the vagina and followed the cord up to the placenta through the dilated cervix into the uterine cavity. At this juncture the left hand steadied the uterus abdominally. On finding the placenta, the ulnar border of the extended fingers abducting and adducting, the placenta was gently and easily detached. This placenta was situated fundo-posteriorly. When the placenta was completely separated, the whole placenta was held by the right hand and gently delivered vaginally taking care to deliver the membranes complete. The procedure completed, the left hand rubbed up a contraction. After the blood was washed off the right hand glove, and the perineum was again sponged with cetavlon solution, the right hand was inserted once more. The whole interior of the uterus was palpated to ascertain that the uterus was completely empty. Care was taken to remove all the membranes and adherent placental fragments. The rubber gloves were so slippery that shreds of membranes could not be grasped. The fingers were covered with gauze to aid in this process. No uterine curretage was performed. There was only minimal bleeding. I.V. Ergometrine 0.5 mg was given with good effect.

POST OPERATIVE CARE:

Routine post-operative management was instituted. She was given 3 litres of I.V. fluids in 24 hours, parenteral pethidine 100 mg 8 hourly for 24 hours, and I.V. ampicillin 500 mg 6 hourly for 5 days. No blood transfussion was given as there was no post-partum haemorrhage. She was allowed to breast-feed her baby

as soon as she was able to do so; and was discharged home in good general conditon after 24 hours observation as there were no puerperial complications.

She was adequately counselled about her future deliveries but declined any contraception. She did not come back for post-natal follow-up.

DISCUSSION:

Delivery of the crying child is the moment of great happiness
to the mother, but this happiness may not last if the next
stage of labour turns abnormal. Many complications are asso-
ciated with the third stage of labour and they can be
davastating and dangerous. Retained placenta is a common
cause of third stage haemorrhage and is responsible for a big
toll of maternal deaths. The purpose herein is to present a
multiparous patient who had repeat retained placenta without
any significant post-partum haemorrhage. She had manual
removal of the placenta done on her under general anaesthesia
follwed by a smooth and un-eventful puerperium.

The incidence of retained placenta at the Kenyatta National
Hospital is unknown. Chahabra and Sandeep (1) found an incidence
of 0.19% of all deliveries elsewhere - 4.33% of these had adherent
type of placenta.

The classical signs that the placenta has been separated and
expelled from the firmly contracted uterine fundus into the lower
segment and vagina are that the uterus rises to the umbilicus and
to the surface; become smaller, as a rule firmer and globular (or
round) rather than wide and flat, and more mobile; the umbilical
cord lengthens, and a gush of vaginal bleeding occurs (2). The
patient presented did not show any of these signs of separation.
Separation of the placenta which takes place through the decidua
spongiosa usually begins as the baby is being delivered and the
uterus contracts. The process is normally complete within a few
minutes of the baby's birth (2,3). Uterine contractions are
responsible for expelling the placenta from the upper uterine
segment into the lower segment and the vagina. Delivery from the
vagina is completed by voluntary bearing-down efforts of the mother
or, as is more often the case, is expressed by the attendant.

The time taken for the placenta to be separated from its uterine
attachment and expelled from the uterus depends on the force of the
uterine contractions and on the firmness of the attachment to the
uterus. The retention of the placenta in the uterus for more than
30 minutes is regarded as abnormal (2,3). The patient under
discussion had spontaneous vertex delivery at 6.50 p.m. and at

7.30 p.m., she was diagnosed as having retained her placenta - a period of 40 minutes. The uterus was not massaged or manipulated. Controlled cord traction was attempted but failed. The uterus was well retracted and since there was only minimal vaginal bleeding, uterine atony was not suspected. Par abdomen there was no indication of uterine abnormality like a bicornuate uterus. Post partum haemorrhage was, however, vigilantly watched for as she had had excessive bleeding after the preceding delivery. Fortunately this did not occur.

Since there was only minimal vaginal bleeding, and the vital signs of the patient were stable, there was no necessity for undue haste, and the patient was prepared for manual removal in the theatre within a reasonalble time span. The patient was not anaemic and did not have any chronic debilitating disease. Her labour was not prolonged and therefore she was not dehydrated. She also did not have pregnancy induced hypertension. However, an intravenous infusion was set up, even though the patient was not shocked.

The recognition of the retained placenta because it was still completely attached was not difficult in the patient presented. The uterus remained retracted at 22 weeks gestation, and it was wide and flat instead of being globula, and the expression of the placenta was not possible. There was only minimal bleeding - both before and after manual removal. She therefore did not need any blood transfusion.

Failure of placental separation may be mechanical or a result of abnormal penetration of the trophoblast into the uterine wall (abnormally adherent placenta) (2,3,4). With a mechanical failure the uterine muscle at the placental site may be relaxed and boggy even though that of the rest of the upper segment is fairly contracted. Because of failure of the muscles at the placental site to contract, the usual mechanism for placental separation does not come into play. It was suspected that in the case presented, the failure was mechanical. With abnormally adherent placenta all or part of the decidua basalis is absent, and the chorionic tissue grows directly into the muscle, thereby eliminating the normal cleavage plane. The term placenta accreta indicates a relatively superficial penetration of the muscle. Deeper penetration is called placenta increta, and placenta percreta indi-

cates that the trophoblast has grown to or completely through the
serosa (4).

The uterine surface after manual detachment of the placenta is
always rugged, it can never be made smooth, even by scraping
- and this is unwise, for there is a distinct danger that by
scraping the placental site too energetically injury may be
done to the uterine wall (3). Cases of weakening of the wall
and rupture in a subsequent labour have followed such a
procedure besides, there is the immediate danger of pushing
the fingers through the wall of the uterus.

Manual removal of the placenta was performed while this patient's
condition was good and stable, and was followed by prophylactic
antibiotic cover. As expected, favourable results was obtained
and she went home with her baby 24 hours after the operation.

The patient was already aware of her recurrent nature of
retained placenta, and would be very willing to deliver in the
hospital always. It must also be remembered that manual
exploration of the uterus after parturition may lead to the
later development of uterine synechia (5), and on occasions
result in rupture of the uterus in a subsequent delivery.

Inspite of so many developments in the field of obstetrics,
retained placenta continues to be a major problem. Mortality
reported by Mathur is 5.76% with 48% having shock, 1.3% by
Attal et al with more than 55% deliveries in the hospital.
The problem appears to be basic state of the patients.
Anaemic patients with added blood loss succumb to the situation.
The mismanagement of third stage and faulty attempts at trial of
removal of placenta add to it. The need of the day appears to
have properly trained mid-wives; vigilance about third stage
problems specially in those who are prone to get availability of
blood and skillful handling of the situation. Place of hysterec-
tomy in the management should not be forgotten.

REFERENCES:

1. Chhabra S. and Sandeep J. Retained Placenta - A Review.
    India Med. J. 83: 37, 1989.

2. Willson J.R., Carrington E.R., Ledger W.J. Obstetrics and
    Gynaecology. 7th Edition. Toronto. 1983.

3. Myerscough P.R. Munro Kerr's Operative Obstetrics.
    10th Edition. Bailliere Tindall. London. 1982.

4. Read J.A., Cotton D.M. and Miller F.C. Placenta accreta:
    Changing clinical aspects and outcome. Obstet. Gynecol.
    56:31, 1980.

5. Lancet M. and Kessler I. A review of Asherman's
    Syndrome, and Results of Modern Treatment. Int. J.
    Fertil. 33: 14, 1988.

6. Mathur and Attal. Quoted by 1.

A PROSPECTIVE STUDY TO ASSESS THE VALUE OF HAEMOGLOBIN CONCENTRATION IN MONITORING PATIENTS WITH PREGNANCY INDUCED HYPERTENSION IN THE THIRD TRIMESTER AT THE KENYATTA NATIONAL HOSPITAL.

SUMMARY:

In Kenya, pregnancy hypertension complicates about 9% of all pregnancies, and constitutes a significant cause of maternal and perinatal morbidity and mortality (5).

Pregnancy induced hypertension (PIH) has been assessed by a large number of clinical and laboratory tests, none of which are completely satisfactory. Even when combined the tests often represent an unsatisfactory basis for assessing the effectiveness of the placental function.

Commonly, a high maternal haemoglobin (Hb) level in late pregnancy is regarded as a sign of favourable outcome of pregnancy. Hitherto, Hb concentration has been measured during pregnancy mainly to detect anaemia, but more recently, interest has been focused on Hb-concentration as an indicator of reduced plasma volume expansion or haemoconcentration; and the relationship of plasma viscosity to systemic arterial hypertension. Increased levels of haemoglobin and plasma viscosity have been associated with complications of hypertension such as stroke, intra-uterine growth retardation or sudden fetal death in-utero by some authors (4,23,30,31,32,33). Studies of haemoglobin levels in PIH should, therefore be worthwhile since maternal Hb levels show a close inverse correlation to maternal plasma volume (30). Therefore in this study an attempt was made to draw attention to the usefulness of the Hb-concentration in monitoring P.I.H. and its relation to the perinatal outcome.

The last maternal haemoglobin (Hb) concentration before delivery was related to the perinatal outcome in 96 women suffering from PIH and to 108 normal pregnant women who acted as the controls. Abnormally high Hb concentrations were found in those patients with placental pathology. 22(22.9%) of the patients with PIH had placental infarcts and their Hb-concentration ranged from 12.5 to 16 g/dl. An inverse correlation was found between the birth weight of the newborn and the last maternal Hb-concentration. Significantlly higher Hb levels were found in pregnancies complicated by low-birth weights, acute fetal distress, sudden reduced fetal movements or un-explained fetal death in-utero as compared with those pregnan-

cies with good outcomes. It is concluded that high Hb levels in pregnancies complicated by PIH may be a sign of impending foetal morbidity and mortality. High viscosity of the mother's blood may impede the uteroplacental circulation causing placental infarction, growth retardation and ultimately fetal death.

This test has the advantage of being rapid, simple and inexpensive compared to the other sophisticated placental function tests. It can thus be appropriate as a monitoring procedure in many hospitals handling prenatal mothers with P.I.H. in developing countries.

INTRODUCTION AND LITERATURE REVIEW:

Hypertensive disorders complicate about 10 per cent of all pregnancies and constitute some of the most significant causes of maternal and perinatal morbidity and mortality. Their effects cover a wide spectrum of pathologic changes in multiple body systems, and they remain an extremely difficult clinical problem of all concerned with the health care of the pregnant patient (1,2,3,4,5). Mati found hypertension in 9% of pregnant women at Kenyatta National Hospital (5).

Hypertension may be premonitory sign of diverse vascular disorders with varied causes that may complicate or be complicated by pregnancy (1,3). The hypertension may be of short or long duration, exist prior to pregnancy, or present for the first time during pregnancy, labour or the puerperium. It may be labile or stable, benign or malignant, primary or secondary (3). Pregnancy-induced hypertension (hypertension in pregnancy in association with the excretion of more than 300 mg of urinary protein per 24 hours after 20 weeks gestation) is associated with a higher rate of intrauterine growth retardation, perinatal distress and perinatal death than is stable chronic hypertension during pregnancy (3,4). Aggarwal et al (1982) found hypertensive disease of pregnancy to be responsible for 4.8% of all perinatal deaths at KNH. 80.7% of these deaths were due to pre-eclamptic toxaemaia (PET), 3.5% were attributed to essential hypertension and 15.8% to eclampsia (6).

Hypertension in pregnancy is a rise in systolic Blood Pressure (BP) of at least 30 mmHg; or a rise in diastolic BP of at least 15 mmHg over the previously known BP, or a BP of at least 140/90

mmHg manifests on at least two occasions 6 or more hours apart
(3,8,9,10). Hypertension that is unique to pregnancy is best
termed pregnancy-induced hypertension (PIH) (8,9). P.I.H
is synonymous with pre-eclampsia-eclampsia syndrome. Eclampsia
being the extension of the pre-eclamptic process (8,9). P.I.H.
is the development of hypertension with proteinuria, oedema, or
both, induced by pregnancy after the 20th week of gestation.
Superimposed P.I.H. is defined as the development of pre-eclampsia
or eclampsia in a woman with chronic hypertensive, vascular or
renal disease. Chronic hypertensive disease is defined as the
presence of persistent hypertension, of whatever cause, before the
20th week of gestation in the absence of hydatidiform mole or exte-
nsive molar changes, or persistent hypertension beyond 6 weeks
postpartum (8,9,19).

P.I.II. is principally a disease of primigravid women and is unique
to humans. It has an association with a large amount of trophoblas-
t; there is a coordination with chronic vascular disease; a genetic
predisposition and a viable fetus is not always present. It more
commonly affects the woman who is at the extremes of her reproducti-
ve age, that is, a teenager or a woman more than 35 years of age
(8,9,10). Ikedife (1980) reported a series of 46 multiparous women
who had developed eclampsia. In no few than 34 of these 46 patients
the affected pregnancy was the first by a new partner (11).

Hypertension in pregnancy of any severity places the patient at
increased risk of accelarated hypertension which in turn may result
in generalised bleeding secondary to a disseminated coagulopathy;
cerebral haemmorrhages; cardiac, renal, or hepatic failure (8,9,12,
13). The risks are present regardless of the cause of the hyperte-
nsion, but may be greater for a given severity of hypertension, in
P.I.H. because of associated generalized vasospasm (13). Foetal
loss is increased at all stages of gestation in hypertensive disea-
se of pregnancy due to spontaneous abortion, premature labour and
placental separation. Less severe placental insufficiency is
associated with fetal growth retardation (13,14). Ebrahim
surveyed 368 cases of P.I.H. and found that 19.8% of them ended in
foetal loss (14). P.I.H. is known to produce arterial spasm
and placental infarction and thereby give rise to foetal malnutri-
tion (8,14). This may be the explanation of the low mean birth
weight and the high incidence of "prematurity" in the newborns. It
is possible that these are the cases of foetal growth retardation

(4). Carey and Liley (1958) related the foetal prognosis more to the duration than to the severity of the pre-eclamptic signs (15).

Eclampsia is nearly always preceded by a clear cut history of PET. but can occur in a woman who a few hours earlier showed only modest hypertension and no proteinuria. It is thus crucial to eclampsia (16).

The principal and most confusing features of PIH is that it is totally asymptomatic. The pre-eclamptic mother feels and looks well even when she has advanced disease. When she begins to feel ill the PIH has already reached its final fulminating stage and a catastrophe is imminent - either eclampsia, a massive placental abruption, or a fetal death. (1,20).

It has been suggested that pre-eclampsia, which used to be called "the disease of theories", could now be called "the disease of cascades", since so many abnormalities are triggered off by a single cause, now thought to be an immunological defect (21,22). One of these abnormalities is lack of pregnancy haemodilution, resulting in reduced plasma volume and hence high haemoglobin levels (32).

Haemoconcentration in women with PIH was emphasized by Dieckmann (1952) in his lengthy monograph, "The Toxemias of pregnancy". More recently, Prichard and co-workers reported measurements of the blood volume in women with PIH. Their findings are consistent with the view that there is reduced blood volume in PIH. The woman of average size can be expected to have a blood volume near 5000 ml during the last several weeks of a normal pregnancy compared to about 3500 ml in the non-pregnant state. With PIH, however, much of the added 1500 ml of blood normally present late in pregnancy can be anticipated to be missing (8,9,10,23).

The near absence of pregnancy-induced hypervolemia could be the consequence of generalized vasocontriction, or it could result from increased vascular permeability, which could account for the features, when compared to normal pregnancy, of too little fluid intravascularly but a marked excess extravascularly (8). Both mechanisms could be involved.

In severe PIH, there is a shift of fluid from the vascular compa-

rtment to the extracellular space. This is manifested by a rising haemoglobin level and hence haematocrit, and elevation of serum proteins and often by increased oedema. The cause of this phenomenon is unknown, but a transient reversal of the shift and temporary clinical improvement may be obtained following haemodilution by administering intravenous salt-free albumen - but a significant fall in haematocrit most often occurs only after delivery (8,23).

It is common practice in antenatal care to consider high haemoglobin (Hb) level of the mother in late pregnancy as a sign of favourable outcome of pregnancy. Hitherto, Hb-concentration has been measured during pregnancy mainly to detect anaemia, but more recently interest has been focused on Hb-concentration as an indicator of reduced plasma volume expansion or haemocontration (30). Several authors have reported that usually high Hb levels are associated with fetal disorders, both growth retardation and intra-uterine death of unknown cause before the start of labour (4,27,30,31,32,33).

The unit of packed erythrocytes is a major determinant of blood viscosity, and the increased viscosity of the maternal blood is detrimental to the utero-placental circulation. Increased viscosity has a particular impact on pregnancy induced hypertension where the uteroplacental circulation is already impaired by vasoconstriction, fibrin deposition and atheromatous lesion (4,5,7). The association between a contracted blood volume and pregnancy induced hypertension has been recognized since the days of Zangemeister in 1903. This study was therefore designed and conducted with the aim of assessing the value of Hb-concentration in monitoring patients with PIH in the third trimester at the KNH.

RATIONALE FOR THE STUDY:

Since the incidence of PIH is high (9%) amongst our pregnant population, and it constitutes a significant maternal and perinatal morbidity and mortality, and the sophisticated placental function tests are not readily available to us, the assessment of a simple test such as measuring the maternal Hb-concentration in monitoring patients with P.I.H. was worthwhile. Maternal Hb levels show a close inverse correlation to maternal plasma volume (33).

BROAD OBJECTIVE:

To determine whether maternal Hb-concentration before delivery
could be used to monitor P.I.H. in the third trimester of pregnancy.

SPECIFIC OBJECTIVES:

1. To measure maternal Hb-levels before delivery in patients
   who presented with P.I.H. and in normal pregnant women
   who acted as controls.
2. To show mode of delivery in patients who had P.I.H. and in the
   controls.
3. To compare gestation at delivery in P.I.H. and the controls.
4. To relate foetal condition at birth to the last maternal
   Hb value.
5. To relate placental condition at birth to the last maternal
   Hb value.

MATERIALS AND METHODS:

STUDY DESIGN:

This was a prospective study to assess maternal Hb-concentration
in Kenyan pregnant women in their third trimester, who had pregna-
cy hypertension. Third trimester was taken from 28 to 42 weeks of
gestation.

STUDY AREA:

The study took place in the Obstetric services at the Kenyatta
National Hospital in the three maternity wards and in labour ward.

STUDY POPULATION:

Patients without a history of hypertension prior to their pregnancy
and who were hypertensive during their third trimester of pregnancy
with or without oedema and/or proteinuria formed the study group.
The hypertension was defined as an increase of 30 mmHg systolic BP
and/or 15 mmHg diastolic BP, over previous determination, that
persisted after 6 hours of bed rest. The first and fourth Korotkoff
sounds were used to correspond to the initial sound of systole and
the last sound of diastole using a manual mercury sphygmomanometer
with a large pressure cuff, (but adequate for arm circumference).
The blood pressures were taken on the right arm which was at heart
level, by the same investigator.

The patients sat quietly with the arm unconstricted for 5 minutes or more prior to measurement. All those patients who were admitted with a diagnosis of PIH and satisfied the above criteria, were recruited in the study after giving informed consent. Those exluded from the study were those patients who were receiving diuretics; those carrying multiple fetuses; and those who presented with fetal death in-utero on admission.

The controls were healthy pregnant women without a history or evidence of either hypertension, or renal disease, during the current pregnancy.

The third normal pregnant woman who was admitted the same day as the patient with PIH was studied after matching for age group and parity as much as was possible. Controlling for day of delivery was not easy. But all the controls were normotensive at delivery.

The subjects were from those attending our ante-natal clinic at KNH and those admitted in the various maternity wards.

STUDY PERIOD:

The period of study was 4 months from October, 1988 to February, 1989.

CLINICAL METHODS:

Patients' data was obtained by direct questioning of the patients themselves. Each patient had a medical history taken and examined by the investigator.

LABORATORY METHODS:

In all cases 1.5 ml of venous blood was obtained from a large vein in the ante-cubital fossa without stasis between 8.00 a.m. and 10.00 a.m. All samples were placed in sequestrene bottles and were processed within 4 hours of collection. The haemoglobin concentration was determined using the coulter-counter model S-plus machine in the Department of Haematology (University of Nairobi) with the help of one Laboratory technician.

DATA MANAGEMENT AND ANALYSIS:

Socio-demographic and clinical data was recorded on separate

semi-structured questionaires for each patient. Laboratory data was also entered on the same questionaires as soon as the results were ready each day.

The statistical analysis were performed manually with the aid of a panasonic solar calculator. Student t-test (paired-tail) statistical analysis was used.

ETHICAL ASPECTS:

This study project was approved by the Research Committee in the Department of Obstetrics and Gynaecology. Permission to carry out the study at the KNH was sought and granted by the KNH Ethical and Research Committee.

All subjects who took part in the study were explained adequately the procedures during the initial interview and the purpose of doing this study as a means of monitoring pregnancy. All the patients who were enrolled gave informed consent for a sample of their blood to be drawn.

CONSTRAINTS:

There were no constraints encountered in this study project. Permission to collect specimen bottles from the laboratories for Hb-estimation was obtained from the Deputy Director KNH; and permission to use the laboratory services was obtained from the Chairman of the Haematology Department (University of Nairobi).

We got full co-operation from the patients, nurses and fellow doctors.

RESULTS:

108 patients with P.I.H. were initially enrolled in the study. 5 patients delivered twin babies, and 7 patients did not deliver at KNH labour ward. Thus these 12 patients were dropped from the study, leaving only 96 patients who were analysed.

108 normal pregnant women acted as controls. None of these had twins, and all delivered at the KNH labour ward, so they were all analysed.

RESULTS:

The majority of the patients with P.I.H. - 63 (65.6%) - were
between 21 and 30 years of age. Mean age was 25.6 years with
the age range of 15 to 40 years. (SD = 5.3)

Of the 108 normal pregnant women who acted as controls, 63 (58.3%)
were aged between 21 and 30 years. Mean age was 25.5 years,
with the age range of 16 - 38 years. (SD = 5.2).

(SD = standard deviation).

FIG. I: Distribution of patients by age.

Age in Years.
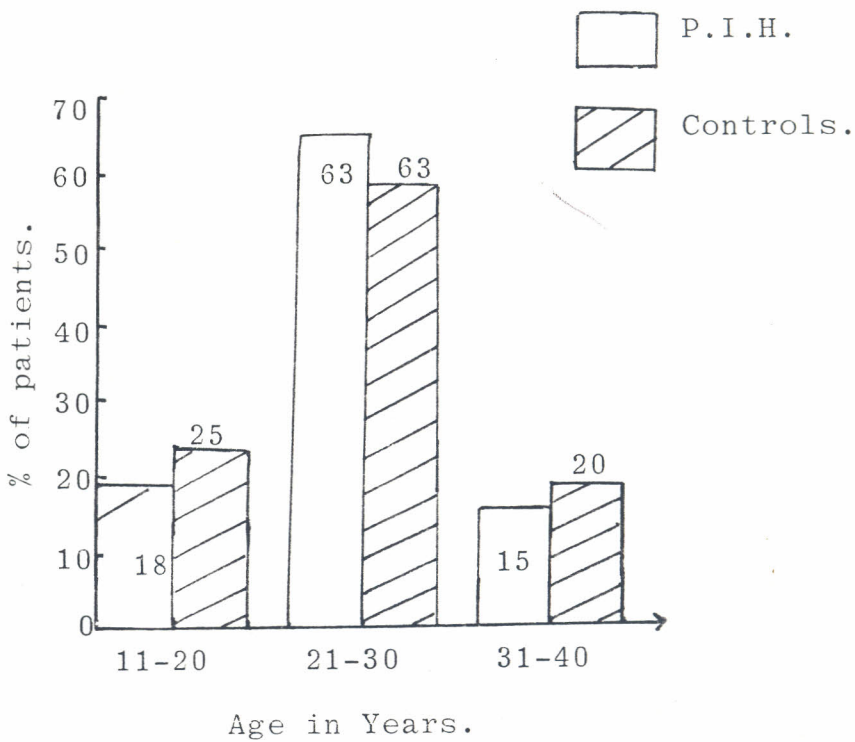
41 (42.7%) patients who had PIH were para 0+0, confirming the
fact that PIH is a disease of the primigravida. No patient was
more than para 7+0. 8 (8.3%) patients were grand-multigravidas
(Para≥5). Only 3 patients admitted that they had new partner for
the index pregnancy. Mean parity was 1.55, and the range was 0-7.
(SD = 2.1).

For the normal pregnant women, 38 (35.2%) were primigravidas.
6 (5.6%) of the controls were grand-multigravidas. None of these
had a new partner for the index pregnancy. Mean parity was 1.52,
and the range 0-7. (SD = 1.5). The difference in means was not
statistically significant. (P>0.05).

FIG. II: Distribution by parity.
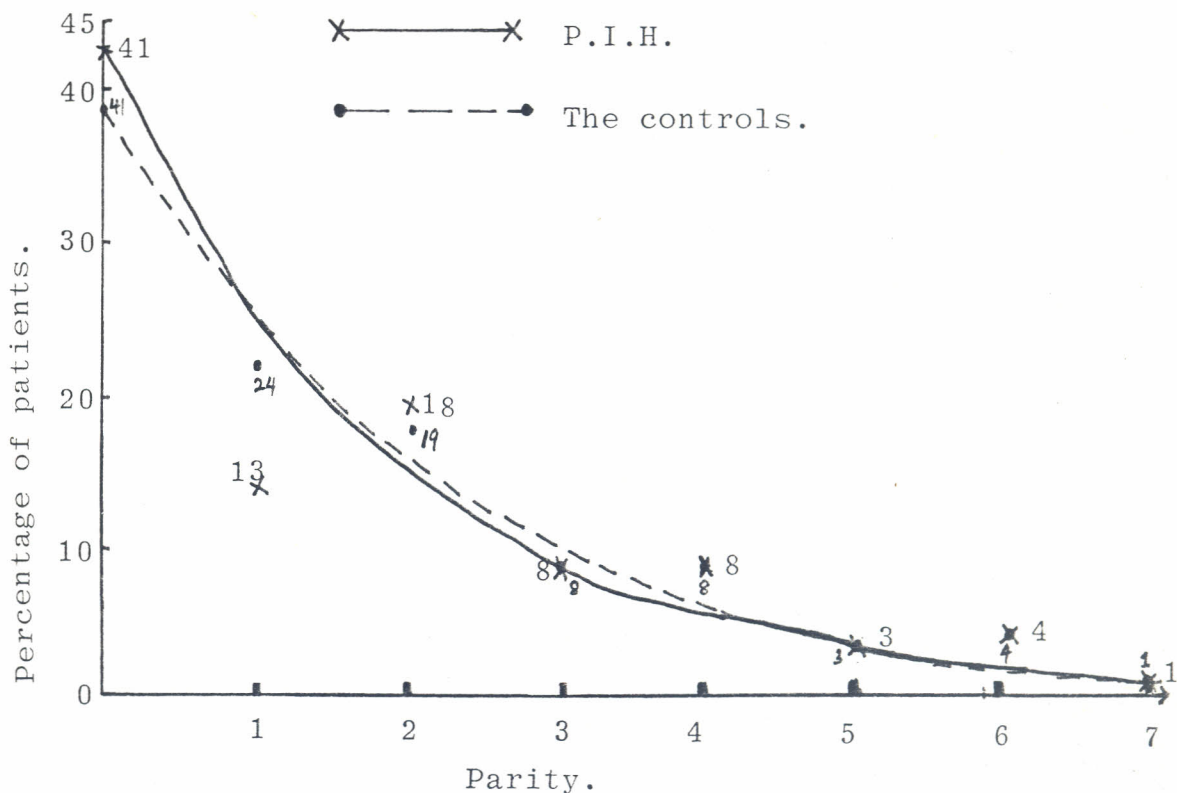
Of those who had P.I.H. 30(31.3%) were house-wives, 8 (8.3%) were nurses and 4(4.2%) were not employed. Only 5(5.2%) were students.

For the controls 73(67.6%) were house-wives 2(1.9%) were nurses and 10 (9.3%) were not employed.

TABLE I: Distribution of patients by Occupation.

| Occupation	P.I.H		Controls	
	No.	%	No.	%
House-wives	30	31.3	73	67.6
Secretary/Clerk	15	15.6	7	6.5
Private business	12	12.5	2	1.9
Teacher	9	9.4	5	4.6
Nurse	8	8.3	2	1.9
Surbodinate staff	7	7.3	4	3.7
Students	5	5.2	3	2.8
Others	6	6.3	2	1.9
None	4	4.2	10	9.3
	96	100	108	100

The majority 55(57.3%) - of those who had PIH had had at least secondary education. Only 4 (4.2%) had had college education. 2 (2.1%) had had no formal education.

TABLE II: Distribution of patients by education level.

	P.I.H.		Controls	
	No.	%	No.	%
None	2	2.1	3	2.8
Primary	29	30.2	57	52.8
Secondary	55	57.3	45	41.7
High school	–	–	1	0.9
College	4	4.2	2	1.8
Total	96	100	108	100

The majority of the patients with PIH peaked at a MAP of 107 and
117 mmHg. (140/90 and 150/100 mmHg respectively). Only one patie-
nt had a MAP of 153 which was the highest in this study population,
and the lowest MAP was 103 mmHg. Mean MAP was 115 (SD = 7.6).
But the height of MAP did not correlate with Hb concentration.
All the controls had a MAP ranging from 73 to 97 mmHg. The mean
MAP was 87.5 (SD = 6.8). The difference in the means was highly
significant (P<0.001). (MAP for the controls is not depicted
graphically as this fell far to the LEFT
of Fig.III and could not be superimposed on it

FIG. III. Mean arterial blood pressure (MAP) at delivery.(P.I.H.)

MAP was calculated from the
diastolic pressure plus one-
third of the pulse pressure.
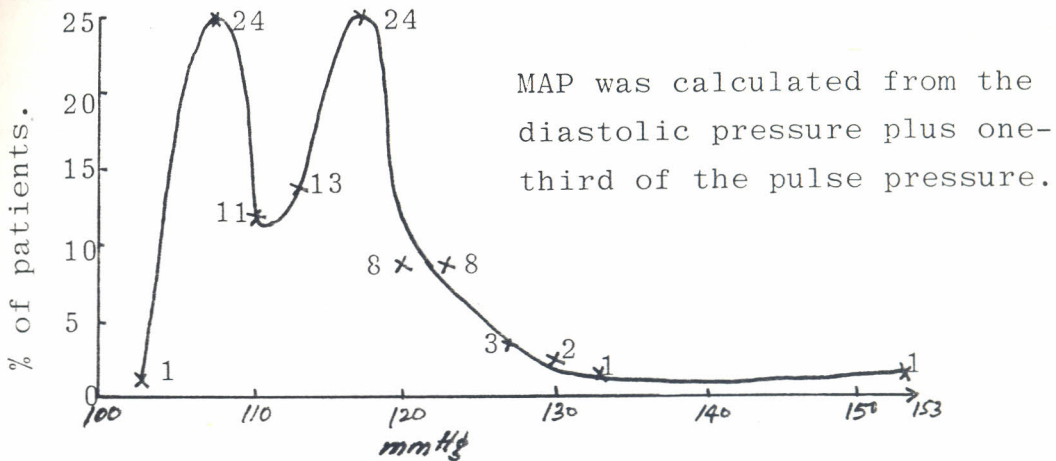
64 (66.8%) patients, who had P.I.H. delivered after 37 completed
weeks. Only 3 (2.1%) delivered at 30 weeks, and 3 (2.1%)
delivered at 42 weeks gestation. Mean gestation at delivery was
37.3 weeks and the range was 30-42 weeks. (SD = 2.8).

For the controls 91 (84.3%) patients delivered at term, and only
3 (2.8%) delivered at 30 weeks. Mean gestation at delivery was
38.4 weeks, and ranged from 30-42 weeks. (SD = 5.1). The differe-
ce in the mean gestational age at delivery was statistically signi-
ficant between the controls and those who had PIH. (P<0.001).

FIG IV. Gestational age at delivery:
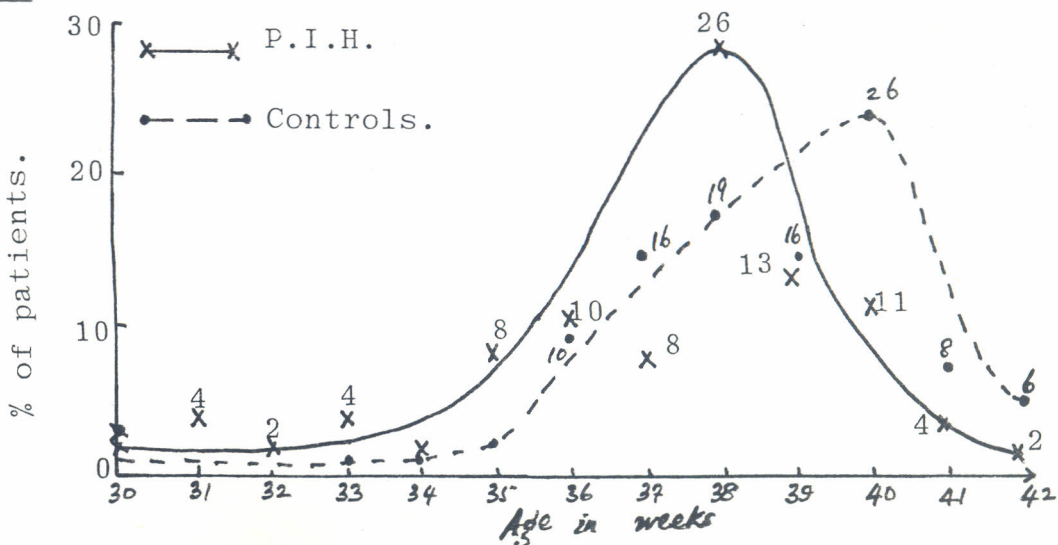
44 (45.8%) patients who had P.I.H. had Hb concentration between 11.5 and 13.4 g/dl (peak). The patients tended to maintain their steady Hb level without showing haemodilution which is characteristic of normal pregnancy. Mean Hb was 11.92 g/dl, with a range of 7.6 - 16.0 g/dl. (SD = 1.8).

40 (41.7%) patients with PIH had Hb levels above 12.5 g/dl. Confirming haemoconcentration which is typical of P.I.H. 19 (19.8%) had their Hb level above 13.5 g/dl; and 8 (8.3%) had Hb values greater than 14.5 g/dl.

In comparison, the controls had peak Hb concentration between 9.5 and 13.4 g/dl, but only 4 (3.7%) had the Hb level above 13.5 g/dl. None had Hb level above 14.5 g/dl. (Figure VI).

The difference in mean Hb was statistically significant. ($0.001 < P < 0.01$).

FIG. VI. Haemoglobin levels obtained before delivery:

Haemoglobin (Hb) concentration in g/dl.
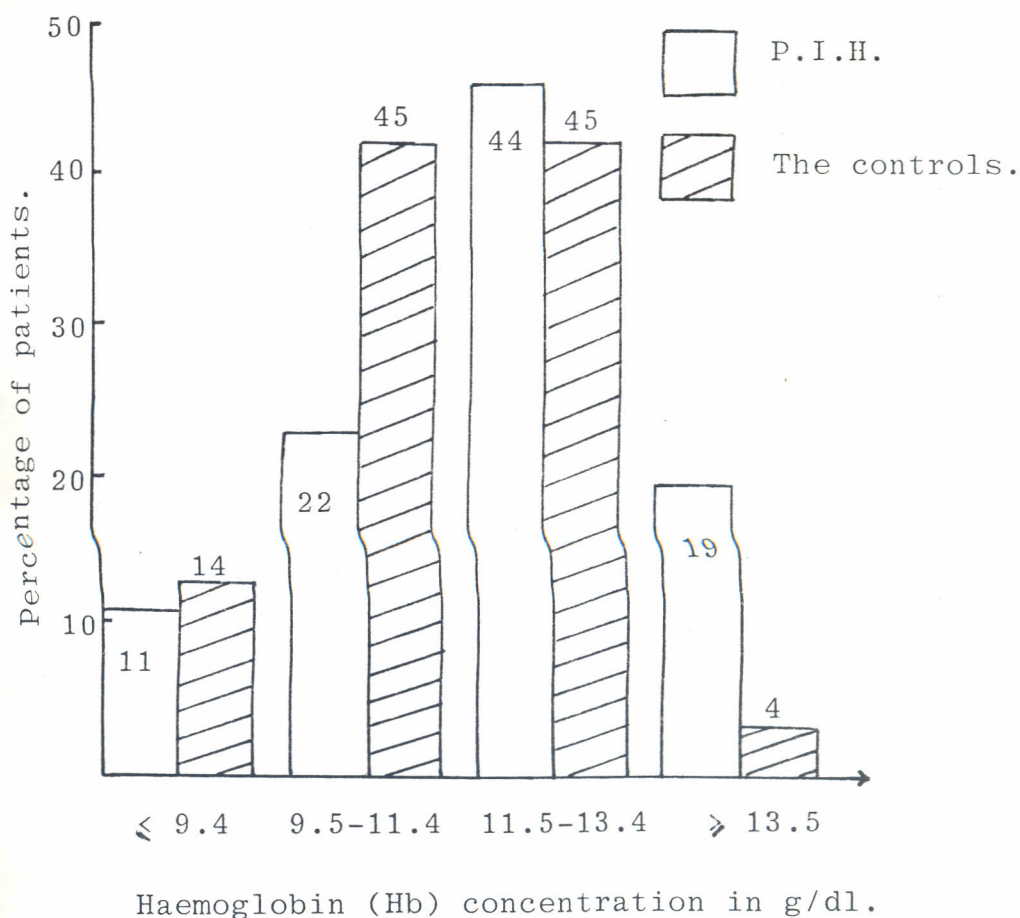
Of those who had P.I.H., 38 (39.6%) delivered by SVD. 17 (44.7%) of these were induced with either amniotomy and oxytocin infusion or prostaglandins. Breech delivery occured in 3 (3.1%) patients.

Elective vacuum extraction after induction of labour was performed in 21 (21.9%) patients. Caesarean section was done in 34 (35.4%) patients - of these, only 7 (20.6%) were elective, the rest were emergency sections, because of the following.

Reduced fetal movements (RFM):	7 (20.6%)
Failed induction of labour (FIL):	6 (17.6%)
Severe P.I.H.:	5 (14.7%)
Fetal Distress (FD):	3 (8.8%)
Breech presentation:	2 (5.9%)
Others (e.g. placenta praevia etc):	4 (11.8%)

* (RFM = Less than ten fetal kicks (as perceived by the mother in 24 hours; FIL = Labour more than 12 hours after amniotomy and start of oxytocin infusion; FD = Fetal bradycardia, irregularity of fetal heart rate and passage of meconium stained liquor; Severe PIH = BP more than 110 mmHg diastolic proteinuria 3+ to 4+, or > 4 to 5g, presence of headache).

For the controls 81 (75%) delivered by SVD, and only 24 (22.2%) were delivered by caesarean sections. 16 (66.7%) were done because of previous caesarean sections; 3 (12.5%) because of breech presentation, and the rest because of other obstetric indications such as cephalo-pelvic disproportion, placenta praevia etc.

TABLE III. Method of Delivery:

	P. I. H.		Controls	
	No.	%	No.	%
Spontaneous vertex delivery (SVD)	38	39.6	81	75.0
Caesarean section	34	35.4	24	22.2
Vacuum extraction	21	21.9	3	2.8
Breech delivery	3	3.1	–	–
Total	96	100	108	100

For the patients who had PIH, 2 (2%) infants were of extreme low birth weight ($\leqslant$ 1kg); 6 (6.3%) were of very low birth weight, and 29 (30.2%) were of low birth weight. Only 59 (61.5%) were of *normal birth weights*. *The smallest fetus was a macerated still-* birth that weighed 450g, whereas the heaviest baby weighed 4570g. The mean birth weight was 2720 gram, with a standard deviation (SD) of 822.

For the controls, none of the infants was of extreme low birth weight, 1 (0.9%) was of very low birth weight; 10 (9.3%) were of low birth weight, and 97 (89.8%) were of normal and appropriate weight. The smallest baby weighed 1380 gram, and the heaviest baby weighed 4400 gram. The mean birth weight was 3087 gram (SD=500).

The difference in the mean ages was statistically significant (P<0.001).

FIG V. Fetal birth weight.

Weight in kilograms.
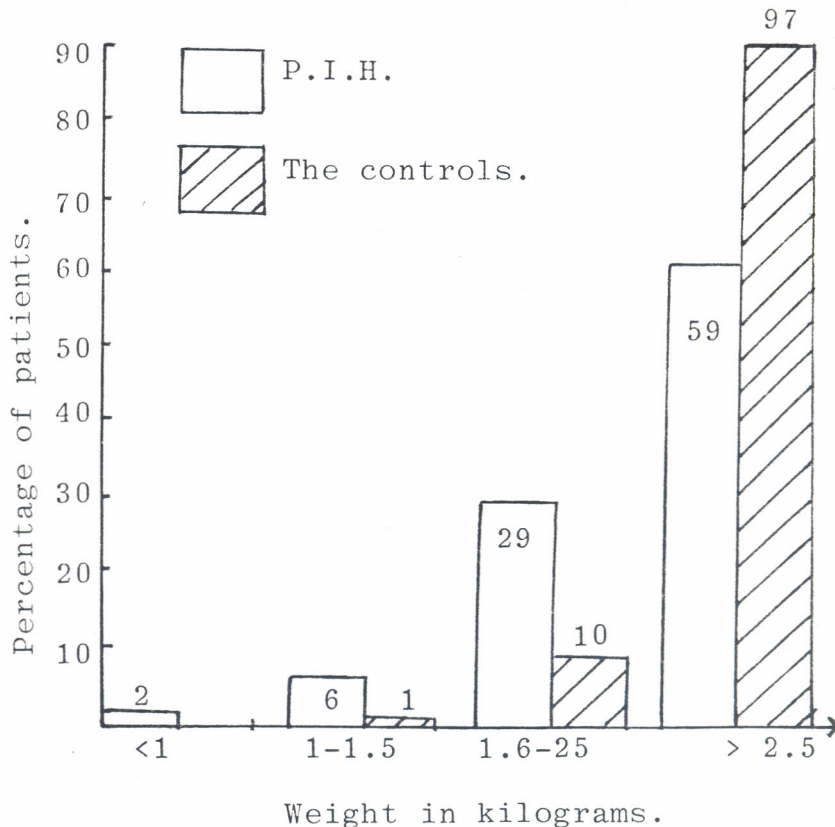
There were 8 still births (8.4%) among the patients who had P.I.H., but none among the controls. The mean birth weight of 2720g was lower than that of 3087g for the controls, which was statistically significant (P<0.001).

TABLE IV.   Fetal outcome.

	Mean Birth Weight/grams	Alive		FSB		MSB	
		No.	%	No.	%	No.	%
P.I.H.	2720	88	91.6	4	4.2	4	4.2
Controls	3087	108	100		–		–

22 (22.9%) of the patients with PIH had placental infarcts. These were usually white and multiple or large. One was also associated with placental calcification at a gestation of 35 weeks indicating premature placental degeneration. The Hb level of the patients who had placental infarcts ranged from 12.5 g/dl to 16.0 g/dl. There were no placental infarcts in patients who had Hb values lower than 12.5 g/dl in both PIH and the controls, but the presence of infarcts increased with the increase in Hb-concentration. This was statistically significant as seen in the table.

TABLE V(a).   Last maternal Hb before delivery. Relation to the presence of placental infarcts.

Hb Conc./g/dl.	PIH			Controls		
	Tot.	Infarcts pres.		Tot.	Infarcts pres.	
	No.	No.	%	No.	No.	%
< 12.4	56	–	–	97	–	–
12.5 – 13.4	21	8	38.1	7	1	14.3
13.5 – 14.4	11	7	63.6	4	1	25.0
> 14.5	8	7	87.5	–	–	–
	96	22	22.9	108	2	1.9

* (Infarcts were defined as circumscribed areas of necrosis of the villi. This definition did not include fibrin deposits or intervillous thrombi, often called red infarcts).

The table below shows a positive correlation between the presence of placental infarcts and high maternal Hb value. The difference between the mean Hb values between those patients who had placental infarcts and those where these were absent was statistically significant.

TABLE V (b).

Placental infarcts	Hb. concentration/g/dl.					
	P.I.H.			Control		
	n	Mean	SD	n	Mean	SD
Absent	74	11.3	1.6	106	11.1	1.2
Present	22	13.9	0.9	2	13.5	0.3

(P<0.001).

Placentas from patients with PIH were generally lighter than those from the controls, and the difference in the mean weights was statistically significant (P<0.001). Placental index in both the PIH and the controls was within the reference range of 16.67 - 20.00.

TABLE VI. Placental weights (gram):

	No.	Mean wt	SD	wt range	Placental index
PIH	96	532	180	100 - 950	19.76
Controls	108	615	148	280 - 1250	19.92

* (Placental index = 100 x placental weight/weight of the newborn).

Patients who had P.I.H., and had Hb levels less than 10.5g/dl. had uneventful labour and delivery as well as those who had Hb levels between 11.5 and 12.4 g/dl. One patient had fetal death in-utero with an Hb of 10.7 g/dl. Perinatal distress (including fetal death in-utero) was more common with higher Hb concentration (i.e $\geq$12.5 g/dl). Only 1 (14.3%) patient who had Hb above 14.5 g/dl had un-eventful course, as compared to more than 7 (85.7%) patients who had either acute fetal distress, sudden reduced fetal movements or *fetal death - in-utero*.

Patients who were anaemic seemed to tolerate pregnancy, labour and delivery surprisingly well.

TABLE VII (a).

Hb COnc. g/dl	P.I.H.						Controls			
	FD + RFM		FDIU		Un-eventful course.		FD + RFM		Un-eventful course.	
	No.	%	No.	%	No.	%	No.	%	No.	%
6.0- 7.4									1	100
7.5- 8.4					4	100			3	100
8.5- 9.4					7	100	1	10	9	90
9.5-10.4					8	100			12	100
10.5-11.4			1	6.7	14	93.3			33	100
11.5-12.4					22	100	1	2.6	38	97.4
12.5-13.4	2	9.5	2	9.5	17	81			7	100
13.5-14.4	1	9.1	3	27.3	7	63.6			4	100
14.5-15.4	4	57.1	2	28.6	1	14.3			-	
15.5-16.4	1	100	-		-				-	

* (FD = Fetal distress.

   RFM = Reduced Fetal Movement.

   FDIU = Fetal Death In Utero).

The table above shows: Last maternal haemoglobin values before delivery. Relation to the state of the newborn.

For those who had P.I.H., high Hb was associated with poor perinatal outcome. They were more prone to intra-uterine fetal demise, intra-partum fetal distress and ante-partum reduced fetal movements. (Table VII).

When the birth-weights were compared to the controls, those who had high Hb levels tended to have low birth weight babies (and this was statistically significant (P<0.001).

TABLE VII (b). Last maternal Hb values before delivery. Relation to the state of the newborn.

| Perinatal state | Haemoglobin concentration. | | | | | | |
| | P.I.H | | | | Controls | | |
	n	Mean	SD	P value	n	Mean	SD
Un-eventful course	80	11.5	1.6	P<0.001	106	11.2	1.3
Perinatal distress (Fetal death inc.)	16	13.9	1.2		2	10.5	1.4
Living infants	87	11.7	1.8	P<0.001	108	11.2	1.3
Intra-uterine death	8	13.5	1.3		0	-	
Weight ≥ 2500g	62	11.6	1.6	P<0.05	98	11.1	1.3
Weight < 2500g	34	12.5	2.0		10	11.3	1.5

* (The designation of perinatal distress was applied to 16 infants including 8 still births, and 8 infants who either had Apgar scores less than 7 at 5 minutes, or had neonatal asphyxia or respiratory distress).

DISCUSSION:

Raised Hb levels are frequently found in severe PIH, probably
because of reduced plasma volume. Fetal growth retardation is
often a concomitant of severe PIH, and it is reasonable to assume
this to be a consequence of increased uterine tonus and exagerated
arteriolar spasm with reduction of the uteroplacental circulation.
To what extent low birth weight is directly correlated with raised
Hb level is not known.

In this study, it seems that high Hb concentration per se during the
third trimester in pregnancies complicated by PIH, may indicate
fetal growth retardation and impending fetal demise in-utero.

We did not separate patients with mild and severe PIH because Hb
levels did not correlate with severity of the disease. Huisman
and Aarnoudse (31) have also shown that a reduced plasma volume,
as indicated by the significantly high Hb-concentration, may
already be present in the second trimester, thus preceding PIH
in the last trimester of pregnancy. None of our patients had low
platelet counts, and because there was virtually no difference
between the Hb-concentration in primigrada and multigravida women,
the two groups were combined. The clinical significance of a
given Hb concentration may vary with the time of onset and duration
of the disease (4). Haemolysis may also occur during severe P.I.H.
leading to reduction in the Hb level. This may partly explain
the low Hb level of 10.7 g/dl of one of our patients who had fetal
death-in-utero, although other tests were not done in her to
document this.

It is common practice in ante-natal clinics to consider high Hb
levels of the mother in the third trimester as a favourable sign
for the outcome of pregnancy. The present study as well as others
(4,27,30,31,32,33) indicate, however, that the opposite may be
true in PIH. The traditional routine in the ante-natal care is
to treat cases of anaemia with standard doses of haematinics.
The philosophy behind this is that raising the Hb level can only
do good.

However, it is shown in this study that higher Hb concentrations
are associated with increased incidence of fetal death in-utero,
thus raising the Hb levels might represent a risk to the fetus.
In this study, pregnant patients tolerated a low Hb level surpris-
ingly well.

The findings may seem to disagree with those of other authors
who have reported an association between a low Hb value and a low
birth weight (34). The present study, however, concerns patients
with PIH, while the others referred to women with Hb below 11 g/dl,
in whom no distinction was made between physiological and patho-
logical reductions in maternal Hb concentration. In addition, the
low birth weights in cases of low maternal Hb concentration were
an effect of preterm labour; there was no increased rate of weight
retardation in infants born at term. Neither was it explicitly
stated in which pregnancy week the Hb tests were made (34).

Viewed in the light of the pathophysiology of PIH, our results
easily fall in place. The correlation between the increase in
plasma volume during pregnancy and the weight of the newborn may
indicate that the increase in plasma volume is of particular
importance to the foetal nutrition (1,3,8,9) and to the uteroplace-
ntal circulation (32).

Another observation may also be relevant. Naeye (27) found that a
falling rate of large fetal placental infarcts was correlated with
the reduction in maternal Hb values. In other words, pregnancy
haemodilution may help to prevent thrombosis of the uteroplacental
circulation, thereby promoting the nourishment and growth of the
fetus. If we accept that growth enhancement, in this particular
connection, is a sign of fetal well-being, then low pregnancy Hb
concentration is a favourable sign - if true anaemia is ruled out.
Placental infarction is usually caused by occlussion of the spiral
arteries in the myometrium or decidua and occasionally by impedi-
ments to venous drainage (27). In the present study, PIH was
associated with significant infarcts. Placental infarcts restrict
fetal nutrition thereby limiting fetal growth, and infarcts appear
to often serve as the nidus for premature placental separation (27)
No such infarcts were found in women with Hb below 12.5 g/dl, and
the frequency increased significantly with increasing Hb. We
interpret this as indicating that the viscosity of the mothers

blood plays a significant role in the genesis of large placental infarcts.

The pathologic lesion in low birth weight for gestational age and fetal death in-utero associated with high Hb levels may well be large placental infarcts (27), which were noted in the present study, (and other studies elsewhere); however, no systemic histo-logic examinations of the placentas were performed. One of our goals in ante-natal care should, therefore, be to prevent unnece-ssary increase of the Hb and the haematocrit caused for instance by uncritical use of diuretic drugs.

Soffronoff et al found marked hypovolaemia in pregnant women with severe pre-eclampsia and the reduction in plasma volume was parti-cularly marked in association with severe placental failure, while the reduction in the erythrocyte volume was less pronounced.

The association between increased maternal Hb concentration and placental dysfunction seems well established. Whether this asso-ciation is simply a statistical one, or whether there is a causal relation, is conjectural.

The reference range of Hb level in normal pregnancy is wider than ordinarily appreciated, being 8.7 - 14.4 g/dl elsewhere. (33), and the Hb concentration in P.I.H. is more dependent on the plasma volume than on the red cell mass. Therefore, a low Hb may signify a large increase in plasma volume while a high Hb may indicate a weak increase. Thus, a high maternal Hb value should be a matter of concern rather than of false reassurance.

CONCLUSIONS:

1. High Hb value in P.I.H.,is associated with increased incidence of acute fetal distress, reduced fetal movements, and sudden fetal death in-utero due to large placental infarcts.

2. Provided that no anaemia exists, estimations of Hb levels during P.I.H. are valuable supplements to other placental function tests.

3. Just as with many other diagnostic tests in medicine, interpretations of haemogram results should include consideration of the patients clinical status and should not be taken in isolation.

RECOMMENDATION:

The measurement of maternal haemoglobin concentration in monitoring P.I.H. is rapid, simple and in-expensive. It can be appropriate as a monitoring procedure in many hospitals handling ante-natal mothers in developing countries, where more sophisticated placental function tests are not readily available.

REFERENCES: 166

1. Sibai B.M. Pitfall in diagnosis and management of pre-eclampsia.
    Am. J. Obstet. Gynecol. 159: 1, 1988.

2. Ihle BU, Long P, Oats J. Early onset of pre-eclampsia:
    recognition of underlying renal disease. Br. Med. J. 294:79,1987.

3. Selman IW and Crenshaw MC. Concurrent Hypertension and
    pregnancy Clin. Obstet.Gynacol. 21: 619, 1987.

4. Sagen N, Koller O, Haram K. Haemoconcentration in severe
    pre-eclampsia. Br. J. Obstet. Gynecol. 89: 802, 1982.

5. Mati JKG. Studies on pregnancy hypertension in Kenya.
    MD. Thesis. University of Nairobi. 1975.

6. Aggarwal VP, Mati JKG. Review of perinatal mortality at
    Kenyatta National Hospital Nairobi. J. Obst. Gyn. East. Cent.
    Afr. 1:1,1982.

7. Sheppard BL and Bonnar J. An ultrastructural study of utero-
    placental spiral arteries in hypertensive and normotensive
    pregnanoy and fetal growth retardation. Br. J. Obstet. Gynecol.
    88: 685, 1983.

8. Pritchard JA, MacDonald PC, Gant NF (Editors) Williams Obstetri-
    cs 17th Edition. Appleton-Century Craft, Nowalk, Connecticut.
    1985.

9. Rivlin ME, Morrison JC, Bates GW (Editors) Manual of clinical
    problems in Obstetrics and Gynaecology. First Little, Brown
    and company. Boston. 1982.

10. Moore MP and Redman CWG. Case-control study of severe-eclampsia
    of early onset. Br. Med. J. 287: 580, 1983.

11. Ikedife D. Eclampsia in Multiparae. Br. Med. J. 280:985,1980.

12. Muia Ndavi. Coagulation studies in hypertensive disease in
    pregnacy. M. Med. Thesis. University of Nairobi, 1985.

13. Galley EDM. Hypertension in pregnancy. The Islamic World
    Medical J. 1: 12, 1984.

14. Ebrahim GJ. Birth weight and Mortality Data in neonates born
    in Toxaemic mother. E.A. Med. J. 46: 34, 1969.

15. Carey HM and Liley AW. Quoted by 14.

16. Khan AQ. Pregnancy induced hypertension: Complications and
    Management. Postgraduate Doctor Africa 9: 20, 1987.

17. Gant NF, Chand S, Worley RJ, Crosby VD, MacDonald PC. A clinical
    test useful for predicting the developement of acute hypertens-
    ion in pregnancy. Am. J. Obstet. Gynecol. 120: 1, 1974.

18. Bansal YP. Pre-eclampsia/Eclampsia: A profile from Pumwani
    Maternity Hospital Nairobi. E.A. Med. J. 62: 691, 1985.

19. Onuoga O. and Rogo KO. The value of Roll-over - Test in the prediction of pre-eclampsia. E.A. Med. J. 65: 22, 1988.

20. Redman CWG. Hypertension in pregnancy. Post Graduate Doctor Africa 1: 91, 1979.

21. Scott J.S. Immunology of pre-eclampsia. Lancet 1: 705-6,1978.

22. Drife JO. The cause of pre-eclampsia. Medicine Digest. 9: 5-11, 1983.

23. Goodlin R.C. Severe pre-eclampsia: Another great imitator. Am. J. Obstet. Gynecol. 125: 747-753, 1976.

24. Philip EE, Barnes J, Newton M, (Editors) Scientific Foundations of Obstetrics and Gynaecology. Third Edition. Williams Heinemann Med. Books Ltd. 1986.

25. Fadel HE, Northrop G, Misenhimer HR. Hyperuricemia in pre-eclampsia: A reappraisal. Am. J. Obstet. Gynecol. 125: 640-647, 1976.

26. Wanjohi JK. Eclampsia in Kenyatta National Hospital. M. Med. Thesis. University of Nairobi, 1984.

27. Naeye RL. Placental infarction leading to fetal or neonatal death. A prospective study. Obstet. Gynecol. 50: 583, 1977.

29. Gibson HM. Plasma volume and glomenular filtration rate in pregnancy and their relation to differences in fetal growth. J. Obstet. Gynecol. Br. Commonw. 80: 1067-1074, 1973.

30. Mathews JD and Mason TW. Plasma Viscosity and pre-eclampsia. Lancet ii: 409, 1974.

31. Huisman A. and Aarnoudse J.G Increased second Trimester Haemoglobin concentration in pregnancies later complicated by hypertension and growth retardation. Acta. Obstet. Gynecol. Scand. 65: 605, 1986.

32. Koller O. The Clinical Significance of Haemodilution during pregnancy. Obstet. Gynecol. Surv. 37: 649, 1982.

33. Sagen N, Nilsen S.T., Kim H.C., Bergsjo P. and Koller O. Maternal Haemoglobin concentration is closely related to Birth weight in normal pregnancies. Acta. Obstet. Gynecol. Scand. 63: 245, 1984.

34. Kaltreider D.F. and Kohls S. Epidemiology of preterm delivery Clin. Obstet. Gynecol. 23: 17, 1980.

35. Dieckmann Quoted by 8.
    Zangemeister Quoted by 4.
    Soffronoff Quoted by 4.

GYNAECOLOGY

SHORT CASES

AND

LONG COMMENTARY.

Name:	G.W.	Parity :	0+0
Age:	29 years	LMP	None
Ip.No:	805648	Admission:	19.8.1988
		Discharge:	19.8.1988

PRESENTING HISTORY:

Miss G.W. was referred to the Kenyatta National Hospital
Gynaecology clinic by a private medical practitioner from
Machakos in 1987 as a case of primary amenorrhoea of unknown
aetiology. She gave a history of not having had any menses since
birth, poor development of her breasts and failure to conceive.
She had no history indicative of cryptomenorrhoea, and had no
gastro-intestinal or urinary tract complaints, and had no skeletal
deformities. She had not experienced headaches or visual
symptoms and her sense of smell was normal.

PAST MEDICAL AND SURGICAL HISTORY:

She had not been hospitalised before, but had been to various
health units for the same problems. No previous surgical procedure
had been performed on her. She had no other significant past
medical history.

PAST GYNAECOLOGICAL HISTORY:

She had not menstruated or conceived. She had had irregular and
infrequent sexual relations with her boyfriend with no problems.

FAMILY AND SOCIAL HISTORY:

She was the second born in a family of seven siblings. Her other
sisters did not have similar problems. Her elder sister was
married and had four children. She had two sisters and four
brothers. She was employed as a house-maid and had been educated
upto standard four. She was still single and had a boyfriend with
whom she had occasional coitus.

PHYSICAL EXAMINATION:

She was a young well nourished female in a good general condition. She had no pallor, jaundice, leg oedema or peripheral lymph node enlargement. Her temperature was 36.6°c, blood pressure was 130/ 80 mmHg; pulse rate of 80 per minute and regular.

The examination of the cardiovascular, respiratory, and central nervous systems was essentially normal. Abdominal examination was also grossly normal.

GENERAL BODY APPEARANCE:

She was of slim build, her height was 162 cm, with an arm span of 140 cm and a weight of 54 Kg. Her general build was rather eunuchoidal. She had feminine features and her voice was moderately high-pitched. She had no evidence of facial dysmorphism or webbing of the neck. The carring angles of her arms were normal. Her breasts were small, symetrically poorly developed with areolae but no nipple development. She had sparsely developed axillary and pubic hair. She had no hirsutism.

PELVIC EXAMINATION:

The vulva: The external genitalia were fairly normal with the labia major marginally infantile - the labia minora appeared more developed. The external urethral meatus was in its normal position within the vestibule. The clitoris was single and normal in shape and size.

The vagina: Was about 5cm long, but ended blindly. There were no distinct fornices. There was no mass felt at the vaginal vault.

The Cervix: was absent.

The Uterus: was not palpated.

No masses were palpated in the adnexae or in the cul-de-sac on recto-vaginal examination.

DIAGNOSIS:

A diagnosis of gonadal and mullerian duct agenesis in a
phenotypic female was made.

INVESTIGATIONS AND RESULTS:

. Haemoglobin   : 13.1 g/dl.
. Haematocrit   : 39.4%.
. Blood urea nitrogen   : 2.3 mmol/l.
. Serum sodium   : 140 mmol/l.
. Serum potassium   : 3.9 mmol/l.
. Serum creatinine   : 69 mmol/l.
. Hormonal profile (on 2 occasions);
    Prolactin: 83 and 290 mU/ml.
    FSH   :   0.65 and 0.6 mU/ml.
    LH   :   1.98 and 0.6 mU/ml.

(Assays of serum 17B - oestradiol, progesterone, dihydrotestos-
terone, and testosterone were not performed because of lack of
reagents).

    . Buccal epithelial smear for single sex chromatin showed
      24% of cells positive for Barr bodies.
    . Intravenous urography was reported as normal.
    . Urine analysis was normal. No unusual red cell antigens
      were present.

(The following investigations were not done because of one reason
or the other: Karyotyping in peripheral lymphocytes; Quinacrine
fluorescence: peripheral blood neutrophil drumstick evaluation;
X-ray series of digits, chest wall and pituitary fossa; Dentition
and bone age; and Dermatoglyphic patterns).

The patient was then booked for diagnostic laparoscopy which was
done on an outpatient basis on 19.8.1988.

DIAGNOSTIC LAPAROSCOPY:

The patient had starved overnight and was admitted to the laparo-
scopy ward on the morning of the day of operation. Pre-operative
vital observations were normal. Consent was obtained for the
operation. The abdomen was shaved and she was premedicated with
atropine 0.6 mg intramuscularly thirty minutes before she was
taken to theatre.

In theatre, she was put on the operation table and given intraven-
ous analgesia (100 mg. pethidine and 10 mg diazepam). She was then
placed in the semi-dorsal lithotomy position with the legs abducted
and supported, on padded special laparoscopy stirrups. Vulvo-
vaginal toilet was done. Patient draped with sterile surgical
towels, and bladder catheterisation done. Pelvic examination
confirmed the earlier findings. A Sim's speculum was introduced,
and the cervix was found to be absent. The rest of the procedure
was as described under "Primary Infertility - investigations".

LAPAROSCOPIC FINDINGS:

A clear view of the pelvis was obtained. No adhesions were found,
and there was no fluid within the pelvis. The uterus and the
fallopian tubes were found completely absent. White looking
streak ovary was seen on the right side - measuring about 3 cm
at its widest diameter. Small rudimentary streak ovary on the
left side was partly covered by omentum. No testis was seen.
These streak ovaries were not removed because the laparoscope
biopsy forceps was not available at the time.

No pelvic kidney was visualized. The renal angles and liver were
normal.

The patient recovered fully from this local anaesthesia and
systemic analgesia, and was discharged home after three hours of
bed rest in the laparoscopy-cum-recovery ward. She was reviewed
one week later and the wound was found to be well healed.

At laparoscopy the diagnosis of gonadal and mullerian duct
agenesis was confirmed.

FOLLOW-UP:

She was subsequently referred back to our Gynaecology clinic
for long-term follow-up. Seen at the clinic 6 weeks later, She
was adequately explained her state of affairs and started on
hormonal therapy.

Developmental failure of the müllerian system as part of gonadal
dysgenesis is an extremely rare phenomenon. The purpose herein
is to present a female with gonadal dysgenesis who was
also found to have complete absence of the fallopian tubes, the
uterus and the cervix. The patient presented with primary
amenorrhoea, poor breast development and primary infertility at
the age of 29 years.

In Kenyatta National Hospital, Müllerian agenesis constitutes
about 44.2% of cases of primary amenorrhoea, and the incidence of
the latter among gynaecological admissions is 0.11% (1).

Developmental failure of müllerian system in association with
gonadal dysgenesis was first described by McDonough et al (2) in a
16 - year old girl with gonadal dysgenesis, duplication of the
Müllerian system, and a normal Karyotype. In 1971, Wong et al (3)
reported a 14 year old girl with a 46, XX Karyotype and clinical
stigmata of Turner's Syndrome with one streak gonad associated with
absence of the uterus. Recently, two patients with gonadal
dysgenesis, müllerian duct abnormalities, and vaginal agenesis, one
with a normal female Karyotype and the other with a 45 X chromoso-
mal complement, were reported by Phansey et al (4). De Leon et al
(5) described a patient with gonadal agenesis and müllerian duct
deficiency in the presence of an isochromosome for the long arm
of the X-chromosome in 1984.

The association of complete gonadal absence with agenesis of müll-
erian ducts has also rarely been reported in indivuduals with
normal male and female Karyotypes (6). The present patient is
described with pure gonadal dysgenesis and müllerian duct deficie-
ncy in presumably normal female karyotype. The basis for müllerian
duct regression in this case remains unclear. Although the possib-
ility exists that there was a small foci of testicular tissue pro-
ducing müllerian inhibiting factor, no testicular tissue was iden-
tified at laparoscopy. It is impossible to ascertain whether
the patients findings represent a rare feature of chromosomal
abnormality, are a manifestation of a localized field defect resul-
ting from an as yet unrecognized cause, or simply were the result
of a random assoçiation (5). This patient did not demonstrate any
of the stigmata of Turner's Syndrome.

Gonadal dysgenesis has classically been associated with Turner's Syndrome or with Turner's stigmata (7). Turner's Syndrome is characterised by a 45, XO chromosome constitution, although it occurs also in patients with a 46, XX karyotype. As is evidenced in our patient gonadal dysgenesis is also found in phenotypic females without the somatic malformations seen in Turner's Syndrome. If bilateral streak gonads are found at laparoscopy, this is referred to as pure gonadal dysgenesis. When a unilateral streak is present, with a contralateral testis, the syndrome is known as mixed gonadal dysgenesis (7).

Most patients with pure gonadal dysgenesis have a normal female or male karyotype (4,6,8), so the finding of streak gonads at laparoscopy does not necessarily imply an abnormal sex chromosome complement. Whatever the chromosomal pattern, patients with pure gonadal dysgenesis are of average height or tall, with a female phenotype, although they may have eunuchoidal proportions. The breasts are undeveloped or small and the axillary and pubic hair is generally sparse. The external genitalia tends to be infantile or fairly normal. Our patient had, in addition to these features, absent fallopian tubes, uterus, cervix and the upper part of the vagina.

Endogenous oestrogen production may be present, although low in amounts. Genadotrophin titres are either very high or within normal limits (7). In our case, the FSH and LH levels were unusually low. This could have been due to errors in measurements or were they unexpected findings? Histologically, the streak gonads do not contain any germinal elements, the basic structure being fibrous tissue which may simulate ovarian stroma. Mesanephric remnants are frequently found and groups of Leydig or hilar cells my be seen (7).

Why patients with normal chromosomal pattern should develope dysgenetic gonads is poorly understood. It seems faily well accepted that gonadal dysgenesis is of genetic nature but the genetic mechanism is unknown (7). The finding of a normal karyotype in most cases differentiate the aetiology of pure gonadal dysgenesis from the aetiology of dysgenetic gonads in Turner's Syndrome, in which chromosomal abnormalities are present in almost all cases. The underdevelopment of the gonads during fetal life may be the result of either a destruction of the genital ridge or

failure of the primitive germ cells to reach the genital ridge (7).

According to Teter and Beczkowki (8) there is an unexpectedly high risk of neoplasia developing in patients with abnormal gonadal development, especially in those with a negative sex chromatin pattern and a Y chromosome. They found that 8 to 26 patients with gonadal dysgenesis without somatic malformation had gonadal tumours It is however, difficult to accept that there is an increased malignant potential in pure gonadal dysgenesis, since both gonads are mainly fibrotic without any active cellular elements (8,9). Yet development of gonadal blastomas in patients with an XY karyotype and pure gonadal dysgenesis has been reported. Therefore it must be accepted that all patients with dysgenetic gonads do run an increased risk of developing neoplasia in these gonads (8). Our patient did not have any signs of masculinization. ·

The approach to therapy in these patients is the replacement of sex steroids for the development of secondary sexual characteristics (5). *This patient will probably need high dose oestrogen* therapy for her breast development.

Intravenous urography done in our patient showed a normal urinary system. This supports the fact that patients with pure gonadal dysgenesis (either XX or XY) or mixed gonadal dysgenesis do not seem to have urinary system anomalies. In contrast over 50 percent of the classic Turner's Syndrome patients have anormalities of the urinary tract. The most common anomalies in this syndrome have been horseshoe kidneys, malrotation, unilateral agenesis, and bifid pelvis. Malrotation problems are the most common abnormality reported (9).

In conclusion, I stress the fact that phenotypic females presenting with primary amenorrhoea require full investigations as was done in our case, to elucidate the underlying pathology. Laparoscopy is required in ALL cases to exclude a unilateral testis which is prone to undergo malignant change. In those with a unilateral testis, removal of the testis and opposite streak gonad is mandatory. In patients with a normal 46 XX karyotype gonadal visualization and biopsy are also required, so as to provide the patient with a correct prognosis and counselling regarding marriage and possible adoption of children.

REFERENCES:

1. Ngumbi P.M. Primary amenorrhoea: Presentation and management at the Kenyatta National Hospital - 8 years period 1967-1974. M. Med. Thesis. University of Nairobi 1976.

2. McDonough PG, Bryrd RJ, Freedman NA. Complete duplication and underdevelopment of the müllerian system in association with gonadal dysgenesis. Obstet. Gynecol. 35: 875, 1970.

3. Wong SR., Lippe BM., Kaplan S. The XX Turner phenotype with unilateral streak gonad and absent uterus. Am. J. Dis. Child. 122: 449, 1971.

4. Phansey SA, Tsai CC, Williamson HO. Vaginal agenesis in association with gonadal dysgenesis. Obstet. Gynecol. 57S: 6, 1981.

5. De Leon FD, Hersh JH, Sanfilipo JS, SCHIKLER KN, YEN FF. Gonadal and Mullerian Duct Agenesis in a Girl with 46, X : (Xq). Obstet. Gynecol. 63: 81S, 1984.

6. Levinson G, Zarate A, Toledano RG, et al : An XX female with sexual infantilism, absent gonads and lack of mullerian duct. J. Genet. 13: 68, 1976.

7. Kaplan E. Gonadal Dysgenesis in a phenotypic Female with an XY chromosomal constitution. S. Afr. Med. J. 53: 552,1978.

8. Teter J. and Beczkowki K. Occurrence of Tumours in Dysgenetic gonads. Cancer 20: 1301, 1967.

9. Buchsbaum HJ and Schrnidt JD. Gynecological and Obstetric Urology. First Edition. W.B. Saunders Company. Philadelphia/ London/Toronto. 1978.

RECURRENT ECTOPIC PREGNANCIES - CONSERVATIVE SURGICAL MANAGEMENT OF THE TUBE.

Name: D.M.   Unit No: 489080.
Age: 30 years   DOA: 12.9.1989.
Parity: 0+2   DOD: 16.9.1989.
LMP: 25.7.1989.

PRESENTING HISTORY:

Mrs. D.M. was admitted to our Acute Gynaecology ward on 12.9.89 at about 4.30 p.m. with a two day's history of lower abdominal pain and vaginal bleeding. The pain had come on suddenly but was now generalized and worse on movement. There was no history of fainting episode or shoulder-tip pain. When the pain started two days earlier, she went to casualty but was treated and discharged home on aspergic acid and buscopan. The next day the pain had become worse. She returned to casualty from where she was admitted to the ward. The vaginal bleeding was only slight. There was no history of passage of any uterine cast.

OBSTETRICS AND GYNAECOLOGICAL HISTORY:

She had her menarche at 14 years, and subsequently had regular periods. She had not used any contraception. She was para 0+2. She had had two previous ectopic pregnancies. The first ectopic pregnancy occurred in 1982 for which left total salpingectomy was done. The second one was in 1983 which was a right cornual preg- nancy, and a right wedge resection was done. The wall of the uterus was stitched in a single layer. The whole right tube was left hebind. At both operations, both ovaries were found normal, and there were no adhesions. Histological examination confirmed the pregnancy in both occasions.

In 1985, she had tuboplasty done on her. This tubal anastomosis was performed in a private Hospital, but she was being followed up in our Gynaecology outpatient clinic.

Her last normal menstrual period was on 25.7.1989 lasting her usual 4 days. She thus had a period of amenorrhoea of seven weeks on admission.

FAMILY AND SOCIAL HISTORY:

She was married and was a nurse at the Kenyatta National Hospital. Her husband was working in the Computer Programming department with the Government of Kenya.

She did not smoke or take any alcohol.

PHYSICAL EXAMINATION:

Her general condition was stable. She had a blood pressure of 90/60 mmHg. Her pulse rate was 88 per minute, regular and not thready. The body temperature was 36.2°c, and the extremities were not cold or clammy. Her respiratory rate was 20 per minute and regular. Her mucous membranes were not dry and the tongue was not shunken. Her skin turgor was normal.

ABDOMINAL EXAMINATION:

The abdomen was slightly distended at the hypogastrium with marked tenderness the same region. Fluid thrill was present. Both guarding and rigidity were conspicuously absent. Rebound phenomenon was positive. There was no organomegaly. Abdominal paracentesis was positive for non-clotting blood.

PELVIC EXAMINATION:

This was performed very gently and cautiously. There was vaginal bleeding, but with no cast. The cervical os was closed. There was marked tenderness at the right adnexum. Cervical excitation test was positive on the right side. The cul-de-sac was full.

DIAGNOSIS:

A diagnosis of ruptured ectopic pregnancy was made, and she was resuscitated and taken to theatre, where a laparotomy was performed; after obtaining the necessary informed consent.

LAPAROTOMY:

After being anaesthetized, she was placed in semi-dorsolithotomy position; vulvo-vaginal toilet was done with savlon lotion; draped

and aseptically catheterized. She was then returned to supine
position, cleansed and draped with sterile surgical towels. The
abdomen was opened via the old pfannestiel incision after
removing the old scar tissue. The following were found:
- haemoperitoneum of about 2 litres of blood with clots
- right tubal ectopic pregnancy which seemingly had been aborted
  and got attached between the ovarian cyst and the right fimbri-
  al end;
- right corpus luteum cyst of about 4 x 4 x 3 cm^3,
- bulky uterus, which was soft and corresponding to an 8 week
  gestation. It had small subserosal fibroids;
- normal left ovary;
- missing left tube;
- no pelvic adhesions.

The following were then done: Haemoperitoneum, blood clots and
the secundines were evacuated from the peritoneal cavity, and
peritoneal toilet done with warm normal saline. There was no
bleeding from the tube, so it was left intact (conserved).
The abdomen was closed in the usual 3 layers.

POST OPERATIVE CARE:

Routine post-operative care was instituted. She did remarkably
well post-operatively. She was discharged home on the third
post-operative day (P.O.D.) to come back for removal of the stitches
on the 7th POD. She did not require any blood transfusion. She
was adequately counselled together with her husband) and she fully
understood the precarious state of affairs she was in. She was
advised to have hysterosalpingograph (HSG) done on her after 3
to 6 months, but she was to be seen in the Gynaecology clinic in
6 weeks time for review.

Haemoglobin was checked on the third post-op day and was 10.6 g/dl.
Seen in the Gynaecology clinic in 6 weeks, she was fully recovered,
and was re-advised as above.

DISCUSSION.

Ectopic pregnancy is defined as the implantation of a fertilized ovum at an aberrant site inconducive to growth and development (1). The commonest site is the fallopian tube (95% of cases). None of the many existing theories fully explain the aetiology. Ectopic pregnancy has been said to occur in association with intra-uterine devices, but is often seen in patients without either. Ectopic gestation is common at the Kenyatta National Hospital, an average of 3-4 cases being seen each week. This represents a considerable source of fetal wastage. Maley and Auma, (2) reported the incidence in Nairobi to be 1:132 total births. Ectopic pregnancy is also an emergency situation in which prompt diagnosis and treatment are necessary.

The following case is rather unusual in that the patient had three consecutive ectopic pregnancies over a period of 8 years and still had her right tube intact.

Unusual variations on ectopic pregnancy have been reported before such as the simultaneous occurrence of two tubal ectopic pregnancies, association of a tubal pregnancy with an intra-uterine pregnancy after bilateral tubal ligation (3). Repeated ectopic pregnancy has an incidence of 9 - 11% (1,4,5,6), but the occurrence of three ectopic gestations in the same woman is rare enough to warrant a discussion in a book like this one. Katsee (1) reported a similar case in 1983, and Kemman et al (4) reported a term uterine pregnancy after four successive tubal pregnancies in the same patient, also in 1983.

Our patient probably did have a history of pelvic inflammatory disease, which might have predisposed her to the two earlier ectopic gestations. Preservation of the tube by reconstructive tuboplasty after the second ectopic pregnancy probably predisposed her to the third one. Reconstructive surgery is attempted to enable further attempts at pregnancy; if a second salpingectomy had been performed her prospects of pregnancy would obviously have been nil save for the invitrofertilization and embryo transfer, which, however, is very expensive and not yet available in Kenya. The intense desire of our patient to procreate and the willingness of her physicians to try to aid her in doing so was clearly evident.

Successful intra-uterine pregnancies occurring after conservative tubal surgery have been reported (4,5,6).

Currently there are three major conservative surgical approaches to tubal pregnancies - milking out (fimbrial expression), linear salpingotomy, and tubal resection and anastomosis (4). At this time it is unclear which of these surgical approaches is best, i.e. which would result in the highest rate of subsequent intra-uterine pregnancies. Our patient had cornual wedge resection and later tubal anastomosis after her second ectopic pregnancy, and natural "milking-out" after her third ectopic pregnancy.

Salpingectomy has long been the treatment of choice for tubal pregnancy and still is and will be a major therapeutic approach. However the choices of therapy are becoming most complex, and conservative surgery receiving more attention.

A repeatedly conservative approach to tubal pregnancy is feasible, but has to be restricted to selected patients who are counselled and whose condition is evaluated appropriately. Because of the high risk of recurrent ectopic pregnancy (estimated to be about 25% (4))it has been suggested that any pregnancy subsequent to conservative management has to be considered "extra-uterine until proven other-wise" (4). Thus a patient must be able to live with the threat of a repeated tubal pregnancy. She must be responsible to contact the physician early once a menstrual period is missed and face the need to undergo perhaps extensive testing each time pregnancy is only suspected. Use of a vaginal probe may improve the diagnostic accuracy of the sonographic study in such a case. A recurrent tubal pregnancy of course would lead to further surgery again. Laparoscopy can also be used as both a diagnostic and therapeatic modality in this respect.

The patient described herein illustrated the risks and opportuni-ties in this trend in ectopic pregnancy management. Although conservative surgery for tubal ectopic pregnancies may increase the chances of a repetition, it must be made clear that successful pregnancies do follow in about one-third (1) of these women. It was unfortunate that our patient had an ectopic pregnancy after conservative surgery, but performance of this procedure was better than sacrificing a salvageable tube because of unwillingness to assume reasonable risks.

Former teaching that leaving blood in the peritoneal cavity
to give patients an extra iron source to build-up their haema-
tocrit post-operatively has no place when fertility is a
consideration, as clotted blood is not completely absorbed,
undergoes organization, and adhesions follow (5).

Use of steroids and anti-histamines to prevent adhesion in
ectopic surgery is controversial. In addition use of intra-
peritoneal high molecular weight dextran to reduce adhesion
formation has not been proven efficacious in ectopic surgery (5),
however, it is theoretically sound. There is no evidence to
support the use of post-operative hydrotubation in ectopic
surgery (5,6), in any case it carries with it the risk of
spreading ascending infection.

I conclude by stressing the fact that efforts to conserve the
fallopian tube were entertained because the patient was under 35
years of age with a stable surgical condition; she had no living
child and she was adamant about retaining the option of future
childbearing; at the same time she realized the risks of this
conservative procedure.

REFERENCES:

1. Khatsee M.H.D. One woman, two tubes, three ectopic pregnancies.
    S. Afr. Med. J. 63: 984, 1983.

2. Maly C.A. and Auma S. Ectopic Pregnancy in Nairobi.
    Kenya. Scot. Med. J. 15: 172, 1970.

3. Muhiu G. and Rogo K.O. Ruptured tubal Pregnancy following
    tubal sterilization. East African Med. J. 64: 333, 1987.

4. Kemman E., Grochmal S.A. and Harrigan J.T. Term uterine
    pregnancy after Four Successive Tubal Pregnancies.
    J.A.M.A. 250: 2673, 1983.

5. Weckstein L.N. Current Perspective on Ectopic Pregnancy.
    Obstet. Gynecol. Surv. 40: 259, 1986.

6. Stromme W." Conservative Surgery for Ectopic pregnancy:
    A Twenty-Year Review. Obstet. Gynecol. 41: 215, 1973.

7. Bruhat M.A., Manties H., Mage G., Pouly J.L.
    Treatment of Ectopic Pregnancy by Means of Laparoscopy.
    Fert. Steril. 33:411, 1980.

MISSED ABORTION-SUCCESSFUL INDUCTION OF LABOUR WITH EXTRA-AMNIOTIC NORMAL SALINE AND PGF_{2a}.

Name;	J.N.	Parity:	3+0
Age:	25 years	LMP:	15.3.1989.
Unit No.	993971	EDD:	22.12.1989.
DOA:	29.9.1989.	DOD:	8.10.1989.

PRESENTING HISTORY:

J.N. was referred to us from Pumwani Maternity Hospital with a diagnosis of missed abortion for further management. They did not have facilities for dilatation and evacuation. She initially had slight vaginal bleeding when she presented to that hospital. By the time of referral she already knew she was carrying a dead fetus inside her, and was overtly anxious to have it come out. There was no ownish vaginal discharge.

OBSTETRIC AND GYNAECOLOGICAL HISTORY:

Her menarche was at 14 years, and subsequently had regular periods lasting 4 to 5 days every month. She never used any form of contraception. Para 3+0. All were spontaneous vertex deliveries, and all were alive and well. She had twins in 1986, during her last delivery. Her last normal menstrual period was on 15.3.1989 which lasted for 5 days, giving her expected date of confinement to be 22.12.89. She was therefore at a gestation of 28 weeks on admission. Her ante-natal care was at Kagundo District Hospital. She attended only 3 times. Last visit was on 25.8.89 - at that time the fundal height was 20 weeks but fetal heart beat was not detected. From April, 1989, she used to have slight vaginal bleeding on and off. She never felt any fetal movements. The fetus probably died sometime during August, 1989, but was retained in utero.

She was admitted to Pumwani Maternity Hospital on 14.9.1989, where induction of labour was tried with intravenous syntocinon infusion but failed, and they accordingly referred her to us.

PAST MEDICAL HISTORY:

Her past medical and surgical history was not remarkable.

SOCIAL AND FAMILY HISTORY:

She was a housewife. Her husband was working as a
casual labourer in Nairobi. She neither smoked or took alcohol.
There was no family history of diabetes mellitus or hypertension.

PHYSICAL EXAMINATION:

Her general condition was satisfactory. Not pale, and there was
no jaundice, oedema, dehydration or palpable peripheral lymph
nodes. She was afebrile.   There were no petechiae or echymoses.
Her blood pressure was 100/60 mmHg; temperature was 37°c, pulse
rate was 80 per minute, and respiratory rate was 22 per minute.

Her cardiovascular, pulmonary and central nervous systems were
grossly normal.

ABDOMINAL EXAMINATION:

Uterine size was 16 weeks. It was globular and firm. There was
no tenderness, and no uterine contractions. Fetal heart beat was
not heard. The liver and the spleen were not enlarged.

PELVIC EXAMINATION:

Her external genitalia was normal, as were the introitus and
the vagina. The cervix was long and rigid. The internal os was
tightly closed. The cervix was un-effaced and hard to the feel.
The uterus was bulky (about 16 weeks). Adnexae and the cul-de-sac
were free.

DIAGNOSIS:

A diagnosis of missed abortion was made.

INVESTIGATIONS AND RESULTS:

. Haemoglobin   : 12.3 g/dl.
. Bed side clotting time : 9 minutes.
. Ultrasound (14.9.89) showed a macerated foetus with excessive
  moulding. Fetal length was 2.5 cm, corresponding to 18.9 weeks.

No amniotic fluid was present.

. VDRL : Negative.

. Random blood sugar: 5.8 mmol/l.

. Pregnancy test   : NEGATIVE

She was then prepared for induction of labour with extra-amniotic prostaglandins, after informed consent was obtained.

EXTRA-AMNIOTIC PGF$_{2a}$ AND NORMAL SALINE:

The patient was placed in lithotomy position, and vulvo-vaginal toilet done. She was then draped with sterile surgical towels and bladder aseptically catheterized. Using a sterile Cusco's bivalve speculum, the cervix was exposed. The anterior lip was grasped with a tenaculum. A Folley catheter FG 18, with a balloon capacity of 30 ml, was introduced through the cervix and retained by means of the blown-up balloon, in the extra-amniotic space at the lower pole of the uterus. 10 mg of PGF$_{2a}$ was diluted to 20 ml with normal saline in a clean plastic syringe of 20 cc. 4 ml of this solution was immediately injected into the catheter at 6.45 a.m. on 4.10.1989, to obliterate the dead space. The rest of the solution was given thereafter at the rate of 2 ml hourly until the solution got finished. On 5.10.89, there was no more PGF$_{2a}$, so for the next 24 hours, no prostaglandin was pushed. On 6.10.89, normal saline was placed in the syringe to substitute for PGF$_{2a}$ after an initial 30 ml was placed in the extra-amniotic space. The rest of the solution was pushed as for PGF$_{2a}$ solution. She expelled a severely compressed and un-recognizable product of conception on 7.10.89 at about 2.00 p.m. Introduction to expelsion time was interrupted and therefore difficult to compute. Evacuation was done after-wards using a Karman canula.

MANUAL VACUUM ASPIRATION WITH KARMAN CANULA:

The patient was placed in lithotomy position, vulvo-vaginal toilet done and draped with sterile surgical towels. After introduction of a Cusco's bivalve speculum and insertion of a tenaculum to the anterior lip of the cervix, the cervix was lightly swabbed with savlon solution and a 12 mm Karman Canula inserted. A vacuumed syringe was then connected, and aspiration/ evacuation completed in a "rotatory - in - out" movement. There was only minimal bleeding.

POST-EVACUATION OBSERVATIONS:

Her general condition was fair. She was up and about. Blood pressure was 110/70 mmHg; pulse rate was 80 per minute, respiratory rate was 22 per minute, and her temperature was 36°c.

She was given prophylactic tetracycline 500 mg 6 hourly for 5 days. She was discharged home on 8.10.1989 in a good general condition, for follow-up at Kagundo District Hospital.

DISCUSSION:

Missed abortion refers to prolonged retention of a fetus who died during the first half of pregnancy. The purpose herein is to present a 25 year old patient whose fetus died in-utero for an indefinite period, and was retained. Successful induction of labour was performed with extra-amniotic prostaglandins and physiologic saline. A missed abortion has been defined as the retention of dead products of conception in-utero for 8 weeks or more (1). The rationale for a time period of 8 weeks as the sine qua non for the diagnosis of a missed abortion is not clear. It certainly serves no useful clinical purpose, as the experience these patients undergo is very traumatizing and emotionally trying, and physicians routinely induce labour as soon as the fetal death is diagnosed to avoid such complications like dissemninated intravascular coagulopathy.

The incidence of missed abortion at Kenyatta National Hospital is unknown. Omuga (1989) found abortions to account for 61.4% of all emergency gynaecological admissions at the KNH (2). These were all incomplete abortions.

The patient presented had first the symptoms and signs of a normal pregnancy. Slight uterine bleeding ensued and a diagnosis of threatened abortion was made. The discharge cleared up temporarily and the pregnancy progressed normally until it became clear to her after some weeks that the uterus was not growing; indeed it became smaller and harder. Thus the death of the fetus seemed to have occurred during the early weeks of the second trimester. Sooner the Vaginal haemorrhage recurred when she was admitted to Pumwani Maternity Hospital. Breast signs retrogressed and pregnancy test became negative. The missed abortion was confirmed in her by ultrasound. The fetus, when finally aborted, was shrivelled and severely macerated. The liquor amnii was non-existence and the placenta was very pale and thin almost un-recognizable. An interesting pathological variant of missed abortion is a carneous mole (3). In this condition the primary disturbance is multiple haemorrhages in the choriodecidual space. These are ultimately so numerous or extensive that they kill the fetus, which, if it is small enough, disintegrates and is absorbed, leaving only a space lined by amnion. The extra- chorionic

haematomas bulge into this space as moulds, and together form
a mass of partly organized blood clot and chorion which const-
titutes the mole.

The reason why some abortions do not terminate after death of the
fetus, while others do, is not clear. A generally accepted
explanation of this retention is that normal progesterone
production by the placenta continues for a relatively long time,
although oestrogen production decreases due to fetal death. This
sequence of homonal changes is thought to suppress myometrial
contractility (4). The effect of progesterone is to fix calcium
at its binding sites; release occurs with a decreasing concentra-
tion of progesterone.

In the patient presented termination of pregnancy was effected by
extra-amniotic instillation of prosta glandin F_{2a} and physiologic
saline via a catheter passed through the cervix. As the cervix
was initially un-ripe and inducible, the 10 mg of PGF_{2a} must have
just primed the cervix, and the later instillation of normal
saline via the same route stimulated the uterus to contract.
Sweeping the membranes by physiologic saline solution is a
mechanical procedure known to damage the decidua and to release
endogenous local prostaglandins and seemed to have low frequency
of side effects (4). Within the cells, prostaglandins are thought
to exert their effects by changes in cyclic AMP; release of cal-
cium ions may be important in some target tissues including the
myometrium (5) and thus opposed the inhibitory effect of progeste-
rone.

Our patient did not manifest any excessive bleeding problem during
the evacuation of the uterus. Clotting defects rarely occur in
association with missed abortion during the first half of
pregnancy, but become progressively more likely as pregnancy
advances (3). The defect develops slowly and is not often seen
unless the dead fetus is retained for at least 4 weeks (1).
In such cases products of placental degeneration (probably
thromboplastin) enter the maternal circulation and cause intra-
vascular clotting. This results in hypofibrinogenaemia and an
increase of the fibrinolysins and fibrin degradation products
(FDP) in circulation. This is sometimes manifested by prolonged
and difficult to control, uterine bleeding at the time the

pregnancy is evacuated, due to failure in uterine retraction.
It is the FDP which cause uterine atony (3). When the uterus is
empty, a fibrinogenaemia cures itself spontaneously in 12-14 hours.

The clotting mechanism should be checked periodically after fetal
death has been diagnosed because coagulopathy usually develops
without any evidence of abnormal bleeding. The greater awareness
and earlier diagnosis of missed abortion and prompt evacuation of
the uterus has made it a rare problem nowadays.

REFERENCES:

1. Prichard J.A., MacDonald P.C., Gant N.F. (ed). William's
   Obstetrics. *17th Edition. Appleton - Century - Crofts.*
   Connecticut. 1985.

2. Tindall V.R. Jeffcoate's Principles of Gynaecology. 5th
   Edition. Butterworth. London. 1987.

3. Omuga B.O.O. Presentation of Abortions and its Preventive
   Problems at Kenyatta National Hospital:
   A Prospective Study. M.Med. Thesis. University of Nairobi.
   1989.

4. Gustavi B. Missed Abortion and uterine contractility.
   Am. J. Obstet. Gynecol. 130: 18,1978.

5. Embrey M.P. Prostaglandins in human reproduction.
   Br.Med.J.283: 1563, 1981.

PRIMARY INFERTILITY - INVESTIGATIONS INCLUDING DIAGNOSTIC LAPAROSCOPY .

Name:	S.A.O.	Parity:	0+0
Age:	21 years	LMP:	7.8.1988
Unit No:	863066	Admission:	22.8.1988
		Discharge:	22.8.1988

PRESENTING HISTORY:

Mrs S.A.O. was admitted to the laparoscopy ward on 22.8.1988 for diagnostic laparoscopy. She was first seen in the Gynaecology clinic on 25.1.1988 complaining of inability to conceive since 1984. She was married in 1984 and was staying with her husband.

PAST MEDICAL HISTORY:

Her past medical history was un-remarkable.

OBSTETRICAL AND GYNAECOLOGICAL HISTORY:

Her menarche was at 13 years with regular menses thereafter for 3 days every 26-30 days. Para 0+0. She had not used any contraceptive methods. Her last menstrual period was on 7.8.88 She had no dysmenorrhoea.

SOCIAL AND FAMILY HISTORY:

She was a housewife. Her husband was a driver. They were staying at Dandora Estate in Nairobi. She did not smoke cigarrettes or take alcohol.

She had regular coital relations about 3 times a week. There was no family history of any major illness such as tuberculosis or diabetes mellitus.

PHYSICAL EXAMINATION:

Her general condition was fair and satisfactory. Not pale. There was no jaundice, oedema or palpable peripheral lymph nodes. Her blood pressure was 110/80 mmHg; pulse rate of 80 per minute, regular. Her body temperature was 36.4°c. The cardio-pulmonary and the nervous systems were grossly normal.

ABDOMINAL EXAMINATION:

There was no distension and no previous therapeutic marks.
There was no tenderness. Both the spleen and the liver were
not palpable. No organomegaly was present. Bowel sounds were
normal.

PELVIC EXAMINATION:

The external genitalia was normal as was the introitus and the
vagina. The cervix was long, firm with a closed nulliparous os.
The uterus was of normal size and anteverted. It was only slightly
mobile. There was no adnexal mass or tenderness. There was no
bleeding or abnormal vaginal discharge.

DIAGNOSIS:

A diagnosis of primary infertility was made and she was worked-up
for laparoscopy on 22.8.1988.

INVESTIGATIONS AND RESULTS:

- Haemogram   : 13 g/dl.
- Haematocrit   : 39.3%.
- White blood cell count   : 6.7 x 10^9/l.
- Pap smear   : Class I.
- Serology (VDRL)   : Negative.
- Hysterosalpingography (HSG) on 31.5.88 - showed normal
  uterine cavity. Both tubes were outlined. Left tube was
  patent. Right tube showed terminal dilatation and locu-
  lation due to fimbrial adhesions.
- Semenalysis (23.6.1988): Quantity 1.0 ml; PH = 8.0;
  colour - creamy white, consistency: viscid; spermatic
  count - 72 million/ml; morphology - most of them showed
  normal morphology; motility > 60% at 1 hour, 40% still
  motile after 2½ hours at room temperature; smear study -
  no pus cells. These findings indicated a normal semen
  examination.

DIAGNOSTIC LAPAROSCOPY:

This was done on 22.8.1988 under pethidine 100 mg, valium 20 mg
and local anaesthesia; after obtaining consent from her.

She was placed in semi-dorsal lithotomy position with the legs
abducted and supported on padded stirrups. Vulvo-vaginal toilet
was done, the patient draped with sterile surgical towels, and
bladder catheterization done. Pelvic examination confirmed the
earlier findings. A Sim's speculum was introduced, and the
cervix was exposed. A volsellum forceps was then applied to the
anterior lip, and uterine elevator inform of a canular inserted
into the cervix and secured in place. This canula was left in
for both manipulation of the uterus and the tubes. The patient
was then placed in the supine position with approximately 5 to
$10°$ Trendelenburg, later increased to as much as $30°$ to $40°$ to
allow full visualization of the pelvis. The abdomen was surgicall-
y prepared and draped as if for laparotomy. A small puncture
wound was made at the sub-umbilical region and the Verres neddle
inserted into the peritoneal cavity. The intraperitoneal place-
ment of this pneumoperitoneum needle was tested by the saline test.
The gas source was then attached and the abdomen was insufflated
until it was clinically protuberant and tympanic to percussion.
After pneumoperitoneum was created with carbondioxide gas, a
trocar and canula were inserted through the above incision in
the sub-umbilical region. The trocar was removed and the
laparoscope passed through the trocar sleeve, which was fitted
with a valve to prevent escape of gas from the peritoneal cavity;
the fibre-optic cable was attached and the pelvic organs were
examined systematically. The patency of the tubes was assessed
by hydrotubation using methylene blue. At the end of the procedure,
the laparoscope was removed and the gas within the abdomen allowed
to escape before the skin was closed in a single sub-cuticular
layer.

LAPAROSCOPIC FINDINGS:

The uterus appeared normal in size, but was tethered down by
adhesions in the cul-de-sac. There were no endometriotic
deposits; there were multiple filmy tubotubal adhesions, tubo-
uterine adhesions, tubo-ovarian adhesions etc; the left tube appea-
red normal however, with its fimbriae; the right tube was slightly
distended at its terminal end; About 30% of both ovaries was
buried within these filmy adhesions - they were however normal
in size. There was no evidence of recent ovulation, but scarring

was present on both ovaries. On hydrotubation, there was both
filling and spilling of dye on both tubes.

The conclusion of the laparoscopy was that the patient was to be
given 6 months before any operation could be performed.

DILATATION AND CURETTAGE:

This was performed at the same sitting. The cervical os was
dilated upto Hegars 6 mm dilator. Sharp uterine curettage
revealed healthy looking endometrial tissue.

The patient was discharged home the same day after resting in the
laparoscopy ward for 4 hours. She was advised to come back after
7 days for removal of stitches.

FOLLOW-UP:

She was seen at the Gynaecology clinic on 28.11.88. She had
no complaints. Histology report of secretory endometrium
had been received. No tubercle bacilli had been cultured.

The findings at laparoscopy were discussed with the patient.
she was advised to come back after 6 months for further follow-
up and for possible salpingolysis if pregnancy had not occured
by then.

At the time of writing, pregnancy had not yet occurred, and she
was already booked for laparotomy and salpingolysis.

DISCUSSION:

Throughout the centuries there have been couples who are unable to produce children, and the inability to conceive and bear children represents a major social problem in the sub-saharan Africa, and Kenya is no exception. The purpose herein is to present a young female aged 21 years who came to us because of primary infertility most probably as a result of pelvic inflamatory disease (PID). She had been married for four years.

Reproductive function is the last body function established and the first body function to fail with advancing age (1). It is especially frustrating for otherwise young healthy men and women to find themselves unable to have children, although barren marriage is as old as mankind.

Infertility is a couple's problem. Of the causes of infertility, 40% can be attributed to the woman, 40% can be attributed to the man, and 20% can be attributed to both partners (1). Investigations of the infertile couple is therefore directed toward establishing the cause (s) of infertility in both partners, and achieving pregnancy. Infertility is defined as the inability to conceive after one year of unprotected sexual intercourse.

Kenya has one of the highest birth rates in the world. Ironically it also has a high infertility rate which is a major public health concern. Although the exact statistics for the whole of Kenya are not known, the magnitude of the problem can be understood by the fact that approximately 60% of all new out-patients at the

Gynaecology clinic of the Kenyatta National Hospital complain of infertility (2), and nearly two - thirds of clinical time is spent seeing such cases (3). It is also usually stated that 10 - 15% of all marriages are infertile.

In Kenya, as in most developing countries, pelvic infections due to sexually transmitted diseases and other infections accounts for most cases of female infertility. This was clearly evident in the case under discussion. Mati et al (2), while investigating primary infertility in this hospital, found that the main cause for this was tubal occlusion following P.I.D. Subsequently. Walton and Mati reveiwing 98 patients with secondary infertility

also in this hospital, found a similar percentage (73%) in that
group showing the same problem of tubal occlusion following
P.I.D. (6).

For the male partner semen analysis remains the major test for
evaluating infertility. In the presented case, this test was
normal. The specimen of semen was produced by masturbation into
a glass vessel (as plastic kills sperms) after a 3 -day abstine-
nce period, and was submitted to the laboratory within one hour
for immediate analysis. In the presence of such a normal semen
analysis, other abnormalities of the man were probably inconse-
quential and no further evaluation was therefore necessary.

For the woman the investigations are many, time consuming and
expensive, but should be restricted to the simplest and cheapest
that will give information on ovulation, tubal patency and
operability.

At the KNH, we rely mainly on thorough history, physical examina-
tion, hysterosalpingography, laparoscopy and diagnostic
currettage to give us the necessary information required.

Since the introduction of the laparoscope at the KNH, it has
been widely used to examine the pelvic organs. Infertility is
the main indication for its use. Laparoscopy gives an excellent
view of the pelvic organs, the posterior surface of the uterus
and the cul-de-sac. Many pelvic pathologies such as pelvic
adhesions which may escape clinical detection can easily be
diagnosed by laparoscopy (4). Laparoscopy was utilized as the
final step in the infertility investigation of this patient -
in the follicular phase of her menstrual cycle. Diagnostic
currettage was done at the same time and biopsy obtained to
ensure the absence of an endometrial lesion such as unsuspected
pelvic tuberculosis, and to ascertain the endometrial response
in the luteal phase.

The importance of diagnostic laparoscopy as an integral part of
a proper infertility work-up has been stressed previously (5).
For accurate tubal evaluation and over-all pelvic assessment,
laparoscopy is superior to hystero-salpingogram (HSG). In

addition to its value in diagnosing unsuspected pelvic pathology, laparoscopy is an essential step when conservative infertility operation is contemplated. The laparoscope can reveal a normal p-elvis, thus eliminating the need for further operation. With the laparoscope, the gynaecologist can begin to define and categorise the degree of pelvic disease and distortion. In some instances the operation can be deferred, and the medical work-up and/or therapy can be continued on a more solid basis when an accurate laparoscopic view of the pelvis is obtained.

In our patient, laparoscopy revealed peritubal adhesive disease. Peritubal adhesions result from inflammatory reactions that compromise the tube from outside and although the lumen appeared patent, motility of the fimbriae might have been impaired. Thus a patent tube does not necessarily mean a functioning tube.

Laparoscopy, however, can be an operative instrument in the pre-ence of minimal pathology, and it permits an "educated delay" in the infertility operation with the possibility that a pregna-cy may ensure (4).

Mati et al (3) established the following criteria for choice of patients for tubal surgery: minimal involvement of the tubes with no masses; few peritubal occlusion as demonstrated by dye filling up the tube. Our patient satisfied the first two, and was already planned for lysis of adhesions at the time of writing. However, the results of tubal surgery are poor, and vary enormously depen-ding mainly on the type of the operation and the experience of the surgeon. One may achieve a patent tube but not necessarily an intra-uterine pregnancy. The chances of successful treatment are highest in cases with peritubal adhesions from previous peri-tonitis not related to any tubal pathology; but the chances are lowest following salpingitis - the tubal epithelium having been irreversibly damaged (5).

From the findings of this patient, it is obvious that our empha-sis in the management of infertility should be on prevention of pelvic sepsis. Cases of sexually transmitted disease should be identified and adequately treated in their initial stages.

REFERENCES:

1. Rivlin M.E., Morrison J.C. and Bates G.W. Manual of clinical problems in Obstetrics and Gynaecology. First Edition. Little, Brown and Company. Boston. 1982.

2. Mathews T., Mati J.K.G., Fomulu J.N. A study of infertility in Kenya: Results of Investigations of the infertile couple in Nairobi. East Afr. Med. J. 58: 288, 1981.

3. Mati J.K.G., Anderson G.E., Carty M.J., McGlashan H.E. A second look into the problem of primary infertility in Kenya. East Afr. Med. J. 50: 94, 1973.

4. Israel R. and March C.M. Diagnostic Laparoscopy : A prognostic aid in the surgical management of infertility. Am. J. Obstet. Gynecol. 125: 969, 1976.

5. Brozens I., Boeck W, Delattin Ph., Puttemans P. and Vasquez G. Salpingoscopy: a new pre-operative diagnostic tool in tubal infertility. Br. J. Obstet. Gynecol. 94: 768, 1987.

6. Walton S.M. and Mati J.K.G. An evaluation of secondary infertility in Kenya. East. Afr. Med. J. 53: 310, 1976.

LAPAROSCOPIC REMOVAL OF A TRANSLOCATED INTRA-UTERINE CONTRACE-
PTIVE DEVICE (IUCD):

Name:	M.A.	Parity:	4+0
Age:	31 years	LMP:	None since her last delivery.
Ip. No.	839291	Admission:	9.8.1988
		Discharge:	9.8.1988

PRESENTING HISTORY:

Mrs M.A. was admitted to the Laparoscopy ward on 9th August, 1988
from the Gynaecology clinic with a diagnosis of a translocated
IUCD. She was for laparoscopic removal of this IUCD. This
coil was inserted in November, 1986, but the strings got missing
sometime in June, 1987. She did not have any other complaints
apart from this "missing IUCD strings".

OBSTETRIC AND GYNAECOLOGICAL HISTORY :

She had her menarche at 16 years of age and subsequently had

had regular periods lasting 3 - 5 days every 26-30 days. She
had not yet resumed her periods since her last delivery. She
was para 4+0. Her first delivery was in 1983, second in 1985,
the third in 1986, and the last delivery occurred on 16th May,
1988. All were spontaneous vertex deliveries. The last pregna-
ncy occurred when the IUCD was already translocated.

PAST MEDICAL AND SURGICAL HISTORY:

She developed pregnancy induced hypertension in 1983 during her
first pregnancy, which subsided soon after delivery. This high
blood pressure had not recured since then.

She had had attempted removal of this IUCD by dilatation and curr-
ettage in theatre under intravenous pethidine 100 mg and
diazeparn 20 mg, on two occasions but were unsuccessful. On
both occasions the uterine cavity was found to be normal in
size, and the cervix was dilated to a size of Hegar's dilator
No.7 and 8 respectively. Uterine currettage combined with the
use of ovum forceps failed to retrieve this IUCD.

FAMILY AND SOCIAL HISTORY:

She was a married lady, and worked as a secretary with the
Kenya Railways Co-operation. Her husband was an engineer, and
also worked with the same Kenya Railways. Both of them were
staying at Valley Road in Nairobi. They had had four children
and all were alive and well. She neither smoked any cigarrettes
nor took any alcoholic drinks.

There was no major illness in the family.

PHYSICAL EXAMINATION:

Her general condition was good. Clinically she was not pale and
not jaundiced. She was well hydrated. No oedema was present.
There were no palpable peripheral lymph nodes. Her blood pressure
was 130/60 mmHg; the pulse rate was regular at 80 per minute, and
the respiratory rate was 18 per minute.

The cardio-pulmonary and central nervous systems were grossly
normal.

ABDOMINAL EXAMINATION:

The abdomen was scaphoid and was moving with respiration. There
were no previous surgical scars. There was no tenderness and no
organomegaly. The uterus was well involuted. Both the liver and
the spleen were not palpable.

PELVIC EXAMINATION:

The external genitalia was normal as was the introitus and the
vagina. The cervical os was parous with a transverse slit. It
was closed. There was no IUCD string. The uterus was normal in
size and shape. The IUCD was palpated to the right of the
posterior fornix in the posterior cul-de-sac. It appeared embedded
to the uterine wall.

DIAGNOSIS:

A diagnosis of a translocated IUCD was made and the patient prepa-
red for laparoscopic removal under local anaesthesia systemic
analgesia.

INVESTIGATIONS AND REPORTS:

. Haemoglobin   : 12.3 g/dl.
. Haematocrit   : 36.2%
. Pelvic Ultrasound   : Uterus had prominent endometrial
                           reaction. The IUCD was seen to be
                           embedded in the myometrium posteriorly.

LAPAROSCOPIC REMOVAL:

The patient arrived to the Laparoscopy ward at 03.00 hours having
starved since mid-night (she had had no solid food for at least
8 hours, and no liquids for more than 6 hours pre-operatively).
Careful explanation of the contemplated procedure was given to
her. The intended operation site was shaved, and consent for the
surgical procedure obtained. She was then asked to empty her
bladder and she was pre-medicated with intramuscular 0.6 mg atro-
pine 30 minutes before the scheduled operation, and she was taken
to theatre. The rest of the procedure was as described under
"Primary Infertility - investigations including laparoscopy".

OTHER LAPAROSCOPIC FINDINGS:

A clear view of the pelvis was obtained. No adhesions were
found, and there was no fluid within the pelvis. The uterus,
the fallopian tubes and both ovaries were seen and were grossly
normal. The IUCD (Nova - T) was seen partly in the right adnexum
posteriorly and the posterior cul-de-sac. It was partly embedded
within the uterine wall. It was not attached to any extra-uterine
structures.

It was caught by the tongs and partly withdrawn into the appli-
cator and removed along with the scope. The scope was re-inserted
and the area visualised to rule out any bleeding. The procedure
was completed after visualizing the other parts of the pelvis
including the adnexa. The abdomen was then compressed to remove
the gas. The incision was closed in one layer, and haemostasis
was adequately achieved.

POST OPERATIVE CARE:

The patient recovered fully from the local anaesthesia and the

systemic analgesia, and was discharged home after three hours of
bed rest in the laparoscopy ward. She was reviewed one week
later, and the wound was found to be well healed.

Both the patient and her husband declined sterilization procedure,
and preferred abstinence and natural methods of family planning
instead, at least for the time being. She was discharged for
follow-up at the family welfare clinic.

DISCUSSION:

The patient presented had intra-uterine contraceptive device
(Nova - T) inserted in November, 1986 following the birth of
her third child the same year. This intra-uterine contraceptive
device (IUCD) got translocated sometime in June, 1987. In the
interval between translocation and removal of this IUCD she had
one pregnancy which progressed to term uneventfully. (Nova - T
contains 200 mm^2 copper wire with a silver core wrapped around
the stem. It also contains polyethylene with barium sulphate
added for visibility on X-rays).

The advantages and adverse effects of IUCDs have concerned
physicians since their original introduction as a method of contra-
ception in 1909 (1). Perforation of the uterine wall is one of
the less common but more serious complications associated with
the IUCD (2). The reported incidence is between 0 and 8.7 per
1000 insertions (1 - 5) but the approximate figure of 0.5 to1
per 1000 is more generally accepted (2). The incidence seems
to be influenced by many factors including the type and size of
the device, the method of insertion, the position of the uterus
the proximity of pregnancy to insertion time, and the skill and
experience of the person performing the insertion (1,2). The
incidence of missing strings is much higher (4).

Synonyms of complete perforation are translocation, migration,
extrusion, wandering loop, ectopic device, ectopic placement,
intraperitoneal location and extra-uterine misplacement; the
synonym of partial is incomplete perforation (3). Occasionally,
as was in our patient, a translocated IUCD may lie in the peritonea-
cavity (including the posterior cul-de-sac) except for its "tip"
which was still within the myometrium for a distance of a milli-
metre or two. This may, for all practical purposes, nevertheless
be considered translocated, since removal by laparoscopy was
easily effected without transerosal incision into the myometrium
to liberate the imprisoned "tip".

Attention was directed to the location of the IUCD in the posterior
cul-de-sac in our patient. This observation supported the fact
that perforation tends to occur more frequently through the
posterior wall of the uterus than the anterior wall.

It is thought that most IUCD perforations occur at the time of insertion - immediate or primary perforation (1,3,4). Secondary perforation occurs when the IUCD has been partially embedded in the uterine wall at insertion, and with time erodes through the wall owing to uterine action (4). Our patient had secondary perforation.

Perforations probably occur more often with the "push-out" rather than the "withdrawal" technique of insertion (1,3,4). Persons with little experience in the insertion of the IUCD or who neglect to use a forceps for straightening and stabilizing the uterus and a sound for determining uterine depth and direction before inser-ion of an IUCD in a patient before the 12th week post-partum and especially from the 4 - 8th week leads to an incidence of perfora-tion four times greater than in the non-postpartal patient (1).

Tail retraction into the uterine cavity probably occurs when the device rotates under the forces of uterine action or when it becomes embedded in the uterine wall. It may also occur when pregnancy supervenes or when the tails are cut too short at the original insertion (4).

The perforated IUCD is most often asymptomatic (except for the rare complications, particularly those involving the intestinal tract etc), and was suspected in our patient by the disappearance of the strings discovered by the patient herself. She did not have any abdominal pain or vaginal bleeding.

The diagnosis of translocated IUCD in our patient was established by bimanual examination (and palpating the IUCD in the posterior cul-de-sac) and by ultrasound. Other procedures such as hystero-scopy, hysterosalpingography, radiography of the pelvis following insertion of a uterine sound or a second IUCD, insertion of a Foley catheter in the urinary bladder and filling of the balloon with radio-opaque material, pelvic pneumography, or fluoroscopy may all provide valuable information, but these are involved procedures and not necessary in most instances (4). Hysteroscopy is a relatively new technique not yet available in our unit. It has already shown itself of great utility in the visualization and removal of the intra-uterine IUCD when, because of missing string or other reason, its presence is uncertain (3).

Dilatation and currettage is a "blind" procedure which was not successful in our case on two occasions. If an IUCD is palpable in the cu-de-sac it can be removed by colpotomy (1). This was not attempted in our case and as part of the IUCD was still within the uterine wall.

The efficacy of laparoscopy as a method for removing translocated devices from the peritoneal cavity is well established (2), and is further confirmed in this patient. The feasibility of IUCD retrieval via the laparoscope depends not only on the ability of the laparoscopist to spot the device within the peritoneal cavity, but also on the degree of attachment of the device to the intrape-ritoneal structures, particularly vascular and intestinal (2). Previous reports on copper-containing IUCD recovery after uterine perforation have suggested laparotomy was necessary: they are usually not found free in the peritoneal cavity. Although laparoscopic removal of copper-containing devices from the perito-neal cavity is not as successful as removal of inert plastic IUCD, the fact that our patient was relieved of her iatrogenic burden and saved the further insult of a laparotomy would indicate that attempted retrieval with the laparoscope appears worthwhile. With 87% of perforations arising from coils inserted in the post-partum or post-abortion period, the increased risk of uterine perforations after pregnancy supports the observations of Ansarii (1974).

To facilitate laparoscopic removal of perforated IUCDs early intervention is desirable, whenever possible, prior to formation of intestinal adhesions (3). Laparoscopic removal of transloca-ted IUCDs has the additional advantage of permitting concomitant procedures, particularly tubal sterilization.

When uterine perforation occurs, copper contained in the devices may produce severe omental and peritoneal reactions. For this reason it is recommended that all translocated IUCDs be removed as soon as perforation is recognized. In a case of missing strings it is best to remove and replace the device, because there is no certainty whether the IUCD is in-situ or has been expelled, putting the woman at risk of pregnancy.

REFERENCES:

1. Gentle G.P. and Siegler M. The misplaced or missing intra-uterine device. Obstet. Gynecol. Survey. 32: 627, 1977.

2. McKenna P.J. and Mylotte M.J. Laparoscopic removal of trans-located intra-uterine devices. Br. J. Obstet. Gynecol. 89: 163, 1982.

3. Zakin D., Stern W.Z., Rosenblatt R., Complete and Partial Perforations and Embedding following insertion of intra-uterine devices: I. classification, complications, mechanism, Incidence and Missing strings. Obstet. Gynecol. Survey, 36: 335, 1981. II. Diagnostic Methods, Prevention and Management. Obstet. Gynecol. Survey, 36: 401, 1981.

4. Ismail R.A. Translocated IUCD and Missing strings. S. Afr. Med. J. 52: 233, 1977.

5. Zakin D. Perforation of the bladder by the intra-uterine device. Obstet. Gynaecol. 39: 59, 1984.

6. Ansarii. Quoted by 2.

GENITAL PROLAPSE IN AN ELDERLY NULLIPARA-VAGINAL HYSTERECTOMY AND PELVIC FLOOR REPAIR.

Name:	M.A.	LMP:	4 years ago
Unit No.	864050	Date of Admission:	25.11.1987
Parity:	0+0	Date of Discharge:	25. 4.1988
Age:	58 years.		

PRESENTING HISTORY:

Mrs. M.A. was referred from New Nyanza General Hospital (Kisumu) with a three months history of a mass coming out through her vagina. She had been an asthmatic since 1974, and was regularly controlled on various bronchodilators. Vaginal examination done at that hospital showed uterine prolapse grade three (procidentia). There was no ulceration of the cervix. She had been reviewed by the anaesthetist and found to be unfit for general anaesthesia. There were no instruments for spinal anaesthesia. So she was referred to us for further management.

Apart from the feeling of a "bulging in the vagina and perineum", she denied any history of urinary stress incontinence, frequency or retention of urine.

PAST MEDICAL HISTORY:

She was an old asthmatic patient, who had been on various broncho-dilators including franol, ventolin, aminophylline and occasional steroids during severe attacks since 1974. She continued, however getting acute asthmatic attacks on and off.

PAST OBSTETRICAL AND GYNAECOLOGICAL HISTORY:

She had her menarche at 14 years of age and subsequently she had regular periods every month till 1983, when she had her last menstrual period. She was para 0+0. She was married at the age of 18 years. She had not gone to any hospital for the investigation of her infertility, and she had never used any contraceptive methods.

FAMILY AND SOCIAL HISTORY:

She was a housewife. Her husband was a peasant farmer at home.
Her husband had another wife with whom they had six children.
The patient was educated upto standard two only. She did not
smoke or did she take alcohol.

PHYSICAL EXAMINATION:

Her general condition was fair and satisfactory. She was an
elderly woman already with grey hair. She was not pale, not
jaundiced and not dehydrated. No oedema was present. There
were no palpable peripheral lymph nodes. Her blood pressure was
120/80 mmHg; pulse rate was 80 per minute and regular and the
respiratory rate was 24 per minute.

The cardiovascular and the central nervous systems were essentially
normal. Clinically there was no evidence of spina bifida occulta.

CHEST EXAMINATION:

Her chest was symmetrical and moving with respiration. Trachea
was central. Air entry was equal on both the right and left sides.
There was no hyper-resonant state of the chest. There were
bilateral occasional rhonchi, but no crepitations.

ABDOMINAL EXAMINATION:

The abdomen was scaphoid. There were no therapeutic marks or
organomegaly. The uterus was not palpable par abdomen. There
were no areas of tenderness, or abnormal swellings.

PELVIC EXAMINATION:

The external genitalia was normal. The vagina was everted. The
cervix was completely outside the vulva, and the slightly enlarged
uterus filling the introitus, but was reducible. There were both
cystocele and rectocele evidenced on straining. The cervix was
however, not ulcerated, nor were there any perineal ulcerations.
The cervix was about 2cm in length with a circular os-typical of
a nulliparous cervix. Vaginal walls were loose. There was no
stress urinary incontinence.

DIAGNOSIS:

A diagnosis of third degree utero-vaginal (genital) prolapse was made, in an old female patient, post-menopausal, who also had chronic bronchial asthma.

INVESTIGATIONS AND RESULTS:

. Haemoglobin   : 15.3 g/dl.
. Platelet count   : 353 x 10^9/l.
. Serum chemistry Na^+ : 141 mmol/l..
                  K^+ : 4.3 mmol/l.
                  BUN   : 4.4 mmol/l.
         Creatinine   : 92 mmol/l.
. M.S.S.U.   : No growth.
. Pap Smear   : CLASS I.

MANAGEMENT:

Both the physician and the anaesthetist were consulted for the adequate control of her asthma and the method of anaesthesia at operation respectively. The physician advised ventolin 8 mg tds, prednisone 10 mg BD and septrin two BD. On 29.1.1988, she was put on Becotide 2 puffs QID and Bricanyl inhaler tds and PRN because her asthmatic attacks had become rather very frequent. The anaesthetist reviewed her and suggested either regional or epidural anaesthesia for vaginal hysterectomy.

She was prepared for the operation adequately. Informed consent was obtained, and she signed the relevant witnessed consent forms.

OPERATION ON 22.4.1988:

Vaginal hysterectomy plus anterior and posterior colporrhaphy were done on 22.4.1988 under epidural anaesthesia. With the patient in lithotomy position, and epidural anaesthesia already given, vulvo-vaginal toilet was done. She was then draped and an indwelling Folley's Catheter F16 put into the urinary bladder. Examination under anaesthesia then completed - this confirmed the earlier findings.

The first incision which was made was racquet-shaped, encompassing the cervix and extending some little way down in the anterior

vaginal wall in the mid-line. The mucosa was reflected and the peritoneal cavity then opened in-front of and behind the cervix (entering the vesico-uterine sac first). The cardinal ligaments were then clamped, cut and ligated, followed by clamping, division and ligation of the uterine vessels bilaterally. After this it was abit difficult to bring down the uterus because of a few subserous fibroids, but with alittle manipulation, the upper broad ligaments, the round ligament, the fallopian tubes and the ligament of the ovary were clamped, divided and ligated. The uterus was then removed leaving the ovaries behind.

<u>Closure of the Vault</u>: A suture was passed through the vaginal mucosa and the uterosacral ligaments, taking up the peritoneum of the cul-de-sac and the fascia that lies external to it and the overlying vaginal mucosa, and then taking up the tissues, on the contralateral side of the pelvis in a reciprocal fashion. The peritoneal cavity was closed by a purse-string suture taking up the round ligaments, the peritoneum of the base of the bladder, the utero-sacral ligaments, and all of the peritoneum of the cul-de-sac. The mucosa of the open vault was closed by a series of continuous, knotted vertical mattress sutures. All the dead space was carefully obliterated, and the wound was completely closed. The first two stitches on the contralateral sides were then tensed and securely tied (to elevate the cul-de-sac to high level).

Both anterior colporrhaphy and the posterior colpoperineorrhaphy were then performed. Both anterior and posterior vaginal walls were divided longitudinally, and carefully disected from the bladder and the rectum in an inverted V-shaped fashion respectively - the apex being near the cervix, and the base inferiorly to the perineum.

The redundant vaginal walls were excised off, and the remaining edges apposed to complete the vaginal repair. A vaginal pack was then left in-situ. The bladder was drained by the indwelling catheter which was put earlier.

POST OPERATIVE CARE:

Routine post-operative care was instituted. She was given analgesia in form of pethidine 100 mg intramuscularly every 8 hours for 48

hours and then put on paracetamol tablets. She was prescribed prophylactic tetracycline for 10 days. The patient was encouraged to get out of bed the next day, and physiotherapy was commenced from the outset. The vaginal pack was removed after 24 hours, and the catheter came out on the third post-operative day. She did not require any blood transfusion as the intra-operative blood loss was minimal. She was able to go home on the fifth post-operative day with no complaints at all, to be followed up at the New Nyanza General Hospital.

DISCUSSION:

The patient presented was an elderly nullipara, but had third
degree genital prolapse. Genital prolapse is a common gynaecologic-
al problem in developed countries (1,2). Cox and Webster (1) report-
ing on genital prolapse amongst the West Pokot tribes of Kenya had
noted that it was relatively uncommon in East Africa as compared
with similar age groups in Europe and the USA. Mwalali (1982) found
an incidence of genital prolapse at the Kenyatta National Hospital
to be 0.10% (3).

Uterine prolapse implies the caudal descent of the uterus from
its normal position in the pelvic floor (4), and is in the nature
of an intussusception. The degree of descent of the uterus
varies and is described as first degree if the cervix reaches the
introitus; second degree if the cervix protrudes through the
introitus; or third degree if the entire uterus protrudes. One
or more of the forms of uterovaginal prolapse may exist in the
individual patient. Extroversion of the vagina with uterine
procidentia is the end stage of all types of genital prolapse.

There are two possible causes of genital prolapse. First, it may
be caused by the supporting structures, where the pelvic diaphragm
have failed to hold the uterus in the correct position. Second,
it may be caused by weakening and stretching of the pelvic
connective tissue, in the cardinal and sacro-uterine ligaments (4).

The aetiological factors in prolapse of the uterus usually have
their origin in the stresses incident to chilbearing (1,2,5). U-
terine prolapse usually occurs in younger middle-aged and older
women who have had children by vaginal deliveries, and it presents
as a gradually developing vulvo-vaginal discomfort. A congenita-
lly deep pouch of Douglas may be a predisposing factor.

Genital prolapse in a nulliparous woman, as occurred in our patient
is an uncommon yet definite entity (5). The patient presented was
an elderly (50 years old) nullipara, who was also post-menopausal.
Lack of oestrogens in the menopause aggravates uterine prolapse
(6). Jacoby BE (5) reported 19 cases in nulliparous patients.

Associated with the prolapse is a relaxation of the pelvic suppo-
rting structures. Predisposing factors vary, and there seems to
be an indication that disturbances with secondary muscular
insufficiency of the pelvic floor, atrophy of the ligaments,
connective tissue, and muscles contributed to the appearance of
the uterine prolapse in the nullipara. One predisposition to
prolapse is congenital, and in these cases where a congenital
retroflexion or retroversion exists, subsequent trauma, poor
nutrition in early life, faulty innervation, or increased intra-
abdominal pressure may be the exciting cause (5). Our patient
had had chronic bronchial asthma for more than 10 years. This
could have contributed to the development of uterine prolapse in
her case. There is an element of infantilism in many cases, and
the fact that these women are infertile is highly suggestive (5).
Spina bifida occulta should be considered as a possible predis-
posing factor in uterine prolapse in nulliparous women (1,5).
Clinically our patient did not have spina bifida occulta nor
varicosities. Old age has no monopoly of genital prolapse althou-
gh it has an interest in it (6).

In the patient presented, the uterus was retroverted. Only when
the uterus is axial to the vagina can descent along the vagina
occur, and this is more easily achieved from the retroverted posi-
tion than from acute anteversion and anteflexion. The supravagin-
al cervix in our patient was not elongated. Enlargement of the
cervix due to gradual prolongation, oedema, and gradual hypertro-
phy is a well recognized association of genital prolapse.

There are two main treatment methods for uterine prolapse. One is
conservative, mainly using a plastic pessary, and the other is to
operate. Age, general physical condition and preservation of the
childbearing functions are all important factors to consider when
deciding the proper method. Whilst a patient is waiting for her
operation, she may temporarily be relieved of her symptoms by
wearing a plastic ring pessary. However, this method should not
be offered as a definite long-term treatment, because, as she
grows older her levator ani muscle tone will diminish and she
will ultimately be unable to retain the pessary (6). It is
suitable for pregnant women suffering from prolapse, but the
pessary should be removed before the delivery. Such a patient
may not then be bothered by the prolapse for some several years,

but she will ultimately need a repair. The patient who is so
old, unfit and unwell that an anaesthetic and operation would
be hazardous, should be fitted with the plastic ring pessary (6).

Another indication for the temporary use of a pessary is
puerperal prolapse. Operation should not be performed for at
least six months after delivery while involution occurs and
pessary support during this time can relieve symptoms and allow
healing to restore the pelvis to normal (6). This conservative
method, especially, use of a pessary, was not selected for our
patient because its chronic irritation may lead to carcinoma of
the cervix, carcinoma of the vagina or even vesico-vaginal fistul-
ae. In any case the use of a pessary is rarely practised in our
unit.

Operative methods used are vaginal hysterectomy with anterior and/
or posterior colporrhaphy, Manchester operation, Le Fort operation
etc. Selection depends on factors such as age, genital state and
physical condition, preservation of the childbearing function, and
sexual activity (5). We managed to do a successful vaginal hyste-
rectomy plus vaginal repair under epidural anaesthesia in our
patient. The success of vaginal hysterectomy with repair lies on
the ability to shorten and fix the stumps of the cardinal ligamen-
nts to the vaginal vault, and at the same time to use the stumps
of the round ligaments for additional vault support.

No serious complications such as post-operative bleeding, haemato-
ma formation, infection or abscess, were seen in our case. There
was no urinary tract infection, pyrexia, thrombosis or emboli.
Residual urine did not occur in the patient presented.

The rate of recurrence is lower for the vaginal hysterectomy than
such other operative methods as colporrhaphy with cervical ampu-
tation, Manchester operation and the Le Fort operation (4,5).
The latter operative procedure is not performed in our unit.

Surgery remains the definitive form of treatment for all but the
most fragile patient. Modern anaesthetic techniques and the
advent of epidural anaesthesia mean that there are very few
patients with genital prolapse in whom surgical repair is
contraindicated.

REFERENCES:

1. Cox PSV and Webster D. Genital prolapse amongst the Pokot.
    E. Afr. Med. J. 52: 694, 1975.

2. Otubu JA and Ezen BU. Genital prolapse in the Hausa/Fulani
    of Northern Nigeria. E. Afr. Med. J. 59: 605, 1982.

3. Mwalali. A Retrospective study of the Genital prolapse at
    Kenyatta National Hospital . M. Med. Thesis. University of
    Nairobi. 1982.

4. Tanaka S., Yamamoto H., Shimato S., Endoli T. and Hashimoto M.
    A vaginal approach to the Treatment of Genital Prolapse.
    Asia-Ocean J. Obstet. Gynecol. 14: 161, 1988.

5. Jacoby BE. Prolapus Uteri in Nullipara.
    Am J. Obstet. Gynecol. 57: 757, 1949.

6. Stallworthy JA. Prolapse. Aetiology, Diagnosis and
    Treatment (in two parts) Br. Med. J. 1: 499 and 539,1971.

URETERO – UTERINE FISTULA WITH PARADOXICAL URINARY INCONTINENCE FOLLOWING CAESEREAN SECTION – BOARI – BLADDER FLAP PROCEDURE.

Name: J.A.O.	**Parity:**	3+1
Age: 29 years	**Last delivery**	25/1/1989
Unit No: 942147	**Admission:**	10/3/1989
	Discharge:	24/3/1989

PRESENTING HISTORY:

Mrs. J.A.O. was admitted to the ward with a history of urinary incontinence and lower abdominal pains on 10.3.89. She had had these symptoms since 5.2.89. There was no fever or chills. Leakage of urine had started 10 days post emergency caesarean section (C.S). She was, however, still able to pass some urine par urethra although she could not feel her urinary bladder full. Lower uterine segment caesarean section (LUSCS) was done on her on 25.1.89 because of pregnancy induced hypertension (PIH) and persistent occipitoposterior position (POPP) after failed induction of labour. At the operation, the uterus was found to be slightly dextrorotated, and the baby to be at the mid-cavity in occipito-posterior position. During the attempt to extract the head, there was an extension of the LUSCS incision on the left side towards the cervix, and profuse bleeding ensued, necessitating summoning of assistance. At the attempts to control the haemorrhage, the left ureter was accidentally ligated together with the bleeders. This accident on the ureter was however, not discovered at the time, or during the seven days she was in the lying-in ward. Intra-operatively she had cardio-pulmonary arrest but was successfully resuscitated. She did not develop any fever, rigours or features of ileus.

PAST MEDICAL HISTORY:

Her past medical and surgical history was un-remarkable.

PAST GYNAECOLOGICAL HISTORY:

Her menarche was at 17 years and subsequently had regular cycles lasting 4 days every 28 days. She did not use any contraceptive method.

PAST OBSTETRICAL HISTORY:

Para 3+0. Her first delivery was in 1978 at Pumwani Maternity
Hospital, at 30 weeks gestation to a male baby weighing 1.5 kg.
The second delivery was at Mater Hospital at 32 weeks gestation
to a female baby weighing 1.7 kg. Both were induced because of
high blood pressure. She had an abortion at 3 months in 1981.
Evacuation was done. The last delivery was by emergency LUSCS
at the KNH as described above. All her pregnancies were thus
complicated by P.I.H.

In the antecedent pregnancy, she attended antenatal clinic at
KNH. The first visit was at 11 weeks gestation. The initial
blood pressure (BP) was 120/80 mmHg, and at that time there was
no proteinuria. At a gestation of 32 weeks, her BP was 130/90
and proteinuria 2+. Her weight gain was however, not excessive,
and there was no peri-orbital oedema. Proteinuria and the eleva-
ted BP persisted till the 38th week when she was admitted for
delivery. In total she had 15 ANC visits. At delivery, the
weight of the baby was 2870 gram. It was a male. Apgar score
was 9 and 10 at 1 and 5 minutes respectively. Placenta weighed
600 gram and was completed with no infarcts. Post delivery her
BP stabilized at 100/60 mmHg.

FAMILY AND SOCIAL HISTORY:

She was married and working as a nurse at the KNH. She did not
smoke or take alcohol.

Her mother had essential hypertension and was maintained on
medical treatment.

PHYSICAL EXAMINATION:

Her general condition was fair, Not pale and afebrile clinically.
There was no oedema or palpable peripheral lymph nodes. Her
BP was 110/60 mmHg; pulse rate was 84 per minute, respiratory rate
was 20 per minute, with a body temperature of 36.0°c.

The cardio-pulmonary and central nervous systems were grossly
normal.

ABDOMINAL EXAMINATION:

The abdomen was not distended. There was a well healed sub-umbilical mid-line scar. The abdomen was soft. Slight tenderness was elicited at the left iliac fossa, but no masses were palpable. The uterus was well involuted.

SPECULUM EXAMINATION:

The external genitalia was normal, as was the introitus and the vagina. The cervix was short with a small transverse slit at the external os. Clear urine was seen to be leaking continuously through the cervical os into the vagina.

PELVIC DIGITAL EXAMINATION:

No excoriation of the vulva was noted. The cervix was firm and os appeared closed. The uterus was normal in size, anteverted and anteflexed. Pouch of Douglas and both adnexae were free.

DIAGNOSIS:

A diagnosis of uretero-cervical fistula was made and the patient worked-up in the ward.

INVESTIGATIONS AND RESULTS:

1. Haemoglobin : 11.0 g/dl.
2. Haematocrit : 32.2%.
3. Platelet count : 568×10^9/l.
4. Serum Electrolytes
   - Na$^+$: 140 mmol/l.
   - K$^+$: 3.2 mmol/l.
   - BUN : 3.0 mmol/l.
   - Creatinine : 80 mmol/l.
   - Uric acid : 268 mmol/l.
5. Urinalysis   pH : 6, Protein - trace, Leucocytes 3+.
6. M.S.S.U. : Contaminats.
7. Intravenous urography showed left hydro-ureteronephrosis and poor excreation of dye from the left kidney. Normal right kidney and right ureter. The patient was then prepared for ureteric re-implantation.

BOARI FLAP PROCEDURE ON 16.3.1989:

The nature of the operation and its possible expected result was explained to the patient fully and the necessary consent obtained from her.

This most useful operation was chosen for the treatment of this ureteric injury because the gap produced was too long for direct re-implantation, but could be bridged by a tubularised bladder flap fashioned from the fundus of the bladder. This was done under general anaesthesia.

Technique: (i) The lower mid-line incision, excising the old scar, gave good exposure. The gut was packed away and self retaining retractor inserted. (ii) The peritoneum was picked up at the brim of the pelvis where it was free and well clear and the fistula, opened with fine scissors and the ureter ident-ified. It was dilated but not friable. It had marked peri-ureteric reaction. Care was taken to avoid damage to the iliac vessels. There was minimal extravasated urine but no pelvic absc-ess, haematoma or urinoma. With a combination of sharp and blunt dissection the ureter was traced down to the fistula at the isthmus, where it was burried in a mass of fibrous tissue. It was not possible to dissect off peritoneum from the ureter, and the peritoneum was instead dissected off with the ureter. (iii) The ureter was raised from its bed and transected well clear of the fibrous tissue i.e. one to two cm away. A balance was made between loss of viable ureter and a clean non-fibrous divided end. (iv) A fine stay suture was passed through the wall of the ureter which was held away. The distal end was ligated. (v) Preparation of the bladder flap: The bladder was steadied with fine stay sutures. The flap was fashioned from full thickness of the fundus of the bladder so that the base was in line with the ureter, and passed through the left broad ligament.

The principles were:

(a) the length of the flap was not more than twice the width of the base to ensure an adequate blood supply. The base was not too large, and was wider than the distal end.

The length was carefully assessed by estimating the length from
the end of the ureter to the base of the flap - it was about 2cm
longer than this, to ensure anastomosis without tension.
(b) the other ureteric orifice was carefully avoided. The flap
was turned up on its base. The width of the flap was about 3
times the diameter of the tube and therefore of the ureter.

The ureter was spatulated by cutting the lower end on its posterior
aspect for 1.5cm. It was then sutured to the upper edge of the
bladder flap using 4 - 0 delayed absorbable suture over an indwe-
lling catheter. A continuous suture was then used to convert
the flap into a tube. The resultant bladder defect was closed in
one continuous layer avoiding transgression of the mucosa (since
foreign bodies are liable to act as a nidus for calculus formation)

Drainage: The site was drained with a corrugated rubber drain
through a separate stab incision, the abdomen closed in layers,
and the bladder and the ureter drained continuously with one
indwelling plastic catheter for 6 days.

POST - OPERATIVE MANAGEMENT:

Routine post-operative care was instituted, and she did quite
well. She was given two litres of intravenous fluids within the
first 24 hours, 100 mg pethidine 8 hourly for 48 hours, and
prophylactic ampicillin for 6 days. She was mobilised the first
post-operative day (POD) and had no complications. Ureteric
catheter was removed the 6th POD, and the patient was discharged
home on the 8th POD, with instructions to attend the Urology
clinic in two weeks time. All stitches were removed on the 10th
POD.

FOLLOW UP:

Seen in the Urology clinic several times, she had no complications,
and soon resumed her normal daily chores without any more urinary
incontinence.

Repeat I.V.U. done on 12.5.1989, showed marked improvement of the
renal function on the left. Decompression of the left calyces and
pelvis had been achieved. The left ureter was still dilated but
no longer tortuous. The right ureter and the right kidney were
normal.

DISCUSSION:

Uretero-vaginal fistulas are not uncommon after radical pelvic operations, but ureteric fistulas following caesarean sections (C.S) are, however, rare. The purpose herein is to present a patient who developed uretero-uterine fistula following an emegency lower uterine segment caesarean section (L.U.S.C.S).

The incidence of ureteric injuries following C.S. at the KNH is unkown. Eisenkop et al (1) reported an incidence of 0.09% elsewhere. Ureteral injuries rarely are reported to occur at the time of CS. Ojo (2) reported 4 cases of obstetrical ureteric injuries were caused at LUSCS. The 7 ureteric injuries reported by Feeney in 1959 included the combined experience of 70 British Obstetricians. Only one of these was recognized intra-operatively. The largest series (8 ureteric injuries) following CS among 15 cases reported by Oliech (3) in 1985 included cases referred from outside the KNH, some of which were operated upon by non-obstetricians.

The ureteric injury that occurred in our patient was associated with a uterine tear at the lateral angle during a difficult LUSCS in a slightly dextrorotated uterus. Of the 7 ureteric injuries reported by Eisenkop et al, 6 were associated with extension of the uterine incision into the broad ligament or the vagina. In our patient, the extension was also into the broad ligament and the cervix. In addition, she had prolonged labour due to persistent occipito-posterior position. Exposure was probably suboptimal, and when haemostasis was obtained, ureteric injury had occurred. Thus, when obtaining haemostasis in a patient with extension of the uterine incision near the base of the bladder or into the broad ligament, the risk of injury to the ureter must always be borne in mind and the operator should be ureterconscious and not ureter-confident. In such cases he should undertake proper steps to document patency of this tube (3).

Ureteric injury is more frequent in obstetric-gynaecological than in surgical operations. This is not because the obstetric-gynaecologist is less careful than his surgical collegue but because of the valnerability of the ureter in the field of obstetrics operations. The pelvic portion of the ureter is not only embryologically connected with the female generative organs

but it is also involved in diseases affecting these organs (2).
Its course in relation to the uterine artery, the vaginal forn-
ices, the broad ligaments adds to the risk of injury to which
the ureter is exposed at the operation for hysterectomy and
during LUSCS extension tears. The ureter is often cut, tied,
stitched or denuded of its blood supply before the operator is
aware of his mistake if at all he becomes aware of it.

It has been said that the venial sin is injury to the ureter but
the mortal sin is failure of recognition (4). In our case injury
seemed not to have been suspected or discovered at the time of CS.
It only became apparent 10 days later upon development of a
uretero-uterine fistula, and a recognition of a left ureterohydrone-
phorosis, and the patient experienced considerable inconvenience
in the interim.

Recognition of ureteral injury may be delayed in many cases on
account of masking of the usual symptoms and signs of ureteral
injury by those caused by the primary operation. Thus, after
CS, pain and tenderness in the abdomen may be attributed to the
abdominal wound. Oliguria or even anuria may be blamed on poor
fluid intake which is usual in the immediate post-operative days.
Vomiting may be dismissed as being a post-anaesthetic complication.

Our patient had high BP before CS, but became normotensive after
delivery. Although this is the usual natural event in pregnancy
induced hypertension, in this case it supports the belief that
acute ureteric obstruction and hydroureteronephrosis will rarely
result in hypertension. In other cases, unilateral ligation of
the ureter without fistula formation may result in silent auto-
nephrectomy and evade detection, and anuria in the early post-
operative period may indicate bilateral ureteral obstruction or
acute tubular necrosis (2).

Injury recognized at operation should be repaired immediately
after the satisfactory completion of the primary operation
provided the patient's condition is good. Injuries discovered
in the period of convalescence call for detailed urological
investigation and reparative operation at an opportune time.
The opinion of a competent urologist is helpful. The pride of
the gynaecologist must be subdued in the interest of the patient.

Feeney has strongly recommended the summoning of a skilled urologist for consultation, consolation, investigation and treatment. Management of ureteral injury at the time of CS is more complicated and must be individualized. Crush injuries and ligations may be managed conservatively if the tissue is not devitalized and ureteral continuity is clearly established (5). One may therefore catheterize the ureter through the ureteral orifice, exteriorize the catheter through the urethra or abdominal wall, and remove it 10 -14 days later (5). If the ureter is transected or devitalized within 5 cm of the bladder, primary re-implantation is the treatment of choice (1). Injuries further removed from the bladder may be managed by either re-implantation with a psoas muscle hitch or end-to-end anastomosis (1). In a patient like ours, who developed a ureteric fistula there was no correlation of the interval between the time or occurence of the injury and repair of the injury to subsequent prognosis. This was hardly surprising since there was free drainage through the uretero-uterine fistula. A Boari-flap (without anti-reflux) procedure was therefore chosen for her.

Injury to the ureter is a serious post-operative complication. The fact that it can cause death further underlies the seriousness of the complication. In addition distressing symptoms such as pain, vomiting, fever and incontinence of urine may occur in the post-operative period in the unrecognized cases. Even after repair of the damage the patient may be left with a legacy of ureteric stricture with consequent hydroureter, hydronephrosis, pyelonephritis and in a few cases calculus formation (1 - 5).

Finally, although it strikes infrequently despite the enormous workload the obstetricians have to contend with ureteric injury is an ever present menace lurking in the background. Vigilant preventive awareness is our best protection against it, keeping in mind that the uterus at term can be pulled up from the pelvis with unexpected ease, and consequently the surrounding structures are displaced from their usual position.

REFERENCES:

1. Eisenkop S.M., Richman R., Platt L.D., Paul R.A. Urinary track Injury During caesarean section. Obstet. Gynecol. 60: 591, 1982.

2. Ojo O.A. Ureteric injury in Obstetric and Gynaecological operations. West Afr. Med. J. 16: 81, 1967.

3. Oliech J.S. Ureteric injuries at Kenyatta National Hospital. Afr. J. Hosp. Med. 7: 49, 1985.

4. Higgins C.C. Ureteral Injuries During Surgery: A review of 87 cases. J.A.M.A. 199: 82, 1967.

5. Raney A.M. Ureteral trauma: Effects of ureteral ligation with and without deligation - Experimental studies and case reports. J. Urol. 119: 326, 1978.

VESICO- VAGINAL FISTULA (VVF) AND RECTO-VAGINAL FISTULA (RVF) - SUCCESSFUL REPAIR.

Name:	G.M.	Parity:	1+0
Age:	23 years	Last Delivery:	1980
Unit No:	726697	Admission:	6.12.1985
		Discharge:	7. 5.1988

PRESENTING HISTORY:

The patient was referred to us from Garisa General Hospital on
6.12.85 with a history of both urinary and faecal incontinence.
She had initially presented herself to that Hospital with a history
of dribbling of urine and passing faeces par vagina-for the
last 5 years following a four-day obstructed labour at home in
July, 1980. She eventually delivered a macerated still birth.
The patient started leaking urine and faeces within 24 hours of
delivery. While at KNH, an attempt at repairing her VVF was
made twice but failed in all occasions. It was only at the 4th
attempt, that a successful repair was achieved.

OBSTETRIC AND GYNAECOLOGICAL HISTORY:

Para 1+0. The delivery was in 1980 to a still-birth as above.
She had not become pregnant again since.

Her menarche was at 13 years and subsequently had regular 30 days
menstrual cycle with normal 3-4 day flow and no dysmenorrhoea.
She did not use any contraceptive method.

During that fateful pregnancy, she never had any antenatal care.

PAST MEDICAL HISTORY:

Her past medical history was not remarkable.

FAMILY AND SOCIAL HISTORY:

She had no formal education, and was once married to a herdsman
who was also illiterate, but had since been divorced because of
her embarrassing predicament.

She was an orphan - her mother having died at her childBirth; and her father died of unknown cause. She was staying with her brother in Garisa. She did not smoke or take alcohol.

PHYSICAL EXAMINATION:

Her general condition was satisfactory. There was no pallor, jaundice, oedema or dehydration. Clinically, she was afebrile, and had no palpable peripheral lymph nodes. Her breasts were normal.

Her blood pressure was 110/70 mmHg; pulse rate was 80 per minute; respiratory rate was 20 per minute, and the temperature was 36.7° c.

The cardio-pulmonary and central nervous systems were grossly normal.

ABDOMINAL EXAMINATION:

The abdomen was scaphoid. There was no tenderness and no organomegaly. Bowel sounds were present. She had multiple therapeutic marks.

PELVIC EXAMINATION:

There was excoriation of the perineal skin and the vulva. Urine leaked freely par vaginum. On digital examination, there were large defects on both the anterior and posterior vaginal wall. Faeces was present on the examining finger. The cervix was small and os closed. The uterus was normal in size and anteverted. Adnexae and the cul-de-sac were free.

DIAGNOSIS:

A diagnosis of vesical-vaginal fistula (VVF) and recto-vaginal fistula (RVF) was made and the patient prepared for work-up in the ward.

INVESTIGATIONS AND RESULTS:

1. Haemoglobin : 13.1 g/dl.
2. Haematocrit : 39.1%

3. White blood cell count : $5.7 \times 10^9/1$ with normal distribution.

4. Serum Electrolytes Na$^+$: 134 mmol/l.

                   K$^+$: 4.6 mmol/l.

                BUN : 5.0 mmol/l.

5. Urinalysis (catheter specimen): No protein or sugar.
   Culture grew E. coli sensitive to nalidixic acid, polymxin
   B and nitrofurantoin.

6. Intravenous urography (IVU) : Normal

7. Blood Group   : "O" Rhesus positive.

Urinary tract infection was adequately treated with nalidixic
acid and repeat urinalysis was normal. She was also given
antiseptic baths. The perineum was smeared with zinc oxide
cream and vaseline when the former was not available. She
was then prepared for examination under anaesthesia.

EXAMINATION UNDER ANAESTHESIA:

In theatre she was given general anaesthesia and placed in
lithotomy position. Vulvo-vaginal toilet was done with
cetavlon and draped with sterile surgical towels. A Sim's
speculum was gently inserted into the vagina and held by an
assistant. With another Sim's speculum, the vaginal walls were
inspected:

- there was a large rectovaginal fistula in the distal posterior
  vaginal wall, measuring about 5 cm in diameter. There was
  enough healthy tissue around it.
- there was a circumferential vesico-vaginal fistula at the
  bladder neck, measuring about 1.5 cm. There was fibrosis around
  this fistula and the surrounding tissues were adherent to pubic
  bone. Urine flowed freely through the fistula.
- the urethra was blocked about 1 cm from the external meatus.
  this stenosis was overcome gently by graduated dilatation
  using metal bougies, and a catheter inserted. Methylene blue
  dye instillation through the urethral catheter showed
  leakage only through the VVF.

A decision was made to fashion a colostomy first before the
repair of the fistulas was undertaken. Both the fistulas were
to be repaired by the vaginal route. VVF was to be repaired in
the knee-chest position, whereas the recto-vaginal fistula was
to be repaired in the lithotomy position. She was reversed from

general anaesthesia and after full recovery was taken back to the
ward to await colostomy.

Pre-colostomy preparation included a three days' daily rectal
wash-outs, low residue diet and laxatives (dulcolax).

TRANSVERSE LOOP COLOSTOMY:

In theatre, the patient was given GA in supine position. She
was then cleaned and draped. Via a right paramedian incision,
the transverse colon was identified and tagged with a nylon tape.
The omentum was dissected from about 8 cm of the bowel and the
colon exteriorized. The loop was then sutured to peritoneum and
fascia circumferentially with interrupted non-absorbable sutures.
The colostomy was opened immediately after the abdominal incision
was closed, and the mucosal stoma was everted and stitched to the
skin, and the incision dressed.

After reversal of GA and full recovery, the patient was returned
to the ward to await fistula repair.

VVF Repair (4th ATTEMPT)

In theatre, the patient was given GA and placed in knee-chest
position with her feet lightly tied to stirrups. A pillow was
placed below the chest and a small sand-bag below the pubis region.
The table was tilted to a head-down position of about 20 degrees
inclination. VVT was done using cetavlon. The buttocks and thighs
were also cleaned and the patient draped with sterile surgical
towels.

The labia were stitched to the thighs using silk sutures to
retract them. With Sim's speculum in posterior vaginal wall the
fistula was exposed. It was surrounded with moderate fibrosis.
The immediate loose tissue was mobilised, and the bleeding
controlled by using adrenaline 1: 100 000 in saline solution
soaked on swabs. A circular incision was made around the fistula
margin using Bishop's knife and the scar tissue excised completely
off its edges. Healthy tissue was thus exposed. First the
mucosal layer was stitched with catgut, and covered with a
second layer before closure of the vaginal wall. Thus the fistula
repair was completed in 3 layers. Extra-chromic catgut 2-0 was

used for suturing. Nelaton catheter was then inserted. During
the operation, the operative site was kept dry by suction.
Haemostasis was achieved. On final instillation of methylene
blue through the bladder catheter, there was no leakage.
The labial sutures were removed, and the Nelaton catheter
secured to the labia majora. Urine had free flow with no
haematuria.

POST-OPERATIVE CARE:

Routine post-operative management was instituted. She was given
parenteral pethidine 100mg 8 hourly for the first 24 hours.
Strict input and output chart was maintained. 3 litres of I.V.
fluids were given in 24 hours. Regular hourly check on urine
output was done for the first 24 hours then 4 hourly, so that any
interference with free drainage due to blood clot could be
reported immediately before any damage could occur to the suture
line. About 30-60 ml of urine was passed hourly. She was
also encouraged liberal oral fluids when she was fully out of
the effects of anaesthesia.

She was given prophylactic ampicillin immediately post-operation,
and this was maintained for five days. Catheter urine specimen
was taken for culture and sensitivity every third day. All
urine specimens showed no growth. Post-operative haemoglobin
was 12.7 g/dl, with a total white cell count of $4.3 \times 10^9/l$.

POST OPERATION EXAMINATION:

This was done on the 21st day in the ward without anaesthesia.
After VVT and draping, about 10 ml of methylene blue dissolved
in 200 ml of normal saline was introduced into the bladder through
a sterile catheter. With Sim's speculum placed against the
posterior vaginal wall gently, the fistula repair site was found
intact with no leakage of dye. The catheter was removed.

She was then planned for repair of her recto-vaginal fistula (RVF).

RVF REPAIR (FIRST ATTEMPT):

In theatre, under anaesthesia, the patient was placed in litho-
tomy position. VVT was done with cetavlon and aseptic

catheterization performed after draping. Retraction stitches were inserted in the labia to the medial aspect of the thighs respectively. With two Sim's speculums the low lying RVF was exposed. The scar tissue was dissected off the edges of the fistula and the rectum separated from the vaginal wall. The rectal mucosa was closed first followed by a second layer to cover the first layer, and the closure of the vaginal wall completed by 3 - layer closure technique. Extra-chromic 2-0 was used for suturing.

Rectal examination revealed a water-tight repair. Haemostasis was adequately achieved.

Barium sulphate x-ray series were done on 5.4.1988 and confirmed the successful repair of the RVF.

Closure of the colostomy was then planned.

CLOSURE OF THE COLOSTOMY:

The bowel was first prepared as for elective colonic surgery: by rectal washouts, laxatives, oral metronidazole and low residue diet - for three days.

This loop colostomy was closed through the previous "local" incision under GA in theatre. An elliptical incision was made round the gut, and deepened to the peritoneum. The gut was then isolated from the sorrounding tissues until the loop was free and could be further lifted out of the abdomen. The colostomy edges were freshened and closed in two layers using absorbable sutures continuous, reinforced with one layer of non-absorbable interrupted sutures. The loop was then replaced inside the abdominal cavity and the incision closed in the 3 traditional layers. Haemostasis was achieved. Post -operatively she did well, with no complications.

She was discharged home on 7.5.1988 with instructions to abstain from sexual intercourse for three months and was told the importance of antenatal care and elective abdominal delivery in all her subsequent pregnancies. She was to be followed-up in Garisa Provincial Hospital.

DISCUSSION :

In many parts of Africa, especially in the more remote rural areas, vesico-vaginal fistula (VVF) is common, and is the most distressing condition which brings women to the hospital. The purpose herein is to present a young woman who developed both VVF and rectovaginal fistula (RVF) at the same time following an obstructed labour of 4 days duration at the tender age of 15 years. Poor childhood nutrition, frequent infection, rudimentary conventional education, and an early start of chilbearing, often before growth is completed, results in nearly 25% of the child-bearing population being stunted leading to obstructed labour due to cephalopelvic disproportion (1). The young primigravida is most prone to VVF, and the patient under discussion is a living testimony of such unfortunate sequalae of neglected labour. Tahzib (1) observed that the young patients tend to have more severe lesions, more often associated with RVFs, third degree perineal tears, and a greater amount of resulting fibrosis in the vagina.

As was in our patient, the chief cause of these fistulas is obstetric trauma (2). Orwenyo (2) found 92.2% to be labour related. Mati (3) found the same in 87.1%. In more remote semi-arid and rural areas malnutrition is rife and ignorance common. Antenatal care (ANC) is sub-standard or non-existence, delivery by untrained traditional birth attendants the order of the day, and survival the name of the game: unfortunately many unattended mothers may suffer an obstructed labour lasting up to 4 days, culminating in a dead fetus and severe pressure necrosis to the bladder base and the rectum as explicitly occurred in our patient. If labour is prolonged in a primigravida, contractions gradually cease, the fetus dies, macerates and is eventually extruded. This process took 4 days in the patient presented, and when she survived the exhaustion and infection, a fistula formation was inevitable. In general, acquired VVF and RVF of obstetric origin is a direct reflection of prevalent inadequate and poor obstetric services in any community. Although poor communication with scarcity of effective transport have a contributory effect on the prevalence of obstetric fistulas, reliance on traditional practices or delay in seeking hospital care appear to be as important as lack of good quality medical practices.

Our patient had mid-vaginal fistula. As to the anatomical site of the fistula, much depends on the site of compression by the presenting part, as well as the degree of cervical dilatation (4). The bladder-neck and the urethra are the commonest sites for VVF. And as a general rule, fistulas resulting from obstetrics trauma are low-lying, while those resulting from surgery and radiotherapy are generally high or juxta-cervical (4). As was seen in our patient, fistula formation can manifest itself within 24 hours of delivery following prolonged labour contrary to popular belief.

The diagnosis of VVF and RVF in this patient was made by means of direct observation, palpation and from the history. As Robertson pointed out, urinary incontinence in women is one of the most frightful afflictions of the human kind, and clear history was readily obtained. Hour by hour, and night and day, a leakage wet, excoriate, and hurt the victim of this misfortune. Clothes were ruined, the bed became a nightmare, sexual intercourse was stopped, a pariah made, and family nearly housed an outcast!

A helpful aid for VVF was the instillation of methylene blue into the bladder during pre-operative examination under anaesthesia (EVA). I.V.U. was essential for the determination of the state of the urinary tract.

Examination under anaesthesia was performed to:
- assess the number, size, site, fixity and the amount of loose tissue or fibrosis associated with fistulas; and to
- determine the best route and position for repair.

VVF and RVF create enormous problems to the patient who is often ostracised from the community to which she rightly belongs and frequently divorced as was evidenced by our patient, and to the surgeon who must find a way of curing these pitiable, miserable patients from their calamity. Although her RVF was successfully repaired at the first attempt, her VVF was only successfully repaired at the fourth attempt. This is hardly surprising as it is well known to every practicing gynaecologist that in repair of VVF closure is not always possible and does not always result in restoration of function (5). Persistence is certainly required. Post-operative urinary tract infection, catheter

blockage and wound infection continue to be the nightmare of a fistula surgeon, yet these are avoidable factors (6).

To ensure successful repair of this patient's fistulas, the following were observed:
- the patient's general condition was improved and the urinary tract infection was eliminated beforehand;
- a temporary transverse colostomy was fashioned in the first instance to reduce infection of the bladder;
- adequate mobilisation of the bladder from the vagina and the rectum from the same, was essential, and broad raw edges were opposed without tension. Only interrupted sutures were used in a layered closure technique.
- excision of scar tissue at the fistula site to leave healthy tissue with adequate blood supply to the repair site;
- gentle manipulation, uncompromised exposure and adequate haemostasis;
- free urinary drainage after repair was of a paramount importance. This was provided for by only one outlet;
- post-operative infection was prophylactically avoided.
  There was no straining and good general nursing care was provided.

In this patient, we used the vaginal closure. This is the classic route used by Sim's in his original work on the Southern States (USA) slaves in 1849. It is the obvious method of choice for low-lying, post obstetric fistulas. The vaginal route offers the following advantages: (i) it is more comfortable for the patient; (ii) bladder sutures may be placed so as not to enter the bladder lumen; (iii) there is less tissue devitalization, and (iv) the vaginal sutures (acting as bladder support) are more accurately inserted (4).

Successful fistula repair requires the separate, tension-less closure of the debrided and healthy tissue of each organ communicating on the fistula. In healthy tissue, this is usually achieved, and success by simple repair was obtained.

In conclusion, a number of biological, social and environmental factors contribute to the high prevalence of obstetric fistulas in developing countries. The hallmark of these lesions is that they are almost all preventable. Prevention is the only satisfactory solution to this traumatic problem. The maternity services should be available, trusted, and used increasingly by

every community. Health education and good obstetric care should
be able *to eradicate VVFs and RVFs.*

REFERENCES:

1. Tahzib F. Epidemiological determinants of vesico-vaginal fistula. Br. J. Obstet. Gynecol. 90: 387, 1983.

2. Orwenyo E.A. Review of vesico-vaginal and recto-vaginal fistulas at Kenyatta National Hospital (1979-1982). M. Med. Thesis. University of Nairobi. 1984.

3. Gunarathe M. and Mati J.K.G. Acquired fistulae of the female lower genital tract: A comprehensive five year review. J. Obst. Gyn. East. Cent. Afr. 1: 11, 1982.

4. Edwards J.N.T. Principles of management of the vesico-vaginal fistulae. S. Afr. Med. J. 62: 989, 1982.

5. John S.G. Factors in the prediction of successful vaginal repair of vesico-vaginal fistulae. J. Obstet. Gynecol. Brit. Cwlth. 76: 741, 1969.

6. Nnatu S. Profile of Obstetric Fistula in a Sub-saharan Centre. J. Obst. Gyn. East. Cent. Afr. 5: 13, 1986.

HIRSUTISM ASSOCIATED WITH AN ADRENAL TUMOUR - UNILATERAL ADRENALECTOMY.

Name:	M.M	Parity:	0+1
Age:	26 years	LMP:	March 1986
Unit No:	956931	DOD:	2.6.1989
		DOD:	24.7.1989

PRESENTING HISTORY:

Mrs. M.M. was referred to us from Aga Khan Hospital with an intra-abdominal mass, hirsutism, secondary amenorrhoea and secondary infertility. The abdominal mass appeared in 1986 and had been progressively enlarging. It was painful on and off. At the time of admission, this mass was extremely painful. She also noticed abnormal hair growth on her chin and the anterior abdomen since 1986. She had no history of change in her voice, enlargement of her clitoris, altered facial appearance, shrinking of her breasts and odorous perspiration. She stopped menstruating the Same year. There was no change in her libido.

OBSTETRICS AND GYNAECOLOGICAL HISTORY

She had her menarche at 15 years of age, and subsequently had regular periods lasting 7 days every 30 days upto 1986 when these periods completely ceased. She therefore had normal menses for a period of 11 years. She did not use any contraception. Para 0+1. The abortion occured in 1982 at 3 months gestation. Dilatation and evacuation was done at a private nursing home.

PAST MEDICAL HISTORY:

Laparoscopy was done on her in 1986 as investigation of her infertility. She had no other previous hospital admissions. No previous illness.

FAMILY AND SOCIAL HISTORY:

She was married since 1983, and was staying with her husband at Kasarani. She was a subordinate staff with the City Education Department (Nairobi). She did not smoke or take alcohol.

PHYSICAL EXAMINATION:

She was sick looking. She had no pallor, cyanosis, oedema or
dehydration. Peripheral lymph nodes were not palpable. Her blood
pressure was 120/80 mmHg; pulse 80 per minute, respiratory rate
was 20 per minute. The temperature was 38°c.

She was a thin looking lady with excessive hair growth (hirsutism)
on her chin (beard) and her anterior abdomen, with a male eschutch-
eon. There was no recession of her scalp hair, and no musculine
features. Her contours were feminine. She had no striae but had
mild acne. Her face, neck and back were not flushed.

BREASTS:

Both breasts were normal looking and symmetrical. They were
Tanner's class V. There were no atrophic changes.

THROAT:

Her larynx was not enlarged.

ABDOMINAL EXAMINATION:

There was a large mass in the left hypochondrium extending to the
left loin. It was about 10 cm in diameter. This mass was firm
and tender. It did not move with respiration. Below this mass,
and attached to this one, was another smaller mass lateral and
adjacent to the umbilicus. This other mass was non-tender and
was kidney-shaped. Both the liver and the spleen were moderately
enlarged.

PELVIC EXAMINATION:

There was excessive hair at the mons pubis, and the labia majora
were more prominent. The introitus was normal. The clitoris
was not enlarged - it was less than 2.5 cm in length. Digitally,
the uterus was normal in size and shape. It was mobile and
anteverted. Both the adnexae and the cul-de-sac were free and
non-tender. Both ovaries were not enlarged.

DIAGNOSIS:

A diagnosis of hirsutism and secondary amenorrhoea probably due

to an adrenal tumour was made, and the patient was worked up
for exploratory laparotomy.

INVESTIGATIONS AND RESULTS:

1. Haemoglobin : 13.9 g/dl.
2. Haematocrit : 40.9%.
3. Serum testosterone : 3.01 mmol/l. (Ref. range 0.35-3.5).
4. Serum chemistry Na$^+$: 139 mmol/l.
                     K$^+$: 4.1 mmol/l.
                     BUN : 3.8 mmol/l.
              Creatinine : 86 mmol/l.
5. Liver Function Tests
          Total protein : 59 g/l.
          Albumen : 38 g/l.
          Bilirubin : 2 mmol/l.
   Alkaline phosphatade : 5.2 KA units.
6. CT scan of abdomen showed a large mixed density mass lesion
   in the left middle abdomen showing streaks of faint calcifica-
   tion. It did not enhance with intravenous contrast medium.
7. Chest X-ray: normal.

Av. Ref. range.

8. FSH : 0.8 mU/ml
   LH : 7.2 mU/ml

	Foll.phase	Ovul.phase	Lut.phase
FSH	2.0 - 8.0	15 - 30	2.0 - 8.0
LH	2.0 - 15	20 - 100	5.0 - 20

9. Abdominal ultrasound showed a large mass displacing the
   spleen medially and the left kidney downwards. The mass
   showed irregular echopattern. The left kidney was normal.
   No pelvic mass was noted.
10. Intravenous urography: The left kidney was displaced to the
    level of L4 vertebra by the mass. There was bilateral
    excretion of the dye. The patient was then routinely
    prepared for exploratory laparotomy.

UNILATERAL ADRENALECTOMY (17/7/1989):

The patient was placed supine on the operation table, anaesthetise-
d, cleaned and draped with sterile surgical towels. The abdomen
was opened via a left roof-top sub-costal incision (anterior

approach) with its apex about two fingers below the tip of the
xiphoid process.   The rectus muscle, transversus muscle and peri-
toneum were incised through a liberal incision.   Additional
exposure was obtained by dividing the internal oblique muscle
in the direction of its fibres out into the flanks, and the
following were   found:

- a large supra-renal mass in the left retroperitoneal area with
extensive venous congestion medially, but only two large arteries
from the abdominal aorta, and a few twigs from the inferior phrenic
artery.   The veins were draining into the left renal vein.

- left kidney was displaced downwards and rotated laterally;
there were no enlarged intra-abdominal lymph nodes; there was no
tumour invasion into the veins; and no metastatic deposits.   Both
the liver and the spleen were enlarged.

- the contralateral right adrenal gland was normal.

TECHNIQUE:

The tumour was carefully mobilized by blunt dissection laterally
with the surgeon's index finger, after carefully grasping the
spleen and dividing the avascular splenorenal ligament so that
the spleen was mobilized.   Dissection was carried medially as
far as the superior mesenteric vein, which gave more mobilization.
It was then easy to incise the distorted Gerota's fascia and the
slightly adherent peritoneum over the tumour.   The prominent
congested adrenal veins were secured one by one, tied and cut,
as the surgeon worked his way about the periphery of the tumour,
ligating all the prominent vessels; and the tumour was finally
extirpaterd in-toto.   Both the left kidney and the spleen were
then returned to the now empty space, and a corrugated rubber
drain left in-situ and the incision closed in four layers.
Haemostasis was adequately achieved.

Estimated blood loss was about 500 ml.   She was however, trans-
fused two units of blood intra-operatively.

GROSS DESCRIPTION OF THE TUMOUR:

The tumour which was solid and roughly spherical in shape
measured approximately 15 x 15 x 10 cm^3. The cut surface had a
yellow colour and showed necrosis and several haemorrhagic
foci in the various septa within it. There was no venous
invasion. The tumour was well encapsulated and weighed about
1.2 kg.

POST OPERATIVE CARE:

Routine post-operative management was instituted. She was given
pethidine 100 mg 8 hourly for 48 hours, and I.V. fluids for the
same period. She did extremely well and was ambulatory on the
second post-operative day, when she was allowed oral fluids.
She had no steroid substitution therapy. The drain was removed
on the 4th post-operative day (P.O.D), and the stitch pulled
out on the 7th P.O.D. - the same day she was discharged home in
good general condition. She was to be followed - up in the
surgical outpatient clinic life-long.

Her post-operation haemoglobin was 11.3 g/dl, with a haematocrit
of 32%.

HISTOLOGY REPORT (Path. No. 2452/89):

There was much necrosis and haemorrhage, but also vital large cell
nests composed of rather uniform large cells with big round
nuclei and much eosinophilic cytoplasm. These features were in
keeping with an adrenal adenoma.

DISCUSSION:

Hirsutism, although is an uncommon presenting complaint of women can occasionally be the first sign of a tumour. The purpose herein is to present a young female patient who came to us because of moderate hirsutism and secondary amenorrhoea associated with a large adrenal adenoma without other features of virilization. This statement was based on the findings of normal clitoris, no change in her voice or fat contours and no male physique. This patient was considered to have a functional tumour because she manifested a clinical syndrome of excess steroid production. Her hirsutism was cosmetically disturbing.

Hirsutism is the growth of terminal, coarse hair on the midline area of the lip, chin, chest, abdomen and back regions of the body (1-3), as was in our patient. Associated features of androgen excess, in order of the degree of androgen overproduction required to induce them are oiliness of the skin, acne, menstrual abnormalities, defiminization and increased libido. The greatest degree of androgen overproduction results in masculinization of women: male hair distribution, temporal balding, clitorimegaly, laryngeal growth and deepening of the voice, increased muscle mass and body habitus (1).

The testosterone in our patient was most likely formed by peripheral aromatization of pre-androgens such as androstenedione, dehydroepiandrosterone (DHEA-S), and its level was at the upper level of the reference range. We do not think that the tumour was producing testoterone directly. The serum level of L.H. was also not elevated. This shows that this tumour was not gonadotrophin responsive.

The adrenal cortex and its tumours are not very efficient at testosterone synthesis (3) and in this patient the plasma testosterone was derived from large amounts of precursors secreted by the adrenal and then converted to testosterone and 17 - ketosteroids. By contrast virilizing tumours of the ovary such as arrhenoblastomas or interstitial cell tumours may synthesize testosterone efficiently, or they can behave as adrenal tumours (3-5). Thus, given a woman with severe virilization, normal urinary 17 - ketosteroids strongly suggest an ovarian tumour, but

except for a few cases, virilizing adrenal adenoma is associated
with markedly elevated 17 - ketosteroids in urine (6), and
presumably elevated peripheral DHEA and DHES-S. In cases where
differential diagnosis between ovarian and adrenal tumours is
difficult to make, adrenal and ovarian venous catheterization via
the femoral vein may be of assistance in localizing the source of
excess androgens (6). However, it is still a specialized
technique not routinely performed in our unit.

There have now been extensive studies of plasma testosterone conce-
ntration in hirsutism and virilization. Virilization has always
been associated with increased plasma testosterone levels.
Since plasma testosterone concentration may be normal or increased
in hirsutism, this measurement may not help in defining the prob-
lem (6). The skin of hirsute women is also highly sensitive to
available testosterone owing to increased activity of 5a-reductase
in these women as compared with non-hirsute women (2).

Let us now return to our patient. The hirsutism accompanied by
secondary amenorrhoea suggested a marked androgenic effect, and
we were rightly amazed at a plasma testosterone concentration
in the normal female reference range. The alteration in the
life cycle of the androgen-responsive hair follicles were either
in ordinately sensitive to normal amounts of androgens or that
they produced androgens intracellularly at an increased rate
(increased skin metabolism of weak androgens to potent effectors).

The effect of excess androgen production on the hypothalamic-
pituitary - ovarian system depends on the sensitivity of that
system to testosterone (2). Thus, some women may be hirsute, but
have regular menstrual cycles. In contrast, our patient had
amenorrhoea. In this patient it was possible that testosterone
was aromatized to estradiol in the hypothalamus - this in-turn
lead to disturbed gonadotrophin secretion, which subsequently
interferred with follicular growth and maturation. Furthermore
testosterone excess inhibits gonadotrophin receptor formation
in the ovary (2). The end result was anovulation manifested by
the secondary amenorrhoea.

The treatment modality employed in our case was surgical extir-
pation of the adrenal adenoma. This tumour was found to be very
large at operation with a diameter of approximately 10 cm. Size
of the tumour has inverse relationship with survival. Those with

tumours of 10 cm in diameter have longer survival than those with tumours less than 10 cm in diameter (7). This paradox may be related to biologically aggressive tumours turning malignant at an early stage while in other, biologically less aggressive tumours attaining large size without becoming malignant. Atrophy of the remaining right adrenal cortex was not suspected. She therefore did not have post-operative adrenal cortical insufficiency as was shown by her speedy recovery. Pre-operative and post-operative substitution steroid therapy was thus not necessary.

The long term results following removal of benign tumours are good, with practically uniform reversion to normal physical status in those cases reported (4). Our patient had had hirsutism for only four years. When hirsutism has been present for a long time, the pattern of hair growth changes slowly following therapy (3). Yet severe hirsutism and masculinization due to an adrenal tumour have been reversed completely by removal of the tumour (1-7). Naturally, our patient had therefore a favourable prognosis with a possibility of a future pregnancy.

In conclusion, the importance of identifying the source of androgen excess in any hirsute patient cannot be overstressed. We recommend that any hirsute woman have an abdominal CT scan performed to search for an adrenal mass as well as an ovarian tumour, before medical treatments with agents such as cyproterone acetate, spironolactone, cimetidine etc can be started empirically.

REFERENCES:

1. Strickler R.C. and Warren J.C. Hirsutism: Diagnosis and Management. Year Book of Obstetrics and Gynaecology. Pitkin M.R. (editor). Year Book Med. Pub. 1979.

2. Badawy S.Z.A. Diagnosis and Management of Hirsute women. Int. J. Fertil. 32: 349, 1987.

3. Lipsett M., Wessler S.W., Avioli L.V. The Differential Diagnosis of Hirsutism and Virilism. Arch. Intern. Med. 132: 616, 1973.

4. Heinbecker P., O'Neal L.W., Ackerman L.V. Functioning and Non-functioning adrenal cortical tumours. Surg. Gynecol. Obstet. 105: 21, 1957.

5. Dolinar R. and Burch W.M. Testosterone-Producing Adrenal Adenoma in a woman with Normal Urinary 17-Ketosteroids Levels. J.A.M.A. 250: 2504, 1983.

6. Chakmakjian Z.H. and Abraham G.E. Peripheral steroid Levels in a patient with Virilizing Adrenal Adenoma. Obstet. Gynecol. 46: 544, 1975.

7. Didolkar M.S., Bescher R.A., Elias E.G., Moore R.H. Natural history of Adrenal Cortical Carcinoma: A clinicopathologic study of 42 patients. Cancer 47: 2153, 1981.